This PDF version is for demonstration only, the text is optimized for reading on a handheld device, the formatting and diagrams and compressed nature of the editing are not standard for this format but is still useful for demonstration purposes. Note: Both formats of the text (.pbd = iSilo and .prc = Mobipocket) can also be read on your desktop computer.

Clinical Medicine Consult - DEMO

Chapters: Allergy | **Cardiology** | Compendium | **Derm** | Endocrine | **ENT** | Geriatrics | **Gastro** | Hem-Onc | **ICU-FEN** | Infectious | **Nephrology** | Neurology | **Ophthalmo** | Ortho | **Pain** | Pediatrics | **Psych** | Pulm | **Rheum** | Trauma & Tox | **Urology** | Womens Health | **Procedures** | Preamble-Abbrev | ***INDEX*** | Studies & On-Call & Sx's | **MEDS** | Surgical | Last updated on **23 Feb 2010**

This demo contains links to all the major sections (there is no separate demo for individual chapters) along with the first few links in each chapter. Included is just the initial 2% of the information in each chapter to give you a feel for the information it contains. Most of the other links are "dead" as the information is not included in the demo.

Available Titles:

The Clinical Medicine Consult (NOTE: includes all 35 of these modules (including this text) as all are pulled from this one highly integrated text, "The Clinical Medicine Consult" for $59 via http://clinicalmedconsult.com - includes 1 year of FREE updates). Clinical Endocrinology, Neurology, Orthopedics, Nephrology, Dermatology, Gastroenterology, ENT, Psychiatry, Urology, Infectious Disease, Women's Health, Hematology / Oncology, Geriatrics-Death & Dying, Rheumatology, Pulmonology, Allergy, Pediatrics, Pain Management, Ethics-Alternative Medicine-Evidence Based Medicine-Communication (Compendium), Cardiology, Ophthalmology, Travel Medicine, Procedures in Medicine, Wound Care & Skin Infections, Sports Medicine, Wilderness, Survival, Alternative and Complementary Medicine, Cancer Medicine, Hospice & Palliative Care. The goal is for these texts is to have >95% of clinical of the information you need at your fingertips. **Disclaimer |**

• **Note:** Information is added as important new information is published. We perform an ongoing review of >520 journals, textbooks, online sources, and many other expert resources. The file size of **The Clinical Medicine Consult** 2010 v3 was 26,946 kb, the last quarterly versions were sized at 24,114 kb, 23,164, 21,901, 17,451, 16,918, 16,516, 15,858, 15,176, 14,580, 13,980, 12,950, 12,276, 11,715, 11,097, 10,752, 10,249, 9,769, 9,298, 8,977, 8,004, 6,805 and 5,364 kb....tons more information for you to use at your fingertips and even more in the very near future.

Ethics, Communication, Evidence Based and Alternative Medicine:

Edited by Jeffrey F. Hankoff MD & A.A. Weber MD, Family Physicians.

Contents: Ethics | **Malpractice** | The Art of Medicine | **Doctor - Pt Relationships** | Placebo | **Pearls** | Preventive Med | **Genetics** | Evidence-Based Medicine | **Stats** | Pharmacotherapy | **Orders & Notes & Coding** | Complementary & Alternative Medicine | **Nutriceuticals (Herbal)** | Dietary Supplements | **Common Conversions** | Disability | **Exercise** | *Herbs* | **Spirituality** | Quackery | **Stress Reduction** | Healthy Living | **Wilderness Medicine** | Survivalist Medicine | **Disaster Medicine** | Environmental and Occupational Med | **Epidemiology** | Travel Medicine |

Principles of Ethics:

Links: Basic Definitions | Professionalism and Oaths | **Confidentiality** | Competency | **Futile Care** | Emergency Procedures | **Refusal of Care / Against Medical Advice** | Impaired Physicians | **Advanced Care Planning** | Informed Consent | See Geriatrics for **Withholding / Withdrawing Care |**

Ethics is the disciplined study of morality. Bioethics concerns morality in the physician-pt relationship. In medicine, our pt's must be assured of confidentiality and that we will tell the truth at all times. Ethics inquires into ethically justified behavior via two key questions: what ought we do and what ought we not do.

Four Fundamental Virtues:

Principlism is a well-established ethical framework for medical decisions in the United States and Western Europe, and it highlights cross-cultural differences that occur along four dimensions: autonomy, beneficence, nonmaleficence and justice

1. Beneficence: balancing risks and benefits of a given action. The duty of the physician to promote good / welfare for the pt. Beneficence emphasizes the benefit of the pt, not the avoidance of harm, as the primary consideration. First, the physician identifies the clinical goods to be sought and the clinical harms to be avoided by use of professional competencies. Second, the physician reaches rigorous judgments about which courses of intervention or nonintervention are reliably expected to produce a greater balance of goods over harms and thus serve the interests of the pt to some reasonable degree. A fiduciary relationship is the basis of The Hippocratic oath, where the physician has a duty to act in the pt's best interest and to refrain from exploiting the pt. Respecting the fiduciary relationship and the trust of the pt is a cornerstone of the ethical physician's practice.

Medical Indication: What is the best tx, what are the alternatives for the pt. We must assist others in furthering their legitimate interest. If they refuse an Rx for a life-threatening situation, especially if low risk, then we must probe why the pt is refusing and if necessary seek legal authorization to treat them against their will. Parenteral religious preferences do not extend to a young child with a life-threatening condition. Many Asian and Native American cultures value beneficence by encouraging pt hope, even in the face of terminal illness.

2. Nonmaleficence: the duty not to inflict harm or injury with a tx. Cultures valuing nonmaleficence, go so far as to protect pt's from the emotional and physical harm caused by directly addressing death and end-of-life care. Primum non nocere, or first do no harm (Hippocrates. Kos. 4th century bc).

3. Autonomy: respecting the decision-making capacity (self-determination) of individual pt's through informed consent. Most pt's in Western counties value autonomy, other cultures have more emphasis on beneficence. Autonomy and self-determination indicate the ability of a person to make a free and informed decision without pressure or coercion. Western societies value autonomy and the belief that competent adults have the right to make virtually any decision pertaining to their health care via facilitated shared decision making.

Pt preferences: what does the pt want? The physician first provides an adequate amount of information to the pt or the surrogate about the pt's condition; about all medically reasonable options for its management, including watchful waiting; and about the

benefits and risks of each management strategy. Second, the physician elicits from the pt or the surrogate their cognitive understanding of the consequences of various management strategies. A pt cannot be forced to receive care contrary to religious preferences (blood transfusion). We must avoid paternalism, which is the practice of over-riding or ignoring a person's preferences in order to "benefit" them. Conflicts can occur between beneficence and autonomy. Pt's' refusals of care may thwart their own goals or cause them serious harm. For example, a young man with an easily treated testicular cancer may refuse tx. Simply to accept such refusals, in the name of respecting autonomy, seems morally constricted. Physicians can elicit pt's' expectations and concerns, correct misunderstandings, and try to persuade them to accept beneficial therapies. If disagreements persist after discussions, the pt's informed choices and view of his or her best interests should prevail. While refusing recommended care does not render a pt incompetent, it may lead the physician to probe further to ensure that the pt is able to make informed decisions.

4. Justice: distributing risks and benefits fairly. The pt should be given what is his/her due. Upholding what is right, with a fair distribution of benefits. Justice in the healthcare provider-pt relationship involves veracity, confidentiality, and is based on allowing pt autonomy and mandatory reporting. Not allowing autonomy is an injustice (upholding what is right), whereas mandatory reporting forms the database for the decisions with regard to the appropriate distribution of benefits or resource allocation.

Social justice: physicians should work actively to eliminate discrimination in health care based on race, gender, socioeconomic status, ethnicity, religion or any other social category.

Substantive justice: concerns what the outcome of the distribution process ought to be, i.e., who ought to get what is due to him or her.

Procedural justice: concerns the distribution process itself, i.e., how the decision-making process about scarce resources ought to be conducted. Just distribution of finite resources.

Contextual issues: what does the pt want, what are the needs of society. Considerable controversy exists over whether physicians should do everything that might benefit each pt without regard to costs or other societal factors, or whether physicians must be forced to consider the bottom line. Traditionalists tend to ignore the fact that financial considerations have always limited the quality of care available to the poor. The question we are now confronting is whether these considerations may legitimately limit the quality of care available to everyone. In caring for individual pt's, physicians should distinguish between providing what the pt wants and what the pt needs.

Quality of life: what impact will the proposed tx or lack of tx have on the pt's life.

Compassion: a is willingness to acknowledge and respond to relieve the suffering and distress of others. It has an intellectual component to know when a pt is in pain, suffering, or distressed. It has a motivating and behavioral component that moves the physician to relieve the pt.

Character: an important consideration for the morality of physicians and health care professionals. Character is addressed in terms of the virtues and vices. Virtues are traits of character that blunt mere self-interest so that one can identify and routinely protect and promote the interests of others. Vices are traits of character that unleash mere self-interest, so that one ignores or even fails to identify the interests of others, much less serve their interests. An ethically justified character is disposed to be concerned primarily with the interests of others and to act primarily for the sake of their interests rather than always our own.

Paternalism: limiting the freedom of another person (usually against their will) with the justification that such interference prevents harm. Paternalism carries with it the danger of intrusive beneficence, ie, my values are better than yours, although paternalisms may be weak or strong and are often valued differently depending on the culture.

Wisdom: being able to see things in a greater variety of ways, resulting in a broader and deeper perspective.

Moral Pluralism: In a pluralistic society such as the USA, there are many sources of morality, including law, historical experience and traditions, religious beliefs, professional education and training, personal experience, professional consensus, authorities and experts, and institutional policies and practices. Sometimes there is disagreement among the sources of morality. This included subject such as abortion and physician-assisted suicide. There can be competing well-made ethical analyses, arguments, and judgments for many of these subjects. This underscores the fact that the results of bioethics are rarely final or certain and therefore are rarely immune to criticism. Nonetheless, well-made rigorous clinical reasoning and judgments are reliable in the sense that physicians, pt's, institutions, and society may act on them with confidence.

Rule of Double Effect: outcomes that would be morally wrong if intentionally provoked are permissible if foreseen but unintended. Specifically, administering opioids to treat a terminally ill pt's dyspnea or pain may be acceptable even if the medication contributes to or causes the pt's death. A harmful effect of tx, even death, is permissible if death is not intended but occurs only as a side effect of a beneficial action. It is not uncommon for dying or terminally ill pt's in the CCU to receive morphine as part of comfort care. It is usually given in small doses (an initial drip of 1 to 2 mg per hour or repeated boluses ranging from 10 to 30 mg/ hour) to alleviate air hunger or dyspnea, supplemented by benzodiazepines to treat anxiety or agitation. The underlying logic is that it is impossible for a person to avoid all harmful actions. Failure to intervene to comfort a dying pt harms the pt by allowing treatable discomfort to continue. If the intent of tx is to relieve suffering, the foreseen but unintended risk of earlier death is permissible.

Professionalism:

The duty to place health care above all other goods. We are guided by pt welfare, pt autonomy and social justice. Health care providers are engaged in the moral enterprise to deliver care in a personal, dignified and compassionate way. We have a personal, nontransferable responsibility to be our pt's advocates this transcends insurance companies, government programs and other markets.

Our responsibilities include: professional competence, honesty with pt's, pt confidentiality, maintaining appropriate relationships with pt's, improving both quality & access to care, just distribution of finite resources, scientific knowledge, and maintaining trust by managing conflicts of interest. The pt's health is always the first concern. The doctor's job is to prevent, cure and treat disease, to alleviate pain and suffering and disability. The doctor should strive to minimize the effect of his personal feelings, inconvenience and discomfort, financial considerations, influence of others such as third party payers and entrepreneurs who would deflect the doctor from placing the pt's welfare above all other considerations. All comers no matter what their background, are viewed as equals, when it comes to a doctor-pt relationship. The physician should not violate the pt's trust. Honesty and forthrightness are expected in all dealings. Any doctor who does not have a special reverence for life should quit practice. Twentieth century history demonstrates that doctors are easily recruited as agents of state sponsored torture, murder and "experimentation." A physician who participates in such acts of his own free will, in direct violation of his duty to preserve life and decrease suffering, deserves to be treated as a criminal. All medical interventions should be undertaken with express informed consent. The doctor has a duty to stay abreast in his

field and continue his medical education. The doctor is expected to perform according to the standards of his specialty, and make reasonable judgments based on the information at hand. All fields of human endeavor are fraught with inevitable human error. The moral imperative is to practice in good faith. A doctor who encourages < scientific patent or mountebank type alternative practices, particularly for their own gain, is working below ethical standards of the modern physician.
Medical professionalism includes the obligation of physicians to live not only an ethical life, but also a healthy one. Physicians should always "practice what they preach" because a physician's personal life as role models is more accessible and relevant to pt's. Professional role models are important, a person would be unlikely to seek advice from a financial advisor who has filed for bankruptcy. Many pt's do not care if the surgeon has a good bedside manner, they just want to know that the surgeon is able to perform the surgery effectively. On the other hand, if the physician is intervening behaviorally, the physician's personal values, attributes, and behaviors are as influential as his or her psychological and educational expertise. However physicians are as imperfect as pt's; therefore, expecting a physician to always follow 100% of the recommendations he or she gives to pt's, while intuitively reasonable, is probably not appropriate (Physicians as Role Models. Commentary by Thomas Schewnk, M.D. April 1, 2007, issue of AFP).
…….END DEMO of this chapter.

Allergy and Immunology:

Edited by Vincent C. Tubliolo MD, Allergist / Internist. And Chris Landon MD, Allergist / Pediatrics.

Contents: General Immunology | **Immune Deficiency** | Transplantation Medicine | **Vaccines / Immunizations** | Immune Complex Dz | ****Common Allergic Disorders**** | Drug Reactions | **Skin Reactions** | Anaphylaxis | **Urticaria** | Asthma | **Allergic Rhinitis** | Foods | **Insect** | Latex | **MEDS** | Angioedema | ***INDEX*** | See Rheum for **Autoantibodies** |

Immunology and the Basics of the Immune System:

Links: Intro | **Autoimmunity** | Immune Cells | **Complement** | Cytokines | **Immunoglobulins** | Gell & Coombs | **Immune Deficiency** | Transplantation Immunology | **Diagnostic Immunology** | Immune Complex Dz | **HLA Typing** |

The immune system is adaptive, specific and communicative in that it recognizes and responds to changes in the environment, and it displays memory by adapting or altering its response to stimuli that it has encountered previously. The complexity of the immune system is extended by the inherint genetic differences among individuals. Adults who slept <7 hours per night were almost three times more likely than longer sleepers to develop a cold after rhinovirus exposure (Arch Intern Med 2009;169:62).....Sleep deprivation can adversely affect immune function.
There are 2 distinct immune systems—innate and adaptive—that act in concert as well as separately in the development of immunity. Both systems have mechanisms for distinguishing self from nonself, therefore, under normal situations they are not directed against the host's tissues and cells.
The innate system provides a first line of defense against a foreign substance. It is nonspecific, rapid, lacks immunologic memory, and is usually of short duration. Neutrophils, monocytes, macrophages, eosinophils, basophils, mast cells, natural killer (NK) cells, complement proteins, and acute-phase reactants. All of these elements have germline-encoded receptors that recognize motifs commonly present on microbes.
The adaptive immune system includes elements such as the B lymphocytes that make antibodies and T lymphocytes that provide the effector elements of antigen-specific cell-mediated immune responses. The adaptive system has exquisite specificity, is slower in development, exhibits immunological memory, and is long lasting. Innate and adaptive immune systems are distinct systems but interact at several levels to develop a complete defense against invading pathogens. Elements of the adaptive immune system display a large repertoire (e.g., tens of millions) of specific antigen receptors that are generated by DNA rearrangements. Each lymphocyte and its clonal descendants express one of the millions of possible antigen receptors. Since numerically there are very few cells at any one time that can recognize newly introduced antigens, B cells and T cells must be appropriately stimulated and induced to divide and produce multiple copies of themselves.
The distinct advantage of the adaptive immune system is its ability to select B cells and T cells that have high-affinity receptors for new antigens and to stimulate them to replicate and provide a specific, fine-tuned response to foreign invaders.
The disadvantage of the adaptive immunes response is that the required expansion process takes time after the first encounter with antigen, in some cases more than 2 weeks. Many infectious agents can cause death or severe disability in less time than it takes the adaptive immune system to mobilize a specific response. This disadvantage leaves a gap in the host defense system.

Autoimmunity:

Link: Risks & Types | General Immunology | **Autoantibodies** |

Autoimmunity is defined as the immune response to antigens of the host itself. This autoimmune response can be demonstrated by the presence of circulating autoantibodies or T lymphocytes reactive with host antigens. A great deal of basic research has been dedicated to unraveling the mechanisms responsible for the body's ability to distinguish its own molecules from foreign molecules. Nevertheless, exceptions to the rules of governing self-/non-self-discrimination are well known. Most autoimmune responses do not result in disease, but when a harmful response occurs, the pathological consequence of the autoimmune response is called autoimmune disease. These disorders can affect virtually any site in the body so that their clinical presentation varies widely. In at least 80 diseases, autoimmunity is now recognized as an important cause or contributor. The immunologic diagnosis of autoimmune disease relies mainly on the demonstration of autoantibodies in the pt's serum.
Risks: A large records study found that childhood adversity (stress) increases the risk for autoimmune disorders in adulthood (Psychosom Med 2009;71:243).....adverse events during childhood (ACEs) included physical, emotional, and sexual abuse and household dysfunction, e.g., domestic violence, substance abuse, mental illness, parental divorce, and criminality (exposure was quantified on a scale of 0–8)....After adjustment for sex and age, individuals with two or more ACEs, compared to those with none, had a 70% greater risk for Th1 disorders, 80% greater risk for TT1 helper (Th1) disorders (e.g., idiopathic myocarditis or insulin-dependent diabetes), and 100% greater risk for Th2 rheumatic disorders.....Childhood adversity has been linked to subsequent increased levels of CRP, an inflammatory biomarker with a possible role in autoimmune diseases....Other investigators have

identified common genetic vulnerabilities for depressive disorders and inflammatory processes.....Clinicians seeing patients with autoimmune diseases should be particularly alert for histories of childhood adversities, and those seeing patients with childhood adversities should similarly be alert to possible adult autoimmune disease.

Types:

A. Connective tissue and rheumatic diseases

Systemic lupus erythematosus: Anti-nuclear. Anti-dsDNA. Anti-nucleaosome. Anti-ribosomal P.
Drug-induced lupus: Anti-histone.
Scleroderma: Anti-nucleolar. Anti-fibrillarin. Anti-scl-70 (topoisomerase). Anti-RNP III.
CREST: Anti-centromere.
Polymyositis/scleroderma: Anti-Ku.
Myositis: Anti-Jo-1.
Rheumatoid arthritis: Rheumatoid factor. Anti-CCP. Anti-RA33.
Sjögren's syndrome: Anti-SS-A (RO). Anti-SS-B (La). Anti-fodrin.

B. Renal disease

Goodpasture's syndrome: Anti-GBM

C. Skin disease

Pemphigus: Anti-desmogleins.
Bullous pemphigoid: Anti-hemidesmosomes.
Vitiligo: Anti-tyrosinase.
Dermatitis herpetiformis: Antiepidermal trans-glutaminase.

D. Nervous system disease

Myasthenia gravis: Anti-acetylcholine receptor. Anti-muscle-specific kinase.
Lambert–Eaton syndrome: Anti-calcium channels.
Peripheral neuropathy: Anti-gangliosides.
Paraneoplastic syndrome: Anti-Purkinje cell cytoplasm. Anti-Hu.

E. Cardiovascular disease

Myocarditis/dilated cardiomyopathy: Anti-myosin heavy chain. Anti-B1 adrenoreceptor. Anti-M2 cholinergic receptor.
Vasculitis/Wegener's granulomatosis: C-ANCA/anti-proteinase. Crescentic glomerulonephritis. P-ANCA/anti-myeloperoxidase.

F. Gastrointestinal disease

Celiac disease: Anti-endomysial. Anti-tissue transglutaminase. Anti-gliaden.
Hepatitis: Anti-smooth muscle. Anti-nuclear. Anti-liver kidney microsomal (LKM).
Primary biliary cirrhosis: Anti-pyruvic dehydrogenase.

G. Endocrine disease

Gastritis: Anti-parietal cell. Anti-intrinsic factor.
Thyroiditis: Anti-thyroglobulin. Anti-thyroperoxidase.
Graves disease: Anti-TSH receptor.
Type 1 diabetes: Anti-islet cell. Anti-GAD. Anti-insulin. Anti-IA-2. Addison's disease (adrenalitis). Anti-21 hydroxylase.

H. Hemotologic disease

Hemolytic anemia: Anti-RBC (Coombs' test).
Thrombocytopenia: Anti-platelet.
Antiphospholipid syndrome: Anti-cardiolipin. Anti-B2 glycoprotien 1. Lupus anticoagulant.
**Ref: (Handbook of Human Immunology. 2008. O'Gorman and Donnenberg. CRC Press)

Immune Cells:

Links: Humoral | **Cellular |** Basophils & Mast | **Phagocytes |** Lymphocytes |

Overview: All cells of the immune system are derived from pluripotent stem cells in the bone marrow (BM). The primary lymph organs are the thymus, BM and fetal liver. The secondary lymphoid organs include the lymph nodes, spleen and gut-associated lymphoid tissue.

Humoral-Mediated Immunity: Link: **Immunoglobulins |** Antibody (Ab) mediated, from B-lymphocytes, which synthesize receptors (immunog-lobulins = **Ig's**) able to recognize and bind specific structures (antigens, determinants, epitopes). The primary defense against bacteria. The human immunoglobulins are a family of proteins that confer humoral immunity and perform vital roles in promoting cellular immunity. 5 distinct classes or isotypes of immunoglobulins (IgG, IgA, IgM, IgD, and IgE) have been identified in human serum on the basis of their structural, biological, and antigenic differences. IgG and IgA have been further subdivided into subclasses IgG1, IgG2, IgG3, and IgG4 or subclasses IgA1 and IgA2 on the basis of unique antigenic determinants Multiple allotypic determinants in the constant region domains of human IgG and IgA molecules as well as kappa light chains indicate inherited genetic markers.

Tests for Humoral immunity: serum levels of IgG, IgA, IgM, and IgE; measurement of isohemagglutinins Ab response to immunization of diphtheria, tetanus, and pneumococcus polysaccharides, IgG subclass determination, B-cell quantitation by flow cytometry and mitogenic stimulation assay. Mounting evidence points to an association between enterovirus infection and induction of beta-cell autoimmunity (J Med Virol 2003;69:91-98.).

Cell-Mediated Immunity: Links: Lymphocytes | **Cytokines |**

Cellular immunity, as opposed to humoral immunity, encompasses a broad spectrum of immune phenomena mediated by several well-characterized cell populations. The latter comprises various subsets of lymphocytes: T, B, and natural killer (NK) cells, which are derived from a common lymphoid progenitor cell in the fetal liver and bone marrow. Monocytes, dendritic cells (DCs), and granulocytes represent hematopoietic cells with lineages distinct from that of lymphocytes, but which also participate in cellular immune reactions. They have the capability to mediate non-major histocompatibility complex (MHC)-restricted cytotoxicity and to release a variety of enzymes and cytokines. Cell-mediated via T-lymphocytes and their cytokines. The primary defense against viruses, fungi, tumors, allograft rejection and intracellular organisms. T lymphocytes (T cells) do not use Ig's as antigen receptors but rather use a distinct set of genes encoding four polypeptide chains (a, b, g, and d), each with variable and constant domains, used to form T-cell receptors (TCRs). The TCR is a heterodimer, either an a b or a g d chain combination, which recognizes and

binds antigen attached to antigen presenting cells. This heterodimer, which is not covalently linked together, is complexed with several other molecules (e.g., CD3, CD4, and CD8), that provide stability and auxiliary functions for the receptor. Unlike immunoglobulin, which can bind to free antigen alone, TCRs bind only to specific combinations of antigen and certain self cell surface molecules. Like Ig's, the TCR chains contain variable and constant domains. The variable domains are encoded by a series of V, J, and sometimes D (b and d chains only) gene clusters that undergo DNA rearrangement, and the constant regions are encoded by constant genes.
Tests for: complete blood cell count (CBC); lymphocyte phenotyping; delayed-type hypersensitivity skin testing. Lymphocyte proliferation in response to mitogens, one-way mixed lymphocyte reaction assay, cytokine production assay and cytotoxicity assay.
Basophils, Granulocytes and Mast Cells:
Have receptors for IgE and release mediators such as Histamine, Leukotrienes, Prostaglandins and Platelet Activating Factor (PAF) in both the immediate hypersensitivity and late-phase type allergic reactions. Mast cells and basophils bear receptors (FceRI) for the Fc portion of unbound IgE, permitting them to use IgE on their own surface as an antigen detector. When antigen binds simultaneously to two or more such IgE molecules on the same mast cell (called bridging), a signal is transmitted into the cell, leading to degranulation and release of a variety of mediators, including histamine, resulting in immediate hypersensitivity (allergic) responses. Neutrophils are drawn to sites of inflammation by cytokines, where their phagocytic activity and production of enzymes and other soluble mediators contribute to the inflammation. Eosinophils are involved in immune responses against large parasites, such as roundworms, and are apparently capable of killing them by direct contact.

Phagocytic Cells:

Macrophages, PMN's and eosinophils all produced in the BM and enter tissue in response to cytokines and other chemotactic factors in response to inflammation. These cells rely on opsonization (Ab coating) and the process of phagocytosis. These cells do not usually recognize antigen alone in its natural form, but rather bind to antigen that has been processed and presented on the surface of appropriate APCs (Antigen-presenting Cells). APCs internalize antigen, enzymatically degrade it into fragments (processing), and put the fragments back onto their surface in association with class I and II MHC molecules (presentation). APCs include monocytes, macrophages, and other related tissue-specific cells that express class II MHC molecules (e.g., astrocytes in the central nervous system, Langerhans cells in the skin, Kupffer cells in the liver, and so forth). In addition, B lymphocytes (which normally express class II) can efficiently process and present antigen. There are some other cells that are capable of transient expression of class II (e.g., vascular endothelium). In addition, a variety of other molecules on APCs and T cells serve to stabilize the contact between the TCR and combination of antigen and MHC molecule. Can test for phagocytic function via a CBC, nitroblue tetrazolium test, phagocytosis assay, bactericidal assay and chemotaxis assay.
Deficiency leads to: neutropenia, chronic granulomatous disease, leukocyte adhesion defect 1 and 2, myeloperoxidase deficiency, leukopenia, dermatitis, impetigo, cellulitis, periodontal disease, recurrent cutaneous abscesses, suppurative adenitis, delayed separation of the umbilical cord, impaired wound healing, lung abscess due to Staphylococci, Pseudomonas, Serratia, Klebsiella, Aspergillus, Candida.

Lymphocytes:

Links: T-cells | **LAK cells** | Helper | **Cytotoxic** | Suppressor | **Transplant Rejection** | Immunoglobulins & B-cells | **Immune Deficiency** |
…….END DEMO of this chapter.

Cardiology

Edited by Anthony DeFrance MD, Cardiologist.

Contents: Exam | **Murmurs** | Athletes & Sudden Death Sydromes | **Valves** | Endocarditis | **ECG's** | *Arrhythmia's* | **A-Fib** | ACLS / BLS | **Palpitations** | Pericarditis, Tamponade and Contusion | **CAD** | Angina | **Infarct / ACS** | Testing & Interventions | **Cardioversion** | Tumors | **Failure (CHF)** | Hypotension | **Vascular Diseases** | Aortic Aneurysm | **Peri-Operative Eval** | Syncope | **Various** | Diagram for Pt's | **Congenital Heart Defects** | Prevention | ***Hypertension*** | Lipids | **MEDS** | Healthy Living | **BNP** | CRP | **PEDIATRIC** | Edema | ***INDEX*** |

Cardiac Exam:

Links: Hx | **Exam** | Auscultation | **S1** | S2 | **S3** | S4 | **Timing of sounds** | Peripheral & Carotid Pulses | **Murmurs** | CXR Eval & References | **CHF** | Venous Waveforms | **CXR** | Valsalva Maneuver | **Ankle Brachial Index (ABI)** | See Neurology for PFO | See Ortho for Pre-participation Cardiac Hx & Exam |

Basic Cardiology Hx:

Hx of CAD (past valvular surgery, PCTA, CABG), dyspnea on exertion (DOE), orthopnea, paroxysmal nocturia dyspnea (PND), peripheral edema, discomfort with exercise/ exertion, weakness/ fatigue, cold hands/ feet, nocturia, faintness/ syncope, h/o joint pains or chorea (St. Vitus' Dance in rheumatic fever), palpitations, skin flushing. ~20–25% of all Westerners have cardiovascular disease (including hypertension), accounting for almost 40% of the mortality from all causes.

Cardiovascular Exam:

Links: Inspection | **Palpation** | Percussion | **Exam** | **Heart Rate** | Auscultation | **Peripheral & Carotid Pulses** | See Ortho for Pre-participation Cardiac Hx & Exam |

Normal Heart Rate:
Newborn 80–180/min.
1 year 80–140/min.
2 years 80–140/min.
6 years 75–120/min.
10 years 50–90/min.
16 years 50–90/min.
Adult 60–100/min.
Geriatric 60–100/min.
Capillary Refill:

Normal: <3 seconds.
Increased: >3 seconds. Seen in low cardiac output or decreased peripheral perfusion.
Normal Blood Pressure:
Newborn 73/55 mmHg.
1 year 90/55 mmHg.
2 years 90/55 mmHg.
6 years 95/57 mmHg.
10 years 95/57 mmHg.
16 years 120/80 mmHg.
Adult 120/80 mmHg.
Geriatric 120/80 mmHg.

Inspection:

Look for clubbing, cyanosis and peripheral edema (C/C/E), splinter hemorrhages (endocarditis), livedo reticularis (cholesterol embolization), retina (papilledema, endocarditis), skin for lipid deposits (xanthomas).

Palpation:

Start at the precordium and suprasternal notch regions for thrills and hypertrophy.
Other points of palpation: radial, brachial, femoral, carotid, popliteal, posterior tibial, dorsal pedal. See Peripheral & Carotid Pulses |
A **thrill** (palpable vibration) best felt with the base of the fingers (distal palm) during systole. If over precordium, it suggests ventricular septal defect (VSD), AS or IHSS. If over suprasternal notch it suggests AS or PS.
Apex beat (Apical impulse): the most lateral palpable ventricular movement/ impulse. Generally synonymous with PMI so long as one is not feeling a dilated pulmonary artery, enlarged RV, aortic aneurysm or ventricular aneurysm.
Normal apical impulse is felt at the 5th IC space (1cm, dime-sized) just left of MCL. It is felt or seen as a brief tap. The best position to palpate is with the pt in the sitting position with their feet on the bed (some sources say in 45 deg L lat decub on exhalation). Locate it with your fingertips (front or behind approach) It sometimes helps to first estimating its location by "visualizing" its location on a standard CXR. LVH likely if its size is >3cm (stethoscope size) along with an S3 that diminishes following extrasystole (S4 is potentiated). It should be located ≤10cm from the midsternal line in adults. An apical impulse more than 10cm from the midline is seen in cardiomegaly.
Point of maximal impulse (PMI): (left ventricular contraction) Usually at the apex, at the 5th ICS in the midclavicular line (MCL), but can be elsewhere if aneurysm or dilation are is present. It is best palpated in the left lateral decubitus, HOB elevated to 30 deg, with R hand from R side of pt as simultaneously palpate R carotid a., initially locate in L lat decubitus position, then feel as pt is supine. Can use a long cotton tip applicator (CTA) or tongue blade to magnify the pulse, which also enhances palpation and S3, S4 and MS murmurs. Left shift = cardiomegaly. Left sternal border = right ventricular hypertrophy (COPD). Reduced in emphysema, hyperinflation. Lobar collapse—shifts toward collapse. Pneumothorax—shifts away from pneumothorax.
With Cardiac enlargement (dilation) --> dilation or increase in chamber volume (overload) with or w/o proportional hypertrophy. Have an enlarged PMI which is **displaced laterally** but of normal duration.
Cardiac hypertrophy --> enlargement of the muscle, not the chamber volume, which remains normal or is encroached upon. Due to pressure overload. Have a **sustained** but normal size & location of the PMI.
Heaving: a sustained impulse, usually due to inc pressure load (AS or HTN).
Hyperkinetic: inc amplitude and inc area, seen with inc volume load (AR and MR).
Pulmonic palpation: 2nd intercostal space at the sterna border. Increased—pulmonary hypertension.

Palpation of Cardiac Impulse:

Normal Cardiac Impulse = Within MCL 4th or 5th LICS.
Hyperkinetic Apical Impulse = Exaggerated thrust at cardiac apex (coincident with S3, if present). **Seen in:** Normal child. Hyperdynamic states. VSD. PDA. Aortic or mitral regurgitation.
Sustained Apical Impulse = Maximal at apex; coincident with S4. **Seen in:** Aortic stenosis. Systemic hypertension.
Hyperkinetic RV Impulse = Maximal at LSB 3rd-4th LICS. **See in:** ASD and pulmonary regurgitation.
Sustained RV Impulse = Maximal at LSB, 3rd-4th LICS = **Seen in:** pulmonary hypertension and pulmonary stenosis.
Ectopic LV Impulse = Maximal over mid-precordium. **Seen in:** ventricular aneurysm.

Percussion:

Used to estimate the size of the heart by locating the left heart border. Relative dullness usually begins at the L mid-clavicular line (MCL) and becomes absolute 2-3 cm medially. Limited by obesity, large muscles, breast tissue and lung dz. Most cardiologist find it quicker and more accurate to palpate the ventricular apical impulse.

Right Atrial Pressure:

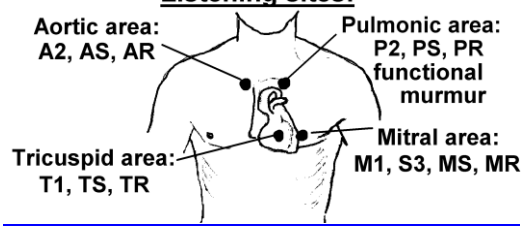
Estimate by measuring vertical height of venous blood in jugular v. and adding center of atrium (= ~5cm from sternal angle) (sternomanubrial jct = 2nd rib). 5+ ht above sternal. nl = 8cm (= 6mmHg). See "JVD" under **CHF |.**
Estimating the Central Venous Pressure on Physical Examination:
The pt should be in the recumbent position with trunk at a 45 degree angle. The central venous pressure in cm H2O = (vertical height of jugular venous distention above the sternal angle in cm) + 5 cm. The central venous pressure in mm Hg = (central venous pressure in cm H2O) / 1.36. Where: for a 45 degree angle, then the vertical height in cm = (distended length along the jugular vein) / SQRT(2). And 5 cm H2O is 3.65 mm Hg. Normally the right atrial pressure is 2.5 to 6.0 mm Hg with a mean of 3.5. At pressures < 3.7 mm Hg the jugular vein is not distended; a pressure of 6 mm Hg would show a jugular distention of 3.2 cm. An elevated central venous pressure may be seen in right heart failure, constrictive pericarditis and obstruction of the superior vena cava. (Droste C, von Planta M. Memorix: Clinical Medicine. Chapman & Hall Medical. 1997. page 50)
Endomyocardial Biopsy: indicated for evaluating possible rejection of a transplanted heart, determining doxorubicin toxicity and myocarditis. Sarcoidosis and alcoholic cardiomyopathy are not s/s.
Dextrocardia: Heart apex is to the right. With situs inversus (39%) have reversed symmetry of the viscera. With situs solitus (34%) have additional anomalies but normal symmetry of the viscera. Situs ambibuus (26%) has abnormal heart symmetry. For the average pt with dextrocardia and situs inversus there are no significant cardiac defects and their life expectancy is normal. ECG with

show pwaves, QRS complex andT waves all inverted in lead I (Int J Cardiol 2003;88:143-55). 20% of the pt's have Kartagener's syndrome (primary ciliary dyskinesia). Some cases X-linked.

Cardiac Auscultation:

Links: S1 | **S2** | S3 | **S4** | S3 Vs S4 | **Abdominal Murmurs** | Pericardial Friction Rub | **Ejection Clicks** | Opening Snap | **Timing of sounds** | Murmurs | **Listening areas** | Murmurs |

Listening sites:



Exam Findings on Inspection / Palpation:

Cardiothoracic reference areas. Each point of auscultation generally correlates with a cardiac valve. Stenotic lesions with be heard at these respective areas, in contrast, regurgitant murmurs can radiate far from the point of origin.
Aortic area (2nd RICS): Pulsation of aortic aneurysm. Thrill from aortic stenosis. Accentuated aortic valve closure (HTN).
Pulmonary area (2nd-3rd LICS): Pulsation of increased pressure/flow in PA. Thrill from pulmonary stenosis. Accentuated pulmonary valve closure (pulmonary HTN).
Tricuspid area = Precordium = RV area (lower half of sternum and subxyphoid area): Sustained systolic lift from RV hypertrophy. Thrill from VSD. Prominent RV impulse in thin pts from fever, anxiety, anemia, hyperthyroidism, pregnancy.
Mitral area = Apex = LV area (5th LICS just past midclavicular line): Thrill of mitral disease. Palpable S3.
Normal Apical impulse: light tap or absent, lasts < half of systole, occupies one interspace
Abnormal Apical impulse: forceful, sustained up to S2, laterally displaced, diffuse (occupies > 1 interspace)
Epigastrium: Increased aortic pulse from aortic aneurysm or aortic regurgitation.
Base = aortic & pulmonic areas.

S1 **-->** Due to closure of AV valves (MV/TV). Best heard at apex or lower left sternal border (LLSB) with the Bell. Due to closure of mitral & tricuspid valve.
Louder --> MS with mobile valve, WPW, sinus tachy, LA myxoma, Short PR, inc contractility.
Softer --> calcified mitral valve (MS with rigid valve), severe HR, LBBB, HTN, MR, AI, pericardial effusion, inc PR interval (1st AVB).
Narrow split --> normal.
Prominent split --> Ebstein's anomaly, TS, Pacing, RBBB.
Differentiating S1 Vs S2: if you have difficulty distinguishing the 1st (S1) and 2nd (S2) heart sounds at the apex, listen carefully at the LUSB, where the 2nd heart sound splits on inspiration and is usually louder than the first. Then while keeping your focus on the rhythm of the heart, "inch" the stethoscope slowly toward the LLSB.

S2 **-->** Due to closure of semilunar valves (AV/PV) (**Aortic =A** is louder and before **Pulmonic =P**), best heard with diaphragm at LUSB (pulmonary area) **in the recumbent position**.
Normally split: single in expiration, split in inspiration (= **physiologic**). Normally it is more obviously split during inspiration (max of 0.06s) (Vs expiration @ 0.03s) (inc thoracic pressure. Split due to **-->** inc venous return **-->** inc R ventricular output delays closure of pulmonic valve). Normal = P2 > A2.
Persistent split (widened S2) --> Does not disappear on exhalation in RBBB, MR, PS, pulmonary embolism, ectopic or pacemaker beats originating in the LV (all cause delayed function of the RV.
Wide split --> RBBB, PVC, PE, pulmonary HTN, PS, VSD, MR, LV pacer (rare).
Fixed split --> ASD, severe RV dysfunction. (an opening snap in MS or midsystolic click in MVP may **simulate** it)
Reverse (Paradoxical, Expiratory) split --> P2 before A2 in organic Heart dz **-->** LBBB, RV pacer, right sided PVC's, HOCM, HTN, WPW, patent ductus arteriosis, angina, AMI, AS, AR.
Single S2 --> Increases with aging, normal variant after age 50yo, AS, PS, HTN, CAD, any cause of paradoxical splits, tetralogy of Fallot.
Loud A2 --> HTN, aortic dilation, coarctation of aorta, AR.
Loud P2 --> pulmonary HTN, thin chest wall.
Narrow split --> pulmonary HTN.
Pseudo-splitting: S2 plus opening snap. S2 plus S3. S2 plus pericardial knock. S2 plus tumor plop.

S3 **-->** "ventricular gallop", ventricular filling. Heard during early diastole. A low-pitched sound during rapid ventricular filling; may arise from either ventricle; typically best heard at apex in left lateral decubitus position; may be normal finding in children and young adults; pathological finding inpts > 40 yr; associated with ventricular dysfunction of any cause, increased early diastolic filling (hyperkinetic states, mitral/tricuspid regurgitation, left to right shunts), restrictive/constrictive pericarditis. Best heard at the apex (mitral area) with bell in left lateral recumbent position at expiration and after exercise or leg elevation to increase venous return. Occurs ~0.12-0.16 sec after the 2nd heart sound. There is a high interobserver variability in detection as it is low frequency and difficult to hear.
Pearl: Palpation of the carotid artery simultaneously with auscultation helps distinguish S3 from S4 as S4 occurs before and S3 after the carotid upstroke. There is a decreasing incidence with age. 70% in age<20yo, 40% age 20-29, considered abnormal in age>40yo. Thus it is physiologic in age <40yo and in during pregnancy. May be an early sign of L-sided heart failure. In pathological state, may represent **high flow states** across AV valves **-->** MR, TR, anemia, thyrotoxicosis, pregnancy, aortic valve fistula, LV/RV dysfunction with increased volume. Also seen in myocarditis, progressive systemic sclerosis, VSD, AR, AS, clinical decompensation of CHF and ischemic heart disease. S3 is more specific for LV dysfunction and superior to S4 (JAMA 2005;293:2238-44) (neither are sensitive).

Kentucky=**S3 -->** cadence sound like: Ken-S1 / tuck-S2 / y-S3.

S4 --> "atrial gallop, presystolic gallop or S4 gallop". A fourth heart sound is a significant finding, and is associated with abnormal ventricular filling, which can assist in the dx of CHF or acute MI. Due to atrial contraction causing blood to enter a stiff ventricle. In normal individuals it is usually too soft and low-pitched to be heard. Best heard at the apex. Physiologic (normal) S4 seen in athletes (especially tall ones) with physiologic hypertrophy or with a concomitant physiologic S3 Normally never present in age <50 or it indicates a myocardial abnormality. Seen in LV & RV hypertrophy, ischemia (1-2 days after AMI), **high flow states**. Almost always pathological in child, signaling dec ventricular compliance from hypertrophy. Absent in A-fib as lose atrial kick.
Tennessee=**S4 -->** cadence sound like: Te-S4 / nne-S1 / ssee-S2.
L-sided (LS4) --> heard at apex in L-lat recumbent position., usually with presystolic apical impulse. Inc with leg raising & exercise. May be normal variant in age 50yo, often from dec LV compliance (HTN, AS, AI, cardiomyopathy), common in AMI, angina attack, AVB, hyperkinetic circulatory states (hyperthyroid, anemia). Not seen in MVP, but often in acute MR due to ruptured cordae tendineae.
Right sided (RS_4) --> heard at LLSB. Due to dec compliance of RV, seen in pulmonary HTN (ASD, VSD, PDA, PE, PPH, PS, cardiomyopathy).
Dx of "Physiologic S4": age <40yo if athletic (otherwise 20yo), should have a physiologic S3, a normal physiologic split S2, a normal apex beat, a venous hum in the neck, a normal ECG (r/o HCM) and normal CXR.
Differentiating the S3 from the S4: they both have the same quality and pitch. Slowing the rate with carotid pressure may help (if not contraindicated). One can wait for the pulse after a PVC as an S4 will be heard preceding the S1 at the end of the pause.
Tennessee=**S4 -->** cadence sound like: Te-S4 / nne-S1 / ssee-S2. **Vs Kentucky**=**S3 -->** cadence sound like: Ken-S1 / tuck-S2 / y-S3.
Auscultation of the fourth heart sound (S4) by attending cardiologists provides significant information on ventricular filling, but the ability to accurately auscultate S4 is significantly lower among physicians with lower levels of experience. For these physicians, computerized acoustic cardiography is of value (Clin Cardiol 2009;32:69-75).....The sensitivity of S4 auscultation was low, ranging from 39% to 46%, and the specificity was fair with auscultation, ranging from 76% to 80% for identifying patients with abnormal measures of left ventricular filling pressure, compared with acoustic cardiography.

Other Cardiac Findings on Exam:

Abdominal Murmurs (Bruits): arterial and venous flow murmurs are normal in age <25yo. Abd murmurs are pathologic in 95% of those >50yo and usually represent renal artery stenosis, aortic atheromatous obstruction, splenic-aortic-SMA or iliac artery aneurysms/ stenosis, hepatic malignancy, alcoholic hepatitis, and portal-systemic vein anastomoses.
Diastolic bruit may indicate renal artery stenosis/ renal vascular HTN.
Systolic bruits are usually normal.
Bruit: an audible vascular sound associated with turbulent blood flow. Although usually heard with the stethoscope, such sounds may occasionally also be palpated as a thrill. Depending on a variety of factors, these bruits may be systolic, primarily systolic with extension into diastole, or continuous. Cranial and orbital bruits are vibrations resulting from turbulence in intracranial or extracranial vessels. Although usually systolic in timing, these bruits may extend into diastole or even be continuous. These sounds may originate within the cranium or be transmitted from arteries in the neck or, occasionally, from cardiac valvular lesions. Cervical bruits and hums may arise from neck arteries or veins, and may be innocuous findings or indicate underlying pathology. Bruits arising in the carotid arteries are produced by intrinsic stenosis or, occasionally, with vascular occlusion from extrinsic compression. The cervical venous hum is auscultated over the internal jugular veins in many normal children. Supraclavicular bruits during systole are a frequent finding in normal children and in adults with subclavian or vertebral artery stenosis.
Pericardial Friction Rub: Single, double or triple throughout the cardiac cycles, very high frequency, best heard with diaphragm at various precordial locations. Usually intensifies when pt leans forward and exhales.
Ejection Clicks: Uncommon, abnormal, high pitch sounds reflecting abrupt opening of AV or PV or distention of the great arteries. They are clicking ejection sounds such as the opening snap of the mitral valve or the systolic click due to mitral insufficiency. Root clicks are abnormal, high pitch sounds reflecting abrupt opening of AV or PV that sound similar in character and timing as a true ejection click from the valve. Seen in AS, HTN, dilated aortic root, PS, pulmonary HTN, dilated pulmonary artery.
Systolic Click: High-pitched sound; early sounds (just after S1) in aortic or pulmonary areas are ejection sounds that may indicate dilation of aorta or main PA, systemic or pulmonary HT, or aortic or pulmonary stenosis (due to opening of stenotic but mobile valve cusps); mid/late systolic clicks heard in mitral valve prolapse syndrome.
Mid to Late Systolic Clicks: common, single or multiple high frequency sounds due to abrupt tensing of chordae tendinea or redundant leaflet scallops during MV prolapse.
Inc : stand, valsalva, amyl nitrite, volume depletion.
Dec: supine, squat, pregnancy.
Opening Snap (OS): Audible opening of the AV valve when stenosed or MV when prolapsed, MS, TS. Heard at apex near xiphoid. Early diastolic high-pitched sound heard in mitral stenosis; valve must be stenotic but mobile; can be confused with widely split S2, but S2-OS interval decreases during inspiration.
Cranial and Orbital Bruits: usually normal, innocent findings in younger persons, occurring in 30 to 60% of normal infants and children under 6 years of age. In adults, nonorganic orbital bruits may be noted in pt's with high cardiac output states, or those with decreased blood viscosity, as with severe anemia. Often systolic in timing and auscultated over both orbits, these bruits are especially frequent in pt's with several simultaneous reasons for increased vascular flow, such as anemia and high fever. If the above conditions are excluded, cranial-orbital bruits in the adult are usually organic (pathologic) and indicate an underlying abnormality. Arteriovenous fistulas, arteriovenous malformations, and intracranial hemangiomas are associated with a prominent bruit in 30 to 50% of cases. Often these are continuous bruits and are appreciated best over the orbits, where osseous dissipation of vibrations is minimized. Atherosclerosis of the arteries in the neck may rarely produce an orbital bruit. With tight internal carotid stenosis, either extracranially or intracranially at the carotid siphon, a bruit may radiate cephalad and be auscultated over the ipsilateral orbit. With total unilateral carotid occlusion, there may be no bruit over the affected carotid and increased flow through the

unoccluded carotid, which can cause a soft bruit over the contralateral eyeball. Other intracranial pathology affecting vascular structures may produce soft systolic orbital bruits.

Timing (Phase of cycle) and Sounds:

Diastolic Heart Sounds/ Murmurs: Normal --> 3rd & 4th heart sounds.
Systolic Sounds --> ejection click, MVP click, pericardial rub, functional murmur, AS, PS, MR, TR.
Due to Abnormal Cardiac Structure --> MS, TS, AR, PR, atrial myxoma, VSD, ASD, MR.
Due to inc Flow --> anemia, sepsis, thyrotoxicosis, RF with volume overload.
Pericardial knock --> a high or low pitched sound that occurs during diastole with constrictive pericarditis. It is due to the abrupt cessation of ventricular filling due to a stiff/ thickened pericardium.
Continuous Heart Sounds --> pericardial rub, machinery murmur, mammary soufflé, PDA **-->** early, mid, late, pan, pre.

Peripheral Pulses:

Links: Grade / Strength & Carotids | **Hyperkinetic** | Hypokinetic | **Pulsus Alternans** | Pulsus Paradoxus | **Pulsus Bisferiens** | Auscultate for Carotid Bruit? | **Peripheral Vascular Disease** | Training Effects on HR | **Pulse Pressure** | Valsalva | **HJR** | JVD | **Venous Waveforms** | Vascular Diseases |

Really conducting 3 examinations. The first is an examination of the cardiac rate and rhythm, the second is an assessment of the characteristics of the pulse as a reflection of cardiac activity, and the third is an assessment of the adequacy of the arterial conduit being examined. The major pulses routinely palpated on physical examination are the radial, brachial, carotid, femoral, dorsalis pedis, and posterior tibial. In special situations, the abdominal aorta and the ulnar, subclavian, popliteal, axillary, temporal, and intercostal arteries are palpated.
Pearls: If a pulse is irregular, it is better to auscultate the heart; some cardiac contractions during rhythm disturbances do not generate a stroke volume sufficient to cause a palpable peripheral pulse. Palpating distal pulses to screen for peripheral artery disease (PAD) is not nearly as accurate as using the Ankle Brachial Index (ABI) and can often be misleading. Look for asymmetry (aortic dissection or vascular obstruction), abnormal rate of rise and hardness of the artery. In assessing the abdominal aorta, it is important to make note of the width of the aorta because an increase suggests an abdominal aortic aneurysm. It is particularly important to palpate the abdominal aorta in older individuals because abdominal aortic aneurysms are more prevalent in those older than 70.
Rate: In many ways, the heart rate reflects the health of the circulatory system. A rapid pulse suggests increased catecholamine levels, which may be due to cardiac disease, such as heart failure; a slow pulse represents an excess of vagal tone, which may be due to disease or athletic training.
Cadence:
Irregularly irregular --> A-fib, frequent multifocal PVC or PAC's.
Regularly irregular --> usually due to PAC's, may be seen in PVC's in a pattern.
Grading Strength Pulses: O to 4+. O= absent, 1+ **-->** markedly reduced (weak pulse). 2+ **-->** slightly reduced. 3+ **-->** normal. 4+ **-->** bounding / hyperdynamic pulse. To assess the characteristics of the cardiac contraction through the pulse, it is usually best to select an artery close to the heart, such as the **carotid**. Bounding high-amplitude carotid pulses suggest an increase in stroke volume and should be accompanied by a wide pulse pressure on the blood pressure measurement. A weak carotid pulse suggests a reduced stroke volume. A low-amplitude, slow-rising pulse, which may be associated with a palpable vibration (thrill), suggests aortic stenosis. A bifid pulse (beating twice in systole) can be a sign of hypertrophic obstructive cardiomyopathy, severe aortic regurgitation, or the combination of moderately severe aortic stenosis and regurgitation. A dicrotic pulse (an exaggerated, early, diastolic wave) is found in severe heart failure. Pulsus alternans (alternate strong and weak pulses) is also a sign of severe heart failure. When evaluating the adequacy of the arterial conduits, all palpable pulses can be assessed and graded on a scale of 0 to 4, where 4 is a fully normal conduit, and anything below that is reduced, including 0—which indicates an absent pulse.
Carotid Pulse: Palpable pulsatile changes in the carotid arterial diameter this is virtually identical to the intraluminal pressure pulse. Characterized by a smooth, relatively rapid upstroke and a smooth, more gradual downstroke, interrupted only briefly at the pulse peak. Usually examined with the pt supine and the trunk of the pt's body slightly elevated. The fingers should be positioned between the larynx and the anterior border of the sternocleidomastoid muscle at the level of the cricoid cartilage. In palpating the pulse, the degree of pressure applied to the artery should be varied until the maximum pulsation is appreciated. Most favor the use of two or three fingers with the examiner sitting or standing comfortably at the pt's or standing at the head of the pt's bed. Because simultaneous palpation of the carotid artery and auscultation of the heart is occasionally beneficial, the examiner positioning himself at the pt's right side seems preferable.
Pulse Contour: rapidity of rise and fall.
Small weak pulse: Decreased stroke volume (CHF, shock), aortic stenosis.
Large bounding pulse: Hyperkinetic states, aortic regurgitation, PDA, increased aortic rigidity (atherosclerosis).
Pulsus alternans: Left-sided heart failure (pulse must be regular).
Bigeminal pulse: Arrhythmia.
Pulsus paradoxicus: Constrictive pericarditis, pericardial tamponade, severe COPD.
Anemia, fever, thyrotoxicosis: Increased amplitude and rate.
Shock: Reduced amplitude.
Aortic stenosis: Slowed upstroke, prolonged duration, pulsus parvus et tardus.
Aortic regurgitation: Rapid rise and runoff, may have 2 peaks.
Obstructive hypertrophic cardiomyopathy: Bisferiens: rapid rise, abrupt peak and plateau.
LV failure: Low amplitude; alternans pulse (strong and weak pulses alternate).
Tamponade or constrictive pericarditis: Pulsus paradoxicus (exaggerated decrease during inspiration).
AV fistula: Rapid rise and runoff.
Hyperkinetic (Bounding) pulses: characterized by an increase in the velocity of the upstroke and amplitude. The water-hammer, or Corrigan's, pulse is characterized by a very brisk upstroke, large amplitude, and rapid collapse; it is an extreme form of the hyperkinetic pulse. Hyperkinetic pulses may be associated with anxiety, exercise, fever, thyrotoxicosis, hypertension, aortic

regurgitation, arterio-venous fistula, and patent ductus arteriosus. Pt's with marked bradycardia may compensate with a large stroke volume clinically manifest by a hyperkinetic pulse. The water-hammer pulse is usually associated with **aortic regurgitation**. Also seen with **inc Pulse Pressure -->** dec Distensibility of arterial system (atherosclerosis, HTN, coarctation of aorta), inc stroke volume (normal, anxiety, exercise, complete AVB, AR), inc CO/ dec PVR (fever, anemia, thyrotoxicosis, hyperkinetic heart syndrome, AV fistula, Paget's, Beriberi, cirrhosis)

Hypokinetic Pulse: (weak pulse/ pulsus parvus), signifies narrowed pulse pressure. Ffound in pt's with a reduced stroke volume. Usually produced by low CO in association with inc peripheral venous return (PVR). This group includes pt's with hypovolemia, left ventricular failure, mitral stenosis, AS and MR.

Decreased pulse pressure --> dec CO (shock, CHF, hypovolemia, AMI, tamponade, constrictive pericarditis), Peripheral vasoconstriction (shock, hypovolemia), Mechanical (valvular dz such as MS, MR, aortic outflow obstruction, coarctation of aorta).

Pulsus Alternans: Pulse waves alternating strong and weak (during a regular rhythm). A beat-to-beat variation in the amplitude of the pressure pulse. It may be accentuated following a premature contraction. This pulse abnormality is usually best appreciated in distal arteries that have a wider pulse pressure than the carotid artery. Pulsus alternans is a manifestation of severe depression in left ventricular systolic function. Best assessed by sphygmomanometer **-->** inflate rapidly to above the systolic blood pressure (SBP), then deflate slowly until sounds first audible. At this point, the beats are heard at ½ of the HR. When the cuff is deflated further, the rate doubles. Seen if LV failure from AMI, PSVT. Poor prognosis.

Pulsus Paradoxus: Weakening of the pulse during inspiration. Seen in asthma (and obstructive airway dz's), cardiac tamponade, constrictive pericarditis, restrictive cardiomyopathy.

Step #1 Inflate BP cuff above SBP as pt is supine.

Step #2: deflate the cuff so that the initial Korotkoff sounds are heard during slow exhalation.

Step #3: check the peak SBP during inspiration. >10mmHg difference suggests pulsus paradoxus. If RV compliance is restricted will see >10mmHg dec in SBP during inspiration.

Quick check: Pulse diminished in amplitude during inspiration. Continuously monitoring pulsus paradoxus (automated pulsus paradoxus monitoring system = AT-PP) can improve the management of pt's with acute asthma (Chest 2006;130:754-765).... can aid in determine disposition among emergency department pt's with acute asthma as able to objectify the work of breathing… The specificity and sensitivity of the AT-PP was 78% for both, similar to the specificity and sensitivity of physicians (89% and 83%). Pulsus paradoxus is among the most reliable of the traditional clinical signs suggesting cardiac tamponade, particularly when faced with a pt with pericardial effusion and the echo is equivocal (JAMA 2007; 297:1810-1818).

Pulsus Bisferiens: double or bifid systolic/ apical impulse. (Latin, "twice beating"). Perceived as two distinct impulses (narrowly separated positive waves) during each heartbeat (systole). Often occurs in pt's with aortic regurgitation and is common in pt's with combined aortic stenosis and aortic regurgitation (AI). A similar pattern may occasionally be noted in other conditions characterized by a hyperkinetic pulse. Itis characteristic of idiopathic hypertrophic subaortic stenosis (HOCM). Also can be seen in AS. Corrigan/ Waterhammer pulse (strong, rapid) also seen in AI.

Inequality: a difference between the pulse amplitude in the two carotid arteries usually reflects atherosclerosis. Other possible explanations include aortic dissection, arteritis, or embolus. Kinking of the carotid artery is occasionally seen in a hypertensive pt and may simulate an aneurysm.

Small and delayed: pulsus parvus et tardus, is characteristic of severe valvular aortic stenosis. It must be recognized, however, that the absence of this finding, particularly in an elderly pt with noncompliant vasculature, does not exclude severe aortic stenosis.

Auscultate for Carotid Bruit?:

The incidence of bruits increases with age, from 3.5% at 45-54 yo to 7% at 65-74yo (Wolf et al 1981). However, only 23% of bruits are found to be associated with a hemodynamically significant stenosis (>=50%), and less than half of significant stenoses occur in the presence of a bruit. There is no correlation between the loudness of a bruit and the degree of narrowing. Thus, a bruit is neither sensitive, nor specific for significant carotid stenosis; still, with the ease of noninvasive imaging, the status of the carotid artery when a bruit is detected is relatively simple. A neck bruit may originate from the carotid arteries or be transmitted from the aortic arch or heart, such as with transmitted murmurs. As of 2008, both the United States Preventive Services Task Force and the Canadian Task Force recommend against routine auscultation of carotid bruits because it is insensitive for severe carotid stenosis and because the management of asymptomatic stenosis is uncertain.

Cons: Found in 4% of adults >45yo, a poor predictor of carotid artery stenosis. A sign of atherosclerotic heart disease, but not generally recommended as a screening as low sen/spec. Only 33% of pt's with a known hemodynamically significant stenosis exhibit a bruit. 10% of pt's with a stenosis <50% have a bruit. Given current data, the time limits of clinical practice, and the greater importance of other risk factors such as hypertension and smoking, we cannot recommend routine carotid auscultation in asymptomatic pt's (J Gen Intern Med. 1998;13:86–90, 140–141)....Rather, we should concentrate our efforts on what we have learned about the efficacy and appropriateness of other medical and surgical interventions to decrease the risk of stroke. There was not an increased risk of stroke in pt's found incidentally to have cervical bruits (Management of asymptomatic carotid bruit. J Vasc Surg. 1987;5:869–873). The auscultation for carotid bruits in screening for underlying arterial disease has been questioned, especially in asymptomatic pt's (Cost-effectiveness of screening for carotid stenosis in asymptomatic persons. Ann Intern Med. 1997;126:337–346). A study with 2736 adults (mean age, 52) found that carotid bruit did not predict the presence of high-grade internal carotid stenosis (OR = 0.9)(J Vasc Surg 2007;46:1215).....the findings cast doubt on the usefulness of routine auscultation of carotid arteries. Carotid bruits are only a weak predictor of an adverse cerebrovascular event, but, as a marker for generalized atherosclerosis, might it predict adverse cardiac events (a prognostic indicator of cardiovascular death and MI according to a meta-analysis of 22 studies (Lancet 2008;371:1587-94).....in the pooled data on 17,295 patients, those with carotid bruits were significantly more likely than those without bruits to experience MIs (3.69 vs. 1.86 per 100 patient-years) or cardiovascular death (2.85 vs. 1.11 per 100 patient-years).....suggests that the presence of a carotid bruit is a coronary risk equivalent (>20% risk of an adverse cardiac event within 10 years), which would call for further measures, such as aspirin and aggressive lipid-lowering, regardless of other risk factors.....but editorialist notes it is unclear whether the finding of a carotid bruit adds important incremental information to conventional risk factors.

Pro's: The presence of an asymptomatic carotid bruit was independently associated with a >6-fold increased risk of stroke in pt's with type 2 diabetes during the first 2 years after the bruit was detected (Stroke. 2003;34:2145)....pt's should receive intensified management of vascular risk factors....The presence of a carotid bruit had no impact on the incidence of stroke beyond this time. In

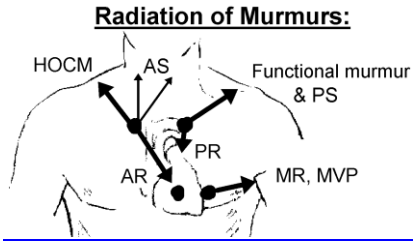
the Framingham cohort, the age-adjusted 2-year incidence of stroke was 2-fold in the presence of an asymptomatic bruit in men and almost 4-fold increased in women (JAMA. 1981;245:1442–1445). A prospective study with 145 pt's found that the sensitivity of carotid auscultation for the detection of a 70%-99% stenosis of the common or extracranial internal carotid artery was 56%, the specificity was 91%, the PPV of a bruit was 27%, and the NPV was 97% (Neurological Research 2002;24705-708)...concluded that carotid auscultation is a useful screening procedure in the detection of carotid stenosis or occlusion, but requires confirmation by carotid ultrasound. Carotid bruits detected by auscultation might serve as markers for heart disease, rather than detect carotid lesions and the subsequent risk of stroke, and could help select patients who would benefit from an aggressive treatment strategy for cardiovascular risk (Lancet 2008; 371:1587-1594).....those with carotid bruits have twice the risk of MI and cardiovascular death than those without carotid bruits.
Auscultation Method: (Vascular Diseases by Pierre J. Vinken. Elsevier Health Sciences. 1989) Should be done with the pt sitting or recumbant. Place the **bell** of the stethoscope over each carotid artery in turn. You may use the diaphragm if the pt's neck is highly contoured. Ask the pt to stop breathing momentarily. Listen for a blowing or rushing sound--a bruit. Do not be confused by heart sounds or murmurs transmitted from the chest.
Tips: Begin in the supraclavicular fossa to elicit subclavian artery bruits and then move cranially along the anterior border of the SCM to the angle of the jaw where the carotids are best heard. Neck rotation can modify carotid bruits and arm positions can modify subclavian bruits. Physiologic bruits typically manifest as a continuous "venous hum. Pathological bruits are typically due to arterial stenosis. and usually appear when the arterial lumen is reduced by at least 50%. The tighter the stenosis, the longer the duration and higher the pitch, Theree is no correlation witbetween stenosis and loudness of the bruit. When the stenosis is very tight, the bruit dampens and disapears wheif occlusion occurs. Cervical bruits can also be due to external carotid a stenosis, dissectiona and to elongation of the carotid artery. The diagnosit significant of carotid bruits has a sensitivity of 47-100%, specificity of 57-100%. PPV 8%, NPV 77% (Sanborn et al 1981 and Von Arbin et al 1983). If both systolic and diastolic it implies severe vascular obstruction or AI. Other arteries may be auscultated under special circumstances, such as suspected temporal arteritis or vertebrobasilar insufficiency.
Observe for carotid pulsations: Place your fingers behind the pt's neck and compress the carotid artery on one side with your thumb at or below the level of the cricoid cartilage. Press firmly but not to the point of discomfort.
Assess the following: The amplitude of the pulse. The contour of the pulse wave. Variations in amplitude from beat to beat or with respiration. Repeat on the opposite side.
Special Maneuvers: Have the pt roll on their left side. Listen with the bell at the apex. This position brings out S3 and mitral murmurs. Have the pt sit up, lean forward, and hold their breath in exhalation. Listen with the diaphragm at the left 3rd and 4th interspace near the sternum. This position brings out aortic murmurs.
Dynamic Maneuvers:
Internal carotid bruit: Breath holding: inc (pitch, intensity, length of bruit). Compression of ipsilateral superficial temporal and facial arteries: inc. Compression of contralateral common carotid artery: Inc.
External carotid bruit: Breath holding: dec. Compression of ipsilateral superficial temporal and facial arteries: Dec. Compression of contralateral common carotid artery: Dec.
Tx: no w/u or tx needed if asymptomatic. Pt's with asymptomatic carotid bruits should not have further diagnostic testing but should be educated about TIA sx's. Mass screening for high-grade asymptomatic carotid artery stenosis is not cost-effective.
Ddx: transmitted heart murmur: differentiation of carotid bruits from transmitted cardiac murmurs sometimes poses a problem. The examiner may slowly "inch" the stethoscope down the carotid; cardiac murmurs should increase in intensity as the stethoscope approaches the precordium. Appropriate and consistent physical findings over the precordium should also clarify this issue. Moreover, cardiac murmurs are only rarely transmitted over the eye, whereas carotid bruits are.
Other: hyperdynamic circulatory states, tortuosity w/o carotid stenosis, stenosis of the subclavian, vertebral or external carotid arteries rather than the common or internal carotid.
ICD-9 Code: 785.9 Other sx's involving cardiovascular system - Bruit (arterial) or Weak pulse
Brachial artery: the preferable site for evaluating the condition of the pt's arterial walls. The hardness and tortuosity of the arterial wall can best be assessed at this site. The heart rate and rhythm are usually assessed by palpating the brachial or radial pulse.
Ref: (Clinical examination of the arterial pulse. Prog Cardiovasc Dis 1967;10:207-35) (Physical examination of the arteries and veins. In: Hurst JW, ed. The heart. New York: McGraw-Hill, 1982;188:93)

Murmurs & Structural Diseases:

Links: Location & Diagram | **Grading** | Tonal quality | **Listening area** | Pearls | **Bedside Maneuvers** | Innocent Murmurs | **Common Pathologic & Congenital Heart Defects** | Common CHILDHOOD Murmurs | **Valves** | Peripheral & Carotid Pulses |

ICD-9 Code: 785.2 Heart murmur NOS. 785.3 Other abnormal heart sounds
Location:

Radiation of Murmurs:



L sided murmurs: (Aortic, Mitral) inc with Expiration.
R sided murmurs: (Pulmonic, Tricuspid) Inc with Inspiration, likely benign.
Diamond-Shaped Murmur: Crescendo-Decrescendo. Ex: AS.
Ejection Murmur: diamond shaped, but ends before the 2nd heart sound.

Graham Steels Murmur: early diastolic caused by pulmonic insufficiency, seen with pulmonary HTN and MS. Best heard at Erb's point.
Flow (Hemic) Murmur: cardiac or vascular murmur heard in anemic pt with no valvular lesion.
Innocent (Functional) Murmur: no anatomic abnormality, benign.

Grading System For Heart Murmurs:

Grade 1 --> 6

1/6=barely audible (only just heard with concentration and optimal conditions.
2/6=soft, but easily audible (no thrill), loud enough to be obvious.
3/6=loud, easily heard even in a noisy child. Louder than grade 2. A murmur **"louder"** is pathologic.
4/6=loud and associated with a **palpable thrill** conducted to the wrist. Louder than grade 3.
5/6=heard with only the edge of the stethoscope in contact with chest wall.
6/6=heard with stethoscope a few cm from the chest wall.
Other: Heart murmur intensity may be graded using heart sounds as an internal reference, which may enhance both accuracy and consistency compared to the standard (Levine) 6-point grading system (Arch Pediatr Adolesc Med. 2005;159:329-334).
Timing: late systolic with coarctation of the aorta, MVP, pulmonary artery stenosis and HCM.
Systolic after AMI: papillary muscle dysfunction, ruptured interventricular septum (VSD, harsh with palpable thrill), MR secondary to cardiac incompetence, misdiagnosis (rub in Dressler's).
Computer-Aided Medical Device (Cardioscan): (Acoustic Cardioscan, made by Zargis Medical Corp.) intended for analyzing heart sounds for the identification of suspected murmurs. The device acquires, processes, and analyzes heart sounds to provide graphical and textual results. Results are provided in both electronic and printable formats, giving physicians a baseline and the ability to detect and track changes over time. Has a sensitivity of 91.8% and a specificity of 68.0%. It also has a 91% sensitivity and at least 92% positive predictivity in the identification of S1 and S2 heart sounds that are key to murmur identification.

Tonal Quality of Heart Sounds:

High-pitched blowing or whooping --> heard with diaphragm --> opening snap, ejection click, MPV click, pericardial rubs, AR, PR, MR, TR.
Mid-range, harsh --> heard with Bell --> Due to high velocity blood flow from high pressure to low. 1st and 2nd heart sounds, AS, PS, functional murmur, HOCM, VSD.
Low-pitched rumbling --> Bell --> 3rd & 4th heart sounds, pericardial knock, MS, TS.
Musical/ vibratory --> nl S1,2, Innocent murmurs.

Listening Areas for Murmurs:

Use a systematic approach when listening to the heart. Start by auscultating the R upper sternal border (RUSB), next the LUSB, then proceed to the LLSB and finally the apex of the heart. Each of these 4 areas corresponds to one of the cardiac valves.
Murmurs: turbulent blood flow due to pressure gradients.
Transmitted murmur: virtually as loud in the area to which it is transmitted as it is in the primary area. Both MR and AS go to the axilla, but AS is much softer than at the apex.
Premature Beat: after a PVC there is a compensatory pause, ventricular filling is prolonged and thus with the next beat the inc EF cause aortic lesions to be louder, but no change with MR.

Intensity: and site of max intensity. **Pitch? Radiation? Quality?**
Apex (PMI) Murmur --> Mitral area, best heard with the bell. MR (pansystolic). MS (diastolic). Can also hear mitral at LLSB.
Aortic Area (2nd R interspace, R Upper Sternal Border, RUSB) --> AS (SEM), AR (diastolic blowing). Use diaphragm.
Pulmonic Area (2nd L interspace and along LUSB) --> PS (SEM), ASD (systolic), PR (diastolic). Use diaphragm.
Erb's Point --> PS and AR (diastolic). 3-4th L sternal border.
Lower Left Sternal Border (LLSB): Tricuspid area best heard with the bell. TS (diastolic), TR, Innocent Murmur, HOCM, VSD, Congenital Pulmonary Stenosis, Tetralogy of Fallot, Idiopathic Dilation of Pulmonary artery.
Mid Left Back --> coarctation of aorta (systolic).

Cardiology Evaluation and Murmur Pearls:

…….END DEMO of this chapter.

Dermatology:

Edited by Gary S. Novatt, MD, Dermatologist.

Contents: Anatomy | **Basic Care** | Morphology, Patterns & Approach | **Topical Steroid** | Dermatitides | **INFECTIONS** | Nails | **Acne** | Sports | **VARIOUS Lesions** | Hyperhidrosis | **Pigmentation** | Keloids & Scars | **Sun** | Malignant / Pre- Lesions | **Signs of Systemic Dz** | Purpura | **Livedo Reticularis** | Varicose / Cosmetic | **Urticaria** | Pruritus | **Bullous / Blister** | PROCEDURES | **Hair** | PEDIATRIC | **Ulcers** | Warts | **Photoaged Skin** | Wound Care & Suturing | **Xerosis** | Allergic Skin Reactions | **Vaginal** | MEDS | **Edema** | *INDEX* | **Allergic Drug Reactions** |

FREE color photos of common derm lesions at http://www.dermnet.com/.
WHY PRESCRIBED THERAPIES DON'T ALWAYS WORK: Researchers investigated the fate of new prescriptions written in a clinical dermatology setting using a national pharmacy tracking system found that 30.7% of the patients did not fill their prescriptions (J Am Acad Dermatol 2008;59:27).....patients with psoriasis were the least compliant, filling only 50% of the new prescriptions.....When conditions do not improve, physicians must ascertain whether patients are using the prescribed therapy or have even filled the prescription.
Anatomy: Our dermis is the largest organ of the body (making up about 10% of the body's weight), it provides the primary defense against external insults to the human body, including chemical, environmental (thermoregulation), infectious and mechanical stressors as well as helps maintain fluid status, and it directly impacts our appearance.
3 Layers of Skin: Epidermis. Dermis. Subcutis (subcutaneous fat).
Layer 1: The Epidermis: 4 cells (see below): Keratinocytes, melanocytes, Langerhans cells, Merkel cells. The primary function is the production of the stratum corneum. The epidermis is purely a cellular structure without blood vessels or nerves. The epidermis

is the thinnest component of the skin (average thickness is 0.1 mm), varying in, thickness from 0.04 mm on the eyelids to 1.6 mm on the palms of the hand. Because the epidermis has no primary blood supply, it depends on capillary loops extending up into the papillary dermis from the subpapillary plexus. The epidermis starts at the basement membrane and extends up through the stratum corneum.

The **4 basic zones (layers)** of the epidermis:

1. Basal cell layer (stratum basalis or stratum germinativum or suprabasal layer).
2. Spiny cell layer (stratum spinosum or ret mucosum).
3. Granular cell layer (stratum granulosum).
4. Cornified layer (stratum corneum). In palmoplantar skin there is an additional zone, the stratum lucidum between the stratum granulosum and the stratum corneum. The combination of the intercellular lipids, matured keratinocytes (corneocytes, with their protein and lipid shells), and intercellular connections between the corneocytes (desmosomes and tight junctions). The mean turnover time of the epidermis is roughly 39 days, with 13 days for the proliferative compartment, 13 days for the maturation of the keratinocytes, and 13 days for stratum corneum transit.

The Stratum Corneum- consists of several rows of flattened cornfield horny cells containing a fatty or waxy material, which are non-nucleated cells. The surface of the cells appears as thin, dry scales. The thickness of this layer varies according to age and site. It is thickest on palms and soles and thinnest on the eyelids. Filaggrin is part of the outermost layers of the skin that help reduce water loss and provide protection against environmental exposures.

Stratum Lucidium - lies on the lower portion of the stratum corneum and consists of a thin clear strip of glistening, translucent, flattened cells without granules or nuclei. Stratum lucidum is present only in the skin of palms and soles.

Stratum Granulosum (granular layer) - lies on the top of the prickle cells and consists of rows of flattened nucleated cells, which contain keratohyaline granules.

Stratum Malpighi (prickle cell layer) - lies on the top of the basal layer and following its direction. This is a very important layer and is involved in most of the pathological conditions of the skin.

Stratum Germinativum (Basal Layer) - is the deeper layer of the epidermis and is composed of a single row of columnar cells arranged vertically to the basement membrane. The cells of the basal layer become modified to form all other cells of the epidermis. The epidermis is maintained by the division of the germinate cells present in the basal layer and differentiate later to keratinocytes in about seven weeks.

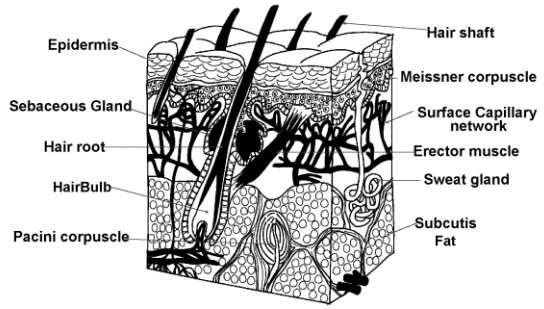
4 Cells of the Epidermis:

Keratinocytes: the largest in number and ultimately form the corneocytes of the stratum corneum. As the keratinocytes move upwards reaching the granular layer of the epidermis, loose their nuclei. Keratinocytes become flattened and heaped on the skin surface as horny dead cells. The thickness of this layer varies according to age and different body sites. Sweat glands produce antimicrobial dermcidin peptides DCD-1 and DCD-1L that can kill certain bacteria and viruses directly (Br J Dermatol 2009;160:243)....these differ from the peptides produced by the keratinocytes, can activate keratinocytes to send signals that recruit nonspecific inflammatory mediators. Activation of the innate immune system as an adjunctive defense may occur when the stratum corneum is disrupted.

Melanocytes: synthesize and secrete melanin-containing organelles called melanosomes that are transported to the end of finger-like projections and are phagocytized by the keratinocytes; this produces the skin's pigmentation and provides ultraviolet protection. Melanocytes are dendritic cells derived from the neural crest and lie on the basement membrane. Melanocytes synthesize melanin from phenylalanine via tyrosine by series of reactions catalyzed initially by tyrosinase giving the skin its color. Pigmentation is related more to melanin synthesis than to the number of skin melanocytes.

Langerhans cells: (leukocytes of bone marrow origin) are located in the midepidermis and send projections between the keratinocytes, spanning from the granular zone above to the dermal/epidermal junction. They are responsible for many immunologic activities and are classified as one of the skin's dendritic cells. As antigen-presenting cells, Langerhans cells can initiate responses in naïve T cells and dictate the net outcome of the interaction. They are important for innate and acquired immunity.

Merkel cells: are low-threshold touch receptors that send projections into the epidermis from their position at the basement membrane. They connect with the developing keratinocytes through intercellular connections, or desmosomal junctions. The nervous system sends unmyelinated nerve endings to the area adjacent to the basement membrane and, at this junction of the dermis and epidermis, Merkel cells connect and thereby communicate with terminal nerve endings.



Layer 2: The Dermis (corium): 2 Layers of the Dermis: **1.** Papillary dermis (thin zone beneath epidermis). **2.** Reticular dermis (bulk of dermis). The dermis contains collagen (types I & III), elastin, proteoglycans, hair follicles, glands (apocrine, eccrine, sebaceous), blood vessels, lymphatics, nerves. The dermis is a dense fibrous layer beneath the epidermis. It is composed mainly of fibrous tissue, elastic fibers and muscle fibers. The upper surface of the dermis is the papillary layer that is a finger like projection into the epidermis and contains the terminal capillaries and sensory nerve endings. Dermoepidermal junction is undulating where ridges of the epidermis, known as ret ridges, project into the dermis. The junction provides mechanical support for the epidermis. The entire surface of the dermis is beset with numerous papillae which are more numerous in some locations such as areola of the nipple and terminal phalanges. Most inflammatory diseases of the skin involve the papillary dermis. The deeper part of the dermis is the

reticular layer, where injury below that level leads to scarring after healing. The dermis contains and supports blood vessels, lymph vessels, nerves, hair follicles, glands and muscle elements.

The dermis is composed of:

Collagen fibers: The dermis is composed mainly of collagen fibers, which are synthesized by fibroblast. The thickness of collagen depends on different factors such as age, sex and body sites. The collagen layer is organized into a smooth superficial layer under the epidermis at the level of papillae and is coarse in the deeper layers. The collage fibers give the skin its toughness and elasticity.

Elastic fibers: are associated with the collagen fibers and both are surrounded by mucopolysaccharides.

Ground Substance: The base of the dermis is a supporting matrix or ground substance which is composed of polysaccharides and protein which are linked to produce macromolecules with a remarkable capacity for holding water in their domain. Within and associated with this matrix are two kinds of protein fibers: collagen, which has a great tensile strength and forms the major constituent of the dermis, and elastin, which forms only a small proportion of the bulk.

The dermis contains the specialized sensory organs and the skin appendages. Below the dermis is a fatty layer known as the **subcutaneous tissue**.

Dermal Cells: derived from the reticulum cell, the primitive mesenchymal cell. The cells of the dermis include :

Fibroblasts: form the fibrous tissue and the matrix (ground substance). Functions in the production of collagen and elastic tissue, synthesize of mucopolysaccharides, metabolize cholesterol and steroids.

Histiocytes: are part of the reticulo-endothelial system. Histiocytes are large phagocytic cells either fixed to the interstitial tissues or wandering .

Mastocytes: are specialised cells that synthesize histamine and heparin. Mast cells are numerous in the papillary layer of the epidermis, in the beds of capillaries, in the reticular layer and in the areolar tissue that surrounds the cutaneous appendages .

Lymphocytes: have an important role in the immune reactions.

Plasma cells: form Immunoglobulins (Ig) ab's.

Eosinophils: inhibit the action of histamine.

Layer 3. The Subcutis (subcutaneous fat): Adipose tissue, large blood vessels, nerves.

Functions of the Skin:

Regulates body temperature. Prevents loss of essential body fluids, and penetration of toxic substances. Protection of the body from harmful effects of the sun and radiation. Excretes toxic substances with sweat. Mechanical support. Immunological function mediated by Langerhans cells. Sensory organ for touch, heat, cold, socio-sexual and emotional sensations. Vitamin D synthesis from its precursors under the effect of sunlight and introversion of steroids.

Facial Pores: Pores in the skin are for the hair follicles. The pore size is related to sebaceous gland size; pores enlarge to accommodate a greater oil flow. Oftentimes acne clears up when the pores enlarge so that sebum is no longer impeded from moving through the pore. The 'skin pore' usually applies to the visible topographic features at the skin surface corresponding to enlarged openings of pilosebaceous follicles. They appear as empty funnel-shaped structures or as cornified cylindrical plugs corresponding to comedones. Many endogenous and exogenous factors are known to cause enlarged pilosebaceous pores. Such factors include sex, genetic predisposition, ageing, chronic UV light exposure, comedogenic xenobiotics, acne and seborrhoea. Increased pore size was significantly associated with increased sebum output level, male sex and age (Size of Facial Pores. Br J Dermatol 2006;155:890-894).

Koebner's Phenomenon:

Koebnerization has been documented to occur between 3 days and 2 years after skin injury, although the average reaction time is 10 to 20 days. First described by the German dermatologist Heinrich Koebner in 1877. The isomorphic phenomenon, as it is also known, is the appearance of a skin lesion as a result of trauma. This phenomenon may occur in about one third of psoriasis pt's, hence its typical association with the disease. After injury to the skin, new psoriatic plaques may flare up at the site of injury or old plaques may spread. Although this response is seen more often in pt's with psoriasis, the phenomenon may also trigger lichen sclerosus et atrophicus, eczema, lichen planus, or vitiligo.

Fingerprints: the skin ridges on volar skin, did not evolve to make the tasks of the FBI easier. The traditional explanation of fingerprints' function is that they increase friction between the skin and grasped objects, decreasing the likelihood that we will drop a dime or a diamond. Neuroscientists also suggest that the ridges give enhancement of the sense of touch (Science 2009 Jan 29;e-pub ahead of print).....Pacinian corpuscles respond to vibrations generated by textured surfaces moving across the fingerprint ridges. Pachyderms one-up man in clever uses of the Pacinian corpuscles as corpuscles in their feet detect low-frequency (20–40 Hz) seismic waves produced by vocalizations and the rumble of the herd on the move, playing a part in elephant communication (J Anat 2007; 211:428).

Basic Skin Care:

See Emollients |

Dry skin is common from October through March. Facial moisturizers & body moisturizers are often needed if the skin is dry, the goal is to maintain the barrier. Eye creams are not needed, why buy tiny containers of very expensive product for just around their eyes, use what the same moisturizer one used on the face around the eyes. No cigarette smoking, no scrubbing or picking the skin. Use a non-drying, non-soap replenishing cleanser. Toners, astringents and clarifiers are excessively harsh and can lead the skin to be stripped. Pt's must have adequate UVA and UVB protection with sun blocks to prevent burn or tan.

Soaps & Cleanser:

The goal is to remove makeup, oil, dirt, and prepare for topical agents. One does not need to have squeaky-clean skin as this dries out the skin. Soap was invented sometime between 600 and 300 BC, but other than the Roman baths, people did not bath often. Throughout the later Middle Ages until the more modern era, soap remained a luxury product for the wealthy until the availability of NaOH in the compounding of soap reduced the cost and increased supply (SKINmed. 2005;4:183-185). Soap is designed to remove oily soils, dirt, sweat, and sebum from skin through the action of surfactants. Surfactants can bind to stratum corneum (SC) proteins, resulting in diminished ability of the SC to bind and hold water. During the 20th century, the marketing became more sophisticated in the promotion of these products, consumer use increased, accompanied by an increase in soap-induced skin irritation by over-drying the skin to promote skin barrier damage, leading to erythema and pruritis. Anionic surfactants (dissociation occurs in a negatively charged ion-anion) are often used in cleansers because of their excellent ability to foam and lather. While high-lathering soap is appealing to consumers, the irritation potential of soap is related to the alkalinity and fatty acid chain length (a palmitate is milder than a laurate) of the formulation. These can leave the skin with a "tight" feeling related to the removal of lipids from skin and

presence of residual surfactant on skin. Most body cleansers on the market today are synthetic detergent products and not true soap. The FDA does not regulate soap, unless a claim such as antibacterial, antiperspirant, or anti-acne effect is made on the label, in which case it is considered a drug.
Facial Soaps: the face skin is different than the rest of the body as it has more sebaceous glands and is constantly exposed to the elements. If have dry skin can use a superfatted soap (containing cocoa butter, cold cream or other moisturizers) or a nonsoap cleanser such as Cetaphil. The Cetaphil can be applied with a cottn both and does not require rinsing (this minimized the evaporative effects of water on the skin). Avoid overwashing the face.
Acne pt's: should never use abrasive products to clean their face. Abrasive sponges, loofahs and scrubs are extremely harsh and can damage hair follicles leading to an exacerbation of the acne. No "I" soaps (Ivory, Irish Spring)...as too drying. Use a non-medicated soap such as Cetaphil liquid Cleanser (non-foaming) or Aquanil liquid cleanser, both fight acne due to their pH balance. Acne Aid soap is likewise very gentle.
If mild inflammatory acne can try Panoxyl Bar 5% soap (although a bit drying) is a great first step (leave on for 30 seconds at least before rinsing..good on any acne prone body parts). Neutrogena acne soap (with Salicylic acid) is helpful for early comedonal and inflammatory acne.
Retinol containing moisturizers (Neutrogena healthy skin) is great for wrinkles and for early mild comedonal acne, as it is non-irritating and if used consistently, will have similar effects as low dose Retin A products.
Mild Bar Soaps:
Alpha-Keri. Basis glycerin. Basis superfatted. Cetaphil. Dove. Neutrogena dry skin. Nivea Crème. Oilatum. Purpose. Shepard's moisturizing.
Soap-Free Cleansers: Commonly used in the management of atopic dermatitis and xerosis.
Aquanil lotion: 8, 16 oz.
Cetaphil lotion: 4, 8, 16 oz.
Moisturel sensitive skin cleanser: 8.75 oz.
Glycerin soap (Neutragena—nondrying): 5.5 oz.
SFC lotion: 8, 16 oz.

Emollients (Lubricants):

Used to help reduce friction and prevent drying by leaving an occlusive smooth film on the skin. All a best applied immediately after washing to trap the moisture from the bath in the skin. Don't waste money on eye creams, these are just regular creams in small containers.
Bath Oils?: Applying emollients directly to the skin after bathing rather than adding oils to the tub is probably better and more cost-effective. For atopic dermatitis, it is probably more effective to rub on skin emollients directly to the skin rather than to soak in them (adding emollients / oil to tub baths) (Drug Ther Bull 2007;45:73)....bath oils make bathtubs slippery, clog septic fields, send oils into wastewater, and, perhaps, threaten the child who drinks bathwater.
There are 2 basic types.
1) Occlusives: Oil and water in various proportions. Coats the skin with a layer of oil to prevent evaporative water. Includes petrolatum, cocoa butter, lanolin, mineral oil, shea butter, waxes, cyclomethicone.
2) Humectants: contains additional ingredients that draw and hold water in the skin. Contains glycerin, glycerol, hyaluronic acid, panthenol, propylene glycol, ammonium lactate or urea. Ex Lac-Hydrin.
For Very dry skin: Safflower oil or vegetable shortening (Crisco), Bag Balm, Johnson's baby oil, cocoa butter, Vaseline petroleum jelly / cream, mineral oil, Elta Swiss Skin care, Blistex lip ointment. See **Xerosis** |
For Dry skin use: A and D ointment with Zinc Oxide. Chapstick lip balm. Eucerin Original moisturizing cream. Jergens Cream. Keri Lotion Deep Original. St Ives Swiss Formula. Ultra-Derm Lotion. Vaseline Intensive Care Healing.
For Normal to oily: Beta Care Cream / Lotion. Biore Hydrating Moisture. CAM Lotion. Droxy Cream. Gerber baby lotion. Johnsons baby lotion. Lubriderm Skin Therapy. Nutrogena Combination Skin Moisture. Corn Husker's Lotion.
For Normal to dry: Acid Mantle Cream. Aqua Care. Aquaderm. Caress Body Lotion. Curel Moisturizing lotion. OML Lotion. Gold Bond Lotion. Jergens Original Scent Lotion. Keri Lotion, Lubriderm Skin Therapy. Neutragena Lotion. Niva Body cream. Ponds Age Defying lotion / cream. Sarna Lotion.
Complex moisturizer ingredients not only retain transepidermal water but may also be able to correct specific deficits in barrier function (Arch Dermatol Res 2009;301:175)....20 healthy volunteers were randomly assigned to apply one of two moisturizers to one side of the body. One moisturizer was a complex formulation of 18 components; the other was a simple oily moisturizer containing mostly lipids, paraffin, and isohexadecane. The moisturizers were applied to one forearm and buttock, twice daily, while the other forearm and buttock served as untreated controls. After 7 weeks, the researchers measured transepidermal water loss, skin capacitance, and blood flow. They provoked irritation at the treated sites by occluding an application of 1% sodium laurel sulfate detergent for 24 hours; the degree of irritation was determined by measuring increases in transepidermal water loss and blood flow.....Transepidermal water loss increased at sites treated with the simple moisturizer (which was assumed to imply barrier damage), but it decreased at sites treated with the complex moisturizer. Sodium laurel sulfate was more irritating to the simple-moisturizer sites than the complex-moisturizer sites. The complex moisturizer decreased messenger RNA expression of cyclin-dependent kinase inhibitor 1A (CDKN1A) but did not affect other assessed genes. The simple moisturizer increased gene expression of involucrin, transglutaminase 1, kallikrein 5, and kallikrein 7, but not CDKN1A. Thus, moisturizers can modify the skin barrier and alter mRNA expression in skin, and some moisturizers actually cause barrier deterioration.
Shampoos: any brand is fine for most scalps. Dandruff is not caused by dry skin, but is due to scalp inflammation and thus these pt's need to shampoo more, not less. See Seb dermatitis. Baby shampoo is not strong enough to kill Pitysporum yeast that colonizes the scalp.
Skin types can be best categorized on the basis of four parameters:
1. Dry or oily. Skin that is dull gray-white in color, rough in texture, and characterized by an elevated number of ridges is considered xerotic or "dry".
2. Sensitive or resistant. Individuals with resistant skin can typically use any kind of skin care product without incurring adverse reactions.
3. Pigmented or nonpigmented.
4. Wrinkled or tight (nonwrinkled).

Combining these four parameters yields 16 possible skin types.
Other: Four commonly used moisturizers promoted skin cancers in mouse studies (applied 5 days a week, for 17 weeks with UV light twice a week for 20 weeks) (Investigative Derm. 2008;published online Aug. 14)....Dermabase increased the total number of tumors by 69%....Dermovan (a wholesale-only product discontinued in 2006) increased the total number of tumors by 95%....Eucerin increased the total number of tumors by 24%....Vanicream increased the total number of tumors by 58%....the authors state that the moisturizers did not cause cancer in the mice, that came from their early-life radiation exposure....But the creams did make skin cancers grow faster and more readily.....team asked Johnson & Johnson to make them a "custom blend" moisturizer without two ingredients previously linked to skin irritation (sodium lauryl sulfate) and tumor promotion (mineral oil).....The custom blend (on which Rutgers University and Johnson & Johnson hold a patent) did not promote skin cancer.....But not all of the products tested use these ingredients, so exactly what — if anything — might be linked to cancer isn't known.....And it's certainly clear that mouse and human skin are very different.

Morphology of Lesions:

Link: Color | Distributions | **Patterns** **|** Diagnostic Hints & Configurations **|** **Hallmark Rashes & Seasonal Variations & Emergencies** **|**
Describe the Lesion: Mnemonic: **LES T. CABS:**
Location & Distribution: Symmetrical, asymmetrical, sun-exposed, flexures/extensors, acral (hands/feet).
Erythema: Erythematous or nonerythematous lesions and/or underlying skin.
Surface Features: Crusting, rough, smooth, scaly, warty.
Type: Cyst, macule, papule, pustule, ulcer, vesicle.
Color: Blue, brown, pink, purple, red, white.
Arrangement: Single or multiple, discrete, unilateral, generalized, disseminated, grouped, annular, gyrate, dermatomal, linear, serpiginous.
Border & Shape: Well- or poorly defined, active edge, round, oval, irregular, pedunculated.
Special Sites/Systemic: Scalp, mouth, nails, genitalia, systemic disease, constitutional symptoms.
Estimating Size: use your own fingernail as a reference if you do not have a ruler handy. Most pinky fingernails are ~1cm across the mid-portion and the middle finger is 1.5 cm.

Primary Lesions:

<1cm: macule, papule, nodule, wheal, vesicle, pustule, purpura.
>1cm: patch, plaque, tumor, wheal, bulla, abscess, ecchymosis.

Secondary Lesions: scale, crust, erosion, ulcer, fissure, scar.

Special Lesions: comedo, cyst, burrows, telangiectasia.

Descriptive Terms:
Atrophy = thinning of the skin.
Sclerosis = scarring, thinning of the skin.
Pigmented = coloring in the lesion, tan/red/brown.
Crusts = impetigo, eczema, trauma (thermal, chemical, factitial), HSV, VZV, eczema herpeticum, pemphigus, stasis dermatitis, pyoderma gangrenosum, keratoderma blephorrhagica, syphilis, yaws.

Lesion Color:

Purple: purpura, vascular malformations, hemangioma, hematoma.
Blue: tattoo, vascular malformation, hemangioma, blue nevus, Mongolian spot.
Bronze: progressive pigmented purpuric dermatosis, resolving hematoma, phytodermatitis.
Green: tattoo, pseudomonas infection, deposition of minocycline, Mongolian spot, resolving hematoma.
Yellow: xanthogranuloma, xanthoma, sebaceous hyperplasia, epidermal inclusion cyst.

Distribution:

Light exposed--> SLE (lupus), drugs, seborrheic dermatitis , acne rosacea , acne vulgaris , acute contact dermatitis, arms.
Generalized--> drug, infection.
Palms and soles--> syphilis (palms), Rocky Mt. Spotted Fever, erythema multiforme.
Intertriginous--> Candida (perlèche), tinea, erythrasma.

Patterns in Children:

Flexural Rash: Atopic dermatitis, infantile seb derm, intertrigo, candidiasis, tinea cruris, inverse psorisis, epidermolytic hyperkeratosis (icthyosis).
Sun Exposed Areas: photo-toxic reaction (sunburn), photo-contact derm, lupus, polymorphus light eruption, viral exanthema, porphyria, xeroderma pigmentosum.
Clothing Covered sites: contact dermatitis, miliaria, psoriasis (summer).
Acrodermatitis: Below elbows and knees. Papular acrodermatitis (viral exanthema), acrodermaittis enterpathica, atopic dermatitis, tinea pedis with id reaction, dyshydrotic eczema, poststeptococcal desquamation. (Pediatric Dermatology, 3rd ed, by Cohen, 2005)

Diagnostic Hints to Consider if Uncertain:

Step #1: Is this a drug eruption? Systemic illness? (see **Signs of Systemic Dz**) Is this factitial? Mycosis fungoides? Occupational or hobbies? Sarcoidosis? Scabies? Syphilis? Lupus erythematosus?
Think of these broad categories?: Infectious (bacterial, fungal, parasitic, spirochetal, viral). Neoplastic. Inflammatory / Autoimmune. Allergic (contact dermatitis). Drug reaction. Metabolic. Genetic.
Did you examine / enquire about: Focus of infection? Foreign travel? Mucosal clues? Nail clues? Over-the-counter, herbal, or natural medicines? Pets? Scalp clues? Tinea pedis (Id reaction)? Cancer / Neoplastic risks? Did you really listen to the pt. Did you ask the pt what they think might be contributing or causing?
Consider Doing: A punch biopsy? Culture? Envision the lesion at another site? Order blood studies? Order imaging studies? Patch test? Perform a KOH?
Warning Signs & Pearls: Changing nevi need punch bipsies to exclude melanoma. A drug rash with blisters or erosions can be life-threatening. A febrile, young child with skin tenderness, flexural erythema and blisters may have staphylococcal scalded skin.

Generalized itching can be a marker of underlying systemic illness. Purpuric rash or nodules on the lower legs may be presentation of systemic vasculitis. Erythema involving 90% of the body surface can lead to death. Urticaria with respiratory symptoms can be life-threatening. Oral and genital mucous membrane ulcers may herald life-threatening disease. Do not forget dermatomyositis in elderly patients with weakness, malaise and photosensitive rash. Non-healing ulcers or crusty nodules may be malignant. Source: (Alarm Bells in Medicine by Nadeem Ali. 2005 by Blackwell Publishing Ltd)

Configuration of Dermatologic Lesions:

Acneiform: acne vulgarus, drug-induced acne (prednisone, Li, INH), Cushing's, chloracne.
Annular: ring shaped lesion. Circular or oval macule or patch, erythematous outer border with central clearing: tinea corporis or ringworm (most common adult cause). Pityriasis Rosea. Granuloma Annulare. Sarcoidosis. Leprosy (Hansen's Disease). Urticaria. Subacute Cutaneous SLE. Erythema Annulare Centrifugum. Erythema Chronicum Migrans (Lyme Disease). Erythema Multiforme. Psoriasis. Nummular Eczema. (Am Fam Physician 2001;64:289-96) (Pediatr Ann 1998;27:136-48). Acute annular urticaria is common in childhood, benign, and self-limiting, but the lesions can be misleading (Pediatrics 2007;119:e1177)...although annular urticaria is ring-shaped, it never forms the true target lesions of EM.
Bulla: raised, fluid-filled lesion >0.5cm.
Circinate: circular (circinate balanitis).
Comedone: Blackhead or whitehead; plugs of sebaceous material in follicle. Example: Acne.
Confluent: lesions fusing together (childhood exanthems).
Cyst: encapsulated material. Epidermal cyst. Trichilemmal (pilar or sebaceous cyst). Milium. Dermoid cyst. Mucous cyst.
Dermatomal: zosteriform (especially metastatic disease), vitiligo, nevus depigmentosus, Becker nevus, café-au-lait spot, port wine stain.
Discoid: disc shaped lesions w/o central clearing (Discoid Lupus).
Discrete: lesions separate.
Excoriations: linear crusts and erosions due to scratching. Scratch marks, causing thinning.
Eczematoid: tendency for erosions and crusting.
Figurate (Patterned): erythema, seen in urticaria, fungal infection, ECM, erythema marginatum, erythema annulare centrifugum, erythema gyratum repens.
Filiform (thread-like): wart, dermatosis papulosa nigra, syringocystadenoma papilliferum, skin tag, pigmented nevus.
Generalized: widespread over much of the body.
Geographic: psoriasis, geographic tongue, nummular eczema, erythema annulare centrifugum.
Grouped / Herpetiform: lesions clustered together (Herpes), zoster, HSV, dermatitis herpetiformis.
Hair in Mole: The presence of one or more hairs in a pigmented lesion does not guarantee that it is benign as presents 3 cases, terminal hair shafts in the presence of invasive melanoma (J Am Acad Dermatol 2007;56:413-416)....dispels the myth of the "benign hair sign," even though it is most likely a congenital melanocytic nevus.
Iris: target or "bulls eye" lesion. (Erythema Multiforme, Lyme).
Keratotic: thickened like a horn (Psoriasis).
Lichenification: (1) an accentuation of normal skin markings commonly associated with thickening of epidermis usually caused by scratching or rubbing. (2) marked by the presence of fine papules.
Linear: line shaped (poison ivy), seen in factitial, nevoid, Koebner phenomenon, autoinoculation, warts, icthyosis, psoriasis, porokeratosis, incontinentia pigmenti, epidermal nevi, contact dermatitis.
Macule: flat lesion, different color from surrounding skin. Suggests viral exanthem.
Maculopapular eruptions: Measles. Atypical measles. Rubella. Scarlet fever. Staphylococcal scalded skin syndrome. Staphylococcal toxic shock syndrome. Meningococcemia. Typhus and tick fevers. Toxoplasmosis. Cytomegalovirus infection. Erythema infectiosum (Parvovirus). Roseola infantum (HHV-6). Enteroviral infections. Infectious mononucleosis. Toxic erythemas. Drug eruptions. Sunburn. Miliaria. Kawasaki disease. Capillaritis (Schamberg's disease) |
Multiforme: more than 1 shape/form (EM).
Nikolsky's Sign: a disadhesion of skin assessed by light pressure at the edge of a preexisting bulla.
Nodule: raised, solid lesion >1cm diameter.
Papule: raised, solid lesion <1cm diameter. Elevated lesion. Example: Wart.
Patch: macule-like, but >1cm. Large macule, greater than 0.5 to 1 cm in diameter. Example: Vitiligo.
Papulovesicular eruptions: Varicella-zoster infections. Smallpox. Eczema herpeticum. Eczema vaccinatum. Coxsackievirus and other enterovirus infections. Atypical measles. Rickettsialpox. Impetigo. Insect bites. Papular urticarial. Drug eruptions. Molluscum contagiosum. Dermatitis herpetiformis.
Papulosquamous Disorders: papules and scaling plaques. A group of dermatoses with a morphologically characteristic primary lesion consisting of a papule, usually erythematous, that has a variable amount of scale on the surface. Plaques or patches form through coalescence of the primary lesions. **Ddx** included pityriasis rosea (PR), lichen planus (LP), seborrheic dermatitis, tinea corporis, pityriasis rubra pilaris, drug eruptions, tinea corporis, secondary syphilis, psoriasis, parapsoriasis and the cutaneous manifestation of AIDS.
Petechiae: A circumscribed deposit of blood less than 0.5 cm in diameter.
Plaque: a patch or small differentiated area of skin, usually raised. Papule-like, but >1cm.
Poikiloderma: a combination of atrophy, variegated hyperpigmentation/ hypopigmentation and telangiectasis.
Purpura: intradermal hemorrhage >3mm in diameter.
Reticulated: Lace-like (Erythema infectiosum), cutis mamorata, livedo reticularis, congenital phlebectasia, reticulated and confluent papillomatotis, erythema ab igne.
Scaling: Abnormal shedding or accumulation of the stratum corneum.
Sclerodactyly: tightness of the skin of fingers & atrophy of soft tissues (scleroderma).
Sclerosis: Induration of the skin, a hardening.

Serpiginous: Snakelike (Cutaneous larva migrans), psoriasis, erythema marginatum, elastosis perforans serpiginosa.
Telangiectatic: Dilated superficial blood vessels (Osler-Weber-Rendu).
Totalis: total body involved (CVD, alopecia totalis).
Vesicle: raised, fluid-filled lesion <0.5cm.
Wheal: Erythematous papule or plaque caused by upper dermal edema. Small, derma edema, <3 days.
Ulcer: Lesion that is deeper than an erosion, caused by breakdown of epidermis and dermis.
Zosteriform: dermatome distribution (HSV, zoster).
Papulovesicular Eruptions: rubeola, acne, varicella, coxsackie (hand-foot-mouth), dermatitis herpetiformis, drugs, HSV, impetigo, vaccinia, insect bites, molluscum, monkeypox, papular urticaria, rickettsialpox, shingles, yaws, smallpox.
Maculopapular Eruptions: HIV, adeno, arbovirus (dengue), rubeola, CMV, drugs, EBV, enterovirus (echo, coxsackie), parvovirus B19, HHV6, rubella, measles, meningococcemia, Kawasaki's, m. pneumonia, roseola, scalded skin synd, scarlet fever (Strep pyog), sunburn, rat-bite fever, reovirus, toxic erythemas, toxic shock, toxo, typhus, typhoid, vaccine reactions.
Referral to a dermatologist: recommended when the primary physician is unable to make a dx, **or** not equipped for **or** not successful with therapy. A dermatologist also is needed if patch testing is indicated.
Pearls: The experienced physician can often immediately diagnose some skin diseases on the spot without hesitation. However, the pt who spent his time and money to have the opportunity to reach the physician to treat his skin problem should have appropriate and appreciated care. He may be disappointed when he finds that his problem was solved within a few minutes especially when that was done in a rush and a non-convincing way. Need more care and more investigations to reach a thorough and convincing dx, otherwise the pt may seek additional opinions else ware.

Hallmark Rashes:

See above for Confiburation and Morphology |
By Location:
Facial: Acne vulgaris. SLE: butterfly rash, women. DLE: sharp marginated, on ears, lower lip, lower eyelid, nose. Sarcoidosis: red-brown papules on eyelid, nostril rim. Rosacea: flushed, perhaps pustular. Tuberous sclerosis: angiofibromas. Facial cosmetic area: cosmetics.
Face spared: Scarlet fever.
Palmar: Palmar erythema (cirrhosis). Meningococcus. Gonococcus. RMSF. 2° syphilis. Erythema multiforme ['target lesions'] (HSV, others).
Ring around scalp: tinea capitis, aka ringworm.
Single dermatome: shingles.
"Christmas tree" on back: pityriasis rosea.
Glove or shoe distribution: allergic reaction.
Between finger digits: scabies.
Diaper area: candida.
Gentials and lips: herpes.
Groin, down upper thigh: tinea curis.
Buttock and leg: Henonch-Schonlein purpura.
Shins: erythema nodosum [infection of fat layer] (OCP, pregnancy, sarcoid, strep, drugs). Capillaritis (Schamberg's disease) |
Foot: tinea pedis, aka athlete's foot.
Legs: erythema ag igne (heat rash).
Extensor side of elbows/knees, scalp, lumbosacral: psoriasis.
Flexor side of elbows knees: para-psoriasis, inherited eczema.
Hands, feet, mouth: hand-foot-mouth.
By Appearance:
Psilver Pscale over Psalmon Pskin: Psoriasis.
Honey crust: impetigo.
Rashes in all stages: chicken pox.
Like measles but finer, pinker: rubella.
Creamy white plaques, scraped to show raw skin: candida.
"Active edge": central clearing, but border of lesion shows scaling: ringworm.
Pinpoint petrochial: CMV.
Clear center "marginosum": rheumatic fever.
Central depression [punctum] in papule: molluscum contagium.
Purple, Peripheral, Pruritic Polygons: lichen Planus.
Dark blue edge around purulent ulcer: pyoderma gangrenosum.
"Carpet tack" when remove skin: DLE.
Cafe-au-lait: Neurofibromatois. Tuberous sclerosis .
Days after fever onset, that rash appears: Rubella. Scarlet fever/ Smallpox. Chickenpox. Measles [with Koplik spots 1 day earlier]. Typhus, rickettsia [variable]. Enteric fever [salmonella]. Roseola: rash appears when fever disappears.
Spread:
Bullseye: Lyme dz. **Centrifugal:** Typhus.
Centripedal: RMSF. **Forehead first:** Rubella: forehead to face to torso to extremities.
Head-to-toe: Measles: both appears, and disappears from head to toe.
Cheeks first: Fifth disease [erythema infectiosum]: "slapped cheeks".
By pre-rash history:
Ate chicken: salmonella. **Bit by tick:** RMSF. **Bit by flea:** typhus.
Born with rash: ToRCHeS: Rubella. CMV. Herpes. Syphilis.
Child: Listeria. Measles. Mumps. Ruebella. Etc.

Very sexually active: Gonococcus. Herpes. Neisseria meningitis. Syphilis.
By sx: **Chorea:** rheumatic fever. **Condyloma lata:** syphilis.
Koplik's spots: measles. **Gross splenomegaly:** EBV [others also].
Nail pitting, onycholysis: psoriasis. **Strawberry tongue:** scarlet fever.
Fluoresce in UV light: **Yellow glow:** pityriasis versicolor. Tinea capitis, aka ringworm.
Always consider in DDx: Contact eczema. Atopic eczema. Drug reaction, esp. penicillins. Allergies. Heat rash. Histiocytosis X.
Peripheral cyanosis: Cold. Obstruction. LVF and shock. Decreased cardiac output.
Central cyanosis: Polycythemia. Altitude. Lung dz. Met-, sulphaemoglobinaemia. Shunt.

Seasonal Variations in Skin Diseases:

Certain dermatoses have an increased incidence in various seasons of the year.
Winter: Atopic eczema. Contact dermatitis of hands. Psoriasis. Seborrheic dermatitis. Nummular eczema. Winter itch and dry skin (xerosis). Ichthyosis.
Spring: Pityriasis rosea. Erythema multiforme. Acne (flares).
Summer: Contact dermatitis due to poison ivy. Tinea of the feet and the groin. Candidal intertrigo. Miliaria or prickly heat. Impetigo and other pyodermas. Polymorphous light eruption. Insect bites. Tinea versicolor (noticed after suntan). Darier's disease (uncommon). Epidermolysis bullosa (uncommon).
Fall: Winter itch. Senile pruritus. Atopic eczema. Pityriasis rosea. Contact dermatitis due to ragweed. Tinea of the scalp (schoolchildren). Acne (flares).

Dermatologic Emergencies:

Vesiculobullous Disorders: Stevens-Johnson syndrome, toxic epidermal necrolysis, pemphigus vulgaris, other generalized blistering disorders (bullous pemphigoid).
Infections: Cysticercosis cutis, hemorrhagic fevers, leprosy (reversalreaction upon tx) mucormycosis, necrotizing fasciitis & other gangrene (gas gangrene, progressive bacterial synergistic gangrene), neonatal herpes simplex, Rocky Mountain spotted fever, Still's disease, trichinosis, tularaemia.
Autoimmune Disorders: Acute eruption, systemic and neonatal lupus erythematosus & dermatomyositis.
Inflammatory Cutaneous Disorders: Acne fulminans, acute drug eruptions (e.g., red man syndrome), acute pustular psoriasis, capillary hemangiomas (affecting respiratory tract or oral cavity, periorbital), exfoliative erythroderma, Kawasaki syndrome, pyoderma gangrenosum, scarring alopecias ("hair emergency").
Environmental: Burns, child abuse, cholesterol emboli, sclerema neonatorum.
Presentation that have a Concerning Ddx:
1. Generalized red rash with fever: Bacterial infections with toxin production. Drug eruptions. Rickettsial exanthems. Viral exanthems.
2. Generalized red rash with blisters and prominent mouth lesions: Bullous pemphigoid. Drug eruptions. Erythema multiforme (major). Toxic epidermal necrolysis.
3. Generalized rash without redness with blisters, erosions, and mouth lesions: Pemphigus.
4. Generalized red rash with pustules: Drug eruptions. Pustular psoriasis.
5. Generalized rash with vesicles: Disseminated herpes simplex. Drug eruptions. Generalized herpes zoster. Varicella.
6. Generalized red rash with scaling over whole body: Exfoliative erythroderma.
7. Generalized wheals and soft tissue swelling: Urticaria and angioedema.
8. Generalized purpura: Drug eruptions. Purpura fulminans. Thrombocytopenia.
9. Generalized purpura that can be palpated: Bacterial endocarditis. Vasculitis.
10. Multiple skin infarcts: Disseminated intravascular coagulopathy. Gonococcemia. Meningococcemia.
11. Localized skin infarcts: Antiphospholipid antibody syndrome. Atheroembolization. Atherosclerosis obliterans. Calciphylaxis. Warfarin necrosis.
12. Facial inflammatory edema with fever: Erysipelas. Lupus erythematosus.
(Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology, edition 5 by Wolff, McGraw-Hill)

Topical Corticosteroid Use:

Links: Amount Needed | **Topical Vehicles & Absorption** | Potency & Dosing | **SE's** | Suggested Strengths | **Class I (Super Potency)** | Class 2 (Very High Potency) | **Class 3 (High Potency)** | Class 4 (Medium Potency) | **Class 5 (Moderate Potency)** | Class 6 (Mild-Mod Potency) | **Class 7 (Low Potency) & COMBO agents** |

Inexpensive options:
Clobetasol 0.05% oint / cream (class 1), 15g @ $11, 30g @ 15. or Solution, 25ml @ $27.
Betamethasone valerate 0.1% oint (class 2) / cream (class 5) [15g tube ~$8, 45g @ $16].
Betamethasone dipropionate 0.05% oint [15g @ $10], cream [15g @ $10, 45g @ $12] lotion [15g @ $10].
Triamcinolone 0.1% (class 4), AAA BID, $3.50 for 15g, $9 for 45g, $15 for 454g jar.
Fluocinolone 0.025% (class 4) oint/cream, 15g @ $12, 60g @ $19.
Solution: Fluocinolone 0.01%, 20ml @ $11, 60ml @ $14, 60g cream $15.
In a pinch or at a free-clinic, can get some relief using a nasal steroid spray on an eczematoid patch or inflamed seborrheic dermatitis. There are many topical corticosteroids are available with variable potency and most are available in generic formulations, but unfortunately, there is a paucity of clinical trial data to assist in choosing a corticosteroid.

Estimation of Amount Needed:

Hand Size = 1% BSA = 0.25g cream/ ointment. (4 hand areas = 1g = 10X10cm area). One fingertip (0.5g) pea-size glob covers both sides of the hand.
Face = 2g/ application. If give BID X 7d ~= 30g (1oz).
Arm or one side of trunk = 3g. BID X 7d = 45g (1.5oz).
Leg = 4g. BID X 7d = 60g (2oz). BID X 30d = 180g (6oz).
Whole body = 30-45g BID X 7d = 450g (=1 lb), BID X 30d = 5lb (#). For a 10 day BID tx need 100g for infant. 150g for child. 300g for adult.

Can use: Triamcinolone 0.1% (class 4) $15 for 454g jar. **Or** Take a 50 ml bottle of Temovate solution and stir into a 1 pound (1/2 kg) jar of cereve cream and apply BID.
The fingertip unit (500 mg): an easy way to estimate how much topical corticosteroid cream/ointment a patient should use. A fingertip unit is the amount of cream or ointment, squeezed out of a standard-sized tube, that covers from the tip to the first crease of the finger (Topical corticosteroid formulations. Prescriber's Letter 2009;25(12):251210).
One fingertip unit is sufficient to cover both sides of an adult hand, with the fingers held together. Below are some more estimates on the amount of body surface that a specific number of fingertip units can cover:
* 2 fingertip units cover one adult foot.
* 2.5 fingertip units cover an adult face and neck.
* 3 fingertip units cover one adult arm.
* 6 fingertip units cover one adult leg.
* 7 fingertip units cover the adult trunk, either front or back.

Topical Med Vehicles and Absorption:

…….END DEMO of this chapter.

Endocrinology:

Edited by Lois Jovanovic, MD, Endocrinologist and Diabetologist.

Contents: Lipids | **Pituitary** | Parathyroid | **Adrenal** | Steroids | **Aldosterone** | Diabetes Mellitus | **Hypoglycemia** | Obesity | **Hirsutism** | Thyroid | **D. Insipidus** | Gynecomastia | **Various & MEDS** | Osteoporosis | **Male Hypogonadism** | Growth & Puberty | **Inborn Errors of Metabolism** | Metabolic Syndrome-X & Hyperglycemia | **Metabolic Bone Dz** | PCOS | ***INDEX*** |

Functions: Regulate cellular activity for: 1. **Metabolism.** 2. **Growth.** 3. **Development.** 4. **Homeostasis.** 5. **Reproduction.**
Hormones: Chemicals derived from amino acids, lipids (e.g., steroids) and peptides are produced by and released from cells and trigger a response in same or other cells by binding to receptors (located inside or outside of cell).
Three major types:
1. **Local hormones (cytokines):** Released into interstitial fluid (e.g., prostaglandins, histamines, growth factors).
a. **Autocrine:** Same cell is affected.
b. **Paracrine:** Neighboring cells affected.
2. **Neurotransmitters** (*Chemical Synapses*).
3. **Circulating hormones**: Released into blood and transported to cells throughout body.
Endocrine Glands: Although every cell may release hormones, certain areas of the body serve as principal endocrine glands that release circulating hormones, resulting in numerous, complex responses by target cells. Hormone actions in general are regulated by **negative feedback systems**.

Cholesterol (Lipid Disorders):

Links: Intro & ICD9 | **Formulas & Ratios** | Goals | **Tx Strategies** | Dietary | **Bile Acid Sequestrant** | Ezetimibe (Zetia) | **Statins** | Niacin | **Fibrates** | Combo Meds | **Arteriosclerosis** | Fredrickson Classification & Skin Findings | **Ddx Lipid Components** | Lipoprotein / Chol Sub-types & & Advanced Lipidology | **Homocysteine** | Metabolic Syndrome-X | **Elderly pt's** | Children & Adolescents | **Omega-3 fatty acids (Fish oil)** |

High cholesterol generally means elevated LDL. High **triglycerides** means elevated chylomicrons, VLDL or IDL. Dyslipidemia is one of the leading treatable causes of lost healthy life world-wide (Lancet. 2002;360:1347–1360). The prevalence of hypercholesterolemia in the USA adult population is 27% (JAMA 1993;269:3009-14). National surveys indicate that 40% of the people in the US between ages 20-74 years have borderline or greater LDL cholesterol,8 and 17-19% of individuals have high LDL cholesterol (Circulation. 2006;113:e85–e151).The prevalence rate of hypercholesterolemia (total chol >240 mg/dL or >6.2 mmol/L) is 17% in the US and Canada (NEJM 2008;358:1842). Adjusting for baseline prevalence of dyslipidemia, the 30-year risks for a 50yo exceeded 80% for borderline-high LDL, 50% for high LDL, and 25% (women) to 65% (men) for low HDL (Am J Med 2007;120:623-630.e1), 20-50% had or developed a low HDL along with a high LDL level. Coronary heart disease is a leading cause of death in the US, accounting for more than one in five deaths each year, or ~ 500,000 fatalities. Of 6704 asymptomatic subjects aged 45 to 84 years in N. Carolina, the overall prevalence of dyslipidemia was 29.3%, and only 54% were taking lipid-lowering drugs, and of those receiving tx, only 40.6% were controlled to goal (Circulation 2006;113:647-656). ....based on the proven benefits of lipid-lowering therapy for primary prevention, dyslipidemia management should be considered among our highest national healthcare quality improvement priorities. A long-term study on 3277 healthy Finnish businessmen found that low cholesterol levels in middle age (30-45yo) predicted lower mortality and better physical quality of life 39 years later (Cardiol Rev 2006;23:13-19)(each mmol/L increase of baseline Tchol was associated with an increased total mortality risk of 11%). Cholesterol levels are closely linked to cardiovascular mortality, especially among middle-age pt's, but not to stroke-related deaths (Lancet 2007;370:1829-39)...For every 1 mmol/L decrease in cholesterol, ischemic heart disease mortality dropped by half among pt's ages 40 to 49, by a third among those 50 to 69, and by a sixth among those 70 to 89 according to a review of observational studies. An analysis of 61 prospective observational studies has failed to find any association of total cholesterol with stroke mortality (Lancet 2007;370:1829-1839). A 10-year analysis of offspring patients in the Framingham Heart Study suggests improvements in lipid profiles, particularly beneficial changes in triglyceride and HDL-cholesterol levels despite an overall increase in BMI (Arch Intern Med 2009;169:279-286).....The findings are counterintuitive....Researchers say they have no certain explanation for these beneficial changes in blood lipid levels....one theory might be based on changes in patterns of food consumption in the US. Recent dietary surveys suggest increased consumption of carbohydrates and a decrease in the consumption of fats, particularly saturated fats. The emergence of the summer sun after a long winter appears to have a positive effect on lipid levels an analysis of data from the PROVE IT-TIMI 22 Study has shown (Am J Cardiol 2009;103:1056-1060).....LDL-cholesterol levels are significantly lower (by 6 mg/d) in summer than in winter.

ICD-9 Codes: 272.5 lipoprotein deficiencies, 272.0 pure hypercholesterolemia, 272.3 hyperchylomicronemia, 272.5 lipoprotein deficiencies, V77.91 screening for lipoid d/o's.
How Often Do We Need to Check Cholesterol Levels: Adherent patients with well-controlled cholesterol levels could be monitored every 3 to 5 years (Ann Intern Med 2008;148:656) (average test variation = 7%)….many apparent increases in cholesterol level may be false positive…takes 3 yrs before plausible changes in true cholesterol values could be comparable with the short-term variation (before signal equals noise).
Note: all units in mg/dL to convert mg/dL to mmol/L, you multiply by 0.02586.
Lipids that can be measured without fasting: Total cholesterol and HDL cholesterol. LDL cholesterol is calculated from the fasting triglyceride level and total and HDL cholesterol levels are calculated.
Cholesterol Ratio: Ratio of total cholesterol to HDL cholesterol: TC/HDL. The goal is to keep the ratio <5:1; the optimum ratio is 3.5:1 (>3.5).
The AHA recommendation is to use the absolute numbers for total blood cholesterol and HDL cholesterol levels rather than the ratio in determining the appropriate tx for pt's (http://www.americanheart.org/presenter.jhtml?identifier=4503). The total cholesterol/HDL cholesterol and apoB/apoA-1 ratios were also strong predictors of ischemic heart disease in all men, and these two lipid ratios were the strongest lipid predictors in men without cardiovascular disease (Arch Intern Med. 2007;167:1373-1378)…Blood lipid levels, other than total cholesterol, are associated with ischemic heart disease in older men…There was no association between total cholesterol and mortality in the overall cohort, total cholesterol levels were positively associated with IHD mortality in men without prior cardiovascular disease. See **Apolipoproteins** |
Triglyceride–HDL Ratio and Insulin Resistance: Trig/HDL: A good surrogate markers for identifying insulin resistance in overweight pt's (Ann Intern Med. 2003;139:802-809)…..The optimal cut-points were 1.47 mmol/L (130 mg/dL) for triglyceride, 1.8 in SI units (3 in traditional units) for the trig–HDL ratio. Trig / HDL >3 is consistent with insulin resistance. In African Americans, TG levels and TG-HDL-C ratio are not reliable markers of insulin resistance (Arch Intern Med 2005;165:1395-1400). According to an analysis of 9,500 data sets apo-B/HDL ratio emerged as the strongest correlate (odds ratio 1.177 per 1 mg/dl increment), followed by the total chol or non–HDL /HDL ratio (odds ratio for each 1.070 per 1 mg/dl increment), followed by the trig/HDL (odds ratio 1.033 per 1 mg/dl increment (A Population-Based, Cross-Sectional Comparison of Lipid-Related Indexes for Sx's of Atherosclerotic Disease. Am J Card 2006;published online 29 August 2006)....neither LDL cholesterol nor the LDL/HDL cholesterol ratio correlated significantly.
LDL:HDL ratio: A study with 9,770 pt's found that those with the lowest LDL:HDL ratios (<1.33) experienced fewer than half as many CV events as those with the highest ratios (2.41) (NEJM 2007 Sep 27; 357:1301).
Formulas:
Total Chol (=TC) = HDL+LDL+VLDL.
Trig/5 =VLDL.
LDL = TC - (HDL+TG / 5). Aim for <130 mg/dL (3.36 mmol/L). For most pt's, total cholesterol of 240 and 200 mg/dL correspond roughly to LDL of 160 and 130 mg/dL. A non-fasting sample may have higher trig's and thus falsely lower LDL as well. If the triglyceride values are higher than 4.52 mm/L (> 400 mg/dL), the LDL-C value will be even less accurate. Direct measurement of LDL in a specialized lab, using ultracentrifugation, may be necessary when significant hypertriglyceridemia persists despite fasting.
Non-HDL cholesterol = **Total Chol –** HDL. Non-HDL-C = the sum of all the cholesterol in the proatherogenic lipoproteins, and apoB, the major apolipoprotein of very low-density lipoprotein, intermediate-density lipoprotein, and LDL particles. This includes all cholesterol present in lipoprotein particles considered to be arthrogenic (LDL, lipoprotein(a), IDL and VLDL) and is a better predictor of CVD mortality, aim for <190mg/dL (Arch Int Med 2001;161:1413). Non HDL-C and the ratio of Total-C to HDL-C were as good or better than apoliporotein fractions in predictiong future cardiovascular events in 15,632 US women (JAMA 2005;294:326-33). Non-HDL cholesterol accounts for LDL cholesterol plus other atherogenic particles but does not require a fasting sample, suggests that non-HDL cholesterol may be at least as useful as LDL cholesterol to initially screen pt's for risk of a first nonfatal MI (Am J card 2005;96:1129-1134). Triglycerides and non-HDL cholesterol correlated with large-artery atherosclerosis in a cohort of patients with stroke or TIA (Neurology 2008;70:841)....These new findings suggest that, for patients with stroke or TIA, we should consider other elements of the lipid profile, including triglycerides and non-HDL cholesterol; the latter encompasses all apolipoprotein-B–containing particles. **See below.**
Combined hyperlipidemia: defined as increased LDL, decreased HDL, and increased triglyceride levels.
Major Cardiac risk Factors: M≥45, F≥55, HLD <35, current smoking, HTN, DM, FHx premature CAD (AMI/sudden death 1st Male relative <55 or 1st Female relative <65yo), homocystinemia.
Minor: Obese, sedentary lifestyle, inc Trig's, Lp(a)-small dense LDL, passive smoking, depression, calcifications in the aortic arch. Anticardiolipin Ab's, other (see Cardiology Chapter).
Pt Explanation of Cholesterol:
HDL= "**H**igher Degree of Living" as a protective "**h**elmet" or "**h**ealthy chol", removes cholesterol from the body by accepting cholesterol from the periphery and delivering it to the liver (reverse's cholesterol transport).
<40 mg/dL = low HDL cholesterol. A major risk factor for heart disease.
40 to 59 mg/dL is better, the higher your HDL level, the better.
≥60 mg/dL = optimal, considered protective against heart disease.
LDL= "**L**ower Degree of Living" as **l**ethal / **l**ousy cholesterol. Transports cholesterol from liver to all peripheral tissues.
A decrease of 6% give a decrease of 12% CAD risk.
<100 mg/dL = Optimal.
100-129 mg/dL = Near optimal / above optimal.
130-159 mg/dL = Borderline high.
160-189 mg/dL = High.
≥190 mg/dL = Very high.
Triglyceride Level Category
<150 mg/dL = Normal.
150-199 mg/dL = Borderline high.
200-499 mg/dL = High.
≥500 mg/dL = Very high. See **Triglyceride** |

Risk of Coronary Heart Disease and Total Cholesterol: (Arch Int Med 2003;163:1966-72).
<200 mg/dl (5.2 mmol/L): 31% lifetime (at age 40-80yo), 3% 10yr cumulative risk if male, 1% if female.
200-239 mg/dL (5.2-6.19 mmol/L): 43% lifetime, 5% 10yr risk if male, 2% if female.
>240 (6.2 mmol/L): 57% lifetime, 12% 10yr risk if male, 5% if female.

Other info: Only trig's and calculated LDL require a pt to be fasting for 12hr. Elevated LDL, Trig, dec HDL, are all independent cardiac risk factors. 35% CAD still occurs in those with Chol <200, thus consider tx even if <200 but have risk factors. 70% of diabetics have inc chol & trig. Tx gives a 30% dec mortality, tx all DM with statin first. Low chol does not protect against smoking related CAD (JAMA 1999;282:2149). C-reactive protein better than LDL cholesterol in predicting first cardiovascular events (NEJM 2002;347:1557-1565,1615-1616). Total chol has a seasonal variation of 4-5 mg/dL (0.1-0.14 mmol/L), with a peak in the winter mo's and greater amplitude in women and those with hypercholesterolemia (Arch Int Med 2004;164:863-70) (related to fat intake, physical activity, mood, light exposure). Analysis from the Women's Health Study confirms that high levels of non-HDL-C > total cholesterol > HDL-C, and LDL-C are significantly associated with an increased risk for ischemic stroke, even in women who are otherwise healthy (Neurology. 2007;68:556-562). An analysis of two studies (Treating to New Targets [TNT] and Incremental Decrease in End Points through Aggressive Lipid Lowering [IDEAL] trials) that contributed to reducing LDL-cholesterol levels to lower and lower targets has shown that on-treatment levels of non–HDL cholesterol and apolipoprotein B were more closely associated with cardiovascular outcomes than levels of LDL cholesterol (Circulation. 2008;117:3002-3009)...."Demonstrating the superiority of non-HDL cholesterol or apoB over LDL cholesterol as a cardiovascular risk predictor during statin treatment".....'When you use LDL cholesterol, you don't take into account the intermediate-density particles, things that have more triglycerides in them but still have a lot of cholesterol"....Compared with individual lipoprotein cholesterol values, the total/HDL cholesterol ratio, and the apolipoprotein B/A-I ratio in particular, were each more closely associated with cardiovascular outcome. This was true when a composite endpoint of coronary death, nonfatal myocardial infarction, resuscitation after cardiac arrest, and fatal or nonfatal stroke, or an endpoint of myocardial infarction alone, was used.

Lipid Tx Goals:

To convert mg/dL to mmol/L, you multiply by 0.02586. See **Tx Strategies** |

Risk factors: cigarette smoking, HTN (BP ≥140/90 mm Hg, even if treated), low HDL, FHx of premature CHD, and age (men >45 years, women >55 years).
Risk Calculator: http://hin.nhlbi.nih.gov/atpiii/calculator.asp

Lower risk:

Without CAD & <2 risk factors = people with zero or one risk factor.
LDL goal <160 mg/dL, TC <240.

Moderately risk:

Without CAD & ≥2 risk factors and a ten-year risk of CHD between 10-20% (www.nhlbi.nih.gov/guidlines/chol).
LDL goal is <130 (3.35 mmol / L)...but you're encouraged to get pt's down to <100 mg/dL (2.6 mmol/L).

High risk:

+ h/o CAD or CAD risk equivalents (diabetes, AAA, PVD) **or** >20% 10-years risk equivalents of CAD for coronary events (www.nhlbi.nih.gov/guidlines/chol).
LDL goal is <100 (2.6 mmol/L)...with encouragement to go to <70 mg/dL. TC (total chol) <200 (2.25 mmol / L). HDL >40 mg/dL. Add 1mg folate qd. Non-HDL Chol <130 mg/dL if TGs >200 mg/dL (Circulation 2006;113:2363-72). Pharmacologic therapy is recommended for all people at high risk. Even if age >65yo, so long as in good health (Clinical Geriatrics 2002;10:9).

Very high risk:

Heart disease plus multiple or poorly controlled risk factors.
LDL goal of <70 mg/dL (1.80 mmol / L). For people at very high risk of MI or death. Very high risk individuals are those with CAD + diabetes, persistent cigarette smoking, poorly controlled HTN, or multiple risk factors of the metabolic syndrome (high trig, low HDL, obesity), and those who recently had an AMI (Circulation 2004;110:227-39). There is no high-quality clinical evidence supporting the recommended tx target of ultralow LDL levels (<70 mg/dL) in high-risk pt's (*Ann Intern Med* 2006; 145:520-530). Dropping LDL from 100 to 70 in 100 pt's for 5 years prevents 1.5 events. Statins used in pt's with extremely low LDL cholesterol levels (<40 mg/dL) and those without documented CAD are safe and may lead to improved survival (Circulation 2007; 116:613-618). Researchers suggest, however, that treating to LDL-cholesterol levels to between 40 and 60 mg/dL confers even more benefit than <70 mg/dL (Circulation 2008;118:672-677)..... proponents of measuring apoB, as it is a surrogate of the number of atherogenic particles, including VLDL and LDL, rather than LDL, which is a surrogate for the cholesterol concentration of the LDL particle.

American Heart Association Guidelines for Women:

1) Initiate statin tx for high-risk women regardless of their LDL levels. Strongly consider if low risk women with LDL ≥190 mg/dL.
2) Maintain an optimal level HDL >50 mg/dL.
3) Initiate niacin or fibrate therapy for low HDL or elevated non-HDL chol levels in high-risk women.
*** High-risk women are those with a 20% risk of developing cardiac disease in 10 years. Intermediate-risk women have a 10-20% risk. Low-risk women have a <10%. (AACE 14th Annual Meeting and Clinical Congress: General Session. Presented May 19, 2005)

Intensive Therapy (Low LDL's): Current recommendations are stillto begin statin therapy at a lower dose and titrate upward, as on average, doubling the dose only reduces LDL levels by an additional 6%.....Neither absolute LDL level nor percentage decrease in LDL appears to be linked with the risk of myopathy or rhabdomyolysis in statin treated pt's (The safety of aggressive statin therapy. Mayo Clin Proc 2006;81:1225-31). Pt's hospitalized for an acute coronary syndrome who were treated with an intensive (80mg, high-dose atorvastatin for a median LDL <62 mg/dL), compared with a moderate (40 mg pravastatin), lipid-lowering statin, showed lower risk of major cardiac events or death from any cause (16% reduction in the hazard ratio) (NEJM 2004;350). Intensive lipid-lowering therapy (LDL goal <80 mg/dL) with atorvastatin (80mg) is more effective than conventional therapy (LDL average was 109) with pravastatin (40mg) at slowing the progression of coronary atherosclerosis (JAMA 2004;291:1071-1080,1132-1134), at 18-mo's, 2.7% of pt's in the pravastatin group had experienced atherosclerosis progression compared with none of the pt's in the atorvastatin group as measured by intravascular U/S, according to the results of the Reversal of Atherosclerosis with Lipitor (REVERSAL) study (also had a 36.4% reduction in CRP compared to 5.2%). More aggressive lowering of LDL (<70 mg/dL) benefits pt's with stable coronary heart disease in terms of risk for stroke and MI (NEJM 2005;352:1425-35) (overall mortality did not significantly). In pt's with prior AMI, high-dose atorvastatin (80mg) was better than standard-dose simvastatin (20mg) as had an

additional 20% reduction in stroke and fewer major cardiovascular events (HR, 0.87) without increased morbidity (The IDEAL study. JAMA. 2005;294:2292-2294) (average LDL 81 Vs 104 mg/dL)…..low dose statins prevent heart attacks and cardiac death, but one additional cardiovascular event is prevented for every 15 high-risk pt's treated for 5 years with high-dose statins. A retrospective analysis of 1800 pt's suggests that intensive lipid-lowering therapy to achieve very low LDL levels is safe and effective (lower-LDL pt's had fewer cardiac events with no difference in liver, muscle, or retinal abnormalities) (J Am Coll Cardiol 2005;46:1411-6) (30% had and LDL 40–60 mg/dL, and 11% had levels of <40 mg/dL). Even pt's with an ACS who have an LDL cholesterol level <80 mg/dL who are not on a statin at hospital admission benefit from initiating statin therapy at discharge based on a retrospective study with 155 pt's (Am J Cardiol 2005;96:1491-1493) (had a lower incidence of death, reinfarction, or stroke at six mo's (29% vs 9.5%; p = 0.005) compared to those pt's who were not discharged on statin therapy. In elderly pt's >65yo, a statistically significant decrease in positive affect (happiness, contentment, energy) emerged in the simvastatin group (if Tchol <148 mg/dL) compared with the placebo group (J Am Geriatr Soc 2006;54:70-6).....aggressive cholesterol lowering might result in subtle affective changes in older adults. Rosuvastatin (40 mg) can regress the atherosclerosis burden (IVUS-documented regression) when get LDL 60.8 ± 20.0 mg/dL (JAMA. 2006 Posted online March 13, 2006) (effect on hard cardiovascular end points pending). Intensive statin therapy (LDL <70 mg/dL) lowers the risk of heart failure hospitalizations by 45% (1.6% Vs 3.1%) in pt's after acute coronary syndrome more than moderate statin therapy does (J Am Coll Cardiol 2006;47:2326-2331). A meta-analysis of 12 RCT's of aggressive lipid lowering found that the relative risks for major adverse coronary events were reduced similarly, by 21% in diabetic pt's and by 23% in nondiabetic pt's (BMJ 2006;332:1115-8)....unfortunatley 70% of adverse cardiovascular events will still occur. A meta-analysis of these four studies showed high-dose statins provide a 16% odds reduction for coronary death or heart attack or any cardiovascular event (J Am Coll Cardiol 2006;48:438-45) (NNT to prevent one cardiovascular event was 29 pt's for 2 yrs)….a trend toward decreased cardiovascular death.

5 Key Lipid Lowering Strategies:

The goal is to treat atherogenic dyslipidemia (ultimately prevent CAD and CVA's), not simply to treat excursions from normative lipid subfraction levels.

#1. Exclude secondary causes (DM, thyroid, nephrotic, liver dz), see below.

#2. Therapeutic Lifestyle Changes: Links: **Dietary** | Functional Foods | **Children and Elderly pt's** | The cumulative effect of multiple therapeutic lifestyle changes may yield up to a 30% reduction in LDL-C and result in a significant increase in HDL-C (J CMS 2006;1;5:308-312). This included engaging dietitians/ nutritionists and diabetes educators in combination with promoting physical exercise.

Exercise: frequency more important than intensity for increasing HDL (Am J Card 2001;87:942). The amount of exercise (total time) might be more important than the intensity of exercise in favorably influencing lipoprotein measures among overweight, sedentary adults (NEJM 2002;347:1483-92).

#3. If inc LDL>20-25% & Trig <500 **or** secondary prevention start with a **Statin**. Statin therapy is associated with regression of coronary atherosclerosis when LDL is reduced to <87.5 mg/dL (2.3mmol/L) combined with an HDL increase of >7.5% (JAMA 2007;297:499-508). Repeat lipids and check LFT's 0, 6, 12wks, then q6-12mo, if profile not improved (<15% dec LDL or inc HDL) **-->** *change drugs* or if HDL still <35 and need a second agent**--> add Niacin.**

If HDL >35**--> add bile sequestrant** first, then niacin PRN as 3rd line. Aim for an HDL of 45mg/dL in men, 55 if women. Current guidelines for the prevention of coronary heart disease focus on lowering LDL-C as the primary target of lipid-modifying therapy (Low HDL-C: A secondary target of dyslipidemia therapy.Am J Med 2005;118:1067-1077).....statins are recommended as first-line drug therapy because of their LDL-C lowering efficacy.

#4. If inc LDL & Trig 200-400. Start a statin, may need to add nicotinic acid/ fibric acid. Or Nicotinic acid**-->** statin/ fibric acid.

#5. Primary Prevention (35-75yo) **-->** Niacin or Bile Acid sequestrant, Statin.

Compliance: Many pt's with high cholesterol don't take their statin medication as prescribed (Pharm J 2004:272:23-26). The risk of dying from a non-cardiovascular cause was nearly 4X higher in the non-compliant group. Compliance-enhancing initiatives such as regular cholesterol monitoring, is a statistically significant predictor of pt compliance. Simple ways of informing patients about their cardiovascular risk (concept of "cardiovascular age" = the gap between their actual age and "cardiovascular age") via sharing a personalized coronary risk profile with patients can make them more likely to adhere to medical advice and reach lipid goals, a study suggests (Arch Intern Med. 2007;167:2296-2303)……(http://www.myhealthcheckup.com/CVDAssessement/CVDTest.aspx).....This information may also assist physicians in treatment selection while improving pt adherence.

Elderly:

Elderly men and women who have higher LDL cholesterol levels are associated with decreasing total mortality risk in women and decreased risk of fatal heart failure in both genders (J Am Geriatr Soc 2005;53:2159-2164). Low total serum chol (<189 mg/dL without taking chol meds) is associated with inc mortality in the elderly and in risk of suicide (Epidemiology 2001;12:168) (J Am Geri Soc 2003;51:991-6), may possibly be a warning sign of occult dz or rapidly declining health. HDL cholesterol level is inversely associated to the risk of thromboembolic stroke in elderly men (Am J Epidemiol 2004;160:150-157), the risk declined consistently with increasing levels of HDL with desirable total cholesterol concentrations. Elevated level of Lp(a) lipoprotein is an independent predictor of stroke and of death from vascular disease or from any cause in elderly men, but not in elderly women (NEJM 2003;349:2108-15). Statin therapy is associated with improved clinical outcomes, reducing 1yr all-cause mortality and death/ hospitalization of frail elderly (J Am Ger Soc 2002;50:1389-95). Although statins lower mortality in AMI pt's between 65 and 80 years old, they may not be as effective in older pt's (J Am Geriatr Soc 2006;54:421-430).....pt's who had LDL cholesterol levels above 130 mg/dL were most likely to benefit from statin therapy, and pt's who had LDL cholesterol levels below 100 mg/dL were least likely to benefit. Very hostile older white men were more likely than their less hostile peers to cope poorly with stress and have lower HDL cholesterol levels (data from 716 men with a mean age of 65yo) (American Psychological Association 115th Annual Meeting. August 17-20, 2007).

Children & Adolescents:

BMI may be a poor marker for hypercholesterolemia in children and adolescents according to the results of a cross-sectional analysis (Arch Pediatr Adolesc Med. 2009;163:716–722)...."ecommendations for targeted screening of obese children and adolescents may require further consideration."

AAP Lipid Screening in Childhood Recommendations from the AAP:

Cholesterol screening of high-risk children should be done before age 10 and fasting lipids should be re-checked every three to five years (Pediatrics 2008;122:198).

…….END DEMO of this chapter.

Ear, Nose, Mouth & Throat:

Edited by R. A. Braganza MD, Otolaryngology, Head & Neck Surgery.

Contents: Exam & Anatomy | Mouth, Tongue, Lips, Salivary | **Neck Mass** | Lymphatics | **Taste & Smell** | Teeth / Dental | **Orofacial Pain** | Facial & Neck - TRAUMA | **Throat** | Ears & Hearing | **Nose** | Hiccups | **Facial Nerve (CN 7) Palsy** | Dizziness | **Cough** | *INFECTIONS* | **CANCER** | Pediatric | **Allergic Rhinitis** | Salivary Glands | **MEDS** | Nasolaryngoscopy | **Regional Anesthesia & Blocks** | *INDEX* |

Exam Techniques:

Link: Nasolaryngoscopy | **Anatomy, Indirect Laryngoscopy & Head Mirror Exam** |

Warning Signs & Pearls: A horse voice for more than 6 weeks could be vocal cord carcinoma. A slowly growing neck lump is suspicious, especially if over 2 cm. An unwell, drooling child (or adult) with difficulty breathing could have imminent airway obstruction due to epiglottitis. Stridor in a child with URI – airway could be at risk. Progressive, unilateral hearing loss may be acoustic neuroma. Unilateral nasal obstruction with bloody discharge or parasthesia – think of sinus tumor (need CT). Recurrent / prolonges unilateral OME in an adult is nasopharyngeal carcinoma until proved otherwise. Ear pain on swallowing is highly suspicious for tumor. A child who does not babble and fails to respond to sound may have a hearing problem. A child with unilateral, smelly nasal discharge could have a Fb. Difficulty in opening the mouth or progressive difficulty swallowing and breathing– think of life-threatening infection. Malocclusion of teeth after facial trauma – suspect facial fracture. Tongue pain that radiates to the ear is likely to be due to an invading cancer. A mouth ulcer that lasts for more than 2 weeks is cancer until proved otherwise. Red or white patches in the mouth could be malignant – even if not ulcerated. A patient under 40 years with sharp, unilateral facial pain, or persistent facial paresthesia, could have sinister trigeminal nerve pathology. Swollen, bleeding gums in an anemic patient could mean leukemia. Progressive facial palsy over a period of weeks is often malignant in origin. Unilateral numb lip or chin needs exclusion of malignancy. Non-healing ulceration of the roof of the mouth could be lethal (Alarm Bells in Medicine by Nadeem Ali. 2005 by Blackwell Publishing Ltd)

Nasolaryngoscopy:

Link: Anatomy, Indirect Laryngoscopy & Head Mirror Exam |

Flexible fiberoptic laryngoscopy examination of the nasopharynx and larynx can be accomplished with either a flexible nasopharyngoscope or a bronchoscope. The nasopharyngoscope is thinner, shorter, and easier to manipulate. Commonly performed by allergists, primary care physicians and most otolaryngologists. A signed informed consent statement is not required, as not an invasive exam (but a good idea).

Indications: Chronic hoarseness (>3wks), nasal obstruction, rhinorrhea, sinus discomfort, postnasal drip, suspected nasal/laryngeal Fb, chronic cough, halitosis. Anosmia. Headaches, facial pain. Epistaxis. Sinusitis. Earache, recurrent or chronic otitis media. Regional adenopathy. Dysphagia or globus. Atypical asthma (laryngeal dysfunction).

Possible Indications: bleeding w/o known site, cancer eval/follow-up, nasotracheal intubation, eval of snoring.

Step #1: Attach the endoscope to its light source, attach the suction to its port (if available), and ensure that both are functioning properly before beginning. Prior to inserting the scope, the eyepiece is adjusted to the operator's visual acuity; it is helpful to check the scope focus on newsprint or a small object at this time. Review of the scope directional controls is also recommended.

Step #2: A reassuring bedside manner with an explanation of the procedure is important. The only special preparations need is to anesthetize and vasoconstrict the naris with 4% lidocaine and epinephrine (or phenylephrine, oxymetazoline, xylometazoline or cocaine). Both medications are delivered by a standard nasal atomizer, 1 or 2 sprays of each drug should be directed straight back and the same amount superiorly and posteriorly at about a 45-degree angle. Warn the pt that the anesthetic has an unpleasant taste, takes a few moments to take effect, and may produce a transient but uncomfortable globus sensation if swallowed. Warm the end of the scope in warm water to help prevent fogging. As and option, the rhinoscope may be lubricated with 2% viscous lidocaine or a water-based lubricant. The examiner should always wear gloves, and in high-risk situations, eye, nasal, and mouth protection (a procedure mask with eye shields).

Step #3: The pt should be seated, with the head placed against a headrest, in the "sniffing" position. Insert the tip of the lubricated scope just inside the naris. A small child may sit in a parent's lap. A series of soft nasal trumpets may be used to dilate the nasal cavity, allowing easier passage of the scope. The movement of the scope against the inside of the nasal passage may be irritating; this discomfort can be minimized by resting the fourth and fifth fingers on the bridge of the pt's nose while stabilizing and guiding the passage of the scope between the thumb and index finger.

Step #4: while looking through the eyepiece, slowly advance the endoscope past the inferior turbinate into the nasopharynx or through the lumen of the trumpet. To clear fogging or mucus off the lens, have the pt swallow, wipe the lens against the pharyngeal mucosa, or use the suction. Once it is in the nasopharynx, direct the tip inferiorly, using the thumb control near the eyepiece. Up-and-down movements of the scope may be accomplished with the thumb control, whereas rotating the scope about its axis and then applying the thumb control provides for lateral movement and visualization. At this point, the base of the tongue and tonsils will come into view. Slide the scope further caudad to bring the larynx into focus. Once again, systematically view the anatomy and function during both respiration and phonation. It is usually convenient to examine structures of the anterior nasal cavity first, followed by examination of the pharynx and larynx. The sphenoethmoidal recess and middle meatus are more difficult to examine and may be less well anesthetized than other structures. Thus, they are generally examined last. Following withdrawal of the endoscope under direct visualization, examination of the other side of the nose is carried out.

Pearl: If the scope will not pass through either naris, pass it through the oropharynx. Properly anesthetize the oropharynx and avoid the posterior tongue to prevent gagging. Cut a 10-mL syringe (without the plunger) in half. Have the pt hold this in the mouth between the incisors. Pass the fragile endoscope through this tube to prevent accidental biting of the scope.

Complications: traumatic abrasions and bleeding anywhere along the path of the laryngoscope. In pt's with head injury, there is always the rare risk of passing the scope intracranially if a basilar skull fx exists, but use of a soft nasal trumpet significantly reduces that risk. The induction of laryngospasm and acute airway compromise is possible in pt's with paraglottic infections. If epistaxis occurs, apply pressure and ice. Occasionally get coughing spasms or syncopal episodes (vasovagal).
Cleaning: clean the endoscope by removing mucus from the endoscope with soap and water and a cotton ball. An isopropyl alcohol wipe is used to remove the soap, and a water wipe is used to remove the alcohol. The endoscope is then soaked in a full strength disinfectant (Envirocide). The endoscope is then allowed to air dry before the next examination.

Anatomy:

Link: Nasolaryngoscopy |
Nasopharynx: the torus tubarius is located on the lateral wall of the nasopharynx, defining and protecting the orifice of the eustachian tube, or tympanopharyngeal duct. Rosenmüller's fossa is a vertical cleft, a potential space between the posterior lip of the torus tubarius and the adenoidal pad. It is considerably important because many of the insidious malignancies of the pharynx have their origin here. The adenoid, or pharyngeal tonsil, is the first collection of lymphoid tissue downstream from the nasal passages and sinuses.
Oropharynx and Hypopharynx: the lingual tonsils are located on either side of the dorsum of the base of the tongue anterior to the epiglottis The median glossoepiglottic fold and the two lateral glossoepiglottic folds extend from the epiglottis to the base of the tongue. The valleculae are cup-shaped spaces, separated by the median glossoepiglottic fold, that are posterior to the base of the tongue and anterior to the epiglottis. To the right and to the left of the larynx are the piriform sinuses, gutter like structures that direct food to the esophagus. Visual screening (with lighting, gauze, gloves, 5min) can detect early oral cancer (Lancet. 2005;365:1905-1906, 1927-1933).
Neck: The neck should be inspected for normal anatomic positions of the hyoid bone, thyroid cartilage, thyroid gland, and presence of the normal cervical lordosis as well as identifying skin markings (e.g., scars, unusual skin pigmentation, or neurocutaneous lesions). Palpation of bony structures in the neck should be done with the pt supine to relax the overlying muscles. To palpate the anterior neck, the examiner should stand at the pt's side and support the neck from behind with one hand, palpating with the other, relaxing the spine as much as possible.
Hyoid bone: The horseshoe-shaped hyoid bone lies above the thyroid cartilage and is at the level of the C3 vertebra. With the index finger and thumb (pincer-like), the examiner should feel the stem of the horseshoe; as the pt swallows, the hyoid bone moves up and then down.
Thyroid cartilage: possesses a superior notch and a flaring upper portion (at the C4 level), whereas the lower border lies at the C5 level.
Cricoid: Below the lower border of the thyroid cartilage, the examiner should palpate the first cricoid ring (opposite the C6 vertebra); this is the upper border of the trachea and is just superior to the site for an emergency tracheotomy. The cricoid ring moves with swallowing. About 2 to 3 cm lateral to the first cricoid ring, the carotid tubercle, the anterior tubercle of the transverse process of C6, can be felt.
Larynx: the framework of the larynx is formed by the thyroid, cricoid, and epiglottic cartilages and by pairs of arytenoid, corniculate, and cuneiform cartilages. The aryepiglottic folds and the arytenoids are located immediately behind the epiglottis. The nodular swellings located medially in the aryepiglottic folds are the corniculate cartilages that sit on top of the arytenoid cartilages. Lateral to the corniculate cartilages are the cuneiform cartilages. The aperture of the glottis (rima glottidis) is formed by the true vocal cords (vocal folds; plicae vocales) and the posterior commissure between the arytenoids. The anterior ligament of the true vocal cords is located at the anterior angle of the vocal cords. Between the true vocal cords and the false vocal cords (vestibular folds; plicae ventriculares) are the laryngeal ventricles. The true vocal cords are anteriorly attached to the thyroid cartilage. Posteriorly, they are attached to the vocal processes of the arytenoid cartilages. The true cords are relatively less vascular and therefore appear whiter than the surrounding mucosa. The strong vocal ligaments are covered by connective tissue and a thin layer of epithelium. Reinke's space is the potential space between the vocal ligaments and the subepithelial connective tissue layer. The mobile arytenoid cartilages move in and out with respiration and phonation.
Muscles Involved in Mastication:
Temporalis: Elevate mandible. Attaches from the temporal fossa to Coronoid process and anterior ramus of mandible. Inervated by deep temporal branches of mandibular nerve.
Masseter: Elevate and protrude mandible. Attaches from the inferior and medial aspects of zygomatic arch to Coronoid process and lateral ramus of mandible. Inervated by mandibular nerve via masseteric nerve.
Other: The carotid arteries lie adjacent to the tubercle, and their pulsation can be appreciated. The transverse process of C1 lies between the angle of the jaw and the styloid process of the skull. Because it is the broadest transverse process of the cervical spine, palpation is facilitated. Normal anatomic movements of the atlanto-occipital and atlantoaxial joints and bony structures can be appreciated by a lateral sliding movement, holding the atlas between the thumb and index finger by the transverse processes.

Indirect Laryngoscopy:

This traditional method is most commonly used by the otolaryngologist. It requires significant eye-to-hand coordination to reflect the light beam off the angulated mirror onto the larynx. When this procedure is properly performed, most pt's are able to tolerate it without anesthesia of the oropharynx. **Step #1:** Have the pt sit erect in the "sniffing position," with the feet flat on the floor and leaning slightly forward. Warm the mirror with warm water or in a flame to prevent fogging, but check the temperature of the mirror with your hand before placing it into the oropharynx. Alternatively, anti-fogging solutions can be applied to the mirrored side.
Step #2: With your nondominant hand, grasp the pt's tongue (after wrapping it with gauze to prevent it from slipping or being injured by the lower incisors). Apply gentle owtward traction to the tongue with your thumb and index finger while lifting the pt's upper lip with your middle finger. Slide the mirror into the oropharynx with the glass surface parallel to the tongue but not touching it. Place the back of the mirror against the uvula and soft palate, smoothly lifting until the larynx is visualized.
Note: While this should not induce gagging, try to make only slight changes in mirror position to inspect the appropriate structures. In pt's who cannot tolerate this procedure without gagging, apply topical anesthetic to aid in the examination. Benzocaine (Hurricaine spray or Cetacaine gargle) or aerosolized tetracaine or lidocaine may be used. One or two quick sprays of benzocaine into the posterior oropharynx is sufficient. Prolonged or repeated spray use may result in methemoglobinemia. Reassure the pt beforehand that although this may make the throat feel as if is swelling or paralyzed, in actuality it is just the numbness that accounts for the sensation. The tendency to gag also can be minimized by having the pt concentrate on his or her breathing efforts

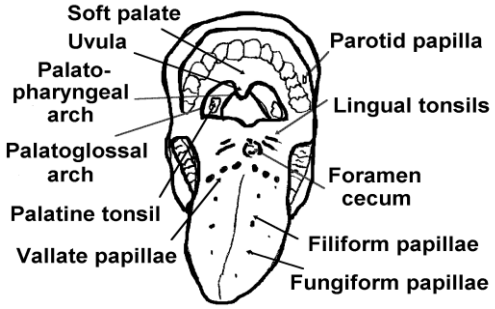
and keep the eyes open, with vision fixed on an object in the distance. Once the pt is anesthetized, repeat the above steps and position the mirror against the soft palate.
Step #3: Rotate the angle of the mirror and systematically inspect the base of the tongue, valleculae, epiglottis, pyriform recess, arytenoids, false and true vocal cords, and, if possible, the superior aspect of the trachea. Observe for masses, evidence of infection, asymmetry, or foreign bodies. Further evaluate the anterior structure of the larynx and function of the vocal cords by having the pt say "eeee" in a high-pitched voice. This should move the epiglottis away from blocking the view of the larynx and bring the true cords together at the midline.
Findings:
Epiglottis: usually has a slightly curved and regular upper edge, but may be acutely curved and conical. It will rise upwards are forward with phonation.
Aryepiglotic folds and Interarytenoid area: looks for swelling and ulceration.
Vocal cords: Examine for color, normal is pearly white. Movement may be restriced by paresis or by infiltration of tumor. Arthritis of cricoarytenoud joint also limits movement. Inspect that the surface is intact and not ulcerated. The edge may be irregular.
False cords: May show swelling or ulceration.
Subglottic space: hard to see. Look for swelling.

