158 Page PANCE/PANRE Review Outline
Containing 500+ Diseases

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### Dysfunctional uterine bleeding

- **General Characteristics**
  - Most likely to occur with anovulation (PCOD, perimenopause, exogenous obesity, adrenal hyperplasia).
  - **Constant, non-cyclic blood estrogen levels:**
    - Stimulates growth & development of the endometrium.
    - Without ovulation & the influence of progesterone, endometrium overgrows & sloughs at irregular times & in unpredictable amounts.
- **Symptoms**
  - Infrequent & light, to frequent & heavy menstrual bleeding.
  - May evolve into amenorrhea.
  - Shortened menstrual cycle (luteal phase defect).
  - Mid-cycle spotting.
- **Diagnostics**
  - Exam:
    - Signs of inflammation or infection of the genital tract, visible changes on cervix or walls of vagina.
    - Palpably abnormal size/shape to the uterus or ovaries.
    - Pap smear abnormalities.
    - Endometrial biopsy: proliferative or hyperplastic endometrium.
- **Treatment**
  - Aim is to convert proliferative endometrium into secretory endometrium.
  - Administer a progestational agent (medroxyprogesterone acetate) for 10 days Q month.
  - OCPs to establish regular, predictable cycles – control acute bleeding episode w/ OCPs (3-4 pill QD for 6-7 d).
  - No response to meds → ablation or hysterectomy.
  - Treat any associated anemia.

### Endometrial cancer

- **General Characteristics**
  - Most common gynecologic malignancy in the US.
  - 2x more common as cervical cancer.
  - Mean age 60 yrs.
  - 4th most common malignancy in women after lung, breast & colorectal cancer.
  - Estrogen-dependent.
  - Associated w/ antecedent endometrial hyperplasia.
  - Age at onset: postmenopausal (75%), perimenopausal (15-20%), menstrual (5-10%).
  - Risk factors: obesity, HTN, DM, nulliparity.
- **Symptoms**
  - Abnormal vaginal bleeding.
  - Post-menopausal bleeding is always abnormal; must be investigated (even a single episode).
- **Diagnostics**
  - Endometrial biopsy.
- **Treatment**
  - Hysterectomy & post-op radiation therapy.

### Endometriosis

- **General Characteristics**
  - Benign condition in which endometrial glands & stroma are present outside the endometrial cavity.
- **Symptoms**
  - Dysmenorrhea.
  - Deep dyspareunia.
  - Dyschezia – painful BM.
  - Pre- & post-menstrual spotting.
  - Ovulatory pain & mid-cycle vaginal bleeding.
  - Pelvic pain.
  - Infertility.
- **Diagnostics**
  - Laparoscopic exam w/ biopsy – only definitive diagnostic tool.
  - Pelvic U/S.
- **Treatment**
  - Medical:
    - Combo estrogen/ progesterone OCPs + NSAIDs.
    - Progesterone therapy (suppresses GnRH).
    - Danazol (testosterone): induces a pseudo-menopause by suppressing both LH & FSH.
    - Lupron (GnRH agonists) down-regulates the pituitary to suppress LH & FSH.
  - Surgical:
| **Leiomyoma** | - Occurs in 30% of women  
- Most common indication for hysterectomy  
- Most common in 3rd decade, 5x more common in African Americans | - Bleeding, most commonly menorrhagia  
- Pain including secondary dysmenorrhea  
- Pressure sx related to size & number of tumor & location of pressure  
- Exam:  
  - Bimanually or abdominally palpable mass – large, irregular contoured, hard, enlarged pelvic mass  
  - Irregularities of the uterine cavity found during curettage | - Pelvic U/S → shadowing  
- Aim is to minimize uterine bleeding  
- Majority do not require surgical or medical treatment  
- Myomectomy – fertility can be preserved  
- Hysterectomy  
- Pharmacologic inhibition of estrogen secretion by suppression of GnRH (Lupron) or medroxyprogesterone (Provera) |
| --- | --- | --- | --- |
| **Prolapse** | - Uterine:  
  - Sagging of uterus into vagina  
  - 1st degree – descent into upper 2/3 of vagina  
  - 2nd degree – cervix approaches the introitus  
  - 3rd degree – structure is outside the introitus  
  - 4th degree or procidentia – entire uterine body is outside the vagina  
- Cystocele:  
  - Posterior bladder protrudes into anterior vagina  
- Rectocele:  
  - Distal sigmoid colon (rectum) into posterior distal vagina | - Feeling of pelvic fullness or heaviness  
- Feeling of something “falling out”  
- Backache: worse w/ prolonged standing or late in the day  
- Difficulty walking  
- Dyspareunia  
- Purulent vaginal discharge, ulceration or vaginal bleeding  
- Urinary frequency, urgency & incontinence  
- Exam:  
  - Vaginal speculum exam w/ & w/o Valsalva maneuver will demonstrate degree of prolapse  
  - Rectal exam may demonstrate rectocele | - Prophylactic:  
  - Treatment of chronic respiratory disease & constipation  
  - Wt. control  
  - Kegel exercises  
  - Non-surgical – pessaries, estrogen  
  - Surgical – hysterectomy, uterosacral or sacrospinous ligament fixation |
| **Ovarian cyst** | - Arise as a result of normal ovarian function  
- When an ovarian follicle fails to rupture, follicular cyst may develop (most spontaneously resolve)  
- **When a follicular cyst ruptures, it may cause acute pelvic pain**  
  - PCOS:  
    - Chronic anovulation w/ infertility  
    - Hx of irregular bleeding | - Mild to moderate unilateral LLQ or RLQ pain (dependent on size of cyst)  
- May have alteration of menstrual interval  
- Exam:  
  - Unilateral pelvic tenderness/pain on exam  
  - Palpable, mobile, cystic adnexal mass  
  - PCOS: hirsutism, obesity, acne, acanthosis nigricans | - U/S: cystic echo on ovary  
- PCOS: mild increase in testosterone, LH:FSH ratio3:1 (normal is 1.5:1), U/S shows bilaterally enlarged ovaries w/ multiple peripheral cysts |
| **Ovarian cancer** | - 5th most common of all cancers in women  
- Mortality rate highest of all gynecologic cancers  
- Mainly ages 40-60 | - Rarely symptomatic until extensive metastasis  
- Possible irregular menses, menorrhagia, or post-menopausal bleeding | - Possible detection of serum tumor marker CA-125  
- Laparoscopic exam  
- Pelvic U/S  
- Surgery – tumor-debulking  
- Chemo – paclitaxel (Taxol), cisplatin |
<table>
<thead>
<tr>
<th>Condition</th>
<th>Symptoms/Exam/Tests</th>
<th>Treatment/Prevention</th>
</tr>
</thead>
</table>
| Abdominal fullness or distention, abdominal or back pain, decreased energy, urinary frequency | -Solid, irregular fixed mass on ovaries  
-May palpate upper abdominal mass or ascites | -Endometrial biopsy & endocervical curettage w/ abnormal bleeding  
-Upper GI or barium enema if GI complaints  
-Mammography for primary in breasts |
| Exam: Solid, irregular fixed mass on ovaries  
-May palpate upper abdominal mass or ascites | | -Cone biopsy – microinvasion  
-Hysterectomy – invasive, no spread  
-Radiation – advanced disease  
-Chemo – cisplatin  
-Prevention: Gardasil vaccine, 3 doses over 6 mo. ages 11-12 (not in pregnant, lactating or immunocompromised) |
| Cervical cancer                   | -Link to HPV  
-Avg. age 45  
-Risk factors: early onset sexual activity, increased # of sexual partners, STIs, DES exposure  
-Squamous & adenocarcinoma (clear cell: linked to DES) | -Post-coital bleeding  
-Watery discharge  
-Pelvic pain | -Pap smear  
-Cone biopsy – microinvasion  
-Hysterectomy – invasive, no spread  
-Radiation – advanced disease  
-Chemo – cisplatin  
-Prevention: Gardasil vaccine, 3 doses over 6 mo. ages 11-12 (not in pregnant, lactating or immunocompromised) |
| Cervicitis                        | -Inflammation of the cervix  
-Has a lot in common w/ urethritis in men & are caused by STIs  
-Non-infectious causes: IUDs, diaphragms, allergic rxn to spermicides or latex condoms | -Abnormal vaginal discharge  
-Abnormal vaginal discharge (may be associated w/ intercourse) | -Wet-mount microscopy  
-Culture for C. trachomatis & N. gonorrhoeae  
-Azithromycin or doxycycline for chlamydia  
-Ceftriaxone IM + azithromycin or doxycycline for gonorrhea  
-Metronidazole for trichomoniasis  
-Erythromycin during pregnancy |
| Cervical dysplasia                | -Caused by HPV  
-Precursor to cervical cancer:  
- Cervical intraepithelial neoplasia I: cancer 7 yrs.  
- Cervical intraepithelial neoplasia II: cancer 4 yrs. | -No classic signs or sx | -Pap smear & colposcopy w/ biopsy  
-Classification by Bethesda system  
-Observation – repeat pap, colposcopy & pap, HPV-DNA  
-Abalve therapy – cryotherapy, laser, electrofulguration  
-Excisional procedure – LEEP, cold-knife cone  
-Hysterectomy – only recurrent CIN 2 or CIN 3  
-Pap smear every 6 mos. For 2 yrs. |
| Incompetent cervix                | -Fetal membranes are exposed to vaginal flora  
-May lead to infxn, vaginal discharge, premature rupture of membranes | -Painless dilation & effacement of cervix  
-Bleeding, vaginal discharge  
-Noted during 2nd trimester | -Based on PE  
-Bed rest  
-Cerclage |
| Vaginal/vulvar cancer             | -Vaginal:  
- Rare, usually 2/2 other cancers  
-Peak age 50s – linked to DES exposure  
-80-90% squamous cell  
- Vulvar:  
- Peak age 70s  
- 90% squamous cell | -Vaginal:  
- Asx  
- Vulvar:  
- Pruritus, red/white ulcerative lesions | -Vaginal:  
- Pap smear, biopsy  
- Vulvar:  
- Biopsy  
-Vaginal:  
- Radiation therapy  
-Vulvar:  
- Surgery, radiation therapy, chemotherapy |
| Vaginal prolapse                  | -Upper portion of the vagina loses its normal shape & sage or drops down into vaginal canal or outside the vagina | -Pelvic heaviness  
-Backache  
-Mass bulging into canal or out of vagina  
-Incontinence  
-Vaginal bleeding | -Exercise, pessary  
-Estrogen replacement  
-Vaginal vault suspension – sacrocolpopexy |
|                            | | | |
Vaginitis

- Caused by weakness of pelvic & vaginal tissues & muscles
- Inflammation of the vaginal vault or tissue
  - Infectious:
    - Bacterial vaginosis – d/t Lactobacillus & overgrowth of G. vaginalis
    - Candida
    - Trichomonas – spread via sexual contact
  - Non-infectious – on-specific vaginitis:
    atrophic vaginitis most common cause in post-menopausal women d/t estrogen-deficient mucosa
    Foreign body – usually a forgotten tampon, diaphragm or condoms, toxic shock caused by staphylococci

Vaginitis

- Bacterial vaginosis – thin, homogenous, white to gray, adherent, often increased amounts, bad odor
- Trichomonas – rancid odor, vulvar pruritus, dysuria, dyspareunia, yellow-green, frothy discharge, strawberry cervix
- Non-specific – vaginal dryness, discharge, itching, burning & dyspareunia

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Bacterial vaginosis</th>
<th>Candidiasis</th>
<th>Trichomoniais</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discharge</td>
<td>Odor worse after sex</td>
<td>Itching</td>
<td>Burning</td>
</tr>
<tr>
<td>Discharge amount</td>
<td>Often increased</td>
<td>Sometimes increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Discharge appearance</td>
<td>Thin, gray-green</td>
<td>White, curdy</td>
<td>Frothy, gray-green</td>
</tr>
<tr>
<td>Vaginal pH</td>
<td>&gt;4.5</td>
<td>Normal</td>
<td>&gt;4.5</td>
</tr>
<tr>
<td>Whiff test</td>
<td>Present</td>
<td>Absent</td>
<td>Possible present</td>
</tr>
<tr>
<td>Microscopic</td>
<td>Clue cells</td>
<td>Hyphae, yeast</td>
<td>Trichomonads</td>
</tr>
<tr>
<td>Treatment</td>
<td>Metronidazole</td>
<td>Fluconazole</td>
<td>Metronidazole or tinidazole</td>
</tr>
<tr>
<td></td>
<td>Clindamycin</td>
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Amenorrhea

- Primary: never menstruate at age 14 w/o secondary sexual characteristics or age 16 w/ secondary sexual characteristics
- Secondary: previously menstruated but has not had menses for the past 6 mos.
  - Most common cause is pregnancy
  - Other causes divided into 3 areas:
    - Hypothalamic-pituitary dysfunction

- Ovarian failure:
  - Hot flashes, mood changes, sleep disturbance, vaginal dryness, dyspareunia
  - Exam: signs of estrogen-deficiency: vaginal dryness, thin vaginal epithelium, thinning &/or flushing of the skin

- If H-P dysfunction:
  - FSH & LH decreased
  - PRL normal in most conditions but elevated in PRL-secreting pituitary adenomas

- If ovarian failure:
  - Progesterone challenge test (PCT) – 100 mg progesterone IM or 10 mg oral medroxyprogesterone acetate (Provera) QD for 10 d
  - Asherman’s syndrome – hysteroscopy (used for dx & treatment)

- Modify functional causal behaviors
- Stimulate gonadotropin secretion – clomiphene citrate, menotropins
- Treat underlying psychogenic, medical (hypothyroidism), or drug-induced causes
- Surgery – tumors
- Ovarian failure:
When pulsatile secretion of GnRH is disrupted or altered in the hypothalamus, the anterior pituitary does not secrete FSH or LH.

- Interference w/ the anterior pituitary disrupts release of FSH & LH
- Other – wt. loss, excessive exercise, obesity, marijuana, tranquilizers
- Neoplastic causes – PRL-secreting pituitary adenoma, craniopharyngioma, hypothalamic hamartoma
- Psychogenic – chronic anxiety, pseudocyesis, anorexia
- Other: head injury, chronic medical illness

- Ovarian dysfunction
  - Follicles are either exhausted or resistant to stimulation by FSH & LH, resulting in increase serum FSH & LH levels
  - Alterations of genital outflow tract
    - Congenital abnormalities of the Mullerian ducts – imperforate hymen, transverse vaginal septum
  - Scarring of the uterine cavity (Asherman’s syndrome)

### Primary amenorrhea:

<table>
<thead>
<tr>
<th>Breasts present</th>
<th>Uterus present</th>
<th>Uterus absent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Imperforate hymen</td>
<td>Mullerian agenesis</td>
</tr>
<tr>
<td></td>
<td>Vaginal septum</td>
<td>Androgen insensitivity</td>
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<td></td>
<td>Anorexia</td>
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<tr>
<td></td>
<td>Pregnancy</td>
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<tr>
<td>Breasts absent</td>
<td>Gonadal dysgenesis</td>
<td>Rare: not clinically relevant</td>
</tr>
<tr>
<td></td>
<td>H-P failure</td>
<td></td>
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</tbody>
</table>

- Dysmenorrhea
  - Painful menstruation which prevents performing normal activities
    - Primary:
      - D/t increased prostaglandins leading to painful uterine muscle-wall activity
      - Common in women in teens & 20s; declines w/ age
    - Secondary:
      - D/t clinically identifiable cause: endometriosis, adenomyosis, adhesion, PID or leiomyomata
      - More common as women ages
  - Diffuse pain in lower abdomen & suprapubic
  - Pain comes & goes, associated w/ N/V, diarrhea, HA, & backache
  - PE: primary = normal, secondary = varies w/ etiology
  - NSAIDs for primary
  - Treat underlying cause for secondary

- If progesterone challenge causes “withdrawal bleeding” the pt is anovulatory or oligo-ovulatory – use exogenous hormone replacement therapy
- If “withdrawal bleeding” does not occur the pt is hypestrogenic or has an anatomic condition such as Asherman’s syndrome or outflow tract obstruction
- Asherman’s syndrome – estrogen therapy to stimulate endometrial regeneration of denuded areas
### Premenstrual syndrome

- Group of physical, mood & behavioral changes which occur in a regular, cyclic relationship to the luteal phase of the menstrual cycle
- PMDD – PMS w/ more severe emotional sx, may require more intensive therapy, 3-5% of women

| Sx occur in most cycles, resolving near the end of menses w/ a sx-free interval of at least 1 wk. | -No definitive physical or laboratory findings to aid dx |
|  | -Must have sx-free follicular phase (approx. 1 wk.) in contrast to the problems during luteal phase |

### Menopause

- Cessation of menses for 1 yr. w/ elevated FSH/LH
- Avg. ages 50-52
- Climacteric 5-10 yrs. prior

| Menstrual cycle alteration | -Exogenous estrogen + progestin: |
| Hot flushes & vasomotor instability | • Use estrogen vaginal cream for vaginal complaints |
| (hallmark signs of perimenopause) | • May use estrogen alone if uterus has been remove – use of unopposed estrogen increases risk for endometrial cancer |
| Sleep disturbances & mood changes | -Collagen & bone loss: |
| Vaginal dryness & genital atrophy – dyspareunia | • Bone mineral analysis |
| Skin, hair & nail changes | • Calcium w/ vitamin D |
| CV lipid changes – increased TC & LDL, decreased HDL | • Bisphosphonates or calcitrol |
| Osteoporosis | • Wt. bearing exercises |
| Skin becomes thin & dry w/ decreased elasticity | • Stop smoking/ETOH |
| Vaginal mucosa dry & thin, pH increased to 7, cervical os stenosed & transformation zone inverted | **Breast abscess**

- Painful collection of pus in the breast
- Often linked to mastitis, but can also be d/t nipple piercings or sores around the nipples

| Localized pain, swelling, & fever | -Painless & may feel mobile early |
| PE: fluctuant mass | -As it grows, border become less distinct & fixation to surrounding tissue occurs |
|  | -Nipple discharge & peau d’orange are late changes & associated w/ poor prognosis |
|  | -80% present as a mass |
|  | -90% are found by the patient |

### Breast abscess

-2nd most common malignancy in women
- Those w/ increased risk factors for breast cancer make up only 25% of breast cancer pts
- Risk factors: BRCA1/BRCA2, 1st-degree relative w/ breast cancer, >65 yrs., never breast fed, no pregnancy after age 35, late (>17) or early (<12) onset

| Surgical drainage |
| -Abx – nafcillin, vancomycin, clindamycin |
| -If fever continues after abx, consider for mastitis |

### Breast cancer

-Local excision & adjunctive therapy
- Surgical resection – lumpectomy, mastectomy, removal or axillary lymph nodes
- Non-surgical – radiation/chemo, tamoxifen, monoclonal antibody therapy
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<th>Condition</th>
<th>Description</th>
<th>Symptoms/Tests</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibroadenoma of breast</td>
<td>-2nd most common cause of type of benign breast disease (10-20% of women)</td>
<td>-Exam: asymmetry, distortion, discoloration or skin/nipple changes</td>
<td>-Ultrasound, mammogram</td>
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<tr>
<td></td>
<td>-Mostly women late teens &amp; early 20s</td>
<td>-Usually 1-2 smooth, well-circumscribed, rubbery lumps</td>
<td>-Mobile, moveable w/ no axillary involvement or nipple discharge</td>
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<td>-Can enlarge in pregnancy &amp; cause discomfort</td>
<td>-Fibroadenoma</td>
<td>-PRL &amp; beta-hCG levels</td>
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<td>-Can be induced by some drugs (spironolactone, cimetidine, estrogens, gonadotropins, marijuana)</td>
<td>-Often asymmetric or unilateral</td>
<td>-If beta-hCG is + then may be testicular tumor or other malignancy (lung or liver)</td>
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<tr>
<td></td>
<td>-Common in teenagers who are very tall or overweight &amp; in elderly men</td>
<td>-Tenderness in glandular gynecomastia</td>
<td>-Serum free testosterone &amp; LH:</td>
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<td>-Seen in 50% of athletes who abuse steroids</td>
<td>-Fatty gynecomastia not tender</td>
<td>-Low serum free testosterone + high LH = primary hypogonadism</td>
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<tr>
<td></td>
<td>-Also seen in Klinefelter syndrome</td>
<td>-Pubertal gynecomastia – tender discoid enlargement of breast tissue 2-3 cm in diameter beneath the areola</td>
<td>-High testosterone + high LH = partial androgen resistance</td>
</tr>
<tr>
<td></td>
<td>-Graded according to severity: I = mild, II = moderate, III = severe</td>
<td>-Worry for malignancy if: asymmetric, location not immediately below the areola, unusual firmness, nipple retraction/ bleeding/discharge</td>
<td>-Serum TSH &amp; FT4</td>
</tr>
<tr>
<td>Galactorrhea</td>
<td>-Milky nipple discharge unrelated to the normal milk production of breast-feeding</td>
<td>-Persistent or intermittent milky nipple discharge that has no trace of blood</td>
<td>-Pregnancy test</td>
</tr>
<tr>
<td></td>
<td>-Usually in women</td>
<td>-One or both breasts affected</td>
<td>-Hormone levels (PRL &amp; TSH)</td>
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<tr>
<td></td>
<td>-Can be d/t hyperprolactinemia (usual cause), prolonged breast feeding, major stress, pituitary tumors, breast lesions (benign, cancer, inflammatory), idiopathic w/ menses &amp; after oral contraceptive use</td>
<td>-Absent or irregular menstrual periods</td>
<td>-Analysis of fluid discharged from nipple</td>
</tr>
<tr>
<td></td>
<td>-Nipple stimulation &amp; nipple rings can increase PRL</td>
<td>-HA or vision problems in large pituitary tumors</td>
<td>-MRI</td>
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<tr>
<td>Mastitis</td>
<td>-Breast infxn</td>
<td>-Sudden onset fever, chills, malaise, &amp; general body aches</td>
<td>-Treatment of underlying cause</td>
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<td></td>
<td>-Mostly in lactating women</td>
<td>-Erythema &amp; tenderness</td>
<td>-Condition will resolve on its own in mild cases</td>
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<td>-Many proceed to generalized sepsis quickly</td>
<td>-Brawny, indurated area to palpation</td>
<td>-Cabergoline or bromocriptine – not to be used postpartum</td>
</tr>
<tr>
<td>Pelvic inflammatory disease</td>
<td>-Any infxn, including STDs, can cause PID – Spontaneous, ascending reproductive-tract infxn</td>
<td>-Almost always unilateral</td>
<td>-Dicloxacillin recommended for sepsis</td>
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<tr>
<td></td>
<td>-May lead to infertility or ectopic pregnancy</td>
<td>-Pelvic pain, dyspareunia, vaginal discharge, dysuria, N/V</td>
<td>-May continue to breastfeed or pump breast</td>
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<tr>
<td></td>
<td>-Risk factors – prior PID, multiple sex partners, not using condoms</td>
<td>-Exam: Lower abdominal tenderness</td>
<td>-Dicloxacillin recommended for sepsis</td>
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<tr>
<td></td>
<td>-Complications:</td>
<td>-Uterine/cervix/adnexal tenderness to palpation &amp; motion “Chandelier sign”</td>
<td>-Outpatient – single-dose ceftriaxone + doxycycline</td>
</tr>
<tr>
<td>Fitz-Hugh-Curtis syndrome (perihepatitis) – d/t peritoneal involvement, hepatic fibrosis &amp; scarring</td>
<td>Fever</td>
<td>Gram-stain + for gram-negative intracellular diplococci</td>
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<td></td>
<td></td>
<td>Temperature &gt;38 C</td>
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<td></td>
<td></td>
<td>WBC &gt;10,000</td>
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<td></td>
<td>Pus on culdocentesis or laparoscopy</td>
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<td>Pelvic abscess on bimanual exam or U/S</td>
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### Contraception
- Extremely effects: IUDs, DMPA, implants, sterilization
- Very effective: OCPs, patch, ring
- Less effective: male/female condom, cervical cap, diaphragm, withdrawal

### Barrier-spermicidal methods:
- Prevent entry of sperm through the cervix
- Do not protect against some STIs
- No systemic SEs
- Failure rate approaches 20%
- Examples: condoms, vaginal diaphragm, spermicides

- Intrauterine contraception:
  - Inhibit sperm transport, cause failure of implantation
  - Contraindications: pregnancy, pelvic malignancy, undiagnosed vaginal bleeding

### Steroid contraception:
- Inhibit midcycle LH surge preventing ovulation, thicken cervical mucosa, alter endometrium
- SEs: fluid retention, increase gall stones, mood changes, depression
- Contraindications: pregnancy, liver disease, vascular disease, smoker, uncontrolled HTN, thrombophilia
- Examples: combination OCPs, combination vaginal ring, transdermal skin patch, progestin-only OCPs, progestin-only injectable, progestin-only subcutaneous implant

### Infertility
- Failure to conceive following 1 yr. of regular, unprotected intercourse
- Primary – never conceived
- Secondary – conceived in the past but unable in the present
- Causes: female
  - Anovulation or ovulatory dysfunction (30%)
  - Anatomic defects
- Causes: male
  - Abnormal spermatogenesis (40%)

### Pregnancy
- Pregnancy:
  - Hx of 1 or more missed periods
  - “Signs” of pregnancy:
    - Chadwick’s sign – cervix also vulva & vagina w/ bluish color, occurs at 8-12 wks. gestation
    - Goodell’s sign – softening of cervix, occurs at 4-5 wks. gestation
    - Hegar’s sign – softening of the uterine isthmus, occurs at 6-8 wks. gestation
    - McDonald’s sign – fundus flexes easily on the cervix, occurs at 7-8 wks. gestation

  - Associated fatigue, N/V, breast swelling/tenderness

  - Exam:
    - Uterus changes
    - Cervix: Chadwick’s & Hegar’s (softening of cervix) signs
    - Ultrasound – detects fetus at 5-6 wks.
    - Fetal heart tones: Doppler at 10-12 wks.
    - Palpable fetal parts
    - Appreciable fetal movements at 16-20 wks. (quickening)

  - Increased urine hCG: test + 4 wks. After conception
  - Increased serum hCG: quantitative hCG
  - Increased serum progesterone

### Contraception
- If anovulation or ovulatory dysfunction:
  - Basal body temperature chart – temperature-rise w/ ovulation, serum progesterone measure mid-luteal phase

  - If anatomic defect:
    - Hysterosalpingogram, diagnostic laparoscopy, or hysteroscopy

  - Abnormal spermatogenesis:
    - Semen analysis for volume, concentration, motility, viscosity, morphology, pH, WBCs