Head Mirror / Light Exam:

The head mirror is convex with a central hole that allows the examiner to see directly along the reflected light beam. Begin by swinging the mirror down over the dominant eye just touching the skin or glasses. Position the light source (a 150-watt bulb works well) over the pt's shoulder on the same side as the head mirror. Keeping your eyes open, adjust the focus of light by adjusting your distance from the pt. Change the direction of the beam by tilting the mirror or turning your head slightly. The electric head lamp also attaches by a forehead strap, and the light should be placed nearly between the examiner's eyes to be maximally effective. The intensity of light in this position is nearly as bright as the head mirror. After securing the head lamp, hold your hands in front of you at a distance that is comfortable for working. Focus the beam of light at that point by adjusting the head lamp into position without moving your head or eyes. This allows for the beam to shine on and follow the area on which your eyes are focused.
Ref: (Key pearls from: Clinical Procedures in Emergency Medicine, W B Saunders; 4th ed 2003)

Mouth:

Links: Congenital | **Ranula** | Masses | **Gingiva** | TONGUE & Oral Lesions | **Pigmented Oral** | Erythematous Oral | **Bullous Oral** | Stomatitis | **White Oral Lesions** | Ulcers | **Xerostomia** | Burning Mouth | **Salivary Glands** | Sialorrhea (Drooling) | **Halitosis (Bad Breath)** | LIPS | **Teeth / Dental** | Taste & Smell | **Throat** | Hoarseness (Dysphonia) | **INFECTIONS** | Cheilitis | **Orofacial Pain** | Candidiasis (Thrush) Stomatitis | **Noma** | **See GI for** Dysphagia |



The fungiform papillae are visible as small red structures and are most densely distributed on the tip and the edges of the anterior two thirds of the tongue. The foliate papillae are located between adjacent folds on the edges of the base of the tongue. The circumvallate papillae are raised circular structures on the rear of the tongue., the largest circumvallate papilla is usually in the middle, with three or four smaller papillae on either side forming an inverted V.
Congenital: A bifid (split) uvula is seen in 1% of the healthy population in the US.
Lip pits: seen in 2%, usually lower lip, may have sinuses. 70% have a cleft lip or palate.
Cleft Palate and Lip: seen in 1/1000 Caucasian births, 1/5000 blacks. 50% are on the lip and palate, 25% on the lip alone (M>F), 25% palate alone (F>M). Due to failure of the maxilla and nasal fusion in midline. Causes a poor suck, malocclusion, chronic otitis, mechanical speech problems. Need to feed with special nipples. Surgically repair at 6 wks if lip alone, 14 wks if palate involved.
Pearls: **Mirror Defogger:** place reflective side down on pt's tongue to wet and warm it.
Lessen Gag Reflex: Avoid posterior 1/3 of tongue. Instruct young pt's to sit on their hands. Use two tongue blades and have pt hold them. Better to have the pt inhale deeply ("take a deep breath") than say "Ah" as this elevates the palate and uvula. Or breath heavily through open mouth/ "pant like a dog" **or** pt hold tongue blade **or** ask pt to yawn. Wet the blade with tap water as dry blade more likely to cause gag reaction.
Pharyngeal Fb Vs a scratch from long departed fish bone or popcorn kernel. Can attempt to view with laryngoscope (#3 or 4 Mac) after hurricane spray, but best to use nasolaryngoscope.
Oral Mucous Cyst = Mucocele = Ranula: Often on inner surface of lower lip or floor of mouth painless, translucent, bluish-white, dome-shape, cystic, tense, fluctuant mass from a saliva filled sac under tongue mucous membrane. From a plugged duct or trauma causing salivary duct rupture. Results from obstruction of a sublingual or minor salivary gland duct in the floor of the mouth. It may be confined to the floor of the mouth above the mylohyoid muscle or may extend below the hyoid muscle (= plunging, deep, or diving ranula).
Dx: incision of early lesion with subsequent leakage of saliva filled contents, often a sticky straw-colored fluid.
Tx: I&D with #11 blade or 18g needle or cryosurgery. May disappear spontaneously. Try marsupialization or excision if recurrent, best to send tissue to path to r/o mucous producing adeno-Ca even though most are benign.

Common Tumors:

Torus Palatini: benign, midline bony protuberance on hard palate. An extosisis. More common in women, Native American, Eskimo and Asians, often autosomal dominant. Often not present until late teen or early adulthood, may continue to slowly enlarge over time. Tx usually not necessary, but can be removed/ chiseled off if bothersome. A **mandibular torus** can be found on the lingula surface of the mandible near the bicuspid teeth.

Epstein's Pearls: white, superficial, mobile nodules, usually midline and paired on hard palate. Common in newborns. Asymptomatic keratin containing cysts, usually exfoliate spontaneously in 2-3 weeks.

Bohn's nodules: are remnants of the dental lamina that appear as cysts on the buccal or lingual aspect of the maxillary and mandibular dental ridges in the newborn. They may appear in the palate but are far removed from the midpalatine raphe and should not be confused with Epstein's pearls. No tx is necessary because they disappear in several weeks.

Dental Lamina Cysts: multiple, or occasionally solitary, nodules on the alveolar ridge of newborn or young infants. They represent trapped remnants of the dental lamina. They are soft and spongy, asymptomatic, and tend to disappear with time or the eruption of teeth.

Eruption Cysts: arise from the pre-eruptive dental sac and appear as a swelling of the alveolar ridge. They are associated with the eruption of primary and permanent teeth. Occasionally, they fill with blood and may be termed eruption hematomas. Tx is unnecessary because the erupting tooth usually emerges within several days. If tx is necessary because of the size of the lesion, excision of the overlying soft tissue to expose the erupting tooth eliminates the problem.**Pyogenic granulomas:** develop as granulation tissue in response to an irritant or trauma. Clinically, they are red, elevated, and usually ulcerated. Initial growth is rapid. Most common on the gingivae and may remain static for a time before becoming fibrotic. Tx consists of simple excision, but recurrence is common unless the causative agent (calculus or foreign body) is removed.

Oral fibroma: aka irritation fibroma, traumatic fibroma, fibrous nodule or focal fibrous hyperplasia. A painless, solitary firm, smooth, round mass in submucosa, slowly enlarging. May be sessile or pedunculated. Often on the tongue, gingiva, labial or buccal mucosa.

Tx: Excision via shave bx (to path) then electrodesiccate base. Local anesthesia can be provided by application of topical anesthetic (benzocaine 20%), then injection of Lido + epi. Send to path for histologic dx.

Ddx: includes mucocele (mucous retention cyst), neurofibroma, granuloma, benign/ malignant salivary gland tumor.

Papillomas: benign lesions, wart-like, elevated lesions.

Squamous cell carcinoma: presents as a mass or ulcer on the border of the tongue. Lesions often only palpated rather than visualized. Associated with ETOH, tobacco use, poor hygiene.

Other Oral Masses:

Soft tissue --> benign (hemangioma, lymphangioma, fibrous epithelial cyst, ranula, thyroglossal duct, brachial cleft), malignant (rhabdomyosarcoma, epidermoid).

Tonsillar: Lymphoid Hyperplasia: a relatively common, benign appearing, yellowish-red, dome shaped, located in the tonsillar fossa.

Gingiva: Mass--> benign (congenital epulis, melanotic neuroepidermal tumor, giant cell reparative granuloma, pyogenic granuloma, gingival hypertrophy, Odontogenic cysts), malignant (Burkitt's lymphoma, fibrosarcoma, osteogenic/ Ewing sarcoma).

Links: Periodontitis / Gingivitis | Pigmented Lesions |

Gingival Hyperplasia:

Common in **anticonvulsants** (50% with Phenytoin, often within 1mo of initiation), also in Valproate, Phenobarbital and Primidone. **Cyclosporine** (30% incidence), **Calcium Channel Blockers** (Nifedipine 38%, Diltiazem 20%, Verapamil 10%, Amlodipine 3% **-->** Usually overgrowth of anterior interdental gingival papillae. Related to dose, duration, plasma levels of the drug. Can lead to problems with speech, mastication, tooth eruption, aesthetics. In pregnancy common to get "**epulis**" or gingival hypertrophy that worsens as gestation progresses and causes easy bleeding with slight trauma (brushing teeth), regresses after pregnancy.

Tx: discontinue med if possible and change to another class, will regress.

Uvular edema:

S/s: pt c/o of a foreign body sensation or fullness in the throat, possibly associated with a muffled voice and gagging. Ask about recent foods, drugs, physical agents, inhalants, insect bites and hereditary angioedema. Upon examination of the throat, the uvula is swollen, pale, and somewhat translucent (uvular hydrops). If greatly enlarged, the uvula might rest on the tongue and move in and out with respiration. There might be an associated rash or a history of exposure to physical stimuli, allergens, or a recurrent seasonal incidence.

Tx: inc risk of hypopharyngeal edema. If pt complains of respiratory difficulty or breathes with stridor, start IV and intubate and cricothyrotomy equipment at the bedside, and a crosstable lateral soft tissue neck x ray to rule out epiglottic swelling. If fever, sore throat and pharyngeal injection are present, culture the throat with a rapid strep screen and give an antibiotic that covers Haemophilus influenzae (e.g., Biaxin, Augmentin, Bactrim). It is reasonable to obtain a complete blood count with a manual differential to demonstrate eosinophilia to support the possibility of an allergic reaction or a high leukocyte count with increased granulocytes and bands to support a bacterial infection. Initially the pt should receive parenteral H1 and H2 antihistamines like hydroxyzine 50-100mg im or diphenhydramine 25-50mg iv along with cimetidine 300mg iv or po or ranitidine 50mg iv or 150mg po. More severe cases should receive repeated doses of epinephrine 0.3ml of 1:1000 SQ every 20 minutes x3. Nebulized isomeric or racemic epinephrine or albuterol are also effective. Parenteral corticosteroids like Solumedrol 125mg iv are also typically used, although efficacy remains unproven. Uvular decompression may be useful in pt's that are resistant to medical therapy or whose sx's progress rapidly. This procedure consists of grasping the uvula with forceps and either making several lacerations with a sterile needle or snipping the distal centimeter as a partial uvulectomy. All pt s should be observed for an adequate period of time to insure that there is either improvement or no further worsening of the swelling before being discharged home. Upon discharge, they should receive 4-5 days of H1 and H2 blockers and steroids if required. (Common Simple Emergencies, 2003 www.ncemi.org, the National Center for Emergency Medicine Informatics)

Tongue:

Links: Codes | **Laceration** | Glossitis | **Geographic Tongue** | Fissured | **Hairy** | Tongue Fasciculations & Other Movement Abn's | **Other** | Mouth, Lips, Teeth, Salivary | **Taste** | Erythematous Oral | **Bullous Oral** | Stomatitis | **White Oral Lesions** | Ulcers | **Pigmented Oral Lesions** | See Peds for **Tongue tie / Ankyloglossia** |

ICD-9 Codes:

529.0 Glossitis
529.1 Geographic tongue
529.2 Median rhomboid glossitis
529.3 Hypertrophy of tongue papillae - Black hairy tongue. Coated tongue and other.
529.4 Atrophy of tongue papillae
529.5 Plicated tongue, Fissured tongue, Furrowed tongue, Scrotal tongue
529.6 Glossodynia
528.6 Leukoplakia of oral mucosa, including tongue
528.7 Other disturbances of oral epithelium, including tongue – Erythroplakia, Leukoedema.
528.8 Oral submucosal fibrosis, including of tongue
528.9 Other and unspecified diseases of the oral soft tissues - Cheek and lip biting. Denture sore mouth. Pyogenic granuloma of oral mucosa. Ulcer (traumatic) of oral mucosa

Glossitis:

Inflammation (acute or chronic) of the tongue that may lead to loss of filiform papillae. Can be a primary disease or a symptom of systemic disease.
Risks: Poor nutrition. Age. Dentures. Piercings. Allergy (asthma, eczema, hay fever). Smoking. Smokeless tobacco (snuff). Alcoholism. Anxiety, stress. Depression. Hormonal disturbances. Immunocompromised.
Etiology:
Vitamin deficiency --> dec Riboflavin (B_2), Pyridoxine (B_6), B_{12}, Iron, Vit-E. Common in malnourished, alcoholics and elderly.
Infection --> viral, Candida, TB and syphilis. HIV. Herpetic geometric glossitis (HGG, have linear fissures on dorsal tongue; geometric pattern is common; lesions usually are absent on other mucosal surfaces).
Irritation --> alcohol, toothpaste, meds, tobacco, citrus.
Trauma --> poor fitting dentures.
Other --> lichen planus, pemphigus, erythema multiforme, neoplasia. Atrphic. Benign migratory glossitis (BMG, see below) or geographic tongue. Median rhomboid glossitis (MRG, have rhomboid-shaped plaque in middle of tongue; hypertrophic or atrophic surface changes).
S/s: variable, tongue often red, smooth-surface due to loss of filiform papillae. Often asymptomatic. Pale if anemic, Swelling with infection, trauma or lichen planus. Ulcerations with HSV, pemphigus or strep. Burning sensation on tongue or sensation of foreign body in the mouth. Paroxysmal ear pain. Swollen or painful submandibular lymph nodes.
W/u: CBC, B12, KOH of scraping if white patches (Candida).
Ddx: Psoriasis. Candidiasis. Reiter syndrome. Lichen planus. Leukoplakia. SLE. Herpes simplex virus. Drug reaction.
Tx: per etiology, if painful give 2% viscous Lidocaine 1-2 tablespoons q4hr or 0.1% Triamcinolone q6 hr PRN. See **Stomatitis** |
If candidiasis, Rx Nystatin oral suspension 100,000 units/mLswish and spit 5 mL q.i.d. or Clotrimazole 1- 2 troches 4-5 times a day.

Migratory glossitis (BMG, Geographic Tongue):

Benign, seen in young adults. Has an inc incidence in DM and psoriasis.
S/s: See annular erythematous patch with gray-white rim. May be atrophic in appearance. Sharply demarcated (annular/ circinate) erythematous depapillation (loss of filiform papillae) areas with white elevated rim/border. Often seen on lateral border or dorsum of tongue, usually anterior. Rare on palate, buccal mucosa. Fungiform papillae become prominent because of a selective loss of filiform papillae/ surface keratin.
Changes in intervals of weeks to mo's.
Tx: Reassurance. Waxes and wanes over lifetime, may resolve spontaneously. Congenital (polygenic), benign. If deep furrows on surface trap food, brush tongue with a toothbrush. Can treat symptomatically with topical lidocaine. If p has burning and tenderness, Rx Kenalog in Orabase 15g, AAA TID ½ hr after eating or 0.025% menthol and/or 0.025% camphor in Orabase (pectin/ gelatin). Avoid spicy, salty, hot, rough (potato chips) foods. If complains of **pain** with acidic/ spicy food, Rx bland diet and antihistamine mouth rinse. If dx in doubt tx empirically with topical antifungal. Diphenhydramine liquid: Rinse with 5-10 mL, holding it over the tongue for a few minutes and then swallowing, 3-4 times a day. Magic Mouthwash swish and spit 5 mL, 3-4 times a day. If HGG, rx acyclovir 200 mg 5 times daily.

Fissured (Scrotal) Tongue:

Lingua plicata. The most common developmental (congenital) defect of the tongue (AD). Benign and asymptomatic worsens with age (elderly). Shallow or deep grooving of the dorsum of the tongue. Tongue fissures occur normally in about 10% of the population. Deep fissures may promote collection of food particles, leading to chronic inflammation and tenderness caused by bacteria and yeast overgrowth. Can be associated with Downs and Sjögren's syndrome.
Tx: Pt needs to splay open tongue and brush the material out with a dentifrice (tongue brush) after meals.

Smooth Tongue:

Absent filiform papillae on ant 2/3 of tongue. Assoc with nutritional deficiency (Plummer-Vinson synd, pellagra, pernicious anemia, sprue and celiac disease). May be associated with Candidiasis.

Lingua Nigra (Black Hairy Tongue):

Elongation of filiform papillae and overproduction of pigment from bacteria. Associated with Abx use, tobacco, exposure to food colorings, Vit def, GI d/o or poor oral hygiene. May have associated glossitis with a burning pain.
Tx: Topical hygiene with toothbrush (brush tongue) to scrape of the keratin. Add dilute 3% peroxide to bleach the pigment. D/c Abx. (Black hairy tongue. Am Fam Phys 1990;41)

Hairy (Furred) Tongue:

Often from changes in bacterial flora from mouth breathing, changes in diet (less fiber), poor fitting dentures or Abx. May be more pronounced in fever, dehydration, smokers.
S/s: hyperkeratotic filiform papillae, "coated" on mid-dorsum.

Tx: reassurance, antimicrobial mouthwash and using toothbrush.

Tongue Fasciculations: An abnormal pattern of nonrhythmical, unorganized contraction of individual muscle fibers across the surface of the tongue. May be observed when the tongue is at rest, or following direct stimulation to the tongue. May also be observed during generalized hypertonicity or hypotonicity affecting the whole body. Ankyloglossia - A structural impairment consisting of a shortened lingual frenulum. Body of the tongue is thinned, with the lateral borders elevated. A heart shaped indention may be noted at the front edge of the tongue. Function is limited if the tongue tip can lift < 1/4" above the lower incisors. Pseudo Ankyloglossia - A functional impairment in which the body of the tongue is thickened and retracted. The lingual frenulum appears as a prominent white fiber at the center of the tongue tip. The end of the tongue is blunt and thick. Fasciculations and atrophy are signs of lower motor neuron lesions. Unilateral tongue weakness causes the tongue to deviate toward the weak side. Tongue weakness can result from lesions of the tongue muscles, the neuromuscular junction, the lower motor neurons of the hypoglossal nerve (CN XII), or the upper motor neurons originating in the motor cortex. Lesions of the motor cortex cause contralateral tongue weakness. Tongue fasciculation can be one of the earliest signs of "extrapyramidal" side effects of drugs like haldol & thorazine.

Benign fasiculations: have no clear etiology. Can appear widespread. They are not associated with weakness, wasting or reflex changes and may remit over time.

W/u: If one has clinical features other than fasiculations an EMG should be considered. Tongue fasiculations are not a specific indicator of ALS. Thyroid function test.

Lip tremor: Rapid, small movements of the lips during purposeful activity, such as lip seal. A mildly abnormal pattern indicating fatigue. Lip retraction - This is an abnormal pattern in which increased abnormal tone pulls the corners of the lips up and back. It may be observed to affect upper lip movement more than lower lip movement. The person with this pattern may be described as "always smiling". The anterior cheek area usually shows a retracted pattern also.

Tongue-Tie (Ankyloglossia):

The tightness of the lingual frenulum varies greatly among normal people. A short frenulum prevents both protrusion and elevation of the tongue.

S/s: Puckering of the midline of the tongue occurring with tongue movement is noted on physical examination.

Tx: When mild, tx consists of reassurance. If the tongue cannot protrude past the teeth or alveolar ridge or move between the gums and cheek, referral to an otolaryngologist for evaluation is indicated. Frenulectomy may be recommended if there is a question of suckling difficulties, dental health (related to the inability to clear food from around the teeth), or articulation problems (Controversies in management. Int J Pediatr Otorhinolaryngol 2000;54:123) (Ankyloglossia: incidence and associated feeding difficulties. Arch Otolaryngol Head Neck Surg 2000;126:36).

Lip purse-string: This is an abnormal pattern in which the corners of the lips are pulled back as the rest of the lip pulls to midline, with an increase in abnormal tone. Increased tone may extend from below the nose to the chin and into the cheeks.

Asymmetrical lip movement: This is an abnormal pattern in which one side of the lip moves with less control than the other side. Abnormal patterns and muscle tone are noted on the affected side.

Hypotonic lips: This is an abnormal pattern in which the lips appear flaccid, with little or no active movement. The lips may look puffy. The lower lip may appear more involved than the upper lip.

Dystonic lip movement: An abnormal pattern characterized by rhythmical, nonfunctional movement of either or both lips, associated with Parkinson's or Parkinson's like sx's. The ability to interrupt the movement is related to the severity of the disease. With less severe involvement, the pattern can be interrupted during functional activities such as eating and speech, and will not be observed during sleep.

Lip Fasciculations: An abnormal pattern of nonrhythmical, unorganized contraction of individual muscle fibers across the lips. May be observed when the lips are at rest, or following direct stimulation of the lips. May also be observed during generalized hypotonicity affecting the whole body.

Normal Tongue Patterns Tongue movements are an integral part of the eating process. The following six normal patterns (suckling, simple tongue protrusion, sucking, munching, tongue tip elevation and lateral tongue movements) are presented in order from primitive to more mature patterns.

Suckling: The primary movement in suckling is extension – retraction. The tongue does not extend beyond the lips. Lateral movement is not observed. The tongue may show a semi-bowl shape (cupping). The tongue remains flat and thin. The movement is accomplished with normal tonal changes with rhythmical cycles of extension - retraction. Jaw opening and closing occur in conjunction with tongue movement. This is a normal but primitive pattern.

Simple tongue protrusion: This is a primitive, normal movement associated with the suckling pattern. The tongue extends between the teeth or gums. The tongue remains flat and thin with no abnormal tonal changes. (In the normal population, this may be called tongue thrust, especially by speech pathologists.)

Sucking: The tongue is flat and thin, movement is up and down and is contained within the mouth. The tongue tip elevates to the anterior hard palate. The movement is rhythmical, up-down cycles, with normal tonal changes. This is the primary pattern for adults. The normal rhythm for nutritive sucking is one cycle/second; non-nutritive suck is faster or slower than that rate. A suck occurs with two kinds of pressure: positive pressure and negative pressure. Positive pressure occurs when the jaw elevates, the tongue elevates to the hard palate, and the lips seal. Negative pressure occurs when the jaw drops, the tongue moves away from the hard palate, the posterior cheeks contract, the soft palate elevates, and the lips remained sealed. More coordination is needed for the negative phase of suck.

Tongue tip elevation: This pattern emerges during suck. The anterior one-third of tongue raises upward to contact the upper teeth or alveolar ridge (gums behind upper teeth). It indicates separation of tongue and jaw movement. This movement continues to develop so that the tongue tip can reach the upper lip, even when the jaw is depressed.

Munching: The primary movement of the tongue is up and down with flattening and spreading. Lateral tongue movements are not observed during this pattern. Tongue movements are accompanied by up and down movement of the jaw for chewing and biting. This is a normal tongue pattern observed in early chewing. Food is positioned on the body of the tongue and raised upward to the palate to break up the food prior to swallowing. Soft, lumpy foods, ground meats, and foods that dissolve in saliva (such as crackers), are tolerated with this chewing pattern. All of these patterns are normal, but do not involve any lateral tongue movement. The person cannot move food between molars for chewing. Since this is needed for chewing more viscous foods, s/he fails to move along the continuum of greater variety and separation of tongue, lip, and jaw patterns. The person is limited to a diet which does not require chewing and grinding, such as a pureed diet.

Lateral tongue movements: The tongue moves to either side, horizontally, to shift food from the center of the mouth to the side. Initially, the tongue may barely shift toward the gum. As skill develops, the tongue will contact the gum or molars. With more control, the tongue will move over the gums or molars. With continued development, the tongue will extend into either cheek. As skills develop, the tongue can move food from one side across the midline to the other side. As movements become more defined, lateral and tongue tip elevations are combined to allow sweeping/cleaning movements of lips, palate, and inside the cheeks. This allows particles of food to be gathered and positioned on the tongue prior to swallowing.
Exaggerated tongue protrusion: The tongue shows extension (forward movement) beyond the border of the lips which is non-forceful. The movement is a rhythmical extension-retraction pattern. It is similar to a suckle pattern, but is mildly abnormal.
Tongue thrust: The tongue is thickened and bunched. The movement is an outward extension beyond the border of the lips. The movement is forceful, and is associated with an abnormal increase in muscle tone. This may occur as part of a total extension pattern of the body, or with hyperextension of the head and neck. The tongue thrust may make it difficult to insert a utensil into the mouth or may cause food to be ejected during feeding. During drinking, the tongue may thrust into the cup or may protrude in a very tight, bunched fashion beneath the cup.
Tongue retraction: In this abnormal movement, the tongue appears thickened and bunched. The movement is retraction, a strong, pulling back of the tongue into the posterior portion of the oral cavity, associated with abnormal increased muscle tone. The tip of the tongue is not forward and even with the lower lip. It is pulled back toward the middle of the hard palate and may be held firmly against the hard or soft palate. Hard approximation of the tongue with the palate may make insertion of utensils extremely difficult and may make it nearly impossible for any food to be placed on top of the tongue for swallowing. Gagging may be increased for the person with this pattern. Severe tongue retraction can partially block the laryngeal airway contributing to added respiratory problems during feeding. Tongue retraction may be associated with other patterns of retraction or extension in the body (i.e., shoulder retraction or neck extension) or it may be an abnormal pattern used as compensation by a person with poor swallowing patterns. When a person has swallowing difficulties, food which moves rapidly or is very thin may be uncontrollable and life threatening when the tongue is more forward. In such cases, the tongue retracts, resulting in reduction of the size of the pharyngeal opening. This pattern is associated with abnormal increased muscle tone.
Asymmetrical tongue placement or movement: The tongue deviates to one side or the other and may show atrophy on the affected side. It may be accomplished by or associated with abnormal tone in the facial musculature. All movements of the tongue are affected. The tongue deviates, or is pushed toward the weak side. If lateral tongue movement is consistently observed only to one side, it may not be active lateral movement, but rather may be asymmetrical movement toward the weak side.
Hypotonic tongue: may appear thickened and shows little or no active movement. Fasciculations, small, uncoordinated movements over the body of the tongue, may be observed when the tongue is at rest. These movements may increase during eating, drinking, swallowing and vocalizations.
Dystonic tongue movement: rhythmical, nonfunctional movement of the tongue associated with Parkinson's or Parkinson's like sx's. The ability to interrupt the movement is related to the severity of the disease. With less severe involvement, the pattern can be interrupted during functional activities such as eating or speech, and will not be observed during sleep.

Other:

Smooth Tongue: "Bald" tongue. Atrophy of filiform papillae due to nutritional deficiency (IDA, pellagra, pernicious anemia), xerostomia, malabsorption, Riley-Day syndrome.
Tongue Lacerations: Tongue biting seen in 25% of generalized tonic-clonic sz's (lateral lacerations). May be seen in TIA's, syncopal episodes (tip of tongue), psychogenic sz's (rare) and hypoglycemic episodes (rare).
Sublingual Varices: seen in 10% age >40yo. See a vascular dilations at the surface to the tongue. Considered a normal finding.
Pigmented Fungiform Papillae: benign, common in dark-skinned individuals. Seen in up to 30% of African Americans. Seen as asymmetrical spots or plaques on the dorsal and lateral aspects. (Black taste buds. Arch Fam Med 2000:9:219-20)
Median rhomboid glossitis: dx based on appearance of the lesion. Also called "central papillary atrophy", due to the loss of the lingual papillae. **Tx:** A corticosteroid paste often helps, usually self-limiting, may be due to a chronic Candidal infection. Consider an HIV test.
Acute Swelling or edema: Often an allergic reaction to food. May be from a bee sting (drinking from open can of sugary soda into which a bee has flown). R/o **Angioedema** (see Allergy chapter).
Tx: Usually resolves with PO antihistamine such as Benadryl.
Fibroma: A focus of hyperplastic fibrous connective tissue representing a reactive response to local irritation or masticatory trauma. Occur in ~1.2% of adults. The most common location is along the occlusal line of the buccal mucosa-an area subject to masticatory trauma-although other locations, such as the tongue, labial mucosa, and gingiva, are possible. Fibromas manifest as asymptomatic, moderately firm, smooth-surfaced, pink, sessile or pedunculated nodules.
Tx: surgical excision, recurrence is rare. The removed tissue should undergo pathologic examination to rule out the possibility of neoplasms that may have a similar appearance.
Traumatic Fibroma: One of the most common lesions in the oral cavity. A firm, benign, flesh-colored papule-nodule on the dorsal lateral tongue. Bx reveals a fibrous-type epidermal hyperplasia. Usually along the buccal mucosal bite line.
Macroglossia: "big" tongue. Associated with multiple myeloma & amyloidosis (also with pinch purpura), lymphoma, hemangioma, acromegaly and Down syndrome.

Pseudomacroglossia:

The tongue appears relatively large but is normal in size. Identification is important, since correction needs to target areas extrinsic to the tongue, rather than the reducing tongue volume itself. Can be seen with habitual posturing of tongue anteriorly, displacement, hypertrophy of tonsils and adenoids, extrinsic cysts or tumors, reduced oral cavity volume, low palatal vault, transverse, vertical or anteroposterior deficiency in the maxillary or mandibular arches, severe mandibular deficiency.
Tx: depends on the cause. Displacement due to enlarged lymphoid tissue, cysts or tumors can be corrected by surgical removal of cause. Orthognathic surgery can be used to advance the mandible, which can increase the oral cavity volume. Anterior posturing of the tongue may be reduced by behavioral counseling. (Am J Orthod Dentofac Orthop 1996;110:170-177)
Sore Tongue Ddx: Geographic tongue, Lichen planus, vitamin deficiency, cancer, Candidosis (red area with papillary atrophy in center of tongue).

****REF:** (Glossitis and other tongue disorders. Dermatol Clin 2003;21:123-134) (Periodontal diseases in the child and adolescent. J Clin Periodontol 2002;29:400-410)

Pigmented Oral Lesions:

Usually physiologic (more common with darker skin), can be anywhere (often gingiva or buccal) in any form. Often see oral melanotic macule on lower lip> gingiva> buccal, usually <1cm, consider Bx if unsure of dx.

W/u: A full history of evolution of the pigmentary changes, as well as inquiring into family history, drug ingestion and systemic sx's of concurrent disease are clearly important in the assessment. The duration, pattern, hue and distribution of colour changes can provide useful diagnostic clues. Special attention is given to newly appearing lesions, or those that have changed significantly in appearance, and biopsy may be needed to validate the clinical impression.

Genetic: African Americans and other people of color can naturally have dark gums (or the gums can darken slowly with the normal aging process).

Peutz-Jeghers: AD, also with benign intestinal polyposis, macular areas on lip, buccal, gingiva, tongue, fingers, macular deposits, no tx needed.

Recklinghausen Neurofibromatosis: see café au lait spots on lips and skin, no tx,

Albright's syndrome: triad of precocious puberty, cutaneous pigments, polyostotic dysplasia. Café au lait on lips and skin, no tx needed.

Carney Syndrome: AD, endocrine overactivity, skin pigmentation, myxoma.

Hormonal:

Addison's: deficient adrenal glucocorticoids **-->** pituitary makes melanocyte stimulating hormone. Can cause gums to turn a fairly even brown or black.

Chloasma: melanocyte stimulation from maternal and fetal corticotropin **-->** macular darkening of skin and mucosa.

Meds / Drugs: quinacrine, quinidine (diffuse pigmentation on hard palate), chlorpromazine, Ketoconazole, OCP's, busulfan, doxycycline, minocycline, Methyldopa, TCA's, doxorubicin, propranolol, terbinafine, risperidone, zidovudine, lomefloxacine, fluoxetine, chlorhexadine. Bismuth from antidiarrheal. Minocycline can discolor the teeth and bone that then appears blue through the gums. Gold salts may cause purplish gingival discoloration. Amodiaquine or quinidine causes blue-black pigmentation. Chloropromazine may cause purple or yellowish brown pigmentation of the skin and mucous membranes.

Foreign bodies: **amalgam tattoo** from silver/ mercury on adjacent tooth, Graphite deposit from pencil stab.

Heavy Metals: Mercury, silver- photography worker.

Amalgam Tattoo --> asymptomatic, blue-gray macules, beneath tongue or on cheeks. Amalgam restorations can generate corrosion products (eg, silver sulfide), leaving a gray-black color in the tooth, especially in large cavity preparations with undermined enamel.

Lead: Chronic lead poisoning can be exposed by the presence of a blue-black line (Burtonia line) at the gingival margin. See a scalloped band at free gingival margin in moonshine distilled in radiators or pica, due to precipitation from sulfide producing bacteria.

Reactive: post-traumatic, under dentures,

Smoker's melanosis: of gingival. Usually anterior mandibular area, brown-black macules. Seen in 5-22% of smokers. Smoking stimulates the melanocytes to manufacture and deposit melanin on the gums and buccal mucosa. If atypical **-->** check Bx).

Other: **nevi** (hard palate> buccal>lip>gingiva>labial> soft palate> tongue), **melanoma** (1%) (palate> maxillary gingiva> buccal> lip> mandibular gingiva> tongue> floor), amalgam tattoo.

Skin diseases associated with mucous membrane manifestations: Pemphigus vulgaris. Lichen planus. Lupus erythematosus. Mucosal candidiasis. Viral diseases such as Koplik's spots of measles and herpes simplex. Syphilis.

Erythematous Oral Lesions:

Inc vascularity of the local tissue. **Erythroplasia =** a nonspecific red patch.

Links: Geographic Tongue | **Candidiasis (Thrush) Stomatitis** | Carcinoma |

Oral Mucositis Ddx: Reactions to exogenous substances (drugs, oral hygiene products, foods, dental material). Stomatitis (aphthous, HSV, herpetiform aphthous). Autoimmune d/o (lichen planus, pemphigoid vulgaris, chronic ulcerative stomatitis, epidermolysis bullosa acquisita, linear IgA dz).

Erythroplakia: A clinical term that refers to a red patch that cannot be defined clinically or pathologically as any other condition. Not a specific histologic entity. Most frequently in older men. Has a premalignant potential, 50% represent invasive carcinoma. Appears as a solitary red patch/ macule of varying size, may be slightly elevated, painless, has a soft velvety texture. Usually on floor of mouth, soft palate or retromolar trigone. Usually asymptomatic, some c/o of sore or burning sensation. Not as common as leukoplakia, but more likely to show dysplasia or carcinoma. Needs Bx.

Ddx: chronic inflammation secondary to trauma, candidiasis, erosive stomatitis, need Bx.

Stomatitis areata migrans: non lingual form of geographic tongue. Buccal mucosa> labial/ oral vestibule> floor> ventral tongue> soft palate> gingiva. Painless, flat, irregularly shaped patches with raises keratotic rims. Heal then reappear.

Tx: same as geographic tongue.

Erythema migrans: multiple flat, irregularly shaped, serpiginous red patches with raised keratotic rims that heal spontaneously and reappear in constantly changing patters. Seen in young adults, usually painless.

Allergy: red lesions from hypersensitivity to topical or systemic drug. EM and lichenoid.

Tx: remove offending agent, symptomatic, steroids.

Plasma cell gingivitis: an atypical gingivostomatitis with diffuse gingival erythema. Some with lesions on lips/ buccal mucosa/ tongue. Represents an allergic reaction to an unknown antigen (often in chewing gum or toothpaste), see plasma cell infiltrate on Bx.

Tx: discontinue offending substance, prednisone 30-40mg/d**.**

Vitamin Deficiencies: loss of filiform papillae.

Riboflavin (B_2 & B_6= pyridoxine) **-->** magenta tongue with fissuring of oral commissures,

Niacin **-->** painful ulceration's, Vit-C **-->** hemorrhagic gingivitis.

Anemia: (micro/ macrocytic) **-->** pallor of mucosa and depapilation of tongue, begins on lateral aspect.
Vascular: may appear as red swellings, bleed easily. Hemangiomas, Kaposi's Sarcoma, AVM (pulsatile), varices and
Genodermatosis: (Osler-Weber-Rendu = hereditary hemorrhagic telangiectasia, see multiple small red macules and papules, Sturge-Weber Syndrome with port-wine stain in trigeminal distribution).
Pemphigus vulgarus: nonspecific erythema in oral cavity, tongue, skin. Small scattered white plaques.
Tx: symptomatic.
Pyogenic Granuloma: a nonspecific term for granulation tissue formed in response to minor trauma with secondary infection. The lesion, which is elevated, pedunculated, or sessile, smooth or verrucal, painless, and soft, occurs most often on the gingiva. Simple excision usually suffices. Lobular capillary hemangioma is also referred to as pyogenic granuloma.
Reparative Granuloma: The cause is unknown, but it is probably secondary to local trauma, such as tooth removal. The peripheral form is a sessile or pedunculated mucosa-covered reddish or bluish mass arising from the gingiva or alveolar mucosa, and it is most common on the anterior mandible. The central form is endosteal, usually anterior to the first molar in the mandible. Radiographically, the lesion appears as a lytic, expansile, unilocular cavity with well-demarcated nonsclerotic margins and a bony cortex, which is thinned but intact. Tx consists of curettage.
Pregnancy tumor: exophytic erythematous gingiva of gravid female, hormonal.

Bullous Oral Lesions:

Erythema Multiforme: often with irregular target lesions on skin. May be caused by a bacterial, viral or fungal infection.
Tx: PO Prednisone 40-60mg/d
Toxic Epidermal Necrolysis (TEN): 90% with MM lesions. Also on conjunctiva and anogenital area.
Tx: Stop offending agent, avoid skin trauma, start IVF **-->** Burn unit for supportive care.
Pemphigus Vulgaris: autoimmune, middle age, affects skin and MM's. Oral cavity may be first presentation.
Dx: Bx with immune complexes (IgA, IgG, IgM) in intraepithelial layer.
Tx: PO Prednisone 60-180mg/d
Bullous Pemphigoid: immune, resembles PV, but usually has skin lesions before oral. Most lesions bullae, there may also be vesicles. Dx: immunofluorescent staining show IgG and C3 in epidermal basement membrane.
Tx: PO prednisone 40-80mg/d
Mucous Membrane Pemphigoid: often involves conjunctiva, may be nearly anywhere. Lesions are vesiculobullous and leave scarring when heal.
Dx: Histological appearance shows subepithelial clefting with deposition of IgG and C3 in the BM.
Tx: emollient mouthwash **-->** Diphenhydramine syrup and PO Prednisone 20-40mg/d.
Paraneoplastic Pemphigus: autoimmune vesiculobullous. Underlying lymphoma, leukemia, carcinoma, thymoma. IgG and complement in intercellular spaces.
Tx: PO Prednisone 60mg/d and Azathioprine 1-2mg/kg/d.
Epidermal Bullosa: Genodermatosis, appears in childhood. Distribution over arms and legs joints.
Tx: Palliative, phenytoin helps.
Erosive Lichen Planus: more common in F, Caucasian, See IgM in colloid bodies.
Tx: PO Prednisone
Dermatitis Herpetiformis: immune mediated, symmetric, pruritic, on extensor surfaces of body, peaks 40's, 80% are gluten sensitive.
Tx: gluten free diet and Dapsone 100-200mg/d.
Other: Lupus Erythematosus (SLE), Linear IgA dz, Chronic ulcerative stomatitis, Graft Versus Host Dz, Secondary Syphilis, Drug induced.

Stomatitis:

Links: Candidiasis (Thrush) Stomatitis | **Ulcers / Mucositis** | Xerostomia | **Soothing Meds** | Herpes | **Coxsackievirus** | Glossitis |

Inflammation of the mucous membranes of the mouth. Often related to malnutrition, lack of proper hygiene of the mouth and vitamin deficiency mainly vitamin C and B complex.
R/o: trauma (cheek biting), thermal injury (hot foods), chemical injury (irritating mouthwash, toothpaste, tobacco).
S/s: The ulcers are punched out, covered by dirty white pseudomembrane covering the mucous membranes of the tongue, lips, buccal mucosa, tonsils, and the pharynx and may involve the whole respiratory tract mucosa.
Tx: Topical analgesia (Viscous 2% Lidocaine and/or Benadryl elixir swish & spit), tobacco cessation, analgesics, oral rinses with ½ strength hydrogen peroxide. Can try Sucralfate (Carafate) suspension @ 1 tsp swish & spit or dab QID. Proper hygiene of the mouth and correction of the predisposing factors. Mild mouth wash antiseptics. Tetracycline suspension orally should be kept for few minutes in the mouth to have a local effect. Zithromax suspensions can be substituted for young age groups.
ICD-9 Codes: 528.0 Stomatitis.
Other: acute necrotizing ulcerative (101), aphthous (528.2), gangrenous (528.1), herpetic (054.2), Vincent's (101)
Oral Bacterial Infection: Clindamycin 300-450 mg PO q6h or Clindamycin 600 mg IV q8h. Can also use AM/CL 875/125 mg PO bid or AM/CL 500/125 mg PO tid or cefotetan 2 g IV q12h.
Buccal Cellulitis: Children >28 days and <5 years usually due to haemophilus influenzae (rare in Hib immunization pt). Tx with Cefuroxime 75-150 mg/kg/day IV div. in q8h or Cefuroxime 10-15 mg/kg PO bid or Cefotaxime 50 mg/kg IV q8h or AM/CL 45 mg/kg div. in bid or TMP-SMX 12 mg/kg div. in bid.
Mouth Care Regimen: brush teeth after meals and HS, Na-bicarb 30ml swish & spit (4 tbls baking soda in 1L NS), Peridex 15ml swish & spit, Nystatin @ 400,000 units = 4ml QID X 10d, swish & spit (or suck a Mycelex touché). Acyclovir 200-800mg 5X/d X 7-14d if herpetic.
Ddx: herpetic stomatitis, Hand-Foot Mouth, denture related, Behçet's, angular stomatitis, uremic, herpangina, pemphigus, Vincent's, squamous cell cancer, aphthous, erythema multiforme, scarlet fever, noma (gangrenous).

Stomatitis Nicotina: Smoker's palate, mostly from pipe, cigars. Hard and soft palate covered with keratotic papules the have elevated red centers, that are inflamed mucous gland orifices (inflamed, highlighted ducts of salivary glands), self limited upon cessation. Only needs biopsy if on soft palate.
Herpetic gingivostomatitis: Children and young age groups are commonly infected with herpes virus leading to herpetic lesions of the mouth, tongue and lips. Painful ulcerations are common which may interfere with feeding and increased salivation.
Gangrenous stomatitis: occurs in children with poor nutrition, lowered resistance and improper dental hygiene. The lesions manifest with mucosal ulcerations of the mouth and genitalia, which rapidly becomes gangrenous. The condition may extend to the adjacent tissues and may be extensive involving the bones and may be fatal.

White Oral Lesions Ddx:

Links: Candidiasis (Thrush) Stomatitis | **Oral Lichen Planus** | Carcinoma | **Leukoplakia** | Leukokeratosis | **Other** | Ulcers |

Oral Candidiasis (Thrush)/ Stomatitis:

An opportunistic mucosal infection caused, in most cases, by Candida albicans. Candida species are commensals in the GI tract. Transmission occurs directly between infected people or on fomites (objects that can harbor pathogenic organisms). Candida is found in the mouth of 31–60% of healthy people (Aust Dent J 1998;43:244–249).
Risk factors: local or systemic immunosuppression, hematological d/o's, broad spectrum antibiotic use, inhaled or systemic steroids, xerostomia, diabetes, and wearing dentures, obturators, or orthodontic appliances.
The four main types of oropharyngeal candidiasis are:
1. Pseudomembranous --> creamy, cheesy white or yellowish plaques adherent to reddish or normal colored mucosa on the palate, buccal , labial mucosa or dorsum of tongue. White exudate, scrapes off leaving erythematous patches. A KOH exam of scrapings confirms Dx. Often seen in diabetics, immunocompromised or with Abx therapy. Pseudomembranous lesion that mimics keratosis, predilection for palate and dorsum of tongue.
2. Erythematous --> red or pink spot / macule on palate, dorsum of tongue or buccal mucosa. Often with a burning sensation.
3. Hyperplastic --> hard white plaques / patches / nodules on posterior buccal mucosa, inner lip commissures or palate.
4. Angular cheilitis --> Perlèche. Cracks or erythematous, scaling fissures with small white plaques radiating from angles of the mouth. **Denture** induced stomatitis, presenting as either a smooth or granular erythema confined to the denture bearing area of the hard palate. Denture stomatitis associated with Candida is prevalent in 65% of denture wearers.
Tx: Topical antifungals. Clotrimazole (Mycelex) troches, 10mg PO 4-5X/d x7-10d. **or**
Nystatin (Mycostatin) oral susp swish & swallow or lozenges (pastilles) @ 400,000 Units (4ml of 100,000 unit/ml suspension) QID X 10d or 200,000 pastilles dissolved in mouth X10d. **Or**
For pt's in whom topical tx is ineffective or poorly tolerated, systemic therapies include:
Ketoconazole @ 200 mg/day PO x 7d.
Itraconazole @100 mg BID x7d.
Fluconazole (Diflucan) PO tabs 200mg X1, then 100mg/d X 7-14d. A multi-center French study has shown that mucoadhesive buccal miconazole gel tablets (50mg qd) is effective for the treatment of radiation-related oropharyngeal candidiasis (Cancer 2008;112:204-211).
Infants: Nystatin @ 1 drop to each side of mouth QID after meals X10d (100,000 unit/ml suspension). Can use Gentian violet, but stains the skin and clothes and is associated with mucosal ulceration (J Fam Pract 2008;57:484). In infants Fluconazole susp (3mg/kg PO qd x7d) more effective than nystatin (Ped Inf Dis J 2002;21:1165-7).
Other: If refractory, may need Ampho-B 0.3mg/kg IV qd.
Compounded Rx Amphotericin B 100 mg Troches: (makes 24) Amphotericin B 2.4 g + Aspartame 500 mg + Silica gel 240 mg + Acacia powder 400 mg + Flavoring oil 3-4 drops + Polyethylene glycol 1450 21.5 g (depending upon the mold used). Note: It is necessary to calibrate the troche mold using the polyethylene glycol 1450 base prior to determining the exact quantity of polyethylene glycol 1450 required for this prescription. Calculate the required quantity of each ingredient for the total amount to be prepared. Accurately weigh/measure each ingredient. Melt the polyethylene glycol 1450 using low heat or a water bath. Comminute and mix the amphotericin B, aspartame, silica gel and acacia powders together in a mortar. Sift into the melted base and mix thoroughly. Cool slightly and add the flavoring oil and mix well. Pour into molds and cool. Package and label.
Compounded Rx Tetracaine Hydrochloride 0.025% and/or Nystatin 250,000 unit Popsicles: (4 Popsicles) Tetracaine hydrochloride 120 mg + and/or Nystatin 1,000,000 units (227 mg) + Aspartame 480 mg + Corn syrup 48 mL + Purified water qs 480 mL + Flavor or KoolAid powder qs. Calculate the required quantity of each ingredient for the total amount to be prepared. Accurately weigh/measure each ingredient. Note: Check the potency of the nystatin to determine the exact quantity to be weighed. Dissolve the tetracaine hydrochloride and aspartame in about half the purified water. Add the corn syrup, nystatin powder and flavor/KoolAid powder and mix well. Add sufficient purified water to volume and mix well. Pour into mold/tray/plastic sleeves and freeze. Package and label. Note: Volume and quantities can be adjusted to mold/tray/plastic sleeve size.
Chronic hyperplastic candidiasis: (candidal leukoplakia) consists of irregular, white, persistent plaques on the tongue or mucous membranes that are difficult to remove; this form of candidiasis occurs especially in male smokers. Soreness, burning, and roughness of the affected areas are the usual sx's. Candidiasis of the tongue can also take the form of median rhomboid glossitis, a diamond-shaped area of atrophic papillae in the central portion of the lingual surface.

Oral Lichen Planus (OLP):

OLP is an autoimmune disorder mediated by T lymphocytes, which recognize keratinocytes as foreign and subsequently secrete tumor necrosis factor a. Up to 4% of the population has OLP, onset usually in mid-life. F > M. 25-35% will also have cutaneous LP. Secondary oral candidal infections common. May get similar lesions with hypersensitivity to dental restorative metals such as mercury. Consider Bx as 2% transform to SCC. See Derm chapter for Lichen Planus.
S/s: lacy, white plaques on buccal mucosa. Presents with multiple striated white, papillated lesions, often misdiagnosed as fungal. Usually symmetrical on buccal mucosa. The most common area of the oral mucosa affected is the posterior buccal mucosa. May present in multiple patterns, the reticular form is the most common.
Dx: 4mm punch Bx shows hyperkeratosis with lymphocytic infiltrate.
Tx: prednisone 20-40 mg/d X 2wks if acute erosive.
Typical Laddered Protocol:

(1) topical corticosteroids, 0.05% fluocinonide gel 2 to 3 times daily + 0.5% clobetasol gel 2 to 5 times daily.
(2) intralesional corticosteroid injection (10 mg/kg; 0.2 - 0.3 mL).
(3) topical 0.1% tacrolimus ointment, 2 to 3 times daily, alternating with clobetasol.
(4) hydroxychloroquine, 200 mg PO BID.
(5) retinoid, including isotretinoin, 80 mg/day, for a 70-kg pt and acetretin, 25 mg/day, for a 70-kg pt.
(6) methotrexate, 2.5 to 12.5 mg/week, based on creatinine clearance. OLP and oral erosive lichen planus (OELP) usually responds to methotrexate (Arch Dermatol. 2007;143:511-515,519-523).
(7) thalidomide, 100 mg every night on average.
Topical Corticosteroids: lead to complete remission in 50% to 75% of pt's, but it is often accompanied by atrophy, telangiectasis and tissue fragility, and can promote infection such as acute candidiasis. Clobetasol (Orabase, high-potency) may be helpful (Oral Dis 1999;5:44). Intralesional injections of corticosteroids may be used to treat localized, recalcitrant lesions. Patients with symptomatic oral lichen planus respond equally well to betamethasone oral mini-pulse therapy (5 mg orally on 2 consecutive days per week x 3 mo, then taper) and to topical triamcinolone acetonide (0.1% oral paste applied TID), but they may respond more quickly to betamethasone according to a randomized comparative study (J Am Acad Dermatol 2008;58:596-602).
Tacrolimus mouthwashes (rinse and spit) @0.1mg/100ml of distilled water QID x6mo showed rapid improvement (Arch Derm 2002;138:1335-38).
Topical isotretinoin gel (0.1% x 8wks) may be an effective alternative to corticosteroids, although relapses often occur after discontinuance of this medication (Arch Dermatol 1986;122:534). Oral erosive lichen planus responds relatively well to applications of 1% pimecrolimus cream, and the tx is well-tolerated (Arch Dermatol 2007;143:472-476).
Hydroxychloroquine (Plaquenil) @ 200-400 mg qd X6mo was effective in 90% in one study (J Am Acad Dermatol 1993;28:609).
Other: Avoid tobacco and excessive ETOH, use sunscreen lip balm, annual exam. Replacing amalgam or gold restorations must be considered in extreme cases. Squamous cell carcinoma is associated with up to 5% of cases, most on the buccal mucosa or tongue. Low-dose excimer laser (308-nm) is effective for the tx (Arch Dermatol. 2004;140:415-420).
Etiology: Possibly due to a humoral and cell-mediated immune response induced by alterations in basal keratinocytes. Hep C is associated with severe cases.
Amalgam: Dental amalgam may cause OLP lesions (Arch Dermatol 2004;140:1434-1438, 1524-1525), removal of the amalgam can resolve the lesions if the patch test to mercury (the most important allergen in amalgam) yields positive results. Removal should be considered based on objective evidence in conjunction with symptomatic lichenoid tissue changes in apposition to or close to a dental metal restoration.
Possible drug induced: beta blockers, NSAIDs, methyldopa, penicillamine, quinidine, and quinine. Less common with ACEi's, sulfonylurea agents, carbamazepine, gold, and lithium (Drug-induced lichen planus. Pharmacotherapy 1994;14:561).

Oral Carcinoma:

Oral cancer makes up ~3% of cancers and 40% of ENT cancers in the USA. 50% have oncogenic HPV DNA. Painless, indurated ulcers with firm, raised borders, may have leukoplakia (a white keratotic plaque that cannot be removed by manual manipulation), erythema/ erythroplakia (red, velvety plaque that cannot be ascribed clinically or pathologically to any other predetermined condition), bleeding or erosions. The oral cavity extends from the skin-vermilion junctions of the anterior lips to the junction of the hard and soft palates above and to the line of circumvallate papillae below. Usually on the dorsum of the tongue, lower lip or floor of mouth. Most head and neck cancers are of the squamous cell variety and may be preceded by various precancerous lesions. Minor salivary gland tumors are not uncommon in these sites. Specimens removed from the lesions may show the carcinomas to be noninvasive, in which case the term "carcinoma-in-situ" is applied. An invasive carcinoma will be either well-differentiated, moderately well-differentiated, poorly differentiated or undifferentiated.
Tumor grading is recommended using Broder's classification:
G1: well-differentiated.
G2: moderately well-differentiated.
G3: poorly differentiated.
G4: undifferentiated.
Squamous Ca: 96% of all oral cavity cancers are SCC. Peaks in 60's, insidious growth, may ulcerate, lateral tongue (40%) > floor> gingiva> buccal> palate. Virtually all oral squamous cell carcinomas arise from a premalignant precursor, studies have found that 16-40% of oral premalignant lesions (OPLs) transform to cancer (Am J Epidemiol 2006;164:556-566). The incidence of oral squamous cell carcinomas potentially related to HPV appears to be increasing in the United States (J Clin Oncol 2008;26:612-619)....HPV-related OSCC appeared more amenable to radiation tx.
Vermillion carcinoma: predilection for the lower lip. Usually in fair-skinned individuals with actinic damage. Often starts as actinic keratosis with atrophy and dry, scaly area, next progresses to ulceration that may appear to partially heal, then recur (pt mistakes them as fever blisters), next it becomes crusted, nontender and indurated ulcer/mass.
Verrucous carcinoma: low-grade variant of oral SCC. 3% of invasive oral Ca. Associated with long-term smokeless tobacco use. Slow growing, no mets.
Risks: tobacco/ ETOH use, genetics, HPV, EBV.
S/s of Malignancy: painless red, white, **or** mixed red and white areas of the oral mucosa. As grows, it may become an exophytic mass with a fungating or papillary surface. It may be ulcerated or indurated. Red & white lesions (erythroplakia) display cellular atypia in 93% of cases and need ASAP Bx. White lesions (leukoplakia) are only malignant/ pre-malignant in 10% of cases and can be closely monitored (Clin Geriatrics 2002;10:2). Pain is not a reliable indicator of malignancy. Using a tongue-blade to depress the tongue actually hides the lesions as most on the post-lat border and ventral (under) surface. Best to use cotton gauze to grasp the tip of the tongue and pull it up and to each side. Chemiluminescent lighting and toluidine blue staining (TBlue630, metachromatic dye) as an adjunct to conventional oral visual examination may improve the identification of oral mucosal lesions and reduce the number of required biopsies (Multidisciplinary Head and Neck Cancer Symposium: Abstract 12. Presented January 19, 2007).
Dysplasia: a histologically abnormal epithelial development, have inc nuclear-cytoplasmic ratio, in rate of mitotic figures, cellular pleomorphism, nuclear hyperchromatism and abnormal progression of basal cells to the more superficial layers. Use of staining with the fluorescein derivative rose bengal along with shade evaluation appears to be useful in finding of precancerous and

malignant oral lesions (Int J Cancer 2007;120:1958-1963)....detected epithelial dysplasia and oral squamous cell carcinoma with a sensitivity of 93.9% and a specificity of 73.7%.
Metastases: usually to ipsilateral cervical nodes. Can be to contralateral side if tumor of the base of the tongue, advanced, near midline. Involved nodes usually enlarged, firm, nontender. If the capsule has been invaded and the surrounding connective tissue is involved (extracapsular spread) node may feel fixed and immovable.
Dx: Tissue Bx. An oral rinse could be used to detect head and neck squamous cell carcinoma by detecting hypermethylated genes in abnormal cells that are shed into the saliva (Clin Cancer Res. 2008;14:97-107). One can also use a brush biopsy kit has a reported spec & sens of 98% & 98% (Int Med News 2009;15 Oct).....using a "OralCDx kit" via ww.sopreventable.com or 1-877-712-7874, cost $17 per kit).
Tx: depends upon the site and extent of the primary tumor and the status of lymph nodes, may include surgery alone, radiation therapy alone, or a combination of the two. Early cancers (stages I and II) of the lip and oral cavity are highly curable by surgery or by radiation therapy, and the choice of tx is dictated by the anticipated functional and cosmetic results of tx and by the availability of the particular expertise required of the surgeon or radiation therapist for the individual pt. The presence of a positive margin or a tumor depth greater than 5 millimeters significantly increases the risk of local recurrence and suggests that combined modality tx may be beneficial. Advanced cancers (stages III and IV) of the lip and oral cavity represent a wide spectrum of challenges, most pt's are candidates for tx by a combination of surgery and radiation therapy.
Ddx: benign lesions (lichen planus, pyogenic granuloma, extragenital chancre, TB, benign papilloma, keratoacanthoma, follicular lymphoid hyperplasia, granular cell tumor, necrotizing sialometaplasia), malignant (melanoma, lymphoma, sarcoma, metastatic tumor, minor salivary gland malignancy).
Stage Information:
The staging systems are all clinical staging, based on the best possible estimate of the extent of disease before tx. The assessment of the primary tumor is based on inspection and palpation when possible and by both indirect mirror examination and direct endoscopy when necessary. The tumor must be confirmed histologically, and any other pathologic data obtained on biopsy may be included. The appropriate nodal drainage areas are examined by careful palpation. MRI offers an advantage over CT scans in the detection and localization of head and neck tumors and in the distinction of lymph nodes from blood vessels. If a pt relapses, complete restaging must be done to select the appropriate additional therapy.
TNM classification:
…….END DEMO of this chapter.

Geriatrics and Death & Dying:

Edited by Erno S. Daniel, MD, PhD, Internal and Geriatric Medicine.

Contents: Intro & Aging | **Approach** | Prevention | **Competency & Driving** | Exercise | **Rx Meds** | Long-Term Care | **Adaptive Equipment** | *Geriatric SYNDROMES* | **Nutrition** | Delirium, Dementia & Memory | **Skin Ulcers** | Advanced Care | **Palliative** | Hospice | **Dying Process** | Bad News | ***INDEX*** |

Introduction to Geriatrics:

Links: Life Expectancy | **Aging Theories** | Self-reported Health Status & Successful Aging | **Physical & Physiologic Changes** | Approach to Elderly | **Geriatric Syndromes** |

Geriatrics is the medical specialty that is concerned with the physiological and pathological aspects of medical problems and care of the aged.
Definitions:
The **young old-->** 65-74yo.
The **elderly (old, "middle age" old) -->** 75-85yo.
The **older-old-->** >85yo. ~1 in 10,000 in the general population lives to be 100 years old.
Supercenturians: age ≥110. Seen in 1 in 3 million. There are roughly 560 of these worldwide and 90% are women.
Frail elderly: older adults or aged individuals who are lacking in general strength and are unusually susceptible to disease or to other infirmity.

Age & Life Expectancy: See Antiaging Strategies |

0 **-->** 77.9yo = the life expectancy of Americans in 2004. Women @ 80.4 yrs; men @ 75.2 yrs.
65 **-->** +18yr = 83 yo.
75 **-->** +12yr = 87 yo.
85 **-->** +7yr = 92 yo.
90 **-->** +4yrs.
100 **-->** +2yrs. (1992 Brody and Cansel).
Average Years Left to Live based on health status:
Men:
Age 70 75 80 85 90 95 years old.
Healthy 18.0 14.2 10.8 7.9 5.8 4.3 years.
Average 12.4 9.3 6.7 4.7 3.2 2.3 years.
Frail 6.7 4.9 3.3 2.2 1.5 1 year(s).
Women
Age 70 75 80 85 90 95 years old.
Healthy 21.3 17.0 13.0 9.6 6.8 4.8 years.
Average 15.7 11.9 8.6 5.9 3.9 2.7 years.
Frail 9.5 6.8 4.6 2.9 1.8 1.7 years. You're only as old as you feel. Your biological age is a much greater determinant of your longevity and vitality than your chronological age, so take control of your health sooner rather than later to lower the age that really matters and to feel younger. Your chronological age is your actual age in years from the date of your birth; however, what really matters is your physiological, or biological, age. This latter age is an estimate of your well-being and general health compared to others of your age and to those who are younger or older than you.
Biomarkers Indicative of Biological Aging:

• Cardiovascular function (blood pressure, heart strength).
• Metabolic activity (blood glucose and cholesterol levels).
• Maximal aerobic capacity (exercise stress testing).
• Muscular strength (hand grip strength).
• Breathing capacity (forced vital capacity and expiratory volume).
• Bone mineral density.
• Skin elasticity.
• Mental function (cognitive abilities, including memory).
• Systemic inflammation (measured by markers in blood).
• Reaction time (nerve conduction velocity).

Info: The US census reports estimated that the number of US citizens over the age of 65 will increase from 12.9% of the population in 2005 to 20% in 2030. The group >85yo is projected to be the fastest growing part of the elderly population. The majority of the older population in the US lives in the farm belt states in the midwest. Social status may influence longevity as Academy Award winners live an average of 4yr longer than other actors/ actresses (Ann Intern Med 2001;134:10). Although males make up only 15% of centenarians, they are better off compared to their female counterparts in terms of physical and cognitive function. Children of centenarians have markedly reduced relative risks for age-related diseases, particularly heart dz, HTN and DM (Ann Int Med 2003;139:445-49). Studies on Scandinavian twins indicate that there is a 25% heritability in life expectancy (75% variation due to environment (J Gerontol Bio Med Sci 1998;53:M441-6). A study on 7th Day Adventists shows that optimal health-related behaviors adds 10 years to the average life expectancy (Arch Int Med 2001;161:1645-52). Individual with exceptional longevity have significantly larger HDL and LDL particle size that is associated with lower prevalence of HTN, cardiovascular dz, metabolic syndrome and increased homozygosity for the 1405V variant in the cholesterol ester transfer protein (CETP) gene (JAMA 2003;290:2030-40). The 10-year mortality rates, regardless of cause, are 50% lower in persons who consume a Mediterranean diet, do not smoke, use alcohol moderately, and are physically active (JAMA 2004;292:1433-9). Modifiable healthy behaviors during the early elderly years, including smoking abstinence, weight management, blood pressure control, and regular exercise, are linked to enhanced life span in men as well as to good health and function during older age according to data from the Physicians' Health Study (Arch Intern Med. 2008;168:262-263, 284-290)....Specific increased risks for death before age 90 were as follows: Smoking, HR 2.10; Diabetes, HR 1.86; Obesity (BMI of at least 30), HR 1.44; Hypertension, HR 1.28; Exercise, HR 0.72; Alcohol intake did not significantly predict survival....The probability of reaching 90 was 54% in the absence of smoking, diabetes, obesity, hypertension, or sedentary lifestyle, the researchers found....Among those with all five factors, the probability of surviving to 90 was 4%. Four simple lifestyle changes -- stopping smoking, limited alcohol use (1-4 units/wk), getting regular exercise, and eating a diet rich in fruits and vegetables (5 servings/d) give an average of 14 years longer than those who didn't adopt any of those lifestyle practices according to data from the EPIC-Norfolk Prospective Population Study with 20,244 men and women (PLoS Med 2008; doi:10.1371/journal.pmed.0050012). Exercise and moderate alcohol intake (<14 drinks/wk) were correlated with lower death rates over a 20-year period according to data on 11,914 participants in the Copenhagen City Heart Study (European Heart Journal 2008; DOI: 10.1093/eurheartj/ehm574)...."neither physical activity alone nor alcohol intake can completely reverse the increased risk associated with physical inactivity and alcohol abstention."

Genetics: How much your genes matter. Your genes apparently determine 20 to 50% of how long you will be alive. In the United States, women live about 5 years longer than men.

Women: In Westernized societies women outlive men by 5 to 6 years on average. Epidemiological evidence suggests that nearly 4 of these extra years are due to intrinsic differences between the sexes while the remining difference has developed during the twentieth century due to extrinsic effects. Women on average live 80.1 years versus 74.8 years in men. Black men on average living 6.2 years < white men and black women 4.4 years < white women. The olded living human recored was 122 yo (Jeanne Clament, a French woman), this max age has unchanged despite the increase in the **average** human ife span. Even though women live longer, they are also more prone to severe chronic illness, but not the life-threatening illnesses than do men. There is a paradox in that although women outlive men they are much more likely to become disabled and dependent in old age. The physical basis for this probably lies largely with sex differences in muscle bulk rather than particular disease entities. There are various hypothesis as to the sex differences in longevity (more active immune system, estrogen levels higher, hetergametic sex [XY men do not have an extra set of nearly 1,000 + genes], men have more oxidative stresses, lower growth hormone and insulin-like growth factor activity) (Gender Med 2006;3:79-92) (comparative biology rules out most except the heterogametic sex hypothesis). A nomogram for exercise capacity specific to women (predicted MET = 14.7 – [0.13 x age]), using a cutoff of 85% of predicted capacity, is predictive of overall and cardiac mortality in both asymptomatic and symptomatic women (NEJM 2005;353:468-475) (confirms the critical importance of fitness to health and longevity). The life expectancy (LE) for US men (74.4 years) and women (79.8 years) at birth rank 26th according to an analysis of 2001 data from 37 countries (MMWR. 2006;55;621-644)...LE was highest among Japanese women (84.9 years) and lowest for Russian men (59.1 years); the difference between the sexes ranged from 4.3 years (Costa Rica, 79.9 vs 75.6 years) to 13.2 years (Russia, 72.3 vs 59.1 years). In the very old (>70yo), IL-6 and the number of carotid plaques (focal widening with protrusion, no the size) are powerful predictors of mortality risk in the years to come (Am J Med 2006;119:519-25) (CIMT and other typical CV risk factors were not predictive). Individuals with long-lived parents have advantageous cardiovascular risk profiles in middle age compared with those whose parents died younger according to data from the Framingham Heart Study (Arch Intern Med 2007; 167:438-444).

Aging Theories:

Links: Antiaging Strategies | **Successful Aging |**

Aging is the decline in physiologic function. The major theories about aging include free-radical damage hypothesis, DNA damage, spontaneous mutagenesis, the neuroendocrine pacemaker and apoptosis theory. Iron is a critical catalyst in free-radical productions, iron deficiency anemia is associated with lower oxidized LDL and subsequently less atherosclerotic plaque production (J Intern Med 1995;237:161-8). "Anti-inflammatory" genotype associated with longevity, men who live to age 100 are more likely than younger men to be homozygous for a single nucleotide polymorphism in a gene associated with transcription of interleukin (IL)-10, either separately or in association with another polymorphism in the gene regulating TNF-alpha (J Med Genet 2003;40:296-299). In nematode and fruit fly models, reducing insulin or insulin-like growth factor (IGF) signaling extends life span (Science 2007;317:369).....Insulin receptor substrates (IRSs) are phosphorylated by an insulin receptor after it is activated by insulin or IGF-1. Phosphorylated IRS activates a variety of downstream signaling pathways, which regulate various cellular functions.....mice with

systemic Irs2 hemizygous deficiency were more insulin-sensitive and glucose-tolerant than wild-type mice, while those with brain-specific Irs2 deficiency were more insulin-resistant, hyperinsulinemic, and glucose-intolerant.....the mutant mice were more active and displayed more "youthful" metabolic characteristics, including optimal levels of superoxidase dismutase, an enzyme that protects cells from oxidative stress.

Apoptosis: Genetically programmed cell death that is characterized as organized nuclear and cellular fragmentation. Nature's way of eliminating unwanted, senescent, and damaged cells from an multicellular organism. Apoptosis has been implicated in various physiologic functions including the remodeling of tissues during development, removal of senescent cells and cells with genetic damage beyond repair, and maintenance of tissue homeostasis. Developmentally this helps sculpture our organs and carve out the interdigital webs of the fingers and toes. The cells are fragmented into small membrane-bound bodies with intact organelles and plasma membrane. (Apoptosis and Liver Disease. Am J Med 2000;108) The best-known marker of aging is the length of telomeres in cells: when telomeres (the spindles at the tips of chromosomes) become very short, the probability of cell death increases greatly (senescent)(Science 2006;311:1257). Many stimuli activate the process of apoptosis, including DNA damage through ionizing radiation, growth factor and nutritional deprivation, activation of certain death receptors (e.g., Fas receptor [FasR] and tumor necrosis factor receptor [TNF-R1]), metabolic or cell cycle perturbations, oxidative stress, and many chemotherapeutic agents.

Antiaging Strategies:

Link: See **Aging Theories** |

For most pt's who are seeking to prevent the effects of aging, healthy lifestyle changes will have more benefit than any ointment, pill, or dietary supplement. Lack of exercise exaggerates the effects of aging. Other strategies for making lifestyle changes include engaging in volunteer work, using mind-body techniques and other forms of self-examination, or simply reducing stress and instilling a feeling of purpose and ease in the world.

Exercise: See Exercise | Following a regular exercise routine through middle age can delay biological aging by up to 12 years (Brit J Sports Med. 2008;Published online April 10)....A 14% increase in risk for dependency is seen with each 1-mL/(kg x minute) decrease in maximal aerobic power....and fatigue is more likely if effort in an 8-hour day demanded more than 40% to 50% of an individual's maximal aerobic power. One of the most important factors influencing the quality of life in very old people was the maintenance of sufficient aerobic power to allow independent living, and depending on the living environment and the degree of social support that was available, the limiting maximal aerobic power was estimated to be 12–15 ml/[kg.min]....During most of adult life, aging is typically associated with a decrease in maximal oxygen intake of about 5 mL/(kg x minute) per decade.

Recombinant human growth hormone (hGH): SQ 3 times a week can increase lean body mass, decreased fat mass, and improved maximum oxygen uptake, however, these benefits are frequently accompanied by significant side effects, including diabetes and glucose intolerance, carpal tunnel syndrome (38%), peripheral edema (39%), elevated trig's, and joint pain and cost up to $400 per month (JAMA 2005;294:16:2086-90) (initially at a dose of 30 ug/kg 3x/wk, then reduced to 20 ug/kg.

Dehydroepiandrosterone (DHEA): a sex steroid that converts to estrogen and testosterone, has shown potential benefit in pt's with depression and adrenal insufficiency and in increasing bone density and libido in postmenopausal women, but no consistent benefit has been found for men and most studies have been small and insufficient and have conflicted with others that suggest various risks from DHEA use.

Caloric restriction (CR): Long been known to extend life and maintain health. Studies in virtually all animals — yeast, worms, flies, mammals — show that severe calorie restriction enhances vitality, lengthens life, and improves memory function. Research on mice and rats has shown that stringent and consistent caloric restriction increases the animals' maximum lifespan by about 30% and protects them against atherosclerosis and cancer. Initially thought to work by slowing down the metabolic process and thereby reducing the incidence of oxidative stress that is a by-product of metabolism. Now shown that it leads to changes in hormone levels in the blood stream and a slowing of aging on all organs. Caloric restriction is the only proven therapy to lengthen the average and max lifespan. It requires a 30-60% reduction in the calories. Long-term calorie restriction (1,400 to 2,000 cal/d x 6yrs giving 7% total body fat Vs 25% in controls) with optimal nutrition has cardiac-specific effects that ameliorate age-associated declines in heart function (based on U/S exam showing improved diastolic function / relaxation, lower CRP & TNFa) (Long-term caloric restriction ameliorates the decline in diastolic function in humans. J Am Coll Card 2006;47:398-402). Calorie restriction (25% of baseline energy requirements) improves biomarkers of longevity (DNA damage and metabolic adaptation)(also gave a reduction in weight, fat mass, fasting insulin, and core body temperature) (JAMA. 2006;295:1539-1548). Calorie restriction causes beneficial metabolic, hormonal and functional change (JAMA 2007;297:986-94). Research with fruits flies suggests that, contrary to previous reports, diet restriction may not increase lifespan as it lowered resistance to Listeria monocytogenes (PLoS Biol 2009;Jul 20)....The authors speculate that diet restriction has a complicated effect on immunity; it increases survival against some microbes but reduces it against others."....Most past studies haven't focused on the effects of diet restriction on immunity, and when they have, they haven't looked carefully at the split between resistance and tolerance." A low-calorie (30% restriction) but nutritious diet gave a group of rhesus monkeys an advantage over their normally fed pals (Science 2009;325:201).....Rates of cancer and cardiovascular disease were 50% lower in the CR monkeys. As expected, the CR monkeys exhibited better insulin sensitivity, lower body weight, much lower fat mass, and slower declines in muscle mass. They also had better preservation of gray matter in parts of the brain that are associated with executive function. Scientists have discovered a gene (S6K1) that may affect longevity and leanness (Science 2009;326:140-144).....In lab tests, the typical life span of mice lacking the S6K1 gene was 9% longer and the maximum life span was 10% longer than mice with that gene intact.

Hormonal therapies with estrogen, testosterone, GH replacement are still investigational and may increase cancer risks. Analysis of 31 studies suggest that growth hormone (ave dose 14 g/kg/day x 27 nweeks) is not a useful tx for "setting back the clock" in elderly individuals as any beneficial changes in body composition the hormone produces are small and side effects are common (Ann Intern Med 2007;146:104-115).

Cell-based therapies with gonadal extracts and cell transplantation are mostly anecdotal, but may have disease specific benefits (BM transplant).

Genetic manipulation after identifying aging-related intracellular RNA, chromosomal and gene damage are still investigational.

Other: Two drugs that are approved to treat absence seizures, trimethadione and ethosuximide can extend the lifespan of the Caenorhabditis elegans worm (trimethadione extended it by 57%) and can improve various critical physiologic functions. Drug concentrations equivalent to those already approved for human use achieved these effects (Anticonvulsant medications extend worm life-span. Science 2005;307:258-62).

Cholesterol ester transfer protein (CETP) gene: associated with longer life as well as increased HDL levels and LDL particle size, is associated with less dementia and improved memory. A study in people of Ashkenazi Jewish descent older than 95 years of age shows a variant of the CRPT VV (1405V homozygosity) genotype is increased 3-fold in those surviving into advanced age and it is protection against age-related cognitive decline (Neurology. 2006;67:2170-2175). Found 25% in a group of subjects with an average age of 100 vs 8% in those aged 30 years or younger. The CETP VV genotype is associated with low plasma levels of CETP, a protein involved in reverse cholesterol transport. It mediates the exchange of cholesterol esters between HDL and apoB-containing lipoproteins, thereby promoting the uptake of cholesterol by the liver. Low CETP is also associated with large LDL and HDL particles. See "HDL" in endocrine module for meds that affect CETP.
Resveratrol: a natural compound found in red wine, grapes, and nuts, has been shown to prevent many adverse metabolic effects of a high-fat diet and to increase survival in mice (Nature 2006; DOI:10.1038/nature05354). Resveratrol is a molecule produced in plants, mimics the effect of caloric restriction in enhancing vitality, protecting against decreased insulin sensitivity, and increasing lifespan in various "simple" animals. Caloric restriction extends the lifespan of many animals, through its effects on enzymes called the sirtuins that are preserved throughout the animal kingdom. See "anti-oxidants" in compendium module. Oligomeric procyanidins are the likely ingredient in red wine's polyphenols that contributes to heart health and longevity (Nature 2006;444:566).
Rapamycin: Mice lived longer (30% extension of life span) when they received this pharmacologic agent — even when it was started late in life (Nature 2009;Jul 8:e-pub ahead of print).....This report identifies another nutrient-responsive molecular pathway (the mTOR pathway) that influences life span in mammals, and it demonstrates that inhibiting the pathway with an available pharmacologic agent (rapamycin) can extend life span.
Common Reversible Functional Causes of Premature Aging: Oxidative stress and impaired mitochondrial function. Glycation (dysglycemia) and insulin resistance. Chronic inflammation. Altered methylation reactions (deficiencies in B6, B12 and folic acid). Lipid atherogenesis (cholesterol). Compromised detoxification capacity (diminished function of liver and GI tract). Poor immune function (food intolerances). Chronic stress response. Hormone imbalance (less DHEA, melatonin, estradiol, progesterone, testosterone). Physical and mental inactivity.
Werner syndrome (Pangeria): a condition featuring multiple signs of premature aging. Telomere loss and consequent genomic instability appears to play a key role (Science 2004;306:1951-1953).
Dyskeratosis Congenita: Features include specific muco-cutaneous traits, bone marrow failure, early graying of hair, dental loss, osteoporosis, and malignancy. It is genetically heterogeneous, with X-linked, autosomal dominant, and autosomal recessive subtypes. Mutations leading to the premature aging syndrome dyskeratosis congenita are associated with low blood levels of telomerase RNA, confirming the underlying role of shortened telomeres in the condition (Proc Natl Acad Sci USA 2008;online on June 2).
Hutchinson-Gilford Progeria Syndrome (HGPS): Certain aspects of aging appear to be accelerated in HGPS. HGPS is a rare genetic disorder that causes rapid, premature aging and early death from cardiovascular disease and stroke (median age at death, 13 years). It is possible, therefore, that the Hutchinson-Gilford progeria syndrome might serve as a model for study of the normal aging process. HGPS is almost always caused by a de novo point mutation in the lamin A gene (LMNA) that produces a truncated mutant protein called progerin. Progerin disrupts the nuclear membrane, alters transcription, and appears to accumulate in cells with normal aging. A prospective study on 15 pt's revealed findings of prolonged prothrombin times, elevated platelet counts and serum phosphorus levels, decreased range of joint motion, farsightedness, low-frequency conductive hearing loss, speech deficits, and oral motor and dental abnormalities (NEJM 2008;358:592)....this is in addition to the classic clinical features of HGPS: sclerotic skin, joint contractures, bone abnormalities, alopecia, decreased body fat, and growth impairment.....Cardiovascular disease, transient ischemic attack, seizures, and arteriosclerosis were also documented in these patients. The accelerated aging results from abnormal lamin A, or progerin, which is produced subsequent to a change from glycine GGC to glycine GGT in codon 608 of exon 11 of the lamin A (LMNA) gene....Progerin disrupts the structural integrity of the inner nuclear membrane in a dominant negative fashion. There is evidence of molecular overlap between the syndrome and normal aging and fibroblasts from an older person contain more progerin than fibroblasts from a younger person.
Stress: The chronic stress that spouses and children develop while caring for Alzheimer's disease pt's may shorten the caregivers' lives by as much as 4 to 8 years (J Immunol 2007;Sept)...."Earlier work by other researchers had shown that mothers caring for chronically ill children developed changes in their chromosomes that effectively amounted to several years of additional aging among those caregivers".
Telomere Length: Twin studies find that adults who are physically active in their leisure time have significantly longer leukocyte telomeres (P<0.001) than those who are more sedentary (Arch Intern Med 2008;168:154-158)....Leukocyte telomere dynamics (telomere length and age-dependent attrition rate) are ostensibly a biological indicator of human aging. Telomeres are areas of genetic material on the ends of a cell's chromosomes. Over time, as a cell divides, those telomeres shorten, losing genetic instructions. The enzyme telomerase normally works to repair that damage to the chromosome. Telomeres are like caps on the chromosome, they insulate and protect the ends of the chromosomes....like frayed rope, if the caps weren't there, the rope would unravel. As we get older, the telomeres shorten and the activity of the telomerase enzyme lessens as part of the normal aging process. Telomeres gradually wear down with each round of cell replication, thereby shortening with age. Processes like oxidative stress and inflammation -- which increases leukocyte turnover -- can speed up the erosion process. The most active individuals were found to have telomeres of the same length as sedentary individuals 10 years younger according to a study study with 2400 subjects (mean age, 49)(Arch Intern Med 2008;168:154)(after controlling for age, sex, body-mass index, smoking and socioeconomic status).....Telomeres are repeated DNA sequences that appear to protect chromosomes from degradation; this process may be central to aging.....Leukocyte telomere length decreased by a mean of 21 nucleotides for each additional year of life….Exercise | Comprehensive healthy lifestyle changes substantially increase telomerase activity, and hence telomere maintenance capacity, in as little as 3 months, according to results of a prospective pilot study (Lancet Oncology 2008;Sept 16)....."Telomere shortness in humans is emerging as a prognostic marker of disease risk, progression, and premature mortality, fully functioning telomerase, preserves not only telomere length, but also healthy cell function and long-term immune function and is inversely associated with major cardiovascular disease risk factors."

Self Reported Health Status:

Link: ADL's | **Approach to Elderly Pt** | Life Exectancy and Aging | **Exercise** | Antiaging Strategies |
Successful aging is defined as an optimal state of overall functioning and well-being, not just the absence of dz or infirmity. Only a happy few (10-13%) age >85 yo meet this criteria (Arch Intern Med 2001;161:2694).

"Excellent" --> have a younger physiologic age.
"Good/fair" --> physiologic age is comparable to chronologic age.
"Poor" --> physiologic age is older than chronologic age. One of the hallmarks of aging is the need for help with personal care or household activities. This occurs in 5% by age 65 and 50% by age 90yo. It is estimated that with each loss of an ADL, there is a dec 2yr survival. While only 20% of people >85yo live in nursing homes, only about 50% at this age range are independent in their activities of self-care. In pt's >75yo, CRP, IL-6 and number of carotid plaques are the most powerful predictors of all-cause and CV mortality risks in the years to come (Prediction of Mortality Risk in the Elderly. Am J Med 2006;119:519-525)...conventional risk scores (the Framingham Risk Score & PROCAM Risk Function) seem to perform unsatisfactorily in this age group.

Successful Aging:

Satisfaction with one's past and present life has been the most commonly proposed definition of successful aging. Successful aging varies from person to person, but there are a few things that are consistent with successful aging. Many people define it as staying engaged with life — being physically and mentally active. People that age successfully are those who are truly resilient and can roll with the waves of life. These people can accept the aging process as well as the diseases or conditions they may have, they optimize their physical and mental abilities, and they are able to move on. To age successfully takes both resilience and motivation. It means practicing acceptance and optimizing what you've got. It doesn't necessarily mean being physically fit beyond your years. Physical activity strengthens the mind and the body. One needs to actively use their mind by taking on challenges and realistically looking at your successes. Having social supports and knowing when to ask for help when you need it. Haveing resilience, the capacity to spring back from physical, emotional, financial, or social challenges. Adhering to a healthy diet and maintaining an optimum weight. Engage in activities that stimulate our minds and challenge us. Sitting around doing a crossword puzzle — if you love doing that — is not a challenge. What you need to do is challenge yourself to the next level. Learn to do crossword puzzles in French, or learn to speak French. Don't just stay in your comfort zone, instead of push out of it both physically and mentally. Older adults' definition of successful aging is multidimensional, encompassing physical, functional, psychological, and social health (Older Adults' Views of "Successful Aging". J Am Geriatr Soc 2004;52:211-216). Overall, 90% of respondents had given previous thought to aging and aging successfully. Overall, about two-thirds (67% of Japanese Americans and 63% of whites) reported that their thoughts about successful aging had changed over the past 20 years. The goal of geriatric therapy is to relieve suffering and to improve function.
75% or more respondents in both cohorts rated this item as important in successful aging: (Japanese American and white). Remaining in good health until close to death. Feeling satisfied with my life the majority of the time. Having friends and family who are there for me. Feeling good about myself. Not feeling lonely or isolated. Being able to meet all of my needs and some of my wants. Being able to act according to my own inner standards and values. Being able to take care of myself until close to the time of my death. Adjusting to changes that are related to aging. Staying involved with the world and people around me. Being able to make choices about things that affect how I age, like my diet, exercise, and smoking. Being able to cope with the challenges of my later years. Remaining free of chronic disease.*
75% or more respondents in the Caucasian-American cohort rated this item as important in successful aging: Continuing to learn new things.
Neutral Items: Living a very long time. Having the kind of genes (heredity) that help me age well. Having a sense of peace when thinking about the fact that I will not live forever. Feeling that I have been able to influence others' lives in positive ways. Having no regrets about how I have lived my life. Being able to work in paid or volunteer activities after usual retirement age (65).
Main constituents of successful ageing: (What is successful ageing and who should define it. BMJ 2005;331:1548-1551)
Theoretical definitions: Life expectancy. Life satisfaction and wellbeing (includes happiness and contentment). Mental and psychological health, cognitive function. Personal growth, learning new things. Physical health and functioning, independent functioning. Psychological characteristics and resources, including perceived autonomy, control, independence, adaptability, coping, self esteem, positive outlook, goals, sense of self. Social, community, leisure activities, integration and participation. Social networks, support, participation, activity.
Additional lay definitions: Accomplishments. Enjoyment of diet. Financial security. Neighborhood. Physical appearance. Productivity and contribution to life. Sense of humor. Sense of purpose. Spirituality. Based on the laws of the universe, all things (including our DNA) tend toward disarray with time. The goal is to compress any significant morbidity into our final year of life such that we can enjoy our good health until then. Part of healthy aging is the acceptance that anti-aging is not the complete solution. It is well known tha cheese, wine, whiskey and other things smooth out and become more complex with time. Very old trees have a certain presence and beauty even though that may wear the battle scars of time.
Factors critical for a long life are: …….END DEMO of this chapter.

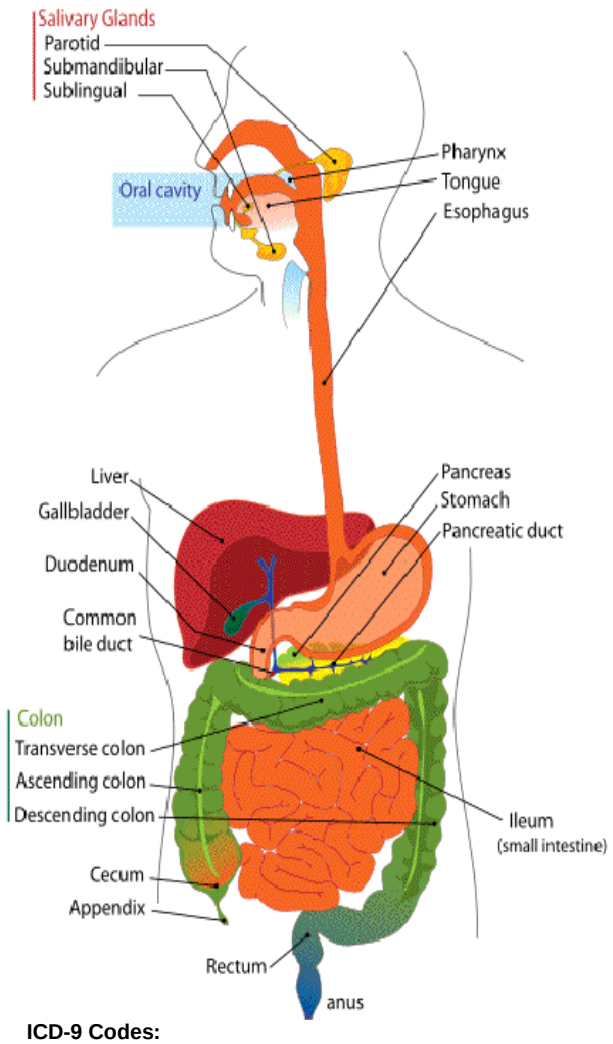
Gastroenterology:

Edited by Brent J. Kovacs MD, Gastroenterologist.

Contents: Abd Pain, Exam & Gut D/o's | **Hernias** | Spleen | **Biliary** | Liver | **Diarrhea & Enteritis** | Malabsorption | **Stomach / Esophagus** | GI Bleed | **Colon / Rectum / Anus** | Colorectal Ca & Polyps | **Inflammatory Bowel / Colitis** | Constipation | **Irritable Bowel** | MEDS | **PEDIATRIC** | Gallbladder | **CANCER** | Ascites | **Hiccups** | Nausea & Vomiting | **Enteral Supplements** | *INDEX* |

Abdominal Pain:

Links: Eval, Exam & Pearls | **Ddx-by location** | Nonsurgical / Chronic | **Pain in Elderly** | Pain in Women | **Distention / Masses** | Appendicitis | **Pancreatitis** | Pancreatic Ca & ERCP | **Gallbladder** | Diverticulitis | **Ischemic Colitis / Mesenteric Ischemia** | Obstructions (Ileus, SBO, Intussusception and Volvulus) | **Aortic Aneurysm** | Smal Bowel Obstruction (SBO) | **Mesenteric Venous Thrombosis** | Splenic Rupture | **Perforation / Peritonitis Abscess** | Psoas / Subdiaphragmatic Abscess | **Mesenteric Lipodystrophy** | SURGICAL Pearls | **Rectal** | Stomach / Esophagus | **Other** | Toxic Megacolon | **Hernias** | Pediatric | **Functional GI D/o's** | Bowel Prep | **Meckel's** | PEDIATRIC | **Lactose Intolerance** | Belching, Bloating & Gas |
GI DIAGRAM FOR Pt's:



ICD-9 Codes:

789.0 Abdominal pain - The following fifth-digit subclassification
- 0 unspecified site
- 1 right upper quadrant
- 2 left upper quadrant
- 3 right lower quadrant
- 4 left lower quadrant
- 5 periumbilic
- 6 epigastric
- 7 generalized
- 9 other specified site multiple sites

789.3 Abdominal or pelvic swelling, mass, or lump

789.6 Abdominal tenderness

Digestive System Physiology:

1. **Function:** Break down food so cells can be nourished.
2. **Mechanical digestion:** Various activities aid in presenting foods to the GI tract for absorption.

a. **Mastication (chewing):** Breaks down large particles, mixing them with saliva.

b. **Deglutition (swallowing):** Moves (via **peristalsis**) materials from mouth, to the pharynx, on to stomach.

c. **Gastric/intestinal motility:** Peristalsis and mixing movements (**segmentation**) facilitate formation of small particles for absorption (requires chemical digestion, too).

3. **Chemical digestion:** Digestive enzymes break down large **macromolecules** (proteins, lipids, carbohydrates) into their constituent parts.
4. **Absorption:** Most food molecules are absorbed in the **small intestine**.

- Some absorption occurs in large intestine (e.g., ions, water).
- Undigested materials (**feces**) are expelled (**defecation**) via the rectum.

5. **Control of digestive processes:** Complex interactions involving **hormones** and **neural reflexes** highly coordinate mechanical and chemical digestion to facilitate absorption.

Evaluation:

Links: Terminology | **Exam** | Peritoneal Signs | **Cancer Signs** | Liver | **Splenic exam** | Other exam | **W/u & Abd X-rays and Radiology Evals** | Flex Sig | **Colonoscopy** | EGD |

Evaluation of the Abdomen

Step #1 Hx: Need a painstakingly detailed hx. Start with a detailed review of the chronological order of events. Location, quality, intensity, duration, aggravating / alleviating factors, radiation, previous episodes, pain scale 1-10. Dysuria, BRBPR (bright-red-blood-per-rectum), N/V/D/C, F/C, PMHx (cardio-pulmonary), meds, ETOH use, GYN Hx (sexually active, LMP, vag d/c), drug allergies.

Onset & duration to max pain:

Minutes--> renal colic (flank or groin depending on level of obstruction, paroxysms last 20-60 min), ruptured viscous, dissection.

Minutes to hours--> pancreatitis, cholecystitis.

Hours--> appy, diverticulitis.

Pain improved by eating (PUD), sitting up (pancreatic), lying still (peritonitis).

Pain worse by eating can be GB, mesenteric ischemia, pancreatic or GERD.

Radiation of Pain: Referred pain is usually aching, close to the surface and has associated inc muscle tone and hyperalgesia.

Ex: gallbladder enters at T5-10, pain is perceived in the scapula, do not get localized RUQ pain until inflammation affects the parietal peritoneum.

Diffuse pain--> visceral pain or un contained peritonitis.

Biliary colic, spleen--> ipsilateral scapula.

Renal colic--> ipsilateral testicle/ groin.

Pancreatitis--> back, improves if lean forward, worse to lay back.

Parietal Pain: due to parietal peritoneal irritation, perceived through segmental somatic fibers. Causes reflex involuntary muscle wall rigidity, hypesthesia of the skin**-->** dull steady ache.

Visceral Pain: early pain, dull crampy, poorly localized secondary to distention/ compression or ischemia of autonomic "stretch" receptors on serosal surfaces. On mucosal surface from substance P released in response to ischemia or inflammation.

Somatic Pain: sharp, well-localized pain secondary to activation of parietal peritoneum receptors.

Referred Pain: pain felt distant to diseased organ because of common embryologic origin. **Ex:** Diaphragmatic irritation **-->** pain in lateral neck or shoulder. Pneumonia **-->** pain in RUQ or LUQ. AMI **-->** epigastric pain.

Terminology: Right Upper Quadrant (RUQ), Right Lower Quadrant (RLQ), Left Upper Quadrant (LUQ), Left Lower Quadrant (LLQ).

Midline: Epigastric, Periumbilical, Suprapubic.

Vomiting: the more proximal the obstruction, the earlier onset and inc frequency. May not occur with distal colonic obstruction and competent ileocecal valve.

Bilious--> nonspecific.

Food--> upper obstruction.

Retching--> torsion of viscus.

Feculent--> pathognomonic for intestinal obstruction (due to overgrowth of bacteria in stagnant small bowel, not regurgitation of feces).

Anorexia--> before pain is characteristic of appy.

Diarrhea--> VGE (viral gastroenteritis), appy in child, early/ partial SB obstruction, acute diverticulitis.

Constipation/ obstipation--> paralytic ileus, acute appy.

Pearls & Warnings: Belching, bloating and nausea are **not** specific for any condition. Fatty foods do **not** worsen GB sx's. Spicy foods do **not** worsen PUD. Giving narcotics or NSAIDs does **not** alter/ delay the dx in adults or children (JAMA 2006;296:1764-74)….. even though it may alter the physical exam. Change in bowel habit – think of colorectal cancer. Dark-red rectal bleeding could be caused by colorectal cancer or colitis. Lethargy and shortness of breath may indicate anaemia due to GI pathology. Weight loss and anorexia – think of abdominal malignancy. Black stool indicates upper GI bleeding. Sudden onset of severe abdominal pain may be a life-threatening perforation. A severe attack of ulcerative colitis with fever and abdominal pain may be life-threatening. A patient who is unwell soon after colorectal surgery has an anastomotic leak until proved otherwise. Lethargy in a patient with an ileostomy may herald dehydration and renal failure. Not all abdominal symptoms are caused by GI disease Abdominal distention in a patient with liver disease is worrying. Clinical deterioration in a patient with ascites – think of spontaneous bacterial peritonitis. Jaundice in the elderly is commonly malignant. A patient with liver disease who has difficulty concentrating may have early encephalopathy. Drowsiness in a patient with cirrhosis may have a life-threatening cause. Pallor and tachycardia in a cirrhotic patient may signal a variceal bleed, even without hematemesis or melena. Bruising in a patient with liver disease could mean deteriorating liver function. Generalized itch can be due to biliary obstruction, even in the absence of jaundice. Intermittent right upper quadrant pain and fever with jaundice – suspect cholangitis. Continuous abdominal pain and fever with retching – examine for guarding in the right upper quadrant. Source: (Alarm Bells in Medicine by Nadeem Ali. 2005 by Blackwell Publishing Ltd)

Step #2: The Exam:

The pt should be lying supine on the exam table and appropriately draped. Begin with inspection, then auscultation. The examination room must be quiet to perform adequate auscultation and percussion. Watch the pt's face for signs of discomfort during the examination. Use the diaphragm to listen for bowel sounds, and characterize as normal, increased, decreased, or absent, then listen for bruits over the renal arteries, iliac arteries, and aorta. Use in conjunction wth vital signs (VS's), orthostatics, look for distention, bowel sounds (BS's), masses, hernias, flank/CVA tenderness, pelvic/ rectal exam.

Check for peristaltic sounds **-->** In bowel obstruction the sounds may initially be high-pitched and in pseudo-obstruction the sounds are infrequent and weak-absent. Abdominal **wall pain** may be distinguished from visceral pain by having supine pt raise both heels off the table by 6 inches, increased pain is virtually diagnostic of abd wall pain. D/o's in the **chest** will often manifest with abd sx's. It is always wise to examine the chest when evaluating an abd complaint.

Step #1: Inspection: Look for scars, striae, hernias, vascular changes, lesions, or rashes. Look for movement associated with peristalsis or pulsations. Note the abdominal contour. Is it flat, scaphoid, or protuberant?
Always check the inguinal/ rectal examination in males and the pelvic/ rectal examination in females. Look for peripheral stigmata of chronic liver dz. Aways examine the chest when evaluating an abdominal complaint as disorders in the chest will often manifest with abd sx's.
Step #2: Percussion: in all four quadrants, categorize what you hear as tympanitic or dull. Tympany is normally present over most of the abdomen in the supine position. Unusual dullness may be a clue to an underlying abd mass.
Liver Span: Percuss downward from the chest in the right midclavicular line until you detect the top edge of liver dullness. Percuss upward from the abdomen in the same line until you detect the bottom edge of liver dullness. Measure the liver span between these two points. This measurement should be 6-12 cm in a normal adult.
Splenic Dullness: Percuss the lowest costal interspace in the left anterior axillary line. This area is normally tympanitic. Ask the pt to take a deep breath and percuss this area again. Dullness in this area is a sign of splenic enlargement.
Step #3: Start with light / superficial palpation: This helps relax the pt for deeper palpation. **Watch the pt's face** for signs of discomfort during the examination. Voluntary or involuntary guarding may also be present.
Ticklish Pt: start by placing their hands on the abdomen in a pain-free area. Place your hands on top and palpate several areas. Next place your hand on the abd with their hand on top of yours.
Deep palpation after surveying the abdomen lightly. Try to identify abd masses or areas of deep tenderness. Use counterpressure by using your free hand to support the pt's lower back.
Palpation of the Aorta: Press down deeply in the midline above the umbilicus. The aortic pulsation is easily felt on most individuals. A well defined, pulsatile mass, >3 cm across, suggests an aortic aneurysm.
Costovertebral Tenderness: CVA tenderness is often associated with renal disease. Warn the pt what you are about to do. Have the pt sit up on the exam table. Use the heel of your closed fist to strike the pt firmly over the costovertebral angles. Compare the left and right sides.
Shifting Dullness: This is a test for peritoneal fluid (ascites). Percuss the pt's abdomen to outline areas of dullness and tympany. Have the pt roll away from you. Percuss and again outline areas of dullness and tympany. If the dullness has shifted to areas of prior tympany, the pt may have excess peritoneal fluid.
Psoas Sign: This is a test for appendicitis. Place your hand above the pt's right knee. Ask the pt to flex the right hip against resistance. Increased abdominal pain indicates a positive psoas sign.
Obturator Sign: This is a test for appendicitis. Raise the pt's right leg with the knee flexed. Rotate the leg internally at the hip. Increased abdominal pain indicates a positive obturator sign.
Peritoneal signs: rebound, rigidity, bed shake sign, heel tap, hip shake, Heal-drop/ Jarring Test (stand on toes for 15sec, then come down with full wt onto heels). Palpation with stethoscope can alleviate anxiety by saying "I'm going to listen to you now" (If true pain the child will react by saying "Ow" or by flinching/ blinking/ tearing.) "Did the ride to the hospital cause you discomfort?, Yes!, especially when we hit bumps."
Rebound tenderness: A test for peritoneal irritation caused by stretching of inflamed parietal peritoneum, and thus does not require deep palpation. Can be elicited by percussion, cough, strain or other subtle change in intra-abd pressure. *****1st warn the pt what you are about to do. Press deeply on the abdomen with your hand. After a moment, quickly release pressure. If it hurts more when you release, the pt has rebound tenderness.
Pancreatitis: Hemorrhagic**--> Cullen's sign**= umbilical bluish discoloration (also seen in hemoperitoneum, cancer and ectopic ruptures).
Grey-Turner sign = bluish discoloration seen in flank.

Cancer signs:

Courvoisier's sign: palpable gallbladder from CA.
Bloomer's shelf: palpable pelvic shelf mass on rectal exam.
Sister Mary Joseph's node: Cutaneous metastases to the umbilical area. Sister Joseph worked at St Mary's hospital and observed the correlation of a lymphatic met from a tumor (usually intra-abd) presenting as a periumbilical mass. This "node" is seen with cancer of stomach, intestine, pancreas, kidney, uterus, ovary, gallbladder, breast, lung and penis. Most metastatic lesions to the umbilicus are irregular firm nodules, occasionally painful, measuring 1–1.5 cm in diameter, although lesions as large as 10 cm have been described. They are rarely ulcerated, may be purulent if secondarily infected, or may show mucoid discharge depending on the tissue of origin (SKINmed 2006;5;5:250-252).
Umbilical nodule Ddx: granuloma (pyogenic, Fb), urachal duct cyst, endometrioma, keloid, hernia, epithelial inclusion cyst, Sister Mary Joseph's node, ventral hernia, omental hernia, epidermal inclusion cyst.
Supraclavicular (SC) Nodes --> lymphoma, lung/ GI/ breast carcinoma, thoracic/ retroperitoneal infection.
Virchow node = L anterior SC node (heralds a thoracic or abd neoplasm). Pt's >65yo with iron deficiency anemia are 31X more likely than the general population to have a GI malignancy (6% if >50yo, 9% if >65yo) (Am J Med 2002;113:276-80).

Liver Span: Link: Exam Liver dz |
Splenic exam: Links: Splenomegaly | **Splenic Rupture |**
Percuss the lowest costal interspace in the **left anterior axillary line**. This area is normally tympanitic. Ask the pt to take a deep breath and percuss this area again. Dullness in this area is a sign of splenic enlargement. Use your left hand to lift the lower rib cage and flank. Press down just below the left costal margin with your right hand. Ask the pt to take a deep breath. The spleen is **not** normally palpable on most individuals.
Size: The normal spleen obeys the "rule of odds": 1 x 3 x 5 inches in size, weighing 7 oz [about 200 g], and lying between ribs 9 and 11. The mean splenic length of 10 cm increased by 0.1 cm for each 1-inch increase in height in women taller than 5 ft 6 inches [168 cm], while the mean splenic length of 11 cm increased by 0.2 cm for each inch taller than 6 ft [180 cm] in men (Sonographic evaluation of spleen size in tall healthy athletes. Am J Roentgen. 2005;184:45-49).
Liver-Spleen Scan: since both contain reticuloendothelial cells that readily take up 99mTc sulfur colloid from the blood, the pattern of uptake reflects the distribution of functioning reticuloendothelial cells as well as hepatic perfusion. The liver normally

phagocytoses 80–90% of the tracer whereas the spleen sequesters 5–10% under normal circumstances. The bone marrow takes in a negligible amount of tracer. Lesions measuring 2–2.5 cm are readily identified. Parenchymal defects demonstrated on this scan are nonspecific and cannot distinguish inflammatory from neoplastic lesions. However, the liver-spleen scan can detect some lesions that are missed by CT or U/S because of isodensity. Causes of a solitary "cold" defect in the liver include hepatocellular carcinoma, metastasis, hemangioma, cyst, abscess, hematoma, and adenoma. Hepatic adenomas typically occur in young women with a history of oral contraceptive use. They usually do not contain a significant number of Kupffer cells and therefore will appear as an intrahepatic "cold" defect. In contrast, focal nodular hyperplasia (FNH) is a benign neoplasm that also occurs often in females. Because FNH contains Kupffer cells that are able to concentrate radiocolloid, it will appear indistinguishable (isodense) from the normal parenchyma or may occasionally appear as "hot" lesions with increased uptake. Multiple focal "cold" intrahepatic defects are commonly due to metastatic disease, multiple cysts, or hemangiomas. Diffuse liver disease may also be detected on liver-spleen scan. Cirrhosis is manifest by a small right hepatic lobe and an enlarged left lobe. Increased colloid uptake in the spleen and bone marrow typically is present. Causes of hepatomegaly with diffusely decreased colloid activity include infiltrative disorders, such as hepatitis or cirrhosis, fatty infiltration, passive congestion, lymphoma, and hemochromatosis.
Causes of splenic "cold" defects include cyst hemangioma, abscess, and malignancy. Splenic infarcts are typically identified by peripheral wedge-shaped defects. Splenomegaly may be due to portal hypertension, hemolytic anemia, lymphoma or leukemia, and infectious causes. The liver-spleen scan also may be used to localize accessory splenic tissue.
Palpation of the Aorta: Press down deeply in the midline above the umbilicus. The aortic pulsation is easily felt on most individuals. A well defined, pulsatile mass, > 3 cm across, suggests an aortic aneurysm.
Costovertebral Tenderness: CVA tenderness is often associated with renal disease. Warn the pt what you are about to do. Have the pt sit up, use the heel of your closed fist to strike the pt firmly over the costovertebral angles. Compare the left and right sides.
Shifting Dullness: a test for peritoneal fluid (ascites). Percuss the pt's abdomen to outline areas of dullness and tympany. Have the pt roll away from you. Percuss and again outline areas of dullness and tympany. If the dullness has shifted to areas of prior tympany, the pt may have excess peritoneal fluid.
Xiphoid process Pain: Usually the pt just needs reassurance that it is just a normal anatomical swelling at the tip of the sternum that occasionally gets painful with minor trauma. Avoid further manipulation. Manage with a tincture of time (massage, heat and exercise).
Work Up of Abd pain: **Lab:** stool guaiac, CBC with diff, amylase, LFT, lytes, glucose, U/A, upright CXR (PA/Lat), KUB, ECG, hCG. Early analgesia for pt's with acute abd pain of unclear etiology does **not** interfere with diagnostic eval (Am Coll Surg 2003;196:18-31). If remains obscure, consider video-capsule endoscopy.

Abdominal Eval:

Links: KUB | CT Scan | **Upper GI, Small Bowel Follow-Through & Enteroclysis** | Barium Enema | **Gallbladder Eval** | Meckel's | **Gastroparesis Eval** | Splenic Scan | **Abd Eval & Exam** | Obstructions (Ileus, SBO, Intussusception and Volvulus) |
Plain Abdominal Films: KUB = Kidney – Ureter – Bladder. The most commonly obtained abd film is the supine view. An upright view of the abd may be helpful in cases of intestinal obstruction. The left lateral decubitus view may be used for suspected pneumoperitoneum in acutely ill pt's when the upright view cannot be obtained. Always include a CXR as 15% of abd sx's arise from a chest abnormality. Should include both supine and erect views, a left lateral decubitus may be substituted for the erect film if the pt is unable, look for pneumoperitoneum. The three-view abd x-ray series includes upright CXR, supine and upright abd x-rays. Costs $225–$350. CT is more accurate than plain films, although CT is more expensive and exposes pt's to higher radiation doses, it makes little sense to order plain films when it's likely that the pt will end up undergoing CT anyway (Radiology 2005;237:114-22).
Indications: Dx of calcium-containing renal tract calculi. Control (scout) film prior to contrast administration. Assess efficacy of stone tx (e.g., shockwave lithotripsy). Check position and status of ureteric stents and foreign bodies / devices in the urinary tract. To evaluate intestinal gas patterns and certain soft tissue abnormalities.
Technique: Antero-posterior (AP) full-length view in full expiration. Lateral films may be required to clarify the position of certain calcifi cations. Bowel preparation prior to test will improve image quality and yield, but is not mandatory. Effective radiation dose is 1.5 mSv.
Interpretation
Step 1: a systematic approach. The renal tract and its anatomical markings are scrutinized. Other intraabdominal and pelvic organs are identified. Abnormal intestinal gas pattern is usually obviously apparent. Look for bony abnormalities in the spine and bony pelvis. Kidneys—renal hilum moves with respiration but tends to lie at the level of L2 lumbar vertebrae. The size (usually 11–13 cm in adults), shape, and position of both kidneys should be observed.
Ureters—the ureteric line is traced inferiorly, along the tip ofthe transverse processes of the lumbar vertabrae, over the sacroiliac joint, then lateral within the bony pelvis towards the ischial spine before sharply turning medially to enter the bladder. Note any calcifications—but non-urinary calcification can often confound matters.
Step 2: Soft tissue abnormalities—It is possible to assess the outline and therefore the shape, size, and displacement—of organs such as the kidneys, liver, spleen, and bladder. Absence of psoas shadow may suggest a retroperitoneal mass or fluid collection.
Step 3: Abdominal gas pattern—A plain film is invaluable in the diagnosis of intestinal obstruction (with bowel dilatation proximal to the level of obstruction) and ileus. Gross fecal loading can be easily verified.
3 Lines: iliopsoas and obturator internus muscles as well as properitoneal fat lines usually show up well.
Pneumoperitoneum: free air in peritoneal cavity. As little as 1ml of air can be seen beneath the right hemidiaphragm. Can visualize both sides of the stomach wall and small bowels. May also see the outline of the ligamentum teres hepatis, falciform ligament (football sign), urachus and/or the lateral umbilical ligaments. False positives with prominent undulations of the diaphragm, abundant fat in the subphrenic region or with colonic interposition. Can get there via douching, coitus, idiopathic, colonoscope (2%), post GI surgery, via abd paracentesis. 10% do not need surgery, need urgent surgery if fever, pain or inc WBC's. Other radiographic evidence of pneumoperitoneum includes (1) Rigler's sign (outline of the inner and outer intestinal wall), (2) football sign (large ovoid air collection), (3) outline of the falciform ligament, (4) triangle of air seen between bowel loops, (5) perihepatic air, (6) urachus sign (outline of the middle umbilical ligament), and (7) inverted V sign (outline of the lateral umbilical ligaments).

Possible Bowel Gas Patterns: Normal. Nonspecific. Adynamic ileus. Mild—localized ileus or "sentinel loop". Severe—"colonic pseudo-obstruction". Small bowel obstruction. Large bowel obstruction.
Intraluminal gas—bowel gas pattern. Free intraperitoneal air—pneumoperitoneum. Other gas collections—biliary, intramural, etc.
Air Fluid Levels: >2 air fluid levels in the small bowel is abnormal.
Colon Cutoff Sign --> a gas-filled segment of the transverse colon abruptly ending in the area of pancreatic inflammation, often accompanied by a sentinel loop.
Sentinel Loop --> dilated, air filled loop of small bowel, most commonly in the LUQ, represents a localized ileus. See in pancreatitis, appy, cholecystitis.
Ischemic Colitis -->Often have a nonspecific bowel gas pattern (NSBGP), moderate distention, air fluid levels, intestinal intramural gas/ gas in portal system (late). Early see diffuse bowel dilation or gas-less abdomen. If severe see submucosal edema (**thumbprinting**, 30%) and thickening. 90% in either descending colon, splenic flexure or sigmoid colon.
Small Bowel Obstruction --> multiple air-fluid levels, dilated loops of small intestine, dec colonic gas. A proximal jejunal obstruction may not have dilatation. With a duodenal ileus (often from pancreatitis) see the **"reversed 3: sign** from air in the 1st-3rd portion of the duodenum with the medial border flattened as the plicae circulares (Kerckring's folds) are effaced by inflammation. The more distal the obstruction, the more **a "step-ladder pattern**" of dilated loops of bowel.
"**String of pearls**" sign due to collections of gas within obstructed loops of bowel.
Stretch sign shows parallel radiolucencies consisting of small collections of gas situated between hypertrophied valvulae.
Pneumatosis intestinalis: gas within the bowel wall, may be primary or secondary. Primary or idiopathic pneumatosis is usually benign and most often involves the descending colon and sigmoid. About 85% of cases are secondary and may be due to intestinal ischemia (mesenteric arterial or venous thrombosis or decreased flow), bowel obstruction, trauma, infection, inflammatory bowel disease, connective tissue disorders, and chronic obstructive pulmonary disease. Primary pneumatosis appears radiographically as clusters of radiolucent cysts along the contour of the bowel wall. More linear collections of gas paralleling the wall of the bowel are seen with secondary pneumatosis. Small bowel involvement is more common with the secondary form.
Abnormal gas collections: may be contained within abscesses. Common locations of intraabdominal abscesses include the subphrenic space, lesser sac, Morison's pouch, and pouch of Douglas. Hepatic, splenic, and pancreatic abscesses may also contain air. Necrotic tumor containing air may suggest a fistulous tract to the bowel. Emphysematous cholecystitis can be diagnosed if gas is seen outlining the gallbladder wall or within the gallbladder lumen. It occurs more commonly in pt's with diabetes and is thought to be due to ischemic changes of the gallbladder initiated by obstruction of the cystic duct by a stone. Gas in the portal venous system is an ominous sign and is usually associated with mesenteric vascular thrombosis with associated intestinal ischemia and necrosis. This leads to the development of intramural gas and subsequent extension of gas through mesenteric veins to the portal vein. Gas in the portal venous system follows centrifugal portal venous flow and will appear as branching tubular lucencies extending into the liver periphery. Gas in the biliary system may result from prior sphincterotomy or surgery, eg, choledochoenterostomy. It may also be seen with biliary-enteric fistula as a result of penetrating duodenal ulcer. Scarring of the ampulla owing to Crohn's disease, parasitic infection, or malignancy are other causes of pneumobilia. Rarely, biliary sepsis resulting from gas-producing bacteria can cause biliary tract gas. Biliary gas appears as centrally located tubular lucencies as it follows biliary drainage into the larger bile ducts.
Step 4: Bony abnormalities—A systematic examination of the lower vertebral column may reveal fractures or obvious bony metastases, as well as provide evidence of neurological disease, including spina bifida and sacral agenesis. Metastases to the bony pelvis is common and can be seen on the KUB film.
Causes of abnormal calcification on KUB include: Renal tract calculi (70–90% are radio-opaque and visible on KUB. Stones smaller than 1–2 mm may be missed). Calcified lymph nodes (common in the elderly). Phleboliths (very common in the pelvis). Calcifi ed costal cartilages. Renal nephrocalcinosis (seen in hyperparathyroidism, renal tubular acidosis, and medullary sponge kidneys). Aortic calcifications. Prostatic calcification (can be seen in chronic prostatitis). Adrenal calcification. Parenchymal calcification within cysts or tumors. Calcifi ed uterine fibroids. Tuberculosis of the kidney. Lamellated calcifications in the right upper quadrant are typical for gallstones. Only 10% of gallstones are radiopaque whereas 90% of urinary tract calculi are radiopaque. Porcelain gallbladder describes sheet-like wall calcifications of the gallbladder and is associated with a high incidence of gallbladder carcinoma, for which prophylactic cholecystectomy is recommended. A focal calcification in the right lower abdomen may represent an appendicolith, which is seen in 10–15% of pt's with acute appendicitis. Mucocele of the appendix may appear as circular or rim calcification in the same location. Enteroliths may also be found in small or large bowel diverticula. Infections such as tuberculosis or Pneumocystis carinii can cause visceral or nodal calcifications. Alternatively, calcified lymph nodes may represent metastatic tumoral involvement. Certain tumors more commonly calcify. Mucinous adenocarcinoma of the stomach, colon, and ovary can cause mottled or punctate calcifications in the primary tumor as well as in metastatic foci. Gastrointestinal tract leiomyomas and lipomas may also calcify. Pancreatic calcifications more commonly occur in association with chronic pancreatitis or pancreatic pseudocyst. Pancreatic tumors that calcify include the rare serous cystadenoma and mucinous cystic neoplasm. Obesity and central abdominal fat are related to calcification in the abdominal and coronary aorta according to data from 3130 participants in the Framingham Heart Study (Am J Cardiol 2009;104:543-547).
Advantages: Cheap. Non-invasive. Will easily identify high-density calcified stones (calcium oxalate, calcium phosphate) as well as low-density struvite and cystine stones. Small radiation dose, therefore can be repeated. Provides useful information about abdominal organs and intestinal obstruction.
Disadvantage: Misses up to 30% of stones (e.g., radiolucent stones such as uric acid and cystine, stones with low calcium content, and small stones). Can be diffi cult to distinguish between calcifi cation within and outside the renal tract. Bowel gas and fecal loading can obscure small calcifications.
CT Scans: A study concluded that CT is more sensitive than U/S for identifying urgent conditions in patients with acute abdominal pain, however, a conditional strategy of U/S in all patients followed by CT only if U/S is negative or inconclusive is the most sensitive strategy (94%; i.e., only 6% of urgent diagnoses missed)(an most expensive), but would substantially lower the number of patients who are exposed to radiation (expose only 49% of patients)(BMJ 2009;338:b2431). See Lab, U/S, CT in Acute Apendicitis |
Systematic Approach to CT Interpretation: "Every Organ in Every Slice".
Solid organs: Liver, spleen, pancreas, kidneys, pelvic organs.
Blood vessels: Aorta and branches, IVC, portal vein.
Bowel: Stomach, duodenum, small bowel, colon, appendix.

Mesentery and peritoneum: Abnormal fluid. Free air (use “lung windows”).
Muscles: Abdominal wall, diaphragm, psoas, and iliac muscles.
Bones: Spine, pelvis (use bone windows).
Thorax: Diaphragm, lungs, heart. (Emergency Radiology Case Studies. 2008. David T. Schwartz, MD. McGraw-Hill)
CT Signs of SBO: Dilated proximal small bowel (> 2.5 cm diameter). Collapsed distal small bowel. Transition zone. Obstructing lesion (adhesions presumed if no lesion seen). Small bowel feces sign.
CT in Ischemic Obstruction: Bowel wall thickening, “target sign,” increased enhancement. Mesenteric congestion—edema, hemorrhage. Ascites—bloody. Infarcted bowel—poorly enhancing, paper-thin bowel wall; intramural gas.
CT in Closed-loop obstruction: Dilated loops of bowel in a cluster or radial distribution. Mesenteric vessels converging to a central point—“Spoke-wheel” sign. C-shaped or U-shaped loop of dilated bowel. Two adjacent collapsed, triangular-shaped loops of bowel. Whirl sign—twisted mesenteric vessels at point of volvulus.
Upper GI study (UGI) series: consists of double-contrast views of the esophagus, stomach, and proximal small bowel followed by single-contrast images of these areas in the prone or upright positions. Single-contrast images are obtained following the administration of low-density barium. Spot compression of portions of the lumen filled with barium will demonstrate filling defects due to thickened folds, large ulcerations, or masses. Abnormalities of motility and contour are well depicted, such as linitis plastica due to scirrhous adenocarcinoma of the stomach. Gastric varices are best seen on single-contrast or partially collapsed double-contrast images. Double-contrast images are best for demonstrating mucosal detail and is more sensitive for detecting erosions, ulcerations, and polyps.
Small Bowel Follow-Through (SBFT): a common technique to evaluate diseases of the small bowel. Usually performed immediately following a double-contrast UGI series, for which the pt has already ingested an effervescent agent and high-density barium. Additional low-density barium is administered for the routine SBFT so as to completely fill the small bowel. Overhead radiographs and compression spot films of small bowel loops are obtained at sequential time intervals. Careful compression under fluoroscopy is essential to separate overlapping bowel loops. The terminal ileum and a portion of the cecum or ascending colon must be imaged before the study is complete. Normal transit of contrast through the small bowel into the colon is variable, most often spanning 1–2 hours. Accelerating agents such as metoclopromide or meglumine diatrizoate mixed with barium can be given in cases of slow transit. The dedicated SBFT is a study performed independently of a UGI series. The pt ingests only low-density barium so that there is more uniform opacification of the small bowel. More accurate evaluation is possible, since artifacts from mixing of gas and barium suspensions of different density are avoided. Usually performed in pt’s with nonspecific sx’s who elicit low clinical suspicion for small bowel pathology. These pt’s often have had negative findings on upper and lower endoscopy.
Enteroclysis (Small Bowel Enema): a method for establishing that the small bowel as normal. Give 10–20 mg of metaclopramide IV and topical anesthesia for the nostril and throat, a special balloon-tipped enteroclysis catheter containing a guidewire is inserted from the nose into the stomach and guided fluoroscopically into the proximal jejunum. Once the tip of the catheter is about 5 cm distal to the ligament of Treitz, the balloon is inflated. Barium followed by methylcellulose is infused at high rates via a pump into the jejunum. Careful compression spot films of each quadrant and overhead films are obtained. Difficulty in tube placement is encountered in pt’s with pyloric or duodenal scarring. Large sliding hiatal hernias or prior surgery, such as gastroduodenostomy and gastrojejunostomy, also may preclude tube placement. Enteroclysis demonstrates excellent mucosal detail, fold pattern, and bowel distension. Through the adjustment of infusion rates, adequate distension of the entire small bowel can be achieved. This makes it easier to recognize areas of subtle narrowing that may not be detected on conventional SBFT.
Peroral Pneumocolon: often performed after a SBFT. Used to specifically evaluate the terminal ileum. The pt ingests low-density barium, after which sequential films are obtained. When the oral contrast reaches the ascending colon to mid-transverse colon, air is insufflated through a rectal tube until the cecum is distended. This air then refluxes into the terminal ileum, providing a double-contrast appearance. Compression spot films are obtained. Occasionally, glucagon is administered to relax the ileocecal valve and to facilitate reflux.

Ddx of Pain by Location:

Links: RUQ | **RLQ** | LUQ | **LLQ & Flank** | Extraperitoneal | **Nonsurgical Causes** | Abd Pain in Elderly | **Abd Pain in Women** | Abd Distention / Masses |

Diffuse: Peritonitis, Pancreatitis, Perforated DU, GU, Perf Sigmoid, Divertic, Gastroenteritis, IBD, IBS, Colitis, Pseudomembranous colitis, Ischemic colitis, Infectious. The dx of angioedema of the intestine induced by ACEi therapy should be considered in any pt on this type of agent who presents with abd pain and thickened small bowel loops with no other obvious cause.
Epigastric: pancreatitis, PUD, Cholecystitis, perforated esophagus, AMI, gastric volvulus (rotation around the long axis in pt’s with long, thin stomachs). Foregut embryonic derivatives such as liver, stomach, gallbladder, pancreas and first two parts of duodenum.
T5-9: esophagus, stomach, duodenum, liver, GB, pancreas.
Periumbilical: T8-L1, midgut derivatives: small intestine, appendix, asc-transverse colon, cecum.
Hypogastrium: T11-L1, hindgut & genitourinary derivatives: distal transverse-rectosigmoid colon, rectum & upper anal canal, reproductive organs, bladder.
RUQ: Cholecystitis (acute, perforated), Biliary Colic, Cholangitis, Hepatitis, CHF, DU, GU, Pancreatitis, Acute Appendicitis, R Subphrenic abscess, Hepatic abscess, AMI, Ischemia, Hepatomegaly, Fitz Hugh Curtis, RLL Pneumonia/ effusion/ Empyema, Renal Stone/colic, Pericarditis, Zoster, Pyelonephritis, UTI.
RLQ: Appendicitis, Crohn’s Dz, Mesenteric Lymphadenitis, PID, Ectopic Preg, Renal Stone, Pyelonephritis, CA cecum/ ascending colon, Intestinal Obstruction, Cholecystitis, Pelvic/ psoas abscess, Rectus Sheath Hematoma, Diverticulitis, Meckel’s Diverticulitis, mittelschmerz (ruptured Graafian Follicle), Ruptured Corpus Luteal Cyst, Ovarian/Fibroid Torsion, Threatened Abortion (TAB), Incomplete Abortion, Münchhausen, Endometriosis, TOA Rupture.
LUQ: Gastritis, DU/GU (inflam, perf.), Splenomegaly, Pancreatitis, Splenic injury / infarct/ Aneurysm, Subphrenic abscess, LLL Pneumonia/ Effusion, LLL Empyema, Renal Stone/colic, Aortic Dissection, Pyelonephritis, UTI, Ischemic Colitis, AMI, ischemia, Pericarditis, Zoster, Intestinal Obstruction.

LLQ: Diverticulitis, Ischemic Colitis, PID, Ectopic Preg, UTI, Pyelonephritis, CA Sigmoid Colon, Intestinal Obstruction, Appendicitis (AP), Pelvic/psoas abscess, Rect Sheath Hematoma, mittelschmerz, Corpus Luteal Cyst, Ovarian/ Fibroid Torsion, Threatened AB, Incomplete Abortion, Renal Colic, AAA (leak, rupture), Fecal Impaction, Münchhausen, Endometriosis, TOA Rupture.
Abdominal Cutaneous Nerve: Entrapment occurs as the nerve emerges through a fibrous ring in the abd wall. Focal tenderness is felt at this site, and pain may be felt over the low abd wall.
Pain worse lying down: pericarditis, epiglottitis, Ludwig's angina, asthma, CHF, GERD.
Pain better lying down: post-spinal tap H-A, retropharyngeal abscess.
Pain better lying on left side: pancreatitis, pregnancy, pyelonephritis on the R or a kidney stone on the L.
Pain better lying on right side: pyelonephritis on the L or a kidney stone on the R.
Back Pain: aortic aneurysm, renal calculus, gallstones, pancreatitis, TB.
Ddx Flank Pain: Acute ureteral obstruction. Stone. Blood clot. Papillary necrosis.Chronic ureteral obstruction. Congenital anomaly. Tumor. Stricture of ureter. Previous surgery. Radiation therapy. Retroperitoneal fibrosis. Acute pyelonephritis. Perinephric abscess. Renal tumor. Renal cell carcinoma. Transitional cell carcinoma. Wilms" tumor. Trauma of kidney. Renal infarction. Vesicoureteral reflux. Gallbladder disease. Appendicitis. Diverticulitis. Chest disease. Salpingitis.
Flank Pain: a sensation of discomfort, distress, or agony in the part of the body below the rib and above the ilium, generally beginning posteriorly or in the midaxillary line and resulting from the stimulation of specialized nerve endings upon distention of the ureter or renal capsule. Flank pain is the presenting sx of many significant diseases of the urinary system. Flank pain that radiates to the ipsilateral groin is usually caused by proximal ureteral or renal pelvic obstruction. Fever suggests infection proximal to the ureteral obstruction. Hematuria is occasionally absent with acute ureteral obstruction, however, and is often absent with chronic obstruction. Renal inflammation usually produces localized pain and is associated with other manifestations of infection such as fever, leukocytosis, and bacteriuria (Renal pain. Urology 1975;6:403-8).
Ptosis of the Kidneys: Age-related drop in the kidneys causing. Dx with upright CT. Can be treated surgically.

Extraperitoneal - Referred Abd Pain (by System):

Cardiac--> AMI, ischemia, myocarditis, pericarditis, pericardial tamponade, pacemaker wire.
Pulmonary--> pneumonia, PTX, PE.
Urologic --> cryptorchidism, cystitis, epididymoorchitis, hydronephrosis, nephrolithiasis, perinephric abscess, prostatitis, pyelonephritis, renal infarction, testicular torsion, ureteral reflux.
OBGYN--> dysmenorrhea, endometriosis, salpingitis, Fitz-Hugh-Curtis Syndrome, Mittelschmerz, Ovarian cyst, adnexal torsion, uterine anomaly, IUP, ectopic, preeclampsia, uterine rupture.
Neurologic--> abd migraine, cutaneous nerve entrapment, epidural abscess, herpes zoster, intercostal neuralgia, MS, spinal cord tumor.
Musculoskeletal--> hematoma of rectus sheath, primary actinomycosis of rectus, pyomyositis of rectus sheath.
Abdominal Wall Pain: The sit-up test: following identification of the site of maximum abd tenderness, the pt is asked to fold his or her arms across the chest and sit up halfway. The examiner maintains a finger on the tender area, and if palpation in the semi-sitting position produces the same or increased tenderness, the test is said to be positive for an abd wall syndrome.
ICD-9 Codes:
789.0 abd pain.
789.00 abd pain, unspecified site.
789.01 abd pain, right upper quadrant.
789.02 abd pain, left upper quadrant.
789.03 abd pain, right lower quadrant.
789.04 abd pain, left lower quadrant.
789.05 abd pain, periumbilical.
789.06 abd pain, epigastric.
789.07 abd pain, generalized.
789.09 abd pain, other specified site.
789.60 abd tenderness, unspecified site.

The Abdominal Compartment Syndrome:

Occurs when raised intra-abd pressure is associated with signs of organ failure in critically ill pt's. It is seen after abd surgery, in injured pt's, with or without abd trauma and in critically ill pt's, including pt's with burns, sepsis or ascites
Dx criteria: (1) elevated intra-abd pressure (>= 25 mm Hg). (2) one or more of the following, indicating organ damage: oliguria, raised pulmonary pressure, hypoxia, dec cardiac output, hypotension, acidosis, abd decompression results in clinical improvement. Pressure in the urinary bladder correlates with the actual intra-abd pressure over the pressure range of 5-70 mm Hg. It can be measured with the pt in the supine position, a Foley catheter in the bladder and a water pressure manometer held at the level of the symphysis pubis.
Complications: (1) failure in one or more organs (heart. lung, kidneys), (2) breakdown of surgical sites, (3) impaired blood flow with decreased visceral perfusion, (4) intestinal obstruction.
Tx: prompt abd decompression when the pressure becomes elevated, prevention through the use of tension-free abd closures. (Intra-abd HTN after life-threatening abd trauma: prophylaxis, incidence and clinical relevance to gastric mucosal pH and abd compartment syndrome. J Trauma 1998;44:1016-1023) (Prevention of the abd compartment syndrome. Lancet 1999;354:1749-1750)

Nonsurgical Causes of Abd Pain:

Links: **Functional GI D/o's** | Abd Pain | **Belching & Gas** |
Obesity: The higher the BMI, the more prevalent GI sx's.
Upper GI pain: in 4% of normal population, 17% if BMI >30, 20% if >40.
Lower GI pain: 4% pop, 20% if BMI >40 kg/m2.
Diarrhea: 17% pop, 41% if BMI >35.
Bloating: 11% pop, 25% if BMI >30.

Constipation: 17% of pop, 26% if BMI >26%.

Chronic Intermittent Abd Pain: consider familial Mediterranean fever, Crohn's, internal hernia, endometriosis, porphyria.
W/u: CBC, ESR, LFT's, amylase, lead level, porphyria screen. Consider abd series, CT. Abd US and/or HIDA scan to r/o GB disease, an UGI with small bowel follow through to r/o obstruction .
Tx: antidepressants, pain clinic referral, TENS, acupuncture. Fructose intolerance may be the cause for unexplained GI sx's in many pt's (Am J Gastroenterol. 2003;98:1348-1353).
Ddx of Chronic or Recurrent Abd Pain: Structural Disorders. Chronic pancreatitis. Abdominal neoplasms (carcinomas, lymphomas, others). Inflammatory bowel diseases. Mesenteric ischemia. Pelvic inflammatory diseases. Endometriosis. Functional Gastrointestinal Disorders. Irritable bowel syndrome. Functional (nonulcer) dyspepsia. Functional abd pain syndrome. Levator syndrome. Biliary pain (gallbladder or sphincter of Oddi dysfunction). Malabsorption syndrome (fructose, gluten, lactose etc). Abdominal adhesions. Toxins / drugs (heavy metals, concretions, drug bezoars etc).
Abdominal migraine (see neurology module). Unexplained upper abd sx's are more common in 99 consecutive pt's with migraine headaches than in healthy controls (81% Vs 37.5%) (Cephalalgia 2006;26:506-510).

Chronic Intractable Abd Pain (CIAP): continuous pain over mo's with no identifiable cause. No features of IBS or non-ulcer dyspepsia. 70% female.
Ddx: somatoform d/o, pain behavior, childhood h/o of abd pain, physical sexual abuse as a child.
Tx: multifactorial.

Abdominal Adhesions: Abdominal adhesions occur in ~90% of pt's following on abd surgery will later develop abd adhesions. Because of the high rate of adhesion formation after laparotomy, surgeons should be aware of the need to prevent postoperative adhesion formation. This can be achieved by careful tissue handling, avoidance of foreign bodies including long sutures and large devascularized tissue stumps distal to ligatures, care in the management of spilled GI content, removal of all gallstones during laparoscopic cholecystectomy, lavage of blood or bile, and avoidance of desiccation of exposed bowel during the surgical procedure. 50% of pt's who underwent a laparotomy for pain and did not undergo adhesiolysis experienced relief of their sx's (Surg Laparosc Endosc Perc Tech. 2003;13:88-94). Laparoscopic adhesiolysis cannot be recommended as a tx for pt's with chronic abd pain, according to Dutch physicians, as it is not evidence-based, a placebo effect (Lancet 2003;361:1247-1251).

Hematologic: Acute intermittent porphyria, lead poisoning, SS crisis, leukemia, lymphoma, thrombocytosis, cyclic neutropenia with ileocecal necrosis, hemolytic uremic syndrome.

Mesenteric Adenitis: painful enlargement of mesenteric lymph nodes. Most cases in age <18yo. Abd pain may be mild ache to severe colic. Usually starts in upper abd or RLQ and eventually localizes to the right side, but no precisely as in appy. Difficult to distinguish from appy, and thus appendix need to be removed as recurrent episodes are common.

Metabolic/ endocrine: acute intermittent porphyria, Familial Mediterranean fever, uremia, hypercalcemia, hyperkalemia, thyrotoxicosis, DKA, pheo, Addisonian crisis, chronic angioedema, acute renal failure.

Inflammatory: rheumatoid vasculitis, SLE, HSP, PAN, periarteritis nodosa, pancreatitis, ARF, IBD, granulomatous enteritis, eosinophilic enteritis, zoster, pneumonia.

Infectious: enteritis (bacterial, viral, TB, MAC, fungal, parasitic), hepatitis, meningococcemia, mono, RMSF, spontaneous bacterial peritonitis.

Toxins/ drugs: heavy metals (arsenic, lead, thallium, cadmium, mercury), caustics, salicylates, Fe, mushrooms, black widow spider, talcosis, concretions, drug bezoars, anticholinergic, sympathomimetics.

Functional: somatization, Münchhausen, malingering (narcotic addiction).

The Functional Abdominal Pain Syndrome: characterized by frequent, recurring, or continuous episodes of abd pain lasting for at least 6 mo. No other gut sx's are present, and there is no evidence of an underlying structural dz after a thorough investigation.
Rome II Criteria: All of the following:
(1) frequently recurrent or continuous abd pain for >= 6 mo's.
(2) incomplete or no relationship of pain with physiological events (eating, defecation, menses).
(3) the abd pain causes some loss of daily social functioning.
(4) no evidence of organic disease to explain the pain despite a thorough exam.
(5) insufficient criteria to establish the dx of an alternative functional GI d/o that would explain the abd pain.
Additional features: (1) The pattern of pain does not fit any known dz.
(2) The pain description may include different pains of different characteristics, often at different sites.
(3) Often associated with abnormal illness behavior.
(4) Often associated with a significant psychiatric d/o.
(5) The pain often does not interfere with sleep or leisure activities, but will prevent the person from working or doing other duties.
(6) The pt may have a history of multiple surgical operations for abd pains.
(7) The onset of pain may be found to coincide with episodes of personal stress.

Common Conditions in Acute Abdominal Pain in the Elderly:

More difficult to dx as have slightly different sx's along with coexisting disease and any are poor personal historians. Morbidity and mortality among older pt's with abdominal pain are high; often requires an expedited eval with admission to the hospital and surgical consultation.

Pearls in the Elderly Pt:

May have the absence of fever / leukocytosis despite a serious bacterial infection or surgical condition. There may be altered pain perception from chronic pain medications. Four times higher likelihood of hypothermic response with a significant intra-abdominal process. Lower likelihood of localized tenderness despite a focal surgical condition; reduced rebound and guarding from decreased abdominal wall musculature. May see suppressed tachycardia from medications or intrinsic cardiac disease

Diseases not to miss: AMI (may present w/ upper GI sx's only), pneumonia, pulmonary embolus, CHF.
W/u: detailed H&P. Check Bld Cx, CXR, ECG, amylase, CBC, lytes, LFT, BUN, Cr, UA and Cx. Drug levels, rectal exam. Consider KUB, Abd U/S or CT. Consider starting Abx. If RUQ get U/S of biliary tract or HIDA. If diffuse pain or any lower quadrant pain check CT.

Ddx: **Links:** **Fecal Impaction** | Pseudo-obstruction | **Abd Pain, Exam & Gut D/o's** |
Intestine--> appy, diverticulitis, infection, infarction, obstruction, perforation.
Stomach--> food poisoning, perforated ulcer.
Gallbladder--> cholecystitis, stones.
Vascular--> aneurysm, thrombosis, trauma.
Renal--> stones, pyelonephritis, UTI.
Cholecystitis: 12% mortality in older pt's. Have a WBC count <10,000 in 30-35%, afebrile in 30-35%. A HIDA scan to dx acute infection is the best test. Pt may appear deceptively well but can crash quickly & needs surgery urgently. 60% of elderly pt's did not have back or flank pain and 5% have no pain at all. >40% do not experience nausea, >50% are afebrile, and 41% have normal white blood cell count. 13% with acute cholecystitis had no abnormal liver function tests, fever, or leukocytosis (Emergency department evaluation of geriatric pt's with acute cholecystitis. Acad Emerg Med 1997;4:51-5).
Perforated Peptic Ulcer: 50% One half *do not* report sudden onset of pain. The pain may be generalized or also in lower quadrants only. Epigastric rigidity may be absent and there may be a lack of free air on plain films in 40%.
Appendicitis: Overall the dx is missed ~50% of time as signs are mild. Delayed presentation is common (20% >3 days). Migration of pain often lacking (shift to RLQ in only 20%), anorexia may be absent, >50% with N/V, afebrile in 40%. Older pt's with appendicitis are more likely to present with generalized pain, longer duration of pain, distention, rigidity, decreased bowel sounds, and a mass. Can present with a painless mass (appendiceal mass). WBC <10,000 in 20%, inc bilirubin common, KUB often misinterpreted as small bowel obstruction.
Large Bowel obstructions: have a 40% mortality. Commonly due to carcinoma, volvulus or diverticulitis. Diarrhea in 19%, lacking obstipation in 50%, vomiting often absent.
Acute Mesenteric Ischemia: often report gradual onset of pain, with prior episodes. Pain is often out of proportion to the physical exam. May be guaiac-negative stool early on. **Ischemic Colitis/ Mesenteric Ischemia:**
Abdominal Aortic Aneurysm: A ruptured aneurysm must be considered in any older pt (even when renal colic is suspected) with acute abd pain **or** back pain. A pulsatile mass will often not be detectable, back pain present in only one half, syncope may be the primary complaint, hematuria may be present. Rupture has a 70% mortality. See Cardiology chapter.
Diverticulitis: WBC may be normal in 50%. Diverticular disease increases with age and is estimated to affect 2/3 of pt's >90yo. (Diagnosis of Acute Abdominal Pain in Older Pt's. Am Fam Physician 2006;74:1537-44)

Chronic Abd Pain in Women:

Link: Nonsurgical / Chronic | IBS is twice as common in women who have chronic pelvic pain as it is in the general population (Obstet Gynecol 2004;104:452-8)....IBS must be considered in women with chronic pelvic pain. Emergency department visits for nonspecific abdominal pain, especially among young women, may be tied to the prevailing level of air pollution, a Canadian team reported (Digestive Disease Week 2009; Presented June 3).....It's possible, that "young women are the most affected cohort because they are at increased risk for functional motility disorders, the most common of which is irritable bowel syndrome."...."Air pollution has been linked to decreased stomach motility in mice" the researcher pointed out.
Endometriosis: child bearing age/ low parity, progressive dysmenorrhea, involvement of the small intestine/ colon causes pain similar to IBS. Dx via laparoscopy.
PID: young with multiple sexual partners, IUD, douching. Pain ranges from mild to mod with bilateral adnexal TTP, cervical motion tenderness (CMT), cervical d/c, fever. Fitz-Hugh-Curtis is a perihepatitis associated with PID.
Ovarian Ca: age >50, FHx, or personal Hx of breast Ca. Vague, dull pain, pressure in pelvic area, bloating.
Dx: exam confirmed by transvaginal U/S.
Metastatic Ca: Hx of Ca. Vague pain suggestive of GI, early satiety, epigastric discomfort.
Chronic Mesenteric Ischemia: extensive atherosclerosis. Pain provoked by eating, wt loss, evidence of maldigestion/ malabsorption.
Dx: mesenteric angiography. Check a postprandial mesenteric Doppler.
Primary Biliary Cirrhosis: middle age, RUQ pain, pruritus, jaundice, pigmentation, xanthelasma. Inc LFT, presence of **antimitochondria**l Ab's,
Dx: liver biopsy. F:M is 10:1. See hepatology section.
Biliary Dyskinesia (Sphincter of Oddi dysfunction): usually follows cholecystectomy or recurrent idiopathic pancreatitis. Inc LFT's, delayed emptying of biliary tree.
Dx: sphincter of Oddi manometry.
Lactose Intolerance: crampy abd pain after ingestion of milk products.
Crohn's: young, familial tendency, pain localized to RLQ, nausea, anorexia, diarrhea.
Dx: small bowel series, CT.
Pancreatic dz: younger pt likely with familial dz, Ca in older.
Upper abd pain radiating to back, relieved by sitting up and leaning forward. Worse with food/ ETOH.
Chronic Functional Abdominal Pain (CFAP): frequently recurrent or continuous abd pain for at least 6mo. Incomplete or no relationship of the pain to physiological events (defecation, menses, eating). Some loss of daily function. No evidence of organic dz or other functional GI d/o to explain.
W/u: Normal CBC, ESR, chem panel and flex sig.
Tx: Tricyclic antidepressants with anticholinergic properties such as Pamelor and Sinequan improve sx's and reduce pain in pt's with non ulcer dyspepsia and irritable bowel syndrome.
Pregnancy: Ultrasound is the imaging modality of choice in pregnant women with acute abd pain, especially in the first trimester. During the first trimester, MRI is not recommended, unless it is absolutely clinically necessary. MRI is a promising modality for evaluating pregnant women with right-lower-quadrant pain in the 2nd and 3rd trimesters (Radiology. 2005;234:445-451).

Causes of Abd Distention / Masses:

Links: Bloating & Other | Obstructions | **Fecal Impaction** | Pseudo-obstruction | **Ileus** | Splenomegaly | **Hepatomegaly |**

Rectus Sheath Hematoma: See Trauma-Tox: Hematoma.
Abdominal bloating and visible distension are common and have similar risk factors according to a population-based study on 2259 subjects (53% females, mean age 62 years) (somatization probably plays a role)(Gut 2008;57:756-763)....The overall age- and sex-adjusted prevalence of abdominal bloating was 19%, and for visible distention it was 8.9%.....female sex and higher overall Somatic Symptom Checklist (SSC) scores and higher scores on each of the items in the SSC significantly increased the odds for bloating and distention compared to controls.....Constipation-predominant irritable bowel syndrome (OR, 2.3), dyspepsia (OR, 1.9), and GI symptom complex overlap (OR, 1.7) also significantly increased the odds for distention compared to bloating alone.

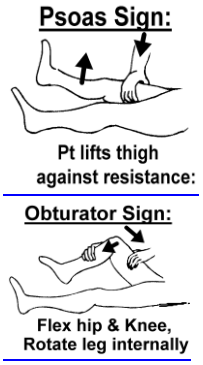
Other:

Abd pain and postprandial bloating: r/o biliary dyskinesis, ck CCK HIDA scan. Also see Belching & Gas |
Abd Wall Pain: usually related to posture, not affected by bowel habits or meals. R/o hernia, cutaneous nerve entrapment, slipping rib syndrome (sharp pain at upper abd due to subluxation of 8th -10th ribs), rectus sheath hematoma or myofascial pain. May have discrete tender points. If not related to a scar and no hernia on exam, can try trigger point injections.
Mass lesions--> hydatid cysts, ameboma of the colorectum, hydronephrosis (schistosomiasis), intra-abd malignancy, ovarian cyst, uterine fibroids.
Dilated bowel--> small intestinal obstruction (Ascariasis, intussusception, intestinal volvulus, inguinal hernia), megacolon (Chagas), toxic megacolon (Shigellosis).
Ascites--> TB peritonitis, hepatic cirrhosis, portal HTN (schistosomiasis, veno-occlusive dz), nephrotic syndrome (Plasmodium malariae), lymphatic filariasis (chylous ascites), malignancy.
Neonates--> Renal (55%): hydronephrosis, Wilms tumor, polycystic kidney dz, distended bladder, renal vein thrombosis, ectopic/ horseshoe kidney, urachal cyst, mesoblastic nephroma.
GI (20%): pyloric stenosis, bowel duplication, intestinal obstruction (imperforate anus, Hirschsprung's, Meconium ileus).
GU (15%): hydrometrocolpos, follicular ovarian cyst.
Biliary: choledochal cyst, hepatic cyst, hemangioma, hydrops of the gallbladder.
Other (10%): adrenal hematoma, sacrococcygeal teratoma, anterior meningomyelocele.
Older Child--> constipation, intussusception, appendiceal abscess, pancreatic pseudocyst, mesenteric or omental cyst, tuboovarian abscess, hematocolpos, ovarian torsion, hepatoblastoma, choledochal cyst, hydrops of gallbladder, neuroblastoma, lymphoma, rhabdomyosarcoma, wandering spleen.
****Ref:** (*A Guide to Physical Examination and History Taking*, 6th ed., by Barbara Bates, Lippincott in 1995) (*Consultant* 2000;1:25-36) (*Principles of Ambulatory Medicine*, 4th ed., 1995, Williams & Wilkins, pp447-56) (Feldman: Sleisenger & Fordtran's Gastrointestinal and Liver Disease, 8th ed., 2006 Saunders)

Appendicitis:

Links: Anatomy | **S/s |** Lab, U/S, CT | **Tx |** Ddx & Surgical Tx |

The most common surgical emergency, due to obstruction (fecalith, gallstone, tumor, parasite) that causes subsequent inflammation. Lifetime cumulative incidence of 7% of general pop, most in age 3-30. 1/ 2,200 pregnant women. The incidence of acute appendicitis peaks in early adulthood, it occurs more frequently in males especially at the time of puberty. 1.3:1 male predominance. 52 cases per 100,000 population. Has a 0.6% mortality if nonperforated, 5% if perf.
Anatomy: The appendix arises as a diverticulum at the apex of the cecum ~2.5cm below the ileocecal junction, in an adult it averages 7.5cm in length, the tip can be retroperitoneal or pelvic. The three taenia coli converge at the junction of the cecum with the appendix and can be a useful landmark to identify the appendix. The appendix can vary in length from <1cm to >30 cm; most appendices are 6 to 9 cm in length. Appendiceal absence, duplication, and diverticula have all been described. Traditionally thought to be a vestigial organ (no function), but really an immunologic organ which actively participates in the secretion of immunoglobulins, particularly IgA. By the age of 60yo, virtually no lymphoid tissue remains within the appendix and complete obliteration of the appendiceal lumen is common. No know sequella after the removal. Contrary to conventional wisdom, the appendix may not be a useless vestigial organ after all (J Theor Biol 2007;Oct 22)....it helps beneficial bacteria survive and repopulate the colon following a pathogen-induced purging event. High levels of air pollution may increase the risk of adult-onset appendicitis, according to results of a study conducted in Canada (73rd annual scientific meeting of the American College of Gastroenterology. 2008;Oct).
S/s: Classic presentation in only 25%. Initially have periumbilical pain, persists after defecation, nausea (85%), vomiting (60%), anorexia (95%), then moves to RLQ 50%) in 1-12 hr (ave of 4-6hr), rebound tenderness (26%), RLQ guarding (21%), low grade fever (68%), dec bowel sounds, have pain and fullness on R with DRE (in 50%). Abd mass (12%), fever (20%), diarrhea (15%). The 3 s/s most predictive are RLQ pain, abd rigidity and migration of pain from periumbilical to RLQ (NEJM 2003;348:236-42). If you can probe all the way to sacral promontory in RLQ and pt is hungry likely **not** appy. Ck rectal exam to r/o other conditions or bleeding.
Child: uncommon, but also have 50% perforation rate difficult to dx. A child with suspected appendicitis should be examined by an attending physician and should undergo appendectomy without imaging if the dx is unequivocal. If diagnostic uncertainty exists, the child should be observed and reexamined after a few hours. If dx remains uncertain, the child should undergo either US (preferred for adolescent females with possible tube-ovarian pathology) or CT with an appropriate dose of radiation (Pediatrics 2004;113:29-34).
Retrocecal appendix (64%): mid abd pain, may have urge to defecate/ urinate.
Pelvic: LLQ pain common, TTP on rectal exam, urge to urinate/ defecate.
Retrocolic (0.5%): typically no mesentery and is retroperitoneal like ascending colon.
Guarding: voluntary contractions (12%). **Rigid** = involuntary.
Rebound: grimace/ pain with sudden release of pressed RLQ.
Rovsing sign: RLQ pain with release of palp of LLQ.
Psoas sign: (12% have) for retrocecal appendix, Inc pain on passive extension (pull backward / hyperextension) of R thigh as apply counter resistance to the R hip as pt on L side. Place your hand above the pt's right knee. Ask the pt to flex the right hip against resistance. Increased abd pain indicates a positive psoas sign.



Obturator sign: inc RLQ pain as supine pt's flexed R thigh/ knee are passively internally rotated at hip as examiner hold the knee. Raise the pt's right leg with the knee flexed. Rotate the leg internally at the hip. Increased abd pain indicates a positive obturator sign.
McBurney's Sign: TTP at 2/3 the distance from the ant iliac spine and umbilicus on the right.
Dunphy's sign: Increased pain in the right lower quadrant with coughing.
Hip flexion sign: Pt maintains hip flexion with knees drawn up for comfort.
Cutaneous hypesthesia: parietal peritoneal inflammation, test with light touch.
Pinch-an-inch test: a form of rebound tenderness, only in reverse. A fold of abd skin over McBurney's point is grasped and elevated away from the peritoneum. The skin is then allowed to recoil back briskly against the peritoneum. If the pt has increased pain when the skin fold strikes the peritoneum, the test is positive and peritonitis is presumably present. If the pain seems excessive just during the initial pinch phase, the pt may have a very low pain threshold, a factor that can be taken into account when deciding if the pt has a surgical abdomen (South Med J. 2005;98:1207-1209).
Lab: WBC 12-15K (yet 10% normals are >10K, has poor specificity), 95% have L shift. The positive and negative predictive values of an elevated WBC in acute appendicitis are 92% and 50%, respectively. 25% have inc WBC on U/A. Abnormal KUB in only 10%.
Dx: Clinical, lab. The frequency of misdiagnosis leading to unnecessary appendectomy has not changed with the introduction of U/S (se a target sign, lack of compressibility, free fluid and edema), CT (streaky fat, free air, phlegmon = periappendiceal inflammation, thick wall, appendicolith in 25%) or laparoscopy, **nor** has the frequency of perforation decreased (JAMA 2001;286:14). CT 97% PPV, 100% NPV, 100% sens, 95% spec, 98% accuracy with rectal contrast. If end up sending pt home under "observation" give them the "appy wrap" in that they need to come back for another exam as soon as possible as it may be an early appy.
U/S: Plane U/S is 89% accurate (sens/spec 85%/95%), appendicitis may be ruled out if see a "normal" appendix, but it is seen in <5%. Color Doppler ultrasound should be the first imaging examination in adult patients with suspected acute appendicitis according to a study on 326 pt's (sensitivity was 74.2% and specificity was 97%)(Am J Roentgenol 2008;190:1300-1306).....The authors recommend that CT be reserved for patients with inconclusive findings on ultrasound and for those with classical signs and symptoms for whom clinical suspicion remains high (sensitivity of 100% and specificity of 98.9%). A diagnostic pathway using ultrasound as the primary study, followed by CT for patients with negative or inconclusive ultrasound findings, had high accuracy for diagnosing appendicitis according to data from 151 patients who presented to a single emergency department with clinically suspected acute appendicitis (J Am Coll Surg 2009;208:434).....avoids the delays and ionizing radiation that are associated with CT scanning.....The protocol had a sensitivity of 100% and a specificity of 86%.....More than half the patients evaluated with this sequential protocol did not require CT scans.
Plain films: worthless unless ruling out free air or bowel obstruction (may see fecalith in 10% or a paucity of RLQ gas).
CT Scans: Liberal use of CT scanning has not improved the accuracy of diagnosing appendicitis (Surg 2003;185:194-7). Focused helical CT with rectal contrast only (88% sens, 100% spec, 100% PPV, 100% NPV) appears superior to the traditional triple-contrast CT (97% sens, 86% spec, 90% PPV, 93% NPV) for diagnosing acute appendicitis (Arch Surg 2004;139:495-500). Non visualization of the appendix on helical CT is associated with low incidence of acute appendicitis in the absence of secondary inflammatory changes given the visualization of more than a scant amount of pericecal fat (Am J Roentgenol. 2004;183:889-892). Unenhanced helical CT can accurately detect acute appendicitis, thereby avoiding the possible allergic reactions and cost associated with contrast enhancement (Br J Surg 2005;91:1641-1645). CT scanning using IV-only contrast is accurate for diagnosing appendicitis and, along with results from several other studies, suggest that routine use of oral contrast is unnecessary…..abandoning oral agents could reduce throughput times by as much as several hours…..however, pt's with little body fat might require enteral contrast, as the fat stranding indicative of appendiceal inflammation might not be as apparent (Emerg Radiol 2006;12:99-102). The pre-op use of CT to make a definitive dx in cases with straightforward s/s of acute appendicitis is linked to a poorer pt outcome than a straight-to-surgery approach (Digestive Disease Week 2007:May 23) (time to surgery was a mean of 3 hours longer and perforation rate was 17% Vs 8%). Helical CT (16-detector MDCT) was more accurate than clinical judgment for diagnosing appendicitis, even in cases of clinically apparent appendicitis according to a study on 157 consecutive patients (Emerg Med J 2008;25:477).....The positive predictive value of the examining physician's clinical impression was 73%, and the negative predictive value was 56%.....Corresponding values for MDCT were 97% and 97%.....Patients with suspected appendicitis, especially women, should undergo abdominal CT scanning to confirm or exclude the diagnosis, with the exception of patients whose diagnosis is considered "certain" by the attending surgeon and who are taken directly to the operating room without imaging. Helical CT was more accurate than clinical judgment for diagnosing appendicitis, even in cases of clinically apparent appendicitis according to a study on 157 consecutive patients who presented to two emergency departments with signs or symptoms that raised concern for possible appendicitis (Emerg Med J 2008;25:477).....The positive predictive value of the examining physician's clinical impression was 73%, and the negative predictive value was 56%. Corresponding values for MDCT were 97% and 97%....editorialist notes that not all radiologists who interpret CT scans are CT specialists, and CT scans are not read by two radiologists in most EDs. CT of the appendix has a significant impact on the management of emergency department patients who are suspected of having appendicitis

(Am J Roentgenol 2008;191:1102-1106).....CT scans led to a change in the treatment plans of 29% of the patients....had a sensitivity of 94%, a specificity and positive predictive value of 100%, and a negative predictive value and accuracy of 99%. Low-dose unenhanced CT that delivers approximately 50% less radiation (4.2 mSv vs 8 mSv) is just as effective in diagnosing appendicitis as standard-dose contrast-enhanced CT according to data from 207 adults (Am J Roentgenol. 2009;193:96–105)(sensitivity of 98.7% vs 100%)(specificity was 95.3% and 93.0%). See **CT Scan** |
MRI is effective in excluding the dx of appendicitis in pregnant women with acute abd pain and in whom a normal appendix is not visualized by U/S (Radiology 2006;238:891-899) (sens 100%, spec 93.6%, NOV = 100% and PPV = 1.4%).
Urine: Scientists using mass spectrometry for proteome profiling of urine samples have identified several markers whose levels are substantially enriched in patients with acute appendicitis according to a study on 67 patients using (Ann Emerg Med. 2009;Published online June 23)....Levels of leucine-rich alpha-2-glycoprotein (LRG), the marker that demonstrated the strongest diagnostic performance.
Tx: Pt's with acute, nonperforated appendicitis should undergo urgent appendectomy (laparotomy or laparoscopy). If clinical presentation does not suggest the need for immediate surgery, observation for 6-10 hours may help clarify the dx. Short delays (12-hour) to avoid surgery in the middle of the night might be acceptable for clinically stable pt's so that surgery can be performed by a well-rested surgeon in the morning (Arch Surg 2006;141:504-7). **Antibiotic therapy alone:** (medical therapy) has an initial success of 95%, but a recurrence rate of 35% with short follow-up.....abx alone have been used in rare situations such as with sailors on long submarine tours. Non-operative management with Abx for two wks is an option if pt is non-toxic with an abscess. Only 1/13 will resolve spontaneously, but 38% of these then recur, most within 1yr.
Prophylactic Abx if unperforated with Ceftriaxone 1g **or** Cefotetan (25mg/kg IV preop **or** Cefoxitin 2g IV **or** Ceftizoxime 2g IV + Metronidazole. Best to hold analgesics until **discuss** with consulting surgeon. Short-acting narcotic analgesics such as fentanyl @ 0.01-0.02 mg/kg are preferred, since they can be reversed by naloxone if necessary. Monotherapy with cefoxitin or piperacillin/tazobactam for appendicitis appears to be a good choice because these drugs are not associated with increased 30-day or 2-year readmission rates, and in some cases they are associated with shorter hospital stays and hospital costs compared to aminoglycoside-based triple therapy (Pediatrics 2007;119:905-11).
If perforated or abscess: Clinda 450-900mg IV **or** Flagyl 500mg IV q6 **AND either** Gentamicin 7mg/kg IV qd **or** Levofloxacin 500mg IV qd. Antibiotics should cover anaerobes, enterococci, and gram-negative intestinal flora, such as monotherapy with tazobactam-piperacillin 3.375 g IV or ampicillin- sulbactam 3 g IV. The wound is usually left open to heal with secondary intention. If get fever or inc WBC of bowels fail to return to function ck CT, drain percutaneously if abscess, if no abscess consider Imipenem 500 mg IV q6. 20% of pt's who undergo ex-lap have abnormal appendix, up to 40% error in advanced age or female sex. In a large meta-analysis, Beta-lactam monotherapy (most often second-generation cephalosporins) was at least as effective as aminoglycoside-based triple-antibiotic therapy for children with ruptured appendicitis (Pediatrics 2007;119:905-11).....Readmission within 30 days of discharge was similar between ABT and monotherapy recipients, except that significantly lower rates were seen in ceftriaxone-monotherapy recipients. Compared with triple therapy, cefoxitin monotherapy was associated with a shorter mean length of stay, lower pharmacy costs, and lower total charges (all differences significant).
Ddx: nonspecific, gyn (PID, torsion, ovarian cyst, endometriosis, ectopic), mesenteric adenitis, Crohn's, cecal/ Meckel's/ sigmoid diverticulitis, acute cholecystitis, bowel perforation, tuberculous/ Yersinia ileitis, ischemic colitis, mono, cancer, obstruction, UTI, pneumonia, parasites, pancreatitis, PUD, epididymitis, nephrolithiasis, epiploic appendagitis (fat globs next to colon inflamed). Recurrent spontaneously resolving appendicitis (Br J Surg 1997;84:110). Anorexia not helpful in differentiating appy from PID.
Prophylaxis during elective surgery: usual microorganism are enterococci, gram negative bacilli, anaerobes. On pre-op day give 4 liter Colyte or GoLYTELY PO over 2 hours and clear liquid diet only. Neomycin 1 g PO and erythromycin base 1 g PO at 1, 2 and 10 hrs PM. NPO starting at midnight.
Non-Ruptured viscus surgery: usual microorganism are enterococci, gram negative bacilli, anaerobes. 30 minutes before surgery give Cefoxitin 1-2 g IV for 1 dose or Cefotetan 1-2 g IV for 1 dose or (cefazolin 1-2 g IV + metronidazole 500 mg IV) for 1 dose. If allergic to penicillin use Vancomycin 1 g IV for 1 dose.
Ruptured Viscus: tx for >5 days with Cefoxitin 2 g IV for 1 dose then 1 g IV q8h and Gentamicin 1.5 mg/kg IV q8h. If allergic to penicillin can use Clindamycin 600 mg IV q6h and Gentamicin 1.5 mg/kg IV q8h.
Ngative appendectomies: represent >15% of all appendectomies performed. Pt factors, such as IBS and anxiety, as well as physician factors, like low CT scan usage, appear to increase the likelihood of an appendectomy with no pathologic evidence of appendicitis (Gut. 2007;56:608-610,655-660)… Multiple logistic regression showed that independent factors in predicting negative appendectomy were IBS (OR = 2.17), degree of anxiety (OR, 1.12), absence of migrating pain (OR, 3.43), absence of muscle guarding (OR, 3.72), lower polymorphonuclear cell percentage (< 75%; OR, 3.05), and no CT scanning (OR, 2.32). Rates of negative appendectomy in Massachusetts General Hospital are 3% (Vs 5.6% if not CT) with routine use of diagnostic CT for suspected appy, a decline from 20% prior to routine CT use (not a statistically significant) (Am J Roentgenol 2005;184:1802-1808).