### Infertility
- Treatment underlying cause
- Ovulation induction w/ clomiphene citrate
- Intrauterine insemination
- In vitro fertilization

### Pregnancy
- Increased urine hCG: test + 4 wks. After conception
- Increased serum hCG: quantitative hCG
- Increased serum progesterone
- Ladin’s sign – softening of the uterus after 6 wks.
- Piskacek’s signs – palpable lateral bulge or soft prominence of one uterine cornu, occurs at 7-8 wks. gestation

**Normal labor/delivery**

- Braxton–Hicks contractions (false labor):
  - Spontaneous uterine contractions occurring late in pregnancy
  - Not associated w/ dilation of the cervix
- Lightening:
  - Fetal head descending into pelvis results in change in shape of abdomen & sensation that baby has become less heavy
- Bloody show:
  - Passage of blood-tinged mucous late in pregnancy
  - Occurs as cervix begins thinning out (effacement) w/ associated extrusion of mucous from endocervical glands
- Ruptured membranes:
  - May present w/ sudden gush of liquid or constant leakage of fluid
- True labor:
  - Contractions that are felt over the uterine fundus w/ radiation of discomfort to the lower back & lower abdomen
- Vaginal examination:
  - Not in the presence of significant bleeding
  - Determine effacement of cervix (%)
- Shortening of the cervical canal:
  - Determine relative position of cervix (anterior, mid-position, posterior)
  - Dilation of cervix (0-10)
  - Fetal station & presentation

**Stage 1:** onset of labor to full dilation of cervix (10 cm), divided into 2 phases:
  - Latent phase: cervical effacement & early dilation
  - Active phase: more rapid cervical dilation, usually beginning at 3-4 cm
**Stage 2:** complete dilation of cervix through delivery of infant
**Stage 3:** begins after delivery of infant & ends w/ delivery of placenta
**Stage 4:** immediate postpartum period of ~2 h after delivery of placenta

**Postpartum:**

- Uterus: ~12 wk. size, barely palpable above symphysis pubis, normal size 6 wks. postpartum
- Lochia serosa (pinkish-brown vaginal bleeding): postpartum days 4-10, resolves by 3 wks.
- Breasts – breast milk on postpartum days 3-5, bluish white

**Postpartum mood disorders:**

<table>
<thead>
<tr>
<th></th>
<th>Postpartum blues</th>
<th>Postpartum depression</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td>70-80%</td>
<td>≥ 10%</td>
</tr>
<tr>
<td><strong>Average time</strong></td>
<td>2-4 d postpartum</td>
<td>2 wks. to 2 mos.</td>
</tr>
<tr>
<td><strong>Average duration</strong></td>
<td>2-3 d, resolve w/in 10 d</td>
<td>3-14 mos.</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td>Mild insomnia, tearfulness, fatigue, irritability, depressed affect</td>
<td>Irritability, labile mood, difficulty falling asleep, phobias, anxiety, worse in the evening</td>
</tr>
<tr>
<td><strong>Mother cares about baby</strong></td>
<td>Yes</td>
<td>May have thoughts about hurting baby</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>None, self-limited</td>
<td>Antidepressants</td>
</tr>
</tbody>
</table>

**Prenatal diagnosis/care**

- Initial screening:
  - Risk assessment & maternal hx
  - Estimated date of delivery (EDD) – 1st day of LMP + 7 d – 3 mos. &/or U/S for gestational age (Naegle’s rule)
  - Complete physical exam
  - Pelvic exam for uterine size
  - CBC, UA, blood group/Rh, antibody screen, serologic tests for syphilis (RPR, VDRL), HIV, hepatitis B, rubella titer, Pap smear, tests for chlamydia & gonorrhea

**Subsequent prenatal visits:**

- BP, wt. & check for edema
- Routine laboratory screening – urine for glucose & protein
- Fundal height measurements
- Glucose tolerance test (24-28 wks.)
- MS α-Fetoprotein – measured at 15-20 wks.
  - Increased in open neural tube defects (ONTD)
  - Decreased in Down’s syndrome (trisomy 21)
- Rho immune globulin (RhOGAM at 28 wks. & w/in 72 h of childbirth)
- Fetal heart tones (120-160 bpm)
- Group B strep (32 wks.)
- Determination of presentation or “lie” – Leopold’s maneuvers

**Abortion**

- Abortion:
  - Termination of pregnancy before 20 wks.
- Estimated at about 50% of all pregnancies
  - Threatened abortion:
  - Possible pregnancy loss
  - Pregnancy
  - Prognosis unpredictable
- Inevitable abortion:
  - Pregnancy cannot be salvaged
  - Prognosis is poor
- Incomplete abortion:
  - Prognosis is poor
- Complete abortion:
  - All products of conception are expelled before 20 wks.
- Missed abortion:
  - Embryo is not viable but is retained in utero for at least 6-8 wks.
- Recurrent pregnancy loss:
  - 3 or more consecutive spontaneous abortions
  - Etiologies: genetic, autoimmune, anatomic
- Elective abortion:
  - Most start at 6 wks. (missed 2nd period)
  - Medical abortion – using oral medication, can be done up to 7 wks. gestation:
    - Mifepristone (RU-486): anti-progestin
    - Methotrexate: anti-metabolite
    - Misoprostol: prostaglandin
  - Surgery should be done before 24 wks. from LMP, 22 wks. gestationally (vacuum extraction most common)

- Vaginal bleeding – bright red & usually significant, saturation of 1 sanitary pad Q hr. is significant
- Low back pain, abdominal cramping
- Passage of products of conception
- Threatened abortion:
  - Slight bleeding before 20 wks.
  - Uterine size compatible w/ dates
  - Cervical os is closed
  - No products of conception are passed
- Inevitable abortion:
  - Moderate bleeding
  - Moderate to severe uterine cramping
  - Cervical os is dilated
  - Uterine size is compatible w/ dates
  - Products of conception are not passed
- Incomplete abortion:
  - Some products of conception are passed before 20 wks.
  - Moderate to severe uterine cramping
  - Uterine contractions have subsided
  - Uterus is normal prepregnancy size
  - Cervical os may be opened or closed
- Missed abortion:
  - No uterine contractions
  - Bleeding may initially be absent, but spotting begins & later becomes heavier

### Abruptio placentae
- 3rd trimester bleeding
- Premature separation of the placenta from uterine wall
- Shares some characteristics w/ placenta previa (vaginal bleeding)
- Interferes w/ oxygenation of the fetus
- Severe abdominal discomfort & painful uterine contractions
- Hemodynamic stabilization & delivery

### Cesarean section
- Delivery of a fetus, placenta & membranes through an abdominal & uterine incision
- Indications: repeat C-section, cephalopelvic disproportion/dystocia (“difficult labor” – labor progresses then stops completely or becomes prolonged), abnormal fetal lie (transverse) & malpresentation (breech), fetal HR abnormalities, placenta previa, preeclampsia
- Complications:
  - Postpartum hemorrhage
  - Endometritis
  - Wound infxn
| eclampsia, placental abruption, multiple gestation, fetal abnormalities, cervical cancer & active genital herpes infxn, OR PATIENT CHOICE |  | Dystocia -Abnormal progression of labor -Shoulder dystocia – anterior shoulder of infant cannot pass below the pubic symphysis -3 categories of abnormality:  - Power – uterine contractions  - Incoordinate uterine activity  - Passenger – position, size or presentation of the fetus  - Passage – pelvis/soft tissue | -Stage 1 of labor: if labor >14-20 h, amniotomy or oxytocin  -Stage 2 of labor: if stage >2 h, increased risk of fetal morbidity & mortality – options include oxytocin or C-section  |
|---|---|---|
| Ectopic pregnancy -Implantation of fertilized ovum outside the uterine cavity -Risk factors: hx of infertility, hx of prior ectopic pregnancy, hx of tubal ligation or reconstruction, hx of PID, hx of IUD use, endometriosis | -1-2 mos. of amenorrhea -Unilateral abdominal pain -Possible mild vaginal bleeding -Malaise & syncope -Referral of pain to shoulder (blood irritates diaphragm) -Atypical presentations – vague or subacute sx, menstrual irregularity -Exam: signs of shock, unilateral abdominal tenderness -Pelvic exam: vagina, cervix, bimanual exam  | -Assess serum beta-hCG levels – should double every 24-48 h  -U/S to determine presence of intrauterine pregnancy  -Culdocentesis: non-clotted blood present  -Definitive dx w/ laparoscopy  -Laparoscopic repair/removal may be necessary to save the reproductive area  -Methotrexate to clear remaining trophoblastic tissue  -Follow up w/ RhoGAM if needed  -Use contraception for at least 20 mos. following  |
| Fetal distress -Fetal monitoring:  - Quantification of fetal activity  - Electronic fetal monitoring  - U/S  - Non-stress test (NST):  - Measures fetal heart rate for 20 min., also monitors fetal movement  - A reactive (normal) test shows fetal HR acceleration  - Contraction stress test (CST):  - Measures response of fetal HR to the stress of a uterine contraction  - A negative (normal) test shows no fetal HR deceleration  | |  |

### Nonstress test (NST) Criteria

<table>
<thead>
<tr>
<th>Reactive ST</th>
<th>Non-reactive ST</th>
<th>Negative CST</th>
<th>Positive CST</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;2 accelerations in 20 min.; increased fetal HR ≥15 bpm &amp; lasting ≥15 seconds</td>
<td>No FHR acceleration or did not meet criteria</td>
<td>No late decelerations are seen in the presence of 3 uterine contractions in 10 min.</td>
<td>Repetitive late decelerations are seen in the presence of 3 uterine contractions in 10 min.</td>
</tr>
</tbody>
</table>

### Assessment

<table>
<thead>
<tr>
<th>Reactive ST</th>
<th>Non-reactive ST</th>
<th>Negative CST</th>
<th>Positive CST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reassuring of fetal well-being</td>
<td>Sleeping, immature or sedated fetus; acidotic, compromised fetus</td>
<td>Reassuring of fetal well-being</td>
<td>Worrisome, especially if nonreactive NST</td>
</tr>
</tbody>
</table>

### Follow-up

<table>
<thead>
<tr>
<th>Reactive ST</th>
<th>Non-reactive ST</th>
<th>Negative CST</th>
<th>Positive CST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repeat weekly/biweekly</td>
<td>Vibroacoustic stimulation</td>
<td>Repeat CST weekly as needed</td>
<td>Prompt delivery</td>
</tr>
</tbody>
</table>

### Note

<table>
<thead>
<tr>
<th>Reactive ST</th>
<th>Non-reactive ST</th>
<th>Negative CST</th>
<th>Positive CST</th>
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<tbody>
<tr>
<td>If still NR: do contraction stress test or biophysical profile</td>
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</table>

### Gestational diabetes

- Glucose intolerance identified during pregnancy – typically subside postpartum -Identified during prenatal screening -Positive if blood sugar >140 mg/dL w/ 1 h glucose challenge -Patient education -Diet -Insulin if not well controlled – does not cross the placenta
**Gestational trophoblastic disease**
- Hydatiform mole (molar pregnancy)
- Neoplasm derived almost entirely from abnormal placental proliferation
- Potential for malignant transformation
- Clinical presentation as a pregnancy
- Uterine size/date discrepancies
- Bleeding (<16 wks.) suggestive of spontaneous abortion
- Profound hormonal changes
- U/S: “snowstorm” appearance & absence of fetal parts
- Removal of intrauterine contents

**HTN in pregnancy**
- Defined as sustained SBP ≥140 mmHg or DBP ≥90 mmHg
- Pathophysiology: maternal vasospasm
- HTN w/ no other sx is called transient or gestational HTN
- Preeclampsia is the development of HTN w/ proteinuria or edema or both (2nd half of pregnancy)
- Eclampsia is the presence of convulsions in a woman who has met the criteria of preeclampsia
- Chronic HTN is HTN present before the 20th week or after 6 wks. postpartum
- Chronic HTN:
  - Not indicated unless SBP >150 or DBP >100
  - Options include methyldopa (DOC), labetalol (bet-blockers are linked to intrauterine growth retardation), or nifedipine
  - Avoid ACE i & diuretics
- Preeclampsia:
  - Rest & frequent monitoring
  - Magnesium sulfate to prevent seizures
  - Hydralazine &/or labetalol for HTN
- Eclampsia:
  - Life-threatening
  - Magnesium sulfate
  - Hydralazine &/or labetalol for HTN
  - Delivery

**Multiple gestation**
- Incidence has risen significantly d/t fertility drugs, superovulation & assisted reproductive technologies
- More likely to be complicated by HTN, gestational DM, anemia, preterm birth, ante- & postpartum hemorrhage & maternal death
- Perinatal mortality is 3-4x higher for twins
- Monozygotic twins (identical) – division of a single fertilized ovum – susceptible to twin-twin transfusion syndrome
- Dizygotic twins (fraternal) – separately fertilized ova
- Same as normal pregnancy
- Uterus larger than expected for dates (>4 cm)
- Excessive weight gain that is not explained by edema or obesity
- Polyhydraminos (10x more common in multiple pregnancy)
- Simultaneous recording of different fetal HR
- Palpation of 1 or more fetuses in the fundus after delivery of 1 infant
- Delivery should be done in the OR in case emergent C-section is needed for twin B
- Postpartum hemorrhage is common → IV ergot or prostaglandin may be required
  - Current recommendations are prophylactic rectal misoprostol in the OR followed by PO misoprostol Q 6 h for 24 h
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placenta previa</td>
<td>- 3rd trimester bleeding</td>
<td>- Sudden onset of painless bleeding at 29-30 wks.</td>
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<tr>
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<td>- Abnormal placement of placenta over – or in close proximity to – the cervical os</td>
<td>- U/S to localize placenta</td>
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<td>- No digital vaginal exam</td>
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<td>- Bleeding spontaneously resolves</td>
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<td>- Hospitalization, bed rest, C-section when fetal maturity is attained</td>
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<tr>
<td>Postpartum hemorrhage</td>
<td>- Common cause of maternal death w/in 24 h of delivery</td>
<td>- Coagulation screen – PT/INR</td>
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<td>- Sequelae include RDS, DIC, pituitary necrosis</td>
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<td>- Most common cause is uterine atony</td>
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<td>- Risk factors: prolonged labor, rapid labor, hx of postpartum hemorrhage, overdistended uterus, operative delivery</td>
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<td>- Fluid resuscitation</td>
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<td>- Blood transfusion if &gt;2000 mL loss</td>
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<td>- FFP if coagulation test results are abnormal, if FFP is unsuccessful then give cryoprecipitate</td>
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<td>- Oxytocin, prostaglandin or ergonovine</td>
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<td></td>
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<td>- Ligate uterine blood supply</td>
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<td></td>
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<td>- Consider hysterectomy</td>
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<tr>
<td>Premature rupture of membranes</td>
<td>- Rupture of chorioamnionic membrane before onset of labor</td>
<td>- Await spontaneous labor</td>
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<tr>
<td></td>
<td>- Associated w/ preterm labor &amp; increased neonatal morbidity &amp; mortality</td>
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<tr>
<td></td>
<td>- Risk factors: STIs, smoking, prior PROM, prior preterm delivery, multiple gestations</td>
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<tr>
<td></td>
<td></td>
<td>- Monitor for infxn</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Chorioamnionitis – infxn of fetal membranes &amp; amniotic fluid → treat w/ abx &amp; delivery</td>
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<tr>
<td>Rh incompatibility</td>
<td>- Several hundred blood group systems</td>
<td></td>
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<tr>
<td></td>
<td>- ABO &amp; Rh most common</td>
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<tr>
<td></td>
<td>- Mother lacks the Rh antigen (Rh-) &amp; father has the Rh antigen (Rh+) – if any mixing of maternal/fetal blood (&amp; fetus is Rh+) there is potential for Rh isoimmunization</td>
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<tr>
<td></td>
<td></td>
<td>- Prophylaxis –Rh antigen is determined during initial assessment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- If not present in mother, RhoGAM (pooled anti-D IgG) is administered at 28 wks. &amp; w/in 72 h of birth</td>
</tr>
<tr>
<td>Condition</td>
<td>General Characteristics</td>
<td>Symptoms</td>
</tr>
<tr>
<td>------------------------</td>
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<td>-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| **Acute bronchitis**   | - Inflammation of large airways of the tracheobronchial tree d/t an infectious agent  
- Most prevalent in winter & early spring  
- Commonly caused by viruses: adenovirus, influenzae, parainfluenzae, coxsackie  
- Bacteria: *Bordatella*, *chlamydia*, *Mycoplasma*  
- Usually follows a URI  
- Risk factor: smoking | - Cough preceded by URI sx – nasal congestion, sore throat, sneezing  
- No findings of pneumonia  
- Dyspnea is rare  
- Sx > 1 wk.  
- Exam: findings of URI w/ possible rales, crackles or wheezing | - CXR normal  
- WBC slight • w/ no L shift | - D/c smoking  
- Supportive – rest, hydration, abx (erythromycin, azithromycin for atypical organisms)                                                                                                                                                                                                                                                                                                                                                                                                                                                                       |
| **Acute bronchiolitis**| - Inflammation & infection of the small airways  
- Usually an illness of young infants & children <2 yrs.  
- Etiology:  
  - RSV > 50% of cases  
  - Other agents associated: adenoviruses, parainfluenza, *Mycoplasma pneumonia* | - Characterized by respiratory distress & wheezing  
- Begins w/ common cold sx  
- Sx • in severity as airways become obstructed  
- Decreased appetite, fever  
- Wheezing, cyanosis, tachypnea  
- Respiratory distress w/ nasal flaring, intercostal retractions & prolonged expirations | - Hypoxemia  
- CO2 retention → impending respiratory failure  
- Identification of RSV by rapid immunofluorescence analysis nasal specimens  
- WBC count normal  
- Radiographic changes:  
  - Hyperinflation  
  - Depressed diaphragms  
  - No discrete infiltrates  
  - Patchy atelectasis  
  - Increased lung volumes | - Supportive care – oxygen, monitor for apnea, mechanical ventilation  
- Bronchodilators – often used but questionable efficacy  
- Ribavirin – w/ hx of congenital heart dz or chronic lung dz  
- Prevention:  
  - Passive immunization – RSV immune globulin, useful in children at high risk for adverse outcomes (premature infants & infants w/ chronic lung dz)  
  - Palivizumab (Synagis) – monoclonal Ab used to prevent RSV                                                                                                                                                                                                                                                                                       |
| **Acute epiglottitis** | - Life threatening supraglottitis/epiglottic infxn – may result in acute airway obstruction  
- Caused by *Strep. pyogenes*, or *Staph. aureus* or *mycoplasma*  
- *H. influenzae* type B is uncommon in N. America as a result of immunization | - Fever  
- Dysphagia  
- Respiratory distress/stridor  
- Symptoms may overlap croup, but toxicity suggests epiglottitis:  
  - Severe sore throat  
  - Drooling  
  - Absence of hoarseness  
  - Child’s insistence on sitting forward w/ neck hyperextended | - Cherry-red epiglottitis on laryngoscopy  
- Tongue depressor or exam of oropharynx may cause acute airway obstruction  
- Lateral neck XR – thumb sign – enlarged epiglottitis  
- Once airway secure obtain blood cx, CBC, culture of epiglottis | - Secure airway – child should not be disturbed until personnel is present & ready to perform intubation or tracheotomy  
- Abx – IV ceftriaxone or cefotaxime for 7-10 days  
- Prevention: vaccination for *H. influenzae* type B, rifampin to eliminate carriers & treat close contacts                                                                                                                                                                                                                                                                                                                |
| **Croup**              | - Viral infxn (parainfluenza viruses most common) of the glottic/subglottic region  
- Epidemiology: age 7-36 mo., very few cases >6 yrs.  
- Common in fall or winter | - Inspiratory stridor (may be dramatic & alarming; usually follows days of milder cold sx, worsens at night, & may be accompanied by cough & hoarseness)  
- Barking cough  
- Retractions | - Neck XR (typically not needed):  
  - Steeple sign  
  Differentiate from epiglottitis w/ lateral XR | - Goal is to reduce airway edema/obstructive sx  
- Humidified air  
- Nebulized epinephrine – may have rebound after several hrs.  
- Corticosteroids  
- Abx not needed                                                                                                                                                                                                                                                                                                                                                                                               |
### Influenza
- Systemic viral illness d/t influenza A & B
- Spread by respiratory droplets in an epidemic pattern – very contagious
- Incubation 1-3 days

- Toxic appearance
  - Abrupt onset fever 101-106 F, chills, rigors, malaise
  - Myalgias, HA, nonproductive cough
  - Coryza & sore throat, clear nasal drainage
  - Exam usually normal

- Leukopenia or normal WBC
  - Rapid antigen detection of nasopharyngeal secretions
  - Viral culture

- Symptomatic – acetaminophen, avoid ASA d/t Reye’s syndrome
- Oral oseltamivir (Tamiflu), inhaled zanamivir (Relenza)
- Amantadine, rimantadine (used for influenza A but AVOID)
- High-risk groups in need of vaccine → elderly, nursing home, hx of COPD/cardiac dz, chronic dz (DM, renal dz, hemoglobinopathies), pregnant in 2nd or 3rd trimester, children 6-60 mos., groups in contact w/ high-risk pts

### Pertussis
- Whooping cough
- Etiology: caused by Bordatella pertussis & Bordatella parapertussis (gram-negative coccobacilli)
- Spread through close contact w/ infected person
- Incubation 4-10 days

- Catarrhal phase – cold-like sx → rhinorrhea, conjunctival injection, cough
- Paroxysmal phase – increasing to severe paroxysmal cough, inspiratory whoop
- Convalescent phase – 2 wks. of gradual resolution

- Marked lymphocytosis (may not be present in infants)
- Organisms cultured on special media
- Direct fluorescent antibody test of nasopharyngeal secretions
- CXR usually normal

**Avoid in pts w/ egg allergy**

### Bacterial pneumonia
- Causes of community-acquired pneumonia:
  - Atypical (make up 10% of CAP)
    - Chlamydia pneumonia – college students
    - Legionella pneumophila – aerosol transmission for contaminated water
    - Mycoplasma – crowded areas
    - Viruses (RSV, influenza)
  - Typical
    - S. pneumoniae - most common cause in all age groups
    - Haemophilus influenza – children usually vaccinated against this
    - S. aureus
    - Gram-negative bacilli
  - Hospital-acquired pneumonia:
    - Very common hospital infxn – incidence w/ length of stay
    - Complications: ARDS
    - Frequently polymicrobial in origin:

- S. pneumoniae:
  - High fever - adults w/103
  - Productive cough
  - Occasionally hemoptysis
  - Pleuritic chest pain
  - Rigors w/in the first few hrs. but are uncommon thereafter
  - Bronchial breath sounds are an early sign

- H. influenzae:
  - Insidious (slow) onset.
  - Fever, cough & purulent sputum production

- Mycoplasma pneumoniae: NUMBER ONE ATYPICAL PNEUMONIA!
  - Chest pain
  - Chills, fever, sore throat
  - Cough, usually dry & w/o hemoptysis
  - Excessive sweating
  - Headache
  - WATERY SPUTUM

- Chlamydia pneumoniae:
  - Usually mild, chills, cough, fever, HA

- CXR
  - S. pneumoniae:
    - Lobar pneumonia w/ consolidation & occasionally effusion
  - H. influenzae:
    - No consolidation, more around heart & periphery
  - Chlamydia pneumoniae:
    - Interstitial findings
  - Legionella pneumophila
    - Lobar, 50% have pleural effusion, often shows patchy alveolar infiltrates w/ consolidation in lower lobes

- Pneumocystis jiroveci
  - Diffuse interstitial dz
  - Interstitial findings: Viruses, “atypical” agents (mycoplasma, chlamydia, rickettsia), fungi
  - Alveolar findings: bacteria (pneumococci, legionella, klebsiella)
  - Sputum culture:
    - Rust colored – pneumococcal infxn

- Community-acquired pneumonia:
  - Outpt - PO Macrolide (azithromycin, clarithromycin, erythromycin) OR oral doxycycline
  - Outpt w/ structural lung dz - PO “respiratory” FQ (levo or moxi) OR β-lactam (amoxicillin, Augmentin, cefpodoxime, cefuroxime PLUS PO macrolide
  - Inpt (if co-morbid conditions)- IV beta-lactam + macrolide or a fluoroquinolone
- Macrolides → Mycoplasma
- Tetracycline → Chlamydia
- Oseltamivir (Tamiflu), zanamivir (Relenza)

- PSI (pneumonia severity index)
  - Outpt in class I (no points) & II (<70) are excellent candidates for outpt oral therapy
  - Inpt in class III (71-90) may be considered for outpt or brief inpt therapy,
Gram-negative bacilli isolated in 47% (most common)
- Anaerobic bacteria in 35%
- S. aureus in 26% - infants, young children & debilitated pts
- Nosocomial gram-negative pneumonias often occur in pts w/ serious acute comorbid dz & are associated w/ high mortality
- Pseudomonas, Klebsiella pneumoniae, & E. coli are the most common gram negative
- Acinetobacter, Citrobacter, Proteus, & Serratia are also important causes b/c often resistant to multiple abx, making effective tx difficult
- Aspiration pneumonia – stroke, dementia, & altered mental status (drugs, alcohol) pts at high risk

- Legionella pneumophila – mainly in immunocompromised or elderly
  - Incubation period of 2-10 days
  - Prodromal mild HA & myalgias followed by high fever, chills & multiple rigors
  - Cough in 90% of the cases – non productive at first
  - Pleuritic chest pain
  - Hemoptysis
  - Non pulmonary sx are prominent early in the dz

- Staphylococcus pneumonia
  - Short prodrome of fever followed by rapid onset of respiratory distress
  - Tachypnea, retractions, cyanosis
  - May also have GI sx
  - May develop after initial viral infxn → seen usually during influenza season

- Pneumocystis jiroveci
  - Fever, dyspnea, nonproductive cough, chills
  - Chest discomfort
  - Weight loss

- Currant jelly sputum – Klebsiella infxn
  - PMNs & monocytes w/o bacteria - Legionella

- depending on clinical judgment.
  - Pts in risk classes IV (91-130) & V (>130) are recommended for hospital admission.

- CRB-65: pts are better served w/ inpt care if they meet more than 1 of the following:
  - Confusion
  - Respiratory rate > 30
  - BP < 90mm Hg systolic or 60mm Hg diastolic
  - Age > than 65 years

-Hospital-acquired pneumonia:
- Ceftriaxone, FQ, ceftazidime, imipenem, aminoglycoside
- Aspiration pneumonia – abx (amoxicillin, clindamycin, FQ)

-Indications for pneumococcal vaccine:
- Healthy persons >65 yrs.
- Underlying lung, cardiac, liver or renal dz
- Immunosuppression or chemotherapy (steroids, etc.)
- Alaskan Natives, Native Americans
- Re-dose in 5 yrs. if severely immunocompromised or original vaccination at age <65 yrs.