ICD-9 Codes:

540.0 Acute appendicitis with generalized peritonitis
540.1 With peritoneal abscess
540.9 Without mention of peritonitis

Approaches to removal of the nonperforated appendix: Can do so through an open incision, usually a transverse right lower quadrant skin incision (Davis-Rockey) or an oblique version (McArthur-McBurney) with separation of the muscles in the direction of their fibers, or a paramedian incision, but this is not routinely done. The incision is centered on the midclavicular line. Occasionally, where the dx is uncertain, a periumbilical midline incision can be used. Once the peritoneum is entered, the appendix is delivered into the field. This can usually be accomplished with careful digital manipulation of the appendix and cecum. It is important to avoid too extensive of a blind dissection. In difficult cases, extending the incision 1 to 2 cm can greatly simplify the procedure. Once the appendix is delivered into the wound, the mesoappendix is sacrificed between clamps and ties. There are several ways to handle the actual removal of the appendix. Some surgeons simply suture ligate the base of the appendix and excise it. Others place a pursestring or Z-stitch in the cecum, excise the appendix, and invert the stump into the cecum. Once the appendix is removed, the cecum is returned to the abdomen, and the peritoneum is closed. The wound is closed primarily in most pt's with nonperforated appendicitis because the risk of infection is <5%. The appendix can also be removed laparoscopically, although operative costs are higher due to a longer procedure and more equipment needed with laparoscopy, the overall costs are lower because the pain is less and pt's can return to work sooner. Most authors report using three ports to remove the appendix--an umbilical port and two others.

The location of the two other ports may vary depending on the surgeon and the pt's body habitus. The appendix can be removed using endoloops or an endoscopic stapling device. The total charges can vary significantly if disposable ports and a stapler are used. The appendiceal stump is not buried when it is removed laparoscopically, and the appendix is removed through one of the port sites. The fascia at the 10-mm port sites is usually closed, as is the skin. Many pt's can be discharged within 24 hours of operation. (Ortega JM, Ricardo AE: Surgery of the Appendix and Colon. Atlas of Ambulatory Surgery. Philadelphia, WB Saunders, 1999.)

Appendectomy Operation Note: The pt was taken to the operating room and placed supine on the operating table. After general anesthesia was established, the abdomen was prepped and draped in the standard surgical fashion. Attention was turned to the right lower quadrant where the location of the anterior superior iliac spine and the umbilicus was identified. A 3 cm transverse incision was made ~ one-third of the way between these two landmarks at McBurney's point. The incision was carried through Scarpa's fascia to the rectus sheath. The external oblique aponeurosis was incised in the direction of its fibers, and the underlying muscle layer was identified and spread in alternating direction longitudinally and transversely until the peritoneum was visualized. The peritoneum was then incised revealing the contents of the peritoneal cavity. The Taenia coil coursing along the right were visualized and followed posteriorly to reveal the cecum. The vermiform appendix in a retrocecal location was gently swept free manually. The appendix appeared edematous, erythematous and necrotic at the tip. No area of perforation was seen. A sample of the turbid peritoneal fluid was taken for culture and sensitivity. The appendix was grasped with the Babcock clamp and elevated. The mesoappendix was ligated in continuity using 2-0 Chromic suture. Once the appendiceal vein and artery were ligated, a clamp was applied at the base of the appendix and it was then doubly ligated using 2-0 Chromic sutures. The appendix was then removed and passed off the field as a specimen. The Bovey electrocautery was then used to cauterize the appendiceal stump. The appendix was then inverted and two figure of eight 3-0 silk sutures were used to close the cecum over the appendiceal stump. The cecum was returned to its normal anatomic position and the remainder of the peritoneal cavity was explored. No other pathology was identified. The operative site was then irrigated with NS.

The peritoneum was then closed with a running 2-0 Chromic suture. The muscle layer was loosely reapproximated with interrupted 2-0 Chromic suture, and the external oblique was similarly closed with 3-0 Vicryl. A few interrupted 3-0 Plain subcutaneous sutures were placed, and a undyed 5-0 PDS subcuticular suture was placed in the skin. Steri-strips were applied and then a sterile dressing. The sponge, needle and instrument counts were correct at the end of the case. The pt was awakened from anesthesia, extubated, and then transported to the recovery room in stable condition.

Chronic or Recurrent Appendicitis: Rare. Intermittent bouts of obstruction of the appendiceal lumen with spontaneous remission may be the cause. Mild local inflammation after a resolving attack of acute appendicitis may result in chronic right lower quadrant discomfort. The dx is made if the following three criteria were met: a history of longer than one month with three or more recurrent attacks of right lower quadrant abdominal pain; localized tenderness in the right lower abdomen without signs of peritoneal irritation or inflammation; or radiologic findings at barium examination consisting of irregular filling of the appendix, nonfilling or partial filling of the appendix after 24 hours, or nonemptying of the appendix after 72 hours.

Interval Appendectomy: when a localized appendiceal abscess from perforated appendicitis can be initially managed with CT-guided percutaneous drainage or limited surgical drainage. When combined with adequate antibiotic and fluid administration, the majority of pt's will respond to this conservative management and can be discharged without fever or abdominal pain. Since up to 15% of the pt's in the delayed group had a recurrent acute episode during the waiting period, despite a slightly higher incidence of wound infection in the early appendectomy group, it appeared safe and cost-effective to remove the appendix early rather than waiting 6 to 10 weeks.

Pregnancy: Misdiagnosis of appendicitis during pregnancy places almost a quarter of affected women at risk of fetal loss (J Am Coll Surg 2007;205:534-540)....inaccurate diagnosis of appendicitis is more likely in pregnant women than in nonpregnant women....30% of pregnant women and 29% of nonpregnant women had complicated appendicitis, which was defined as acute appendicitis with generalized peritonitis or peritoneal abscess. Pregnant women had a significantly higher rate of negative appendicitis (23% versus 18%)....Laparoscopic appendectomy (performed in 14% of pregnant women) was associated with a fetal loss rate of 7% compared with 3% in pt's who had open appendectomy.

The overall diagnostic accuracy for appendicitis in women was 75.3%, but varied between 50% and 96.7% (CMAJ 1995;152:1617-1626).

****Ref:** (The natural history of appendicitis, *Ann Surg* 1997;225:252) (Does this pt have appendicitis, *JAMA* 1996;276:1589) (Am J Surg 2002;183:609)

Acute Pancreatitis:

Links: Drugs Causing | **S/s** | Lab | **Tx** | Prognosis | **Complications & Cysts & Enlargement** | Ddx Amylase & Lipase | **Chronic Pancreatitis** | Pancreatic Ca & ERCP | **Pancreatic Enzymes** |

Exocrine (95% of mass), endocrine (5%) portions.

ICD-9 Codes:

577 Diseases of pancreas
577.0 Acute pancreatitis
577.2 Cyst and pseudocyst of pancreas
577.8 Other specified diseases of pancreas

Etiology / Risks: Most from either ETOH (65%) or cholelithiasis (30%, ~5% of those with gallstones develop pancreatitis, alk phos >180 U/L is suggestive). Pancreatitis is about three times more common in patients with type 2 diabetes than in people without diabetes. **Smoking** appears to be associated with an increased risk for acute and chronic pancreatitis according to the results of an observational, population-based cohort study on 9573 women and 8332 men in Denmark who underwent a physical examination and completed self-administered questionnaires about lifestyle habits at baseline, and who were followed up for a mean of 20.2 years (Arch Intern Med. 2009;169:603–609). ....There was a dose-response association between smoking and risk for acute and chronic pancreatitis in both men and women. Among those who smoked 15 to 24 g of tobacco per day, the hazard ratio (HR) of developing pancreatitis was 2.6 for women and 2.6 for men independent of alcohol and gallstone disease.....In this cohort, smoking accounted for approximately 46% of cases of pancreatitis.

…….END DEMO of this chapter.

Hematology-Oncology:

Edited by Greg Newman, MD and Ra'id Marji, MD, Hematologist-Oncologists

Contents: **Cancer** | **Venous Thromboembolism (VTE)** | Warfarin | **Hypercoagulability** | Bruising | **Bleeding** | Platelets | **DIC** | Blood Products | **Electrophoresis & Protein** | Peripheral Smears | **WBCs** | Erythrocytes (RBC's) | **Anemia(s)** | Porphyria | **Lead** | Sickle Cell | **Bone Marrow Transplant** | Edema | **Anticoagulations and Blood Thinners** | *INDEX* |

Cancer:

Links: **Definition & Warning Signs and Physiology** | Screening & Types | **Causes / Incidence** | Risks & PREVENTION | **Tumor Markers** | Ca of Unknown Primary | **METS** | Fatigue | **Wasting & Wt loss** | Derm Signs | **Radiation Therapy** | Oncology EMERGENCIES | **Paraneoplastic Syndromes** | Cancer Tx & CHEMO AGENTS | **Supportive Agents** | Leukemia | **Lymphoma** | Myelodysplastic | **Plasma Cell Dyscrasias (MM, Amyloid...)** | Thymic | **Nasopharyngeal** | PEDIATRIC | **Neutropenic fever** | GI | **Bone & Sarcoma** | Amyloid | **Multiple Myeloma** | Lymphadenopathy | **Urologic** | Gyn | **Lung** | Skin | **Chemo Toxicity** | Thyroid | **Brain Tumors** | Chemo agents in Alphabetical Order |

Common ICD-9: Lung Ca = 162.9. Breast Ca = 174.9. Colon Ca = 153.9. Prostate Ca = 185. Bladder Ca = 188.0.
Hematology deals with the normal functions and d/o's of the formed elements in the blood (erythrocytes, leukocytes, and platelets) and the plasma factors governing hemostasis. Oncology deals with cancers.

Warning Signs & Pearls: So-called "alarm symptoms" (hematuria, hemoptysis, dysphagia, or rectal bleeding) may help to alert general practitioners to clinically relevant diagnoses according to the results of a cohort study (BMJ. 2009;339:b3094).....The proportions of patients diagnosed with either cancer or noncancer diagnoses within 90 days were 17% presenting with hematuria, 25.7% for hemoptysis, 17.2% for dysphagia, and 14.5% for rectal bleeding.

1 Generalized itch may be the first hint of a myeloproliferative disorder.
2 Painful mouth ulcers could signal life-threatening neutropenia.
3 'Pulled calf muscle' in a recent traveller could be a DVT.
4 Easy bruising of recent onset may indicate a platelet problem.
5 Constipation with back pain – think of myeloma.
6 An Afro-Caribbean child with an acute abdomen may have sickle cell crisis.
7 Tired and lethargic – check for anemia.
8 Do not forget hematological malignancy as a cause for weight loss.
9 Night sweats could herald lymphoma.
10 Pain on drinking alcohol is a feature of Hodgkin's disease.
Source: (Alarm Bells in Medicine by Nadeem Ali. 2005 by Blackwell Publishing Ltd)

Cancer Info:

Cancer is any monoclonal neoplasm, most of which invade surrounding cells, often metastasizes, may recurs after resection, may cause death if inadequately treated. Histologically identified on the basis of invasiveness and anaplastic changes (loss or orderly maturation, loss of polarity of nuclei). Cancer is the second leading cause of death in developed countries (Lung > Colorectal > Breast > Prostate > Ovarian) and the leading cause of death worldwide (Hepatocellular > Cervical > Gastric). It accounts for about 900,000 new cases and 400,000 deaths per year in USA. 50% of males and 30% of females will be diagnosed with a cancer in their lifetime. Cancer causes 25% of deaths in USA (it is second leading cause of all deaths, lung Ca is #1, causing 28% of cancer deaths/yr), 2nd only to CV dz. Smoking is responsible for 85% of male and 75% of female cancer deaths, nonsmoking spouse had a 30% inc risk of lung cancer. Colon Ca is #2 (10% of cancer deaths/yr), inc fat intake **-->** colon and prostate Ca. The American Cancer Society estimates that in 2005, an estimated 1,372,910 new cancer diagnoses and ~570,280 cancer deaths (1,500 per day) are expected in the United States, where cancer causes one out of every four deaths. The five-year survival rate for all cancers is now 64%, up from 50% in the 1970s. Errors in cancer dx were seen in up to 11.8% of cases, in a substantial proportion of cases, the error caused some degree of harm for the pt (Cancer 2005;Oct) (up to 50% were due to pathologic misinterpretation with the remainder being due to poor tissue sampling).

Types of Cancers:

Carcinoma: a malignant neoplasm derived from epithelial tissue. Skin, lung, prostate, intestine, breast. Cells of epithelial cancers tend to progress from hyperplasia to dysplasia to carcinoma in situ to carcinoma to invasive carcinoma. here are 4 major types of epithelium in the body (Glandular, squamous, transitional, and pseudostratified). Some types are only found in a few select organs such as the lung (pseudostratified) or urinary bladder (transitional).

Sarcoma: a malignant neoplasm derived from connective tissue (mesodermal cells such as fat, nerves, muscle, bone and cartilage, fibroblasts).

Adenocarcinoma - tumors of glandular (epithelial) tissue. The term **adenoma** is applied to any benign epithelial neoplasm that forms glandular patterns. Those that form large cystic masses, as in the ovary, are referred to as cystadenomas. Some tumors produce papillary patterns that protrude into cystic spaces and are called papillary cystadenomas. The malignant form is called cystadenocarcinoma.

Lymphoma - tumors of lymph nodes, usually B or T lymphocytes. Derived from the white blood cells that are present in the lymphoid tissues of the body. These sites most commonly include the lymph nodes and spleen. However, lymhomas may arise from any organ and body site.

Leukemia - tumors of blood cells, usually granulocytes or lymphocytes.

Carcinosarcoma - mixed tumor types.

Teratoma - tumors of progenitor cells with multipotent differentiation capacity.

Poorly differentiated or undifferentiated malignancy: There are times when cancer cells display such a distorted and grotesque growth, that a recognizable growth pattern such as a carcinoma or sarcoma cannot be given. In some of these cases, additional studies and special stains or immunohistochemistry may aid in the final dx.

Cancer Warning Signs:

Links: Fatigue | **Wasting & Wt loss** | Derm Signs | **Ca of Unknown Primary** | Mets | **Causes of Ca** |

Cancer may present with certain clinical signs which should prompt a clinical exam.
General: (1) New sx's that persist for more than 2-3 weeks should be checked by a physician.
(2) The earlier a cancer is diagnosed, the better the chances of successfully treating it.
(3) The sx's associated with cancer can also be caused by conditions other than cancer.
Sx's which should be checked: rapid wt loss without apparent cause. A scab, sore or ulcer that does not heal within 3 wks. A blemish or mole that grows, bleeds or itches. New or progressive indigestion or difficulty swallowing. Nagging cough or hoarseness. Coughing up bloody phlegm. Thickening or lump in the breast or any other part of the body. Blood in urine without pain during urination. Change in bowel or bladder habits (JAMA 1998;280:1548).
4 Sx's That Predict an Increased Likelihood of Cancer: Hematuria, dysphagia, hemoptysis, and rectal bleeding are sx's to be taken seriously (BMJ 2007;334:1040)....analysis of 762,325 pt's. The likelihood ratios for a specific cancer ranged from 78 for colorectal cancer in women with rectal bleeding to a high likelihood ratio of 348 for esophageal cancer in men with dysphagia. At 3 years after presentation, those likelihood ratios dropped significantly: However, predictive values increased dramatically with age; for instance, the positive predictive value of hemoptysis in men aged 75 to 84 was 17%.
Cancer Systemic Sx's:
1. Fevers - mainly IL6, IL1, possibly tumor necrosis factor alpha (TNFa).
2. Weight Loss - mainly TNFa (formerly called "cachexin"), catabolic state.
3. Night Sweats - IL6, IL1.
4. Anorexia - chronic inflammation.
5. Induction of acute phase reactants - catabolic state.
6. Cancer Pain - often associated with bony metastases, but may vary.
Other: Horse voice with difficulty breathing is suspicious for a mediastinal tumor. Unexplained weight loss of more than 10% in a middleaged person could be a sign of malignancy.

Malignant Tumor Progression:

(NEJM 2002;347:1593)
1. Progression from normal to malignant cell is called transformation which is believed to occur over 5 or more years (>15 years in some)
2. Tumor simply refers to an abnormal mass of cells. Benign tumors enlarge but to not invade surrounding tissues while malignant tumors, have the capacity to invade normal tissue. Malignant tumors also have the capacity to break off from primary tumor, migrate around the body through blood or lymph, and establish distant growths; these distant growths are called metastases. Many tumors progress from benign growths to malignant.
3. Death from cancer usually due to interference of normal tissue/organ function by metastatic tumors.
Cell Growth Histopathology:
1. The suffix "plasia" means growth.
2. Dysplasia means abnormal growth of a specific cell type. Dysplastic cells retain some histological and molecular markers of original cell type.
3. Metaplasia refers to growths which are abnormal for specific anatomic area, but do not possess invasive or metastatic potential. Metaplastic cells usually appear completely different from the original cell type.
4. Neoplasia, or "new growth", refers specifically to tumors (benign or malignant).
Most neoplasms arise from a single clone of transformed (malignant) cells: Over 5 years is required for growth of cloned transformed cell into apparent tumor. Chronic inflammation may aide in tumor initiation and/or progression. Additional time is required for tumor mass to overwhelm the host, this additional time is highly variable depending on the aggressiveness of the tumor. There are thought to be five distinct alterations for neoplastic transformation of human cells.
Acquired Capabilities of Cancer Cells:
a. Evading apoptosis. b. Self-sufficiency in growth signals (autocrine and paracrine). c. Insensitivity to anti-growth signals. d. Limitless replicative potential. e. Sustained angiogenesis. f. Tissue invasion and metastasis.
Overview of Tumor Growth Stages: Clonal cell population grows to a mass 3-4mm in diameter called primary tumor. Primary tumors >3-4mm in diameter require specific blood supply for further growth, this is called Angiogenesis, which is a critical step in tumor growth. As malignant tumors increase in size, they invade basement membrane and/or stroma, this requires specific enzymes and receptors to degrade surrounding tissue. Tumors can also enter blood vessels and/or lymph nodes. Tumor metastasis to LN is a "marker" for development of cells which have left primary tumor site. It is not known whether tumor cells in normal LN are able to escape the node.
Most small (<0.5mL or <5x108 cells) tumors are curable by surgical removal (NEJM 2000;343(22):1627).
Most small neoplasms are contained (Stage 1), in some tumors, even small neoplasms have invaded tissues and metastasized. Tumors as small as 1-2 mm in diameter have the potential to metastasize. Possible that a 1cm tumor can shed 3-5 million cells into blood every 24 hours. Detection of small tumors, however, nearly always improves survival, thus, development and use of tests for screening for early stage (small tumors) is a major priority in cancer research. Tumors of $>10^8$ to $>10^9$ cells usually invade surrounding tissue: This requires various proteases and other destructive enzymes. Matrix metalloproteinases and other enzymes have been implicated in the process. Tumors then enter stromal areas where they invade blood vessels and LN
Telomeres and Telomerase: (NEJM 2000;342(17):1282) Many cancer cells are believed to be immortal, normal cells die after ~50-60 generations, at least in part due to shortened telomeres. Cancer cells appear to have overcome telomere shortening during cell division. About 90% of cancers express telomerase, which replicates telomeres. Telomerase is normally only expressed in germ cells and early embryo cells, it consists of an RNA component and a set of proteins. Inhibition of telomerase activity may provide a novel mechanism for cancer therapy
Tumor Angiogenesis: New blood vessel growth (angiogenesis) is essential to tumor growth. Tumors secrete a variety of angiogenic factors including TGFbeta (NEJM 2000;342(18):1350), local endothelial cells are activated and express integrin alpha v beta 3 (aVbeta3), Neovascularized tumors can then grow. Angiogenesis inhibitors are being evaluated for anticancer activity
Tumor Metastasis: Once tumor cells are in blood, they "home" to specific organs based on surface receptors, this homing requires typical adhesion molecules including integrins and others, after that tumors bind to endothelium in organ to which they have homed. They traverse endothelium (destruction and/or migration) and establish new sites. Extracellular matrix breakdown is required for

metastatic invasion. Most tumors establish multiple metastases and Immune system may help select for metastatic tumor phenotypes (Lancet 1999;354(9194):1989). Metastatic tumor cells display marked genetic heterogeneity (Lancet 2002;360:683).
Molecular Biology of Cancer: (Lancet 1998;351(suppl II):1).
Progression of normal cells to metastatic tumor caused by dysfunction of ~5 pathways (Retinoblastoma (pRB) pathway, p53 pathway, Telomerase (hTERT) pathway, Ras pathway and Unknown pathway in humans equivalent to SV40 small t antigen). The acquired capabilities discussed above are provided by dysfunction in these pathways, thus, cancer is now known to be a disease of gene mutation and dysregulation.
In general, mutations in genes regulating growth, cell cycle, and differentiation occur (JAMA 2001;285:588). Two major classes of genes involved in cancer have been identified; Oncogenes and Tumor suppressor genes (anti-oncogenes), mutations in these genes may lead to abnormal growth and cell cycle control. Genomic profiling has aided in understanding gene expression changes in various cancers (NEJM 2001;334:539 and NEJM 2001;334(8):601). Cancers will likely be reclassified in the future based on gene expression profiles
Causes of DNA Mutation:
1. Loss of DNA replication fidelity during (normal) aging.
2. Chronic inflammation and reactive oxygen species (Am J Med 2000;109:33).
3. Environmental carcinogens.
4. Various chemotherapeutic agents that damage DNA.
5. Ionizing radiation.
6. Dysfunction of endogenous DNA repair systems (due to genetic or acquired mutations).
Oncogenes: Mutations in these genes have been shown to cause or contribute to tumors. In humans, most oncogenes are mutated versions of normal genes. In some cases, viruses contain genes which are "oncogenic", for example, hepatitis B virus contains one or more genes which can cause liver cancer. Ras, a normal signal transduction protein which binds GTP, is mutated in >50% of human tumors. A large variety of oncogenes with various functions have been identified. In some cases, specific chromosomal translocations cause oncogene activation; these chromosomal translocations are most common in leukemias and some lymphomas. In general, single copies of specifically mutated oncogenes cause cancer ("dominant").
Carcinogen: Substances that can cause changes to a cell's that can lead to cancer. Some carcinogens do not act on DNA directly, but lead to cancer in other ways, such as causing cells to divide at a faster rate, which could increase the chances that DNA changes will occur. Carcinogens do not cause cancer in every case, all the time. Substances classified as carcinogens may have different levels of cancer-causing potential. Some may cause cancer only after prolonged, high levels of exposure. And for any particular person, the risk of developing cancer depends on many factors, including the length and intensity of exposure to the carcinogen and the person's genetic makeup. As of 2008, there are about 900 likely candidates in one of the following groups:
Group 1: Carcinogenic to humans
Group 2A: Probably carcinogenic to humans
Group 2B: Possibly carcinogenic to humans
Group 3: Unclassifiable as to carcinogenicity in humans
Group 4: Probably not carcinogenic to humans
(http://www.cancer.org/docroot/PED/content/PED_1_3x_Known_and_Probable_Carcinogens.asp)
Tumor Suppressor Genes (TSG): Inactivating mutations in TSG have been shown to cause or contribute to tumors. DNA methylation reduces gene expression and may down regulate TSG mRNA (Ann Intern Med 2001;134:573). Some TSG such as FHIT occur at fragile sites and are inactivated by chromosome breaks (JAMA 2001;286:2441). In humans, most TSG are inactive versions of normal cell cycle inhibitory genes and in some cases, TSG are DNA repair genes. In other cases, viruses contain proteins which directly inactivate TSG, for example, human papilloma virus E6 protein binds to and inactivates p53, a TSG. In general, both normal copies of TSG must be inactivated in order to develop cancer
As tumors increase in size, specific molecular genetic defects accumulate: Tumors are characterized by highly unstable DNA replication and repair. Chromosome numbers and ultrastructure are highly abnormal. In late stage tumors, essentially all tumor cells are aneuploid. DNA proofreading and repair capabilities are essentially lost, therefore, germ line DNA in tumors is highly abnormal. Disseminated single metastatic cells show marked genetic heterogeneity. Alterations in five pathways are likely sufficient to transform human cells.
Metastatic Potential: (NEJM 2002;347:1999) Originally believed that tumors acquired metastatic potential as they grow. Whole genome analysis of early breast cancers (Stage I or II) suggests metastatic potential via hematogenous dissemination is a property of early tumors. Primary tumor gene expression profile used to predict clinical outcome. Out of 25,000 genes, a 70-gene prognosis profile was created, this prognosis profile was superior to all standard systems, including LN status. Data indicate that primary tumor contains critical metastatic potential
Certain Viruses Clearly Cause Human Cancers:
1. Hepatitis B Virus (HBV) - hepatocellular carcinoma
2. Hepatitis C Virus (HCV) - hepatocellular carcinoma
3. Epstein-Barr Virus (EBV) - lymphoreticular malignancy, nasopharyngeal carcinoma
4. Human Herpesvirus 8 - Kaposi's Sarcoma
5. Human Papilloma Virus (HPV) - cervical cancer, some vaginal cancer
6. HTLV-I and HTLV-II - adult T cell leukemia
Helicobacter Pylori: (Am J Med 1999;106:222 and NEJM 2002;347:1175) Chronic bacterial infection in stomach linked with peptic ulcer disease. Associated cancers include gastric Carcinoma and Mucosa-Associated Lymphoid Tissue (MALT) Neoplasms.

Screening For Cancer:

Link: Prevention | See Compendium for Recommended Adult Screening & Preventive Services |
The motivating factor behind regular cancer screening is the theory that the earlier one detects a malignancy or premalignancy, the more likely treatment is to be effective in increasing lifespan while minimizing harms caused by the therapy. The goal is to develop sensitive and specific screening tests which should be inexpensive, easily administered, and reduce mortality/morbidity. For many neoplasms, there is controversy over screening. Pt's with a FHx of specific cancers are automatically not low risk. Screening in pt's >65 years old is generally of unclear benefit. Whole-body 19F-flurodeoxyglucose PET scanning has been used to detect occult

disease. Of note, not all cancers progress and thus early detection is not always a benefit and thus early detection may not reduce mortality (Ex. prostate and neuroblastoma) (BJM USA 2004;4:276).

Screening Tests With Proven Cancer Death Reduction:

- Breast Cancer - mammography and possibly self examination.
- Colon Cancer - screening sigmoidoscopy and stool occult blood testing.
- Prostate Cancer - serum PSA test, digital rectal examination.
- Cervical And Vaginal Cancer - Pap Smear, pelvic and external genitalia exam.

Overdiagnosis of cancer: refers to cancers that grow so slowly that the patient dies of other causes before it produces symptoms or to cancer that is dormant or even regresses. Overdiagnosis is a widely recognized problem in prostate cancer screening and some suggest this may be true for breast cancer in many western countries. Cancer screening frequently yields false positives — with resulting invasive procedures according to a study on nearly 68,500 adults, aged 55 to 74, who underwent up to 14 screenings over 3 years in the Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial (Ann Fam Medicine 2009;7:212-222).....Tests included digital rectal examination plus prostate-specific antigen measurement; chest x-ray; flexible sigmoidoscopy; and cancer antigen 125 testing plus transvaginal ultrasound.....The risk for having one false positive after four tests was 37% among men and 26% among women; after 14 tests, risks rose to 60% and 49%, respectively.....The risk for undergoing a false-positive–prompted invasive procedure after four tests was 17% among men and 12% among women; after 14 tests, risks were 28% and 22%, respectively.

Cancer Staging:

Definitions here are generalized; specific staging has been developed for each tumor
Stage 0 - no clinical evidence for cancer; screening test for specific cancer positive
Stage 1 - single tumor mass, no lymph node (LN) or distant metastases (M)
Stage 2 - tumor mass (often larger than stage 1), local LN spread may be present
Stage 3 - tumor mass of any size with distant LN spread and/or other organ invasion
Stage 4 - any tumor mass size, any LN spread, but with distant M disease
Survival decreases with increasing tumor stage

Causes of Cancer (2008) in the US:

Link: See **Risks & Prevention** **|**

3 events in cancer development: initiation, promotion and progression.

Basic Screening / early detection: Mammogram, PAP smears, skin exams, Digital rectal exam (DRE) for prostate, PSA, flex sigs/ colonoscopy. (See prevention chapter for specifics).

New Cancer Cases in Male: Prostate (25%) > Lung & bronchus (15%) > Colon & Rectum (10%) > Bladder (7%) > Melanoma (5%) > Non-Hodgkin Lymphoma (5%) > Kidney & renal pelvis (4%) > Oral Cavity (3%) > Leukemia (3%) > Pancreas (3%), other (18%). Prostate cancer rates are 1.5 times higher in African American men than in white men.

Male Cancer Deaths: Lung (31%) > Prostate (10%) > Colon & Rectum (8%) > Pancreas (6%) > Liver and Intrahepatic bile duct (4%) > Leukemia (4%) > Esophagus (4%) > Non-Hodgkin Lymphoma (3%) > Bladder (3%) > Kidney (3%) > Stomach > other sites (21%).

New Cancer Cases in Female: Breast (26%) > Lung (14%) > Colon & Rectum (10%) > Uterine (6%) > Non-Hodgkin Lymphoma (4%) > Melanoma (4%) > Thyroid (4%) > Ovary (3%) > > Kidney & renal pelvis (3%) > Leukemia (3%) > Pancreas (2%) > Other sites (20%). Breast cancer rates are about 20% higher in white women compared to African American women.

Female Cancer Deaths: Lung (26%) > Breast (15%) > Colon & Rectum (9%) > Pancreas (6%) > Ovary (6%) > Leukemia (3%) >Non-Hodgkin Lymphoma (3%) > Uterine (3%) > Brain & other nervous system (2%) > Liver & Intrahepatic bile duct (2%) > Melanoma > Thyroid > Urinary > Stomach > other sites (24%).

Diet and CA: Overnutrition**-->** in Prostate, breast, Ov, endom, colon. Undernutrition**-->** Stomach, liver, esoph, oropharynx. Nitrates (stomach), animal fat (colon and possibly breast & prostate).

Bacteria and Cancer: if grow *Streptococcus bovis/ sanguis* (Group D, non-enterococcus, alpha-hemolytic) in blood or *Clostridium septicum* in a cellulitis check a colonoscopy.

Other: Cancer will affect 1 in 2 men and 1 in 3 women in the United States, and the number of new cases of cancer is set to nearly double by the year 2050 due to aging and the growing US population (Oncologist. 2007;12:20-37).....Much of this increase in the number of new cancers will be in the 65-to-84-year age group, and this will reach a peak at 2030, as the baby-boomer generation ages. From 1993 to 2001, death rates from common cancers fell in the United States, but this decline was primarily confined to people with at least 16 years of education (J Natl Cancer Inst 2008;100)....subjects with this level of educational attainment, colorectal cancer mortality fell by 2.4% to 4.8% annually from 1993 to 2001. Cancer incidence and mortality rates in the United States are declining, it is still the second leading cause of death, coming after cardiovascular disease and has a total of 1,479,350 new cancer cases and 562,340 cancer-related deaths projected in 2009 (CA Cancer J Clin. 2009;Published online before print May 27).....Between 1990 and 2005, the overall cancer death rate decreased by 19.2% in men and by 11.4% in women. In men, the decline in morality rates was largely due to decreasing rates of lung (37%), prostate (24%), and colorectal (17%) cancers, which accounted for almost 80% of the total decrease. In women, the decreased mortality rate was largely driven by declining rates in breast (37%) and colorectal (24%) cancers, which accounted for 60% of the total decrease. Death rates from cancer have been decreasing in all age groups in recent years, but the steepest declines have been among younger people (Cancer Research 2009;69:Aug. 15).....The youngest age groups experienced the steepest average rate of decline at 25.9% per decade, the researchers say. But even the oldest groups recorded a 6.8% decline per decade.

*****Ref:** (CA Cancer J Clin 2009;59(4):221-36)

Lifetime risks for the 5 most common cancers:

Cancer Type: Diagnosis (%) **/** Risk of Death (%):
Prostate: 18.5 / 3.6.
Breast: (women)**-->** 12.6 / 3.6.
Lung: (men) **-->**8.6 / 7.1 (women) **-->**5.4 / 4.2.
Colorectal: (men) **-->** 6.2 / 2.6. (women) **-->** 5.9 / 2.6.
Bladder: (men) **-->** 3.3 / 0.7. (women) **-->** 1.2 / 0.4.

Prognosis:

Evidence on the strength of cancer prognostic factors may be biased by the selective publication and dissemination of studies and by selection of specific analyses that yield stronger estimates for the importance of the prognostic factor (J Natl Cancer Inst 2005;97:1023-1025,1043-1055). The cancer death rate for all sites combined has dropped in the US (J Natl Cancer Inst 2005;97:1407-1427), the cancer incidence has remained stable in men, but a slight increase has occurred in women.

WBC Count: There is a dose-dependent association between WBC count and cancer mortality in older individuals (Arch Intern Med. 2006;166:188-194)...the risk is greater with diabetes mellitus and hyperglycemia and lower with regular aspirin use. The RR for all cancer mortality for the highest quartile of WBC count (>/=7,400 cells/µL) vs the lowest quartile (</=5,300 cells/µL) was 1.73.

Conditional-Survival (CS): a calculation that paints a clearer picture of lont-term survivors' prognosis. Cancer-survival statistics are usually calculated from the time of dx, but for pt's who have survived for some time already. Most pt's' prognosis improves after they survived longer periods of time from when they were first diagnosed and treated. For example, in the head and neck squamous cell carcinomas (HNSCC) population, the 5-year CS increased from 47.8% at dx to a plateau of 64.4% within 3 years and after 6 years there is an increasing risk of HNSCC pt's dying from other disease processes (such as heart disease or stroke) rather than their primary cancer.

Cancer Transmission: There have been cases of transmission from organ donors to transplant recipients of both solid and nonsolid malignancies, as well as oncogenic viruses such as Kaposi's sarcoma–associated herpesvirus and Epstein-Barr virus. There have also been reports of cancer transmission via needle-stick injury or by surgical instruments, which demonstrates the ability of malignant cells to be transplanted to and graft onto immunocompetent hosts. Blood transfusions from donors with undiagnosed cancer do not appear to increase the risk for a future malignancy in recipients according to an analysis of 12,012 recipients who had been exposed to blood products from donors who eventually developed cancer (Lancet. 2007 369:1724–30, 1670-1671).

Misinformation: There appears to be a lot of misinformation about cancer risks according to a phone survey conducted by the American Cancer Society of 957 U.S. adults with no history of cancer (Cancer 2007; doi 10.1002/cncr.22880) (86.3% had at least a high school education).

The false statements, with percent marked "true" and "don't know," included: The risk of dying from cancer in the U.S. is increasing (67.7% true, 9.8% don't know). Living in a polluted city is a greater risk for lung cancer than smoking a pack of cigarettes a day (38.7%, 18.8%). Some injuries can cause cancer later in life (37.2%, 20.9%). What someone does as a young adult has little effect on their chance of getting cancer later in life (24.8%, 7.1%). Personal hygiene products, like shampoo and deodorants, can cause cancer (13.7%, 15.3%). Getting a mammogram can cause cancer of the breast (10.2%, 16.1%). You cannot get skin cancer from using a tanning booth (6.2%, 18.3%).

Proportions of Cancer Deaths Attributed to Preventable Factors:

Link: Lifestyle | **Environmental** | Smoking | **Diet** | NSAIDs & Other | **Annual & Medical Radiation Exposure** |

Range of estimates as to the causes: Tobacco: 25-40%. Alcohol: 2-4%. Diet: 10-50%. Food Additives: 1-2%. Reproductive/sexual behavior: 1-13%. Occupation: 2-8%. General pollution: 1-5%. Industrial products: 1-2%. Medical procedures: 0.5-3%. Geophysical factors: 2-4%. Infections: 1-10% (Cancer Causes & Control 1996;7:Suppl 1). Black Americans have a 33% higher death rate for all cancers than whites.

9 Key Modifiable Risk Factors:

Affect 35-39% of cancers deaths worldwide. Preventing cancer is preferable to early detection and tx...this is a far more effective and advantageous on a population level to prevent a disease than searching out cancers and destroying them.

Smoking: lung and many others. Smoking alone caused an estimated 21% of all cancer deaths worldwide. In high-income countries, 52% of risk-factor–attributable cancer deaths were from lung cancer, compared with 30% in low-income countries.

Obesity: endometrial, breast, and colorectal cancers.

Low fruit and vegetable intake: lung, esophageal, stomach, and colorectal.

Physical inactivity: breast and colorectal.

Alcohol use: breast, liver, oropharyngeal, and esophageal. Even low to moderate alcohol consumption significantly increases the risk for cancer in women, both overall and at specific sites according to results from the Million Women Study (J Natl Cancer Inst. 2009;101:296-305).....Consuming 1 drink per day increased this to an extra 15 cancers per 1000 women, and 2 drinks a day increased it to an extra 30 cancers per 1000 women.....Most were breast cancers, but the risk of cancer was also increased for the liver and rectum, and for the mouth and throat in women who also smoked.

.....in the United Kingdom, these estimates suggest that around 13% of cancers of the breast, upper aerodigestive tract, rectum, and liver — that is, 7000 cases annually — can be attributed to alcohol.

Unsafe sex: cervical (HPV).

Urban air pollution and indoor smoke from coal fires: lung.

Contaminated therapeutic injections: hepatitis-related liver cancers (healthcare settings). (Causes of cancer in the world: Comparative risk assessment of nine behavioural and environmental risk factors. Lancet 2005;366:1784-93)

33% of common adult cancers (and 24% of all cancers) may be preventable in the U.S. and that doesn't even count cancers that could be prevented by not smoking [World Cancer Research Fund (WCRF) and the American Institute for Cancer Research (AICR)].....eating a nutritious diet, being physically active, and keeping body fat under control may prevent:

•38% of breast cancers
•45% of colorectal cancers
•36% of lung cancers
•39% of pancreatic cancers
•47% of stomach cancers
•69% of esophageal cancers
•63% of cancers of the mouth, pharynx, or larynx
•70% of endometrial cancers
•24% of kidney cancers
•21% of gallbladder cancers
•15% of liver cancers

•11% of prostate cancers
Among the Recommendations:
•New developments should be designed to encourage walking and cycling.
•Government and school cafeterias should provide healthy foods and drinks.
•Food and drink industries should price healthy fare competitively with other products and stop promoting sugary drinks and unhealthy foods to kids.
•Workplaces should have policies and environments that are supportive of breastfeeding.
•Media should promote cancer prevention and flag misleading cancer claims.
Other Risk Factors:
1. Environmental factors are more important than heritable factors (EJM 2000;343:78).
2. Genetic Contribution / Family History: 5-40% of risk for major cancers (NEJM 2000;343:78).
3. Smoking and Smoke Exposure.
4. Chronic Viral Infections (such as hepatitis viruses B and C, human papilloma virus).
Xenotropic murine leukemia virus-related virus (XMRV): A retrovirus. A type of virus known to cause leukemia and sarcomas in animals has been found in malignant human prostate cancer cells, and this infectious agent might be responsible prostate cancer (PNAS 2009;Published online September 7).....Researchers from the University of Utah found evidence of XMRV in 27% of the prostate cancer samples that were examined, and the virus was associated with the more aggressive tumors.....The viral proteins were found almost exclusively in malignant prostate cells, suggesting that the XMRV infection is linked directly to the formation of tumors, the researchers note.....Detection of XMRV in 6% of the controls could indicate that the virus causes cancer only after a long induction period, the researchers suggest. Alternatively, these men might have cancer in an unsampled area of the prostate.
5. Chronic bacterial infections (such as Helicobacter pylori).
6. Exposure to electrical currents and magnetic fields does not cause cancer (Lancet 2001;357(9263):1143).
7. Chronic exposure to high doses of electrical currents may increase risk of childhood leukemia (Ahlbom A and Feychting M. Lancet 2001;357(9263):1143).
8. **Higher birth weight:** may increase the risk of some adult cancers (Int J Cancer 2005) (13% increase in the rate of digestive cancers and a 17% increase in the rate of lymphatic/hematopoietic cancers).
9. Infection: in 2005, 17% of new cancer diagnoses worldwide will be attributable to infection. (Ex HPV, HIV, EBV). The proportion of total cancer deaths attributable to infectious agents is estimate to be about 20-25% in developing countries and 7-10% in more industrialized countries (CA Cancer J Clin 2006;56:69-83).
10. **Immunodeficiency:** Analysis of studies involving 444,172 pt's with HIV/AIDS and 31,977 pt's with solid organ transplants (97% renal) finds that there is an increased cancer incidence in any immunosuppressed pt (Lancet 2007;370:59-67).....significantly increased incidences of cancers known to be related to EBV (lymphoma), human herpesvirus 8 (Kaposi sarcoma), HBC & HCV (liver cancer), H pylori (stomach cancer), and HPV (anogenital cancer, plus several other cancers possibly related to HPV). Lung cancers and leukemia were also increased in both groups. Thyroid and colorectal cancers were increased in transplant pt's only. Breast, prostate, and ovarian cancers were not increased in either group.
11. **Chronic Inflammation:** recurrent or persistent inflammation may induce, promote or influence susceptibility to carcinogenesis by causing DNA damage, inciting tissue reparative proliferation and/or creating a stromal "soil" that is enriched with cytokines and growth factors (CA Cancer J Clin 2006;56:69-83). Examples include prostatitis, COPD, colitis, chronic diffuse infiltrating lung diseases (asbestosis, silicosis), GERD, hepatitis, HPV, EBV and H pylori.
Other: Having more than a high school education is associated with a lower risk of dying from cancer (J Natl Cancer Inst 2007;99:1384-94,1356-57).... men with 12 years of education or less die of cancer at more than twice the rate of those with more years of schooling.
Lifestyle Modification: …….END DEMO of this chapter.

ICU - Fluids - Electrolytes - Nutrition:

Edited by Andy S. Binder, MD, Pulmonologist, Critical Care.

Contents: Electrolytes | **Fluids** | Dehydration | **Shock** | Nutrition | **Nutritional Support** | Common Conversions | **Therapeutic Levels** | Hypoxemia | **Hypoxia** | Pulse Oximetry | **Hypercapnia** | Intubation | **Mechanical Ventilation** | Sedation | **Vascular Access** | PA Cath Patterns | **Acid-Base Physiology** | Admission and Notes | **Healthy Living** | Common Nutrients / Vitamins | **Orders & Notes** | Withholding / Withdrawing Care | ***INDEX*** |

Most Common Reasons for ICU-CCU Admissions:
1. Respiratory failure: Acute respiratory distress syndrome (ARDS). Pneumonia. Exacerbation of chronic lung disease (COPD, asthma, bronchiectasis). Pulmonary embolism (PE).
2. Shock: Hypovolemic (GI bleed and diarrhea). Cardiogenic (acute MI). Distributive (sepsis and anaphylaxis). Obstructive (massive PE and cardiac tamponade).
3. Cardiac disease: Acute MI. Unstable angina. Decompensated heart failure. Valvular heart disease. Arrhythmia with hemodynamic instability.
4. Trauma: massive, involving multiple organs.
5. Burns.
6. CNS injury: Stroke. Closed head injury. Intracranial hemorrhage. Status epilepticus.
7. Infections: Septic shock. Toxic shock syndrome. Meningitis. Neutropenic fever.
8. Metabolic derangements: Acute renal failure/uremia. Diabetic ketoacidosis. Severe hypo- or hypernatremia. Severe hypo- or hyperkalemia. Severe hypophosphatemia. Intentional or accidental poisoning.
9. GI diseases: GI hemorrhage. Acute liver failure.
10. Hematologic disease: Severe anemia/thrombocytopenia. Hemolytic uremic syndrome (HUS)/thrombotic thrombocytopenic purpura (TTP). Disseminated intravascular coagulation (DIC).
11. Psychiatric diseases: Suicide attempt. Neuroleptic malignant syndrome (NMS). ICU patients whose care is managed by intensivists have a higher hospital mortality rate than those managed otherwise according to an analysis of 101,832 patients from

123 ICUs across the US (Ann Int Med 2008;148:801-809). Factors Predicting Post-ICU Blues Intensive care patients who suffered multiple organ failure, underwent surgery, or received high doses of benzodiazepine were at significantly increased risk for depressive symptoms six months after ICU discharge (Crit Care Med 2009;DOI: 10.1097/CCM.0b013e31819fea55).
Care of a patient post-ICU hospitalization: (Am Fam Physician. 2009;79:459–464).
• Often have generalized weakness or fatigue, which mandates assessment for possible anemia, nutritional deficits, sleep disturbance, muscular deconditioning, medication adverse effects, and/or neurologic impairment such as myopathy or polyneuropathy. Impaired mobility may lead to falls, difficulty with stair climbing, and/or sexual dysfunction. Possible psychological comorbidities may include posttraumatic stress disorder (PTSD), anxiety disorder, and depression. Families and caregivers may also have depression and anxiety.
• **W/u:** CBC, iron panel, serum chemistries, serial body weight measurement, and/or consultation with physical therapy or possibly neurology.
Specific key clinical recommendations for practice, all rated level of evidence C, are as follows:
• Post-ICU patients, especially those who are older, women, or have a history of sepsis or malignancy, should be monitored for the development of anemia.
• Risk for PTSD is higher in post-ICU patients who are younger, have received sedatives, have been on prolonged mechanical ventilation, and/or have a history of acute lung injury or traumatic brain injury.
• Patients who had delirium during their ICU stay should be evaluated for anxiety.
ICU Errors: Parenteral administration of drugs "is a weak point in patients safety in intensive care" according to a study (BMJ 2009;338:b814).....There were 75 errors per 100 patient-days.....Three quarters were errors of omission (drug given at wrong time or dosage missed).....Most mistakes occurred not during crises or unusual circumstances, but during routine situations.....Roughly 1% of the population suffered permanent harm or death due to an error.....Error likelihood rose in the face of higher patient/nurse ratios and dropped in the presence of a critical incident reporting system.
GI Ulcer Prophylaxis: There's new concern about overuse of proton pump inhibitors (PPIs) and H2-blockers in hospitalized patients. There's no proof acid-suppressing drugs prevent GI bleeding in hospitalized patients and they may cause harm as PPIs may increase the risk of C difficile diarrhea or hospital-acquired pneumonia (Prescriber's Letter 2009;25(7):250701).....PPIs and H2-blockers might increase infection risk by increasing gastric pH and allowing bacterial overgrowth in the GI tract and airways.....Stop prophylaxis when patients leave the ICU.
Infections: Chlorhexidine bathing daily (4 oz. of 4% solution mixed in warm water) significantly reduced the acquisition of MRSA and VRE, and the incidence of VRE bacteremia, in ICU patients (Crit Care Med 2009;37:1858).
Risk for Invasive Candidiasis in the ICU: (Crit Care Med 2009;37:1624) A study on 1107 nonneutropenic adults found that 20% of the patients were neither colonized nor infected with Candida, 75% were colonized at 1 site, and 5% developed IC. Most IC cases were detected during patients' second or third ICU week. IC occurred in 2% of patients with a CS <3 and in 9%, 17%, and 24% of those with a CS of 3, 4, and 5, respectively. The association between increasing CS values and IC incidence was highly significant. Among patients with Candida colonization or invasive fungal infection, (1–3)-beta-D-glucan serum levels were measured for 240, and anti-Candida antibody titers were determined for 323. A CS 3 and a (1–3)beta-D-glucan serum level >75 pg/mL were independent predictors of IC; anti-Candida antibody titers were not useful in this regard.

Serum Electrolytes:

Links: CO2/bicarb | Sodium (Na) | **Magnesium (Mg)** | Potassium (K) | **Calcium (Ca)** | Phosphate (P) | **Fluids |** Chloride | See Cardiology for **Intensive Glucose Control & GIK |**

Always repeat to r/o artifact (laboma). Laboratory errors are common. Results should be double-checked when the clinical picture and the lab data conflict. When altered, fluids and electrolytes should generally be corrected in the following order: (1) volume; (2) pH; (3) potassium, calcium, magnesium; and (4) sodium and chloride. Re-establishment of tissue perfusion often re-equilibrates the fluid-electrolyte and acid-base balance. Because the osmolarity of normal saline matches that of the serum, it is an excellent fluid for volume replacement. Abnormalities should be corrected at the same rate they developed; however, slower correction is usually safe unless the condition warrants rapid and/or early intervention (ie, hypoglycemia, hyperkalemia).

ICD-9 Codes:
269.3 Mineral deficiency, not elsewhere classified
276.9 Electrolyte and fluid d/o's not elsewhere classified - chloride

Bicarbonate: Major extracellular anion with Cl.
Basic Science: The kidneys and lungs maintain daily acidbase balance. Bicarbonate and carbonic acid constitute the major buffer pair in body fluids. Carbonic acid dissociates into hydrogen ion and bicarbonate with a dissociation constant of 7.95 x 107. Carbonic acid also maintains an equilibrium with H2O and CO2. We produce ~1 mEq/kg daily of hydrogen ions (derived from metabolism of proteins primarily). The kidney normally excretes this daily acid load. Failure of excretion forces the reaction of H+ and HCO3, resulting in a decrease of bicarbonate concentration.
Serum Total Carbon Dioxide: Measururing serum total CO2 content rather than just serum bicarbonate includes the serum bicarbonate as well as available forms of carbon dioxide (i.e., dissolved CO2 and carbonic acid). Generally, the serum bicarbonate comprises about 95% of the total CO2 content; thus we can use this measurement as an excellent estimator of serum bicarbonate. The total CO2 content normally equals 23 to 30 mEq/L of serum. Arterial blood gas (ABG) reports generally include a bicarbonate value. The blood gas machine measures pH and pCO2 and then calculates a bicarbonate value using the HendersonHasselbalch equation. Generally, a concurrent venous total CO2 content will exceed this value by < 2 to 4 mEq/L, of which 1 to 2 mEq/L represents the difference between venous and arterial blood; the remaining difference comes from dissolved CO2.
Sodium Bicarb: [325 mg (3.8 mEq), 650 mg (7.6 mEq) tabs. Powder: 120, 480 g. Injection: 4.2% (0.5 mEq/mL) (10 mL), 7.5% (0.89 mEq/mL), 8.4% (1 mEq/mL) (10, 50 mL). Injection, premixed: 5% (0.6 mEq/mL) (500 mL)] Each 1 mEq bicarbonate provides 1 mEq Na+. One g sodium bicarbonate provides 11.9 mEq each of sodium and bicarbonate. An alkalinizing agent and electrolyte.
Correction of metabolic acidosis: Calculate pt's dose with the following formulas.
Adult: HCO3– (mEq) = 0.2 x weight (kg) x base deficit (mEq/L), OR HCO3– (mEq) = 0.5 x weight (kg) x [24 – serum HCO3– (mEq/L)].

Neonate, infant, and child: HCO3– (mEq) = 0.3 x weight (kg) x base deficit (mEq/L), OR HCO3– (mEq) = 0.5 x weight (kg) x [24 – serum HCO3– (mEq/L)].
Urinary alkalinization (titrate dose accordingly to urine pH): Child @ 84–840 mg (1–10 mEq)/kg/24 hr PO divided QID. Adult @ 4 g (48 mEq) x 1 followed by 1–2 g (12–24 mEq) PO Q4 hr. Doses up to 16 g (192 mEq)/24 hr have been used.
Info: Contra in respiratory alkalosis, hypochloremia, and inadequate ventilation during cardiac arrest. Use with caution in CHF, renal impairment, cirrhosis, hypocalcemia, hypertension, and concurrent corticosteroids. Maintain high urine output. Monitor acid-base balance and serum electrolytes. May cause hypernatremia (contains sodium), hypokalemia, hypomagnesemia, hypocalcemia, hyperreflexia, edema, and tissue necrosis (extravasation). Oral route of administration may cause GI discomfort and gastric rupture from gas production. For direct IV administration (cardiac arrest) in neonates and infants, use the 0.5 mEq/mL (4.2%) concentration or dilute the 1 mEq/mL (8.4%) concentration 1:1 with sterile water for injection and infuse at a rate no >10 mEq/min. The 1 mEq/mL (8.4%) concentration may be used in children and adults for direct IV administration. For IV infusions (for all ages), dilute to a maximum concentration of 0.5 mEq/mL in dextrose or sterile water for injection and infuse over 2 hr using a maximum rate of 1 mEq/kg/hr. Sodium bicarbonate should not be mixed with or be in contact with calcium, norepinephrine, or dobutamine.
Cardiac arrest: Bicarbonate administration in this situation may be detrimental. Administer according to results of arterial blood pH and PaCO2 and calculation of base deficit. Flush IV lines before and after use.
Adults - A rapid IV dose of 200 to 300 mEq of bicarbonate, given as a 7.5% or 8.4% solution.
Infants (2 years of age or younger) - 4.2% solution for IV administration at a rate not to exceed 8 mEq/kg/day to guard against the possibility of producing hypernatremia, decreasing CSF pressure, and inducing intracranial hemorrhage.
Initial dose - 1 to 2 mEq/kg/min given over 1 to 2 minutes followed by 1 mEq/kg every 10 minutes of arrest. If base deficit is known, give calculated dose of 0.3 × kg × base deficit. If only 7.5% or 8.4% sodium bicarbonate is available, dilute 1:1 with 5% dextrose in water before administration.
Severe metabolic acidosis: Administer 90 to 180 mEq/L (approximately 7.5 to 15 g) at a rate of 1 to 1.5 L during the first hour. Adjust to patient's needs for further management.
Less urgent forms of metabolic acidosis: Sodium bicarbonate injection may be added to other IV fluids. The amount of bicarbonate to be given to older children and adults over a 4- to 8-hour period is approximately 2 to 5 mEq/kg, depending on the severity of the acidosis as judged by the lowering of total CO2 content, blood pH, and clinical condition. Initially, an infusion of 2 to 5 mEq/kg over 4 to 8 hours will produce improvement in the acid-base status of the blood.
Estimates of the initial dose of sodium bicarbonate may be based on the following equation:
0.5 (L/kg) × body weight (kg) × desired increase in serum HCO3- (mEq/L) = bicarbonate dose (mEq) or 0.5 (L/kg) × body weight (kg) × base deficit (mEq/L) = bicarbonate dose (mEq). The next step of therapy is dependent on the clinical response of the patient. If severe symptoms have abated, reduce frequency of administration and dose. If the CO2 plasma content is unknown, a safe average dose of sodium bicarbonate is 5 mEq (420 mg)/kg. It is unwise to attempt full correction of a low total CO2 content during the first 24 hours, because this may accompany an unrecognized alkalosis due to delayed readjustment of ventilation to normal. Thus, achieving total CO2 content of about 20 mEq/L at the end of the first day will usually be associated with a normal blood pH.
Neutralizing additive solution: One vial of neutralizing additive solution added to 1 L of any of the commonly used parenteral solutions including dextrose, sodium chloride, Ringer's, etc, will increase the pH to a more physiologic range (specific pH may vary slightly). Some products such as amino acid solutions and multiple electrolyte solutions containing dextrose will not be brought to near physiologic pH by the addition of sodium bicarbonate neutralizing additive solution. This is due to the relatively high buffer capacity of these fluids. Avoid adding sodium bicarbonate to parenteral solutions containing calcium, except where compatibility is established; precipitation or haze may result. Norepinephrine, dopamine, and dobutamine are incompatible.

Sodium (Na) Metabolism:

Links: FENa & PP | **Hyponatremia** | SIADH | **Hypernatremia** | See Nephro module: **Na intake (foods) & HTN** |

Major determinant of body tonicity, primarily an extracellular cation. Serum Na reflects body water.
Na requirements: 3 mEq/L ECF = 0.6 X wt in kg X 3. Adults need ~75-150 mEq/d. Child needs 3-5 mEq/kg/d. 1 teaspoon table salt = 100 mEq of Na. 1g salt = 17 1 mEq of Na. 1g Na = 43.5 mEq of Na.

ICD-9 Codes:
276.0 Hyperosmolality and/or hypernatremia
276.1 Hyposmolality and/or hyponatremia

FENa: [Ur_{Na} X S_{Cr}/ S_{Na} X U_{Cr}] X 100**-->** <1 = pre-renal. > 1= renal (ATN). FENa is the fraction of Na in the urine filtered by the glomerulus and not reabsorbed.
Corrected Na for Blood Sugar = [0.016 x (BS-100)] + Na (measured).
As Na falsely decreases 1.6 mEq/L for each 100mg/dL inc glucose over 100mg/dL.
As blood glucose rises from 200 to 1200 mg/dL in an untreated diabetic pt, the plasma sodium concentration would be expected to fall from 140 to 124 mEq/L (1.6 mEq/L x 10 = 16 mEq) without a change in total body water and electrolytes. Conversely, tx with insulin and lowering of the blood sugar from 1200 to 200 mg per dL in this diabetic pt would result in comparable osmotic water movement from the ECF into cells and a return of plasma sodium concentration to 140 mEq/ L without any change in total body water or electrolytes.
PP: Serum sodium concentration and serum osmolarity normally are maintained under precise control by homeostatic mechanisms involving stimulation of thirst, secretion of antidiuretic hormone (ADH), and renal handling of filtered sodium. ADH (antidiuretic hormone = **AVP** = arginine vasopressin) is released by the posterior pituitary in response to either volume depletion or an osmolar stimulus (inc in plasma osmolarity >280-290 mOsm/kgH2O). Below this stimulus the urine should be maximally dilute (Osm <100 mOsm/kg/H2O). ADH tells the kidneys to excrete Na to save water (opposite of aldo effect to “save salt”). Serum sodium concentration is regulated by stimulation of thirst, secretion of ADH, feedback mechanisms of the renin-angiotensin-aldosterone system, and variations in renal handling of filtered sodium. Increases in serum osmolarity above the normal range (280-300 mOsm/kg) stimulate hypothalamic osmoreceptors, which, in turn, cause an increase in thirst and in circulating levels of ADH. ADH increases free water reabsorption from the urine, yielding urine of low volume and relatively high osmolarity and, as a result, returning serum osmolarity to normal. ADH is also secreted in response to hypovolemia, pain, fear, nausea, and hypoxia. Aldosterone, synthesized by the adrenal cortex, is regulated primarily by serum potassium but also is released in response to

hypovolemia through the renin-angiotensin-aldosterone axis. Aldosterone causes absorption of sodium at the distal renal tubule. Sodium retention obligates free water retention, helping to correct the hypovolemic state. The healthy kidney regulates sodium balance independently of ADH or aldosterone by varying the degree of sodium absorption at the distal tubule. Hypovolemic states, such as hemorrhage or dehydration, prompt increases in sodium absorption in the proximal tubule. Increases in vascular volume suppress tubular sodium reabsorption, resulting in natriuresis and helping to restore normal vascular volume. Generally, disorders of sodium balance can be traced to a disturbance in thirst or water acquisition, ADH, aldosterone, or renal sodium transport.

Hyponatremia:

Links: Etiology | S/s | **W/u |** Spot Ur Na | **Ur Osm |** Tx | **Hypotonic (Hyposmotic, can be hypo-hyper or isovolemic) |** SIADH | **Isotonic (Isosmotic) |** Hypertonic (Hyperosmotic) | **Volume State | See GI Module for** Post-op Hyponatremia |

Too much extracellular water relative to Na. **Na <136** mEq/L (normal = 136-145). Hyponatremia implies a relative excess of total body water to sodium and is seen in a variety of medical conditions including congestive heart failure, liver disease, the syndrome of inappropriate antidiuretic hormone, and as a result of medications (eg, thiazide diuretics, psychotropic agents, and chemotherapeutic agents). Hyponatremia is most common in the extremes of age; these groups are less able to experience and express thirst and less able to regulate fluid intake autonomously. Hyponatremia is the most common electrolyte abnormality in hospitalized individuals, affecting up to 30% of patients in some series (Am J Med. 2006;119(7 Suppl 1):S30–S35).....The risk of death during hospitalization is increased by more than 50% in patients admitted with hyponatremia compared with normonatremia, but the magnitude of the risk according to the actual degree of hyponatremia has not been well studied.
Mild hyponatremia: serum Na 130–134 mmol/L. Hyponatremia, even when mild (Na 130-134 mEq/L), is associated with increased mortality in hospitalized pt's (hazard ratio 1.25)(Am J Med 2009;122:857-865).
Marked hyponatremia: serum Na <130 mmol/L.
Na^+ deficit: 0.6 X (wt in kg) X (140- Na). The tonicity (osmolality) is usually low (hypotonic), but may be normal or high.
Causes: Dilutional hyponatremia, which includes euvolemic and hypervolemic hyponatremia, is the most common form of the condition, and occurs when retained water dilutes serum sodium content. It often results from elevated levels of AVP, which regulates water and salt balance in the body.
Drugs: ACEi, SSRI's, diuretic (thiazides a common cause), Carbamazepine (Tegretol), chlorpromazine (Thorazine), vasopressin analogs, Indapamide (Natrilix), theophylline, amiodarone (Cordarone), ecstasy (3,4-methylenedioxymethamphetamine). SIADH in the elderly can be caused by CNS agents such as TCA's, SSRI's, NRI's, Mirtazapine, phenothiazines(Crit Care Clin 2001;17:11-230). Antineoplastic agents (vincristine, vinblastine, cyclophosphamide). Oxytocin, Chlorpropramide. Clofibrate. Narctotics.
Renal Na loss: diuretic, adrenal insufficiency, RTA, salt losing nephritis.
Nonrenal Na loss: emesis, diarrhea, pancreatitis, rhabdo, burns.
Water excess: SIADH, secondary adrenal insuf, hypotonic fluids.
Hypovolemic: Saline excess with dec ECV**-->** CHF, liver cirrhosis, nephrotic syndrome.
Euvolemic hyponatremia: TBW increases while total sodium remains normal. The ECF volume is increased minimally to moderately but without the presence of edema.
Hypervolemic hyponatremia: Total body sodium increases, and TBW increases to a greater extent. The ECF is increased markedly, with the presence of edema.
Redistributive hyponatremia: Water shifts from the intracellular to the extracellular compartment, with a resultant dilution of sodium. The TBW and total body sodium are unchanged. This condition occurs with hyperglycemia or administration of mannitol.
Pseudohyponatremia: The aqueous phase is diluted by excessive proteins or lipids. The TBW and total body sodium are unchanged. This condition is seen with hypertriglyceridemia and multiple myeloma.
Saline Excess w/o dec ECV--> ARF, CRF.
Meds enhancing ADH effect: nicotine, high dose morphine, epi, cyclophosphamide, tolbutamide, chlorpropamide, NSAIDs, SSRI's. Older pt's with depression who are being treated with paroxetine may be at increased risk of hyponatremia (12% incidence, mean time to development is 9.3 days) (Arch Intern Med 2004;164:327-332).
Postoperative hyponatremia: due to alterations in ADH, renal function, excessive fluid administration, or drug effects occurs in ~4.4% of all surgical pt's (Arch Intern Med 1986;146:333–336). See GI – Surgery module.
Cerebral Salt Wasting (CSW): The mechanism by which intracranial disease leads to CSW is not well understood. The most probable process involves the disruption of neural input into the kidney and/or the central elaboration of a circulating natriuretic factor. Atrial natriuretic peptide and BNP produce several effects that could lead to the clinical syndrome of CSW. Making an accurate dx between SIADH and CSW in pt's undergoing neurosurgery who develop hyponatremia is important because the tx of each condition is quite different. Vigorous salt replacement is required in pt's with CSW because of ECV contraction and thus they require volume replenishment with intravenous saline or administration of salt tablets instead of fluid restriction until the transient (3–4 weeks) condition of renal salt wasting resolves. Drugs. Diseases associated with SIADH.
Aging causes: (Clini Geri 2009;9:34-39) 7% incidence. Aging leads to hyponatremia due to physiologic changes (diminished total body water, decr renal sodium conserving ability, altered tubular function), increased water intake, decreased sodium intake, increased sodium loss (renal disease, GI loss via vomiting, diarrhea, GI suctioning). Idiopathic SIADH of the elderly person (age >80, race other than African American).
S/s: muscle twitching, inc DTR, sz, HTN (secondary to inc ICP), salivation, lacrimation, watery diarrhea. Clinically significant hyponatremia tends to be nonspecific in its presentation; therefore, the physician must consider the dx a pt presenting with vague constitutional sx's or with altered level of consciousness. Pt's with acute hyponatremia (developing over <48 hr) tend to have more severe degrees of cerebral edema (and thus sx's) for a given serum sodium level.
Sx's include: lethargy, apathy, disorientation, muscle cramps, anorexia, nausea, agitation. Some can have a Na <120 w/o sx.
Signs include: abnormal sensorium, depressed deep tendon reflexes, Cheyne-Stokes respiration, hypothermia, pathologic reflexes, pseudobulbar palsy, seizures.
W/u: Repeat blood draw (confirm accuracy). Examine pt for acute neurologic changes. Clinically determine the extracellular fluid volume status, get initial wt, daily wt's. R/o hyperglycemia or other **etiology**.
Labs: Check serum osmolality, Ur Osm, Chem 7, Alb, Ca, Mg, spot urine Na/ K/ Cl/ Cr, FENa [= (U_{Na} X $Plasma_{Cr}$ X 100)/ (P_{Na} X U_{Cr})]. Step 1 (serum Osm), Step 2 (voume status), step 3 (urine osm), step 4 (other labs)(SEE ALL BELOW).

Step #1: Assess:
Serum Osmolality: a major determinant of total body water homeostasis, is measured by the number of solute particles present in 1 kg of plasma. It is calculated in mmol / L: = 2 X [sodium] + [urea] + [glucose].
A. Normal (280-295) = isoosmotic hyponatremia (pseudohyponatremia) and is due to either:
1. Hyperproteinemia or 2. Hyperlipidemia. 3. infusion of glucose or mannitol.
B. Elevated (>295) = hypertonic hyponatremia and is due to either:
1. Hyperglycemia (no osmolar gap) or 2. Hypertonic infusions (mannitol, glycine, glucose or contrast agents).
C. Low (<275-280) = hypotonic hyponatremia. ---> rules out pseudohyponatremia or unmeasured solutes.
Step #2: Assess volume status and Ur-Na. Link: **Volume State |**
A. Hypovolemic (ECF volume dec) = total body depletion of Na disproportionately to water losses, due to:
1. Urine Na >20 = renal loss from osmotic diuresis (urea, mannitol, glucose), salt-losing nephropathy, diuretic, proximal RTA, adrenal insufficiency, vomiting with bicarbonaturia and obligate Na loss (RTA, metabolic alkalosis). Urinary obstruction, ketonuria.
2. Urine Na <10-20 = extrarenal loss. GI loss (vomiting, diarrhea, NG suction), skin loss (burns, sweating), lung losses, third space loss, traumatized muscle, post-diuretic therapy.
B. Hypervolemic = retention of free water.
1. Urine Na >20 = renal failure (acute or chronic).
2. Urine Na <20 = nephrotic syndrome, CHF, cirrhosis.
C. Euvolemic = a mixed bag.
1. Urine Na >20 = renal failure, SIADH, hypothyroidism, potassium loss, various drugs (amitriptyline, carbamazepine, clofibrate, Cytoxan, morphine, vincristine), selective glucocorticoid deficiency, reset osmostat, stress (pain, emotion, post-op).
2. Urine Na <20 = water intoxication.
Calculating the Na (mEq) deficit: Not very accurate as it assumes a closed system (kidneys clamped), in real life the kidneys assist in correcting the deficit much quicker.
Na deficit = 0.6 x (wt kg) x (desired [Na] - actual [Na]).
Ex: if wt is 64kg and serum Na is 113**-->** 0.6 (113) X 140-113 = 38.4 X 27 = ~1040. If have 154 mEq Na in NS, then 1040 / 154 = ~6.7 L NS needed to correct. To estimate the effect of 1L of infusate on serum Na:
Change in Na = [(infusate Na +K) - serum Na] / (total body water - 1).

Spot Ur Na in Hyponatremia:

Interpret based on estimated volume status. Ur Na reflects aldo effect.
1. Inc Extracellular Volume (Hypervolemic):
<10-20 mEq/L --> CHF, Cirrhosis, Nephrotic, hypo-albumen.
>20-40 mEq/L --> Renal Failure (acute or chronic).
2. Dec Extracellular Volume (Hypovolemic):
<10-20 mEq/L --> Emesis, diarrhea, pancreatitis, lung/ skin loss (burns).
>20-40 mEq/L --> Renal Loss: Na-wasting nephropathy, osmotic diuretics, obstruction, RTA, adrenal insufficiency.
3. Isovolumic (Euvolemic):
<10-20 mEq/L --> Water intoxication.
>20-40 mEq/L --> Renal failure, hypothyroid, pain, SIADH, emotion, adrenal insufficiency, post-op states. Variable Urine-Na concentration with decreased solute intake, psychogenic polydipsia and resetting of osmostat (pregnancy, psychiatric d/o's).

Step #3:

Urine Osmolality: Reflects ADH effect. Amount of solutes in urine, depends on fluid & solute intake, kidney function and water intake. The appropriate response to hyponatremia is maximally dilute using (N100 mOsm/kg H2O). Limited value as nearly all pt's with hyponatremia do not maximally dilute their urine except in psychogenic polydipsia or reset osmostat.
"Normal" is >700-1400 mOsm/kg. If 600-700 then minimal impairment in renal concentrating ability.
If 400-600 moderate impairment.
If <400 mOsm/kg then severe impairment.
In complete diabetes insipidus (DI) Ur Osm 50-200. In partial DI 250-500.
In nephrogenic DI 100-200. In primary (psychogenic) polydipsia Ur Osm are 700-1200 mOsm/kg. In high-set osmoreceptor DI it is 700-1400.
Step #4: test thyroid and adrenal function if clinical suspicion. Serum uric acid and BUN are helpful in differentiating euvolemic SIADH from hypovolemic (low levels of either are compatible with euvolemia and SIADH.
Tx: Correct underlying d/o with volume and correct the cause. Correct any hypokalemia as Na-K ATPase regulate intracellular concentrations. See **SIADH |**
Sample Outpatient Tx Plan: If mild or chronic and tolerating PO's. See Fluid intake reccomendations | Tx the **Etiology** such as stopping HCTZ etc. If appropriate, give Furosamide 20-80mg PO qd x 3-8 days. See workup. Repeat labs in 3-6 days. Water restricting to <800-1000 ml/day will ameliorate all forms of hyponatremia, but it is **not** considered the optimal therapy as it is uncomfortable to the pt and can lead to dehydration with orthostatic sx's.
Info: A 4g "salt" diet is not the same as a 4g "Na" diet (there is 1g of Na in 2.5 g of salt based on the molecular wt of Na and Cl). There are 3.5g of Na in 1L of NS (154 mEq/L x 0.023g in 1 mmol).
NaCl (Salt) tablets: Typical Rx is 1g/tab, take 2 tabs TID for 7 days.
Loop diuretics: with K and Na replacement in hypervolemic forms. Furosamide 40-80mg PO/IV.
If symptomatic--> Hypertonic saline (3-5%) @ 2ml/kg/hr, to inc Na by a max 1-2 mEq/h, up to serum Na of 125-130. The initial rate of correction can be 1-2 mmol/L/hr in pt's with severe sx's, but the maximum rate of correction should not exceed 8 mmol/L on any day of tx. Max rate of infusion of 100ml of 5% saline/h to avoid **central pontine myelinolysis** (demyelination, think hy**po** – **po**ns, hyp**e**r = **e**dema- cerebral). A lag time of 1 to 2 days usually is required before mental status clears. A case of Klüver Bucy syndrome with bitemporal myelinolysis is presented as an unusual consequence of excessively rapid correction of severe hyponatremia (107mmol/L, with an increase of 30mmol/L over 36h) (La Presse Médicale 2008;37:975-977).

Rule of thumb: 1ml/kg 3%NS **raises serum** Na by 1mEq/L. Correct slowly over 48hr. To raise the sodium 1mEq/hr infuse at rate of**-->** (wt in kg X 0.6) / (0.513 mEq/L X 1hr). Or take the amount of NS calculated from the deficit and divide by 24hr. The pt usually correct faster as the calculations are based on the kidneys being clamped, so re-check in 4-6hr to make sure not correcting too quick as you may need to add free water (½NS or D5W).

Vaprisol (Conivaptan): [20mg/4ml ampule, costs $450/dose] loading dose of 20 mg IV followed by 20 mg administered as a continuous infusion over 24 hours. Following the initial day of tx, it is administered for an additional 1 to 3 days as a continuous infusion of 20 mg/day. If serum sodium does not rise at the desired rate, Vaprisol may be titrated upward to a daily dose of 40 mg, again administered in a continuous infusion. An arginine vasopressin (AVP) antagonist for the IV tx of euvolemic hyponatremia in hospitalized pt's, results in increased urine output without loss of valuable electrolytes such as Na and K ("aquaresis") to correct serum Na levels. FDA approved in 12/05 to treat euvolemic hyponatremia and in 3/07 as a tx for hypervolemic hyponatremia. The safetly and effectiveness of Conivaptan has not been compared directly with hyertonic saline and in most cases the hyponatremia resolves with fluid restriction alone (Am Fam Phys 2008;78:984-89)….typically 40mg/d will increase the serum Na after 24 hours by 6.4 mEq/L (6.4 mmol/L) more than placebo, after 72 hours this rises to a total Na increase of 6.9 mEq/L.

SE: infusion site reactions, hypokalemia, H-A, thirst and vomiting. **Contra:** pt's who have hypovolemic hyponatremia, co-administration with potent CYP3A4 inhibitors, such as ketoconazole, itraconazole, clarithromycin, ritonavir, and indinavir. Pregnancy Class C.

Tolvaptan: Not FDA approved as of 2/08. Start @ 15 mg/d, increased to 30 or 60 mg/d if necessary. An oral vasopressin-receptor antagonist. At baseline, mean serum sodium concentration was 129 mmol/L, at day 4, it was significantly higher in the tolvaptan group than in the placebo group (135 vs. 130 mmol/L) (NEJM 2006;355:2099-112). Findings were similar at 30 days. On a standardized survey of general health, scores on the mental, but not the physical, component were improved in the tolvaptan group compared with the placebo group. Dry mouth and thirst were the most common side effects of tolvaptan use…no effect on renal function, heart rate, electrolyte imbalance, or blood pressure. Closely follow serum sodium and daily measurements of body weigh. Tolvaptan is safe in hospitalized acute decompensated heart failure (ADHF) pt's who have reduced LVEFs (The EVEREST trial. JAMA 2007;297:1332-43)....tx lowered body weight and corrected hyponatremia better than placebo, but it did not improve survival or overall pt well-being. In 5/09, the FDA has approved tolvaptan tablets for use in hyponatremia associated with heart failure, cirrhosis, and the syndrome of inappropriate antidiuretic hormone secretion. A vasopressin-receptor antagonist, will be dispensed with a boxed warning that it only be started in hospitals, where sodium levels can be monitored closely because if sodium levels rise too quickly, osmotic demyelination syndrome can result.

Prognosis: Patients with hyponatremia are twice as likely (9.1% vs 4.1%) to suffer a major fracture as those who do not have low sodium levels according to emergency-department records on 364 patients over the age of 65 with a sustained fracture [of the hip, pelvis, or femur] and a sodium level that was considered mild to severe hyponatremia (National Kidney Foundation 2009 Spring Clinical Meetings: Abstract 75. Presented March 26, 2009)....The overall sodium level was 131 ± 1.9 mEq/L, and females were 1.6 times more likely than males to suffer fractures. The mean age of the fracture group was 79.2 years and of the nonfracture group was 82.8 years. 24% of patients in the fracture group were taking selective serotonin reuptake inhibitors, whereas none of the patients in the nonfracture group were taking that class of medication.

1. Hypotonic (Hyposmotic hyponatremia):

Plasma Osm <280 mOsm/kg (normal = 280-295).
Check Uosm **-->** if <150, then psychogenic polydipsia as appropriate dilution.
If Uosm >150**-->** assess renal function to r/o primary renal dz. If normal renal function, assess extracellular volume status.

W/u: **Check** urine Osm, if ≤150 mOsm/kg then psychogenic polydipsia. If >150 mOsm/kg assess renal function. If inc BUN or Cr, then primary renal dz. If normal renal function, assess extracellular fluid volume, check Ur_{Na}. Consider the Cosyntropin Stimulation Test for adrenal insufficiency.

Etiology of Hypotonic Hyponatremia:

1. Excessive water intake: primary polydipsia, accidental intake of water (swimming lessons), dilute infant formula, Na-free irrigant solutions during surgery, multiple tap-water enemas.

2. Impaired capacity of renal water excretion. Salt-wasting nephropathy can be seen in acute or chronic renal insufficiency, in which the patient may be unable to excrete adequate amounts of free water.

Cerebral salt-wasting syndrome: can be seen in patients with traumatic brain injury, aneurysmal subarachnoid hemorrhage, and intracranial surgery. Must be distinguished from SIADH because both conditions can cause hyponatremia in neurosurgical patients, and yet the pathophysiology and treatment are different.

A. Decreased Volume of Extracellular Fluids (Hypovolemic): Hypovolemic hyponatremia occurs when total body water (TBW) decreases; total body sodium (Na+) decreases to a greater extent. The extracellular fluid (ECF) volume is decreased. Loss of isotonic fluids or replacement with inadequate volume of excessively hypotonic fluid. GI loss (vomit, diarrhea), skin, lungs, kidneys (diuretics, RTA, diuretic phase of ATN, adrenal insufficiency, hypo-aldo), third spacing.
Tx: 3% NS.

B. Increased Volume of ECF (Hypervolemic): Due to fluid retaining states such as CHF, nephrotic syndrome, hepatic failure (cirrhosis), malnutrition, acute/ chronic renal failure, pregnancy.
Tx: water restrict, consider diuresis.

C. Normal ECF Volume (Isovolumic): Iatrogenic free water overloading, **SIADH** (unable to dilute urine in response to a water load), renal insufficiency, dec K (sensitizes kidney to ADH), thiazides, hypothyroid, adrenal insufficiency.
Tx: restrict water, consider hypertonic saline if sx's.

Other: **Oxytocin:** Used to induce labor, has an ADH effect, mimics SIADH. Avoid giving Oxytocin with hypotonic fluids.
Tx: D/c Oxytocin.

Cyclophosphamide: has antidiuretic effect and pt's encouraged to drink fluid to avoid chemical cystitis.
Tx: 3% NS and Furosemide.

Psychotic Self Induce Water Intoxication: Polydipsia (>1L/hr).
Tx: monitor diurnal wt for early detection. Avoid antidiuretic meds, water restrict. R/o SIADH.

Marathon Runners: A preventable condition that can be managed by runners weighing themselves before and after training to determine overall hydration strategy. Has been associated with the use NSAIDs before race and female. Pain and stress + ECF depletion due to salt loss (minimal contributor as if lose 9L sweat, only lose 1/17 of total body sodium), sugar water consumed during race + volume expansion from lots of fluid (excess hydration with plain water) **-->** promotes ADH (vasopressin) release**-->** hyponatremia and risk of cerebral edema and sz. Whenever an athlete collapses, the first step is to place in Trendelenburg, then check a rectal temperature (if >39C, treat as heat illness), before tx ck serum Na as hypernatremia with hyperosomolaslity can occur despite adequate fluid intake (Arch Path 2005;129:2227-30). Hyponatremia occurs in a substantial fraction of marathon runners and may be severe, according (NEJM. 2005;352:1516-1518, 1550-1556)....488 runners tested at the 2002 Boston Marathon found that 13% had hyponatremia (Na ≤135 mmol/L), and 0.6% had critical hyponatremia (≤120 mmol/L).....was associated with substantial weight gain (1kg = 1L)….. Composition of fluids (even if drinking 100% water), female sex, and use of NSAIDs were **not** associated with hyponatremia….. Determinants of weight gain included intake of ≥3 L of fluid, taking fluids every mile, racing time >4 hours, female sex, and BMI < 20 kg/m2. 75% of runners reported water loading, and 50% reported use of NSAIDs. Mean race duration was 3.3 hours for men and 4.0 hours for women.
Tx: 3% NS if sz's. Isotonic saline and water restriction if milder sx's. If post race Na >130, then observe pt. If 126-130 give NS IV by estimating ECFV. If <126 give 100ml of 3%NS IV qh until Na >130 (takes 3-5h).
Maternal hyponatremia is "not uncommon" following delivery of full-term infants (BJOG 2009;116:552-561).....Hyponatremia was found in 26% of the 61 mothers who received more than 2,500 ml of fluid during labor, of which two-thirds was ingested orally.....A decrease in plasma sodium concentration during labor correlated with duration of labor and the total fluid volume the mother received. Hyponatremia correlated significantly with fluid volume received but not with oxytocin administration or epidural analgesia.....Hyponatremia correlated significantly with prolonged second stage of labor, use of instruments for delivery and emergency cesarean section for failure to progress....."Women should not be encouraged to drink excessively during labor. Oral fluids, when permitted, should be recorded, and intravenous administration of hypotonic fluids should be avoided," the investigators advise.

Syndrome of Inappropriate Antidiuretic Hormone (SIADH):

Criteria for Dx of SIADH:
Essential: Plasma osmolarity <275 mOsm/kg. Urinary osmolarity >100. No signs of volume depletion, no edema. Urinary sodium greater >40. Normal thyroid function, normal adrenal function. No diuretics.
Supplemental: Uric acid <4. BUN <10. Fractional excretion of sodium >1%. Failure to correct hyponatremia with 0.9% saline. Correction of hyponatremia with fluid restriction. Low AVP levels (Clinical practice: The syndrome of inappropriate antidiuresis. NEJM. 2007;356(20):2064–2072).
Other Info:
Typically see: 1. Hypotonic Hyponatremia (Na <125 mEq/L) and hypo-osmolarity (<270-280 mOsm/kg). Hypo-osmolarity = <270-280 mOsm/kg.
2. Urine osmolality > appropriate (>75-100 mOsm/kg = 75-100 mmol/kg, usually >400 mOsm/kg, but < maximally dilute) for the degree of systemic hypotonicity.
3. Urinary Na excretion is high, usually >30-40 mEq/d. May go to 154 if pt treated with NS.
4. Absence of volume contraction (no extracellular fluid depletion). Euvolemic appearing (really volume expanded as have low uric acid level).
5. Uric acid is **low** in SIADH (<2.5) (Hypouricemia in SIADH. NEJM 1979;301:528).
6. R/o other causes: normal renal, adrenal, thyroid, cardiac, hepatic function.
Etiology of SIADH: Under normal conditions, ADH secretion is suppressed when osmolality is low and rises with increasing osmolality, resulting in reduced free-water excretion and lowered osmolality. In SIADH, secretion persists despite low osmolality.
Common Causes of SIADH --> Amiodarone (Cordarone), Carbamazepine (Tegretol), cerebral d/o's (tumor, meningitis), Chest d/o's (pneumonia, empyema), Chlorpromazine (Thorazine), ectopic antidiuretic hormone secretion, SSRI's, Theophylline.
Cancer --> lung (small cell) >pancreas> duodenum> bladder> prostate> lymphoma/ leukemia> thymoma> mesothelioma> Ewing's.
Any pulmonary lesion--> TB, CF, any type of pneumonia, abscess, asthma, positive pressure mechanical ventilation, acute respiratory failure. **Endocrine-->** hypothyroid, glucocorticoid def (hypopituitarism).
Neurologic d/o--> psychosis, any CNS lesions (neoplasia, vascular, trauma), stroke, Guillain-Barré, infection (meningitis, abscess), hydrocephalus, MS.
Drugs--> chlorpropamide (Diabinese), tolbutamide, clofibrate, antineoplastic (vincristine, cyclophosphamide, vinblastine), antidiuretic hormones (oxytocin, DDAVP, Vasopressin), tricyclics, Haldol, nicotine, phenothiazines, carbamazepine (Tegretol), opiates, bromocriptine, NSAIDs, SSRI's. **Other-->** Idiopathic, surgical (post-op)/ emotional stress, pain, nausea, acute intermittent porphyria, delirium tremens, AIDS, decreased intake of solutes (beer potomania, tea-and-toast diet).
Infections--> viral pneumonia, bacterial pneumonia, pulmonary abscess, tuberculosis, aspergillosis, meningitis.
Postoperative--> vasopressin secreted in response to surgical stress, free water from hypotonic IV fluids.
Tx: 3% saline and Furosemide (Lasix makes the kidneys excrete ½NS as get dec Na in the urine). Avoid hypotonic fluids (D5W, 0.45% saline) and excessive volumes of isotonic (LR, NS). The osmostat often resets to the lower serum Na level. See **Fluids** |
Demeclocycline: 150-300mg PO BID-QID, indefinitely. It is a tetracycline derivative that increases free water excretion by partially antagonizing the action of ADH. It inhibits the formation and action of cyclic adenosine monophosphate in the renal collecting duct. The onset of action is 3-6 days after tx is started. The dose needs to be decreased to the lowest level that keeps the serum Na+ within the desired range, with unrestricted water intake; this dose is usually 300-900 mg daily. The drug should be given 1-2 hours after meals, Ca, aluminum and Mg-containing antacids should be avoided. Polyuria tends to make pt's noncompliant. Skin photosensitivity may occur and in children, tooth or bone abnormalities may result. Nephrotoxicity also limits the drug's use, especially in pt's with underlying liver dz, in whom the hepatic metabolism of demeclocycline may be impaired.
Other: Increasing in solute excretion. A loop diuretic, when combined with high sodium intake (2-3 g additional NaCl), is effective. A single diuretic dose (40mg furosemide) is usually sufficient. The dose should be doubled if the diuresis induced in the first 8 hours is < 60% of the total daily urine output. Administration of urea to increase the solute load increases urine flow by causing an osmotic diuresis. This permits a more liberal water intake without worsening the hyponatremia and without altering urinary concentration. The dose is usually 30-60 g of urea daily. The major limitations are GI distress and unpalatability.

Urea: Children with hyponatremia due to chronic SIADH can be safely and effectively managed with oral urea therapy (J Pediatr 2006;148:128-131)....started on a 30% to 50% oral urea solution, with dose titration to a maximum of 2 g/kg/day, until normal serum sodium levels were achieved...then fluid intake was liberalized.

Pearls on "Fluids": All fluids are restricted in SIADH. Virtually all liquids are hypotonic relative to our salt content, so they all "count" against the fluid restriction. Tell patients any fluid is included - sports drinks, gatorade, water, juice, milk, Ensure/Boost etc. The only exception is broth and bouillon, to which one can encourage them to add salt liberally. Encourage them to take in lots of soup / broth / bouillon to make up for their "fluid" intake. Gatorade has 110mg sodium in 8oz of fluid…that amounts to about 280mg of salt in 0.23 liters, or about 1.2 g/dL which is equivalent to about 0.13 NS. Any administration of hypotonic fluids will have a tendency to worsen the hyponatremia and since no fluid out there is even close to iso-osmotic, we fluid restrict, loosen their salt intake, and, very useful, is actually giving NaCl tablets to "balance" out whatever hypotonic fluid intake. An attempt should be made to reduce/remove the stimulus for ADH secretion, esp. careful med review for SSRIs, thiazides, etc. Volume depletion should not be present in patients with SIADH, as they are euvolemic. Salt-wasting may be the reason for the hyponatremia or hypocortisolism if they're showing signs of vol. depletion with hyponatremia. SIADH and DI are water issues (since volume is normal). You can have low, normal, or high volume status with either low, normal, or high volume. That is because *water content* determines your sodium concentration but *salt content* determines your volume status with virtually no interplay in general.

2. Isotonic (Isosmotic hyponatremia):

(250-280 mOsm/L) "**Pseudohyponatremia**", see in presence of inc Trig (serum Na decreases by 1mmol/L for each inc serum lipids of 4.6g/L), inc protein. Or isotonic infusions of glucose, mannitol, glycine, glycerol, sorbitol, ethanol, methanol.
Plasma triglycerides (g/L) x 0.002 = mEq/L decrease in plasma Na.
Plasma protein level minus 8 (g/dL) x 0.025 = mEq/L decrease in plasma Na.
Due to lab artifact as plasma water as smaller fraction than normal leading to an underestimation of true Na concentration. Suspect when the serum is milky appearing. Need to compare measured plasma osmolarity with calculated or measure plasma Na with a direct-reading Na electrode. This occurs when the solid phase of plasma, primarily lipids and proteins (usually 6-8%) is greatly increased, as in severe hypertriglyceridemia and paraproteinemic d/o's. This falsely low reading is a consequence of the flow photometry methods that measure the concentration of Na+ in whole plasma and not only in the liquid phase. It is estimated that a rise in plasma lipids by 4.6 g / L or in plasma proteins of 10 g / dL lowers serum Na concentration by 1 mEq / L. This is not a problem when undiluted serum is analyzed with an ion-specific electrode that measures the concentration of sodium in serum water.

3. Hypertonic (Hyperosmotic hyponatremia):

Posm >285. Due to a non-Na hyperosmotic infusion of a substance that has an intracellular water osmotic redistribution (glucose, mannitol, glycine, isopropyl, ethylene glycol, sorbitol). For each 100mg/dL > 100, serum Na decreases by 3 mEq/L.
Hyperglycemia--> anticipate 3 mEq/L inc Na for every 200mg/dL reduction in BS. IV Mannitol**-->** water shift from ICF to ECF as with hyperglycemia. Should be rapidly excreted if normal renal function. With hyperglycemia, for each 100 mg per dL rise in blood glucose, a 1.6 mEq per L fall in plasma sodium concentration will occur as water moves out of cells into the ECF. For example, as blood glucose rises from 200 to 1200 mg per dL in an untreated diabetic pt, the plasma sodium concentration would be expected to fall from 140 to 124 mEq per L (1.6 mEq/L x 10 = 16 mEq) without a change in total body water and electrolytes. Conversely, tx with insulin and lowering of the blood sugar from 1200 to 200 mg per dL in this diabetic pt would result in comparable osmotic water movement from the ECF into cells and a return of plasma sodium concentration to 140 mEq per L without any change in total body water or electrolytes.
IVIG--> maltose in the solution acts like mannitol.
Irrigant absorption--> seen in prostatectomy and intrauterine surgery. The solute (glycine, mannitol or sorbitol) is initially confined to ECF causes dec Na with little change in plasma osmolarity. Glycine is a neurotoxin and causes blindness, it is metabolized to ammonia and may cause encephalopathy.

Isovolemic hyponatremia: is characterized by a small gain in free water, but not enough to be clinically detected (~ 5 L of excess water is necessary to produce detectable peripheral edema in the average-size adult). In this situation, the major d/o's to consider are inappropriate (nonosmotic) release of ADH and acute water intoxication (psychogenic polydipsia). The urine sodium and urine osmolality will help distinguish between these two d/o's.
Urine Sodium Urine Osmolality: Inappropriate ADH >20 mEq/L >100 mOsm/kg H2O. Water intoxication <10 mEq/L <100 mOsm/kg H2O
The inappropriate (nonosmotic) release of ADH is characterized by an inappropriately concentrated urine (urine osmolality above 100 mOsm/kg H2O) in the face of a hypotonic plasma (plasma tonicity below 290 mOsm/kg H2O). This condition can be seen in certain groups of "stressed" pt's, such as pt's who have undergone recent surgery. It can also be produced by a variety of tumors and infections. This latter condition is known as the syndrome of inappropriate ADH (SIADH), and it can be accompanied by severe hyponatremia (plasma sodium below 120 mEq/L).

Hypervolemic hyponatremia: represents an excess of sodium and water, with the water gain exceeding the sodium gain. In this situation, the urine sodium can sometimes help identify the source of the problem.
Common Urine Sodium: Heart failure <20 mEq/L. Renal failure >20 mEq/L. Hepatic failure <20 mEq/L. The urine sodium can be misleading if the pt is also receiving diuretics (which are commonly used in these conditions). The clinical picture is usually helpful, although these conditions can co-exist in critically ill pt's.
Management: Link: **W/u:** | Determined by the state of the ECV (low, normal, or high) and by the presence or absence of neurologic sx's. Symptomatic hyponatremia requires more aggressive corrective therapy than asymptomatic hyponatremia. However, to limit the risk of a demyelinating encephalopathy, the rate of rise in plasma sodium should not exceed 0.5 mEq/L/hour and the final plasma sodium concentration should not exceed 130 mEq/L (17). The general management strategies based on the ECV are as follows:
Low ECV: Infuse hypertonic saline (3% NaCl) in symptomatic pt's, and isotonic saline in asymptomatic pt's.
Normal ECV: Combine furosemide diuresis with infusion of hypertonic saline in symptomatic pt's, or isotonic saline in asymptomatic pt's.
High ECV: Use furosemide-induced diuresis in asymptomatic pt's. In symptomatic pt's, combine furosemide diuresis with judicious use of hypertonic saline. Persistent hyponatremia independently predicted mortality as well as rehospitalization in pt's hospitalized with CHF (Arch Intern Med 2007;167:1998)....After 6 months of follow-up, baseline hyponatremia was associated with a higher risk

for death (hazard ratio, 1.23 per each 3 mEq/L decrement in serum sodium)....editorialist notes that unfortunately, however, therapies specifically targeting hyponatremia in HF (e.g., tolvaptan) have been disappointing, suggesting that, until an effective treatment is developed, hyponatremia only identifies pt's who require relatively aggressive use of evidence-based HF therapies and closer follow-up.
****Ref:**(Hyponatremia. NEJM 2000;342:21) (Ann IM 1990;113:417-19) (J IM 1994;235:497) (Hyponatremia: age-related risk factors and therapy decisions. Geriatrics. 1998;53:32-8) (Beer potomania: two cases and review of the literature. Clin Nephrol. 1996;45:61-4) (Severe hyponatremia in the polydipsia-hyponatremia syndrome. J Clin Psychiatry. 1994;55:355-61) (Acute hyponatremia in ultra-endurance athletes. Am J Emerg Med. 1994;12:441-4) (Management of symptomatic hyponatremia: dependence on the duration of development. J Intern Med. 1994;235:497-501) (Hyponatremia: epidemiology, pathophysiology, and therapy. Curr Opin Nephrol Hypertens. 1993;2:636-52) (Hyponatremia in hospitalized patients epidemiology, etiology and symptomatology. J Am Soc Nephrol. 2004;15:561(A) (Hyponatremia treatment guidelines 2007: expert panel recommendations. Am J Med. 2007;120(11 Suppl 1):S1-21)

Hypernatremia:

Links: Etiology | **S/s** | Labs | **W/u & Tx** |

Na >145-150 mEq/L (mmol/L). Free water deficit **or** water loss (>Na loss) **or** a Na gain with free water restriction. Always associated with a hyperosmolar state (hypertonic hyperosmolality) leading to at least a transient state of cellular dehydration. The most frequent cause of hypernatremia is a decrease in total body water due to decreased intake or excessive loss. An osmolarity increase of 2% sets off thirst to prevent hypernatremia. Morbidity and mortality are highest in infants and the elderly, who may be unable to respond to increased thirst.
Etiology:
1. Net Water Loss: neurogenic/ nephrogenic diabetes insipidus, renal diuresis (osmotic, Loop diuretics), postobstructive diuresis, polyuric phase of ATN, intrinsic renal dz, GI or cutaneous losses.
Poor/ decreased fluid intake--> mental or physical disability (coma), limited access to fluids (infant not given water), inadequate thirst response (hypothalamic dysfunction).
Free Water loss--> osmotic diuresis (DM, DI), diuretic use, febrile illness (unreplaced insensible losses).
Water Shift out of Extracellular Fluid Compartment--> sz/ extreme exercise (water shifts into muscle cells), GI bleed with intraluminal protein catabolisms (water shifts into lumen).
2. Hypertonic Sodium Gain: Salt poisoning--> inappropriate administration of hypertonic salt solution (hemodialysis pt). Hypertonic NaHCO3 infusion, hypertonic feeding preparations, ingestion of salt or seawater, hypertonic saline enemas, primary hyperaldosteronism, Cushing's syndrome.
S/s: restlessness, sz, coma, delirium, mania, sticky mucous membranes, dec salivation/lacrimation, inc temp, red/swollen tongue, thirst, weakness. In infants it causes hyperpnea, weakness, high-pitched cry, insomnia, lethargy and coma. The sx's of hypernatremia usually begin when the osmolarity is >350 mOsm/L. Irritability and ataxia occur at osmolarities >375 mOsm/L. Lethargy, coma, and seizures occur with osmolarities >400 mOsm/L. In elderly there may be few sx's until Na >160. Brain shrinkage may cause vascular rupture leading to a cerebral bleed or a SAH.
Lab: Elev Na (>145 mEq/L), inc serum Osm, inc Ur Osm (if extrarenal losses), inc Ur Na (if renal losses), dec GFR. Check ADH (vasopressin) level on admission labs (low in central DI, high in nephrogenic DI).
Step #1: assess volume status. This helps to determine underlying cause.
A. Hypovolemia--> usually from Na (and hence H2O) losses with H2O losses predominating.
Urine Na >20 meq/L reflects renal losses from diuretics, glycosuria, mannitol, renal failure, etc. Urine volume also tends to be high with high osmolality.
Urine Na <10 meq/L reflects extrarenal losses (sweat, GI, insensible). Urine volume is low with high osmolality.
B. Isovolemia--> reflects loss of free water. R/o diabetes insipidus (DI), consider extrarenal losses-skin & respiratory insensible losses.
C. Hypervolemia--> usually from net Na gain, can be iatrogenic-IVF with hypertonic saline or NaHCO3, NaCl tablets, hypertonic IVF (e.g. cryoprecipitate with NA citrate anticoagulant), dialysis with hypertonic solutions or from mineralocorticoid excess-primary hyperaldosteronism, Cushing's disease, congenital adrenal hyperplasia
Tx: Links: Step #1: FWD | **Step #2: Volume Status** | Hypovolemic | **Isovolumic** | Hypervolemic |

Basics: correct free water deficit slowly with ½ the calculated amount over 8hr, then reassess and give remaining ½ over 16-24hr. If too rapid correction, cerebral edema may result. Hypotonic fluids such as D5W, dextrose are metabolized in the liver to leave electrolyte free water. May need to stop GI losses, control pyrexia, hyperglycemia and treat the possible associated inc Ca and dec K. Isotonic fluids if there is significant depletion of extracellular fluid as to cause hemodynamic compromise.
Step #1: Ck serum Na, Cr, serum osmolarity, urine lytes (Na and K), urine Osm, urine SG.
Free (total body) Water Deficit (FWD) in Liters:
FWD in Hypernatremia: FWD = [Serum Na (measured) - serum Na (normal) x 0.6 Wt in kg/ normal serum Na].
Also written as, FWD = [(Na-140)/140] baseline wt X F. (**F** = fraction of wt that is water, 0.6 for male, 0.5 for females).
Also written as, FWD = FWD = 0.6 X wt in kg X (1- 140/Na). Kg is estimated wt when fully hydrated. Generally, about half of deficit can be replaced in the first 24 hours; rest over 1-2 days.
Pearls: As a rule, each liter of water deficit causes the Na+ to increase 3-5 mEq/L. Treat any perfusion deficits with NS or LR, then switch to 0.5 NS after a urine output of 0.5 mL/kg/h is reached. Avoid lowering the Na+ >10 mEq/L/day. Avoid correcting the serum Na concentration >1mEq/L/hr. If no urine output is observed after NS/LR rehydration, rapidly switch to 0.5 NS; unload the body of the extra sodium by using a diuretic (Furosemide @ 20-40 mg IV). In children with a serum Na+ >180 mEq/L, peritoneal dialysis using high glucose low Na+ dialysate should be considered in consultation with a pediatric nephrologist.
Step #2: …….END DEMO of this chapter.

Infectious Disease:

Edited by Stephen Hosea, MD, ID Specialist.

Contents: **Emerging & Occupational** | Fever | **Sepsis** | Acute Phase Reactants | **Bacteria** | Antibiotics | **Osteomyelitis** | CNS | **TB** | Leprosy | **Parasites & Insects** | Zoonotic | **TRAVEL & Wilderness** | *Viral* | **HIV-AIDS** | Hemorrhagic Fevers | **Skin** | Botulism | **Wounds** | STD's | **Lymphadenopathy** | Fungal | **Bio-Chem-Nuke Weapons** | PEP / Needlestick | **Pediatric** | ENT | **Pneumonia** | Urologic | **Vaccines & Related Dz's** | Immune Deficiency | **Vaginal** | Hepatitis | **Peritonitis-Abd Abscess** | Peri-partum | **Septic Joint** | Endocarditis | **H. pylori** | Diarrhea | **ENT** | Urology | **Infection Control** | *INDEX* |

See pulmonary chapter for pneumonia and bronchitis. See ENT for sinusitis, URI and Otitis. See Urology for prostatitis and UTI. See women's health for vaginitis. Infectious disease hospitalizations in the United States increased by more than 6% between 1998 and 2006 (Clin Infect Dis 2009;49:1025-1035).....However, hospital deaths due to infections have been declining.....The largest proportion of hospitalizations for infectious diseases occurred in the winter months, and lower respiratory tract infections were the most common diagnoses.

Emerging Infections:

Links: Occupational Infections | **Disease / Problem Healthcare Worker Restriction – Duration** | Pandemic | See Compedium module for Epidemiology | **Infection Control** | ID EMERGENCIES |

An emerging infection is defined as infections that have newly appeared in a population or have existed previously but are rapidly increasing in incidence or geographic range. Infectious diseases are emerging more quickly and spreading faster around the globe than ever and becoming increasingly difficult to treat according to the World Health Organisation (Annual WHO World Health Report;2007;Aug 23)....with people moving around the planet, an outbreak or epidemic in one part of the world is only a few hours away from becoming an imminent threat somewhere else....new threats have been identified at an "unprecedented rate" of one or more every year, meaning that nearly 40 diseases exist today which were unknown just over a generation ago. Since the mid 80's in the US this includes severe acute respiratory syndrome (SARS), Lyme disease and HIV. Re-emerging infections include West Nile virus, dengue fever and drug resistent TB. Agents of bioterrorism are also a category of emerging infectious diseases, in 2001, the deliberate release of anthrax by terrorists resulted in 22 infections and 5 deaths. Even though malaria was officially eradicated in this country in the early 1950s, 1337 cases were reported in 2002, including 8 deaths….of these cases, all but 5 were acquired in malaria-endemic countries. The 2 species of mosquitoes capable of transmitting malaria are still widely prevalent, making the disease a lurking risk. The world is becoming a "global village", 53 million Americans journeyed outside the United States in 2003 and while most international travel occurs among developed countries, an estimated 107 million people from the United States and Canada traveled to developing countries in 2000. According to 2004 US census bureau data, 58.2% of the people living in Miami, Fla, and 35.5% of the people living in New York City were born outside the US. See **Viral** | Despite extensive investigations, a microbiological cause cannot be found in about 50% of the pt's with infectious disease (Curr Opin Inf Dis 2005;18:413–419). New infectious diseases emerged at an increasing rate over the past six decades according to a study that charted global outbreaks since the 1940s (Nature 2008;451:990-93)....human population density is a key player in their emergence.....arising in "hot spots" such as Asia, Latin America and tropical Africa....The emergence of new diseases reached a peak in the 1980s, probably as a result of the HIV pandemic, which left millions of people more susceptible to novel infections, the researchers said.....More than half (54.3%) of the pathogens involved were bacterial or rickettsial, while viral or prion pathogens caused only 25.4% of the recorded events. During the study period, 60.3% of emerging infectious diseases were caused by zoonotic pathogens and 71.8% of these zoonotic events arose from wildlife. Vector-borne diseases account for 22.8% of all emerging disease over the study period, but 28.8% in the 1990s, possibly because changing climate patterns have altered some organisms' historic ranges. Diseases caused by drug-resistant microbes (20.9% of the diseases in the database) have significantly increased with time, possibly because of an increase in the use of antimicrobial drugs. **Infection Control** |

Factors that contribute to the emergence of certain infections:

1. Man-made or natural changes in the environment (eg, accounting for Lyme disease and HPS).
2. Demographic shifts in populations (eg, accounting for the spread of HIV).
3. Increased international travel and commerce (eg, the rapid global spread of severe acute respiratory syndrome = SARS)
4. Technologic and industrial changes (eg, accounting for the rise of hemolytic uremic syndrome in the Northwest US in 1993).
5. Adaptation of microbes (eg, accounting for antibiotic-resistant organisms).
6. Lapses in the public health system (eg, accounting for infections due to water-borne Cryptosporidium).

Internet Resources:

American Society of Tropical Medicine and Hygiene: http://www.astmh.org
Promotes world health by prevention and control of tropical diseases through research and education of health professionals and the public.

CDC: http://www.cdc.gov
Provides information regarding emerging infectious diseases, epidemiology, outbreak status, links to state health departments, vaccines and immunizations, infection control guidelines, and the journal Emerging Infectious Diseases.

FDA: http://www.fda.gov/oca/sthealth.htm
Provides links to state health departments in 50 states.

International Society for Infectious Diseases: http://www.isid.org
Provides Pro-Med, www.promedmail.org, the global electronic reporting system for outbreaks of emerging infectious diseases and toxins and publishes the International Journal of Infectious Diseases.

National Center for Infectious Diseases: Traveler's Health: http://www.cdc.gov/travel/diseases.htm
Provides travel fact sheets, vaccination information, listings of travel medicine clinics, and Department of State health updates.

National Notifiable Diseases Surveillance System:

http://www.cdc.gov/epo/dphsi/nndsshis.htm

Reporting of nationally notifiable diseases to CDC by the states is voluntary. Reporting is currently mandated (i.e., by state legislation or regulation) only at the state level. The list of diseases that are considered notifiable, therefore, varies slightly by state. All states generally report the internationally quarantinable diseases (i.e., cholera, plague, and yellow fever) in compliance with the World Health Organization's International Health Regulations.

Notifiable diseases: AIDS. Anthrax. Arboviral neuroinvasive and non-neuroinvasive diseases. California serogroup virus disease. Eastern equine encephalitis virus disease. Powassan virus disease. St. Louis encephalitis virus disease. West Nile virus disease. Western equine encephalitis virus disease. Botulism. Brucellosis. Chancroid. Chlamydia trachomatis, genital infections. Cholera. Coccidioidomycosis. Cryptosporidiosis.
Cyclosporiasis. Diphtheria. Ehrlichiosis. Giardiasis. Gonorrhea. Haemophilus influenzae, invasive disease. Hansen disease (leprosy). Hantavirus pulmonary syndrome . Hemolytic uremic syndrome, post-diarrheal. Hepatitis, viral (A,B and C), acute or chronic. HIV infection. Influenza-associated pediatric mortality. Legionellosis. Listeriosis. Lyme disease. Malaria. Measles. Meningococcal disease. Mumps. Pertussis. Plague. Poliomyelitis. Psittacosis. Q Fever. Rabies. Rocky Mountain spotted fever. Rubella. Salmonellosis. Severe Acute Respiratory Syndrome-associated Coronavirus (SARS-CoV) disease. Shiga toxin-producing Escherichia coli (STEC). Shigellosis. Smallpox. Streptococcal disease, invasive, Group A. Streptococcal toxic-shock syndrome . Streptococcus pneumoniae, drug resistant, invasive disease. Syphilis. Tetanus. Toxic-shock syndrome (other than Streptococcal). Trichinellosis (Trichinosis). Tuberculosis. Tularemia. Typhoid fever. Vancomycin - intermediate Staphylococcus aureus (VISA). Vancomycin - resistant Staphylococcus aureus (VRSA). Varicella (morbidity). Vibriosis. Yellow fever.

Occupational Infections:

Links: Needlesticks / Occupational Exposures | Infection Control | Respiratory Protection | Universal Precuations |

Airborne Transmission attack rates: TB (20-50% attack rate, nurses, pathologist, lab worker, housekeeper), Varicella (5-14% attack rate, all, varicella >> zoster), Measles (physicians, nurses), Influenza (4-45% attack rate, nurses & physicians), Rubella (13%, all), Mumps (pediatricians, dentists), Pertussis (43%, all), Parvovirus B19 (30-45%, nurses, teachers), RSV (50%, all), Adenovirus (25-30%, all HCW's), influenza (45%).
How Contagious (Risk of infection)?: (NEJM 2003;348:1256-66)
TB: at nursing home, 1 in 20, home 1 in 3, dormitory 1 in 5, social club 1 in 10, school/work varies, casual contact 1 in 100,000.
N meningitidis: home 1 in 50 to 1 in 400, social club or dorm, increase slightly, school/work 1 in 200 to 1 in 3,000, casual contact 1 in 100,000.
Influenza: high in all situations, moderate if casual social contact.

Occupations and Associated Infectious Diseases:

Health care worker: C difficile, hepatitis A, B, C, and delta, HSV, HIV, influenza, lice, measles, Mycobacterium tuberculosis, Neisseria meningitidis, parvovirus, RSV, Salmonella, scabies, VZV
Research lab worker: Campylobacteriosis, filoviruses, hepatitis A, herpes simiae, influenza, leptospirosis, lymphocytic choriomeningitis, ornithosis, Q fever, rat-bite fever, ringworm, Salmonella, toxoplasmosis, West Nile virus
Microbiology lab worker: Brucellosis, campylobacteriosis, coccidioidomycosis, Salmonella, tularemia
Butchers / meat handlers: Anthrax, brucellosis, cryptosporidiosis, erysipeloid, infectious diarrhea, leptospirosis, Mycobacterium bovis, Newcastle disease, orf, ornithosis, Q fever, Streptococcus suis meningitis, toxoplasmosis, tularemia, vesicular stomatitis.
Animal handlers: Bite wounds, rabies
Cattle: Babesiosis, bovine papular stomatitis, brucellosis, cryptosporidiosis, erysipeloid, Mycobacterium bovis, pseudocowpox, Q fever, ringworm, vesicular stomatitis
Fish/crustaceans: Erysipeloid, leptospirosis, Mycobacterium marinum, nanophyetiasis
Pet shop employees: Bite wounds, Mycobacterium marinum, Newcastle disease, ornithosis, ringworm, toxoplasmosis, rat bite fever.
Poultry: Campylobacteriosis, erysipeloid, histoplasmosis, infectious diarrhea, Newcastle disease, ornithosis, ringworm.
Sheep/goats: Brucellosis, cryptosporidiosis, echinococcosis, erysipeloid, louping ill, orf, ornithosis, Q fever, tularemia.
Swine: Erysipeloid, influenza, ringworm, Streptococcus suis meningitis.
Veterinarians: Anthrax, bite wounds, brucellosis, cryptosporidiosis, echinococcosis, herpes simiae, leptospirosis, Mycobacterium bovis, Newcastle disease, orf, ornithosis, Q fever, rabies, ringworm, toxoplasmosis, tularemia, vesicular stomatitis
Zoo personnel: Bite wounds, herpes simiae, Mycobacterium marinum, ringworm, sealpox.
Archeologists: Coccidioidomycosis.
Construction workers: Coccidioidomycosis, histoplasmosis, tetanus.
Daycare providers: CMV, hepatitis A, infectious diarrhea, upper respiratory tract infections.
Farmers: Brucellosis, campylobacteriosis, coccidioidomycosis, cryptosporidiosis, histoplasmosis, hypersensitivity pneumonitis, orf, Q fever, ringworm, sporotrichosis, tetanus, toxoplasmosis, tularemia, vesicular stomatitis
Foundry workers: Acinetobacter pneumonia.
Hide processors: Anthrax, tularemia.
Military personnel: Adenovirus, coccidioidomycosis, hepatitis A, leptospirosis, Neisseria meningitidis, ringworm, tetanus, upper respiratory tract infections
Miners: Leptospirosis, ringworm.
Office workers: Influenza, legionellosis.
Outdoor/rural occupation: Babesiosis, leptospirosis, Lyme disease, Q fever.
Plant and plant product exposure: Hypersensitivity pneumonitis, sporotrichosis.
Sewer workers: Hepatitis A, leptospirosis, tetanus.
Sandblasters, miners, quarry workers, ceramic workers: Silico-tuberculosis.
Management of Job-Related Illnesses and Exposures:

Disease/Problem Healthcare Worker Restriction - Duration:

Conjunctivitis - Restrict from patient contact and contact with the patient's environment until discharge ceases.
Cytomegalovirus infections - No restriction.
Diarrheal diseases - Acute stage (diarrhea with other symptoms): Restrict from patient contact, contact with the patient's environment, or food handling until symptoms resolve. Convalescent stage, Salmonella spp. Restrict from care of high-risk patients until symptoms resolve; consult with local and state health authorities regarding need for negative stool cultures.
Hepatitis A - Restrict from patient contact, contact with patient's environment, and food handling until 7 days after onset of jaundice.

Herpes simplex Hands (herpetic whitlow) - Restrict from patient contact and contact with the patient's environment. Until lesions heal
Measles - Active: Exclude from duty until 7 days after the rash appears. Postexposure (susceptible personnel): Exclude from duty From the 5th day after the 1st exposure through the 21st day after last exposure.
Meningococcal infections: Exclude from duty until 24 hours after start of effective therapy.
Mumps - Active: Exclude from duty until 9 days after onset of parotitis. Postexposure (susceptible personnel): Exclude from duty from 12th day after 1st exposure through until 25 days after last exposure or until 9 days after onset of parotitis.
Pertussis - Active: Exclude from duty from beginning of catarrhal stage through 3rd week after onset of paroxysms or until 5 days after start of effective antimicrobial therapy. Postexposure (asymptomatic personnel): No restriction, prophylaxis recommended. Postexposure (symptomatic personnel): Until 5 days after start of effective antimicrobial therapy.
Rubella - Active: Exclude from duty until 5 days after rash appears. Postexposure (susceptible personnel): Exclude from duty from 7th day after 1st exposure through 21st day after last exposure.
Staphylococcus aureus infection - Restrict from contact with patients and patient's environment or food handling until any active, draining skin lesions have resolved.
Tuberculosis - Active disease: Exclude from duty until proved noninfectious. TST (PPD) converter - No restriction.
Varicella Active - Exclude from duty until all lesions dry and crust. Postexposure (susceptible personnel): Exclude from duty from 10th day after 1st exposure through 21st day (28th day if VZIG given) after last exposure. Viral respiratory infections, acute febrile: Consider excluding from the care of high risk patients or contact with their environment during community outbreak of RSV and influenza until acute symptoms resolve.
Zoster - Localized, in healthy person: Cover lesions; restrict from care of high-risk patients until all lesions dry and crust. Generalized or localized in immunosuppressed person: Restrict from patient contact until all lesions dry and crust. Postexposure (susceptible personnel) - Restrict from patient contact from 10th day after 1st exposure through 21st day (28th day if VariZIG given) after last exposure or, if varicella occurs, until all lesions dry and crust. (Bennett & Brachman's Hospital Infections, 5th Ed. 2007. Lippincott Williams & Wilkins)

Fever:

Links: Definition, ICD9 & Assessment Techniques | **Mechanisms** | Benefits | **ID EMERGENCIES** | Hyperthermia | **Types of Fevers** | *FUO* | **Drug Fevers** | Fever and Rash | **Acute Rheumatic Fever** | Returned Traveler | **Sepsis** | Acute Phase Reactants (ESR, CRP) | **Nosocomial** | ICU Fever | **Cancer/ HIV** | Neutropenic fever | **Pediatric** | Relative Brady / pulse-temp dissoc | **Infection Control** | Hereditary / Periodic Fever's | **Blood Cx** |

Humanity has but three great enemies: Fever, famine and war. Of these, by far the greatest, by far the most terrible, is fever. -Sir William Osler.
Fever = Elevation of core body temperature **>38C (100.4F).** A sign of inflammation, not just infection, nor the severity of it.
Rectal (core) >100.4° F (38.0° C). The normal rectal temp is ~0.6C (1F) higher than oral.
Oral >99.5° F (37.5° C).
Axillary >98.6° F (37.0° C). Axillary temp measurements are unreliable but are often used in acutely ill pt's.
Tympanic >100.0° F (37.8° C). TM temps are typically 0.4° C (0.7° F) below sublingual readings and have even greater variability.
Pyrexia: an abnormal elevation of body temperature via either a fever or hyperthermia. Fever enhances the inflammatory response, inhibits bacterial growth and creates an undesirable physiologic environment for infection. Heat is a byproduct of all metabolic processes. 70% of the bodies heat is dissipated via conduction, 30% via evaporation. Thermal control is from the preoptic nucleus of the anterior hypothalamus.
C= 0.5 (F-32).
F=(1.8C)+32.
*** Pearls *:** Always have a high level of suspicion of the pt looks sick, age >55 or other subtle changes in vital signs (hypotension, tachycardia, tachypnea). Hypothermia can also be a significant sign of infection. The magnitude of fever reduction from antipyretics does not distinguish those with serious infections from those with simple, uncomplicated illnesses.
ICD-9: 780.60 Fever, unspecified
780.61 Fever presenting with conditions classified elsewhere
780.62 Postprocedural fever
780.63 Postvaccination fever
780.64 Chills (without fever)
780.65 Hypothermia not associated with low environmental temperature
Rectal Temperature Assessment:
Step #1: Draw curtains around the patient's bed and/or close the door. Keep the patient's upper body and lower extremities covered. Assist patient into Sims' position, with the upper leg flexed to expose anal area. Children may be placed in a prone position.
Step #2: Squeeze a liberal amount of water-soluble lubricant onto a tissue. Apply lubricant to the thermometer. With the nondominant hand, raise the patient's upper buttock to expose the anus. Full retraction of the buttocks completely exposes anus.
Step #3: Gently insert the thermometer into the anus in the direction of the umbilicus.Insert the thermometer 11/2 in. (3.5 cm) for adults and 1/2 in. (1.2 cm) for infants or children.Do not force the thermometer. Ask the patient to take a deep breath and blow out. Insert the thermometer as the patient takes in a breath. If resistance is felt, immediately remove the thermometer. Hold the thermometer in place for a minimum of 2 minutes. It may be necessary to hold patient's legs. Holding the thermometer in place prevents injury to the patient.
Axillary Temperature:
Step #1: Assist the patient into a supine or sitting position. Move clothing away from the patient's shoulder and arm. Be sure the axillary area is dry.
Step #2: Place the thermometer into the center of the patient's axilla, lower the arm over the thermometer, and place the patient's forearm across the chest. Hold the thermometer in place for; 5 to 10 minutes. Remain with the patient and hold the thermometer in

place if the patient is unable to do so. Wash the thermometer in tepid, soapy water, rinse in cold water, and dry. Shake down the thermometer and return it to its container.

Electronic Thermometers:

Oral Temperature:

Step #1: Attach the oral probe to the electronic display unit. Grasp the top of the stem, using caution not to apply pressure to the eject button.

Ejection button releases the plastic cover from the probe. Slide a clean disposable plastic cover over the temperature probe until it locks in place.

Step #2: Ask the patient to open mouth and gently place the probe under the tongue, on either side of the frenulum. Have patient keep the probe in place until the alarm on the electronic thermometer sounds and the temperature reading appears on the digital display.

Step #3: Electronic units register the body temperature within seconds. Remove the probe from under the patient's tongue, then push the eject button on the thermometer probe to discard the plastic probe cover into the proper receptacle.

Rectal Temperature:

Attach the rectal probe (usually the red tip). With the nondominant hand, raise the patient's upper buttock to expose the anus. Apply lubricant to the probe end of the thermometer. Gently insert the lubricated thermometer as previously described.

Axillary Temperature: Attach the rectal probe (red tip) and cover slip. Insert the probe into the center of the axilla, lower the patient's arm over the thermometer, and place the arm across the patient's chest.

Electronic Tympanic Temperature:

Step #1: Attach a disposable probe cover to the thermometer unit. Different size sheaths fit adults and children. Select the ear opposite the side on which the patient has been lying aslying against a surface can build up heat.

Step #2: use the right hand to hold the thermometer when using the patient's right ear, the left hand for the left ear to achieve the proper angle for a good seal. Gently pull the pinna upward and back for children older than age 3 and adults; straight back for children younger than age 3 to straighten the ear canal.

Step #3: Center the probe and gently advance into the ear canal to make a firm seal, directing the probe toward the tympanic membrane. Press and hold the temperature switch until the green light flashes and the temperature reading is displayed. Remove the thermometer and discard the probe cover.

Mechanism of Fever: Body temperature is regulated by the hypothalamus, and prostaglandin E2 acts on this region to stimulate fever. Fever is the result of the body's thermostat being reset by exogenous pyrogens, such as bacteria, bacterial endotoxins, antigen-antibody complexes, and viruses, which stimulate the production of endogenous pyrogens. Endogenous pyogenic cytokines **-->** IL-1, TNF-alpha, INF-gamma, IL-6. These pyrogens bind to receptors in the preoptic nucleus of anterior hypothalamus (near the floor of the third ventricle) to inc prostaglandins which **-->** (thermal set point. The pyrogens are activated by endotoxins, microorganisms, tissue injury, immune complexes and phagocytosis.

Normal diurnal variation in temp (up to 1.3C or 2.4F). The nadir is @ 4-8am ~97.0F (36.1C). Peak is at 4-6pm. 99.9F (37.7C) in PM. The hypothalamus regulates body temperature with a typical diurnal variation. The "normal" range of temperature varies among individuals.

Benefits of Fever: in the pre-antibiotic era temperatures up to 107F (41.6C) seemed tolerated w/o evidence of tissue damage. Fever decrease the serum iron levels that bacteria need to grow. Some bacteria are heat sensitive. Fever shifts metabolism to increased fat and protein utilization as bacteria need glucose. Attempts to lower elevated body temperature is advisable only when it is above 40.5C (105F) unless the pt is having hypoxemia, heat shock, malignant hyperthermia, acute neuro dz or other related problems. Antipyretic therapy prolongs the illness in pt's with influenza (Pharmacotherapy 2000;20:1417)

ID Emergencies:

Links: Pandemic | **Bio-Chem-Nuke Weapons** | Many infections such as pneumonia and bacterial meningitis need immediate Abx at time of dx, yet some infections are particularly life-threatening.

1. Superantigen toxin-mediated syndromes: includes toxic shock syndrome, streptococcal toxic shock-like syndrome and recurrent toxin-mediated perineal erythema. Kawasaki disease (KD) may be a superantigen toxin-mediated disease, as well.

2. Meningococcemia.

3. Post-splenectomy sepsis.

4. Acute bacterial endocarditis.

5. Pus under pressure: ascending cholangitis, pyelonephritis with hydronephrosis, abscesses.

6. Flesh-eating bacteria & Polymicrobic wound infections: Meleney's synergistic gangrene, Fournier's gangrene, necrotizing fasciitis have a common pathophysiology and all require prompt surgical debridement as well as Abx's. Cellulitis after a cut in an aquatic environment can be devastating.

Infections and Psychiatric Diseases:

HIV and Dementia: Due to the human immunodeficiency virus. Termed HIV-associated cognitive/motor complex (CMC) or HIV-associated dementia. HIV-1 infection has been associated with mood disorders such as mania and depression.

Treponema and Neurosyphilis: The neuropsychiatric symptoms of tertiary syphilis include impaired cognitive performance, memory loss, confabulation, pseudobulbar palsy, mania, depression, and psychosis. If left untreated, it typically progresses toward "general paresis" (chronic progressive dementia); "tabes dorsalis" (peripheral neuropathy, ataxia, autonomic dysfunction); mutism; spasticity; seizures; and eventually death within 3 to 5 years.

Streptococcal Infection: Rheumatic fever can present with Sydenham's chorea, which can have psychiatric symptoms, such as emotional lability, hyperactivity, impulsivity, anxiety, tics, Tourette's-like signs, obsessions, and compulsive behavior. Sudden onset or exacerbation of OCD or tics has been reported following recent group A beta-hemolytic streptococcal infections in children who did not have rheumatic fever or Sydenham's chorea and is associated with PANDAS. The OCD symptoms of PANDAS can precede the chorea by days or several weeks, and tend to wax and wane in parallel with the plasma level of antineuronal antibodies.

Borna disease virus (BDV): BVD is a nonsegmented, negative-stranded, enveloped RNA virus that was first identified during the 19th century as the cause of severe neurologic diseases in domestic animals. Psychiatric patients who had BDV have been found to have major depression, OCD disorder, panic disorder, or organic mood disorder (J Virol 1996;70:7713-7724). However, the precise relationship between the virus and specific mental disorders, if any, needs to be determined.

Chronic Fatigue Syndrome: Characterized by chronic debilitating fatigue and other nonspecific complaints of headache, myalgias, joint pain, recurrent pharyngitis, and cognitive deficits. The etiopathogenesis of CFS remains unknown. A theory of an infectious etiology is based on sudden onset preceded by an acute infection with flu-like symptoms. Suggested infectious agents have been suspected at different times to play an etiologic role in CFS, including EBV, CMV, HSV, retroviruses, spumaviruses, and Borrelia burgdorferi, the agent of Lyme disease. No specific immunologic dysfunction has been found in these patients.

Hyperthermia:

An unregulated rise in the body temperature (unlike fever with a set point increase) due to a failure in the thermal control mechanism. Pyogenic cytokines are not involved, and it does not respond to standard antipyretics. It is due to uncontrolled heat production, inadequate dissipation of abnormal hypothalamic thermoregulation.

Mechanism of Hyperthermia:

A. Increased Hypothalamic Set Point: infection, malignancy, CVA, trauma, sarcoid, neuroleptic malignant syndrome (NMS), idiopathic.
B. Heat Production Exceeds Loss: excessive environmental temp, hyperthyroid, malignant hyperthermia, salicylate OD, NMS, status epilepticus, pheo, lethal catatonia, generalized tetanus, delirium tremens, heat stroke.
C. Defective Heat Loss: anticholinergic drug poisoning, ectodermal dysplasia, heat stroke, dehydration, autonomic dysfunction.

Main types of fever:

Links: | FUO | **Drug Fevers** **|** Fever and Rash | **Acute Rheumatic Fever** | Hereditary Fever |

…….END DEMO of this chapter.

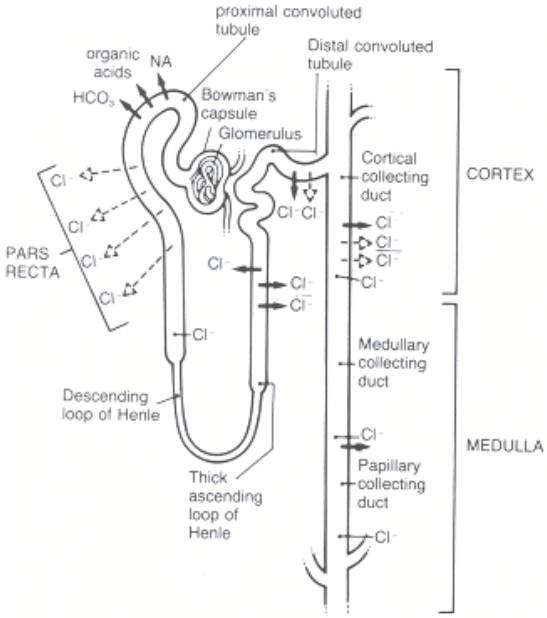
Nephrology (Renal):

Edited by Darol D. Joseff, MD, Nephrology & Hypertension.

Contents: Info & Pearls | Hematuria | **Proteinuria** | Acute Renal Failure | ***Intrinsic Renal Dz's** | Chronic Renal Kidney Dz / Failure | **Dialysis** | Rhabdo | **RTA** | Various Renal Disease & Masses | **Urinalysis** | Kidney Biopsy | **Acid-Base Physiology** | *Hypertension* | **HTN Meds** | Edema | **Drug Dosing in RF** | Cystic Disease | **Renal Drug Toxicity & ICD-9** | ICU-FEN | **Electrolytes** | Labs in Renal Dz's | **Kidney Transplant** | Tx of Complications of CRI | **Drug Dosing in RF** **|** Radiocontrast Nephropathy | **Urology** **|** *INDEX* |

The kidneys four main functions are:
1. Regulation of electrolyte and acid-base balance.
2. Disposition of metabolic byproducts and drugs.
3. Maintenance of volume status.
4. Organ-specific endocrine and metabolic effects.
1. **Function:** Maintain **blood homeostasis**: i.e., pressure, pH, ionic balance and conserve nutrients while eliminating wastes.
2. **Nephron:** Functional unit of kidney found in cortex/medula. **Two major portions:**
a. **Renal corpuscle:** Blood enters **Bowman's capsule** via **glomerulus** where a filtrate is formed.
b. **Renal tubules:** Glomerular filtrate enters proximal convoluted tubule **-->** loop of Henle **-->** distal convoluted tubule **-->** collecting duct where it is now urine.
3. **Tubular secretion and reabsorption:**
a. Glomerular filtrate entering **renal tubules** consists mostly of plasma minus proteins.
b. 180 L (47 gallons) of filtrate are produced per day.
c. Nearly all the plasma (and nutrients) must be reabsorbed by capillaries that follow the renal tubules.
d. The **loop of Henle** dips deep into the medulla of the kidney, which has an **interstitial fluid** laden with solutes to help the blood vessels reabsorb water.
e. **Electrolytes** may be actively and passively removed from the filtrate to help maintain ion balance of the blood and interstitial fluid of the kidney.
f. Collecting tubule's permeability to water can be increased by **antidiuretic hormone** (ADH), which allows for more water reabsorption and more concentrated urine.

Blood Filtration: The kidneys are responsible for the maintenance of total body chloride balance. They maintain homeostasis because each adult kidney is composed of ~1 million functional units, nephrons (glomeruli). Glomeruli are complex structures comprising a series of capillary loops that start with the afferent arteriole and end with the efferent arteriole. The three resident glomerular cell types- the mesangial cell, the glomerular endothelial cell, and the podocyte (visceral epithelial cell)-serve distinct functions. The normal glomerular filtration rate (GFR) is 180 L/day. ~30% of the plasma flow is filtered at the level of the glomerulus. The major barrier to filtration of plasma constituents is the glomerular capillary wall, which comprises the endothelial cells, the glomerular basement membrane (GBM), and the podocyte. The normal rate of protein excretion is <150 mg/ day, of which < 20 mg is albumin. The normal filtration barrier limits passage of molecules by size and charge selectivity. The negative charge of the glomerular endothelial cells, GBM, and podocytes limits the passage of anionic substances, such as albumin. (Current status of the structural and functional basis of glomerular filtration and proteinuria. Semin Nephrol 1991;11:390)



The Top 10 Things Nephrologists Wish Every Primary Care Physician Knew:

(Mayo Clin Proc 2009;84:180-6)

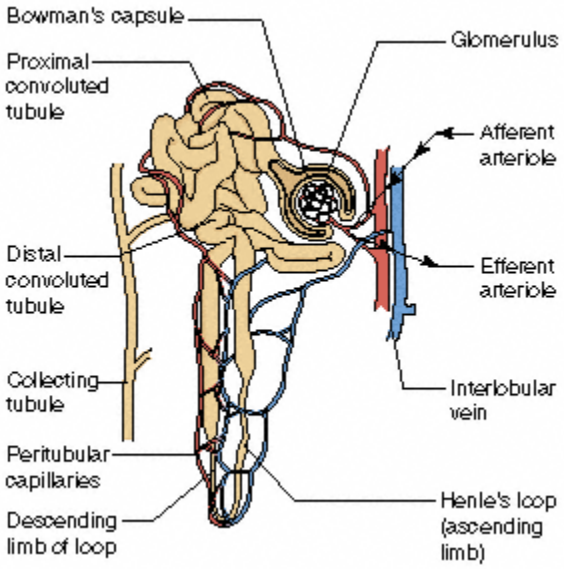
1. A "Normal" Serum Creatinine Level May Not Be Normal.

A. Normal SCr may vary among different laboratories, and some patients with SCr levels within the “normal” range may have substantial reduction in kidney function.
B. A 66-year-old man with a SCr of 1.4 mg/dL (to convert to µmol/L, multiply by 88.4) would have an estimated glomerular filtration rate (eGFR) of 54 mL/min per 1.73 m2.....thus this perceived “normal” serum creatinine value actually corresponds to stage 3 CKD. Likewise, the rate of increase or trend of serum creatinine levels can also indicate CKD. S 66-year-old man with a SCr of 0.8 mg/dL (eGFR = 103) would have an eGFR of <64 should the SCr level stabilize at 1.2 mg/dL.....This would represent at least a 40% reduction in GFR.
C. Estimates of GFR can only be based on steady-state SCr values and cannot be used to accurately estimate GFR when it is changing. A rapid increase in the SCr from 0.8 to 1.2 mg/dL within 8 hours could reflect a GFR approaching zero in a patient with acute renal failure.
D. The interpretation of SCr level also depends on muscle mass, age, sex, height, and limb amputation. A patient with an amputation and a high-normal SCr may actually have advanced CKD.
2. Know the Medications That Spuriously Elevate the Serum Creatinine Level. The SCr level can increase without reflecting a change in the actual GFR if taking the antibiotic trimethoprim-sulfamethoxazole and the H2-blocker cimetidine. This can result in a self-limited and reversible increase in the SCr of as much as 0.4 to 0.5 mg/dL (depending on baseline serum creatinine level). Famotidine and ranitidine can likewise cause an increase but to a lesser degree. The antibiotic cefoxitin can spuriously increase the Scr by interfering with the colorimetric assay used to measure SCr levels. In both instances, the blood urea nitrogen (BUN) typically does not change. As such, an increase in creatinine level suggests a true decrease in GFR only if accompanied by a corresponding increase in BUN levels.
3. Patients With Decreased GFR or Proteinuria Should Be Evaluated to Determine the Cause. Positive Urine Dipstick Test Results for Protein Should Be Followed Up With a Spot Urine Protein to Urine Creatinine Ratio. Chronic kidney disease is defined as a kidney abnormality that persists for more than 3 months. Persons with a normal GFR may have CKD if they have persistent proteinuria or hematuria of renal origin. Proteinuria is initially detected by the urine dipstick test, which reflects the concentration of albumin in the urine. Because variations in urine flow and concentration can affect the semiquantitative dipstick determination, a quantitative estimation of proteinuria is required. The preferred method for quantitative estimation of protein excretion is the spot urine protein to creatinine ratio because it is accurate and more convenient than a 24-hour urine collection. In patients at risk of proteinuria (eg, patients with diabetes), a spot urine albumin to creatinine ratio is helpful for detecting microalbuminuria, thereby guiding further therapy. In a patient with a urine protein to creatinine ratio of 1 or more, the risk of progression of CKD is higher. In fact, any patient with an elevated ratio (=1) should be evaluated for causes of glomerular disease, including diabetes, collagen vascular disease (eg, SLE), malignancy (eg, multiple myeloma), infections (eg, HIV, syphilis, hepatitis B and C), and use of medications such as NSAIDs. The key to the diagnostic assessment of patients with these conditions includes, most importantly, a careful history and physical examination, followed by renal Doppler ultrasonography to detect renal artery stenosis, obstruction, and/or cysts.
4. In Patients With Early-Stage CKD, Periodic Evaluation and Intervention Are Appropriate to Slow the Progression of Renal Disease and Avoid Its Complications. Nephrotoxic drugs, such as NSAIDs, aminoglycoside antibiotics, and radiocontrast agents, should be used with caution or avoided completely. Systemic BP should be monitored frequently and controlled with a goal BP <130/80 mm Hg. A lower BP may be desirable in patients with proteinuric CKD. A spot urine protein to creatinine ratio should be obtained periodically, with a ratio of 1 or more suggesting a higher risk of CKD progression. ACEIs and ARBs may slow the progression of CKD, especially in patients with proteinuria. In addition to lowering systemic blood pressure, ACEIs and ARBs also lower glomerular capillary blood pressure and protein filtration, which may contribute to their beneficial effect in slowing progression. Patients with progressive CKD are at risk of renal osteodystrophy. Phosphorus, calcium, and parathyroid hormone levels should be monitored closely in all patients with stage 3 to 4 CKD. Abnormalities in these values may indicate the need for dietary phosphate restriction, administration of oral phosphate binders, and/or the administration of vitamin D. Patients with CKD also have an increased risk of cardiovascular complications and thus aspirin (eg, 81 mg/d) and aggressive lipid-lowering strategies should be used, including statins, with a goal low-density lipoprotein cholesterol (LDL-C) level of 100 mg/dL or less (to convert to mmol/L, multiply by 0.0259). All patients with a GFR of less than 30 mL/min per 1.73 m2 (stages 4-5) should be referred to a nephrologist.9
5. Do Not Automatically Discontinue an ACEI or ARB Solely Because of a Small Increase in the Serum Creatinine or Potassium Level. ACEIs or ARBs not only to optimize BP control, they are also the drugs of choice to prevent progression of proteinuric CKD. Increases of 30% in SCr are acceptable, it is important to confirm that the SCr level stabilizes at the higher value and does not continue to increase. A serum potassium level of up to 5.5 mEq/L (to convert to mmol/L, multiply by 1.0) is acceptable as long as it is stable and as long as the patient is aware of the need for dietary potassium restriction and will not be exposed to additional medications, such as spironolactone, that may exacerbate hyperkalemia. Follow-up SCr and potassium levels should be ordered within 1 week. In patients with an increase in SCr of more than 30% or in those with uncontrollable hyperkalemia, the ACEI or ARB should be discontinued or titrated to a lower dose. Diuretics that work with reduced GFR, such as furosemide or metolazone, are useful agents in the treatment of hypertension and hyperkalemia in CKD. However, it is important to note that volume depletion may make a patient more susceptible to an ACEI- or ARB-induced increase in the serum creatinine level.
6. Anemia in Patients With CKD Should Be Treated With Erythrocyte-Stimulating Agents Such as Recombinant Human Erythropoietin (EPO) But Should Not Be Overtreated. Target Hb should be between 11 and 12 g/dL (to convert to g/L, multiply by 10) and should not exceed 13 g/dL. All patients with anemia should be evaluated for other reversible causes, including vitamin deficiencies and iron deficiency. Routine evaluation would include a reticulocyte count and measurement of serum vitamin B12 and folate, serum iron, ferritin, and total iron-binding capacity. Patients (especially males and nonmenstruating females) with iron deficiency should undergo a gastrointestinal evaluation with endoscopy and colonoscopy. Iron deficiency must be corrected if erythrocyte-stimulating agents are to be effective.
7. Phosphate-Containing Bowel Preparations Should Be Used With Caution. Patients with CKD or chronic heart failure and those taking NSAIDs or diuretics would also appear to be at higher risk of acute phosphate nephropathy.
8. Patients With Severe CKD Should Avoid Magnesium- or Aluminum-Containing Oral Preparations. Concomitant Use of Citrate-Containing Preparations and Aluminum-Containing Oral Preparations (eg, Maalox and Mylanta) Is Potentially Hazardous Because It Can Lead to Acute Aluminum Toxicity. Moreover, certain medications, such as calcium citrate, potassium citrate, and sodium citrate, markedly enhance aluminum absorption from the gut. A number of studies have suggested that the concomitant use of citrate-containing preparations and aluminum hydroxide is potentially hazardous.

9. Although Most Patients With Hypertension Should Not Be Screened for Secondary Hypertension, Certain Clinical Clues May Suggest the Presence of an Underlying Cause That, When Addressed, May Resolve or Improve the Patient's Hypertension. Secondary hypertension is relatively uncommon (<5%). Severe or difficult-to-control hypertension, hypertension that suddenly develops or suddenly worsens, or hypertension that is associated with other clinical findings may indicate secondary hypertension. Hypokalemia may suggest primary aldosteronism. Headaches, palpitations, and sweats may suggest pheochromocytoma. Moon facies and/or striae may suggest Cushing syndrome, and a history of snoring in an obese patient may suggest obstructive sleep apnea. A bruit on one side of the abdomen may indicate renal artery stenosis. Worsening blood pressure or renal function on initiation of an ACEI or ARB may also suggest renal artery stenosis. Patients with peripheral vascular, cardiovascular, or cerebrovascular disease are at risk of also having renovascular disease. Other potentially correctable systemic conditions that can cause hypertension include hypothyroidism and hyperparathyroidism.

10. In Patients With Recurrent Stone Disease, an Indepth Metabolic Evaluation Is Needed to Identify and Treat Modifiable Risk Factors, Thereby Preventing Further Episodes and/or Promoting Stone Dissolution. Patients with a family history of stones, concomitant gastrointestinal disease such as inflammatory bowel disease, frequent urinary tract infections, or a history of nephrocalcinosis should be referred to a nephrologist for further evaluation. Patients without these risk factors should have a simplified evaluation that includes a dietary history, a review of medications that can promote stone development, urinalysis, and measurement of levels of serum calcium, phosphorous, electrolytes, and uric acid. Compositional stone analysis is an integral part of the metabolic evaluation because it can provide guidance for therapy. The first-time diagnosis of a single calcium-containing kidney stone in a patient without a family history of kidney stones and with normal findings on an initial metabolic evaluation can be managed by increasing fluid intake to maintain a urine output of about 2 L/d. If a uric acid or cystine stone is detected on stone analysis, further metabolic evaluation with a 24-hour urine stone risk profile (eg, UroRisk or StoneRisk Panel [Mission Pharmacal Company, San Antonio, TX]) is warranted to guide both dietary and medical interventions. Likewise, in patients without a stone available for analysis or in patients with multiple calcium stones, further metabolic evaluation, including the 24-hour urine stone risk profile, is recommended.

11. Cyclosporine and Tacrolimus, Drugs Commonly Used in Patients With Renal Allografts, Have Many Drug-Drug Interactions. Every new medication (prescribed, over-the-counter, and herbal) that is given to a patient with a renal allograft should be reviewed to determine if it will interact with his or her transplant medications (eg, tacrolimus or cyclosporine). If a drug must be used that has a considerable interaction, dose adjustment may be needed with careful follow-up of the calcineurin inhibitor levels.



Drug Related Renal Syndromes:

Link: NSAID toxicity | **Radiocontrast Nephropathy** | Drug Dosing in RF | **Specific Medication Dosing** |

Renal tubular dysfunction: renal potassium wasting and acidosis.

Nephrotic Syndrome: due to glomerular dysfunction from gold, NSAIDs, penicillamine, INF, captopril. Usually improves if stop the med.

Chronic Renal Failure: usually from drugs causing AIN. Seen with chronic analgesic abuse, Li, cisplatin, cyclosporine, 5-ASA, ifosfamide, mesalamine, nitrosourea and Chinese herbs. Occasional or moderate/ sensible use of aspirin, acetaminophen, or other NSAIDs (≥2500 pills over 14 years ~= 178/yr ~= 15/mo) is not associated with a decline in kidney function (GFR or Cr) (Am J Kidney Dis 2003;42:234-244).

Acute Renal Failure: an inc in Cr >0.5 mg/dL above baseline or if >1.5 mg/dL. May be prerenal, intrinsic or obstructive.

Drugs -- prerenal: diuretics, NSAIDs, ACEi, IL-2, high-osmolar radiocontrast media, cyclosporin and tacrolimus. Usually resolves when stop the drug.

Triple whammy: Combining an ACEi or ARB + diuretic + NSAID leads to riks for worsening renal function, increased BP, inc potassium or edema especially in pt's who are at risk for dehydration (during acute viral illness, period of low oral intake, excessive heat)….. was prescribed in 4.7% to 7.9% of pt's in one study (Br J Clin Pharmacol 2005;59:239-43). Low-dose aspirin (≤325 mg/day) aren't likely to cause renal problems. Glomerular filtration is impaired by the inhibition of prostaglandin synthesis by NSAIDs, thus they can cause azotemia in persons with hypovolemia, heart failure, or the nephrotic syndrome. ACEi's can cause azotemia in those with hypovolemia, heart failure, and bilateral renal artery stenosis (or renal artery stenosis in one kidney).

Drugs -- intrinsic: ATN | AIN | **Thrombotic Microangiopathy** | Rhabdo |
Drugs -- obstructive: Crystal formation obstructing tubules seen with acyclovir, sulfonamides, MTX, indinavir, triamterene, mega-dose Vit-C. Occasionally guaifenesin and ephedrine can cause stones to form. Elderly pt's who receive high doses of ciprofloxacin may be especially vulnerable to ciprofloxacin-induced crystal nephropathy according to a report on 2 cases (Am J Kidney Dis 2007;50:330-5)....should be considered as a potential cause of acute renal failure.
Benign urine sediment: containing few formed elements should alert the physician to the possibility that obstruction is present.
Retroperitoneal fibrosis with methylsergide, bromocriptine, methyldopa, hydralazine and beta-blockers. Risk with volume depletion. Urine sediment contains RC's, WBC's and crystals. Alkalizing the urine prevents sulfonamide, MTX and triamterene crystals. (Drug-induced nephrotoxicity. Clev Clin J Med 2002;69:4)

ICD9 Codes:

585.x Chronic Kidney Disease Stage 1 = .1, Stage 2 = .2, .3 and .4.
585.1—CKD, stage 1: Kidney damage with normal or increased GFR ≥90 mL/min/1.73m2.
585.2—CKD, stage 2 (mild): Kidney damage with mild decrease in GFR, 60-89 mL/min/1.73m2.
585.3—CKD, stage 3 (moderate): Kidney damage with moderate decrease in GFR, 30-59 mL/min/1.73m2.
585.4—CKD, stage 4 (severe): Kidney with a severe decrease in GFR, 15-29 mL/min/1.73m2.
585.5—CKD, stage 5: Kidney damage with GFR of l<15 mL/min/1.73m2 or kidney failure with GFR <15 mL/min/1.73m2 and the pt is not on dialysis.
585.6—End-stage kidney disease. The pt has stage 5 disease and is on dialysis.
585.7—For chronic renal insufficiency and chronic renal failure not otherwise specified.
585.9 Chronic Kidney Disease NOS
285.2 Anemia of Chronic Kidney Disease (**CKD**)
403.01 Hypertensive Kidney Disease, malignant with chronic kidney disease
403.11 Hypertensive Kidney Disease, benign with chronic kidney disease
404.13 Hypertensive Heart & Kidney Disease, with heart failure and chronic kidney disease
403.00 Hypertensive CKD, malignant, with CKD stage I through stage IV, or unspecified
403.01 Hypertensive CKD, malignant, with CKD stage V or end stage renal disease
403.10 Hypertensive CKD, benign, with CKD stage I through stage IV, or unspecified
403.11 Hypertensive CKD, benign, with CKD stage V or end stage renal disease
403.90 Hypertensive CKD, unspecified, with CKD stage I through stage IV, or unspecified
403.91 Hypertensive CKD, unspecified, with CKD stage V or end stage renal disease
404.00 Hypertensive heart and CKD, malignant, without heart failure and with CKD stage I through stage IV, or unspecified
404.01 Hypertensive heart and CKD, malignant, with heart failure and with CKD stage I through stage IV, or unspecified
404.02 Hypertensive heart and CKD, malignant, without heart failure and with CKD stage V or end stage renal disease
404.03 Hypertensive heart and CKD, malignant, with heart failure and with CKD stage V or end stage renal disease
404.10 Hypertensive heart and CKD, benign, without heart failure and with CKD stage I through stage IV, or unspecified
404.11 Hypertensive heart and CKD, benign, with heart failure and with CKD stage I through stage IV, or unspecified
404.12 Hypertensive heart and CKD, benign, without heart failure and with CKD stage V or end stage renal disease
404.13 Hypertensive heart and CKD, benign, with heart failure and CKD stage V or end stage renal disease
404.90 Hypertensive heart and CKD, unspecified, without heart failure and with CKD stage I through stage IV, or unspecified
404.91 Hypertensive heart and CKD, unspecified, with heart failure and with CKD stage I through stage IV, or unspecified
404.92 Hypertensive heart and CKD, unspecified, without heart failure and with CKD stage V or end stage renal disease
404.93 Hypertensive heart and CKD, unspecified, with heart failure and CKD stage V or end stage renal disease

Microhematuria:

Links: Info & ICD9 | **Definitions & Red Flags** | Hx | **W/u** | Nephrology Eval | **Etiology** | Drug Induced | **Gross** | References | **Urinalysis** | Pediatric | **Hematospermia** |

Found in up to 5-10% of asymptomatic normal adults and 1-2-4.1% of school children in the general population. Normally up to 2 million RBC's are excreted in the urine daily. Routine screening of urine is not recommended by the USPSTF or Canadian Task Forces.
ICD-9: 599.70 Hematuria, unspecified
599.71 Gross hematuria
599.72 Microscopic hematuria
Definition:
>3-5 RBC's/ HPF (some labs vary) on 2 of 3 properly collected specimens.
≤3 RBC's = normal regardless of +dipstick, which has more **False +'s** (= micro with no RBC's, also seen in rhabdomyolysis and hemolytic anemia).
False Negatives: seen with air exposed strips, ascorbic acid (>2g/d), pH<5.1, formaldehyde. Risk factors for significant dz should always be taken into account.
Red Flags: Smoking history. Occupational exposure to chemicals or dyes (benzenes or aromatic amines). Age >40 years. History of urologic d/o or disease. History of irritative voiding sx's. History of UTI. Analgesic abuse. History of pelvic irradiation.
W/u: If have a UTI, tx the infection and repeat the UA in 6 weeks.
If low clinical suspicion for disease **-->** Repeat the UA with micro (look at sediment) to see if 2 of 3 UA's are positive. No eval needed if no hematuria on repeat and no **risk factors for bladder cancer** (smoker, exposure to toxins, occupational chemicals, irritative voiding sx's).
If **gross** with dysuria/ pyuria/ WBC casts, get culture and give Abx empirically for cystitis, re-check in 14-42 days.
Lab: BUN/Cr, U/A & C&S, ± Chem 18, ±24hr Ur Protein if 2-3+ on dipstick, CT. U/S is the least expensive and safest choice for evaluating microscopic hematuria.
Micro exam: if nl, eval for hemoglobinuria or myoglobinuria.