## Etiologies of pneumonia:

<table>
<thead>
<tr>
<th>Factor</th>
<th>Etiology</th>
<th>Factor</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking, COPD</td>
<td>S. pneumoniae H. influenzae</td>
<td>Recent hx of influenza</td>
<td>S. aureus S. pneumoniae H. influenzae</td>
</tr>
<tr>
<td>Nursing home resident</td>
<td>S. pneumoniae Gram-negative bacilli H. influenzae TB</td>
<td>Cystic fibrosis</td>
<td>Pseudomonas aeruginosa</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>S. pneumoniae Klebsiella pneumoniae</td>
<td>IVDA</td>
<td>S. aureus Anaerobes TB</td>
</tr>
<tr>
<td>Exposure to bats</td>
<td>Histoplasma capsulatum</td>
<td>Aspiration</td>
<td>Anaerobes</td>
</tr>
<tr>
<td>Exposure to birds</td>
<td>Cryptococcus neoformans</td>
<td>HIV/AIDS</td>
<td>Pneumocystic jirovecii</td>
</tr>
<tr>
<td>------------------</td>
<td>-------------------------</td>
<td>----------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>Young, healthy adults</td>
<td>Mycoplasma</td>
<td>Water source</td>
<td>Legionella</td>
</tr>
</tbody>
</table>

Respiratory syncytial virus
- Most important cause of lower respiratory tract infection in children
- Peak season: cold weather in temperate climates
- Epidemics in late fall & early spring
- Leading cause of bronchiolitis
- Complications: secondary bacterial infection of middle ear
- First rhinorrhea & pharyngitis, then cough, wheezing & fever
- Tachypnea, difficult feeding
- Exam: hyperinflation, crackles, wheezing, prolonged expiration
- If conjunctivitis present, think Chlamydia trachomatis – also no fever or wheezing
- Positive RSV antigen nasal or pulmonary
- Imaging: diffuse hyperinflation & peribronchial thickening
- Oxygen
- Bronchodilator
- Ribavirin

TB
- Etiology:
  - *Mycobacterium tuberculosis*
  - Transmitted by respiratory droplets
  - Most exposed pts do not progress to clinical illness
- Cough, fever, chills, night sweats, anorexia, weight loss, & fatigue
- Positive PPD:
  - ≥5 mm
  - >10 mm
  - >15 mm

| HIV + | Travel to high prev. country | No risk factors |
| Contact w/ active TB | | |
| Contact w/ active TB | IVDA | |
| Old, healed TB | Work/resident of jails, nursing home/hospital, homeless shelter | |
| Organ transp. | Clinical condition that places pt at high risk | |
| Immunecomp. | <4 yrs. or exposed to adults in high-risk setting | |

- Latent TB:
  - INH 2x/wk. for 9 months
  - Rifampin QD for 4 months (6 months in children)
- Culture positive TB:
  - Initial: RIPE – 7 days a wk. x 8 wks.
  - Continuation:
    - INH/rifampin – 7 days a wk. x 18 wks.
- Isoniazid – bactericidal, inhibits synthesis of mycolic acid in mycobacterial cell wall
- Side effects:
  - Hepatitis – monitor AST/ALT
  - Peripheral neuropathy: rare, but minimize w/ pyridoxine (B6)
  - Abdominal pain, n/v
  - Increases toxicity from phenytoin & theophylline
- Rifampin – bactericidal, inhibits DNA-dependent RNA polymerase
**Carcinoid tumors**
- Low-grade malignant neoplasms – slow growing & rarely metastasize
- Most pts <60 yrs.
- Hemoptysis, cough, focal wheezing, recurrent pneumonia
- Carcinoid sx (flushing, diarrhea, wheezing, hypotension) are rare
- CT scan
- Octreotide scintigraphy for localization of tumor
- Surgery, resistant to chemotherapy & radiation therapy

**Lung cancer**
- Leading cause of cancer death
- Two groups: non-small cell (adenocarcinoma, squamous cell, large cell) & small cell
- Risk factors:
  - Cigarette smoking (10x greater risk than nonsmokers)
  - Asbestos exposure; radon exposure
  - Exposure to uranium, arsenic, chromium & nickel
  - History of COPD
  - Idiopathic pulmonary fibrosis
- Squamous cell:
  - Cough, chest pain, weight loss, dyspnea, hemoptysis
- Adenocarcinoma:
  - Lymphadenopathy, hepatomegaly, & clubbing
- Small cell:
  - Paraneoplastic syndromes (15-20%)
    - SIADH, Cushing’s syndrome, Lambert-Eaton syndrome
  - No clubbing
  - Non-small cell:
    - Severe pain in shoulder & along inner side of arm & hand
    - Atrophy d/t invasion of brachial plexus
    - Horner syndrome (drooping eyelids, sweating, pupil contraction) d/t invasion of paravertebral sympathetic chain & stellate ganglion
- Squamous cell:
  - CXR (hilar or peripheral masses, cavitation, effusions), cytology, bronchoscopy
  - Hypercalcemia
- Adenocarcinoma:
  - CEA
  - Bronchoscopy
  - CXR – small peripheral masses
- Small cell:
  - Electrolytes; bronchoscopy
  - CXR – hilar mass, wide mediastinum
  - Non-small cell:
    - CXR, CT, MRI
- Non-small cell:
  - Surgery
  - RT & chemotherapy before surgery b/c of location

**Side effects:**
- Hepatitis
- Induces CYP450 & metabolism of many drugs (Coumadin, BCP)
- Orange discoloration of body fluids
- Pseudomembranous colitis
- Pyrazinamide – bactericidal, unknown mechanism, works best in acid environment
- Side effects:
  - Live toxic, hyperuricemia, not used in pregnancy
- Ethambutol – bacteriostatic, inhibits the enzyme involved in synthesis of cell wall, eliminated by the kidneys
- Side effects:
  - Optic neuropathy – diminished visual acuity, loss of color vision
**Pulmonary nodules**

- Non-small cell:
  - Pancoast tumor
  - Mass in extreme apex of lungs – invades other tissues (lymph nodes, nerves, & ribs) leading to other sx

- Solitary pulmonary nodule:
  - 25% are primary bronchogenic cancer
  - 10% are mets

- Round, oval, sharp lesion up to 3 cm in diameter (>3 cm = a “mass”)
- Central cavitation & calcification may occur

- Biopsy:
  - Benign: has not enlarged in >2 yrs., calcified
  - Malignant: occasionally symptomatic, age >45, size >2 cm, rarely calcified, indistinct margins

- Exploratory thoracotomy or thoracoscopy

**Asthma**

- Chronic inflammatory disorder of the airways that is reversible
  - Three major characteristics:
    - Obstruction of airflow
    - Bronchial hyper-reactivity
    - Inflammation of airways
  - Status asthmaticus:
    - Unremitting asthma w/ rapidly increasing severity
    - D/t diffuse bronchial obstruction leading to hypoxia &/or respiratory muscle fatigue

- Persistent wheeze, chronic episodic dyspnea, chronic cough, & • respiratory rate
- Asx periods

<table>
<thead>
<tr>
<th>Step</th>
<th>Class</th>
<th>Freq.</th>
<th>Nocturnal sx</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mild intermit.</td>
<td>&lt;2x per wk.</td>
<td>&lt;2x per mo.</td>
</tr>
<tr>
<td>2</td>
<td>Mild persistent</td>
<td>&gt;2x per wk.</td>
<td>&gt;2x per mo.</td>
</tr>
<tr>
<td>3</td>
<td>Moderate persistent</td>
<td>Daily</td>
<td>&gt;6x per mo.</td>
</tr>
<tr>
<td>4</td>
<td>Severe persistent</td>
<td>Cont.</td>
<td>Frequent</td>
</tr>
</tbody>
</table>

- Status asthmaticus:
  - Acute onset chest tightness, SOB, cough
  - PE: tachycardia, tachypnea, cyanosis, accessory muscle use, intercostal retractions, no wheezing

- Decreased FEV1, improved w/ bronchodilator (10% •)
- Positive methacholine challenge
- Eosinophilia, • IgE levels
- ABG: hypoxemia (<60) & hypocarbia (<40), oxygen saturation ~90%

- Status asthmaticus:
  - Hypoxemia, hypercapnia
  - Decreased peak flow & FEV1
  - CXR hyperinflation

- Acute:
  - Corticosteroids
  - Beta-adrenergic agonists – short-acting
  - Oxygen

- Chronic:
  - Corticosteroids
  - Beta-adrenergic agonists – long-acting
  - Leukotriene antagonists (zafirlukast, zileuton, montelukast)
  - Mast cell stabilizers (cromolyn, nedocromil)
  - Theophylline, vaccines

- Status asthmaticus:
  - Oxygen: maintain O2 sats at 92-94%
  - Bronchodilators every 2 h
  - Corticosteroids: high dose IV
  - Mechanical ventilation
**Bronchiectasis**

- Abnormal & persistent dilation of major bronchi & bronchioles
- D/t destructive changes as a result of severe recurrent infxn – TB, fungal, lung abscess, pneumonia
- **Etiology:**
  - Cause unknown in ½ of cases
  - Allergic bronchopulmonary aspergillosis
    - In those w/ asthma sx who don’t respond to bronchodilators
  - Cough: productive w/ sputum/mucus plugs
  - Cystic fibrosis

- Chronic purulent sputum (foul-smelling)
- Hemoptysis (blood-tinged)
- PE: crackles
- CXR: interstitial markings, linear atelectasis
- CT: dilated, tortuous airways, “signet sign”
- Goal is to minimize sx & prevent complications
- Abx, bronchodilators, chest PT
- Vaccines

**Chronic bronchitis**

- Excessive sputum production w/ chronic or recurring cough on most days for a minimum of 3 mos. of the yr. for at least 2 consecutive yrs.
- **Pathology:**
  - Smooth-muscle hypertrophy
  - Inflammation
  - Mucosal edema
  - Narrowing of airways
  - Mucus plugging
  - Peribronchial fibrosis

- Dyspnea on exertion
- Thick, yellow, copious sputum
- Blue bloater
  - Obese, cyanotic
  - Tachypnea, tachycardia
  - JVD, right-sided heart failure
  - Hepatomegaly, ascites
- PE: coarse rhonchi, wheeze, prolonged expiration, edema
- PFT: obstructive pattern (FEV1)
- CXR: normal or signs of CHF
- ABGs: hypoxemia
- Smoking cessation (dz seen after >10 yrs. of smoking)
- Anticholinergic agents (ipratropium)
- Beta agonist (albuterol)
- Methylxanthine (theophylline)
- Steroids
- Mucokinetic (guaifenesin)
- Oxygen
- Vaccines
**Emphysema**

- **Enlarged air spaces d/t destruction of alveolar septa**
  - Etiology unknown:
    - Possibly d/t proteolytic enzymes
    - Smoking
    - Alpha-1-antitrypsin
- **Minimal cough, non-productive**
- **Dyspnea**
- **Weight loss**
- **Pink puffer:**
  - Thin, cachectic
  - Pursed lip breathing
  - Barrel chest
  - Tachypnea
  - Decreased breath sounds
  - Prolonged expiration
- **PFTs: obstructive pattern (• FEV1)**
- **CXR: hyperinflation, flat diaphragm**

**COPD Summary**

<table>
<thead>
<tr>
<th>Description</th>
<th>Emphysema</th>
<th>Chronic bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major complaint</td>
<td>Pink puffer</td>
<td>Blue bloater</td>
</tr>
<tr>
<td>Age onset</td>
<td>&gt;50</td>
<td>30s to 40s</td>
</tr>
<tr>
<td>Lung exam</td>
<td>No adventitious signs</td>
<td>Rhonchial present</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>Normal</td>
<td>Elevated</td>
</tr>
<tr>
<td>Blood gases</td>
<td>pO₂ NL/↓</td>
<td>pCO₂ NL/↓</td>
</tr>
<tr>
<td>Chest x-ray</td>
<td>Hyperinflated, flat diaphragm</td>
<td>Increased interstitial markings and normal diaphragm</td>
</tr>
</tbody>
</table>

**Cystic fibrosis**

- Progressive, multi-system dz
- Autosomal recessive mode of transmission
- Most common lethal recessive dz affecting Caucasians
- CF gene – located on long arm of chromosome 7, directs production of the cystic fibrosis transmembrane conductance regulator protein (CFTR)
- Multiple complications related to viscous mucus, malabsorption & infection
- Abnormally thick bronchial secretion leading to chronic infection/recurrent pneumonia, inflammation of the respiratory tract, bronchiectasis, respiratory failure
- *Chronic productive cough*
  - Wheezing
  - Dyspnea
  - Hemoptysis
  - Pneumothorax
  - Cor pulmonale
  - Progressive respiratory failure
  - Nasal polyps
- Pancreatic:
  - Insufficiency of exocrine pancreatic function (85–90% of cases)
  - Causes malabsorption & failure to thrive
- Chronic diarrhea, abdominal bloating, ravenous appetite, steatorrhea (fatty malodorous stools), rectal prolapse, excessive flatulence, 2/2 vitamin deficiency (A, D, E, K)
- Respiratory: allergic bronchopulmonary aspergillosis
- GI: meconium ileus
- Hepatobiliary dz: prolonged neonatal jaundice, cholelithiasis
- Reproductive: infertility in men, reduced fertility in women
- **Sweat chloride test elevated >60 mEq/L**
- **Genotype testing:**
  - Offered for up to 62 of the most common mutations
  - Appropriate for screening in families of a pt w/ CF
- **Diagnostic:**
  - One or more phenotypic features of CF
  - Sibling w/ CF or + newborn screening
  - + Sweat chloride test
  - 2 mutations known to cause CF
- Acute exacerbations:
  - Abx – guided by results of ongoing sputum cultures, cover *Pseudomonas & Burkholderia*
  - Corticosteroids – used short-term or prolonged to treat airway inflammation
  - NSAIDs – ibuprofen, may slow progression of inflammatory process
  - Chest physiotherapy
  - Mucous-thinning drugs: N-acetylcysteine, deoxyribonuclease (DNase), may reduce frequency of acute exacerbation
  - Lung transplantation – when forced expiratory volume is stable at 30–40% of predicted value
  - Pancreatic insufficiency – pancreatic enzyme supplements, fat-soluble vitamins A, D, E, K
### Pleural effusion

Transudates (from vessel leakage into pleural space):
- CHF
- Nephrotic syndrome
- Malnutrition
- Cirrhosis
- Ascites
- PE

Exudates (from an inflammatory process):
- Infxn
- Malignancy
- PE
- Collagen vascular (RA, SLE)
- Trauma
- Uremia
- Pancreatitis

**BOLDED ARE 3 MOST COMMON CAUSES OF PLEURAL EFFUSION**

<table>
<thead>
<tr>
<th>Exudates</th>
<th>Transudates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>&gt;3 g/dL</td>
</tr>
<tr>
<td>Pleural/serum protein</td>
<td>&gt;0.5</td>
</tr>
<tr>
<td>LDH</td>
<td>&gt;200 IU/L</td>
</tr>
<tr>
<td>Pleural/serum LDH</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>Glucose</td>
<td>&lt;60 mg/dL</td>
</tr>
</tbody>
</table>

- CXR: blunting of costophrenic angle, loss of landmarks:
  - Unilateral – think exudate
  - Bilateral – think transudate

- Thoracentesis if large amount of fluid causing SOB, chest pressure or breathing problems
- Diuretics if d/t CHF

### Pneumothorax

- Accumulation of air in pleural space
- Entry through opening in visceral or parietal pleura

- Causes:
  - Spontaneous:
    - Primary – thin males, smokers, those w/ no underlying lung dz
    - Secondary – those w/ underlying lung dz
  - Traumatic – penetrating/nonpenetrating chest injury
  - Tension – chest wound/pulmonary laceration
    - Pressure + in pleural space throughout respiratory cycle
    - Occurs during mechanical ventilation or resuscitation

- Tall, thin men at greatest risk
- Acute onset ipsilateral chest pain & dyspnea
- PE: tympany on percussion, breath sounds, hyperresonance

- CXR: presence of pleural air
- Hypoxemia

- Thoracentesis if large amount of fluid causing SOB, chest pressure or breathing problems
- Oxygen
- Chest tube w/ pleurodesis

-20-30% resolve spontaneously (depends on size)
<table>
<thead>
<tr>
<th>Cor pulmonale</th>
<th>Pulmonary embolism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RVH &amp; failure d/t pulmonary dz/hypoxia</strong></td>
<td>- D/t thrombi in the venous circulation or right side of heart - 80-90% originate in deep veins of lower extremities</td>
</tr>
</tbody>
</table>
| - Etiology:  
  - COPD (most common)  
  - Pneumoconiosis  
  - Pulmonary fibrosis | - Risk factors:  
  - Hypercoagulable states: malignancy, thrombophilia  
  - Pregnancy/BCP  
  - Surgical procedures: orthopedic surgery  
  - A. fib  
  - Major trauma |
| - Both from pulmonary dz & heart effects - Chronic productive cough, exertional dyspnea, wheezing & weakness - JVD, edema, hepatomegaly, ascites | - Pleuritic chest pain (74%) - Dyspnea (85%), cough (53%), hemoptysis (30%) |
| - Polycythemia, O2 saturation - EKG: right axis deviation, deep S waves in V6, prominent P waves in II, III, AVF (atrial enlargement) - PFT: underlying lung dz - Echo: RV dilation | - ABC: hypoxemia, hypocapnia, wide A-a gradient  
  - EKG: sinus tachycardia (S1Q3T3) d/t right heart strain (cor pulmonale)  
  - S wave in lead I  
  - Q wave in lead III  
  - Inverted T waves in III  
  - CXR: normal  
  - Westermark sign (vascularity)  
  - Hampton hump (wedge-shaped infiltrate = pulmonary infarction)  
  - D-dimer (normal result = no PE in low-risk pts)  
  - Ultrasound LE  
  - V/Q scan  
  - Uses radioactive material to compare ventilation & perfusion  
  - Scoring system:  
    - Normal < 1% PE rate  
    - Low prob. 14% PE rate  
    - Intermediate 30% PE rate  
    - High prob. 87% PE rate  
  - Angiography – gold standard – very invasive & not easily available  
  - Spiral CT:  
    - 95% sensitive for large PE  
    - 75% sensitive for subsegmental PE - Wells Probability System:  
    - DVT S/S → 3 points  
    - PE as or more likely → 3 points  
    - HR > 100 → 1.5 points  
    - Immobilization/surgery → 1.5 points  
    - Previous DVT/PE → 1.5 points  
    - Hemoptysis → 1 point  
    - Malignancy → 1 point |
| - Treat the pulmonary problem  
  - Oxygen, salt & fluid restriction  
  - Diuretics  
  - No use for digoxin  
  - Prognosis: 2-5 yrs. | - If hemodynamically stable:  
  - IV or LMW heparin & oral anticoagulation (warfarin) 5-7 days  
  - Oral anticoagulation (warfarin) for at least 6 mos.  
  - If anticoagulation contraindicated → inferior vena cava filter  
  - If hemodynamically unstable:  
  - Thrombolytic therapy  
  - If anticoagulation contraindicated → pulmonary embolectomy & interrupt inferior vena cava |

<table>
<thead>
<tr>
<th>Wells score</th>
<th>Pretest prob.</th>
<th>Likelihood of PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2.0</td>
<td>Low</td>
<td>9%</td>
</tr>
<tr>
<td>2.0-6.0</td>
<td>Moderate</td>
<td>30%</td>
</tr>
<tr>
<td>&gt;6.0</td>
<td>High</td>
<td>68%</td>
</tr>
<tr>
<td>Pulmonary HTN</td>
<td>Idiopathic pulmonary fibrosis</td>
<td>Pneumoconiosis</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------------------------</td>
<td>---------------</td>
</tr>
</tbody>
</table>
| - Idiopathic pulmonary HTN:  
  - Young, middle-aged females  
  - Progressive dyspnea, fatal  
  - Secondary pulmonary HTN  
  - Reduction in cross-sectional area of pulmonary arterial bed  
  - Increased pulmonary venous pressure  
  - Increased pulmonary blood flow  
  - Increased blood viscosity  
  - COPD most common cause  
  - Pathology: high blood flow, low pressure, & low resistance  
  - Present when pulmonary artery pressure rises to a high level inappropriate for the level of cardiac output | - Progressive parenchymal scarring & loss of pulmonary function  
- Etiology unknown  
- Men > women, 40-50 yrs. of age | - Chronic, fibrotic, occupational lung dz  
- Caused by inhalation of foreign particle  
- Examples: coal worker’s lung, farmer’s lung, silicosis, asbestosis  
- Appears 20-30 yrs. after constant exposure | - Multi-systemic dz of unknown cause  
- Noncaseating granulomatous inflammation in affected organ (lung, nodes, eyes, skin, liver)  
- Seen in N. American blacks  
- Adults ages 20-30 |
| - Dyspnea, fatigue, chest pain, & syncope on exertion  
- PE: narrow splitting of second heart sound w/ loud pulmonary component | - Gradual onset exertional dyspnea, nonproductive cough  
- Fine bibasilar inspiratory crackles, clubbing  
- Cyanosis & cor pulmonale late features | - Usually asx  
- Dyspnea, inspiratory crackles, clubbing, cyanosis, cough  
- Exam: unremarkable, occupational hx | - Cough, dyspnea, fatigue, chest discomfort |
| - RVH on CXR & EKG  
- Cardiac echo  
- Pulmonary arteriogram | - CXR: bilateral reticular opacities in periphery & lower lobes  
- CT: subpleural honeycombing, ground-glass appearance  
- Lung biopsy | - Asbestosis  
- Coal worker’s Lung  
- Silicosis | - Leukopenia, eosinophilia, elevated ESR, hypercalcemia  
- Elevated ACE levels  
- Biopsy: non-caseating granulomas  
- CXR: symmetric bilateral hilar & right paratracheal adenopathy; diffuse reticular infiltrates |
| - Oxygen  
- Treat underlying disorder  
- Lung transplant  
- CCBs (idiopathic) | | | - Steroids  
- Methotrexate |

<table>
<thead>
<tr>
<th>Material</th>
<th>Occupation</th>
<th>CXR</th>
<th>PFT pattern</th>
<th>Complications</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Asbestos | Brake lining workers  
Insulators  
Shipyard workers  
Mining, milling | Reticular, basilar predominance | Restrictive | Mesotheliomas  
Bronchogenic CA  
(RA)  
Caplan syndrome (RA)  
Lung cancer • risk TB | Supportive care  
Steroids | }

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Coal miners  
Glass makers  
Sandblasters  
Pottery workers | Supportive care  
Steroids |

Other treatments: beta agonist, steroids, anticholinergic agents, oxygen.