If isolated glomerular microscopic hematuria: Blood originating from the nephron is termed glomerular or nephronal hematuria. These RBCs can be trapped in Tamm-Horsfall mucoprotein and will be manifest in the urine by RBC casts. Acanthocytes or RBC casts [pathognomonic are seen on micro. Follow q6 x2, then q12mo X3yrs (@6, 12, 24, 36mo) with U/A, BP, BUN/Cr, ±cytology to check for the development of proteinuria or renal insufficiency (Nephron 1996;72) (NEJM 2003;348:2330-8). The presence of proteinuria helps support a glomerular source of blood loss. See **Etiology** |
If isomorphic (nl shape) red cells: likely non glomerular hematuria, obtain helical CT. If negative obtain cytologic analysis of 3 serial first morning specimens.
Get cytology if >50yo to r/o TCC (grade 1-25% yield, grade 4-75% yield), will need to repeat q6mo until hematuria resolved or 3yrs have passed. Urine cytology studies alone may provide sufficient evaluation of the lower urinary tract in certain low-risk pt's. But older pt's and those who have identifiable risk factors for urothelial neoplasms merit referral to a urology subspecialist for cystoscopy.
Cystoscopy if positive cytology, also consider if no current infection and age ≥50yo or if <50 with risk factors.
CT Urography (CTU): CTU offers a comprehensive evaluation of the kidneys, urinary tract, and bladder, it may obviate the need for cystoscopy in low-risk pt's with hematuria as has a sens / spec 79% & 94% (vs cystoscopy at 95% & 92%) with a PPV 75% (vs 72%) and NPV of 95% (vs 99%) (RSNA. doi:10.1016/S0300-7073(07)70326-2).
IVP **(IVP:)** had been the gold standard as the best initial functional and anatomical study. By itself it has limited sensitivity in detecting small renal masses and thus it is being replaced by spiral CT and U/S. Get a renal U/S if renal insufficiency (Cr> normal) or contrast allergy. Start with a spiral CT if any acute abd process. If have a non contrast scan demonstrating urolithiasis in a pt with low risk of malignancy, can stop the eval, if no calculus detected then need a contrast study or U/S. If complex cyst or solid renal mass on U/S get CT of Abd/ pelvis. CT more accurate than IVP to asses the cause of asymptomatic hematuria and requires less additional imaging (J Urol 2002;168:2457-60).
Confounding: rhubarb, Vit-C, Pyridium, Coumadin (high INR, nl level may unmask lesion), clots = nonglomerular origin.
Get Nephrology Eval If: Renal insufficiency or **proteinuria** >500mg/ 24hr, dysmorphic RBC's (often >80%) **-->** Glomerular dz labs. Get 24hr protein. Check a serum "IgA fibronectin level" as 60% positive if IgA nephropathy.
If find dysmorphic RBC's, RBC casts and / or proteinuria --> get ANA, ANCA, anti-GBM, ASO, anti-hyaluronidase, RPR, HIV, Chem 17, 24hr urine, cryoglobulins, C3, C4, CH50, hep serology. Consider SPEP and SIEP. May need renal Bx especially if abnormal renal function or proteinuria >1.5g/d.
Consider --> throat Cx, ANA, serum complement (GN), renal Bx. Most cases are idiopathic, Benign Familial Hematuria (= thin basement membrane nephropathy, non progressive), Bergers dz (IgA nephropathy), post-Step GN or **Alport's** (chronic hereditary nephritis). 70% of cases of Alport's are discovered by age 6yo, 70% have proteinuria, 50% with SNHL (4,000-8000Hz), all males progress to ESRD.
Isolated hematuria: consult Urology if age> 40-45, Nephrology if <40yo, r/o AV fistula (renal), Burger's dz (IgA nephropathy).
Pediatric / Children: Asymptomatic children with normal physical exams and urinalysis except for microscopic hematuria should be followed regularly but do not require diagnostic evaluation. The prevalence of asymptomatic hematuria is ~0.5 to 2% in children. Diagnostic studies are warranted in children with HTN or other urine abnormalities such as gross hematuria (The clinical significance of asymptomatic gross and microscopic hematuria in children. Arch Pediatr Adolesc Med 2005;159:353-5). Of 570 children evaluated, no cause was discovered in 80%, hypercalciuria (which presumably causes hematuria through formation of crystals or small calculi) was discovered in 16%. Four children (1%) had post-streptococcal glomerulonephritis (which resolved spontaneously in all), four had minor structural abnormalities of the urinary tract, one had vesicoureteral reflux, one had IgA nephropathy, and one had membranoproliferative glomerulonephritis. No child had a UTI. 53 pt's have a renal bx (thus very little yield for the costs and risks).
Gross Hematuria: The child with gross hematuria who is suspected of having a nephrologic process, typically glomerulonephritis, needs evaluation for hypertension, edema, and oliguric renal failure. The child whom the provider suspects has bleeding from outside the nephron needs an immediate imaging study, typically U/S of the kidneys and bladder, because bleeding may stem from anywhere in the urinary tract.
Etiology: Infection accounts for 25% of all cases and stones another 20%, yet despite work-up, a urologic cause of hematuria is not discovered in ~10% (Med Clin North Am. 1997;81:641-52 and Emerg Med Clin North Am. 2001;19:621-32).

1. Nonglomerular:

Exercise "sports hematuria" --> bladder trauma (up and down movement of the bladder), up 18% of normals may have after prolonged exercise, re-check post cessation of exercise X3d). Often see dysmorphic red cells and, in up to 30% of cases, red cell casts and mild proteinuria.
Polycystic kidney (up to 10% have cerebral aneurysm). Exercise-induced hematuria is a relatively common, benign condition that often is associated with long-distance running. Results of repeat urinalysis after 48 to 72 hours should be negative in pt's with this condition.
Papillary necrosis (low blood flow) **-->** necrosis, in DM, TB, SS, obstruction, analgesics, pyelo). Other: Ca, Vascular dz (malignant HTN, renal v thrombosis, atheroemboli), loin pain hematuria, hypercalciuria (young adult/ child).
Medullary sponge kidney --> assoc. with hematuria, hypercalcemia (50%), nephrocalcinosis, Ca stone dz and hemihypertrophy. Does not cause renal failure. Dx by IVP showing small cystic outpouchings of the renal papillary ducts.
Ddx Nonglomerular causes: Renal (tubulointerstitial) Acute tubular necrosis: Familial. Hereditary nephritis. Medullary cystic disease. Multicystic kidney disease. Polycystic kidney disease. Infection: pyelonephritis, tuberculosis (e.g., travel to Indian subcontinent), schistosomiasis (e.g., travel to Africa). Infection: syphilis, toxoplasmosis, cytomegalovirus, Epstein-Barr virus.
Systemic disease: sarcoidosis, lymphoma, Sjögren's syndrome. Loin pain-hematuria syndrome. Hypercalciuria. Hyperuricosuria. Renal cell carcinoma. Solitary renal cyst. Vascular disease. Arteriovenous malformation. Malignant hypertension. Renal artery embolism/thrombosis. Renal venous thrombosis.
Secondary to systemic disease: Congenital abnormalities. Endometriosis. Factitious. Foreign bodies.
Infection: prostate, epididymis, urethra, bladder
Inflammation: drug or radiation induced. Perineal irritation. Posterior ureteral valves. Strictures. Transitional cell carcinoma of ureter, bladder.

Trauma: catheterization, blunt trauma. Tumor. Exercise hematuria. Menstrual contamination. Sexual intercourse. Prostate cancer is detected in a high percentage of men between the ages of 50-79 with persistent hematuria (BJU Int 2006;98:1221-1224) (4.7% if macroscopic haematuria vs 8.5% of those with microscopic haematuria)....suggest with PSA testing and prostate biopsy if indicated.
Meds: Aminoglycosides. Amitriptyline. Analgesics. Anticonvulsants. Aspirin. Busulfan (Busulfex). Chlorpromazine (Thorazine). Cyclophosphamide (Cytoxan). Diuretics. Oral contraceptives. Penicillins (extended spectrum). Quinine (QM-260). Vincristine (Oncovin). Warfarin (Coumadin).
Drug induced Interstitial nephritis: penicillins, cephalosporins, diuretics, nonsteroidal anti-inflammatory drugs, cyclophosphamide (Cytoxan), chlorpromazine (Thorazine), anticonvulsants.
2. Glomerular: have systemic illness with fatigue/ arthralgias see dysmorphic RBC's/ casts **and proteinuria** check ANA, complements, anti GBM, ANCA, ASO, cryoglobulins, (+RPR, HIV, HBsAg, anti-HCV, SPEP). Need ASAP Nephrology consult if nephrotic or have impaired renal function.
IgA Nephropathy --> gross hematuria within 1-2 days of URI/ skin infection.
Thin BM Nephropathy (TBMN) / Disease: A common cause of asymptomatic hematuria in children. Have dysmorphic RBC and/or RBC casts. Check serum lytes, BUN, Ca, uric acid, complement panel, IgA, IgG, Ur Cr, prot, Ca, renal U/S. Often have a +FHx (AD), persists long time. Have variable thickness on renal Bx. Good prognosis. (Clin Pediatr 2001;40:607-13).
Alport's Syndrome (Hereditary Nephritis, X-linked defect in collagen, hearing loss (HL), lens abnormalities, often ESRD by 16-35), **also:**
Post infectious GN --> hematuria 6-21 days **after** URI/ skin infection. Other **-->** membranoproliferative, RPGN, SLE, HSP, Goodpasture's, DM, FSGN.
Ddx of Glomerular causes: Fabry's disease. Hereditary nephritis (Alport's syndrome). Nail-patella syndrome. Thin basement-membrane disease. Primary glomerulonephritis. Focal segmental glomerulonephritis. Goodpasture's disease. Henoch-Schönlein purpura. IgA nephropathy (Berger's disease). Mesangioproliferative glomerulonephritis. Postinfectious glomerulonephritis. Rapidly progressive glomerulonephritis. Secondary glomerulonephritis. Hemolytic-uremic syndrome. Systemic lupus nephritis. Thrombotic thrombocytopenic purpura Vasculitis.
3. Extrarenal: Infection, calculi, neoplasm, BPH, Meds (cyclophosphamide **-->** hemorrhagic cystitis), trauma, extra-GU (anal fissure, vaginal prolapse, endometriosis), Schistosoma haematobium.
4. Hematologic: coagulopathy, SS, anticoagulants.
5. Malignancy: risks: age >40, tobacco use, h/o pelvic irradiation, exposure to cyclophosphamide or occupational toxins such as dyes, benzene, aromatic amines, chimney sweepers. Analgesic abuse.
Other causes:
Renal: Arteriovenous malformation. Hypercalciuria. Hyperuricosuria. Loin pain-hematuria syndrome. Malignant HTN. Medullary sponge kidney. Metabolic causes. Papillary necrosis. Polycystic kidney disease. Renal artery embolism. Renal vein thrombosis. Sickle cell disease or trait. Tubulointerstitial causes. Vascular cause.
Urologic causes: Benign prostatic hyperplasia. Cancer (kidney, ureteral, bladder, prostate, and urethral). Cystitis/pyelonephritis. Nephrolithiasis. Prostatitis. Schistosoma haematobium infection. Tuberculosis. Urologic hematuria is distinguished from other etiologies by the absence of proteinuria, dysmorphic RBCs, and erythrocyte casts. Even significant hematuria will not elevate the protein concentration to the 2+ to 3+ range on the dipstick test. Up to 20% of pt's with gross hematuria have urinary tract malignancy; a full work-up with cystoscopy and upper-tract imaging is indicated in pt's with this condition. In pt's with asymptomatic microscopic hematuria (without proteinuria or pyuria), 5-22% have serious urologic disease, and 0.5-5% have a genitourinary malignancy.
Trauma: contact sports, running, Foley catheter.
Pregnant women: those with dipstick-positive hematuria can be reassured that their pregnancy is unlikely to be affected by the condition (Am J Kidney Dis 2005;45:667-673), however, these pt's should be followed up after delivery to identify those with glomerulonephritis.
Athlete's (Exercise-induced) hematuria: both hematuria and albuminuria are benign, self-limiting conditions occurring in many athletes. Microscopic or gross hematuria and mild albuminuria are most likely in the first urine voided after exercise and should resolve within 48 hours (Scientific American medicine. 2001: p CTM: I: 1–32). Severe or slowly resolving hematuria or albuminuria indicate the need for careful urinary tract evaluation.

Drug Induced Hematuria:

Pseudohematuria (Rare) --> phenytoin, ibuprofen, levodopa, nitrofurantoin, rifampin, quinine.
Glomerulonephritis --> mercury, gold, penicillamine, heroin, probenecid, antivenom.
Vasculitis --> allopurinol, colchicine, diphenhydramine, furosemide, hydantoins, isoniazid, penicillins.
Thrombotic microangiopathy --> chemo agents, cyclophosphamide.
Interstitial nephritis --> penicillins (Methicillin 10%, Nafcillin 3%), rifampin, ibuprofen, sulfonamides, phenindione, phenytoin, cyclophosphamide, danazol, mitotane, busulfan, chloroform, NSAIDs, olsalazine, Silvadene, TMP/ SMX, Furosemide, Cipro, cephalosporins, Omeprazole.
Chronic interstitial nephritis --> analgesics, Li, chemo agents.
Intrarenal obstruction --> methotrexate, sulfonamides, tumor lysis syndrome.
Extrarenal obstruction --> methysergide.
Hemorrhagic cystitis --> Cyclophosphamide, Ifosfamide, Mitotane.
Nephrolithiasis --> triamterene, Vit-D, acetazolamide, Ritonavir, Indinavir, Mirtazapine, Dichlorphenamide.
Urinary tract carcinoma --> analgesic abuse. (*Am Fam Phys* 1999; 60:4)
Ddx: **myoglobinuria** (muscle fiber necrosis, has inc CPK, nl haptoglobin), porphyria, oxidizing agent (bleach), provodone Iodine, narcotic seeker, munchausens's, red diaper syndrome, drugs (rifampin, phenothiazines, Flagyl), fruits and veges with plant dyes (beets, berries, rhubarb), hypotonic urine (sg <1.010), urine left out >30min.

Gross Hematuria:

Need urgent consult if RBC casts, renal dysfunction or high grade proteinuria is present.
Hx: **clots:** large = bladder, small stringy -upper tract.

Gross hematuria: suggests distal urethra to the urogenital (UG) diaphragm. >100 RBC/ HPF **-->** red color. Pinks up at 30-30 RBC's. If long Hx of smoking, then inc risk for neoplasia.
The old "Three Glass Test" was historically used before cystoscopy.
#1: Terminal: prostate or bladder neck (e.g. varices), urethritis, trigone irritation.
#2: Total: bladder or upper tract: stone, tumor, TB, nephritis.
#3: Initial: anterior urethral lesions, urethritis, stricture, meatal stenosis.
W/u: r/o infection, especially if female. Check IVP if normal renal function. Consider CT scan ±cystoscopy. If h/o bone marrow transplant r/o hemorrhagic cystitis from adenovirus. Even pediatric pt's need a thorough diagnostic eval (Arch Ped & Adol 2005;159:353-55).
Labs: CBC, BUN, Cr, UA with micro, Urine C&S. Use of CT urography to evaluate macroscopic hematuria possibly due to bladder cancer obviates the need for intravenous urogram and U/S and can help determine whether cystoscopy is warranted (BJU Int 2006;98:345-348)….of 200 consecutive pt's >40yo, compared with the histopathological findings obtained with cystoscopy, CT urography yielded one false-positive and three false-negative findings, resulting in a sensitivity of 93%, a specificity of 99% for bladder cancer dx.
Consider: PT, PTT, peripheral smear, urine cytology, ANA, anti-dsANA, C3, C4, anti-GBM, ANCA, renal U/S, 24h protein and creatinine, renal Bx.

Trauma: if **gross hematuria** or >10 RBC/ HPF, follow closely re-check U/S in 6wks, if still + **-->** IVP.
*****Ref:** (Microscopic hematuria NEJM 2003;348:2330-8) (Evaluation of asymptomatic hematuria in adults. Urology 2001;57:599) (Evaluation of asymptomatic microscopic hematuria. *Urol Clin North Am*. 1998;25:661-76) (Workup and management of traumatic hematuria. *Emerg Med Clin North Am*. 1998;16:145-64) (*Med Clin NA* 1997;81:641) (*Kidney Int* 1996;49:222) (Nephron 1996;72:125) (Management of nephropathy, *Kidney Int* 1999;70:S56-70) (Exercise induced hematuria. *Am Fam Phys* 1996;53:3) (A practical primary care approach to hematuria in children. *Pediatr Nephrol.* 2000;14:65-72) (The pt with hematuria. *Practitioner*. 1999;243:564-70) (Evaluation of asymptomatic microscopic hematuria in adults. *Am Fam Physician*. 1999;60:1143-52)

Proteinuria:

Links: Quantity | **Definitions** | Microalbuminuria & Screening for | **Ur Prot-Cr Ratio** | W/u | **Tx** | Etiology - Ddx & Considerations | **Benign Causes & Orthostatic** | Overflow | **Tubular** | Glomerular | **Child** | Diabetic Nephropathy | **References** |

…….END DEMO of this chapter.

Neurology:

Edited by Robert D. Harbaugh, MD, Neurologist.

Contents: Neuro Exam | **Weakness Disorders** | Peripheral Neuropathy | **Brachial Plexus / UE / LE** | Movement Disorders | **Parkinson's Dz** | Spells | **Seizures** | Strokes | **Cephalgia (H-A)** | Coma & Encephalopathy | **Cervical-Thoracic-Lumbar Spine** | Pseudo-Neurologic Syndromes | **Various Other & Anatomy** | Diagrams | **Pain** | Delirium, Dementia & Memory | **CNS Infections** | Facial Nerve (CN 7) Palsy | **Diplopia / Ocular Nerve Lesions** | ENT | **Sleep D/o's** | Brachial Plexus | **Dizziness** | PEDIATRICS | **Head Injury** |

The Neurology Exam:

Links: Neurology pearls / Approach | **Quick Exam** | Cranial Nerves | **Sensory** | Motor | **Reflexes** | Pyramidal Tract Dz/ Babinski / Soft Signs | **Cerebellar Testing** | GCS | **EEG & Evoked Potentials** | EMG/NCS | **Imaging Studies, Biopsies & Other Tests** | Gait Disturbance | **Neurologic Patterns / Alterations** | Neuro Levels | **Pseudo-Neurologic Syndromes** | See Rheum for **Forearm (exercise) Ischemic Test** | See Psych for **Mental Status Exam** | See ID module for **LP** |
*** For mental status exam see psychiatry text, for memory, dementia testing/ evaluation/ tx, see Geriatrics.

Basic Neuro Hx: "OPQRST" :

Onset (and evolution),
Provocation (exacerbating and relieving factors),
Quality, **R**adiation(site), **S**everity (best, worst, average, current on 1-10 scale),
Time (constant, intermittent, predictable).
Other: Prior tx's, current meds, drugs/ETOH use, how pain interferes with life, why seek tx now, pt's ideas as to cause.

Neurology Pearls:

#1 Ask the basic questions:
A. Is it neurologic?
B. Where is it in the nervous system (most likely anatomic site)?
From distal to proximal: Muscle. Neuromuscular junction. Peripheral nerve. Root. Spinal cord. Brain stem. Cerebellum. Subcortical brain. Cortical brain. Begin distally (with the muscle) and to ask the patient questions about each part of the neurologic anatomy, working backward (proximally) from the muscle, through the brain.
C. What is the pathophysiology (etiological cause)?
D. What diagnostic tests to order to support your opinion or excluding other possibilities? The differential diagnosis.
E. What can we do about managing the problem?
#2 "History is everything": take another history if uncertain ("the second visit rule"); interview observers before establishing a working dx; use the pt's own words in describing sx's (dx comes later). MSE can be incorporated into the Hx by asking questions about ADLs', are they reading, writing, balancing check book. Only after the lesion is localized by history-taking should the physical exam begin. If localization of the lesion is still unclear after a careful history, do not begin the physical exam-take a better history.
#3 Use neurological localization tools in making a dx. Most serious or threatening neurological d/o's follow anatomic and physiologic principles of localization and function; however, human diversity exists and "outliers" are not uncommonly encountered. Embellishment is common, it doesn't necessarily reflect malingering. Use objective test (reflexes) to confirm subjective abnormalities.

#4 Always think of the most threatening diagnoses, but do not feel compelled to pursue an exhaustive investigation unless clinical grounds support such … consider "following" the challenging pt with a slowing evolving d/o once reasonable diagnostic efforts have been exhausted (e.g. indolent neuropathy, dementia, subjective numbness, etc.)
#5 Customize the neurologic exam – the exam serves to 1) confirm or exclude suspected deficits by the history; 2) unearth subclinical signs (e.g. Babinski sign); 3) provide a baseline for future comparisons. Taylor the exam to the complaint. Use the pt's own body as the control by comparing L-to-R.
#6 A few words about therapeutics: 1) there is no fixed dosing for any primary neurological d/o – thus, titration to optimal efficacy is always performed – "start low, go slow, but don't stop".
2) human diversity has led to a need for rational therapeutic trials for many neurological d/o's.
3) always define your therapeutic goals with the pt – reassess if the goals are not met.
#7: The Neuro Differential:
"**Vindi Itch MD"-->**
Vascular, **I**nfectious, **N**eoplastic, **D**rugs, **I**mmune.
Idiopathic, **T**rauma/ **T**oxic, **C**ongenital, **H**ereditary.
Metabolic **D**egenerative/ Demyelinating.
Remember: Atypical presentations of common things are still common! In agitated and uncooperative patients, neurological examination is some times best achieved by careful observation of the pt's behavior. Transient, bilateral loss of vision suggests raised intracranial pressure. All oculomotor (third nerve) palsies are urgent, whether the pupil is involved or not. Worsening neurological symptoms in the evening suggests myasthenia gravis. Episodic loss of consciousness without provoking factors may be due to epilepsy. Numb chin – think of metastatic carcinoma. Progressive headache with neck stiffness, fever, vomiting, with or without rash, is bacterial meningitis until proved otherwise. Do not forget viral encephalitis in the elderly with pyrexia and altered level of consciousness. Sensory disturbance on neck flexion suggests an inflammatory lesion of the cervical spinal cord. Thunderclap headache or focal neurology during pregnancy or post partum may mean cerebral venous sinus thrombosis. Sudden onset of headache – think of subarachnoid hemorrhage. 2 Reduced level of consciousness after head injury may be due to extradural hematoma. 3 An elderly patient with confusion, hemiparesis or deteriorating consciousness level after a fall – exclude chronic subdural hematoma.
Continuing complaints of headache, worse in the morning, may indicate raised intracranial pressure. New onset of seizures in an adult means a brain tumur until proved otherwise. Low back pain with sphincter disturbance is cauda equina syndrome until proved otherwise. Sudden onset of headache, visual deterioration and reduced mental state may be pituitary apoplexy. Acute alcohol withdrawal can cause serious neurological problems. Headache with eye movement abnormality requires a scan. Clear fluid leaking from the nose or ear after trauma may be CSF. A patient with difficulty walking – check for a sensory level.

The 2-Minute Neuro Exam:

For the young & healthy to r/o significant dz.
Observation during interview: arousal, speech, language, mood, behavior, vision, hearing, cognition, memory, thinking, personality, facies, grooming, tics, mannerisms, movements, postures, pain behaviors, signs of acute distress (pallor, edema, respirations).
Step #1: Stand with feet together, arms out in front, palms up, eyes closed**:** look for drift/ sway/ unsteadiness.
Step #2: "Close eyes tightly"**:** look for weakness/ asymmetry. "Touch your nose with each outstretched hand".
Step #3: Open eyes, follow my finger with your head held still**:** look at tracking and pupils.
Step #4: Sit pt on table**:** Check fundi, Check reflexes, look for Babinski sign.
Other: draw intersecting pentagons, check proximal muscle strength.

Cranial Nerves:

Links: CN 1,2,3,4 | **CN 5 (Trigeminal) to 7** **|** CN 8-12 | See ENT for **Cavernous sinus thrombosis** **|**
Most cases are examined by merely observing the pt. Pay special attention to the ocular fundus it is a window to the brain to directly assess the bodies vasculature. Check VA by reading the print on a 4X4 or alcohol prep at 15-18 inches. Compare visual fields by finger counts in each 4 quadrants of each eye. Use the whisper test to check hearing. Stick out tongue and say "Ah", move neck from side to side against mild resistance, shrug shoulders.
Anatomy: All CNs originate from the brainstem, except CNs I and II. The location of CNs III and IV nuclei are in the midbrain; CNs V, VI, VII, and VIII are in the pons; and CNs IX, X, XI, and XII are in the medulla. CNs I, II, and VIII are purely sensory. CNs III, IV, VI, XI, and XII are purely motor; and the remainder are mixed. CNs III, VII, IX, and X have parasympathetic fibers.
CN #1 (Olfactory) = smell. Not usually done. Have the pt occlude one nostril and identify a common scent (eg, coffee, oranges, flavored gum such as peppermint, cinnamon) placed under the other nostril. The stimulus should be nonirritating. Can be difficult to know whether pt's who cannot identify a particular scent have a disturbance of olfaction or whether they are just not familiar with that scent.
Ddx: trauma, olfactory groove meningioma.
Pearls: Most rarely test this nerve; it is typically only tested if the pt complains of a problem. Ask patients whether they have difficulty with smell or taste. Anosmia is the inability to recognize scent, and hyposmia is diminishment of smell. The sense of smell diminishes with increasing age. The most common causes of bilateral anosmia/hyposmia are the common cold and trauma to the nose and head. Neurological diseases that are associated with hyposmia include Parkinson's, dementia and B12 deficiency. Consider a subfrontal tumor in cases of unilateral anosmia. Hyposmia is commonly associated with a taste problem. The odor of ammonia is recognized through the trigeminal nerve (CN V); therefore, the pt who has no sense of smell, including to ammonia, should be considered to have a functional disorder (conversion).
CN #2 (Optic) = Acuity, fields, fundi. Lesion causes loss of vision (monocular bitemporal hemianopsia). See Ophthalmology section. Pupillary light reflex. Have the pt cover his or her left eye. Stand facing the pt from two arm's lengths away, close your right eye, and stretch your arms forward and to the sides so that your hands are at about "1:30" and "10:30" and just barely visible in your peripheral vision. Wiggle the finger on either the left, right, or both hands, and ask the pt to identify where the movement occurs while looking directly at your nose. Acuity via a hand-held visual acuity card 14 inches in front of the pt, have them read the 20/20 line with each eye. See Optho chapter for **Comlpete Eye Exam** **|**
Ddx: optic neuritis, pituitary tumor, ischemic optic neuropathy, optic nerve glioma, Leber disease.

CN #3 (Oculomotor) = eye movement, pupils, eyelid opening. Lesions causes weakness of adduction, elevation, and depression of eye, ptosis, a dilated, nonreactive pupil. Pupils can be large in pediatric pts and small in elderly. Pupillary asymmetry (up to 0.5 mm) is seen in 20% of normal individuals. See Optho module for **Pupils** |
Ddx: trauma, microvessel ischemia, compression by aneurysm or mass, brain stem stroke or tumor, orbital tumor.
CN #4 (Trochlear) = Sup oblique m. Lesions causes Weakness of depression and intorsion of eye.
Ddx: trauma, microvessel ischemia, brain stem stroke or tumor, orbital tumor.
CN #5 (Trigeminal) = muscles of mastication, tooth sensation, facial sensation, corneal reflex (sensory component). Lesion causes weakness of chewing and other jaw movements with loss of facial sensation and corneal reflex.
Motor component contracts the masseter & temporal muscles for clenching, and lateral movement of the jaw by the pterygoids. Feel the contraction of the masseter and temporalis muscle while the pt is clenching the teeth.
Jaw jerk: Have the pt open the mouth slightly and relax the chin. Place your finger on the chin and tap your finger gently with a percussion hammer. Most nor-mal individuals have no jaw jerk. Increased, or "brisk," jaw jerk is seen in an upper motor neuron lesion, with localization of the lesion above the foramen magnum.
Sensory component consists of three divisions: I. Ophthalmic, II. Maxillary, III. Mandibular. Ask the pt to close his eyes and test each of these divisions simultaneously on both sides. Apply both the sharp and the dull side of a pin for pain perception, and cotton swab for fine touch. If you detect any sensory deficit then do hot and cold object test for temperature perception. Use tubes containing ice-water and warm water. Sensation at the angle of the jaw is not mediated by the trigeminal nerve.
Corneal reflex (CR): not routinely checked. To check the integrity of corneal reflex (blinking conjunctiva), lightly touch the cornea with the edge of a fine threaded cotton swab-the sensory response is relayed by the CN V to the facial motor nucleus producing contraction of the orbicularis oculi-muscle. Always check on pt's with Bell's palsy, comatose patients, or in patients suspected to have brainstem or cavernous sinus lesion. The afferent limb of this reflex is supplied by CN V and the efferent limb by CN VII. Check with a cotton wisp and not a Q-tip or paper. Explain to the pt what you are going to do; it is best to demonstrate by touching the pt's face with cotton. Touch the edge of the cornea while the pt is looking to the other side, or you can touch the bottom of the cornea while the pt is looking up. You can check CR by blowing air onto the eye (this is a particularly useful technique in comatose patients and/or patients unable to keep their eyes open). Note an ipsilateral and contralateral eye blink, and ask the pt whether the sensations of both corneal touches were the same or different.
Pearl: Do not touch the cornea too briskly because you will get a reflex blink response. It is important that you touch the cornea and not the sclera, because you may not get a response (common mistake by students). The corneal reflex is decreased or absent if the pt is wearing contact lenses. Interpretation: When CR is absent bilaterally, consider a trigeminal nerve (CN V) or a pontine lesion; if it is absent unilaterally, a facial nerve or cere-bellopontine lesion.
Ddx: Trigeminal neuralgia (tic douloureux), scleroderma and other connective tissue diseases, tumor of petrosal bone.
Sensory loss in the distribution of a single branch—V1: herpes, cavernous sinus thrombosis; V2: trauma; V3: basal tumor or meningitis.
Sensory loss in the distribution of all branches: geniculate ganglion, sensory root or nucleus lesion, for example, basilar meningitis or pontine lesion.
Sensory loss of light touch only: sensory root lesion.
Sensory loss of pain and temperature: brainstem lesion.
Sensory loss in the distribution of V2 or V3: metastasis.
Sensory loss around the mouth: syringomyelia.
CN #6 (Abducens) = lateral rectus m for lateral gaze. Lesion causes weakness of abduction of eye.
Ddx: Trauma, raised intracranial pressure, microvessel ischemia, brain stem stroke or tumor, orbital tumor.
CN #7 (Facial) = facial expression, ant 2/3 of tongue taste, salivation, tearing, hyperacusis (stapedius). When one entire side of the face is weak, the lesion is usually peripheral. With a central lesion (such as a stroke in one cerebral hemisphere), the forehead muscles are often spared because the portion of the facial nerve nucleus representing the forehead typically gets input from the motor strips of both cerebral hemispheres. The portion of the facial nerve nucleus representing the lower face does not have the same bilateral input; its input is predominantly from the contralateral cortex. Look for asymmetry between the nasolabial folds, forehead wrinkles, blinking, or smiling. Ask the pt to show the teeth, whistle, or wrinkle their forehead and again look for asymmetry. Gently check eye resistance of closure muscles. Note if there is weakness of the lower face and forehead muscles on the same side. Bell's phenomenon is when the eyes roll up while the pt is trying to close the eyes. The sensory portion of the facial nerve supplies the external auditory canal up to the tragus of the ear.
Sensory supplies perception of sour, sweet, salty and bitter taste from anterior 2/3 of the tongue. Testing the sense of taste is not routinely performed. If the pt complains of lack of taste, check each side of the anterior two-thirds of the tongue and the posterior one-third separately with normal saline and sugar solution.
Pearls: Slight facial asymmetry without facial weakness is insignificant; similarly, asymmetry that disappears on voluntary expression or smiling is also insignificant. A flat face in Parkinson's disease is not indicative of a facial nerve lesion. Facial nerves pull down the eyelids and close the eyes; CN III, on the other hand, keeps the eyes open. Therefore, ptosis is not because of a facial nerve lesion.
Ddx: Bell palsy, Lyme disease, sarcoidosis, herpes zoster, cerebello-pontine angle tumor, pontine stroke or tumor.
Lower facial and forehead muscle weakness is consistent with lower motor neuron (LMN) weakness:
Isolated unilateral LMN facial weakness: Bell's palsy.
Bilateral LMN weakness: sarcoidosis, Lyme disease neuropathy, GuillainBarré syndrome.
Lower facial muscle weakness with relative preservation of the forehead muscles is consistent with upper motor neuron (UMN) weakness:
UMN weakness plus ipsilateral, appendicular neurological deficit: supratento-rial lesion, for example, stroke or tumor.
UMN weakness plus contralateral appendicular neurological deficit: brainstem lesion, pontine.
Bilateral UMN weakness with dysarthria, dysphagia, inappropriate laughing and crying: corticobulbar tract lesion or pseudobulbar palsy, for example, bilateral strokes.
CN #8 (Acoustic) = hearing, equilibrium. Lesion causes unilateral deafness, vertigo, nystagmus. Note if the pt has any hearing impairment or complains of hearing problems with normal conversation. Rub your fingers together on one side while moving the

fingers noiselessly on the other. Whisper test. Ask the pt to tell you when and on which side they hear the rubbing. If abnormal, proceed with the Weber and Rinne tests.
Rinne's test: done to compare bone conduction (BC) from air conduction (AC). Place the tuning fork (512 Hz) over the mastoid firmly until the vibration noise disappears, and then, place the tuning fork in front of the ear. Normally, the pt should still hear the humming.
Weber's test: Place the tuning fork over the head vertex and ask the pt if he or she hears humming equally in the two ears; normally, this is equal. In patients with conductive deafness, Weber is lateralized to the deaf ear.
Ddx: Vestibular neuronitis, acoustic schwannoma. When you find hearing loss for the first time, formal audiological testing is mandatory.
Conductive hearing loss: BC>AC, and Weber is lateralized to the deaf side. This is commonly seen in middle ear disease or with a buildup of ear wax.
Sensorineural hearing loss (SNHL): AC>BC, but BC>AC in the other ear. This is seen in many conditions such as Ménière's disease, meningitis, cerebellopontine lesions, or brainstem disease.
Vestibular System: Ask the pt whether he or she experiences dizziness and note the presence of nystagmus. If the pt presents with acute vertigo, you want to now whether the vertigo is because of inner ear (vestibulopathy) disease or a brainstem lesion, and then you may proceed with the head-tilt test (Nylen-Barany maneuver or Hallpike's test). See ENT module.
CN #9 (Glossopharyngeal) = motor of pharynx (Gag), taste to posterior 2/3 of tongue. Lesion causes dysphagia, weakness of elevation of palate, loss of gag reflex.
Ddx: Motor neuron disease, jugular foramen tumor, nasopharyngeal carcinoma, metastases to skull base, glossopharyngeal neuralgia.
Examining the Mouth CNs IX, X, and XII:
Tongue: Inspect the tongue for atrophy and fasciculation. Then, ask the pt to protrude the tongue and note any tongue deviation. Ask the pt to protrude the tongue and move it side to side and note for asymmetry of movement. Ask the pt to push the tongue into each cheek, one side at a time.
Note: Small, tremor-like movements of the tongue on protrusion should not be mistaken for fasciculation. Fasciculation occurs when the tongue is resting in the mouth. In patients with unilateral facial muscle weakness, the tongue appears to be deviated. Do not mistake this for CN XII lesion. This can be cor-rected by lifting up the weak facial muscle.
Info: In an LMN lesion, the tongue deviates toward the side of the lesion, and in a UMN lesion, the tongue deviates away from the side of the lesion and is often associated with hemiparesis. Bilateral LMN lesions present with tongue weakness and are often associated with tongue atrophy and fasciculation. Bilateral UMN lesions present with tongue weakness and are often associated with hyperactive gag reflex and labile affect (pseudobulbar palsy).
Tongue deviation: Lesion involving the nucleus of the axons of cranial nerve XII cause the tongue to deviate toward the site of the lesion. If the pt can move the tongue side to side but cannot protrude the tongue, this is because of an upper motor neuron lesion (corticobulbar tract involvement).
Pharynx: Ask the pt to open the mouth and note the position of the uvula spontaneously and/or by saying "ah" and note the movement of the soft palate and uvula. Check the gag reflex when a pt has dysarthria or dysphagia. For the gag reflex, touch the pharyngeal wall with a cotton applicator and note symmetry of palate movements and movements of the uvula. Ask the pt about any differ-ence of sensation between the sides. The afferent arm of the gag reflex is CN IX and efferent is CN X.
INTERPRETATION: Uvula moves to one side: CN X lesion. Absence of gag reflex is seen in LMN lesion. Hyperactive gag reflex is seen in UMN lesions.
Larynx: Ask the pt to cough and drink a glass of water. For direct vocal cord function, ear, nose, and throat consultation is required.
INTERPRETATION: Poor cough is usually seen in vocal cord palsy. Unilateral recurrent laryngeal nerve palsy (branch of vagus: CN X) presents with hoarseness and is commonly seen after thyroid surgery. Dysphagia followed by cough (choking) is seen in CN X palsy. Dysarthria, dysphagia, hoarseness, Horner's syndrome, and contralateral sensory loss with ipsilateral cerebellar dysfunction are indicative of brainstem lesion (lat-eral medullary syndrome).
CN #10 (Vagus) = motor to vocal cords, heart, GI. Lesion causes dysphagia, dysarthria, weakness of elevation of palate. loss of gag reflex. Sensory to bronchus, heart, GI, larynx, ear, visceral parasympathetic, larynx, taste. Listen to the pt's voice (hoarse or nasal), ask pt to swallow, ask pt to **say "Ah"**. Watch the movements of the soft palate and the pharynx. Test gag reflex.
Ddx: Motor neuron disease, jugular foramen tumor, metastases to skull base, nasopharyngeal carcinoma.
CN #11 (Accessory): Lesion causes weakness of sternocleidomastoid and trapezius muscles. Look for atrophy or asymmetry of the trapezius muscles, ask pt to shrug shoulders against resistance or **"touch your ear with your shoulders"**. CN XI, or the spinal accessory nerve, has branches from the medulla and cervical spinal roots of C2–C4, and innervates the sternomastoid and the trapezius muscles. Look at the neck for sternomastoid and trapezius muscle wasting and fas-ciculation or shoulder drops. Ask the pt to shrug the shoulders: Check the resistance against your pushing down on the shoulders, and note any asymmetry. For the sternomastoid, ask the pt to hold his or her neck against your push to the right, and then the left jaw angles (temperomandibular joint). Note the strength and bulk of the sternomas-toid muscles, and observe any asymmetry.
Ddx: Trauma (surgical and other), jugular foramen tumor.
Ipsilateral weakness of the trapezius and sternomastoid muscles is indicative of peripheral CN XI palsy. If this is associated with ninth and tenth nerve palsy, con-sider foramen jugular lesion.
Weakness of the sternomastoid on one side and trapezius on the other side is seen in ipsilateral UMN lesion.
Unilateral inability to shrug is because of a contralateral UMN lesion.
Bilateral weakness and atrophy of sternomastoid muscles is usually seen in myotonic muscular dystrophy, fascioscapulohumeral muscular dystrophy, or motor neuron disease.
Ipsilateral weakness of the trapezius (inability to shrug) and hemiparesis is sug-gestive of internal capsule infarction in the distribution of the anterior cerebral artery.
Unilateral sternomastoid weakness is rare and usually is posttraumatic.
CN #12 (Hypoglossal) = motor to tongue and neck strap muscles. Listen to the articulation of the pt's words, **"stick out your tongue"** and move tongue from side to side.
Ddx: Motor neuron disease, skull base tumor, carotid artery dissection or trauma.

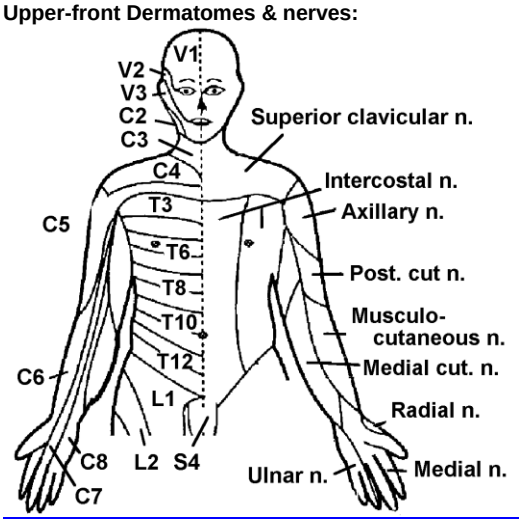
Pearls:

1) The presence of CN dysfunction implies a lesion above the foramen magnum; therefore, a spinal cord lesion does not give rise to symptoms and signs of CN abnormalities.
2) CN abnormalities plus ipsilateral appendicular motor or sensory deficits is consistent with a supranuclear lesion, such as a stroke.
3) CN abnormalities plus contralateral, appendicular, motor, or sensory deficits imply a brainstem lesion, such as a stroke or tumor.
4) Unilateral CNs V–VIII dysfunction plus contralateral appendicular motor or sensory deficit is indicative of a cerebellopontine angle lesion, such as a tumor.
5) Unilateral CNs IX–XI dysfunction without appendicular deficit is seen in foramen jugular lesions, such as a tumor.
6) Unilateral abnormality of CNs III, IV, V, and VI is suggestive of cavernous sinus lesion, such as a thrombosis.
7) Unilateral abnormality of CNs IX, XI, and XII is indicative of bulbar palsy, such as a stroke.
8) Dysfunction of CNs can arise from: Supranuclear lesion. Brainstem lesions. Cranial nerve lesions. Neuromuscular junction. Surrounding structures, such as the cerebellum.
*****Ref:** (Practicing Neurology by Rahman Pourmand, MD. 2008 Humana Press)

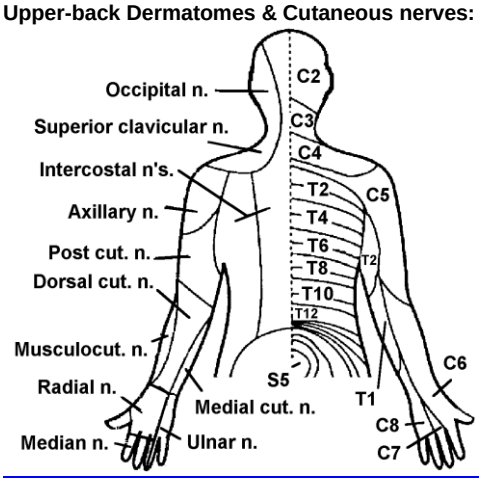
Sensory Exam:

Links: Basics & Initial Exam | **Upper-front Dermatomes & Cutaneous nerves** | Upper-back | **Lower-front** | Lower-back | **Dermatomal Testing & Other Tests** | Neuro Levels | **Sensory Patterns** | Cervical radicular pain | **Lumbosacral Root Compression** | Nerve Injury Grade |

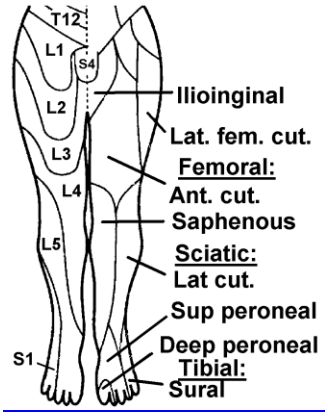
Upper-front Dermatomes & nerves:



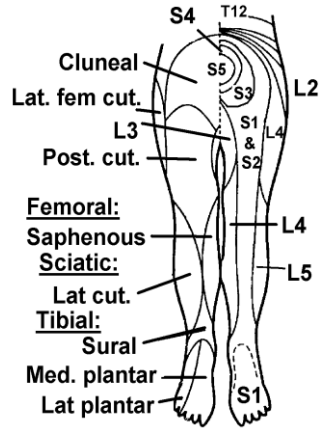
Upper-back Dermatomes & Cutaneous nerves:



Lower-front Dermatomes & Cutaneous nerves:



Lower-back Dermatomes & Cutaneous nerves:



Basics: Least reliable part of the evaluation as it is unfortunately, always subjective and difficult to quantify. Can be challenging and confusing because responses are indirect and represent a pt's interpretation of the test and test questions. It is important that the pt clearly understands the object of the test, and attention and cooperation are imperative. Use the sensory exam to clarify clinical hypotheses posed by the history and other elements of the exam. First have the pt outline the area of loss. The pt's eyes should be closed during the actual testing. Should be done in an organized manner. Start the sensory testing from an abnormal area, moving toward the normal area. You need to establish whether the deficit is in the distribution of one nerve or multiple nerves or root; whether the sensory deficit is symmetric, or asymmetric or distal versus proximal; and, most importantly whether the deficit is segmental (level). Start by doing light touch, vibration, and joint position sense first, because they are less disturbing and you are assessing the pt's reliability. Compare symmetrical areas on the two sides of the body. Also compare distal and proximal areas of the extremities, recognizing better discrimination is found distally, hands more sensitive than feet. Articulate persons can usually discriminate areas/ regions of altered sensation, excepting those with non-dominant parietal lesions (e.g. anosognosia). See **Approach & Type of nerve** |

Sensory Modalities: Pain and temperature (extroceptive). ibration, joint position sense, light touch (proprioceptive). Stereognosis, graphesthesia, two-point discrimination, double-simultaneous stimulation (higher cortical sensory). **Start with:** Dermatomal Testing & Other Tests |

Cape-like hypesthesia: central spinal cord syndrome, syringomyelia. Presents with diminishment or loss of sensation for pinprick and temperature across the upper back, shoulders, and upper arms resulting from lesions that involve spinothalamic neurons as they cross in the anterior commissure in the cervical cord. The Ddx includes tumor, syrinx, and demyelination.

Stocking-glove hypesthesia: peripheral neuropathy. Seen in length dependent axonal neuropathies. Typically due to diabetes mellitus, alcohol, Vit-12 deficiency, syphilis, HIV, Lyme disease, uremia, chemo, vasculitis, paraneoplastic neuropathy or amyloidosis.

Ddx: mononeuritis multiplex, GBS, chronic inflammatory demyelinating polyneuropathy, demyelinating lesion in the cervical cord or cervical spondylosis with cord impingement

Mixed sensory/ opposite motor: partial transection of spinal cord (Brown-Sequard lesion).

Paresthesias: Central: CVA, TIA, tumor, trauma, brain abscess, encephalitis, SLE, B12 def, MS.
Peripheral: neuropathy.

Localization Pearls:

UE: Sensation travels from the thumb and index finger via the C6 nerve root, from the middle finger via the C7 root, and from the fourth and fifth fingers via the C8 root. For the remainder of the upper extremity, the root innervation "fans out" from the hand. These innervation patterns cover both the anterior and posterior aspects of the upper extremity.

LE: The L5 dermatome includes the large toe and the lateral lower leg; the S1 dermatome includes the small toe and sole. Roots L4, L3, and L2 cover the region fanning out medially and proximally from the L5 dermatome. The S2 dermatome extends from the S1 dermatome up the back of the leg.

Dermatomal Testing:

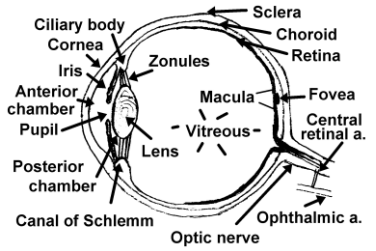
…….END DEMO of this chapter.

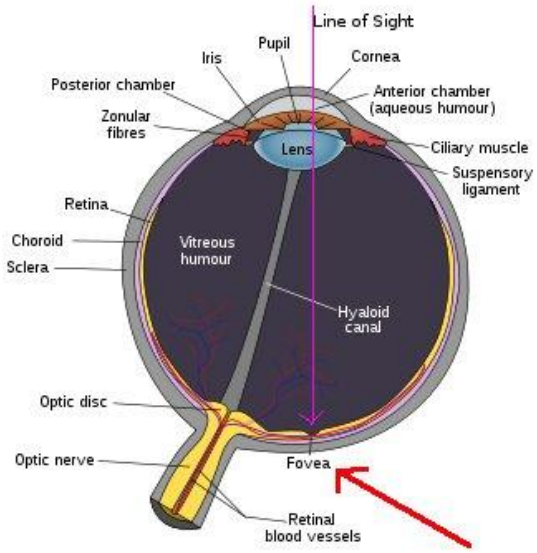
Ophthalmology:

Edited by Steven D. Zelko MD, Ophthalmologist.

Contents: Exam | **Pupils** | Fundus & Internal Eye | **External** | Eyelids | **Various S/s & Abn's** | Trauma | **Dry Eyes** | Red Eye | **Glaucoma** | Impaired Vision | **Cataracts** | Sudden Vision Loss | **Diplopia / Nerve Lesions** | Nystagmus | **MEDS** | Ophth Abbr.'s | **Terminology** | INFECTIONS | **Cornea** | Pediatric | **Pain** | *INDEX* |

Cross Section Diagram:





Despite its relatively small size, the eye is one of the most complex organs of the human body. It is derived from neural crest cells, neuroectoderm, surface ectoderm, and mesoderm. The white tissue of the eye, the sclera, is a rigid connective tissue that encloses intraocular contents except at the anterior and posterior poles, where it meets the cornea and optic nerve respectively. The conjunctiva is a diaphanous tissue that lines the inner aspect of the eyelids (tarsal conjunctiva) and reflects in the fornices to cover the sclera (bulbar conjunctiva), consists of an outer epithelial layer and underlying loose connective tissue. As a result of its rich vascularization, the conjunctiva can demonstrate dramatic injection and edema (chemosis) in response to irritation, allergies, or infection. Goblet cells within the conjunctiva secrete the mucous component of the tear film.

The cornea (more specifically, the air-tear film interface) is responsible for ~ two thirds of the eye's refractive power. Although like the sclera, the cornea is composed primarily of connective tissue (with outer epithelial and inner endothelial layers), it is transparent. This transparency is attributable to the unique characteristic arrangement of type I collagen fibrils in a regular array held in place by a proteoglycan ground substance. When damaged, corneal stroma undergoes further deposition of type I collagen; however, this secondary process occurs without fibrillar organization, and opacification (scarring) of corneal stroma occurs. When the cornea is not clear, this indicates that a pathologic event has occurred or is occurring.

Extraocular muscles attach to the globe underneath the conjunctiva and act as pulleys to effect ocular rotations. Each extraocular muscle is yoked with a muscle in the other eye, causing complementary binocular function. A simplified schematic approach to eyelid anatomy would divide the eyelid into two segments (anterior/external and posterior/internal) separated by a thin fibrous structure, the orbital septum. The anatomic segments external to the septum consist of skin, loose subcutaneous connective tissue in which eyelash follicles originate, and orbicularis oculi muscle. Internal to the septum lie the levator muscle, tarsus, Müller's muscle, and tarsal conjunctiva. Both segments of the eyelid contain sebaceous glands that contribute to the lipid component of the trilayered tear film. Zeis' glands are associated with cilia (eyelashes), and meibomian glands located within tarsus excrete secretions through orifices in the inner eyelid margin. The nasolacrimal system consists of inflow and outflow components. The lacrimal gland, a bilobed exocrine gland located under the temporal upper eyelid, produces aqueous secretions in response to reflex and psychogenic stimuli. Basal aqueous secretions are produced by the eccrine glands of Wolfring and Krause located adjacent to tarsus in the eyelids.The nasolacrimal outflow tract originates at the inferior and superior puncta of the nasal aspect of the eyelids. They open into the superior and inferior canaliculi, which join to form a common canaliculus that opens into the lacrimal sac. The nasolacrimal duct exits the sac inferiorly and ends at the inferior meatus under the inferior turbinate within the nose.

Physiology of Vision:

1. **Optics:** Light travels in a straight line until a new medium is encountered - it may then bend or be **refracted**.
a. The refractive tissues of the eye form a convex surface.
b. The distance at which the bent light converges to a focal point creates three conditions:
- **Emmetropia:** Focal point hits retina.
- **Myopia:** Focal point is in front of retina creating nearsightedness. Corrective lenses or surgery may correct refractive abnormalities.
- **Hypermetropia:** Focal point is behind retina, creating farsightedness.
2. **Accommodation:** Objects closer than six meters (20 ft.) generally have light rays that must be refracted greatly, thus requiring the eye to accommodate or adjust. Involves three major actions:
a. **Lens shape**: Ciliary muscle contraction regulates the shape of the lens. **Presbyopia** occurs when lens loses elasticity with age, which decreases ability to accommodate.
b. **Pupil size:** Pupillary dilator muscles relax while pupillary constrictors contract to eliminate divergent light rays, making refraction easier to accomplish.
c. **Eye convergence:** Eyes turn medially to focus light on the **fovea** or area of greatest visual acuity.
- **Near point:** Minimum distance from eye that object can be focused using accommodation.
- **Far point:** Distance from eye that does not require accommodation; generally six meters (20 ft.)
• 20/20 vision is normal focusing at 20 ft.
• 20/40 is only focusing objects at 20 ft. that a normal eye can focus at 40 ft. • 20/10 is focusing objects that a normal eye could only focus at 10 ft.
3. **Photoreceptors of the retina**
a. **Rods:** Respond to light levels, but not color; low threshold; good in dim light (i.e., night); common in peripheral areas of retina.
b. **Cones:** Respond to light levels and color (red, green, blue); high threshold; good in bright light conditions (e.g., day); common in fovea for visual acuity.

Common Principles about the Eye:

Reading in dim light does not harm the eyes. Excessive use of the eyes does not harm them. Sitting too close to the TV does not harm the eyes. The eyes clean themselves, healthy eyes do not require eyedrops. Action video games heighten contrast sensitivity (the ability to detect subtle differences in shading) — indicating a possible method to improve eyesight in pilots or those with impaired vision (Nat Neurosci 2009;12:549).....the benefit generalizes to different situations, not just playing other video games.
Ophthalmology referral if: blepharitis unresolved after 3wks tx, chemical exposure, ciliary flush, conjunctivitis of unknown etiology or not resolved in 3d of tx, constricted or irregular pupil, fixed pupil, herpetic lesions, ocular pain or photophobia, vision loss, trauma other than superficial abrasion / foreign body or if unsure about dx / tx.

Complete Eye Exam:

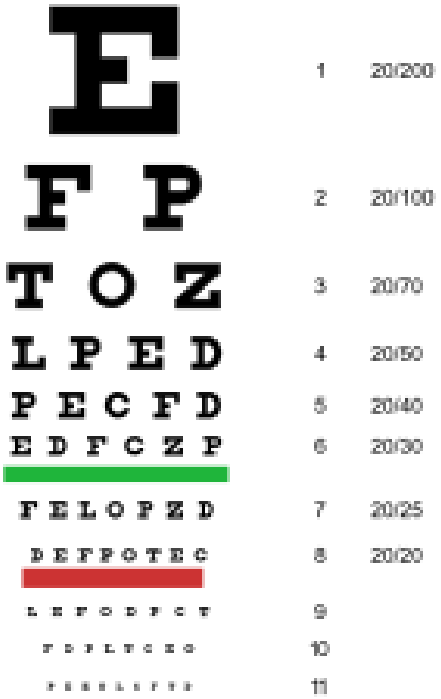
Links: Cross Section Diagram | **Visual Acuity** | External | **Pupils** | EOM | **Anterior Segment & Ophthalmoscopy** | Posterior Segment | **IOP** | Visual Fields | **Malingering** | Color Vision | **Nystagmus** | Exam in Children | **Phorias & Tropias** | The Slit Lamp | **Refraction** | Maddox Rod Test | **Lensometer** | Fundus & Internal Eye | **External Eye** | Cornea | **Eyelid Fb** | Fundus & Internal Eye | **Fluorescein Staining** | Tonometry | **Diplopia / Nerve Lesions** | Ptosis |

8 steps, ~8 minute exam.
1. Visual Acuity (VA). 2. External & Adnexa. 3. Pupils.
4. Extra Ocular Movements (EOM's). 5. Anterior segment.
6. Posterior segment. 7. Intraocular Pressure (IOP). 8. Visual Fields.
CPT: 99173 for screening eye test.

1. Visual Acuity (VA):

The #1 vital sign of the eye. See **Sudden Vision Loss** | Exam in Children |
Check VA in a well-lighted room. Always ask the pt whether he or she wears glasses; if so, test VA while pt wears their glasses (correction). Determine the visual acuity of each eye with the other eye completely covered. Although measurements are obtained at distance and near, with and without refraction (glasses), the most important determination of general eye condition is the best-corrected-distance visual acuity, usually assessed with a Snellen chart. Distant visual acuity (DVA) is usually measured at 20 feet (6 m). The largest letter should be seen at 200 feet (60 m) by a person with normal vision. If a pt can see that letter at a distance of 20 feet and is unable to see any of the smaller letters, vision is 20/200. If the line read by a person with normal sight can be seen at 20 feet, and the pt is 20 feet from the chart, the vision is 20/20. Reported as a pair of numbers (20/20) where the 1st number is how far the pt is from the chart and the 2nd number is the distance from which the "normal" eye can read a line of letters. For example, 20/40 means that at 20 feet the pt can only read letters a "normal" person can read from twice that distance. 20/400 eye at 20 feet sees what a normal eye can see at 400ft. Typically uses the Snellen chart (eye chart), which is a large card or lighted box that displays rows of letters in smaller and smaller sizes. The card is read from a standard distance. The degree of visual acuity is determined by the size of the row of letters that the person can read. For those who are unable to read, a modified chart can be used in which the letters are represented by an upper case "E," which is rotated randomly. The person is asked to describe the way the letter is facing. Examination of children can also be done with pictures.
A corrected acuity of <20/30 is abnormal. If unable to read the top line can count fingers (CF), hand motion (HM), light perception (PL), color. Wiggling a finger, raise one or two fingers (unilaterally or bilaterally) and have the pt state how many fingers (total, both sides) they see. To test for **neglect**, on some trials wiggle your right and left fingers simultaneously. If assessment of distant visual acuity is not possible, near acuity should be tested with a reduced Snellen chart or standardized reading test types. The pt must be wearing an appropriate reading correction. Vision is not a true fraction. In other words, 20/40 vision does not mean 50% of normal vision but that the pt can see at 20 feet what a person with normal sight can see at 40 feet (12 m). If the pt can see the 20/20 line wearing glasses, his or her functional vision is probably just as good as that of someone who sees the line without glasses.

Pinhole: Punch 5 holes in a 2-3 cm D circle in the middle of a 3'x5' card with 18g needle. If refractive error **-->** This improves vision. If organic dz, then the vision will not improve. Pinhole only corrects about 3 diopters of refractive error, may not improve with pinhole due to macular disease or hand tremor. **Driving:** must have at least 20/40 in the better eye. See **Visual Impairment** **|**



2. External & Adnexa exam:

The examination of the eyes begins by sitting or standing in front of the pt. Hold penlight 12-14 inches, observe for asymmetry of the eye globe, redness, scaling, swelling, discharge (brows/ lids/ conjunctiva, orbit). Calcium deposits in the sclera are common in the elderly and manifest as hyperpigmentation.

Arcus Senilis = a narrow, white band of lipid infiltration separated from the limbus by a clear zone that occurs in most >80yo and those with elevated triglycerides <40yo.

Links: Foreign Body (see below) | External | **Eyelids** **|** Cornea | **Eye irrigation** **|** Fluorescein Staining | **Eye Patching** **|** Lid Laceration | **Other Findings / Abn's** **|** Iris Color |

Eye / Conjunctival Foreign Body:

Everting the eyelids is critical in the evaluation of possible "lost" contact lens or other objects such as sand particles. Many low-velocity projectiles, like wind blown dust particles, can be loose in the tear film or lodged in a conjunctival sac. Likely superficial if something "fell" or "blew" into the eye, but at risk for a globe perforation if pt sustained a high-velocity wound to the eye (drilling or grinding metal, blasting rock). Always consider a **Ruptured Globe.** Also see Orbital Fb |

Equipment: Topical anesthetic. Sterile cotton-tipped applicators. Fluorescein strips. Magnification with loupes plus a Wood lamp or a slit lamp. Eye spud or 25-ga needle attached to a 1- or 3-mL syringe or to the tip of a cotton-tipped applicator. Dilator drops, such as homatropine 5% or cyclopentolate 1%. Antibiotic ointment, such as erythromycin. Eye patches. Tape (nonallergenic paper tape is preferable).

Step #1: A drop of both topical anesthetic.

Step #2: Inner Lid exam: Have the pt gently (not tightly) close their eyes and look up. To examine the lower sac, pull the lower lid down with your finger while the pt looks up.

Step #3: To examine the upper sac, hold the proximal portion of the upper lid down with a cotton-tipped swab while pulling the lid and forward (out) and up by its lashes, **everting upper lid**, as the pt looks down. Using your nondominant hands, grasp the upper eyelashes and gently pull the lid inferiorly, make an inferior and posterior movement with the swab to everting the eyelid. Push the cotton swab downward to help turn the upper conjunctival sac "inside out." The stiff tarsal plate usually keeps the upper lid everted after the swab is removed, and as long as the pt continues looking down. The cotton swab acts as a fulcrum at the upper border of the tarsus (~10mm from the edge of the lid) over which the lid can be everted. With simple lid eversion it is still not possible, sweep the anesthetized fornix with a wetted applicator as the upper lid.

Step #4: Looking up reduces the lid to its usual position. A loose foreign body usually adheres to a swab lightly touched to the surface of the conjunctiva, or will be washed out by copious irrigation with saline. The Fb can then be removed with the moistened cotton swab or sterile saline wash.

Embedded corneal Fb: best removed with a commercial "spud" device or a short 25- or 27-ga needle on a small-diameter syringe (insulin or "TB" syringe) or a cotton-tipped applicator. The applicator or the syringe serves as a handle for the attached needle. The pt should be positioned such that the head is well secured (preferably in a slit lamp frame). When removing a foreign body, it is useful to have an assistant present to help the pt keep the head properly positioned and the eye opened. The pt must be instructed to gaze at an object in the distance (the practitioner's ear when a slit lamp is used) to stabilize the eye further. The device is held

tangentially to the globe, and the foreign object is picked or scooped out. During removal, the physician should brace his or her hand against the pt's face. It also may be helpful to brace the elbow with a pad or half-full tissue box to provide further support to the arm as the foreign body is removed.
Step #5: Next fluorescein is applied to the inside of the lower lid. See **Fluorescein Staining** |. First add a drop of anesthetic to the eye or the strip itself). View the eye with a cobalt blue lens or Wood's lamp looking for a corneal abrasion that will appear as a greenish-yellow fluorescent defect in the epithelium (corneal abrasion caused by the foreign body). On exam, normally occurring white papules inside lids can be mistaken for foreign bodies, and transparent foreign bodies can be invisible in the tear film (until outlined by fluorescein dye). Examine the anterior chamber and tear film with a bright light (best done with a slit lamp) and examine the conjunctival sacs. Do not overlook an eyelash that has turned in and is rubbing on the surface of the eye. Sometimes you may see a lash sticking out of the inferior lacrimal punctum.
Aftercare: An Abx ointment is applied. The most frequent problem is incomplete removal of the foreign body. In such cases the epithelium has difficulty healing over the affected area, and thus the eye stays inflamed. Eventually, the diseased epithelium either sloughs off and heals or heals over the foreign body remnants, which are gradually absorbed. In either case, the adverse effects on the eye are minimal; a minute scar on the cornea, even directly in the center, will rarely affect the vision. Nonetheless, incomplete removal of a corneal foreign object warrants ophthalmologic follow-up.
Conjunctivitis may develop after removal of an extraocular foreign body.
Pearls: Glass embedded in the cornea may be particularly difficult to detect.
Rust Rings: a common problem with metallic foreign bodies is rust rings. These can develop within hours because of oxidation of the iron in the foreign body. There are 2 preferred techniques for removal of a rust ring. The most direct method is to remove it at the same time as the foreign body, either with repeated picking away with a spud device or with a rotating burr. The second approach is to let the iron of the rust ring poison and kill the surrounding epithelial cells during a 24- to 48-hour patching period. At that time, the rust ring will be soft and often comes out in one solid plug. After rust ring removal, the eye must be patched again for another 24 hours to allow the residual corneal defect time to heal. Generally, a small rust ring produces little visual difficulty unless it is directly in the line of sight. The rust ring, if large, may delay corneal healing.
Multiple Foreign Bodies: should be referred to an ophthalmologist as often need to denude the entire epithelium with alcohol and remove the superficial foreign bodies. The deeper ones gradually work their way to the surface, sometimes years later.

Fluorescein Staining:

A key part of the evaluation of all cases of eye trauma and infection. It is a quick and easy technique that is crucial for the proper dx and management of common eye problems. The fluorescein-stained cornea and conjunctiva should be viewed under a "blue" light and ideally in conjunction with slit lamp magnification.
PP: Sodium fluorescein is a water-soluble chemical that fluoresces--it absorbs light in the blue wavelengths and emits the energy in the longer green wavelengths. It fluoresces in an alkaline environment (such as in the Bowman membrane, which is located below the corneal epithelium) but not in an acidic environment (such as in the tear film over an intact corneal epithelium). Thus, it is useful in revealing even minute abrasions on the cornea. To prevent contamination by bacteria, paper strips are impregnated with fluorescein.
Indications: all suspected abrasions, foreign bodies, and infections of the eye. This may include "simple" cases of conjunctivitis, which may actually be herpetic keratitis.
Contra: Fluorescein may permanently stain some soft contact lenses, do not reinsert contact lenses for 4-6 hour to avoid staining. Vagal reactions can occur similar to examining the ear. The dye may enter the anterior chamber of the eye in the presence of deep corneal defects, but this intraocular fluorescein accumulation is generally nontoxic…see a "fluorescein flare" on slit-lamp evaluation.
Step #1: Put a drop of topical anesthetic onto end of the paper strip. Ideally one should not use topical anesthetic because some pt's will develop a superficial punctate keratitis from the anesthetic, which can confuse the dx. However are uncomfortable and the exam would be nearly impossible if a topical anesthetic is not first used.
Step #2: The wetted strip is then placed gently onto the inside of the pt's lower lid. The strip is withdrawn, and the pt is instructed to blink. The pt's blinking spreads the fluorescein over the eye. The key to a good examination is a thin layer of fluorescein over the corneal and conjunctival surfaces. If the strip is heavily wetted before application in the lower fornix, the eye may become flooded with the solution, thus making evaluation difficult. On the other hand, use of a dry strip in the unanesthetized eye may be irritating. If too much dye accumulates, the pt can remove the excess by blotting the closed eye with a tissue.
Step #3: use a Wood lamp, the blue filter of a slit lamp, or simply a penlight with a Cobalt blue light filter to examine the eye in a darkened room. Check for areas of bright apple green fluorescence on the corneal and conjunctival surfaces. Record size and location of abrasion. Small defects may not be seen with the naked eye; ideally, a slit lamp should be used to examine the stained cornea before ruling out a pathologic process. After completion of the fluorescein examination, excess dye should be irrigated from the eye to minimize damage to the pt's clothing from dye-stained tears.
Interpretation: conjunctival abrasions pick up the stain, most of the staining on the conjunctiva represents patches of mucus rather than a real pathologic condition. Corneal staining is more specific for injury, and the pattern of injury often reflects the original insult.
Abrasions: usually occur in the central cornea because of the limited protection of the pt's closing eyelids. The margins of the abrasions are usually sharp and linear if seen in the first 24 hours.
Foreign bodies: Circular defects are seen about embedded foreign bodies and may persist for up to 48 hours after removal of a superficial foreign object. Deeply embedded objects may be associated with defects persisting for longer than 48 hours. Objects under the upper lid (including some chalazions) often produce vertical linear lesions on the upper surface of the cornea. When vertical lesions are noted, a diligent search for a retained foreign body under the upper lid should be made.
Hard contact lens overuse: this diminishes the nutrient supply to the cornea. The central cornea receives the most injury and thus fluoresces brightly when stained.
Herpetic lesions: may develop anywhere on the cornea. Classically, these lesions are dendritic, although ulcers may also be punctate or stellate.
Radiation Injury / Ultraviolet Keratitis: From UV light exposure, snow blindness, or welding flash. Fluorescein staining may be negative or show diffuse superficial punctate staining pattern (corneal haziness) (discerned as a punctate keratopathy = SPK, under slit lamp exam). See diffuse corneal punctate staining with fluorescein slit lamp exam. The central cornea is the least protected by the lids, and a central horizontal band-like keratitis can result.

Superficial Punctate Keratitis (SPK): With fluorescein stain see multiple small pinpoint corneal epithelial defects (punctate lesions).
Infectious Keratitis: Any area of corneal staining with an infiltrate or opacification beneath or around the lesion should alert the practitioner to the possibility of a viral, bacterial, or fungal keratitis. Urgent ophthalmologic consultation should be obtained so that cultures of the possible etiologic agents can be procured and tx initiated.
Pearls: The presence of fluorescence before the instillation of fluorescein in the red eye should suggest the possibility of a pseudomonal infection. Topical fluorescein can interfere with immunofluorescence tests for chlamydial culture of conjunctiva. Fluorescein angiography is a diagnostic test using IV injected fluorescein to highlight vascular abnormalities in the eye, most commonly in the fundus.

Eye Patching:

A simple patch may be used to protect the dilated eye from bright light. Simple corneal abrasions do not require patching to aid healing or for reduction of pain.
Pressure patching as discussed in this section is the use of a patch to hold the eyelid closed to facilitate healing of a corneal defect, by limiting eyelid movement over an injured area.
Indications: whenever the surface of the cornea has been injured. This can occur after a significant mechanical abrasion. Chemical damage, damage from prolonged hard contact lens use, and ultraviolet light injuries.
Contra: when the corneal epithelial loss results from an active infection--such as a corneal ulcer--rather than from an abrasion. Abrasions associated with soft contact lens use should be considered infected and patching avoided until ophthalmologic evaluation to avoid Pseudomonas keratitis. A protective cup is the preferred covering in such cases pending ophthalmologic consultation.
Equipment: Gauze eye patches (2). Tape (1-in. paper tape, preferably nonallergenic). Abx ointment (sulfacetamide 10%). Dilator eyedrops (cyclopentolate [Cyclogyl] 1% or homatropine 5%). The tape should be precut into 4-in. strips and should be kept within reach.
Step #1: apply both a dilator drop and an antibiotic ointment. The dilator must be a cycloplegic to relax the ciliary muscle spasm that accompanies corneal abrasions. Both cyclopentolate 1% and homatropine 5% last ~ 24 hours. The pt should be checked for a narrow anterior chamber before the drop is instilled. Abx ointment is used prophylactically, although it is rare for abrasions to become infected.
Step #2: have the pt shut both eyes and should remind him or her to keep them shut throughout the entire procedure. Two patches are used. The vertically positioned first patch is doubly folded and placed over the closed lids. The unfolded second patch is then put horizontally over the first. An effective patch must be put on tightly enough to keep the lids shut. The tape strips are stretched diagonally from the center of the forehead to the cheekbone. The physician can pull up the skin of the cheek; when he or she lets go, the tape will be even tighter. If the tape completely covers the patch, slippage of the patch and resultant eye movement are avoided . The tape should not extend onto the angle of the mandible, because mastication loosens the tape in such a situation. Can paint the skin with tincture of benzoin to help secure the tape.
Step #3: F/u: Do not leave a pressure patch on for >24 hours. If repatching needed, a clean patch should be applied to avoid infection. Some corneal defects are extensive and may require 3 to 5 days for healing…re-eval for ulcer formation. Document the size of the corneal defect at each visit. If healing does not occur in a progressive fashion, ophthalmologic consultation should be obtained.
Commercial Products: An adjustable elastic strap pressure patch (e.g., Presspatch II, Precision Therapeutics, Inc, Las Vegas) may be useful in this setting. The Donaldson Eyepatch (Keeler Instruments, Inc, Broomall, Pa) is a commercial product used for enforced eyelid closure that avoids the bulkiness of pressure patching. The device has 2 components: one adheres to the upper lid, and the other is a circle that adheres to the ipsilateral cheek. A tab on the upper component connects by Velcro to the lower component. This patch may be advantageous in some settings, but its design may encourage some individuals to release the lid closure prematurely.
Pearls: Because of conjugate eye movement, patching only one eye does not totally immobilize the globe. When all movement must be stopped, bilateral eye patches should be applied. The use of bilateral patches may not be accepted by all pt's, and one must seek both pt understanding and family support for pt home activities if dual eye patching is to be successful.
Complications: if patch the pt's lashes in between the lids they abrade the cornea….occurs if the pt partially opens the eye during the procedure. The pt should be instructed to rest the uninjured eye. Reading should be discouraged, because involuntary movement of the patched eye will result. Watching television from a distance of 10 feet or more promotes eye fixation and is acceptable.

3. Pupils: Link: Pupils |

4. Extra Ocular Movements (EOM's):

Links: EOM Diagram | **Relex Eye Movement Tests (Dolls Eye & Calorics) |** Nystagmus |
Basically tested by checking to see if the two eyes correctly aligned. Stay about one arm's length from the pt to avoid strain on convergence. Ask the pt to follow your finger or penlight with the eyes, without mov-ing the head. You may hold the pt's chin gently with the other hand. Move your finger slowly upward, downward, to the left, and then to the right. A misalignment of the visual axes under binocular viewing conditions is known as a manifest deviation, or tropia. Use a penlight held 16 inches, the light reflection should be centered in both pupils. Have pt follow light to extreme ends of an "X". Look for smooth pursuit. Get jerky (saccadic) if frontal or cerebellar dysfunction. Upgaze is usually limited in the elderly.
An eye movement abnormality in which the two eyes move conjugately but have limited movement in one direction is called a gaze palsy. It is due to malfunction of one of the "gaze centers" (cortical and brainstem regions responsible for conjugate gaze) or to interruption of the pathways leading from them. When the lesion is in a brainstem gaze center, the neurons there cannot be activated either voluntarily or by reflex (such as the oculocephalic reflex or "doll's eyes response"). This is called a nuclear gaze palsy. When the lesion is in a cortical gaze center, only voluntary gaze is impaired; reflexes can still activate the brainstem neurons responsible for gaze. This is called a supranuclear gaze palsy.
Nystagmus: a rhythmic oscillation of the eyes, can be vertical, horizontal or rotary, named for fast component. Horizontal nystagmus is described as being either "leftward" or "rightward" based on the direction of the **fast** component. Normal individuals have a moderate horizontal nystagmus at end-gaze laterally. **Link:** Nystagmus |

Other: A routine check for saccadic vs. pursirt vergence is necessary. In the unconscious pt and in the pt suspected of having supranuclear palsy or ocular myopathy (fixed ophthalmoplegia), EOMs can be assessed by oculocephalic ("doll's eyes") or oculovestibular (caloric) reflexes (see below).

Eye Movement Abnormalities:

1. From a lesion affecting supranuclear gaze centers, which includes the frontal lobes, occipital lobes, and cerebellovestibular pathway: stroke, progressive supranuclear palsy, Parkinson's disease.
2. From a brainstem lesion affecting CNs III, IV, and VI nuclei: stroke, brainstem glioma.
3. From a brainstem lesion affecting internuclear (CNs III, IV, and VI) connections by involving the medial longitudinal fasciculus, known as internuclear ophthal-moplegia: multiple sclerosis, stroke, brainstem glioma.
4. From a lesion affecting cranial nerves:compressive CN III palsy,diabetic ophthalmoplegia, ophthalmoplegic migraine.
5. From a neuromuscular disorder: ocular myasthenia gravis.
6. From primary eye muscle disease: myotonic muscular dystrophy, oculopharyngeal muscular dystrophy, mitochondrial myopathies.

Pearls: Diplopia is the prime symptom of many acquired EOM disorders. Supranuclear and internuclear lesions, and primary ocular myopathies, however, usually do not manifest as diplopia. Diplopia is maximum in the direction of paretic muscle. Horizontal diplopia is a result of CN III or VI nerve dysfunction and vertical is a result of CNs III and IV. Isolated cranial nerve palsy could be because of diabetes, migraine, vasculitis, neuritis, aneurysm, or trauma. Bilateral internuclear ophthalmoplegia is because of a medial longitudinal fasciculus lesion. Consider multiple sclerosis or pontine glioma in a young pt and brainstem stroke in the elderly.

Horizontal gaze palsy: The pt looks away from the paralyzed side but is looking toward the site of lesion. Gaze palsy is correctable by the doll's eye maneuver or caloric reflex. This is commonly seen in hemisphere stroke.

Disconjugate gaze palsy: seen in a brainstem (pontine) lesion, and the eye does not move by the doll's eye maneuver.

Upward or downward gaze palsy: usually a result of a pontomesencephalic lesion.

Supranuclear palsy: seen in progressive supranuclear palsy and Parkinson's disease. The eyes move with the caloric test (eyes move by reflex induction and not voluntarily).

Reflex Eye Movements: Dolls eyes **and** Caloric testing.

Caloric Testing: = **Oculovestibular reflex**. Involves inhibition of the functio of one of the vestibular canals by infusing ice water against the tympanic membrane. Have HOB @ 30 deg, irrigate the canal with ~60ml of ice water. A comatose pt with an intact brainstem will have tonic conjugate eye deviation to the same side as the cold stimulus, they will not have a fast (cortical) component. Calorics are not helpful with drug induced comas. "Cold opposite, warm same". First r/o TM perforation before performing.

Dolls Eyes: = Oculocephalic reflex. Passive movement of the head stimulates reflex eye movement (reflects the integrity of the pons). The eye movements are due to stimulation of the semicircular canals. Normally in the awake pt the eyes will move with the head, unless the movement in **slow** and the pt is fixating on an object, you will see a conjugate movement. In a comatose pt you will see also see a +Dolls eye (conjugate eye movement) if there is an intact brainstem. As the head is passively turned to R, eyes will deviate to L (keep facing original direction) = +, a good prognosis (preserved brain stem despite lack of cortical control). Do not perform unless the c-spine has been cleared. In a metabolic coma, dolls eyes may be lost or retained. The lack of inducible eye movement in the setting of preserved pupillary reactivity is virtually diagnostic of drug toxicity. (Arch Neurology 1989;46:561-3)

Other eye movements in coma:

Ocular bobbing --> repetitive rapid vertical deviations inferiorly with a slow return to midline is seen with pontine lesions.

Windshield wiper eye --> random, nonlocalizing movements seen with CN 3 nucleus lesions.

Ping-Pong gaze --> side-to-side deviation every 35 sec seen with bilateral cerebral dysfunction.

5. Anterior segment:

Link: **Cornea** | Fundus & Internal Eye | **Fluorescein Staining** |

The anterior segment is made up of the conjunctiva/ sclera/ cornea/ iris/ lens/ ant chamber. Look for clarity & transparency of cornea, observe ant chamber depth (for predisposition of < closure glaucoma) with light held temporally (normal < light will illuminate entire iris, get a crescent shape shadow if narrow angle).

Leukocoria: alteration in the pupillary light reflex causing the pupil to appear white. May be due to corneal opacity, blood (hyphema), or other material in the anterior chamber, cataract, vitreous opacity or retinal dz such as retinoblastoma.

Blue sclerae: osteogenesis imperfecta, Marfan's, Ehlers-Danlos syndrome, pseudoxanthoma elasticum.

6. Posterior segment:

Ophthalmoscopy:

***Direct ophthalmoscope*:** a handheld device like a small flashlight with magnifying lenses that shines a light into the eye to examine the cornea, lens, and retina.

Three Lenses: Your Lens. Diopter Wheel. Pt's Lens. A mirror reflects light onto the retina. The diopter wheel focuses the image.

Three Controls: The Diopter Wheel -- Outside Edge. The Aperture Control -- On Front. The Brightness Control -- On Handle.

Step #1: Darken the room as much as possible. Adjust the ophthalmoscope so that the light is no brighter than necessary. Adjust the aperture to a plain white circle. Set the diopter dial to zero.

Step #2: The pt looks straight ahead (ask them to pick a spot in the distance and stay focused on it) as the beam of light is shone into the eye. Dilating the pupil, which allows a better view. It is useful to detect the changes that occur in the retinal blood vessels in people with high blood pressure, arteriosclerosis, and diabetes mellitus. It can also be used to diagnose elevated pressure within the brain, which results in a swelling (pushing-out) of the normally cupped optic disk (papilledema). Tumors on the retina can be seen with ophthalmoscopy. With direct ophthalmoscope check for **red reflex** (spoke cataract seen in DM, post-subcapsular seen in steroid intake & DM). Best to check in a well-darkened room, if still poor, helpful to dilate the eye, may be hard to see with pigmented fundi. In child, if reflex is still not bright or symmetric, need urgent referral.

Indirect ophthalmoscope: a binocular device is placed on the doctor's head and a handheld lens is used in front of the person's eye to focus the image inside the eye. This method gives a three-dimensional view, allowing a better view of objects that have depth, including a detached retina or a swollen optic disk. It also allows a brighter light source to be used, which is important if the interior of the eye is cloudy, for instance due to infection or cataract. The indirect ophthalmoscope also allows a much wider field of view than a regular ophthalmoscope, so that the doctor can examine more of the retina at once.

Optic cup: Normal 0.2-.3 optic cup **-->** disc ratio, if asymmetric or >0.5 r/o glaucoma. There is usually a pinkish color of healthy neural tissue surrounding the cup. If see a band of whitish wispy tissue = **peripapillary atrophy**, often seen in normal aging, consider syphilis, ischemia, toxic.
Vitreous: makes up 80% of the volume of the ocular globe. Mostly water and soluble proteins interwoven by a network of collagen fibrils. Minimally scatters light and is acellular except for a small number of modified macrophages called hyalocytes. Acellular debris from the embryonic hyaloid vascular system are responsible for most floaters.
Fundus & Internal Eye:
Retinal blood vessels: normal A-V ratio of 2:3. (smaller arteriole diameter seen in HTN retinopathy) Arteriole is brighter red and has a ribbon of light reflex along the top of the vessel (increases in sclerosis of arteriole).
Macula: yellow-red area 2 disc diameters temporally from the nerve.

7. Intraocular Pressure (IOP):

Tono-Pen: pen device that is easy to use.
Step #1: touch the button to turn it on.
#2: press button 2X to calibrate (need to rotate upside down as calibrate). Should say "good" in LED readout.
#3: Anesthetize the eye. Lightly touch cornea until hear beep and get reading. Should have <5 error.
Schiotz tonometer is difficult to use as many are not properly oiled. In a pinch can attempt the
Finger-tension (tactile pressure) technique: place index fingers on the upper closes lids of pt as pt looks down. Slightly indent globe for 20 sec with the R index finger, then locate the sclera bulge with the L index finger. IOP< 20mmHg if able to feel the bulge. This is only recommended for experts.

8. Visual Fields: See Loss of Field |

The visual field is the entire area of vision that one sees out of each eye, including the corners (peripheral vision). The field of vision is that portion of space in which objects are visible at the same moment during steady fixation of gaze in one direction. The monocular visual field consists of central vision, which includes the inner 30 degrees of vision and central fixation, and the peripheral visual field, which extends 100 degrees laterally, 60 degrees medially, 60 degrees upward, and 75 degrees downward. A normal visual field is an island of vision measuring 90 degrees temporally to central Fixation, 50 degrees superiorly and nasally, and 60 degrees inferiorly.
Confrontation field testing: There are different confrontation techniques when doing a visual field (VF) evaluation. For outer visual fields, most use the **simultaneous finger counting technique:** pt to cover one eye and look directly at you. Sit about one arm length from the pt, and make sure the pt fixates the eyes. It is best to check each eye separately. The person must fix his vision on the doctor's face. Face the person and gradually move a finger from the left and the right at face level in toward the center of vision. The person tells the doctor when the moving finger is first detected. Examiner hold fingers of both hands up in an imaginary tic-tac-toe grid for the pt to count (examiner is the base-line for comparison). Helps to place metal end of reflex hammer on the tip of your nose to inc pt's concentration. This bedside technique only picks up large defects. If the pt is unable to count fingers, then hand motions, and light perception can be tested in each quadrants. Confrontation fields are an essential part of the eye exam. It is not uncommon for pt's to mistake homonymous visual loss for ipsilateral monocular visual loss. Can challenge the pt as raise one finger with your left hand and two fingers with your right hand; then hold up two fingers with your left hand and one on the right. If the pt first sees only one finger and then in the second part of the test sees only the hand with two fingers, you may suspect a nasal field defect of the right eye.
Hold both hands in the hemifield under suspicion (in this case, the nasal field of the right eye) and flash the fingers above and below the horizontal meridian, thereby testing the upper and lower portions of the affected field of vision.
Automated Testing: commonly use a hemisphere perimeter (Humphry's) test, which evaluates the central and peripheral visual fields **-->** pt stares at a center dot, a computer flashes spots in the pt's visual field and the pt pushes a button if they see it. The visual field may be measured more precisely with a "tangent screen" or a Goldmann perimeter. With these tests, the person stares at the center of a black screen or a hollow, white, spherical device (which resembles a small satellite dish). An object or a light is moved slowly from the periphery toward the center of vision from many different directions. The person indicates when he first sees the light out of the corner of his eye.
Amsler grids are the easiest method for home monitoring or for the detecting central field abnormalities (get metamorphopsia or waviness of lines) due to macular disease as the will start to lose the central 10-15 deg of visual field. It is a hand-held black cross-hatched card to test the central 20 deg of vision. Looks for zones of distortion (Retinal dz). Amsler testing is performed monocularly with the near correction in place, if applicable. The grid consists of a black card covered with a white grid and with a white dot in its center. Looking through one eye, the person notes any distortion in the lines of the grid, while staring at the white dot. If a person cannot see an area of the grid, an abnormal blind spot may exist. (There is a normal but very small blind spot in the area where the optic nerve leaves the eye; however, people are not aware of it.) Wavy lines suggest a possible problem with the macula.
Blindspot: is represented on a visual field chart by an absolute scotoma and corresponds anatomically to the scleral canal through which the retinal nerve fibers leave the eye at the optic disk.
VF defect Pearls: If VF defect is limited to one eye, consider ocular, retinal, optic nerve lesion, or conversion disroder. If bilateral, the lesion is at or behind the chiasm or bilateral anterior to chiasm. Tunnel vision is generally indicative of nonorganic disease. Bitemporal hemianopia is caused by a chiasmatic lesion. Homonymous quadrantopia is caused by a temporoparietal lobe lesion. Homonymous hemianopia when incongruousis indicative of an optic tract lesion; when it is congruous, it is suggestive of a lesion behind the lateral geniculate body; and when there is macular sparing, this is suggestive of an occipital cortex lesion.
Optic Chiasm Lesion: produces bitemporal hemianopia. Lesions of the optic tract produce homonymous hemianopia; specifically, incongruous homonymous hemianopia. Incongruity refers to asymmetry of the visual field defects. Within the optic tract the crossing nasal fibers and the uncrossed temporal fibers are relatively separated anatomically. Hence, corresponding points of the visual field from each eye are not closely aligned. Visual field loss from lesions of the optic tract or the lateral geniculate body affect each eye differently, resulting in asymmetric field loss in each eye.
Optic Tract Lesion: These fibers synapse at the lateral geniculate body and project backward as the geniculo-calcarine radiations. All the retrogeniculate fibers sweep laterally and inferiorly around the temporal horn of the lateral ventricle. The most anterior-inferior fibers form Meyer's loop, which contains projections of the inferior retinal fibers. Hence, lesions of Meyer's loop, located primarily in

the temporal lobe, produce congruous, superior homonymous quadrantanopias; more superiorly located parietal lobe lesions produce the inverse defects, inferior homonymous hemianopias or quadrantanopias.
Visual Cortex Lesion: occupies the medial and posterolateral surfaces of the occipital lobe. Striate cortex can be found above, below, and even within the walls and floor of the calcarine fissure itself. A lesion here will produce a homonymous, paracentral hemianopic scotoma. Such defects involve the central 5 to 10 degrees of vision and may spare the remainder of visual field if the remainder of the striate cortex is spared. The opposite-type field defect, a homonymous hemianopia "with macular sparing," occurs with occipital lesions that spare the posterolateral striate cortex.

9. Malingering:

Pinhole testing --> Punch 5 holes in a 2-3 cm D circle in the middle of a 3'x5' card with 18g needle. If refractive error **-->** This improves vision. If organic dz, then the vision will not improve.
Pt's Claiming No Light Perception Determine whether each pupil reacts to light: When one eye has no light perception, its pupil will not react to light. The pupil should not appear dilated unless the pt has bilateral lack of light perception or third nerve involvement.
Pt's Claiming Hand-Motion to No Light Perception:
Test for an afferent pupillary defect: A defect should be present in unilateral visual loss to this degree. If not, the dx of nonphysiologic visual loss is made.
Mirror test: If the pt claims unilateral visual loss, cover the better-seeing eye with a patch; otherwise leave both uncovered. Ask the pt to hold eyes still and slowly tilt a large mirror from side to side in front of the eyes, holding it beyond the pt's range of hand-motion vision. If the eyes move, the pt can see better than hand motion.
Optokinetic test: Patch the uninvolved eye when unilateral visual loss is claimed. Ask the pt to look straight ahead, and slowly move an optokinetic tape in front of the eyes (or rotate an optokinetic drum). If nystagmus can be elicited, vision is better than hand motion. Or, can pass a tape measure or newspaper in front of and instruct pt to look at. It proves that near vision is intact. If presents, strongly suggests a psychogenic coma.
Worth four-dot test: Place red–green glasses on pt, and quickly turn on four-dot pattern and ask pt how many dots are seen. If pt closes one eye (cheating), try reversing the glasses and repeating test. If all four dots are seen, vision is better than hand-motion.
Pt's Claiming 20/40 to 20/400 Vision:
Visual acuity testing: Start with the 20/10 line and ask the pt to read it. When the pt claims incompetence, look amazed and then offer reassurance. Inform the pt you will go to a larger line and show the 20/15 line. Again, force the pt to work to see this line. Slowly proceed up the chart, asking the pt to read each line as you pass it (including the three or four 20/20 lines). Make the pt feel incompetent. By the time the 20/30 or 20/40 lines are reached, the pt may in fact read one or two letters correctly. The visual acuity can then be recorded.
Fog test: For example, in a pt feigning visual loss on the right. If pt wears glasses, dial pt's correction into phoropter, if not dial in plano. Add +4.00 to the left. Put pt in phoropter with both eyes open. Tell pt to use both eyes to read each line, starting at the 20/15 line and working up the chart slowly, as described previously. Record visual acuity (this should be visual acuity of the right eye). Close phoropter over the right eye. Ask pt to read the same line as before; work up the chart until pt is able to read line. Record VA.
Retest visual acuity in the supposedly "poorly seeing eye" at 10 feet from the chart: Vision should be twice as good (e.g., a pt with 20/100 vision at 20 feet should read 20/50 at 10 feet). If it is better than expected, record the better vision. If it worse, the pt has nonphysiologic visual loss.
Test near vision: If normal near vision can be documented, nonphysiologic visual loss or myopia has been documented.
Visual field testing: Goldmann visual field tests often reveal inconsistent responses and nonphysiologic field losses.
Tx: Pt's are usually told that no ocular abnormality can be found that accounts for their decreased vision. Hysterical pt's often benefit from being told that everything is going to be all right and that their vision can be expected to return to normal by their next visit. Psychiatric referral is sometimes indicated. If it's a child, tell them that there is an eye abnormality, but the strong drops about to be administered will cure it. Dilate the child's eyes (e.g., tropicamide, 1%), and retest the visual acuity in 40 minutes. Children, as well as adults, sometimes need a "way out."

Color Vision:

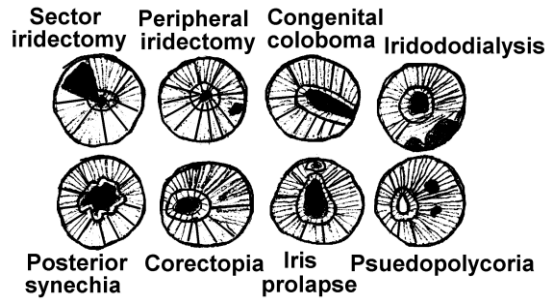
Links: Loss | **Increased** | Blue Tinge | **Brown Tinge** | Green Tinge | **Red Tinge** | Orange Tinge | **Other** | Iris Color |

3 primary: R-G-B. (**ROYGBIV** = red, orange, yellow, green, blue, indigo, violet) Deficiency in ~8% of Caucasian males (Vs 0.5% F) carry such a trait. 4.6% Green weak = Deuteranomaly. 1.4% Green blind = Deuteranopia 1% Red blind =Protanopia, 0.8% Protanomaly. The gene for color vision is found on the X chromosome, deficiencies are a genetically recessive trait, meaning that if at least one gene for color vision is normal, the individual will have normal color vision, thus males who have a single X chromosome (Vs two in women) are much more likely to be color deficient. Most of these individuals are not "color blind", rather their perception of color varies from normal individuals. These individual might be called "**color weak" (trichromats**, see all three primary colors, but have a relatively fewer number of cones for one type of color.). **Dichromat (deutan):** have a complete absence of cones perceiving one of the primary colors, they make up about 2% of the male population, there are three types of dichromats, protanopes, deuteranopes and tritanopes. Protanopes have an absence or deficiency in red receptor cones, and thus are red-green deficient. Deuteranopes carry a deficiency of green receptors and are also red-green deficient. Tritanopes have a deficiency in blue receptor cones and have difficulty with blue-yellow distinctions. All dichromats are at risk for temporary monochromatic vision if looking through tinted lenses or glass.
Certain dz's are associated: optic nerve inflammation, glaucoma, cataracts, multiple sclerosis, central serous retinopathy, cataracts and toxicity from drugs or poisons. Yellowing of the lens of the eye with age may also alter color perception. Blue Blocker sunglasses, which are not recommended for pilots. (See the Sunglasses Selection part of the VFS article on Optimum Vision for Pilots)
Dx: inability to reach a passing score on one of a number of color vision tests. These tests may require the ability to perceive an number within a circle of dots of varying shades of dots or a test as basic as naming a color projected from a lamp. Color vision testing is subject to error if improper lighting is used or the examiner gives incorrect instructions.
Testing: A variety of tests can be used to detect a reduced ability to perceive certain colors (color blindness).

Ishihara color plates: (14 plate, 24 plate and 38 plate editions) are a commonly used, effective means of screening color vision. Pt's with vision as poor as 20/200 may still be able to see the control plate of the Ishihara. The Ishihara plates are patterns of small, colored circles crowded together on a white background to form a large circle. The small circles are usually arranged so that people with normal color vision see a particular number. Those who have color blindness see another number or no number, depending on the type of color blindness.
Other: In pt's who are congenitally red-green defective, the Hardy-Rand-Rittler plates may be useful. Asymmetry in the speed of recognition of the color plates may be significant. Farnsworth lantern (FALANT), Keystone orthoscope, Keystone telebinocular, the LKC Technologies Incorporated, ATT-5 color vision tester, the Opti tech 2000 vision tester, the Titmus vision tester, the Titmus II vision tester and the Titmus 2 vision tester. The military uses pseudoisochromatic plates and the Farnsworth lantern. The Farnsworth Panel D-15, and Farnsworth-Munsell 100-hue test are more involved color vision tests that allow determination of the axis of dyschromatopsia. The latter test also indicates the severity of the dyschromatopsia.
Color Loss --> Alcoholism, Dihydrostreptomycin (loss of green), Amodiaquine, Dimenhydrinate, Carbon disulfide, Ethambutol, Cisplatin, Ibuprofen, Contraceptive hormones, Lysergic acid diethylamide (LSD), Deferoxamine, Ranitidine.
Color Vision Disturbances: "washed out or grayed" seen in optic neuritis, migraine H-A, age related changes of the macula.
Increased Color Perception --> Dronabinol, Mescaline, Ethionamide, Oxygen, Hashish, Psilocybin, Lysergic acid diethylamide (LSD), Tetrahydrocannabinol (THC), Marijuana.
Blue Tinge (Cyanopsia) --> Digitalis, Lanatoside, Alcohol, Digitoxin, Methylene blue, Amodiaquine, Digoxin, Nalidixic acid, Amphetamine, Gitalin, Oral contraceptives, Chloroquine, Hydroxyamphetamine, Ouabain, Deslanoside, Hydroxychloroquine, Quinacrine, Viagra.
Brown Tinge --> Acetophenazine, Diethazine, Methdilazine, Butaperazine, Ethopropazine, Methotrimeprazine, Carphenazine, Fluphenazine, Perazine, Chlorpromazine, Mesoridazine, Pericyazine, Perphenazine, Propiomazine, Trifluoperazine, Piperacetazine, Thiethylperazine, Triflupromazine, Prochlorperazine, Thiopropazate, Trimeprazine, Promazine, Thioproperazine, Promethazine, Thioridazine
Green Tinge --> Acetyldigitoxin, Cyclobarbital, Hydroxychloroquine, Allobarbital, Cyclopentobarbital, Iodine and iodides, Amodiaquine, Digitalis, Barbiturates, Digoxin, Epinephrine, Methohexital, Nalidixic acid, Griseofulvin, Ouabain, Chloroquine, Primidone, Radioactive iodides, Quinine, Talbutal.
Red Tinge --> digitoxin, Methylergonovine, Sulfamethizole, Atropine, Methysergide, Sulfamethoxazole, Belladonna, Ouabain, Quinine, Sulfacetamide, Sulfanilamide, Ergonovine, Sulfadiazine, Homatropine.
Orange Tinge --> Digitalis, Acetaminophen, Amyl nitrite, Benzthiazide, Acetophenazine, Aspirin, Butalbital, Barbiturates, Carbon dioxide, Carbon disulfide, Digoxin, Methaqualone, Chloroquine, Fluphenazine, Furosemide, Colloidal silver, Nitrofurantoin, Hydrochlorothiazide, Phenacetin, Streptomycin, Sulfacetamide, Thiabendazole, Primidone, Thiamine deficiency, Thiocyanate, Thioridazine.
White Tinge --> Capreomycin, Paramethadione, Trimethadione, Chlorothiazide, Phenytoin.
Violet Tinge --> Dronabinol, Marijuana, Quinacrine, Hashish, Nalidixic acid, THC.
****Ref:**(General ophthalmologic examination. Emerg Med Clin North Am. 1995;13:521-38) (A Guide to Physical Examination and History Taking, Sixth Ed, by Barbara Bates, Lippincott in 1995) (Practical problems of defective color vision. Practitioner. 1985;214(1283):654-660)

Pupils:

Links: Exam | **Hippus** | Dilated Pupil | **Mid position** | Constricted | **Common of Abnormalities** | Afferent Defect | **Marcus Gunn** | Argyll Robertson | **Adie's** | Pharmacologic Pupil | **Anisocoria** | Horner's Syndrome | **Iris Color** |



ICD-9 Codes:
379.40 Abnormal pupillary function, unspecified
379.41 Anisocoria
379.42 Miosis (persistent), not due to miotics
379.43 Mydriasis (persistent), not due to mydriatics
379.45 Argyll Robertson pupil, atypical
379.46 Tonic pupillary reaction - Adie's
379.49 Other – Hippus - Pupillary paralysis
Exam: Examine the pupil in a semidark room and do not stand in front of the pt; have the pt fixate the eyes on a distant object while you shine the light obliquely at the pupils. Examined for absolute and relative size and reactions to both light and accommodation. In neurophysiologic terms, applying light to the pupils is equivalent to striking major tendons with a reflex hammer. The normal pupil size in adults varies from 2 to 4 mm in diameter in bright light to 4 to 8 mm in the dark. The pupils are generally equal in size. They constrict to direct illumination (direct response) and to illumination of the opposite eye (consensual response). The pupil dilates in the dark. Both pupils constrict when the eye is focused on a near object (accommodative response).

Size & shape estimate in millimeters (if dark brown iris, shine light from side to see better). Pupils are typically small in infants and elderly and large in adolescents. Check **Reactivity**, direct and consensual (indirect) **-->** observe each sequentially as can't look at them both at same time. (if can't see a reaction, dim the room lights or hold the lid open). PERRLA (pupils equal round reactive to light and accommodation).
Check **Near Reaction (accommodation) --> Pupillary Reactions to Accommodation:** Hold your finger about 10cm from the pt's nose. Ask them to alternate looking into the distance and at your finger. Observe the pupillary response in each eye. Ask pt to look afar then at end of own nose, or have them focus on your finger at ½ meter and bring it close to nose, as eyes converge the pupils constrict. (Not a routine test, most abnormalities due to one eye losing fixation = loss of binocular convergence). If you did not specifically check the accommodation reaction use the term PERRL. Accommodation is constriction of pupils when pt quickly shifts fixation from far object to tip of nose. Physiologic anisocoria is a common cause of unequal pupils that react normally. A non-black pupil suggests opacification of refracting media (cataract). Afferent pupillary defect (APD= paradoxical dilation with light) suggests optic nerve or retinal lesion. See below.
Anatomy: The afferent pathway for pupillary light reflex is the optic nerve (CN II), and for the accommodation is the frontal lobes, but the efferent pathway for both are the parasympathetic fibers of the CN III.
Dilating the eye: see **Cycloplegics (Dilation)** |
Hippus: intermittent (rhythmic contraction) pupillary dilation and constriction independent of light, convergence reflex or psychological stimuli. A normal phenomena when shining light in the eye. Often can get dilation during forced voluntary expiration and contraction during expiration.
Pupillary sparing: the clinical situation where there is complete ptosis and paralysis of ocular elevation, depression, and adduction, but normal pupillary size and movement. Pupillary reactivity with partial ptosis and/or partial ophthalmoparesis does not constitute true pupillary sparing. These pt's have partial involvement of the third nerve, including the pupillomotor fibers, and frequently harbor a space-occupying lesion in the parasellar fossa. Pt's with third nerve paralysis and true pupillary sparing can be followed clinically. If the pupil retains normal size and reactivity after 1 week of observation, they need not undergo CT or cerebral angiography in search of a cerebral aneurysm. Almost assuredly, they will spontaneously improve within 3 months. If not, further analysis is indicated.

Dilated (Mydriatic) Pupil:

Reactive **-->** atropine, sympathomimetics.
Unreactive **-->** supratentorial lesion, ocular trauma, high dose atropine, high dose dopamine.
Ddx: Mydriasis: Oculomotor paralysis **-->** aneurysm, diabetes, trauma, tumor, syphilis. Tonic pupil. Drug induced (topical or systemic), traumatic, essential iris atrophy, absent sphincter muscle.
…….END DEMO of this chapter.

Orthopedics:

Edited by Richard K. N. Ryu, MD, Orthopedic Surgeon.

Contents: Essential Ortho Conditions | **Fractures** | Wrist & Hand | **Fingers** | Elbow & Arm | **Shoulder** | Thoracic Outlet | **Costochondritis** | Gait Eval | **Hip-Thigh** | Knee | **Leg** | Ankle | **Foot** | *Sports Med* | **Compartment Syndrome** | Metabolic and Bone Dz, Tumors and Sarcomas | **Rehabilitation** | Joint Aspiration & Injection | **Pediatric** | Osteomyelitis | **Cervical-Thoracic-Lumbar Spine** | Exercise | **Rheumatology** | OA | **Septic Joint** | Carpal Tunnel | **Acute / Chronic Arthritis, Joint Eval & Exam** | Amputations | **Peri-Operative Eval** | *INDEX* |

Essential Orthopedics Conditions & Definitions:

For individual conditions see anatomical location.
Links: Ortho Emergencies | **Stretching & Flexibility** | Strains & Sprains | **Overuse Injuries** | Bursitis | **Tendonitis** | Muscle Cramps & Soreness | **Joint ROM** | Rehabilitation | **Ganglion Cyst** | Splinting | **Hand Tendons & Nerves** | Extensor & Flexor Tendon Injury and Extremity Lacerations | **Tendon Ruptures** | **See Trauma for** Splinting & Cast application |

On average an adult human has 206 bones (these numbers can vary slightly from individual to individual). The human skeleton consists of both fused and individual bones supported and supplemented by ligaments, tendons, muscles and cartilage. It serves as a scaffold which supports organs, anchors muscles, and protects organs such as the brain, lungs and heart. The biggest bone in the body is the femur and the smallest is the stapes bone in the middle ear. In an adult, the skeleton comprises around 14% of the total body weight, and half of this weight is water. Fused bones include those of the pelvis and the cranium. Not all bones are interconnected directly: There are three bones in each middle ear called the ossicles that articulate only with each other. The hyoid bone, which is located in the neck and serves as the point of attachment for the tongue, does not articulate with any other bones in the body, being supported by muscles and ligaments. The skeleton is the site of hematopoiesis, which takes place in red bone marrow. Marrow is found in the center of long bones.

Orthopedic Emergencies: A septic joint is usually hot, swollen, and very tender with all movements restricted because of pain. Pt's with objective neurological deficit due to nerve root compression or due to other spinal pathology should be referred immediately. A major joint dislocation should be reduced as soon as possible particularly if there is no distal circulation or sensation to the limb. Compartment syndrome caused by swelling in a myofascial compartment leading to a critically impaired circulation to the enclosed muscles in that compartment. In older pt's, aways consider zoster as a cause of pain or the life-threatening conditions of a leaking abd aortic aneurysm (AAA) presenting as back pain, aortic dissection presenting as inter-scapular pain, perforation/peritonitis presenting as shoulder tip pain and acute myocardial infarction (MI) presenting as shoulder or arm pain. A painful, swollen finger could be septic flexor tenosynovitis – a surgical emergency. Pediatric trauma may be due to child abuse. An adolescent with a limp, hip or knee pain could have slipped upper femoral epiphysis. Uncontrollable limb pain after injury may be compartment yndrome. Wrist pain after injury – do not miss scaphoid fracture. A skin break overlying any fracture means an open fracture until proved otherwise. A cut on the knuckle is a fight bite until proved otherwise. Not all acute back pains are mechanical.

Unexplained bone pain, especially at night, is ominous. A painful, swollen stiff digit or hand after a high-pressure injection injury needs urgent exploration.

Stretching:

Some people stretch to reduce risk of injury, others stretch to enhance athletic or sporting performance, and yet others stretch because it gives them a sense of well-being. Athletes are typically told to warm up to begin to sweat prior to stretching. While stretching is believed to prevent muscle soreness and injury and enhance performance, several reviews of the medical literature have concluded there is insufficient evidence to support these claims. Stretching for about 5-10 minutes per session showed no significant effect on either muscle soreness or injury (Effects of stretching before and after exercising on muscle soreness and risk of injury: Systematic review. BMJ 2002;325:468-70), the ave subject would need to stretch 23 yrs to prevent one injury. Stretching reduces the muscles force and power by 2-5%, but other reports indicate that it may increase the running speed by 2-5% (Phys Sports Med 2005;33:22-26). No protective effect was seen for stretching and warm-up programs, as assessed in three trials (Prevention of sports injuries: Systematic review of randomized controlled trials. Arch Intern Med 2007;167:1585-92). Stretching before or after exercise won't spare either elite athletes or weekend warriors from muscle soreness one to three days later according to a review of 10 studies (Cochrane Database of Systematic Reviews 2007, Issue 4. DOI: 10.1002/14651858.CD004577.pub2)....."Some evidence suggests that once muscle soreness has developed stretching may provide a transient relief of soreness….The current review does not provide any evidence of an effect or otherwise of stretching on risk of injury, performance, or well-being." In many studies, the major predictor of joint injury was previous injury and joint laxity, not necessarily inadequate flexibility.

Types of Stretching:

Passive stretching: Partner for external force. Slow sustained stretch.

Static stretching: Self imposed force. Hold for 15–60s. 2 sets for each muscle group.

Ballistic stretching: Repetitive jerking movement. Rapid movements. Possibly injurious.

Flexibility: defined as the ability of the muscle to relax and yield to a stretching force. It is also defined as the active or passive range of motion of a muscle group. Flexibility exercises are used to increase length of the musculotendinous unit. The term flexibility exercise is often used synonymously with stretching exercise. Stretching is employed in the exercise program of injured athletes after evaluation reveals flexibility deficits. A contracture is a shortening of a muscle or other tissues that cross a joint that results in loss of motion. Tightness is a nonspecific term used to describe mild shortness of the musculotendinous unit that does not result in loss of joint motion. Tightness is common in multijoint muscles such as the hamstrings, rectus femoris, and gastrocnemius. Tightness can be improved by self-stretching or flexibility exercises. Stretching exercises are indicated when the athlete demonstrates limited ROM in the subacute or chronic phase of healing. It is necessary to regain the full ROM required for athletic activity to avoid interference with sports-related skills. The underlying cause of restricted flexibility or joint motion must be defined before a treatment can be recommended. The normal flexibility of different muscle groups is sport-specific. For example, a ballet dancer can usually take their foot, with the leg straight, right up to their shoulder, while a marathon runner may be able to lift the straight leg to only 60 degrees. Symmetrical, bilateral apparent muscle tightness, which does not cause symptoms, may be considered as a functional adaptation to the sport being performed. However, if there is an obvious asymmetric flexibility or the athlete's movements are painful, mobilization or other treatments may be indicated. Tight thigh muscles (hamstring) and hip-flexing (iliopsoas) muscles can not only cause pain in those muscles but also back problems, disturbed core stability and similar symptoms. Tight calf muscles may prevent squats, whereby weight will be transferred to the lower back, causing pain there. Chronic muscle tightness can cause fatigue, pain and dysfunction. Hypermobility can cause as many problems as it can solve. There are different types of stretching but the difference in effect among the various methods is small. The most important thing to make the stretching as effective as possible is that the injured player 'finds' the middle of the muscle bulk they need to stretch. Stretching in this position should be held for about 10 to 15 seconds, followed by a few seconds of relaxation, repeated two to three times. Stretching should always be performed in warmed-up muscles.

Strain and Sprains:

Links: Groin Strains | **Hamstring Strain** | Patellar Tendonitis | **Iliotibial Band** | Patellofemoral | **Achilles Tendon** | Splinting & Taping | **Wrist Sprain** | Rehabilitation |

Strain: An injury of the musculo-**t**endinous unit (pearl, the "**t**" in **s**train stands for "**t**endon", Vs ligament with sprain).

Mild (first-degree): Local pain, mild pain on passive stretch and active contraction of the involved muscle; minor disability. Mild spasm, swelling, ecchymosis; local tenderness; minor loss of function and strength. Pain on stretching.

Moderate (second-degree): + pop/snap with spasm. Local pain, moderate pain on passive stretch and active contraction of the involved muscle, moderate disability. Severe spasm, swelling, ecchymosis, hematoma, tenderness, loss of muscle function; palpable defect may be present.

Severe (third-degree): Dramatic onset, often with audible snap/pop, then pain. Severe pain, disability. Moderate spasm, swelling, ecchymosis, local tenderness; impaired muscle function and strength.

Sprain: A ligament injury. Ligaments stabilize joints by preventing abnormal motion.

Grade 1: no laxity and good end point.

Grade 2: laxity, but good end point.

Grade 3: no end point, needs referral for repair within 5 days. Can evaluate with stress radiographs. Rehab with isometric exercises that are begun immediately. Goal is to re-establish strength and motion while protect from re-injury.

Tx of Strain/ Sprains:

Tx: NSAIDs, consider muscle relaxer. **"PRICEMS"**

Protection (padding and changes in technique to avoid further injury.

Rest. **I**ce (cold reduces inflammation, heat in an acute injury may augment inflammation).

Compression.

Elevation. **M**odalities (U/S, electrical stimulation, heat)/ **M**eds (PRN NSAIDs [may impair healing], steroid injections).

Support (braces). Rehab to re-establish both strength and flexibility together. Early mobilization after acute upper & lower limb injuries in adults decreases pain, swelling and stiffness based on a systematic review of 49 clinical trials (J Fam Prac 2004;53:706-

12). Avoid overuse of common OTC sports creams such as Bengay or Icy Hot as deaths have occured due to high levels of methyl salicylate, an anti-inflammatory commonly found in the creams. Methyl salicylate poisoning is uncommon and deaths are rare.

General Patient Advice:

1) Apply ice for 20 minutes 3 to 4 times a day during the first 48 hours of any injury. Use gentle range of motion exercises as tolerated.

2) Take non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprophen 400-600mg 3 times daily or Naproxen twice daily or Aspirin 650mg 4 times daily as needed for pain. If get an upset stomach, stop, may need to take Omeprazole (Prilosec) 20mg daily to protect the stomach.

3) After the first 48 hours can start to add in or alternate with heat so long as it does not worsen the symptoms.

Compression: Most effective means of stopping hemorrhage. Elastic dressings for periods of 2-4hr, never sleep with them on. Apply selectively to injured structures. Best to place a padding underneath the wrap such as 6-8 ABD pads or diapers or ½ felt for focal compression.

Elevation: Raise above level of the heart. Avoid dependency, which increases swelling.

Rest: Cease all activity, not just painful activity.

Active --> avoid exacerbating activities. Early, controlled mobilization is superior to immobilization for soft-tissue injuries.

Alternative training: to maintain fitness while the injury is mending. Wear a life vest and run in place in a pool. See "physical rehab".

Childhood injuries: PRICE (instead of RICE): protection, rest (active rest, play something that doesn't hurt), iced, compression and elevation. Children rarely sustain sprains or torn ligaments but they are at risk for fx's or separations in the growth plates, which are generally "the weakest link" in the musculoskeletal system in children.

Applying a Moist Compress:

To reduce pain, swelling, and warmth.

Step #1: Prepare moist compress by soaking gauze pads in selected solution (hot/cold). Make sure gauze pads are big enough and plentiful enough to cover area desired. Make sure they are thoroughly soaked before applying to area.

Step #2: Don gloves. Cover with dry covering, preferably a plastic bag or linen saver, and mold it to the body area.

Step #3: Check area every 15 minutes to ensure heat is not "cool" and cold is not "warm." Apply moist compress for approximately 15 to 30 minutes four to five times per day. Avoid overexposure to moist compress, which may impair skin integrity. Keep the patient as dry as possible—check to make sure you have dried area after moist compress has been completed.

Overuse Injuries:

Link: Grade | **Injury in Youth & ICD9 |** Overtraining Syndrome | **Strains & Sprains |** Bursitis | **Tendonitis** | Physical Therapy and Rehabilitation | **Nerve & Brachial Plexus |** Carpal Tunnel | See Prev Med: Ergonomics |

Get bone, muscle, or tendon microtrauma caused by repetitive stress without adequate healing time. Those who play multiple sports involving same body part are at higher risk for overuse injury vs those who play multiple sports involving different body parts. Examples in youth include traction apophysitis of elbow in a baseball pitcher, spondylolysis in a gymnast, and rotator cuff tendonitis in a swimme…these injuries are more likely during peak growth velocity.

Grade 1: no pain with activity, some pain after (immediately, during evening or following day).

Grade 2: some discomfort during activity, but not enough to interfere with performance.

Grade 3: discomfort with activity that interferes with performance.

Grade 4: discomfort so intense that the activity cannot be performed at all.

Over training Syndrome: a marked decrease in performance, inc fatigue, persistent muscle soreness, mood disturbances and feeling "burn out"….see above link.

W/u: CBC, LFT, lytes, glucose, TSH, ECG, echocardiogram. (Phys Sport Med 2001;29:5)

Overuse Injury in Youth:

Overuse injuries are those caused by repetitive minor trauma that overwhelms the individual's healing capacity. When evaluating children and adolescents who report pain with sports activity, one must always keep in mind that other systemic problems can masquerade as sport injuries. For example, a persistently swollen ankle may be the first sign of juvenile rheumatoid arthritis; a swollen tender tibial tubercle or iliac crest may be due to inflammation secondary to infection. The first sign of leukemia may be fatigue with activity; and the early stages of osteogenic sarcoma may be confused radiographically with a healing stress fx. An adolescents may no longer wish to continue to spend the long hours in training, but fearing rejection and chastisement from disappointed parents, cannot admit to them that they just do not want to participate anymore (physician must "read between the lines" to help the athlete deal with the situation in an acceptable manner).

Hx: type of sporting activity, duration and frequency of training, previous injuries, any new equipment changes, and type and timing of the pain.

Shoulder injuries seen with swimming, baseball, and tennis. Elbow injury common in baseball, racquet sports, weight lifting, and gymnastics. Wrist injuries are seen in sports that involve repetitive loading to the dorsiflexed wrist, such as gymnastics.

Baseball and softball injuries: decreased by interventions such as breakaway bases, batting helmets, face shields, and lighter mass balls. Curve ball, slider, and increased number of pitches thrown in a game and during a season to be associated with an increased risk of shoulder and elbow pain.

****Ref:** (Overuse injuries in pediatric athletes. Curr Opin Ped 2004;16:47-50)

ICD-9 Codes:

840.0 Sprains and strains of Acromioclavicular (joint) (ligament)
840.4 Sprains and strains of Rotator cuff (capsule)
840.9 Sprains and strains of shoulder and upper arm NOS
841.9 Sprains and strains of elbow and forearm NOS
842.10 Sprains and strains of Hand NOS
843.9 Sprains and strains of hip and thigh NOS
844.9 Sprains and strains of knee and leg NOS
845.00 Sprains and strains of ankle NOS

847.1 Sprains and strains of Thoracic
847.2 Sprains and strains of Lumbar
E927.0 Overexertion from sudden strenuous movement
E927.1 Overexertion from prolonged static position
E927.2 Excessive physical exertion from prolonged activity
E927.3 Cumulative trauma from repetitive motion
E927.4 Cumulative trauma from repetitive impact
E927.8 Other overexertion and strenuous and repetitive movements or loads
E927.9 Unspecified overexertion and strenuous and repetitivemovements or loads

Bursitis:

Links: Hip Bursitis | **Knee Bursitis** | Shoulder |

Inflammation of a bursa. There are over 150 bursae in the body. They allow skin, tendons and ligaments to smoothly glide over one another, preventing friction during motion over bony prominences, tendons and fibrous tissue. Most are closed and sac-like, and only a few cell layers thick. The most common cause is repetitive microtrauma or macrotrauma. Once inflamed they produce fluid and swell, the wall thickens.
Common bursitis syndromes include subacromial (shoulder), olecranon (elbow), iliopectineal (inguinal), trochanteric (lateral hip), prepatellar (ant knee), infrapatellar (adjacent to patellar tendon), anserine (medial knee), ischiogluteal (buttock), retrocalcaneal (heel), calcaneal (heel).

ICD-9 Codes:
727.3 Bursitis NOS
727.00 Synovitis and Tenosynovitis
726.90 Tendinitis/Capsulitis
729.1 Myofascitis of Upper Extremity Musculature
729.1 Myofascitis of Lower Extremity Musculature
S/s: Pt has pain on movement and at rest with dec active but normal passive ROM. If chronic can scar up with irregular areas that feel like loose cartilage (joint mice) or get calcium deposits. In exam see tenderness, warmth and erythema. See below for knee and hip bursitis.
Basic Tx nonseptic of Bursitis: If superficial with obvious swelling, it should be **aspirated first** before corticosteroid is injected. If deep, aspiration is likely of limited value. Splint to limit motion for 5-7 days (relative rest), this prevents progression to a chronic bursitis. NSAIDs, PRICEMS. If not improved in 2-3 weeks give another corticosteroid injection after aspiration of contents. Can return to play with padding when swelling is controlled. Consider night splint if not improving. Advise pt to watch for signs of infection. Consider immediate aspiration to r/o infection (<8,000 WBC/mm^3 with negative Gram stain and Cx is reassuring) or an X-ray to r/o fx.
Septic bursitis: should be suspected if there is surrounding cellulitis, erythema, fever and inc WBC, it is usually due to Staph aureus/ epidermidis. If early cellulitis give Keflex 500mg TID or Dicloxacillin 500mg QID, if lymphangitis, diabetes, immunocompromised will need parenteral Abx with repeated aspirations. Initial daily aspirate and Tx with Abx for a minimum of 3 weeks. Nafcillin or oxacillin 2 g IV q4h or Dicloxacillin 500 mg PO qid. Can also use Cefazolin 2 g IV q8h or Cephalothin 2 g IV q4h or Vancomycin 1 g IV q12h or (ciprofloxacin 750 MG PO bid or 400 mg IV) + rifampicin 300 mg PO bid.
Ddx: avascular necrosis, arthritis, arthrosis, joint effusion, crystal induced, palindromic rheumatism, other infections (i.e., tubercular, fungal, viral), tendonitis, bursitis, juvenile rheumatoid arthritis.

Tendonitis:

Links: Elbow & Arm | **Shoulder** | Wrist & Hand | **Knee** | Leg | **Tendon Ruptures** | Triceps Tendonitis | **Biceps Tendonitis** |
Overuse injuries account for 40% of all sports injuries. Tendons are interposed between muscles and bones.
"**Big Four**" tendons are the Achilles, patellar, lateral elbow (ext carpi radialis brevis) and rotator cuff.
Commonly seen in: hamstrings (origin), quads (insertion), patellar (origin), Achilles (insertion, sheath, mid substance), posterior tibial (mid substance), rotator cuff (supraspinatus insertion), De Quervain's/ trigger finger (sheath, pulley of abductor pollicis longus and long finger flexors), Lateral epicondylitis (common wrist extensor origin).
Sx: gelling type pain, pain with use after a period of rest (AM pain but subsides in minutes of initial use)
Multifactorial cause: from overuses, cumulative trauma, repetitive strain (inc duration, frequency intensity).
Risk: age related degeneration and dec vascular supply/ healing, inc stiffness, poor technique, equipment problems, muscle imbalance/ weakness, inflexibility, malalignment. Quinolone induces tendinopathy. Vs **Tenosynovitis** (tendon sheath)
ICD9:
726.90 Tendinitis/Capsulitis
727.00 Synovitis/Tenosynovitis
729.1 Myofascitis of Upper Extremity Musculature
729.1 Myofascitis of Lower Extremity Musculature
3 stages of injury:
1. Peritendinitis (inflammation of only the paratendon**-->** swell, pain, local TTP, warmth, dysfunction).
2. Peritendinitis with tendinosis (some intratendinous degeneration**-->** palpable tendon nodule, swell, inflammatory signs).
3. Tendinosis (Tendonopathy): loss of integrity of collagen. Acute inflammatory tendinopathies are common, but most pt's seen in primary care will have chronic sx's suggesting a degenerative condition that should be labeled as tendinosus or tendinopathy (recovery time of 2-3 days Vs 2-3 mo's).
Degeneration due to atrophy from aging/ microtrauma/ vascular compromise**-->** ±palpable nodule, disordered collagen structure, swelling is absent. Majority of athletic injuries are truly tendinosis, this includes Achilles "tendonitis", patellar "tendonitis", lateral epicondylitis and rotator cuff "tendonitis". Has longer recovery than tendonitis, may take mo's.

S/s: visible or palpable swelling of the tendon sheath, ± crepitance/ dec ROM. Consider X-ray to r/o degenerative arthritis, stress fx, calcified/ ossified tendon, traction spur, bone cyst.

Tx of Grade 1 Overuse Injuries: Relative rest, decrease the intensity or duration of activity by 25%, then gradually increase as sx's permit. Same for assembly line workers, commonly seen with inc overtime.

Tx of Grade 2: dec intensity or duration by 50%, ice, stretch, NSAIDs, consider PT for eccentric strengthening exercises. The majority of chronic tendinopathies are not inflammatory, few data exist to support the use of NSAIDs over analgesics without anti-inflammatory effects (Med Sci Sports Exerc 1998;30:1183-90) and (Cochrane Database Syst Rev 2001;(4):CD003686).

Tx of Grade 3: rest, some activity OK, PT to speed recovery, strengthen, increase endurance, restore coordination/ proprioception. Steroid injection, NSAIDs, ice.

Tx of Grade 4: Complete rest with sling or splint. Above tx, consider surgery.

Tx of Tendinosis:

#1: Education: A tendon only has 13% the O_2 uptake of muscles and it requires >100 days to make collagen. Expect 6-8 wks to recover from an acute injury and 12-16 wks (3-4mo) for a chronic injury.

#2: Relative rest. Pain as the guide, rest until no pain with ADL's, then start strengthening. Need to use massage and eccentric muscle training/ strengthening, as treating them as pure inflammatory "tendonitis" is flawed. Relative rest, change in technique, ergonomics. Load-decreasing devices such as braces and supports. Intermittent splinting (qHS) is sometimes useful. Modalities such as ice, heat, U/S, NSAIDs. PT to correct tightness/ imbalances with emphasis on eccentric strengthening. Local steroid injections (caution if Achilles or patellar, wt bearing areas, avoid intratendinous inj), max of 2-3 with 3mo in between. Avoid strenuous activity for 2-3 wks after injection to avoid rupture during the phase of maximal steroid atrophy. Surgical consult if persists >4-6mo.

Enthesopathy: disorders of peripheral ligamentous or muscular attachments. Any abnormality of the site where the tendon joins to the bone. Abnormalities that can occur includes inflammation and calcification.

ICD9: 726.8 Other peripheral enthesopathies. 726.9 Unspecified enthesopathy.

Examples: Adhesive capsulitis of shoulder. Rotator cuff syndrome of shoulder and allied disorders. Periarthritis of shoulder. Scapulohumeral fibrositis. Enthesopathy of elbow region. Enthesopathy of wrist and carpus. Bursitis of hand or wrist. Periarthritis of wrist. Enthesopathy of hip region. Bursitis of hip. Gluteal tendinitis. Iliac crest spur. Psoas tendinitis. Trochanteric tendinitis. Enthesopathy of knee. Enthesopathy of ankle and tarsus. Other peripheral enthesopathies. Unspecified enthesopathy.

Tx: Rest, activity modifications. PRN NSAIDs. Cortisone inj's.

Large-Tendon Rupture or Avulsion:

Links: **Achilles Tendon** | Biceps Tendonitis & Rupture | **Pectoral Injury** | Pattelar Rupture |

Forceful eccentric contraction may cause a partial or complete rupture of a vulnerable large tendon, especially in middle-aged men. A history of local or systemic corticosteroid or anabolic steroid use may lead to poor tendon quality and increased risk for rupture. Ultrasound or MRI may help confirm the dx. Tx is generally surgical with anatomic repair. Return to sport depends on the pt's age, lifestyle, tendon involved, and medical comorbidities.

Risk Factors: Chronic hemodialysis with secondary hyperparathyroidism. Current fluoroquinolone use. Diabetes mellitus. Excessive body weight. Heavy weight lifting. History or current use of anabolic steroid. History or current use of local corticosteroid injections. Immobilization of a specific joint. Inflammatory arthropathies. Male sex. Muscle weakness and imbalance. Smoking. Spondyloarthropathies.

S/s: Functional impairment—careful physical examination and evaluation are required. Mechanism of injury usually involves a violent eccentric contraction. MRI is highly sensitive and specific for dx. Pt usually reports feeling and hearing a "pop". Possibly associated with systemic disease—a thorough medical history is always required.

Tx: surgical, with anatomic repair (only with Achilles tendon rupture is nonsurgical management a reasonable option).

Muscle Cramps:

Links: See Psych module: **Nocturnal Leg Cramps** | Neuro module: **Movement Disorders** |

Cramps are involuntary, irrational, painful contractions of voluntary muscles. Usually caused by sports or occupational muscle injury. The affected muscle feels hard and tense. See below for Ddx and Muslce Soreness.

Idiopathic: ordinary cramps (ubiquitous and tend to be little more than a benign nuisance to the sufferer), nocturnal myoclonus (Rx Klonopin), RLS.

Physiologic Fasciculations: repetitive twitching of a mucles, ofen the thigh, peri-orbital or biceps. Seen after exercise or with fatigue.

True Cramp: Due to motor unit hyperactivity. Seen **with "ordinary" cramps,** commonly due to imbalance of electrolytes (Ca, K, Mg, Na), slight muscle train, inadequate warm-up, recent dietary changes (inc caffeine, new energy bar or electrolyte drink). Seen with high intensity exercise of long duration.

Leg cramps: incidence increases with age. Up to 50% of people attending a general medicine clinic have had leg cramps within 1 month of their visit, and >2/3 of people >50 yo have experienced leg cramps (J Gen Intern Med 1998;13:600–606). Very little is known about the causes of leg cramps. Risk factors include pregnancy, exercise, salt depletion, renal dialysis, electrolyte imbalances, peripheral vascular disease (both venous and arterial), peripheral nerve injury, polyneuropathies, motor neuron disease, muscle diseases, and certain drugs. Other causes of acute calf pain include trauma, deep venous thrombosis (see thromboembolism), and ruptured Baker's cyst. Ordinary muscle cramps are characterized by a painfully hard, palpable contraction of explosive onset, at times preceded or followed by brief twitches, which usually involves one muscle at a time. They may be precipitated by volitional movement of trivial extent or more forceful contraction in a muscle already shortened (as may occur during or after exercise). The cramps can be terminated by passive stretching of the muscle or activation of its antagonist. In severe cramps, the muscle may remain sore and tender for days.

ICD-9 Codes:

729.82 Cramp

728.85 Spasm of muscle

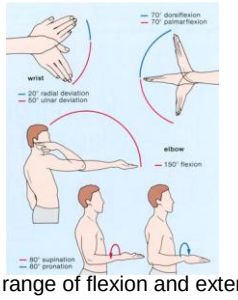
Tx of Exercise-Related: immediately stop exercise, immediately hydrate with fluid containing electrolytes, then apply pressure and massage area, finally stretch the muscle, then warm soaks. Prevent by being well conditioned, regularly stretching, adequate

nutrition. Decrease the intensity and duration of exercise. Try a high-carb meal prior to activity. Ice for acute cramps, heat & stretching to prevent. Empiric evidence suggests that supplementation with high sodium & high acetic acid = vinegar) via pickle juice or mustard may help.
Pycnogenol: Pycnogenol is an extract from the bark of the French maritime pine tree. Pycnogenol (100 BID + 1.5 L of water daily) was effective in reducing the requency and severity of cramps and muscle pain at rest and before and after exercise among 66 healthy athletes and pt's with chronic venous insufficiency, intermittent claudication, and diabetes with microangiopathy (Angiology. 2006;57:331-339). Pycnogenol is a naturally occurring compound found in French maritime pine bark. Chemically, it is a combination of procyanidins and phenolic acids and is purported to have significant antioxidant effects, in part by enhancing the actions of vitamins C and E. There were no adverse events associated with tx. One study found that taking pycnogenol 200 mg daily for 30 days significantly improves treadmill exercise capacity in recreational athletes (Improved endurance by use of antioxidants. Eur Bull Drug Res 1999;7:26-9).
Cherry Juice: Drinking cherry juice (12-ounce, each bottle of juice contained the equivalent of 50–60 cherries and provided at least 600 mg of phenolic compounds and 40 mg of anthocyanins) before and after exercise decreases some indicators of exercise-induced muscle damage, notably pain and loss of strength (Br. J. Sports Med. 2006;40:679–83) (strength was lost by 22% and 4% of theplacebo and juice groups, respectively)….Tart cherries contain cyclooxygenase-inhibiting flavonoids and anthocyanins with high levels of antioxidant and anti-inflammatory activity.
Caffeine eases the muscle pains of exercising according to a study on 25 fit, college-aged men given a placebo pill an hour prior to the high-intensity exercise alternating with a caffeine pill (amount = 2.5 to 3 cups of coffee) prior to exercise (Int J Sport Nutr Exercise Metabolism. 2009;April).
Other Etiology/ Ddx: Generalized severe cramping, usually a systemic metabolic disturbance.
Congential--> McArdle dz, Autosomal dominant cramping dz.
Neoplasm--> invasion of nerves.
Endocrine--> thyroid (hypo / hyper), Diabetes mellitus.
Myotonia and hypothyroidism: the pt may perceive an uncomfortable but painless muscle tightness or stiffness, often made worse in the cold. Pt notes difficulty releasing a grip (be it a handshake, doorknob, or cooking pan) that improves with repeated contractions.
Metabolic--> Dec BS, Dec Na/ Ca/ Mg, Inc/dec K. Alkalosis due to any cause (eg, severe vomiting or hyperventilation) may decrease ionized calcium and cause muscle cramping and paresthesias.
Toxins--> strychnine, lead, tetanus, Black Widow Spider. Tetanus is due to the suppression of spinal inhibitory neurons by an exotoxin of the bacterium Clostridium tetani. Clinically, involuntary spasms triggered by movement, sensory or emotional stimuli are superimposed on a state of virtually continuous stiffness. The sx's may begin in the vicinity of a wound and remain localized or, more often, become generalized. Even in the localized form, one may note mild trismus due to the susceptibility of the masseter innervation.
Vascular--> claudication (vascular insufficiency) (worse with any walking).
Neuro--> LMN dz or myotonic dystrophy (check EMG's), nerve root compression (ck MRI), spinal stenosis (worse with walking down stairs or down hills as extend the back). "Stiff man" syndrome, rare.
Drugs--> Nifedipine, beta-Agonists (Terbutaline), ETOH, Penicillamine, Nicotinic acid, diuretics, Li, Cimetidine, Clofibrate, Morphine withdrawal, Phenothiazines, cholestyramine. Extended use of Prilosec. Avoid Earl Gray Tea.
Other--> diarrhea, liver dz, sarcoid, pregnancy related, occupational, nocturnal calf-muscle cramps.
Contracture--> Metab myopathy, hypothyroidism, McArdle disease.
Tetany--> Hypo- Ca, Mg, K, Hyperkalemia, Respiratory alkalosis.
Myalgia: a "crampy" feeling, or aching discomfort in the muscles without any visible or palpable hardening. Common in childhood, but can also be seen in a variety of neuromuscular and systemic illnesses, as well as in the context of generalized pervasive fatigue without objective muscle weakness in depressed pt's.
Fasciculations: spontaneous visible muscle twitches due to contraction of a motor unit. Can be seen in several neuropathic disorders associated with cramping. Can be a sx of cervical or lumbar spine degenerative disc disease. Can also appear as a benign condition with muscle cramps, particularly in people with large muscles.
Focal dystonia: a movement disorder manifest by the assumption of involuntary abnormal postures. Pt's may relate spasmodic, even painful, bending or twisting movements of an extremity, neck, face or trunk that remain in a persistent attitude (e.g., overextended/overflexed posture of hand, inversion of foot, pulling of the head to one side). The slower onset, inappropriate contraction of many opposing muscles, and unusual postures evoked help to distinguish dystonia. Occupational in athletes (golf, tennis), writers, miners, musicians. Writer's Cramp, Focal Hand (Occupational) Dystonia.
Get cramping pain in forearm and hand with activity. Tx with sensory discriminative retraining, home exercise program, sensory exercises, posture correction, relaxation exercises, range of motion and strengthening exercises, cardiovascular fitness program. Botulism Toxin Type A local injection. (J Hand Ther 2000;13:289-301) (Br J Med 1993;50:91-4)
Tetany: a cramplike syndrome consisting of painless spasms, most notable for their carpopedal distribution but potentially involving other muscles. The hand typically assumes a posture of tonic adduction of thumb and fingers, flexion of metacarpophalangeal joints, with extension of interphalangeal joints. The toe becomes plantar flexed and the ankle inverted. The spasms, commonly precipitated by hyperventilation, are preceded by tingling paresthesias of the mouth, fingers, and toes, a discriminative feature from dystonia. Tetany is seen in states that cause alkalosis or reduction in ionized calcium and magnesium, which appear to render the peripheral nerves hyperirritable.
Carpopedal or Carpal Spasm: often associated with tetany. May have flexion at wrist/ MCP joints/ thumb against palm with extension of PIP joints and DIP joints and adduction of fingers (forms a cone). Trousseau's Sign.
Exercise-Related Delayed-Onset Muscle Soreness (DOMS): occurs during activity (acute soreness surrounding activity, known as "the burn") or is delayed in onset. DOMS starts 8-24 hours after exercise, reaches a maximal intensity at 1 to 3 days, and then gradually subsides in 5 to 7 days. This delayed soreness serves as the best available guide to maximize training and avoiding injury.
DOMS is thought to be caused by two mechanisms: Exercise that exceeds a muscle's stress tolerance can lead to mechanical damage to the muscle fibers with disruption of Z lines and metabolic release of cytosolic enzymes into the blood. The more muscle damage that occurs, the more soreness is present. Increased environmental temperature and eccentric contractions (e.g., running downhill, squats) are factors that increase soreness. The muscle volume is increased by edema. The muscles are firm and tender

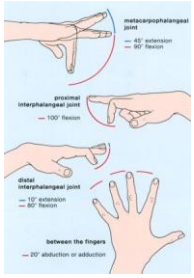
and cannot contract with their maximum force. Continued exercise and pressure increases soreness. Muscles subjected to eccentric loading (e.g. running downhill) are particularly susceptible to this condition. Unconditioned individuals are more likely to develop DOMS after strenuous activity.
S/s: Onset is usually within 1–2 days after exercise and peaks at 2–3 days, with resolution within 1 week of the offending activity. Sx's include muscle pain, fatigue and tenderness.
Tx: symptomatic, consisting of relative rest and PRN NSAIDs, which do not hasten healing and may mask sx's and increase susceptibility to injury. Tart cherry juice (12 fl oz BID x 8 days) decreases sx's of exercise-induced muscle damage, according to a RCT (Br J Sports Med. Posted online June 21, 2006) (cyclo-oxygenase inhibitory flavonoids and anthocynanins with high antioxidant and anti-inflammatory activities, ~45 cherries a day) (strength loss averaged over the four days after eccentric exercise was 22% with the placebo but only 4% with the cherry juice).
Exertional Rhabdomyolysis: due to significant muscle damage, most probable in untrained athletes with predisposing risk factors for dehydration such as drug use (e.g., alcohol, diuretics), concomitant illness, or a hot environment. Athletes can present with sx's in a single muscle group (biceps, triceps) after an overstrenuous lifting session. The athlete presents with muscle weakness, tenderness, and swelling which can develop rapidly. Typical lab findings include a markedly elevated CK (up to 10 to 40 times normal) and an elevated creatinine. Urinalysis can show a dark brown urine with casts and myoglobin. Hb may or may not be present. Acute renal failure can develop secondary to direct tubular injury from the myoglobin. Hydration is very important for recovery. If myoglobinuria is present, admission to an inpatient unit for vigorous hydration and alkalinization of the urine with bicarbonate to maintain renal function are indicated. Administration of mannitol to encourage diuresis may be necessary.
****Ref:** (Cramp: a review. J R Soc Med 1982;75:546-49) (Cramps. NEJM 1971;285:31-40)

Normal Joint Range-of-Motion (ROM) in degrees:

Link: See Rheum Module for **Hypermobility Syndrome(s)** |

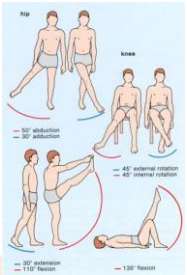


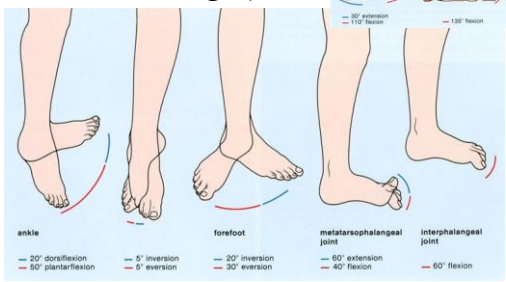
Shoulder: flexion @158-180 deg. Ext @53-60, Abduction @170-180 (first 120 deg are strictly from the shoulder, last 50-60 deg are due to scapular motion). Adduction @50 deg. Horizontal flexion @135. Arm at side (internal rotation = IR) @68-70 deg (@90 deg adduction, 90 deg). External rotation (ER) @68-70deg. Arm in 90 deg: Abd IR @70, ER @90. Both passive and active ROM is markedly decreased with glenohumeral arthritis and frozen shoulder. Active ROM is decreased but normal passive ROM with impingement, rotator cuff tear, bicipital tendonitis.
Elbow: flex @146, hyperextension @0.
Forearm: pronation @71, supination @84.
Wrist: ext @71, flex @73, ulnar deviation @33, radial deviation @19. The normal range of flexion and extension are 90º and 80º, respectively.



Thumb: abd @58, IP flex @81, MP flex @53, MC flex @17, IP ext @17, MP ext @8, MC ext @20.
Fingers: DIP flex @, MIP flex @100, MP flex @90, no DIP or MIP ext, MP ext of 45.

Hip: Flexion 113-120 degrees. Abduction 45-48 degrees. Adduction 20-31 degrees. Ext rotation 45 degrees. Int rotation 40 degrees. Ext @28. Horizontal flex @60. Hip in flexion IR & ER @45, hip in ext IR @35, ER @48. In the standing position. Flexion = move your leg forwards. Extension = move your leg backwards. Abduction = move your leg away from your side. Adduction = move your leg towards to other leg. Internal Rotation = rotate your foot towards the other (toes pointing towards each other). External Rotation = rotate your foot away from the other (toes pointing outwards).
Knee: flex @ 135, hyperextension @10.
Ankle: plantar-flexion @48-70, dorsiflex @10-18.
Hind Foot (subtalar joint): inversion & eversion @5. Supination 20, pronation 10.
Forefoot: inversion @33, eversion @18.





Great Toe: IP flex @60-90, MP flex @37-45, MP ext @60-90.
2nd-5th Toes: DIP flex @55, MIP flex @38, MP flex @35, ext @40.

Cervical Spine: flex at 48, ext @38, lateral bend @43, rotation @45.
Thoracic Spine: flex @85, ext @30.
Lumbar Spine: Flexion @ 40, Ext @ 15, Lateral bend @28-30, Lateral rotation @38-40 to each side.
Joint instability: present when the joint has greater-than-normal movement in any plane. Subluxation refers to a joint in which there is partial displacement of the articular surfaces but still some joint surface-to-surface contact. A dislocated joint has lost all cartilage surface-to-surface contact. Instability is best determined by supporting the joint between the examiner's hands and stressing the adjacent bones in directions in which the normal joint does not move. The pt must be relaxed during the examination, because muscle tension may serve to stabilize an otherwise unstable joint. For example, a knee with a deficient ligament might appear stable if the pt contracts the quadriceps muscles during evaluation.

Joint Motion Terminology:

Abduction - the act of drawing a body segment away from the median line of the body.
Adduction - the act of drawing a body segment toward the median line of the body.
Eversion - the act of rotating the pronated foot externally on the ankle.
Inversion - the act of rotating the pronated foot internally on the ankle.
Extension - the act of drawing a body segment toward a straight line position with its proximally conjoined body segment.

Flexion - the act of drawing a body segment away from a straight line with its proximally conjoined body segment, or toward that joint's smallest acute angle.
Hyperextension - in excess of normal extension.
Hyperflexion - in excess of normal flexion.
Pronation - the act of rotating the hand or foot internally on its long axis.
Supination - the act of rotating a hand or foot externally on its long axis.

Anatomical Positions Terminology:

Anterior - the front of the body or body part.
Collateral - from lateral meaning side, parallel.
Diarthrodial ioint - a ball and socket joint.
Distal - farthest from a center, from the midline, or from the trunk. Farthest from a point of reference (opposite of proximal).
Dorsal - upper surface.
Dorsum - the back of a body part.
Inferior - toward the bottom of the body or body part.
Insertion - muscle attachment to a bone that moves.
Lateral - away from the middle of the body. Pertains to the side (in relationship of position from the midline of the body).
Medial - toward the midline of the body.
Origin - the fixed end or attachment of a muscle.
Plantar - ventral aspect of the foot (sole of the foot).
Posterior - the back of the body or body part.
Prone - face-down, horizontal position of the body.
Proximal - closest to the midline or center of the trunk.
Nearest to the point of attachment, origin, or other point of reference.
Superior - toward the top of the body or body part.
Supine - lying on the back, face upwards, opposed to prone.
Valgus - position of a body part that is bent outward.
Varus - position of a body part that is bent inward.
Ventral - bottom surface.

Body Size and Habitus:

Describes the physical characteristics of an individual and include such considerations as physique, general bearing, and body build.
Height: the length from the plantar surface of the foot to the crown of the head. Heights that fall below the 3rd percentile or above the 97th percentile may require investigation.
Weight: the total weight of the body. Body weight should be measured on beam scales with nondetachable weights. Spring scales are not sufficiently accurate. The scale should be calibrated to zero. The pt should be dressed in light clothing without shoes.Weights > 120% of "ideal" suggest obesity, while weights < 70% of "ideal" may indicate severe malnutrition.
Body proportions: At birth the normal upper to lower segment ratio is 1.7:1. The legs grow more rapidly than the trunk, and by age 10 the segments are equal and remain so in adults. The arm span is the distance between the tips of the middle fingers with the arms fully extended. In adults the arm span should equal the height. Severe depletion of energy stores is indicated by values below the 30th percentile. Midupper arm circumference is used to calculate midupper arm muscle circumference. Muscle circumferences < the 30th percentile suggest severe depletion of protein stores.

Fractures (Fx's):

Links: Intro & Eval of Fx's | Anatomy | **Salter-Harris & Other Classification** | Open Fx's & Traction | **Types of Fx** | Fx Healing, Abx & ICD9 Codes | **Casting & Splinting** | STRESS Fx's | **Wrist Fx's** | Finger Fx | **Hip (femur) Fx** | **Sesamoiditis / Fx** | Other Foot & Toe Fx | **Ankle Fx** | Arm Fx | **Splinting** | Leg Fx | **Shoulder and Clavicular Fx's** | Compartment Syndrome | **See Hem-Onc for** Fat Embolism | See Trauma for **Pelvic** | Spine | **See Wilderness Module for Fx in Survival Situations** | **See Pulm for** Rib Fractures | **Sternal Constusion & Fx** |

Before reaching 65 years of age, half of Americans will sustain a fx that is referred for orthopedic services. Except for rib fractures, all types of fracture were more common among women than among men.
Stable fx's: tend to maintain their position and alignment; unstable fx's tend to displace.
Unstable fx's: require early immobilization to prevent this result. If there is doubt about the stability of a particular fx, it should be treated as unstable until consultation is obtained. There are relatively few types of fx's that do not require urgent (1-2 days after immobilization) or immediate orthopedic referral. In general urgent referral is needed for any unstable fx or one compromising a neurovascular structure.
Hematoma Block: to provide anesthesia for reduction of fx's, particularly of the distal forearm and hand. Preparing the skin over the fx site, inserting the needle into the hematoma (confirmed by aspirating blood), and slowly injecting from 5-15 mL of plain 1-2% lidocaine, into the fx cavity and around the adjacent periosteum. Onset of adequate anesthesia occurs in about 5 minutes and may last for several hours. This procedure should not be performed through dirty skin, into open fx's, or in small children.
Bone contusions (bone bruises): traumatic lesions resulting in edema, hemorrhage, and microfracture of subchondral trabecular bone. Bone contusions are radiographically occult, but their appearance MRI has been well described.
Pseudofractures: Anatomical variants (congenital anomalies). Developmental findings in children (growth plates, ossification. centers)—need comparison views. Degenerative changes (elderly patients), old trauma. Nutrient artery foramina. Imaging artifacts (e.g., Mach band adjacent to overlapping bones). Suboptimal or incorrect positioning.
Indications for ORIF of fx's:
Absolute: Unable to obtain an adequate reduction. Displaced intra-articular fx's. Certain types of displaced epiphyseal fx's. Major avulsion fx's where there is loss of function of a joint or muscle group. Non-unions. Re- implantations of limbs or extremities.

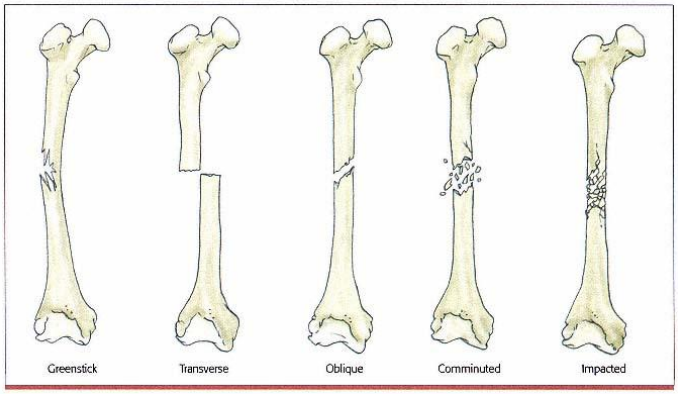
Relative: Delayed unions. Multiple fx's to assist in care and general management. Unable to maintain a reduction. Pathological fx's. To assist in nursing care. To reduce morbidity due to prolonged immobilization. For fx's in which closed methods are known to be ineffective. Fx's accompanying nerve of vessel injury. Open fx's. Cosmetic considerations. Economic considerations.
Indications for ORIF of metastatic lesions: Life expectancy >1-2 mo's. Continued pain after radiotherapy. Lesion >2.5 cm diameter (described for the femur). Destruction of 50% or more of the cortex of a long bone. Adequate bone quality. Procedure would improve mobilization and independence.

Definitions/ Classifications:

Link: Types of Fx |

1. Evaluation of fx: Examine the bone for instability. Examine the soft tissue for associated injury. Look for any associated injuries to viscera, nerves, blood vessels, tendons, and overlying skin must be considered in the evaluation of any fx. Document neurologic and vascular status. Pain (if neurologically intact) and loss of function are common. Perform and document a neurovascular examination by testing capillary refill/pulses and sensation and immediately address any deficit.
Carefully inspect: Wounds that may indicate an open fracture. Tenting of the skin over a displaced fracture. Devitalized skin that may necrose. Common fracture findings (e.g., swelling, ecchymosis). Less common fracture findings (e.g., deformity, fracture blisters). Palpate for point tenderness. Briefly evaluate nearby joints and structures. Be alert for signs and symptoms of compartment syndrome, including early symptoms such as disproportionate pain
Deformity--> change in length, angulation, rotation and displacement.
Dislocation--> occurs when the articular surfaces of a joint are no longer in contact. Subluxation (partial dislocation) is a less severe condition that occurs when the orientation of the surfaces is altered but they remain in contact. Both active and passive motion are limited in all dislocations. There is usually a loss of normal joint contour. The incidence of neurologic injuries is much higher with dislocations than with fx's.

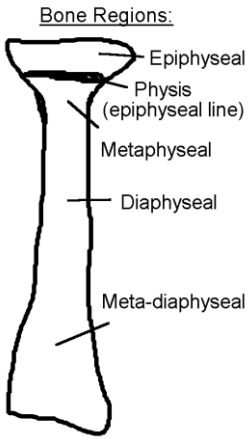
2. Direction of fx line: the radiographic direction of the fx.
Transverse--> perpendicular (right angle) to the long axis of the bone.
Oblique--> fx is at an angle to the bone (fx line in same plane as bone).
Spiral--> secondary to torsional stress (an oblique fx line extending circumferentially around the bone)
Stellate--> fx line radiated from a central point.
Open Vs closed--> an open fx has the bone protruding through the skin. A closed fx has the skin intact over fx sight.
Buckle fx--> one cortex is broken while the other remains intact.



3. Number of fragments: simple (two) or **comminuted** (fx with more than two fragments), **segmental** (prox & distal fragments with middle devascularized segment).

4. Displacement: angulation & distance in centimeter distal fragment with respect to proximal.
Varus = away from body, Valgus = towards, Dorsal = posterior, Volar = anterior.
Anteversion: anterior twist or angulation of the femoral head away from the frontal plane.
Adduction: the movement toward the midline of the body in the coronal plane.
Valgum Angulation: of a bone distal to a joint or in a part of a bone away from the midline. Genu valgum results in knock-knees, because angulation distal to the knees is away from the midline.
Varum Angulation: of a bone or within a bone toward the midline. Genu varum results in bowlegs.
Imaging techniques: Most fx's are adequately visualized on plain radiographs, but some fx's require special imaging techniques for prompt dx and optimal tx. Radionuclide bone scanning is a very sensitive but nonspecific tool in the evaluation of fx's, especially for suspected occult or stress fx's that are not apparent on plain radiographs. Tomography is used to evaluate suspected fx's in bones that are frequently obscured by overlying structures. CT scanning is particularly helpful in confirming fx's of the pelvic and facial bones. MRI is useful to diagnose associated injuries to cartilage, ligaments, and tendons.

5. Anatomical Location:



Epiphysis--> the bony area atop the physis where the cartilaginous area become filled with bone. It serves as a support for the articular cartilage.
Physis--> a cartilaginous growth area in the epiphysis that contains the matrix upon which new bone is formed. It is a relatively weak area compared to the stronger ligamentous attachments between the bones. Thus younger children are more likely to have a fx than a sprain.
Metaphysis--> the flared out region between the shaft and the physis.
Diaphysis--> the tubular shaft of a long bone.
Wormian bones: "Pork Chops": Pyknodysostosis. Osteogenesis imperfecta. Rickets in healing phase. Kinky hair syndrome. Cleidocranial dysplasia. Hypothyroidism / hypophosphatasia. Otopalatodigital syndrome. Primary acro-osteolysis (Hadju-Cheney) / pachydermoperiostosis. Syndrome of Down.
Simple bone cyst: "unicameral" or "solitary" bone cyst. Typically lytic, unilocular central, meta-diaphyseal. The humerus is the most common site. Typically in age 0 - 10 yo. Ddx: bubbly bone lesions.
Joint mice: ossicles within joint space. Can be seen in synovial osteochondromatosis.
6. Salter-Harris (SH) Classification of physeal fx's: For children only. For fx involving epiphysial growth plates.
"SALTER"
S= Slide, straight through the growth plate, Type **1** (fx through physis, between the epiphysis and metaphysis), best prognosis.
A= above, Type **2** (fx through physis, involving the metaphysic / with an attached metaphyseal fragment), 75% of fx's, has good prognosis unless ankle or knee.
L= Low to the growth plate, Type **3** (fx through physis, involving the epiphysis). Cleavage through physis with an attached epiphyseal fragment (fx exits into joint). Good prognosis if nondisplaced.
T= Through the growth plate above and below, Type **4** (fx through physis, involving the metaphysis and epiphysis [across the growth plate]).
ER= Erasing the growth plate, Type **5** = Crush injury to physis, between the epiphysis and metaphysis, worst prognosis.
Ogden VI: Peripheral fx across the physis remaining extra-articular.
Ogden VII: Fx of the epiphysis.
Ogden VIII: Fx of the metaphysis with disruption of the blood supply to the physis.
Ogden IX: Periosteal damage affecting later growth.
Etiology of premature partial growth plate arrest:
Salter-Harris Type 1: 5%
Salter Harris Type 2: 5%
Salter Harris Type 3: 5%
Salter Harris Type 4: 85%
Salter Harris Type 5: 0% ?
Other: Infection: 10%. Tumor: 5%. Iatrogenic (pins, stapes): 2%. Irradiation: 2%. Burns: 1%.
Location of physeal arrest: Distal Femur: 39%. Proximal Tibia: 18%. Distal Tibia: 30%. Distal Radius: 5%. Distal Ulna: 3%. Distal Fibula: 1%. Proximal Humerus: 1%. Proximal Phalanx Great Toe: 1%. Pelvis (tri-radiate): 1%. Distal radius physis is injured most frequently but is an uncommon site of physeal arrest. The distal femur and proximal tibia account for only 3% of physeal injuries but are the most frequent sites of physeal damage and growth disturbance. Physeal fx of the distal femur has at least a 30% chance of developing a growth arrest, 36% have a leg length discrepancy of more than 2cm. Growth disturbance may not become evident for several / many years after the insult, particularly if the injury occurred when the physis was small. (J Ped Orthop; 1982).
Classification Diaphyseal Fx's: (AO)
Type A: Simple fx's with cortical disruption for at least 90% of the bone circumference.
Type B: Multi-fragmentary, wedge fx, always some contact however between the two main fragments.
Type C: Multi-fragmentary, complex fx's with no contact between the two main fragments.
With A, B or C further divided on the basis of the fx configuration:
A1 spiral - C1 complex spiral.
A2 oblique - C2 complex segmental with 50% contact each end.
A3 transverse - C3 complex irregular (number or irregular intermediate fragments).
Articular Fractures:
Type A: Extra-articular, may be intra-capsular but do not involve the joint surface.

Type B: Part of the articular surface involved, the remainder being still connected to the diaphysis.
Type C: Complete articular fx with the articular surface disrupted and completely separated from the diaphysis.

Open Fractures:

30% of pt's with an open fx have other life threatening injuries. Evaluate the extremities for neurovascular function, possible compartment syndrome, and soft-tissue injury, then obtain appropriate X-rays. A widely displaced dislocations of the ankle, subtalar joint, knee, or elbow, are best reduced immediately after pt is given IV pain meds. Open fx-dislocations of the hip and shoulder almost always require general or regional anesthesia. Photograph & cover wound.

Gustilo Open Fracture Classification: (J Bone Joint Surg Am 1976;58A:453)

Type 1: An open fx with a wound < 1cm long and clean.

Type 2: An open fx with a wound more than 1 cm long without extensive soft tissue damage,flaps or avulsions.

Type 3: An open fx with extensive soft tissue damage. An open segmental fx. A traumatic amputation.

IIIa: Adequate periosteal cover of the bone despite extensive soft-tissue damage; high-energy trauma with small wound.

IIIb: Extensive soft-tissue loss with periosteal stripping and bone exposure; usually associated with massive contamination.

IIIc: Arterial injury requiring repair.

Type 4: Adequate soft tissue coverage of a fractured bone despite extensive soft tissue laceration or flaps, or, high energy trauma irrespective of the size of the wound. Extensive soft tissue loss with periosteal stripping and bone exposure-usually assoc with massive contamination. Open fx assoc with arterial injury requiring repair. Injuries in a farmyard or when heavily contaminated by soil are associated with Clostridium perfringens, and wounds exposed to the environment of a lake or a river carry the risk of infection by Pseudomonas aeruginosa or Aeromonas hydrophilia. The neurovascular status of the limb should be carefully evaluated. The distal arterial pulses, capillary refill and overall colour of the limb, and the presence of active bleeding from the wound must be recorded.

Info: Gustilo types IIIb and c frequently require flap coverage of exposed bone. The proximal third of the tibia or knee can often be covered by a pedicled hemigastrocnemius flap. The middle third of the tibia is often covered by a pedicled hemisoleus flap. Large defects of the distal third of the tibia generally require coverage by free muscle transfer.

Tx: After resuscitation and stabilization splint the pt's fx's and cover open wounds with sterile compression dressings. Soak the dressing immediately adjacent to the wound with a dilute povidone-iodine solution, particularly if the pt will not be undergoing immediate debridement. Use traction for femoral fx's. Administer tetanus toxoid (±tetanus immune globulin). Start IV Abx such as cefazolin, the average adult needs a loading dose of 1-2 g, followed by 1 g every 8 h. If farmyard injuries or other open fx's with inc risk of Clostridium, give 4-5 million units of penicillin q 6 h as well. If grossly contaminated wound may need to add IV Gentamycin at 3-5 mg/kg of lean body weight qd divided doses at 8-h (Chapman's Orthopaedic Surgery, 2001 Lippincott Williams & Wilkins). Most common initial contaminants are skin flora (Staph Epidermidis, propionibacterium acnes, Corynebacterium species, Micrococcus) Give IV Abx's for 48-72 hours post injury and again for 48-72 hours each time a further procedure is performed. The open fx should be dealt with in the operating theatre as soon as possible, preferably within six hours of the injury. Care should be taken to avoid the introduction of gross contamination into the intramedullary canal.

Classification Types:

1. Wound less <1cm and compound from within out (0-2% infection).
2. Wounds >1cm clean with little or no devitalized tissue (2-7% infection).
3. Moderate to massive wound, high velocity injuries and those with significant contamination. Adequate soft tissue cover (7% infection). Extensive soft tissue injury bone exposed (10 - 50% infection). Associated with vascular injury needing repair (25 - 50% infection).

Amputation indicated if: Type IIIC with division of posterior tibial nerve. Type IIIC with extensive muscle loss and tissue damage resulting in poor functional expectation. Warm ischemia of >8 hours.

Operative: Debridement and copious irrigation. Small wounds should be extended and excised to allow adequate exposure. Unattached bone should be discarded. For type II and III fx's irrigate with 5-10L of saline. Repeat debridement at 48 hourly intervals. Aim for soft tissue coverage of the wound as early as possible to avoid infection, optimize the milieu for bone healing. Plate and screws are useful in displaced intraarticular fx fixation. External fixation has the advantage of ease of application with minimal operative trauma. Early soft-tissue cover with a muscle flap by a combined orthopedic and plastic surgery service via a radical debridement of the wound outside the zone of injury, skeletal stabilization and early soft-tissue cover with a vascularized muscle flap (Fix and flap: the radical orthopaedic and plastic tx of severe open fx's of the tibia. J Bone Joint Surg [Br] 2000;82-B:959-66). (A review of the management of open fx's of the tibia and femur. J Bone Joint Surg (br) 2006;88:281-289) Severe lower extremity fracture wounds treated with prophylactic negative pressure wound dressing (VAC device costing $400-$500) to increase blood flow had nearly half the rate of infections and wound breakdown as those treated with standard dressings (AAOS meeting 2008;March:Abstract 499) (248 patients with calcaneus, pilon, and tibial plateau fractures as Madigan Army Medical Center).

Traction:

Produces a reduction through the surrounding soft parts which align the fragments by their tension. The spasms and elastic retraction of muscles surrounding the bone tends to produce over-riding of the fragments. Continuous traction generated by weights and pulleys enables maintenance of alignment. Traction may be applied through traction tapes attached to skin by adhesives or by direct pull by transfixing pins through or onto the skeleton. Traction must always be apposed by counter traction or the pull exerted against a fixed object, otherwise it merely pulls the pt down or off the bed. Incr. risk of thromboembolism, decubiti, pneumonia and atelectasis. Once the fx is reduced a decreasing amount of weight is required to maintain a reduction once the muscle stretch reflex has been overcome and the fx immobilized. Need ~ 8-10lbs for a femoral fx.

Skin Traction: used only as a temporary measure as skin is designed to bear compression forces and not shear. If much more than 8lbs is applied for any length of time it results in superficial layers of skin pulled off.

Skeletal Traction: a pin transfixing bone, max 18kg(40lb) can be used.

Traction by Gravity: applies only to fx's of the upper limb (hanging cast).

Fixed Traction: the length of the limb remains constant and there is continuous diminution of traction force as the tone in the muscles diminishes and no further stimuli results in activation of the muscle stretch reflex.

Balanced Traction: The pull is exerted against an opposing force provided by the weight of the body when the foot of the bed is raised.

Combined Traction: May be used in conjunction with fixed traction where the weight takes up any slack in the tapes or cords while the splint maintains a reduction.
Sliding Traction: fastening pt to the raised foot of the bed, the amount of weight is determined by the inclination of the bed.

7. Other Types of Fx's:

Impaction Fx: distal fragment driven/ imbedded into proximal one.
Avulsion Fx: bone pulled out of point where tendon/ ligament/ capsule inserts.
Complete fx: disrupt the entire cortex.
Incomplete fx: only one cortex disrupted.
Stress fx: microfracture due to repetitive submaximal stress.
Greenstick (incomplete) Fx: disruption of one cortex with resultant angulation due to bending of bone. Often associated with a plastic deformity. Younger bone has a decreased density. The cortex and periosteum are disrupted on one side of the bone, but intact on the other.
Torus / Buckle / Cortical (compression) Fx: axial load (longitudinal compressive force) causes cortex to wrinkle or form a bump (often seen in distal radius of child). Involve a bulging or buckling of the bony cortex, usually of the metaphysis. Radiographs show no or subtle disruption of cortex. They are not typically angulated, rotated, or displaced, so reduction is rarely necessary. A compressive force causes a failure in the bone, usually at the metaphyseal-diaphyseal junction of the radius or tibia. These fx's are very subtle clinically and radiographically. Pt's have point tenderness over the fx site and soft tissue swelling. Splinting or casting in a position of function for 3 to 4 weeks with orthopedic follow-up is recommended.
Plastic deformity or Bowing fx: occurs when the bone is bent beyond its ability to recoil and thus it remains in a curved position. Very subtle, need comparative films to dx. Plastic deformities are seen in the forearm and lower leg in combination with a completed fx in the companion bone. The diaphyseal cortex is deformed, but the periosteum is intact.
Pathological Fx's: may be due to: Defective bone (brittle or marble bones). Disused bone (Post traumatic, rheumatoid, paralysis). Diseased bone (Paget's, osteomyelitis, syphilis). Displaced bone (cysts, deposits, fibrous dysplasia etc.). Disordered bone (Osteoporosis, osteomalacia, hyperparathyroid).

Most common fx in child: Physeal (growth plate) and metaphyseal (ends of long bones). These are the weakest areas as ossification is not yet complete, most commonly in clavicle, distal radius/ ulna

Healing of Fractures: A broken bone normally heals within 2 to 3 mo's, it can take longer as people get older. As of 4/05, Medicare will pay for U/S tx's to help mend broken bones in any beneficiary whose fx's won't heal, not just pt's who have undergone failed surgery.
Inflammatory phase: lasts 5-7 days. F/u radiograph at 1-2 wks to asses alignment.
Reparative phase: lasts 4-40 days as a bone callus forms. Radiographically expect to see a callus between day 7-28, it is objective evidence that the fx healing process is occurring as expected. If no callus at the 4wks mark, consider delayed union or nonunion and refer to Ortho.
Remodeling phase: last phase, takes up 70% of healing time and can take up to 1yr. The periosteal callus is converted into lamellar (mature) bone.
Union--> uniting.
Nonunion--> failure to unite, often has sclerotic fx borders.
Delayed Union--> healing occurring after expected time period.
Malunion--> healing with rotational or angular deformity. First fx is associated with increased risk of new fx's (J Pediatr 2005;146:286-8), authors offer sensible and broad guidelines to avoid recurrent fx's: good nutrition (including Vit-D supplementation), avoiding obesity and smoking, and exercising regularly and safely. Exposure to cigarette smoke delays the chondrogenic phase of fx repair as well as ligament healing in mice (J Orthop Res 2006;24).
Ultrasound Device (Exogen 4000+ Bone Healing System): a low-intensity pulsed U/S device that is externally applied 20 minutes a day over the site of a broken bone. It is the only bone stimulator using U/S technology approved to treat fx's that have failed to heal and to heal specific fresh fx's faster. FDA approved in 6/06 as a bone healing system for the noninvasive tx of established fx's excluding skull and vertebra. The low-intensity pulsed U/S device is applied externally for 20 minutes/day over the site of the broken bone. According to a company news release, the system is clinically proven to accelerate healing of indicated fx's by 38% and is effective for 86% of hard-to-heal bone fx's.
Extracorporeal shock wave therapy (ESWT) is a noninvasive and effective method of treating resistant stress fx's in athletes (Am J Sports Med 2007;35:1188-1192)....Successful bone union was achieved in all cases roughly 3 months after ESWT, and the subjects were able to return to their sports between 3 and 6 months after ESWT.

Indications for operative tx: failure of closed methods to reduce the fx, multiple injuries, early mobilization (associated medical problems and high cost can be avoided by early mobilization). Fractured and displaced articular surfaces. Operative tx also minimizes the risk of late degenerative changes.

Abx Prophylaxis prior to Ortho Surgery:

Hip Arthroplasty or Spinal Fusion: usual microorganism are Staph. epidermidis, staph. Aureus. 30 minutes before surgery, customarily stop after hemovac is removed. Cefazolin 1 g IV for 1 dose or q8h for 1-2 dose or Cefuroxime 1.5 g IV for 1 dose or q12h for a total of 6 g. If allergic to penicillin or risk MRSA use Vancomycin 1 g IV for 1 dose.
Total Joint Replacement (other than hip): Cefazolin 1-2 g IV for 1 dose or q8h for 1-2 dose. If allergic to penicillin or risk MRSA use Vancomycin 1 g IV for 1 dose.
Open Reduction of Closed Fractures with Internal Fixation: 30 minutes before surgery give Ceftriaxone 2 g IV/IM for 1 dose. If allergic to penicillin or risk MRSA use Vancomycin 1 g IV for 1 dose.

ICD-9 Codes:

802 Fracture of face bones
802.0 Nasal bones, closed
802.1 Nasal bones, open
802.20 Mandible, closed
805.0 Cervical Fx, closed
805.2 Dorsal [thoracic], closed

805.4 Lumbar, closed
807.00 Rib Fx(s), closed
810. Closed fx of clavicle, .01 sternal end, .02 shaft, .03 acromial end
812 Fracture of humerus
812.00 Fx of humerus - upper end, closed
812.20 Unspecified part of humerus
812.21 Shaft of humerus
813.40 Lower end of forearm, unspecified
814.01 Fx navicular [scaphoid] of wrist
815.00 Fracture of metacarpal bone(s), site unspecified
816.00 Fx phalanx or phalanges, unspecified
824.0 Fx of Medial malleolus, closed
824.2 Lateral malleolus, closed
824.8 Ankle NOS
825.0 Fracture of calcaneus, closed
825.20 Fx unspecified bone(s) of foot [except toes] closed, open 829.1
826.00 Fx of one or more phalanges of foot
829.0 Unspecified bone, closed
733.81 Malunion of fx
733.82 Nonunion of fx
V13.51 Personal history of pathologic fracture
V13.52 Personal history of stress fracture
V15.51 Personal history of traumatic fracture
Fracture of Phalanges of Foot (Toe),(one or more) - Closed 826.0. Open 826.1
Fracture of One or More Tarsal and Metatarsal Bones - calcaneus including heel bone, os calcis - closed 825.0, open 825.1.
Astragalus / talus - closed 825.21, open 825.31
Navicular [scaphoid], foot - closed 825.22, open 825.32
Cuboid - closed 825.23, open 825.33.
Cuneiform, foot - closed 825.24, open 825.34
Fracture of Clavicle including collar bone, interligamentous part of clavicle
Sternal end of clavicle - closed 810.01, open 810.11
Shaft of clavicle - closed 810.02, open 810.12
Acromial end of clavicle - closed 810.03, open 810.13
Unspecified part (clavicle NOS) - closed 810.00, open 810.10
Fracture of Phalanges of Hand (including fingers, thumb; one or more)
Middle or proximal phalanx or phalanges - closed 816.01, open 816.11
Distal phalanx or phalanges - closed 816.02, open 816.12
Multiple sites - closed 816.03, open 816.13.
Phalanx or phalanges, unspecified - closed 816.00, open 816.10
Fracture of Lower (Distal) End of Forearm: Colles' or Smith's fracture - closed 813.41, open 813.51
Other fractures of distal end of radius (alone) - including Dupuytren's fracture (radius) - closed 813.42, open 813.52
Distal end of ulna (alone) - including head, lower epiphysis, styloid process - closed 813.43, open 813.53
Radius with ulna, lower end - closed 813.44, open 813.54
Torus fracture of radius 813.45. Unspecified - closed 813.40, open 813.50
Fracture of Upper (Proximal) End of Radius and/or Ulna: Olecranon process of ulna - closed 813.01, open 813.11.
Coronoid process of ulna - closed 813.02, open 813.12.
Monteggia's fracture - closed 813.03, open 813.13
Head of radius - closed 813.05, open 813.15
Neck of radius - closed 813.06, open 813.16
Fractures of Shaft of Radius and/or Ulna
Closed: radius (alone) 813.21, ulna (alone) 813.22, radius with ulna 813.23, unspecified 813.20
Open: radius (alone) 813.31, ulna (alone) 813.32, radius with ulna 813.33, unspecified 813.30

Casting:

…….END DEMO of this chapter.

Pain Management:

Edited by Robert D. Harbaugh, MD, Neurologist.

Links: Physiology | **Classification** | Pain Hx, Scales, Eval & Acute Pain | **Chronic Pain** | W/u & Neuropathic pain | **Tx of PAIN** | Muscle Relaxers | **Narcotics** | Non-Malignant Pain & Long-term Opioids | **Substance Abusers** | Cancer Pain | **RSD / CRPS** | Anesthesia | **NSAIDs** | Adjuvants | **Weak Opioids** | Combo Agents | **Strong Opioids** | Fibromyalgia | **Topical & Local ANESTHESIA** | Sedation | **Erythromelalgia** | Cervical-Thoracic-Lumbar Pain | **Substances of Abuse / Addiction** | Opiate Abuse / Withdrawal | **Post-Herpetic Neuralgia** | Children | See optho for Pain in eye | **Oral-Facial Pain** | Abd Pain | **OBGYN Pain** | Psychosocial Factors & Dr Sarno | ***INDEX*** |

Approximately 20% of primary care patients experience chronic pain, and pain screening is intended to improve the quality of pain management by systematically identifying patients with pain in clinical settings, but currently there is no commonly accepted gold standard for clinically important pain. Pain as the **"fifth vital sign"** (or 6th if you count pulse Ox): The inclusion of pain level as a vital sign had no impact on pain management, which remained unchanged before and after the implementation of pain as the fifth vital

sign in a group of ~500 pt's reporting severe pain (J Gen Intern Med June 2006;21:607-12)… The authors conclude that using pain as the fifth vital sign alone is not sufficient for improving pain care, and that other measures to improve evaluation and tx of pain are needed. Pain is a leading reason that people seek medical care. Annually, pain is estimated to burden the U.S. economy with $100 billion in direct costs and $61 billion in productivity losses. These losses, which amount to a mean of 4.6 hours weekly, are largely due to diminished performance at work (JAMA 2003;290:2443).

Physiology: Pain is an unpleasant sensory or emotional experience associated with actual or potential tissue damage. Pain is a blend of the physical and nonphysical components and varies depending on whether the pain is acute or chronic in nature and with the pt in whom the pain is occurring. Pain is thought to be an evolutionary protective mechanism, by creating anxiety and limiting activity, pain serves to forewarn of physiologic excess and to prevent tissue damage and organ dysfunction. Pain is a complex integration of noxious stimuli, affective traits and cognitive factors. Remember that "pain is what the pt says it is". The multifaceted, complex nature of pain poses numerous challenges for the clinician engaged in treating pt's suffering from painful conditions. No two pt's will have the same experience of pain, each person who experiences pain does so in a unique, individual way, even if the physical injury is identical to that afflicted upon another. There are no lab tests or objective diagnostic tools to detect the presence of pain, to assess the intensity of pain, or even to determine the source of pain. Opium is made from the dried juice of the opium poppy (Papaver somniferum), with morphine being the principal alkaloid of opium. Understanding the sometimes-striking individual differences in pain response can be elusive, suggested differences can be explained by a biopsychosocial model of pain that includes genetic, social, and psychological factors (American Pain Society 28th Annual Meeting: Abstract 105. Presented May 8, 2009)....."Abundant evidence shows that pain and tissue damage are poorly related, and there are significant differences among individual patients and their perception of pain that extend beyond pathology.....psychosocial factors consistently account for significant variance in pain reports among these patients."

.....too many patients focus on the pain, lie around, become increasingly sedated on medications, and get into a downward spiral that can affect every aspect of their lives.....Patients can become socially isolated, start doing less and less, and may even gain weight.....Some really should get out, get some physical exercise, and get their mind off the pain."

Mediators of pain:

AA's --> glutamate, aspartate.

Peptides --> substance-P, histamine, bradykinin, prostaglandins, calcitonin gene-related peptide (CGRP).

Opioid Receptors:

Mu (u) --> supraspinal analgesia, respiratory depression, euphoria, dependence, bradycardia, decreased GI transit.

Kappa (k) --> supraspinal analgesia, miosis, sedation, respiratory depression, decreased GI transit.

Sigma (o) --> spinal analgesia, respiratory depression, excitation, dysphoria, hallucinations.

Omega --> effects dysphoria, delirium, hallucinations, tachycardia and HTN.

N/OFQ receptor: initially called the opioid-receptor-like 1 (ORL-1) receptor or "orphan" opioid receptor has added a new dimension to the study of opioids.

For acute pain management, women respond better to a kappa agonist (butorphanol) than to a mu agonist (morphine, which men responded better to) (South Med J. 2004;97:35-41), this difference may extend to other mixed-action opioids, such as pentazocine and nalbuphine. HTN may help protect against chronic musculoskeletal complaints, possibly by decreasing pain sensitivity (hypalgesia) (Arch Intern Med 2005;165:916-922)....compared with normotension, a high systolic and diastolic BP was linked to a 10-60% lower prevalence of chronic musculoskeletal complaints. The endogenous opioid dynorphin A can activate non-opioid bradykinin receptors, leading to neural excitation, whichmay be important in the maintenance of chronic pain (Nat Neurosci 2006;Dec). A study based on FDG-PET imaging suggest that arthritic pain has a strong emotional component that is not present with experimental pain conditions (induced by heat application) (Arthritis Rheum 2007;56:1345-1354)…goal for new classes of analgesics for arthritic pain that specifically target the medial pain system = cingulate cortex, thalamus, and the amygdala -- brain regions known for the role in processing fear and emotions. Full agonists also do not antagonize the effects of other full agonists given at the same time. Partial agonists have less of an effect than full agonists at opioid receptors. Partial agonists, such as buprenorphine (Buprenex), are also less effective analgesics because they are limited by the dose-related ceiling effect. Mixed agonist-antagonists block or are neutral at one type of opioid receptor, and are active at another opioid receptor. These mixed agonist-antagonists, such as pentazocine (Talwin), butorphanol tartrate (Stadol), dezocine (Dalgan), and nalbuphine hydrochloride (Nubain), are contraindicated for use in patients receiving an opioid agonist because they can precipitate withdrawal and increase pain. These mixed agonist-antagonists are also less effective analgesics because they are limited by the dose-related ceiling effect.

Ascending Spinal Tracts Relaying Pain Information: Spinoreticular tract. Post-synaptic dorsal column fibers.

1. Spinothalamic Tract: The concept that pain is carried by anterolateral spinothalamic tract alone is rather over-simplified, since this is not the sole tract for the carriage of pain signal as taught in undergraduate medical school curriculum.

2. Spinomesencephalic Tract: This tract arises from the posterior horn and mainly ascends contralaterally. Destination areas include tegmentum, periaqueductal grey matter, superior colliculus and cuneiform regions.

3. Spinoreticular Tract: This ascending tract has wide projections to areas of the brainstem (between medulla oblongata and the midbrain).

4. Post-Synaptic Dorsal Column Fibers: The existence of this pathway, which was first described in 1968 via reports of pain abortion via commissural myelotomy.

Gate Theory of Pain: Pain is modulated at the level of the dorsal column of the spinal cord through a "gate" system, which can be "opened" or "closed" based on: Peripheral pattern of sensory afferent firing. Spinal, supraspinal and/or central influences (modulation) through the effect of the descending endogenous opiate system (DEOS) (Pain 1999;6(Suppl):121–126).Normally, when peripheral nociceptors are stimulated, the evoked response travels through the smaller diameter C and A-delta afferents to the spinal cord, which are in turn relay to higher centers and perceived as pain via pain-transmitting cells (T-cells).

Physiological Responses to Acute Pain: Peripheral vasoconstriction. Increased HR and cardiac workload, sometimes precipitate a cardiac event. Increased sweating. Increased respiratory rate. Increased hospitalisation. n Acute nociceptive pain is sharp and intense (as opposed to visceral pain, which is dull and poorly localized).

Pain as a multidimensional experience: This is especially chronic pain. Pain produced by patterns of nerve firing so-called "neurosignatures" that originate from a widely distributed network some have called body-self neuromatrix. Chronic pain arises from the output of a widely distributed neural network from the brain, as opposed to persistent peripheral sensory output evoked from sensory afferents. This neuromatrix is modified by numerous factors including somatic sensory afferent firing, an individual's

sensory experience and genetic factors. Neurons in our CNS have a capacity to change their connectivity, chemical profile, and hence function.....This neuronal plasticity is believed to contribute to the altered sensitivity to pain (J Neurosci Nurs 1997;29(3):179–186). The cellular-molecular reflection of the pain central sensitization process at the cellular level includes: Increased spontaneous firing of neurons. Increase after discharge to stimuli. Increased degree of response to stimuli. Increased receptive field (thus even nearby areas are sensitized).

Mechanism of "Centralization" in Chronic Pain: Persistent pain can result in sensitization of neurons in the spinal cord dorsal horn and produce physiological changes in sites such as the thalamus that receive projections from the dorsal horn.

Psychogenic Pain: once centralization occrs, the pain is much more difficult to control and thus some of the methods used by psychiatrists in managing chronic pain, such as cognitive-behavioral methods may help. Tertiary amines such as TCA's act to decrease reuptake of serotonin and NE at spinal cord brainstem and dorsal horn nuclei receptors.

Emory Pain Estimate Model:

1 - Low pathology, high behavioral disturbance. These patients may have an element of symptom exaggeration. High verbalization. Occasional drug or substance abuse.

2 - Low pathology, low behavioral disturbance. These patients tend to have no/little identifiable pathology and rather ill-defined complaints.

3 - High pathology, high behavioral disturbance. These patients do have pathology, but their adoption of sick role is at the same time quite dominant or significant.

4 - High pathology, low behavioral disturbance. These individuals have organic pathology and know how to cope well with their condition.

Pharmacodynamic Properties of Opioids:

CNS Effects: Suppression of pain; analgesia. Drowsiness and decreased mental alertness; sedation. Respiratory function depression (at the same dose that produce analgesia). Euphoria. Psychotomimetic effects (nightmares, hallucinations). Suppression of cough; codeine is used primarily as antitussive. Miosis, mediated by parasympathetic pathways. Nausea and vomiting, by activating the brain stem chemoreceptor trigger zone. Antimuscarinic effects by meperedine.

Peripheral Effects: Increased intracranial pressure. Hypotension, if cardiovascular system is stressed. Bradycardia. Decreased peristalsis; constipation. Decreased gastric acid secretion. Inhibition of fluid and electrolyte accumulation in intestinal lumen. Increased tone of intestinal smooth muscle. Increased tone of sphincter of Oddi; increased biliary pressure. Increased tone of detrusor muscle and vesical sphincter. Decreased uterine tone. Stimulation of the release of antidiuretic, prolactine, and somatotropine hormones. Inhibition of luteinizing hormone release. Skin flushing and warming; sweating; itching. Immune system modulation.

Drug Interactions of Some Opioids:

Morphine - MAO Inhibitors: Increased effects of Morphine; anxiety, confusion, respiratory depression, coma

Morphine - Quinidine: Increased toxicity of opiates.

Morphine – Fluoxetine: Fluoxetine attenuated morphine analgesia.

Morphine – Ginseng: Ginseng inhibited the analgesic activity,tolerance to and dependence on morphine.

Pentazocine - Amitriptyline: respiratory depression may be increased by their concomitant use.

Codeine - Glutethimide: when wither is in the high therapeutic range get a possible toxic synergistic effect.

Meperidine - Isoniazid: Isoniazid inhibits monoamine oxidase causing hypotensive episode or CNS depression.

Bisphosphonates: The FDA called attention on Jan. 7, 2008 to evidence suggesting that bisphosphonates (Fosamax, Actonel, Boniva etc) may be associated with the sudden onset of otherwise unexplained severe musculoskeletal pain. Symptom onset varies from a few days to years after initiating (range of one day to 52 months [mean, 91 days; median, 14 days]). In the most severe cases, pain was described as extreme, disabling, or incapacitating, and for some patients the pain was so severe that they were unable to continue their normal activities and required aids for walking. Many patients had relief after the bisphosphonate was discontinued, but while relief was immediate after drug discontinuation in some patients, others had a slower, partial resolution.

Classification of Pain:

Links: Defintions & ICD9 | Pain Hx, Evaluation & Acute Pain | **Chronic Pain** | W/u & Neuropathic pain |

Pain, as defined by the International Association for the Study of Pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP Task Force on Taxonomy. Classification of Chronic Pain, Second Edition. IASP Press;1994). Pain is an experience that comprises a physical sensory component and an affective cognitive component. The physical element involves a nociceptive, neuropathic, or inflammatory stimulus that is transmitted via the spinal cord to the thalamus and then to areas of the cerebral cortex, where it is perceived. Acute pain serves a biological purpose as an alert to potential, immediate, or ongoing injury.

1. Psychogenic pain: lacking organic cause, such as somatoform pain d/o. This is not the same as the emotional response to pain.

2. Organic pain: Has an identifiable cause, two kinds.

A. Nociceptive pain: Tissue damage (cutaneous/ deep tissue or organs) from direct stimulation of intact peripheral afferent nerve via noxious chemical (cytokines, substance P)/ thermal/ mechanical stimuli. Nociceptive pain is typically the result of a musculoskeletal or visceral injury or disease and includes somatic and visceral mechanisms. Usually responds to opioids.

Fast pain is mediated by A-delta fibers, tells us where the pain is and how intense.

Slow pain is mediated by unmyelinated, paleospinothalamic tract, tells us the extent of the injury and modulates the suffering perceived.

Somatic pain--> from skin, muscle or bone. A constant, dull, easily identified/ localized aching or throbbing. Often due to arthropathies (OA, RA, gout), nonarticular inflammatory d/o's (polymyalgia rheumatic, bone mets), skin/ mucosal ulcerations, myalgias (myofascial pain), fx, cuts and ischemic d/o's. Pain is derived from receptors located in the parietal peritoneum and is triggered by such stimuli as touch, cutting, ischemia, pressure, heat, or inflammation, pain is exacerbated by movement. The processing of somatic pain occurs in the cortex and, as a result, has specific localization that is perceived at the respective dermatomal distribution.

Visceral pain -->poorly localize, dull, deep, aching, cramping, twisting, tearing. Often due to AMI, liver mets, gallbladder, constipation, endometriosis or bowel obstruction. Visceral pain is derived from receptors located in the visceral peritoneum and is

triggered by such stimuli as smooth-muscle contraction or spasm, distension or stretching, and ischemia, but not to physical palpation or temperature. Typically, there is no exacerbation or change in pain quality or quantity with movement. Sx's associated with visceral pain include salivation, nausea, vomiting, and sweating. Oxycodone (possessing some effect at the k-opioid receptor) appear to have a better effect in visceral pain than morphine, which probably has lesser effect at the k-opioid receptor (Opioids in modulation of experimental pain in skin, muscles and viscera. Poster presented at the 11th IASP World Congress of Pain. Aug 2005)
Chronic Widespread Pain (CWP): this is the cardinal sx of fibromyalgia. Tends to co-occur with chronic fatigue syndrome, joint pain, headache, irritable bowel syndrome and psychiatric disorders and appears to be mediated by unmeasured genetic and family environmental factors (Arch Int Med 2006;166:1649-54). Always consider osteomalacia (Vit-D def) and hypermobility syndrome (soft tissue laxity in axial skeleton and joints).
Dysesthesia: An unpleasant abnormal / altered sensations, whether spontaneous or evoked.
Paresthesias: An abnormal sensation, whether spontaneous or evoked. Often an electric shock sensation.
Hyperalgesia: An increased response to a stimulus that is normally painful. An extreme exaggerated/ sensitivity to painful stimuli. A heightened appreciation of pain. A pinprick can cause severe pain in a hyperalgesic area.
Hyperpathia: An abnormally painful and prolonged reaction to a stimulus. An appreciation of pain in response to a non-noxious stimulus. A single pinprick may cause minimal pain, but repetitive pinpricks may cause explosive and worsening pain as time passes due to a summation or aftersensation phenomena.
Allodynia: pain with touch or thing that do not normally provoke pain (wearing socks, cold).
Causalgia: A syndrome of sustained burning pain, allodynia, and hyperpathia after a traumatic nerve lesion, often combined with vasomotor or pseudomotor dysfunction and later trophic changes.
Central pain: Pain initiated or caused by a primary lesion of dysfunction in the central nervous system.
Neuralgia: Pain in the distribution of a nerve or nerves.
Neuropathic pain: Pain initiated or caused by a primary lesion or dysfunction in the nervous system.
Noxious stimulus: A stimulus that is damaging to nerves.
Peripheral neuropathic pain: Pain initiated or caused by a primary lesion or dysfunction in the peripheral nervous system
Ref: (Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms. 2nd ed Seattle: IASP Press, 1994)
ICD-9 Codes:
338.0 Central pain syndrome,
338.11 Acute pain due to trauma,
338.12 Acute post-thoracotomy pain,
338.18 Other acute postoperative pain,
338.19 Other acute pain,
338.21 Chronic pain due to trauma,
338.22 Chronic post-thoracotomy pain,
338.28 Other chronic postoperative pain,
338.29 Other chronic pain,
338.3 Neoplasm related pain (acute) (chronic),
338.4 Chronic pain syndrome,
780.96 Generalized pain.
Women: Women are two to three more times more likely than men to suffer from chronic pain and are more likely to have conditions associated with ongoing pain, Conditions such as headache, pelvic pain, fibromyalgia, rheumatoid arthritis, and facial pain are more common in women than in men; accordingly, women are two to three times more likely than men to suffer from chronic pain. Compared with men, women also tend to perceive pain as more severe and to report lower pain thresholds and higher pain ratings in research studies. Such sex differences could be secondary to the influence of hormones, family history, traditional sex roles, cognitive factors, and how the central nervous system processes pain (Sex-related influences on pain: A review of mechanisms and clinical implications. Rehabil Psychol 2003;48:165).
Patients in workmen's compensation cases who are in pain are particularly prone (1.58%) to want to take it out on their physicians in one way or another, even to the point of having thoughts of homicide (AAMP Meeting 2008; Abstract 102). ....the chronic pain pt most likely to harbor the hostile wish is involved in litigation -- and most frequently that is a worker's compensation legal tussle. These patients are forced to see physicians against their will -- often because of dictates of the litigation, and patients don't trust the doctors.....A person with chronic pain, in rehabilitation programs and involved in litigation is 10 times more likely than a healthy person to harbor a wish about killing the doctor, are 8 times more likely to think about suing.
W/u: Neuropathic Pain: …….END DEMO of this chapter.

Pediatrics:

Edited by David Medina, MD, Pediatrician.

Contents: Well Child Visits | **Vaccines** | Fever & Infections | **ENT** | Common Emergencies | **PALS** | Neonates & Babies | **Dermatology** | Behavioral & Developmental Issues | **SYNDROMES** | Endocrine & Growth | **Sleep** | Urology | **Ortho** | Gastro | **Dehydration** | Nephro | **Cardio** | Pulm | **Neurol** | Adolescence, Teen & Puberty | **MEDS** | Child Abuse | **Death of Family Members** | Asthma | **Obese** | Oncology | ***INDEX*** |

Vital Signs & H&P:

<1 d old: HR 93-154/min (mean = 126); RR 40-60/min; BP 67/41.
1-2 d old: HR 91-159/min; RR 40-60/min; BP 67-74/41-43.
3-6 d old: HR 91-166/min; RR 40-60/min; BP 71-76/42-47.
1-3 wk old: HR 107-182/min; RR 40-60/min; BP 65-103/52-71.
1-2 mo: HR 121-179/min (mean = 152); RR 23-39/min; BP 84-106/51-69.
3-5 mo: HR 106-186/min (mean = 139); RR 23-39/min; BP 86-111/53-69.
6-11 mo: HR 109-169/min (mean = 136); RR 23-39/min; BP 93-112/53-73.

1-2 yo: HR 89-151/min (mean = 121); RR 22-31/min; BP 99-109/57-65.
3-4 yo: HR 73-137/min (mean = 98); RR 20-27; BP 100-111/61-71.
5-7 yo: HR 65-133/min; RR 18-24/min; BP 103-116/65-76.
8-11 yo: HR 62-130/min (mean = 86); RR 17-22/min; BP 108-124/71-83.
12-15 yo: HR 60-119/min; RR 14-21/min; BP 116-135/75-86.
15-18 yo: HR 60-120/min; RR 13-21/min; BP 125-140/79-89.
>18 yo: HR 60-100/min; RR 12-20/min; BP <140/90 if healthy, <130/85 if diabetic.
Pregnant: HR 66-120/min; RR 12-20/min; BP <140/90.

History & Physical:

General: age and birth date, gender, and race, the relationship of the child and informant, and some indication of the mental state or reliability of the informant. Ethnic background and telephone numbers of the informants.
Chief Complaint: a brief statement of the reason why the pt is being seen. "Why did you bring the child in?"
HPI: in chronologic order. "The child was well until ____ days before this visit…..followed by a daily documentation of events leading up to the present time, including signs, sx's, and tx, if any. For the well child, a simple statement such as "No complaints" or "No illness" suffices. History of exposures such as school attendance may be pertinent.
PMHx: varies depending on the age of the pt, may be significant either now or later to the well-being of the child, but also to provide evidence of children who are at risk for health or psychosocial problems.
Prenatal Hx: health of the mother during the pregnancy, especially in regard to any infections, other illnesses, vaginal bleeding, toxemia, or exposure to animals, any of which can have permanent effects on the embryo and child. The time and type of fetal movements should be determined. The record should include the number of previous pregnancies and their results; whether radiography was performed, what medications were taken, and whether the mother smoked or abused drugs or alcohol during the pregnancy; results of serology and blood typing of the mother and baby; and results of other tests such as amniocentesis. If the mother's weight gain was excessive or insufficient.
Birth Hx: duration of pregnancy, the ease or difficulty of labor, and the duration of labor may be important. The type of delivery (spontaneous, forceps-assisted, or cesarean section), type of anesthesia or analgesia used during delivery, attendance by other family members at delivery, and presenting part (if known) are recorded. Birth order (if multiple births) and birth weight.
Neonatal Hx: Apgar scores. Jaundice, anemia, convulsions, dysmorphic states, and congenital anomalies or infections in the mother or infant.
Feeding Hx: breast- or bottle-fed and how well the baby took the first feeding. Poor sucking at initial feeding may be sleepiness, but also is a warning sign of neurologic abnormality, which may not become manifest until much later in life. By the second or third feeding, even brain-damaged children usually nurse well. If the infant has been bottle-fed, inquire about the type of formula used and the amount taken during a 24-hour period. Nature of bonding, eye-to-eye contact, the baby's patterns of crying, sleeping, urinating, and defecating. Ages at which solid foods were introduced and supplementation with vitamins or fluoride took place, as well as the age at which weaning occurred and the method used to wean. In addition, note the age at which baby foods, toddlers' foods, and table food were introduced, the response to these, and any evidence of food intolerance or vomiting. If feeding difficulties are present, determine the onset of the problem, methods of feeding, reasons for changes, interval between feedings, amount taken at each feeding, vomiting, crying, and weight changes. With any problem, evaluate the effect on the family by asking, "How did you manage the problem?"
Developmental Hx: physical growth rate is important. Attempt to ascertain the birth weight and the weights & lenghts at 6 mo's, 1 year, 2 years, 5 years, and 10 years (plotted on growth chart). Any sudden gain or loss in physical growth may correspond to the onset of organic or psychosocial illness. May be helpful to compare the child's growth with the rate of growth of siblings or parents. Ages at which major developmental milestones were met (holding the head erect, smiling responsively, reaching for objects, transferring objects, sitting alone, walking with support and alone, speaking the first words and sentences, and experiencing tooth eruption. Ages of dressing self, tying own shoes, hopping, skipping, and riding a tricycle and bicycle, grade in school and school performance). Age at which bowel and bladder control was achieved.
Behavior Hx: The amount of sleep and sleep problems and habits such as pica, smoking, and use of alcohol or drugs. The informant should state whether the child is happy or difficult to manage and should indicate the child's response to new situations, strangers, and school. Temper tantrums, excessive or unprovoked crying, nail biting, and nightmares and night terrors. Question the child regarding masturbation, dating, dealing with the opposite sex, and parents' responses to menstruation and sexual development.
Immunization Hx: types, number, dates, sites given, and reactions.
Hx of Past Illnesses: include any screening tests and any history of infectious or contagious diseases, or any other illness, as well as specific tx, results, and residua. The history of any drug reactions, food allergies, hay fever, asthma, accidents, injuries, and poisonings.
ROS: If information has been obtained previously, simply state, "See HPI" or "See history of past illnesses." Questions concerning each system may be introduced with a question such as, "Are there any sx's related to . . . ?" (head, eyes, ears, nose, mouth, neck…..).
FHx: A genetic type chart (family tree) is easy to read and very helpful. It should include parents, siblings, and grandparents, with their ages, health, or cause of death. Pertinent negative answers should be included also.
Social Hx: family unit, include number of people in the habitat and its size, the presence of grandparents, the marital status of the parents, the significant caretaker, the total family income and its source, and whether the mother and father work outside the home. The family's attitude toward the child and toward each other, the type of discipline used, and the major disciplinarian.
Hx from the Child: Even young children should be asked about their sx's and their understanding of their problem. This also provides an opportunity to observe the child interact with the parent. For most adolescents, it is important to take part of the history from the adolescent alone after asking for his or her approval.

Physical Exam: Link: **Neonate Physical Exam** |

The order of the examination should fit the child and the circumstances. Only the part that is being examined needs to be uncovered and then it can be reclothed.
Vital Signs: see above norms. Temperature is taken in the axilla or rectum in the young child and by mouth after 5 or 6 years of age,

The pulse rate can be obtained at any peripheral pulse (femoral, radial, or carotid) or by palpation over the heart. The respiratory rate should be determined by observing the movement of the chest or abdomen or by auscultating the chest. The blood pressure should be measured with a cuff, with the bladder completely encircling the extremity and the width covering one-half to two-thirds of the length of the upper arm or upper leg. To obtain height and weight recordings, the infant should be measured supine up to the age of 2 years and standing thereafter. Head circumference should be measured in all infants younger than 2 years and in those with misshapen heads.
General: alertness, distress, general development, and nutrition of the child. Mental status, activity, unusual positions, or apprehension. Note any unusual odor.
Skin: color and turgor, the type of any lesions, and the condition of body and scalp hair and nails.
HEENT: shape, symmetry, and any defects of the head; the distribution of hair; and the size and tension of the fontanelles. The fontanelles normally are flat. The posterior fontanelle closes by 2 mo's of age, and the anterior fontanelle closes by 12 to 18 mo's of age. Coarse facies occur with storage diseases. Epicanthal folds occur in a number of syndromes, including trisomy 21.
Eyes: Test vision grossly. Evaluate for strabismus by noting the position of the reflection of light on the cornea from a distant source. Evaluate the range of eye movements, the conjunctivae, the cornea, the size and shape of the pupils, the color, and the response of the iris to light and accommodation. Funduscopic examination, use a zero lens and note the presence of a red reflex, or hemorrhages or pigmented areas, and the size of the veins compared to the arteries.
Ears: Low-set ears may suggest the presence of renal agenesis. Tags and deformities frequently are associated with other minor or major anomalies. Grossly evaluate hearing. Pull the earlobe up and anteriorly. Grasp an otoscope equipped with a bright light so that the hand rests on the child's head and moves with any movement of the head, and use the largest speculum that will fit into the canal. The canal should be clear, and the drum should be pearly gray in color and concave. A cone of light, the malleus, and sometimes the incus can be identified.
Nose: Raise the tip of the nose and look up the nose, record discharges, bleeding, or deformities of the septum.
Mouth: this part of the exam is where the young child usually is most resistant, so perform near the end of the visit. Count the number and note the condition of the teeth. Similarly, note the condition and color of the tongue, buccal mucosa, palate, tonsils, and posterior pharynx. After the age of 2 years, children should not drool (chronic suggests mental deficiency, acute onset is a grave sign of epiglottitis or poison ingestion).
Neck: lymph nodes are normally nontender and up to 1 cm in diameter in both the anterior and posterior cervical triangles. Feel the trachea in the midline. Feel for the thyroid.
Lymph Nodes: palpate inguinal, epitrochlear, supraclavicular, axillary, and posterior occipital nodes. Normally, inguinal nodes may be up to 1 cm in diameter; the others are nonpalpable or < 5 mm.
Chest: shape and symmetry. Respirations are predominantly abd until about 6 years of age, when they become thoracic. Note suprasternal, intercostal, and subcostal retractions.
Breasts: normally are hypertrophied at birth; they regress within 6 mo's and develop with the onset of puberty.
Lungs: palpation, percussion, auscultation. To auscultate the lungs in children, listen with a small bell in small children and with the diaphragm in older children. Normal breath sounds are bronchovesicular and inspiration is twice as long as expiration in young children; breath sounds are vesicular and inspiration is three times as long as expiration in older children.
Cardiac: rate (pulse) and rhythm, and the blood pressure, note the size, shape, sound quality, and presence of murmurs.
Abdomen: Auscultate before percussing or palpating.
Genitalia: note child's stage of pubertal development (estimated from the presence of pubic hair). Inspect the genitalia for urethral discharge.
In boys, testes should be in the scrotum after birth.
Rectum: Inspect for fissures, inflammation, or lack of tone.
Extremities: note any asymmetry, anomalies, unusual size, pain, tenderness, heat, and swelling deformities of the extremities and back.
Neuro: note any abnormalities in gait, position at rest, abnormal movements such as tremors, twitchings, choreiform movements, and athetosis. Cranial nerves can be tested. Examination of tendon reflexes (biceps, triceps, patellar, and Achilles) is less important than is observation of general activity.

"Well-Child" Checks:

Includes: Growth & Developmental **Milestones**, Anticipatory guidance, Screening labs, Safety, Immunizations and Vital Signs (VS).
Links: AAP Guidelines & Pearls | Premature | **Newborn** | 1mo | **2 mo** | 3 mo | **4 mo** | 6 mo | **9 mo** | 12 mo | **15 mo** | 18 mo | **24 mo** | 3-4yr | **5 yr** | 6-12 yo | **Adolescent (>12yo)** | Leading Accidents & PV | **Vaccines** | Growth | **Preparticipation Sports Exam** | Behavioral & Developmental Issues | See ENT for **Hearing Loss Screening** |
The monitoring of children's development is one of the most fundamental activities of pediatric primary care. The process by which pediatric practitioners conduct such monitoring is commonly termed developmental surveillance, and is the means by which providers identify children who have developmental delays or are at high risk of developing such delays. About 18% of children <18 years of age have either developmental delays or chronic physical, behavioral, or emotional conditions that place them at risk for such delays. Greet each family member and introduce self. Always ask: "Do you have any concerns about your child's development?", parents' concerns about their children's development identify children with developmental delays with a sens/spec similar to those for physician-administered developmental screening tests. Have a low threshold for referring children with possible developmental delays, one is are far more likely to leave parents with un addressed concerns about their children's development than to introduce unwarranted concerns. May not be able to screen at every visit, best to conduct screenings in particular areas at key visits. For example: * Family psychosocial risk factors: detailed screening at prenatal/newborn visit, 3 years. * Temperament: detailed screening at the 4-month visit. * Behavior: detailed screening at 2 and 4 years. * Development: detailed screening at 18 mo's and 2½ years. Young children given immunizations during sick visits are 45% less likely to return for well-child visits, potentially affecting their overall preventive care according to a retrospective study (Pediatrics 2008; 121:898)....The authors suggest physicians counsel parents at sick visits "regarding the non-vaccine-related benefits of well-child care.".....many caregivers consider vaccination as the primary reason for well-child visits rather than discussing safety, nutrition, development, and behavior.
Handouts: See http://www.med.umich.edu/1libr/pa/pa_index.htm.
AAP 2007 Guidelines Update: Periodicity Schedule For preventive pediatric healthcare:

(http://practice.aap.org/content.aspx?aid=1599&nodeID=4003)
1. All newborns should be evaluated within 2 to 3 days after discharge.
2. Three routine visits at ages 30 months, 7 years, and 9 years have been added.
3. Calculation of BMI is now recommended to begin at age 2 years.
4. In addition to developmental surveillance, developmental screening has been added back to the schedule and is recommended at ages 9, 18, and 30 months.
5. Autism screening is recommended at ages 18 and 24 months. NOTE: The USPSTF concluded that the evidence was insufficient to recommend screening for speech and language delay.
6. Urinalysis is no longer required.
7. Dental referral (now listed under oral health) should start at age 12 months.
8. Cholesterol screening is now listed as dyslipidemia screening and involves risk assessment (based on family history and physical examination) at ages 2, 4, 6, 8, and 10 years, and then annually through age 21, with dyslipidemia screening performed sometime between ages 18 and 21 years.
9. Sexually transmitted diseases are now referred to as sexually transmitted infections (STIs). All sexually active patients should be screened for STIs.
10. Every visit should be considered an opportunity to update and complete a child's immunizations.

Key Elements to Review in an Annual Preschool Child Exam:

Rhythmicity and daily patterns: Child makes healthy food choices. Family shares meals. Family maintains nighttime rituals. Child regularly brushes teeth.

Emotional growth: Family manages anger and resolves conflicts. Family shows affection. Child makes choices as appropriate. Parents praise good behavior and accomplishments. Family avoids power struggles. Parents set clear and consistent limits. Child has opportunities to play with other children of the same age. Child is provided with transitional objects. Family uses night-light (unless shadows increase a child's fears). Parents provide reassurance if nightmares occur.

Cognitive growth: Parents limit television for child to 1 to 2 hours per day. Parents talk with child to develop vocabulary. Parents read to child to support language. Parents provide toys that child can use creatively. Parents listen with care and respond actively. Parents allow the child to explore.

Self-care: Family encourages self-feeding. Parents anticipate child's interest in genital differences. Parents promote toilet training and hygienic habits. Parents promote physical activities in safe places. Family insists on use of a car seat.

Strength and coordination: Child can exercise big muscles. Child sings and dances to music. Parents provide outdoor play opportunities in safe areas.

Make use of Internet-based resources:

* National Information Center for Children and Youth with Disabilities (NICHCY)http://www.nichcy.org - In addition to its parent-friendly materials for specific developmental conditions, NICHCY maintains State Resource Sheets for all US states and territories, including public education resources, agencies serving children and youth with disabilities, state chapters of disability organizations and parent groups, and parent training and information projects.
* Head Start Program:http://www.acf.hhs.gov/programs/hsb/hsweb/index.jsp - Families with limited incomes may be eligible for center-based services from Head Start, Early Head Start, or Migrant Head Start. Head Start programs reserve 10% of their positions specifically for children with disabilities.
* Pediatric Development and Behavior Homepage http://www.dbpeds.org & * First signs http://www.firstsigns.org - Both of these sites contain references for providers, materials for parents, and links to a number of developmental, mental health, parent training, and community-based resources. For providers, these sites also provide detailed descriptions of quality developmental screening tools, including links to publishers' web sites. (Developmental surveillance in pediatric primary care. Curr Opin Ped 2003;15:624-629).

Anticipatory guidance (Parenting advice): consists of the information that clinicians give families about what they should expect in their child's development, what they should do to promote this development, and the benefits of these healthy lifestyles and practices. It is distinct from counseling, which is advice given in response to specific problems. Covers areas such as parent-infant interaction, sleep patterns and injury prevention. Research indicates that anticipatory guidance about parent-infant interaction, sleep patterns, injury prevention, and reading at home during early childhood is associated with improved child and family functioning (Curr Opin Ped 2003;15630-635). These are "touch points" in pediatric development allow clinicians to help parents through difficult and predictable phases of infant growth. Often, before each burst of development there is a short period in which a baby experiences periods of frustration, is irritable, and displays regressive eating and sleeping behaviors (American Academy of Child & Adolescent Psychiatry 54th Annual Meeting: Dr. Brazelton. October 23-28, 2007).

Interviewing Caregivers Advice: ask about: Location; hours and days open. Openings available. Transportation provided. Costs and financial assistance available. Number and ages of children in care. Meals and snacks provided. Licensing, accreditation or other certification. Convenient time to visit.

When on visits, look for: Positive interactions between caregiver and children. Evidence that children are getting individual attention, are happily involved in activities, and are comfortable with their caregivers. Clean, safe, and healthy indoor and outdoor environment; areas for naps, meals, and toileting. Toys and learning materials that contribute to children's growth and development.

Ask caregiver about: visiting your child during the day. Discipline, sick children, emergencies. Training of staff and substitutes. Immunizations required for children and staff. Their license or other certification (ask to see a copy). Substitute or back-up caregivers. A list of parents who use or have used their care. Ask other parents who use the caregiver about the caregiver's reliability, discipline methods and their child's experience with the caregiver. Choose the best place for your child to be happy and grow. The caregiver who will best meet your child's needs and whose values are compatible with yours. The care must be accessible, affordable and makes you feel good about your decision. Try and talk with the caregiver daily and always talk with your child daily about how the day went. The caregiver should keep you informed about your child's growth and development while in care.

Vaccine (Vac): IPV = inactivated polio. **PCV** = Pneumococcal-7 vaccine. **Hep A** for high risk areas only.

Premature:

Infant born before 37 wk (<259 days) estimated gestational age (time since conception). Chronologic or birth age = time since birth. Corrected age (corrected for prematurity). Premature birth occurs in ~13% of all births in the United States, and that number is

increasing despite extensive public health and private prevention initiatives. ~2% of all premature infants are very premature, defined as birth before 32 weeks' gestation.
Chance of normal to borderline cognition 26% if <24wks. 52% @25wks, 74% @26wks. Very low birth weight may predispose to respiratory problems (chronic cough, wheezing, and asthma) later in life (Arch Dis Child 2003;88:135-138). Deciding whether to take extraordinary measures to try to save an extremely premature infant born at 22 to 25 weeks of gestation may be simplified by a risk algorithm devised by investigators here (http://www.nichd.nih.gov/neonatalestimates)(NEJM 2008;358:1672-81). Daily skin application of sunflower oil significantly reduces (41%) nosocomial infections for preterm infants compared with no tx or Aquaphor (Lancet. Posted online March 3, 2005). Prematurely born infants also are classified by birth weight. MMR and varicella vaccine responses in infants who are extremely preterm are similar to those of term children (Pediatrics 2007;119:e574-e579)...giving these shots on time is the best way to protect these children from these diseases.
Low birth wt --> <2,500g (5 lb 8oz). Low birth weight appears to run in the family.
Very low (VLBW) --> <1,500g (3# 5oz). Most need resuscitation at birth, 60% require endotracheal intubation and 7% receive resuscitation medications. Younger and older mothers (aged ≤15 yo or ≥45 yo) are at increased risk of giving birth to VLBW infants (MMWR. 2005;54:1201-1228).
Extremely low birth wt (ELBW) --> <1,000g (2lb, 3oz).
A very preterm birth is generally defined as <32 weeks of gestation. In the US in 2002, ~12% of live births were born before 37 weeks, 1.96% occurred at a gestational age <32 weeks. 80% of preterm deliveries occur spontaneously as a result of preterm labor (50%) or preterm rupture of membranes (30%), whereas intervention for maternal or fetal problems account for the remaining 20%. Complications typically result from anatomic or functional immaturity and include hypothermia, respiratory abnormalities, patent ductus arteriosus, intracranial hemorrhage, hypoglycemia, necrotizing enterocolitis, infection, and retinopathy of prematurity. The prognosis "remains grim" for ELBW infants who require protracted mechanical ventilation (J Pediatr 2005;146:798-804)....survival rates for ELBW infants ventilated <28 days (85%) than for infants who required ventilation for at least 60 days (75.8%), for more than 90 days (54.2%), or for 120 days or longer (50%). Once they reach their early 20s, most individuals who were born at ELBW are just as likely as their normal-weight counterparts to be working or in school and living independently according to a Canadian study (JAMA 2006;295:667-675). One of the most difficult clinical decisions is whether to initiate intensive care for extremely premature infants. The most important determinants are gestational age and parental wishes, but ascertainment of gestational age is not an exact science.
<25wks Gestation: 14-28% mortality. 6-125-fold risk of intraventricular hemorrhage. 30% risk of chronic lung dz. 3-9% risk of necrotizing enterocolitis. 30% risk of nosocomial sepsis. 7% risk of periventricular malacia. 12% risk of retinopathy of prematurity. Ultimately 79% are normal neurologically, 15% with cerebral palsy, 6% with sz d/o, 4% with severe hydrocephalus. 5% have vision impairment, 9% with hearing impairment (JAMA 2001;286:12). Cognitive impairment is common (41%) at school age for children born extremely premature (birth before 26 wks of gestation) (NEJM. 2005;352:9-19), nearly all extremely preterm infants will have moderate or severe neurocognitive disabilities as children (disabling in 12%, severe in 22%, moderate in 24%, and mild in 34%).
In a report of 1,925 premature infants 23-29 weeks gestation with birthweight >500 g, the gestational age-specific survival was 28%, 51, 68, 78, 90, and 92% for each completed week from 23 to 29 weeks, respectively (Pediatrics 1998;101:975). In those with birthweight 501-600 g, survival increases from 31% at birth to 58, 67, and 75% by days 7, 14, and 28, respectively. Pasteurized donor human milk (milk banks) as a substitute for mothers' own milk offers no short-term benefit over a commercial preterm formula (Enfamil Premature Formula) for extremely premature infants (Pediatrics 2005;116:400-406) (mothers of extremely premature infants often have difficulty providing enough breast milk to meet the needs of the infants throughout their long hospitalization).
Skin-to-skin contact between a mother and her infant in the neonatal intensive care unit (NICU) can reduce stress response (Pediatrics 2005;116:1105-1113) (drop in salivary cortisol levels, heart rate, and pain scores). Umbilical cord milking reduces the need for red blood cell transfusion and for circulatory and respiratory support in very preterm infants (Arch Dis Child Fetal Neonatal Ed 2008;93:F14-F19).....Nearly two thirds of the infants in the milked group required no transfusion at all, whereas 70% in the control group required at least one.....Other benefits associated with cord milking were reduced duration of mechanical ventilation or oxygen supplementation, higher initial blood pressure, and earlier milk feedings. A study of 3702 infants born in 19 U.S. centers that received intensive care found that at an age of 18 to 22 months, 42% had died, 53% had died or had profound neurodevelopmental impairment, and 67% had died or had any neurodevelopmental impairment (NEJM 2008;358:1672).....Factors that reduced the risk for death, profound neurodevelopmental impairment, or any neurodevelopmental impairment among infants who received intensive care were use of antenatal corticosteroids, singleton birth, female sex, and increased birth weight (per 100-g increment).....At an estimated cost of US$3400 per day for neonatal intensive care, finding a way to prevent prematurity would be better.
VS Ranges: Wt in # (kg): 2.2 # (=lb) (1). **HR:** 145. **RR:** 40. **SBP:** 42 ±10. **DBP:** 21 ±8.
Wt in # (kg): 2.2-3.3# (1-2). **HR:** 135. **RR:** 40. **SBP:** 50 ±10. **DBP:** 28 ±8. Retinopathy of prematurity may occur if born @<32wk, get eye screening exam at 4-6wks chronological age.
Vitamin K prophylaxis: The AAP ecommendation of a dose of 0.3 mg/kg of vitamin K1 for preterm infants of birth weight < 1,000 grams. A dose of 0.2 mg (or a dose of 0.3 mg/kg birth weight) vitamin K administered IM - lower than the 1 mg typically recommended - is adequate for maintaining vitamin K sufficiency in preterm infants during at least the first postnatal month or until they have been established on milk feeds (Pediatrics 2006;118:e1657).
Recommendations for small-for-gestational-age (SGA) children: (J Clin Endocrinol Metab. Published online January 2, 2007) Defined as a weight or length -2 SDs or less from the mean, and further subclassified for weight, length, or both. The dx of SGA should be based on accurate anthropometry at birth, including weight, length, and head circumference. Early surveillance in a growth clinic is recommended for those infants who do not catch up quickly. At-risk children should have early neurodevelopmental evaluation and interventions because some children born SGA have cognitive impairment. Although endocrine and metabolic disturbances are recognized in the SGA child, they are infrequent. There is no evidence to recommend routine investigation of all SGA children. For the 10% of SGA children who lack catch-up, tx with growth hormone can increase linear growth. For SGA children with severe growth retardation, defined as height -2.5 or less SD score at age 2 to 4 years, early intervention with growth hormone should be considered at a dose of 35 to 70 µg/kg per day. Long-term, systematic surveillance is essential for those treated with growth hormone. Growth and development monitoring in premature infants should be based on adjusted age, defined as age in months from term due date. Poor growth, especially of the head, is linked to higher risk for neurodevelopmental problems. Preterm growth charts can be used until age 2 years. After discharge from the hospital, premature infants should gain 20 to 30 g/day, human breast milk remains the recommended source of nutrition, but in the short term, the use of enriched preterm formula or

commercially available liquid or powdered breast milk fortifier may improve growth parameters (Am Fam Physician. 2007;76:1159-1164).
Problems associated with premature birth: higher risk for growth retardation; developmental delays; and complex medical issues such as bronchopulmonary dysplasia (BPD) or chronic lung disease, apnea and bradycardia, cryptorchidism, gastroesophageal reflux disease (GERD), sudden infant death syndrome (SIDS), ventriculomegaly, and hernias. At risk for visual disturbances including retinopathy, strabismus, and significant refractive error. Also at greater risk for sensorineural hearing loss and should have universal newborn hearing screening before discharge from the NICU. Most routine vaccination should be administered based on chronologic age.... except for hepatitis B in infants weighing less than 2000 g (70.55 oz), in whom immunization against hepatitis B is dependent on maternal hepatitis B status. Cranial ultrasonography for intraventricular hemorrhage and periventricular leukomalacia is recommended for infants born less than 30 weeks at 7 to 10 days' and 36 to 40 weeks' postmenstrual age. Infants born before 33 weeks gestation are more likely to show physical disabilities and cognitive deficits as they grow, with the degree of impairment linked to gestational age at birth (Lancet 2008;371:813-20).....40% of 5-year-olds who were born at 22 to 32 weeks gestation showed some degree of disability and 34% required special healthcare resources vs only 12% of a comparison group born at full term (39-40 weeks gestation) experienced disabilities and just 16% required special care.....children born at 24 to 28 weeks gestation had generally poorer outcomes than those born at 31 to 32 weeks. Among the specific differences: Overall disability rates, 48% versus 34%. Care in specialized center since birth, 29% versus 12%. Special care at present, 41% versus 29%. Occupational therapist at present, 13% versus 4%. Cerebral palsy, 14% versus 6%.....In the very preterm group, 21% had IQ scores of 70 to 84 vs 8% in the control group (P<0.0001). Skin barrier therapy in the form of topical application of sunflower seed oil or Aquaphor reduces mortality in very premature neonates in hospital settings (Pediatrics 2008;121:522-529)....at the end of the first month, survival rates were 29.4% in controls, 34.2% in the sunflower seed oil group and 45.8% in the Aquaphor group....beneficial effects of emollients could be through improvements in the skin barrier resulting in protection from bacteria and reduced water and heat loss. A study of postural interventions to improve bradycardia and intermittent hypoxia in preterm infants found no advantage to placing infants in a 15-degree head-up tilt or a three-level position (elevation of the thorax) over a standard horizontal position (Arch Dis Chil Fetal Neonatal Ed 2008;93:F289-F291). Research confirms that extreme prematurity (gestational age of 28 weeks or less and birthweight less than 1250 g) is a risk factor for permanent hearing loss ((3.1% at 3 years of age) and goes on to show that the loss is often delayed in onset and progressive (Pediatrics 2009;123:e797-e807).....prolonged supplemental oxygen use is a key predictor of permanent hearing loss, especially severe/profound loss (odds 4.61-fold).....10% had delayed-onset loss and 28% had progressive loss. One child had auditory neuropathy and 29 children (73%) had multiple disabilities.
Compared with those born full term, the most premature babies were more likely to be disabled in adulthood:
Cerebral palsy: 0.1% versus 9.1% (RR 78.9).
Mental retardation: 0.4% versus 4.4% (RR 10.3).
Disorders of psychological development, behavior, and emotion: 0.2% versus 2.5% (RR 10.5).
Other major disabilities such as blindness, epilepsy, or hearing loss: 0.2% versus 4.1%, (RR 19.6).
Receipt of a disability pension: 1.7% versus 10.6% (RR 7.5)(NEJM 2008;359:262-273). The death of a child in the neonatal intensive-care unit (NICU) has lifelong psychosocial consequences for surviving siblings (J Pediatr. 2009;Published online April 3).....Half of the participants reported having repeated death-related nightmares....study subjects perceived being allowed to hold the baby as a great privilege.....surviving siblings have to be considered part of family-centered care.
Newborn: **Milestones:** lies flexed, turns head side to side, head sags on vertical suspension. Fixates to light and close objects. Check for femoral click/ corneal light reflex/ red reflex (all up to 12mo age) to screen for retinoblastoma --leukocoria (white pupillary reflex) is the most common presenting sign (54%), followed by strabismus (19%) (Pediatrics 2003;112:1248-55)..
VS Ranges: Wt in # (kg): 5-7# (2-3). **HR:**125. **RR:** 40. **SBP:** 60 ±10. **DBP:** 37 ±8. **Vac:** Hep B #1.
Link: Basic Care & Pearls |
2 wks: **Milestones:** stares at face and tracks 90 deg horizontally, turns to voices, symmetrical movements. Social: none, language: none.
Parenting advice: normal incr in crying which peaks at 6 weeks. Not all crying represents hunger. Exprected intake volumes of 150-200ml/kg/d in 7 to 10 feedings. Infants regain or exceed birth wt by 2 weeks of age then grow about 1 oz (30 g) per day.
1 mo: **Milestones:** more extended legs, responsive smile (6-8wks), follows moving objects. Check for femoral click/ corneal light reflex/ red reflex (all up to 12mo age).
Hazards: Crib safety (slats < 2-3/8" apart), Scald burns (water-heater temp <120F = <48.8C), MVA injuries (rear facing car seats until >20lb or 9.1kg). Do not leave unattended. Increased risk of foreign body aspiration (keep small toys & objects out of reach).
VS Ranges: 1 mo --> Wt in # (kg): 9# (4). **HR:** 85-200. **RR:** 24-35. **SBP:** 80 ±16. **DBP:** 46 ±16.
Vac: Hep B #1 or Hep B #2.
2 mo: **Milestones:** Raises head slightly further as prone, follows moving objects 180 degrees, smiles on social contact, listens to voice and coos. Check for response to noise, persistent heart murmur, fixing gaze on face, femoral click/ corneal light reflex/ red reflex (all up to 12mo age).
Vac: Hep B #1 or Hep B #2, DTaP #1, PCV, Hib #1, IPV.
Parenting advice: impossible to spoil infant by picking up when cries. Introduce crib mobiles. Expect them to stat reaching, kicking and stretching.
3 mo: **Milestones:** tonic neck posture predominates, reaches forward, but misses objects. Check for femoral click/ corneal light reflex/ red reflex (all up to 12mo age). At 3 months, a baby should, with help, be able to support his or her weight on legs and forearms.
4 mo: **Milestones:** lifts head/ chest up, can pull to sit with head lag, holds rattle then lets go. Rolls front to back (may not occur until later), reaches for objects and "rakes" them in, cooing, stares at one hands, crumples paper joyfully, recognizes sounds and is excited by parents' voice. Posterior fontanelle closes (anterior at 9-18mo). Check for lack of bonding, femoral click/ corneal light reflex/ red reflex (all up to 12mo age). Regards stranger with interest or pleasure.
Vac: Hep B #2, DTaP #2, PCV, Hib #2, IPV.
Parenting advice: Put to bed while still drowsy, do not rock to sleep. Avoid infant walkers if there are stairs in the home. Around this time the baby's visual angle suddenly changes from 18 inches to the whole room. Add some belly playtime. Milk volume is up to 32 oz/d. Do not give juice yet (wait until 6 mo's). Start iron fortified cereal (portion size at 2-4 T BID).

6 mo: **Milestones:** Firm grasp of objects, passes cube from one hand to another. Sits w/o support, pulls, bears some wt on legs, babbles, may laugh out loud, show displeasure if social contacts broken, looks after a fallen object. Lower incisors erupt, then central (all primary by age 2yr). Check for strabismus, 180 deg following objects. By 6 months, the baby should sit independently. Hold the infant up by the shoulders to test neck and truncal tone. Tone is impaired if the baby slips down instead of holding himself up. Another test of tone is horizontal suspension—hold the baby face down by placing a hand under the thorax; if her head or buttocks droop on either side, tone is impaired. Another tip is to look for an inverted V shape to the baby's lips, this indicator of lower facial strength is a subtle sign.
VS Ranges: 6 mo --> Wt in # (kg): 16# (7). **HR:** 100-190. **RR:** 24-35. **SBP:** 98 ±29. **DBP:** 60 ±10. **Vac:** Hep B #3, DTaP #3, PCV, Hib#3, Polio. **Links:** **Teeth** | Teething |
Parenting advice: Routines important, sleep and separation problems may begin. Ok to add 100% juice (2-4 oz/d). Avoid "screen time" (TV, computers). Start baby food vegetables (portion size at 2T BID), then add new food in 3-4 day intervals (observe for allergy).
9mo: **Milestones:** bears wt, sits up alone w/o support, stands holding on to things, pokes and picks up things with forefingers, crawls (may walk first), says "mama/ da-da". Plays peek-a-boo or pat-a-cake, waves bye-bye, inspects objects, then mouths them. Bangs cubes, grabs crayons efficiently.
Labs: Hb to screen for anemia, consider lead screen, nystagmus. Should be able to sit alone, able to babble.
Parenting advice: Avoid feeding battles, give child spoon, continue finger foods. Offer a variety of finger foods and table foods (~850 kcal/d). Watch out for choking hazards. Fruit-veg serving size of 1-2 T or 1/8 cup (1 oz) BID. Variety of protein / lean meat. Cereal 6-8 T/day. Limit 100% juice to 3 oz via a cup. Wash hands often; clean toys. Avoid or limit TV viewing. Reinforce bedtime routine. Supervise near water; empty buckets, tubs, pools. Limit sun; use sunscreen, hat. Turn pot handles to back of stove. Childproof home (dangling cords, poisons, medicines, guns). Supervise near pets, mowers, driveways, streets. Use stair gates, safety locks. Never leave child alone in home or car. Keep poison center number handy. Discuss first aid. Healthy snacks daily. Encourage child to feed self, drink from cup. Let child decide what to eat, don't force eating. Avoid choke foods, limit sugar. Don't use food to comfort, reward. Practice good family oral health habits (brushing, flossing). Brush child's teeth with soft toothbrush, water only. Don't put child to bed with bottle. Allow assertiveness within limits. Keep discipline brief. Expect curiosity about genitals. Spend time with each child. Help child express emotions. Keep family outings short, simple. Don't expect child to share all toys. Help siblings resolve conflicts. Allow older children their own space, toys. A child's language development at age 6-18mo's may actually suffer from early exposure to DVDs and videos (J Pediatr 2007;151:epub)...a paucity of evidence has not stopped producers and marketers of DVDs or videos for babies from making claims of benefits.
Hazards: Poison ingestion's (restrict access to such substances), high mobility (protect from open windows, electric cords, wall sockets, stairways and water hazards.
12 mo: **Milestones:** walks holding hand, rises independently and takes several steps, pincer response, 3 words other than "mama" or "dada" (Jargoning). Plays simple ball games. Willing to explore new environments from safety of parents lap. Looks for hidden object. Releases object to other person on request of gesture. Should be able to transfer objects hand-to-hand, bear wt alone.
Parenting advice: Give 3 meals and 2 snacks per day. Offer a variety of foods but watch out for choking hazards. Limit 100% juice to 4-6 oz/d. Avoid sweetened drinks. Expect finicky eating as child loses baby fat (may only appear to eat one good meal per day. Consider limiting child to 2 milk 8oz bottle of milk/d to stimulate appetite. Start to face car seat forward when >20 lb. (=9.1kg, ~1yo) (rear facing car seats until. Avoid excessive fruit juices as bad for teeth, ok to drink water. Parents or caregivers who suspect a child has ingested a toxic substance should first consult with a local poison center by calling (800) 222-1222. Pediatricians and other healthcare professionals should advise parents to dispose of ipecac in their homes (Pediatrics 2003;112:1180-1185).
Labs: UA and lead level and PPD (if high risk).
VS Ranges: 1 yr --> Wt in # (kg): 22# (10). **HR:** 100-180. **RR:** 20-30. **SBP:** 96 ±30. **DBP:** 66 ±25.
Vac: Hep B #3, Polio, Hib #4, MMR #1, Var #1.
15 mo: **Milestones:** stands, then walks alone, can build tower of 3 cubes and scribble. Follows single-step command without gestures. Responds to names, speaks at least one word. Can indicate needs by pointing, hugs parents. Reads parents expressions to see if safe to explore, uses objects correctly in play. Check grasp/ pincer grip, able to pull self to stand. Crawls upstairs.
Vac: Hep B #3, Polio, Hib #4, MMR #1, Var #1, DTaP #4.
Parenting advice: Equate discipline with teaching, give frequent praise. Providing choices reduces power struggles. Appetite tends to decrease. Aim for 2 cups (16 oz) of whole milk daily. 4-6 oz of 100% juice. 3 meals and 2 light snacks/day. Give a variety of foods.
18 mo: **Milestones:** starts to run, can climb stairs with hand held. Walks backwards. 4-6 block tower, kicks ball, knows 6-10 meaningful words (not just echolalic). Works wind-up toys, understands cause and effect such as on-off buttons. Feeds self, calls for help when needed. Indicates desired objects with index finger not whole hand. Soon able to follow 2-step commands; makes horizontal and circular strokes with crayon; imitates adults. Check closed anterior fontanelle, walks alone, constructive play, spont vocalization.
Vac: Hep B #3, Polio, DTaP #4, Var #1.
Parenting advice: As above. Aim for 5 fruits or vegetables per day. Family mealtime. Avoid excessive juices and sweets / desserts. Best to use water in the sippy cups or bottles if "sucking" on them in order to avoid cavities. Do you have questions/ concerns about his behavior? How is toilet training going? Tell me about his/her eating and sleeping habits. How does he get along with other children?
24 mo (2yr): **Milestones:** climbs stairs/ runs well, jumps, uses spoon well, 7 block tower, 2-3 word sentence/ phrases. Refers to self by name, listens to stories and follows pictures. Should have recognizable words. Points to at least one body part. Understands substitution (in play one object stands for another).
Labs: lead Level, cholesterol.
VS Ranges: 2-3 yr --> Wt in # (kg): 25-30# (12-14). **HR:** 90-160. **RR:** 20-30. **SBP:** 99 ±25. **DBP:** 64 ±25.
Vac: Hep A #1.
Parenting advice: Pick battles selectively to minimize power struggles. Increase in clingy behavior. Can now switch to 1-2% milk. Meal time can be fun if cut foods into shapes. Teach child to wash hands, wipe nose with tissue. Limit TV, watch programs together.

Reinforce bedtime routine. Use bike helmet. Supervise play, ensure playground safety. Provide healthy snacks daily. Eat meals as a family. Offer variety of healthy foods, let child decide, avoid struggles. Don't force eating. Provide child-size utensils. Begin brushing child's teeth with fluoridated toothpaste. Praise good behavior and accomplishments. Hug, talk, read, play together. Learn how to help with fears, nightmares. Begin toilet training when child is ready.

30 mo (2.5yr): Milestones: Walks up and down steps independently, copies, mostly self dresses. Daytime diaper dryness (average age). Combines play actions (able to rock doll and put to bet), names several body parts, able to follow two prepositional commands. Jumps in place; kicks ball; rides tricycle; knows name, age, sex; copies circle, cross; has self-care skills; shows early imaginative behavior.

36 mo (3yr): Milestones: rides tricycle, can briefly stand (balance) on one foot, jumps with both feet off the floor. The preschool child should be able to ride a tricycle and walk on tiptoe. Copies a circle, counts to 3, uses plurals, knows entire (full) name, age and sex. Can build 10 block towers, understands taking turns, can engage in parallel play. Separates from caregivers more easily. Night time diaper dryness (average age).

Check intelligible speech to strangers (3-4 word sentence). Check BP, dental exam, objective VA & hearing (re-check in 6mo in uncooperative).

VS Ranges: 2-3 yr --> Wt in # (kg): 25-30# (12-14). **HR:** 90-160. **RR:** 20-30. **SBP:** 99 ±25. **DBP:** 64 ±25.

Parenting advice: Clean potty chairs after each use. Switch to belt-positioning booster seat in back seat when child weighs 40 lbs. Never place child in front seat with a passenger air bag. Test smoke alarms, change batteries yearly. Limit sun; use sunscreen, hat. Use helmet for biking. Ensure playground safety. Teach stranger safety. Schedule dental appointment. Explain that certain body parts are private. Provide choices, reinforce limits, use "time out". Handle anger constructively, help siblings resolve conflicts. By age 3 years, all 20 primary teeth have erupted.

4 yr: Milestones: Balances on one foot for four to five seconds, understands taking turns, arbitrates dispute with words not hitting. Draws a person with three or more body parts, can engage in role assignments in play. Speaks in sentences, grammar is usually correct, asks questions. Sings songs; knows about things used at home (food, appliances); distinguishes fantasy from reality; gives first and last name; talks about daily activities, experiences; rides a bicycle with training wheels; throws overhand ball. The child can skip and hop on one foot and can throw / catch a ball.

Parenting advice: Teach pedestrian, playground, stranger, neighborhood safety. Know where child is at all times. Limit candy, chips, soft drinks. Serve low-fat dairy products. Model good eating habits. Schedule dental appointment if not already done. Assign chores (toys, setting table). Visit parks, museums, libraries. Encourage assertiveness without aggression. Provide structured learning (preschool, Sunday school). Be sure child learns how to swim. Keep cigarettes, matches, poisons, alcohol, guns, electrical tools locked up and/ or out of reach. Minimize 100% juice to 4-6 oz/d. Give healthy snacks. See: **TV/Video** | Token Economy & House Rules |

5 yr: Milestones: Dresses self without help; knows address and phone number; can count on fingers; copies triangle or square; draws person with head, body, arms, legs; recognizes many letters and can print some; plays make-believe; may be able to skip. Can hop on one foot (5-10sec), cut with scissors, copies a cross, copies square and triangle shape. Understands under/ in front of (prepositional directives), plays cooperatively with others in a group. Dresses with little help, plays out imaginary scripts. Beginning to understand and question surrounding world, correct use of "I" and "me", past tense and plurals.

Parenting advice: Hazards: Unsafe use of toys (teach playground safety), bites from "pets" (teach appropriate respect for animals), Auto / Pedestrian (teach traffic safety accidents). Teach hygiene, hand washing after toileting and before meals. Use helmet for biking, skating. Emphasize pedestrian, neighborhood, stranger, playground safety. Reinforce water safety rules. Teach child emergency phone numbers, home safety rules. Limit high-fat, low-nutrient foods. Limit TV, video, computer time. See: **TV/Video** |

Labs: consider UA, PPD and Hb.

VS Ranges: 4-5 yr --> Wt in # (kg): 35-40# (16-18). **HR:** 60-140. **RR:** 20-30. **SBP:** 99 ±20. **DBP:** 65 ±20.

Vac (4-6yo): Hep A #1 or 2, DTaP #5, MMR #2.

6 yr: Milestones: balances on foot, walks backward, heel-to-toe walking, begins sport and dance, copies diamond shape/ square can draw a man. Counts to 10, uses 10 syllable sentences. Knows primary colors, can identify coins, dresses self, prints letters with help. Simple math, counts 5 items, knows names of all letters, recognizes a few words. Understands rule-based games, has best friend. Anal sphincter control. Permanent teeth start to erupt (32 by teens). Frontal & sphenoid sinuses become aerated.

Hazards: Bicycle accidents (helmets), Sports injuries (protective equip, supervised sports), Water safety (drowning).

Handguns: many 8-12yo boys will handle one if they find it, keep them stored in a manner that renders them inaccessible to children.

Parenting advice: Check that able to perform self care (hand wash, simple dressing, daytime toilet). Ensure adequate sleep. Promote physical activity. School-age youth should have 60 minutes or more of moderate to vigorous physical activity daily (J Pediatr. 2005;146:719-720, 732-737). Limit TV, computer time. Reinforce personal care/ hygiene. Supervise tooth brushing, flossing, consider sealants. Teach family rules, respect for authority, right from wrong, how to resolve conflicts, handle anger. Assign chores and provide personal space. See: **TV/Video** |

VS Ranges: 6-8 yr --> Wt in # (kg): 45-80# (20-36). **HR:** 60-130. **RR:** 18-25. **SBP:** 100 ±15. **DBP:** 60 ±10.

7 yo: Milestones: Understands same and different concept, knows left from right. Masters addition, defines simple adjectives and pronouns, tells more complex stories.

Parenting advice: increase responsibilities at home, assign specific chores, often attach positive outcome to carrying out chores.

8 yo: Milestones: Spells most one syllable words, increasingly complex stories. "Tell me some things you're really good at."

Parenting advice: Encourage physical activity. Teach healthy choices, including fruits and vegetables, limit high-fat, high-sugar foods. Know child's friends and their families. Limit sun; ensure sunscreen use. Home safety. Test smoke alarms. Regular car seat when child is >8yo and 4'9" tall, but back-seat only until >12yo.

9-10 yo: Milestones: Plays sports, repeats four digits in reverse, lists days of the week backwards, has three object recall at 10 minutes. Simple addition, subtraction, multiplication and division calculations, understands time concept, spells simple two syllable words. Parenteral involvement in the adolescents life is critical for keeping high-risk behaviors under control.

Vac (9 to 26yo): Vaccines against HPV.

Parenting advice: Counsel child about avoiding alcohol, tobacco, drugs and inhalants. More critical than ever to know the child's friends and their families. Encourage physical activity. Anticipate some errors in judgment, increased risk-taking. Reinforce water/ biking/ skating safety, protective sports gear use. Limit sun exposure. Set reasonable standards for TV, music, video, computer time. See: **TV/Video** | Teach importance of delaying sexual behavior. Promote interaction/ friendships through team or group activities.

11-12yo: Vaccination recommendations for preteens: The 3 vaccines include meningococcal conjugate (MCV4), tetanus-diphtheria-acellular pertussis (Tdap), and for girls, human papillomavirus (HPV). Can also provide an opportunity to catch up on other vaccines, such as chickenpox, hepatitis B, and measles-mumps-rubella (MMR).

12 yr+ (Adolescents): …….END DEMO of this chapter.

Psychiatry:

Edited by James Moghtader, MD, Psychiatrist.

Contents: Evals, Tests & Definitions | **Psychosis** | Extrapyramidal & Drug SE | **Mood D/o** | Suicide | **Anxiety D/o** | OCD | **Somatoform D/o's** | Eating D/o | **ADHD** | Personality D/o | **Substances of Abuse** | Sleep D/o's | **Domestic Violence** | Factitious Illness | **Various Other D/o (Sexual, Impulse-Control Etc)** | MEDS & ICD-9 | **Violent Pt's** | Psych Emergencies | **Stress Management** | Depression | **Delirium, Dementia & Memory** | Terminology | **PEDIATRIC** | *INDEX* |

Mental Health:

Links: When to Refer to Psychiatry, Involuntary Commitment, Duty to Warn, etc. | **Forms of Therapy** | Bathe Technique, CBT & Medical Hypnosis | **Formal Mental Status Exam (MSE) & Definitions** | Axis Classification & GAF | **Common Terminlogy** | Formal Testing |

The DSM-IV-TR defines mental illness/disorder as a clinically significant behavioral or psychological syndrome or pattern associated with distress or disability…with increased risk of death, pain, disability and is not a reasonable (expectable) response to a particular situation. Mental health is defined as a state of successful performance of mental function, resulting in productive activities, fulfilling relationships with other people, and the ability to adapt to change and cope with adversity (US Surgeon General Report, Dec 1999). Psychiatric d/o's can be defined as a set of functional impairments that result from disturbance of interrelated factors including social & environmental conditions, biologic function (neurotransmitter dysfunction, nutritional deficiencies or toxic agents), psychodynamic adaptation (intrapsychic motivations) and learned behavior (A neurobiologically informed perspective on psychotherapy. Br J Psychiatry 2000;177:117). About half of all Americans will have a mental illness during their lifetime, with sx's beginning in the teen years for many (Arch Gen Psychiatry. 2005;62:593-613).....Lifetime prevalence estimates were 46.4% for any d/o, 28.8% for anxiety d/o's, 20.8% for mood d/o's, 24.8% for impulse control d/o's, and 14.6% for substance use d/o's. Median age at onset was much earlier for anxiety or impulse-control d/o's (11 years) than for substance use (20 years) and mood d/o's (30 years). Of all lifetime cases, half start by age 14 years and three quarters by age 24 years. The majority of people with lifetime d/o's eventually make tx contact. However, this is more often the case for mood d/o's (88.1% - 94.2%) than for anxiety (27.3% - 95.3%), impulse control (33.9% - 51.8%), or substance (52.7% - 76.9%) d/o's. An NIH household survey of 9,282 English-speaking people who were 18 years of age and older showed that 50% of all lifetime cases of mental illness had begun by the age of 14 and 75% had begun by age 24 and that 45% of people with one mental d/o met the criteria for two or more d/o's (Press Release. June 6, 2005. [http://www.nih.gov/news/pr/jun2005/nimh-06.htm]).

The psychiatric dx is like the dx of other medical conditions, there are certain criteria the pt must have, which are listed in the Diagnostics and Statistics Manual (DSM-IV-R). Despite this system, it is often difficult to lump a dimensional mental illness into a neat category. There is a high interrelater reliability with which most psychiatric diagnoses are made, which at times leaves the impression that psychiatrists always disagree. Many primary care pt's who meet a DSM criteria do so only transiently. These "distressed" pt's do not have a psychiatric d/o, often not even an adjustment d/o (DSM utility in primary care. J Clin Psych 2005;7:91-9). In one study, 20% of pt's with at least one psychiatric d/o recovered within 3 mo's (Soc Psych Psych Epidem 1992;27:234-41). 40% of mental health in the USA is provided by primary care. Highly educated workers may be more likely to have mental health problems in comparison to the general public (Am J Health Promot 2003;17). The human brain is extremely complex with 10-20 billions cells, 75 know neurotransmitters (300 putative). The term "mental d/o's," as defined in the DSM-IV, encompasses a broad range of conditions characterized by patterns of abnormal behavioral and psychological signs and sx's that result in dysfunction. Mental d/o's are highly prevalent in medical practice and may present either as a primary d/o or as a comorbid condition. Perceived nervousness and anxiety may predict early mortality and morbidity (J Epidemiol Community Health. 2005;59:794-798) (negative emotions may be risk factors for CAD as well as deliberate self harm). 24% of adults admitted to community hospitals in the United States had mental-health and substance-abuse disorders (Agency for Healthcare Research and Quality, 2007. HCUP Fact Book No. 10. AHRQ Publication No. 07-0008)...Mood disorders were most common in women, whereas substance-related disorders were most common in men....3% of all hospital stays involved a dual dx (mental-health disorder + a substance-related problem). This was most often seen in men (55% of cases) and in individuals aged 18 to 44 years (60% of cases). A CDC study concludes that there is a strong association of mental illness (depression and anxiety) with chronic diseases and related adverse behaviors (2-fold incidence in asthma, CVD and diabetes) (American Psychiatric Association 59th Institute on Psychiatric Services: Poster 166. Presented October 13, 2007). The prevalence of current depression was 8.7%; a lifetime diagnosis of depression was present in 15.7%, and a lifetime diagnosis of anxiety was seen in 11.3%. Depression is on a par with smoking when it comes to increasing risks for mortality, although anxiety may counteract some of this increased risk according to a study (Br J Psychiatry. 2009;195:118–125).....With respect to the association between anxiety and mortality, the researchers found a U-shaped curve with both high and low levels of anxiety symptoms linked to increased risk.....If you're depressed and have no anxiety, that might be a very bad set-up because you're doing very badly physically and you're not worried enough to take measures." Common mental health disorders such as depression and anxiety are strongly linked to an increased risk for obesity — an association that grows stronger as individuals age research on 10,166 civil servants aged 35 to 55 years suggests (Br J Psychiatry. 2009;195:149–155).

The one-year incidence rates per 100 person-years:

Alcohol abuse was 1.03, while the rate for alcohol dependence was 1.72. Blacks are 40% less likely than whites to develop alcohol abuse and Hispanics are half as likely as whites to develop generalized anxiety disorder (Molecular Psychiatry 2008; DOI: 10.1038/mp.2008.41).
Drug abuse was 0.28 and drug dependence was 0.32.
Major depressive disorder was 1.52.
Bipolar I and bipolar II were 0.53 and 0.21, respectively.
Panic disorder was 0.62.
Social phobia was 0.32.
Specific phobia was 0.44.
Generalized anxiety disorder was 1.13 (based on invterviews of 43,093 Americans. Molecular Psychiatry 2008; DOI: 10.1038/mp.2008.41).
Routinely ask friendly questions: How is work going? How are things at home? Are you sleeping well? How is you appetite?
General Eval for a medical cause of psychiatric illness:
Ddx: B12 Def, Thyroid (can take mo's to see effects resolve with Rx), Multiple Sclerosis, Post concussive Syndrome, Drugs, Infection, Lupus Cerebritis, Vasculitis, Syphilis (must do LP with VDRL), Hepatic/Renal Failure, Adrenal Insufficiency, Hypo/Hypercalcemia (depersonalization), Pancreatic / Colon CA.
Ck CBC, comprehensive metabolic panel (glucose, lytes, LFT, Cr), TSH, RPR, B12, ESR.
When to refer to a psychiatrist?: Acute mania or psychosis. When the pt has a dual dx with one being substance abuse. Suicidal. Consider a mental health assessment if you have changed tx's $\geq$3X for the same complaint and the condition still does not improve as the pt is not responding to your therapy. Whenever there is concern of the dx or you are wanting a second opinion.
Involuntary Commitment: Civil commitment as opposed to arrest. Temporary admission. A majority of persons suffering from severe mental illness show limited insight into their illness. Schizophrenic pt's, in particular, may show no recognition that they have a mental illness or need tx. ntreated depression, mania, and psychosis can have devastating effects on both the affected individual and those around him or her: suicide, assaults on others, inadvertent tragedies stemming from delusional thinking, financial and social ruin, and inability to adequately care for one's own needs. Laws for civil commitment vary from state to state. However, most are based on: Danger to self. Danger to others. Grave disability (Inability to provide for oneself their basic needs i.e., food, clothing or shelter). Pt's who are suicidal and have comorbid psychiatric or medical problems, a history of violent or near-lethal suicide attempts, a poor response to outpatient tx, or limited family or social support should be considered for inpatient hospital admission. The term grave disability refers to an inability to care adequately for one's own needs without the assistance of others. some include those without basic needs of food, clothing, shelter, or essential medical care. A pt who was admitted voluntarily and then wishes to leave can be converted to an involuntary pt. A family member or close friend may transport the pt; if the pt refuses, local law enforcement should be contacted. For pt's who require admission due to impaired decision making capacity (minors, mentally handicapped, organic brain disease, toxic states, and psychiatric conditions including suicidality and homicidality). Pt's retain their rights, including the right to refuse medications and other medical tx. Does not imply incompetence, adults are presumed competent unless deemed incompetent by a court of law. (Except in Utah). Does not imply that it is necessary to restrain a pt. Restraint is based on immediate need. Pt's must have access to legal counsel; case may be brought before a judge who can order the pt's release. Periodically case must be reviewed to allow for potential release. Involuntary hospitalization are generally limited to a brief period, usually 3–5 days; the period ranges from only 24 hours in a few states to 20 days in New Jersey. Many states explicitly provide for outpatient commitment, whereas others simply do not prohibit the extension of civil commitment to outpatient programs. Pt's appropriate for outpatient commitment include those who have shown a good response to psychiatric medications in the past, but are noncompliant with medications and other aspects of tx without continued coercion. Involuntary outpatient tx also is indicated for pt's who require considerable structure to their lives and support from others to maintain adequate functioning outside the hospital. For outpatient commitment to be realistically tenable, the facility, often a mental health center, should be capable of adequate outreach. Involuntarily hospitalized pt's may refuse to take medications unless they are specifically found legally incompetent by a court of law. Many states have provisions in their statutes that specifically allow for refusal of electroconvulsive therapy (ECT). Insanity involves judgment of lack of criminal responsibility based on particular standard specified by statute.
Insanity defense: The response of a criminal defendant requesting to be [entirely] excused for misconduct on the basis of mental condition. The "not guilty by reason of insanity" (NGRI) verdict usually involves the conclusion that concealment was not evidence of rational behavior, but that such concealment is regressive behavior driven by psychosis. Gender- and relationship-specific stereotypes often predispose juries to deliver NGRI verdicts for women who kill their children or for children who kill their parents and result in guilty verdicts for men who kill. The legal definition of insanity/mental illness applies the M'Naghten Rule, formulated in 1843 and derived from English law. It says that: a person is innocent by reason of insanity if at the time of committing the act, [the person] was laboring under a defect of reason from disease of the mind as not to know the nature and quality of the act being done, or if he did know it, he did not know that what he was doing was wrong. There are variations of this legal definition by state, and some states have abolished the insanity defense.
Imperfect self-defense: The response of a criminal defendant requesting to be excused for misconduct on the basis of a mental state of self-defense which is itself substantially influenced by a mental disorder or defect.
Diminished capacity: The response of a criminal defendant requesting to be partially excused from mis-conduct on the basis of mental condition. A partial insanity defense that when successful, usually results in a conviction on a lesser charge rather than outright acquittal. For example, a person found to have lacked the mental state necessary to commit first-degree murder (i.e., premeditation) may be convicted of second-degree murder or involuntary manslaughter.
Duty to warn: US case law prescribes that a physician has a "duty to warn" a potential victim if a pt communicates an intent to harm a specific individual. Law enforcement must also be notified. Often above decisions are not clear-cut; in such cases consultation with facility legal team or risk management is warranted.
Not-For Profit Internet Sites for Pt's: For depression and bipolar see www.dbsalliance.org. National Insitute for mental health @ www.NAMI.org. National Mental Health Assoc @ www.NMHA.org. National Institute of Mental Health @ WWW.NIMH.nih.gov

Causes of Abnormal Behavior:

These merely predispose persons to abnormal behavior:

• **Maternal Deprivation:** Especially, orphans who are institutionalized and/or are abandoned by their natural parents.
• **Pathological Family Patterns:** Maladaptive family behavior (e.g., faulty role model) which the child imitates and internalizes; faulty relationship between parent and child (e.g., over-protective, domineering, alcoholic and/or emotionally abusive parents, etc.)
• **Psychic Trauma (Especially in the psychoanalytic view):** An early-repressed childhood traumatic experience is repressed (e.g., abandonment, death or divorce, sexual abuse, etc.) and then resurfaces later as a disorder.
• **Pathological Interpersonal Relationships:** Stressful, anxiety-causing, maladaptive personal relationships (e.g., co-dependent, narcissistic, or control-obsessive), or no relationships at all.
• **Severe Stress:** Extreme pressure on the coping mechanisms which take a destructive toll on psychological processes (e.g., post-traumatic stress disorder resulting from combat, captivity, torture, natural disasters, being kidnapped or terrorized, etc.).
Causes: *Proposed causal explanations for neuroses include the following*:
• **Behaviorist:** Stress is a stimulus for anxiety. The neurotic behavior functions as a negative reinforcer for the amelioration of stress; thus, the resultant reduction in anxiety prompts further avoidance responses which further reinforce the neurotic behavior, thus initiating a vicious cycle.
• **Psychoanalytic:** An unacceptable id impulse triggers anxiety, eliciting the utilization of a defense mechanism as an avoidance mechanism for the anxiety, once again instigating a vicious cycle. Freud claims phobic reactions occurred when anxiety was displaced from an unacceptable object to a neutral object; whereas, behaviorists argue that phobia is caused by classical conditioning. Behavior modification is the most effective treatment for phobias.
• **General Proposed Eclectic Causes:** Faulty personality development due to parental dysfunction or toxic social conditions. Negative self-concept feedback from primary group in early development. Faulty learning and conditioning of maladaptive behavior patterns. Interpersonal theorists argue that a conflict between peer-group norms and parental norms can also cause neurotic behaviors.
Info: As humans attempt to adapt to their environment, maladaptive behavior causes abnormal behavior. The psychological perspective assumes that both normal and abnormal adaptive patterns are learned, not inherited. Thus, treatment consists of substituting healthy adaptive behaviors for maladaptive behaviors. The goal of this approach is to explain abnormality in terms of inefficient and ineffective coping mechanisms. A psychological treatment works only if the patients actively participate in their treatment; the approach is usually ineffective in the case of involuntary institutionalization.
• **Neo-Freudians:** Concentrate on the power of the ego, instead of the id. Emphasized social interactions in the formation of personality, deemphasized instincts and biological factors. In psychotherapy, they emphasize the present, not the past, and tend to advocate shorter periods of treatment. Specific problems that could be readily treated are focused upon.

Main forms of Psychotherapy:

Links: Group | **Supportive |** Client Centered | **Psychodynamic psychotherapy |** Interpersonal | Dialectical-behavioral therapy | **Family Therapy |** Bathe Technique Medical Hypnosis | **Cognitive Behavioral Therapy (CBT) |**
A declining number of office-based psychiatrists in the United States are providing psychotherapy, according to results from a national 10-year survey (Arch Gen Psychiatry. 2008;65:962-970).....dropped from 44% in 1996–1997 to 29% in 2004–2005....the authors note that there are many more types of antipsychotic medications available now and "A recent study showed that psychiatrists in insurance plans cannot afford to provide psychotherapy as third-party reimbursement to psychiatrists is 41% less for one 45-minute psychotherapy session than for three 15-minute "med checks", so insurance policies have a built-in disincentive for psychiatrists to provide psychotherapy"...."Patients may be receiving more psychotherapy from other providers, such as psychologists, social workers, or other therapists.".....Given the pressure to see large numbers of patients briefly and the minimal reimbursement for non–patient-contact activity, true coordination of pharmacologic and psychosocial therapies is rare.....This situation, combined with the vigorous marketing of medications to patients and practitioners, is altering psychiatric practice and, perhaps, the psychiatrist's professional identity.....as psychotherapy becomes less relevant to the actual practice of most psychiatrists, it is evolving into a theoretical topic in residency education.
Group Therapy: viable alternative to costly individual therapy. Best with 6-8 members and 1-2 group leaders. Offers supportive networks. Good for pt's with social skill deficits and interpersonal difficulties. Pt needs to be comfortable with self-disclosure.
Supportive: focuses on offering more direct help such as education or advice. Supports areas of adequate functioning to gain self-esteem, initiate constructive changes or ventilate emotions. Can be brief, but often open ended. The role of the therapist is typically parental, they used empathic reflection, unconditional positive regard and tend to give concrete advice.
Client Centered: based on the premise that humans are essentially good, rational and free. Given a loving, nurturing environment, people will grow in healthy and fulfilling ways. Insight oriented, focuses on the here-and-now experience rather than the past. Goal is for the pt to become fully functioning through the emergence of a positive self-concepts. Some say it lacks scientific concepts. Good for personal growth problems, self-esteem problems, career uncertainty.
Short-term psychotherapies: cognitive-behavioral, interpersonal, and dialectical-behavioral therapies.
Psychodynamic psychotherapy: Long-term, the psychotherapeutic approach most widely used by psychiatrists, often focuses on interpretation of transference and resistance. People with complex mental disorders seem to improve with long-term psychodynamic psychotherapy and suggests that a greater number of sessions, rather than a longer time since baseline assessment, is associated with better outcome according to a meta-analysis of 23 trials (JAMA 2008;300:1551).....long-term psychotherapy can be particularly useful for patients with personality disorders or complex, chronic disorders.....editorialist notes that "Unfortunately, the evidence base is not yet adequate to support the use of this approach as a standard first-line intervention (as many psychodynamically oriented clinicians do)".....more than 50 sessions had better overall outcomes than 96% of patients receiving shorter-term psychotherapy.

Interpersonal Psychotherapy (IPT): based on the theory that psychiatric d/o's derive principally from current interpersonal experience. Improvement in communication and interpersonal skills are assumed to improve self-concepts and lead to more effective coping strategies and better connectedness in relationships. Goal is to minimize conflict among various interpersonal roles, mastering grief reactions, resolve disputes and correct communication deficits. Good for anxiety d/o mood d/o, role changes (career, divorce), interpersonal deficits / conflict and grief / loss. The therapies assumes the role of a coach to empower the pt and adjust interpersonal relationships. Techniques used include primary inventory, problem identification and exploration of interpersonal options. IPT is derived from theories in which interpersonal function is recognized to be a critical component of psychological adjustment and well-being. It is also based on empirical research which has linked change in the social environment to the onset and maintenance of depression.

Cognitive-behavioral (CBT):

Psychodynamic: Focuses on increasing self-awareness and personal development. It assumes that problems in living occur as a result of internal and often unconscious conflicts, which may date back to early childhood. Therapist takes a neutral role (no self-disclosure) to understand both sides of internal conflict. They attempt to make the pt conscious of these unconscious conflicts by interpreting the pt's relationship with the therapist, any resistance to the therapy and links to past relationships and conflict. Good for personality d/o, relationship problems, early childhood & adult trauma, those who fail CBT & interpersonal therapy. The therapist assumes the role of researcher and analyst to assist the pt in developing insight, adopt mature defenses and achieve self acceptance. Techniques include family origins, uncovering defenses and transference.

Dialectical Behavior Therapy (DBT):

DBT is a complex treatment based upon a provisional 'Biosocial Theory'. DBT is usually an outpatient treatment that typically it runs for one year although the program may be repeated. It has four main elements. Three of these are therapeutic activities which involve the pt, namely weekly sessions with an individual therapist, weekly skills training in a group and telephone contact for skills coaching between sessions. DBT is a team treatment and the fourth element is a weekly consultation group in which the team meets together to discuss the patients and the program. Arguably, one of the strengths of DBT is the clear supportive framework that it supplies for patients and clinicians alike. However, the conceptual underpinnings of DBT are varied. Dialectical thinking emphasizes the wholeness and interconnectedness of the world and the potential for the reconciliation of opposites. Includes ideas and techniques drawn from Zen Buddhism. The key concept is that of mindfulness. The person with BPD is seen as having difficulty in being at all detached from her experience and as being frequently overwhelmed by it. Developing the capacity for mindfulness and living in the moment increases the potential for feeling appropriately in charge of the self. Paradoxically, greater mastery is achieved through an increased ability to be detached. The overall stance of the therapist in DBT is that of being a 'consultant to the pt'. The relationship between the pt and the therapist should be quite explicitly a working partnership.

Behavior Therapy Reaction to the unscientific, and unobservable, nature of psychoanalysis and introspective psychology. This approach emphasizes the scientific method in dealing with abnormality, and only observable phenomenon are taken into consideration. Behaviorism ordinarily presupposes that all behaviors are learned through conditioning. Cognitive behaviorism recognizes non-observable phenomenom that also affect behavior, such as thoughts and ideas (it is argued that these can be considered observable phenomena as long as we allow that the observation can be made by "an audience of one,'' namely, the person having the thought or idea). Behaviorism is totally deterministic and denies the existence of free will; its treatment methods are most applicable to specific disorders (e.g., phobias). Behavior therapy includes any technique based upon conditioning principles/theory. Theory does not appeal to any cognitive causes to explain behavior unless reference to cognitive causes can be translated away operationally or "anchored'' to overt observable behavior. "Exemplars'' are frequently utilized as models of research strategy (e.g., Waton's "Little Albert experiment''; Pavlov's classical conditioning of dogs; Skinner's operant conditioning of pigeons, etc.). Behavior therapy is increasingly cognitivistic in its approaches as a response to the tacit recognition that appeals to some mentalistic variables which seem necessary to successfully address some disorders. Thus, systematic desensitization employs techniques of mental imagery; rational emotive therapy studies the maladaptive affect of irrational ideas. Still, these techniques seem best suited for behaviors which can be narrowly operationally specified phobias, sexual dysfunctions, etc.

Family Therapy:

Techniques for increasing insight:

1. Clarification: In using the technique of clarification, the therapist asks the family to elucidate their understanding and/or emotional reactions to present and past events.

2. Confrontation: Confrontation is the pointing out of contradictory aspects of the pt's behavior, often between verbal and nonverbal behavior.

3. Interpretation: Interpretation is the elucidation by the therapist of links between present contradictory behavior and present or past distortions that are out of the awareness of the pt. In makinginterpretations, often immediately preceded by clarification and confrontations, the therapist provides a conceptual link for pt and family. It should be timed so as to maximize cognitive and emotional impact, between current behavior (as distorted or guided by internal templates) and past experiences (distorted by anxiety and defense mechanisms).

Objectives of the first phase:

1. Detail the primary problems and nonproductive family patterns.
2. Clarify the goals for treatment.
3. Solidify the therapeutic contract.
4. Strengthen the therapeutic relationship.
5. Shift the focus from the identified pt to the entire family system.
6. Decrease guilt and blame.
7. Increase the ability of family members to empathize with one another.
8. Assess the family's strengths.
9. Assess the family's preferred style of thinking and working.
10. Define who is in the family.
11. Obtain a clear idea of what ethnic and cultural issues are part of the family's functioning.
12. Determine the life cycle phase for each individual and for the family.

Bathe Technique:

Topics: Therapeutic alliance | **Cognitive Behavioral Therapy (CBT)** | Medical Hypnosis | **Formal Mental Status Exam & Definitions** |

The Bathe technique is a form of psycho-therapeutic intervention for primary care providers that fits into 15min appointment.

Background **-->** What is going on in your life? / Tell me what has been happening? Elicit the context of the pt's visit.

Affect **-->** How do you **feel** about that? / many things happening now, out of all these problems what is the most troublesome for you? (To allow pt to report and label their current state of their feelings).

Troubles **-->** Listen, I understand how you're feeling, but what **troubles** you? **Or** What **troubles** you most (about the situation)? The goal is to focus on subjective meaning of the situation or illness, because you get some sense of why they're there and what you can do to help if you know what it is that's troubling them.

Handling **-->** How are you **handling** that/ it? (the problem). The goal is to asses the pt's functioning and provides direction for an intervention.

Empathy **-->** That must be difficult. / I can understand that you would feel this way. (To legitimize the pt's reaction to the situation). Reassure the pt that he/she has coped well to this point and then express an interest in learning about or understanding the strategies that the pt has developed thus far.

Characteristics of brief psychological intervention: Brief, single session intervention. Suitable for more complex problems, such as in secondary care. Delivered with or soon after clinic attendance. Integrated with usual care. Uses cognitive understanding of health anxiety. Minimizes negative aspects of pt experience. Reinforces positive aspects of pt experience. Provides explicit explanation and reassurance (ABC of psychological medicine. BMJ 2002;325:33-35).

Provider --> ask nothing but bathe Q's, don't allow them to elaborate at length, just summarize briefly and ask the next Q. Don't analyze of give advice, your job is not to fix the problem, just provide support and clarification. If a psychosocial problem is discovered ask them to return to discuss it further, say "Tell me what's been happening since I last saw you?" Consider referral to psychotherapist if not improving in a couple of visits.

Emotionally expressive writing assignments: Pt's with a variety of chronic illnesses should be encouraged to keep journals or engage in other types of writing activities to decompress negative emotions and resolve their negative feelings about the illness. To give full vent (deliberate, expressive, and explicit) to their emotions when they write. Stream-of-consciousness / automatic writing clears away the obstacle and inspires the journal writer to even deeper levels of self-expression. Pt's are asked to write for twenty minutes on each day about a particular subject.....they must not stop writing and must have no concern with spelling, grammar, or sentence structure. Later, they can refer to their own work and gain insight and clarity from their own words.

Therapeutic Relationship / Alliance:

Concerned with the attitudes and the relationship between the mental health professional (MHP) and the client. This relationship comes out of the creation of a safe environment, conducive to communication and trust. An alliance is formed when the professional and the client are working together cooperatively in the best interest of the client. The therapeutic relationship begins the moment the MHP and client first meet.

A therapeutic alliance is essential for positive patient encounters and outcomes. The most important factors for achieving a sound therapeutic alliance are listening to patients sensitively, being empathetic, and building trust and respect. **3 main components:**

1. Agreement on goals, which are the desired outcomes of the therapeutic process
2. Agreement on tasks, which are the steps that will be undertaken to achieve the goals.
3. Bond between client and therapist, which encompasses Rogerian aspects such as trust, respect, genuineness, unconditional positive regard, and empathy.

Core Elements:

1. Communication/rapport – It is important to establish a connection before a relationship can develop. Encouraging the client to speak, using open-ended questions, is helpful. Asking general (not personal) questions can relax the client in an initial session. It is important to project a caring, nonjudgmental attitude.

2. Trust – A core element of a therapeutic relationship. Many clients have experienced disappointment and unstable, even abusive, relationships.

Trust develops over time and remains part of the process. Without trust, a therapeutic relationship is not possible. Other important elements are confidentiality, setting boundaries, and consistency.

3. Dignity/Respect – Many clients have been abused and humiliated and have low self-esteem. If treated with dignity through the therapeutic relationship, clients can learn to regain their dignity.

4. Empathy – Empathy is not sympathy (caught up in client's feelings) but is, rather, open to understanding the "client's perceptions" and helps the client understand these better through therapeutic exploration.

5. Genuineness – Genuineness relates to trust because it says to the client: I am honest, and I am a real person. Again, it will allow the client to get in touch with her/his "real" feelings and to learn from and grow from the relationship.

Cognitive Behavioral Therapy (CBT):

Proposes that sx's of anxiety and depression are mediated by erroneous thoughts (for example, "My heart is pounding—I must be having a heart attack") and maladaptive behaviors (such as avoiding situations that "make my heart pound" to prevent a heart attack). Changing these thoughts (as in, "When my heart pounds, it means that I'm anxious, not that I'm having a heart attack") and behaviors (for example, by using relaxation strategies and facing situations that create anxiety) can alleviate anxiety and depression. The initial focus of CBT is to increase awareness of thoughts and behaviors. Cognitive therapy is a tx process that enables pt's to correct false self-beliefs that can lead to negative moods and behaviors. Focuses on developing new responses and ways of thinking through active learning. Any cognitive therapy recognizes the reciprocal role of cognitions (mental representations in the form of thoughts or images), affect and behavior. The way we think affects the way we feel and behave, which then affect the way we think. Simply put, if our cognitions or interpretations are valid, we feel and react appropriately: if our interpretations are skewed or distorted, we feel and behave in ways that do not reflect reality and can cause difficulties. The aim of CBT is first to help the client to identify the cognitions that underpin problem behaviors and/or emotional states, and then to help that person to reappraise these cognitions. Insights that are evolved in this way are then 'tested', in that the client is encouraged to check out the veracity of the new belief. Insights are developed using guided discovery (or 'Socratic questioning'), often combined with self-monitoring in the form of 'daily thought records'. Clients are taught the technique of appraising automatic thoughts and images, identifying cognitive distortions and substituting statements (or images) that carry greater validity and which do not promote the

problem affect/behavior. Clients are also encouraged to use structured data collection and behavioural tests to evaluate all new perspectives. ABC process is that activating events (A) trigger maladaptive beliefs or behaviors (B), which produce negative emotional consequences (C) such as anxiety and depression. First, the pt accepts that some of his or her perceptions and interpretations of reality may be false (because of past experience or hereditary or biological reasons) and that these interpretations lead to negative thoughts. Next, the pt learns to recognize the negative (surface or "automatic") thoughts and discovers alternative thoughts that reflect reality more closely. The pt then decides internally whether the evidence supports the negative thought or the alternative thought. Ideally, the pt will recognize distorted thinking and "reframe" the situation.6 As cognitive therapy progresses, it focuses more on reframing deeply held or "core" beliefs about self and the world. CBT models suggests that thoughts and behaviors are modifiable and that the pt can learn more rational ways of thinking and more efficient and adaptive behaviors via "cognitive restructuring". Ex: depression pt is taught to monitor and ultimately dispute disparaging views of self and the world and in the future promote more favorable mood states. There therapist role is typically that of a teacher, they identify / challenge cognitive distortions, challenge / replace assumptions and modify behavior. Techniques include graduated exposure, breathing techniques, progressive relaxation and cognitive restructuring. Homework is often assigned to the pt. Frequent visits, but usually time limited. Pt need to be motivated. Good for mood, anxiety, somatoform, substance abuse, personality d/o, sexual dysfunction. Beck Institute for Cognitive Therapy and Research @ http://beckinstitute.org American Institute for Cognitive Therapy @ http://www.cognitivetherapynyc.com

Features of CBT: The pt and therapist actively work together. Tx is directive, time limited, structured, and problem focused. The focus is on the present. Various techniques are used to help pt's modify thoughts, behaviors, and physical responses. CBT helps pt's challenge their thinking by treating their thoughts as hypotheses. This promotes identification of erroneous thoughts, examination of evidence for and against particular beliefs, acquisition of specific skills for challenging maladaptive thoughts, and development of alternative beliefs. Pt's are taught skills that they can continue using after the tx ends to achieve further gains and prevent relapse.

CBT techniques: One initiates CBT by making a functional assessment of the pt's beliefs or thoughts, emotions, physical reactions, and behavior patterns. The information is obtained from the pt's responses to direct questions, the use of self-monitoring, and standardized self-report scales.

Questions can be as simple as, "What was happening in your life when you first noticed sleeping problems/loss of appetite/etc?" To better understand the antecedents of sx's, ask pt's to keep a daily log of sx's relating to mood and anxiety. The information gathered in these first meetings defines tx goals and guides tx planning, including decisions about medications and specialist referrals. For example, if a pt's sleeping problems and weight loss began a month earlier when a friend died, a discussion of a therapy referral might be appropriate.

Tx CBT: involves educating the pt about sx's and teaching self-monitoring techniques. When therapy begins, pt's are taught to identify the 3 components of mood (thoughts, behavior, physical response) and to recognize the ways in which they interact. This is also a time when the clinician and pt can collaborate on developing tx goals. Next, tx options are selected from a range of intervention techniques used in CBT. Not all approaches are appropriate for all pt's or all problems.

Self-monitoring: increases pt's' awareness of their sx's and allows them to keep track of their progress throughout tx. First, the pt needs to be able to identify a troubling situation and recognize concurrent thoughts, physical feelings, and behaviors. The pt then records this information in a daily log. It is important that the record keeping conform to the pt's comfort level and ability: A simple form with check boxes may work well for some pt's. At the beginning of tx, self-monitoring provides information regarding the targeted symptom, such as rapid heartbeat or headaches. Between visits, it records the use and success of implemented interventions, such as medications, the exercise routine, and/or diet changes

Format: List symptom that one in monitoring. List the situation that is occurring when the symptom starts. Describe in details the all physical and emotional sx's. Describe what thoughts are going on while having the symptom. Describe what behaviors are going on while having the symptom.

Review: the pt's physical and emotional sx's and to understand antecedents, consequences, and tx implications. Appropriate tx for a pt who has headaches only when he or she argues with a family member will differ from that for someone who has headaches continuously with a consistent constellation of sx's.

Physical response: to anxiety or depression (including sx's such as muscle tension, rapid heart rate, chills, perspiration, fatigue, and/or restlessness, or just the way the body "feels") are often the easiest sx's for pt's to identify. Breathing retraining and progressive muscle relaxation (systematic tensing and relaxing of muscles) can be helpful ways to cope with physiologic responses. Can teach breathing retraining. See **Stress Management** |

****Ref:** (Bathe Technique. *Am Fam Phys* 1998;57:9) (Writing about stressful events produces sx reduction in asthmatics and rheumatoid arthritis. JAMA 1999;281:1304-1309). (Emotionally expressive writing assignments. APA Annual Convention: Session 2347. Presented Aug. 19, 2005).

Medical Hypnosis:

Link: Self Hypnosis |

Medical hypnosis is the art and science of suggestive therapy. The use of hypnosis and trance-like states date back as far as 3,000 BC in the earliest writings in China, India, Tibet, Greece and Egypt. In 1958, the AMA officially approved the use of hypnosis in medicine and dentistry.

Common Indications: addiction, amnesia, eating d/o, phobias (fear of flying, public speaking etc), pain management, trichotillomania, cephalgia, gagging, dyspareunia and axiety.

Contra: uncooperative pt, language barrier, lack of practitioner experience, paranoid schizophrenia, psychosis.

Equipment: quiet room, comfortable chair, props as needed (pen, pencil or card with yellow & blue rectangles).

Step #1: Preparation: inform the pt that they cannot lose control and that hypnosis is just a guide or catalyst to help them do something they want to do….analogy of the physician as the orchestra conductor and the pt as the musician. The state of hypnosis simply increases ones relaxation and concentration and allows for increased susceptibility to suggestion.

Step #2: Induce a trance: …….END DEMO of this section.

CPT: 90880 Medical hypnotherapy. Further info see the National Procedures Institute @ www.npinstitute.com or www.mdhypnosis.com

The Sedative-Hypnotic Interview:

A rapid and safe method for distinguishing and treating the functional factors that contribute to several types of sx complexes including the following: To resolve conversion sx's to avoid their "crystallization" and permanence. To treat acute panic states following such traumatic events as rape, catastrophic loss, or disaster. To diagnose and treat mute and unresponsive pt's (benign stupor) or pt's with acute hysterical amnesia. To diagnose malingering. To reveal suicidal ideation. To gain information in criminal cases (although this is of presumed dubious merit or legal worth). To differentiate between organic illness or organic psychosis and functional psychosis. To differentiate functional psychiatric d/o's, such as multiple personality d/o's or pseudodementia, from other psychiatric d/o's.

Medical Contraindications: Porphyria or allergy to barbiturates. Severe liver, cardiac, or renal disease. Severe HTN or hypotension (only if >500 mg is used).

Psychiatric Contraindications include a history of barbiturate addiction, under influence of depressant drugs, paranoid reaction, unwilling pt, overeager pt.

Meds used: Sodium amobarbital, thiopental and mixtures of thiopental and amobarbital. Amobarbital sodium is supplied as a dry powder that must be reconstituted with sterile water. In reconstituting the amobarbital, it is important to rotate, not shake, the ampule. Five hundred milligrams of amobarbital sodium should be prepared as a 5%-by-weight solution by diluting 500 mg of the powder in 10 mL of sterile water. Note that because amobarbital has relinquished its role as a mainline anticonvulsant drug, many hospitals no longer keep it in stock. Its main use now is for the neurologic diagnostic Wada test (i.e., direct anesthesia of the hemicortex).

Step #1: …….END DEMO of this section.

Mental Status Exam (MSE):

Links: Format | General | **Behavior |** Orientation | **Language |** Mood | **SI/HI |** Perceptual (Hallucinations etc) | **Thought & Delusions |** Impulse | **Judgment |** Cognition | **Mini-MSE (MMSE) |** Axis Classification and Pearls | **Terminology |** Formal Testing | **Bathe Technique |**

The MSE is a structured assessment of the pt's behavioral and cognitive functioning. The key to accurate neuropsychiatric dx is a comprehensive MSE. Many pt's resist certain questions in formal mental status testing, thus it is frequently omitted unless there is a specific need or, when necessary, defer formal testing until the rest of the examination is done. One can ask a screening question that may be a little too tough. If the pt gets the correct answer, skip the easier questions. If the pt doesn't get the correct answer, gradually reduce the difficulty.

Begin: best to start with a general, open-ended question such as "How can I help you?" or "What brings you here today?" and then allow the pt to talk freely for several minutes before interposing further questions.

Write and Exam up Format:

Identification: name, age, marital status, sex, occupation, language if other than English, race, nationality. Note if any previous admissions for the same or a different condition. Some like to include with whom the pt lives.

CC: a verbatim recording of the pt's reason for seeking tx or evaluation. Conveys information about the person's capacity for insight and self-observation.

HPI: Start evaluation with the history of the present illness. A chronological description of how sx's in the current episode have unfolded over time.

PMHx: meds, surgery, allergies and ongoing problems (with tx's).

ROS: weight changes, appetite, concerns, recent changes.

FHx: including past psych Hx.

Past Psychiatric Hx: all previous episodes and sx's whether treated (and what tx's) or not in chronological order.

Social Hx: Any unmanageable problems in your life, financial, stresses, interpersonal relationships (loner, leader, follower). Describe events of major significance throughout a person's life -- childhood, birth / development, puberty, marriages, education, occupational Hx (summary of the jobs held, the length of time in each and the reasons for leaving), current social situation.

Daily Activities: who do you talk and interact with, religion, work, sleep. What they do with their free time.

Chemical Dependence: ETOH use per week/day. Recreational/ street drugs and method of intake.

CAGE Q's. See **Substances of Abuse |** below.

Expanded Personal Hx:

Early childhood (0-3): Prenatal history and mother's pregnancy and delivery. Any birth trauma / defects, whether pt was planned and wanted.

Feeding habits such as breast-fed or bottle-fed, eating problems. Early development: maternal deprivation, language development, motor development, signs of unmet needs, sleep pattern, object constancy, stranger anxiety, separation anxiety. Toilet training: age, attitude of parents, feelings about it.

Sx's of behavior problems: thumb sucking, temper tantrums, tics, head bumping, rocking, night terrors, fears, bed wetting or bed soiling, nail biting, masturbation. Personality and temperament as a child (shy, restless, overactive, withdrawn, studious, outgoing, timid, athletic, friendly patterns of play, reactions to siblings). Early or recurrent dreams or fantasies.

Middle childhood (3-11): feelings about going to school, early adjustment, gender identification, conscience development, punishment; social relationships, attitudes toward siblings and playmates.

Later childhood (adolescence): Peer relationships: number and closeness of friends, leader or follower, social popularity, participation in group or gang activities, idealized figures; patterns of aggression, passivity, anxiety, antisocial behavior. How far the pt went, adjustment to school, relationships with teachers—teacher's pet or rebellious—favorite studies or interests, particular abilities or assets, extracurricular activities, sports, hobbies, relationships of problems or sx's to any school period.

Psychosexual history: early curiosity, infantile masturbation, sex play. Acquiring of sexual knowledge, attitude of parents toward sex, sexual abuse. Onset of puberty, feelings about it, kind of preparation, feelings about menstruation, development of secondary sexual characteristics.

Adolescent sexual activity: crushes, parties, dating, petting, masturbation, wet dreams and attitudes toward them.

Attitudes toward same and opposite sex: timid, shy, aggressive, need to impress, seductive, sexual conquests, anxiety.

Sexual practices: sexual problems, homosexual and heterosexual experiences, paraphilias, promiscuity.
Adult sexuality: Premarital sexual relationships, age of first coitus, sexual orientation.
Marital history: common-law marriages, legal marriages, description of courtship and role played by each partner, age at marriage, family planning and contraception, names and ages of children, attitudes toward raising children, problems of any family members, housing difficulties if important to the marriage, sexual adjustment, extramarital affairs, areas of agreement and disagreement, management of money, role of in-laws
Sexual sx's: anorgasmia, impotence, premature ejaculation, lack of desire. Attitudes toward pregnancy and having children; contraceptive practices and feelings about them. Sexual practices: paraphilias such as sadism, fetishes, voyeurism, attitude toward fellatio, cunnilingus; coital techniques, frequency
Religious background: strict, liberal, mixed (possible conflicts), relationship of background to current religious practices
Ocupational history: choice of occupation, training, ambitions, conflicts; relations with authority, peers, and subordinates; number of jobs and duration; changes in job status; current job and feelings about it
Military history: general adjustment, combat, injuries, referral to psychiatrists, type of discharge, veteran status
Social activity: whether pt has friends or not; is he or she withdrawn or socializing well; social, intellectual, and physical interests; relationships with same sex and opposite sex; depth, duration, and quality of human relations.
Value systems: whether children are seen as a burden or a joy; whether work is seen as a necessary evil, an avoidable chore, or an opportunity; current attitude about religion; belief in heaven and hell

1. General Description / Appearance: Age (apparent vs. stated age = old/young looking), sex, general health, clothing, grooming, facial expressions, posture (erect/kyphotic), and eye contact (direct/furtive). It is also important to observe the pt's interactions with their family, their speech, movements, posture and behaviors. Is the pt overdressed / underdressed. Wearing excessive make-up. Disheveled, unkempt, ungroomed or immaculate. Cooperative, oppositional, hostile, seductive, impassive. Any unusual movements such as smacking / chewing motions, tremor or pacing. Note any pallor, jaundice, labored breathing or dilated pupils. Pt's with dementia, delirium, or frontal lobe syndromes may appear disheveled and unkempt. Pt's with schizophrenia-like illnesses or secondary mania may be eccentric or flamboyant. Pt's with unilateral neglect following lateralized brain injury may ignore objects or people in one hemi-universe and fail to dress or groom one half of the body. Signs of incontinence may be evident in pt's with frontal lobe syndromes or dementia. May see evidence anxiety (hyperthyroidism), impulsivity (orbitofrontal syndromes), or apathy (medial frontal lobe syndromes). Pt's with delusional d/o's may be suspicious and establish poor rapport. Pt's with orbitofrontal dysfunction may be unduly familiar with the examiner. Pt's with delirium or frontal lobe d/o's may be disinhibited and distractible.

2. Behavior/ Attitude Toward Examiner: Ability to interact with the interviewer. **-->** Agitation, level of consciousness, (AAO X4), drowsy, somnolence, inattentive, distracted, hypervigilant, lethargic, withdrawn, friendly, hostile, evasive, pleasant, calm, bizarre, anxious, effeminate, apathetic, combative, gestures, picking, twitches, psychomotor retardation, evasive, seductive.
Behavioral Analysis: helpful in determining certain aspects of neuropsychiatric condition
Manics - Wear decorative, bright, and colorful clothing. They have reddish cheeks during manic episodes.
Schizophrenic And Chronically Ill Pt's - Appear ectomorphic (slender with fragile physique) wear poor-fitting, unmatched, or soiled clothes.
Parkinson's Disease Or Neuroleptics - have mask like facial expression.
Depressed Or Anxious Pt - may have furrowed forehead over the bridge of their nose.
Sociopath - Have a mesomorphic (large muscle and skeletal mass) appearance, wear offensive tattoos, or similar outfit.
Alcoholics - Have plump, red nose with ruddy complexion, lots of spider nevi on thorax. Nystagmus.
Rashes- Can be due to allergens, psychotic, infection or medications.
Marijuana Intoxication- Produces red streaked conjunctiva secondary to capillary dilatation.
Opiate Intoxication- Associated with pinpoint pupils.
Tardive Dyskinesia - Manifests with nonrhythmic chewing, puckering, and flailing tongue, mouth, or cheek. Can be secondary to neuroleptics.
Dental Hygiene - Poor in mentally retarded and chronic psychiatric pt's.

3. Orientation: Full name, age, date, where are you right now, please tell me what is happening to you at this moment. Degree of eye contact, level of consciousness.
Alertness and Attention: Can test with serial 7's, spelling WORLD backwards, reverse digit span, and reciting the days of the week or mo's of the year in reverse order. 3 item recall to test short-term memory. Have the pt listen to a string of letters in which one letter is repeated frequently but randomly and to tap each time that letter is heard, for example, "Please tap each time you hear the letter K." L K B K M N Z K K T K G B H W K L T K . . .The number of errors the pt makes is noted. Another test might be to have the pt count the number of times a given letter appears on a page full of randomly ordered letters.
Levels of hypoarousal descend from **drowsiness** (fatigued and falls asleep when unstimulated) to **obtundation** (reduced alertness with slow responses and diminished interest in the environment) to **stupor** (must be vigorously stimulated to be aroused and engaged in conversation, which then returns when stimulation ceases) and then **coma** (unarousable unresponsiveness). Seen in toxic-metabolic encephalopathies, increased intracranial pressure, large strokes, encephalitis and traumatic brain injury.
Twilight state: s a well-defined interruption of the continuity of consciousness. Consciousness is clouded and sometimes narrowed. Despite the d/o of consciousness the pt is able to perform certain actions, such as dressing, driving, or walking around. Subsequently, there is amnesia for this state.
Oneiroid state: the pt experiences narrowing of consciousness together with multiple scenic hallucinations. Oneiroid states may occur in schizophrenia, but are also observed in pt's under intensive care who have to be totally passive and dependent on others. The atmosphere is perceived as strange and dreamlike. Accordingly pt's may be aloof and behave like dreamers. Unlike twilight states, the contents of oneiroid states are often remembered.
Hyperarousal with anxiety: autonomic hyperactivity (tachycardia, tachypnea, hyperthermia), and exaggerated startle responses may occur. Seen in withdrawal from alcohol, opiates or sedatives.
Akinetic mutism: a state of silent, alert-appearing immobility that is seen with large frontal lobe injuries, bilateral cingulate gyrus damage and midbrain pathology. Pt may have occasional brief movements to adjust posture.

Locked-in syndrome: occurs with bilateral pontine lesions that render the pt mute and paralyzed. Intellectual function is not impaired and the pt's can communicate by eye movements or eye blinks (blinking once for "yes" and twice for "no").
Catatonic stupor: the pt is mute and motionless. Waxy flexibility may be elicited. Occurs in a wide variety of neurological, toxic-metabolic, and psychiatric d/o's including frontal lobe syndromes, hypoglycemia, schizophrenia and depressive d/o's.

4. Language and Speech: The left perisylvian cortex mediates most aspects of language function in 99% of right-handed individuals and over half of left-handed individuals. Thus, an aphasia implies damage to the left hemisphere about 95% of the time. Basic examination of language function should include an assessment of spontaneous speech, comprehension of spoken commands, reading ability, reading comprehension, writing, and repetition. Confronting (naming), repetitions = (no ifs ands or buts), comprehension, (3 stage command), write a simple sentence.
Note the pt's **speech** rate, quality and quantity, tone, associations, volume, pitch. Described as blunted, restricted, flat, labile, broad, dramatic, loud, mumbled, monotonous, pressured, rapid, hesitant, logical, impaired (stutter, slur, lisping), talkative, whispered. Flow of ideas (relevant, logical, goal directed).
Fluency: using phrases and sentences are of normal length during conversation, are spoken effortlessly and at a normal rate, and have normal grammatical structure). Can be tested by having the pt generate as many words in a given category as he or she is able in a fixed time period. Standard tests ask for such things as "items found in a supermarket" or "words beginning with the letters F, then A, then S."
Comprehension: First the pt is asked complex yes and no questions such as, "Do you take off your clothes before taking a shower?" thereby minimizing the need for motoric and speech acts. Second, questions where gesture alone can be an adequate response are asked, for example, "Point to where people may sit down in this room." finally, the pt is asked to follow a command with a motor response: "Squeeze my fingers" or "Touch your nose."), two steps (eg, "Touch your nose, then stick out your tongue"), and three steps (eg, "Touch your nose, then stick out your tongue, and then raise your right foot"). Complex grammatical structures such as passive voice or possessive may be useful (eg, "Is my aunt's uncle a man or a woman?" "If a lion was killed by a tiger, which one is still alive?").
Word-list Generation: ask the pt to think of as many members of a specific category (category fluency) or as many words beginning with a specific letter (semantic fluency) as possible in 1 minute. Name as many animals names as possible in 1 minute or as many words beginning with the letter "F" as possible in 1 minute (excluding numbers, proper names like France or Frank, and repetitions of words with the same core such as fix, fixed, fixing, and fixation). Normal individuals produce between 12 and 24 animals names in 1 minute (mean: 18, <12 is abnormal) and between 10 and 20 words beginning with the letter "F" (mean: 15; <10 is abnormal). These tests sensitive to anomia and word-finding disturbances, lexical search abnormalities associated with frontal-subcortical systems dysfunction (dementia) and psychomotor retardation.
Reading is tested by having the pt read out loud, listening for errors and testing reading comprehension by having the pt follow a written command, for example, "Close your eyes." Standardized short stories are available that pt's can be asked to read and then later recall. These are scored on the remembrance of key items.
Writing is tested by having the pt sign his name, generate spontaneous sentences, or describe an object in writing.

5. Mood & Affect: Mood refers to an internally experienced pervasive emotion; affect is the outward emotional display. How has your mood been lately / right now (stated mood). Subjective internal emotional tone of the pt (emotion that colors the pt's perception of the world) **-->** ambivalent, angry, anxious, apathetic, aroused, ashamed, awed, depressed, disbarring, distrustful, dysphoric, ecstatic, elated, empty, euphoric, euthymic, frightened, grieving, guilty, happy, helpless, hopeless, hostile irritable, perplexed, optimistic, pessimistic, sad.
Affect: pt's external expression of emotional responsiveness (facial expression, tone of voice, body movements). Pt's display a range of affect that may be described as broad, restricted, labile, or flat. Affect is inappropriate when there is no consonance between what the pt is experiencing or describing and the emotion he is showing at the same time (e.g., laughing when relating the recent death of a loved one). Appropriateness (congruent with mood). Blunted or restricted, labile. Full/ wide range (normal).
Pseudobulbar Affect (PBA): AKA emotional lability, emotionalism, emotional dysregulation. Pt's may manifest with pathological (uncontrolled) laughing and crying (PLC)…without apparent motivating stimulus or in response to a stimulus that would not have elicited such a response before their underlying neurologic d/o. PBA can be a consequence of neurological diseases such as ALS, dementias, MS and traumatic brain injury. The pt appears emotionally normal most of the time will unpredictably burst into tears, grimaces and vocalizes at the slightest provocation (seen with stroke pt, AD, ALS, MS). Also called emotional incontinence / lability, they are generally tx with SSRI (citalopram 10-20mg qd, fluoxetine 20mg qd) or TCA (Am J Geri Pharm 2005;3:S5-14). Dextromethorphan 30mg + quinidine 30mg improved 52% in one study (Mayo Clin Proc 2006;81:1482-86). Not the same as "essential crying", a lifelolng and probably hereditary tendancy to easily cry. Witzelsucht is a when a person has a peculiar addiction to trivial joking and find or seek humor in things that others do not as they constantly laugh, make childish, facetious or sarcastic remarks….often seen in those with right frontal lobe injury. Kluver-Bucy-like syndromes of a marked deficit in affective responding can be seen in bilateral amygdala lesions (due to endephalitis, trauma, dementia, ischemia or porphyria etc). A phase 3 trial found benefits using lower doses of an investigational drug combining dextromethorphan and quinidine (Zenvia, 20/10 mg) in the treatment of pseudobulbar affect (PBA).

6. Suicidal Ideation and Homicidal ideation (SI/HI): …….END DEMO of this chapter.

Pulmonary Medicine:

Edited by Andy S. Binder, MD, Pulmonologist and Intensivist.

Contents: Exam & Testing | **Effusion** | Atelectasis | **Pneumothorax** | Trauma | **Dyspnea / ARDS** | Pleurisy | **Mass / Nodule** | Hemoptysis | **Cough** | Cystic Fibrosis | **Sleep Apnea** | Sarcoid | **Interstitial Lung Dz** | Pulmonary HTN | **Asthma** | Vocal Cord Dysfunction | **COPD** | Bronchitis | **Pneumonia** | Pneumonitis | **Pneumoconiosis** | Transplant | **VARIOUS & Pearls** | Venous Thromboembolism (VTE) / PE | **PFT's** | Lung Ca | **Alpha-1 Antitrypsin** | Pulm Embolism | **O2 therapy** | Hypoxemia | **Hypoxia** | Pulse Ox | **Hypercapnia** | Intubation | **Mechanical Vent** | Disseminated Fungal (Aspergillosis, ect) | **TB** | Smoking | **Costochondritis** | Sleep D/o's | **Bronchiolitis (RSV)** | Hyperventilation Syndrome | ***INDEX*** |

MEDS: Asthma | **COPD** | Cough | **Pneumonia** | O2 therapy |

An early life history of bronchitis, pneumonia, or asthma is associated with 57% higher respiratory disease mortality in adulthood, according to a longitudinal study in the UK (Thorax 2008;Feb). Patients with chronic respiratory illness are at increased risk of CVD, diabetes, and hypertension (Eur Respir J 2008;32:962-969)....>half of patients with COPD have at least one of these comorbid conditions.
Acetaminophen use: is associated with risk of asthma, COPD, and decreased lung function, according to the results of a cross-sectional analysis (Am J Respir Crit Care Med. 2005;171:966-971)(deplete pulmonary antioxidant levels)(OR of asthma was 1.4 it took 6-29X/mo and 1.75 if took >30X/mo [OR = 1.16 for COPD])...concomitant use of aspirin attenuated the prevalence of asthma in acetaminophen users. Retrospective results suggest a relation between acetaminophen use in infancy and development of asthma (OPR = 1.46)(Lancet 2008;372:1039).....Use in the previous 12 months showed a significant dose-response association with asthma symptoms (ORs ranged from 1.61 for use once in the past year to 3.23 for monthly use).....Acetaminophen use also was associated with increased risk for eczema and rhinoconjunctivitis symptoms.....However, an association does not necessarily prove cause and effect as the analysis could not control for two important variables — reporting bias (parents of wheezing children might be more likely to give them acetaminophen) and recall bias (parents of children with asthma might be more likely to recall giving the drug).
Past Nutrition: A lack of fruit and fish in teenagers' diets may keep them from attaining full lung capacity and set them up for later respiratory problems (Chest 2007;132:238-245) (18% of teens at risk of not getting enough antioxidant-containing foods, OR of increased risk of chronic bronchitic sx's, wheeeze and asthma at 1.37, 1.34 and 1.68).
Psych Info: There appears to be a high prevalence of anxiety and depression among pt's with chronic breathing d/o's (Chest 2005;127:1205-1211) (of 862 pt's screened with PRIME-MD, 65% had both, 80% had either, 10% had anxiety only, and 5% had depression only).
Neck Pain: The results of a study support the hypothesis that patients with chronic neck pain could have a predisposition toward respiratory dysfunction (Cephalalgia 2009;29:701-710)....."Abdominal and chest breathing was related to neck pain.....Patients with neck pain had a statistically significant decreased maximal voluntary ventilation compared to controls.....provides support to the belief that the clinician should have a 'holistic view' of the patient, as other systems could have also been influenced."
Pearls: Hemoptysis should be investigated in smokers over 40. Early morning headache in a patient with lung disease may be due to carbon dioxide retention. A stuffy nose with chest symptoms could indicate Wegener's granulomatosis. An asthmatic with nasal polyps might be hypersensitive to NSAIDs. Adults with a history of low birth weight (LBW) are more prone to respiratory illnesses requiring hospitalization than are adults without such a history according to a population-based, case-control study (Am J Respir Crit Care Med 2009;180:176-180).

Respiratory System Physiology:

1. **Function:** Exchange O2 and CO2.
2. **Mechanics of breathing:** Ventilation of the lungs occurs by muscular **contractions/relaxations** that alter pressure within the thoracic cavity.

Gas Exchange:
1. **Dalton's Law:** The pressure of a gas mixture is equal to the sum of the separate or partial pressures (e.g., Air = 1 atmosphere or 760mm Hg: pN2 = 78% or 593mm Hg; pO2 = 21% or 160mm Hg).
2. **Henry's Law:** Each gas in a mixture will dissolve proportionally to its partial pressure.
3. **Alveolar air:** The composition of air reaching the alveoli helps determine the dynamics of gas exchange with the blood.
4. **O2 transport in blood:** Only 3% can dissolve in plasma; 97% of O2 is transported by hemoglobin.
- The binding of O2 to Hb is influenced by several factors:
• pO2, pH (**Bohr effect**), pCO2, temperature.
• **Active tissues** have low pO2, low pH, high pCO2 and high temperatures, all of which increase oxygen delivery.
5. **CO2 transport in blood:** Only 7% can dissolve in plasma; 23% binds to hemoglobin; 70% transported as HCO3.

The Pulmonary Exam:

Links: Physical Exam & Abnormalities | **Breath Sounds** | Pulmonary Function Testing & Spirometry | **Bronchoscopy / BAL** | Labs & Capnography | **CT** | PET Scan | **CXR Analysis & Common CXR Lesions** | Thoracentesis | **Arterial Blood Gas (ABG)** | G-stain | **Sputum** | Sleep Monitoring |

Physical Exam of the Chest:

Links: Respiratory Rate | **Common Disorders** | Inspection | **Sternum** | Accessory Inspiration | **Palpation** | Percussion | **Auscultation** | Auscultation Errors | **Disease Processes** | Breath Sounds |
4 Steps: Inspection. Palpation. Percussion. Auscultation.
Assessing Respiratory Rate:
Equipment: A watch with a second hand.
Step #1: Place the patient in a position of comfort, preferably sitting. Place the patient's arm in a relaxed position across the abdomen or lower chest, or place your hand directly over the patient's upper abdomen.
Step #2: Observe the complete respiratory cycle (consists of one inspiration and one expiration). Once a cycle is observed, monitor the watch's second hand and begin to count the rate of respirations.When the second hand reaches a number on the dial, count "one" to begin the first cycle. Timing of the respirations begins with a count of 1. Respirations occur more slowly than the pulse; therefore, the count begins with 1.
Pearls: For adults, count the number of respirations in 30 seconds, then multiply by 2. For infants or young children, count the respirations for 1 full minute.
The respiratory rate is equivalent to the number of respirations per minute. If an adult's respirations have an irregular rhythm or they are abnormally fast or slow, count for 1 full minute. While counting, note whether the depth of respirations is shallow, normal, or deep and whether the rhythm is regular or altered. The character / pattern of ventilatory movements may indicate specific alterations in respirations or disease states.
Normal Ventilation Frequency (rate)—The number of breaths per minute:
Term infant: 30–80.
6-month-old: 30–60.

Pediatric: 20–40.
Adolescent: 15–25.
Adult: 12–20.
Muffled voice (supraglottic abscess), hot potato voice (oral abscess, Ludwig's angina), barking cough (laryngotracheobronchitis), monotone, hurried sentence with inspiratory pause or suppressed laughter/cough (bilateral vocal cord paralysis).

Summary of Typical Physical Findings of Common Disorders:

Bronchial asthma (acute attack): Inspection: Hyperinflation; use of accessory muscles. Palpation: Impaired expansion; decreased fremitus. Percussion: Hyperresonance; low diaphragm Auscultation: Prolonged expiration: inspiratory and expiratory wheezes.
Pneumothorax (complete): Inspection: Lag on affected side. Palpation: Absent fremitus. Percussion: Hyperresonant or tympanitic Auscultation: Absent breath sounds.
Pleural effusion (large): Inspection: Lag on affected side. Palpation: Decreased fremitus; trachea and heart shifted away from affected side. Percussion: Dullness or flatness Auscultation: Absent breath sounds.
Atelectasis (lobar obstruction): Inspection: Lag on affected side. Palpation: Decreased fremitus; trachea and heart shifted toward affected side. Percussion: Dullness or flatness Auscultation: Absent breath sounds.
Consolidation (pneumonia): Inspection: Possible lag or splinting on affected side. Palpation: Increased fremitus. Percussion: Dullness Auscultation: Bronchial breath sounds; bronchophony; pectoriloquy; crackles.

1. Inspection: Sternum | **Ventilatory Patterns** | Evaluate the contours.

Kyphosis --> extensive flexion of the thoracic spine (hump-back). ~80% of cases of kyphoscoliosis are idiopathic. Idiopathic kyphoscoliosis commonly begins in late childhood or early adolescence and may progress in severity during these years of rapid skeletal growth. The incidence of kyphoscoliosis in females is four times higher than that in males. The remaining 20% of cases of kyphoscoliosis are found in association with neuromuscular d/o's (e.g., syringomyelia, neurofibromatosis, or poliomyelitis), congenital vertebral defects (e.g., hemivertebrae), acquired vertebral abnormalities (e.g., tuberculous spondylitis or osteomalacia), or deforming chest wall processes (e.g., sequelae of empyema or as a result of thoracoplasty). Severe deformities (scoliotic angle > 100 deg) can be associated with prominently restricted lung volumes; typically, total lung capacity is reduced to 50% or less of the predicted value. Such restriction may lead to chronic alveolar hypoventilation, hypoxemia, pulmonary HTN, and, ultimately, cor pulmonale.

ICD-9 Codes:
738.3 Acquired deformity of chest and rib
737.10 Kyphosis (acquired) (postural)
737.20 Lordosis (acquired) (postural)
737.30 Kyphoscoliosis and scoliosis
737.9 Unspecified curvature of spine

Deformities of the sternum: seen in <5% of the general population and appears to be more common in pt's with other evidence of structural or connective tissue disease, such as scoliosis, Marfan syndrome, Poland syndrome, or Pierre Robin syndrome. Surgical correction if evidence of lung restriction or cardiovascular dysfunction. Pectus excavatum: an inward concavity of the lower sternum. Pectus carinatum: an outward protuberance of the upper, middle, or lower sternum.

Pectus carinatum (Pigeon chest): sternum is displaced forward, seen in Marfan's, rickets and normals.
Link: Ventilatory Patterns |

Pectus excavatum (Funnel Chest): A posterior angulation, funnel chest or inward depression of lower sternum (measure the indentation from the surface to its deepest depression.). It may push heart to the L simulating an erroneous ventricular enlargement. Affects 2% of the pop 1/3 genetic, M:F, 3:1, r/o Marfan's, 15% have scoliosis. May lead to mild decrease in total lung capacity, significant if have diminished exercise tolerance, easy fatigability, inability to take a deep breath, inc frequency of URI's. Can cause heart displacement that leads to fatigue, dyspnea with mild exertion, chest discomfort and tachycardia.
W/u: of excavatum: Check PFT's (best at max exercise) to r/o restrictive lung dz. Consider ECG as rare association with WPW syndrome, may see Q waves. Echo if have murmur (r/o MVP or cardiac compression).
Tx: Reassurance that many will cosmetically improve as one develops more pectoral muscle mass at puberty. Usually only progresses in a small percentage. Must consider Marfan's, Rickets, scoliosis and congenital heart disease. If asymptomatic can follow through puberty with annual CXR and PFT's looking for restriction of either lungs (volumes) or heart (AP diameter). Hold any surgery until after 4 yo, correction during pre-pubertal period is ideal. The decision for surgery to quantify the cardiorespiratory complications are PFT's, echocardiogram and MRI with breath-holding. Scarring from surgery can limit thoracic expansion, newer endoscopic, minimally-invasive procedures may be best. (J Ped Surg 2002;37:437-45) A modified Ravitch repair with minimal cartilage resection is effective for all variations of pectus excavatum in pt's of all ages, resulting in a stable postoperative chest wall, relatively mild postoperative pain, and a short recovery with good physiologic and cosmetic results (Ann Surg 2004;240:231-235). Surgery to correct pectus excavatum can result in significant improvements in cardiovascular function according to a meta-analysis (Chest 2006;130:506-516). Surgical repair of pectus excavatum can dramatically improve body image as well as physical and psychosocial functioning according to a report (Pediatrics 2008;122:1218-1222)....Overall, 97% of patients thought that the surgery improved the appearance of their chest,....the patients' mean body image score improved from 2.30 to 1.40 and the physical difficulty score from 2.11 to 1.37.

Barrel chest --> AP diameter > Lat, seen in COPD.

Kyphoscoliosis: a combination of scoliosis, a lateral curvature with rotation of the vertebral column, and kyphosis, an anterior flexion of the spine—are usually found in combination. 80% of cases are idiopathic, which commonly begins in late childhood or early adolescence and may progress in severity during these years of rapid skeletal growth. F:M is 4:1. ~20% of cases of kyphoscoliosis are found in association with neuromuscular d/o's (syringomyelia, neurofibromatosis, or poliomyelitis), congenital vertebral defects (hemivertebrae), acquired vertebral abnormalities (tuberculous spondylitis or osteomalacia), or deforming chest wall processes (sequelae of empyema or as a result of thoracoplasty). See ortho section.

Thoracic Insufficiency Syndrome: in 2004, FDA approved an implantable metal rod device (Vertical Expandable Prosthetic Titanium Rib), to correct chest wall instability in pediatric pt's aged six mo's to vertebral maturity (14 years in girls, 16 years in boys) with thoracic insufficiency syndrome caused by severe deformities of the chest, spine, and ribs. The curved metal rod is attached by hooks at both ends to ribs near the spine. By straightening the spine and separating the ribs, the device allows expansion of the

chest cavity for breathing and lung development. The implanted device must be lengthened or replaced every six mo's as the pt matures.

Anatomy: The bony thorax consists of 12 ribs. The 11th and 12th ribs are "floating" ribs and are not attached directly to the sternum. Ribs 1 to 5 are directly attached to the sternum by costal cartilages. The lower ribs (6 to 10) coalesce by way of the costal cartilages into the costal arch. The first rib is flat and travels from the first thoracic vertebra to the manubrium at the manubrium-clavicular junction. Through this relatively small area pass the great vessels, trachea, esophagus, and nerves. The remaining ribs gradually slope downward. The intercostal muscle layers assist with respiration and protect the thoracic structures. The extrinsic muscles of the chest, the latissimus dorsi muscle, serratus anterior muscle, the pectoralis major and minor muscles, and the cervical muscles (sternocleidomastoid, scalene muscles) attach to the bony thorax and protect the chest wall itself.

Accessory Inspiration:

Sternocleidomastoid, scalenes, abd muscles.

Supraclavicular Retraction: indrawing of soft tissue above clavicle.

Intercostal: indrawing of skin between ribs.

Subcostal: on or just below the costal margin. Distinguished from **chest wall indrawing** that is normal inward mov't of ribs during inspiration in age <2yo as more compliant, best viewed laterally.

Seesaw (paradoxical) breathing: abd moves out as chest moves inward as inspire. Have a paradoxical movement of chest wall, see a down and inward movement of the costal margin (often see in with airway obstruction or stridor) as inspire. (Vs nl up and out as diaphragm moves).

Nasal flaring during inspiration, **grunting** during expiration (partially close vocal cords to inc lung volume and alveolar pressure, suggesting impending respiratory failure. **Listen** to front, back and **sides** of infants. Best finding for r/o pneumonia in child is absence of tachypnea, observation is the most important exam, auscultation is unreliable.

2. Palpation: chest expansion @ nipple level >2" in adults, respiratory mov't with thumbs meeting in front of chest, mediastinal position per tracheal position in suprasternal notch.

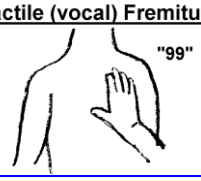
Tactile (vocal) Fremitus "99, 99" to check vibratory conduction through lung tissue with ulnar border of *same* hand:

Inc --> consolidation, pneumonia.

Dec --> effusion, empyema, atelectasis, pleural thickening, PTX, cavitary lesion, emphysema.

Soft tissue crepitus --> subcut/ mediastinal emphysema.

Tactile (vocal) Fremitus:



3. Percussion: with middle fingers, check normal lung resonance.

Resonance: Normal air/tissue density.

Increased (hyperresonance): cavity, emphysema, air trapping, pneumothorax.

Decreased: Flatness: effusion (usually highest at the axillary line), empyema, consolidation (pneumonia, tumor, infarct), pleural thickening or pneumonectomy. **Dullness:** Consolidation? Atelectasis? Pleural fluid?

Tympanic =-ringing, over air in stomach or distended intestines.

4. Auscultation:

Links: Wheeze | **Rhonchi** | Crackles (Rales) | **Stridor** |

Alterations in lung mechanics with postural changes tend to be small in healthy individuals (Chest 2008;133:729-736)....Body position is therefore not a major variable in the measurement of lung sound intensity in this population.

Breath sounds: pt should breath at normal tidal volume as inspiration of the residual volume yields abnormal findings in 50% of normals. Check the quality and intensity. Then ask them to take deep breaths as if trying to make the paper towels dangling in the dispenser to move. The diaphragm detects higher pitched sounds, and the bell lower pitched, though if the bell is tightly pressed against the body, the taught underlying skin itself may serve as a "diaphragm" and improve perception of higher pitches. Full contact with the skin is necessary for best listening, which may pose a problem in a pt whose intercostal spaces are sunken from weight loss; in addition, the skin or hairs may brush against the diaphragm and produce a sound that resembles a pleural friction rub.

Normal Sounds: A quiet expiration and lack of any egophony or bronchophony are critical.

1. Vesicular: Low pitched and soft with inspiration longer than expiration (3:1 insp:exp ratio). Low freq., soft, absent in last 2/3 or expiration. Normal over most of the lung fields.

2. Bronchial (tubular): Harsh, loud and higher pitched with expiration longer than inspiration. Normal over the manubrium, loud, less harsh, hollow, 1:1 insp:exp ratio (both equally loud). High freq., loud, well preserved in last 2/3 of exp.

3. Broncho-vesicular: Moderate intensity and pitch with equal inspiratory and expiratory phases. Over sternum (mainstem bronchi) and lung apices. Soft, high pitched 1:1 insp:exp ratio. Well preserved in last 2/3 of exp, heard in the normal lung in the interscapular area. Occasionally from incomplete consolidation or a partial atelectasis.

4. Tracheal: Heard over trachea, high freq., loud, course/ harsh, equal duration during insp/ exp.

Common Errors In Auscultation to Avoid:

1. Listening through pt's gown **-->** best to auscultate directly against the pt's chest wall.
2. Allowing the tubing to rub against the bed rail or pt's gown **-->** keep tubing free of contact with any objects.
3. Attempting to auscultate in a noisy room.
4. Interpreting chest hair sounds as adventitious lung sounds **-->** wet chest hairs if thick.
5. Auscultating only the "convenient" areas **-->** ask pt to sit up, roll comatose pt on to side to auscultate posterior lobes.

Electronic Stethoscope (VRIXP): FDA approved in 9/07 for monitoring and recording lung sounds. A digital collection module uses the lung sounds to create dynamic images of the lungs, which may be useful in the detection and assessment of lung disorders.

Lung sounds are obtained from sensors applied to the pt's back, and the data are converted to images that are quickly delivered to the physician. The data can be stored in a system database for future reference.

Disease Processes:

Normal lung: midline trachea, normal fremitus, resonant to percussion, vesicular breath sounds, adventitial breath sounds with late insp crackles at bases that resolve with deep breaths, absent transmitted voice sounds.
Bronchitis: normal **-->** hyperresonance to percussion, vesicular early insp crackles, possible rhonchi/ wheeze.
Effusion: trachea shifted to opposite side, absent fremitus, flat percussion, bronchial sounds immediately above the effusion, absent over the effusion, transmitted voice sounds may be present above/ absent below the effusion.
Pulmonary fibrosis: normal/ inc fremitus, resonant to percussion, bronchovesicular breath sounds, late insp "velcro" crackles.
Consolidation (pneumonia/ hemorrhage): inc fremitus, dull to percussion, bronchial sounds, late insp crackles, transmitted voice sounds present.
Emphysema: dec fremitus, hyperresonance percussion, diminished vesicular, absent adventitial.
Atelectasis: trachea shifted to same side, inc to absent fremitus, dull percussion, bronchial to absent breath sounds, absent adventitial sounds.
PTX: trachea shifted to opposite, absent fremitus, tympanitic to percussion, absent sounds.
Bronchiectasis: mid inspiratory crackles.
Amorphic (cavernous) sounds: resonating quality, pathognomonic of a cavity.
****Ref:** (DM 1995;9:580-636) (Textbook of Pulmonary Diseases, 6th ed., 1998, Lippincott-Raven)

Abnormally Transmitted Breath/ Voice Sounds in Diseases:

Links: ICD-9 and Various | Wheeze | **Rhonchi |** Crackles (Rales) | **Stridor |** Diphragmatic Paralysis | **Exam & Testing |** Ventilatory Patterns |

…….END DEMO of this chapter.