<table>
<thead>
<tr>
<th>Asbestosis</th>
<th>Coal Worker’s Lung</th>
<th>Silicosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asbestos</td>
<td>Coal dust</td>
<td>Silica</td>
</tr>
</tbody>
</table>
| Brake lining workers  
Insulators  
Shipyard workers  
Mining, milling | Coal miners  
Glass makers  
Sandblasters  
Pottery workers | Reticular, basilar predominance | Restrictive |
| Small nodules upper lung field | Nodular, upper lobes | Nodular, upper lobes |
| Restrictive | Obstructive | Restrictive |
| Mesotheliomas  
Bronchogenic CA  
(RA)  
Caplan syndrome (RA)  
Lung cancer • risk TB | Supportive care  
Steroids  
Steroids | Supportive care  
Steroids  
Steroids |
| Supportive care  
Steroids | Steroids  
Methotrexate | Steroids  
Methotrexate |
### Acute Respiratory Distress Syndrome
- Increased permeability of alveolar-capillary membrane & pulmonary edema
- Follows systemic or pulmonary insult w/o evidence of heart failure
- Etiologies: sepsis, aspiration, trauma, drugs, multiple transfusions, pneumonia, burns, pancreatitis, DIC
- Rapid onset (12-48 h) after event
- Labored breathing, tachypnea, intercostal retractions, crackles
- CXR: diffuse or patchy bilateral infiltrates, spares costophrenic angles, air bronchograms
- Swan-Ganz catheter: normal cardiac output & capillary wedge pressure, pulmonary artery pressure
- Treat underlying cause
- Positive end-expiratory pressure (PEEP) to stabilize the lung

### Hyaline Membrane Disease
- Newborns – predisposing factors: premature, mother w/ DM, + family hx
- Deficiency of surfactant – alveoli collapse
- Dyspnea, tachypnea, nasal flaring, retractions, grunting respirations, cyanosis
- CXR: diffuse reticulogranular pattern & air bronchograms
- Corticosteroids
- CPAP

### Foreign Body Aspiration
- Accidental inhalation of foreign material into airway or esophagus
- Epidemiology:
  - Highest incidence in children <3 d/t limited mastication skills & commonly place objects in mouth in active exploration
  - Aspiration in older children – improperly chewed food, habitually chewed objects (pen tops, erasers, small toy parts)
  - Site where foreign bodies lodge: trachea, major bronchus most common location, smaller airways, esophagus (common in younger children, causes tracheal compression, stridor or cough)
- Varies according to location, size & shape of object
- Occluded upper airway – sudden & severe respiratory distress
- Peripheral airway – chronic cough
- Supraglottic region or larynx – dyspnea, stridor, retractions, croupy cough, drooling
- Small airway – asymmetric breath sounds, wheezing cough
- Trachea – dyspnea, stridor/wheezing, retractions, cough
- Bronchus – asymmetric chest movement, unilateral wheezing, cough
- Normal CXR in 80%
- Inspiratory/expiratory films may be helpful in cases of partial obstruction
- R & L decubitus films – may show partial airway obstruction
- Removal of foreign body → manual, rigid of flexible bronchoscopy, thoracotomy, abx (when evidence of secondary infxn)

**Coughing or choking episode followed by wheezing, think airway foreign body**
<table>
<thead>
<tr>
<th>General Characteristics</th>
<th>Symptoms</th>
<th>Diagnostics</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| **Candidiasis** | - Most common opportunistic fungal infxn  
- Normal flora in GI & GU tract  
- D/t *Candida albicans* | - Oropharyngeal:  
  - White plaques on buccal mucosa  
  - Erythematous base  
- Vulvovaginitis:  
  - Women child-bearing years  
  - Pruritus, curd-like discharge  
- Cutaneous:  
  - Intertinginosus areas  
  - Erythematous lesions w/ distinct borders & satellite lesions  
- Disseminated – endocarditis, candidemia:  
  - Hepatosplenic infxn in leukemic pts | - KOH prep  
- Culture | - Mucocutaneous – clotrimazole, fluconazole in AIDS  
- Vulvovaginitis – miconazole, clotrimazole  
- Disseminated – fluconazole, amphotericin B |
| **Cryptococcosis** | - Most common cause of fungal meningitis  
- Organism – *Cryptococcus neoformans*  
- Predisposing factors – Hodgkin’s, steroid therapy, HIV | - HA, mental status changes, menimgismus, visual abnormalities | - CSF: • glucose, • protein, • WBC (lymphs)  
- India ink prep  
- Latex agglutination titer | - Amphotericin B + fluocytosine for CNS infxn  
- Fluconazole PO for pulmonary disease |
| **Histoplasmosis** | - *Histoplasma capsulatum* (dimorphic fung)  
- Endemic to Mississippi & Ohio river valleys, eastern Canada, Mexico & C. America  
- Related to bird droppings & bat exposure – inhale spores | - Most asx  
- Respiratory illness  
- May disseminate in immunocompromised – leukemia, steroids, HIV | - Bone marrow +  
- Urine antigen test  
- Skin test  
- CXR: pneumonia, miliary pattern | - Itraconazole (mild to moderate dz)  
- Amphotericin B (severe dz) |
| **Pneumocystis** | - A protozoan – noted in AIDS pts w/ CD4 <200 | - Fever, chills, sweats, fatigue, dyspnea, nonproductive cough  
- Exam: bibasilar crackles or normal | - CXR: bilateral diffuse interstitial disease w/o hilar adenopathy, apices are typically spared  
- Partial pressure of oxygen  
- LDH  
- Dx made w/ bronchoalveolar lavage & special lung tissue stains | - Trim-sulfa  
- Pentamidine  
- Prednisone if PaO2 <70 mmHg  
- Clindamycin + primarquine in milde cases |
| **Acute rheumatic fever** | - Inflammatory disease in response to group A strep infxn  
- Causes inflammatory lesions on connective tissue (heart, joints, SC tissue)  
- Mechanism unkown  
- Time from infxn to onset of sx is 1-5 wks.  
- Peak incidence ages 5-15 | - 2 major or 1 major & 2 minor criteria  
- Major (Jones) criteria:  
  - Carditis – murmur apical systolic  
  - Polyarthritis – swollen, warm, red, tender, migratory, large joints (knees, ankles, wrist, elbows)  
  - Chorea – rapid, purposeless movements  
  - Erythema marginatum – macule or papule w/ central clearing | - Minor criteria (labs:  
  - Elevated sed rate  
  - • CRP  
  - EKG – prolonged PR interval  
- Supporting - + strep screen, elevated or  
  • ASO titer | - Abx do not modify course of disease  
- Anti-inflammatory drugs suppress the signs & sx |
| Botulism | Subcutaneous nodules – firm, painless, on bony surface & tendons  
- Minor criteria (clinical):  
  - Arthralgia  
  - Fever | Sudden onset of cranial nerve paralysis (CN III, IV, VI, VII, IX)  
- Diplopia, dysarthria, dysphagia, dysphonia (4 Ds)  
- Progressive muscle weakness (floppy baby syndrome)  
- Fixed & dilated pupils in 50%  
- Children: irritability, weakness & hypotonicity | ID toxin in serum or food  
- Removal of toxin from gut – lavage or cathartics  
- Trivalent antitoxin or specific antitoxin (A, B or E)  
- Support |
| --- | --- | --- |
| Chlamydia | Risk Factors:  
- Age <25  
- Hx of prior C. trachomatis infxn  
- New or multiple sex partners  
- Nonwhite race or ethnicity  
- Lower socioeconomic status  
- OCP use (cervical ectopy) | Lymphogranuloma venereum:  
- Initial vesicular or ulcerative lesions – 1st stage  
- Inguinal buboes – 2nd stage – may have fever, malaise, chills, swollen lymph nodes usually in femoral inguinal areas  
- 3rd stage – genitoreal, proctitis, women who have this may get fistulas & drainage  
- Urethritis/cervicitis:  
  - Men – dysuria, urinary frequency, meatal itching, urethral discharge  
  - Women – vaginal discharge, lower abd pain, dyspareunia, cervical ectopy | Nucleic acid amplification of urine or swab specimen  
- Cultures take a long time to grow  
- Doxycycline DOC (azithromycin if pregnant)  
- If not treated in women they can run the risk of becoming infertile |
| Cholera | Caused by *Vibrio cholerae*  
- Acute diarrheal illness leading to profound hypovolemia & death  
- Epidemics: crowding & famine  
- Acquired from contaminated water & food | Stool: liquid & grey in color (rice water)  
- Dark field microscopy  
- Stool culture | Fluids & electrolytes  
- Water & food safety  
- Vaccination if travel to endemic areas – not usually indicated  
- Abx: tetracycline/doxycycline or macrolides may shorten duration of vibrio excretion |
| Diphtheria | D/t *Corynebacterium diphtheriae*  
- Humans are only known reservoir  
- Incubation 1-7 d – spread via respiratory droplets  
- Primarily infects the respiratory tract  
- May develop myocarditis, conduction disturbances, neuro impairment | Sore throat, fever, discrete white exudate (bleeds when removed)  
- Marked cervical adenopathy – bull-neck appearance | Antitoxin – equine diphtheria antitoxin  
- PCN or erythromycin  
- Prevention – immunization (2, 4, 6, 15-18 mos. w/ a preschool booster at age 4-6) |
| Gonorrhea | Venereal disease – *Neisseria gonorrhoea*  
- Purulent, profuse urethral/cervical discharge | Gram stain/culture  
- HIV, RPR | Ceftriaxone/cefixime/cefpodoxime |
<table>
<thead>
<tr>
<th>Disease</th>
<th>Symptoms</th>
<th>Complications</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram – diplococci</td>
<td>Fever, skin lesion, tenosynovitis, monoarticular arthritis (knee, ankle, wrist)</td>
<td>Others: conjunctivitis, pharyngitis, proctitis, endocarditis, meningitis</td>
<td>Treat also for chlamydia (doxycycline/azithromycin)</td>
</tr>
<tr>
<td>Salmonellosis</td>
<td>-Nausea, HA, fever&lt;br&gt;-High-volume diarrhea (pea soup), no blood in stool&lt;br&gt;-Craming, abdominal pain 12-48 h after ingestion&lt;br&gt;-Rash: rose spots (2-3 mm salmon-colored maculopapule on trunk)</td>
<td>-Normal or low WBC count&lt;br&gt;-Stool culture, + for fecal WBCs&lt;br&gt;-Blood cultures: bacteremia rare</td>
<td>Fluids &amp; electrolyte replacement&lt;br&gt;-Abx: Fluoroquinolones &amp; ceftriaxone in sickle cell, immunosuppression, &amp; bacteria&lt;br&gt;-Chloramphenicol useful except for SEs&lt;br&gt;-In other pts will reduce sx by 1-2 d</td>
</tr>
<tr>
<td>Shigelloides</td>
<td>-Fever, malaise, toxic-appearing (+ BP)&lt;br&gt;-LLQ cramping, abdominal pain w/ blood diarrhea&lt;br&gt;-Tenesmus &amp; rectal prolapse</td>
<td>-Leukocytosis &amp; + fecal WBC&lt;br&gt;-Stool culture&lt;br&gt;-Blood cultures often +</td>
<td>Supportive care&lt;br&gt;-Abx – based on sensitivity patterns: Ciprofloxacin – DOC if sensitivity unknown&lt;br&gt;-Trim-sulfa&lt;br&gt;-Ampicillin</td>
</tr>
<tr>
<td>Tetanos</td>
<td>-Stiffness of neck &amp; other muscles (trismus), dysphagia, irritability, hyperreflexia</td>
<td>-Complications: airway obstruction, cardiac failure</td>
<td>Supportive care &amp; wound cleaning&lt;br&gt;-Immunization&lt;br&gt;-Abx: PCN, metronidazole&lt;br&gt;-Benzodiazepines for muscle spasm&lt;br&gt;-Booster every 10 yrs. at mid-decade ages (15, 25, 35, etc.)</td>
</tr>
<tr>
<td>Atypical mycobacterial disease</td>
<td>MAC: fever, weight loss, anorexia, diarrhea&lt;br&gt;-MAC: blood cultures, bone marrow, DNA probes</td>
<td>MAC: multiple drugs (rifabutin, azithromycin, clarithromycin, ethambutol)&lt;br&gt;-Leprosy: months or years (dapsone, rifampin, clofazimine)</td>
<td>MAC: fever, weight loss, anorexia, diarrhea&lt;br&gt;-MAC: blood cultures, bone marrow, DNA probes</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>-Cough, fever, chills, night sweats, anorexia, weight loss &amp; fatigue&lt;br&gt;-Hemoptysis</td>
<td>-Positive PPD&lt;br&gt;- ≥ 5 mm: HIV-positive, those who have had contact w/ clinically active TB, organ transplant, immunosuppressed&lt;br&gt;- ≥ 10 mm: recent arrival from high prevalence countries, IVDA, work/resident of high-risk setting, clinical condition that places pt at high risk, children &lt;4 or exposed to adults in high risk setting&lt;br&gt;- ≥ 15 mm: no known risk factors</td>
<td>Latent TB:&lt;br&gt;- INH 2x/week for 9 months&lt;br&gt;- Rifampin QD for 4 months (6 months in children)&lt;br&gt;-Culture positive TB:&lt;br&gt;- Initial: RIPE – 7 days a week x 8 weeks&lt;br&gt;-Continuation:&lt;br&gt;- INH/rifampin – 7 days a week x 18 weeks&lt;br&gt;-Isoniazid – bactericidal, inhibits synthesis of mycolic acid in mycobacterial cell wall</td>
</tr>
</tbody>
</table>
All positive PPDs need CXR & if abnormal, AFB culture & smears

- AFB cultures (gold standard) & smears
- CXR:
  - Abnormalities such as cavitation often seen in apical or posterior segments of upper lobe
  - May have unusual appearance in HIV
  Cannot confirm dx of TB

- Side effects:
  - Hepatitis – monitor AST/ALT
  - Peripheral neuropathy: rare, but minimize w/ pyridoxine (B6)
  - Abdominal pain, n/v
  - • toxicity from phenytoin & theophylline

- Rifampin – bactericidal, inhibits DNA-dependent RNA polymerase
  - Side effects:
  - Hepatitis
  - Induces CYP450 & • metabolism of many drugs (Coumadin, BCP)
  - Orange discoloration of body fluids
  - Pseudomembranous colitis
  - Pyrazinamide – bactericidal, unknown mechanism, works best in acid environment
  - Side effects:
    - Live toxic, hyperuricemia, not used in pregnancy
  - Ethambutol – bacteriostatic, inhibits the enzyme involved in synthesis of cell wall, eliminated by the kidneys
  - Side effects:
    - Optic neuropathy – diminished visual acuity, loss of color vision

---

### Helminth infestations

- **Hookworms:**
  - *Necator americanus* – a nematode
  - Moist tropics & SE U.S.
  - Life cycle:
    - Penetrates skin
    - Migrates to lung; ciliary action brings organism to mouth; swallowed
    - Move to upper bowel; mature & release eggs
  - *Ascaris lumbricoides*:
    - Roundworm – 2-3 cm long, reside in small intestine
    - Oral egg ingestion – contaminated soil

- **Skin penetration:** ground itch
- **Lungs:** dry cough, blood-tinged sputum
- **GI:** anorexia, diarrhea, vague abdominal pain
  - *Ascaris lumbricoides*:
    - **Asx** – fever, cough, GI distention

- **Iron deficiency anemia**
- **Stool + blood**
- **O&P for dx**
  - *Ascaris lumbricoides*:
    - **O&P, eosinophil**

- **Albendazole DOC**
- **Mebendazole**
- *Ascaris lumbricoides*:
  - Albendazole or mebendazole

### Tapeworms:

- **Albendazole**
<table>
<thead>
<tr>
<th><strong>Disease</strong></th>
<th><strong>Taenia solium</strong></th>
<th><strong>Taenia saginata</strong></th>
<th><strong>Diphyllobothrium latum</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
<td>Mexico, S. &amp; C. America, Africa, SE Asia, India</td>
<td>Worldwide</td>
<td>Europe, Canada, Alaska &amp; Japan</td>
</tr>
<tr>
<td><strong>Intermediate host</strong></td>
<td>Pig</td>
<td>Cow</td>
<td>Freshwater fish</td>
</tr>
<tr>
<td><strong>Signs/symptoms</strong></td>
<td>Asymptomatic</td>
<td>Asymptomatic</td>
<td>Bloating, abdominal pain, diarrhea</td>
</tr>
<tr>
<td><strong>Labs</strong></td>
<td>Eosinophilia</td>
<td>Eosinophilia</td>
<td>Eosinophilia &amp; vitamin B12 deficiency</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Stool O&amp;P</td>
<td>Stool O&amp;P</td>
<td>Stool O&amp;P</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Praziquantel or niclosamide</td>
<td>Praziquantel or niclosamide</td>
<td>Praziquantel or niclosamide</td>
</tr>
<tr>
<td><strong>Prevention</strong></td>
<td>Adequate cooking</td>
<td>Adequate cooking</td>
<td>Adequate cooking</td>
</tr>
</tbody>
</table>

**Malaria**
- Hx of travel to endemic area
- Transmitted by anopheles mosquito
- Complications:
  - Hemolytic anemia
  - Cerebral malaria – *P. falciparum*
  - Blackwater fever – *P. falciparum*
- Episodes of fever, chills & sweating
  - HA, myalgias, splenomegaly, anemia, & leukopenia
  - *Plasmodium*:
    - Vivax: fever every 48 h
    - Malaria: every 72 h
    - Ovale: every 48 h
    - Falciparum: continuous
- Parasite in the blood → thin & thick smear
- Varies w/ *Plasmodium* species
- Chloroquine resistance options:
  - Atovaquone
  - Artemether, lumefantrine
  - Quinine sulfate + tetracycline or clindamycin
  - Mefloquine
- Chloroquine-sensitive options:
  - Chloroquine
  - Hydroxychloroquine

**Pinworms**
- *Enterobius vermicularis*
- Humans are the only host; found worldwide
- Life cycle:
  - Adult worms inhabit the ceum
  - Females migrate to anus to lay eggs
  - Patient then auto-infects
  - Eggs hatch in duodenum, larva migrate to cecum
- Perianal pruritus (night)
  - Restless sleep
- No eosinophilia
- Scotch tape test
- Patient + family
- Mebendazole single dose – repeat in 2 wks.

**Toxoplasmosis**
- Caused by *Toxoplasma gondii* (a protozoan)
- Cats are the host
- Infxn results from ingestion of cysts in raw or undercooked meat (pork/lamb) or cat feces (cat litter)
- Congenital transmission can lead to infxn in the fetus
- Asx in immunocompetent patients
  - Immunocompromised – d/t reactivation of latent disease → fever, malaise, HA, cervical lymphadenopathy, myalgia, arthralgia, stiff neck, sore throat
- Detection of organism in body fluids
  - Serology: IgM antibodies
  - CT: ring-enhanced lesions w/ contrast
- Pyrimethamine + folinic acid + sulfadiazine or clindamycin
- Proper cooking
- Avoid cat litter

**Lyme disease**
- Most common vertor-borne infxn in the US – *Ixodid tick*
- Agent: *Borrelia burgdorferi* – fastidious, microaerophilic spirochete
- Location – Massachusetts to Maryland
- Animal hosts – white-footed mouse, white tail deer
- 3 stages:
  - Stage 1 – 3-30 d after tick bite, flu-like sx, HA, fever/chills, & skin rash (erythema migrans)
  - Stage 2 – wks. to mos. later: Bell’s palsy, AV block, meningitis
  - Stage 3 – mos. to yrs. later: migratory polyarthritis
- ELISA testing
- If minor – doxycycline or amoxicillin
- Rash facial palsy, arthralgias – doxycycline
- Heart block, meningitis, myocarditis – serious manifestations of heart & neurologic, IV ceftriaxone
**Rocky Mountain spotted fever**
- D/t tick bite exposure in endemic area
- Tick from *Dermacentor* family
- *Rickettsia rickettsii*
- Incubation 2-14 d
- Influenzal prodrome followed by chills, fever, severe HA, myalgias
- Classic triad: abrupt onset HA, fever & rash
- Red, macular rash w/ onset btwn days 2-6 of fever
- Rash on hands: petechial or purpuric, then moves to center
- Leukocytosis, proteinuria, hematuria, thrombocytopenia
- Serologic tests – acute & convalescent
- Doxycycline DOC
- Chloramphenicol

**Syphilis**
- Primary:
  - Caused by *Treponema pallidum*
  - Hx of sexual contact
- Secondary:
  - Noted 4-8 wks. after chancre
- Late (tertiary): Infiltrative tumors of skin, bones or liver
- Neurosyphilis:
  - Can be noted at any time during course of disease
- Painless ulcer on genitalia, perianal area, rectum or pharynx
- Chancre resolves in 3-6 wks.
- Enlarged regional lymph nodes
- Generalized maculopapular rash
- Fever, meningitis, hepatitis, arthritis, iritis
- Aortitis, aneurysms, aortic regurgitation, CNS disorders
- Meningitis may present w/ HA, n/v, stiff neck, cranial nerve palsies, hearing loss
- Meningovascular meningitis can lead to hemiparesis, hemiplegia, aphasia, & seizures
- Argyll Robertson pupils: small irregular pupils that react normally to accommodation but not light
- Dark field examination
- RPR (75-100%)
- VDRL (75%)
- FTA-ABS (90%)
- VDRL (99%)
- FTA-ABS (100%)
- +CSF, VDRL
- Benzathine PCN
- Doxycycline/tetracycline in PCN allergic
- Same as primary
- PCN
- Neurosyphilis:
  - PCN

**CMV**
- Member of the Herpes family
- Leading cause of blindness in AIDS pts
- Neonates → hepatosplenomegaly, purpura, CNS changes
- Immune incompetence → mono-like illness w/ fever, myalgias, hepatosplenomegaly, no pharyngitis
- Immunocompromised → AIDS retinitis, pneumonitis, meningoencephalitis, chronic diarrhea
- Leukopenia w/ + lymphps
- IgM antibody is diagnostic
- Cytopathology – “owl eye”
- Cell culture
- Antibody detection
- Supportive care – prophylactic tx in HIV pts
- Ganciclovir – used in children & adults
- (SE = neutropenia)
- Foscarnet – used in adults
- (SE = renal toxicity)
- Cidofovir – 2nd or 3rd line d/t renal toxicity

**EBV**
- Member of Herpes family
- Causes infectious mono
- Transmitted by saliva, incubation period 5-15 d
- Peak age 14-18
- Complications:
  - Splenic rupture
- Fever, pharyngitis, malaise, posterior cervical lymphadenopathy, rash, splenomegaly
- Lymphocytosis w/ atypical lymphps
- Elevated LFTs
- +Heterophile antibody – Monospot
- +EBV specific serology
- Supportive care
- Ampicillin may cause rash

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Time of appearance</th>
<th>Persistence</th>
<th>% of IM patients w/ Ab</th>
</tr>
</thead>
<tbody>
<tr>
<td>VCA-IgM</td>
<td>At presentation</td>
<td>1-2 mos.</td>
<td>100%</td>
</tr>
<tr>
<td>Conditions</td>
<td>VCA-IgG</td>
<td>At presentation</td>
<td>Lifelong</td>
</tr>
<tr>
<td>-----------------</td>
<td>------------</td>
<td>-----------------</td>
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</tr>
<tr>
<td></td>
<td>EBNA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hepatitis</td>
<td></td>
<td>3-6 wks.</td>
<td>Lifelong</td>
</tr>
</tbody>
</table>

- VCA: viral capsid antigen
- EBNA: Epstein Barr nuclear antigen
- VCA-IgM is the best indicator of primary infxn
- Presence of VCA-IgG & EBNA indicates past infxn

**Erythema Infectiosum**
- AKA 5th disease
- Most common under age 10
- D/t parvovirus B19 – spread by respiratory droplets
- Complications → miscarriage, aplastic crisis

**Herpes Simplex**
- Humans’ only natural reservoir
- Transmission by direct contact w/ infected secretions
- Recurrences precipitated by stress, trauma & sun
- Type 1:
  - Any mucosal surface
  - Painful & lasts 5-10 d
  - May become latent in sensory nerve root ganglion
  - Herpes whitlow: HSV of finger or nail region
- Type 2:
  - Genital herpes
  - Incubation period 5 d after sexual contact
  - Painful, multiple lesions
  - May have systemic sx such as fever & myalgias
  - Paresthesias may be noted 12-24 h prior to lesions

- Malaise, HA & pruritus
- Little or no fever
- Rash: **fiery red “slapped cheek,”** circumoral pallor & a subsequent lacy, maculopapular truncal rash (no rash on palms or soles)

- Recurrent grouped small vesicles on an erythematous base
- Perioral or perigenital
- Primary infxn → fever, regional lymphadenopathy, aseptic meningitis
- Primary infxn → proctitis, esophagitis, keratitis
- Tzanck smear
- Direct fluorescent antibody
- Culture

- Supportive
- NSAIDs

- Acyclovir – IV for encephalitis
- Famciclovir
- Valacyclovir
HIV

- Risk factors: sex, IVDA, transfusions
- Retrovirus – changes viral RNA to viral DNA w/ the aid of reverse transcriptase
- Opportunistic infxn (CD4 count)
  - 700-1500: normal
  - >500: lymphadenopathy
  - 200-500: TB, thrush, zoster, lymphoma, Kaposi
  - 100-200: PCP, histoplasmosis
  - 50-100: toxoplasmosis, Cryptococcus
  - <50: MAC, PML, CMV retinitis

- Acute: viral-like illness
- Rapid test
  - ELISA (screening test)
  - Western blot (confirming test)
- HIV RNA viral load

<table>
<thead>
<tr>
<th>HIV RNA</th>
<th>ELISA</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neg.</td>
<td>Neg.</td>
<td>Infxn not confirmed</td>
</tr>
<tr>
<td>Pos.</td>
<td>Neg.</td>
<td>Acute infxn</td>
</tr>
<tr>
<td>Pos.</td>
<td>Pos.</td>
<td>Chronic infxn</td>
</tr>
</tbody>
</table>

Prophylaxis for opportunistic infxn:

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Primary prophylaxis</th>
<th>Secondary prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumocystis</td>
<td>TMP/SMX</td>
<td>Dapsone</td>
</tr>
<tr>
<td>Toxoplasmosis</td>
<td>TMP/SMX</td>
<td>Pyrimethamine + sulfadiazine + folic acid</td>
</tr>
<tr>
<td>TB (INH-sensitive)</td>
<td>INH</td>
<td>Rifampin</td>
</tr>
<tr>
<td>TB (INH-resistant)</td>
<td>Rifampin + pyrazinamide</td>
<td>Rifabutin</td>
</tr>
<tr>
<td>MAC</td>
<td>Azithromycin/clarithromycin</td>
<td>Rifabutin</td>
</tr>
<tr>
<td>CMV retinitis</td>
<td>Valganciclovir</td>
<td>Ganciclovir + foscarnet</td>
</tr>
<tr>
<td>Cryptococcal</td>
<td>Fluconazole</td>
<td>Amphotericin B</td>
</tr>
<tr>
<td>Histoplasmosis</td>
<td>Itraconazole</td>
<td>Amphotericin B</td>
</tr>
</tbody>
</table>

HPV

- Genital warts (condyloma acuminatum)
- More than 30 types of HPV can infect the genital tract
- Spread by direct skin-to-skin contact
- Types 16, 18, 31, 33 & 35 → strongly associated w/ cervical neoplasia (vaccine), associated w/ squamous intraepithelial neoplasia
- Visible genital warts usually HPV types 6 or 11
- May be infected simultaneously w/ multiple types

- Most are axial or subclinical
- Usually present w/ <10 visible genital warts
- Fleshy growths on vulva, vagina, cervix, urethral meatus, perineum & anus

- Biopsy only if:
  - Dx uncertain
  - Lesions do not respond to standard therapy
  - Disease worsens during therapy
  - Pt is immunocompromised

- Guidelines for starting:
  - CD4 <350 or viral load >55,000 by PCR-RNA

Influenza

- RNA virus (Orthomyxoviridae)
- Influenza A & B
  - A = highly infectious, institutional settings
  - B = noted in schools & military
- Spread by respiratory droplets
- Incubation 1-3 d
- Outbreaks every winter
- Community outbreaks

- Abrupt onset fever (101-106)
- Myalgias, HA, nonproductive cough
- Coryza & sore throat
- Exam usually normal

- Leukopenia or normal WBC
- CXR normal
- Viral culture

- Influenza A → amantadine, rimantadine (fewer CNS SEs)
- A or B → oseltamivir, zanamivir – duration of sx if given w/in 48 h
- Abx if secondary bacterial infxn
- Immunization: elderly, respiratory dz, pregnant women, cardiac dz, health care workers, immunosuppressed
- No ASA d/t Reye’s syndrome
<table>
<thead>
<tr>
<th>Virus</th>
<th>Amantadine</th>
<th>Rimantadine</th>
<th>Zanamivir (Relenza)</th>
<th>Oseltamivir (Tamiflu)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virus A</td>
<td>Oral</td>
<td>Oral</td>
<td>Inhalation</td>
<td>Oral</td>
</tr>
<tr>
<td>Virus A &amp; B</td>
<td>≥1 yr.</td>
<td>≥13 yrs.</td>
<td>≥7 yrs.</td>
<td>≥1 yr.</td>
</tr>
<tr>
<td>Virus A &amp; B</td>
<td>≥1 yr.</td>
<td>≥1 yr.</td>
<td>Not licensed</td>
<td>≥13 yrs.</td>
</tr>
<tr>
<td>Administration</td>
<td>Oral</td>
<td>Oral</td>
<td>Inhalation</td>
<td>Oral</td>
</tr>
<tr>
<td>Licensed ages</td>
<td></td>
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</tr>
<tr>
<td>Prophylaxis</td>
<td></td>
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</tr>
<tr>
<td>Adverse</td>
<td>CNS, anxiety</td>
<td>CNS, anxiety</td>
<td>Bronchospasm</td>
<td>N/V</td>
</tr>
<tr>
<td>effects</td>
<td></td>
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</tr>
</tbody>
</table>

**Measles**

- Transmitted by infected droplets – very contagious
- Incubation periods 10-12 d
- Complications – secondary infxn otitis media, encephalitis
- Prodrom of fever (40-41 C), coryza, cough, conjunctivitis, photophobia
- Rash:
  - Brick-red, irregular maculopapular rash
  - 2-4 d after prodrome
  - Head to trunk to extremities
  - Fever concurrent w/ rash
- Koplik’s spots – white/blue gray lesions on red base opposite upper molar
- Primary immunization after age 15 mos. 2nd dose at age 4-6
- Isolate for 1 week after rash