Rheumatology:

Edited by Mark A. Bookspan, MD, Rheumatology & Critical Care.

Contents: **Basics** | Gout | **CPPD** | Acute / Chronic Arthritis, Joint Eval & Exam | **Synovial Fluid** | Seronegative Spondylopathy (Psoriatic, Enteropathic, Reactive, AS) | **OA** | Rheumatoid | **DMARDs** | Connective Tissue Diseases (SLE, Scleroderma etc) | **Autoantibodies** | Raynaud's | **Relapsing Polychondritis** | Vasculitis (TA, PMR, Wegners, etc) | **Myositis / Myopathy** | Fibromyalgia | **Fatigue** | Pain | **PMR** | Rehab Modalities | **Metabolic and Bone Dz |** MEDS | **Ankylosing Spondylitis |** Hypermobility Syndrome | ***INDEX* | See Pulm for** Sarcoidosis | See ID for **Rheumatic Fever |**

Rheumatology Basics:

Links: Rheum Emergencies | **Pearls in the Rheum Exam** | Rehab Modalities | **Joint Ranges of Motion** | Joint Aspiration & Injection | **Acute / Chronic Arthritis Eval** |

Rheumatic diseases are syndromes of pain and/or inflammation in articular or periarticular tissues. More than 21% of U.S. adults have arthritis or other rheumatic conditions, and by 2030 the number is projected to increase by nearly 40% as the population ages according to a national study (Arthritis & Rheumatism 2008;58:15-35)....Age-adjusted disease rates were higher for women than for men (24% versus 18%) and for whites and African-Americans (about 22% for both groups) compared with Hispanics (16.5%).

Rheumatologic Emergencies:

RA: C-spine subluxation (atlanto-axial, need decompression and fusion), cricoaryterititis, pericarditis/ tamponade, hyperviscosity syndrome.
Scleroderma: malignant renal crisis **-->** manifests as dramatically rising serum Cr (1mg/d), HTN, bland sediment, microangiopathic anemia, mild thrombocytopenia, eosinophilia, retinal hemorrhages.
Relapsing polychondritis: tracheobronchial collapse.
Dermatomyositis: respiratory failure.
Other: Temporal Arteritis, **Septic** arthritis, episcleritis, vasculitic complications (alveolar hemorrhage in SLE).

Key Emergency Sx's:

Hx of significant trauma: Fx, compartmental syndrome, rhabdomyolsis.
Systemic sx's (fever, weight loss): Infection (septic arthritis, osteomyelitis, endocarditis, necrotizing fasciitis).
Focal Weakness: Radiculopathy, entrapment neuropathy, compartmental syndrome, motor neuron disease, mononeuritis multiplex.
Global or progressive weakness: Spinal cord compression, myelopathy, transverse myelitis, myositis, response to toxin.
Neurogenic pain (burning, numbness, paresthesias): Radiculopathy, entrapment neuropathy
Point tenderness: Fx. Red, hot, swollen joint: Septic arthritis, gout, pseudogout.
Asymmetrical painful, swollen leg: DVT.
Diffuse shoulder / hip pain, H-A in elderly pt: Temporal arteritis.
Bowel or bladder dysfunction: Cauda equina syndrome.

Gouty Arthritis:

Links: PP, Risks & ICD9 | Clinical Phases | **Dx, X-ray & ACR criteria** | Polarized Microscopy | **Acute Tx** | Under secreters: Uricosurics (Sulfinpyrazone (Anturane) & Probenecid (Benemid) | **Overproducers: Allopurinol & Febuxostat (Uloric)** | Purine Content of Food & Hyperuricemia | **Prevention** | CPPD (Pseudogout) & Other | **Serum Uric Acid |** See Urology for **Hyperuricuria (Uric Acid Stones) & Urate nephropathy |**

Gout is a common form of inflammatory arthritis, often causing recurrent episodes of pain and swelling of certain joints. More than 90% of cases are due to inefficient excretion of uric acid. Gout is a metabolic dz resulting from tissue deposition of monosodium urate crystals from supersaturated extracellular fluids. Gout is seen in 1-2% of men in Western countries, M:F ratio is 8:1. Increased incidence with rising metabolic syndrome (obesity, HTN, DM). The clinical manifestations include acute gouty arthritis, aggregates

of crystal in connective tissues (tophi), urate urolithiasis, and, rarely, gouty nephropathy. Gout is the most common inflammatory joint dz in men >40yo.

PP: Urate crystals form when the serum urate concentration exceeds 0.42mmol/L. When the local solubility limits of uric acid are exceeded, monosodium urate crystal deposition in the joints, kidneys, and soft tissues causes clinical manifestations, including arthritis, soft tissue masses (i.e., tophi), nephrolithiasis, and urate nephropathy. The deposition of urate crystals that leads to neutrophil activation and synovial inflammation. Trauma, post-surgical state and other factors can precipitate the attack (see below). Local factors that contribute to this deposition are changes in pH level (e.g., from perioperative ketosis in surgical pt's); lower body temperature, explaining nocturnal attacks; and the level of articular dehydration (e.g., from initiation of diuretic therapy). However, most persons with elevated serum uric acid levels do not develop gout. The annual incidence of gout is 0.5% in persons with a uric acid level between 7 and 8.9 mg/dL (415 and 530 µmol/L), and the annual incidence is 4.5% in those with a level $\geq$9 mg/dL (535 µmol/L) (Am J Med 1987;82:421-6). The prevalence of gout increases with age and is higher in black pt's than white. Increased adiposity and insulin resistance are both associated with hyperuricemia (Ann Int Med 2005;143:499-516). A high incidence occurs in Filipinos and South Pacific Islanders, in whom it is associated with obesity. Other risk factors include alcohol use, lead exposure, diuretic use, HTN, and renal insufficiency. HTN, thiazide & loop diuretics, obesity and alcohol intake all contribute in an additive manner (NEJM 2003;349:1467-55) (Arch Intern Med. 2005;165:742-748). The ARB losartan (Cozaar) has a uricosuric effect that is not seen or is less pronounced with other ARBs or ACEi's, thus it may provide a safe means to blunt the thiazide-induced increase in uric acid level, while potentiating the antihypertensive action of the diuretic (J Hypertens 2001;19:1855). Alcohol intake increases the risk of gout, intake of $\geq$2 twelve-ounce beers daily increased risk 2.5-fold; 2 shots of liquor increased risk 1.6X; and consumption of two 4-ounce glasses of wine did not increase risk (Lancet. 2004;363:1277-1281). Gout appears to be associated with the occurrence of subsequent cardiac arrhythmia, especially if have HTN (J Rheumatol 2004;31:1152-1158). Alcohol consumption (even a light-to-moderate amount) triggers recurrent gout attacks, this effect is likely to occur within 24 hours after its consumption (Am J Med 2006;119:e13-18) (regardless of which type of alcoholic beverage). Osteoarthritis may trigger the localized deposition of monosodium urate crystals (Ann Rheum Dis 2007;66:1374-1377)...they often occure in the same joints, especially at the first MTP joint, mid-foot, knee, and finger DIP joints. Researchers have identified two new genetic loci, and confirmed a third, that show association with uric acid concentration and risk of gout (Lancet 2008;Sept 30)....."Heritability of serum uric acid concentration is 63%, through regulation of uric acid synthesis, excretion, or reabsorption."....identified three candidate missense mutations in SLC2A9, ABCG2, and SLC17A3, genes associated with renal urate transport and expressed in the apical membrane of renal proximal tubule cells.....The presence of all six risk alleles "was associated with up to 40-fold increased risk of developing gout, which is substantially higher than that for environmental risk factors."

ICD-9 Codes:

274.0 Gouty arthropathy
274.11 Uric acid nephrolithiasis
274.81 Gouty tophi of ear
274.82 Gouty tophi of other sites

4 Clinical phases:

#1. Asymptomatic hyperuricemia: serum urate (uric acid) >7mg/dL (416 umol/L) = the level urate is supersaturated in the plasma. Yet 5% of adult males have serum urate >7, thus 2/3 will remain asymptomatic. Hyperuricemia may develop secondary to numerous conditions (renal insufficiency, myeloproliferative diseases, obesity, alcohol consumption, insulin-resistance / metabolic syndrome, and drug intake). Pt's with persistent marked hyperuricemia (>10 mg/dl) or hyperuricosuria (>1,000 mg/24 hr) should be followed carefully for manifestations of gout or renal stones. ~30% of people with uric acid >10 mg/dl will have gout over 5 yr (Arch Int Med 1987;147:645-9).

#2. Acute gouty arthritis: peak incidence of 1st attack is at 30-40yo. If onset is before 30yo in male or pre-menopause in female suspect an inherited enzyme defect in purine metabolism or renal parenchymal dz. In women who are normal producers/ under secreters, gout occurs exclusively postmenopausal. 60% have 2nd attack within 1yr, 78% in 5 yrs. Only 7% do not have a recurrence in 10yrs. The initial attack of gout is monoarticular in 85-90% of pt's. In 75% of pt's, the 1st joint at presentation is the 1st metatarsophalangeal (MTP) joint = **Podagra**, MTP is ultimately affected in 90%. Pain peaks in intensity at 24-36hr. The acute gouty arthritis generally presents with the rapid onset of incapacitating, throbbing pain in the affected joint. The overlying skin is tender, warm, and erythematous and may be confused with a sprain, septic joint, or cellulitis. Fever and systemic sx's may occur. If untreated, it clears after days to weeks. As the swelling subsides, the skin takes on a violaceous hue and desquamation occurs.

Elderly: Older pt's tend to have more upper extremity and more often **polyarthritis** at presentation. In older women in particular, involvement of the small joints of the fingers (previously affected by osteoarthritis) is more commonly seen earlier in the course of the disease.

Other sites effected: other MTP's> instep/forefoot> ankle> knees> wrist> fingers. Polyarticular involvement becomes more common over time. May mimic other forms of arthritis (i.e. ~30% may have low titers of RF). Initial attacks last 3-6 days, 2wks in later dz, and eventually continuous if untreated adequately. Uric acid crystals can be found in the joint even after resolution of the sx's, the higher the serum uric acid, the more likely crystals will be seen. 20% will have normal serum urate during acute attack. Axial gout was found in 14% of 64 pt's who underwent CT scans of the spine, often in the absence of back pain (J Rheum 2009;36:609-13)(usually manifested as facet erosions, located in lumbar in 78% and thoracic in 30%, none found in cervical).

#3. Intercritical gout: asymptomatic intervals between acute attacks. Low-level inflammation persist during remissions of acute flares (Pathogenesis of Gout. Ann Int Med 2005;143:499-516).

#4. Chronic tophaceous gout: persistent sx's of soreness & aching of affected joints **or** tophi (asymmetrical, nontender soft tissue masses). Tophi are commonly on the joints of the hands/ feet, less commonly on the helix of the ear, olecranon bursa and Achilles tendon. Often contain flecks of calcification. May occasionally precede acute gouty attacks in the pulps of the fingers. The lack of marked inflammation in tophi of the subcutaneous and dermal layers of the skin has not been clearly explained, they may drain a clear fluid with white flakes of urate, or a thick chalky material. A more rapid onset can be seen in tophi associated with

myeloproliferative d/o's and tophi in infants with deficiencies of enzymes involved in purine metabolism. If painful, large, and inflamed, they may regress quickly with an intralesional steroid injection as well as proper systemic medication. Tophi tend to have a heterogeneous appearance with both hyper and hypoechoic areas on U/s. Tophi can also occur in places such as lumbar facet joints at or adjacent to the temporomandibular joint.
S/s of Gout: Usually monoarticular (polyarticular if chronic disease), rubor (heat), often febrile, pain, erythema, swelling. Will subside in 3-10d even w/o tx. Tophi typically appear ~10yr after 1st episode, they indicate prolonged hyperuricemia with a heavy burden of urate.
In the elderly, M=F, **atypical gout** common, it involves any joints (upper extremities common), often polyarthritis, tophi at presentation common—most are underexcreters (JAMA 2003;289:21).
Dx Gout: Links: **Polarized Microscopy** | Serum Uric Acid | Combo of risk factors, clinical features and intracellular urate crystals. Always consider infection. Definitive dx with aspiration of synovial fluid or tophus showing characteristic needle shaped monosodium urate crystals within WBC's on conventional light microscopy ("Toothpick pierced through an olive") is **always** found during an acute attack. Best place to "tap" the great toe is at the top, just under the extensor tendon (put air in the syringe and "spit" out the contents onto a slide, may see crystals or whitish clumps even without polarized light). Consider X-ray if first attack of unclear of dx.
Criteria of American College of Rheumatology (Wallace. Arthritis Rheum 1977;20:896**):**
Dx of acute gouty arthritis if **one or more** of the following are present:
(1) presence of urate crystals in the joint fluid. The gold standard for gout dx, with a sens of 84% and a spec of 100% (Ann Rheum Dis 2006;65:1301–11).
(2) a tophus shown to contain monosodium urate (MSU) crystals by chemical analysis or light microscopy (**polarized microscopy**).
(3) 6 or more of 12 clinical, lab and radiographic features:
(1) more than 1 attack of acute arthritis.
(2) max inflammation developed within 1 day.
(3) monoarthritis attack.
(4) redness observed over joints.
(5) first MTP joint painful or swollen.
(6) unilateral first MTP joint attack.
(7) unilateral tarsal joint attack.
(8) tophus proven or suspected.
(9) hyperuricemia.
(10) asymmetric swelling within a joint as seen on X-ray.
(11) subcortical cysts without erosions on X-rays.
(12) joint fluid Cx negative during the attack. 97.8% of gout pt's in study group were correctly identified (Arthritis Rheumatism 1977;20:895-900).
Accuracy of Key Findings in the Dx of Acute Gout: (Arthritis Rheum 1977;20:895-900)
Asymmetric swelling shown on a radiograph: Sens & Spec = 42 & 90%.
Hyperuricemia: Sens & Spec = 92 & 91%.
Monosodium urate crystals in synovial fluid: Sens & Spec = 84 & 100%.
Podagra (first metatarsophalangeal joint involvement): Sens & Spec = 96 & 97%.
Tophi confirmed: Sens & Spec = 30 & 99%.
X-ray: sharply defined, variable size erosions with overhanging edges (sclerotic margins) and bony cysts, not present for 6-8 yrs after disease is present. No osteopenia. Mild joint space narrowing with late dz. Bony erosions occur relatively late in gout, and many pt's with repeated acute attacks show neither bone nor soft tissue lesions. The major value of radiographic findings include sharply "punched-out," round, or oval (apple-core) defects situated in the marginal areas of the joint and surrounded by a sclerotic border suggest gout. Bony (intraosseous) tophaceous lesions almost always antedate subcutaneous tophi and may be seen in joints that have never been clinically inflamed. Gouty erosions in bone often manifest as an elevated margin extends outward into the soft tissue covering the tophaceous nodule, this "overhanging margin" of bone, which may result from bony resorption beneath the enlarging tophus and periosteal apposition of the involved cortex, helps to distinguish gouty erosions from the "pocketed" erosions seen in RA. preservation of joint space initially and an absence of periarticular demineralization are also important clues.

10 Diagnostic Guidelines:

(Ann. Rheum. Dis. 2006;65:1312–24)

1. Clinical sx's of rapidly developing severe joint pain and swelling, especially with overlying erythema, are highly indicative of crystal formation but have low sensitivity (23%) for a gout dx without further evidence.
2. Monosodium urate monohydrate (**MSU**) crystals in synovial aspirate have both high sensitivity and high specificity, although variability in lab expertise may affect the accuracy of the sample findings.
3. A clinical dx of classic recurrent podagra with hyperuricemia can be reasonably assumed to be gout but requires MSU crystal confirmation.
4. All pt's with inflamed joints should have their synovial fluid examined for MSU crystals.
5. MSU testing can also be performed between bouts of inflammation, because urate crystals persist in intercritical periods in up to 70% of pt's.
6. Because gout and sepsis may occur simultaneously, all possible gout pt's should also have Gram staining and culture of synovial fluid. The test should be performed even if the fluid is positive for MSU crystals, because septic arthritis can progress rapidly and carries a significant risk of morbidity and mortality.
7. Serum uric acid can't be used exclusively as a diagnostic tool. Many people with high serum uric acid don't develop gout, and some pt's with confirmed MSU crystals have normal serum uric acid.
8. Gout pt's under age 25 years who have a FHx of the disease or who have renal calculi should have a 24-hour urinary uric acid/creatinine ratio done. The 24-hour screen appears to be more accurate and cost effective than an early morning spot sample.
9. Radiographs might be useful for a differential dx, but they can't confirm gout. There are radiographic changes in all stages of gout, but many affected joints can be radiographically normal. Pt's with intradermal tophi are more likely to show severe radiographic changes.

10. Male gender, diet, alcohol use, and diuretics increase the risk of gout, but don't ignore other important risk factors. These include hypertension (relative risk of 4, compared with controls), coronary heart disease (odds ratio, 1.75), chronic renal failure (odds ratio, up to 5), and obesity (odds ratio, 3.8).
Serum Uric Acid: Hyperuricemia | Normal range = 2–7 mg/dl (120–420 µmol/L). The risk of developing gout is directly related to the degree of hyperuricemia. The serum urate concentration may be normal in 40% of people measured during the acute attack. The annual incidence of gout is <0.5% in serum uric acid 7-8.9 mg/dL. Have an incidence of gout of ~5%/yr if level >9.5mg/dL. Should w/u and start treatment male if >12 or a female >10mg/dL, even is asymptomatic. Most people with asymptomatic **hyperuricemia** (= serum UA >7mg/dL [>0.42 mmol/L] in males **or** >6 mg/dL [>0.36mmlol/L] in women) do not need further workup or tx. Since <10% of hyperuricemic pt's are "overproducers", a 24hr uric acid secretion not typically done unless pt has kidney stones, strong FHx of gout or gout at a young age.
UA elevated in: Renal failure, gout, excessive cell lysis (chemo, radiation, leukemia, lymphoma, hemolytic anemia), hereditary enzyme deficiency (hypoxanthine-guanine-phosphoribosyl transferase), acidosis, myeloproliferative disorders, diet high in purines or protein, drugs (diuretics, low doses of ASA, ethambutol, nicotinic acid), lead poisoning, hypothyroidism, Addison's disease, nephrogenic diabetes insipidus, active psoriasis, polycystic kidneys. Among asymptomatic moderate-risk men, high UA levels (7.1 mg/dl) were independently associated with (3-fold risk) coronary artery calcium in subjects with metabolic syndrome (Am J Card 2007;99:42-45). A genetic risk score based on three genes (single nucleotide polymorphisms = SNPs) that influence serum levels of uric acid are associated with a 40-fold increased risk for gout (Lancet 2008;DOI: 10.1016/S0140-6736(08)61343-4).
UA decreased in: Drugs (allopurinol, high doses of ASA, probenecid, warfarin, corticosteroid), deficiency of xanthine oxidase, syndrome of inappropriate antidiuretic hormone secretion, renal tubular deficits (Fanconi's syndrome), alcoholism, liver disease, diet deficient in protein or purines, Wilson's disease, hemochromatosis
UA Levels During Acute Gout Attacks: Data from 339 pt's suggest that one third of pt's with acute gouty arthritis have levels <8 mg/dL (J Rheumatol 2009;36:1287).....14% had serum uric acid levels 6 mg/dL, and 18% had levels between 6 mg/dL and 8 mg/dL.
Other Info: Middle-aged men with gout and elevated uric acid levels have an increased risk for cardiovascular disease (CVD) mortality (30% greater risk) (Arch Intern Med. 2008;168:1104-1110). Elevated UA seen in severe pulmonary HTN and can be used as a marker for dz severity (Resp Med 2003;97:130-33). In middle-aged men, elevated serum US levels are a strong and independent predictor of death from cardiovascular disease and all causes (>2.5-fold) (Arch Intern Med 2004;164:1546-1551). Most pt's (>75%) with gout have an elevated uric acid (hyperuricemia) never develop gout. No tx if asymptomatic. A 5 year longitudinal study of 223 asymptomatic men with hyperuricemia estimated 5 year cumulative incidence of gout to be 10.8% for those with baseline serum urate of 0.42–0.47 mmol/L, 27.7% for baseline urate 0.48–0.53 mmol/L, and 61.1% for baseline urate levels of 0.54 mmol/L or more (J Rheumatol 2000;27:1501–1505) The risk of stroke and myocardial infarction is nearly doubled in pt's with the highest serum uric acid levels compared with those with the lowest levels (Stroke 2006;37:1503-1507).....the risk of cardiovascular disease seems to be linear, there is no 'natural' threshold value, presumably by causing renal disease that leads to hypertension, which in its turn leads to stroke and myocardial infarction. A population-based study with 3604 men between the ages of 45 and 74 years found that high uric acid levels are independently associated with both all-cause and cardiovascular disease (CVD) mortality (Arterioscler Thromb Vasc Biol 2008;28:1186-1192)....For men in the highest versus lowest quartile to uric acid level, the hazard ratio for CVD mortality was 1.44 and for all-cause mortality it was 1.40, after adjustment for conventional cardiovascular risk factors, C-reactive protein levels and diuretic intake. A population-based study with 1387 men with a BMI <30 found that after adjustment for smoking, BMI, intake of total energy, dairy protein and alcohol, and other potential confounders, the team found a significant association between greater intakes of total vitamin C and lower serum uric acid concentrations (J Rheumatol 2008;35:1853-1858).....The multivariable odds ratio for hyperuricemia for the highest intake of vitamin C (>1000 mg/d) compared to the lowest (<90 mg/d) was 0.34 (p for trend < 0.001). Elevated uric acid levels are associated with an increased risk (increases linearly) for chronic kidney disease (J Am Soc Nephrol 2008;Dec).....Slightly elevated uric acid levels (7.0 to 8.9 mg/dL) were associated with an unadjusted odds ratio of 1.49 for the risk of developing stage 3 CKD; with elevated uric acid levels (9.0 mg/dL and higher), the odds ratio was 2.49....."normotension as well as uric acid levels < 6mg/dL (women) and < 7 mg/dL (men) should be recommended, and treatment -- e.g., diet low in purines, drugs (allopurinol, probenecid, antihypertensives), endurance exercise, and weight reduction -- should be initiated from a preventive medicine viewpoint." In middle-aged Chinese adults, hyperuricemia (>7 mg/dL) is an independent risk factor for all-cause mortality (HR = 1.16), cardiovascular disease (CVD) (HR = 1.39) and ischemic stroke (HR = 1.35) according to a prospective cohort study conducted in Taiwan on 2,000 men and 49,000 women aged 35 years and older (mean 51.5 years)(Arthritis Rheum 2009;61:225-232).....High levels of uric acid may act as a pro-oxidant, leading to vascular endothelial dysfunction, inflammation, nitric oxide production impairment, atherosclerosis, and thrombogenesis."

Tx: Acute Gout: Topics: **NSAIDs |** Corticosteroids | **Colchicine |**

Oral colchicine and/or NSAIDs are generally first-line tx. The goal is to terminate the acute painful attack, **prevent** further recurrences and reverse any complications of urate deposition. If untreated, 27% will experience spontaneous resolution after 7 days. Ice therapy and joint rest are good adjunctive tx's for acute gout (Geriatrics 2008;63(7):17).
NSAIDs: Standard therapy for acute attacks. ASA is avoided because of the paradoxical effects of salicylates on serum urate. Can use Ibuprofen 600-800mg TID **or** Naprosyn 750-1000mg mg/d x3, then 275mg TID **or** Sulindac (Clinoril) 200mg BID x3d, then taper. Can also use Toradol IM/IV or even selective COX II inhibitors such as Celecoxib (Celebrex) 400mg x1, then 200mg qd. **Indocin** 50mg PO TID-QID X3d, then 25mg PRN q8hr. **Contra:** PUD, systemic anticoagulation, CHF, nephropathy. A pure PGE inhibitor, thus 100X better than other NSAIDs for Gout.
Steroids: An option instead of NSAIDs, particularly if renal insufficiency, age >65, HTN, CHF, anticoagulation, hepatic dysfunction or any GI problems where NSAIDs are contraindicated.
Intra-articular: the preferred route for monoarticular involvement and those pt's who cannot tolerate pharmacotherapy. Typically use 10-40mg Triamcinolone in large joint, 2-20mg in small joint. If Depo-Medrol use 5-10mg for small joint, 10-20 for medium and 40-80mg for large joints.
Oral Prednisone: Some need 40-60mg, most get by on 30mg. Rx 5mg #42, dose @ 30mg qd X5d (0.5mg/kg), then taper by 25mg x2d, 20mg x2d, 15mg x1, 10mg, then 5mg and stop (12 days total). IV methylprednisolone @ 50-100mg/d X3d, then oral taper. For short term use only. A RCT with 90 pt's with acute gout found that prednisolone (30mg PO qd x 5) was as effective as indomethacin 50mg TID x 2, then 25mg TID x 3) and was associated with considerably fewer adverse effects (Ann Emerg Med 2007;49:670-7)....should be considered as first-line therapy. For the acute pain of monoarticular gout over four days, it's a tossup between oral prednisolone (35 mg qd x 5) and naproxen (Aleve, (500 mg BID x 5 days), a randomized trial found (Lancet

2008;371:1854-1860). .....In addition to better safety, the direct drug costs would also be less if systemic corticosteroids were the first-line drug choice..... this trial "will satisfy both rheumatological purists and front-line pragmatists that short-term oral corticosteroids are as equally effective as NSAIDs in the initial treatment of acute gout and gout-like syndrome."
Corticotropin (ACTH): 400 IU IM x 1. Max = 40-80 Units IM gluteal q6-12hr, max 4 doses. Need repeat injections, may cause volume overload via mineralocorticoid effects. 100 UI produces about 100mg of cortisol over 16h. Not usually used.
Colchicine (Colcrys, ColBENEMID, Proben-C, Col-Probenecid): [0.6mg tab] For acute attacks @ two 0.6mg tablets followed by just 1 tab one hour later for a gout flare (= "new" regimen). About 25% of patients on the lower dose get GI effects, compared to almost everyone on the higher doses. Based on 2009 approval of Colcrys, the first FDA approved colchicine tablet in which they updated the dosing at 117 deaths have been reported over the past 30 years related to the older dosing method. Also approve for the treatment of Familial Mediterranean fever (FMF) in pt's >4yo.
"Old" regimen: two 0.6 mg colchicine tablets followed by 1 tab qhr until pain subsides or GI toxicity occurs (80% experience GI SE's)….Stop if get N/V/D or pain is resolved or reached a max of 7-10 tabs per 24hr or 30 per 21days. Others suggest 1.2mg initially, then repeat in 3-4 hrs and a final 0.6mg at bedtime. A lower dose @ 0.6 mg TID may be appropriate and effective in elderly or those prone to GI SE's.
Drug Interactions: Avoid with inhibition of CYP3A4 such as macrolides (erythromycin, telithromycin and clarithromycin) (azithromycin is safe), simvastatin and cyclosporine as they lower the secretion and may lead to toxic doses…. particularly in the elderly with age-related decreases in renal function. For pt's on moderate CYP3A4 inhibitors (verapamil, etc), have patients take just 2 colchicine tabs and not repeat for at least 3 DAYS. For strong CYP3A4 inhibitors (clarithromycin, etc), have patients take 1 colchicine tab, then HALF a tab an hour later if needed and not redose for at least 3 days. Colchicine can cause myopathy in some when combined with statins, fibrates, cyclosporine, or digoxin. Advise patients on these combos to report unexplained muscle pain or weakness. Isolated and anecdotal cases of myopathy have been reported in pt's receiving concomitant tx with fluvastatin sodium and colchicine, the cause is unknown.
Notes: 66% respond within hours if given within the 1st 24hrs after the onset of arthritis. Less effective once sx's have persisted >2days. Use ½ dose if GFR<50 (see below). Avoid in those who recently received prophylaxis with Colchicine, the elderly and those with renal insufficiency as risk BM failure or death. Antimitotic, derived from roots of Colchicum autumnal, it inhibits microtubule assembly, which interferes with granulocyte mobility and the inflammatory response to precipitated crystals. **IV** @ 2mg, then 1-2mg q6-12hr, 4mg/24hr, this is discourage as associated with high complication rate such as bone marrow suppression. IV colchicine should be diluted in 10-15 mL of NS solution and administered slowly during 5 minutes through a free-flowing IV route to decrease the chance of infiltration and soft-tissue necrosis. Fatalities have been reported with IV administration. Reduction in dosage is necessary in liver or renal impairment.
For maintenance prophylaxis: after 2-3 attacks use 0.6 mg PO BID if CC >50ml/min.
@ 0.6mg qd if CC 35-49 ml/min.
@ 0.6mg q2-3d if CC 10-34 ml/min. Avoid if CC <10, on hemodialysis or hepatobiliary dz. Reduce dose by ½ if age >70yo. Best used in combination with effective hypouricemic therapy as may may simply "sweep under the rug" acute attacks and chronic crystalline precipitation may progress to tophi, articular damage or nephropathy without the warning of sentinel attacks.

Prevention of Gouty Attacks:

Link: Allopurinol | **Diet Mods: Purine Content of Food & Hyperuricemia** | Colchicine |
Recurrence rates are 62% after 1 year, 78% after 2 years and 84% after 3 years. Weight control, limits on red meat consumption and daily exercise are the foundations of lifestyle modifications in pt's with hyperuricemia (Ann Int Med 2005;143:499-516). Of 4,166 new users of urate lowering drugs (97% allopurinol), 70% had one or more gaps in their therapy (based on pharmacy refills) during the first year alone, 50% returned to therapy within 8 months and 75% by 4 yrs (presumably due to recurrence of sx's) (Am J Med 2010;123:54-59).
Tx comorbid conditions to correct the metabolic condition: Metabolic syndrome, hyperlipidemia, hypertension, hyperglycemia, obesity, and alcohol use should be addressed as all linked to elevated serum uric acid.
Indications for Hypouricemic (uric acid lowering) Tx: Repeated painful or disabling attacks (>2-3/ yr), tophaceous deposits in soft tissue, severe hyperuricemia (≥10-11mg/dL female, ≥12-13 male), urate nephropathy, recurrent urate nephrolithiasis, pt's with high tumor burden of leukemia-lymphoma about to receive cytotoxic chemo (tumor lysis syndrome) or a documented state of uric acid overproduction. Never give these medications for an acute attack. The goal is to maintain the serum uric acid <6.5 mg/dL. It is helpful, but not essential to 1st determine if over secreter **or** under secreter. Check a 24hr Urine Uric Acid level (nl <1g on a normal diet or <600mg on a purine-restricted diet), during intercritical gout.
Rx: Colchicine 0.6mg qd **or** QOD (see above) or NSAIDs such as Indomethacin 25 mg BID or others are probably effective and may be associated with a lower frequency of side effects. It is recommended to give Colchicine 0.6-1.2mg PO qd with a urate-lowering drug until serum urate level is stable and no acute attacks for 3-6mo. 85% effective. Use of antihyperuricemic agents and a decline in serum urate concentration (to at least 6mg/dL) are significantly associated with a reduction in gouty arthritis attacks (Arthritis Rheum 2004;51:321-325). In pt's without tophi prophylaxis can generally be safely discontinued 6-12 mo's after normal serum urate values have been obtained. Pt's with tophi are usually continued until resolution of these lesions occurs, or it is clear that tophaceous deposits will not resolve despite persistent normouricemia. For pt's with severe renal insufficiency, a modest lowering of serum urate is a potentially beneficial side-effect of tx of hyperphosphatemic pt's with phosphate binding agents (calcium containing antacids or sevelamer). Failure to adhere to a urate-lowering medication regimen is exceedingly common in patients with gout, and this appears to be particularly true of younger patients (Arthritis Res Ther 2009;Apr 17).....during the first year of therapy 56% of the patients were nonadherent.....being under the age of 45 years was a major predictor of poor adherence (odd ratio, 2.43).....Other important predictors included fewer comorbidities and no gout-related provider visits before starting medication.

Undersecreter (Underexcretors) of Uric Acid (90%):

<600-800 mg/d on 24h urine (4.76mmol/d). = low due to reduced renal filtration/ secretion. Can also use Allopurinol.
Etiology: enhanced reabsorption. From intrinsic renal dz, **drugs** (diuretics? [see PV below], low dose ASA, ethambutol, pyrazinamide, Cyclosporine, Levodopa, Nicotinic Acid (Nicolar), dehydration, lactic acidosis, ketosis, obesity, sarcoid, toxemia of pregnancy, HTN, hyper/ hypothyroid, lead, ethanol. Even pt's with high levels of urate excretion (overproducers) demonstrate a relative decrease in urate clearance compared to pt's with normal levels of uric acid production (Arthritis Rheum 2002;47:610).

Uricosuric: (if GFR>50, fluid intake >2L, no Hx of nephrolithiasis, avoid salicylates): The uricosuric drugs are weak organic acids that promote renal clearance of uric acid by inhibiting the urate-anion exchanger in the proximal tubule that mediates urate reabsorption. At the beginning of therapy encourage pt to drink lots of water, consider urinary alkalinization may help avoid nephrolithiasis if Hx of stone. Give oral Na-bicarb at night until uric acid is stabilized in the desired range (takes ~2-6 wks). Avoid Na-bicarb in CHF as salt load

Contra to Uricosuric Tx: overproducers, renal insufficiency, tophaceous gout, allergy, recurrent urolithiasis, enzyme defect.

1. Probenecid (Benemid, ColBENEMID, Proben-C, Col-Probenecid): [500mg tab] 250mg BID X7, then 500mg BID + (Na-bicarb Antacid X30d to inc Ur pH). Increase to 500-1000mg BID for maintenance. Max dose of 2-3g/d, titrate until serum urate <6mg/dL (335 mol/L). CrCl must be >60ml/min to be effective. Maintenance colchicine should be given during the first 3 mo's of probenecid therapy and then can be stopped if the pt is asymptomatic. Probenecid is not useful in acute attacks of gout. **SE:** rash, GI. A uricosuric agent that inhibits renal tubular resorption of organic acids, including uric acid (increases UA secretion).

Avoid: if daily ASA. Creatinine clearance <40 mL/min (probenecid not effective) or in on low-dose aspirin or diuretics (negate its effects). Probenecid is relatively contraindicated in patients with kidney stones because probenecid competitively inhibits the active reabsorption of uric acid at the proximal convoluted tubule. This excessive uric acid excretion increases of risk of kidney stones. Probenecid also reduces renal tubular secretion of medications such as NSAIDs, most cephalosporins, penicillin, methotrexate, and others. Probenecid's effectiveness is also reduced by salicylate. A large alkaline urine output should be maintained, especially during the first week of therapy, to prevent urate renal stones. A history of renal calculi is a relative contraindication. May alter the metabolism of other drugs by decreasing their excretion (indomethacin, ampicillin), reducing their volume of distribution (ampicillin), or delaying metabolism (heparin). Probenecid is antagonized by low-dose salicylates (<3 g/d).

2. Sulfinpyrazone (Anturane): [100 mg tab; 200 mg cap] Start @ 50-100 mg BID with meals, and increase gradually during several weeks to a maintenance dosage usually of 300-400 mg/d in three to four divided doses. Maximum recommended daily dose is 800 mg. Goal to keep Uric acid <6. Has added natural antiplatelet benefits, thus good for cardiac pt's. Maintenance colchicine should be given for the first 3 mo's of therapy to prevent precipitation of an acute attack of gouty arthritis. A uricosuric agent that inhibits renal tubular reabsorption of organic acids, used for hyperuricemia associated with refractory or recurrent gouty arthritis. It is rapidly and completely absorbed, with peak plasma levels in about 1 hour. Sulfinpyrazone is 98% protein-bound. The kidney excretes about 40% unchanged. **SE:** GI irritation is common. Because sulfinpyrazone is similar in structure to phenylbutazone, it should be avoided in pt's with known sensitivity to phenylbutazone. A large alkaline urine output should be maintained, especially during the first week of therapy, to decrease the risk for urate calculi formation. Sulfinpyrazone inhibits platelet aggregation and potentiates the action of protein-bound drugs such as oral hypoglycemics and oral anticoagulants. The action of sulfinpyrazone is antagonized by low-dose salicylates, and the drug is ineffective in the presence of renal failure. CrCl must be >45ml/min to be effective.

Overproducers of Urate (10-15%):

Links: See below for Allopurinol & Febuxostat (Uloric) | Serum Uric Acid | **Diet Mods: Purine Content of Food & Hyperuricemia** **|** High levels of urate excretion, usually >800mg/24hr in urine, (usually significantly >800mg/d). Caused by increased purine synthesis. Uric acid overproduction with impaired renal clearance of uric acid. Often have Hx stones, tophi, renal insufficiency. Uricosuric agents (Probenecid & Sulfinpyrazone) are contraindicated in overproducers.

Etiology: familial metabolic d/o, excess dietary purine or fructose consumption, inc nucleic acid turnover (myoproliferative dz, tumor lysis, psoriasis, hemolytic), ETOH, tissue hypoxia, sustained exercise, pernicious anemia, obesity, hypertriglyceridemia.

Tx: For the management of recurrent gout (two or more attacks per year)6 or chronic gout, lifelong uric acid lowering therapies are warranted with a therapeutic goal of <6 mg/dL (Guideline for the management of gout. Rheumatology 2007;46:1372-4).....Allopurinol is the first drug of choice....Probenecid can be used as an alternative to allopurinol in patients with normal renal function.

Allopurinol (Zyloprim, Xyloprim, Lopurin): [100, 300mg tabs, $12-16/#100, 20 mg/mL elixir] Start @ 50-100mg/d, titrate slowly per renal function until serum urate <6mg/dL (335 umol/L). The average dose is 200-300mg/d, use the lowest does to achieve a serum UA (SUA) <6. Some start @100mg qdX7, then 200mg qd X7, then 300mg qd. Max = 800mg/d. Others say "start low, go slow" to avoid the rare but potentially life-threatening allopurinol hypersensitivity sydrome, begin with 100 mg/day and increase by 100 mg/day every 2–4 weeks. Commonly used even in those pt's who do not overproduce urate (works for all forms of gout), used in overproducers or underexcretors. A xanthine oxidase inhibitor (inhibits nucleic [uric] acid synthesis). Takes 2wks for peak effects. Can be used with NSAIDs or colchicine 0.6mg qd-BID X 3mo (until serum urate equilibrates) as likely to flare or even precipitate gout.

Indications: tophaceous gout, uric acid renal stones, prophylaxis of tumor lysis syndrome, psoriasis induced gout.

Caution: renal stones, or **renal insufficiency:** If the GFR (Cr Clearance = CC) is >100 mL/min maintenance dose = 300mg/d. [CC 60-100]: 200-250 mg daily. [40-60]: 150-200mg daily. [20-40]: 100-150mg daily. [10-20]: 100mg q24. [<10 ml/min]: 100 mg q24-48h. Alternatively some day if: [>50 ml/min]: No changes. [20-50]: 100-300mg q24h. [10-20]: 100-200mg q24h. [<10]: 100mg q24-48h. If 0-10 ml, then 100mg q3d.

IV: [10-20 ml/min]: 200 mg/day. [3-10 ml/min]: 100 mg/day. [<3 ml/min]: 100 mg/day at extended intervals.

Hemodialysis: 100mg q24-48h (give dose after dialysis on dialysis days). Allopurinol therapy significantly decreases serum UA levels in hyperuricemic pt's with chronic kidney disease, which seems to help preserve kidney function (Am J Kidney Dis 2006;47:51-59).....by diminishing serum UA levels, it may serve as an agent to decrease glomerular hydrostatic pressure indirectly and thus help alleviate renal damage. Adjusting allopurinol doses according to creatinine clearance, as recommended by published guidelines, may not provide adequate control of hyperuricemia in many pt's with gout (J Rheumatol 2006;33:1646-1650)…."hypersensitivity reactions were not associated with higher than recommended allopurinol doses."

SE: maculopapular rash (the most common SE, occurs in 2-3% of pt's), GI, H-A, urticaria, inc LFT, inc eosinophils, fever. Hypersensitivity reaction (eosinophilia, hepatitis, fever, TEN, worsening renal function, erythematous, desquamating rash, usually only with high doses). Pt's with normal renal function who cannot tolerate allopurinol may do well with a uricosuric drug such as probenecid or sulphinpyrazone or low-dose colchicine (0.5–1.0 mg daily).

Do not need to renal dose the first few days of prophylactic therapy for urate nephropathy prior to chemo, typically giving 600-800 mg/d for 2-3 days, then based on CC. **Avoid ETOH, ASA, thiazides (?, see below). Prophylactic colchicine (0.6 mg BID x6mo) when starting Allopurinol can reduce the risk for an acute flare of gout (33% Vs 77% with placebo), particularly if tophi are present (J Rheumatol 2004;31:2429-32).

Desensitization is possible, usually start with oral doses of 10-50 micrograms, then inc dose over 30 days (Arthritis Rheum 2001;44:231) (tab dissolve in 1% methylcellulose, day 1-3 50 ug, 4-6 100, 7-9 200, 10-12 500ug, 13-15 @ 1mg, 16-18 @ 5mg, 19-21 @ 10mg, 22-24 @ 25mg, 25-27 @ 50mg, 28+ @ 100mg). For mild disease, 200-300 mg qd is usually adequate, but the dosage should be individualized to achieve the desired serum urate level. Allopurinol is counterproductive and is to be avoided in acute attacks of gout. For severe tophaceous gout, 400-600 mg qd can be used. The maximum single dose should be 300 mg. Total daily doses in excess of 600 mg are associated with increased toxicity. It is advisable to prescribe colchicine (0.6 mg PO daily or BID) during the first 3 mo's of therapy as prophylaxis against acute gout. To prevent uric acid nephropathy during the tx of neoplastic disease, a 600-800 mg qd may be required, with the maintenance of large volumes of an alkaline urine.
Drug interactions: potentiation of the immunosuppressive and cytolytic effects of 6-mercaptopurine and azathioprine. Bone marrow suppression in pt's receiving alkylating agents, such as cyclophosphamide. Increase in the likelihood of an ampicillin-induced skin rash. Caution if use with uricosurics.
Febuxostat (Uloric): [40 and 80mg green tab] Start at 40 tab PO qd. If the uric acid level is not reduced to <6 mg/dL after 2 weeks, a dosage increase to 80 mg daily is recommended. Can be taken with or without food. A more selective xanthine oxidase inhibitor than allopurinol, but it's not known if this will offer more effective control of gout flares.
SE: (40 mg vs 80 mg) Liver function abnormalities (6.6% vs 4.6%), nausea (1.1% vs 1.3%), arthralgia (1.1% vs 0.7%), and rash (0.5% vs 1.6%).
Contra: should not be co-administered with xanthine oxidase substrate drugs such as Theophylline, Mercaptopurine, and Azathioprine because this can increase plasma concentrations of these drugs, leading to toxicity. Uloric should not be used to treat an acute gout attack. Pregnancy Cat C.
Monitoring: baseline serum uric acid level, LFT, and CBC are recommended. Follow-up of these tests should be done 2 and 4 months after starting Uloric, and then periodically (Prescriber's Letter. 2009;25(4):250408). Goal serum uric acid level is <6 mg/dL.
Info: A xanthine oxidase inhibitor. No dose adjustment needed if CC >80ml/min. Mainly metabolized in the liver by both oxidation via cytochrome P450 (CYP) enzyme including CYP1A2, 2C8, and 2C9 and non-P450 enzymes and conjugation via various uridine diphosphate glucuronosyltransferase (UGT) enzymes. Costs about $160/month...much more than allopurinol. Febuxostat is a novel nonpurine selective inhibitor of xanthine oxidase, is a potential alternative to allopurinol for pt's with hyperuricemia and gout. Febuxostat (80 mg or 120 mg qd) was more effective than allopurinol (300 mg PO qd) in lowering serum urate (NEJM 2005;353:2450-61)....both had similar reductions in gout flares and tophus area. Continue to prescribe allopurinol first for most patients (titrate the dose up to 800 mg/day if needed to get serum uric acid below 6 mg/dL) and save Uloric for pt's who can't take, or don't do well on, allopurinol (Prescriber's Letter. 2009;25(4):250408).

Modification:

A purine restricted diet is often unpalatable most as it is a vegetarian-type diet. In general, try increasing dietary protein intake from low fat dairy products and to decrease the amount of red meat and fish ingested. Ask the pt to decrease, meats (No organ meats), and ETOH. The hyperuricemic effect of alcohol has since been observed in many studies. Eating fruits with lot's of Vit-C is beneficial (see below). Best to drink 8 glasses of water per day.
MEDS: Avoid thiazides (?see below), full dose ASA, Niacin, Ethambutol and pyrazinamide.
Diuretics: If the pt is on a thiazide diuretic and has an acute attack of gout: 1) discontinue the diuretic, 2) re-check a uric acid level after the attack and assess the need for prophylaxis….3) restart or use another antihypertensive agent if uricosuric prophylaxis is not tolerated / not indicated (NEJM 2009;361:2161). Traditional teaching has been that thiazides and loop diuretics can increase UA levels by decr renal excretion Although many pt's present with gout for the first time while they are being treated with diuretics, it is the medical conditions that indicate diuretic use, not the diuretics themselves, that cause gout according to a Dutch study with 3,764 pt's (Ann Rheum Dis 2006;65:981-982,1080-1083)…..The incidence rate ratio (IRR) for gout was significant for HTN (IRR 3.9) and CHF (IRR 4.1), but the IRRs for diuretic use and MI were 0.6 and 1.5, respectively, neither of which was statistically significant….the authors recommend that diuretics should not be withheld from pt's because of a risk of gout and that diuretic therapy should not be switched if a pt's gets gout. The editorialists note that most of the information on hyperuricemia and gout seen after the introduction of diuretics to pt's who already have these predisposing conditions is drawn from observational data and has received little critical attention….they recommend further study into how the kidney handles urate when comorbidity exists, either by calculating the fractional excretion of urate or the urate clearance, along with measurement of serum uric acid. Recent diuretic use more than triples the risk of recurrent gouty arthritis (J Rheumatol 2006;33:1341-1345) (lower for thiazide diuretics -- 3.2-fold -- and slightly higher for loop diuretics -- 3.8-fold)….. could reduce risk of recurrent gout attacks by avoiding the use of thiazide and possibly loop diuretics in persons with preexisting gout.
Other Drugs that Lower Uric Acid: Cozaar (Losartan) has a mild uricosuric effect, it lowers serum urate 0.4mg/dL (Curr Ther Research 2001;62:79-91) (no effect with other ARBs). Fenofibrate can help lower uric acid levels by increasing its excretion. Losartan plus fenofibrate in combination in pt's with hyperuricemia lowered the average sUA from 7.6 mg/dl down to 4.9 mg/dl (J Cardiov Pharm 1999;34:60-63). Serum uric acid levels are significantly lowered by atorvastatin, but not simvastatin, in patents with primary hyperlipidemia (Am Heart J 2004;148:635-640). Amlodipine increases urate excretion (Neph Dial Transpl 2003;18:22147-53).
Vit-C: Data from several small studies have suggested that Vit- C (500mg qd) exert a uricosuric effect by reducing uric acid levels an average of 1.5 mg/dL (Arthritis Rheum 2005;52:1843-7) (uricosuric effect). If pt's ask about using this or cherry juice, tell them it might help increase renal excretion of uric acid and reduce gout flares. Suggest 500 to 1000 mg/day from food or supplements. A prospective cohort study vitamin C intake and risk of gout was evaluated in 46,994 men over a 20 year period found that compared with men with vitamin C intake less than 250 mg per day, absolute risk reductions of gout incidence associated with doses of vitamin C 500 mg to 999 mg, 1000 mg to 1499 mg, and 1500 mg per day or more were 27, 51, and 69 cases per 10,000 persons per year, respectively (Vitamin C intake and the risk of gout in men. Arch Intern Med 2009;169:502-7)…..thought to work by decreasing uric acid reabsorption in the kidney's proximal tubule. Warn pt's to not take over 2000 mg per day as high doses of vitamin C may lead to an increased risk of osmotic diarrhea and GI upset and kidney stone formation in patients with renal impairment or history of kidney stones.
Coffee: Long-term consumption of any coffee (not caffeine intake) is associated with reduced risk for gout in men older than 40 years (Arthritis Rheum. 2007;56:2048-2054)....multivariate RRs for 0, <1, 1 to 3, and 4 or more cups per day were 1.00, 0.83, 0.67, and 0.73, respectively.

Purine Content of Foods:

In pt's with normal dietary habits, the mean serum urate concentrations decrease ~1 mg/dL (59 µmol/L), this may be significant mostly in the management of pt's with severe renal insufficiency or those intolerance to pharmacologic therapy.
Extremely High: herring & roe, mussels, sardines, yeast, brain, sweet meats (thymus).
Very High: anchovies, crab, lobster, trout, salmon, turkey, veal, beef, bacon, pheasant, venison.
High: bass, halibut, oysters, tuna, chicken, pork, ham, duck. Each additional daily serving of meat was associated with a 21% increase in the risk of gout, and each additional weekly serving of seafood was associated with a 7% increase in risk (NEJM 2004;350:1093-103).
Note: Certain vegetables are high in purine but this form is **not** associated with gout as not as well absorbed (low bioavailability). This includes asparagus, cauliflower, kidney/ lima beans, spinach, whole grains, mushrooms, peas. High consumption of meat or seafood is associated with an increased risk of gout in men, but this is not the case for high consumption of vegetable protein or purine-rich vegetables (NEJM 2004;350:1093-103).
Fructose: Fructose, a common dietary sweetener causes hyperuricemia by accelerating the catabolism of adenine nucleotides, the ingestion of 5 apples causes a 35% increase in serum urate levels within 6 hours of consumption (Free Radic Biol Med 2004;37:251-8). Consumption of fructose from sugar-sweetened soft drinks and other sources increases risk for gout according to 12 year data from 46,393 male health professionals (BMJ 2008;336:309)(RR = 1.45 for one drink and RR = 1.85 for 2 or more)……consuming five to six drinks a week had a 29% increased risk; one a day, a 45% increase; and two or more daily, an 85% increase).....Other major contributors to fructose intake such as total fruit juice or fructose rich fruits (apples and oranges) were also associated with a higher risk of gout).....the risk of gout posed by the highest fifth of fructose intake was comparable to that seen with alcohol intake of 30 g to 50 g daily.....consumption of diet soft drinks was not associated with gout. A diet high in fructose (table sugar and high-fructose corn syrup) can increase uric acid levels (Perez-Pozo S, et al "Excessive fructose intake raises blood pressure in humans" AHA BPRC 2009;Abstract P127).....typical source is from sugary drinks sweetened with high-fructose corn syrup.
The best diet: whole grains and cereals, low fat dairy, eggs, nuts, fruits, peanut butter, tomatoes, green vegetables, milk products, fruit juices, lettuce and tomatoes. Low-fat dairy product consumption is associated with a decreased risk of gout. Other food that are low in purine, but not necessarily reccomended include refined / white breads, pasta, flour, sugar, sweets, cheese, gelatin, carbonated drinks, macaroni, tea/ coffee, chocolate, butter, soft drinks. (Recent developments in diet and gout. Curr Opin Rheum 2006;18:193–198)
Under secreter and Overproducer: Seen with alcohol, hypoxemia & tissue underperfusion, glucose 1-phosphate def and fructose 1-phosphate –aldolase def.
Info: Mildly elevated levels of uric acid have been linked to a 5-fold increased odds of high volume of white-matter hyperintensities in aging adults (Neurology. 2007;69:1418-1423)....confirms the role of UA in increasing the risk for cerebrovascular disease and that may also explain the link between elevated UA and mild cognitive impairment (MCI) in aging adults.

Ddx of Hyperuricemia:

Hyperuricemia (i.e., serum uric acid concentration greater than 6.5 mg per dL [385 µmol per L]) is common in the general population. If asymptomatic and <8, then most need no tx. It is most often caused by a combination of a high purine diet, alcohol use, diuretic therapy, and reduced renal clearance. It can be due to an overproduction of uric acid, undersecretion of uric acid, cancer chemo, Glycogen storage disease, Gout, hyperparathyroidism, primary HTN, Hemoglobinopathies, Ketoacidosis, Hemolytic anemia, Diabetic, Lesch-Nyhan syndrome, Starvation, Myeloproliferative d/o's, Lactic acidosis, Multiple myeloma, Alcoholism, Myeloid metaplasia, Preeclampsia, Waldenström's macroglobulinemia, Lead nephropathy, Neoplastic diseases, Chronic Renal failure, Lymphoma, Polycythemia, idiopathic, Psoriasis, Down's syndrome, Hypoparathyroidism, Obesity, Paget's disease, Sarcoidosis.
Urate underexcretion: primary/ idiopathic. Secondary (dec renal function, inhibition of urate secretion from acidosis), unknown (HTN, lead neuropathy, drugs such as ASA and ethambutol).
Urate overproduction: primary hyperuricemia (phosphoribosyl pyrophosphate synthetase overactivity, hypoxanthine-guinine phosphoribosyl transferase def), secondary (diet, high nucleotide turnover, inc ATP degradation from vigorous muscle exertion).
Combined: G6-PD def, tissue hypoperfusion. Inc alcohol consumption.
Drugs causing inc Uric Acid--> Acetazolamide, Isoprinosine, Amiloride, Isotretinoin, Bumetanide, Ketoconazole, Chlorthalidone, Levodopa, Cisplatin, Metolazone, Cyclophosphamide, Phencyclidine, Ethacrynic acid, Piretanide, Ethambutol, Pyrazinamide, Furosemide, Thiazide diuretics, Indapamide, Vincristine.

Primary Hyperuricemia

A. Increased production of purine:
1. Idiopathic.
2. Specific enzyme defects (eg, Lesch-Nyhan syndrome, glycogen storage diseases).
B. Decreased renal clearance of uric acid (idiopathic).

Secondary Hyperuricemia

A. Increased catabolism and turnover of purine:
1. Myeloproliferative disorders.
2. Lymphoproliferative disorders.
3. Carcinoma and sarcoma (disseminated).
4. Chronic hemolytic anemias.
5. Cytotoxic drugs.
6. Psoriasis.
B. Decreased renal clearance of uric acid:
1. Intrinsic kidney disease.
2. Functional impairment of tubular transport:
a. Drug-induced (eg, thiazides, probenecid).
b. Hyperlacticacidemia (eg, lactic acidosis, alcoholism).
c. Hyperketoacidemia (eg, diabetic ketoacidosis, starvation).

d. Diabetes insipidus (vasopressin-resistant).
e. Bartter's syndrome.
Ref:(Am Fam Phys 1999;59:4) (Textbook of Rheumatology, 5th ed., Kelly, 1997, WB Saunders) (Rheum Dz NA 1990;16:539-50) (NEJM 1996;334:7) (Postgrad Med 1999;106:4)

Calcium Pyrophosphate Dihydrate (CPPD) (Pseudogout):

Links: W/u | **Tx** | Ddx | …….END DEMO of this chapter.

Trauma & Toxicology:

Edited by Chris J. Flynn MD and Bob Gayou MD, Emergency Physicians.

Contents: ATLS | **ACLS / BLS** | PALS | **TRAUMA** | Intubation | **Asthma** | Anaphylaxis | **Oncologic** | Psychiatric | **Toxicology** | Toxidromes | **Toxic Food** | Inhalational | **Marine** | Bites / Stings | **Burns** | Radiation | **Environmental Exposures** | Rape | **Substance Abuse** | Foreign Bodies | **PEDIATRIC** | Wounds & Suturing & Coding | **Coma** | Bio-Chem-Nuke Weapons | **Status Epilepticus** | Cervical-Thoracic-Lumbar Pain | **Pregnancy** | Wilderness | **Disaster Medicine** | Sedation | ***INDEX*** |

Advanced Trauma Life Support (ATLS):

Links: Triage | Primary Survey & MAST Suits | **Secondary Survey** | Step #2: **Trauma Eval & Tx** | **ACLS / BLS** | PALS | **Splinting & Bracing** |

External hemorrhage should be controlled by direct manual pressure. Blind clamping in a wound should not be performed, but clamping bleeding vessels under direct visualization is acceptable and effective. The careful use of a tourniquet is reserved for cases of amputation or when other measures have been ineffective.
Triage: Triage in a mass casualty incident assumes that not all persons requiring care can be readily accommodated. The rationale behind the triage is to sort pt's into classes according to the severity of injury, for the purpose of expediting clinical care and maximizing the use of the available clinical services and facilities. Focuses on the **salvage of the maximum number of pt's based on current resources** by prioritizing pt's into 4 main categories: **Pt's are generally tagged, with the tags color-coded as follows:**
Red = Emergent. Need immediate tx as will die 15 min- 6 hrs after the injury, due to a cardiovascular and/or pulmonary collapse. This usually includes: Asphyxia. Respiratory obstruction from mechanical causes. Sucking chest wounds. Tension pneumothorax. Maxillofacial wounds in which asphyxia exists or is likely to develop. Shock caused by major external hemorrhage. Dislocations. Severe burns over <25% of BSA. Lesser fx's. Major medical problems that are readily treatable.
Yellow = Urgent / Delayed tx priority (must be treated within 1-2 hours): Major fx's (if able to stabilize). Visceral injuries or evisceration. Cardiopericardial injuries. Massive muscle damage. Severe burns over >25% of BSA. Vascular injuries requiring repair. Wounds of the genitourinary tract. Thoracic wounds without asphyxia. Closed cerebral injuries with increasing loss of consciousness. Spinal cord injuries requiring decompression. Suspected spinal cord injuries without neurologic signs. Eye injuries. Maxillofacial injuries without asphyxia. Complicated major medical problems.
Green = Non urgent. Minor medical problems that can be put off until later or healthy.
Black = Dead or very severely injured and not expected to survive (the most difficult to assign because of the obvious ominous implications, these pt's clearly, and without a doubt, are so severely injured that no degree of medical help will relieve them as have a fatal disruption of the great vessels, heart, and lungs, or a major disruption of body cavities)
Other info: Also need to consider vital signs and pre-hospital clinical course, mechanism of injury, age and known or suspected comorbid conditions. Accelerated workup indicated if multiple injuries, extremes of age, evidence of severe neurological injury, unstable vital signs, and preexisting cardiac or pulmonary disease.
Other Types of triage:
Category I (obvious emergency): Physician must examine the pt as soon as possible. Case examples include cardiac arrest, acute severe chest pain, massive vomiting of blood, sudden loss of consciousness, and major trauma with hypotension. Asphyxia. Respiratory obstruction from mechanical causes. Sucking chest wounds. Tension pneumothorax. Maxillofacial wounds in which asphyxia exists or is likely to develop. Shock caused by major external hemorrhage. Major internal hemorrhage. Visceral injuries or evisceration. Cardiopericardial injuries. Massive muscle damage. Severe burns over more than 25% of body surface area. Dislocations. Major fx's. Major medical problems readily correctable. Closed cerebral injuries with increasing loss of consciousness.
Category II (strong potential for emergency): Pt will need a full evaluation and tx by a physician. Case examples include acute dyspnea, acute abd pain, acute chest pain, acute confusion, and severe pain.
Category III (potential emergency): Consider the possibility of an occult or pending emergency condition. Physician must examine the pt during the ED visit, because an emergency cannot be excluded by an initial screening examination.
Category IV (**non emergent):** There is no reason to think that these pt's have an emergency medical condition or are at risk of developing one.
Other Info: 5 readily available clinical criteria help identify pt's who need immediate advanced trauma care at a trauma center were found to be need for an emergent airway intervention, initial GCS score <11, blood transfusion, initial SBP <100 or >220 mm Hg, and initial RR <10 or >32 (Prehosp Emerg Care 2007;11:154-63). For pt's with certain life-threatening emergencies, mortality increases with increasing distance from the emergency department (Emerg Med J 2007;24:644-653)...relative risk for death increased by 2% with each additional kilometer traveled. Determining retrospectively whether patients had injuries that were serious enough to require the resources of a major trauma center often is based on anatomic injury measured by the Injury Severity Score (ISS), but it does not correlate well with the need for specialized trauma resources (Prehosp Emerg Care 2008;12:451).....A better determination of "trauma center need" should be developed; one approach might be to combine use of specialized trauma resources and the ISS.
The Manchester triage system: A computerized complaint-based algorithm with 52 pathways (49 suitable for children) that uses six key discriminators (life threat, pain, hemorrhage, acuteness of onset, level of consciousness, and temperature) to sort patients into one of 5 urgency categories, corresponding to maximum waiting time:
Immediate — 0 minutes.

Very urgent — 10 minutes.
Urgent — 60 minutes.
Standard — 120 minutes.
Nonurgent — 240 minutes.
The Manchester triage system has moderate validity for pediatric ED patients according to an a prospective observational study of 17,600 children (age, <16 years) (BMJ 2008;337:a1501).....triage is particularly challenging in patients with medical (rather than traumatic) problems and in infants younger than 3 months.

Guidelines for Field Triage of Injured Patients Recommendations of the National Expert Panel on Field Triage:
(MMWR 1009;8(RR01);1-35)

Step 1: Physiologic Criteria: To guide EMS personnel in identifying critically injured patients rapidly through measuring their vital signs and assessing their level of consciousness.

The Panel recommended transport to a trauma center if any of the following are identified:
• Glasgow Coma Scale of <14,
• SBP <90 mmHg, or RR <10 or >29 breaths per minute (<20 in infant aged <1 year).
• Other Physiologic Observations: Abnormal pulse rate and skin findings should prompt EMS providers to seek other physiologic indications of severe injury.

Transition from Step One to Step Two: Patients meeting the physiologic criteria of Step One have potentially serious injuries and should be transported to the highest level trauma center (i.e., Level I, if available).

Step Two: Anatomic Criteria: recognizes that certain patients, on initial presentation to EMS providers, might have a severe injury and need care at a high-level trauma center but have physiologic parameters that do not meet the criteria of Step One

The Panel recommended transport to a trauma center if any of the following are identified:
• all penetrating injuries to head, neck, torso, and extremities proximal to elbow and knee;
• flail chest;
• two or more proximal long-bone fractures;
• crushed, degloved, or mangled extremity;
• amputation proximal to wrist and ankle;
• pelvic fractures;
• open or depressed skull fracture; or
• paralysis.

Step Three: Mechanism-of-Injury Criteria: A patient who does not meet Step One or Step Two criteria might still have severe, but occult, injury.

The Panel recommended transport to a trauma center if any of the following are identified:
• falls: Adults: fall >20 feet (one story = 10 feet). Children aged <15 years: fall >10 feet or two to three times child's height;
• high-risk auto crash: Intrusion: >12 inches to the occupant site or >18 inches to any site. Ejection (partial or complete) from automobile. Death in same passenger compartment. Vehicle telemetry data consistent with high risk of injury;
• auto versus pedestrian/bicyclist thrown, run over, or with significant (>20 mph) impact; or
• motorcycle crash >20 mph.

Step Four: Special Considerations: EMS personnel must determine whether persons who have not met physiologic, anatomic, or mechanism-of-injury criteria have underlying conditions or comorbid factors that place them at higher risk for severe injury.
The Panel recommended that transport to a trauma center or specific resource hospital be considered if any of the following are identified:
• age: Adults aged >55 years. Children aged <15 years;
• anticoagulation and bleeding disorders;
• burns: Without other trauma mechanism: triage to burn facility. With trauma mechanism: triage to trauma center;
• time-sensitive extremity injury;
• end-stage renal disease requiring dialysis;
• pregnancy >20 weeks; or
• EMS provider judgment.

Primary Survey:

Mnemonic ABCDEF: airway, breathing, circulation/hemorrhage, disability, exposure/environment, fluids. See **Secondary Survey & FAST** |

#1 Airway: look for obstruction of the airway due to direct injury, edema, or foreign bodies, and the inability to protect the airway due to a depressed level of consciousness. Tx may be simply secretion control with suctioning or may require endotracheal intubation or placement of a surgical airway (cricothyroidotomy, emergent tracheostomy). If pt is talking clearly then airway adequate (at least for that moment).

#2 Breathing (ability to ventilate and oxygenate), look for absence of spontaneous ventilation. R/o causes of rapid deterioration --> absent or asymmetric breath sounds (PTX or endotracheal tube malposition), dyspnea, hyper-resonance or dullness to chest percussion (tension PTX or hemothorax), and gross chest wall instability or defects that compromise ventilation (flail chest, sucking chest wound). **Identify and treat the following potentially fatal conditions that require immediate attention:** Flail Chest | **Hemothorax** | Pneumothorax | and sucking chest wounds / Tracheobronchial disruption (Chest trauma). Most of these treated with a tube thoracostomy (Chest Tube).

#3 Circulation, look for hypovolemia, cardiac tamponade, and external sources of hemorrhage. Usually need LR via 2 large-bore, peripheral, IV catheters. Place them preferentially in the upper extremities. Treat cardiac tamponade by pericardiocentesis, or place a subxiphoid pericardial window (then surgery to explore and repair the source of bleeding). Control any external hemorrhaging with direct pressure.

Asses Blood Loss:
Class I: 0-15%--> minimal tachycardia. This degree of blood loss is usually fully compensated by transcapillary refill.
Class II: 15-30%--> tachycardia, tachypnea, dec pulse pressure, anxiety. A positive tilt test. Need 2-3L of fluid give crystalloid in a 3:1 ratio per ml blood loss.

Class III: 30-40%--> get mental status changes and dec SBP & urine output (onset of hypovolemic shock. Transfuse ASAP if signs of end-organ hypo-perfusion.
Class IV: >40%--> narrow pulse pressure, severely hypotensive, skin is pale and cold, needs immediate transfusion as harbinger of circulatory collapse, evidence of organ failure. If pt does not respond to initial respond to initial fluid bolus consider ongoing blood loss. As a guide, if the pt remains hemodynamically unstable after 2-3 L or 40-50 ml/kg of crystalloid, blood should be started. Fully crossmatched blood is preferable, but it may take 30-45 min to obtain it, thus type-specific blood is a safe alternative and is usually ready in 5-15 min. Use an autotransfusion device if available. A platelet count <100,000/mm3 in the face of bleeding is an indication for platelets. Efforts should be made to prevent hypothermia, use of warm blankets, heat lamps, and blood warmers for all transfused blood or saline.
Estimate blood loss: a standard field dressing holds about 0.25 liter (one-fourth canteen) of blood. A soaked T-shirt holds 0.5 to 0.75 liter.
#4 Disability, gross mental status and motor examinations, look for serious head or spinal cord injury. Assess the gross mental status using the AVPU scale (response to stimuli: alert, responding to voice, pain, or unresponsive) or the Glasgow Coma Scale. Examine the pupils for size, symmetry, and reactiveness to light. Obtain an early assessment of spinal cord injury by observing spontaneous movement of the extremities and spontaneous respiratory effort. Pupillary asymmetry or dilation, impaired or absent light reflexes, and hemiplegia or weakness suggest impending herniation of the cerebrum through the tentorial incisura due to an expanding intracranial mass or diffuse cerebral edema. These findings indicate the need for emergency tx of intracranial HTN, including administration of IV mannitol, sedatives, and paralytics, after the establishment of a definitive airway.
#5 Exposure and control of the immediate environment, remove pt's clothes for a thorough exam. Simultaneously, initiate tx to prevent hypothermia (warmed IV fluids, blankets, heat lamps, and warmed air-circulating blankets).
#6 F: Fluids/ Resuscitation phase: aim for stable vital signs, most need several liters of crystalloid over the first 24 hours to sustain intravascular volume, aim for urine output of 0.5-1.0 ml/kg/h. Any shock pt needs 2 large bore IV's, ≤16g.
Pain: Oral transmucosal fentanyl is useful for pain control in trauma pt's without immediate hospital access, such as in a combat situation as rapid-onset (15min) as minimal SE's (Ann Emerg Med 2004;44:121-127).
Vascular Shunt (Temporary Limb Salvage Shunt): FDA approved in 2/07 for critical war injuries. Expected to reduce the number of arm and leg amputations. The plastic shunt can be used to connect the ends of severed blood vessels, thereby providing a bridge across the damaged area and restoring blood flow to the injured limb until the pt can be transported to surgery. Has a self-sealing elastomer membrane that allows direct injection of medications into the shunt, beveled ends for quick and effective placement, graduated markings for visual confirmation of proper placement, and extra reinforcement in the center of the device so that it can be cut to a shorter length if needed.
Pneumatic Antishock Garment (PASG): widely available and commonly used, both by emergency medical technicians and paramedics in the out-of-hospital care of hypotensive pt's and, to a lesser extent, by hospital emergency departments and critical care personnel. In its commercially available forms—**MAST Suit** (military antishock trousers) and Gladiator antishock pants, resembles a pair of high-wasted men's trousers. The devices are constructed from two layers of an opaque, airtight fabric sewn into three independently inflatable chambers. The increase in blood pressure that results from application of PASG is due to at least three effects: enhanced venous return (autotransfusion), increased total peripheral resistance, and reduced volume loss from control of hemorrhage (acting as a pneumatic splint).
Indications: Hypovolemic shock, Spinal shock, Drug overdose, Septic shock, Anaphylaxis, "Prophylactic" use (GI bleeding, aortic aneurysm), Stabilization of fx's, Compression of external bleeding, Postoperative hemorrhage, Coagulation defects.
Contra: pulmonary edema, CHF / Cardiogenic shock and penetrating thoracic trauma. Relative contra includes pregnancy, evisceration, impaled foreign body, lower extremity compartmental injury, lumbar spine instability. Do not use the abd portion in 3rd trimester of pregnancy.
Technique:
Step #1: Lay suit out & transfer the supine pt onto it. The pt can be "logrolled" onto the opened trousers. Alternatively, with one person standing on either side and elevating the pt's legs, the trousers can be slid beneath to the buttocks. Then the pt's hips are elevated slightly, and the upper border of the trousers is placed at the costal margin. The cervical spine must be stabilized in those at risk for cervical injury.
Step #2: The medial portion on each leg binder is brought between the legs, and the Velcro fasteners are closed over each leg and over the abdomen. Wrap legs & abdomen, avoiding distortion of the suit and secure with ties.
Step #3: Attach pump device and check that valves are open. Gradually inflate legs then abdomen until BP is sufficiently raised. Close both valves in each tubing, as this allows for pump to be disconnected without deflating the suit with sudden loss of BP. If the pt is very small or a child, place a folded towel/sheet between legs and use either one leg compartment or the abd compartment to wrap both legs together, then inflate as above.
Pearls: never suddenly deflate the suit as you may suddenly lose blood pressure. When IV replacement has been begun, slowly reduce the pressure one compartment at a time, starting with abdomen; hold whenever the BP falls more than 5mmHg to allow fluid replacement to catch up. Saphenous vein IV's in the feet may be used with the suit fully inflated; although they may require pressure bags.
Complications: suit works by decreasing afterload and will rapidly worsen CHF. Once the suit is inflated the abdomen and lower extremities are covered and easily forgotten - keep them in mind. The cost for a PASG, pump and other equipment ranges from $500 for the adult size without gauges and $6-800 for the adult size with gauges. The cost for the pediatric size ranges from $450-800 for a unit without gauges and from $500-900 for a unit with gauges.

Secondary Trauma Survey:

(2 min's), after starting the resuscitation phase. Conducting a thorough head-to-toe exam (fingers or tubes in every orifice, log roll the pt, palpate for any tenderness, neuro, all bones, CV, X-rays). Splint major fx's (briefly to prevent further soft tissue injury). See **Step #2: Trauma Eval Tx** | FAST U/S scan |
Cushing Reflex: inc BP, dec HR, dec RR. Look for basilar skull fx, battle sign (bilateral mastoid ecchymosis), raccoon eyes (bilateral periorbital ecchymosis). CSF rhinorrhea, otorrhea or hemotympanum. "fingers and tubes in every orifice"
Check: ECG, ABG, CBC, pulse oximetry. Once the airway is secured, place NGT to decompress the stomach to minimize aspiration risk (contra if a basilar skull fx). Insert a Foley catheter to facilitate measuring the response to fluid resuscitation (contra if urethral injury, blood at the meatus, ecchymosis in the scrotum or labium majora, or a high-riding prostate, if yes, a retrograde

urethrogram to rule out urethral injury). Review the vital signs, and perform a quick repeat of the primary survey, review the pt's history, including reports from pre-hospital personnel and from family members or other victims. If the pt is awake, collect data on PMHx, current meds and allergies, tetanus shot, time of last meal, and events surrounding the injury. Link: **Splinting |**
Rectal: A study with 1032 blunt trauma pt's found that spinal cord injury was diagnosed in 5.2% overall, 2.7% of those with normal rectal exams and in 27.3% of those with abnormal exams (Am J Emerg Med 2006;24:113-7), the digital rectal exam had a sensitivity of 50%, specificity of 93%, positive predictive value of 27%, and negative predictive value of 97%.....not an outstanding track record, but not provide sufficient evidence to abandon a fairly simple, minimally invasive procedure.
Point-of-care limited U/S (PLUS) in the emergency department evaluation of trauma pt's reduced time to surgery, length of stay in the hospital, and costs (Ann Emerg Med 2006;48:227-35) (used the FAST technique).
The Focused Abdominal Sonogram for Trauma (FAST) and Handheld U/S for Cardiac Scanning:
Advantages of theFAST include bedside performance, lack of ionizing radiation, ability to perform serial examinations, and 70% to 90% sensitivity overall for detecting hemoperitoneum. Complements the portable chest and pelvis films, this has supplanted diagnostic peritoneal lavage (DPL) for rapid assessment of unstable trauma pt's. CT imaging of the abdomen, pelvis, and head is the most sensitive and accurate noninvasive diagnostic tool for identifying soft-tissue injury. Urine dip to rule out occult hematuria. Urine screens for drugs, Blood alcohol and glucose. Has a sensitivity of 70-98% and a specificity of 95-99% when performed by trauma surgeons with appropriate training. Can be performed in an average of 2–4 minutes. The handheld FAST provides an extremely rapid and reliable test for free intraperitoneal fluid in severely injured pt's, which after penetrating trauma almost always indicates significant injury (Am J Surg 2004;187:660-5). A negative examinations be followed by more extensive diagnostic evaluation because of the limited ability of handheld FAST to reliably detect intra-abd injuries.
Indications: all trauma pt's, after the initial primary survey, who do not already require emergent operative intervention.
Equipment: U/s probe and monitor. Water-soluble hypoallergenic transmission gel.
Step #1: position pt supine. Done after initial stabilization and primary survey, warmed U/S gel is applied to 4 key areas in the thoracoabdominal region (Pericardial area, RUQ, LLQ, Pouch of Douglas = suprapubic area).
Step #2: the transducer is oriented for sagittal sections and then placed first over the subxiphoid region to examine the heart and pericardial sac for blood. The transducer is placed over the right midaxillary line between the eleventh and twelfth ribs. Examine the sagittal section of the liver, kidney, and diaphragm. Examine Morison's pouch for blood. The transducer is placed over the left posterior axillary line between the eleventh and twelfth ribs. Examine the spleen and kidney. Examine the splenorenal recess and subdiaphragmatic space for blood. Lastly, the transducer is placed 4 cm superior to the symphysis pubis, directed for coronal sections, and then moved inferiorly in a side-to-side fashion to examine the bladder and pouch of Douglas for blood.
Limitations: A FAST exam is helpful when the findings are positive; however, a negative study must be evaluated further (CT scan, repeat evaluations, observation).
A falsely negative FAST exam: Too little time has elapsed since the trauma for the blood to accumulate. Bleeding may be retroperitoneal and not visible on FAST exam. Solid organ injury with encapsulated bleeding is present. Hollow viscus injury is present. Inadequate volume of fluid. As little as 50 cc of fluid can be detected; however, it may take up to 250 cc of fluid to create an unequivocally positive FAST exam.
Traumatic pneumothorax (PTX): Results of a prospective study suggest that lung ultrasonography performed in the emergency department may be nearly as accurate as CT in detecting occult traumatic PTX and its size (Chest 2008;133:204-211)...."We believe that echocardiographic evaluation of the thorax should be performed during the primary survey as an extension of the FAST (focused assessment with sonography for trauma) examination in every trauma patient."
Hemoperitoneum: FAST when performed by radiology residents had low sensitivity for detecting hemoperitoneum in hemodynamically unstable trauma patients (J Trauma 2009;67:323)....a negative FAST result in patients with signs of hemorrhagic shock owing to intraperitoneal trauma should lead to diagnostic peritoneal lavage — the critical aspect being the initial peritoneal aspirate — or to CT, if CT can be performed rapidly and safely in the confines of the trauma bay.
Hand-carried U/S (HCU): Real-time cardiovascular imaging with a HCU enhances the physical examination of pt's with cardiovascular disease (Chest 2004;126:693-701). The HCU is particularly useful in detecting global left ventricular dysfunction, but may be less valuable for less experienced operators in diagnosing segmental wall motion abnormalities, right ventricular dysfunction, and valvular lesions.
The HCU is battery-operated, lightweight, and about the size of a laptop computer. It is particularly suitable in three settings, emergency and critical care medicine, community screening and remote areas. The American Society of Echocardiography and the American College of Cardiology/American Heart Association recommend a minimum level 2 training in echocardiography (defined as at least 150 studies performed and 300 studies interpreted under supervision) for the use of an HCU device as an extension of the physical examination. Bedside echocardiography trauma/critical care (BEAT) examination was found to be effective for assessing cardiac function in in 22 patients using a 5- to 2-MHz cardiac ultrasound probe (J Trauma 2008;65:509)...Estimates of stroke volume and inferior vena cava diameter correlated with pulmonary artery catheter–derived measurements of cardiac index and central venous pressure.....The exam required approximately 20 minutes to perform (once examiners had conducted at least 5 exams), and the bulk of time was spent on calculations, not the exam itself.
Cardiac Scan: Intensivists and emergency department physicians currently use hand-carried ultrasound echocardiography (HCUE) to make rapid point-of-care patient assessments. Researchers at a 500-bed teaching hospital in Chicago designed a brief HCUE training program to teach procedures for assessing six important cardiac abnormalities (J Hosp Med 2009;4:340): Left ventricular (LV) systolic dysfunction; Severe mitral regurgitation; Moderate-to-severe left atrial enlargement; Moderate-to-severe LV hypertrophy; Medium-to-large pericardial effusion; Dilated inferior vena cava.....Trainees received a 2-hour lecture from a cardiologist on the basic principles of HCUE, 20 hours of hands-on training (on at least 30 patients) from the instructor, and feedback from a cardiologist investigator (total training period, 27 hours).....Positive likelihood ratios for HCUE tests ranged from 2.5 (for LV hypertrophy) to 21 (for pericardial effusion); negative likelihood ratios ranged from 0 to 0.4. These likelihood ratios changed the baseline odds 5-fold for three cardiac abnormalities (LV systolic dysfunction, mitral regurgitation, and pericardial effusion) and 2-fold for the other three cardiac abnormalities. For example, the positive likelihood ratio for LV systolic dysfunction was calculated as 6.9, and the negative likelihood ratio was 0.2: The HCUE result could change the probability of actual LV systolic dysfunction (pretest probability, 50%) to 15% or 85%.
Cardioscan: a computer-aided medical device is intended for use by clinicians in the analysis of heart sounds for suspected murmurs. A graphical display includes the median energy level, timing, and duration of suspected heart murmurs during specific

segments of the systolic and diastolic intervals of heartbeats recorded through the device. The display is intended to better represent the distribution of acoustic energy at each recorded cardiac location, thus aiding in the characterization of suspected murmurs with respect to onset, duration, and loudness.

Splinting:

Link: Fiberglass & Plaster Splints | Common Splints Used | **Slings** | Taping and Wrapping | See Travel Medicine Module for Wilderness Medical Kit | **Wilderness Splinting, Litters & Carries** | Rescue Carries |

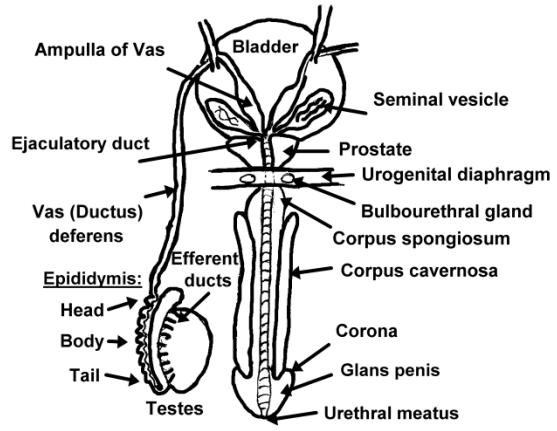
…….END DEMO of this chapter.

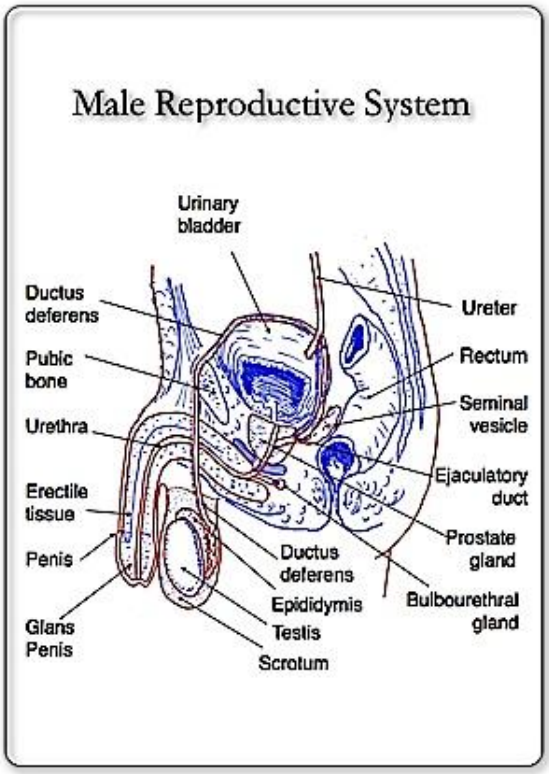
Urology:

Edited by Alex J. Weinstein MD, FACS, Urologist.

Contents: Anatomy | **Renal Stones** | INFECTIONS | **Urinary Retention** | Priapism | **Trauma** | CANCERS | **Penile** | PEDIATRIC | **Scrotal Pain** | Testicular Mass | **Erectile Dysfunction** | Sexual Disorders | **Male Hypogonadism** | Peyronie | **Prostate** | Bladder | **Nocturia** | Incontinence | **Catheters** | Interstitial Cystitis | **Male Infertility** | Hematuria | **Proteinuria** | Urinalysis | **Urodynamics and Studies** | PSA Testing | **Urethra** | Vasectomy | **MEDS** | BPH | **Pelvic Relaxation** | Lower Urinary Tract Sx's (LUTS) | **Nephrology** | Various Renal Disease & Masses | ***INDEX*** |

Male Anatomy: Link: See Womens Health: **Reproductive / Anatomy** |





Male Genital Organs

Scrotum: an outpouching of the lower part of the anterior abdominal wall. It contains the testes, the epididymides, and the lower ends of the spermatic cords. The wall of the scrotum has the following layers:
1. Skin: The skin of the scrotum is thin, wrinkled, and pigmented and forms a single pouch. A slightly raised ridge in the midline indicates the line of fusion of the two lateral labioscrotal swellings. In the female, the swellings remain separate and form the labia majora.
2. Superficial fascia: This is continuous with the fatty and membranous layers of the anterior abdominal wall; the fat is, however, replaced by smooth muscle called the dartos muscle. This is innervated by sympathetic nerve fibers and is responsible for the wrinkling of the overlying skin. The membranous layer of the superficial fascia (often referred to as Colles' fascia) is continuous in front with the membranous layer of the anterior abdominal wall (Scarpa's fascia), and behind it is attached to the perineal body and the posterior edge of the perineal membrane.
3. Spermatic fasciae: These three layers lie beneath the superficial fascia and are derived from the three layers of the anterior abdominal wall on each side. The external spermatic fascia is derived from the aponeurosis of the external oblique muscle; the cremasteric fascia is derived from the internal oblique muscle; and, finally, the internal spermatic fascia is derived from the fascia transversalis. The cremaster muscle is supplied by the genital branch of the genitofemoral nerve. The cremaster muscle can be made to contract by stroking the skin on the medial aspect of the thigh. This is called the cremasteric reflex. The afferent fibers of this reflex arc travel in the femoral branch of the genitofemoral nerve (L1 and 2), and the efferent motor nerve fibers travel in the genital branch of the genitofemoral nerve. The function of the cremaster muscle is to raise the testis and the scrotum upward for warmth and for protection against injury. Normal spermatogenesis can occur only if the testes are at a temperature lower than that of the abdominal cavity.
Testis: a firm, mobile organ lying within the scrotum. The left testis usually lies at a lower level than the right. Each testis is surrounded by a tough fibrous capsule, the tunica albuginea. Extending from the inner surface of the capsule is a series of fibrous septa that divide the interior of the organ into lobules. Lying within each lobule are one to three coiled seminiferous tubules. The tubules open into a network of channels called the rete testis. Small efferent ductules connect the rete testis to the upper end of the epididymis.
Epididymis: a firm structure lying posterior to the testis, with the vas deferens lying on its medial side. It has an expanded upper end, the head, a body, and a pointed tail inferiorly. Laterally, a distinct groove lies between the testis and the epididymis, which is lined with the inner visceral layer of the tunica vaginalis and is called the sinus of the epididymis.
Vas Deferens: a thick-walled tube about 18 in. (45 cm) long that conveys mature sperm from the epididymis to the ejaculatory duct and the urethra. It arises from the lower end or tail of the epididymis and passes through the inguinal canal. It emerges from the deep inguinal ring and passes around the lateral margin of the inferior epigastric artery. It then passes downward and backward on the lateral wall of the pelvis and crosses the ureter in the region of the ischial spine. The vas deferens then runs medially and downward on the posterior surface of the bladder. The terminal part of the vas deferens is dilated to form the ampulla of the vas deferens. The inferior end of the ampulla narrows down and joins the duct of the seminal vesicle to form the ejaculatory duct.
Seminal Vesicles: two lobulated organs about 2 in. (5 cm) long lying on the posterior surface of the bladder. On the medial side of each vesicle lies the terminal part of the vas deferens. Posteriorly, the seminal vesicles are related to the rectum. Inferiorly, each seminal vesicle narrows and joins the vas deferens of the same side to form the ejaculatory duct. Each seminal vesicle consists of a much-coiled tube embedded in connective tissue.
Spermatic Cord: a collection of structures that pass through the inguinal canal to and from the testis). It begins at the deep inguinal ring lateral to the inferior epigastric artery and ends at the testis. The structures are as follows: Vas deferens. Testicular artery. Testicular veins (pampiniform plexus). Testicular lymph vessels. Autonomic nerves. Remains of the processus vaginalis. Genital branch of the genitofemoral nerve, which supplies the cremaster muscle.
Ejaculatory Ducts: The two ducts are each less than 1 in. (2.5 cm) long and are formed by the union of the vas deferens and the duct of the seminal vesicle. The ejaculatory ducts pierce the posterior surface of the prostate and open into the prostatic part of the urethra, close to the margins of the prostatic utricle; their function is to drain the seminal fluid into the prostatic urethra.
Prostate: a fibromuscular glandular organ that surrounds the prostatic urethra. It is about 1.25 in. (3 cm) long and lies between the neck of the bladder above and the urogenital diaphragm below. The prostate is surrounded by a fibrous capsule. The somewhat conical prostate has a base, which lies against the bladder neck above, and an apex, which lies against the urogenital diaphragm below. The two ejaculatory ducts pierce the upper part of the posterior surface of the prostate to open into the prostatic urethra at the lateral margins of the prostatic utricle.
Abx Prophylaxis prior to Urologic Surgery: need to cover Streptococci and G- bacilli. 30 minutes before surgery give Cefazolin 1 g IV q8h for 1-3 dose then (TMP-SMX or nitrofurantoin) PO for 10 days or until a catheter is removed.
Transrectal Prostate Biopsy: 30 minutes before surgery give Ciprofloxacin 500 PO 12 hrs prior and after biopsy.

Urinary Calculi (Stones / Urolithiasis / Nephrolithiasis):

Links: PP, Risks & ICD9 | S/s | **Lab & Radiology |** Tx | **PV |** W/u Recurrent | **Underlying Etiology |** Hypercalciuria | **Hyperuricuria (Uric Acid Stones) |** Hyperoxaluria | **Hypocitruria |** Struvite | **Cystinuria |** Calcium-Phosphate | **Bladder & Prostate Stones & Ref's |**

A relatively common problem, has a 5-15% lifetime risk worldwide, M: F, 3:1, most between ages 20-50, has a 50% recurrence rate within 5 years. The first episode in men tends to occur at an average age of 30 yo, where as women have a bimodal age of onset, with episodes peaking at 35 and 55 years. Peak incidence is the early AM due to hyperconcentration and low urine output. 55% have a FHx of stones. Kidney stones are solid deposits that form in the kidneys from substances excreted in urine. Children under 16 years of age constitute ~ 7% of all cases of renal stones with a 1:1 sex distribution. ~80% of pt's with urolithiasis form calcium stones, most of which are composed primarily of calcium oxalate, and less often, calcium phosphate.
PP: The precise cause of urinary stone formation is unknown. It requires three elements: supersaturation, lack of inhibitors, and stasis. The process of stone formation depends on urinary volume; concentrations of calcium, phosphate, oxalate, sodium, and uric acid ions; concentrations of natural calculus inhibitors (e.g., citrate, magnesium, Tamm-Horsfall mucoproteins, bikunin); and urinary pH.[4] High ion levels, low urinary volume, low pH, and low citrate levels favor calculus formation. When waste materials in urine do not dissolve completely, microscopic particles begin to form and over time grow into kidney stones. Supersaturation of urine **-->** crystallizes, and then aggregates. May have dec amount of urinary stone inhibitors (primarily citrate). Nanobacteria (small

intracellular bacteria that from a Ca-phosphate shell) were present in the central nidus of 97% of kidney stones in ones study, but this was never replicated by others, thus likely was a contaminent (Kidney Int 1999;56:1893). A polymorphism of the Vit-D receptor gene (Fok-1) appears to be associated with some aspects of calcium urolithiasis (OR = 2.15) (BJU Int 2007;99:1534-1538)(higher first-void morning urinary calcium levels)...They were also significantly younger than the median age of 45 years at the onset of the first episode (OR = 3.23).

Risks: Hot weather and high humidity. Occupations associated with a sedentary lifestyle or with a hot, dry workplace. FHx of stones (seen in ~50%) (3-fold risk, J Am Soc Nephrol 1997;8:1568), prolonged immobilization, recurrent urinary infections, **drugs** (Indinavir, acetazolamide, triamterene, ephedrine, felbamate, topiramate and zonisamide, guaifenesin, calcium + Vit-D, or sulfadiazine). Bowel disease (chronic diarrheal states) promotes low urine volume, acidic urine (depletes available citrate) and hyperoxaluria. Diet high in protein, refined carbohydrates, and sodium. Excess dietary meat (including poultry) creates acidic urinary milieu, depletes available citrate; promotes hyperuricosuria. Excess dietary oxalate promotes hyperoxaluria. Excess dietary sodium promotes hypercalciuria. Low urine volume allows stone constituents to supersaturate. White > African American. Obesity may promote hypercalciuria; other results similar to excess dietary meat. Primary hyperparathyroidism creates persistent hypercalciuria. Prolonged immobilization leads to bone turnover, whick creates hypercalciuria. Renal tubular acidosis (type 1) give an alkaline urine promotes calcium phosphate supersaturation; loss of citrate. Idiopathic hypercalciuria (autosomal dominant). Cystinuria, Lesch-Nyan syndrome, hyperoxaluria types I and II (all autosomal recessive). Ehler-Danlos syndrome, Marfan syndrome and Wilson disease. **Systemic illnesses** that may increase the risk for kidney stone formation or otherwise affect the clinical course include primary hyperparathyroidism, renal tubular acidosis, cystinuria, gout, diabetes mellitus, inflammatory bowel disease, renal insufficiency, sarcoidosis, and medullary sponge kidney. Insulin resistance leads to ammonia mishandling; alters pH of urine. Gout promotes hyperuricosuria. Hypercalcemia (due to hyperparathyroidism, sarcoidosis, malignancy, Pagets disease or multiple myeloma). Chronically sleeping **on** the side at risk of calculi formation can be seen in pt's with unilateral stone dz (J Urol 2001;165:1085), consider attaching a tennis ball to sleep ware to break this habit. HTN in 32.8% (Semin Nephrol 1995;5:519–525). Men who work in the steel industry and are exposed to high temperatures have an increased risk of developing urinary lithiasis (Urology 2005:65:858-861) (related to dehydration and hypocitraturia). The risk of developing kidney stones rises with the number of traits of metabolic syndrome a patient has (obesity, hyperglycemia, hypertriglyceridemia, low HDL-cholesterol levels, and hypertension) (Am J Kidney Dis 2008;51:741-747).....the prevalence rose from 3% with no traits to 9.8% with five traits. As temperatures across the U.S. increase because of global warming, the prevalence of kidney stones is expected to grow (PNAS 2008;July)....Assuming a mean temperature increase of 2.38 C, climate change is expected to create 1.61 to 2.25 million new cases of nephrolithiasis by 2050, an increase of 7% to 10.4% from 2000.....The "stone belt" would spread from the Southeast northward into the Midwest, covering much of the southern half of the country and all of California by 2050. Roux-en-Y gastric bypass surgery is associated with an increased postoperative risk for kidney stones according to the results of a study of insurance claims data (OR = 1.71)(J Urol. 2009;181:2573-2577).

Urine pH: Calcium oxalate stones are not pH-dependent. Most people excrete an acid urine which favors uric acid precipitation which may promote the formation of calcium stones. Calcium phosphate stones form in a relatively alkaline urine.

ICD-9 Codes:

592.0 Calculus of kidney - Nephrolithiasis NOS
592.1 Calculus of ureter
592.9 Urinary calculus, unspecified
594.1 Other calculus in bladder - bladder stone
594.9 Calculus of lower urinary tract, unspecified
788.0 Renal colic

S/s: Urinary stone sx's are varied and may include: Acute renal or ureteral colic—the classic symptoms include colicky flank pain with radiation to the groin, ipsilateral testicle, or labia. Mild abdominal or flank discomfort. Nausea and vomiting. Hematuria (microscopic or gross). UTI. High-grade fevers or sepsis may ensue. Irritative LUTS—distal ureteric stones may present with frequency, urgency, and dysuria.

Renal colic: a misnomer, aching flank pain that is usually constant, sometimes waxes and wanes. No position of comfort (**unlike** Appy, diverticulosis, salpingitis/ peritoneal inflammation (makes pt want to lay still). Pain starts as a colicky flank pain radiating to the groin / testicle or labia and can be accompanied by N/V and hematuria. Character of the pain changes as is passes down the ureter and may diminish even if stuck as transition from a hyperperistaltic to an aperistaltic ureter. At renal calyces is a deep, dull flank/back ache. At the renal pelvis it is a sharp pain radiating to the ipsilateral abd quadrant. At the upper-mid ureter it is sharp, bandlike radiating to the mid-low abd. At the distal ureter it is boring that radiates to the ipsilateral groin. Urgency and frequency can occur if stone is close to bladder. In the bladder it may produce a suprapubic pain with stranguria, urgency, frequency and/or dysuria. Pain resolves with passage of stone, may persist temporarily from ureteral edema.

Ddx: appendicitis, PID, pyelonephritis, diverticulitis, dissecting AAA, cholecystitis, ovarian pathology and ectopic pregnancy. Renal cell carcinoma. Acute intestinal obstruction.

Lab: U/A, BUN **or** Cr. ±CBC, lytes, Ca, P. Gross or microscopic hematuria in 90-95%. Non-contrast helical CT is the imaging study of choice to evaluate patients with suspected stone disease, in the acute setting (emergency department). If known history of nephrolithiasis is present, a KUB, U/S, or both may provide initial low-cost confirmation of a recurrent stone that requires further radiographic evaluation. **Link:** W/u Recurrent |

X-ray / KUB: A plain abd x-ray will identify radiopaque stones such as calcium, struvite, and cystine stones, but will miss radiolucent uric acid stones, may miss small stones or stones overlying bony structures, and will not detect obstruction. Best study if PMHx of stones. 90% contain calcium and are **radiopaque** (yet only 30% seen in ER, up to 70% with IVP), if >2-3 mm should see on KUB. (r/o calcified mesenteric lymph node, phlebolith, fecalith, renal calcification, barium), misses 10% as uric acid, struvite calculi and cysteine are **radiolucent** or poorly visualized on plain film radiography. Has no role when helical CT is available. Helpful in documenting the number, size, and location of stones. Will identify calcium stones >1–2 mm in diameter. Will miss most uric acid and some cystine calculi. Will identify nephrocalcinosis—seen in hyperparathyroidism, primary hyperoxaluria, renal tubular acidosis, or sarcoidosis.

Spiral (helical) CT: without contrast (unenhanced) now considered **best** overall study to establish dx (96% sensitive), takes < 5 minutes, no risk of contrast reaction, detects all stone types, rules out other pathologic processes (r/o **AAA** if age >55yo). Takes 0.5mm cuts from top of bladder to top of kidneys (nl abd CT take 1cm cuts). To distinguish ureteral stones from phleboliths

overlying the course of the ureter, look for the soft tissue "rim" = circumferential edema from a ureteral stone (Radiology 1997;202:709). Noncontrast helical CT consistently has outperformed IVP in studies of pt's with suspected ureteral stones. Because helical CT has other advantages in this setting (no use of contrast material, visualization of other intra-abd causes of sx's), it is becoming the imaging procedure of choice for these pt's (Ann Emerg Med 2002;40:280-6). An unenhanced low-dose abd CT (LDCT) protocol should replace the initial abd plain film in pt's with a clinical suspicion of renal colic (Urology. 2006;67:64-68) (sensitivity of 95-100% with a 2.1-mSv radiation dose to women and 1.6 mSv to men) (the mean effective dose [all testing done] was 3.5 mSv in group 1 and 6.9 mSv in group 2). The accuracy and speed (26 versus 69 minutes) of diagnosis of helical CT vs IVP appear to outweigh its cost ($600 versus $400) (Am Fam Physician 2001;63:1329). If the obstruction is acute, proximal ureter and renal pelvis is dilated to the level of obstruction and perinephric "stranding" is seen. If chronic, renal atrophy may be noted.
IVP: Often the only test required for making management decisions in stone patients. Can be performed by a non-radiologist in an emergency. Provides precise information on the relationship of the calculus to the pelvicalyceal system and the ureter. Computed tomography: Helical CT more sensitive than KUB, IVU, or U/S. Will detect radiolucent calculi. Other conditions that mimic ureteral colic can be easily identified. May be useful in determining some composition. Poor detection of Indinavir stones. Delayed images required if high-grade obstruction (delayed "blush" if obstructed). IVP was previously the diagnostic procedure of choice, but is no longer because of potential contrast reactions, lower sensitivity, and higher radiation exposure and has now been replaced by non-contrasted helical CT as the test of choice.
Ultrasound: has a lower sens/spec, but it is the preferred imaging modality in pregnant women. Useful as a screening tool for hydronephrosis or stones within the kidney or renal pelvis. Provides information on renal parenchyma in an obstructed kidney. May detect radiolucent calculi. Useful to rule out other causes of abdominal pain. Middle and distal ureter are not clearly visible. Useful for follow-up evaluation in recurrent nephrolithiasis. The procedure of choice for patients who should avoid radiation, pregnant women and possibly women of child bearing age where gynecologic process or cholecystitis is a prominent consideration. U/S can detect radiolucent stones missed on KUB; however, it may miss small stones and ureteral stones (Ann Emerg Med 1989;18:556). Distal ureteral stones may be detected by transvaginal ultrasonography. May need an alternative study if ultrasound is negative and the clinical suspicion remains high.
Three places stones get caught: ureteropelvic junction (UPJ), midureteral (at iliac vessels), ureterovesical junction.
Stone Types seen in the USA:
Mixed Ca-oxalate and Ca-phosphate@37**%.**
Calcium oxalate@26**%.**
Calcium phosphate@7**%.**
Uric acid@5**%.**
Struvite@22**%.**
Cystine@2**%.**
(Kelly's Textbook of Medicine. New York, Lippincott Williams & Wilkins, 2006, pp 1243-1248).
Fetuin-A: a glycoprotein that inhibits the precipitation of hydroxyapatite from supersaturated solutions of calcium and phosphate in vitro; and is considered one of the more powerful inhibitors of hydroxyapatite formation. Fetuin-A concentrations are decreased in acute inflammation and end-stage renal disease. Patients with urolithiasis have lower levels of urine fetuin-A than do healthy controls, according to a case-control study (BJU Int 2008;101:1151-1154).

Tx: Links: **Basic Tx & Stone Size Considerations |** Medical Expulsive Therapy | **Consult Indications, F/u & Surgical Options** **|** PV |

No urgent intervention needed unless the upper urinary tract is obstructed and infected, the renal function is compromised, or there is intractable pain or vomiting. Most pt's can be managed conservatively / expectantly with analgesics...NSAIDs like Motrin 600-800mg TID, adequeat hydration, strain urine, local heat with an electric blanket at lateral abd & lower back. Takes an average of 8 days to pass, if not passed by then or fever or increased pain etc, need to monitor serum Cr. For acute pain IV Toradol better than morphine.
4 Scenarios of Acute Management: (Clev Clin J Med 2009;76;592-98)
• **CT shows obstruction or hydronephrosis and the pt has signs of infections (WBC >15, chills, sweats):** Admit. Start broad spectrum Abx. Drain kidney via percutaneous nephrolithotomy with ureteral stent.
• **CT shows obstruction or hydronephrosis but the pt has no signs of infections:** Send home. Rx oral pain med. Rx an alpha-blocker. F/u with urologist.
• **CT shows no obstruction but pt has a solitary kidney or persistent pain or vomiting:** Admit. IV fluid at maintenance rate. Control pain. Drain kidney if it is solitary.
• **CT shows no obstruction andt pt has 2 kidneys and no persistent pain or vomiting:** Send home. Rx oral pain med. Rx an alpha-blocker. F/u 1 week if not passed stone for check of serum Cr.
NSAIDs: Pt's treated with NSAIDs for renal colic report better pain relief and are less likely to need further analgesia than those treated with opioids (and less N/V) (BMJ 2004;328:1401-4), based on a pooled analysis of 10 trials. NSAIDs lower requirement for rescue analgesia and thus lower incidence of adverse effects such nausea and vomiting. Of note, in ED's, NSAIDs discourages opiate-seeking patients who may spuriously present with symptoms of renal colic. Caution is advised in patients with preexisting renal disease, consider Sulindac. A combination of IV morphine 5mg + Ketorolac 15 mg (both repeated in 20min) was superior to either drug alone for tx of acute renal colic (Ann Emerg Med 2006;48:173-81).
Other: Hydration (no benefit to stone passage), strain urine, NSAIDs, Abx if evidence of infection. Local heat with an electric blanket at lateral abd & lower back (42 C) decreases the pain, anxiety and nausea of renal colic (J Urol 2003;170:741-44).
Stone size: The likelihood of spontaneous stone passage is directly related to the size of the stone and the time needed for passage.
If stone ≤5 mm: 98% of proximal and distal stones pass spontaneously (J Urol 1997;158:1915–1921) (within 4 weeks, may take up to 40 days).
If 5-10mm: ~53% pass spontaneously. Observation and periodic intervention for pt's with newly diagnosed stones <10 mm in diameter and controlled sx's, with drugs offered when necessary to facilitate stone passage, is an appropriate tx option (AUA 2007 Annual Meeting: AUA/EAU Guidelines Update: Ureteral Stone Management. Presented May 21, 2007).
≥10mm: ~20% pass and thus pt will likely need an intervention.

Time: Takes an average of 8 days to pass if ≤2mm, 12 days if 2-4mm and 22 days if ≥4mm (Time to stone passage. *J Urol* 1999;162:688-91). If no stone movement has occurred after a 4-6 weeks, intervention is warranted because the incidence of complications (renal deterioration, sepsis, ureteral stricture) is increased.

Medical Expulsive Therapy (MET): Indicated in most pt's with ureteral stones measuring <1 cm who are candidates for observation, especially those with stones in the distal ureter. Medical tx costs just a fraction of the average cost of ureteroscopy ($2,645) or shock wave lithotripsy ($4,225). The estimated cost for medical tx ranges from $10 to $74 for a 28-day course of Cardura (doxazosin) to $104 to $141 for a 42-day course of Flomax. The likelihood of spontaneous urinary stone passage can be increased significantly by tx with calcium channel blockers or alpha-blockers, according to meta-analysis of 9 RCT's with nearly 700 pt's with urinary calculi (Lancet 2006; 368:1171-79) (chance of expulsion is 65% greater) (Risk Ratio: CCB or alpha blockers @ 1.65. Alpha blockers @ 1.54. CCB @ 1.51. CCB + steroids @ 1.90). There are large numbers of alpha-1 adrenoceptors in the distal ureter, these blockers inhibit basal ureteral tone and peristaltic frequency and decrease the intensity of ureteral contractions. A meta-analysis of 22 trials, tamsulosin (alpha-blockers) and nifedipine (calcium-channel blockers) increased the rate of spontaneous expulsion of moderate-sized (5mm) distal ureteral stones (Ann Emerg Med 2007;50:552)....recipients were more likely than controls to pass stones without need for additional intervention (risk ratio, 1.59).....four pt's had to be treated to prevent one case of failure to pass a stone....editorialist notes that the potential benefits of these generally well-tolerated drugs probably outweigh the potential harms for most pt's.

Tamsulosin (Flomax): 0.4 mg qd x 5-7 days. Instead of immediately performing cystoscopy or lithotripsy, new research suggests that pt's with distal ureteral stones should be given a trial of MET (J Urol 2005;174:167-172)....210 pt's were randomized to receive home tx with tamsulosin, phloroglucinol, or nifedipine **+ oral corticosteroid + Abx prophylaxis**. Injectable diclofenac was given on an as-needed basis. All were encouraged to drink 2 L of water daily. The expulsion rate for tamsulosin was 97.1%, whereas phloroglucinol and nifedipine had rates of 64.3% and 77.1%, respectively. Pt's treated with tamsulosin achieved stone passage in a shorter period of time and were less likely to be hospitalized and has less renal colic than pt's treated with the other agents. In another study, stone expulsion was noted in 80%, 85%, and 43%, in the nifedipine, tamsulosin, and control groups, respectively. With lower ureteral stones < 1cm in diameter, tx with nifedipine or tamsulosin increases the rate of ureteral stone expulsion and decreases the need for analgesic therapy (J Urol 2004;172:568-571). Stone expulsion in 80% on nifedipine (ave 9.3 days) and 85% with tamsulosin (ave 7.9 days) Vs 43% of controls (ave 12 days) after 4 weeks. The authors conclude that for lower ureteral stones nearest to the bladder the most effective tx is tamsulosin with cortisone, Nifedipine plus cortisone is useful for stones which are lower-ureteral but further away from the bladder (Nifedipine versus tamsulosin for the management of lower ureteral stones. J Urol 2004;172:568-71). Adjunctive tx with tamsulosin after ureteroscopic laser lithotripsy for large renal and ureteric calculi improves the stone-free rate as well as pt's' quality of life (AUA annual meeting. Abstract 1707. May 26, 2006)(4.3% experienced ureteric colic episodes Vs 23.4% of controls and 94.6% were stone-free Vs 83.1%). Studies directly comparing nifedipine and tamsulosin have reported similar rates of stone passage, however, tamsulosin showed a somewhat faster stone passage and fewer hospitalizations and procedures (Lancet 2006;368:1171). Other alpha blockers appear to be similarly effective (The comparison and efficacy of 3 different alpha1-adrenergic blockers for distal ureteral stones. J Urol 2005;173:2010). 4 weeks of alpha blockers to facilitate spontaneous stone passage in patients with stones 10 mm in diameter. Re-image pt if spontaneous passage has not occurred. Tamsulosin 0.4 mg daily as first-line medical therapy is not associated with improved rates of ureteral stone expulsion or time to passage of the stone in patients with ureteral stones with a mean diameter of 3.1 mm (European Association of Urology 23rd Annual Congress: Abstract 313 . Presented March 27, 2008). Tamsulosin 0.4 mg taken for 10 days after unsuccessful treatment with tamsulosin and deflazacort for 10 days is associated with improved rates of ureteral stone expulsion of 80% (vs 49% for placebo), but not with shorter time to expulsion or less pain from renal colic, for stones with a mean diameter of 5.9 mm (European Association of Urology 23rd Annual Congress: Abstract 314. Presented March 27, 2008). A RCT treated 69 consecutive patients who each had a distal ureteral calculus measuring 8 mm (mean size, 4 mm) with either the selective 1a-blocker alfuzosin (Uroxatral) or placebo until passage of the stone (J Urol 2008;179:2244).....The rate of spontaneous stone passage was similar in the alfuzosin and placebo groups (74% and 77%), but stones passed significantly faster with alfuzosin than with placebo (mean time to stone passage, 5.2 vs. 8.5 days).....Alfuzosin modestly (but statistically significantly) lowered pain scores compared with placebo.

Calcium channel blockers (Nifedipine SR 30mg qd = Procardia XL or Adalat CC): x 7-28 days, and oral steroids can facilitate stone passage, both may inc the success rate & also dec the pain after ESWL (Urology 2002;59:835-38). Nifedipine 40mg qd + oral methylprednisolone 16 mg qd x 45 days lead to stone passage in 87% of pt's Vs 65% who received methylprednisolone alone. Stone passage occurred at 11.2 ±7.5 days in the pt's treated with combo tx Vs 16.4 ±11.0 days in pt's treated with methylprednisolone alone (J Urol 1994;152:1095-98). A RCT with an oral corticosteroid and nifedipine compared with corticosteroid alone found an improved stone expulsion rate (79% Vs 35%), faster expulsion time (7 Vs 20 days) as well as a decreased requirement for analgesia (Urology 2000; 56:579–583).

Consult Urology: Immediate intervention is indicated in a pt with a high grade (anuria) obstructed solitary or transplanted kidney, bilateral obstruction or infected upper urinary tract with signs of sepsis. Urgent intervention needed if impending renal deterioration or intractable pain or vomiting. Patients with stones >10 mm in diameter, and patients with significant discomfort or who have not passed the stone after 4 to 6 weeks, should be referred to urology for potential intervention. Consider early referral if an upcoming away trip from home, persistent hydronephrosis, staghorn calculus or certain occupations (pilot or bus driver). Women who develop nephrolithiasis during pregnancy have nearly double the risk of preterm delivery as pregnant women who do not (Obstet Gynecol 2007;109:1099-1104).

F/u: If fail to pass stone in 5-7 days, fever, unrelenting pain, vomiting. Check radiograph of kidney and upper bladder (KUB), UA and serum Cr.

Urine strainer: can use a coffee filter or place a small square cut from a pair of nylon panty hose (rinse & re-use) over the urethra while voiding to catch the stone for analysis. Send passed or removed stone for analysis. (Acute renal colic from ureteral calculus. NEJM 2004;350:684-93)

Tx Options for the Urologist:

For treating urolithiasis, shock wave lithotripsy, ureteroscopy, and percutaneous nephrolithotomy have replaced open surgery (BMJ. 2007;334:468-472).....Shock wave lithotripsy is effective in approximately 80% to 85% of simple renal calculi. For complex renal calculi, percutaneous nephrolithotomy is the tx of choice. Staghorn calculi should be treated, preferably with percutaneous nephrolithotomy in most pt's. For pt's who are pregnant, morbidly obese, or have coagulopathy, ureteroscopy is the preferred tx. Consider immediate referral for patients with larger calculi, (>1.5 cm) stone of harder composition (eg, cystine or calcium oxalate

monohydrate) or for stones in complex renal or ureteral locations (lower pole calyx or mid-ureter), SWL is only successful in approximately 50% of cases and thus endoscopic stone fragmentation with a percutaneous or ureteroscopic approach is preferred.
1. Emergent Decompression: With either a nephrostomy tube (percutaneous nephrolitholapaxy = PCNL) or ureteral stent. Septic pt's with obstruction should have nephrostomy tube placed (can be done with local anesthesia), drainage can be monitored as opposed to internal stent that might not be draining properly.
2. Extracorporeal Shock Wave Lithotripsy (ESWL): Usually under mild IV sedation although some machines require general or regional anesthesia Shock waves break up the stone into small fragments that pass out on own. Best choice for renal, proximal and mid ureteral stones <1-2 cm. 85% overall success rate. Occasionally a fragment can obstruct the ureter and require intervention. Repeat tx required 10-20%. Lower-pole stones have been consistently associated with decreased stone-free rates following SWL when compared with upper and middle-pole stones. Cystine, brushite and calcium oxalate monohydrate stones have demonstrated a high degree of resistance to fragmentation by SWL owing to their high density, whereas less dense stones (calcium oxalate dihydrate, hydroxyapatite and uric acid) are more susceptible to SWL (Prediction of shockwave failure in pt's with urinary tract stones. Curr Opin Urol 2006;16:88-92). Can gait the shocks to the pt's ECG (heart rate) such that arrhythmia potential is lowered… R-wave triggered mode, where ESWL can trigger the atrial output pulse and the subsequent inhibition of the following ventricular pulse if pt has a dual pacemakers to the VVI or VOO mode prior to ESWL to prevent this occurrence. The rate of clearance of renal stones after ESWL is lower in elderly pt's than in younger pt's (BJU Int 2007;100:392-395)(stone-free rate after ESWL: in the >60 year age group at 37.6% Vs in the 40-and-under at 54% and 41-60 at 43%)....suggest that "more endoscopic procedures should be offered to the elderly population." A slower rate of shock wave delivery can reduce renal injury during shock wave lithotripsy (BJU Int 2007;100:624-628)...Kidneys treated with 120 shock waves/minute showed focal to extensive subcapsular hematomas and parenchymal lesions that extended, in some cases, across the full thickness of the kidney....In contrast, kidneys treated with 30 shock waves/minute showed no surface bleeding, and parenchymal hemorrhage was limited to papillae in the region of the focal treatment area...and a majority have found a significant effect of rate of better stone breakage. Extracorporeal shock wave lithotripsy (ESWL) alters renal perfusion, particularly in elderly patients (BJU Int 2008;101:731-735).....causes considerable renal parenchymal damage, resulting in disturbances in renal perfusion on MRA. Contrary to an earlier report, shock wave lithotripsy does not increase the risk of diabetes according to 6-yr f/u data on 1947 patients treated with ESWL (Urology 2009;73:4-8).
Pneumatic lithotripter: Can generate contact pressures of up to 2.9 MPa at the tip of the probe. The device is powered by a carbon dioxide cartridge that can deliver at least 80 shocks via a spring-loaded hammer that drives a projectile into contact with the probe. The "StoneBreaker" is a rapid and effective means of fragmenting urinary stones (BJU Internat 2007;100:629-631)...Hand-held portable, appears to offer much faster intracorporeal lithotripsy compared with other ballistic lithotripters.
3. Ureteroscopy / ureterorenoscopy (URS) Stone Removal: Stone fragments are removed using suction, graspers, or basket extraction. Endoscopic (laser) lithotripsy can be successful in up to 95% for distal stones. Tx of choice for distal stones or proximal stones > 1-2cm. Indicated for ESWL failures. Ureteroscopy traditionally requires the placement of a stent in order to minimize the risk of flank pain secondary to ureteric edema and facilitate the passage of residual fragments. The presence of a ureteric stent, however, has been shown to reduce quality of life in up to 80% of pt's and thus many authors have shown that it is not necessary on a routine basis (The management of ureteric colic. Curr Opin Urol 2006;16:71-76)….risk factors for complication included renal pelvic stone location, bilateral procedure, lithotripsy, history of urolithiasis, diabetes mellitus, recurrent/recent infection, operative time of 45 min or more plus lithotripsy and operative time of 45 min or more plus ureteral dilatation. Treatment of ureteric lithiasis with retrograde ureteroscopy and holmium:YAG laser lithotripsy appears generally more successful than ESWL (BJU Int 2009;104:1144-1147).....At 3 weeks an overall success rate of 96.4% vs 48% of the patients (NNT 2). Staged flexible ureteroscopy/holmium laser lithotripsy (URS) is a less costly alternative to percutaneous nephrostolithotomy (PNL) for renal stones 2 to 3 cm in diameter (J Urol 2009;182:1012-1017)....."Although PNL has a higher stone-free rate, it is more invasive, has a higher rate of second stage procedures, and typically requires at least one night of hospitalization, while URS has very good success rates and is much less expensive.".....The estimated cost of PNL was $19,845, significantly higher than the estimated cost of $6,675 for URS, according to their report.
4. Percutaneous Lithotripsy: Best for large/staghorn renal calculi, can be followed by ESWL for residual fragments. Conventional PCNL is recommended in this situation only when there is a significant stone burden or when external shock-wave lithotripsy fails. Minimally invasive percutaneous nephrolithotomy (mPCNL) is an effective and safe strategy for dealing with upper urinary tract calculi in transplanted kidneys (BJU International 2007;99:1467-1471)....should be the initial approach for most cases of upper urinary tract stones in transplanted kidneys, except for simple and small stones in the middle or lower calyx
Complications of Urolithiasis: renal failure, ureteral stricture, infection/ sepsis, urine extravasation, perinephric abscess, xanthogranulomatous pyelonephritis. Stone bulk, dilated pelvicalyceal systems, and previous UTI have strong associations with UTI after kidney stone surgery, while infected stones, pelvic urine, and cloudy urine are predictors of urosepsis after percutaneous nephrolithotomy (EAU 22nd Annual Congress: Abstract 876. Presented March 23, 2007). Patients who develop kidney stones may be at increased risk (60% more likely) of developing chronic kidney disease a cohort study on 4,066 residents of Olmsted County who had a first kidney stone from 1986 through 2003 (mean age 44, 59% males) (ASN Meeting 2008; Abstract F-FC202).....Some potential explanations for the relationship proposed by other researchers are that patients with kidney stones are predisposed to urinary tract infections or that lithotripsy could lead to kidney damage during the elimination of stones.
Retained Stones: likely no problems if in the renal pelvis. Consider annual KUB to monitor for size changes. Non-obstructing ureteral stones need annual monitoring of renal US.

Prevention of Ca stones:

Normalization of body weight (BMI) and cardiovascular risk factors, sufficient physical activity, balanced nutrition and sufficient circadian fluid intake would be the appropriate measures to avoid new calculus formation. Up to 85% of all stone pt's could anticipate lower risk of stone recurrence with basic changes in lifestyle and dietary habits, ~15% of pt's require additional specific pharmacological prevention (Curr Opin Urology. 2005;15:119–126).
Fluids: Increase fluid intake with the goal to produce at least 2 liters of urine per day to increase the urine flow rate and lower the urine solute concentration, both of which protect against stone formation to lower the 5 yr recurrence rate from 27% to 12% (J Urol 1996;155:839). A low urinary volume increases urinary supersaturation, thus fluid therapy is safe, cheap and effective: Increased water (8-10 cups a day) will dec recurrence by 55%. Try to maintain 2L urine output a day or try to maintain a clear-colored urine rather than yellow urine. Lemon juice/ lemonade increases citrate excretion, particularly in the hypocitraturic stone former. Lemon

juice (4 ounces of concentrate per day in water or as lemonaid) resulted in increased urinary citrate levels in >92% of pt's (average increase of 142 to 346 mg/day (J Urol 1996; 156:907). Can add lemon juice or diluted potassium citrate to just plain water. The type of fluid consumed may be important; grapefruit juice may enhance the risk of stone formation (Ann Intern Med 1998;128:534), whereas pt's who ingest one bottle of beer a day may reduce the risk of stone formation by 40% (Am J Epidemiol 1999;150:187). Consumption of Gatorade neither increases nor decreases urinary stone risk factors according to research supported by the Gatorade Sports Science Institute (J Urology 2009;182:992-997)....."water consumption (>2 L /day), which still remains first-line treatment for the prevention of nephrolithiasis."

Diet: Low salt, low protein. Increase fruit and vegetable intake, which are rich in potassium, result of increasing citrate excretion (Kidney Int 2004;66:2402). A 5 years study on 120 men found a significantly lower risk of stone recurrence among the group assigned to the normal-calcium (1200 mg/day = 30 mmol/day), low animal-protein (52 g/day), low-salt diet (2900 mg/day = 50 mmol/day NaCl) (unadjusted relative risk of 0.49)(NEJM 2002;346:77).

The (DASH) diet: (See ICU-FEN Module: DASH Diet) **--> high:** fruit/ veges/ fiber/ Ca/ Mg/ K, +low fat dairy = 2100 kcal/d, 27% fat, 6% saturated fat, 31g/d of fiber, 4,700mg/d of potassium, 500mg/d Mg, 1,240mg/d Ca). DASH consists of an eating plan low in total fat, saturated fat, and cholesterol and rich in fruits, vegetables, and low-fat dairy products. The underlying nutrient targets are reduced fat, saturated-fat, and cholesterol intake and increased protein, fiber, calcium, magnesium, and potassium intake The DASH diet may help protect against kidney stones according to the results of a prospective study (J Am Soc Nephrol. 2009;Published online August 13)(decreases the risk of calcium oxalate stone recurrence by 51%)....."Although we think it reasonable for calcium oxalate stone formers with high levels of urinary oxalate to avoid intake of some individual foods very high in oxalate (such as spinach and almonds), our data do not support the common practice of dietary oxalate restriction in calcium stone formers, particularly if such advice results in lower intake of fruits, vegetables, and whole grains.

High Dietary Phytates: High phytate diet (myoinositol hexaphosphate) best. Phytate is found in cold cereal, dark bread, wheat fiber, bran, rice, nuts and beans. Dietary phytate (the most abundant form of phosphate in plants) binds tightly to double charged ions such as calcium (and zinc, iron, magnesium and manganese) in the GI tract to reduce absorption and enhance urinary excretion, which may reduce stone formation (37%) (Arch Intern Med 2004;164:885-891). Calcium oxalate crystal formation is strongly inhibited in vitro by phytate.

Calcium: A normal-calcium, low animal protein and low-salt diet reduce the risk of recurrence (NEJM 2002;346:77-84). A low calcium intake, which acts by increasing the absorption and subsequent excretion of oxalate due to less complexing with calcium in the intestinal lumen (J Am Soc Nephrol 2004;15:1567).....The effect on oxalate more than counterbalances the decrease in calcium absorption and excretion. Avoid soft drinks (promotes natriuresis and hypercalcuria). Dietary calcium and supplemental calcium are not associated with increased risk of kidney stones and may even be protective in younger (27-44 yo) women (Arch Intern Med 2004;164:885-891). Recommendation for calcium intake cannot be generalized since the effect of calcium intake on stone formation depends on the type of stone, oxalate intake, presence of stones and the efficiency of calcium absorption from the bowel. Calcium restriction recommended in pt's who have moderately to severely elevated intestinal calcium absorption and urinary calcium levels. Pt's with calcium phosphate stones may need to carefully monitor their calcium dietary intake. Except for absorptive hypercalciuria, calcium restriction in nephrolithiasis pt's is not recommended (Curr Opin Urology. 2005;15:119–126).

Limit Protein: A high animal protein diet, which can lead to hypercalciuria, hyperuricosuria, hypocitraturia, and elevated urinary acid excretion. Increased intake of oxalate-containing foods is controversial and likely varies considerably from person to person. Reducing animal protein intake will reduce urinary calcium, uric acid, and citrate excretion as the metabolism of sulfur-containing amino acids increases the daily acid load by generating sulfuric acid. Vegetable protein has a lower sulfur content.

Limit Salt intake: Calcium is reabsorbed passively in the proximal tubule down, thus, a low sodium diet (80-100 meq/day) can enhance proximal sodium and calcium reabsorption, leading to a reduction in calcium excretion (NEJM 2002;346:77).

Thiazied Diuretic: Thiazide therapy can lower calcium excretion by as much as 150 mg/day (3.75 mmol/day). Start at 25 mg/day of chlorthalidone or hydrochlorothiazide, some will require 50 to 100 mg/day to achieve adequate lowering of the urine calcium.

Avoid excess: Protein (restrict to 1g/kg/d or ~<52g/d), salt (restrict to 2-3g/d by avoiding processed foods), oxalates (if have Ca-oxalate stones, particularly if have hyperoxaluria on testing – **not** the same as oxalate stones), prevent recurrent infections. A high salt diet, which increases urinary calcium excretion. Obesity and weight gain increase the risk of kidney stones (JAMA. 2005;293:455-462) as a larger body size may result in increased urinary excretion of calcium, oxalate, and uric acid. Higher sucrose intake is associated with an increased risk of stone formation in younger and older women (Arch Intern Med 2004; 164:885). High fructose intake is associated with an increase risk (37% with women, 27% with men) of kidney stones compares to those with lower intake (Kidney Int 2008;73:207-12)(avoid excessive soda's, fruit punch, apple juice, canned fruits etc). Cranberry juice does not seem to increase urinary citrate levels. Orange juice is not reccomended at it does not lower calcium excretion, it modestly raises oxalate excretion; and the increase in caloric intake (J Urol 1993; 149:1405). High dose vitamin C appears to increases urine oxalate excretion in certain individuals, thus high dose supplements should be avoided (J Urol 2003;170:397).

Oxalobacter formigenes: a Gram-negative, anaerobic microbe common in normal adults. Presence of O formigenes in the intestinal tract is associated with a 70% decreased likelihood of developing calcium oxalate kidney stones according to a case-control study with 247 patients with a recurrent calcium oxalate stones (J Am Soc Nephrol 2008;March).....The bacteria were more prevalent in healthy controls than in patients with a history of calcium oxalate stones (38% versus 17%).....The bacterium consumes oxalate, and its absence could result in "higher oxalate excretion in the urine and thus predisposition to [calcium oxalate] calculus formation"....Among the healthy controls, prevalence of O. formigenes was lowest (29%) in those who took antibiotics to which the bacterium is sensitive -- for example, macrolides, tetracyclines, chloramphenicol, rifampin, or metronidazole -- at any time in the past. Prevalence was highest (59%) in controls who had never used these antibiotics or taken any others in the past five years -- the non-users. This bacterium might one day be used as a probiotic to prevent stone formation. O. formigenes metabolizes oxalate in the gastrointestinal tract, which could reduce its absorption.

W/u: All pt's should have stone analysis. Detailed metabolic evaluation should be postponed until after the acute stone event has resolved.

Limited evaluation if first stone: Because the rate of recurrence is variable, most recommend only a limited evaluation after a first stone. Ck routine blood chemistries, including multiple measurements of serum calcium and phosph. A low serum phosphorus warrants further eval as well. Complete evaluation after the first stone may be warranted in individuals willing to make dietary changes or to take medical therapy (if warranted by the work-up), because of the potentially high rate of recurrence and potential morbidity from recurrent stones (BMJ 2004;328:1420).

Further eval indicated if: Recurrent, multiple stones, younger, FHx or pt requests eval. Pt's with stones composed of cystine, uric acid, calcium phosphate or struvite.
Patients at moderate to high risk for recurrent disease: Middle-aged, white males with a family history of stones. Patients with chronic diarrheal states and/or malabsorption, pathologic skeletal fractures, osteoporosis, urinary tract infection, and gout.
Tests to order: 24-hour urine collections: urine volume, pH, and excretion of calcium, uric acid, citrate, oxalate, sodium, creatinine, urinary supersaturation. At lease 2 or 3 separate collections may required given the high variability of this test or to obtain all of this information. If there is a substantial discrepancy between two results, a third collection may be required. Pt should maintain their usual diet and physical activities. Some even suggest waiting several months after the initial stone episode since the pt may have temporarily modified their diet. A 2-day food diary may be helpful.
Check serum for: Ca, P, uric acid, creatinine, alkaline phosphatase, intact PTH. A recent study found no significant association between stone recurrence and initial phosphate levels in serum or urine, thus may not be necessary to check (Scan J Urol 2002;36:368-72). In pt's with first-time kidney stones, conservative therapy (dietary modification only) is the most cost-effective strategy, but for pt's with recurrent kidney stones, empiric therapy (dietary modification and potassium citrate) and a modified simple metabolic evaluation (a single 24-hour urine collection for renal stone risk factors, with potassium citrate and HCTZ for pt's with hypercalciuria and potassium citrate alone for pt's with normocalciuria) are equally cost-effective (J Urol 2004;172(6 pt 1):2275-81).
UroRisk & StoneRisk diagnostic profiles: measures 10 urine risk factors for stone formation (Calcium oxalate - Brushite - Sodium urate - Uric acid). It offers a urine collection and preservation system and is recommended for initial and recurrent stone formers who test positive for a UTI. Calculates the "relative supersaturation. Give a choice of three customized profiles. (800) 330-6565, ext. 7308

Normal values:

Calcium: For a pt with a history of renal stone formation, urinary calcium results will be more meaningful if the pt's usual diet is followed for 3 days before specimen collection. Do not stop medications. If the urine calcium test is done because of a metabolic disorder, the pt should eat a low-calcium diet and calcium medications should be restricted for 1 to 3 days before specimen collection. Obtain multiple measurements of serum calcium (at least 2 in patients with high-normal values) to r/o primary hyperparathyroidism, which is a surgically correctable disease While stone analysis is not a good clinical guide to diagnosis of hyperparathyroidism, hypercalcemia is prevalent in this curable disease and should be monitored closely (BJU Int 2009;103:670-678)....."The hypercalcemia of hyperparathyroidism was modest, but hypercalciuria was far more marked than in (stone formers without systemic disease), because the fractional calcium excretion of hyperparathyroidism exceeded that of (stone formers without systemic disease)."....."Surgical cure of hyperparathyroidism did not completely eradicate either hypercalciuria or hypophosphatemia, suggesting that these patients have some additional mineral disorder."....."When faced with severe hypercalciuria (above 300 mg/day) it is prudent to get at least a few fasting serum calciums."
Normal diet: Men <300 (7.5 mmol/day). Women <250 (6.25 mmol/day). Child <4mg/kg.
Low-calcium diet: 50–150 mg/24 h or 1.25–3.75 mmol/d. The bulk of calcium excreted is eliminated in the stool. Increased calcium in urine results from an increase in intestinal calcium absorption, a lack of renal tubule reabsorption of calcium, resorption or loss of calcium from bone, or a combination of these mechanisms.
Ddx increased urine Ca: Hyperparathyroidism (30% to 50% of cases). Sarcoidosis. Primary cancers of breast and bladder. Osteolytic bone metastases. Multiple myeloma. Paget's disease. Renal tubular acidosis. Fanconi's syndrome. Vit-D intoxication. Idiopathic hypercalcuria. Diabetes mellitus. Crohn's disease and some cases of ulcerative colitis. Thyrotoxicosis. Increased urinary calcium almost always accompanies increased blood calcium levels. Ca excretion levels > calcium intake levels are always excessive; urine excretion values >450 mg/24 hours are reliably abnormal. Increased Ca excretion occurs whenever Ca is mobilized from the bone, as in metastatic cancer or prolonged skeletal immobilization. When Ca is excreted in increasing amounts, the situation creates the potential for nephrolithiasis or nephrocalcinosis, especially with high protein intake.
Falsely elevated Ca levels: drugs (calcitonin, vitamins A, K, and C, corticosteroids), meals high in Ca (milk), increased exposure to sunlight, immobilization (especially in children).
Ddx decreased urine Ca: Hypoparathyroidism. Familial hypocalciuria hypercalcemia. Vit-D def. Preeclampsia. Acute nephrosis, nephritis, renal failure. Renal osteodystrophy. Vit D–resistant rickets. Metastatic carcinoma of prostate. Malabsorption syndrome—celiac-sprue disease, steatorrhea. Urine calcium decreases in late normal pregnancy.
Falsely decreased Ca levels: increased ingestion of phosphate, bicarbonate, antacids. Alkaline urine. Thiazide diuretics. Oral contraceptives, estrogens. Lithium.
Uric acid: Men <800 (4.8 mmol/day). Women <750 (4.5 mmol/day). High uric acid is thought to act as a nidus for calcium stone formation in calcium oxalate-stone formers.
Oxalate: Men <45 (0.5 mmol/day). Women <45 (0.5 mmol/day).
Citrate: normal is 320-1300mg for men and women. Alkalinization raises citrate excretion by diminishing the uptake of filtered citrate by the proximal tubular cells (Kidney Int 1990;38:728)(raising the pH in the tubular lumen converts the divalent form into the less reabsorbable trivalent form).
Phosphorus (360-1600mg).
Na (52-380mEq).
Mg (28-180mg/d).
Urinary Creatinine excretion: permits assessment of the completeness of the 24-hour collection. Low level suggest an incomplete collection (need repeat) except for older subjects or malnourished pt's with decreased muscle mass.
Normal in <50 yo: 20-25 mg/kg (177-221 µmol/kg) lean body weight in men and 15-20 mg/kg (133-177 µmol/kg) in women.
Normal in >50yo: 10-12 mg/kg/d for men. 8-10mg/kg/d in women.
If normal, calculate the ratio of "measured-to-estimated creatinine clearance". If <0.75 then repeat as inaccurate.
If 0.75-0.9 the inaccurate, but still may be useful. If 0.9-1.1 then accurate urine collection.
If 1.1-1.25 then inaccurate, but still may be useful. If >1.25 then inaccurate, needs repeat.
Sodium excretion: increased sodium intake can contribute to hypercalciuria.
24hr Urine (commercial kits are available) initially while pt on a random diet. Evaluate for **volume** (>2L), pH (Ca-phos stones form if pH >7, may be due to distal RTA if low urinary citrate, vegetarian diet if high citrate. If pH >7.5 suspect infection. If pH <5.5 suspect uric acid, cysteine, chronic diarrhea or strenuous physical exercise). Can repeat 24hr Ur after modified diets (Ca restriction to see if hypercalcuria is dependent on dietary intake), acid load test or on meds.

Bicarb: A low plasma bicarbonate concentration is suggestive of type 1 renal tubular acidosis or chronic diarrhea.

Underlying Causes of Stones:

Links: Hypercalciuria | **Hyperuricuria (Uric Acid Stones)** | Hyperoxaluria | **Hypocitruria** | Struvite | **Cystinuria** | Calcium-Phosphate |

Urine pH: Uric acid and cystine stones associated with acid urine. Renal tubular acidosis (type I), and infection related stones associated with alkaline urine. Tx of uric acid and cystine stones includes alkalinization of urine, limit proteins

Hypercalciuria:

(50%) (>300mg/d in M, >250 in F), causes 45% of Ca stones. Idiopathic hypercalciuria (**IH**) is the most common cause of calcium-containing kidney stones. IH is defined as excessive urinary calcium excretion (hypercalciuria) in the setting of normocalcemia and in the absence of secondary causes of hypercalciuria. Determining the specific etiology of hypercalciuria with special diets often not that helpful since it makes little difference in tx. The disorder is familial, was initially thought to exhibit an autosomal dominant pattern of inheritance, but may be polygenetic.

May be due to: IH consists of a spectrum of these disorders, with considerable overlap among these potential mechanisms.

1. Absorptive hypercalciuria: Excessive intestinal calcium absorption. Inc intestinal absorption causes inc serum Ca (high normal), which suppresses PTH, dec P.

2. Renal Hypercalciuria: Decreased renal tubular calcium reabsorption. Defective resorption of Ca in kidney, have dec serum Ca and inc PTH, no change with fasting state. Due to Ca leaking from distal tubules.

3. Primary Hyperparathyroidism: Enhanced bone demineralization. Inc PTH **-->** inc bone resorption of Ca and inc 1,25(OH)2D to stimulate intestinal absorption. More common in women. R/o if high-normal serum Ca. The intact hormone assay is the best choice for proving subtle HPT. This need to be treated with Alendronate, Calcitonin of Mithramycin.

4. Unclassified Hypercalciuria: normal PTH and serum Ca with inc urinary Ca and no change with fasting.

Other --> RTA, Addison's, sarcoid, hyperthyroid, Vit-D intoxication, milk-alkali syndrome. Only 5% have associated dz (RTA or hyperparathyroidism).

Tx of Hypercalciuria: Limit dietary protein, oxalate and Na (<2g/d). High calcium diet (low intake increases the risk of stones). A low-sodium, low-protein diet in place of a traditional low-calcium diet is best for hypercalciuric men who form calcium oxalate stone (NEJM 2002;346-84).

Thiazide diuretic: Start HCTZ @25-50mg qd, increase up to 50mg BID as dictated by 24hr urine.

Potassium Citrate: Adding K-citrate 20-30mEq BID will inc excretion of citrate (a stone inhibitor) as well as prevent dec K. A retrospective cohort study of 503 patients who experienced recurrent kidney stones and who received potassium citrate therapy due to a combination of hypocitraturia, low urine volume, hypercalciuria, and gout found that at an average tx duration of 41 months (range, 6–168 months), the stone formation rate dropped from a baseline of 1.89 stones annually to 0.46 stones annually (J Urol 2009;181:1145)(mean urinary citrate levels rose from 470 to 700 mg, and mean urinary pH rose from 5.9 to 6.5).

Other: Can also add a K-sparing diuretic. 1/3 of females with Ca stone have medullary sponge kidney. Inc risk of osteoporosis in hypercalciuria: thiazides are beneficial. Ca-citrate is the preferred Ca supplement in postmenopausal females with stones. Can also use Bendroflumethiazide 2.5 mg BID, Trichlormethiazide 2-4mg qd, Na-cellulose phosphate 2.5-5g TID or Orthophosphate 500mg BID. A high calcium diet, if not accompanied by higher intake of fluids, potassium, magnesium and phosphate, may increase the risk for calcium oxalate stones, results of a metabolic investigation suggest, the risk is increased further if calcium is increased by using supplements rather than by diet based on urinary Ca excretion (J Urol 2003;169:470-474). (*J Urol* 1997;158) (*Am J Clin Nutr* 1994;60).

ICD-9 Code: 275.4 Hypercalcinuria.

Hyperuricuria:

(15% of stones)(>800mg/d M, >750mg F) Uric acid stones are more difficult to diagnose than other types of kidney calculi because they are radiolucent and therefore don't show up on standard abdominal x-rays. Seen in those with underlying hyperuricemia or gout and have abnormally low urine pH due to low ammonia excretion. Frequency of stone formation increases with increasing serum uric acid levels and urinary uric acid excretion rates. Up to 20% of patients with gout will form uric acid stones. Risks include decreased urine pH, diminished urinary volume, excessive urinary uric acid, acute diarrheal states and inflammatory bowel disease, diabetes mellitus, metabolic syndrome, and the use of probenecid and aspirin. Can be seen in chronic diarrheal states that cause loss of water and bicarb., hypermetabolic states, myeloproliferative d/o's, medications such as thiazides. Uric acid can serve as a nidus for Ca-oxalate stones (12% of pt's). Uric acid stones may be suspected on the basis of a history of uric acid stones or of gout (present in ~20% of pt's with uric acid stones). The typical pt has normal amounts of uric acid in an acidic urine; this condition increases the likelihood of uric acid crystallization. Pure uric acid calculi are **radiolucent** on plain imaging but visible on U/S or CT. Other radiolucent stones that should be considered in appropriate clinical settings include matrix stones (which are made of organic material and are occasionally seen in pt's with urease-producing bacteria) and indinavir stones. Pure uric acid stones primarily occur in pt's in whom a persistently acid urine (pH <5.5) promotes uric acid precipitation. That the incidence of nephrolithiasis is increasing, both in the US and worldwide, particularly for those composed of uric acid (Am J Kidney Dis 2006;48:897-904)....HTN, obesity and diabetes are significantly associated with a dx of urate nephrolithiasis (OR = 1.22).

Labs: Urine pH <6 (Nitrazine paper). Uric acid crystals in urine. Serum uric acid often normal, especially with low urine pH <6.2. Often have hematuria. Stone analysis shows uric acid or mixed uric acid with calcium oxalate or calcium phosphate. 24-hour urine collection shows urinary uric acid often >4800 umol/d in men and >4400 umol/d in women or uric acid:creatinine ratio >530 umol/mmol (hyperuricosuria). Hyperoxaluria is often seen in pt's with kidney stones who have undergone bariatric surgery using currently accepted methods (J Urol 2007;177:565-569). Pt's with elements of metabolic syndrome are more likely to develop highly acidic urine, increasing their risk of forming uric-acid kidney stones (Clin j Am Soc Nephrol 2007;2:883-888). Pure uric acid stones primarily occur in patients in whom a persistently acid urine (pH <5.5) promotes uric acid precipitation. ~10-20% of pt's with gout overproduce uric acid, resulting in increased uric acid excretion. Uric acid stones may be seen with other causes of chronic overproduction of uric acid or in chronic diarrheal states in which bicarbonate loss and volume depletion lead to a concentrated, acid urine.

Tx: Uric acid stones are unique in that they can be managed medically. Increase fluid intake, aim for urine output 1.5-2 L/d. Normalize renal uric acid excretion. Abx tx of urinary tract infection. Avoid Purine rich foods, restrict animal protein to 5-7oz of meat/

fish per day) ± Allopurinol (Zyloprim) @100-300 mg/d to dec uric acid production(especially if hyperuricemia). Normalize urine pH…..Alkalinizing the urine (inc Ur pH, maintain at 6.0-7.0) with bicarb or potassium citrate 10-30 mEq (~20 mmol) TID to inc solubility of uric acid and dissolves pure uric acid stones (goal is pH of 6.5-7, pt can measure with Nitrazine paper, higher than 7 may cause CaP to precipitate). At a urinary pH below 5.5, uric acid is poorly soluble; solubility increases at a pH above 6.5. Can dissolve them at a rate of 1cm/mo, imaging can be repeated at one month to determine whether dissolution has occurred. Unless a stone is pure uric acid, however, oral dissolution therapy is not possible. If oral dissolution therapy fails, tx should proceed as for a radiopaque stone.

Purine Content of Foods:

Very-very high: herring & roe, mussels, sardines, yeast.
Very high: anchovies, crab, lobster, trout, salmon, turkey, veal, beef, bacon, pheasant, venison.
High: bass, halibut, oysters, tuna, chicken, pork, ham, duck, asparagus, cauliflower, kidney/ lima beans, spinach, whole grains, mushrooms, peas.
Low: non whole grains (breads, cereals), cheese, eggs, nuts, fruits, peanut butter, tea/ coffee, chocolate, butter, soft drinks.
ICD-9 Code: 274.11 Uric acid nephrolithiasis. 274.10 Gouty nephropathy, unspecified.

Urate Nephropathy:

Renal parenchymal damage / dysfunction associated with disordered uric acid metabolism. There is a 5-10% prevelence of hyperuricemia, a 1% prevelence of gout and a 0.1% prevelence of uric acid nephrolithiasis 0.1% in the US.
Types:
1. Hyperuricemic acute renal failure: Precipitated by distal tubular obstruction resulting from acute massive elevation of serum uric acid such as that seen with cell lysis during induction chemotherapy or radiation. Risks include volume depletion, pre-existing acute or chronic renal insufficiency, large tumor burden, LDH >1500 IU, extensive bone marrow involvement, elevated tumor sensitivity to chemotherapeutic agents. Labs show serum uric acid >15-20 mg/dL (0.88-1.18 mmol/L), rising BUN and creatinine, urinary uric acid-to-creatinine ratio >1; ratio of 0.6-0.75 suggests another cause of renal failure and uric acid crystals in urine. Tx with IV hydration. Hemodialysis in severe cases. See Hem-Onc Module for **Hyperuricemia & Urate Nephropathy** |
2. Uric acid nephrolithiasis: See above.
3. Hyperuricemia of chronic renal failure: due to retention of uric acid resulting from decreased tubular secretion or altered postsecretory reabsorption, or both. Secondary gout occurs in <1% of all cases of CKD. Labs show serum uric acid usually 7-10 mg/dL and is rarely >10 mg/dL due to compensatory increase in GI secretion of uric acid. Serum uric acid remains normal until GFR <20 mL/min.
4. Chronic urate nephropathy: Renal insufficiency attributed to parenchymal damage secondary to medullary urate deposition. Hyperuricemia has been linked to intrarenal vascular disease (Nephrology 2004;9:394-399).

Hyperoxaluria:

(15%) (>40mg/d of oxalate). Hyperoxaluria which may be present in up to 40% of male and 15% of female stone formers and is typically only slightly elevated; in comparison, marked hyperoxaluria is usually associated with inflammatory bowel disease and/or malabsorption or primary hyperoxaluria. Hyperoxaluria is present in roughly 20% of patients with calcium oxalate kidney stones (Urology 2009;73:484-489).
75% of all stones are composed of some CaOxalate, Mixed Ca-Oxalate-P in 34% but most of these pt's do not have hyperoxaluria (see **PV**). 15% of those with hyperoxaluria is related to dietary consumption of oxalate or inc Vit-C. Also seen in Crohn's, chronic pancreatitis, celiac sprue, Ca restriction, primary hyperoxaluria or other ileal dz because of inc absorption as Ca is bound to free fatty acids.
Recurrence rate for untreated Ca-Oxalate stones **-->** 10% at 1 year, 35% in 5yr, 50% recur in 10yr. If inc urine output to >2L/d will cut in ½ the recurrence rate. If serum Ca >10.3 **-->** check PTH.
Tx: Ca or **Mg-oxide** 200-400mg BID supplements + **Pyridoxine** 25-100 mg qd (as many deficient) with meals to bind oxalates in the gut.
K-Mg-Citrate: for recurrent Ca-Oxalate stones, less GI SE's than K-citrate (10-30 mEq qd). Dose of 42mEq K + 21mEq Mg + 63mEq citrate qd (J Uro 1997:158).
Calcium: By binding dietary oxalate, calcium supplements or calcium-containing foods may be useful in the management of hyperoxaluria (Urology 2009;73:484-489).....of 22 patients with hyperoxaluric stone formation, 10 were instructed to increase intake of calcium-containing foods and 12 were given the same advice and told to take calcium citrate with meals.....in each group, oxalate excretion fell by roughly 13 mg/day from about 58 mg/day.....Neither group showed increases in urinary calcium excretion, the researchers report.
Diet: In general, avoid excess oxalates (spinach/ leafy dark greens, nuts, rhubarb, instant coffee, tea, chocolate, berries, purple grapes, Tofu, wheat germ). Dietary oxalate intake is only a modest risk factor for kidney stones according to a large prospective study based upon food frequency questionnaires of three large cohorts: the Health Professionals follow-up study and the Nurse's Health studies I and II found that compared individuals in the highest quintile for dietary oxalate intake were compared to the lowest quintile, there was a mild increase in the risk of stones for men (RR of 1.22) and for older women (RR of 1.21)(Oxalate intake and the risk for nephrolithiasis. J Am Soc Nephrol 2007;18:2198)(8 or more servings of spinach per month accounted for >40% of oxalate intake).....risk was higher in men with a decreased dietary intake of calcium.
Other less efficacious: Cholestyramine 4g TID, Mg-citrate 10 mEq BID. Probiotics with Oxalobacter formigenes may reduce the ocalate exretion (J Urol 2001;166:1487-91).

Oxalate Content of Foods:

High content: beets, black tea, chocolate, cocoa, dried figs, ground pepper, lamb, lime peel, nuts, parsley, poppy seeds, rhubarb, sorrel, spinach, swiss chard.
Moderately high: beans, all types of berries, carrots, cashews, celery, coffee, concord grapes, okra, green onions, oranges, green peppers, sweet potatoes, tomatoes. Strawberries, cranberries, raspberries, plums, and apples. This is somewhat confusing, however, because several of these have been shown to decrease incidence of kidney stones even though they contain significant amounts of oxalate! In addition, some studies have shown that eating oxalate- and calcium-containing foods together may actually decrease stone risk (The influence of diet on kidney stone disease. J Urol 1996;155:432–440). A large cohort study of more than

85,000 women showed that those who consumed black tea had an 8% decreased risk of developing kidney stones (Arch Intern Med 1998;128:534–540).

Hypocitruria:

(20%)(<450-600mg/d M, <650-800 in F) citrate is a stone inhibitor. Hypocitraturia can be marked in pt's with chronic metabolic acidosis; however, mild hypocitraturia occurs in a significant proportion of stone formers in the absence of academia.
Tx: K-citrate (Urocit-K) [5, 10 mEq tab] start @ 5mEq BID with food, titrate to 10-20 mEq TID-QID (0.5-1mEq/kg/d). **Contra:** hyperkalemia or renal insufficiency is present, monitor serum K and Cr. Avoid Na-citrate unless K-citrate not tolerated. The dosage should be adjusted to maintain a urine pH 6.5-7, it usually needs to be continued indefinitely. A retrospective cohort study of 503 patients who experienced recurrent kidney stones and who received potassium citrate therapy due to a combination of hypocitraturia, low urine volume, hypercalciuria, and gout found that at an average tx duration of 41 months (range, 6–168 months), the stone formation rate dropped from a baseline of 1.89 stones annually to 0.46 stones annually (J Urol 2009;181:1145)(mean urinary citrate levels rose from 470 to 700 mg, and mean urinary pH rose from 5.9 to 6.5).
Causes: Often idiopathic, may be RTA, met acidosis, high protein/ salt diet, dec K or Mg, UTI, carbonic anhydrase inhibitors, renal insufficiency, dehydration, thiazides, diarrhea. People subject to recurrent calcium renal stones tend to excrete low amounts of citrate in their urine (Am J Kidney Dis 2006;48:546-554).....Their analysis showed that hypocitraturia is associated with urinary potassium level, and may reflect low levels of potassium in the diet, the chief source of which is fruit. Topiramate therapy can induce profound declines in urinary citrate levels, which may lead to urinary stones (Urology 2008;72:29-33).....Mean urinary citrate levels declined by 65% after a 50-mg dose and by 52.9% after a 75-mg dose of topiramate, the authors report.....In the two patients taking 100 mg of topiramate, urinary citrate levels declined by 74% and 62% from baseline....."The average final citrate level was 215.5 mg/day, well below the 320 mg/day generally accepted as defining hypocitraturia the investigators point out".

Struvite:

(8%): (Mg-P-NH_3). Also known as infection or triple-phosphate stones, consist of magnesium, ammonium, and calcium phosphate. Infection of the urinary tract with organisms that secrete the ectoenzyme urease can greatly increase the urinary ammonia concentration and pH. From urea splitting organisms (*Proteus, Pseudomonas, Providencia, U. urealyticum*). Struvite stones only form in pt's with a chronic UTI due to a urease producing organism such as Proteus or Klebsiella. Affected pt's often have multiple magnesium ammonium phosphate crystals in the urine sediment. The stone may grow rapidly over a period of weeks to mo's and, if not adequately treated, can develop into a staghorn or branched calculus involving the entire renal collecting system. They occur more often in women than in men and are the leading cause of staghorn calculi. Struvite stones only form in patients with a chronic urinary tract infection due to a urease producing organism. Affected patients often have multiple magnesium ammonium phosphate crystals in the urine sediment. The stone may grow rapidly over a period of weeks to months and, if not adequately treated, can develop into a staghorn or branched calculus involving the entire renal collecting system.
Tx: Struvite stones typically require stone removal with percutaneous nephrolithotomy of the entire stone. If the pt is febrile or presents with signs of systemic infection, surgical manipulation should be delayed until Abx tx has been administered and the pt has been afebrile for at least 48 hours. After surgical intervention, medical therapy should focus on preventing recurrent UTIs. Retained residual fragments increase the risk of recurrent UTI and future calculi. Acetohydroxamic acid (Lithostat, 250mg TID) is an irreversible inhibitor of urease and can prevent the crystallization of struvite stones, however, because of side effects (including deep venous thrombosis), it generally is reserved for use in pt's who cannot tolerate surgical intervention. Consider Amoxicillin suppressive therapy 250mg PO qd.

Cystinuria:

(3%): consists of two cystine molecules linked with a disulfide bond. Cystinuria is an autosomal recessive d/o in which excessive urinary excretion of the dicarboxylic amino acids (cystine, ornithine, lysine, and arginine) results from impaired transport. Cystine stones develop in pt's with cystinuria due to the insolubility of cystine in the urine. Only homozygote pt's form cystine calculi and often present with stones during childhood. Cystine stones only develop in patients with cystinuria (an autosomal recessive disorder) due to the poor solubility of cystine in the urine. The diagnosis of cystinuria is made by identification of the pathognomonic hexagonal cystine crystals on urinalysis, and by measurement of urinary cystine excretion of >250 mg/liter.
Dx: The normal rate of cystine excretion is 30 mg/day (1.3 mmol/day). In contrast, pt's with cystinuria excrete >400 mg/day and sometimes up to 3,600 mg/day. The dx of cystinuria is made by identification of the pathognomonic hexagonal cystine crystals on urinalysis (which can be seen in the initial urinalysis in about 25% of pt'), and by measurement of urinary cystine excretion of >250 mg/liter.
Tx: Stones can form rapidly increase fluids to 4-5L/d (round the clock). Alkalinize the uring to pH>7-7.5 with K-citrate 10-30mEq TID, Pyridoxine 50mg/d. If these measures are not effective, administration of cystine binders such as penicillamine (Cuprimine) and tiopronin (Thiola) can help prevent cystine calculi. Bind sulfhydryl groups to other molecules (chelation) by adding: D-Penicillamine 250mg QID, Captopril 50mg BID or Mercapto-propionylglycine 250mg TID to interfere with disulfide bond formation. Dietary manipulation with a low-methionine diet is difficult and rarely successful.

Calcium Phosphate (CaP):

(7%): often have an acidification disorder such as Distal RTA, giving an alkaline urine. Can also be due to primary hyperparathyroidism, excessive alkalinization, and sarcoidosis. F>M. Same risks as Ca-Oxalate stones (other than hyperoxaluria and hyperuricosuria)… One exception is that calcium phosphate stones are more typical of Type I (distal) renal tubular acidosis in which the urine pH is persistently >5.3, even after an acid load.
Tx: K-citrate to increase the inhibitor level to normal to correct the hypercalciuria. Care must be taken to avoid excessive alkalinization, because high urinary pH can increase the urinary supersaturation of calcium phosphate salts. If hypercalciuria persists, addition of a thiazide diuretic is indicated.
HypoMg-uria: <50mg/d and no diarrhea d/o. **Triamterene:** found in diuretics and Indinavir.

Bladder Stone:

Vesical calculi affect men predominantly and account for 5% of urinary calculi in the Western world(Schwartz, 2000). Most occur in men older than 50 years and are often associated with bladder outlet obstruction. The majority of bladder calculi are struvite, but calcium oxalate and uric acid stones are also encountered. Uric acid stones are identified in about 50% of pts with bladder stones

(Urology 1991;37:240). Calcium phosphate stones are also noted. Usually a single stone, but may be multiple, especially when bladder diverticulum is present. Pediatric bladder calculi in endemic areas are ammonium acid urate and/or calcium oxalate
Risk factors: bladder outlet obstruction; neurogenic bladder; chronic bacteriuria (urea-splitting organisms); foreign bodies; bladder diverticula; and, rarely, upper tract stones. In adults, commonly associated with bacteriuria, intravesical obstruction, and neuropathic bladder, and may occasionally develop secondary to calculi migrating from the upper urinary tract (Urology 2002;59:688). Children at risk for ammonium urate bladder stones in underdeveloped nations where a low-protein, high-carbohydrate diet and chronic dehydration predispose to endemic stones. Pediatric stones related to low phosphate, high oxalate, and high acid diet (J Urol 1988;140:461; BJU 1976;48:617; Am J Clin Nutr 1974;27:877).
S/s: Bladder calculi are often found incidentally during evaluation of pt's with obstructive or irritative voiding sx's. Terminal hematuria, interrupted urine stream related to obstruction from the stone. Recurrent UTIs are common and are an identified risk factor. Bladder stones may be voided spontaneously, with larger calculi precipitating acute urinary retention. Dysuria, gross hematuria, and suprapubic pain may occur. Interruption of the urinary stream from impaction of the stone at the bladder neck or urethra is not uncommon. Bladder calculi are often radiolucent, may be single or multiple, and can be diagnosed as filling defects on the cystogram phase of an IV urogram or as an incidental finding on CT scan. On U/S, a hyperechoic, mobile bladder mass with shadowing is suspicious.
W/u: UA, c + s. If stone is calcium-containing, may be identified on plain film of the abdomen/pelvis; plain film is not a reliable method for detecting bladder stones in spinal cord injury pts (J Spinal Cord Med 2004;27[5]:438). Depending on the size, the stone may be identified on US as a hyperechoic, mobile lesion with shadowing. Cystoscopy confirms presence and size of stone(s).
Tx: Cystoscopy is essential to evaluate the bladder and the bladder outlet, both to determine the etiology of the stone and to plan the most appropriate tx approach. Percutaneous suprapubic cystolithotripsy to remove bladder calculi can be safely accomplished under local anesthesia (Urology 2006;68:38-41) (2% lignocaine (lidocaine) injected 3 to 4 cm above the symphysis pubis. Lignocaine gel was also used for urethral lubrication). Pts with chronic indwelling catheters who are at increased risk for stone formation may use an irrigation of 0.25-0.5% acetic acid solution 2-3 times/d.

Prostatic Calculi:

Deposition of calcareous material on corpora amylacea; develop in the tissues or acini of the gland. May occur secondary to infections and may be assoc with ochronosis (Urology 1991;37:240). Rare in boys. Frequently identified in males over age 50. Often noted incidentally on routine x-rays. Composed of calcium phosphate trihydrate and carbonate (BJU 1974;46:533)
S/s: Usually asymptomatic. Sx may be secondary to BPH, urethral stricture, or prostatitis. Calcifiation on plain film of abdomen and pelvis, may have terminal hematuria. If infected, may lead to recurrent infection
Diffdx: Bladder calculi
W/u: UA, c + s. Demonstrated on plain films of pelvis or on TRUS of the prostate. May not be identified on cystoscopy.
Tx: If asymptomatic, no rx necessary. If pt has sx, rx incl TURP or suprapubic prostatectomy. In the setting of multiple symptomatic prostatic calculi and intractable infections, total prostatectomy and bilateral seminal vesiculectomy are usually curative (Campbell's Urology 1992;6:2085).
****Ref:**(*Campbell's Urology*, 7th ed., 1998, WB Saunders) (*Urologic Clin NA* 1997;24:1, pp50-122) (*NEJM* 1992;327:1141-52) (Prevention of recurrent nephrolithiasis. *Am Fam Physician*. 1999;60:2269-76) (Nephrolithiasis. *Semin Nephrol*. 1999;19:381-8) (Urolithiasis. *Uro Clin North Am* 2000;27:2) (Acute Renal Colic from Ureteral Calculus. NEJM 2004;350:684-93) (An evidence-based literature review of the management of urolithiasis. BMJ. 2007;334:468-472)

Urologic Infections:

Links: UTI's | **Pyelonephritis** | Urethritis | **Balanitis** | Epididymitis | **Orchitis** | Prostatitis | **Fournier's** | Sexually Transmitted Dz's (STD's) | **Dysuria** |

Urinary Tract Infections (UTI):

Links: **S/s & Decision Rule** | Labs & Dx | **Bugs** | Risks & Complications | **Tx** | PV in Women | **Recurrent** | Prophylaxis | **Referral Indications & Abscess** | Asymptomatic | **Men** | Pregnant | **Ddx** | Hospital Acquired & Fungal / Candidal | **Elderly** | Pediatric | **Hemorrhagic, Eosinophilic, Emphysematous, Glandularis Cystitis, Malacoplakia & References** | STD's | **Pyelonephritis** | Urethritis | **Dysuria** | Catheterized Pt's | **Lower Urinary Tract Symptoms (LUTS)** | Prostatitis | **Balanitis** |
…….END DEMO of this chapter.

Surgical Pearls:

Links: Safety | **Etiquette, Knots & Intruments** | Post-Op Confusion | **Hypotension & Fever** | Oliguria & Short-of-Breath | **Hyponatremia, Navel Jewelry & Other Pearls** | Drains | **NGT** | Feeding Tubes | **PEG** | PEGJ | **DPEJ** | | Abd Pain, Disorders & Exam | **Appendicitis** | Post-op ileus & Bowel Recovery | **Surgical Hemostasis Issues** | Peri-Operative Eval | **PV of Thromboembolism** | Antibiotic Prophylaxis | **Surgical Notes & Orders** | Abdominal Incision & Closure | **General Anesthesia** | Wounds & Suturing & Coding | **TRAUMA** | Amputations | **Survival Situations (Leg Amputation, Appy)** | Procedures | **Gastro** |

Laparoscopy = endoscopy = minimally invasive surgery. After initial exploratory laparotomy, repeat laparotomy is often performed. Although "on-demand" relaparotomy is no better than planned relaparotomy at reducing morbidity or mortality in pt's with severe peritonitis, it does cut healthcare utilization and medical costs, according to findings reported by the Dutch Peritonitis Study Group (JAMA 2007;298:865-873,923-924)(reduce the relaparotomy rate to 42% vs. 94%)...The morbidity rate in the planned surgery group was 44% compared with 40% in the on-demand surgery group. The corresponding mortality rates were 36% and 29%.
Oxygen: Use of 80% oxygen during surgery is no more effective than 30% oxygen (administered during surgery and for 2 hours afterward) in preventing surgical wound infection according to a RCT on 1386 patients who underwent acute or elective laparotomy (JAMA 2009;302:1543-1550,1588-1589)...Moreover, no difference was seen in rates of pulmonary complications.....Surgical site infection within 14 days after surgery occurred in 19.1% in the high-oxygen group and 20.1% in the control group, a nonsignificant difference.

Bowel Prep: Cleansing a bowel in preparation for colorectal surgery increases, rather than decreases, the chances of infection and leakage (Am Soc Colon and Rectal Surg. Annual Meeting: Poster 10. Presented June 5, 2006) (might be due to compromised microcirculation of blood in the bowel wall).Mechanical bowel preparation prior to colonic surgery for large lesions (> 3 cm) increases the risk of surgical site infections (OR = 1.40), and therefore should be abandoned according to a meta-analysis (Ann Surg 2009;249:203-209). Mechanical bowel preparation (with polyethylene glycol and bisacodyl or with sodium phosphate solution) before colorectal surgery doesn't prevent anastomotic leaks (Lancet 2007;370:2112)...researchers suggests that bowel prep be abandoned in most, but not all, patients....may still be needed for patients requiring low rectal anastomoses, which generally have a higher leak rate than intraperitoneal procedures.

Post-Op- Iatrogenic Problems to avoid: Fluid overload from forgotten or inappropriate IV's. Hypokalemia. Contraction hypokalemic hypochloremic metabolic alkalosis when not replacing NG tube output. Fecal impaction. Urosepsis from old Foley. Medication side effects when drugs are not adjusted for renal failure, hepatic failure or advanced age. Deafness or ATN after losing track of aminoglycoside or vancomycin levels. Failure to recognize respiratory distress because it had been ignored by an earlier shift. Failure to recognize sepsis because charted vital signs look rosier than the pt. Wound infection in unmonitored surgical wounds. Not considering pulmonary emboli or MI when the pt is having problems. Sacral decubitus. A simple maneuver at the end of surgery attenuated post-laparoscopic shoulder pain and nausea signficantly (Obstet Gynecol 2008;111:1155)....pt's placed in the Trendelenburg position (30 degrees), after which the anesthesiologist performed five manual pulmonary inflations (maximum pressure, 60 cm H2O) and held the fifth inflation for 5 seconds as the surgeon kept the trocar sleeve valve fully open, afterward, the patient was returned to a flat position, the trocar was removed, and the abdominal incisions were closed.

Standardized operating room (OR) Briefing: a short meeting that introduces the surgical team members and verifies critical components of the operation is seen as a means to help prevent wrong-site surgery (J Am Coll Surg 2007;Jan) ("do we have the correct pt?" and "is the correct side or site marked?"). Rates of death and major complications declined with use of a simple 19-item checklist for surgical safety (the death rate decreased from 1.5% to 0.8%, and the rate of inpatient complications decreased from 11.0% to 7.0%)(NEJM 2009;360:491)..... Checklist items were as follows: patient verifying his or her own identity, surgical site, and procedure; marking surgical site; using pulse oximetry; formal pause for team members to introduce themselves by name and role; reviewing anticipated critical events; confirming that prophylactic antibiotics were administered <60 minutes before incision; confirming needle and sponge counts; and, at completion, reviewing key concerns for the patient's recovery and care.

World Health Organization (WHO) Safety checklist for surgical teams to use in operating rooms:

(WHO Surgical Safety Checklist (First Edition) http://www.who.int/patientsafety) In industrial countries, rate of major complications ranges from 3% to 16%, and rates of permanent disability or death range from 0.4% to 0.8%. In the developing world, death rates for major surgery may reach 5% to 10%, with mortality from general anesthesia approaching 1 in 150 in parts of sub-Saharan Africa. The checklist covers 3 phases of a surgical procedure: before anesthesia is induced, before skin incision, and before the patient leaves the operating room. For each phase, a checklist coordinator confirms that the team has completed the designated tasks before the next phase of the operation occurs.

Before induction of anesthesia, key components of the checklist, using the mnemonic "Sign In," are as follows:

• Check that the patient has confirmed their identity, the surgical site, and the procedure to be done and that the patient has given informed consent.
• The surgical site should be marked, if applicable.
• The anesthesia safety check should be completed.
• The pulse oximeter should be placed on the patient and functioning.
• Check to see if the patient has

(1) A known allergy. If so, these should be documented.
(2) An anatomically difficult airway to intubate or aspiration risk. If so, additional equipment and assistance should be available.
(3) Risk of more than 500-mL blood loss in adults or 7 mL/kg in children. If so, provision should be made for adequate intravenous access and fluids.

Before skin incision, the checklist uses the mnemonic "Time Out" for the following components:

• Confirm that all team members have introduced themselves both by name and by their role on the surgical team.
• The surgeon, anesthesia professional, and nurse should verbally confirm the patient's identity, surgical site, and procedure to be performed.
• Anticipated critical events to be reviewed by the surgeon are any critical or unexpected steps, estimated operative duration, and anticipated blood loss.
• Anticipated critical events to be reviewed by the anesthesia team are whether there are any patient-specific concerns.
• Anticipated critical events to be reviewed by the nursing team are confirmation of sterility of the tools, supplies, and field (including indicator results); documentation and discussion of any equipment issues or concerns; whether antibiotic prophylaxis has been given within the last 60 minutes, if applicable; and whether essential imaging is displayed, if applicable.

Before the patient leaves the operating room, the checklist uses the mnemonic "Sign Out" for the following components:

• The nurse verbally confirms with the team the name of the procedure to be recorded and verifies instrument, sponge, and needle counts, if applicable; labeling for the surgical specimen, including patient name; and whether there are any equipment problems to be addressed.
• The surgeon, anesthesia professional, and nurse review the key concerns regarding recovery and management of the specific patient.

The WHO notes that the checklist is not intended to be comprehensive but encourages specific modifications and additions appropriate for each local practice.

Info: Surgery patients who are injured as a result of their operation (Iatrogenic pneumothorax, Selected infections resulting from medical care, Postoperative hemorrhage or hematoma, Postoperative physiologic and metabolic derangements, Postoperative respiratory failure, Postoperative pulmonary embolism or deep vein thrombosis, Postoperative sepsis, Postoperative wound dehiscence after abdominopelvic surgery, Accidental puncture or laceration) are 7 times more likely to die while hospitalized and are also much more likely to be readmitted to hospital within 3 months of being discharged than are surgery patients who are not injured (Med Care. 2009;47:583–590).....2.6% of patients had at least 1 safety event....At 3 months, the readmission rate was about 17% for those with no safety event, but about 25% for those who had a safety event.....The in-hospital death rate was 1.3% with no safety event, but 9.2% with a safety event.

Common OR Etiquette and Instruments:

Links: Knots | **Instruments** |

Where possible rings should be removed as the incidence of glove perforation is higher when they are present, and there is an increased risk of skin reaction below rings due to the concentration of scrub solution. Earrings may be left in place as long as they are contained under the theatre hat. The hair should be covered with a hat or hood. Few items, if any, should be kept in the pockets as they may injure patients during transfers. Appropriate footwear should be clean, well-fitting and provide protection from falling items. Scrubs and shoes should never be worn outside the OR area and if for any reason you leave the area, the scrubs must be changed on your return.

Pre-Scrubbing: ensure all the hair is covered. The face mask is contoured over the nose to prevent steaming up of the eye protection. Masks and eye protection styles vary depending on the type of surgery being performed. If your own spectacles are worn they should be well-fitting, and if necessary, secured with tape. Should additional protection to the eyes from the side be required; this can be achieved by using a visor. Other types of Personal Protective Equipment may be necessary, for example, lead coats and thyroid protection for cases using X-rays, or specialist goggles for laser surgery. Prior to scrubbing, any small cuts or lesions should be covered with an occlusive waterproof plaster. Chipped nail varnish or false nails can harbour resistant strains of bacteria and must be avoided.

The scrub room should have: A mirror to check that all hair is covered, and the eye protection and mask are correctly fitted. A selection of antiseptic lotions. Sterile scrubbing brushes. A clock to check on the time taken to perform each phase of the scrub procedure.

Scrubbing: If a local scrub policy is displayed in the scrub room, it must be strictly adhered to.

Turn the sleeves up to allow access to the elbows.

Set the water temperature allowing the water to flow for a while to let the temperature stabilize.

Set the water flow fast enough to rinse, but not to splash your scrubs, as when they become wet bacteria from the upper body will be drawn through the damp scrub material onto the outer surface.

Wet the arms to the elbows.

Take 5-10 millilitres of your chosen antiseptic lotion using your elbow to operate the dispenser. It is advisable once selected to continue with this lotion for the remainder of the procedure, as mixing of the lotions is not recommended by the manufacturers. Apply the lotion rapidly to the hands, forearms and elbows ensuring all areas are covered. Remember it is the time the lotion is in contact with the skin that is important, not how hard you rub it in. Use the sterile scrubbing brush with antiseptic lotion applied only to clean under the nails. This should be done in a downward direction. This complete section takes approximately 2 minutes. Unless moving directly from one procedure to another, subsequent hand-washing should follow the same pattern using the brush on each occasion as this reduces confusion and increases compliance.

Rinse the arms from the finger tips to the elbows moving the hand upwards at all times to allow the water to drain at the elbow. Manipulating the fingers and rotating the arm assists in rinsing off all the lotion. More than one pass through the water may be required, but at no time should the arm be drawn backwards allowing dirty water to flow back towards the hand. Take another 5-10 mls of lotion and apply rapidly from the hands up to the elbows. Once fully covered concentrate mainly on the wrists and hands ensuring all web spaces, thumbs and palms are given particular attention as demonstrated. After approximately 1 minute the hand and forearms are rinsed as before. For the final application take 5-10 mls of lotion and apply from the hand up to the mid forearm. Once again concentraing on the wrists and hands as demonstrated. This final stage should take 2 minutes approximately. Rinse the whole arm as before. To aid drying allow excess water to run off from the elbows before moving to the gowning trolley.

Hand Drying: If the gown pack is unopened look for the folded corners, which when pulled systematically allow you to open the pack, without contaminating the contents. Lift up the first towel and keep it folded. If an unfolded towel is used by the time you have reached the elbow, which is now classed as “dirty”, the towel will be wet and allow contamination from the elbow straight onto the palm of your “clean hand”. The drying is carried out by dabbing the skin, not rubbing. Rotating the arm being dried helps to dry the whole arm. Drying must be carried out only from hand to elbow - never go in the reverse direction from elbow to hand. Ensure the hand holding the towel does not come into contact with the hand being dried. Discard the towel.

Gowning: Locate the neck of the gown and apply a pincer grip with the thumb and forefinger to each side, being careful not to touch the outside of the gown. With the remaining fingers pick up the folded gown and move away from the trolley. While retaining the pincer grip at the neck of the gown allow it to unfold. Do not shake the gown, as this causes turbulence resulting in debris rising from the floor which can contaminate the hands or the front of the gown. Moving your hands and arms outwards locate the sleeves and move your hands and arms into them. Be aware of your surroundings during this procedure. An assistant will loosely tie your gown at the back being careful not to touch the front of your gown. At no time should you allow your hands to exit the cuffed end of the sleeve. Now fold a portion of the cuffs inwards to allow you to perform the closed gloving method. Finally, to secure the gown an assistant will adjust any loose ties at the back. The waist ties vary in position, but generally you should hold the tie that you can see is attached at the side. Straighten the piece of card prior to handing this with its tie firmly attached to an assistant. Turn around slowly and grasp the tie prior to pulling it from the card. It is important to develop an awareness of sterile OR theater environment. Care needs to be taken when moving in the locality of sterile trolleys and other members of the gowned theater team. When passing either personnel or items always face those that are sterile, with your back towards the unsterile, thus avoiding potential contamination.

Gloving: Request your preferred glove size and type being aware that some specialties, for example use ultra sensitive gloves. In Orthopaedics you will probably be required to double glove (please note you should use gloves half a size larger than your normal size and don them first).

Open the sterile packet on the sterile field and create a fold along the bottom edge to stop the pack folding in on itself.

As demonstrated here using the enclosed left hand, turn the glove onto the upward facing right hand.

Open the cuff of the glove with the left hand and slide the finger tips of the right hand into the cuff.

Take the double layer of glove on top of the right hand and pull this over the back of the right fist and thumb.

Take the ribbed single edge of the glove and pull this down over the wrist.

Pull the sleeve of the gown down from front and back of the gloved hand allowing the fingers to just emerge.

Take hold of the finger tips of the glove and stretch them out to exactly align the fingers and thumb of the hand.

Holding the sleeve and the gown at the wrist pull downwards until the glove is in the correct position.

Now Repeat the Procedure for the Left Hand.

Intra-operative Glove Changing: If you need to change a glove during a procedure an unscrubbed assistant, wearing unsterile gloves, should take hold of your glove and the underlying gown at the wrist, ensuring they do not touch the sleeve directly. As you move your hand backward into the sleeve, the assistant should ease the glove forward and remove it. This will leave you with your hand in the correct position to re-glove using the closed method.

Post Op Protocol: The conclusion of a case, you should observe the following procedures for removal of gown and gloves. Once the ties have been undone, with your right hand hold the left shoulder of the gown, and take the right shoulder of the gown with your left hand. Pull the gown forwards making sure your gloves remain in place. Fold the gown inwards on its self, thus keeping any contaminants contained, roll up and place in the laundry bag provided. With the right hand take hold of the other glove just below the left thumb and pull forwards and remove. Run the fingers of the left hand below the glove of the right hand, and pull the gloves inside each other. If safety glasses have been worn remove them by the legs rather than by the front of the glasses. Remove the mask by pulling the ties at the back of the head. The mask should then be disposed of, holding it by the ties only, in the correct rubbish bag. Wash your Hands. If you need to apply hand cream make sure it is non-irritant and water-based, as some types of cream cover the hands with a protective layer that impedes the action of the antiseptic lotion used in the next scrub.

Various Surgical Knots:

Slip Knot: created by applying the consecutive throw onto the one limb of a ligature or suture and sliding it down a stem created by slight traction on the held limb. Two forehand index finger throws are applied in succession and both slipped down the opposite strand which is held, under slight tension to facilitate the process. The knot has to be completed with a square knot, if knot stability is to be achieved

Square (Reef) Knot: With the hand in a prone posture, the end of the suture is picked up between the thumb and the long finger and pulled towards the surgeon. In doing so, it is laid over the index finger.. The other limb of the knot is then crossed over the index finger and using the back of the nail of the index finger, the ligature is passed through the loop created and passed back into the holding hand. This maneuver requires purposeful wrist rotation. The free end is then pulled towards the surgeon. The fingers are then placed on top of the suture, the hand supinated - placed palm up, the suture crossed over the fingers of the tying hand, and again the free end delivered back into the fingers of the holding hand, bringing the end through the loop so created and this time the free end is pulled away from the body, thus creating a square knot. This knot will be repeated with the free end of the ligature or the suture pointing towards the surgeon and the needle end away from the surgeon. The pick-up is again important, using the index finger and thumb, with the hand fully pronated, the wrist is supinated, and the free end is crossed over the fingers and again delivered into the fingers of the holding hand. On this occasion the ligature is drawn away.

The knot is then completed by placing the free end, holding it in the thumb and long finger, on top of the index finger and repeating the first manoeuvre of the previous knot using the nail of the index finger to loop into the holding hand.

Knot Tying with Instruments: Often, when suturing skin, it may be useful to perform an instrument tie since the needle holder will already be in your hand. In this demonstration, the tail of the suture is lying on the knot board and the rubber tubes simulate the two wound edges to be approximated. Grasp the bight of the suture with the left hand and hold the shaft of the needle holder above the suture and parallel to the wound with the right hand. Form one wind of suture around the needle holder then take hold of the tip of the tail. The first throw is then tightened, with the left hand moving away from the body. The second throw is made with the needle holder again held parallel to the wound and above the suture. This is tightened with the left hand moving towards you, resulting in a secure reef knot.

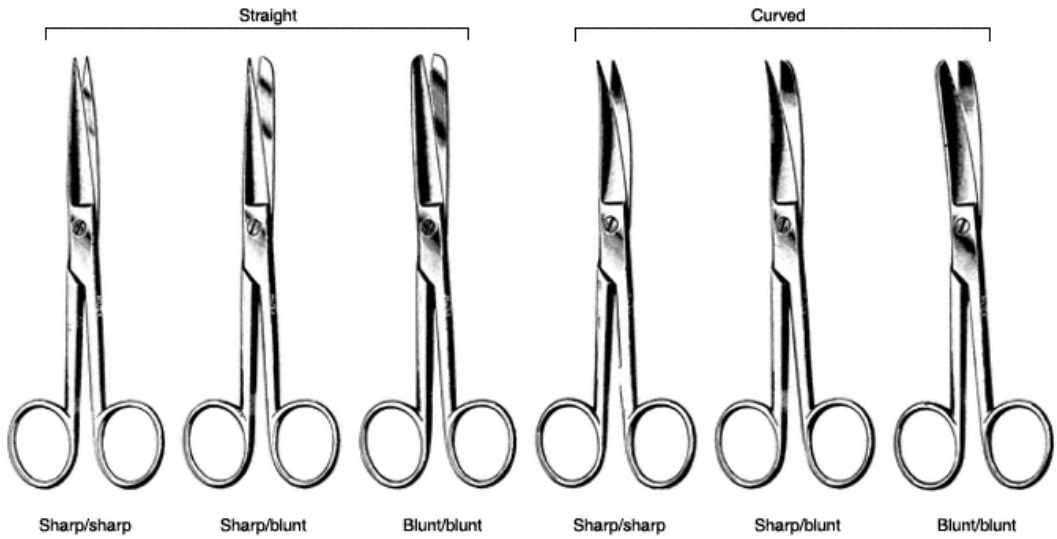
Aberdeen Knot: After forming a continuous monofilament suture, it is necessary to use an Aberdeen knot for termination. Once the last bite has been made through the tissue, the suture is not pulled all the way through, leaving a loop through which the fingers of one hand can be passed. The bight of the suture is then handed to the fingers through the loop, creating a second loop. The first loop is tightened down by a see-saw action between the two hands holding the new loop and the single strand. The bight is again passed through the loop, forming another loop, with the previous one being tightened down as before. This maneuver is repeated at least 6 times. The final step is to bring the suture and needle through the loop and tighten firmly. Remember to leave a slightly longer tail when dividing the suture, due to the memory of the monofilament material.

Two handed Surgeon's knot: Begin by crossing the right thread over the left and form a figure 4 shape by extending the right forefinger. At the crossing point, grasp both ends with the left hand and form a half hitch by twisting the right end under the left. The end of the right thread is held again and the action repeated to create a double half hitch. The first throw is snugged down with the left thread moving away from the body so that the knot is square.

Surgical Instruments:

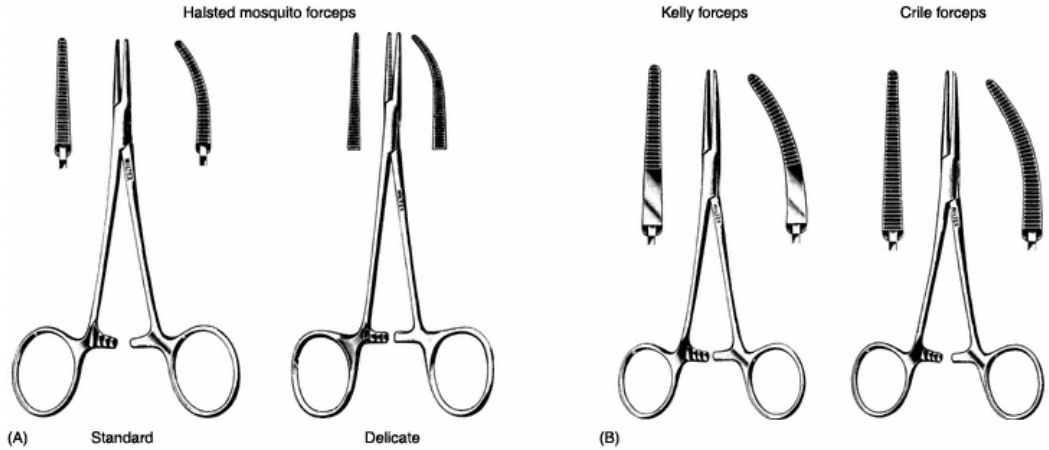
Scissors - Dissecting: It is important to use the sensitive pulp of the fingers to hold the loop of the scissors. Also, you should deploy the tripod technique using the thumb and ring finger with the index finger down the shaft of the scissors in order to stabilise them. Be aware that it is easy for the index finger to slip down the shaft leading to instability of the scissors. It is therefore important to keep the index finger down the shaft as shown. In addition, do not push the thumb all the way through the loop of the scissors as this too can render them unstable. Effective and safe cutting with scissors is achieved by cutting near the tips of the instrument rather than at the hinge end. The blades approximate more closely here and you are much less likely to inadvertently cut an unseen, distal structure by using the tips. In general terms, you should always try to have the scissor tips curving upwards so that you can see the end of the scissors rather than curving downwards when, once again, you may unintentionally cut a structure that you cannot see. When using scissors down a deep cavity it is helpful to use the index finger of your left hand to stabilise the scissors prior to cutting.

Scissors - Straight Suture: These are sharp-tipped scissors that should only be used for surface work and not within a body cavity. Compare these to the blunt-tipped scissors that we usually use for cavity work. Scissors are all designed for right-handed people and intuitively we push away with the thumb which brings the two blades together. However when you try to push away with the thumb of the left hand, you open up the blades making cutting more difficult. It is easier sometimes to cut with the palm technique using the thumb and index finger on one side and the middle and ring finger on the other side of the scissors. This however is slightly unstable because it is not a tripod technique and you might have to use the index finger of your right hand for additional stability. Again with this technique you push upwards with your middle and ring finger to get the blades to come together to cut. Note how the index finger is used to stabilise the scissors.



Scalpel insertion and removal of blade: The scalpel blade should be removed from the pack using the needle holder. With the handle held like this and the blade remaining stationary, the handle is placed on to the blade. To remove the blade, the index finger of the left hand is used to help push up the needle holder and take the blade off the lip of the handle. Then, holding the blade with the needle holder, the handle is removed in a backwards direction, the blade remaining stationary and pointing down. This reduces the chance of the blade flying off and causing injury. Effectively you should keep the blade stationary and move the holder on and off the blade.

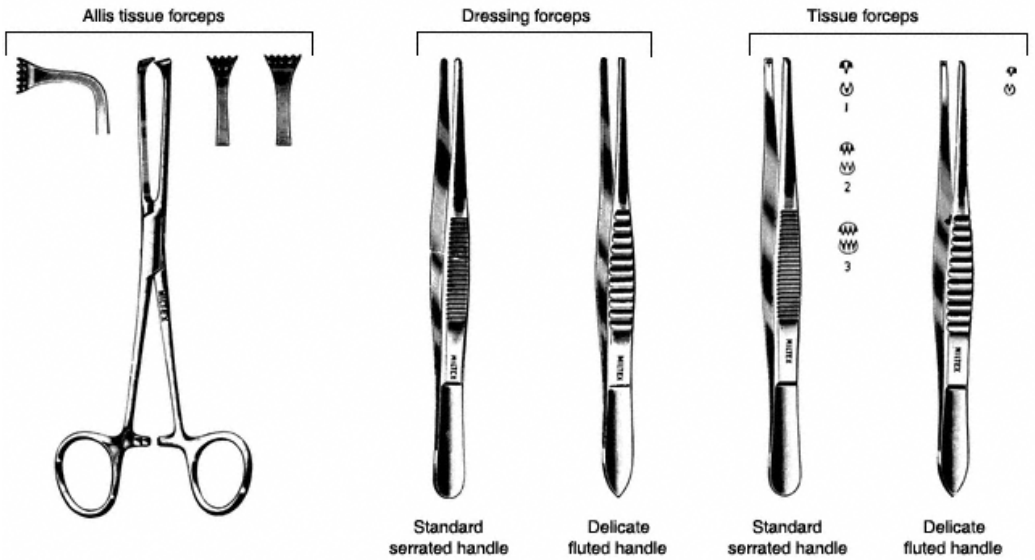
Scissors - Potts: Potts scissors are used in vascular work. You can see that the actual blades are at an angle to the shaft of the scissors. These scissors come with a variety of tips with angles ranging from 30 to 90 degrees and are useful for cutting straight along an arteriotomy. Once an initial stab incision is made, the scissors can be placed into the arteriotomy, their design allowing the blades to be kept parallel to the artery wall allowing straightforward and accurate division of the wall.



Toothed forceps: have V-shaped inter-digitating teeth. These tend to be used for handling skin as they are relatively atraumatic compared with non-toothed forceps.

Non-toothed forceps: have serrations at the tips of the shaft to help grip needles, suture material and needles. Forceps should be held in a pen-like grip. A common mistake is to hold them with the hand over the top of the forceps, which is an unstable way of holding them.

Hemostats: When placing hemostats on a vessel it is important that the curve of the jaws faces upwards to ease placement of ligatures and knot tying. Grasp the vessel in distal part of the jaws. In some cases, the section of the jaw near the hinge does not have serrations, making it difficult to grasp tissue adequately. In addition, grasping tissue closer to the hinge of the jaws creates difficulties when trying to tie a ligature around the vessel. When passing a length of suture for ligation in continuity, the assistant should hold the thread on a hemostat. The surgeon positions another hemostat beneath the vessel to be ligated, with the jaws slightly open. The assistant then positions the tips of the jaws of their hemostat just below that of the surgeon and the ligature is held under tension. This positioning helps the surgeon to pick up the ligature in order to pass it around the vessel. If ligature is not presented under tension, it is very difficult to pick up, so it is important to hold it taut.



Hemostats - Curved: come in various sizes ranging from small mosquito forceps to these criles, sawtels, and mayos. Held in a tripod fashion using the thumb and ring finger with the index finger extended down the shaft. he ratchet mechanism keeps the hemostat jaws tightly approximated and is unlocked by pushing the thumb laterally. All these hemostats are designed for right-handed individuals and, in a similar fashion to scissors, it is quite difficult when held in the left hand, to unlock the hemostats as one intuitively pushes away with the thumb. If you cannot master pulling the thumb towards the hand, the alternative technique is to use the open palm technique utilising the thumb and index finger on one loop of the haemostat and the middle and ring fingers on the other loop, opening the haemostat like this. Be aware that although most hemostats have serrations over the entire area of the jaws there are some, such as sawtels, where the serrations do not go all the way to the hinge end of the jaws. You should not grasp tissue at the very tip of the hemostat but rather slightly proximal to the tip. This allows suture material to be readily passed around the tip of the hemostat for subsequent ligation of the structure.

Hemostats - Rubber Shod: a straight instrument with small rubber tubes placed over the serrated jaws. Commonly used in fine vascular surgery allowing delicate monofilament sutures to be held in an atraumatic fashion avoiding fracturing of the suture material.

Needle Holder: held in a similar tripod fashion using the thumb, index finger and ring finger. The ratchet mechanism generally requires just one click to securely hold the needle without causing damage or bending the needle with too much force. An alternative, “palm technique” is where the thenar eminence is used to open the ratchet mechanism. Again it is important to try to keep the index finger down the shaft of the needle holder to maintain stability. After opening the suture pack, the needle is presented ready for mounting in the needle holder. Grasp the needle with the tip of the needle holder, two thirds along the shaft from the needle tip. When removing the suture from the pack, it is often useful to use your little finger to take up the slack in the suture. The needle can either be held two thirds away from the tip and perpendicular to the needle holder. Or, when using a half curved needle, it can be held half way along the shaft at a slight angle. It is important that the needle is grasped at the tip of the jaws of the needle holder.

Endoscopic Surgery: The Endoscopic Stack

Camera Box: Ensure this is switched on before starting work and note that all controls for this hardware are accessed by the three rubber buttons of the camera head itself. White balancing is achieved by turning up the light source, holding the scope near to a white piece of paper and then pressing the centre rubber button on the camera, the button nearest to you, and then the centre button again. White balancing only needs to be carried out when you change a scope or light cable; it is not necessary to white balance at the start of every practical

Light Source: Please note that the endoscopic light sources should be turned on when you begin your first session in the skills centre and not switched off during the day. Repeatedly switching the Xenon bulbs on and off drastically shortens their life therefore they should be turned up and down between sessions using only the rotary control on the front of the unit. Please set this rotary control to around the “11 o’clock” position; setting it higher is unnecessary and merely causes the iris of the camera to close and bring the screen brightness to the same level as before.

Monitor: The flat panel monitors are height adjustable; please ask a member of the laboratory staff to help you adjust your monitor if necessary.

Cameras and Rigid Endoscopes: The equipment you will have access to in the Skills Centre is the latest produced by Karl Storz. The stacks include flat panel monitors and Image 1 three-CCD cameras. We use both forward viewing and angled 'scopes and will encourage you to work with these throughout the session. It is vital you know how to hold these scopes properly and understand how to orientate the scope to allow you to view the target tissue. Firstly, the camera must always be held in such a way that it provides an absolutely horizontal view. With the Image 1 cameras this is assured by always keeping the three rubber control buttons on the camera body at the “12 o’clock” position. Secondly, the angle of view of the scope is determined by the position of the light cable’s junction with the scope. For example with a 30 degree scope, when the light cable is in the “12 o’clock” position, the scope is looking directly down at 30 degrees; when in the “6 o’clock” position, it is looking directly up; when in the “3 o’clock” position, it looks to the left, and when in the “9 o’clock” position, the view is to the right at 30 degrees. You will find holding the camera and scope quite easy if the camera is held in the supinated non-dominant hand and the light cable controlled with the dominant hand.

Visual problems: The surgical image is magnified, 2 Dimensional and unnatural. The image may be distorted due to various factors

epending on the skill of the camera operator, the image may be uncontrolled. Take responsibility for the situation and slow down your actions. Ensure that your cameraman is properly trained and practice together as much as possible. Finally, make sure that you have selected the correct equipment for the tasks you intend to perform and that it will be available at the time of the procedure. Optical axis-to-target view angle. (Errors increase as optical angle increases; aim to provide optical target to view angle of 90 degrees for best task performance). Direction of view (Altered view angle increases accessible field, but can create blind areas). Illumination - imaging relationship (Angled scopes provide lower illumination than end viewing scopes).

There are three important angles to be considered: 1. Azimuth angle: the angle between optical axis and instruments. Ideally these angles should be equal (30 deg) and allow comfortable movement within the visual field. 2. Manipulation angle: The angle between the axis of the right and left hand instruments. Ideally this angle should be 60o and allow comfortable movement within visual field. 3. Elevation angle: The angle between the target, and the axis of the instrument. Ideally this angle should be between 45-60 degrees and allow comfortable movement within visual field. Remember that this angle may be adjusted by table height. Swording occurs when the telescope or the shaft of the assistant's instrument obstructs the operator's instruments. If this occurs you may need to consider: repositioning retracting instruments, rotation of an angled telescope allowing alteration of the position of the end of the telescope, withdrawal of the telescope, transposition of the operator's instruments, additional port placement, changing the instruments to a different port. On-axis operating is where the scope and instruments approach the target from the same direction and is to be desired for all MAS tasks. Off-axis operating occurs when the scope and instruments approach the target from different directions. Proficiency during this mode of operating is dictated by intellect, access, anatomy, and pathology. In all cases, off axis operating is much more difficult and should be avoided if possible (usually by moving the camera port and or monitor).

Safe Dissection in Minimal Access Surgery: Remember that when operating laparoscopically, the image you are viewing is apparent and magnified between approximately 2x to 8x. Your view is 2-dimensional leading to problems with depth perception as well as physical and psychological fatigue. The view is also unnatural in a number if ways: the colour rendition may be altered, the view distance is of the order of one tenth of that in open surgery, and there is a loss of peripheral vision. All these aspects are important when it comes to dissecting in the MAS environment. Using long instruments with small jaws and with very little tactile feedback also means that dissection is often one of the main challenges in a MAS procedure. The types of dissection are the same as used in conventional surgery:

Blunt Dissection: The most common instruments used for blunt dissection are the suction/ irrigation probe, atraumatic graspers and the pledget or peanut swab. The non-dominant or assisting instrument is vitally important as the counter-traction applied to the tissues will enable effective dissection to take place.

Sharp Dissection: Scissors are useful instruments as both sharp and blunt dissectors. Remember that you should always see both blades before cutting any structure and that the non-dominant hand is important in that it provides counter-traction and tissue stability. Common errors that have been noted in scissor dissection include ineffective use of non-dominant hand, too much sharp and not enough blunt dissection, and also using scissors without clear view of field. Applying monopolar of electrosurgery to scissors is useful but do remember that when used on open scissors it has a tendency to blunt them.

Clip Application: Before proceeding to apply clips you must familiarize yourself with how to properly load the clip if using a non-disposable applicator. Most applicators have their jaws angled relative to the shaft of the instrument and so it is important to offer the jaws of the applicator to the clip at the correct angle. This normally means that the shaft of the applicator will be slightly off the perpendicular relative to the clip. Once the clip is loaded, the instrument should be carefully introduced into the abdomen taking care not to dislodge it by accidentally squeezing the handle. Once inside the abdomen, you must ensure that you visualize both jaws of the clip applicator before proceeding. If necessary, turn the shaft of the instrument, alter the amount of distraction of the structure with the non-dominant hand, or ask the camera operator to change the angle at which the scope is viewing the structure (if using an angled scope). Now, begin to close the jaws so that the tips of the clips approximate to form a ring but do not completely close the clips at this point. You should then carefully place the clip in the desired position, check that you have not trapped any tissue deep to the clip unintentionally, and only then completely close the clip applying it transversely across the structure. Adequate pressure should be exerted to ensure the clip is securely closed around the structure. Common errors in clip application include ineffective use of non-dominant hand, incorrect clip placement, applying clip without clear view, and accidental dislodgement of the clip

Endoloops: Commercial endoloops are made utilizing the Melzer knot which developed out of the slip knot and are useful for a variety of applications such as the reinforcement of clipped structures, closing the hole in a perforated gall bladder, ligating the base of the appendix, or securing a bleeding vessel being isolated by grasper. To properly deploy the loop you should ensure that before inserting it into a port, the loop is drawn back inside the applicator; this is important if using non-disposable ports. You must also ensure that you have a good ergonomic set-up and that the port is not so distant from the target that you are unable to reach it with your endoloop. For ease of use, you should insert the endoloop down the port at your dominant hand and have a pair of graspers down the assisting port in your non-dominant hand. The grasper should then be put through the loop and the structure picked up (this is easier than trying to poke the structure through the loop). Before proceeding any further, you should hand the grasper to an assistant as loop application is a two-handed task. You should then place the loop on the target tissue and locate the approximate desired position. To properly apply a knot, you should put the tip of the pushrod at the point you would like the knot to be tied and then pull back on the ligature. By combining traction on the ligature and slight pressure on the pushrod to keep its tip against the target tissue you will find the loop is placed accurately at the desired point. You must ensure adequate tightening down of the knot by applying firm traction to the ligature before proceeding to cut it leaving a tail of not less than 1cm.

Post-Op Confusion:

Post-op delirium is characterized by incoherent speech and thought, disorientation and impaired memory and attention and has bee reported in 5.1% to 61.3% of older pt's after surgery (Br J Anesth 2001;87:608-24) (reversible in >99%).

Step #1 Consider: Bleeding, Fever, Pneumonia, Hypoglycemia, Hypoxia (very common), Hypercapnia, Hypotension, DTs, PE, MI, Sepsis (often hypothermic before febrile). Hyponatremia. UTI.

Med SE: Insulin, oral hypoglycemics. Benzodiazepines, opiates. Lidocaine. H2-blockers - cimetidine, ranitidine.
Consider the utility of: 02, D50, naloxone.

W/u to consider: pulse ox, ABG, lytes, finger stick glucose, CXR, blood cultures. Consider a CT of the head early and always get a CT before a spinal tap.

Step 2: Other ddx: Thyrotoxicosis. Porphyria. Sedative withdrawal. CHF. Uremia. Dementia. Hepatic coma. Hypercalcemia.

If lateralizing signs or abnormal CSF: CVA. Post concussive. Meningitis. Encephalitis. Post ictal. Subdural hematoma. CNS tumor. CNS abscess.
If lateralizing signs or an abnormal CT, call neurosurgery before the spinal tap. Definitely include your resident and attending.
ICU psychosis and Sundowning are dx of exclusion. As always, treat underlying condition and avoid sedatives. Correct any fluid or electrolyte imbalances. Antipsychotics may be useful and most suggest Haloperidol @ 0.25mg to 2mg 1-2 hours qHS, repeat q4 hrs for severe agitation. See geriatric module: Delirium.
Risks: Cognitive dysfunction in the early postoperative period is common in all age groups, however, older patients are more likely than younger patients to exhibit cognitive dysfunction. Also see with low educational level, polypharmacy, alcohol & sedative withdrawal, impaired vision or hearing, sleep deficiency, anxiety, depression, dementia, use of physical restraints, malnutrition, bladder catheter, use of >3 new meds. Older surgical pt's who carry at least one copy of the E4 (ApoE4, a risk factor for early-onset AD) allele are an at increased risk (4-fold)) for postoperative delirium according to a study with 190 pt's, mean age 72.5 (Anesthesiology 2007;107;3). In a subset of geriatric (>70yo) pt's undergoing abdominal surgery who are at high risk for in-hospital delirium, adverse outcomes correlated with key clinical variables, such as hyperglycemia (glucose >150 mg/dL) and poor nutritional (albumin level <3 mg/dL [<0.003 g/dL]) and functional states (Arch Surg. 2007;142:1072-1078)....risk for postoperative delirium, the overall incidence of delirium, in-hospital mortality, and prolonged length of stay is 60%, 20%, and 32%, respectively. Older age, less education, and previous stroke were among factors predicting cognitive dysfunction at 3 months post major noncardiac surgery in 1064 nondemented adults who received general anesthesia (Anesthesiology 2008;108:18-30).....At hospital discharge, the incidence of cognitive dysfunction was 37%, 30%, and 41% in young (age range, 18–39), middle-age (age, 40–59), and older (age, 60) groups, respectively.....At 3 months, the incidence of cognitive dysfunction was 6%, 6%, and 13% in the three age groups, respectively.....By comparison, the incidence of cognitive dysfunction among controls was about 5% in all age groups at both time points. Independent predictors of cognitive dysfunction at 3 months were older age, lower educational level, history of stroke, and cognitive dysfunction at hospital discharge. Patients with cognitive dysfunction at discharge and at 3 months had higher 1-year mortality than other patients.....The relative contributions of drug side effects, exacerbation of previous subtle cognitive deficits, or perioperative brain injury are unclear. See Geriatrics module: **Etiology of Delerium in Hospitalized pt's** |

Post-op Hypotension: .....END DEMO of this chapter.

Women's Health: Obstetrics and Gynecology:

Edited by Robert A. Reid MD, Obstetrician and A A Weber, Family Physician.

Contents: Contraception | **Cervix - Ovary - Vagina** | Sexual Issues | **Menstrual - Uterine** | Breast | **Menopause** | Osteoporosis | **Infertility** | Pregnancy | **Cancer** | Pain | **Hormonal Agents** | INFECTIONS | **Eating D/o** | Domestic Violence | **Rape** | Lesbian | See Urology: **Urine Incontinence** | *INDEX* |
Commonly Used Meds:
Links: Meds & Pregnancy | **Gestational Diabetes** | Infections | **Menopause** | Osteoporosis | **Contraception** | PMS | **Dysmenorrhea** | PCOS | **Endometriosis** | Fibroids |

Contraception:

Links: Failure Rates | **Choices for Specific Medical Problems** | Oral Contraception | **Emergency Contraception** | Other Contraception (Patch, Ring, Condoms…) | **Depo-Provera (DMPA)** | Lunelle Shots | **Dermal Implants** | Intrauterine Device (IUD) | **Permenant Methods (Tubal, Essure, Quinacrine)** | Fertility Awareness | **Diaphragms** | Pregnancy | **Abortion** | See Sexual Issues |

Approximately 49% of U.S. pregnancies are unintended, a rate that compares unfavorably with rates in countries such as France (33%) and Scotland (28%)(Obstet Gynecol 2007;110:1493)……65% of cases occur in women <20yo. Female sterilization is the most common method (25.6% using) followed by OCP's (24.9%), male condom (18.9%), male sterilization (10.1%), no method (7.5%), withdrawal (2.9%), injectable (3%), diaphragm (2%), implant (1.5%), IUD (1%) in 1995.
The FDA has approved a non-surgical sterilization technique for women. The Essure system is performed transcervically w/o incision or general anesthesia. Is it performed via hysteroscopy, placing two soft metallic coils into the proximal fallopian tubes that creates a local fibrotic response to occlude the tubes over 3 mo's.
Codes: V25.01 Contraceptive management.
First-year Fertility (Failure) Rates: Biological factors appear to increase the likelihood that a woman will engage in intercourse during her 6 most fertile days (24% more frequent) rather than at other times, thus a single episode of unprotected sex may be more risky than chance alone would dictate (Hum Reprod 2004;June 10th online issue).
Methods --> Lowest observed failure rate (%) Vs Failure rate in typical user (%):
Tubal sterilization **-->** 0.3%.
Vasectomy **-->** 0.4%.
Injectable progestin (DEPO) **-->** 0.3.
Progestin implant (Norplant) **-->** 0.09.
Combined oral contraceptive pill **-->** 0.1-3.
Progestin-only pill **-->** 0.5-3.
Copper T-380A intrauterine device **-->** 0.6-0.8.
Quinacrine (QS) Pellets **-->** 1-2%.
Male Condom **-->** 3-12.
Female Condom **-->** 5-21.
Diaphragm (with spermicide) **-->** 6-18.
Patch **-->** 1-3.
Ring **-->** 0.7-3.
Cervical cap **-->** 11.5-18.
Sponge **-->** nulliparous (16%) & parous (32%)
Foams, creams, spermicides, etc. **-->** 6-21.

Coitus interruptus (withdrawal) **-->** 16-23.
Fertility awareness techniques (e.g., rhythm) (periodic abstinence) **-->** 2-24.
Douche **-->** 40.
Chance (no method of birth control) **-->** 85.
Compliance: Women who are considered to be at high risk of pregnancy appear more likely to continue with use of OCP's than with a transdermal contraceptive patch (Obstet Gynecol J 2006;108:378-386)...date from 1230 women found the pregnancy rate per 100 woman-years of contraceptive use was 3.62 for the pill users and 14.84 for the patch users. A substantial minority of women (40.6%) do not refill prescriptions for hormonal contraceptives after 30 days (Obstet Gynecol 2008;112:782-787).....over 70% of women fail to refill their prescriptions for contraceptive products on a timely basis for even one year. Rates of unintended pregnancy were <1% for both OCP's and the contraceptive patch according to data on 375,000 women in the U.K.(Contraception 2009;80:142).
Prevalence of Contraceptive Methods in 2002: Oral contraceptive @ 30.6%. Female sterilization @ 27%. Male condom @ 18%. Vasectomy % 9.2% Three-month injectable @ 5.3%. Withdrawal @ 4%. Other @ 4%. Intrauterine device % 2% (Adv Data 2004;350:18).
Ensure system (Conceptus): Hysteroscopic sterilization using the Ensure system can be performed safely and effectively on an outpatient basis without general anesthesia or sedation, with high rates of pt satisfaction (BJOG 2007;114:676-683). The system involves transcervical placement of two microinserts into the fallopian tubes to achieve permanent contraception. Successful bilateral placement of the microinserts was achieved in 92% and was deemed optimal in 88%. The complications seen in 5% of pt's were mainly minor vasovagal reactions, and all of the pt's were discharged within 4 hours of their procedure.

Contraception in Women with Medical Problems / Conditions:

See Common Problem & Solutions with OCPs |
DIABETES MELLITUS: The risk for birth defects is directly related to glycemic control, so effective family planning for women with diabetes is especially important.3 All forms of contraception can be used safely by otherwise healthy diabetic women. Hormonal contraception does not appear to affect maternal glycemic control or to accelerate end-organ effects. However, be cognizant of additional cardiovascular risk factors including tobacco use, obesity, age older than 35, hypertension, retinopathy, neuropathy, or vascular changes. In general, diabetic women with additional risk factors should avoid using estrogen-containing products and instead should consider progestin-only methods (Depo-Provera [depot medroxyprogesterone acetate, DMPA], Implanon [etonogestrel implant], minipills), barrier methods, levonorgestrel-releasing or copper IUDs, or sterilization.
MIGRAINE: Women who experience migraine with aura have a higher baseline risk for stroke than do women who have migraine without aura.5 Therefore, women who have migraine with aura should not use combination estrogen-progestin contraceptives. For women who have migraine without aura, the data are unclear because not all investigators have classified migraine history similarly — however, as the absolute risk for stroke in this group is minimal, these women can use combination hormonal contraceptives if they are otherwise healthy and younger than 35. Because age is an independent risk factor for thrombosis, women 35 or older who have migraine without aura should use a contraceptive method without estrogen. Regardless of migraine type, progestin-only methods, barrier methods, IUDs, and sterilization are safe.
OBESITY: Obese women can safely use progestin-only methods, barrier methods, IUDs, or sterilization. Combination hormonal contraceptives should be used with caution as obesity is an independent risk factor for deep venous thrombosis (DVT), and exposure to estrogen in this population can increase DVT risk further. However, because DVT risk is greater in pregnant women than in those using combination hormonal contraceptives, the WHO MEC state that the benefits offered by this form of contraception generally outweigh the risks in otherwise healthy obese women. Be mindful of comorbidities (being 35 or older or having hypertension or diabetes) that would preclude obese patients from using estrogen-containing contraceptives. Hormonal contraception has not been studied extensively in obese women, and there is some concern that it might be less effective than in women of normal weight.9,10 Nevertheless, any form of contraception prevents more pregnancies than does no contraception. Encourage the use of a dual method (e.g., hormonal contraception plus condoms) if hormonal contraception is planned, or consider methods in which efficacy is unaffected by body weight (DMPA, IUD, or sterilization).
THROMBOSIS: Combination hormonal contraceptives increase risk for thrombosis. For combined oral contraceptives (OCs), both the estrogen dose and the progestin type contribute to this increased risk. However, as previously noted, DVT risk associated with pregnancy far exceeds that related to hormonal contraception. Estrogen's effect is dose-dependent: Oral contraceptives containing 50 µg ethinyl estradiol are associated with a higher risk for thrombosis than are formulations containing 20–30 µg ethinyl estradiol. Women with known thrombogenic mutations (e.g., Factor V Leiden) should not use combination hormonal contraceptives. For other conditions such as obesity or immobility that increase the risk for thrombosis, an individualized approach should take into account other risk factors such as age and general health, and alternatives to estrogen-containing methods should be considered. Although use of progestin-only preparations causes only minor effects on coagulation and fibrinolysis,15 class labeling of progestin-only methods includes the risk for thrombosis. In women with a known thrombophilia or a prior clot, the WHO MEC do not restrict use of progestin-only methods (DMPA, etonogestrel implant, progestin-only pill, or levonorgestrel-secreting intrauterine system); the advantages of these methods generally outweigh the risks in such women.1 However, an active clot is a contraindication for any estrogen-containing contraceptive. Barrier methods, copper IUDs, and sterilization are safe to use.
HYPERTENSION: Combined OCs increases blood pressure by as much as 8 mm Hg systolic and 6 mm Hg diastolic. Depot medroxyprogesterone acetate (DMPA; Depo-Provera) does not significantly affect blood pressure. A trial of combination contraceptives may be appropriate as long as the pt is otherwise healthy, shows no signs of end-organ vascular disease, and does not smoke. Progestin-only contraceptives and the levonorgestrel-releasing intrauterine system (Mirena) are appropriate options in women with hypertension. In most cases, this increase is not clinically significant. Women should have their BP checked before and periodically after initiating a combined hormonal method. If a clinically significant increase in BP occurs, the contraceptive should be discontinued to see whether BP reverts to baseline. According to the ACOG guidelines, otherwise healthy women 35 or younger with well-controlled hypertension on medication can use a combined hormonal method as long as BP remains controlled.2 In contrast, the WHO MEC state that the risks of a combined method outweigh the benefits in such women. Progestin-only methods do not raise BP and can be used safely by hypertensive women. Barrier methods, IUDs, and sterilization are also safe.
ANTIEPILEPTIC DRUGS FOR EPILEPSY OR BIPOLAR DISORDER: Some antiepileptic drugs (AEDs) induce hepatic enzyme activity, thus increasing the metabolism and impeding the efficacy of OCs, patch, ring, and contraceptive implants. When taken as single pills, OCs containing <30 µg estrogen should be avoided by women taking hepatic enzyme inducers; conversely, increasing

the OC dose (e.g., taking 2 pills per day) and eliminating the hormone-free interval have been proposed as methods to maintain contraceptive efficacy. Hormonal contraceptives can reduce blood levels of AEDs, necessitating dose titration of the latter. DMPA and IUDs (hormonal and copper) are excellent alternatives; emergency contraception also can be used safely.
LIPID D/o: The estrogen component of enhances removal of LDL and increases levels of HDL cholesterol. Oral estrogen also increases triglyceride levels, but in women with both increased HDL and decreased LDL levels, this moderate increase in triglyceride levels does not increase the risk of atherogenesis. Formulas with less androgenic progestins increase HDL levels more and triglyceride levels less than formulas with more androgenic progestins.
SMOKER: ACOG recommends that physicians prescribe combination oral contraceptives with caution, if at all, in women older than 35 years who smoke.
BREAST Ca: The risk of benign breast disease is lower in women who use oral contraceptives compared with non-users. The use of oral contraceptives before age 30 and use for more than five years are associated with an increased risk of breast cancer in women with the BRCA1 gene, but not in those with the BRCA2 gene. Because oral contraceptive use reduces the risk of ovarian cancer, these drugs offer significant benefits for women with BRCA mutations.
UTERINE FIBROIDS: Combination oral contraceptives reduce menstrual blood loss in women with normal menses and those with menorrhagia. Oral contraceptives also reduce dysmenorrhea and may decrease bleeding disorders in women with uterine fibroid tumors. The levonorgestrel-releasing intrauterine system also reduces menorrhagia associated with uterine fibroid tumors.
POSTPARTUM: Women remain in a hypercoagulable condition for weeks after childbirth. Product labeling for combination oral contraceptives advises deferring their use until four weeks postpartum in nonbreastfeeding women. Because DMPA and progestin-only contraceptives do not contain estrogen, these methods may be safely initiated immediately after delivery.
BREASTFEEDING: The use of combination oral contraceptives in well-nourished breastfeeding women does not result in infant developmental problems, and these drugs may be initiated once milk flow is established. DMPA and progestin-only contraceptives do not impair infant development or lactation and, in fact, may increase the quality and duration of lactation. Because DMPA and progestin-only pills do not have a procoagulation effect and are safe in breastfeeding women, their use at 6 weeks postpartum in lactating women and immediately after delivery in nonlactating women is reasonable.
ATHLETES: Ten weeks of resistance exercise training resulted in 40% less gain in lean mass among users of oral contraceptive pills than in nonusers (Experimental Biology 09 FASEB 2009; Abstract 4197).....The reduced muscle development in OC users was associated with significantly lower levels of androgenic hormones and insulin-like growth factor 1 (IGF-1), as well as elevated levels of the stress hormone cortisol....."women who are active or competing, and want to achieve the highest level of performance need to consider whether birth control is a negative influence on that."
*****Ref:** (World Health Organization. Medical eligibility criteria for contraceptive use. Third Edition, 2004. Accessed: Dec 4, 2007. (http://www.who.int/reproductive-health/publications/mec/)(ACOG Committee on Practice Bulletins-Gynecology. ACOG Practice Bulletin No. 73: Use of hormonal contraception in women with coexisting medical conditions. Obstet Gynecol 2006 Jun; 107:1453)

Oral Contraceptive Pills (OCP):

Links: Contra & SE | **Benefits** | Starting | **Common Problem & Solutions** | Missed Pills | **E-dominant** | P-dominant | ****The Pills**** | Continuous | **Progestin-only pill** | OCP's to HRT |

Highly effective at preventing pregnancy, <1 per 100 women with perfect use get pregnant each year (5/100 typical users). Currently contain 2-5x less estrogen and 5-10x less progesterone than the initial pills in the 1960's.
Mechanisms: Suppress FSH so no follicle or LH surges, changes the cervical mucous. 55% of women stop using within 1yr, most often from irregular bleeding in 1st 3mo's. Less effective (60% inc risk of OCP failure) if pt weighs >70.5 kg (155lb) (Obstet Gyn 2002;99:820). Can be safely prescribed in many women >35yo until menopause (Ann Int Med 2003;138:54-64).
First Generation Pills: pills approved before 1970, 50 mcg pills. Have a failure rate of <1 per 100 women-years of use (Pearl Index <1).
Second generation Pills: 35 mcg, low-dose pills. Have a Pearl Index of <1.5.
Ultra-low dose pills: Have a Pearl Index of <2 in clinical trials. Failure rates are higher in "real life," and may be 8% during the first year of use (Am Fam Phys 2004;69:853-60). Noncompliance is an issue outside clinical trials. Women with a BMI >35 are usually excluded from clinical trials. An increase in pregnancies with the newer pills that contain <30 mcg of estrogen. There are about 2 pregnancies per 100 women per year on these lower-dose OCs...compared to < 1 pregnancy with 50 mcg OCs. The lower-dose pills are usually better tolerated and less likely to cause thrombosis than the 50 mcg pills. But also less "forgiving" of a missed dose. Lower-dose pills might not work as well in heavier women (Prescriber's Letter 2007;23(3):230303)....Slightly less effective. Examples include Ethinyl estradiol 30 mcg (e.g., Nordette) and 20 mcg ethinyl estradiol (e.g., Alesse). Estrogenic side effects (headache, nausea, weight gain, breast tenderness) are nearly comparable to placebo. Cause irregular bleeding more commonly than higher dose pills.
Contra: liver dz (active hepatitis, severe cirrhosis, liver tumors, h/o cholestatic jaundice of pregnancy), CVA/ CAD (h/o stroke or ischemic heart dz), uncontrolled HTN (SBP >160 or DBP >99 mmHg), smoking >15cig's/day **if** age >35yo, h/o DVT/ PE, pregnancy, postpartum <6wks & breast-feeding, DM with any complication (vascular, nephropathy, neuropathy or retinopathy), current breast Ca, migraine with aura at any age, severe H-A's with neurologic manifestations. Major surgery with prolonged immobilization, complicated valvular heart disease.
All should be advised to stop smoking. Age >40 **-->** small inc risk DVT/PE, avoid if smoker, lipid d/o, HTN, migraines, **Factor Five Leiden (FVL) mutation** (30X risk with OCP use, 8X w/o, 3.5X with normal female and OCP use, 8X with normal female and pregnancy). Screen for FVL with the APC test (+ if <2 have, if borderline check an FV genotype).
Relative contra: classic migraine with age ≥35yo (ok so long as no focal neuro sx's), HTN (inc both SBP & DBP by 4-9 mHg), planned surgery (stop 4 wks before a major surgery, stop to reduce risk of VTE), SLE, inc trig's. St. John's wort (induces CYP3A4 enzymes, this increases the metabolism of ethinyl estradiol and reduces the effectiveness of OCs). A study suggest that thrombophilia screening is warranted before oral contraceptives are administered to adolescents with family history risk factors (J Pediatr Adolesc Gynecol 2007;20:221).....individuals who have had an unexplained thromboembolic event before age 45, FHx of clotting disorders (DVT/PE), multiple miscarriages, or stillbirths....however, screening is expensive, and it can delay the start of hormonal contraception for several weeks.

Overweight women: are more likely than lean women to become pregnant while using oral contraceptives (58% higher risk if BMI >27.3) (Obstet Gynecol 2005;105:46-52).The NuvaRing contraceptive appears to be as effective in women who weigh more than 89.9 kg (198 lbs) as in women who weigh less based on retrospective analysis of four phase III studies (2005 anual meating of the Am Coll of Obst and Gyn). Higher body weight and BMI are not associated with reduced efficacy in Ortho Tri-Cyclen Lo users (Poster presentation at: 54th Annual Clinical Meeting of The Am Coll Obst Gyn, May 6-10, 2006).
SE: breast tenderness, nausea, H-A (Can usually con't dose as improves with time. Am J Ob Gyn 2005;193:636-49), spotting in 1st 3mo. OCP use may increase the risk of infection and pain following the removal of impacted wisdom teeth (Brit Dent J 2003;194;453-455). The use of modern, low-dose OCP's containing ≤50 mcg of estrogen does not appear to appreciably raise the risk of ischemic stroke in healthy women (Stroke 2003;34:4332). The association between use of low-dose OCPs (<50 mcg of ethinyl estradiol) and stroke cannot be concluded with certainty (Arch Intern Med 2004;164:741-747). Low-dose oral (2nd and 3rd gen) contraceptives increase the risk of both cardiac (RR 1.84) and vascular arterial events (RR 2.12 for stroke) (J Clin Endocrinol Metab 2005;90:3863-3870)….if limited to healthy women and restricted in time, should not increase noticeably the incidence of these adverse outcomes, which might be outweighed by the benefits of contraception," the investigators conclude. Increase the risk of osteoporosis...consume at least 1000 mg/day of calcium to prevent bone loss (J Clin Endo and Metab 2005;90;221). A large prospective study concludes that low-dose OCP's do not increase MI risk (Fertil Steril 2007;88:310-6)....this study did not address the risk for stroke or venous thromboembolism. Oral contraceptives may raise arterial atherosclerosis 20% to 30% for every decade of use according to a random sample of 1,301 apparently healthy women ages 35 to 55 in Belgium who underwent ultrasound imaging of atherosclerosis of the bilateral carotid and femoral arteries (AHA meeting 2007; Abstract 3614). Neither former nor current OC users had a higher risk for MI than did never users during an 11-year period according to a population based study with 48,000 Swedish women (Fertil Steril 2007;88:310). Use of oral contraceptives raised the odds of incident urinary incontinence moderately (odds ratio = 1.27)(J Urol 2009;181:2170).....Longer duration of use was associated with progressively greater odds of incident incontinence.....Only urge incontinence was associated with ever use of OCs (OR, 2.48).
VTE: The risks of using oral combination contraceptives include: a 3- to 6-fold increase in the incidence of VTE apparent by the fourth month of use and highest between six and 12 months of use; a 2- to 6-fold increase in the risk of ischemic stroke among women with a history of migraine; and an increase in cervical cancer risk after five or more years of use (Arch Intern Med 2004;164:1965-76). Women with hereditary deficiencies of protein S, protein C, or antithrombin are at higher risk of VTE from combined oral contraceptives than women without such deficiencies, according to a retrospective family cohort study (Arch Intern Med 2007;167:282-289). Excess risk for venous thrombosis was lowest for OCs containing levonorgestrel as well as for those with the least amount of estrogen (20-µg doses) (BMJ 2009;339:b2890).....Levonorgestrel-containing OCs were associated with nearly fourfold VT risk (odds ratio, 3.6); OCs containing gestodene (OR, 5.6), drospirenone (OR, 6.3), cyproterone (OR, 6.8), or desogestrel (OR, 7.3) were associated with even higher risk (gestodene- and cyproterone-based OCs are not available in the U.S.).....High-risk or older women might consider a progestogen-only method of contraception. In young, healthy, normal-weight women, use of third-generation oral contraceptives is associated with elevated levels of high-sensitivity C-reactive protein (CRP), potentially placing these women at higher risk for venous thromboembolism and cardiovascular disease (Obstet Gynecol 2008;111:857-864). Many women are using brands of oral contraceptives that have been linked to greater risk for venous thrombosis according to a Dutch population-based study (BMJ. 2009;339:b2890, b2921, b3164).....Compared with nonuse of oral contraceptives, use of currently available oral contraceptives was associated with a 5-fold increase in the risk for venous thrombosis (OR, 5.0).....Compared with other progestogens, levonorgestrel was associated with the lowest thrombotic risk (with a nearly 4-fold increased risk for venous thrombosis vs nonusers of oral contraceptives; OR, 3.6).....increased 5.6-fold for gestodene, 7.3-fold for desogestrel, 6.8-fold for cyproterone acetate, and 6.3-fold for drospirenone.
....."For women of normal weight and without known genetic predispositions, we recommend a low dose combined pill as first choice for contraception," the study authors conclude. "For women genetically predisposed to venous thrombosis who still want hormonal contraception, however, a progestogen only pill or hormone releasing intrauterine device seems to be the appropriate first choice."
Benefits / Indications: decreases acne. Help regulate menstruation as decreases DUB/ PMS/ dysmenorrhea, shortens the duration & amount of flow and increases regularity. Prevent anemia (IDA), dec ovarian Ca (40-60%), dec endometrial Ca (50-56%, persists after discontinuation), dec risk of PID, dec risk of ectopic pregnancy, and dec 50% benign breast dz's. Decreased the erratic hormone fluctuations in peri menopausal period and relieves most sx's (HRT is 4-10X less potent). Prevent osteoporosis. Effective, only a 3% accidental pregnancy rate. No inc risk of breast Ca (NEJM 2002;346:2025). The use of oral contraceptives, but not estrogen replacement therapy, lowers the risk of developing RA (J Rheumatol 2004;31:207-213), most estrogen supplements used for postmenopause have < one-sixth the potency of low dose OC. OCP use does not influence premenstrual mood in most women (Am J Obstet Gynecol 2003;189:1523-1530). Progestin-only contraceptives don't increase risk for breast cancer (Contraception 2004;69:353-60). OCP's (low-dose, monophasic, combined) generally improve body composition (BMD) in female athletes with or without amenorrhea (J Clin Endocrinol Metab 2004;89:4364-4370). Women of child-bearing age who use oral contraceptives face a lower risk of lower urinary tract dysfunction than women of similar ages who do not (Fertil Steril. 2009;92:428-433)....."positively influences bladder and urethral function".....Use of oral contraceptives was associated with a 43% lower risk of stress urinary incontinence, a 48% lower risk of mixed urinary incontinence, and a 64% lower risk of urgency urinary incontinence.
Cancers: Contraceptive pills do not increase the overall risk of cancer and may even reduce some risks, according to a huge British cohort study (BMJ 2007: doi:10.1136/bmj.39289.649410.55)....the risk for gynecological, colon, and other cancers actually decreased, the risks of cervical, central nervous system, and pituitary cancers increased. OCP use is associated with an increased risk of cervical cancer (Lancet 2003;361:1159-1167), possibly due to acquisition of HPV. OCP use is associated with increased risk (OR of 1.19) for premenopausal breast cancer according to a meta-analysis (Mayo Clinic Proc. 2006;81:1287, 1290-1302). Combination oral contraceptives seem to reduce the risk for ovarian cancer for many years after their use is discontinued (Lancet 2007;369:1703-10). Data from the U.K. National Health Service (NHS) showed that ever-users of OCs had statistically significant relative risk reductions of 12% for any type of cancer and 30% for gynecologic cancers compared with never-users (BMJ 2007;335:651)....Prolonged use of OCs (>8 years) reduced ovarian cancer risk by 62% but increased the risk for any cancer by 20%. In particular, prolonged OC use more than doubled the risk for cervical cancer (risk ratio, 2.73) and increased 5-fold the risk for central nervous system and pituitary cancers (RR, 5.51). Use of OCP's for 5 or more years is associated with the increased risk for invasive cervical cancer and CIN3/carcinoma in situ according to analysis of pooled data from 24 studies (Lancet. 2007;370:1591-1592, 1609-1621)...Each year of use increased the relative risk by a factor of 1.07 equivalent to a change in relative risk per 5 years

of 1.38.....the risk diminishes with years from last use and reaches the level of never-users by 10 years. OC use had neither a beneficial nor a harmful effect on breast cancer mortality according to a 15-year follow-up study with 1473 women (Obstet Gynecol 2007;110:793). OC use had neither a beneficial nor a harmful effect on breast cancer mortality according to a 15 yr study on 1473 women who died from breast cancer (Obstet Gynecol 2007;110:793). Risk for cervical cancer increased in current users of hormonal contraception but declines after use stops according to analysis of individual data for 16,573 women with cervical cancer and 35,509 women without (Lancet 2007 Nov 10; 370:1609). Compared with never-users of OCs, ever-users had an overall relative risk for ovarian cancer of 0.73 (Lancet 2008;371:303).....Those who had ceased OC use within the past decade had the steepest decline in relative risk (29% per 5 years of use), but a reduction in risk (15% per 5 years of use) still was observed 20 to 29 years after OC discontinuation.....An editorialist writes that these "compelling" findings "set a new standard in primary prevention for a deadly cancer, noting that the decline in ovarian cancer risk outweighs small increases in risk for other cancers that are associated with oral contraceptive use. Use of the OC pill or hormone therapy does not seem to increase risk for a second primary breast cancer according to a multicenter, population-based, case-control study that compared 708 women with asynchronous bilateral breast cancer (cases) with 1399 women with unilateral breast cancer (controls) (J Clin Oncol 2008 Feb 4;e-pub ahead of print)....editorialist notes that "Although these results run counter to conventional wisdom, they are relevant to breast cancer survivors who have no evidence of disease beyond regional lymph nodes and who are considering use of OCs or HT". Oral contraceptive use is associated with an increased risk of breast cancer according to data from the Case-Control Surveillance Study with 907 case women (731 white, 176 black) and 1711 controls (1152 white, 559 black) (Am J Epidemiol 2009;169:473-479).....On multivariate analysis, the odds ratio (OR) was 1.5 for 1 year or more of oral contraceptive use relative to less than 1 year.....The association between oral contraceptive use and breast cancer risk did not differ according to the hormone receptor status of the tumor.

Drug Interactions:

Some drugs will dec efficacy --> TCN, Doxy, PCN, Amox, Rifampin, Clinda, Griseofulvin, CBZ, Lamotrigine, phenobarbital, Primidone, Felbamate, antacids, anti-HIV agents (Ritonavir), Bactrim, St John's Wort and metronidazole. Women who take OCP's should avoid eating >10g/d of licorice can trigger edema from the glycyrrhizic acid (gives licorice its distinctive taste), which depletes the body of minerals such as potassium, zinc and magnesium. Antiepileptics such as Lamictal (lamotrigine) increase OC metabolism, best to prescribe a an OC with at least 50 mcg of estrogen or consider switching to Neurontin, Keppra, Gabitril, or Depakote if appropriate as these antiepileptics don't affect OCs.

Drug concentrations affected by OCP's: Lamotrigine (Lamictal), Mephenytoin (Mesantoin), Selegiline (Eldepryl), Chlorpromazine (Thorazine), Clozapine (Clozaril), Amprenavir (Agenerase), Fosamprenavir (Lexiva), Alprazolam (Xanax), Cholrdiazepoxide (Librium),
Clonazepam (Klonopin), Diazepam (Valium), Flurazepam (Dalmane), Midazolam (Versed),
Prazepam, Quazepam, Triazolam (Halcion), Lorazepam (Ativan), Oxazepam (Serax),
Temazepam (Restoril), Theophylline, Hydrocortisone, Methylprednisolone (Medrol),
Predisolone (Delta-Cortef), Prednisone (Deltasone), Cyclosporine (Neoral, Sandimmune), morphine, aspirin, tricyclic Antidepressants (TCAs).

Anticonvulsants that decrease serum steroid levels: (ACOG Practice Bulletin No. 73. Obstet Gynecol 2006;107:1459) Barbiturates (including phenobarbital and primidone [Mysoline]). Carbamazepine (Tegretol) and oxcarbazepine (Trileptal). Felbamate (Felbatol). Phenytoin (Dilantin). Topiramate (Topamax). Vigabatrin (Sabril, not available in the US).

Anticonvulsants that do not decrease serum steroid levels: Ethosuximide (Zarontin). Gabapentin (Neurontin). Lamotrigine (Lamictal). Levetiracetam (Keppra). Tiagabine (Gabitril). Valproic acid (Depakene). Zonisamide (Zonegran).

Anti-infective agents that decrease serum steroid levels: Rifampin (Rifadin).

Anti-infective agents that do not decrease serum steroid levels: Ampicillin. Doxycycline (Vibramycin). Fluconazole (Diflucan). Metronidazole (Flagyl). Miconazole (Monistat). Quinolone antibiotics. Tetracycline antibiotics.

Antiretrovirals that decrease serum steroid levels: Lopinavir/ritonavir (Kaletra). Nelfinavir (Viracept). Nevirapine (Viramune). Ritonavir (Norvir).

Antiretrovirals that increase serum steroid levels: Amprenavir (Agenerase). Atazanavir (Reyataz). Delavirdine (Rescriptor). Efavirenz (Sustiva). Indinavir (Crixivan)

Starting OCP: OCs can be started at **any** time after the onset of the next period (Quick-Start) during the menstrual cycle without affecting bleeding patterns (Fertil Steril 2003;79:322-9). Immediate initiation of OCP's before start of the next menses (start on the date of prescription) improves continuation of OCP's use according to a RCT (Obstet Gynecol. 2007;109:1270-1276).....a previous meta-analysis demonstrated no significant teratogenic potential associated with the use of OCs in early pregnancy….will decrease time required by clinic staff explaining when to begin OCs. Take pill at same time each day (qHS), do monthly breast exam the day she starts her new pack, need a back-up birth control method for 1st 7 days. ** Perform pregnancy test if pt does not have a normal period after completing first package of pills. In the past, to avoid disruption of menstrual patterns, pt's have typically been told to begin OCs on the first Sunday after their next menstrual period begins, and this delay contributes to failure to begin taking OCs.

Old: "start" on the 1st Sunday of cycle (to avoid menstruating on weekends) (start after period started in order to ensure that they are not pregnant and in an attempt to minimize breakthrough bleeding). The pt can expect to have "period" to start ~2 days after a "day" they start (Ex: if start on Saturday, menses starts on a Monday 3 weeks later).

If miss 1 pill --> take missed pill immediately and next one at regular time. Or Take 2 the next day.

If miss 2 pills --> take 2 qd X 2d, but use barrier method for rest of month or withdraw then restart fresh pack in 5 days.

Disp: #84, refills #3 =1yr. 1st and last pills the most important. Better to have <7day pill free interval, can inc effectiveness if go to 4-5 days. Continue to use condoms to prevent STD's. There may be advantages to providing a year's supply of OC pills as breaks in continuity of supply of OCs ("running out of pills" for any reason) was one of the top three reasons for contraceptive failure (Obstet Gynecol 2006;108:1107-14).

Common Problems with OCP's:

First few mo's: it is normal to have dec sex drive, H-A, breast tender, spot, wt gain, nausea, depression, break-through bleeding (main reason why pt stops).

Breakthrough bleeding / Spotting = ovulation: If the pt recently started taking combination OCP's and is having abnormal uterine bleeding after 3mo's, give a trial of Ibuprofen 600-800mg TID for 1-2 weeks or try supplemental estrogen for 1-2 weeks before switching to a pill formulation with either higher estrogen or a different progestin. Encourage women to try a pill for at least 3 months

before deciding if breakthrough bleeding is a problem. Explain that missed pills also lead to spotting. If the problem persists, try a pill with more estrogen for early (first week after menses) breakthrough bleeding or Rx one with more estrogen and progestin for mid-cycle (days 14 to 21) bleeding. Consider one with more progestin for late breakthrough bleeding. **Other:** stop pills 1-2 mo's and change brands to monophasic or inc estrogen dose, shorten the pill free interval from 7 to 5 days. Check hCG. Stop smoking (antiestrogenic effect) **Or** add .625mg of estrogen during the spotting.
If periods stop: check hCG, change brands or add 0.625 Premarin to 1st ½ of 1mo.
Steadily worsening cramps: Use continuous OCP's X3-4mo.
Acne: two triphasic formulations, Ortho Tri-Cyclen and Estrostep, and one monophasic extended-cycle pill, Yaz, are FDA approved for acne. But most OCs are helpful because estrogen decreases testosterone levels. Use of the 3-mg drospirenone/20-µg ethinyl estradiol 24/4 combined OCP is effective in significantly reducing acne lesion counts in girls and women aged 14 to 45 years according to a RCT (by 46.3% vs 30.6% in the placebo group at 3-6 mo's)(Obstet Gynecol. 2008;112:773-781).
Weight gain: Estrogen may initially cause weight gain due to water and sodium retention, but long-term weight gain isn't likely. Pills with androgenic activity sometimes stimulate appetite. If this is suspected, try a pill that is less androgenic.
Dysmenorrhea or PMS: may improve with extended or continuous-cycle pills such as Seasonale, Seasonique, Lybrel, as the hormone-free interval is shorter or absent.
Bloating: Consider Yasmin or Yaz for pt's as these contain drospirenone...a progestin that blocks aldosterone and has a mild diuretic effect.
Menstrual migraine: Extended or continuous-cycle pills can be helpful...by preventing the drop in estrogen that triggers migraines. But avoid using the pill in women who have migraines with aura because of an increased risk of stroke.
Mood: The emergence of mood symptoms is a common adverse event among OC users and accounts for as many as 20% of discontinuations. Nevertheless, some OC formulations can alleviate physical symptoms and mood complaints in patients with premenstrual syndrome (PMS) or premenstrual dysphoric disorder (PMDD). Rates of mood and anxiety disorders were similar for current and past OC users aaccording to a study on 118 women (mean age, 25)(Contraception 2009;79:50).
Indications for Estrogen Dominant: acne, alopecia, hirsutism, scanty periods, dec breast development, depression, early cycle spotting or break through bleeding, Monilia, DM. **-->** **-->** Enovid 10 **>** Enovid E > Enovid 5 > Brevicon/ Modicum > Ovulen/ Ortho Novum 1/80, Norinyl 1/80. Ortho Tri-Cyclin proven to dec acne.
Too much Estrogen: N/V/ bloating, fluid retention, mastalgia (breast tenderness), HTN, vascular H-A, chloasma, melasma, mucorrhea, fibroid growth, visual changes, late cycle spotting, irritability, fibrocystic breasts.
Too little estrogen: early or mid-cycle breakthrough bleeding, increased spotting, hypomenorrhea.
Indications for Progesterone Dominant: cervical erosion, fibroids, N/V, fibrocystic dz of the breast, dysmenorrhea, hypermenorrhea, unreliable pt, late cycle spotting **-->** Ovral/ Ortho Novum 10/ Norlestrin 10/ Lo Ovral **>>** Desogen/ Ortho-Cept/ Ortho Cyclin> Nordette/ Levlen/ Alesse. Mircette (by Organon) good for migraines/ dysmenorrhea, very low estrogen and short hormone free interval.
Too much progesterone: Breast tenderness, H-A, fatigue, depression, changes in mood, breast regression, oligomenorrhea, amenorrhea, early cycle spotting, acne, moniliasis, wt gain, inc appetite, insulin resistance, hirsutism, dec libido, regression of breast tissue and occasionally breast tenderness.
Too little progestins: late breakthrough bleeding.
Too much androgen: Increased appetite, weight gain, acne, oily skin, hirsutism, decreased libido, increased breast size, breast tenderness, increased LDL, decreased HDL.
***Some progestins have less androgenic effects and thus may improve hirsutism and acne, these include **Norgestimate** (NGM, includes Triphasic, Ortho-Cyclen, Ortho Tri-Cyclen also have NGMN = metabolite of NGM called **Norelgestromin**, found in the Ortho Evra patch, both types have favorable lipid profiles) and **Desogestrel** (DSG – but may give inc incidence of DVT's, includes Desogen, Ortho-Cept, Mircette, Cyclessa). **Drosopirenone** (DRSP, found in Yasmin) is a spironolactone analogue.
SE of too much androgens: increased appetite, weight gain, acne, oily skin, hirsutism, decreased libido, increased breast size, breast tenderness, increased LDL cholesterol, decreased HDL cholesterol.

Missed Pills: **Step #1:** determine how long it has been since the person missed taking the last pill.
• If one or more pills are missed in WEEK ONE, recommend taking one pill as soon as possible and then continue with the pack. Also recommend backup contraception for 7 days...and consider emergency contraception for unprotected sex in the past 5 days.
• If one or two pills are missed during WEEK TWO or THREE, recommend taking one pill ASAP and continuing the current pack. But tell women to skip the placebo pills and go straight to a new pack without the usual hormone-free interval.
• If three or more pills are missed in WEEK TWO or THREE, recommend the same routine...one pill ASAP, finish the active tabs, then start a new pack. In addition, recommend backup contraception for 7 days...and consider emergency contraception if a woman has unprotected sex before 7 consecutive active pills have been taken.
• For extended or continuous hormonal contraceptives, tell women not to worry about special precautions unless they miss more than 7 days... AFTER at least 21 days of continuous use.
If the time missed is <= 12 hours: (1) take the missed pill now, (2) take the remainder of the pills as usual.
If the time missed is >12 hours: take 2 qd X 2d, but use barrier method for rest of month. **Or**
Withdraw then restart fresh pack in 5 days. **Or**
(1) take the most recent missed pill now,
(2) discard any previous pills in the pack (going backward),
(3) count the number of pills left in the packet (going forward), after the most recent missed pill has been taken and the other pills have been discarded. If <7 pills present, then complete the packet and start the next packet the next day, without a break. If >= 7 pills present, then complete the pack and leave the usual 7 day break before starting the next packet.
(4) Use extra precautions for the next 7 days (condom, abstinence, etc.)
****Ref:** (Emergency postcoital contraception. NEJM 1997;337:1058-1064) (Combination Oral Contraceptives. NEJM 2003;349:1443-50)

Oral Contraceptive Pills (OCP):

Links: Monophasic | **Tri** | Bi | **Estro phasic** | Continuous | **Progestin-only pill** | **See** Oral Contraception |

Name: estrogen (**E** in mcg) content / Progestin (**P** in mg), active tab **color**(s). ****Genera 1/50 and 1/35 are available from Physicians Total Care and costs <$10/mo. Codes: V25.01 Contraceptive management. V25.41 is surveillance of previously prescribed OCP.

Phasic Pills: Combined oral contraceptives were first developed as monophasic pills; biphasic and triphasic pills were later developed in order to reduce dose-dependent adverse effects of the progestin. They were designed to more closely mimic fluctuations in estrogen and progesterone levels during the menstrual cycle. A Cochrane review was unable to determine if there is a difference in efficacy between monophasic and biphasic pills. Based on the results of a single qualified study, the reviewers found no major difference in bleeding pattern between a monophasic pill (Loestrin) and a biphasic pill (Ortho-Novum 10/11) (The Cochrane Library 2003;Issue 3)....a review compared biphasic and triphasic pills and found that cycle control was worse with a biphasic pill containing norethindrone (Ortho-Novum 10/11) than a triphasic pill containing levonorgestrel (Triphasil).....The reviewers concluded that the choice of progestin is more important than the phasic formulation.

Very Low Dose OCP's: Mircette, Alesse, Levret, Loestrin 1/20, Estrostep.

An ACOG statement says that a woman should be informed of any change in her contraceptive prescription (Obstetrics and Gynecology 2007;110:447-448) (whether for cost, insurance coverage, compliance, or any other reason, the pt should always be notified about the substitution to avoid confusion and poor compliance due to confused by new packaging, fear they have received the wrong pill, or lack confidence in generics).

Combination Monophasic:

Low-Dose:

Alesse, Aviane, Lessina, Levlite, Lutera: EE 20 mcg / Levonorgestrel 0.1 mg. Low estrogen; low progestin; low androgen. Low estrogen dose may cause more spotting and less margin of error for missed pills. Good choice to minimize risk of estrogen side effects like nausea, breast tenderness, vascular headache.

Junel 1/20, Junel Fe 1/20, Loestrin 1/20, Fe 1/20, Microgestin 1/20, Fe 1/20: EE 20 mcg / Norethindrone 1 mg. Low estrogen; high progestin; medium androgen. Low estrogen dose may cause more spotting and less margin of error for missed pills. Good choice to minimize risk of estrogen side effects like nausea, breast tenderness, vascular headache.

Levlen, Levora, Nordette, Portia: EE 30 mcg / Levonorgestrel 0.15 mg. Low estrogen; medium progestin; medium/high androgen. Good choice to minimize estrogen side effects like nausea, breast tenderness, vascular headache. Good choice to minimize spotting and/or breakthrough bleeding.

Cryselle, Lo/Ovral, Low-Ogestrel: EE 30 mcg / Norgestrel 0.3 mg. Low estrogen; medium progestin; medium/high androgen. Good choice to minimize estrogen side effects like nausea, breast tenderness, vascular headache and to minimize spotting and/or breakthrough bleeding.

Junel 1.5/30, Junel Fe 1.5/30, Loestrin Fe 1.5/30, Microgestin 1.5/30, Microgestin Fe 1.5/30: EE 30 mcg / Norethindrone acetate 1.5 mg. Low estrogen; high progestin; high androgen. Good choice to minimize estrogen side effects like nausea, breast tenderness, vascular headache.

Apri, Desogen, Ortho-Cept, Reclipsen, Solia: EE 30 mcg / Desogestrel 0.15 mg. Low estrogen; high progestin; low androgen. Increased risk of DVT with desogestrel over other progestins (controversial data). Good choice to minimize spotting and/or breakthrough bleeding; and to minimize androgenic effects. Has favorable lipid profile.

Yasmin: EE 30 mcg / Drospirenone 3 mg. Low estrogen; progestin potency unclear; anti-androgenic and antimineralocorticoid activity. Does not appear to cause cyclic fluid retention. May be good choice for women with PMS, premenstrual dysphoric disorder, acne, hirsutism, or PCOS. Can increase potassium: Avoid in renal/hepatic dysfunction or renal insufficiency. Check potassium during first cycle if another potassium-sparing drug (NSAID, ACE inhibitor, angiotensin receptor blocker, potassium-sparing diuretic; aldosterone antagonist) is given.

Demulen 1/35, Kelnor 1/35, Zovia 1/35: EE 35 mcg / Ethynodiol diacetate 1 mg. Medium estrogen; high progestin; low androgen. Good choice to minimize androgenic effects.

Ortho-Cyclen, Mononessa, Previfem, Sprintec: EE 35 mcg / Norgestimate 0.25 mg. Medium estrogen; low progestin; low androgen. Good choice to minimize spotting and/or breakthrough bleeding and to minimize androgenic effects. Has favorable lipid profile.

Necon 1/50, Norinyl 1 + 50, Ortho-Novum 1/50: Mestranol 50 mcg / Norethindrone 1 mg. Medium estrogen; medium progestin; medium androgen.

Ovcon-35, Femcon Fe chewable: EE 35 mcg / Norethindrone 0.4 mg; total of 8.4 mg/cycle.6 Medium estrogen; low progestin; low androgen. Good choice to minimize androgenic effects. Has favorable lipid profile.

Brevicon, Modicon, Necon 0.5/35, Nortrel 0.5/35: EE 35 mcg / Norethindrone 0.5 mg; total of 10.5 mg/cycle. Medium estrogen; low progestin; low androgen. Good choice to minimize androgenic effects. Has favorable lipid profile.

Necon 1/35, Norethin 1/35, Norinyl 1+35, Nortrel 1/35, Ortho-Novum 1/35: EE 35 mcg / Norethindrone 1 mg; total of 21 mg/cycle. Medium estrogen; medium/high progestin; medium androgen.

High Dose:

Avoid pills with more than 35 mcg of ethinyl estradiol in hypertensive women; lower doses of estrogen can be used in non-smoking women age 35 years or younger who have well-controlled hypertension, and no evidence of end-organ vascular disease. Also avoid pills with more than 35 mcg of ethinyl estradiol in women with controlled hyperlipidemia; use alternative contraceptive if LDL cholesterol >160 mg/dL or multiple additional risk factors for coronary heart disease.

Ovcon-50: EE 50 mcg / Norethindrone 1 mg. High estrogen; medium progestin; medium androgen.

Ogestrel 0.5/50: EE 50 mcg / Norgestrel 0.5 mg. High estrogen; high progestin; high androgen.

Demulen 1/50, Zovia: EE 50 mcg / Ethynodiol diacetate 1 mg. High estrogen; high progestin; medium/high androgen.

(Ethinyl Estradiol (EE) / Norethindrone):

Loestrin 1/20: 20 (E) /1 (P),White.

Loestrin 1/20: 20 (E) /1 (P),White.

Loestrin 1.5/30: 30 (E) /1.5 (P),Green.

Loestrin Fe 1.5/30: 30 (E) /1.5 (P),Green.

Loestrin 24 Fe 1-mg (E) / 0.02-mg (P) 24 days of active hormonal therapy and 4 days of iron-containing placebo pills.

Brevicon: 35 (E) /0.5 (P),Blue.

Norlestrin 1/35: 35 (E) /1 (P),White.
Genera, Nelova 1/35: 35 (E) /1 (P),same.
Norinyl 1+35: 35 (E) /1 (P),Yellow-green.
Ortho Novum 1/35: 35 (E) /1 (P),Peach.
Ovcon 35: 35 (E) /0.4 (P),Peach. See below for chewable form.
Ovcon 50: 50 (E) /1 (P),Yellow.
(EE / Levonorgestrel):
Alesse: 20 (E) /0.1 (P),Pink.
Levlite: 20 (E) /0.1 (P),Pink.
Levlen: 30 (E) /0.15 (P),Orange.
Nordette: 30 (E) /0.15 (P),Orange.
(EE / Norgestrel):
Lo/ Ovral: 30 (E) /0.3 (P),White.
Ovral: 50 (E) /0.5 (P),White.
Low-Ogestrel: 33ug EE/0.3 norgestrel
(EE / Ethynodiol diacetate):
Demulen 1/35: 35 (E) /1 (P),White.
Demulen 1/50: 50 (E) /1 (P),White.
(Mestranol / Norethindrone):
Genera, Nelova 1/50: 50 (E) /1 (P), same.
Norinyl 1+50: 50 (E) /1 (P),White.
Ortho Novum 1/50: 50 (E) /1 (P),Yellow.
(EE / Desogestrel):
Desogen: 30 (E) /0.15 (P),White.
Ortho-Cept: 30 (E) /0.15 (P),Orange.
(EE / Norgestimate):
Ortho-Cyclen: 35 (E) /0.25 (P), Blue.
(EE / Drospirenone): The risk for thromboembolism is similar in young, relatively healthy women starting ethinylestradiol 0.03 mg/drospirenone 3 mg vs other oral contraceptives according to a study (Obstet Gynecol. 2007;110:587-593).
Yasmin (EE 30 mcg/ Drospirenone 3mg): Low-dose, the progesterone component called drospirenone is a spironolactone analogue with both anti-mineralocorticoid & anti-androgenic activity. Helps counteract fluid retention, may be good for acne. May inc serum K, especially if on NSAIDs or ACEi/ ARB, contra if renal, adrenal or hepatic insufficiency. May ease sx's of PMDD. Drospirenone (DRSP): possesses progestogenic, antiandrogenic and anti-mineralocorticoid activity. It increases sex hormone-binding globulin (SHBG) 3-4-fold. Being structurally related, a 3mg dose is equivalent to ~25mg of spironolactone (ck K 3wks after starting if on any other potassium altering meds). Some initial wt loss due to fluid loss, but does not reduce body fat or mass. Due to its favorable influence on bone metabolism, an OC that contains the progestogen drospirenone may be of particular benefit in women with risk factors for osteopenia and osteoporosis who require oral contraception (Obstet Gynecol 2005;105:53-60). There seems to be a higher thrombosis risk with drospirenone and desogestrel compared to levonorgestrel and norethindrone, but the increased risk is very small...about one more clot for every 7400 women taking drospirenone or desogestrel compared to the others (Prescriber's Letter 2009;25(10):251001).
YAZ (EE 20 mcg / Drospirenone 3mg: Each pack of YAZ contains 24 active hormone pills and 4 inactive pills (24/4) instead of the usual 21/7 OC dosing schedule. The first pill to combine 20 mcg of ethinyl estradiol with drospirenone, FDA approved in 3-06. May be used for tx of the emotional and physical sx's of premenstrual dysphoric d/o (PMDD). The 24-day active hormone pill regimen of YAZ combined with the 30-hour half life of drospirenone results in less hormonal fluctuation between cycles, compared to traditional OC's with 21 days of active pills and 7 days of placebo.
Ovcon 35 Chewable: a spearmint-flavored contraceptive tablet that can be chewed and swallowed. It contains a progestin (norethindrone) and an estrogen (ethinyl estradiol). The directions for use tell women that the pill may be swallowed whole or chewed and swallowed. If the pill is chewed and then swallowed, the woman should drink a full glass (8 oz) of liquid immediately afterwards so that the full dose of medication reaches the stomach and no residue is left in the mouth. Each package contains 21 round, white tablets, with norethindrone and ethinyl estradiol followed by seven reminder green (inactive) tablets to complete a 4-week cycle. **Ovcon 35:** 35 (E) /0.4 (P).

Biphasic:

(EE / Norethindrone):
Gencept 10/11, Necon 10/11, Ortho-Novum 10/11: EE 35 mcg / Norethindrone 0.5 mg x 10 days, 1 mg x 11 days. High estrogen; medium progestin; low/medium androgen. Poor cycle control compared with levonorgestrel triphasic pill (Triphasil). Ortho-Novum 10/11 with 10 white,11peach. Jenest 28 with 7 wht,14 peach.
(EE/ Desogestrel):
Mircette, Kariva: 20/ 10 (E) /0.15 / - (P),21 white, 5 yellow. EE 20 mcg x 21 days, placebo x 2 days, 10 mcg x 5 days / Desogestrel 0.15 mg x 21 days. Low estrogen; high progestin; low androgen. Shorter hormone-free interval may help menstrual migraine, dysmenorrhea, PMS. Increased risk of DVT with desogestrel over other progestins (controversial data).

Triphasic:

(EE/ Norethindrone)
Ortho Novum 7/7/7, Nortrel 7/7/7, Necon 7/7/7: 35/ 35/ 35 (E) /0.5/ -.75/ 1 (P),7 white, 7 light-peach, 7 peach. EE 35 mcg x 21 days / Norethindrone 0.5 mg x 7 days, 0.75 mg x 7 days, 1 mg x 7 days. Total of 15.75 mg/cycle. Medium estrogen; medium progestin; low/medium androgen.
Tri-Norinyl, Aranelle, Leena: 35/ 35/ 35 (E) /0.5/ 1/ 0.5 (P),7 blue, 9 green,5 blue. EE 35 mcg x 21 days / Norethindrone 0.5 mg x 7 days, 1 mg x 9 days, 0.5 mg x 5 days. Total of 15 mg/cycle. Medium estrogen; medium progestin; low/medium androgen.

Estrostep Fe: EE 20 mcg x 5 days, 30 mcg x 7 days, 35 mcg x 9 days / Norethindrone 1 mg x 21 days. Low estrogen; high progestin; medium androgen. FDA-labeled for acne. Good choice to minimize estrogen side effects like nausea, breast tenderness, vascular headache; and to minimize spotting and/or breakthrough bleeding.

(EE/ Norgestimate):

Ortho Tri-Cyclen, Trinessa, Tri-Previfem, Tri-Sprintec: 35/ 35/ 35 (E) /0.18/ .215/ .25 (P),7 white, 7 light blue, 7 blue. EE 35 mcg x 21 days / Norgestimate 0.18 mg x 7 days, 0.215 mg x 7 days, 0.25 mg x 7 days. Medium estrogen; low progestin; low androgen. FDA-labeled for treatment of acne.

Ortho Tri-Cyclen Lo: EE 25 mcg x 21 days / Norgestimate 0.18 mg x 7 days, 0.215 mg x 7 days,0.25 mg x 7 days. Low estrogen; low progestin; low androgen. Good choice to minimize spotting, breakthrough bleeding, and androgenic effects. Has favorable lipid profile.

(EE/ Levonorgestrel):

Enpresse, Tri-Levlen, Triphasil, Trivora: 30/ 40/ 30 (E) /.05/ .075/ .125 (P),6 brown, 5 white, 10 yellow. EE 30 mcg x 6 days, 40 mcg x 5 days, 30 mcg x 10 days / Levonorgestrel 0.05 mg x 6 days, 0.075 mg x 5 days, 0.125 mg x 10 days. Total of 1.925 mg/cycle. Medium estrogen; low progestin; low/medium androgen. Better cycle control than with norethindrone biphasic pill (Ortho-Novum 10/11).

(EE / Desogestrel):

Cyclessa, Cesia, Velivet: 25 mcg/ 25/ 25 (E), 0.1mg/ 0.125/ 0.15 (P). Low total cycle estrogen. EE 25 mcg x 21 days / Desogestrel 0.1 mg x 7 days, 0.125 mg x 7 days, 0.15 mg x 7 days. Low estrogen; high progestin; low androgen. Increased risk of DVT with desogestrel over other progestins (controversial data). Better cycle control and less weight gain than Ortho-Novum 7/7/7.

Estro phasic:

(EE/ Norethindrone):

Estrostep Fe: 20/ 30/ 35 (E)/ 1/ 1/ 1 (P),5 white triangle, 7 squares, 9 round. Approved for tx of acne in age >15yo.

Androgenicity of Progestins Used in Oral Contraceptives:

Level of Androgenicity. Progestin --> Brand Name of Products.

High: Norgestrel --> LoOvral.

Levonorgestrel --> Alesse, Levlite, Nordette, Trilevlen, Tri-Phasil.

Moderate: Norethindrone --> Genora 1/35, Norinyl 1/35, OrthoNovum 1/35, Ovcon 35.

Norethindrone acetate --> Loestrin 1/20, Loestrin 1.5/35.

Low: Ethynodiol diacetate --> Demulen 1/35.

Norgestimate --> Ortho-TriCyclen, Ortho-TriCyclen Lo.

Desogestrel --> Desogen, Ortho-Cept. Drosperinone --> Yasmin.

Continuous / Extended Cycle OCP's:

See Below for: Seasonale: **Seasonique:** Yaz: **Loestrin-24 FE:** Lybrel:

Shortening the pill-free interval: The pill suppresses endometrial thickening so monthly bleeding is not necessary to slough it off. Longer cycles may increase the contraceptive efficacy. Beneficial especially if having sx's during this time such as migraines, dysmenorrhea, menorrhagia, PMS sx's, also dec endometrial Ca and anemia. It may be more expensive for the meds, but cheaper than tampons/ pads, Motrin and Imitrex. Continuous combined hormone therapy with ethinyl estradiol @0.02 mg and desogestrel @0.15 mg/d may be appropriate for women with endometriosis pain not controlled by cyclic therapy as 80% are satisfied or very satisfied with this therapy (Fertil Steril. 2003;80:560-563). Continuous tx with a levonorgestrel/ethinyl estradiol (LNG/EE, 90/20mcg PO 365 d/yr) oral contraceptive can safely eliminate menstrual cycles in 79% of women (Contraception 2006; Dec 13). The cyclic administration of the OCP was based on dogma to provide a "natural menstrual cycle" rather than physiology, but the pill-free interval has been associated with rebound and increased ovarian activity, which may predispose to user and contraceptive method failure. Continuous vs cyclic OC did not reduce bleeding days for a 168-day observation period but provided greater ovarian and endometrial suppression and improved symptoms, according to the results of a RCT (J Clin Endocrinol Metab. Published online December 4, 2007). Continuous treatment with oral contraceptives may ease pain, heavy bleeding, and other menstrual symptoms compared with standard cyclic contraceptives (J Clin Endocrinol Metab 2008;93:420-429)....However, continuous suppression of the ovaries did not reduce the overall days of bleeding (mean 31.5 versus 35.1 days), likely because of an increase in unpredictable breakthrough bleeding with continuous contraception because the pill makes the lining of the uterus thinner and more fragile.

Bicycling and Tri cycling (three 21 day packs in a row) where 2-3 cycles of active pills taken consecutively and the placebo pills are discarded. After 6-9 wks, the pt stops taking active pills for 4 days to allow for withdrawal bleed, then resumes the cycling. Another option is to shorten the pill-free interval to only 4 days/mo, best done by having the pt resume pills on the 1st day of bleeding (3-4 days after stopping). Best to use a monophasic pill to reduce the chance of spotting. An alternative is Mircette has 3wks of active meds and only 2 days of placebo to reduce the estrogen withdrawal sx".

Seasonale: (levonorgestrel plus ethinyl estradiol 0.15-mg/0.03-mg tablets) A 91-day oral contraceptive regimen. Tablets containing the active hormones are taken for 12 weeks (84 days), followed by 1 week (7 days) of placebo tablets. Contains a progestin (levonorgestrel, 0.15 mg) and an estrogen (ethinyl estradiol, 0.03 mg) (similar to Levlen, Levora and Nordette). The number of expected menstrual periods that a woman usually experiences is reduced from about once a month to about once every 3 mo's. In the first few cycles of use, may see more unplanned bleeding and spotting between the expected menstrual periods than women taking a conventional 28-day cycle oral contraceptive. Cost ~$1 a pill, which is comparable to other branded oral contraceptives. Generic version approved by FDA in 9/06. They accomplish the same thing with other monophasic pills. Advise them to take the active pills for 12 weeks straight, then skip a week before starting another cycle.

Seasonique: levonorgestrel/ethinyl estradiol tablets 0.15 mg/0.03 mg and ethinyl estradiol tablets 0.01 mg. Limits the number of menstrual periods from 12 to 4 a year. With Seasonale, women take a placebo for 7 days, while for Seasonique, women would take a low-dose estrogen (10 mcg of ethinyl estradiol) for 7 days. The difference could mean less bleeding for women on Seasonique. Worth a try for women who get menstrual migraines as estrogen during the placebo days to prevent migraines.

Yaz: EE 20 mcg / Drospirenone 3 mg. FDA-approved for premenstrual dysphoric disorder and moderate acne. Follows 24/4 day cycle and contains ingredients of Yasmin, but with lower estrogen dose (20 mcg EE compared to 30 mcg EE in Yasmin). Also see Yasmin comments.

Loestrin-24 FE: EE 20 mcg / Norethindrone 1 mg. Low estrogen; high progestin; medium androgen. Low estrogen dose may cause more spotting and less margin of error for missed pills. Good choice to minimize risk of estrogen side effects like nausea, breast tenderness, vascular headache. 24/4 day cycle combination may be helpful for women wanting to stay on a 28-day cycle, but minimize duration of withdrawal bleeding and menstrual-related symptoms.
Lybrel: 90 mcg / 20 mcg of levonorgestrel / ethinyl estradiol. Take qd without a placebo interval, resulting in the intended temporary interruption of menses. The potential benefit of amenorrhea should be weighed against the inconvenience of unscheduled bleeding or spotting. Up to 50% of women have 7 or more days of bleeding or spotting during the first 3 months, but this decreases with continued use. The approval was based on data from two 1-year clinical studies (n = 2457), showing that 59% of women achieved amenorrhea without bleeding or spotting; 20% experienced spotting not requiring sanitary protection; and 21% of women required sanitary protection due to breakthrough bleeding.
Data from 187 women (mean age, 30.4 years) who took Lybrel continuously for a median of 364 days found that within the first 30 days after stopping Lybrel, 38.5% of women reported a return to menses or became pregnant, which increased to 92.5% within 60 days and to 99% by 90 days (Am Coll Obst Gyne annual clinical meeting. May 2006). As with other oral contraceptives, the continuous-use product is linked to increased risks for blood clots, myocardial infarction, and stroke. Other monophasic pills can be used continuously too…such as Alesse (very similar) which has less expensive generics.

Progestin only pill (POP):

Have dec DVT risk, H-A, HTN and less depression than regular OCP's. No estrogen risks/ SE or benefits. However, they tend to cause more irregular bleeding and thus are generally reserved for **breast-feeding** women and those whom estrogen is contraindicated or poorly tolerated.
2 kinds in USA:
Norethindrone (Micronor, Nor-QD, Camila, Errin, Jolivette, Nora-BE): 0.35mg qd, lime green tabs.
Norgestrel (Ovrette): 0.075mg qd, yellow.
If pt recently started taking the POP and is having abnormal uterine bleeding after 3mo give a trial of Ibuprofen 600-800mg TID for 1-2 weeks. If bleeding persists, either change to combination OCP or MPA injections or Norplant.

Emergency (Postcoital) Contraception (EC):

Links: Info & Telephone Triage | **Rx's used** | Other / Abortion | **Intrauterine Device (IUD)** |

Best to **give Rx before it is needed** (especially if use the barrier method) or over the phone as best to take 1st dose ASAP ("morning-after pill").
Codes: V25.03, which denotes rncounter for EC counseling and Rx. A single act of unprotected intercourse occurring 1-2 days prior to ovulation is associated with an 8% chance of pregnancy in all women, yet in age 19-26yo the chance may be as high as 50%. EC will reduce the risk of pregnancy to 1-2% (Emergency contraception. NEJM 2003;349:1830-5). Women given easy access to EC use it more than do women with restricted access, however, EC availability did not change sexual habits or use of other contraceptive methods (JAMA. 2005;293:54-62). www.not-2-late.com.
States that allow pharmacist to provide: As of 12/08: AK, CA, HW, ME, MA, NH, NM, VT, WA.
Phone Rx if "Yes" to the following 3 q's: Unprotected sex or problem with birth control during past 3 days (max 120 hrs). LMP < 4wks ago, normal length & timing. This method does not cause "abortion" as can't interrupt an established pregnancy, just prevents implantation of the egg. Adolescents account for 10% of sexually active women age 15-44, but have 26% of all abortions and 77% of all unintended pregnancies. No true contraindications, Ok even if migraine, breast Ca, DVT, severe liver dz. Check hCG as may already be pregnant. Effective up to 72hr post-coital, won't inhibit established pregnancy or prevent STD (continue to use ongoing contraception). Advise that tx can fail (4% chance of pregnancy), lowers the risk of pregnancy by 75%, but since only a 20% chance anyway, now 4% **-->** check pregnancy test if period delayed >3wks.
After 72hr: insertion of IUD (ParaGard T-380A, $359) can be performed, up to 7days later and is 99% effective.
Rx: Helps to use Phenergan 25mg prior to the doses to avoid redosing. Pretreatment with the inexpensive antiemetic metoclopramide 10mg BID lessens nausea and cramping associated with high-dose estrogen/progestin emergency contraception (Am J Obstet Gynecol 2003;188:330-333). Pregnancy rates are ~1.77% with two-dose Levogestrel, 1.62% with all levonorgestrel, 1.55% with Mifepristone and 1.47% with single-dose levonorgestrel (Lancet 2002;360:1807).
1. Levonorgestrel 0.75mg (aka "Plan B"): = **Progestin Mini Pill:** 1 tab PO, repeat in 12hr is more effective and has dec nausea, ~$24. 1.1% pregnancy rate Vs 3.2% with standard 4pills. **Or** Can take 1.5mg (2 tabs at once) = 40 Ovrette pills x 1 (single dose), same efficacy, improved compliance (Lancet 2002;360:1803-10). **Or** Each dose = 20 tabs of Ovrette (Norgestrel) to get 0.75mg. Available from the Women's Capital Corp (800-330-1271), $21. In 4/09, the FDA has agreed to allow 17-year-olds to purchase levonorgestrel (Plan B) birth control pills over-the-counter (rather than behind the counter) due to a ruling from the U.S. District Court for the Eastern District of New York.
Next Choice: a generic version of the two-dose levonorgestrel ECP product. Available to women 17 and older without prescription. Priced about $10 less.
Plan B One-Step: a single tablet that contains 1.5 mg of levonorgestrel. In 8/09, the FDA has approved this as a single-dose formulation of the emergency contraceptive pill (ECP). Available without prescription (but behind the counter) for women 17 and older and by prescription only for women younger than 17. Cost US$45 to $50. The original EC product, Plan B, consists of two 0.75-mg levonorgestrel tablets taken 12 hours apart. Like its predecessor.
2. The "Preven Kit": has 4 tabs: 0.05mg ethinyl estradiol (50ug) + 0.25mg Levonorgestrel take 12hr apart and Urine pregnancy test. If vomit within 1hr after dose need to repeat, consider prophylactic **anti-nausea med** 1hr before (20% incidence). **-->** 75% dec in pregnancies. ~$20. In 2004, there are six states (California, Alaska, Washington, New Mexico, Hawaii, and Maine) where it can be obtained without prescription. **Or**
3. Two doses 12hr apart of OCP's: OCP's used | **Or**
4. Ethynyl Estradiol (EE): 2.5 mcg BID X 5d. High dose estrogen is only licensed in the Netherlands. Can be used up to 72hr after intercourse.
Or

5. Mifepristone (RU 486) 10mg X1 dose (this dose not available in the US). (Lancet 2002;360:1803-10) Mifepristone (10 mg) is as effective as levonorgestrel (two doses of 750 mcg given 12 hrs apart) for emergency contraception (Obstet Gynecol. 2004;104:1307-1313).

OCP's used for Emergency Contraception:

…….END DEMO of this chapter.

Procedures in Medicine:

Links in alphabetical order: A-B: **C:** D-E: **F-K:** L-O: **P-Q:** R-S: **T-Z:** See Studies & On-Call & Sx's |

Unretrieved device fragments (UDFs): a medical device fragment that has separated unintentionally and remains in the pt after a procedure. Patients are often unaware that a UDF may be the cause of their difficulty. Fragments have caused local tissue reaction, infection, perforation and obstruction of blood vessels, and even death. Contributing factors may include biocompatibility of the device materials, location and potential migration of the fragment, and individual pt anatomy, according to an alert sent from MedWatch in 1/08.

A:

Abscess I&D:
Abd X-ray Analysis:
ACLS / BLS:
Acid Fast Stain:
ACTH Stimulation Test:
Acute Urinary Retention:
Alcohol withdrawal:
Allergy: Skin Testing:
Alternating Cover Test:
Ambulatory BP Monitoring:
Anesthesia - topical and local |
Anesthesia – regional blocks |
Ankle Dislocation:
Ankle Brachial Index (ABI)
Anoscopy:
Aortic Balloon Pump:
Apt test:
Arterial Blood Gas (ABG):
Arterial Lines
Arthrocentesis:
ATLS:
Automated External Defibrillator (AED):
Baby Delivery |
Bartholin Gland I&D:
Biceps Tendonitis:
Biological & Chemical Weapons:
Blood Cx:
Bladder Tap:
Bone Marrow (BM) Sampling:
Brain Death Eval
Breast Exam:
Botox Injections:
Bronchoscopy |

C:

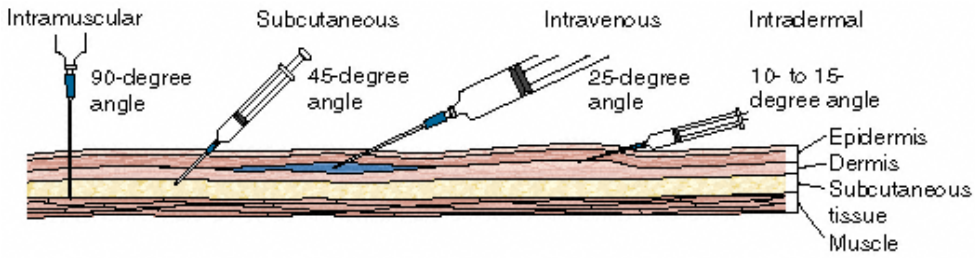
Caloric Testing - Reflex Eye Mov't |
Canalith Repositioning:
Cardiac "Clearance":
Cardiac Testing
Cardioversion:
Carpal Tunnel Injection:
Cauliflower Ear:
Central Lines:
Cerumen
Chalazion Excision:
Chest Tube:
Circumcision |
CXR Analysis:
Colposcopy:
Compartment Pressure Measuring:
Costochondritis:
Cricothyroidotomy:
Cryosurgery:
C-spine X-rays
Culdocentesis:
Curettage & Electrodesiccation:
Cystourethrography |

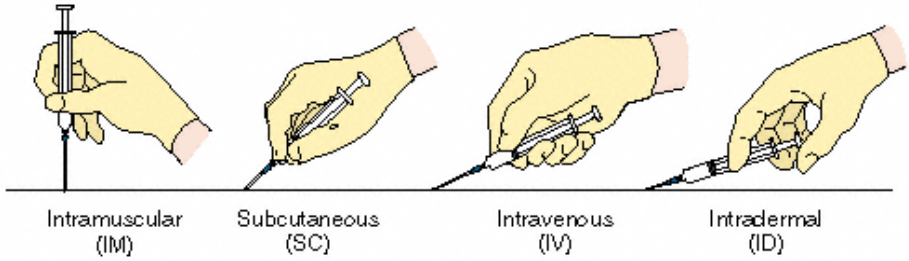
D:
D&C:
Defibrillation:
Delivery of a Baby |
De Quervain's Wrist Injection:
Dermatologic:
Dermabond:
Dermatophyte Test Medium (DTM):
Dermoscopy |
Dexamethasone Suppression Test:
Dialysis:
Diaphragm Fitting:
Digital Mucous Cysts |
Dilation - eye |
Dislocated Knee:
Dislocated Mandible:
Ear Fb:
Ear Lac:
ECG Interpretation:
EGD |
Elbow Dislocation:
Electrosurgery:
Emergency Airways:
Endometrial Bx
Enteral Feeding:
Epidermal cyst excision:
Epistaxis:
Esophageal Fb:
Evacuation of Thrombosed Hemorrhoid:
Excisional Bx:
Exercise (Treadmill) Stress Test (EST):
Extensor Tendon Repair:
Extubation:
Eyelid Fb:
Eye irrigation |
Eye Patching |
F:
Fecal Occult Blood (FOB):
Feeding Tubes:
Femoral Artery Cath:
Finger-Tip Repair:
Fish Hooks:
Flexible Sigmoidoscopy:
Fluorescein Staining - eye |
FNA Breast:
FNA Thyroid:
Foley - urinary catheter:
Forearm Ischemic Test:
Foreign Bodies:
Four Cup Test (prostatitis):
Ganglion Cyst:
Grams Stain:
Heat Stroke:
Hepatojugular Reflux Test (HJR):
Hernia, inguinal reduction:
Hip Bursitis & Injections:
Hypodermoclysis:
Infection Control
Infertility Eval:
Ingrown Nail |
Inguinal Hernia Reduction:
Injections:
Instruments and Sutures:
Insulin Drips
Intra-aortic Balloon Pump (IABC):
Intercostal Anesthesia:
Intraosseous infusion |
Intubation:
Joint Aspiration & Injection:
Jugular Venous Distension (JVD):

Kidney Biopsy:
Kleihauer-Betke Test:
Knee Aspiration:
Knee injection:
KOH:
L:
Laryngeal Mask Airway:
Lensometer:
Lip Laceration:
Lipoma Removal:
Liver Bx:
Lumbar Puncture:
Maddox Rod Test:
Mechanical Ventilation:
Mental Status Exam:
Myocardial Perfusion Study:
Nail Bed Bx |
Nasal Fb:
Nasopharyngeal swab |
Nasogastric Tube (NGT):
Nasolaryngoscopy
Needlestick Eval:
Nerve Blocks |
Nursemaids Elbow:
Olecranon Bursa Aspiration:
Oropharyngeal Fb:
Osmotic Fragility Test:
P:
Pacer's Transvenous & Transthoracic:
PALS:
Pap Smear:
Paracentesis-abd:
Paraphimosis Reduction:
Peak Flow:
Pelvic Exam:
Penile Nerve Block:
Pericardiocentesis:
Perineal Laceration:
Peri-operative Medical Management:
Peripheral Parenteral Nutrition (PPN):
Peripheral Smears:
Peritoneal Lavage:
Peritonsillar Abscess: .0
Pilonidal Abscess:
Plantar Fasciitis:
Pleurodesis:
Peri-operative Clearance:
Pessary Fitting:
Potassium Hydroxide (KOH):
PPD Test:
Priapism:
Pulmonary Artery Cath:
Pulmonary Function Testing (PFT's)
Pulse Pressure:
Punch Bx:
R:
Radiology |
Rape:
Rectal & Vaginal Fb:
Refraction:
Retrograde Urethrogram/ Cystogram:
Ring Removal:
Schiotz Tonometry:
Sclerotherapy - skin |
Screening PPD:
Sedation:
Semen analysis |
Sengstaken-Blakemore Tube:
Sexual Assault Eval:
Shave Bx:

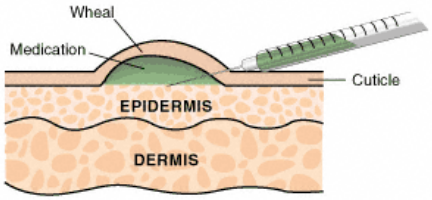
Shoulder Reduction Techniques:
Skin cyst excision:
Skin Grafts |
Skin Testing:
Slit Lamp:
Smears |
Spirometry
Splinting:
Sputum G-stain Analysis |
Steroid Injection to joints:
Stress Management:
Stool Osmotic gap:
Subacromial Injection:
Subungual Splinter:
Subungual Hematoma:
Suturing Techniques:
Swallowed Fb:
T:
Tenckhoff Catheter Placement:
Tendon Repair:
Terminal Pt & Withdrawing Care:
The Slit Lamp Exam (SLE):
Thoracentesis:
Thoracotomy:
Thrombolytics in AMI:
Tick Removal |
Tonometry:
Total Parenteral Nutrition (TPN):
Toxic Exposure Eval:
Tracheostomy Placement and Care:
Transvenous Pacer:
Trichogram
Trigger Point Injections:
Tympanocentesis:
Tympanometry
Tzanck Prep:
Umbilical Lines |
Unna Boot:
Urethral Swabs |
Urinary Catheter:
Urinalysis & Sediment:
Vagal Maneuvers:
Valsalva Maneuver:
Varicose Veins:
Vascular Access:
Vasectomy:
Venipuncture |
Venous Waveforms:
Vestibular Rehab
Violent Pt's:
Viscosupplementation of Knee:
Wart Tx:
Wedge Resection of Toenail:
Wound Care:
Wound Débride:
Wound Dressings:
Woods Lamp:
Wright Stain:

Typical Injections:





ID (intradermal): Clean skin with alcohol. 0.1ml into the upper layer of the skin, just beneath the epidermis. Used with PPD's.



IM (intramuscular):

Indications: Immunizations and other medications.
Complications: Bleeding, infection, allergic reaction, nerve injury.
Procedure: Clean skin with alcohol. Pinch muscle with free hand and insert 1-inch, 23-or 25-gauge needle until the hub is flush with the skin surface. For deltoid and ventral gluteal muscles, the needle should be perpendicular to the skin. For the anterolateral thigh, the needle should be 45 degrees to the long axis of the thigh. Aspirate for blood, then inject medication. The maximum volume 5 ml in the gluteal region & 2 ml in the deltoid of the arm. Have pt lean forward with the elbows on the exam table, feet slightly wider than the shoulder width apart and toes pointed as far inward as comfortable. Aim for upper outer quadrant of one of the butt cheeks. To find the gluteal site, form a triangle by placing your index finger on the anterior iliac spine and your middle finger on the most superior aspect of the iliac crest. The injection should occur in the middle of the triangle formed by the two fingers and the iliac crest.
A CT study of 100 consecutive pt's found that substantial number of pt's have enough fat in their gluteal region to make it unlikely that a typical needle will reach muscle (BMJ 2006;332:637-8).....injections into fat, instead of muscle, could have implications for absorption and the clinical effect of some medications....depth of adipose tissue at the ventrogluteal site ranged from 2.5-62.6 mm (mean, 19 mm). The maximum penetrating depth of commonly used long needles is 35mm. When receive IM injections in the buttocks via 23 gauge needle that was 3 cm, most of the dosage may remain in SQ fat.
Children use 1" 23-25g if infant give @ ant-lateral thigh (ALT = vastus lateralis muscle) if child give @ ALT or upper arm (deltoid). Max volume to be injected is 0.5 mL in a small infant, 1 mL in an older infant, 2 mL in a school-age child, and 3 mL in an adolescent. Avoid IM injections in a pt with a bleeding d/o or thrombocytopenia.

SC (Subcutaneous):

Indications: Immunizations and other medications.
Complications: Bleeding, infection, allergic reaction, lipohypertrophy/lipoatrophy after repeated injections.
Procedure: Clean skin with alcohol. 0.5ml-2.0ml into the outer surface of the upper arm, anterior surface of the thigh, abdomen or buttock. Vaccine in child use ½ - 5/8" 25-27g @ outer upper thigh or upper arm. Enter the subcutaneous layer at a 45-degree angle to the skin. Aspirate for blood, then inject medication.

Capillary Puncture:

Capillary blood is preferred for a peripheral blood smear and can also be used for other hematology studies. Indicated for blood sampling for lab studies unaffected by hemolysis.
Site: Obtain capillary blood from fingertips or earlobes (adults) or from the great toe or heel (infants).
Complications: Infection, bleeding, osteomyelitis.
Procedure: Do not squeeze the site to obtain blood because this alters blood composition and invalidates test values.
Step #1: Warm heel or finger or placing it in a dependent position may facilitate specimen retrieval. Clean with alcohol.
Step #2: Puncture finger using a lancet on the palmar lateral surface of the finger near the tip. Wipe away the first drop of blood, then collect the sample using a capillary tube or container. Puncture heel using a lancet on the lateral part of the heel, avoiding the posterior area. Alternate between squeezing blood from the leg toward the heel (or from the hand toward the finger) and then

releasing the pressure for several seconds. Earlobe sticks hurt < lateral thumb (10 fingers, 20 sites, minimize pain by pushing with thumb to create a pad), which hurts < the pad of the fingers.
Aftercare: Apply small dressing or adhesive strip to site. Evaluate puncture site for bleeding or oozing. Apply compression or pressure to the site if it continues to bleed.

Medications:

Links: ACLS | **Acne** | ADHD | **Allergy** | Alzheimer's | **Anxiety D/o & Benzo's** | AOM | **Antibiotics** | Antifungals | Antihistamine | **Antiarrhythmics** | Anitemetics | **Antiplatelet** | Antipsychotics | **Arthritis** | Aspirin (ASA) | **Asthma** | Benzo's | **Bladder Relaxants** | Calcium | **Cardiology** | Chemo | Clopidogrel (Plavix) | **Colchicine** | **Constipation** | Contraception | **COPD** | **Corticosteroids** | Cough | **Coumadin** | Decongestants | Depression | **Derm** | Diarrhea | **Diabetic** | **Diuretics** | DMARDs | **Endocarditis** | ENT | **Epogen** | Erectile | **Geriatric** | Gastro (GI) | **Gout** | H-A | **Hemorrhoids** | Heparins | **Hepatic encephalopathy** | Herbs | **HIV** | HTN | **Incontinence** | Influenza | IVIG | **Insomnia** | **Insulin** | Iron | **Lipid d/o** | **Lithium** | Joint steroids | **LMWH** | Magnesium | **Menopausal** | Mucolytics | **Muscle Relaxer** | Narcotics | **Nausea & Vomiting** | Nitro | **NSAIDs** | Obesity | **Ophtho** | Osteoporosis | **Parkinson's** | Pain | **PEDIATRIC** | Pharmacotherapy | **Phosphate** | Pneumonia | **Potassium (KCL)** | Prostate | **Proton Pump** | PSYCH | **Sedation** | Seizure (AED) | **Sinusitis** | Snake Antivenom | **Steroids - anabolic** | Steroid - cortico | **Steroids - topical** | Thyroid-hypo | **Thyrotoxicosis** | Topical & Local Anesthesia | **Transplantation** | URI | **Urology** | UTI | **Vitamins & Nutrients** | Warfarin | **Extrapyramidal SE** | Neuroleptic Malignant Syndrome | **Malignant Hyperthermia** | Viscosupplementation | **Pharmacotherapy** |

Preamble and List of Abbreviations:

Links: Disclaimer | **Labs & Study Values** | Abbreviations | **INDEX** | Studies & Sx's | **Medications** | Diagrams | **Radiology** | Customer Book Reviews |

Written by Carl G. Weber MD, board certified, Internal Medicine, edited by the board certified specialist listed in each section.

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Quotes: "If you can't explain it simply, you don't understand it well enough." - Albert Einstein. "Sharing what you have is more important than what you have" –Albert M Wells Jr. Life is a journey, enjoy the ride!

Purpose of Book:

We believe this is the first and only electronic textbook available that adequately summarizes medical information in a concise and easy to read format. This book is directed at practicing clinicians. It is also useful to residents and medical students doing clinical rotations. It includes differential dx, basic pathophysiology, clinical pearls, physical diagnostic findings as well as evaluations and tx's of a multitude of medical conditions. Our hope is to accumulate and share information that will allow you to practice high quality and cost-effective medicine. This book will also allow primary care physicians to interface with the specialist, and assist in deciding **when** referral is necessary.

Book Disclaimer:

Medicine is a constantly changing science. Much of the information contained herein may be time sensitive. There is an increasingly better understanding of disease processes and newer diagnostic and tx modalities emerging almost every day. The authors, editors, and publisher of this text have given their best efforts to provide accurate, up-to-date, information that is considered to be within current medical standards. The approaches to medical problems are referenced with supporting studies, and further reading is encouraged. Human error is always possible and the authors, editors, and publisher of this text do not warrant the information as being absolutely complete, nor are they responsible for omissions or errors in the text or for the results of using this information. This book should not take the place of your own clinical judgment or the advice of specialists. We have tried with great fervor to limit errors, misinformation, and copyright infringement in the text. All attempts towards current medical practice has been made. We would greatly appreciate feedback regarding any problems you discover while perusing these pages. The presenters of this text take no responsibility nor liability for clinical outcomes based on information in this text. This text must be used at your own risk, it is not intended as a decision tool, nor is it a complete reference. It is simply a brief guide and memory jogger. We strongly advise users to obtain a more complete text, particularly with drug information as not all side effects and contra-indications are listed, nor are drug interactions included. We do not assume and expressly disclaim any obligation to obtain an include any information in this document. We do not advocate the use of any product described in this document, nor are we responsible for misuse of a product due to typographical or other errors in the document, user negligence or otherwise. The user agrees to seek additional information on any product from the manufacturer; and it will use this text only as a reference aid, and that such information is not intended to be (nor should it be used as) a substitute for the exercise of professional judgment. In view of the possibility of human error, or changes in medical science, user should confirm the information in the document through independent sources. User agrees and acknowledges that user will, at all times, advise pt's to seek professional dx and tx for any medical condition, and to discuss information obtained from the document with other healthcare providers. Any personal distribution or replication of this document is strictly forbidden. This information is an informational resource designed to assist licensed healthcare practitioners in caring for their pt's and/or to serve consumers viewing this service as a supplement to, and not a substitute for, the expertise, skill, knowledge and judgment of specialist medical practitioners. Furthermore, over some subjects there are differing opinions, or differing degrees of certainty. We have usually not attempted to discuss these here because the aim has been to provide an immediate and brief guide. In all areas, prior medical knowledge is assumed.

Other Available titles:

The Clinical Medicine Consult (NOTE: includes all 30 of these titles (including this text) as all are pulled from this one highly integrated text, "The Clinical Medicine Consult" for $59 via http://clinicalmedconsult.com/, includes 1 year of FREE updates). Clinical Endocrinology, Neurology, Orthopedics, Nephrology, Dermatology, Gastroenterology, ENT, Psychiatry, Urology, Infectious Disease, Women's Health, Hematology / Oncology, Geriatrics-Death & Dying, Rheumatology, Pulmonology, Allergy, Pediatrics, Pain Management, Ethics-Alternative Medicine-Evidence Based Medicine-Communication (Compendium), Cardiology, Ophthalmology, Travel Medicine, Procedures in Medicine, Wound Care & Skin Infections, Sports Medicine, Alternative and Complementary Medicine, Cancer Medicine, Hospice & Palliative Care. The goal is for these texts is to have >95% of clinical of the information you need at your fingertips. **Disclaimer |**

• **Note:** Information is added as important new information is published. We perform an ongoing review of >520 journals, textbooks, online sources, and many other expert resources. For example, in 6/08, the current file size of the CMC 2009v3 is 16,918 kb, the last quarterly versions were sized at 16,516, 15,858 kb, 15,176 kb, 14,580 kb, 13,980 kb, 12,950 kb 12,276 kb, 11,715 kb, 11,097, 10,752, 10,249, 9,769, 9,298, 8,977, 8,004, 6,805 and 5,364kb....tons more information for you to use at your fingertips and even more in the very near future. Expect to see additional huge improvements with upcoming versions! We do not advertise this textbook, it is purely by word of mouth.

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Radiology:

Links: U/S | **Nuclear Med** | CT Scan | **CT calcium score** | MRI | **CXR** | GI Tract / Abd X-rays | **Mammography** | Imaging Studies in Strokes | **Radionuclide Techniques in Cardiology** | Bone Scans in Ortho | **PET Scan** | Typical Medical Radiation Exposure Doses | **Fractures** | Swallowing Eval |

Cost is an important consideration when evaluating techniques. Cost is generally about 20% more for MRI ($1200) compared with CT ($1000) and about twice as expensive as U/S ($500). Obesity contributes to a rising trend in poor image quality and interferes with the interpretation of many radiological studies (Radiology 2006;240:435-439)....most frequently with abd U/S followed by CXR, abd X-rays, abd CT, chest CT, and MRI reports.....this increases overall healthcare costs as additional studies needed.

ICD-9: 793.91 Image test inconclusive due to excess body fat.

Myelography: Since the advancement of the CT scan and MRI, myelography is rarely utilized. A water-soluble contrast agent is injected into the subarachnoid space. A myelogram is an invasive procedure and carries some degree of risk includ-ing back pain, post-spinal tap headache, infection, and, occasionally, seizures. A myelogram is performed more in neurosurgery or orthopedic departments. In general, if an MRI is not available and you are looking for spinal root and cord compression, a myelogram followed by a CT scan is a reasonable approach.

Ultrasound (U/S):

Link: See Trauma for FAST U/S scan in Trauma Pt's & Handheld Cardiac Scans | See Nephro for **Renal U/S** **| See Womens Health for** U/S in Pregnancy **|**

An U/S is a sound wave of a frequency > can be heard by the human ear, 20,000 Hz. A RUQ U/S costs ~$430 and a full Abd U/S costs ~$570. U/S is based on the emission of high-frequency sound waves and their subsequent reflection as they pass through the tissue. The amplitude of the reflections of the sound waves varies according to differences in the acoustic impedance of the various tissues.

• US relies on a dual-mode transducer, which first sends high frequency sound waves into the pt and then receives the reflected waves to generate an image.

• Images may be displayed as a two-dimensional gray-scale image or as a color Doppler.

• Frequencies used in medical sonography range from 2 to 10 MHz.

• The higher the frequency, the better the resolution, but the poorer the depth of penetration. Small part probes for scanning testes and the penis operate at a frequency of between 7 MHz and 9 MHz, while an abdominal scan in an obese individual requires a 2.5–7 MHz transducer.

Ultrasound scanning is cheap, easily accessible, painless, safe for the pt, and provides excellent real-time anatomical as well as functional information.

Color-flow Doppler capabilities has made it possible to display the speed and direction of blood flow. This methodology encodes the frequency of pulsed Doppler signals by color, which are then overlaid on B-mode sonograms with their two-dimensional spatial information. Detection of velocities of < 1 cm/s, in combination with increased spatial resolution (high-frequency US), allows visualization of very small vessels, so that the vascularity of areas can be assessed. The introduction of power Doppler US, displaying the strength of the Doppler signal in color, increases the gain by 10 to 15 dB, resulting in an improved sensitivity for detection of color flow. US contrast media presently under development can, in principle, increase the Doppler signals by up to 25 dB.

Ultrasound Techniques in the Urgent Care Setting:

Step #1: Turn the scanner "on" and allow for any hardware warm-up and self-testing. Drape the pt to expose the appropriate area to be scanned and apply a liberal amount of acoustic gel (acts as a transduction medium for the ultrasound waves and serves as a lubricant to allow the transducer to slide over the skin).

The Gallbladder and Cholelithiasis: The normal gallbladder is a cystic structure whose echogenic walls surround dark anechoic bile. The normal fasting gallbladder is usually easy to visualize. Position the pt supine, expose the right upper quadrant and epigastic area, and apply acoustic gel. The US transducer is placed under the right costal margin or in an intercostal space. The liver serves as an acoustic window over the gallbladder. Obtain images in both longitudinal (long-axis) and transverse (short-axis) planes. Gallstones are highly echogenic and cast acoustic shadows. Calculi as small as 1 mm may be seen within the gallbladder lumen. Intraluminal stones are easier to see than those lodged in the cystic duct, common bile duct, or the neck of the gallbladder. The acoustic shadow produced by the stone is sometimes easier to see than the stone itself. Other US signs suggesting acute cholecystitis include the sonographic Murphy's sign (tenderness over the gallbladder with probe pressure), pericholecystic fluid, and wall thickening greater than 3 mm.

Abdominal Aorta: US can diagnose AAA. In symptomatic pt's who are hemodynamically stable, bedside US can provide a rapid dx before presurgical imaging (CT scanning), thus expediting tx, including notifying a surgical consultant, mobilizing the operating room, or arranging transportation to an appropriate facility. The US examination will demonstrate widening of the aorta (aneurysm) but will not demonstrate actual leakage. (A contrast-enhanced CT scan will demonstrate this.) The abdominal aorta is visualized by scanning with a convex transducer from the diaphragm to the aortic bifurcation. The IVC is normally located to the right of the aorta. In contrast to the IVC, the aorta is identified by its thicker walls and narrower diameter. The aorta is pulsatile, although the IVC has reflected pulsations from the right ventricle. If the aorta has a cross-sectional diameter (measured from the outer walls) of greater than 5 cm, an AAA is present.

Traumatic Hemoperitoneum: US provides a rapid assessment of the presence or absence of fluid in the thorax and pericardial spaces. In an unstable pt, US not only is accurate in detecting hemoperitoneum, but may be preferred over diagnostic peritoneal lavage.

Free fluid flows to the dependent areas of the abdominal cavity, including Morrison's pouch (the potential space between the liver and the right kidney), the left upper quadrant (either in the left hemithorax, subdiaphragmatic space, splenorenal space, or paracolic gutter), and the pelvis in the region of the cul-de-sac. Fresh, unclotted blood appears anechoic. A black "stripe" in these locations is indicative of fluid. If the pt has sustained a recent abdominal trauma, this fluid is presumed to be blood. The US screening examination for hemoperitoneum assesses four areas for free fluid. The convex scanner is placed in the subcostal area to look for pericardial blood. The right upper quadrant is then scanned for evidence of free fluid in the right hemithorax, Morrison's pouch, and the right paracolic gutter. The left upper quadrant is scanned for fluid in the left hemithorax, subdiaphragmatic space, splenorenal space, or left paracolic gutter. Placing the pt in the Trendelenberg position will allow fluid to flow into Morrison's pouch and the splenorenal space, maximizing the sensitivity of the scan. Finally, the cul-de-sac is assessed for free fluid. Optimal visualization of the pelvis is aided by bladder filling.

Cardiovascular: The pericardium is highly echogenic and serves as the sonographic border of the cardiac image. Ultrasound is highly sensitive for detection of pericardial fluid, which is recognized as an anechoic area (black stripe) between the echogenic pericardium and the heart. The simplest US view of the pericardium is obtained by placing the transducer (3.5 MHz works best) in the subcostal area just to the left of the xiphoid process, and angling toward the RIGHT scapula. Although visualization of all the individual heart chambers can be difficult in this view, a pericardial effusion is easily appreciated. Clotted blood within the sac is often somewhat echogenic. Anechoic pericardial fat and pleural effusions can occasionally be confused with small pericardial effusions.

Pelvic Applications: Ultrasound scanning is an invaluable tool in the dx of obstetric and gynecologic conditions. In the urgent care setting, the simplest application of pelvic US scanning is for the dx of an intrauterine pregnancy (IUP) or for the presence of free pelvic fluid. A convex transducer (3.5 or 5 MHz) is generally adequate for these applications, although a transvaginal transducer (if available) will provide a superior image in the first trimester of pregnancy. Ultrasound imaging should be an adjunct to pt history, physical examination, and measurement of a qualitative (quantitative if available) HCG level. A full bladder provides an acoustic window and landmark for transabdominal scanning of the pelvis. A gestational sac of an IUP will appear as an anechoic structure within the uterus with highly echogenic borders. As the pregnancy progresses, the gestational sac will obviously enlarge and fetal

structures (head, body, limbs) will become evident. Transabdominal scanning will generally reveal a fetal heartbeat by 7 to 8 weeks of pregnancy. Free fluid in the cul-de-sac appears as an anechoic stripe behind the uterus. If the pt has a measurable HCG level, has an empty uterus, and fluid in the cul-de-sac, urgent consultation is advised to rule out the possibility of ectopic pregnancy.
Physics: US equipment uses the piezoelectric effect for both generating US waves and receiving the reflected echoes. The trans-abd wave is 3.5 megahertz, trans-vaginal, 5 megahertz. U/S uses the principle of echography, locating objects by receiving echoes from reflecting surfaces. The area of greatest reflection is at an acoustic impedance interface where 2 tissues meet. Areas that reflect a lot of sound waves (bone) are called sonodense, while areas transmit sound waves are sonolucent. Diagnostic equipment transmits U/S waves then listens for sound waves that are reflected. Based on the time and location of the reflected waves, it locates an anatomic structure in space. Piezoelectric crystals located in the transducer produce the sound waves. These waves enter water soluble gel placed on the skin, to increase transmission into the body. Equipment is real time, meaning it sees motion as it is occurring. The piezoelectric effect has two components, both of which are essential to the operation of diagnostic US.
First: when a piezoelectric substance is compressed, as by a returning US wave, an electric current is produced.
Second: when an alternating current is applied to the piezoelectric element, the object vibrates at a stable frequency. That frequency is a characteristic of the material and its thickness. These two properties allow for the transmission and reception of US waves by an US probe or transducer. The two-dimensional image on an US screen is produced from the electrical information that is generated by the transducer.
RUQ Abd Pain: Evaluate for gallstones.
Kidneys, adrenals, and ureter: Possible upper tract obstruction. Suspected renal/adrenal mass. Investigation of renal failure. Investigation of hematuria. Monitor renal cystic disease. Diagnosis of urinary stone disease. Aids access to kidneys for interventional procedures. Color doppler to demonstrate vascular lesions.
Bladder: Bladder outflow obstruction—measurement of residual urine. Investigate intra-vesical mass (e.g., clots, stone, tumor). Aid suprapubic aspiration.
Prostate and seminal vesical: Suspected prostate cancer (+biopsy). Investigation for chronic pelvic pain.
Scrotum: Eval of: Lump. Pain. Trauma.
Penis: Investigation of erectile dysfunction. Peyronie's disease. Diagnose high-fl ow priapism. Can aid visualization of urethral strictures.
Doppler technology: The Doppler effect is a physical phenomenon which demonstrates a change in the frequency of a signal due to the relative movement of the signal source compared to the observer. The Doppler signal is usually in the audible range. Application of an ultrasound beam will allow the measurement of flow direction and velocity of any structure which moves (e.g., flow of RBC in a blood vessel).
Simple continuous-wave Doppler can detect velocity but not depth of the signal source.
Pulsed Doppler, on the other hand, uses intermittent bursts of ultrasound and is capable of assessing depth as well as velocity and can adequately display the flow characteristics at a desired depth.
The flow, direction, pulsatile rhythm, and resistivity of flow can be displayed either graphically (spectral trace) or by color.
Color Doppler imaging (CDI): currently the technique of choice for the evaluation of blood flow and directional information in vessels and works by superimposing color over the conventional gray-scale image. Red demonstrates blood flow toward the transducer, while blue demonstrates flow away. CDI, however, is angle dependent and lacks sensitivity when imaging small, deep blood vessels or areas of slow blood flow.
Power Doppler imaging PDI): attempts to overcome the limitations of CDI, by displaying the amplitude (or strength) of the Doppler signal rather than simply the change in frequency. Power Doppler imaging works by detecting the density and movement of red blood cells and as such is independent of blood flow velocity and direction. It is 3–5 times more sensitive in the study of small, deep, slow-flowing blood vessels.
Dupplex scans: combine real-time anatomic scanning with pulsed Doppler hemodynamic information and permits blood velocity estimation at different sites within a two-dimensional field.
Contrast-enhanced ultrasound (CEUS): Used in the diagnosis and characterization of hepatic lesions. However, due to the inherent vascular nature of the kidneys, CEUS allows excellent spatial and temporal resolution, as well as assessment of renal perfusion. Contrast agents (SonoVue, Levovist, Optison, Definity, Sonazoid) are intravenously injected compounds which consist of microbubbles containing a gas (air or hydrophobic gas) stabilized by a shell. Thus the agents acting as vascular tracers neither leave the blood vessels nor are subjected to renal fi ltration, and therefore dramatically increase the signal intensity from blood. Although modern contrast agents are more stable, the microbubble shells are thin and fragile, breaking easily as they oscillate under the infl uence of the ultrasonic accoustic pressure wave. Even the destruction of the microbubbles can aid in their detection and allow for quantifi cation of renal perfusion.
Transcranial Doppler (TCD): an ultrasound technique that measures the blood flow and noninvasively images the velocity of cerebral circulation in the distribution of major intracranial arteries. In experienced hands, this proce-dure is safe, fast, and reliable. Combined duplex and TCD increases the sen-sitivity and specificity of the test when performed by skilled technician.
Indications: To detect stenosis more than 65% in major intracranial arteries. To assess the collateral circulation of the intracranial arteries. To assess and monitor response to tx of cerebral vasospasm follow-ing subarachnoid hemorrhage. To detect arteriovenous malformations, assess their arteries and flow, and evaluation of their obliteration following embolization (injecting particles into the arteriovenous malformation). TCD may soon become the procedure of choice to assess suspected brain death and monitoring patients for increased intracranial pressure.

Common Nuclear Medicine Studies:

Isotopes of an element share the same atomic number (and therefore the same biochemical characteristics) but differ in their mass number (as well as their energy states). An example is the isotopes of iodine (123I, 131I, 125I). When an isotope has radioactive properties, it is called a radioisotope or radionuclide. Radionuclides undergo spontaneous disintegration, while emitting high—penetrating, electromagnetic gamma rays (measured in electron volts [eV]). Radionuclides are used to label a compound, with a specific interaction with the target organ (e.g., kidney, bone) and the resulting ionizing radiation is detected and quantified by a gamma camera. A nuclear medicine image, therefore, is a map of where the tracer has accumulated, and is dependent on blood flow to the target organ as well as tracer metabolism by the organ (an indicator of function). The half-life of a radiopharmaceutical (radionuclide + labeled compound) is determined by its natural rate of nuclide decay and metabolism/handling by the body. Decay is measured in Becquerals (Bq) and 1 Bq equals 1 disintegration per second. Typically, diagnostic procedures result in a dose delivery

of 106 Bq (1 MBq) and therapeutic interventions in 109 Bq (1 GBq). The ideal radiopharmaceutical should have the following characteristics: 1. Easily and cheaply generated. 2. Radiochemically pure. 3. Non-toxic. 4. Should emit only gamma rays (100–200 keV energy). 5. Half-life long enough to complete investigation. 6. Half-life short enough to minimize pt radiation risk.
Positron emission tomography (PET) is an emerging radionuclide technique which has recently enjoyed a migration from the research setting into clinical practice. The most commonly used radionuclide in PET scanning isfl uorine-18 which binds to a D-glucose analog.
Bone scan with 99m Tc-MDP: to dx benign or metastatic bone d/o's. Used for unexplained bone pain, child abuse. Delayed imaging at 3-4 hr. Localizes at sites of osteoblastic or osteoclastic activity. Three-phase bone scan (99m Tc-MDP) Infection or inflammation (osteomyelitis, septic arthritis), AVN, fx No preparation Immediate blood flow, extracellular phases. Delayed imaging at 3-4 hr Blood flow and extracellular information helpful in infection or inflammation
Gallium scan (67 Ga citrate): Helps evaluate infections (FUO). Some tumors (Hodgkin's lymphoma). Imaging times dependent on indication or interference from bowel activity (24, 48, 72, 96 hr) Ga2+ is an Fe2+ analog, binds to serum proteins. Partial excretion in colon.
White blood cell scan (111 In-labeled WBCs): for infections. WBC labeling, reinjection. Imaging at 24 and/or 48 hr. Localizes active inflammation or infection. No bowel excretion.
Brain blood flow scan (99m Tc-DTPA): used as an adjunct in assessment of brain death. Absent perfusion is compatible with brain death. Brain scan (99m Tc-ECD or 99m Tc-HMPAO): used to help in localization of epileptic foci. Ictal foci show increased flow. Tracer follows blood flow.
Cisternography (111 In-DTPA): helps dx hydrocephalus or CSF leak and assess patency of VP, LP, VA shunts. Tracer injected into subarachnoid space. Tracer follows CSF flow.
VQ scan (99m Tc-MAA perfusion,133 Xe ventilation): Assess congenital pulmonary or vascular anomalies, pulmonary embolus. IV-injected MAA trapped in the pulmonary capillary bed. 133 Xe gas inhaled.
Gastric emptying or reflux study (99m Tc-sulfur colloid): Quantitative assessment of solid or liquid emptying. Gastric reflux. Tracer is not absorbed in the GI tract. Assess for GERD or pulmonary aspiration.
Hepatobiliary (99m Tc-mebrofenin): Neonatal hepatitis versus biliary atresia. Biliary leak after trauma or transplant. Phenobarbital 2.5 mg/kg Q12hr 3-5 days before imaging. Phenobarbital increases bile secretion; in hepatitis, helps in tracer transit. In leaks, tracer extravasates into peritoneal cavity.
Liver-spleen scan (99m Tc-sulfur colloid): Defining organs in congenital anomalies (splenosis, asplenia). Assess splenic function. Tracer taken up by RES in liver (Kupffer's cells), spleen, bone marrow.
Meckel's scan (99m Tc-pertechnetate): Gastrointestinal bleeding (Optional) pentagastrin, cimetidine, glucagon. Tracer localizes to mucinous cells in Meckel's diverticula.
Testicular scan (99m Tc-pertechnetate): Suspected testicular torsion versus epididymitis. Absent flow in torsion; increased in epididymitis
Radionuclide cystogram (99m Tc-pertechnetate): to dx vesicoureteral reflux and functional bladder capacity. Direct infusion of tracer via bladder catheter. Dynamic imaging of bladder filling and voiding. Sensitive in detection of VUR
Renal scan: 99m Tc-DTPA or 99m Tc-MAG3. To asses renal function, renovascular HTN (RAS), ureteral obstruction. Initial 30-min imaging. Lasix washout 30 min (optional) 99m Tc-DTPA is excreted by filtration (GFR). Captopril challenge for RAS. Lasix washout slower in obstruction. 99m Tc-DMSA or 99m Tc- glucoheptonate Renal cortical scarring. Pyelonephritis 99m Tc-DMSA: Imaging at 4 hr. Glucoheptonate: Initial 2-hr delayed imaging. Tracers localize in the renal tubules, providing an indicator of function. There are three important radiopharmaceuticals currently in clinical use for radionuclide renography. First, I123 orthoiodo-hippurate (I123 hippuran) is a tracer that replaced I131 hippuran because of its lower radiation dose and better imaging properties. Like para-aminohippuric acid, it is almost completely cleared by the renal tubules and is therefore closely related to the effective renal plasma flow. Second, technetium-99m diaminotetra-ethyl pentaacetic acid (DTPA) is a tracer that is cleared only by glomerular filtration and therefore can be used as a direct measure of GFR, although problems related to protein binding may cause significant inaccuracies. For many years, technetium-99m DTPA was used almost universally for dynamic radionuclide renography. However, it is progressively being replaced by technetium-99m mercaptoacetyltriglycine (MAG3) for this purpose. Finally, technetium-99m MAG3 is, like hippuran, largely excreted by the proximal renal tubules but with the significant advantage of improved images because of the higher renal concentrations that occur as a result of higher extraction rates. This may be particularly important in situations in which there is impaired renal function. Technetium-99m MAG3 is now the radiotracer of choice for dynamic radionuclide renography.

Lab Values and Tests:

Links: See Below: On Call and Outpatient Problems | **Labs** | Radiology | **Urinalysis** | Procedures | **ACLS / BLS** | Medications | ***INDEX*** | Diagrams |

Various "On Call" Problems:

Links: Lab abnormality (see below) | ACLS / BLS | **Acute Renal Failure** | Airway and Intubation | **Allergic Reactions** | Angina – Chest pain | Coma | **Diarrhea & Enteritis** | Dyspnea / SOB | **Fever** | GI Bleed | **GI Tubes and drains** | HTN Urgency | **Hypotension** | Mental Status Changes & Delirium | **Musculoskeletal / Ortho** | Nausea & Vomiting | Needlestick | **Oncologic Emergency** | Pain | **Pleurisy** | Post-Op Complications / Surgical Pearls | **Psychiatric Emergency** | Seizure | **Sudden Vision Loss** | Transfusion Reactions | Urinary Catheters | **Urinary Retention** |

Common Outpatient Conditions:

Abd Pain | Anemia | **Bites/ Stings** | Breast d/o's | **Bruising & Bleeding** | Chest Pain | **Claudication** | Constipation | **Cough** | Cramps (muscle) | **Delirium & Dementia** | Dermatitides | **Diabetes Mellitus** | Diarrhea | **Dizziness** | Dry Eyes | **Dry Mouth** | Dysphagia | **Dyspnea** | Dysuria | **Edema** | Epilepsy | **Fatigue** | Fever | **Flushing** | Foreign Bodies | **GI Bleed** | GERD | **Gynecomastia** | Heat | **Hematuria** | Hepatitis | **Hoarseness** | Hot Flashes | **Infections** | Joint Pain | **Hearing Loss** | Hypertension | **Hypoglycemia** | Hypothermia | **Jaundice** | Lower Urinary Tract Symptoms (LUTS) | **Movement D/o** | Musculoskeletal / Ortho |

Neck Mass | Neck Pain | **Obesity** | Orofacial Pain | **Otitis** | Proteinuria | **Psychiatric** | Red Eye | **Palpitations** | Pleurisy | **Scrotal Mass/ Pain** | Seizures | **Sexual D/o** | Skin Reactions | **Skin Lesion** | Sleep D/o | **Spells** | STD's | **Strokes** | Syncope | **Syndromes** | Tinnitus | **Trauma** | Urology | **URI** | UTI | **Vaginal / Pelvic** | Vision Loss | **Wasting & Wt loss** | Weakness |

Ddx based on Labs:

Acute Phase Reactants | Albumin | **Alk Phos** | Ammonia | **Amylase** | ANA | **ANCA's** | Anemia Labs | **Anemia Type** | AST / ALT | **Auto-Ab's** | **b-hCG** | Bili | **Blood Cx** | BM Sample | **BNP** | BRCA gene | BUN | **BUN/Cr** | Calcium | **Cancer Ag's** | **CEA** | Ceruloplasmin | **CK** | Complement | **Coombs Test** | Creatinine | **Creatinine Clearance** | CRP & ESR | **Cryoglobulins** | D-dimer | **Erythrocytes** | Female Hormones | FENa | **Ferritin** | GFR | **GGT** | H/H | **Haptoglobin** | HbA1c | **Iron** | LDH | **"LFT's"** | Lipase | **Magnesium** | MCV | **Met-Hb** | Normocytic | **Osmolar Gap** | Osmolarity | **Osmotic Fragility Test** | Peripheral Smears | **Phosphate (P)** | **Pituitary** | Platelets | **Potassium (K)** | Protein | PSA | **PT** | RBC's | RDW | **Retic** | RF | **Protein (serum)** | Sodium (Na) | **Spot Ur Na** | Synovial Fluid | **Thyroid Labs** | TIBC | **Transferrin** | Transferrin sat | **Troponin** | Tumor Markers | **Ur Osm** | Uric Acid (serum) | **Urinalysis** | Urinary Electrolytes | WBCs |

Studies:

ABG | **Bleeding pt's Labs** | Blood Cx | **BM Sample** | Bronchoscopy/ BAL | **Cardiac Testing** | Cosyntropin (ACTH) Stim Test | **CRH Stim Test** | Dexamethasone Suppression Test | **ECG's** | EEG | **Electrophoresis** | EMG / NCV | **Fecal Occult Blood (FOB)** | Grams Stain | **Hypercoagulability** | Pap Smear | **PFT's** | Radiology | **PPD** | Serology | **Sputum Analysis** | Stool Osmotic gap | **Synovial Fluid** | Tzanck |

Common Lab Values:

Links: Hematology | **Endocrinology** | ***Most laboratories set their own "normal" ranges, thus check with your own lab. This list is for your convenience to give you a general range, thus use at your risk! No warranty to accuracy or correctness implied or otherwise.

http://labtestsonline.org/: a nonprofit web site sponsored by the American Association for Clinical Chemistry, offers detailed but easily understood information on lab work and test results.

Reference Range: are method- and lab-specific. In practice, they often represent test results found in 95% of a small population presumed to be healthy; by definition, then, 5% of healthy pt's will have an abnormal test result. Slightly abnormal results should be interpreted critically—they may be either truly abnormal or falsely abnormal. The practitioner should also be aware that the more tests ordered, the greater the chance of obtaining a falsely abnormal result. For a healthy person subjected to 20 independent tests, there is a 64% chance that one test result will lie outside the reference range. All abnormal labs should be interpreted in the context of the pt's baseline values because up to 5% of the general population without disease will display lab values outside the statistically assigned "normal" reference range. Just as a substantial / significant change in a "personal normal" may also still be in the "normal" reference range.

ICD-9: 790.6 = Lab Abnormality

Relationship between the number of tests and the probability that a healthy person will have one or more abnormal results.

Number of Tests **-->** Probability That One or More Results Will Be Abnormal.
1 **-->** 5%.
6 **-->** 26%.
12 **-->** 46%.
20 **-->** 64%.