**Mumps**

- Spread via respiratory droplets
- Incubation 12-24 d
- Viral culture in saliva
- Antibodies after week 2
- Immunization – vaccine at 12-15 mos. then at age 4-5
- Supportive

**Rabies**

- Rhabdovirus – bullet-shaped
- Transmitted by infected saliva
- Hx of animal bites – bats, bears, skunks, foxes & raccoons, dogs & cats in developing countries
- Paresthesias, hydrophobia, rage
- Convulsion, paralysis, thick saliva, muscle spasms
- Supportive, wash & clean the wound
- Observe the animal
- Post-exposure immunization – rabies immune globulin (RIG) → full dose around wound, do not give if previously immunized
- Vaccine → dose on days 0, 3, 7 & 14 & 28, if previously immunized then on days 0 & 3

**Roseola**

- AKA 6th disease
- Etiology is human herpes virus 6 & 7
- Incubation 10 d
- Typically under age 5 (6-16 mos.)
- Complications: febrile seizures, encephalitis
- High fever (41 C) for 3 d, resolves, then rash appears
- Faintly erythematous, macular, & diffusely disseminated (starts on trunk)
- Supportive
- Fever control

**Rubella**

- Systemic illness, transmitted by infected droplets
- Incubation 14-21 d
- Complications:
  - Thrombocytopenia
  - Postinfectious encephalopathy
  - Congenital rubella – growth retardation, cataracts, deafness
- Cervical, suboccipital, posterior auricular lymphadenopathy 5-10 d before rash
- Fever, malaise, & coryza & then 2-3 d later, maculopapular rash develops
- Forchheimer spots: petechial lesions soft palate
- Active immunization after age 15 mos. 2nd dose at age 4-6
<table>
<thead>
<tr>
<th>PDA, check immune status in pregnant pts</th>
<th>Rash to face, trunk then extremities (rapidly fades, lasts 3 d)</th>
<th>Joint pain common in young women</th>
</tr>
</thead>
</table>

**Varicella-zoster**

- Acute varicella – chicken pox, incubation 10-21 d
- Reactivation – zoster/shingles, dermatomal distribution
- Complications:
  - Bacterial infxn (most common)
  - Pneumonia
  - Encephalitis, Guillan-Barre syndrome
  - Post-herpetic neuralgia
- Acute → fever, malaise, rash *(pruritic, centripetal, popular rash, vesicular & pustular before crusting)*, first lesion is “drop on rose petal,” lesions in all stages at any given time (macules, papules, vesicle, pustules)
- Reactivation → vesicular rash w/ preceding pain

- Supportive: lotions, antihistamines
- Antiviral: acyclovir, valacyclovir, famciclovir
- Immune globulin: pregnancy, immunosuppressed
- ACIP recommends routine vaccination of all persons age ≥60 yrs. w/ 1 dose of zoster vaccine
### General Characteristics

**Dermatitis**
- Atopic eczema:
  - Probable immune dysregulation
  - Positive family hx
  - Hx of other allergies/asthma
  - Contact dermatitis:
    - Irritant 80%, allergic 20%
  - Perioral dermatitis:
    - Eczema or acne-like perioral rash — often resembles rosacea & pt may have both conditions
    - Women > men; ages 16-45
    - Cause unknown — fluorinated-corticosteroid use on face, fluorinated toothpaste, mint/cinnamon products
  - Stasis dermatitis:
    - Eczema-like eruption on lower legs, secondary to PVD
    - Mechanism — venous incompetence > hydrostatic pressure & capillary damage > leaking w/ extravasation of RBCs into skin
    - Seborrheic dermatitis:
      - Oversecretion of sebaceous material & hypersensitivity to Pityrosporum ovale

**Dyshidrosis**
- Hands/feet
- Difficulty to manage
- Associated w/ stress/metal
- Differentials:
  - Tinea
  - Pustular psoriasis, contact dermatitis

**Lichen simplex chronicus**
- Itch-scratch-itch cycle
- Solitary patch usually >1cm
- Scale +/-

**Drug eruptions**
- Always consider for any generalized rash
- Occur w/in 1-4 weeks
- Differentials: viral exanthema, contact dermatitis
- Common drugs: allopurinol, beta-lactam abx, sulfa meds, anti-

### Symptoms

**Dermatitis**
- Atopic eczema:
  - Red, scaling patchy rash w/ lichenification, very itchy
- Nummular eczema:
  - No central clearing, sharply defined
  - Coin-shaped plaques, very pruritic
  - Dorsal hand, feet & extensor surfaces
  - Contact dermatitis:
    - Papules/vesicles — acute
    - Scaly, lichenified — chronic
  - Perioral dermatitis:
    - Irregularly grouped, discrete red papulopustules on a red base
  - Perioral but spares the vermillion border
  - Seborrheic dermatitis:
    - Moist papules, transparent to pink-red patches that are macerated
    - Dry (dandruff) to inflammatory, scaly plaques
    - Distribution — scalp, ears, face (nasolabial folds), chest, groin, “greasy” scale: cradle cap

**Dyshidrosis**
- Tense pruritic vesicles, especially lateral digits
- Cracking/fissuring, erythema, scale

**Lichen simplex chronicus**
- Solitary patch usually >1cm
- Scale +/-

**Drug eruptions**
- Usually morbilliform, urticarial, sudden, symmetric & “bright red”
- May present w/ fever

### Diagnostics

**Dermatitis**
- Eczematous: Clinical, KOH to rule out fungal, family hx
- Contact dermatitis: clinical based on hx, exposure to metals, etc. distribution & pattern
- Stasis dermatitis: clinical (skin changes, varicosities often seen, swelling w/ pitting edema)

**Dyshidrosis**
- Superpotent corticosteroids
- Tar soaks
- Oral erythromycin
- Derm referral

**Lichen simplex chronicus**
- Avoid itching
- Steroids

**Drug eruptions**
- Drug hx crucial

### Treatment

**Dermatitis**
- Eczematous:
  - Moisturize w/ bland emollients & mild cleansers
  - Topical corticosteroids & antipruritics
  - Sedating antihistamines
  - Keys to effective tx: pt education, follow-up, itch control
- Contact:
  - Avoidance, bland emollients, topical or systemic corticosteroids, sedating antihistamines
- Stasis dermatitis:
  - Support stockings, elevation, ankle/calf exercises, weight loss, topical corticosteroids/bland emollients
  - Seborrheic dermatitis:
    - OTC dandruff shampoo, OTC/Rx ketoconazole shampoo, corticosteroid shampoo, ketoconazole cream, hydrocortisone cream (flares)

**Dyshidrosis**
- Superpotent corticosteroids
- Tar soaks
- Oral erythromycin
- Derm referral

**Lichen simplex chronicus**
- Avoid itching
- Steroids

**Drug eruptions**
- Discontinue medication
- Oral antihistamines
- Antipruritics
- Topical or oral corticosteroids
<table>
<thead>
<tr>
<th>Condition</th>
<th>Etiology</th>
<th>Lesions/Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lichen planus</strong></td>
<td>Etiology unknown</td>
<td>-5 Ps: purple, planar, polygonal, pruritic, papules -2-10 mm lesions, flat-topped, violaceous, papule w/ irregular angulated border</td>
<td>Punch biopsy - Corticosteroids (topical or oral) are mainstay of treatment</td>
</tr>
<tr>
<td><strong>Pityriasis rosea</strong></td>
<td>Etiology unknown – can mimic secondary syphilis (do an RPR in sexually active patient)</td>
<td>-“Herald patch” precedes breakout of smaller lesions (Christmas tree pattern) – typical lesion is round or oval following skin lines w/ light erythema; salmon-colored</td>
<td>Topical mid-potency corticosteroids &amp;/or antipruritics (if needed) - UVB (may hasten resolution)</td>
</tr>
<tr>
<td><strong>Psoriasis</strong></td>
<td>Etiology unknown: • T-cell mediated disease • Genetic susceptibility - Various types: • Palmar pustular psoriasis • Guttate psoriasis post strep infxn • Inverse psoriasis • Erythrodermic psoriasis</td>
<td>-Primary lesion is the erythematous plaque or papule w/ development of silvery, plate-like scale on extensor surfaces</td>
<td>Topical: corticosteroids, retinoids, calcipotriene, tars, salicylic acid, bland emollients, shampoo for scalp psoriasis - Systemic: UV light, methotrexate - Biologicals – alefacept, etanercept, infliximab - Avoid systemic corticosteroids - If extensive → refer</td>
</tr>
<tr>
<td><strong>Erythema multiforme</strong></td>
<td>Immunologic rxn in skin to antigen stimulus • Multiple agents implicated • Etiologies: meds (NSAIDs, sulfa, quinolones, ASA, allopurinol, colchicine), HSV or mycoplasma</td>
<td>-Lesions: plaques, blisters, “targetoid” on extensor surfaces of arms, mucous membrane involvement in severe form (SJS)</td>
<td>Treat infectious cause - Discontinue responsible med - Antihistamines</td>
</tr>
<tr>
<td><strong>Stevens-Johnson syndrome</strong></td>
<td>Severe form of erythema multiforme - Often starts w/ targetoid lesions - Mucocutaneous drug-induced or idiopathic rxn - &lt;10% epidermal detachment</td>
<td>-Painful, tender oral &amp; skin lesions</td>
<td>Systemic steroids: value controversial, supportive care (hydration, prevent secondary infxn, pain relief) - Viscous lidocaine for oral lesions - Mortality rates 0-15%</td>
</tr>
<tr>
<td><strong>Toxic epidermal necrolysis</strong></td>
<td>Severe form of SJS - &gt;30% epidermal detachment - Mortality rate 30% due to sepsis, GI hemorrhage or fluid/electrolyte imbalance - Early dx &amp; discontinuation of meds is crucial - Reexposure to causative agent results in a more rapid &amp; severe rxn than initial episode → medical alert bracelet</td>
<td>-Usually high fever</td>
<td></td>
</tr>
<tr>
<td><strong>Bullous pemphigoid</strong></td>
<td>Autoimmune attack on basement membrane causing subepidermal blistering</td>
<td>-Mild redness, itching or irritation</td>
<td>Punch biopsy - Topical if oral lesions or oral corticosteroids - Methotrexate</td>
</tr>
</tbody>
</table>
### Acne vulgaris
- One of the most common skin disorders
- Common between age 10-15
- More common in women but more severe in men
- Lasts 5-10 yrs.
- Mechanism:
  - Increased sebum production
  - Growth of sebaceous glands
  - Increased Propionibacterium acnes
- Inflammatory:
  - Papules, pustules & nodules/cysts
- Non-inflammatory:
  - Consists of open (blackheads) & closed (whiteheads) comedones

<table>
<thead>
<tr>
<th>Severity</th>
<th>Papules/pustules</th>
<th>Nodules</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Few to several</td>
<td>None</td>
</tr>
<tr>
<td>Moderate</td>
<td>Several to many</td>
<td>Few to several</td>
</tr>
<tr>
<td>Severe</td>
<td>Many &amp;/or extensive</td>
<td>Many</td>
</tr>
</tbody>
</table>

- Comedones: benzoyl peroxide, retinoids (Tretinoin)
- Papules/pustules: above plus abx
- Nodules/cysts: isotretinoin (Accutane) → teratogenic, elevated lipids; steroids (triamcinolone or prednisone)
- Hormonal therapy: BCP, spironolactone

### Rosacea
- "Adult acne"
- Can progress to rhinophyma
- No comedones, blushing hx
- Erythematous/telangiectases

- Avoid triggers: hot beverages, weather changes, spicy food
- Topical abx: metronidazole, sulfacetamide, clindamycin

### Actinic keratosis
- Noted on chronic sun-exposed areas
- Rough, "sandpaper" patch of scaly papules to hypertrophic crusted lesions; pigmented variety

- If lesion is thick or recurrent, shave biopsy to rule out SCC
- Liquid nitrogen
- Topical 5-FU
- Regular follow-up
- Sun protection & avoidance

### Seborrheic keratosis
- Benign; epidermal neoplasm
- Usually noted after age 30
- 0.2-0.3 cm in diameter, flat or raised, smooth or velvety
- "Stuck-on" appearance

- If lesion is thick or recurrent, shave biopsy to rule out SCC
- Liquid nitrogen, curettage, shave removal

### Lice
- Head, body & pubic lice:
  - Close personal contact, contact w/ personal articles
  - Onligate human parasite; cannot survive longer than 10 days (adult) to 3 weeks (eggs) w/o host
  - Life cycle from egg to egg approximately 1 month; eggs incubate & hatch 8-10 days, thus need to retreat

- Blue-gray macules (pubic)
- Nit cemented to hair shaft, unlike hair casts
- "Crawling" sensation

- Clinical: requires close, not cursory inspection of individual hairs
- Microscopic exam
- Permethrin
- Lindane: neurotoxic
- Pyrethrins
- Malathion: flammable
- Bactrim BID x3d repeat in 7-10d

### Scabies
- Due to Sarcoptes scabei
- Consider w/ any generalized itchy rash
- Worse in immunocompromised

- Web spaces, flexor surfaces or wrist, nipples, anterior axillary, genitals
- Itchy papules/nodules on scrotum are scabies until proven otherwise
- Infants – palms & soles

- Clinical
- Scabies prep: scrape multiple lesions, especially "burrows"
- Permethrin 5%
- Ivermectin – resistant cases
- 6-10% sulfur in petrolatum (infants, pregnancy)
- Treat close contacts, launder bedding, clothes, etc.
- Avoid lindane d/t toxicity (seizures, HA)

### Spider bites
- Black widow:

- Black widow:
| Basal cell carcinoma | - Most common skin cancer 
- Common after age 40 
- Risk factors: **UV exposure**, ionizing radiation | - Common on face, scalp, ears & neck 
- **Pearly-white, translucent, rolled border** 
- Can ulcerate & bleed 
- Variety of colors | - Shave or punch biopsy | - Surgical removal 
- 5-FU 
- Follow-up: frequent clinician skin exam, self skin exam every month, sun protection/avoidance |
|----------------------|-----------------------------------------------------|---------------------------------------------------------------------------------|-------------------------------------------------|------------------------------------------------|
| Kaposi sarcoma | - Tumor caused by human herpesvirus 8 
- Transmitted through saliva 
- AIDS defining illness | - Skin lesions that are nodules/papules that are red, purple, brown or black 
- May also be found in the mouth, GI tract, & respiratory tract | - Biopsy: presence of spindle cells 
- Physical exam | - Not curable but lesions will shrink w/ antiretroviral therapy 
- Alpha interferon |
| Melanoma | - Rates increasing faster than any other cancer 
- Most common cancer in women ages 25-30 
- **Know ABCDs but remember E (evolution)** 
- Increased risk: 
  - Hx severe blistering sunburns 
  - Intermittent intense sun exposure 
  - Fair skin 
  - Family hx 
  - Dysplastic nevus syndrome | - Itching, tenderness 
- Bleeding, ulceration | - Patient hx 
- Biopsy: excisional or punch | - Excision w/ adequate margins, interferon reduces recurrence 
- Follow-up: depends on size & stage of lesion |
| Squamous cell carcinoma | - Second most common skin cancer 
- **Chronic sun exposure**, arsenic, radiation, HPV | - Most common on lips, hands, neck, & head 
- Red, scaly papule or plaque 
- Nodular (lips) 
- Hyperkeratotic | - Clinical 
- Skin biopsy to confirm before tx | - Wide local excision – MOHS if very large or in cosmetically sensitive areas 
- Radiation therapy 
- Follow-up: frequent clinical skin exam, sun protection/avoidance |
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
</table>
| Alopecia           | - Alopecia areata: 
  - Autoimmune attack on hair matrix is probable
  - Acute onset
  - Alopecia totalis & universalis – course variable; relapses
  - Telogen effluvium – loss of hair in response to stress
- Androgenetic alopecia:
  - Typically starts after puberty & ends by age 40
  - Patterns in men: frontotemporal (does not respond to topical or finasterid), crown (does not respond to tx, response variables); wigs, hair transplants |
| Onychomycosis      | - Fungal infxn of nail plate
- Distal plate is yellow or white
- KOH prep |
| Paronychia         | - Acute:
  - Usual cause is trauma or manipulation of cuticle
  - Usually S. aureus
- Chronic:
  - Usually Candida, but multiple organisms possible |
| Condyloma acuminatum | - Common, flat, plantar, digitate, genital, or anal
- Etiology: HPV
- Pale white to pink, rough, barely raised papules
- Smooth, velvety & moist |
| Exanthems          | - Diffuse skin eruption associated w/ bacterial or viral illness
- Different forms: erythoderma (scarlatiniform), maculopaupules (morbilliform, measles-like), or vesicles that may evolve to pustules
- Viral: (most common) rubella, rubeola, parvovirus B19, adenoviruses, cytomegalovirus, Epstein-Barr virus, herpes simplex virus 6 (exanthem subitum) & 7 (roseola infantum), enteroviruses, HIV, Colorado tick fever, & many others
- Bacterial: Group A streptococcus (scarlet fever), staphylococcus (toxic | - Scarlet fever:
  - Maculopapular erythematous blanching eruption on the neck, axillae, & groin that later becomes generalized
  - Skin may feel like fine sandpaper
  - Rash heals w/ desquamation beginning around the nails
- Measles:
  - Coalescing erythematous macules & papules that begins behind the ears & in the hairline area that spreads over the rest of the skin in a few days
- Rubella:
  - Macular or maculopapular rash that spreads in a cephalocaudal pattern
- Clinical diagnosis
- Vaccination against MMR & chickenpox
- Treat underlying cause |
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Treatment/Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock syndrome, leptospirosis, meningococcemia</td>
<td>Rickettsial: Rocky Mountain spotted fever, rickettsial pox, &amp; typhus.</td>
<td>- Erythema infectiosum:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- “Slapped cheeks”</td>
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<td></td>
<td></td>
<td>- Macular or urticarial exanthema 1-4 d after slapped cheek eruption mainly</td>
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<td></td>
<td></td>
<td>over proximal extremities</td>
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<td></td>
<td></td>
<td>- Roseola infantum:</td>
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<tr>
<td></td>
<td></td>
<td>- Rosey pink, nonpruritic macular rash predominantly on the neck &amp; trunk</td>
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<td>- EBV:</td>
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<td>- Bright-red morbilliform eruption 5-9 d after exposure to amoxicillin or</td>
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<td></td>
<td></td>
<td>ampicillin</td>
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<tr>
<td></td>
<td></td>
<td>- Varicella:</td>
</tr>
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<td></td>
<td></td>
<td>- Erythematous pruritic macules which develop into papules &amp; fluid-filled</td>
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<tr>
<td></td>
<td></td>
<td>vesicles “dewdrops on a rose petal”</td>
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<td></td>
<td></td>
<td>- Hand-Foot-Mouth disease:</td>
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<td>- 2-8 mm painful, oval, gray vesicles on the palmar &amp; plantar skin, buccal</td>
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<td></td>
<td></td>
<td>mucosa &amp; tongue after 1-2 d</td>
</tr>
<tr>
<td>Herpes simplex</td>
<td>- Dermatomal pattern:</td>
<td>- Clinical</td>
</tr>
<tr>
<td></td>
<td>- HSV1: &gt;80% above waist</td>
<td>- Confirmed w/ Tzanck smear or viral culture</td>
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<td>- HSV 2: &gt;80% below waist</td>
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<td></td>
<td>- HZV: unilateral</td>
<td></td>
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<tr>
<td>Molluscum contagiosum</td>
<td>- Poxvirus</td>
<td>- May resolve spontaneously</td>
</tr>
<tr>
<td></td>
<td>- Spread via skin-to-skin</td>
<td>- Cryosurgery, curettage</td>
</tr>
<tr>
<td></td>
<td>- May itch &amp; vary in number – in number w/ immunocompromised pts</td>
<td></td>
</tr>
<tr>
<td>Varicella-zoster</td>
<td>- Acute varicella – chicken pox, incubation 10-21 d</td>
<td>- Supportive: lotions, antihistamines</td>
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<td></td>
<td>- Reactivation – zoster/shingles, dermatomal distribution</td>
<td>- Antiviral: acyclovir, valacyclovir, famciclovir</td>
</tr>
<tr>
<td></td>
<td>- Complications:</td>
<td>- Immune globulin: pregnancy, immunosuppressed</td>
</tr>
<tr>
<td></td>
<td>- Bacterial infxn (most common)</td>
<td>- ACIP recommends routine vaccination of all persons age &gt;60 yrs. w/ 1 dose</td>
</tr>
<tr>
<td></td>
<td>- Pneumonia</td>
<td>of zoster vaccine</td>
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<td></td>
<td>- Encephalitis, Guillan-Barre syndrome</td>
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<td>- Post-herpetic neuralgia</td>
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</tr>
<tr>
<td></td>
<td>- Acute fever, malaise, rash (pruritic, centripetal, popular rash, vesicular &amp;</td>
<td></td>
</tr>
<tr>
<td></td>
<td>putular before crusting, first lesion is “drop on rose petal,” lesions in</td>
<td></td>
</tr>
<tr>
<td></td>
<td>all stages at any given time (macules, papules, vesicle, pustules)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Reactivation → vesicular rash w/ preceding pain</td>
<td></td>
</tr>
<tr>
<td>Verrucae</td>
<td>- Usually asx</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Verrucous papules anywhere on the skin or mucous membranes usually no</td>
<td></td>
</tr>
<tr>
<td></td>
<td>longer than 1 cm in diameter</td>
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<tr>
<td></td>
<td>- “Recurrences” are frequent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Tenderness on pressure w/ plantar warts</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Periungual – dry, fissured &amp; hyperkeratotic &amp; may resemble hangnails</td>
<td></td>
</tr>
<tr>
<td>Cellulitis</td>
<td>- Deep dermis &amp; subcutaneous tissue infxn</td>
<td>- Oral abx w/ staph or strep coverage</td>
</tr>
<tr>
<td></td>
<td>- Fever, chills, malaise, pain</td>
<td></td>
</tr>
</tbody>
</table>
| Erysipelas | - **Infxn of the epidermis & dermis**  
- Strep pyogenes infxn (group A beta streo)  
- Facial cellulitis in infants/toddlers consider H. influenzae | - Malaise, fever, chills, nausea, adenopathy  
- Elevated, sharply demarcated borders | - Dicloxacillin, amoxicillin/clavulanate, oxacillin, first-generation cephalosporins |
| --- | --- | --- | --- |
| Impetigo | - Superficial bacterial infxn of epidermis  
- usually S. aureus, rarely group A strep  
- Transmitted via skin-to-skin contact | - Honey-colored crusting  
- Inflammation  
- Sometimes vesicular | - Mupirocin 2% TID x 7-10 days  
- Dicloxacillin, macrolide if PCN allergy |
| Candidiasis | **Risk factors:**  
- Moist areas, abx/corticosteroid use, DM, HIV, poor hygiene  
- ~80% of diaper dermatitis  
- Usually the organism is chronic paronychia | - "Beefy red" w/ satellite papules/pustules  
- KOH prep | - Topical nystatin or azoles  
- Oral fluconazole or ketoconazole (widespread or recalcitrant infxn)  
- Dry environment & repair skin barrier |
| Dermatophyte infections | **Types:**  
- T. capitis: scalp  
- T. faciei: face  
- T. corporis: body (ringworm)  
- T. manuum: palms  
- T. pedis: feet (athlete's foot)  
- T. cruris: groin (jock itch)  
- T. unguium: nails  
- T. versicolor – d/t Malassezia furfur | - Many small, circular, white, scaling papules w/ raised border  
- T. versicolor: **scaly patches & plaques of various color**, "fine scale" mostly truncal  
- Clinical, but KOH or culture to confirm (short hyphae & spores in versicolor)  
- Antifungals: ketoconazole, clotrimazole  
- Oral agents: griseofulvin DOC for capitis (not effective for nail dz)  
- Terbinafine (Lamisil) or itraconazole (Sporanox) for hair & nail infxn  
- Selenium sulfide, itrconazole or fluconazole for T. versicolor | - 12% lactic acid lotion/cream  
- Check blood sugar & insulin levels periodically  
- Weight loss  
- Treat associated disorders |
| Acanthosis nigricans | **Causes:** hereditary, obesity, endocrine (DM), meds (OCPs, nicotinic acid)  
- Sudden onset &/or in non-obese pt suspicious for internal malignancy (lymphoma, adenocarcinoma) | - Localized hyperpigmentation – thick, "velvety" plaques on neck, axillae, inframammary folds, inner thighs/inguinal  
- Clinical | - Third-degree:  
- Skin grafting needed unless burn is small (<1 cm in diameter)  
- Requires debridement & reconstruction of tissues  
- Pre-hospital care:  
- Evaluate for signs of inhalation injury – includes dyspnea, burns on mouth & nose, singed nasal |
| Burns | **First-degree:**  
- Minor epithelial damage of epidermis  
- Most common causes are flash burns & sunburn  
- **Second-degree:**  
- Superficial partial-thickness burn  
- Deep partial-thickness burn | **First-degree:**  
- Redness, tenderness & pain are present, no blistering  
- **Second-degree:**  
- Superficial partial-thickness burn  
- Skin appears pink, moist & soft & thin-walled blisters are present, very tender  
- Deep partial-thickness burn  
- Skin appears red & blanched white w/ thick-walled blisters  
- Rule of nines:  
- Adults BSA  
- 9% head & neck  
- 9% each upper extremity  
- 18% anterior portion of trunk  
- 18% posterior portion of trunk  
- 18% each lower extremity  
- 1% to perineum & genitalia  
- Children BSA | - Third-degree:  
- Skin grafting needed unless burn is small (<1 cm in diameter)  
- Requires debridement & reconstruction of tissues  
- Pre-hospital care:  
- Evaluate for signs of inhalation injury – includes dyspnea, burns on mouth & nose, singed nasal |
- Involves epidermis & extends into the lower dermis layer

- Third-degree:
  - Full-thickness burn that destroys epidermis & dermis
  - Caused by immersion scalds, flame burns, chemical & high-voltage electrical injuries

- Fourth-degree:
  - Full-thickness destruction of skin, subcutaneous tissue, fascia, muscle, bone & other structures
  - Due to prolonged exposure to causes of 3rd-degree burns

- Third-degree:
  - Skin is white or leathery w/ underlying clotted vessels & is numb

- 18% head & neck
- 9% each upper extremity
- 18% anterior portion of trunk
- 18% posterior portion of trunk
- 13% each lower extremity
- 1% to perineum & genitalia