Nondisease Factors That Can Cause Lab Values to Fall Outside the Normal Range: Pregnancy, lactation. Athletics and exercise. The neonatal period and prematurity. Posture. Issues requiring medical judgment (i.e., hematuria). Food intake and nutritional state, dietary supplements, vitamins. Prescription drugs, over-the-counter preparations. Oral contraceptives. Ethanol. Hemolysis or clotting of the specimen. Specimen that is aged or exposed to sunlight. Physiologic variations based on circadian rhythms, diurnal variations, time of day. Sampling problems, including drawing blood during IV therapy or near (from) the IV infusion site, blood stasis from a tourniquet, capillary puncture, arterial specimens or unusual sites of venous sampling, phlebotomy errors to include using the wrong collection tube. Lipemic specimens. Analytic instrument breakdown, loss of calibration, limits of precision, reagent quality, technician error, lab methods, reaction temperatures, instrumentation accuracy. Variations resulting from age, gender, race, genetic makeup, stress, menstrual cycle, menopause, body weight. The population used to determine the normal range. Application of the reference range from one lab to results from another lab. State of hydration. Socioeconomic environment, geographic location, altitude. Coexisting disease. Clerical error. (Textbook of Family Practice 6th ed. Rakel. By W B Saunders

Chem 7 = basic metabolic panel, 17 = comprehensive, which includes electrolytes, renal and liver function tests.

Medicare Lab Panels:

Medicare Organ- or disease-oriented panels:

Electrolyte Panel: #4 items: Na, K, Cl, CO2.

Basic metabolic panel: CPT 80048. #8 items. Or an "old" Chem 7 (SMA-7) with Ca. Calcium (82310). Carbon dioxide (bicarbonate) (82374). Chloride (82435). Creatinine (82565). Glucose (82947). Potassium (84132). Sodium (84295). Urea Nitrogen (BUN) (84520).

Comprehensive metabolic panel: CPT 80053. #14: BMP + Alb, AP, AST, ALT, Tb, Dbili. Albumin (82040). Bilirubin, total (82247). Calcium (82310). Carbon dioxide (bicarbonate) (82374). Chloride (82435). Creatinine (82565). Glucose (82947). Phosphatase, alkaline (84075). Potassium (84132). Protein, total (84155). Sodium (84295). Transferase, alanine amino (ALT) (SGPT) (84460). Transferase, aspartate amino (AST) (SGOT) (84450). Urea Nitrogen (BUN) (84520).

Renal Function Panel: CPT 80069. #10 BMP + Alb & PO4. Test Includes: Albumin (82040). Calcium (82310). Carbon dioxide (bicarbonate) (82374). Chloride (82435). Creatinine (82565). Glucose (82947). Phosphorus inorganic (phosphate) (84100). Potassium (84132). Sodium (84295). Urea Nitrogen (BUN) (84520). Requires 5ml of serum in a red-top tube or gel-barrier tube. Need to separate serum from cells within 45 minutes of collection. Refrigerate. Pt should fast for 12 hours preceding collection of specimen.

Extended CMP (ECMP) #17: CMP +Mg, P, LDH.

Acute hepatitis panel: CPT 80074. Hepatitis A antibody (HAAb), IgM antibody (86709). Hepatitis B core antibody (HbcAb), IgM antibody (86705). Hepatitis B surface antigen (HbsAg) (87340). Hepatitis C antibody (86803).

Hepatic function panel: CPT 80076. Albumin Bilirubin, total Bilirubin, direct Phosphatase, alkaline (84075). Protein, total (84155). Transferase, alanine amino (ALT) (SGPT) (84460). Transferase, aspartate amino (AST) (SGOT) (84450).

Acetoacetate: 0.3-2.0 mg/dl.

Acid phosphatase: 0-0.8 U/ml (0-5.5 U/L).
Acid phosphatase, prostatic: 2.5-12.0 IU/L.
Delta-aminolevulinic acid (ALA): <200 mcg/dl
Alpha-Fetoprotein, serum: <25-40 mcg/dl.
Albumin (Alb): 3.0-5.5 g/dl.
Aldolase: 1-6 IU/L.
Alkaline phosphatase (AP, ALP): 15-20 years @ 40-200 IU/L (U/L), 20-101 years @ 35-125 IU/L.
0-30 days: 60-320 U/L. 1-12 mo's: 70-350 U/L. 1-3 years: 125-320 U/L. 4-6 years: 150-370 U/L. 7-9 years: 150-440 U/L. 10-11 years: 150-470 U/L. 12-13 years: 160-500 U/L. 14-15 years: 130-530 U/L. 16-19 years: 60-270 U/L. 20 years and older: 40-120 U/L.
Alkaline Phosphatase Isoenzymes: at least nine recognized isoenzymes. Each has varying properties of heat stability, chemical inhibition, and electrophoretic mobility. They include fast (pre-liver), liver, bone, placental, Regan, Nagao, renal, intestinal, and PA. Of primary interest in the lab is distinguishing bone from liver, and for this purpose, heat inactivation is the most convenient method. Of secondary interest is the presence of the more heat-stable "tumor" isoenzymes: Regan, Nagao, and renal. **See LFT's.**
Alpha-1 antitrypsin: 200-500 mg/dl.
Aminotransferases: AST (SGOT): 0-35 U/L (IU/L), ALT (SGPT): 0-40 U/L.
Ammonia: Normal range = 10–80 µg/dl (6–50 µmol/L) = 80-110 mcg/dL. High in hepatic failure, hepatic encephalopathy, Reye's syndrome, portacaval shunt, drugs (diuretics, polymyxin B, methicillin). Low in tx with drugs (neomycin, lactulose, tetracycline), renal failure.
Amylase: 2-40 U/L.
Agiotensin converting enzyme (ACE level): nl is <40 nmol/ml/min (<670 nkat/L) High in sarcoidosis, primary biliary cirrhosis, alcoholic liver disease, hyperthyroidism, hyperparathyroidism, diabetes mellitus, amyloidosis, multiple myeloma, lung disease (asbestosis, silicosis, berylliosis, allergic alveolitis, coccidioidomycosis), Gaucher's disease, leprosy.
Anion gap: 8-12 mEq/L (mmol/L).
Ascorbic acid: 0.4-1.5 mg/dl.
Antimitochondiral Ab: <1:20 titer. High in primary biliary cirrhosis (85% to 95%), chronic active hepatitis (25% to 30%), cryptogenic cirrhosis (25% to 30%).
Anti-streptolysin O titer = Anti-streptococcus O titer = ASO Titer: Latex agglutination test to identify serum Ab to streptolysin O (Group A Streptococcus Oxygen labile hemolysin) Sensitive for acute streptococcal infection. Early Penicillin prevents ASO rise. Increases within one week of infection. Peaks 2-4 weeks after infection. Normal: Age 5-12 years: <333 I.U. Young Adults: <200 I.U. Increased in Streptococcus respiratory infection, Acute Rheumatic Fever (80%), Acute Glomerulonephritis (95%), Membranoproliferative Glomerulonephritis (20%), Acute Poststreptococcal Glomerulonephritis (70%).
AST: 5-40 IU/L.
Bilirubin: Total @ 0.2-1.2 mg/dl, Direct @ 0-0.4 mg/dl, Indirect @ 0.2-0.7 mg/dL.
Calcium: 8.7-10.6 mg/dl.
Carbon dioxide: 18-30 mEq/L (mmol/L).
Carcinoembryonic antigen, serum (CEA): <25. mcg/dl.
Carotene (carotenoids): 50-300 mcg/dl (0.9–4.6 µmol/L). High in carotenemia, chronic nephritis, diabetes mellitus, hypothyroidism, nephrotic syndrome, hyperlipidemia. Low in fat malabsorption, steatorrhea, pancreatic insufficiency, lack of carotenoids in diet, high fever, liver disease.
C3 complement: 55-120 mg/dl. C4 @ 14-51 mg/dl.
Ceruloplasmin: 15-60 mg/dl.
Chloride: 95-106 mEq/L (=mmol/L). High in dehydration, excessive infusion of normal saline solution, cystic fibrosis (sweat test), hyperparathyroidism, renal tubular disease, metabolic acidosis, prolonged diarrhea, drugs (ammonium chloride administration, acetazolamide, boric acid, triamterene).
Low in CHF, SIADH, Addison's disease, vomiting, gastric suction, salt-losing nephritis, continuous infusion of D5 W, thiazide diuretic administration, diaphoresis, diarrhea, burns, diabetic ketoacidosis
Cholesterol Total: <200 mg/dL, 12-19 yo @ 120-230 mg/dl,
20-29 yo @ 120-240 mg/dl, 30-39 yo @ 140-270 mg/dl,
40-49 yo @150-310 mg/dl. 50-59 yo @ 160-330 mg/dl.
HDL: 30-90 mg/dL, **LDL:** 50-160 mg/dL.
Complement, serum: C3 85–175 mg/dL= 0.85–1.75 g/L. C4 15–45 mg/dL= 150–450 mg/L. Total hemolytic (CH50) 150–250 U/mL= 150–250 U/mL.
Copper: 100-200 mcg/dl.
C-peptide: nl = 0.8-4 ng/mL.
Creatine kinase, total (CPK): 20-200 IU/L (25-145 U/L).
Creatine kinase, isoenzymes: CK-MB <3-5%. MM fraction @ 94%-95%, MB @ 0%-5%, BB @ 0%-2%.
Normal values in the heart @ 80% MM, 20% MB, in the brain @ 100% BB, in the skeletal muscle @ 95% MM, 2% MB.
CK-BB elevated in: cerebrovascular accident, subarachnoid hemorrhage, neoplasms (prostate, GI tract, brain, ovary, breast, lung), severe shock, bowel infarction, hypothermia, meningitis
CK-MB elevated in: MI, myocarditis, pericarditis, muscular dystrophy, cardiac defibrillation, cardiac surgery, extensive rhabdomyolysis, strenuous exercise (marathon runners), mixed connective tissue disease, cardiomyopathy, hypothermia. CK-MB exists in the blood in two subforms. MB2 is released from cardiac cells and converted in the blood to MB1. Rapid assay of CK-MB subforms can detect MI (CK-MB2 =1.0 U/L, with a ratio of CK-MB2 /CK-MB1 =1.5) within 6 hours of onset of sx's.**CK-MM elevated in:** crush injury, seizures, malignant hyperthermia syndrome, rhabdomyolysis, myositis, polymyositis, dermatomyositis, vigorous exercise, muscular dystrophy, IM injections, acute dissection of aorta
Creatinine (Cr): Female adult @ 0.5-1.3 mg/dl, Male adult @ 0.7-1.5 mg/dl.
CEA: <2.5 mcg/dL (<5.0 mcg/dL smoker).
CA-125: < 30 mcg/dL.
Dehydroepiandrosterone
Males 0.2–2.0 mg/24 h = 0.7–6.9 µmol/d .

Females 0.2–1.8 mg/24 h= 0.7–6.2 µmol/d.
Ferritin: 15-200 ng/mL.
Folate: 1.9-14.0 ng/ml.
Gamma glutamyl transpeptidase (GGT or GGTP):
Male @ 12-38 IU/L, Female @ 9-31 IU/L.
Gastrin: 150 pg/ml. See ZE syndrome.
Glucose, serum (fasting, blood sugar = FBS): 70-115 mg/dl. Subtract 15-20% if a plasma value.
Glucose-6-phosphate dehydrogenase: 5-10 IU/g Hb.
G6PD screen, qualitative: Negative.
Haptoglobin: 100-300 mg/dl. Hemoglobin A2 @ 0%-4% of total Hb.
Hemoglobin F @ 0%-2% of total Hb.
Immunoglobulin, quantitation: serum, adult
IgG @ 700-1500 mg/dl = 6.4–13.5 g/L,
IgA @ 70-400 mg/dl, = 0.70–3.1 g/L,
IgM @ Male = 30-350 mg/dl = 0.90–3.5 g/L,
Female = 30-300 mg/dl,
IgD @ 0-40 mg/dl = 0.0–60 mg/L.
IgE 0.0–430 ng / dL = 0.0–430 mcg/L.
Iron-binding capacity: 250-400 mcg/dl (250-450 mcg/dL) (45–82 µmol/L. TIBC elevated in iron deficiency anemia, pregnancy, polycythemia, hepatitis, weight loss. Decreased in anemia of chronic disease, hemochromatosis, chronic liver disease, hemolytic anemias, malnutrition (protein depletion).
Iron Saturation: 20-45.
Iron, total, serum (Fe): 40-150 mcg/dl (80-180 mcg/dL).
Lactic acid: 0.6-1.8 mEq/L.
LDH: 20-220 IU/L. LDH isoenzymes:
LDH1 @ 20%-34%,
LDH2 @ 28%-41%,
LDH3 @ 15%-25%,
LDH4 @ 3%-12%,
LDH5 @ 6%-15%.
Leucine aminopeptidase (LAP): 30-55 IU/L.
Lipase: 4-24 IU/dl (49-220 U/L).
Magnesium (Mg): 1.6-2.6 mg/dL.
5'-Nucleotidase: 0.3-3.2 Bodansky units.
Osmolality: 285-295 mOsm/kg serum water.
Phenylalanine: 3 mg/dl.
Phosphorus (P): 2.5-4.5 mg/dL.
Potassium, plasma: 3.1-4.3 mEq/L.
Potassium, serum: 3.5-5.2 mEq/L.
Protein: 2-55 yo @ 5.0-8.0 g/dl, 55-101 yo @ 6.0-8.3 g/dl.
Protein electrophoresis, serum (SPEP): Albumin @ 3.2-5.2 g/dl,
Alpha-1 @ 0.6-1.0 g/dl, Alpha-2 @ 0.6-1.0 g/dl,
Beta @ 0.6-1.2 g/dl, Gamma 0.7-1.5 g/dl.
PSA: < 4.0 ng/mL (see Urology section).
Sodium (Na): 136-145 mEq/L.
Sulfate: 0.5-15 mg/dl.
T3 uptake: 25%-45%.
T4: 4-11 mcg/dl.
Triglycerides (Trig): <60 mg/dL. 2-29 yo @ 10-140 mg/dl,
30-39 yo @ 20-150 mg/dl, 40-49 yo @ 20-160 mg/dl,
50-59 yo @ 20-190 mg/dl, 60-101 yo @ 20-200 mg/dl.
Trop-I: NL <0.4. 0.4-2.0 possible early MI. >2.0 MI likely.
Urea nitrogen, serum (BUN): 2-65 yo @ 5-22 mg/dl.
Uric acid: 10-59 yo: Male @ 2.5-9.0 mg/dl, Female @ 2.0-8.0 mg/dl. 60-101 yo: Male @ 2.5-9.0 mg/dl, Female @ 2.5-9.0 mg/dl.
Viscosity: 1.4-1.8 (serum compared to H2O) (1.10–1.22 centipoise). **Inc in:** Monoclonal gammopathies (Waldenström's macroglobulinemia, multiple myeloma), hyperfibrinogenemia, systemic lupus erythematosus, rheumatoid arthritis, polycythemia, leukemia.
Vitamin A: 0.15-0.60 mcg/ml.
Vitamin B12: 200-850 pg/ml.
Lead, blood
Adults <25 mcg / dL <1.2 µmol / L
Children <15 mcg / dL <0.7 µmol / L
Lead, urine <80 mcg / 24 h <0.4 µmol / d
Mercury, urine <30 mcg / 24 h <150 nmol / d

Hematology Labs:

Complete blood count: Male / Female.
Hematocrit (%): 40-52 / 38-48.
Hemoglobin (g/dl): 13.5-18.0 / 12-16.
Erythrocyte count (x 10-12th cells/L): 4.6-6.2 / 4.2-5.4.
Reticulocyte count (%): 0.6-2.6 / 0.4-2.4.

MCV (fL): 82-98 / 82-98.
MCH (pg): 27-32 / 27-32.
MCHC (g/dl): 32-36 / 32-36.
WBC (x 109 cells/L): 4.5-11.0 / 4.5-11.0.
Segmented neutrophils: 1.8-7.7 / 1.8-7.7. Ave %: 40-60 / 40-60.
Bands (cells): 0-0.3 / 0-0.3. Ave (%): 0-3 / 0-3.
Eosinophils (cells x 10-9th/L): 0-0.5 / 0-0.5. Ave %: 0-5 / 0-5.
Basophils (cells x 10-9th/L): 0.4% to 1% of total WBC; 40–100/mm3. Elevated in leukemia, inflammatory processes, polycythemia vera, Hodgkin's lymphoma, hemolytic anemia, after splenectomy, myeloid metaplasia, myxedema. Decreased in stress, hypersensitivity reaction, steroids, pregnancy, hyperthyroidism, postirradiation.
Subsets, Whole Blood, Heparinized Antigen(s) Expressed Cell Type// Percentage Absolute Cell Count
CD3 Total T cells 56%–77% //860–1880.
CD19 Total B cells 7%–17% //140–370.
CD3 and CD4 Helper-inducer cells 32%–54% //550–1190.
CD3 and CD8 Suppressor-cytotoxic cells 24%–37% //430–1060.
CD3 and DR Activated T cells 5%–14% //430–1060.
CD2 E rosette T cells 73%–87% //1040–2160.
CD16 and CD56 Natural killer (NK) cells 8%–22% //130–500.
Helper / suppressor ratio: 0.8–1.8.
Monocytes (cells x 10-9th/L): 0-0.8 / 0-0.8. Ave %: 2-6 / 2-6.
Platelet count (cells x 10-9th/L): 150-350 / 150-350.

Coagulation Normal Values:

Antithrombin III: 81% to 120% of normal activity; 17–30 mg/dl. Decreased in hereditary deficiency of antithrombin III, disseminated intravascular coagulation, pulmonary embolism, cirrhosis, thrombolytic therapy, chronic liver failure, postsurgery, third trimester of pregnancy, oral contraceptives, nephrotic syndrome, IV heparin >3 days, sepsis, acute leukemia, carcinoma, thrombophlebitis. Increased in warfarin drugs, post-myocardial infarction.
Template bleeding time: 3.5-7.5 min.
Clot retraction, qualitative: Apparent in 30-60 min; complete in 24 hr, usually in 6 hr.
Coagulation time (Lee-White), in glass tubes: 5-15 min, in siliconized tubes: 20-60 min.
Euglobulin lysis time: 120-240 min.
Factors II, V, VII, VIII, IX, X, XI, or XII: 100% or 1.0 unit/ml.
FACTOR I = Fibrinogen. From the liver. T1/2 = 120 hr. Substrate for fibrin clot.
FACTOR II = Prothrombin. From the liver. T-1/2 60hr. A serine protease.
FACTOR V = Proaccelerin, labile factor. From the liver. T-1/2 = 12–36hr. A cofactor.
FACTOR VII = Serum prothrombin conversion accelerator, proconvertin. From the liver. T1/2 = 6hr. A serine protease.
FACTOR VIII = Antihemophilic factor or globulin. From endothelial cells and (') elsewhere. T-1/2 = 12hr. A cofactor.
FACTOR IX = Plasma thromboplastin component, Christmas factor. From the liver. T-1/2 = 24hr. A serine protease.
FACTOR X = Stuart-Prower factor. From the liver. T-1/2 = 36hr. A serine protease.
FACTOR XI = Plasma thromboplastin antecedent. Likely from the liver. T-1/2 = 40–84hr. A serine protease.
FACTOR XII = Hageman factor. Likely from the liver. T-1/2 = 50hr. A serine protease contact activation.
FACTOR XIII = Fibrin-stabilizing factor. Likely from the liver. T-1/2 = 96–180hr. A transglutaminase.
Fibrin degradation products: <10 mcg/ml or titer 1.4.
Fibrinogen: 200-400 mg/ml.
Partial thromboplastin time, activated (aPTT): 20-40 sec.
Prothrombin time (PT): 11-14 sec.
Thrombin time: 10-15 sec.
Whole blood clot lysis time: >24 hr.
Acid hemolysis test (Ham): No hemolysis.
Carboxyhemoglobin: Nonsmoker @ <1%, Smoker @ 2.1%-4.2%.
Cold hemolysis test: No hemolysis
Erythrocyte life span: Normal @120 days, 51Cr-labeled half-life @ 28 days.
Erythropoietin by radioimmunoassay: 9-33 mU/dl.
Ferritin (serum): Male @ 15-200 mcg/L, Female @ 12-15O mcg/L.
B12: 200 - 1000 pg/mL.
Folate, RBC: 120-670 ng/ml.
Folate: 5 - 12 ng/mL.
Fragility, osmotic: Hemolysis begins @ 0.45%-0.38g% NaCl, completed @ 0.33%-0.30% NaCl.
Haptoglobin: 50-300 mg/dl. Itis depleted in all forms of hemolytic anemia, in liver failure and in individuals with a genetic absence of the protein or a defect in binding sites. It goes up in inflammatory states. It is an alpha-2 globulin that is cleared by the liver.
Hemoglobin: Hemoglobin A1C @ 0%-5% of total, A2 by column @ 2%-3% of total, fetal @ <1% of total, plasma @ 0%-5% of total, serum @ 2-3 mg/ml.
Iron: Male @ 75-175 mcg/dl, Female @ 65-165 mcg/dl.
Iron-binding capacity, total (TIBC): 250-450 mcg/dl.
Iron turnover rate (plasma): 20-42 mg/24 hr.
Leukocyte alkaline phosphatase (LAP) score: 30-150. Elevated in polycythemia vera, myeloid metaplasia and some inflammatory diseases. Dec in CML, PNH, Wilson's dz and occasionally in Hodgkin's.
Methemoglobin: <1.8%.
Schilling test: urinary excretion of radiolabeled vitamin B12 after "flushing" IM injection of B12 @ 6%-30% of oral dose within 24 hr.
Sedimentation rate: Male / Female. Wintrobe: 0-5 mm/hr / 0-15 mm/hr. Westergren: 0-15 mm/hr / 0-20 mm/hr.
Transferrin saturation, serum: 20%-50%.

Volume: Male / Female.
Blood: 52-83 ml/kg / 50-75 ml/kg.
Plasma: 25-43 ml/kg / 28-45 ml/kg.
Red cell: 20-36 ml/kg /19-31 ml/kg.
Differential cell count of bone marrow:
Myeloid cells: Neutrophilic series: **Myeloblasts:** 0.3%-5.0%, **Promyelocytes:** 1%-8%, **Myelocytes:** 5%-19%, **Metamyelocytes:** 9%-24%, **Bands:** 9%-15%, **Segmented cells:** 7%-30%, **Eosinophil precursors:** 0.5%-3.0%, **Eosinophils:** 0.5%-4.0%, **Basophilic series:** 0.2%-0.7%.
Erythroid cells: Pronormoblasts: 1%-8%, Polychromatophilic normoblasts 7%-32%, Megakaryocytes: 0.1%.
Lymphoreticular cells: Lymphocytes: 3%-17%, Plasma cells: 0%-2%, Reticulum cells: 0.1%-2.0%, Monocytes: 0.5%-5.0%, Myeloid/erythroid ratio: 0.6-2.7.

Endocrine Labs:

Laboratory Parameter: SI=Conventional (C)
Acetoacetate, plasma: <100 µmol/L=<1 mg/dL
Adrenocorticotropin (ACTH): 15-100 pg/ml
Adrenal Steroids, plasma:
Aldosterone, supine, saline suppression: <240 pmol/L=<8.5 ng/dL
Aldosterone, upright, normal diet: 140-560 pmol/L=5-20 ng/dL
Cortisol: 8 AM: 140-690 nmol/L=5-25 mcg/dL
4 PM: 80-330 nmol/L=3-12 mcg/dL
Overnight dexamethasone suppression: <140 nmol/L=<5 mcg/dL
Dehydroepiandrosterone (DHEA) : 7-31 nmol/L=2-9 mcg/dL
Dehydroepiandrosterone Sulfate (DHEAS) : 1.3-6.8 µmol/L=500-2500 ng/ml
11-Deoxycortisol: <30 nmol/L=<1 mcg/dL
17-Hydroxyprogesterone:
Women, follicular phase: 0.6-3 nmol/L=0.2-1 mcg/L
Women, luteal phase: 1.5-10.6 nmol/L=0.5-3.5 mcg/L
Men: 1.8-9 nmol/L: 0.6-3 mcg/L
Adrenal Steroids, urine:
Aldosterone: 14-53 nmol/d=5-19 mcg/d
Cortisol, free: 55-276 nmol/d=20-100 mcg/d
17-Hydroxycorticosteroids: 5.4-27.6 µmol/d=2-10 mg/d
17-Ketosteroids:
Men: 25-88 µmol/d=7-25 mg/d
Women: 14-53 µmol/d=4-16 mg/d
Aldosterone suppression: sodium depletion test (urine aldosterone collected on day 3 of 200 mEq day/sodium diet) @ <20 mcg/24 hr.
Ammonia, as NH3 , plasma: 6-47 µmol/L=10-80 mcg/dL
Angiotensin II, plasma: 10-60 ng/L=10-60 pg/mL
Arginine Vasopressin (AVP), plasma:
Random fluid intake: 0.0-2.8 pmol/L=1-3 pg/ml
Dehydration 18-24 h: 5.5-13 pmol/L=4-14 pg/ml
B-HCG levels: 0-2 wks @ 0 - 250 mIU/ml.
2-4 wks @ 100-5000.
4-8 wks @ 4000-200,000.
8-2 wks @ 8000-100,000.
C-peptide: 0.28-0.63 pmol/ml. Inc in insulinoma, DM 2. Dec in exogenous insulin administration (high insulin levels), DM 1. Formed during the conversion of proinsulin to insulin.
Calciferols (as Cholecalciferol, Vitamin D3) plasma:
1,25-Dihydroxycholecalciferol [1,25(OH)2 D] : 36-144 pmol/L=15-60 pg/mL
25-Hydroxycholecalciferol (25-OH-D) : 20-100 nmol/L=8-40 ng/mL
Calcitonin: Basal @ 0.15-0.35 ng/ml, stimulated @ <0.6 ng/ml.
Calcitonin, plasma:
Normal: <19 ng/L=<19 pg/mL. Normal <100 pg/ml (<100 ng/L).
Elevated in: Medullary carcinoma of the thyroid (particularly if level >1500 pg/ml), carcinoma of the breast, APUDomas, carcinoids, renal failure, thyroiditis.
Medullary cancer: >100 ng/L=>100 pg/mL
Calcium:
Ionized serum: 1-1.4 mmol/L=4-5.6 mg/dL
Total serum: 2.2-2.6 mmol/L=9-10.5 mg/dL
Catecholamines, urine:
Free Catecholamines: <590 nmol/d=<100 mcg/d
Epinephrine: <275 nmol/d=<50 mcg/d or <10 mcg / 24 h <55 nmol / d
Metanephrines: <7 µmol/d=<1.3 ng/d or 0.1–1.6 mg / 24 h 0.5–8.1 µmol / d
Norepinephrine: 89-473 nmol/d=15-89 mcg/d
Vanillylmandelic Acid (VMA) : <40 µmol/d=<8 mg/d
Chloride, serum: 98-106 µmol/L=98-106 mEq/L
Cholesterol, total plasma:
Desirable: <5.20 mmol/L=<200 mg/dL

Borderline: 5.20-6.18 mmol/L=200-239 mg/dL
Undesirable: =6.21 mmol/L==240 mg/dL

Cholesterol, HDL, plasma:
Desirable: >1.55 mmol/L=>69 mg/dL
Borderline: 0.9-1.55 mmol/L=35-60 mg/dL
Undesirable: <0.9 mmol/L=<35 mg/dL

Cholesterol, LDL, plasma:
Desirable: <3.36 mmol/L=<130 mg/dL
Borderline: 3.36-4.11 mmol/L=130-159 mg/dL
Undesirable: =4.14 mmol/L==160 mg/dL

Chorionic gonadotropin (hCG): Pregnancy 1st mo @ 10-10,000 mIU/ml, 2nd-3rd @ 10,000-100,000 mIU/ml. 2nd trimester @ 10,000-30,000 mIU/ml, 3rd trimester @ 5000-15,000 mIU/ml. Nonpregnant @ <3 mIU/ml.

Cortisol: 8 AM @ 5-25 mcg/dl, 8 PM @<10 mcg/dl. Cosyntropin stimulation @ >10 mcg/dl rise over baseline (30-90 min after 0.25 mg cosyntropin IM/IV). Overnight suppression (8 AM after 1 mg dexamethasone PO at 11 PM) @ 5 mcg/dl. Urine @ 20-70 mcg/24 hr.

11-Deoxycortisol: Basal @ 0-1.4 mcg/dl. Metyrapone stimulation (30 mg/kg PO 8 hr prior to level) @ >7.5 mcg/dl.

Corticotropin (ACTH) plasma, 8 AM: 2-11 pmol/L=9-52 pg/mL

Estrogens, urine: inc during pregnancy, dec after menopause. Male / Female. Total @ 4-25 mcg/24 hr / 5-100 mcg/24 hr. Estriol @ 1-11 mcg/24 hr / 0-65 mcg/24 hr. Estradiol @ 0-6 mcg/24 hr / 0-l4 mcg/24 hr. Estrone @ 3-8 mcg/24 hr / 4-31 mcg/24 hr.

Etiocholanolone, serum: <1.2 mcg/dl.

Epinephrine, plasma: <35 pg/ml.

Fatty Acids, Free (nonesterified) (FFA), plasma: 0.4-0.7 nmol/L=10.6-18 mg/dL

Follicle-stimulating hormone (FSH): Male @ 2-18 mIU/ml. Female Follicular phase @ 5-20 mIU/ml. Peak midcycle @ 30-50 mIU/ml. Luteal phase @ 5-15 mIU/ml. Postmenopausal @ >50 mIU/ml.

Free thyroxine index: 1-4 ng/dl.

Gastrin, plasma: <120 ng/L=<120 pg/mL

Gastrin, serum (fasting): 30-200 pg/ml.

Glucagon, plasma: 50-100 ng/L=50-100 pg/mL

Glucose, plasm:
Overnight fast, normal: 4.2-6.4 mmol/L=75-115 mg/dL
Overnight fast, diabetes mellitus:
National Diabetes Data Group: >7.8 mmol/L=>140 mg/dL
American Diabetes Association: >7.0 mmol/L=>126 mg/dL
72-h fast, normal men: >2.8 mmol/L=>50 mg/dL
72-h fast, normal women: >2.2 mmol/L=>40 mg/dL

Glucose Tolerance Test, 2-h postprandial plasma glucose:
Normal: <7.8 mmol/L=<140 mg/dL
Impaired glucose tolerance: 7.8-11.1 mmol/L=140-200 mg/dL
Diabetes mellitus: >11.1 mmol/L=>200 mg/dL

Glucose tolerance test: serum glucose after 100 g glucose PO. 60 min after ingestion @ <180 mg/dl. 90 min @ <160 mg/dl. 120 min @ <125 mg/dl. Add 10 mg/dl for each decade over 50 years of age.

Growth hormone suppression: glucose tolerance test (serum growth hormone after 100g glucose orally after 8 hr fast) @ <5 ng/ml within 2 hr.

Glucocorticoid suppression: overnight dexamethasone suppression test (8 AM serum cortisol after 1 mg dexamethasone orally at 11 PM) @ 5 mcg/dl.

Glucocorticoid stimulation: cosyntropin stimulation test (serum cortisol 30-90 min after 0.25 mg cosyntropin IM or IV) @ >10 mcg/ml more than baseline serum cortisol.

Gonadal Steroids, plasma:
Androstenedione:
Women: 3.5-7.0 nmol/L=1-2 ng/mL
Men: 3.0-5.0 nmol/L=0.8-1.3 ng/mL
Dihydrotestosterone:
Women: 0.17-1 nmol/L=0.05-3 ng/mL
Men: 0.87-2.6 nmol/L=0.25-0.75 ng/mL
Estradiol: Estradiol-17-beta, adult
Women, basal: 70-220 pmol/L=20-60 pg/mL Follicular @30–100 pg/mL = 110–370 pmol/L. Ovulatory@ 200–400 pg/mL 730–1470 pmol/L. Luteal @50–140 pg/mL = 180–510 pmol/L.
Women, ovulatory surge: >740 pmol/L=>200 pg/mL
Men: <180 pmol/L=<50 pg/mL
Progesterone:
Women, luteal phase: 6-64 nmol/L=2-20 ng/mL
Women, follicular phase: <6 nmol/L=<2 ng/mL
Men: <6 nmol/L=<2 ng/mL
Testosterone:
Women: <3.5 nmol/L=<1 ng/mL
Men: 10-35 nmol/L=3-10 ng/mL

Gonadotropins, plasma:
Follicle-Stimulating Hormone (FSH) :
Women, basal: 1.4-9.6 IU/L=1.4-9.6 mIU/mL
Women, ovulatory surge: 2.3-21 IU/L=2.3-21 mIU/mL

Women, postmenopausal: 34-96 IU/L=34-96 mIU/mL
Men: 0.9-15 IU/L=0.9-15 mIU/mL
Luteinizing Hormone (LH) :
Women, basal: 0.8-26 IU/L=0.8-26 mIU/mL
Women, ovulatory surge: 25-57 IU/L=25-57 mIU/mL
Women, postmenopausal: 40-104 IU/L=40-104 mIU/mL
Men: 1.3-13 IU/L=1.3-13 mIU/mL

Growth Hormone (GH), plasma:
After 100 g glucose orally: <2 mcg/L=<2 ng/mL
After insulin-induced hypoglycemia: >9 mcg/L=>9 ng/mL

17-Hydroxycorticosteroids, urine: Male @ 2-12 mg/24 hr. Female @ 2-8 mg/24 hr.

5'-Hydroxyindoleacetic acid (5'-HIAA), urine: 2-9 mg/24 hr.

Human Chorionic Gonadotropin beta Subunit (beta-hCG), plasma:
Men and nonpregnant women: <3 IU/L=<3 mIU/mL

beta-Hydroxybutyrate, plasma: <300 µmol/L=<3 mg/dL

Insulin, plasma:
Fasting: 35-145 pmol/L=5-20 uU/mL. Insulin levels should drop to basal level 2-3hr post-prandial and be <20. Can indicate significant insulin resistance if insulin levels are 100, even with a normal blood glucose.
During hypoglycemia (plasma glucose <2.8 nmol/L <50 mg/mL) : <35 pmol/L=<5 uU/mL

Insulin C Peptide, plasma: 0.5-2 mcg/L=0.5-2 pg/mL

Insulin-Like Growth Factor I (IGF-I, Somatomedin-C) :
Women: 0.45-2.2 kU/L=0.45-2.2 U/mL
Men: 0.34-1.9 kU/L=0.34-1.9 U/mL

17-Ketosteroids, urine: <8 yo @ 0-2 mg/24 hr. Adolescent @ 0-18 mg/24 hr. Adult Male @ 8-18 mg/24 hr. Female @ 5-15 mg/24 hr.

Lactate, plasma: 0.56-2.2 mmol/L=5-20 mg/dL

Luteinizing hormone (LH) stimulation: gonadotropin releasing hormone (GnRH) test (serum LH after 100 mcg GnRH IM/IV) @ 4- to 6-fold rise over baseline.

Levodopa stimulation test: serum growth hormone after 0.5 g (500mg) levodopa PO while fasting @ >5 ng/ml rise over baseline within 2hr.

Magnesium, serum: 0.8-1.30 mmol/L=1.8-3.0 mg/dL

Metyrapone test: 8 AM serum deoxycortisol after 30 mg/kg metyrapone PO at midnight)@ >7.5 mcg/dl. Metyrapone is no longer available in the US in 2007.

Osmolality, plasma: 285-295 mmol/kg=285-295 mOsmol/L

Oxytocin, plasma:
Random: 1-4 pmol/L=1.25-5 ng/L
Women, ovulatory surge: 4-8 pmol/L=5-10 ng/L

Parathyroid Hormone, serum (Intact PTH using IRMA assay): 10-65 ng/L=10-65 pg/mL. C-terminal @ 150-350 pg/ml. N-terminal @ 230-630 pg/ml.

Phosphorus, inorganic, serum: 1-1.5 mmol/L=3.0-4.5 mg/dL

Pregnanediol, urine: Female Follicular phase @ <1.5 mg/24 hr, Luteal phase @ 2.0-4.2 mg/24 hr, Postmenopausal @ 0.2-1.0 mg/24 hr. Male @<1.5 mg/24 hr.

Progesterone: Female Follicular phase @ 0.02-0.9 ng/ml, Luteal phase @ 6-30 ng/ml. Male @ <2 ng/ml.

Prolactin, serum:
Nonpregnant women and men: 2-15 mcg/L=2-15 ng/mL. Nonpregnant day @ 5-25 ng/ml, night @ 20-40 ng/ml. Pregnant @ 150-200 ng/ml.

Proinsulin: for the dx of insulinoma. Also been shown to be elevated in non-insulin dependent diabetics (NIDDM), newly diagnosed insulin dependent diabetics (IDDM), some older pt's, pregnant or obese diabetics, functional hypoglycemia and hyperproinsulinemia (a rare syndrome). Proinsulin is a 9000 MW protein that is synthesized in the beta cells of the pancreas and is the precursor molecule for insulin. Most proinsulin is converted to insulin and c-peptide, which are secreted in equimolar amounts into the blood. About 15% is not converted and is released as proinsulin. The biological activity of proinsulin is only about 10% of insulin, but the half life of proinsulin is three times as long as insulin. By calculating the molar ratio of proinsulin to true insulin (P/I), a better assessment of beta cell function can be made.
Proinsulin: 2 years and over: 2.1-26.8 pmol/L. Insulin, Fasting: 4-27 µIU/mL. Note: To convert from µIU/mL to pmol/L, multiply by 6.9. The reference intervals are based on a 75 g glucose challenge.

Proinsulin/Insulin Ratio Calculation: Proinsulin/Insulin Ratio as %:
Normal fasting adults: 3.4-21. Normal fasting children: 6.4-16

Pyruvate, plasma: 39-102 µmol/L=0.3-0.9 mg/dL

Radioactive iodine (131I) uptake (RAIU): 5%-25% at 24 hr (varies with iodine intake). Suppression test (RAIU on day 7 after 25 mcg tri-iodothyronine PO 4 times daily) @ <10% to <50% baseline.

Renin Activity, plasma, normal-sodium intake:
Supine: 3.2 ± 1 mcg/L/h=3.2 ± 1 ng/mL/h.
Standing: 9.3 ± 4.3 mcg/L/h=9.3 ± 4.3 ng/mL/h.
Elevated in drugs (thiazides, estrogen, minoxidil), chronic renal failure, Bartter's syndrome, pregnancy (normal), pheochromocytoma, renal hypertension, reduced plasma volume, secondary aldosteronisms.
Decreased in adrenocortical hypertension, increased plasma volume, primary aldosteronism, drugs (propranolol, reserpine, clonidine).

Sodium, serum: 136-145 mmol/L=136-145 mEq/L

Testosterone, total plasma: Bound in adolescent male @ <100 ng/dl. Adult male @ 300-1100 ng/dl. Tanner stage I (7mo-9yo @ <30 ng/dL in male, <10 in female. Tanner Stage 2 (10-13yo) @ <150 male, <30 females. Tanner Stage 3 (14-15yo @ 100-320 in

males, <35 in female. Tanner Stage 4-5 (16-19yo) @ 400-1080 in male, 15-40 in female. Adult Female @ 4-90 ng/dl. Unbound adult male @ 3-24 ng/dl. Female @ 0.09-1.30 ng/dl. To convert to nmol/L, multiply ng/dL by 0.0347.

Thyroid Function Tests:

Free thyroxine estimate: 9-26 pmol/L=0.7-2.0 ng/dL
Radioactive iodine uptake, 24 h: 0.05-0.30=5-30%
Resin T3 uptake, serum: 0.25-0.35=25-35%
Reverse triiodothyronine (rT3), serum: 0.15-0.61 nmol/L=10-40 ng/dL
Thyroid hormone-binding ratio (THBR) : 0.85-1.10=85-110%
Thyrotropin (TSH), serum: 0.5-5 mU/L=0.5-5 µU/mL
Thyroxine (T4), serum: 64-154 nmol/L=5-12 mcg/dL Total @ 4-11 mcg/dl, Free @ 0.8-2.4 ng/dl.
Triiodothyronine (T3), serum: 1.1-2.9 nmol/L=70-190 ng/dL
Thyroxine-binding globulin capacity: 15-25 mcg T4/dl

Thyroid-stimulating hormone (TSH) stimulation: thyrotropin-releasing hormone (TRH) stimulation test (serum TSH after 400 mcg TRH IV) @ >2-fold rise over baseline within 2hr.

Triglycerides, plasma: <1.80 mmol/L=<160 mg/dL

Vanillylmandelic acid (VMA), urine: 1-8 mg/24 hr.

Lab Profiles:

MENOPAUSAL PROFILE
Follicle Stimulating Hormone (FSH)
Luteinizing Hormone (LH)

OVARIAN FUNCTION PROFILE
Dehydroepiandrosterone (DHEA) Sulfate
Estradiol
Follicle Stimulating Hormone (FSH)
Luteinizing Hormone (LH)
Progesterone
Prolactin

POST MENOPAUSAL PROFILE
Estradiol
Follicle Stimulating Hormone (FSH)
Luteinizing Hormone (LH) 2 mL. Serum

TESTICULAR FUNCTION PROFILE
Follicle Stimulating Hormone (FSH)
Luteinizing Hormone (LH)
Prolactin
Testosterone

ANEMIA PROFILE
Iron, Total
Ferritin
Folic Acid
Vitamin B12

COMPREHENSIVE ANEMIA PROFILE
Complete Blood Count
(with zinc protoporphyrin and platelet count)
Iron, Total
Iron Binding Capacity, Total (TIBC)
Percent Transferrin Saturation
Ferritin
Folic Acid
Vitamin B12 3 mL. EDTA (lavender) Whole Blood
Reticulocyte Count

MACROCYTIC ANEMIA PROFILE
Complete Blood Count
(with zinc protoporphyrin and platelet count)
Folic Acid
Vitamin B12 3 mL. EDTA (lavender) Whole Blood
Reticulocyte Count

MICROCYTIC ANEMIA PROFILE
Complete Blood Count
Iron, Total
Iron Binding Capacity, Total (TIBC)
Percent Transferrin Saturation
Ferritin
Reticulocyte Count

PRENATAL PROFILE, BASIC
Complete Blood Count
ABO Blood Group
Rhesus (Rh) Blood Type
Irregular RBC Antibody Screen

Syphilis - Treponema palliduin Antibodies (with reflex RPR on reactive specimens)
Rubella Immunity (qualitative lgG Ab's)
Hepatitis B Surface Antigen (HbsAg)

PRENATAL PROFILE, BASIC, WITH URINALYSIS

Complete Blood Count
(with zinc protoporphyrin, WBC differential and platelet count)
ABO Blood Group
Rhesus (Rh) Blood Type
Irregular RBC Antibody Screen
Syphilis - Treponema pallidum Antibodies (with reflex RPR on reactive specimens)
Human Immunodeficiency Viruses (HIV) 1 & 2 Antibodies
Rubella Immunity (qualitative lgG Ab's)
Hepatitis B Surface Antigen (HbsAg)
Urinalysis with Microscopic Examination

COMPREHENSIVE PRENATAL / OBSTETRIC PROFILE

Complete Blood Count
(with zinc protoporphyrin, WBC differential and platelet count)
Erythrocyte Sedimentation Rate (ESR), Wintrobe
Glucose, Fasting
ABO Blood Group
Rhesus (Rh) Blood Type
Irregular RBC Antibody Screen
Sickle Cell Screen
Syphilis - Treponema pallidum Antibodies (with reflex RPR on reactive specimens)
Rubella Immunity (qualitative lgG Ab's)
Human Chorionic Gonadotropin (hCG), Quantitative, Total and Beta-Subunit Specific
Human Immunodeficiency Viruses (HIV) 1 & 2 Antibodies
Hepatitis B Surface Antigen (HbsAg)
Hepatitis B Surface Antibody (Anti-HbsAg)
Hepatitis B Core Antibodies, Total (lgG + lgM)
Hepatitis B Core lgM Antibody
Hepatitis C Antibodies, (Anti-HCV)
Hepatitis A Antibodies, Total (lgG + lgM)
Hepatitis A lgM Antibody
Urinalysis with Microscopic Examination
Neisseria gonorrhoeae DNA probe
Chlamydia trachomatis DNA probe

URINE DRUG SCREEN

Amphetamines / Methamphetamines
Barbiturates
Benzodiazepines
Cannabinoids (Marijuana)
Cocaine Metabolite (Benzoylecgonine)
Methadone
Methaqualone (Qualuude)
Opiates (Codeine, Morphine, Heroin)
Phencyclidine (PCP)
Propoxyphene (Darvon)
Alcohol, Ethyl

URINE ABUSED DRUG SCREEN

Amphetamines / Methamphetamines
Barbiturates
Cannabinoids (Marijuana)
Cocaine Metabolite (Benzoylecgonine)
Opiates (Codeine, Morphine, Heroin)

BLOOD LEAD SCREENING PROFILE

Lead, Blood 1 mL. Metal-
Zinc Protoporphyrin

HEPATITIS A,B,C DIAGNOSTIC PROFILE

Hepatitis B Surface Antigen (HbsAg)
Hepatitis B Surface Antibody (Anti-HbsAg)
Hepatitis B Core Antibodies, Total (lgG + lgM)
Hepatitis B Core lgM Antibody
Hepatitis C Antibodies, (Anti-HCV)
Hepatitis A Antibodies, Total (lgG + lgM)
Hepatitis A lgM Antibody

ACUTE HEPATITIS MARKER SCREEN

Hepatitis A lgM Antibody
Hepatitis B Surface Antigen (HbsAg)
Hepatitis B Core tgM Antibody

Hepatitis C Antibodies
HEPATITIS B PROFILE
Hepatitis B Surface Antigen (HbsAg)
Hepatitis B Surface Antibody (Anti-HbsAg)
Hepatitis B Core Antibodies, total (lgG + lgM)
Hepatitis B Core lgM Antibody
ARTHRITIS PROFILE
Calcium, Total
Phosphorus, Inorganic
Uric Acid
C-Reactive Protein (CRP)
Rheumatoid Factor (RA Factor)
Anti-Streptolysin 0 (ASO)
Erythrocyte Sedimentation Rate
ARTHRITIS/AUTOIMMUNE PROFILE
Calcium, Total
Phosphorus, Inorganic
Uric Acid
C-Reactive Protein (CRP)
Rheumatoid Factor (RA Factor)
Anti-Streptolysin 0 (ASO)
Erythrocyte Sedimentation Rate (ESR)
Anti-Nuclear Antibody (ANA)
Anti-double-stranded DNA (ds-DNA)
Anti-single-stranded DNA (ss-DNA)
Anti-Jo1
Anti-SSA
Anti-SSB
Anti-Sm (anti-Smith)
Anti-Sm/RNP (anti-Smith/Ribonucleoprotein)
Anti-Scl-70 (Scleroderma)
AUTOIMMUNE PROFILE
Anti-Nuclear Antibody (ANA) Titer and HEp-2 Staining Pattern if Positive
Anti-double-stranded DNA (ds-DNA)
Anti-single-stranded DNA (ss-DNA)
Anti-Jo1
Anti-SSA
Anti-SSB
Anti-Sm (anti-Smith)
Anti-Sm/RNP (anti-Smith/Ribonucleoprotein)
Anti-Scl-70 (Scleroderma)

Reviews:
From Handango:

Link to Reviews posted at Handango (palm version):
http://www.handango.com/PlatformProductDetail.jsp'productType=2&jid=15X5265C5CE795X7E386X3AXB671FF9X&platformId=1&siteId=1&productId=95693&sectionId=0&catalog=1&authorId=70507&review=1
Link to Reviews at Handango (Pocket PC version):
http://www.handango.com/PlatformProductDetail.jsp'siteId=1&jid=15X5265C5CE795X7E386X3AXB671FF9X&platformId=2&productType=2&catalog=0&sectionId=0&productId=95694&review=1

All inclusive resource.
A reviewer from Boise, ID, 2006-06-06 16:03:56
"I have used this software all through Family Medicine Residency and find it to be an all-inclusive resource that is indispensible on the wards and in clinic. The frequent updates ensure that the information I reference is up to date and evidence based. I highly recommend this software."
Your first crutial medical reference to guide you
A reviewer from forward, deployed, 2006-02-13 21:23:00
…….”This document contains all of the knowledge we were looking for then and reinforce with for today, but in our pocket. It"s all text and you"ll need Isilo to run it but you will find yourself using no other reference first. If Dr. Weber"s program doesn"t solve your issue it will point you in the right direction. Besides, we did acquire a higher level of medical knowledge but you can"t regurgitate all of it verbatim. This is your second opinion, reinforcing document, and idle learning tool for your daily activities. It"s all in there, with references to the documents from which they came."
Only reference you'll only need
A reviewer from Las Vegas NV, 2005-08-27 10:43:33
"This reference is unbelievable. I am a board certified family physician who has been working more urgent care over the last year. Thanks to your guide I successfully removed a fish hook from a finger, diagnosed a case of neurosyphilis, and the list goes on. I have on my PDA [many competing products] and I might as well delete them all due to the beauty of this work of art. Thanks again."

Very Helpful
A reviewer from Lakeland, OH, 2005-06-04 12:28:24
"I have purchased many other texts and find this to be the all-around best! No other text has the succinct, complete and clinically relevent material. Most are just a bunch of fluff and pomp. This one give unabashed and clear, concise workups and detailed tx options listing immediate references to evidence based resources. This is the one all-around text to have in one's pocket for nearly every clinical encounter."

Extremely good
mark hand from newcastle, uk, December 2, 2004
"bought this after reading other reviews. had considered 5MCC and others which seemed too expensive and limited coverage of topics. i work in A&E and am training for general practice, this proves to be infinitely useful. the hyperlinks are superb. makes looking up any topic fast and interesting. top marks."

Excellent product
A reviewer from Batavia, IL, October 14, 2004
"This product has served me well both in subspecialty rotations as an internal medicine resident at an academic medical center and now that I'm doing primary care in the community. A truly unique combination of depth, breadth, and accessibility of information. Also kept up to date with journal citations of relevant information."

Forget the rest Get the best ! (Not been paid to say this)
Chief Webmaster from London, England, UK, September 5, 2004
"If you ignore the cosmetic issues, compared with its competition, this text is robust, comprehensive and very usable. Its update policy (unless you understand it carefully) can leave you with non-funtioning software. Some may think it to be overpriced, but in balance probably not, if you consider the range of topics it covers. No doubt, if you are looking for an answer on your PDA you are most likely to find it here. It would be nice occationally to be able to transfer some of its contents to a doc format for teaching purposes. The surgical component is somewhat lacking. But in conclusion, if you are in clinical practise or learning medicine and use a PDA to help you provide clinical information then why have you not tried this brilliant resource yet?"

An outstanding clinical resource!
Elizabeth Ross, MD from Newton, MA, October 9, 2004
"This is the best all-around medical reference program I've found for the Pocket PC. It is easy to search, quite comprehensive, and well-referenced. If I had to buy one program, this would be the one I'd choose."
The one reference I can't live without
Edwin Scott, MD from Augusta, GA, July 1, 2004
I use this reference (not just Clinical Ortho, I have all of them) at least five times a day. It has saved my skin numerous times both in the office and the hospital. It is way better than the Washington Manual, and it is better than 5 Minute Clinical Consult. Excedingly well organized. The author has been most helpful when I have had a problem. You can't afford not to use this if you are in Primary Care.

excellent product
D Lane from tucson, arizona, June 30, 2004
As a primary care provider, this has been an easy to use in clinical settings as such an excellent product. I'd rate it 5 stars, however, receiving updates is problematic. As such I'm rating it 3.

Simply the best
A reviewer from Atlanta, GA, June 8, 2004
Hands down the best Internal medicine program out there. May not have the bells and whistles of other medical programs but more than makes up for it with consistently the most useful and up to date information. I couldn't imagine practicing medicine without it any more.

Wonderful!
Lloyd P. Hitchings, M.D. from Newport News, Virginia, April 14, 2004
I use this every day in my practice and love the quick referral to other specialty areas. This replaces many textbooks that I could not carry to my clinical offices. I cannot imagine being without this on a daily basis.

2 Thumbs Up!
B. Sills, MD from Fort Smith, AR, April 3, 2004
I don't know how the author does it, but he has written a concise, but very comprehensive medical resource for the Palm that is head and shoulders above the "commercial" texts I have seen. Every Family Practitioner should have a copy on their Palm. It's money well spent.

Outstanding Medical Text!
C Rhoades, MD, FACP from Portland, Oregon, February 24, 2004
This is an outstanding product. The program is easy to navigate and very fast on my clie, even when running entirely off of my memory card. This text the most frequently used text in my PDA library (much more useable CLINICAL info than Harrisons or the 5-Minute). Clinical content is solid, at least qd this text has helped me with disease management, dx and tx to make me more productive (and smarter). Great job!

Fantastic!!
A reviewer from MD, Europe, January 25, 2004
It's the best reference program out there. Much more information and clinical relevant pearls. Free updates for 1 year!! New chapters on procedures which is both extensive and up to the point.

Fantastic!
A reviewer from MD, Norway, January 24, 2004
After just a few minutes I had come to a conclusion, this is the best medical referance software for PDA made EVER. This software shouldn't have only 5 stars, but 10!! So much relevant clinical information bundled together in such a way that this software deserves several "medical Oscars".

This text is a "must" for primary care MD's who use PDA's.
Chris Vincent, MD from Seattle, WA, December 9, 2003

The Clinical Medicine Consult is well researched and comprehensive. References are included in the text so you can always find the original source if you need to. On my Sony Clie I also have the Merck Manual, Washington Manual, PEPID, MicroMedix, and 5 Min Clinical Consult, but find I use Dr. Weber's text the most. Very easy to find info on almost any topic, runs from the expansion card, and doesn't require "auto update" software or multiple files. I have used it for 2 years, the updates are frequent and easy to download (sent as an imbedded web link in an email message). This text is a "must" for primary care MD's who use PDA's.

My "Go To" Reference

Bernard Buffett MD from Nova Scotia , Canada, November 2, 2003

This is THE program if you want update concise information NOW.

Best Value for Money

Dr Adrian Loh from Singapore, October 17, 2003

I have used other medical software from "famous names" - at great expense! But none of them compares. Dr Weber's CMC is intuitive and user-friendly, suitable for both quick reference and serious study. Other programmes usually are good for only one or the other. The program is also friendly to the device, not taking up much memory in my Pocket PC relative to the in-depth info packed within. And surprisingly - the info is supported by useful illustrations! I am impressed with how up-to-date the medical info within is; and although from the US, Dr Weber has crafted an integrated text that can be used around the world without bias. If you had to get one program, this is the ONE! Don't waste your money elsewhere!

Superior Medical Application

A reviewer from Whiteville, NC, October 3, 2003

Dr. Weber has created an extremely useful tool for anyone in the medical field. I am an administrator and use this application to keep on top of what my doc's are doing every day in the practice. It helps me to relate better. I find it extremely useful as a reference guide. You don't have to be a doctor to find use in this software. For doctor's this is a must!!! For administrator and office managers the like ..... this is a great way to stay on top of the subject matter. Thanks Dr. Weber!

A solid PDA medical data base that is easy and intuitive to use.

A reviewer from Walnut Creek, CA, May 2, 2003

I am impressed with the volume of information available to me with this textbook. The hyperlinks are great, but you need to have the iSilo document reader to use them. It is well worth the investment. I can search and access information rapidly because it is formatted in an intuitive and logical manner. As a PDA medical textbook it is FAR SUPERIOR to another similar app that I also bought. The latter is tedious and difficult to use on my Tungsten T.

Clinical Medicine Consult is well organized and very thorough.

Julie Toponce PA-S from Albuquerque, New Mexico, February 4, 2003

This is the most complete medical reference I have encounted for handheld computers. As a physician assistant student I have used many different programs to assist my studies while on clinical rotations. Clinical Medicine Consult has been the most useful and by far the most thorough for all of my rotations, ranging from primary care and peds to OBGyn and ER. Thank you for developing such a wonderful tool.

A superb text!

Bryan F. Connelly MD from Needville, TX, July 15, 2003

I am a bit of a reference junkie, and I find myself always going back to the Clinical Medicine Consult as the gold standard. The most complete medical reference I have encounted for handheld computers. The information is concise, up to date, and comprehensive. It is really helpfull and the information is searched very quick. I used to have the Washington Manual and 5MCC with me, that has been changed by CMC! Love the product!!! keep it up!

Worth every penny

Andy Garden MD from Denver, April 16, 2003

I use the daily in my practice. I have found this text to be very useful not only in the clinic, but during hospital admissions/ rounds. Full of very useful and easy to find clinical information. No other text is as complete, useful and yet concise as this. Strongly reccomended. I previously used the iSilo version that had been out for years, but find the mobireader to have many more options such as text size and better quallity illustrations. A must have for any clinician! Keep up the good work -Pacific Primary Care.

The best medical reference guide to have !!!

Fred Witham MD from Portsmouth, VA, July 14, 2002

Packed full of clinically relevent information. Like having a hoard of specialists in my pocket. I look to this first before all other references (both paper and electronic)! Like the frequent updates.

Simply the best--bar none

A reviewer from Atlanta, GA, January 8, 2003

Best clinical palm application around. Extremely easy to use with best informations. Updated regulary to include the latest, useful knowledge. I use this everyday in my practice.

Comprehensive, easy and fun to use, up to date. First-rate!

M.Towbin, M.D. from Weed, California, October 27, 2002

Having been in practice for 20+ years, it is a pleasure to use such a comprehensive, current, interesting and fast program as Clinical Medicine Consult. It is tailor-made for my busy rural ER practice, and has exactly what I'm looking for in the wee hours of the morning when a difficult case comes in. It runs smoothly on my palm m130, and the graphics and links quickly give me the answers I need on a variety of topics. This is truly an excellent point of contact reference for busy clinicians.

Easy, fast and comprehensive

dmarkbeatty@hotmail.com from milwaukee,wi, June 20, 2002

I find the program very useful and access it frequently. Sometimes it broadens my approach to medical problems, and also lets me know of therapeutic options I may not have considered.

Solid PDA medical data base that is easy and intuitive to use.

A reviewer from Walnut Creek, CA, May 2, 2003

I am impressed with the volume of information available to me with this textbook. The hyperlinks are great, but you need to have the iSilo document reader to use them. It is well worth the investment. I can search and access information rapidly because it is formatted in an intuitive and logical manner. As a PDA medical textbook it is FAR SUPERIOR to another similar app that I also bought. The latter is tedious and difficult to use on my Tungsten T.

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Julie Toponce PA-S from Albuquerque, New Mexico, February 4, 2003
This is the most complete medical reference I have encounted for handheld computers. As a physician assistant student I have used many different programs to assist my studies while on clinical rotations. Clinical Medicine Consult has been the most useful and by far the most thorough for all of my rotations, ranging from primary care and peds to OBGyn and ER. Thank you for developing such a wonderful tool.
A very good on-the-ward medical information. I would recommend it.
John Do from Orange County, CA, October 5, 2002
I use it daily for lookup and refresh medical information while I am at the hospital and rounding with my attending. John Do, MS4. Osteopathic Medicine
Best medical text on the PDA market
RA. Smith MD from Pensacola, FL, September 25, 2002.
I have found this very useful, frequent updates, easy to search. I always look to this text before any other source, even consultants. Very practical.
It's an excellent program
khiemlai@hotmail.com from Santa Ana, CA, April 14, 2002
docflyingace@aol.com from Chicago, September 1, 2001
It is easy to get detailed, practical info on most any clinical dz. It has succinct reviews of not only the pathophysiology and physical exam finding, but incredible detail of how-to work-up and treat almost everything. Stuff even my attendings and most specialists lack. I also use this in the ER and in clinic evaluating pts and while writing admission orders and progress notes. It is full of detailed tx and differential dx. It also has details of meds, labs and studies. This new index has taken it to a new level, very easy to navigate. I highly recommend this to any fellow or resident in internal medicine. It has much more detail and a wider range of topics than the 5MCC or any other text on the market. My only only complaint is that I wish I had this a few years ago!
Ray Ralston MD from Seattle, WA, August 24, 2001
CMC is a fabulous program! I have tried the Wash manual and 5MCC and neither compare to the CMC. The information is more relevant and thorough and much, much better indexed. It is full of pearls by speacialists. It is definitely worth the money. Everytime a new topic comes up during a discussion, I am always amazed to find the answers (plus a great deal of "extras") in CMC. It is a great program- I highly recommend it.
Anonymous, August 2, 2001
Incredible value, a must have for all primary care providers.
Elegant
TLWilkensMD@aol.com from San Diego, CA, August 4, 2001
I utilize this so frequently that I feel lost without this text on the handheld in my pocket. This text has nearly every detail to manage 99% of what any primary physician sees. It covers all the basics with incredible subspecialty details on even unusual topics and physical findings. A must for every resident and medical student as well. Dr. Weber and his team of speacialist have outdone any medical text on the market, including those in print! Keep up the great work.

From PalmGear.com:

PalmGear Review Link: http://www.palmgear.com/index.cfm'fuseaction=software.showsoftware&SID=2416B773-CF18-ADC9-1E9137B05BC293F2&PartnerREF=&prodid=64588
27-Aug-05 by Jeff Marcovitch
This reference is unbelievable. I am a board certified family physician who has been working more urgent care over the last year. Thanks to your guide I successfully removed a fish hook from a finger, diagnosed a case of neurosyphilis, and the list goes on. I have on my PDA a Harrison's, a Cecil's, a Merck manual, a Harriet Lane, etc and I might as well delete them all due to the beauty of this work of art.Thanks again.
19-Aug-05 by Jay Baker MD
Very useful text. I strongly recommend this to any practicing physician. It gives clear, current and concise recommendations on most any medical problem imaginable. It goes far beyond the superficial recommendations that most of the other "leading" textbooks contain. I find the navigation of this text to be simple and intuitive. This text has superior content and a usefulness value for any primary care physician or student in medicine, even a benefit to the sub specialist looking for information outside their area of expertise.
11-Feb-05 by Bilal Naseer
This is simply the best and most upto date medical database that I've ever used on my palm. The best thing is that there is hardly any topic that is not covered. Every topic is reviewed in detail with references to medical journals or other resources.
It has great images with so many topics.
I highly recommend it to everyone, especially those interested in internal medicine. I use it all the time during our busy residency rounds and calls and I don't have to carry any books with me anymore. I would have given it ten stars if there was an option here.
22-Sep-04 by Richard Carpenter DO
CMC is fantastic. I have been using it for two years and I would be lost without it. I can review the latest tx's on a disease or get up to speed on a rare condition in a few seconds between rooms. The quarterly updates keep CMC on the cutting edge.
02-Jul-04 by Jeff White,MD
I purchased the entire Clinical Medicine Consult after first using the Cardiology module, which was very impressive. The hyperlinked format is remarkably intuitive--I ALWAYS find the subject or information I'm seeking without having to back-track or search again. The amount of information is much more complete than my 5MCC, although significantly more scrolling is required. Excellent resource to brush up on those clinical subjects that one sees infrequently in primary care.
16-Apr-04 by Joshua Morganstein
I reviewed this application several years ago after it's first inception and thought I'd post something a bit more updated. There isn't much I can say that hasn't already been noted by the many reviewers before me. There is simply no other medical reference available for the handheld platform that comes close to the thoroughness, accuracy, clinical applicability, and usefulness of Clinical Medicine Consult. The outstanding index feature, detailed procedures, as well as the highly-referenced and regularly updated

content make this a must have for any medical professional using a PDA in practice. And, the price is staggeringly low for a resource of this caliber. You'll use the Clinical Medicine Consult more than all your other handheld resources combined!

01-Feb-04 by Jeffrey Shames MD

Very well written e-book. I have print copy of 5-minute clinical consult which I have used in my office for some time, and I wasn't sure that the transition to a palm-based program would be useful. I'm glad I made the switch. The coverage of medical topics is very broad and in-depth, and the hyperlinks make it almost impossible to put the darn thing down!

30-Jan-04 by Jeff Williams MD

I cannot count how many times this text has assisted my in the care of my pt's. I have found this text to be indispensable! It is the most up to date, user friendly, concise and yet comprehensive text on the market. Having the Clinical Medicine Consult in my pocket puts the fun back in practicing medicine. The specialist who have contributed to this text hold nothing back, which is very rare in current times. This texts allows me access to evaluate and treat both complex and common complaints and with so many usable pearls. It is a must for all who practice medicine! The frequent updates are a plus. I cannot say enough.Thanks.

29-Oct-03 by David Pitts MD

After using this anything else is disappointing. This is THE program to have on your PDA. All the info, all the pearls are here.

03-Oct-03 by M. Syno

Dr. Weber has created an extremely useful tool for anyone in the medical field. I am an administrator and use this application to keep on top of what my doc's are doing every day in the practice. It helps me to relate better. I find it extremely useful as a reference guide. You don't have to be a doctor to find use in this software. For doctor's this is a must!!! For administrator and office managers the like ..... this is a great way to stay on top of the subject matter. Thanks Dr. Weber!

29-Jun-03 by Maurice Bernstein MD

The amount, variety and usefullness of the content is surely worth the price. I teach first and second year medical students and I have found the program of great value to quickly look up the answer to a student question I had forgotten or never even learned. Best of all, Dr. Weber's updates, gives me confidence that the answers will be reasonably current and accurate.

11-Apr-03 by Phillip Burrer

This IS the BEST medical software I have ever seen on a handheld device. No with Isilo for Windows you can view Dr. Weber's CMC on your desktop. Since purchasing this product I barely (if ever) look at all my Skyscape software. If you only got the bucks for one reference than THIS IS IT!

04-Apr-03 by Dr.Deborah Hilterman

Definitiley the most comprehensive and clinically based program out there. I have trialled them all and since from New Zealand didn't want a program with too much US influence in terms of the drug trade names offered. This suited me best. I hope that when my year is up Carl Weber and the team withh endeavour to make purchasing future versions at a reduced price for those of us who have shown support.

27-Oct-02 by Martin Towbin

Having been in practice 20+ years, it is a pleasure to use such a comprehensive,up to date, fast, and interesting reference as Clinical Medicine Consult. It is tailor-made for my busy rural ER practice, and provides me with the answers I need on a variety of topics when a difficult case comes in. Dr. Weber not only has nicely addressed many of the little details I'm always looking up in reference books that someone else has just "borrowed", but he also has neatly included nuances about the latest common tx regimens. The program runs smoothly on my Palm m130 and the graphics,links and bookmark functions make it a breeze to use. It is an excellent point of contact reference for any busy clinician.

25-Oct-02 by Jan Cornell

I've used this reference for quite a while now and I just wanted to write & say it's the most complete and practical of any I've tried yet. I am a bit of a reference junkie, and I find myself always going back to this as the gold standard. This is laid out in a very functional manner, and the indexing is superb. Consider me a believer. Jan Cornell, M.D.

03-Oct-02 by Victoria Abdelnur

It´s an extremely useful tool! Practical, complete. I use it almost every day (for more than a year now) at the hospital. I´m a medicine student in her final year. I also like that its updates come thru e-mail. Thank you for developing such a wonderful product.

11-Sep-02 by Andrew Bowman

A wonderful product. I am the charge nurse in a busy emergency department and have found this to be a great teaching tool at the bedside for pt's and myself. It is nice to be able to review a medical condition before going to see the pt. Thanks Dr Weber!

02-Jun-02 by David Ragan

I have been in practice 15 years, and this software is excellent. Practical advice, referenced, succinct. Now, when studying a topic I read what CMC says first.Great for hospital work; updates are a plus.

15-Apr-02 by Joshua Morganstein

Unbelievable! Dr. Weber has continually updated this masterful, concise etext with relevant, well-referenced information. I've been using CMC since it's inception and I am more impressed with each revision. The latest, v2.2, adds a section on procedures (step-by-step!) as well as various studies, their uses, and differential diagnoses for various abnormal labs and studies. Outstanding!!! I look forward to each update and, because it uses iSilo and words flawlessly from memory cards, I never run out of room, despite the amount of valuable data Dr. Weber packs into this thing! I have tried and purchased dozens of medical references, utilities, etc. I can say, without hesitation or reservation that, for the money, Dr. Weber's etext is hands-down the best medical reference available for the PalmOS handhelds!

14-Apr-02 by Roland Leon

As a defense attorney in the area of medical malpractice/personal injury, I use this program daily in my law practice. CMC is the best medical reference in the market--and I have tried them all! Thank you Dr. Weber for an outstanding product at a reasonable price.

16-Jan-02 by John Tapp MD

I am a second year Internal Medicine resident who has been using this program for several mo's. I find that it is indispensable and far superior to any other references that I have on my Palm. I use this program several times a day and the Index feature make this product very user friendly. I also appreciate the fact that the program is updated and has current references at the end of most topics. This program is well worth the price.

12-Jan-02 by Christopher Gn

Awesome!! I run a family practice with a heavy case load and this program gets all thumbs up in terms of clinical content and concise practical information. And its all relevant. I am constantly being amazed by the amount of information and clinical pearls included in it. It is an invaluable tool to me in my Family practice and its like a hoard of consultants in my pocket! Keep up the great work!

12-Nov-01 by Matt Wilkinson MD

As a med-peds resident, CMC is easily my most valuable medical program - I usually use it several times a day and rarely ever refer to 5MCC anymore. It is outstanding, thorough, current, and has a great index! Cheers! Dr. Weber has my full support. Thanks!!

05-Nov-01 by George Valdez

It is well established that textbooks are several years outdated by the time they come out. The Clinical Medical Consult series is constantly updated. I have used the Palm OS for years, and am familiar with most every reference text available. This one approaches ideal for its purpose - to be used on the go. When I look through the text, I am able to rapidly find, learn and make better-educated decisions. Indexes and hyperlinks bring it to a whole new level of efficiency. I come away from this reference feeling like I made a quick call to the specialist. Usually after a major effort looking through a text or journal, I am able to cull a few useable tidbits that almost relate to my question. Who's got time for that? And, on those times I refer to the specialist, I can pose better-educated questions. There is no text that is continuously revised and updated, which covers such a vast array of subjects, at such a cheap price.

24-Sep-01 by Catherine Shoff, DO

This program is outstanding! I am a medicine resident and use it frequently during rounds and morning report. I am amazed by the wealth of information I can access now - it is easier to navigate than the Wash Manual and goes into more depth too. Now that clinical med consults is indexed, I can find what I'm looking for in seconds! I have spread the word to other residents in my program and would recommend this software to anyone who wants the most information possible at their fingertips for a great price!

28-Aug-01 by Ryan Smith

I have been using CMC for a few mo's in my medicine residency. I am amazed how a few clicks often gets me the info I'm looking for, even right in the middle of rounds, or admitting in the ED. This is the way of the future, and Carl Weber is ahead of the curve. The text is fairly well referenced, and specialists apparently have edited the text. There are things I've looked for that aren't in the text, but there is an amazing amount that is present. The hyperlink function is great, and it actually can search. I have it on a visor with the text stored on removable flash rom memory. That way, it takes up nearly no system memory. This is still slightly unstable, though, since one database file on iSilo has to remain on the system memory, and DON'T try to edit anything on a flash rom.

06-Aug-01 by William Medrano

I am a Family Practitioner in Austin, Tx. Have been in practice 17 years. I see pretty much the full spectrum of diseases/illness including infants to the elderly. The more I use this program, the more I am amazed at its comprehensiveness and detail. It is well thought out and easy to use and fast. The only program I use more is Epocrates and I have a large number of programs I have been trying out (ie 5MCC/5MPC/Current Medical Theraputics/Common Simple Emergencies/MedCal/Peddose etc). It works great using MemPlug/PiDirect (I am using 64meg SmartMedia card). Dr Weber gets my vote for best full spectrum medical program for the palm. Thank you Dr Weber!

04-Aug-01 by Ben Weilix

Truly an exceptional compelation! This progam makes my handheld invaluable. It is encyclopedic, yet precise and up to date in how to manage everything from the emergency to the mundane to the unusual d/o's. Great work in creating this.

07-Jul-01 by C Worley, MD

CMC is a fabulous program! I have tried the wash manual and 5-MCC and neither compare to the CMC. The information is more relevant and thorough and much, much better indexed. It is definitely worth the money- I carry no other texts (or reference cards) in my pockets during rounds - an amazing feat for a resident. Everytime a new topic comes up during a discussion, I am always amazed to find the answers (plus a great deal of "extras") in CMC. It is a great program- I highly recommend it.

07-Aug-01 by Chris Lewis, RN, MSN, ARNP

This program is a marvel in my practice. I use it every day. It incorporates detailed and thorough information that I can access in a few clicks, and get what I need. The detail is fabulous, and up to date with the latest in criterion for dx and tx's. I have recommended this software to my entire graduating class of MSN students, as well as every physician that I have worked with. I highly recommend this software to any and all in the medical field, from RN and ARNP to PAs and Physicians.

Commonly Used Abbreviations:

Links: C | E | J | N | S |

~ = ~

1/2 NS = 0.45% saline solution
5-HIAA = 5-hydroxyindoleacetic acid
5-HT = serotonin
17-OHCS = 17-hydroxcorticosteroids
AAA = apply to affected area, abd aortic aneurysm
ac = ante cibum (before meals)
ABG = arterial blood gas
Ab = antibody
ABI = ankle brachial index (in PVD)
Abx = Abx's
ac = before meals
ACD = anemia of chronic dz
ACS = Acute coronary syndromes
ACT = activated clotting time
ACTH = adrenocorticotropic hormone
Ad = R ear (aurio dextra)

ad lib = as needed or desired
AD = autosomal dominant. Alzheimers Dz. Atopic Dermatitis.
ADH = antidiuretic hormone
ADL = activities of daily living
AF = atrial fibrillation
AFB = acid fast bacillus
AK = actinic keratosis
Aka = otherwise know as
AP = alkaline phosphatase
AR – autosomal recessive
ARR = absolute risk reduction
ALL = acute lymphocytic leukemia
ALT = alanine amino-transferase
am = morning
AMA = against medical advice
AMI = acute myocardial infarction
AML = acute myelogenous leukemia
amp = ampule
AMV = assisted mandatory (mode) ventilation
ANA = antinuclear antibody
ante = before
AP = anteroposterior, alk phos
AR = autosomal recessive
ARB = angiotensin receptor blocker
ARDS = adult respiratory distress syndrome
ARF = acute renal failure
ASA = acetylsalicylic acid, aspirin
ASO = antistreptolysin
AST = aspartate amino-transferase
AVB = atriovenous block
AVM = atrial venous malformation
AVN = avascular necrosis
BAL = blood alcohol level
BBB = bundle branch block
BCC = basal cell carcinoma
BID = bis in die (twice a day)
B12 = vitamin B-12
BM = bowel movement, bone marrow
BMD = bone mineral density
BMI = body mass index
BMR = basal metabolic rate
BMT = bone marrow transplant
BNP = B-type natriuretic peptide
BP = blood pressure
BPH = benign prostatic hypertrophy
BS = bowel sounds
BUN = blood urea nitrogen
BSA = body surface area
BSO = Bilateral Salpingo-Oophorectomy
Bx = biopsy

C

CA = cancer
Cal = calorie (kilocalorie)
c/o = complaint of
c cum (with)
C / S or C & S = culture and sensitivity
C = centigrade
Ca = calcium
CAD = CAD = coronary heart disease (CHD)
CAM = Complementary and alternative medicine
CAP = capsule, community acquired pneumonia
CBC = complete blood count
CBT: cognitive behavioral therapy.
CBZ = carbamazepine
cc = cubic centimeter, creatinine clearance
CCB = calcium channel blocker
CCU = coronary care unit
CF = cystic fibrosis
CFU = colony forming units
Chem 7 = basic metabolic panel, 17 = comprehensive, which includes electrolytes, renal and liver function tests.

New Medicare Labs:
Electrolyte Panel (#4): Na, K, Cl, CO2.
Basic Metabolic Panel (BMP): #8 items: Chem 7 (SMA-7) with Ca.
Renal Panel: #10 BMP + Alb & PO4.
Comprehensive Metabolic Panel (CMP) #14: BMP + Alb, AP, AST, ALT, Tb, Dbili.
Extended CMP (ECMP) #17: CMP +Mg, P, LDH.
Hepatic Panel: Alb, Tbili, Dbili, AP, Tp, ALT, AST.
cm = centimeter
CMV = cytomegalovirus
CNS = central nervous system
CO2 = carbon dioxide
COPD = chronic obstr pulm dz
CP = chest pain
CPD = cephalopelvic disproportion
CPK-MB = myocardial-specific CPK
Cr = creatinine
CrCl = creatinine clearance
CRI = Chronic renal insufficiency = CRF = ESRD
CRF = chronic renal failure = CRI
CSF = cerebrospinal fluid
CT = computerized tomography
CTA = cotton tip applicator
CTD = connective tissue disease
CTX = contraction
CV = cardiovascular
CVA = cerebrovascular accident, costovertebral angle
CVD = cardiovascular disease
CVP = central venous pressure
Cx = culture
CXR = Chest x ray
DA = dopamine
d/c = discharge or discontinue
D5W = 5% dextrose water solution
DBP = diastolic blood pressure
dec = decrease or decreased
DIC = dissemin. Intravasc Coagulation
Diff = differential cell count
DHP = dihydropyridine, a CCB
DJD = degenerative joint disease
DKA = diabetic ketoacidosis
dL = deciliter
DM = diabetes mellitus
DNR = do not resuscitate
D/o = Disorder(s)
DOC = drug (tx) of choice
DOE = dyspnea on exertion
DOT = directly observed therapy
Doxy = doxycycline
DT's = delirium tremens
DTR = deep tendon reflex
DVT = deep vein thrombosis
Dx = dx
Ddx = differential dx
DUB = dysfunctional uterine bleeding
Dz = disease
EBV = Epstein Barr virus
ECG = electrocardiogram = EKG
ECT = electroconvulsive therapy
ED = Emergency Department
EDC = estimated date of confinement (due date)
EE = ethinyl estradiol
EEG = electroencephalogram
EGA = estimated gestational age
ELISA = enzyme-linked immunoabsorbent assay
EM = erythema multiforme
EMB = endometrial biopsy
Emyc = erythromycin
EN = erythema nodosum
EPO = erythropoietin
EPS = extra pyramidal sx's

ERCP = endoscopic retrograde cholangiopancreatography
ERT = estrogen replacement therapy
ESR = erythrocyte sedimentation rate
ESRD = End-stage renal dz
ESLD = End-stage liver dz
ETT = endotracheal tube
ETD = eustachian tube dysfunction
ETOH = alcohol
Fb = foreign body
FBS = fasting blood sugar
F/C = fever and chills
FEV1 = forced expiratory volume (1 sec)
FHT/ FHR = fetal heart tones/ rate
FiO2 = fractional inspired oxygen
FOB = fecal occult blood
FSP = fibrin split product
FVC = functional vital capacity
Fx = fx(‘s)
G = gram(s), G- = Gram negative.
GC = gonococcal; gonococcus
GBS = GBBHS = Group B Beta Hem Strep
GFR = glomerular filtration rate
GH = growth hormone
GI = Gastrointestinal
gm = gram
GN = glomerular nephritis
gt = drop
gtt = drops
GU = genitourinary
h or hr = hour
H20 = water
HA = H-A
Hb = hemoglobin concentration
HCO3 = bicarbonate
HCG = human chorionic gonadotropin
HCT hematocrit
HCTZ = hydrochlorothiazide or hydrocortisone
HCW = Health Care Worker
HDL = high-density lipoprotein
HF = heart failure
Hg = mercury
HI = homicidal ideation
HIV = human immunodeficiency virus
hr = hour
H/o = history of.
HOCM = HCM = hypertrophic cardiomyopathy
HPF = high power field
HR = heart rate
HRT = hormone replacement therapy
HS = hora somni (bedtime)
HSM = hepato-splenomegaly
HSP = Henoch-Schönlein purpura
HTN = hypertension
HUS = hemolytic uremic syndrome
Hx = history
I & D = incision and drainage
ICP = intracranial pressure
IBD = inflammatory bowel disease
IBS = irritable bowel syndrome
IBW = ideal body weight
ICP = intracranial pressure
ICU = intensive care unit
IDA = iron deficiency anemia
ILD = interstitial lung disease
IM = intramuscular
inc = increase or increased
IgM = immunoglobulin M
IMV = intermittent mandatory ventilation
INH = isoniazid
INI = if not improved, RTC.

INR = International normalized ratio
I & O = intake and output
IOP = intraocular pressure
IU = international units
IV = Intravenous
IVD = IV drug
IVF = IV fluids, in-vitro fertilization
IVP = IV pyelogram, IV piggyback
JRA = juvenile rheumatoid arthritis
K = potassium
kcal = kilocalorie
KCL = potassium chloride
Kg = kilogram
KOH = potassium hydroxide
KUB = x-ray of abdomen
L = liter
lb = pounds (1kg = 2.2 lb)
LBBB = left bundle branch block
LPB = lower back pain
LDH = lactate dehydrogenase
LDL = low-density lipoprotein
LE = lower extremity
LFT = liver function tests
liq = liquid
LLSB = left lower sternal border
LLQ = left lower quadrant
LMN = lower motor neuron
LMP = last menstrual period
LN2 = liquid nitrogen
LOC = loss of consciousness
LP = Lumbar Puncture
LR = lactated Ringer's
LV = left ventricle
LVH = left ventricular hypertrophy
MAP = mean arterial pressure
mEq = milliequivalent
MD = muscular dystrophy
Mg = magnesium, milligram, myasthenia gravis
MgSO4 = Magnesium Sulfate
MI = myocardial infarction
MIC = minimum inhibitory concentration
mL = milliliter
mm = millimeter, multiple myeloma, morbidity & mortality (M&M)
MOM = Milk of Magnesia
MR = mitral regurg
MRI = magnetic resonance imaging
MS = mitral stenosis, multiple sclerosis, mental status, morphine sulfate
MSE = mental status exam
MTX = methotrexate
MVP mitral valve prolapse
Na = sodium
NaHCO3 = sodium bicarbonate
NE = norepinephrine
Neuro = neurologic
NCV = nerve conduction velocity
NGT = nasogastric tube
NKA = no known allergies
NM(J) = neuro muscular (junction)
NMT = no more than (maximum dose)
NLT = no < (minimum age to use drug)
NPH = neutral protamine, normal pressure hydrocephalus
NPO = nulla per os (nothing by mouth)
NS = normal saline solution (0.9%)
NSAIDs = nonsteroidal anti-inflammatory drugs
NTG = nitroglycerine
N/V/D = nausea, vomiting, diarrhea
NYHA = New York Heart Association, class heart failure
NWB = non-weight bearing.
OCD = obsessive compulsive d/o
OCP = oral contraceptive pill

OD = right eye, overdose, optometrist
ODT = orally disintegrating tablet
oint = ointment
OR = odds ratio or operating room
OS = left eye (oculus sinister)
Osm = osmolality
OT = occupational therapy
OTC = over the counter (no Rx med)
OU = each eye
oz = ounce
p = post after, Phosphate
pc = post cibum (after meals)
PA = posteroanterior, pulmonary artery
PAC = premature atrial contraction = APB (atrial premature beat) or SVE (supraventricular ectopic) or Pulmonary artery catheter
PaO2 = arterial oxygen pressure
pAO2 = partial pressure of oxygen
pc = after meals
PCA = pt controlled anesthesia
PCN = penicillin
pCO2 = partial pressure of carbon dioxide
PE = pulmonary embolism, physical exam
PEEP positive end-expiratory pressure
per = by
PFO = patent foramen ovale
PFS = patella-femoral syndrome
PFT = pulmonary function test
PG or PGE = prostaglandin
pH = hydrogen ion concentration (H+)
PID = pelvic inflammatory disease
PM = afternoon
PND = paroxysmal nocturnal dyspnea
PO = orally, per os
pO2 = partial pressure of oxygen
Post cib = after meals (post cibos)
polys = polymorphonuclear leukocytes
PP = pathophysiology
PPD = purified protein derivative
PPI = proton pump inhibitor
PR = per rectum
PRN = pro re nata (as needed), as the occasion arises.
PT = physical therapy, pro-thrombin time
Pt = P atient pt's = p atient's
PTCA = percutaneous transluminal coronary angioplasty
PTT = partial thromboplastin time
PV or Px = prevention
PUD = peptic ulcer disease
PVC = premature ventricular contraction
PVD = peripheral vascular disease
q = (every) q2h = every 2hours
qac: before every meal
QID = quarter in die (four times a day)
qAM = every morning
qd = quaque die (every day)
qh = every hour
qHS = every night before bedtime
QID = 4 times a day
Ql = as much as desired (quantum libet)
qmo: every month
qnoon: every noon
QOD = every other day
qs = quantity sufficient
qwk: every week
qt = quart
R = right
RAD = right axis deviation
RAE = right atrial enlargement
RCT = randomized clinical trial
R/O = rule out
RA = rheumatoid arthritis, room air, right atrial
RAST = radioallergosorbent test

RCT = randomized controlled trial, can be single or double blinded.
RF = rheumatic fever
RMSF = rocky mountain spotted fever
RPR = syphilis test
RR = Respiratory rate
RRR = relative risk reduction
ROM = range of motion
RSD = reflex sympathetic dystrophy
RTA = renal tubular acidosis
RTC = return to clinic (pt to come back)
RV = right ventricle, residual volume
s = sine (without)
s/p = status post
sat = saturated
SBE: subacute bacterial endocarditis
SBP = systolic blood pressure or Spontaneous Bacterial Peritonitis
SC = SC = SQ
SD = standard deviations
SE = side effect
SED = SE discussed (chart if start a new med), ESR
SES = socioeconomic status
s.g. = specific gravity (on a U/A)
SI = suicidal ideation
SIADH = syndrome of inappropriate antidiuretic hormone
SJS or SJ = Stevens Johnson syndrome
SK = seborrheic keratosis
SL = sublingually under tongue
SLE = systemic lupus erythematosus, slit lamp exam
SMA-7, 10, 17 = sequential multiple analysis
SMX = sulfamethoxazole
SOB = shortness of breath = DOE
sol = solution
SPEP = serum protein electrophoresis
SQ = under the skin, sub-cutaneous = SC.
SR = sustained release
S(S)RI = selective serotonin reuptake inhibitor
S/s = signs and sx's of disease
SSC = squamous cell carcinoma
STAT = statim (immediately)
Statin = HMG CoA-reductase inhibitor such as simvistatin.
STD = sexually transmitted disease
susp = suspension
T-½ = half life (of a drug)
Td = tetanus-diptheria vaccine.
TCN = tetracycline
TID = ter in die (three times a day)
T4 = Thyroxine level
tab = tablet
TB = tuberculosis
Tbsp = tablespoon
TCA = tricyclic antidepressant
Temp = temperature
TFT's = thyroid function studies.
TIA = transient ischemic attack
TKO = to keep open, an infusion rate (~500 mL/24h) just enough to keep the IV from clotting, **not** the same as saline lock or Heplock.
TL = toxic level
TMJ = temporomandibular joint
TMP = trimethoprim
TMP-SMX = trimethoprim-sulfa-methimazole
TPA = tissue plasminogen activator
TS/ TR = tricuspid stenosis/ regurge
TSH thyroid-stimulating hormone
tsp = teaspoon
TPN = total parenteral nutrition
TSS = toxic shock syndrome
TT = thrombin time
TTE = trans-thoracic echocardiography
TTP = tender to palpation
TWI = T-wave inversion.

Tx = T reatment
U = units
UA = uric acid
U/A or Ua = urinalysis
UC = ulcerative colitis
UD = as directed
UDS = urine drug screen (tox)
mcg = microgram
UFH = unfractionated heparin
ULN = upper limits of normal
um = micrometer
UO = urine output
URI = upper respiratory infection
UPEP = urine protein electrophoresis
U/S = Ultra-sound
USPSTF = The U.S. Preventive Services Task Force
UTI = Urinar y tract infection
UV = ultraviolet light
V = vitamin, Vit-C, Vit-E, V-B6 etc.
VAC vincristine, adriamycin, and cyclophosphamide
vag = vaginal
VC = vital capacity
VDRL = Venereal Disease Research Laboratory
V fib = ventricular fibrillation
VGE = viral gastroenteritis
VLDL = very low-density lipoprotein
Vol. = volume
VS = vital signs
VSD = ventricular septal defect
VT = ventricular tachycardia
VTE = venous thromboembolism (PE, DVT)
WBC = white blood count
w/o: without
W/u = work-up (labs and studies to order)
X = times
Yo = years old = years of age.
Zn = zinc

Pirated Copies
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Diagrams (Partial Listing):

Neurology | **Facial Sensory Nerve Roots** | Upper-front Dermatomes & Cutaneous nerves | **Sensory – upper extremity -back** | Sensory – Lower extremity -front | **Sensory – Lower extremity-back side** | Sensory Patterns | **Pyramidal Tract Dz** | Brachial Plexus | **Carpal Tunnel Injection** | Cerebral Circulation Diagram | **Migraine** | Cervical radicular pain diagram | **Lumbosacral Root Compression Diagram** | Piriformis Syndrome | **Sacroiliac Dysfunction** | Cardiac auscultation | Heart Murmur location | Male Urology Anatomy | **Penile** | **Penumonia CXR Findings** | **Bone anatomy** | Finger anatomy | Wrist Bones | **Shoulder** | Knee Aspiration | **Knee Exam** | Ankle exam | Pain Scale | Peripheral Smears | **RBC Inclusions** | **WBC Abnormalities on Smears** | FNA Breast | **Bartholin Abscess** | Canalith Repositioning | **Vascular Access Anatomy** | PA Cath Patterns | **Skull & Birth Injuries** | ECG Interpretation | Fish Hooks | Four Cup Test (prostatitis) | Peak Flow | Nursemaids Elbow | Blepharitis | **Electrophoresis & Protein** | **Foot Bones** | **Acetaminophen Nomogram** | **Salicylate Nomogram** | Normal Fundus | **Glaucoma** | Eye Movement | Entropion & Ectropion | Visual Fields | Abdominal Organs | Coronary Artery | Heart Diagram | Early Repolarization | **ECG in Pulmonary Embolism** | RBBB | **LBBB** | **QRS ECG Axis** | **ST-T** | Physical Child Abuse | **Penile Nerve Block** |

Alphabetical INDEX:

Links: A: B: C: D: E: F: G: H: I : J: K: L: M: N: O: P: Q-R: S: T: U: V: W: XYZ: Studies & Sx's | **Procedures** | ACLS / BLS | **MEDS** | Abbreviations | **Diagrams** |

A: Links: **Ad:** **AL:** **An:** **Ao:**

A-1 Antitrypsin |
AAA – 12 - Steps |
A1c |
A-a Gradient |
Abd Distention / Masses |
Abd Pain |

Abd X-rays |
Abdominal Aortic Aneurysm (AAA) |
Abducens Palsy (CN 6) |
Aberrant Origin of Coronary Artery |
Abortion |
Abscess - abd & rectal |
Abscess - Brain/ Spinal / Epidural
Abscess - Breast |
Abscess - Skin |
Acanthosis Nigricans |
Accessory Navicular |
Acclimatization |
ACEi |
Acetaminophen-tox |
Acetaminophen-peds |
Achalasia |
Achilles Tendon |
Achondroplasia |
Acid Fast Stain |
Acid-Base Physiology |
AC Joint |
ACL Tear |
ACLS Drugs |
ACLS |
Acne Keloidalis Nuchae |
Acne Rosacea |
Acne Vulgaris |
Acne |
Acoustic Neuroma |
Actinic Keratosis |
Activated Charcoal |
Acute Arthritis |
Acute Cholecystitis |
Acute Otitis Media |
Acute Phase Reactants |
Acute Radiation Syndrome |
Acute Renal Failure |
Acute Respiratory Failure |
Acute Rheumatic Fever |
Acute/ ascending Cholangitis |
AD |
Adaptive Equipment |
Adeno-Keratoconjunctivitis |
Adenosine |
Adenovirus |
ADHD |
Adies |
Admissions |
Adnexal Mass |
Adolescence & Puberty |
Adrenal Insufficiency |
Adrenal Mass |
Adrenal |
Adult Pancytopenia |
Advanced Care |
Adverse Drug Reactions |
Afferent Pupillary Defect |
A-Fib |
A-Flutter |
African Sleeping Sickness |
Agglutination Assays |
Aggressiveness |
Aging Theories |
AIDS |
AIN |
Air Travel |
Akathisia |
Al |
Albumin |
Alcohol |

Alcoholic Liver Dz |
Alcoholic Hepatitis |
Alcoholic Ketoacidosis |
Aldosterone |
Alkylating Agents |
ALL |
Allergic Alveolitis/ Pneumonitis |
Allergic Conjunctivitis (SAC) |
Allergic Rhinitis |
Allergy Shots |
Allergy |
Alopecia Areata |
Alpha Blocker |
Alpha-1 Antitrypsin |
Alport's |
ALS |
Alternative Medicine |
Alternating Cover |
Altitude Sickness |
Alveolar (Air Space dz) Infiltrates |
Alveolar Hemorrhage Syndromes |
Alzheimer's Disease (AD) |
Amaurosis Fugax |
Amebic Abscess |
Amebic |
Amenorrhea |
American to SI |
AMI |
Aminoglycosides |
Amiodarone (Cordarone) |
AML |
Ammonia |
Amnesia |
Amphetamines |
Ampho |
Amputations
Amylase & Lipase |
Amyloid |
AN |
ANA |
ANA Patterns |
Anabolic Steroids |
Anaerobes |
Anaerobic/ Aspiration |
Anagen Effluvium |
Anal Ca |
Anal Fissure |
Anal Fistula |
Anal Skin Tag |
Anaphylactoid |
Anaphylaxis |
ANCA's |
Andropause |
Androstenedione |
Anemia of Chronic Dz |
Anemia |
Anesthesia |
Aneurysm - Intracranial |
Aneurysm – Aortic and Peripheral |
Angina |
Angina Pain |
Angioedema |
Angry Pt |
Animal Bites |
Animal Allergy |
Anion Gap (AG) |
Anisakiasis |
Anisocoria |
Ankle Brachial Index |
Ankle |

Ankylosing Spondylitis |
Ann Arbor/ Cotswold Stage |
Anorexia |
Anorgasmia |
Antacid |
Anthrax VAC |
Anthrax |
Antiarrhythmic Drugs |
Antibiotic & URI's |
Antibiotic Resistance |
Antibiotics |
Antidotes |
Antifungals |
Antihistamine |
Antimotility |
Antioxidants |
Antiphospholipid |
Antitussive |
Antivirals |
Anxiety D/o |
Ao |
Aortic Aneurysm |
Aortic Dissection |
Aortic Stenosis
AP |
Apgar |
Aphasic Pt |
Aplastic |
Appendiceal Torsion |
Appendicitis |
Aphthous |
Apolipoproteins
AR / AI |
Autosomal Recessive (AR) |
ARB |
ARDS |
Arenaviridae |
Argyll Robertson |
Aromatase Inhibitors |
Arrhythmia |
Art of Medicine |
Arterial Blood Gas (ABG) |
Arterial Lines |
Arterial Ulcers |
Arteriosclerosis |
Artificial Sweeteners |
AS |
ASA |
Asbestosis |
Ascites |
Ascitic Fluid Analysis |
Ascorbic Acid (Vit-C) |
Aseptic Meningitis |
Aspergillosis |
Aspirin (acetylsalicylic acid) |
AST/ALT |
Asteatotic Dermatitis |
Asterixis |
Asthma |
Asystole |
A-Tach |
Ataxia |
Ataxia-Telangiectasia |
Atelectasis |
Athetosis |
Athlete Collapse's |
Athletes |
ATLS |
ATN |
Atopic Dermatitis (Eczema) |

Atrial Fibrillation |
Atrial Septal Defect |
Atrophic Vaginitis |
Atypical Anti psychotics |
Audiometry |
Autism |
Autoimmunity |
Autoantibodies |
Autoimmune Hepatitis |
Autoimmune Hemolytic Anemia |
AVB |
AVNRT & AVRT |
Axis Classification |
Azoospermia |
B | Links | Bi | Br |

BNP |
B12 |
Babesiosis |
Bacteria- common |
Bacterial Overgrowth |
Bacterial Vaginosis |
Bacteriology Report |
Bad Breath
Bad News |
Baker Cyst |
BAL |
Balanitis |
Bands / Metamyelocytes |
Barbae |
Barbiturates and Other Drugs
Barotrauma |
Barrett's |
Bartholin Gland |
Bartonella |
Basal Body Temp |
Basal Cell Carcinoma |
Basal Metabolic Rate |
Basic Wound Care |
Basophil |
Basophilia |
Basophilic Stippling |
Bathe Technique |
BCG |
Bed Bugs
Bedside Clues |
Behavioral & Developmental Issues |
Behçet's |
Belching |
Bell's Palsy |
Benign Transient Global Amnesia |
Benzo's |
Bereavement |
Berylliosis |
Beta-blocker |
b-hCG |
Bezoars |
Bl |
Biceps Tendonitis |
Bicycling |
Bile Acid Sequestrant |
Bili |
Biliary Colic |
Biliary |
Binswanger |
Biofeedback |
Biologic Response Modifiers |
Biological Weapons |
Biotin |
BIPAP |

Bipolar |
Birth Injuries |
Bite Wounds- animal |
Bites/ Stings- insect |
Black Widow |
Bladder Relaxants |
Bladder Retraining |
Bladder Scan |
Bladder Tap |
Blakemore-Sengstaken Tube |
Bleeding |
Blepharitis |
Blepharospasms |
Blister Cells |
Blistering Lesions |
Blister- sports |
Blood / Transfusions |
Blood Cx |
Blood Tubes |
Blowout Fracture |
Blue Bloater |
BM Sample |
BMI |
BNP **|**
Body Dysmorphic d/o |
Body Piercing |
Boerhaave's |
Bone Marrow Transplant |
Bone Mineral Density |
Bone Tumors |
BOOP |
Botulism |
Botox |
Boutonniere Deformity |
Bowel Irrigation |
Bowel Prep |
Bowels |
Boxer's Fx |
BPH |
BPV |
Br |
Brachial Plexus |
Brady |
Brain Abscess |
Brain Tumors |
Brain Death |
Brain Mets |
Branchial Cleft Cysts & Fistulas |
BRCA gene |
Breast Cancer |
Breast Exam |
Breast Feeding |
Breast Mass |
Breast Pain |
Breast |
Breast cancer in men
Breath Holding |
Breath Sounds |
Breech Presentation |
Bretylium |
Bronchiectasis |
Bronchiolitis Obliterans (BO) |
Bronchiolitis |
Bronchitis |
Brown recluse Spider |
Brucellosis |
Bruising |
Bruxism |
BSA |
Budd Chiari |

Buerger's Dz |
Brugada Syndrome / Criteria |
Bulbar Palsy |
Bulimia |
Bulling |
Bullous Oral |
BUN |
Bunions |
Bunyaviridae |
Burning Mouth |
Burns |
Burr |
Bursitis |
Butalbital Combo |

C | Links | CC | Chi | Co | Cr |

C. difficile |
CABG |
Cabot Rings |
CAD |
Café au Lait Spots |
Café Coronary |
Caffeine |
CAH |
Calcific Tendonitis |
Calcinosis cutis |
Calciphylaxis |
Calcitonin |
Calcium (Ca) |
Calcium Channel Blocker |
Calcium- nutrition |
Callus |
Caloric Density |
Calorics |
Campylobacter
Canalith Repositioning |
Cancer Pain |
Cancer-Gyn |
Cancer-all types |
Cancidas (Caspofungin) |
Candida-vaginal |
Candida-invasive |
Candidiasis |
Candida Hypersensitivity |
Candidiasis (Thrush) Stomatitis |
Captopril Test |
Carbamazepine OD |
Carbapenems |
Carbohydrates |
Carbon Monoxide |
Carbuncle |
Carcinoid |
Carcinoma of Unknown Primary |
Cardiac Contusion |
Cardiac Death, Sudden |
Cardiac Risk Stratification |
Cardiac Testing |
Cardiology |
Cardiogenic Shock
Cardiomyopathy |
Cardioversion |
Carditis & Tamponade |
Carotids and Carotid artery stenosis |
Carotid & Vertebral Dissection |
Carotid (Hypersensitivity) Sinus Syndrome |
Carpal Dislocations |
Carpal Tunnel |
Castleman's |
Cat Scratch Dz |
Cataracts |

Cathartics |
Catheter Sizes |
Catheterization |
Catheters |
Catheter Infection |
Cauda Equina |
Cauliflower Ear |
Cavernous Sinus Thrombosis & Syndrome
Cognitive Behavioral Therapy (CBT) |
CC |
CCU Admit |
CDL Physicals |
CEA |
Celiac Sprue |
Cellulitis |
Central Alpha-agonists |
Central Lines |
Central Retinal Vein Occlusion |
Cephalgia (H-A) |
Cephalosporins |
Cerebellar Testing |
Cerebral Palsy |
Cerumen |
Cervical Cancer |
Cervical Incompetence |
Cervical Spinal Stenosis |
Cervical Spondylotic Myelopathy |
Cervical-Lumbar Pain |
Cervix, Ovary & Vagina |
Cervix |
Cestode |
Chaga's Dz |
Chalazion |
Challenge Testing |
Challenging/ Problem Person |
Chancroid |
Charcot Foot |
Charcot-joint |
Charcot-Marie-Tooth |
Cheilitis |
Chemical Sensitivity Syndrome |
Chemical Burns |
Chemo Acronyms |
Chemo COMPLICATIONS |
Chemotherapy |
Cherry Angiomas |
Chest Tube |
Chest X-ray Analysis |
Chi |
Chiari |
Chickenpox |
Chiggers/ Mites
Chikungunya |
Child Abuse |
Childhood |
Chimerism |
Chlamydia |
Chlamydia Pneumonia |
Chloride |
Chocolate |
Cholangitis |
Choledocholithiasis |
Cholesterol D/o |
Cholesterol Emboli |
Cholera- VAC |
Chondromalacia Patellae |
Chorea |
Chorionic Tumors |
Chromium |
Chronic Angina |

Chronic Bronchitis |
Chronic Cholecystitis |
Chronic Daily H-A |
Chronic Fatigue Syndrome |
Chronic Granulomatous Dz |
Chronic Mucocutaneous Candidiasis |
Chronic Pancreatitis |
Chronic Renal Failure |
Chronic Vaginitis |
Churg-Strauss |
Ciguatera |
Ciliary Dyskinesia
Circumcision |
Cirrhosis |
Claudication |
Clavicular Fracture |
Claw Hand |
Cleft Lip & Palate |
CLL |
Clomiphene |
Closed Angle Glaucoma |
Clostridium Infections |
Club Drugs |
Club Foot |
Clubbing |
Cluster H-A |
CML |
CMV |
CNS |
CNS Vasculitis |
Co |
Coag Path |
Coal Worker |
Coarctation of the Aorta |
Coarctation |
Cobalt |
Cocaine |
Coccidioidomycosis |
Cockroaches |
Coenzyme Q10 (Ubiquinol)
Cogan |
Cold Agglutinin |
Colic |
Collagenous Colitis |
Collateral Ligaments |
Colloid |
Colonoscopy |
Colony Stimulating Factors |
Color Vision |
Colorectal Ca |
Colposcopy |
Coma Cocktail |
Coma |
Combined Immunodef |
Combined Variable |
Comfort Care |
Compartment Syndrome |
Compendium |
Competency |
Complement Levels |
Complement System |
Complementary & Alternative Medicine |
Compliance |
Compression stockings |
Comvax |
Concussion |
Conduct D/o |
Confidentiality |
Conflict Managing |
Congenital Heart Defects |

Congenital Heart block |
Congenital Nevi |
Congenital Pulmonary Stenosis |
Connective Tissue Diseases |
Constipation |
Constrictive Pericarditis |
Contact Dermatitis |
Contraception |
Contrast Nephropathy |
Contusions |
Conversion D/o |
Conversions |
COPD |
Copper |
Cornea |
Cor Pulmonale |
Cord Compression |
Corneal Abrasion |
Corneal Ulcer |
Corns |
Corporis & Cruris |
Costochondritis |
Cosyntropin Stim Test |
Cough |
Coumadin |
Counseling |
Coxiella Burnetii |
CPAP |
CPK Elevation |
CPPD |
CR |
Crackles (Rales) |
Cranial Nerves |
CN 3 (Occulomotor) Palsy |
CN 4 and CN 6 Palsy
CN 7 (Facial Nerve) Palsy |
Creatine |
Creatinine |
CREST Syndrome |
Cri du Chat |
Cricothyroidotomy |
Crigler-Najjar |
Crohn's Dz |
Croup |
CRP |
Crutches |
Crying |
Cryptococcus |
Cryoglobulinemic |
Cryoprecipitate |
Cryosurgery |
Cryotherapy |
Cryptorchidism |
Cryptococossis |
Crystalloid |
CSF Studies |
C-spine X-rays |
Cubital Tunnel Syndrome |
Culdocentesis |
Cushing's |
Cutaneous Larva Migrans |
Cutaneous Signs of Systemic Dz |
Cutaneous Vascular Lesions /(birthmarks) |
CVP |
Cyanide
Cyanosis |
Cycloplegics (Dilation) |
Cyclothymic |
Cystatin C |
Cystic Fibrosis |

Cystic Renal Disease |
Cysticercosis |
Cystinuria |
Cystometry |
Cystosarcoma |
Cystoscopy |
Cystourethrography |
Cytochrome P-450 System |
Cytogenic |
Cytokines |
Cytology |

D | Links | Di | DK |

D&C |
D/c Note |
Dacryoadenitis |
Dacryocystitis |
Dacryostenosis |
Dalteparin |
DASH Diet |cutc
Dawn Phenomena |
DC Cardioversion |
D-dimer |
De Quervain's |
Deaf Pt |
Debridement
Decongestants |
Defective Cell Wall |
Dehydration-Peds |
Dehydration |
Dehydroepiandrosterone (DHEA) |
Delirium |
Delta Gap |
Delusions of Parasitosis |
Demanding Pt's |
Demented/ Rambling Pt |
Dementia |
Demyelinating Diseases |
Dengue
Dental Appliances |
Dental Blocks
Dental Infection |
Depo-Provera |
Depression |
Dequervain's Thyroiditis |
Derm |
Dermabond |
Dermatitides |
Dermatitis artefacta |
Dermatitis Herpetiformis |
Dermatofibroma |
Dermatology |
Dermatomal Testing |
Dermatophytes |
Dermatophyte Test Medium (DTM) |
Dermatophytid (Id) |
Developmental Dysplasia of HIP |
Dexamethasone Suppression Test |
DHEA |

DI |

Diabetes Insipidus |
Diabetes Mellitus |
Diabetic Nephropathy |
Dialysis |
Diaper Dermatitis |
Diaphragms - Contraception |
Diaphragm & Pleura |
Diphragmatic Paralysis |
Diarrhea Pediatric |
Diarrhea Adult |
Diarrhea |

DIC |
Dietary Supplements |
Diets, “special” |
Dieulafoy |
Diffuse Esophageal Spasm |
DiGeorge Anomaly |
Digoxin (Lanoxicaps, Lanoxin) |
DIP Dislocation |
Diphtheria |
Diplopia |
Disability |
Disabled Pt |
Discitis |
Discoid SLE |
Disequilibrium |
Dislocated Knee |
Dislocated Mandible |
Dissociative Fugue |
Dissociative Identity D/o |
Distorted Vision |
Disulfiram (Antabuse) |
Diuretics |
Diverticulitis |
Dix-Hallpike |
Dizziness |
DK |
DKA |
DLCO |
DM Foot Care |
DM Infections/ Foot/ Ulcer |
DM neuropathy |
DMARDs |
DNR |
Doctor-Pt Relationships |
Dolls Eye |
Domestic Violence |
Donepezil (Aricept) |
Double Vision |
Downs |
DPEJ |
Dracunculiasis |
Drains |
Drowning |
Drug Abuse
Drug Fevers |
Drug Induced SLE |
Drug Reactions |
Dry Eyes |
Dupuytren’s Contracture |
Dust Mites |
DVT |
Dwarf Tapeworm |
Dyshidrotic Eczema (Pompholyx) |
Dying Process |
Dysarthria |
Dysentery |
Dyslexia |
Dysmenorrhea |
Dyspareunia |
Dyspepsia/ GERD |
Dyspepsia |
Dysphagia |
Dysplastic Nevi |
Dyspnea/ ARDS |
Dyspnea-Terminal |
Dysthymic |
Dystonia |
Dysuria |
E | Links | EM | **ER |**
Ear Foreign Body |

Ear Lac |
Ear |
Early Repolarization |
Eating D/o |
Eaton-Lambert |
EBV |
ECG |
Echinococcus |
Echo |
Eclampsia |
ECT |
Ecthyma Gangrenosum |
Ectodermal Dysplasia Syndromes |
Ectoparasites in travelers |
Ectoparasites |
Ectopic ACTH |
Ectopic Pregnancy |
Eczema Variants |
Edema |
EDTA Test |
EEG |
Ehlers-Danlos |
Ehrlichiosis |
Eisenmenger's Syndrome |
Ejection Clicks |
Elbow Dislocation |
Elbow |
Elder Abuse |
Elderly Abuse |
Elderly Falling |
Electrical |
Electrolytes |
Electronystagmography |
Electrophoresis |
Electrophysiologic Testing |
ELISA |
EM |
E-mail |
Emergency Airways |
Emergency Contraception |
Emergency Procedures |
EMG
Empathy |
Emphysema |
Empyema |
Encephalitis |
Encephalopathy |
Encopresis
End the Visit |
Endemic Typhus |
Endocarditis |
Endocrine |
Endometrial Bx
Endometrial Ca |
Endometriosis |
Endophthalmitis |
Endocrine tumors |
Enemas |
Enoxaparin (Lovenox) |
ENT |
Entamoeba histolytica = Amebic |
Enteropathic |
Enterovirus
Enuresis
Environmental/ Exposures |
Eosinophilia |
Eosinophils |
Eosinophilic Esophagitis |
Eosinophilic Pneumonia Syndromes |
EP Study |

Epidemic Typhus |
Epidermal inclusion cyst |
Epididymitis |
Epiglottitis |
Epiglottitis-pediatric |
Episcleritis |
Episiotomy |
Epispadias |
Epistaxis |
EPO |
EPS |
ERCP |
ER |
Erectile Dysfunction |
Ergots |
ERVL |
Erysipelas |
Erythema ab igne |
Erythema Multiforme |
Erythema Nodosum |
Erythematous Oral |
Erythrasma |
Erythrocytes |
Erythroderma |
Erythromelagia
Esophageal Ca |
Esophageal Cancer |
Esophageal Disease |
Esophageal Diverticula |
Esophageal Dysphagia |
Esophageal Hypermotility D/o |
Esophageal Varices |
Esophagitis |
ESR |
Essential Thrombocythemia (ET) |
Estrogen Patches |
Ethambutol |
Ethnic Groups |
Ethylene Glycol |
ETOH Abuse |
Eustachian tube Dysfunction |
Euthanasia |
Evidence-Based Medicine |
Exam Liver dz |
Exercise (Treadmill) Stress Test (EST) |
Exercise Induced Asthma |
Exercise Radionuclide Cardiac Angiography (RNCA) |
Exercise Stress Echo |
Exercise |
Exercise Physiology |
Exfoliative Dermatitis |
Exophthalmos (Proptosis) |
Expectations |
Exposure Keratopathy |
External Eye |
Extrapyramidal Side Effects |
Extravasation |
Eyelid Fb |
Eyelid Dermatitis |
Eye Movement Desensitization and Reprocessing (EMDR) |
Eye Twitching |
Eyelid Abnormalities |
Eyelid Swelling |
Eyelids |
Eyelid Dermatitis
F | Links | Fi | FO |
FAB Class |
Fabry Disease |
Facial Nerve Palsy |
Facial Trauma |

Factitious Illness |
Factors |
Failure (CHF) |
Failure/ Encephalopathy |
Falling |
Familial Hypocalciuric Hypercalcemia |
Familial Mediterranean Fever |
Family Therapy |
Fanconi |
Fasciolopsis |
72hr Fast |
Fat Embolism |
Fat Necrosis |
Fatigue |
Fatigue-Cancer |
Fatty Acids |
Fb- rectal & Vaginal |
Fb-wound |
Febrile Seizure |
Fecal Discoloration |
Fecal Impaction |
Fecal Incontinence |
Fecal Occult Blood (FOB) |
Feeding Tubes |
FEF |
Felon |
Female Athlete Triad |
Female Hormone Ranges |
Femoral Artery |
Femoral Neuropathy |
FENa |
Fentanyl |
Ferritin |
Fertility Awareness Monitoring |
Fetal Alcohol |
Fetal Testing |
FEV |
Fever and Rash |
Fever |
FFP |
Fi |
Fibrates |
Fibroadenoma |
Fibrocystic Breast |
Fibroids |
Fibroids |
Fibromyalgia |
Fick Principle |
Filamentary Keratopathy |
Filarial |
Filoviridae |
Finasteride |
Fingers |
Fish Hooks |
Fish Ingestions |
Fish Tapeworm |
Fish |
Flail Chest |
Flat Feet |
Flatulence |
Flaviviridae |
Flesh Eating |
Flex Sig |
Floaters |
Flow Volume Loops |
Fluconazole |
Fluids |
Fluoride |
Flushing
Flying & Diving |

FO |
Folate |
Folic Acid |
Folliculitis |
Food Allergy |
Food Poisoning |
Food Pyramid |
Foot |
Foot Drop |
Foot Nerves |
Forearm Ischemic Test |
Foreign Bodies |
Foreign Body Sensation |
Fournier's |
Fox-Fordyce Dz |
Fractures |
Fragile X |
Fredrickson Classification |
Free Water |
Freiberg's Infarction |
Friction Blisters |
Frostbite |
Frozen RBC |
Frozen Shoulder |
Fundus & Internal Eye |
Fungal -Disseminated |
Fungal Rhinosinusitis |
Fungal Sinusitis |
Fungal tx-topical |
FUO |
Furuncle |
Futile Care |
FWBC |
G | Link | GF |
G- aerobic bacilli |
G- aerobic coccobacilli |
G- aerobic diplococci |
G+ aerobic bacilli |
G6PD |
Gait Eval |
Galactocele |
Galantamine (Reminyl) |
Gallbladder |
Gallstones |
Gambling D/o |
Gamekeeper's Thumb |
Ganglion Cyst |
Gas Gangrene |
Gas Embolism – Venous / Arterial |
Gastric Cancer |
Gastric Lavage |
Gastrinoma (ZE Syndrome) |
Gastritis |
Gastrocnemius |
Gastroparesis |
Gaucher's disease **|**
Gonorrhea-GC |
Glasgow Coma Scale = GCS |
Gell & Coombs |
Generic Drugs |
Genetics |
Genital Warts |
Geographic Tongue |
Geriatric Syndromes |
Geriatrics & End of Life |
Gestational DM |
Gestational HTN |
GF |
GFR |
GGT |

GH Deficiency |
GI Bleed |
GI Cancers |
GI Decontamination |
GI Meds |
GI |
Giardiasis |
Gilberts |
GIK |
Gingiva |
Glare |
Glaucoma |
Gleason Score |
Globulins and Protein |
Glossitis |
Glucagonoma |
Glucose Monitoring |
Glucose Control in CCU/ICU |
GnRH Analogs |
Goiter |
Gonorrhea |
Goodpasture's |
Gout |
GP IIb/ IIIa Blocker |
Gram + Cocci = GPC |
Grams Stain |
Granuloma Annulare |
Granuloma Inguinale |
Granuloma |
Granulomatous |
Graves |
Grief Reaction |
Griseofulvin |
Groin Strains |
Growth Hormone Excess (acromegaly) |
Growth |
Guillain-Barré |
Gut Endocrine tumors |
GVHD |
Gyn Pain |
Gynecomastia |
H | Links | Hi | Hyp |
H Flu |
H. pylori |
H/H |
H2RA |
H-A Meds |
Habitual Abortion |
Haglund's Heel |
Hair |
Haldol |
Halitosis |
Hallux Limitus |
Halo Nevus |
Hamstring Strain |
Hand arthritis |
Hand Dermatitis |
Hand nerves-muscles |
Hand-Foot & Mouth (Coxsackievirus) |
Hand-Foot Syndrome – Chemo tox |
Hangover |
Hantavirus |
Haptoglobin |
HARRT |
Hashimoto's |
Hep-A = HAV |
Hazardous Herbs |
HbA1c
Hb C |
Hb E |

Hb SC |
hCG |
HDL |
Hep-B = HBV |
Hep-C = HCV |
Hep-D = HDV |
Heel Cord Tightness |
Hearing Loss |
Heart Failure |
Heart Transplantation |
Heat Edema |
Heat Injury |
Heat Rash |
Heat Stroke |
Heat Therapies |
Heinz |
HELLP |
Hemarthrosis |
Hematoma - abd |
Hematoma - auricular |
Hematoma - intracranial |
Hematoma - Septal |
Hematoma - Subungual |
Hematoma - Birth Injuries |
Hematospermia |
Hematuria |
Heme-Onc |
Hemianopia |
Hemochromatosis |
Hemolytic |
Hemophilia |
Haemophilus Influenza (HiB)- VAC |
Hemoptysis |
Hemorrhagic Fevers |
Hemorrhoids |
Henoch Schönlein Purpura |
HEPA |
Heparin
Heparin Bridging |
Hepatic Cysts |
Hepatic Encephalopathy |
Hepatitis A- VAC |
Hepatitis B-VAC |
Hepatitis |
Hepatomegaly |
Hepatopulmonary Syndrome |
Hepatorenal Syndrome |
Herbal Meds |
Hereditary Fever |
Hernias |
Herniation - ICP |
Herpes Simplex Virus |
Herpes Zoster Ophthalmicus
Hep E = HEV |
Hep-G = HGV |
HHNKS |
Hi |
Hiatal Hernia |
HiB Infections |
Hiccups |
Hidradenitis Suppurativa |
High Altitude Sickness |
Hip Avulsion Injury |
Hip Bursitis |
Hip Fx |
Hip Pain |
Hippus |
Hip-Thigh |
Hirschsprung's |
Hirsutism |

Histiocyte |
Histiocytosis |
Histo |
HIT |
HIV Meds |
HIV-AIDS |
HIV Dementia |
HJR |
HLA & Disease |
HLA Typing |
Hoarseness |
Hodgkin's |
Homocysteine |
Hookworm |
Hordeolum/ Stye |
Hormonal Agents |
Hormone Replacement Regimens |
Horner's Syndrome |
Hospice |
Hospital D/c Summary |
Hospitalist Care |
Hot Flashes |
Howell-Jolly |
HPV - warts |
HPV - cervical |
Hysterosalpingography-HSG |
HSP |
HTN Meds |
HTN urgency & Emergency |
Human Bites |
Human Parva B19 |
Humoral Hypercalcemia |
Hunter's & **Hurler's syndrome** |
Huntington's Disease |
HUS |
Hydrocele |
Hydromorphone (Dilaudid) |
Hydrocephalus |
Hydronephrosis |
HYHA Class |
Hymenoptera |
Hyp |
Hyper IgE Syndrome |
Hyperaldosteronism |
Hyperbaric Medicine |
Hypercalciuria |
Hypercapnia |
Hypercoagulability W/u |
Hypercoagulability |
Hyperemesis gravidarum
Hypereosinophilic Syndrome |
Hyperhidrosis |
Hyper-IgM |
Hyperkalemia |
Hypermagnesemia |
Hypermobility Syndrome |
Hypernatremia |
Hyperoxaluria |
Hyperphosphatemia |
Hyperpigmentation |
Hypersensitive Pneumonitis |
Hypersensitivity Syndrome (AED's) |
Hypersomnia |
Hypersplenism |
Hypertension & Eclampsia |
Hypertension |
Hyperthyroid |
Hypertrophic Cardiomyopathy |
Hypertorphic Pyloric Stenosis |
Hyperuricemia & Urate Nephropathy |

Hyperuricuria-Gout |
Hyperventilation Syndrome
Hyperviscosity Syndrome |
Hypervolemia |
Hyphema |
Hypoaldosteronism |
Hypocalcemia |
Hypochondriasis |
Hypocitruria |
Hypodermoclysis |
Hypoglycemia |
Hypogonadism in Boys |
Hypogonadism in Girls |
Hypokalemia |
Hypomagnesemia |
Hypomotility D/o |
Hyponatremia |
Hypo-parathyroid
Hypophosphatemia |
Hypopigmentation |
Hypopituitarism |
Hypospadias |
Hypotension |
Hypothenar Hammer Syndrome |
Hypothermia |
Hypothyroid |
Hypovolemia |
Hypoxemia |
Hypoxia |
Hysterectomy |
I | Link | In |
Inspiratory Capacity |
ICD = Implantable Cardioverter-Defibrillator
Ichthyosis |
ICU Notes |
ICU-FEN |
Iliopsoas Tendonitis / bursitis |
Infectious Dz |
IDA |
Idiopathic Fibrosing Interstitial Pneumonia |
Id Reaction |
Indirect immunofl assay- IFA |
IgA def |
IgA Nephropathy |
IgG Subclass Def |
IL-2 & 3 |
Ileus |
Iliotibial Band Syndrome |
IM Estrogen |
Immune Complex Dz |
Immune Deficiency |
Immuno (Western) blot |
Immunoelectrophoresis |
Immunofixation |
Immunoglobulins |
Impaired Physicians |
Impaired Vision |
Impetigo |
Impingement Syndrome |
IN |
Intermittent Mandatory Ventilation -IMV |
Incompetent Cervix |
Incontinence |
INF |
Infarct |
Infection Control |
Infertility |
Inflammatory Bowel |
Influenza- Flu VAC |
Influenza |

Informed Consent |
INH |
Inhalants |
Inhalational |
Injections |
Innocent Murmurs |
INR Monitoring & Adjustments |
Insect Bites |
Insect Allergy |
Insomnia |
Instruments and Sutures |
Insulin Drips |
Insulin |
Insulinoma |
Integrative |
Intercostal Anesthesia |
Intermittent Claudication |
Internuclear Ophthalmoplegia |
Intersection Syndrome |
Interstitial Cystitis |
Interstitial Infiltrate / Parenchymal Patterns |
Interstitial Lung Dz |
Intertrigo |
Intestinal Obstructions |
Intra-aortic Balloon Pump (IABC) |
Intraductal Papilloma |
Intranasal Lidocaine |
Intrauterine Device |
Intraosseous infusion |
Intron-A |
Intubation |
Intraocular Pressure |
Intussusception |
Ipecac |
Iodine Def |
Iodine |
Iris Color |
Iron |
Irritable Bowel
Ischemic Colitis |
Isotretinoin (Accutane) |
ITP |
Itraconazole |
IV Immune Globulin (IVIG) |
IV Solutions |
IVP |
J |
Jammed Finger |
Japanese B Encephalitis |
Jaundice |
Jehovah Witness |
Jersey Finger |
Jet Lag |
Jodbasedow disease |
Joint Aspiration & Injection |
Joint Ranges of Motion |
JRA |
Junctional Nevi |
Junctional Rhythm |
JVD |
K |
Kaposi's Sarcoma
Katayama fever
Kartegeners Syndrome
Kawasaki's |
KCl |
Kegels |
Keloids |
Keratocanthoma
Ketoconazole |

Keratosis pilaris |
Kidney Biopsy |
Kidney Transplant |
Kienbock's Disease |
Killip Class |
Klebsiella |
Kleptomania |
Knee arthritis |
Knee Aspiration |
Knee injection |
Knee Bursitis |
Knee Effusions |
Knee |
Köhler's Dz |
Korsakoff Syndrome |
Kyphosis |
L | Lf | LM |
Labor |
Labyrinthitis |
Lactic Acidosis |
Lactose Intolerance |
Lacunar |
LAFB |
Laryngitis |
Lymphangioleiomyomatosis (LAM) |
Language Barriers |
Lantus (Glargine) |
Laryngeal Mask |
Lateral Epicondylitis |
Latex |
LBBB |
L-Carnitine |
LDH |
Lead |
Left Ventricular Hypertrophy (LVH) |
Leg Cramps |
Legg-Calve-Perthes |
Leg-Length Discrepancy
Leg Ulcers |
Leg Pain/ Injury |
Legionella |
Leiden |
Leishmaniasis |
Lemierre's |
Lens Dislocation |
Lensometer |
Leprosy |
Leptospirosis |
Lesbian
Lesser-Toe Deformities |
Leukemia |
Leukocyte Poor (Washed) RBC |
Leukocytoclastic |
Leukocytosis |
Leukokeratosis |
Leukopenia |
Leukostasis |
Lewy Body |
LF |
LFT's |
Lice |
Lichen Nitidus |
Lichen Planus |
Lichen planus- vaginal |
Lichen Sclerosis |
Lichen Simplex Chronicus |
Lichen striatus |
Lid Laceration |
Light Reflex |
Lightening |

Lightheaded |
Limping |
Lipoid Pneumonia |
Lip Laceration |
Lip Lesions |
Lipedema |
Lipids |
Lipoma |
Lipoprotein/ chol Types |
Lips Eczema |
Lispro (Humalog) |
Listeriosis |
Lithium |
Lithium OD |
Livedo Reticularis |
Liver Bx |
Liver Mets |
Liver Palpation |
Liver Transplant |
Liver Tumors |
Liver |
Living Will |
LM |
LMWH |
Loop Diuretics |
Low Back Pain |
Lower GIB |
LPFB |
Ludwig's |
Lumbar Neuropathy |
Lumbar Puncture |
Lung Cancer |
Lung Volumes |
Lupus |
LVG |
Lyme Disease |
Lyme VAC |
Lymphadenitis |
Lymphadenopathy |
Lymphangitis |
Lymphatics |
Lymphedema |
Lymphocytes |
Lymphocytic Colitis |
Lymphocytic choriomeningitis virus (LCMV)
Lymphocytosis |
Lymphoma |
M | MC | **Mi** | **MR |**
Macrolides |
Macular Degeneration |
Maddox Rod Test |
Magnesium (Mg) |
Magnesium |
Majocchi's Granuloma |
Majocchi's |
Malabsorption |
Malaria |
Male Hypogonadism |
Male Infertility |
Malignant Ascites |
Malignant Effusions |
Malignant Hyperthermia |
Malignant/ Pre- Lesions |
Malingering |
Mallet Finger |
Mallory-Weiss |
Malnutrition |
Malpractice |
Mamillary Duct Ectasia |
Mammo Report |

Mammography |
Mandible Fx |
Manganese |
March anemia |
Marcus Gunn |
Marfan |
Marfan's Syndrome-cardiac |
Marijuana |
Marine Injuries & Bites |
Mass Hysteria |
Massive Hemothorax |
Mastitis |
Mastocytosis |
Mastoiditis |
Maximal Breathing Capacity |
MC |
MCV |
MDI Use |
Measles |
Mechanical Ventilation |
Meckel's |
Meconium |
Medial Epicondylitis |
Medial Tibial Stress |
Median Nerve |
Mediastinal Mass |
Medically Unexplained Sx's |
Mediterranean Diet |
Meds & Pregnancy |
Megaloblastic |
Melanoma |
Melatonin |
Melioidosis |
Membranous-GN |
Memory |
MEN |
Ménière's |
Meningitis |
Meningococcal VAC |
Meningococcal |
Meniscal Injury |
Menopause |
Menstrual & Uterine |
Menstrual Cycle |
Mental Retardation |
Mental Status Exam |
Mentally Retarded Pt |
Meperidine (Demerol) |
Meralgia Paresthetica |
Mesenteric Venous Thrombosis |
Metabolic Acidosis |
Metabolic Alkalosis |
Metabolic and Bone Dz |
Metatarsal Stress Fx |
Metatarsalgia |
Methadone |
Methanol |
Met-hemoglobin |
Methotrexate (Rheumatrex, MTX) |
Mets |
MGUS
Mi |
Microalbuminuria |
Microangiopathic |
Microscopic Colitis |
Microscopic Polyangiitis |
Migraine |
Mild Hypothermia |
Milk Alkali |
Milk Allergy |

Milk Thistle |
Mineralocorticoid Excess |
Miotics |
Mitochondrial Inheritance |
Mitochondrial Myopathy
Mixed Connective Tissue D/o |
MMR |
MMSE |
Molds |
Mold Hypersensitivity |
Molluscum |
Molybdenum |
Mongolian Spots |
Monkeypox
Monobactams |
Monoclonal Ab's |
Monocyte |
Monocytosis |
Mood D/o |
Moraxella |
Morphine Sulfate (MS) |
Morton Neuroma |
Motor |
Mouth |
Movement Disorders |
MPGN |
MR |
MR - Mitral Regurge |
MRSA |
MS Contin |
Morphine Sulfate |
MSOF / MODS |
Mucolytics |
Mucormycosis |
Multiple Cranial Nerve Lesions |
Multiple Myeloma |
Multiple Sclerosis |
Munchausen |
Murmurs |
Muscle Cramps |
Muscle Relaxer |
Muscular Dystrophy |
Mushrooms |
MV |
MVP |
Myasthenia Gravis |
Mycobacterium avium |
Mycoplasma |
Mycosis Fungoides |
Mycotoxin |
Myelodysplastic Syndrome |
Myeloproliferative |
Myiasis |
Myocarditis |
Myocardial Perfusion Study |
Myoclonic Jerks |
Myoclonus |
Myoglobin |
Myositis/ Myopathy |
Myositis |
Myxedema Coma |
N | Ng |
N-Acetylcysteine (Mucomyst) |
Nail Dyschromia |
Nails |
Nandrolone decanoate |
Narcolepsy |
Narcotics |
Nasal Foreign Body |
Nasal Fx |

Nasal Irrigation
Nasal Polyps |
Nasal Trauma |
Nasal Vestibulitis |
NASH |
Nasopharyngeal Cancer |
Natrecor |
Nausea & Vomiting |
Nausea |
Nebulizer |
Neck Mass |
Neck Pain |
Necrotizing Enterocolitis |
Necrotizing Infections |
Needlestick |
Nematodes |
Nephrocalcinosis & Nephrogenic Systemic Fibrosis |
Nerve Conduction Velocity (NCV)
Neonates & Babies |
Nephritic |
Nephritis - lupus
Nephrology |
Nephropathy |
Nephrotic |
Nerve Blocks |
Neuro Exam |
Neurofibromatosis |
Neurogenic Claudication |
Neuroleptic Malignant Syndrome |
Neurology |
Neuropraxia |
Neuropathic Ulcers |
Neuropathy
Neurotic Excoriations |
Neutropenia |
Neutropenic fever |
Neutrophilia |
Neutrophils |
Newborn Jaundice |
Ng |
NGAL |
Nasogastric Tube |
Non Gonococcal Urethritis |
Non-Hodgkin's Lymphoma |
Niacin (B3) |
Niacin |
Nicotine |
Nicotine OD |
Night Blindness |
Night Sweats |
Night Terrors |
Nightmares |
NIH Scale |
Nipple Disorders |
Nitro Drip |
Nitro |
Nitrogen Narcosis |
Nitrous Oxide – NO2 |
Nits |
NMS |
Neuroleptic Malignant Syndrome |
Nocturnal Enuresis |
Nocturnal Leg Cramps |
Nodules
Normal Pressure Hydrocephalus |
Normocytic |
Norplant |
Nose |
NQMI |
NSAID toxicity |

NSAIDs |
Nucleated |
NUD |
Nursemaids Elbow |
Nutcracker |
Nutriceutical (Herbal) Agents |
Nutrition & Vitamins |
Nutrition in Elderly |
Nutritional Support |
Nystagmus |
Nystatin |
O | OS |
O157 |H7 |
O2 therapy |
OA |
Obesity |
OBGYN- Notes |
Integrative Medicine |
OBGYN- Notes |
Obstructions-GI |
Obstructive-Lung |
Occipital Neuralgia |
Occult GIB |
OCD |
Oculomotor Palsy (CN 3) |
Odor – foot & bodyt |
Off-Label Drug Use |
Olecranon Bursitis |
Omega-3 fatty acids
Onc Emergency |
Oncogens |
Oncologic |
Oncology Emergencies |
Onychomycosis |
Open Angle Glaucoma |
Opening Snap |
Ophthalmology |
Opioids |
Opioid Abuse / Withdrawal |
Optic Disc Atrophy |
Optic Neuritis |
Oral Allergy Syndrome |
Oral Contraception |
Oral Estrogen |
Oral Leukoplakia |
Oral Lichen Planus |
Oral Rehydration |
Oral Ulcers |
Orbital Cellulitis |
Orbital Emphysema |
Orchitis |
Orders & Notes |
Orf |
Orofacial Pain |
Oropharyngeal Dysphagia |
ORT |
Ortho |
Orthoses |
Orthostatic Hypotension |
Orthostatic |
OS |
Osgood-Schlatter Dz |
Osmotic Laxatives |
Osteochondritis Dissecans-knee |
Osteochondritis Desiccans (OCD) |
Osteochondritis Desiccans (foot) |
Osteolysis of Distal Clavicle |
Osteomalacia |
Osteomyelitis |
Osteonecrosis (AVN) |

Osteoporosis |
Ostomy |
Otalgia |
Other Shoulder Problems |
Other Vaginitis |
Other |
Otitis Externa (OE) |
Otitis Media with Effusion |
Ovalocyte |
Ovarian Cancer |
Ovarian Cyst |
Ovarian Failure |
Ovarian Torsion |
Overactive Bladder |
Overflow Incontinence |
Oversedation |
Overtraining |
Overuse Injuries |
Oxymorphone |
P | Pe | Ph | PL | Pr | Ps |
P wave |
PA Cath Patterns |
PA Cath |
PAC |
Pacemaker |
Paget's |
Pain and Substance Abusers |
Pain Management |
Palindromic Rheumatism |
Palliative |
Palpitations |
PALS |
PAN |
PANDAS |
Pancreatic Ca |
Pancreatic Enzymes |
Pancreatitis |
Panniculitis |
Pantothenic Acid |
Pap Results |
Papilledema |
Papillitis |
Pappenheimer |
PAR |
Paracentesis |
Paragonimus |
Paralysis |
Paraneoplastic Syndromes |
Paraphimosis |
Parasites & Insects |
Parasitic Cholangitis |
Parasomnias |
Parathyroid |
Parkinson's Dz |
Paronychia |
Parotid |
Paroxysmal |
Patellar Dislocations |
Patellar Subluxation |
Patellar Tendonitis |
Patellofemoral Syndrome |
Patent Ductus Arteriosus |
Patent Foramen Ovale |
PAWP |
PBC |
PCOS |
PCP |
PCTA |
Pe |
PEA |

Peak Flow |
Peanut |
Pectus excavatum |
Pediatric Exanthems |
Pediatric Limp |
Pediatric Emergencies |
Pediatrics |
PEEP |
PEG |
Peg-Intron |
PEGJ |
Pelvic Examination |
Pelvic Inflammatory |
Pelvic Relaxation |
Pemphigoid |
Pemphigus Vulgaris |
Penicillins |
Penile |
Perforated TM |
Perforation/ Peritonitis |
Performance Fuel |
Pericardial Friction Rub |
Pericardiocentesis
Perineal Laceration |
Perineal Lesions |
Periodic Limb Movements of Sleep (PLMS) |
Perioperative |
Perioral Dermatitis |
Periorbital Contusion |
Peripartum Emergencies |
Peripheral Neuropathy |
Peripheral Smears |
Peripheral Vascular Disease |
Peritoneal Lavage |
Peritonsillar Abscess |
Peroneal Nerve |
Peroneal Tendonitis |
Personality D/o |
Pertussis |
Pessary |
PET Scanning |
Peyronie |
Pfiesteria |
PFO |
PH |
PH - Acid-Base |
Pharmacologic Pupil |
Pharmacotherapy |
Pharyngeal Foreign Body |
Pharyngitis |
Pheo |
Pheochromocytoma |
Phimosis |
Phosphate (P) |
Phosphorus |
Photoaged Skin |
Photophobia |
Photosensitivity |
Pica |
Picks |
PID |
Piercing - body |
Pigmentation |
Pigmented Oral |
Pigmented Villonodular Synovitis |
Pilomatrixoma |
Pilonidal Abscess |
Pilonidal Cyst |
Pinguecula/ Pterygium |
Pink Puffer |

Pinworms |
PIP Injury |
Piriformis Syndrome |
Pitted Keratolysis |
Pituitary |
Pityriasis Alba |
Pityriasis Rosea |
Pityriasis rubra pilaris |
Pl |
Placebo |
Placenta |
Plague |
Plant Alkaloids |
Plantar Fasciitis |
Plantar Laceration |
Plant Dermatitis |
Plaque |
Plasma Cell Dyscrasias |
Platelet Concentrate |
Platelet Pheresis |
Platelets |
Plavix (Clopidogrel) |
Pleural Effusion |
Pleurisy |
Pleurodesis |
Plica Syndrome |
Plummer-Vinson |
PML |
PMR |
Pneumococcal (Pneumovax) |
Pneumoconiosis |
Pneumomediastinum |
Pneumonia |
Pneumonitis |
Pneumopericardium |
Pneumothorax |
PNH |
POEMS |
Polarized Microscopy |
Poliomyelitis |
Post Polio Syndrome |
Pollen Count |
Pollen |
Polyarthritis |
Polycystic Kidney Disease |
Polycystic Ovary Syndrome |
Polycythemia Vera |
Polymyalgia Rheumatica (PMR) |
Polymyositis / Dermatomyositis |
Popliteus Tendinitis |
Porphyria Cutanea Tarda |
Porphyria |
Portal HTN |
Portal Vein Thrombosis |
Post Coital Test |
Posterior Interosseus Syndrome |
Postmenopausal |
Postpartum Hemorrhage |
Postpartum Mood D/o |
Post Polio |
PostStrep-GN |
Post-Thrombotic Syndrome |
Postterm Pregnancy |
Posturing |
Postural Drainage & Controlled Coughing |
Potassium (K) |
Potassium |
Pouchitis |
Power of Attorney |
PPHN |

PPI |
PPN |
PR |
PR |
Prader-Willi |
PRBC |
Preeclampsia |
Pregnancy Related Dermatosis |
Pregnancy |
Premature Beats |
Premature CAD |
Premature Ejaculation |
Premature Infants |
Premature Rupture of the Membranes |
Premature Sexual Development |
Premenstrual Syndrome |
Preseptal Cellulitis |
Pressure Ulcers (Decubiti) |
Pre syncope |
Preterm Labor |
Preventive Med |
Prevnar (PCV, PCV-7) |
Priapism |
Primary Biliary Cirrhosis (PBC) |
Primary Sclerosing Cholangitis (PSC) |
Prions |
Probiotics |
Procedure Note |
Procedures-Derm |
Proctalgia |
Progestin-only pill |
Prokinetic |
Prolactinoma |
Prolotherapy |
PROM |
Pronator Teres Syndrome |
Prostate |
Prostate Cancer |
Prostatitis |
Prostatodynia |
Protamine |
Prot C & S |
Protein - dietary |
Protein-Losing Enteropathy |
Protein - serum |
Proteinuria |
Prothrombin G2 |
Proven |
Prune Belly |
Pruritus Ani |
Pruritus
PS |
PS – pulmonary stenosis |
PSC |
Pseudocoma |
Pseudocyesis |
Pseudofolliculitis Barbae |
Pseudo-HTN |
Pseudo-Neurologic Syndromes |
Pseudoseizure |
Pseudotumor Cerebri |
Psittacosis |
Psoas Abscess |
Psoriasis |
Psoriatic arthritis |
PSVT |
Psych Emergencies |
Psych Eval |
Psych Meds |
Psych |

Psychiatric |
Psychocutaneous Dz |
Psychosis |
PT |
Pt's with Lists |
Ptosis |
PUD |
Puffer Fish |
Pulm |
Pulmonary & Cardiac Parameters |
Pulmonary Contusion |
Pulmonary Edema |
Pulmonary Embolism |
Pulmonary HTN |
Pulmonary Mass |
Pulse exam |
Pulse Oximetry |
Pulse Pressure |
Pulsus Alternans |
Pulsus Bisferiens |
Pulsus Paradoxus |
Pupils |
Purine Content of Food |
Purpura |
PVC's |
PVRV |
Pyelonephritis |
Pyloric Stenosis |
Pyoderma Gangrenosum |
Pyoderma/ other infections |
Pyogenic Granuloma |
Pyogenic Liver Abscess |
Pyramidal Tract Dz |
Pyridoxine (B6) |
Pyridoxine |
Pyruvate Kinase |
PZA |
Q-R | Red | Rh |
Q-fever |
QT |
Quackery |
Quinolones |
Q-wave |
R wave |
Rabies VAC |
Rabies |
Rabdo – see Rhabdo
Radial Nerve |
Radial Tunnel Syndrome |
Radiation Therapy (XRT) |
Radiation -exposure |
Radiocontrast Nephropathy |
Radiology |
Radionuclide Ventriculography/ MUGA |
RAI Class |
Ramsay Hunt |
Ramsay Scale |
Ranula |
Rape |
Rat Bite Fever |
Raynaud's |
RBBB |
RBC Inclusions |
RDW |
Reactive Arthritis |
Refeeding Syndrome |
REAL Class |
Rebetron |
Rebound H-A |
Rectal Abscess |

Rectal Exam |
Rectal Prolapse |
Rectal |
Rectum/ Anus |
RED |
Red Eye |
Refeeding Syndrome |
Reflex Eye Movements |
Reflex Sympathetic Dystrophy |
Reflexes |
Refraction |
Refusal of Care/ AMA |
Rehabilitation |
Reiter's |
Rejection | -transplant
Relapsing Fever (Borrelia) |
Relapsing Polychondritis |
REM Sleep Behavior Disorder |
Renal Cell Ca |
Renal Drug Toxicity |
Renal Failure Index |
Renal Parenchymal |
Renal Stones |
Renal Vein Thrombosis |
Renovascular |
Repellents |
Respiratory Acidosis |
Respiratory Alkalosis |
Restless Leg Syndrome (RLS) |
Restraints |
Restrictive |
Retic |
Retinal Detachment |
Retinal Hemorrhage |
Retinitis Pigmentosa |
Retinopathy-DM |
Retinopathy - HTN |
Retrograde Ejaculation |
Retropharyngeal |
Retroperitoneal Fibrosis |
Rh |
Rh Negative Pregnancy |
Rhabdo |
Rheumatoid |
Rheum |
Rhinitis
Rhomboid Muscles |
Rhonchi |
RIA |
Rib Fractures |
Rib Notching |
Ribavirin |
Riboflavin (B2) |
Rickettsial Diseases |
Rickettsialpox |
Rifampin |
Ring Removal |
Rivastigmine (Exelon) |
RMSF |
Rodenticides |
Rotary Chair Test |
Rotavirus |
Rouleaux |
Roundworms |
RPGN |
RSV |
RTA |
Rubella |
Rumination Syndrome |
Running |

Ruptured Globe |
RV |
S | SE | Sh | Sp | Su |
S aureus |
S. pneumo |
SAAG |
Sacroiliac Dysfunction |
SAD |
Salicylate |
Salivary Glands |
Salmonella |
Salter-Harris |
SAR |
SARS |
Sarcoid |
SBP |
Scabies |
Scaphoid |
Scapular Winging |
Scheuermann's dz |
Schilling's |
Schiotz Tonometry |
Schirmer test |
Schistocyte |
Schistosomiasis |
Schizophrenia |
Schmorl's nodes |
Sciatica |
Scleritis |
Scleroderma |
Scoliosis |
Scorpions |
Scotoma |
Scrofula |
Scombroid |
Scrotal Pain |
Scrub Typhus |
SE |
Sea Sick
Sea Snakes |
Sebaceous Hyperplasia |
Seborrheic Dermatitis |
Sedation |
Seizures |
Selenium |
Sensory |
Sentinel Node |
Sepsis |
Septal Hematoma |
Septic arthritis |
Serology |
Seronegative Spondylopathy |
Serotonin Syndrome |
Serum Fe |
Serum Sickness |
Sesamoiditis / Fx |
Severs Apophysitis |
Sexual Abuse |
Sexual Disorders |
Sexual Disorders - psychosexual
Sexual Dysfunction- Female |
Sh |
Shaken Baby |
Shellfish |
Shigella |
Shin Splints |
Schistosomiasis
Shock |
Shoe Wear / Orthoses |
Short Stature |

Shoulder Dystocia |
Shoulder |
SIADH |
Sialadenitis |
Sialolithiasis |
Sialorrhea |
Sialorrhea |
Sick Building |
Sick Euthyroid |
Sickle Cell |
SIDS |
Signal Averaged ECG |
Silicosis |
SIMV |
Sinus Arrhythmia |
Sinus Pauses |
Sinusitis |
Sjögren's |
Skiing |
Skin Infections |
Skin Tag |
Skin Ulcers |
Skin |
SLE |
Sleep Apnea |
Sleep D/o |
Sleep Walk |
Sleep |
Slipped capital femoral epiphysis |
Slit Lamp |
Small Intestine Motility D/o |
Smallpox |
Smoke |
Snake |
Snapping Hip Syndrome |
Snoring |
SOB |
Sodium (Na) |
Sodium |
Somatic & Visceral Pain |
Somatization d/o |
Somatizing |
Somatoform D/o |
Somogyi Effect |
Sore Throat/ Strep |
Sotalol (Betapace AF) |
Sp. |
Spacers |
Spanking |
Spasms |
Spasticity |
Speech Therapy |
Spells |
Spermatocele |
Spherocyte |
Spiculated RBC's |
Spiders |
Spina Bifida |
Spinal Stenosis |
Spirituality |
Spirometry |
Spleen |
Splenic Deficiency (Asplenia) |
Splenic exam |
Splenic Rupture |
Splenomegaly |
Splinting |
Spondylopathy |
Spondylosis |
Sponges |

Spontaneous bacterial peritonitis |
Sporotrichosis |
Sports Med |
Sports |
Spot Ur Na |
Spur |
Squamous Cell |
SSI |
SSS |
Statins |
Statistics |
Stats |
Status Epilepticus |
STD's |
Sternum |
Steroids |
Stevens Johnson Syndrome |
Sticky Eye |
Sticky Platelet Syndrome |
Stiff Man |
Stills Disease |
Stimulant & Irritants |
Stomach/ Esophagus |
Stomatitis |
Stomatocyte |
stool Osmotic gap.
Stool Softener |
Stowed Finger |
Strabismus |
Strains & Sprains |
Strep pneumonia |
Streptomycin |
Stress (SI) |
Stress Fractures |
Stress Reduction |
Stress Management |
Stricture |
Stridor |
Stroke Scale |
Stroke Syndromes |
Strokes |
Strongyloides |
Structural Analogs & Antimetabolites |
Struma ovarii |
Struvite |
Strychnine |
ST-T Wave |
Sturge-Weber |
Stutters |
Su |
Subacromial Injection |
Subacute Thyroiditis |
Subclavian Steal |
Subconjunctival Hemorrhage |
Subcortical dementia |
Substance Abuse |
Substances of Abuse |
Subungual Hematoma |
Subungual Splinter |
Sugar Substitutes |
Suicide |
Suicide |
Sulfonamides |
Sulfonylurea- OD |
Sulfur |
Sun Exposure |
Sunburn |
Sunscreens |
Supportive Agents-chemo |
Suprascapular Neuropathy |

Supraventricular |
Surgical Notes |
Surgical Pearls |
Survival |
Survival Medicine |
Suturing Techniques |
SVC Syndrome |
Swan Neck Deformity |
Swan Orders |
Sweating - hyperhidrosis |
Sweets Syndrome |
Syncope |
Syndromes-Child |
Syndrome-X-cardiac |
Syndrome-X-endocrine |
Synovial Fluid |
Syphilis |
Syringoma & Sweat Glands |
Syringomyelia |
Systemic Sclerosis |

T | Th | To |

T-2 Mycotoxin |
Tachycardia |
Tacrine (Cognex) |
Tacrolimus- topical |
Takayasu's |
Tamponade |
Tanner Stages |
Target Cell |
Tarsal Coalition |
Tarsal Tunnel |
Taste & Smell |
Tay-Sachs disease |
TB Meningitis |
TB |
Tear Drop |
Tearing |
Technetium-99m Sestamibi (Cardiolite) |
Teeth |
Teeth |
Teething |
Telogen effluvium |
Temp Conversion |
Temper Tantrums |
Temporal arteritis |
Tendonitis |
Tendon Ruptures |
Tenosynovitis |
TEN |
TENS |
Tension H-A |
Teratogens |
Terbinafine |
Terminal Delirium |
Terminal Sedation |
Testicular Cancer |
Testicular Mass |
Testicular Torsion |
Tetanus |
Tetracyclines
Tetralogy of Fallot |

Th |

Thalassemia |
Thalidomide (Thalomid)
Thallium |
Theophylline |
Therapeutic Levels |
Therapeutic Tap |
Thiamine (B1) |
Thiazide |

Thigh Pain |
Thoracentesis |
Thoracic (upper) Back |
Thoracic Aortic Aneurysm |
Thoracic Outlet Syndrome |
Threadworm |
Threatened & complete Ab |
Throat |
Thromboangiitis Obliterans (Buerger's Dz) |
Thrombocytopenia |
Thrombocytosis |
Thrombolytics in AMI |
Thrombolytic-PE/DVT |
Thromboembolic Renal Dz |
Thymic Cancer |
Thyroid Cancer |
Thyroid Nodules |
Thyroid Storm |
Thyroid |
Thyroiditis |
Thyrotoxicosis factitia |
TIBC |
Tic |
Ticks |
Tilt Table Testing |
Tinea Capitis |
Tinea Incognito |
Tinea pedis |
Tinea Versicolor |
Tinnitus |
TIPS |
TLC |
TMJ Disorders |
To |
Tobacco/ Nicotine |
Tocolysis
Togaviridae |
Toilet Train |
Tomographic imaging procedures (SPECT) |
Tongue & Oral Lesions |
Tonsillectomy |
Topical Anesthesia |
Topical Steroid |
Torsades |
Torticollis |
Tourette's Syndrome |
Toxic Adenomas |
Toxic Food |
Toxic Epidermal Necrolysis |
Toxic Megacolon
Toxic Shock |
Toxic Synovitis |
Toxicology |
Toxidromes |
Toxocariasis |
Toxoplasmosis |
TPN |
TR |
Tracheostomy |
Trachoma |
Tramadol |
Transferrin sat |
Transferrin |
Transfusion Reactions |
Transient Tachypnea |
Transplantation |
Transposition of the Great Vessels |
Transvenous Pacer |
Transverse Myelitis |
Trapezius Sprain/Strain |

Trauma & Tox |
Trauma and Pregnancy |
Trauma |
Travel Medicine |
Travel Medicine |
Traveler Diarrhea |
Travelers Vaccines |
Trematodes |
Tremors |
Trench foot |
Trench mouth/ Vincent's |
Trendelenburg Test |
Treponomes |
Tri 13 |
Tri 18 |
Triangular Fibrocartilaginous Complex |
Trichinosis |
Trichomonas |
Trichomycosis Axillaris |
Trichotillomania |
Trigeminal Neuralgia |
Trigger Finger |
Trigger Point Therapy |
Triglyceride |
Triptans |
Tropical Sprue |
Trypanosomiasis |
TS |
T-score |
TTP |
Tuberous Sclerosis |
Tularemia |
Tumor Lysis |
Tumor Markers |
Turf Toe |
TV |
T-wave |
Twinrix |
Two foot, one hand dz |
Tympanometry |
Typhlitis |
Typhoid Fever |
Tzanck |
U |
UAP/NQMI |
UC Vs Crohn's |
Ulcer, genital- Ddx |
Ulcerative Colitis |
Ulnar Nerve |
Ultram |
Ultraviolet Radiation |
Unexplained Sx's |
Unfractionated Heparin |
Unilateral Cover |
Unna Boot |
Upper GIB |
Uprima |
Ur Osm |
Uremic |
Urethra |
Urethral Catheterization |
Urethral Hypermobility |
Urethral Suppository |
Urethritis |
Urge (UI) |
Uric Acid |
Urinalysis |
Urinary Electrolytes |
Urinary Incontinence |
Urinary Retention |

Urine Drug Testing |
Urodynamics and Studies |
Uroflow |
Urology |
Urticaria |
Urticarial Vasculitis |
Uterine Bleeding |
UTI |
Uveitis (Iritis/ Iridocyclitis) |
V | Ve |
Vaccines/ Immunizations |
Vagal Maneuvers |
Vaginal Estrogen |
Vaginismus |
Valsalva |
Valve Replacement |
Valves |
Vancomycin |
VAREC |
Variant Angina |
Varices - Esophageal
Variceal Bleeding |
Varicella (Chicken Pox) |
Varices |
Varicocele |
Varicose |
Various Abx |
Various Other D/o |
Various Other Neuro |
Vascular Access |
Vascular Anatomy |
Vascular dementia |
Vascular Derm Lesions |
Vascular Diseases
Vasculitis |
Vasectomy |
Vasopressin |
Vasopressors |
Vasovagal (Neurocardiogenic) |
VC |
Ve |
Vectors |
Vegetarians |
Vegetative State |
Vena Cava Filter |
Venous cutdown |
Venous Thromboembolism |
Venous Ulcers |
Vent Modes |
Ventricular from SVT |
Ventricular Septal Defect |
Ventricular |
Venturi |
Vertebral Body Fx's |
Vertebrobasilar Dz |
Vertigo |
Vesicoureteral Reflux |
Viagra |
Vibrio sp |
Violence |
Violent Pt's |
VIPoma |
Viral Rhinosinusitis (Colds) |
Viral |
VISA |
Visceral larva migrans |
Vision Loss |
Visual Acuity |
Visual Fields |
Vit B-12 |

Vit E |
Vit K |
Vitamin A |
Vitamin D |
Vitamin Deficiencies |
Vitamin Toxicity |
Vitamins & Nutrients |
Vitiligo |
Vocal Cord Dysfunction |
Von Hippel-Lindau |
Zoster
VP Shunt |
VQ |
VREF |
Vulvar Ca |
Vulvar Lesions |
Vulvar Vestibulitis |
Vulvodynia |
vWb Dz |
W |
Waddell's (Exaggeration) Signs |
Waldenström's macroglobulinemia
Warfarin |
Warfarin OD |
Warts- genital |
Warts-skin |
Wasting Syndromes & Wt loss |
Water Disinfection |
Waterborne Bacteria |
WBC Abnormalities on Smears |
WBCs |
Weakness Disorders |
Web Searching |
Web |
Wedge Resection-nail |
Wegener |
Weight Conversion |
Wernicke-Korsakoff Syndrome |
West Nile Virus |
Wheeze |
Whiplash |
Whipple's |
Whipworms |
White Oral Lesions |
White-coat |
Williams (William-Beuren Syndrome) |
Wilson's Dz |
Winging, Scapular |
Wiskott-Aldrich Syndrome |
Withholding / Withdrawing Care |
Womens Health |
Wound Care |
Wounds- burn care |
Wound Dressings |
Wounds |
Wound care |
WPW |
Wright Stain |
Wrist & Hand |
XYZ |
Xanthelasma |
Xerosis |
Xerostomia |
X-link |
X-Linked Agammaglobulinemia |
X-Linked Lymphoproliferative Syndrome |
Ximelagratan |
Yellow Fever |
Yellow nail syndrome |
Zenker's |

Zollinger-Ellison (Gastrinoma) |
Zinc |
Zoonotic Infections |
Zoster |
Z-score |