- Hospital care:
  - Fluid resuscitation:
    - Parkland formula:
      - Uses lactated ringers
      - Total volume given is 4 ml/kg/% body surface area burned in first 24h (1/2 is given in first 8h, rest given over next 16h) → % body area burned includes only 2nd/3rd degree burns
  - Pain management
    - Requirement for pain meds is inversely related to depth of burn injury
    - Full thickness are painless
    - Morphine is medication of choice

- Hidradenitis suppurativa
  - Inflammatory disorder of the apocrine glands
  - Women > men 3:1; noted after puberty
  - Predisposing factors: obesity, acne hx, apocrine gland occlusion

- Double open comedones pathognomonic
- Boil-like lesions, scarring

- Lipomas
  - Most common soft tissue tumors
  - Benign & presents in adulthood (40-60) as a small circumscribed mass
  - Relatively spares the head, hands & feet
  - Many different subtypes: adeno-, angiolipoleiom, angio-, etc.
  - May be a genetic condition – familial multiple lipomatosis

- Painless, slowly enlarging mass involving the SC tissue of the trunk, neck or proximal extremities
- Usually <5 cm

- Histology: circumscribed, encapsulated, & composed of mature white adipose tissue
- Usually not necessary unless painful or restricting movement
- Simple excision under local anesthetic

- Epithelial inclusion cysts
  - Common, benign growths of the upper portion of the hair follicle
  - Common in Gardner syndrome
  - Favor the face & trunk & may complicate nodulocystic acne vulgaris

- Firm dermal papule or nodule
- Overlying black comedone or “punctum”
- Expressible foul-smelling cheesy material
- May become red & drain & mimic & abscess

- None if asx
- I&D or intralesional triamcinolone

- Melasma
  - Acquired brown pigmentation of the face & neck
  - "Mask of pregnancy"

- Brown pigmentation of the face & neck
- Discontinue meds or end of pregnancy
- Sun protection/avoidance

- Hidradenitis suppurativa
- Weight loss, avoid friction
- Antibacterial cleansers
- Abx (clindamycin)
- Corticosteroids (flares)
- Surgery

- Lipomas
- None if asx
- I&D or intralesional triamcinolone

- Epithelial inclusion cysts
- None if asx
- I&D or intralesional triamcinolone

- Melasma
- None if asx
- I&D or intralesional triamcinolone
| Pilonidal disease | -Acute or chronic recurring abscess or chronic draining sinus over the sacrococcygeal or perianal region  
-Incidence highest in white males btwn 15-40, peak btwn 16-20 yrs. (generally heavy hirsute males who perspire profusely)  
-Acquired infxn of natal cleft hair follicles  
| -Pain, tenderness, purulent drainage, inspissated hair, induration  
-Midline pits or abscesses on or off the midline near the coccyx or sacrum  
| -I&D under local anesthesia |

| Pressure ulcer | -Classifications:  
- Stage I – non-blanchable hyperemia  
- Stage II – extension through epidermis  
- Stage III – full thickness loss  
- Stage IV – full thickness wounds w/ extension into muscle, bone or supporting structures  
- Ulcers in which the base is covered by slough (yellow, tan, gray, green or brown) or eschar (tan, brown or black) are considered unstageable  
- Majority develop during hospital stay for acute illness  
- Risk factors: immobility, reduced sensory perception, moisture (urinary or fecal incontinence), poor nutritional status, & friction/shear forces  
-Red skin that worsens over time, the area forms a blister then an open sore  
-Most commonly found on buttocks, elbow, hips, heels, ankles, shoulders, back and back of head  
| -Remove necrotic debris & maintain moist wound bed  
-Pressure-reducing devices improve healing rates  
-Prevention:  
- Specialized support surfaces  
- Patient repositioning  
- Optimizing nutritional status  
- Moisturizing sacral skin |

| Urticaria | -Hypersensitivity rxn mediated by IgE & mast cells  
-Individual lesions last <24h → if >24h, bx is mandatory to rule out vasculitis, connective tissue dz, drug eruption  
-Acute <6 wks  
-Chronic >6 wks, 90% of the time cause not found, leading causes are meds, infxn, foods  
| -Wheals w/ clearly defined edges and central blanching  
-Itching  
| -H1 blockers (start w/ non-sedating) add another as needed  
-H2 blockers  
-TCAs (doxepin) |

| Vitiligo | -Autoimmune attack on melanocytes  
-Most prominent on face, hands and wrists  
| -Depigmentation of patches of skin  
-Wood’s lamp will accentuate hypopigmentation (if none then  
| -Topical corticosteroids  
-Phototherapy |
<p>| Best chance for repigmentation is in hair-bearing areas | probably pityriasis alba or post-inflammatory hypopigmentation |</p>
<table>
<thead>
<tr>
<th>General Characteristics</th>
<th>Symptoms</th>
<th>Diagnostics</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| **Generalized anxiety disorder** | - Lifetime prevalence 5%  
- Women > men 3:2  
- Decreases w/ advancing age  
- Impairs daily living | - Primary sx: anxiety, fatigue, motor tension, autonomic hyperactivity  
- Cognitive vigilance – irritability, easy startle response  
- Motor tension usually manifests as shakiness, restlessness, & HA  
- Worry: “chain of thought & images, negative affect, laden & relatively uncontrollable” | - DSM-IV criteria:  
• The excessive anxiety or worry occurs in majority of days in 6-month periods (about a number of events or activities)  
• Patient finds it difficult to control the worry  
• The worry is associated w/ 3 or more of the following:  
  - Restlessness  
  - Fatigue  
  - Difficulty concentrating  
  - Irritability  
  - Muscle tension  
  - Sleep disturbance  
• Focus of the worry is not confined to another axis I d/o  
• Anxiety/worry or clinical sx causes significant impairment  
• Condition is not d/t a substance or medical condition | - SSRIs – paroxetine & escitalopram  
- SNRIs – venlafaxine (long-term)  
- TCAs – imipramine  
- Buspirone  
- Benzodiazepines  
- Beta-blockers  
- Psychotherapy |
| **Panic disorder** | - Recurrent, severe panic attacks  
- Epidemiology:  
  • Prevalence 3-8% of general population  
  • F > M 2:1  
  • Onset from late adolescence to early adulthood  
  • Strong evidence of familial trait (4-7 fold risk in first degree relative)  
  • Most initial attacks occur w/o environmental trigger | - Criteria:  
• Sx not d/t a substance or medical condition  
• Sx not better explained by another mental d/o  
• Recurrent, unexpected panic attacks (at least 2) – not related to persistent situational trigger as in phobias or PTSD  
• At least one of the panic attacks has been followed by 1 month of ≥1 of the following:  
  - Concern about more attacks  
  - Worry about implications of the attacks  
  - Significant change in behavior related to the attacks  
• Presence or absence of agoraphobia (anxiety about being in a certain place or situation & avoidance of these situations) | - SSRIS – first line DOC: paroxetine, sertraline, fluoxetine  
- Benzodiazepines (watch for abuse)  
- TCAs – imipramine  
- Cognitive-behavioral therapy, relaxation, desensitization |
| **Phobias** | - Social phobia: | - Social phobia DSM-IV criteria: | - Social phobia: |
- Marked or persistent fear of social or performance situations in which person is exposed to scrutiny by others
  - Person fears that he/she will act in a way which will be embarrassing
  - Exposure to social situation provokes anxiety & may cause panic attack
  - Person recognizes the fear is unreasonable
  - Social situations are either avoided or endured w/ intense anxiety or distress

-Specific phobias:
  - Anxiety d/o characterized by intense fear of a particular object or situation
  - Women > men; typical onset in childhood

-If panic attacks are experience they are expected
  - Estimated 1/3 of pts will also have a depressive d/o
  - Pts able to describe how they avoid contact w/ phobic situation

-Social phobia:
  - Must experience a marked, persistent fear that is recognized by the pt to be excessive or unreasonable
  - The avoidance interferes significantly w/ person's routine
  - Exposure to the social situation provokes anxiety and may cause panic attack
  - Person recognizes the fear is unreasonable
  - If a general medical condition or another mental d/o is present, the fear is unrelated

-PTSD
  - Prevalence 8-9% of population
  - <10% of those exposed to a traumatic event will develop PTSD
  - 30% of those diagnosed will develop chronic disease
  - Recovery most pronounced in the first year following the trauma
  - Most common group is young adults:
    - Trauma for men is often combat experience; can be life threat by violence in other (non-military) high-risk groups (urban violence)
    - Trauma for women is usually assault or rape
    - Adult survivors of child sexual abuse
    - Person has been exposed to traumatic event in which there is:
      - Actual or threatened death or serious injury/violation to self or others
        &
      - The response may involve helplessness, dissociative sx, autonomic arousal, avoidance of associated stimuli, & emotional numbing

-Frequently associated w/ feelings of guilt, rejection & humiliation
  - May cause memory, attention impairments
  - Aggression/violence
  - Poor impulse control
  - Comorbid disorders: depression, panic, substance-related disorders

-DSM-IV criteria:
  - The trauma is persistently re-experienced (>1 month) as 1 or more of the following:
    - Recollections
    - Distressing dreams
    - Acting/feeling as if event were recurring
    - Physiological distress when exposed to cure that symbolize the trauma
  &
  - Avoidance of stimuli:
    - Thoughts, feelings or conversations
    - Activities, places or people
    - Memory lapse about certain aspects of event
      - Interest in activities
      - Feelings of detachment
      - Restricted range of affect
      - Sense of foreshortened future
  - Sx of • arousal (2 or more of the following):
    - Difficulty falling or staying asleep
    - Irritability, hypervigilance

-Refer to psychiatry
  - Meds – SSRIs (paroxetine, sertraline (DOC)); TCAs, MAOIs
  - Cognitive-behavioral therapy – psychotherapy or counseling
  - Combo of meds/treatment is more effective than either one alone
  - • risk of suicide

-Beta-blockers – propranolol and atenolol (good for performance anxiety, not 1st-line)
-SSRIs – paroxetine
-MAOI – SEs are a problem
-Benzodiazepines
-Specific phobia:
  - Childhood phobias may remit spontaneously w/ age
  - Rarely cause disability
  - Treatment of choice is exposure therapy
  - Medications not indicated
### ADD/ADHD
- Duration longer than 6 mos.
- Onset before age 7
- M > F 4:1
- Distractibility, short attention span (sometimes w/ hyper-focus), hyperactivity, impulsivity → present in more than one setting
- Duration of disturbance (dreams, physical disturbance) > 1 mo.
- Behavior modification
- Stimulant drugs:
  - Methylphenidate (Ritalin)
  - D-amphetamine (Adderall)
  - Pemoline (Cylert)
  - Strattera (Atomoxetine)
- Referral for neuropsychologic testing as age, language skills allow
- Possible referral to psych for meds for behavioral control, mood disorders
- Treatment modalities emphasize behavioral strategies

### Autism
- Pervasive developmental d/o (axis I d/o)
- Presentation usually in context of school dysfunction or behavioral acting out
- Characterized by severe, pervasive impairment of several areas of development:
  - Reciprocal social interaction skills
  - Communication skills
  - Presence of stereotyped behavior, interests, & activities
  - May be associated w/ some mental retardation (coded on axis II)
- Referral for neuropsychologic testing as age, language skills allow
- Possible referral to psych for meds for behavioral control, mood disorders
- Treatment modalities emphasize behavioral strategies

### Anorexia nervosa
- Disturbance in perception of body shape & wt.
- Loses wt.
- Introverted
- Takes pride in wt. control
- Less sexually active
- Feels in control w/ food
- Abuses laxatives
- Amenorrhea if severe
- DSM-IV Criteria:
  - Refusal to maintain body wt. at or above a minimally normal wt. for age & height (eg, wt. loss leading to maintenance of body wt. <85% of that expected, or failure to make expected wt. gain during period of growth, leading to body wt. <85% of that expected
  - Intense fear of gaining wt. or becoming fat, even though underweight.
  - Disturbance in the way in which one's body wt. or shape is experienced, undue influence of body wt. or shape on self-evaluation, or denial of the seriousness of the current low body wt.
  - In postmenarcheal females, amenorrhea, (a woman is considered to have amenorrhea if her periods occur only following hormone, eg, estrogen administration)
- Medical treatment of complications
- Supportive psychotherapy or cognitive behavioral therapy

<table>
<thead>
<tr>
<th>BMI</th>
<th>Weight Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;14</td>
<td>At risk for refeeding syndrome</td>
</tr>
<tr>
<td>&lt;17.5</td>
<td>Anorexic range</td>
</tr>
<tr>
<td>&lt;18.5</td>
<td>Underweight</td>
</tr>
<tr>
<td>18.5-24.9</td>
<td>Normal wt.</td>
</tr>
<tr>
<td>&gt;22</td>
<td>At risk for DM, HTN, CHD</td>
</tr>
<tr>
<td>25-29.9</td>
<td>Overweight</td>
</tr>
<tr>
<td>&gt;30</td>
<td>Obese</td>
</tr>
<tr>
<td>&gt;40</td>
<td>Morbidly obese</td>
</tr>
</tbody>
</table>
### Bulimia nervosa
- Disturbance in perception of body shape & wt.
- Repeated binge eating, followed by behavior to prevent wt. gain (vomiting or laxative abuse)
- Minor wt. changes
- Extroverted
- Has shame
- Sexually active
- Feels out of control w/ food

#### DSM-IV Criteria:
- Recurrent episodes of binge eating characterized by both of the following:
  - Eating, within any 2-h period, an amount of food that is larger than most people would eat during a similar period of time & under similar circumstances
  - Sense of lack of control over eating during the episode
- Recurrent inappropriate compensatory behavior in order to prevent wt. gain, such as self-induced vomiting, misuse of laxatives, diuretics, enemas, or other meds; fasting, or excessive exercise.
- The binge eating & inappropriate compensatory behaviors both occur, on avg., at least 2x/week for 3 mos.
- Self-evaluation is unduly influenced by body shape & wt.
- The disturbance does not occur exclusively during episodes of anorexia nervosa.

#### Treatment
- Same as anorexia
- Pharmacologic therapy (not effective in anorexia) → fluoxetine

### Obesity
- BMI > 30, affects 30% of American adults
- Results in coronary heart disease, 3-4 fold risk for DM, 5-6 fold risk for HTN & 2x risk for hypercholesterolemia
- Also associated w/ degenerative joint disease, cholecystitis, GERD, hear failure, thromboembolic disorders, respiratory impairment (apnea)
- Strong genetic component
- Medications are an important cause (clozapine, onlanzapine, risperidone, amitriptyline, cyproheptadine, valproate, carbamazepine, gabapentin, insulin, & thiazolidinediones

#### Lifestyle modifications (diet & exercise)
- Behavioral therapy & social support
- Medications – amphetamines, benzphetamine, phendimetrazine, phentermine, diethylpropion, mazindol, sibutramine & orlistat → only sibutramine & orlistat are approved by the FDA for long-term use
- Surgery:
  - Duodenal switch
  - Biliopancreatic diversion
  - Gastric band/bypass (Roux-en-Y bypass is the most common bariatric procedure in the U.S.)

### Adjustment disorder
- Occurs when an individual is unable to adjust or cope w/ a particular stressor or major life event
- May also be known as situational depression but differs because usually
- Emotional sx: sadness, hopelessness, lack of enjoyment, crying spells, nervousness, anxiety, worry, desperation, trouble sleeping, difficulty concentrating, feeling overwhelmed & thoughts of suicide

#### DSM-IV Criteria:
- The development of emotional or behavioral sx in response to an identifiable stressor(s) occurring w/in 3 mos. of onset of the stressor(s)

#### Treatment
- Psychotherapy – goal is to relieve sx & behavior change
- Cognitive behavioral therapy
- Benzodiazepines or antidepressants
<table>
<thead>
<tr>
<th>Bipolar disorder</th>
<th>Depressive disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Has a variety of presentations which must incorporate a current or prior manic, hypomanic, mixed or depressive episode</td>
<td>One or more episodes of major depression</td>
</tr>
<tr>
<td>Manic episode: a period of abnormally &amp; persistently elevated, expansive or irritable mood lasting at least 1 week</td>
<td>5-10% of primary care patient population</td>
</tr>
<tr>
<td>Lithium – for acute manic or hypomanic episode or for the prevention of future episodes of mania &amp; depression</td>
<td>Women &gt; men 2-3:1</td>
</tr>
<tr>
<td>TCAs, SSRIs</td>
<td>1/4 of cancer patients &amp; women (at some point have clinical depression)</td>
</tr>
<tr>
<td>Quetiapine/olanzapine</td>
<td>- prevalence w/ family hx, substance abuse, chronic pain, chronic illness &amp; severe or unanticipated stress</td>
</tr>
<tr>
<td>DSM-IV criteria:</td>
<td>- Depressed mood (core sx)</td>
</tr>
<tr>
<td>Goal is to deal w/ stressors more adaptively</td>
<td>- Diminished interest or pleasure (core sx)</td>
</tr>
<tr>
<td>First-line therapy in mild to moderate depression that is nonpsychotic &amp; not chronic/highly recurrent</td>
<td>- Significant wt. change (&gt;5%)</td>
</tr>
<tr>
<td>Electroconvulsive therapy – indicated if suicidal or worried about drug S/Es</td>
<td>- Insomnia or hypersomnia</td>
</tr>
<tr>
<td>-SSRIs – fluoxetine, paroxetine, citalopram, sertraline, escitalopram</td>
<td>- Psychomotor agitation or retardation</td>
</tr>
<tr>
<td>-Anti-OCD SSRIs – fluvoxamine</td>
<td>- Feelings of worthlessness/guilt</td>
</tr>
<tr>
<td>-Anti –OCD (not SSRI) – clomipramine</td>
<td>- concentration/ indecisiveness</td>
</tr>
<tr>
<td>-TCAs – Elavil, nortriptyline, imipramine</td>
<td>- Recurring thoughts of death or suicide</td>
</tr>
<tr>
<td>-Aminoketones – bupropion</td>
<td>- energy/fatigue</td>
</tr>
<tr>
<td>-Atypical SSRIs – mirtazapine – more sedating at lower doses</td>
<td>- Somatic sx: HA, constipation, skin changes, chest pain, abdominal pain, cough, SOB</td>
</tr>
</tbody>
</table>

- These sx or behaviors are clinically significant as evidenced by either of the following:
  - Marked distress that is in excess of what would be expected from exposure to the stressor
  - Significant impairment in social or occupational (academic) functioning
- The stress-related disturbance does not meet the criteria for another specific Axis I d/o & is not merely an exacerbation of a preexisting Axis I or Axis II d/o
- The sx do not represent bereavement
- Once the stressor has terminated, the sx do not persist for more than 6 mos.
<table>
<thead>
<tr>
<th>Personality disorders</th>
<th>Mild, chronic form of major depression</th>
<th>Change in appetite &amp; sleep, fatigue, concentration, hopelessness</th>
<th>At least 2 yrs. of chronically depressed mood most of the time</th>
<th>Similar to major depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>- 10–15% of the general population</td>
<td>- Organized into 3 clusters</td>
<td>• Cluster A: Social detachment w/ unusual behaviors</td>
<td>• Distrust &amp; suspiciousness</td>
<td>• Psychodynamic psychotherapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Weird symptoms, odd, or eccentric</td>
<td>• Unforgiving of enemy or friend, fear confiding in others, perceive threats; socially isolated; avoid intimacy</td>
<td>• Goal is to help patients recognize how they’re responsible for the turmoil in their lives &amp; learn healthier ways of reacting to people &amp; problems</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cluster B: Drama</td>
<td>• Detached w/ limited emotional expression</td>
<td>• Individual, group, &amp; family therapy can all be helpful</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Wild symptoms, dramatic, emotional, erratic</td>
<td>• Indifferent, little interest in sex, work alone, no close friends</td>
<td>• Cognitive behavior therapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Emotional &amp; impulsive</td>
<td>• Embrace unusual beliefs to a degree that exceeds the norm</td>
<td>• Goal is to actively retrain the way patients think about problems, which in turn improves their emotions &amp; behaviors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cluster C: Anxiety &amp; fearful</td>
<td>• Embrace unusual beliefs to a degree</td>
<td>• Dialectical behavior therapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Whiny, anxious, fearful, dependent</td>
<td>• Highly defended against any challenge of the self-protective, rigid way they approach social interactions</td>
<td>• Goal is to focus on coping skills</td>
</tr>
<tr>
<td>- Paranoid Personality DO (Cluster A)</td>
<td></td>
<td></td>
<td>• Pride themselves as being rational &amp; objective</td>
<td>• Most often used to treat borderline personality d/o</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Differential diagnosis: long-standing psychotic symptoms</td>
<td>• Appear to others as unemotional, affectively restricted, &amp; hypervigilant</td>
<td>• Medications may help alleviate related conditions but they can’t cure underlying d/o</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Illness experience &amp; illness behavior</td>
<td></td>
<td>• Antidepressants: SSRIs such as fluoxetine (Prozac), sertraline (Zoloft), citalopram (Celexa), paroxetine (Paxil), &amp; escitalopram (Lexapro), or the related antidepressant venlafaxine (Effexor)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Highly defended against any challenge of the self-protective, rigid way they approach social interactions</td>
<td>• Anticonvulsants: may help suppress impulsive &amp; aggressive behavior</td>
<td>• Antidepressant venlafaxine (Effexor) or the related antidepressant venlafaxine (Effexor)</td>
</tr>
<tr>
<td>- Schizoid Personality DO (Cluster A)</td>
<td></td>
<td></td>
<td>• Pride themselves as being rational &amp; objective</td>
<td>• Carbamazepine (Tegretol) or valproic acid (Depakote)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Differential diagnosis - may suffer brief psychotic decompensation</td>
<td>• Appear to others as unemotional, affectively restricted, &amp; hypervigilant</td>
<td>• Topiramate (Topamax) may aid in managing impulse-control problems</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Illness experience &amp; illness behavior</td>
<td></td>
<td>• Anticonvulsants: may help suppress impulsive &amp; aggressive behavior</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Strong emotions they can’t deal w/</td>
<td>• Unstable self-image, relationships</td>
<td>• Carbamazepine (Tegretol) or valproic acid (Depakote)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Enjoy solitary pursuits</td>
<td>• Despair, unstable affect, rage, relationship instability, empty feeling</td>
<td>• Topiramate (Topamax) may aid in managing impulse-control problems</td>
</tr>
<tr>
<td>- Schizotypal Personality DO (Cluster A)</td>
<td></td>
<td></td>
<td>• Highly defended against any challenge of the self-protective, rigid way they approach social interactions</td>
<td>• Antipsychotics: patients w/ borderline &amp; schizotypal personality disorders are at risk of losing touch w/ reality</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Differential diagnosis - schizophrenia</td>
<td>• Embrace unusual beliefs to a degree</td>
<td>• Antipsychotic meds such as risperidone (Risperdal) &amp; olanzapine (Zyprexa) for distorted thinking</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Illness experience &amp; illness behavior</td>
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</tr>
</tbody>
</table>
then dx of conduct d/o; overlaps w/ other personality DO traits

- Illness experience & illness behavior
- Excessively self-assured, entitled, hostile
- May appear charming & engaged

- Borderline Personality DO (Cluster B)
- Differential diagnosis - may have brief psychotic episodes
- Illness experience & illness behavior
- Have difficulty distinguishing reality from fantasy
- Substance abuse is a major comorbidity
- Rapid mood swings, impulsivity, chronic boredom

- Histrionic Personality DO (Cluster B)
- Differential diagnosis - deeply affected by perceived frailties
- Illness experience & illness behavior
- Threatens sense of physical attractiveness
- Overly concerned w/ physical appearance
- Speech impressionistic & vague
- May be perceived as vain

- Narcissistic Personality DO (Cluster B)
- Differential diagnosis - difficult to distinguish from need for admiration
- Illness experience & illness behavior
- Threatens their image of well-being
- Blindly ambitious, self-serving
- Hypersensitive to issues of self-esteem

- Dependent Personality DO (Cluster C)
- Differential diagnosis - distinguish from other dependencies
- Illness experience & illness behavior
- Fear that illness leads to helplessness & abandonment
- Clingy, demanding

- Avoidant:
  - Desire relationships but avoid them
  - Have intense feelings of inadequacy, very sensitive to criticism, fear rejection & humiliation

- Obsessive-Compulsive:
  - Perfectionists who require a great deal of order & control
  - Attention to minutiae impairs ability to finish projects; cold & rigid in relationships; make frequent moral judgments

- Other medications
  - Haloperidol (Haldol) for severe behavior problems
  - Anti-anxiety meds such as alprazolam (Xanax) & clonazepam (Klonopin)
  - Mood stabilizers such as lithium (Eskalith, Lithobid) may relieve symptoms associated w/ personality disorders
| Delusional disorders | - Paranoid d/o  
|                     | - Onset later in life, cause is unknown  
|                     | - Different types:  
|                     |   - Erotomantic type: delusions that another person, usually of higher status, is in love with the individual  
|                     |   - Grandiose type: delusions of inflated worth, power, knowledge, identity, or special relationship to a deity or famous person  
|                     |   - Jealous type: delusions that the individual’s sexual partner is unfaithful  
|                     |   - Persecutory type: delusions that the person is being malevolently treated in some way  
|                     |   - Somatic type: delusions that the person has some physical defect or general medical condition  
|                     |   - Mixed type: delusions characteristic of more than one of the above types but no one theme predominates  
|                     | - Non-bizarre delusions about things that could happen in real life – being followed, poisoned, etc.  
| DSM-IV criteria: | - Nonbizarre delusions (i.e., being followed, poisoned, infected, loved at a distance, or deceived by spouse or lover, or having a disease) of at least 1 mo.  
|                     | - Criterion I for schizophrenia has never been met  
|                     | - Note: Tactile & olfactory hallucinations may be present in delusional d/o if they are related to the delusional theme  
|                     | - Apart from the impact of the delusion(s) or its ramifications, functioning is not markedly impaired & behavior is not obviously odd or bizarre.  
|                     | - If mood episodes have occurred concurrently w/ delusions, their total duration has been brief relative to the duration of the delusional periods.  
|                     | - The disturbance is not d/t the direct physiological effects of a substance or medical condition  
| Treatment: | - Atypical antipsychotics  
|                     | - Psychotherapy & family therapy |
Schizophrenia
- 1% of population
- Most present between ages 15-45
- Men > woman (slight •) but men earlier onset
- Greater than 6 months duration
- Risk factors:
  - Environmental
  - Genetic
    - Prenatal infection & starvation
    - Obstetric complications
    - Born in winter or urban setting
    - Head injury
    - Drug use
    - Family history
    - Environmental
    - Genetic
      - Prenatal infection & starvation
      - Family history
      - Obstetric complications
      - Paternal age
      - Genetic syndromes
      - Born in winter or urban setting
      - Head injury
      - Drug use
    - Specific susceptibility genes
  - Paternal age
  - Born in winter or urban setting

- Paranoid
  - Preoccupied with delusions of persecution or grandeur; usually tense, suspicious, guarded, & reserved
- Disorganized
  - Regressed to primitive, disinhibited, & unorganized behavior; provide inappropriate emotional responses (disorganized speech & inappropriate affect)
- Catatonic
  - Marked disturbance in motor function; stupor, negativism, rigidity, posturing &/or motoric immobility (waxy flexibility or catalepsy)
- Undifferentiated
  - Criterion for schizophrenia but no criteria for paranoid, disorganized, or catatonic subtypes
- Residual
  - Socially withdrawn, eccentric, emotionally blunted, illogical; have mild loosening of associations

- DSM-IV criteria:
  - 6 months of illness w/ 1 month of acute sx
  - Active-phase sx: auditory hallucinations, bizarre delusions OR
  - 2 or more of the following:
    - Delusions
    - Hallucinations
    - Disorganized speech
    - Disorganized or catatonic behavior
  - Negative sx: social withdrawal, lack of emotional expression, communication, & reactivity
  - Must also have functional decline
  - No major depressive, manic or hypomanic episodes have occurred
  - Sx are not d/t substance or medical condition
  - If there is dx of autism, schizophrenia dx is made only if prominent delusions or hallucinations are present for at least 1 mo.

- Psychosocial rehab
- Antipsychotics – DA receptor antagonists:
  - 1st generation – chlorpromazine or haloperidol (SEs = extrapyramidal sx)
  - 2nd generation – clozapine, risperidone, olanzapine (1st-line DOC)
    - Clozapine: possible agranulocytosis (2nd line drug)
    - Risperidone: prolactin
    - Olanzapine: marked wt. gain

<table>
<thead>
<tr>
<th>Hallucinations</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual</td>
<td>Can be simple (flashing light) or complex (face). More common in deliria &amp; dementias</td>
</tr>
<tr>
<td>Auditory</td>
<td>Can be a sound or voice. Voice is often in the 3rd-person talking about the pt (&quot;He is such a loser.&quot;) or can be command hallucinations.</td>
</tr>
<tr>
<td>Olfactory</td>
<td>Often have the character of stench. Also common in temporal lobe epilepsy</td>
</tr>
<tr>
<td>Tactile</td>
<td>Sensation of being touched or insects on skin. Also seen in cocaine &amp; amphetamine use.</td>
</tr>
<tr>
<td>Somatic</td>
<td>Sensation arising from w/in the body (noting movement of the brain). Are common &amp; obvious</td>
</tr>
<tr>
<td>Gustatory</td>
<td>Very rare. Can be part of persecutory delusion (tasting poison in food).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Delusions</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persecutory</td>
<td>A person or force is interfering w/ observing, &amp; wishing to harm the pt</td>
</tr>
<tr>
<td>Reference</td>
<td>Random or innocuous events take on personal significance</td>
</tr>
<tr>
<td>Control</td>
<td>Some agency takes control of a pt’s thoughts, feelings &amp; behaviors</td>
</tr>
<tr>
<td>Somatic</td>
<td>Part of the body is diseased or malfunctioning, or physically altered</td>
</tr>
</tbody>
</table>
| Somatoform disorders | -Somatization disorder:  
  • Chronic w/ significant distress  
  • Women > men 10:1, onset age <30  
  • Multiple physical complaints  
  • Preoccupation w/ medical or surgical therapy – poly-surgery is a feature  
-Conversion disorder:  
  • Loss or change in sensory or motor function suggestive of a physical d/o but caused by psychological factors – sx is not intentionally produced  
  • Extreme psychosocial stress may be the most important precipitating factor  
-Pain disorders:  
  • Formally called somatoform pain d/o  
  • Mainly women, ages 40-50  
-Hypochondriasis:  
  • Preoccupation w/ the fear or belief that one has a serious undiagnosed dz  
  • Men = women, 5% of the population  
  • Ages 20-30  
  • Doctor shop; use of OTCs/herbals  
-Factitious disorders:  
  • Typically women w/ medical background  
  • Munchausen – present in exaggerated or dramatic fashion  
  • Munchausen by proxy – parent creates illness in child to maintain relationship w/ clinician | -Somatization disorder:  
  • 4 different pain sx  
  • 2 GI sx  
  • 1 sexual sx  
  • 1 pseudoneurological sx other than pain  
  • Sx must cause significant role impairment or result in medical attention | -Hypochondriasis – group therapy |
<table>
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</thead>
<tbody>
<tr>
<td>Grandeur</td>
<td>Unrealistic belief in one’s power &amp; abilities, can be obvious or subtle</td>
<td></td>
</tr>
<tr>
<td>Nihilism</td>
<td>Exaggerated belief in the futility of everything</td>
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</tr>
<tr>
<td>Delusion of love</td>
<td>Feel loved by another, often higher status, but is merely an innocent bystander</td>
<td></td>
</tr>
<tr>
<td>Jealousy</td>
<td>Somebody is suspected of being unfaithful; typical for alcoholics</td>
<td></td>
</tr>
<tr>
<td>Delusion of doubles</td>
<td>Believes a family member or close friend has been replaced by an identical double</td>
<td></td>
</tr>
</tbody>
</table>

| Somatoform disorders | -Somatization disorder:  
  • Shortness of breath  
  • Dysmenorrhea  
  • Burning in sex organ  
  • Lump in throat (difficulty swallowing)  
  • Amnesia  
  • Vomiting  
  • Painful extremities | -Somatization disorder (2 or more + = high likelihood of dx):  
  • Paralysis, aphonia, seizures, gait issues, blindness, anesthesia  
  • Commonly have depression, anxiety, schizophrenia, & personality d/o  
-Pain disorders:  
  • Long hx of severe pain not constant w/ anatomic clinical signs – pain is source of all life troubles  
  • Pain exists in 1 or more sites & leads to distress  
-Hypochondriasis:  
  • Absence of a physical d/o, persistence of fear despite reassurance, significant distress or role impairment, & duration at least 6 months  
-Pain disorders:  
  • Self-induced sx or false physical or lab findings  
  • May include self-mutilation, fever, hemorrhage, hypoglycemia, seizures | -Hypochondriasis – group therapy |

<table>
<thead>
<tr>
<th>Mnemonic</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatization</td>
<td>Shortness of breath</td>
</tr>
<tr>
<td>Disorder</td>
<td>Dysmenorrhea</td>
</tr>
<tr>
<td>Besets</td>
<td>Burning in sex organ</td>
</tr>
<tr>
<td>Ladies</td>
<td>Lump in throat (difficulty swallowing)</td>
</tr>
<tr>
<td>&amp;</td>
<td>Amnesia</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Physicians</td>
<td>Painful extremities</td>
</tr>
</tbody>
</table>
### Substance Abuse

- Persons adversely affected by ETOH & substance use >20% of PCP patients
- Substance use disorders:
  - At-risk alcohol use
  - Problem alcohol use
  - Dependent alcohol use
  - Tobacco use
  - Caffeine, steroids, chemicals, adrenaline, ephedrine
  - Illicit drug use
  - Rx drug use

#### Signs of abuse:
- Substance taken in larger amounts over longer periods of time than intended
- Efforts to cut down or stop are unsuccessful
- Large amount of time spent seeking the substance or recovering from its effects
- Disruption of work, social or recreation activities
- Substance use continues despite realization that it impacts health

#### DSM-IV criteria:
- Maladaptive pattern of use of a substance which has abuse potential leading to significant functional impairment in at least 1 of the following areas over a 12 mo. period:
  - Non-fulfillment of important responsibilities
  - Recurrent use in physically dangerous situations
  - Legal entanglements
  - Use in spite of social problems caused

#### CAGE screening for alcohol:
- Have you felt you ought to CUT down on your drinking?
- Have people ANNOYED you by criticizing your drinking?
- Have you felt GUILTY about your drinking?
- Have you ever needed an EYE OPENER in the morning to steady your nerves?
- Positive if 2 or more yes responses

### Substance Dependence

- AKA drug addiction
- Compulsive need to use drugs in order to function normally
- When such substances are unattainable the pt suffers from withdrawal

#### DSM-IV criteria:
- Maladaptive pattern of substance use leading to significant distress or impairment > substance use; must be manifested by at least 3 of the following areas over a 12 mo. period:
  - Signs of tolerance
  - Need for increasing amounts to achieve desired effect
  - Diminished desired effect w/ continued use of the same quantity of the substance

#### There is no one “fit all” tx
- If >45 at least 7 wks. required to partially normalize brain dysfunction

#### Detox:
- Must have hx of withdrawal seizures or delirium tremens, pregnant or coexisting medical/psychiatric dx

#### Pharmacotherapy
- 3 meds approved by FDA for use only in pts who have stopped drinking & are attempting to maintain abstinence
  - Disulfiram (antabuse) – aversive therapy if ETOH is consumed w/ this, acetaldehyde accumulates → tachycardia, skin flushing, intense sweating, dyspnea, N/V
  - Naltrexone (Revia) – blocks the “high” DO NOT USE W/ HEPATITIS OR SEVERE LIVER DZ
  - Acamprosate (Campral) – reduces physical distress & emotional discomfort

#### Should be adjunctive tx w/ other intervention programs
- Counseling, meds, evaluation for concurrent mental illness
### Substance withdrawal

#### Narcotics:
- Euphoria
- Drowsiness
- Respiratory depression
- Constricted pupils
- Nausea
- Constipation

#### Overdose:
- Respiratory depression
- Clammy skin
- Convulsions
- Coma
- Possible death

#### Withdrawal:
- Watery dilated eyes
- Rhinorhea
- Yawning
- Anorexia
- Irritability
- Restless
- Tremors
- Panic
- Cramps
- Nausea & vomiting
- Chills & sweating
- Diarrhea

#### Hallucinogens:
- LSD
- Altered perception of time and distance

#### Overdose:
- Increased body temperature
- Electrolyte imbalance
- Cardiac arrest

#### Withdrawal:
- Muscle aches
- Drowsiness
- Depression
- Acne

#### MDMA & Analogs:
- Heightened senses
- Diuresis
- Dehydration

#### Overdose:
- Few if any

#### Withdrawal:
- LSD & Other

#### Depressants:
- Hospitalize
- Flumazenil
- LA benzodiazepine substitution
- IV Valium, Ativan or phenobarbital.
- Thiamine for alcohol withdrawal
- Patients going through withdrawal may look “bad,” very anxious & like they may die!

#### Overdose:
- Anxiety
- Insomnia
- Tremors
- Delirium
- Seizures
- Death

#### Withdrawal:
- Anxiety
- Insomnia
- Delirium
- Psychosis
- Seizures
- CV collapse

#### Stimulants:
- Benzodiazepines
- Antipsychotics
- Medical & psychological support

What is serotonin syndrome: HTN, tachycardia, agitation, heavy sweating, diarrhea, headache, hot flushing

#### Effects
- Increased alertness
- Excitation
- Euphoria
- Increased pulse & BP
- Insomnia
- Anorexia

#### Overdose:
- Agitation
- Increased body temp
- Hallucinations
- Seizures
- Death

#### Withdrawal:
- Apathy
- Hypersomnia
- Irritability
- Depression
- Disorientation

#### Cannabis:

#### Anabolic steroids:

---

- 12 step meetings (not tx but support system)

- Narcan for overdose
- Hydration
- **Clonidine 0.1mg** every 8 hours for 3-10 days during withdrawal
- **Lorazepam 1-2mg PO** every 4-6 hours for 5 days
- Opioid rotation
- Avoid short-acting
- **Loperamide 2mg PO** three times a day as needed for diarrhea
- Narcotics anonymous

- Hospitalize
- Flumazenil
- LA benzodiazepine substitution
- IV Valium, Ativan or phenobarbital.
- Thiamine for alcohol withdrawal
- Patients going through withdrawal may look “bad,” very anxious & like they may die!

- Benzodiazepines
- Antipsychotics
- Medical & psychological support

What is serotonin syndrome: HTN, tachycardia, agitation, heavy sweating, diarrhea, headache, hot flushing
### Acute reaction to stress

- Like PTSD
- Sx for duration <1 mo.
- Other criteria less stringent than PTSD

- Initial counseling/psychotherapy
- If persistent, same meds as for PTSD
- Treatment may vary according to acuity of patient

### Child abuse

At Risk:
- Parent w/ mental illness or substance abuse
- Unwanted pregnancy
- Children w/ behavioral issues
- Low socioeconomic status, unemployment, intimate partner violence, low education
- Chronically ill children

- Metaphyseal, posterior rib, long bone & flat bone fx
- Subdural hemorrhages
- Cigarette burns
- Low wt.
- Developmental delays
- Aggression & impulse control
- Poor school performance
- Torn frenulum
- Bruises on face & soft tissue
- Dental neglect
- Belt & bite marks
- Burns around the genitals & soles of feet

- Texas Department of Family & Protective Services (DFPS)
  - Texas family code requires everyone to report suspected child abuse, including medical professionals
  - HIPAA authorizes disclosure of protected health information to DFPS
  - Reporting in good faith
  - Immune from civil or criminal liability
  - Report is confidential

### Known Sexual Abuse & Female Genital Exam Findings:

<table>
<thead>
<tr>
<th>Category 1 (50%)</th>
<th>Category 2 (26%)</th>
<th>Category 3 (45%)</th>
<th>Category 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal appearing genitalia</td>
<td>No findings</td>
<td>Nonspecific findings (possible evidence)</td>
<td>Specific findings (strong evidence)</td>
</tr>
<tr>
<td>• Inflammation</td>
<td>• Lacerated hymen</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Scratching</td>
<td>• Hymen opening &gt; 1 cm</td>
<td></td>
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<tr>
<td>• Redness</td>
<td>• Laceration to rectal mucosa</td>
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<td></td>
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<tr>
<td>• Submucosal erythema</td>
<td>• Bite marks on vulva</td>
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</tr>
<tr>
<td>• Small skin fissures</td>
<td>• Lab evidence of STD</td>
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</tr>
</tbody>
</table>
Known Sexual Abuse Anal Findings:
- Erythema, swelling, laxity (looseness), dilation
- Fissures, skin changes
- Hematoma, bruising
- Infection

Elder abuse
- Most common reporters are family members & social services agency staff
  - 2-10% of elderly may be victims of moderate to severe abuse
- 3 categories – domestic elder abuse, institutional elder abuse, & self-neglect or self-abuse
- Failure to Self-Report:
  - Fear of losing care giver
  - Shame
  - Cognitive impairment
  - Strong coercion
  - Isolation

Neglect Indicators:
- Dehydration
- Malnutrition
- Untreated medical conditions
- Inadequate clothing for the season
- Unclean environment

Healthcare provider is legally obligated to report to appropriate authorities (Adult Protective Services)

Conduct disorders
- Group of behavioral & emotional problems in youngsters
- Children & adolescents have great difficulty following rules & behaving in a socially acceptable way
- Contributing factors: brain damage, child abuse or neglect, genetic vulnerability, school failure, & traumatic life experiences

- Intolerable disruptive behavior, dangerous at home, in school or in the community

-DSSM-IV criteria:
- Repetitive/persistent pattern in which basic rights of others or major age-appropriate rules are violated (manifested by 3 (or more) of the following criteria in the past 12 mos., w/ at least 1 criterion in the past 6 mos.:
  - Aggression to people & animals
    - Bullies/threatens/ intimidates others
    - Initiates physical fights
    - Has used a weapon that can cause serious harm to others
    - Has been physically cruel to people or animals
    - Has stolen while confronting a victim
    - Has forced someone into sexual activity
  - Destruction of property
    - Has engaged in fire setting w/ intentions of causing serious damage
    - Has destroyed others’ property (other than by fire setting)
  - Deceitfulness or theft
    - Has broken into someone else’s house, building, or car

- Too complex to be treated by a single method
- Individually tailored combos of biological, psychosocial & ecological interventions are most effective
- Head Start programs at school
- Methylphenidate to • defiance, oppositionalism, aggression, & mood changes in pts age 5-8 yrs.
- Divaloprex to • hyperarousal, anger, & aggressiveness
- Lithium
| Domestic violence | -90-95% of victims are women
-Characterized by physical & sexual attacks as well as psychological & economic coercion (threats, throwing objects, verbal abuse, hitting/beating, progressive social isolation, deprivation of things such as food/money/transportation or access to health care)
-Abuse is usually accompanied by shame & guilt so it is often not reported
-Phases:
  - Tension-building phase – arguing & blaming as anger intensifies
  - Battering phase – verbal threats, sexual abuse, physical battering, use of weapons
  - Honeymoon phase – abuser may deny the violence, make excuses, apologize, buy gifts & promise never to do it again until cycle starts over
| Women may report chronic pelvic pain from sexual abuse
-Persistent multiple bodily complaints such as chronic HA, palpitations, abd complaints, sleep/appetite disturbances
-Eating disorders
-Somatoform d/o – condition for which there are no organic findings or physiologic mechanisms
-Depression/suicidal ideations
-Anxiety/sleep d/o/PTSD
| -Screening assessment (direct questioning):
  - "I would like to ask you a few questions about physical, sexual, & emotional trauma b/c we know that these are common & affect women's health."
  - Has anyone close to you ever threatened to hurt you?
  - Has anyone ever hit, kicked, choked, or hurt you physically?
  - Has anyone ever hit, kicked, choked, or hurt you physically?
  - Has anyone, including your partner, ever forced you to have sex?
  - Are you ever afraid of your partner? |
| Domestic violence | -Prevention – offer the pt shelter if she is afraid for her safety or the safety of her kids
-Establish a safety plan
-Education about domestic violence & its consequences – let the pt know it is not their fault & acknowledge/document the trauma
-List of referral resources
-Refer for psychiatric screening/counseling
-Flumazenil (anxiety med withdrawal/OD) |
| Grief reaction | -Types:
  - Anticipatory grief – rxn that occurs in anticipation of an impending death
- Anticipatory grief – depression, heightened concern for the dying person, rehearsal of the death, attempts |
| Grief reaction | -Grief counseling
- Antidepressants and psychotherapy in complicated grief |
loss, provides family members w/ time to gradually absorb the reality of the loss

- Normal or common grief – gradual movement toward an acceptance of the loss and manage to continue w/ basic daily activities although it may be difficult
  - Most pts will experience sx less frequently over time
  - Stages: denial, anger, bargaining, depression and acceptance

- Complicated grief:
  - Inhibited or absent grief – little evidence of the expected separation distress, seeking, yearning, or other characteristics normal grief
  - Delayed grief – sx of distress, seeking, yearning, etc. occur at a much later time than is typical
  - Chronic – prolonged duration of grief sx
  - Distorted – extremely intense or atypical sx

to adjust to the consequences of the death

- Normal grief – emotional numbness, shock, disbelief, and/or denial often occurring immediately after the death, self-esteem is intact

<table>
<thead>
<tr>
<th>Suicide</th>
<th>Clinical judgment</th>
<th>Needs emergent psychiatric evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>-80% who commit suicide have seen a physician or other provider w/in 2 weeks before death</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Single most important element is constant awareness of the possibility that it exists</td>
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</tr>
<tr>
<td>-Risk factors:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Age – peak attempts: men 25-30 &amp; women 45-50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Ethnicity – Caucasians &gt; others</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Religion – Protestants</td>
<td></td>
<td></td>
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<tr>
<td>- Lives alone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Bereaved</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Unemployed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Poor health status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Impulsive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Rigid in thinking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Faces stressful events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Has made direct verbal warnings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Has made plans or past attempts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Clinical judgment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Needs emergent psychiatric evaluation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- If pt is at risk of hurting themselves or others, laws would allow holding pt until full evaluation is complete</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
- Suffers from depression
- Feels hopeless
- Has anxiety
## Complex regional pain syndrome
- Rare characterized by autonomic & vasomotor instability
- Most cases preceded by direct physical trauma, often of relatively minor nature, to the soft tissues, bone, or nerve
- Any extremity can be involved, but hand is most commonly affected & associated w/ ipsilateral restriction of shoulder motion (shoulder-hand syndrome)
- Intense, burning pain often worsened by minimal stimuli, such as light touch
- Localized pain
- Swelling of involved extremity
- Disturbances of color & temperature in affected limb
- Dystrophic changes in overlying skin & nails
- Limited ROM
- Radiographs: generalized osteopenia

## Treatment
- Nortriptyline
- Gabapentin
- NSAIDs for mild cases
- Prednisone for severe cases
- PT, regional nerve blocks, dorsal-column stimulation

## Peripheral neuropathies
- **Charcot-Marie-Tooth Disease:**
  - Autosomal dominant can be X-linked
  - May have foot deformities or gait disturbances in childhood or early adult life
  - Onset btwn 20-40 yrs.
- **Diabetic neuropathy:**
  - Suspected that blood vessels that supply peripheral nerves may be blocked
  - **Toxin neuropathy:**
    - Chemo drugs, anti-TB drugs, organophosphates, heavy metals, alcohol, lead (motor), acrylamide (sensory & motor), glue sniffing (sensory & motor), arsenic (sensory initially), thallium (sensory initially)
    - Seen in pts w/ chronic EtOH use w/ secondary nutritional deficiencies
- **Vitamin deficiencies:**
  - B1 deficiency – thiamine deficiency (AKA beriberi), sensorimotor polyneuropathy, caused by EtOH & anorexia
  - B6 deficiency – pyridoxine deficiency, sensory loss, can be caused by isoniazid (TB drug)
  - B12 deficiency – cobalamin deficiency, sensory loss, numbness & tingling to fingers & toes
- **Charcot-Marie-Tooth:**
  - Pes cavus: higher arches, hammer toes, trouble walking
  - Slow progression leads to typical features of polyneuropathy w/ distal weakness & wasting that begins in legs, a variable amount of distal sensory loss & depressed/absent tendon reflexes
  - May develop foot drop “champagne bottle” legs
- **Diabetic neuropathy:**
  - Early – loss of vibratory sense, pain & temperature sensation
  - Distal symmetric polyneuropathy → MOST COMMON, stocking/glove pattern (usually bilateral), symmetrical
  - Diabetic amyotrophy – severe pain to anterior thigh
  - Painful neuropathy – burning pain that is worse at night
- **Toxin neuropathy:**
  - Braces can help the foot drop
  - **Diabetic neuropathy:**
    - Amyotrophy – pain control, gabapentin, TCAs → may take up to 18 months to improve
    - Painful – control sugars, have pt look at feet daily to avoid diabetic foot ulcer
- **Toxin neuropathy:**
  - Prevent further exposure to causative agent
  - Replace nutrients, quit drinking EtOH
- **Vitamin deficiencies:**
  - B1 deficiency – banana bag: 100mg thiamine, 1mg folic acid, 1L normal saline & 10mL multivitamin
  - B6 deficiency – pyridoxine supplementation (esp. w/ anti-TB tx)
  - B12 – replace B12 (monthly IM injection)

## Cluster headache
- ≥5 attacks that are:
  - Severe, unilateral, supraorbital, &/or temporal
- ≥1 of the following on the side of pain:
  - Conjunctival injection

## Treatment
- Oxygen
- Ergotamine
<table>
<thead>
<tr>
<th><strong>Tension headache</strong></th>
<th><strong>Migraine</strong></th>
<th><strong>Encephalitis</strong></th>
<th><strong>Meningitis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>-On average &gt;15/month for 6 months  &lt;br&gt;-Most common cause of headache – by stress  &lt;br&gt;-Not common in the elderly – women &gt; men, onset in second decade of life</td>
<td>-Women &gt; men; ages 15-45  &lt;br&gt;-Genetic predisposition  &lt;br&gt;-4-72 hrs. in length if untreated  &lt;br&gt;-Common triggers:  &lt;br&gt;-Stress  &lt;br&gt;-Menses  &lt;br&gt;-Oral contraceptives  &lt;br&gt;-Alcohol  &lt;br&gt;-Food – cheese, chocolate  &lt;br&gt;-Lack of sleep  &lt;br&gt;-Glare  &lt;br&gt;-Changes in weather  &lt;br&gt;-Physical exertion  &lt;br&gt;-Fatigue  &lt;br&gt;-Head trauma</td>
<td>-Infection of the brain parenchyma  &lt;br&gt;-Causes include herpes (#1), enterovirus, EBV, CMV, measles, Eastern &amp; Western equine, St. Louis, varicella, West Nile</td>
<td>-Inflammation or infection of the meninges  &lt;br&gt;-Bacterial etiologies:  &lt;br&gt;- Neonates – gram negative bacilli, streptococci, listeria</td>
</tr>
<tr>
<td>-Physical exam:  &lt;br&gt;- Fever, malaise, stiff neck, nausea, altered mentation  &lt;br&gt;- Signs of upper motor neuron lesion (exaggerated DTRs, spastic, paralysis)</td>
<td>-&gt;5 attacks w/ 2 of the following:  &lt;br&gt;- Unilateral  &lt;br&gt;- Pulsating  &lt;br&gt;- Moderate to severe  &lt;br&gt;- Aggravated by activity  &lt;br&gt;- 1 of the following:  &lt;br&gt;- Nausea/vomiting  &lt;br&gt;- Photo/phonophobia  &lt;br&gt;- Aura (may or may not be present):  &lt;br&gt;- Scotoma  &lt;br&gt;- Numbness  &lt;br&gt;- Paresthesias  &lt;br&gt;- Paralysis</td>
<td>-Physical exam:  &lt;br&gt;- Fever, malaise, stiff neck, nausea, altered mentation  &lt;br&gt;- Signs of upper motor neuron lesion (exaggerated DTRs, spastic, paralysis)</td>
<td>-Bacterial:  &lt;br&gt;- Headache, nuchal rigidity, fever, change in mental status, seizures  &lt;br&gt;- Brudzinski or Kernig sign  &lt;br&gt;- Rash: petechial, think Neisseria</td>
</tr>
<tr>
<td>-No vomiting  &lt;br&gt;-&lt;1 of the following:  &lt;br&gt;- Phonophobia  &lt;br&gt;- Photophobia  &lt;br&gt;- Nausea</td>
<td>-No vomiting  &lt;br&gt;-&lt;1 of the following:  &lt;br&gt;- Phonophobia  &lt;br&gt;- Photophobia  &lt;br&gt;- Nausea  &lt;br&gt;-At least 2 of the following:  &lt;br&gt;- Pressing/tightening  &lt;br&gt;- Band-like pain  &lt;br&gt;- Mild or moderate severity  &lt;br&gt;- Bilateral pain  &lt;br&gt;- No aggravation by physical exertion</td>
<td></td>
<td>-Bacterial:  &lt;br&gt;- Headache, nuchal rigidity, fever, change in mental status, seizures  &lt;br&gt;- Brudzinski or Kernig sign  &lt;br&gt;- Rash: petechial, think Neisseria</td>
</tr>
<tr>
<td>-CSF: • lymphocytes, glucose normal or *  &lt;br&gt;- PCR</td>
<td>-CSF: • lymphocytes, glucose normal or *  &lt;br&gt;- PCR</td>
<td>-Supportive (acetaminophen)  &lt;br&gt;-Acyclovir: herpes simplex, varicella-zoster  &lt;br&gt;-Ganciclovir or foscamet: CMV  &lt;br&gt;-Avoid steroids</td>
<td>-S. pneumonia:  &lt;br&gt;- 1st choice – PCN G, ampicillin or ceftriaxone  &lt;br&gt;- 2nd choice – vancomycin or chloramphenicol</td>
</tr>
</tbody>
</table>

- Prophylactic – recommended if headache limits normal activity >3 days/month, is severe or has complications:  <br>- ASA  <br>- Propranolol  <br>- Amitriptyline  <br>- Clonidine  <br>- Verapamil  <br>- Abortive:  <br>- Cafergot  <br>- Sumatriptan (5-HT receptor agonist) – avoid in HTN or CAD  <br>- Analgesics: NSAIDs (avoid opiates)  <br>- Simple analgesics: acetaminophen, NSAIDs  <br>- Prophylactic treatment: TCAs (amitriptyline)  <br>- Supportive (acetaminophen)  <br>- Acyclovir: herpes simplex, varicella-zoster  <br>- Ganciclovir or foscamet: CMV  <br>- Avoid steroids  <br>- S. pneumonia:  <br>- 1st choice – PCN G, ampicillin or ceftriaxone  <br>- 2nd choice – vancomycin or chloramphenicol.
- Children <15 – H. influenzae type B, N. meningitidis, S. pneumoniae
- Adults >15 – S. pneumoniae, N. meningitidis, gram negative bacilli, listeria (age >60)

<table>
<thead>
<tr>
<th>CSF Analysis:</th>
<th>Bacterial</th>
<th>Viral</th>
<th>Fungal</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Press.</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>&gt;100</td>
<td>&gt;50</td>
<td>&gt;50</td>
<td>&lt;5</td>
</tr>
<tr>
<td>PMN</td>
<td>&gt;80%</td>
<td>&lt;50%</td>
<td>&lt;50%</td>
<td>None</td>
</tr>
<tr>
<td>Lymphs</td>
<td>&lt;20%</td>
<td>&gt;50%</td>
<td>&gt;50%</td>
<td>100%</td>
</tr>
<tr>
<td>Protein</td>
<td>&gt;100</td>
<td>&gt;50</td>
<td>&gt;50</td>
<td>&lt;50</td>
</tr>
<tr>
<td>Glucose</td>
<td>&lt;30</td>
<td>&gt;50</td>
<td>&lt;30</td>
<td>&gt;50</td>
</tr>
</tbody>
</table>

-N. meningitidis:
- 1st choice – PCN G or ampicillin
- 2nd choice – ceftriaxone
-H. influenzae:
- 1st choice – ampicillin or ceftriaxone
- 2nd choice – 3rd generation, ceph, or chloramphenicol
-Listeria:
- 1st choice – ampicillin or PCN G
- 2nd choice – TMP/sulfa
-Staph aureus:
- 1st choice – nafcillin
- 2nd choice - vancomycin
-Gram negative bacilli:
- 1st choice – cefotaxime or ceftazidime plus aminoglycoside
- 2nd choice – meropenem + aminoglycoside
-Pseudomonas:
- 1st choice – cefepime + tobramycin
- 2nd choice – meropenem + aminoglycoside
-Empiric treatment:
- Neonates:
  - 1st choice – ampicillin + cefotaxime; 2nd choice – ampicillin + gentamicin
- Children:
  - 1st choice – cefotaxime or ceftriaxone + vancomycin
  - 2nd choice – ampicillin + meropenem
- Adults:
  - 1st choice – cefotaxime or ceftriaxone + vancomycin
  - 2nd choice – meropenem
- Elderly:
  - 1st choice – cefotaxime or ceftriaxone + ampicillin + vancomycin
  - 2nd choice – cefotaxime + vancomycin + TMP/sulfa

*Dexamethasone day 1 in all pts & for 4 days in confirmed cases*
| **Essential tremor** | - Cause is uncertain – sometimes inherited autosomal dominant  
- Begins at any age; typical onset age 60s  
- Alcohol improves tremor | - Involves hands &/or head – lacks hypokinetic features & rigidity of Parkinson’s disease | - Propranolol |
|---------------------|-----------------------------------------------------|---------------------------------------------------|----------------|
| **Huntington’s disease** | - Autosomal dominant  
- Onset age 30-55 w/ gradual chorea & dementia  
- Progressive | - Personality changes, impulsiveness, aggressive behavior, depression, & paranoid psychosis are noted (may precede the motor manifestations)  
- Motor manifestations:  
  - Flicking movements of the extremities  
  - Lilting gait  
  - Inability to sustain a motor act  
  - Facial grimacing  
  - Ataxia  
  - Dystonia | - CSF normal  
- CT scan: cerebral atrophy  
- Diagnosis by genetic testing | - Supportive only  
- Genetic counseling  
- Anti-dopaminergic agents (haloperidol) may work early  
- Fatal 15-20 yrs. after diagnosis |
| **Parkinson’s disease** | - Idiopathic, slowly progressive, degenerative CNS disorder d/t dopamine depletion in basal ganglia  
- Insidious onset in older pt; men > women  
- Pill-rolling tremor, rigidity, bradykinesia, postural instability, micrographia  
- Physical exam:  
  - Mask-like facies, cog wheeling of extremities, DTR normal, shuffling gait, may have intellectual deterioration  
  - Dementia (50%) & depression common | - Levodopa  
- S/Es: nausea, vomiting, hypotension  
- Dopamine agonists:  
  - Bromocriptine, ropinirole, pramipexole  
- S/Es: orthostatic hypotension  
- Monoamine oxidase type B inhibitors (*intracellular dopamine):  
  - Selegiline, rasagiline  
- S/Es: orthostasis, confusion, headache  
- Catechol-O-methyltransferase (COMT) inhibitors prevent breakdown of dopamine:  
  - Entacapone  
- S/Es: GI upset, brown urine discoloration | - Nimodipine  
- Surgery |
| **Cerebral aneurysm** | - Weakness in the wall of a cerebral artery or vein causes a localized dilation or ballooning of the vessel  
- Asx until expansion or rupture – sx vary depending on site  
- Rupture characterized by sudden, severe headache, altered mental status, photophobia, vomiting  
- Focal neuro signs (unusual)  
- CT, MRA or angiography | - Lower BP to a MAP of <130 mmHg via nicardipine  
- Correctly underlying coagulopathy if any  
- Surgery if hemorrhage > 10 mL:  
  - Craniotomy & clot evaluation |
| **Intracranial hemorrhage** | - Pathologic accumulation of blood w/in the cranial vault  
- 8-13% of all strokes & more likely to results in death or major disability  
- Causes: hypertensive damage to vessel walls, reperfusion injury, cold exposure, altered mental status  
- N/V  
- HA  
- Seizures  
- Focal neuro deficits | - CT |
| Stroke | -Hx of atherosclerotic heart dz, HTN, diabetes, a. fib  
-Deficits >24 hrs.  
-Reduced hemostasis, hemorrhagic necrosis (tumor/infection), thrombosis, trauma  
-Endoscopic evacuation  
-Sterotactic aspiration w/ thrombolytic agents  
-Modify risk factors  
-Anticoagulation therapy – heparin for those w/ recurrent stroke  
-Surgical therapy – carotid endarterectomy  
-Thrombolysis – tissue plasminogen activator within 3 hrs.  
-Rehab  
-Complications:  
  -Pneumonia – d/t aspiration & hypoventilation  
  -Hypovolemia – d/t lack of fluids often b/c of dysphagia  
  -Hyponatremia – inappropriate ADH, diuretics, poor intake  
  -Seizures – excitable partially injured cerebral tissue  
  -Depression – organic mental changes, discouragement  
  -Shoulder dislocation – lack of proper care of paralyzed limbs  
  -Peripheral nerve injury – improper positioning of paretic limbs  
  -Decubitus ulcer – immobility  
  -UTI – indwelling catheter, bladder distention  
  -Bleeding, brain or systemic – excessive anticoagulation  
  -CHF – fluid overload  
  -Hypotension – excessive use of antihypertensives  
 | -Sudden onset neuro complaint:  
  -Focal weakness, sensory abnormalities, visual changes, language defect, altered mentation  
  -Neuro exam  
  -Cardiac/carotids  
  -Non-contrast CT scan  
  -CBC, sed rate, electrolytes, glucose  
  -VDRL, lipid profile, coagulation panel, thyroid function  |
| Transient ischemic attack | -Acute, focal neuro deficit  
-Clinical deficit resolves completely in 24 hrs.  
-Emboli is an important etiology; some sources:  
  -Cardiac: a. fib, rheumatic heart dz, mitral valve dz, infective endocarditis, atrial myxoma, MI, atrial septal defects  
-Neuro exam  
-Cardiac/carotids  
-Non-contrast CT scan  
-CBC, sed rate, electrolytes, glucose  
-VDRL, lipid profile, coagulation panel, thyroid function  |
| | -Amaurosis fugax – transient monocular blindness when internal carotid artery is affected  
-Dysphagia  
-Weakness & heaviness of contralateral arm, leg or face  
-Numbness/paresthesias  
-Carotid bruit  
-CBC, FBG, cholesterol, homocysteine, syphilis, blood cultures if endocarditis suspected  
-Chest radiograph  
-CT/MRI  
-Carotid duplex U/S  
-Conventional cerebral arteriography  |
| | -Anticoagulants  
-IV heparin  
-Warfarin  
-ASA  
-Carotid endarterectomy |
### Altered LOC

- Any measure of arousal other than normal
- Prolonged unconsciousness is a medical emergency
- Level of consciousness is a measurement of a person’s arousability and responsiveness to stimuli form the environment
- Causes: poisons/toxins, insufficient oxygen or blood flow in the brain, excessive pressure in the skull
- Lethargy – mildly depressed level of consciousness or alertness – these patients may be aroused with little difficulty
- Coma – inability to make any purposeful response

<table>
<thead>
<tr>
<th>Conscious</th>
<th>Normal</th>
<th>Check orientation to name, location and date or time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confused</td>
<td>Disoriented; impaired thinking &amp; responses</td>
<td>People who do not respond quickly w/ info about their name, location and the time = obtuse or confused Can be caused by sleep deprivation, malnutrition, allergies, pollution, drugs and infxn</td>
</tr>
<tr>
<td>Delirious</td>
<td>Disoriented, restless, hallucinations, sometimes delusions</td>
<td>Marked deficit in attention</td>
</tr>
<tr>
<td>Somnolent</td>
<td>Sleepy</td>
<td>Excessive drowsiness and responds to stimuli only w/ incoherent mumbles or disorganized movements</td>
</tr>
<tr>
<td>Obtunded</td>
<td>Decreased alertness, slowed psychomotor responses</td>
<td>Person has decreased interest in their surroundings, slowed responses, and sleepiness</td>
</tr>
<tr>
<td>Stuporous</td>
<td>Sleep-like states (not unconscious), little/no spontaneous activity</td>
<td>Only respond by grimacing or drawing away from painful stimuli</td>
</tr>
<tr>
<td>Comatose</td>
<td>Cannot be aroused; no response to stimuli</td>
<td>No response to stimuli, have no corneal or gag reflex and they may have no pupillary response to light</td>
</tr>
</tbody>
</table>

### Cerebrovascular

- Cerebrovascular
- Hypotension
- Polycythemia, sickle cell, hyperviscosity

### Hypotension

### Polycythemia, sickle cell, hyperviscosity

### Cerebral palsy

- Group of motor syndromes resulting from disorders of early brain development – delay in normal motor milestones
- Secondary to brain lesion acquired pre- or perinatal – usually d/t hypoxic-ischemic insult, infxn, hemorrhage, or brain maldevelopment
- Intellectual ability may be spared

<table>
<thead>
<tr>
<th>Cerebral palsy</th>
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<th>Cerebral palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spastic diplegia</td>
<td>Spastic diplegia</td>
<td>Spastic diplegia</td>
</tr>
<tr>
<td>Spasticity in both legs w/ relative sparing of arms related to bilateral hypoxic-ischemic lesion</td>
<td>Spastic hemiplegia</td>
<td>Spastic hemiplegia</td>
</tr>
<tr>
<td>Unilateral cerebral hemispheric damage w/ spasticity in arm &amp; leg on same side w/ undergrowth of</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Spastic diplegia</td>
<td>-Spastic hemiplegia</td>
<td></td>
</tr>
<tr>
<td>-Maximize mobility</td>
<td>-Provide team of various specialties – PT, OT, speech</td>
<td>-Spasticity treated w/ oral dantrolene sodium, benzodiazepines, &amp; baclofen</td>
</tr>
</tbody>
</table>

### Glasgow Coma Score

<table>
<thead>
<tr>
<th>Eye Opening (E)</th>
<th>Verbal Response (V)</th>
<th>Motor Response (M)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4=Spontaneous</td>
<td>5=Normal conversation</td>
<td>6=Normal</td>
</tr>
<tr>
<td>3=To voice</td>
<td>4=Disoriented conversation</td>
<td>5=Localizes to pain</td>
</tr>
<tr>
<td>2=To pain</td>
<td>3=Words, but not coherent</td>
<td>4=Withdraws to pain</td>
</tr>
<tr>
<td>1=None</td>
<td>2=No words……only sounds</td>
<td>3=Decorticate posture</td>
</tr>
<tr>
<td></td>
<td>1=None</td>
<td>2=Decorticate</td>
</tr>
</tbody>
</table>

Total = E+V+M

-Dextrose if low blood sugar
-Treat underlying cause
<table>
<thead>
<tr>
<th>Limbs &amp; abnormal posturing related to trauma or vascular event</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Choreoathetosis</td>
</tr>
<tr>
<td>- Initial hypotonia evolving into choreoathetosis &amp; dystonia (uncontrolled jerking, writhing &amp; posterior movements)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Concussion</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Transient loss of consciousness followed by complete recovery – implies an injury which resolves 7-10 days w/o complications</td>
</tr>
<tr>
<td>- Headache, dizziness, N/V, sleepiness</td>
</tr>
<tr>
<td>- Short period of amnesia is often related</td>
</tr>
<tr>
<td>- No focal neurologic findings</td>
</tr>
<tr>
<td>- Brain contusion – brain damage w/ prolonged coma, amnesia, &amp; focal signs → may suffer from chronic impairment</td>
</tr>
<tr>
<td>- CT</td>
</tr>
<tr>
<td>- Monitor for return to activity</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Alzheimer's disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Very common cause of dementia – 60-80% of cases, more common in women</td>
</tr>
<tr>
<td>- Probable Alzheimer's:</td>
</tr>
<tr>
<td>- Clinical evidence of progressive dementia</td>
</tr>
<tr>
<td>- No disturbance of consciousness</td>
</tr>
<tr>
<td>- Absence of systemic or another brain disease which causes dementia</td>
</tr>
<tr>
<td>- Definite Alzheimer's:</td>
</tr>
<tr>
<td>- Requires neuropathologic confirmation or clinical diagnosis</td>
</tr>
<tr>
<td>- Pathology:</td>
</tr>
<tr>
<td>- Cortical atrophy &amp; ventricular enlargement</td>
</tr>
<tr>
<td>- Microscopic – neurofibrillary tangles (occupy neuronal cell body) &amp; senile plaques (amyloid in microglia, astrocytes)</td>
</tr>
<tr>
<td>- Genetics:</td>
</tr>
<tr>
<td>- Autosomal dominant in some families</td>
</tr>
<tr>
<td>- Loci on chromosomes 1, 14, 19, 21</td>
</tr>
<tr>
<td>- Prognosis: life expectancy 3-15 yrs., avg. 7 years</td>
</tr>
<tr>
<td>- Anterograde amnesia is dominant sx</td>
</tr>
<tr>
<td>- Early stage:</td>
</tr>
<tr>
<td>- Loss of recent memory – inability to learn &amp; retain new information</td>
</tr>
<tr>
<td>- Language problems (word finding)</td>
</tr>
<tr>
<td>- Mood swings</td>
</tr>
<tr>
<td>- Personality changes</td>
</tr>
<tr>
<td>- Diminished ability to perform activities of daily living (drawing &amp; driving)</td>
</tr>
<tr>
<td>- Intermediate stage:</td>
</tr>
<tr>
<td>- Inability to learn &amp; recall new information</td>
</tr>
<tr>
<td>- Diminished memory of remote events</td>
</tr>
<tr>
<td>- Behavioral changes – agitation, hostility, aggressiveness, may wander</td>
</tr>
<tr>
<td>- Loss of all sense of time &amp; place – gets lost &amp; • risk of falls</td>
</tr>
<tr>
<td>- Aphasia &amp; apraxia</td>
</tr>
<tr>
<td>- Requires assistance to perform ADLs</td>
</tr>
<tr>
<td>- Severe:</td>
</tr>
<tr>
<td>- Inability to walk or perform ADLs</td>
</tr>
<tr>
<td>- Total incontinence</td>
</tr>
<tr>
<td>- Complete loss of recent &amp; remote memory</td>
</tr>
<tr>
<td>- Possible inability to swallow &amp; eat</td>
</tr>
</tbody>
</table>

- Exclude other causes of dementia: check CMP, B12, TSH, CSF studies |
- Watch for depression (can mimic early Alzheimer's) |
- Definitive diagnosis only at autopsy |
- Medications to reduce cognitive problems: |
  - Acetylcholinesterase inhibitors – racine, rivastigmine, galantamine and donepezil |
  - SEs – N/V, muscle cramps, bradycardia, decreased appetite |
  - NMDA receptor antagonist - memantine |
  - Antipsychotics for aggression and psychosis |
  - Psychosocial interventions |
Dementia

- Epidemiology:
  - 1% at age 60, doubles every 5 yrs.
  - 30-50% at age 85
  - 4th leading cause of death in elderly
  - Life expectancy after dx = 3-15 yrs.
  - More prevalent among women but this is due to their longer life expectancy

- Initial presentation:
  - Slight forgetfulness
  - Attention and concentration deficits
  - Inconsistencies in usual behavior

- Late presentation:
  - Impaired judgment
  - Inability to abstract
  - Personality change with rigidity, preservation, irritability, & confusion
  - Affective disturbances may be prominent with loss of personality and self care (hygiene)

- History from patient and a relative, friend or nurse
  - CBC, CMP, thyroid function, syphilis serology (RPR), folate & Vitamin B12 (rare to see B12 dementia, but if it is the cause it is reversible)
  - MRI is preferred but CT scan will suffice, no contrast is necessary
  - If indicated: Objective: recognize when additional diagnostic testing is needed
    - Neuropsych testing
    - HIV (if sexually active)
    - Brain biopsy
    - SPECT or PET scan – to discriminate specific clinical circumstances such as differentiating between Alzheimer’s disease and frontotemporal dementia in a patient who has symptoms of each
    - Lumbar puncture
    - EEG → PET scan, MRI

- Treatable causes of dementia:
  - Medications (including OTC meds)
  - Alcohol abuse
  - Delirium
  - Depression: pseudodementia – elderly present with depression much more different than younger patients; they are much more withdrawn and lose interest in usual activities
  - Tumors (especially in the frontal lobe)
  - Chronic heart/lung disease
  - Metabolic disorders (thyroid and hypercalcemia)
  - Head injury
  - Infection
  - Vision/Hearing problems

5 Most Common Types of Dementia:

<table>
<thead>
<tr>
<th>Alzheimer’s Disease</th>
<th>Vascular dementia (Multi-infarct dementia)</th>
<th>Diffuse Lewy Body Disease</th>
<th>Fronto-temporal Dementia (FTD)</th>
<th>Normal Pressure Hydrocephalus</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Slowly progressive - Linear decline in cognition → short-term memory impairment is the first symptom - CT shows atrophy with larger black spaces representing CSF</td>
<td>- Caused by a series of small strokes - Stepwise loss of cognition with each infarct - Symptoms depend on location of stroke - CT will show multifocal ischemic change</td>
<td>- Looks like Alzheimer’s but much more rapid progression - Associated with Parkinson-like movement disorder - Does not respond much to L-Dopa - Patients often have detailed hallucinations - DON'T USE NARCOLEPTICS because it can risk of stroke in dementia and can put patient into a coma</td>
<td>- Equal to Alzheimer’s disease as a cause of dementia in patients &lt; 60 - Focal and asymmetric atrophy of the frontal &amp; anterior temporal lobes - Group of diseases which includes behavioral-variant FTD, Semantic dementia, and progressive nonfluent dementia</td>
<td>- Wet, Wacky and Wobbly → urinary incontinence, gait disturbance (ataxia; patient falls a lot), and dementia - CT shows enlarged lateral ventricles - CSF drainage will improve gait</td>
</tr>
</tbody>
</table>

Delirium

- Acute confusional state w/ clouding of consciousness usually as a result of systemic problems
- Rapid onset
- Retrograde amnesia
- Electrolytes, glucose, BUN/creatinine, LFTs, thyroid function, ABGs, CBC, N-acetylcysteine
- Correct medical problem
- Avg. duration is about 1 week w/ full recovery in most cases
- Marked deficit of short-term memory & recall
- Extrinsic (can be changed) – medications (Beer’s list of meds that are detrimental esp. to elderly), new or exacerbated systemic dz process
- Can coexist w/ dementia
- May be hypoactive, hyperactive or mixed
- Can be caused by drugs/alcohol or another general disorder
- Terminal delirium can occur at the end of life – may be related to multiple medical causes including organ failure

<table>
<thead>
<tr>
<th>Guillan-Barre syndrome</th>
<th>Progressive, ascending, symmetric weakness w/ variable paresthesia or dysesthesia</th>
<th>Progression of symptoms, including weakness in ascending pattern, loss of proprioception</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Demyelinating injury</td>
<td>-Physical exam:</td>
<td>-Labs:</td>
</tr>
<tr>
<td>-Associated w/ viral infxn, stress, campylobacter jejuni enteritis</td>
<td>-DTRs, weakness in ascending pattern, loss of proprioception</td>
<td>- Spinal tap: total protein, normal cell count</td>
</tr>
<tr>
<td>-Physical exam:</td>
<td></td>
<td>- EMG consistent w/ demyelination</td>
</tr>
<tr>
<td>-DTRs, weakness in ascending pattern, loss of proprioception</td>
<td></td>
<td>- Watch for involvement of respiratory muscles</td>
</tr>
<tr>
<td>-Labs:</td>
<td></td>
<td>- IV immunoglobulin</td>
</tr>
<tr>
<td>- Spinal tap: total protein, normal cell count</td>
<td></td>
<td>- Plasma exchange</td>
</tr>
<tr>
<td>- EMG consistent w/ demyelination</td>
<td></td>
<td>- Steroids are not effective</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Multiple sclerosis</th>
<th>-Slowly progressive CNS disease w/ disseminated patches of demyelination in brain &amp; spinal cord – probably autoimmune in nature</th>
<th>-CSF studies: mononuclear cell pleocytosis, elevated levels of total Ig, presence of oligoclonal Ig</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Women &gt; men, onset &lt;50, Northern European descent</td>
<td>-Episode sx:</td>
<td>-MRI: abnormal hyperintense arease in white matter, found in brainstem, cerebellum &amp; spinal cord</td>
</tr>
<tr>
<td></td>
<td>- Sensory abnormalities, <strong>blurred vision (optic neuritis)</strong>, sphincter disturbances, weakness (heat may worsen sx)</td>
<td>- Modify dz course:</td>
</tr>
<tr>
<td></td>
<td>- Lhermitte’s sign positive – sensation of electricity down back w/ passive flexion of the neck</td>
<td>- Corticosteroids</td>
</tr>
<tr>
<td></td>
<td>- Fatigue, depression, bladder urgency, weakness, impaired balance &amp; coordination</td>
<td>- Interferon-β1b (Betaseron), IFN-β1a (Avonex), IFN-β1a (Rebif)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Flatiramer acetate (Copaxone)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Natalizumab (Tysabri)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>All of the above are for RELAPSING &amp; REMITTING MS</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Mitoxantrone (Novantrone) – for WORSENING forms of MS &amp; secondary progressive MS</td>
</tr>
</tbody>
</table>

**Calcium/phosphorus/magnesium/B12/folate, blood cultures, UA**

-EEG/CT/MRI may also be helpful

-MMSE, mini-cog exam, etc.

-D/c drugs that may be contributing to problem: analgesic, corticosteroids, cimetidine, lidocaine, CNS depressants

-Meds only for behavioral control or subjective distress

-If alcohol/substance withdrawal – benzo diazepine

-Haloperidol if no withdrawal
### Myasthenia gravis
- Due to block of neuromuscular transmission – Ach receptors, antibody-mediated autoimmune
- All ages affected, mainly young women
- Fluctuating weakness/fatigue of voluntary muscles – proximal muscles affected more than distal
- Diplopia, dysphagia, ptosis, facial weakness, difficulty swallowing
- Early stage affects the eye muscles, activity • weakness
- Ach-receptor antibodies
- Short-acting anticholinesterase (Tensilon test: edrophonium) → improvement in weakness, cholinergic side effects: nausea, diarrhea, bradycardia
- EMG
- Anticholinesterase drugs – pyridostigmine or neostigmine
- Thymectomy
- Plasmapheresis to relieve sx
- Corticosteroids – long term tx
- Aminoglycosides worsen dz so should be avoided

### Post-concussion syndrome
- Presents immediately or months after injury
- Headache, dizziness, sensitivity to light or noise, blurred vision, irritability, anxiety, depression, or change in personality
- EMG

### Seizure disorders
- Sudden, excessive, & disorderly discharge of cerebral neurons which causes abnormal movements or perceptions
- Peak incidence in childhood
- Etiologies (VITAMINS mnemonic):
  - Vascular
  - Infection
  - Trauma
  - Autoimmune
  - Metabolic
  - Idiopathic
  - Neoplasm
  - Psychiatric
- Partial seizures:
  - Simple
    - Focal motor or somatosensory sx
    - Consciousness is preserved
    - May be followed by transient neuro deficit (Todd’s paralysis)
  - Complex partial
    - Impaired consciousness may be preceded, accompanied or followed by aura sx
    - Motor dysfunction: chewing movements, lip smacking
- Generalized seizures:
  - Absence (petit mal)
  - Myoclonic
  - No loss of consciousness
  - Single or multiple myoclonic jerks
  - Tonic-clonic
    - Sudden loss of consciousness
    - Rigid, fall to ground
    - Urinary incontinence (common)
- EEG: most important
- MRI: rules out other causes
- Partial simple: phenytoin, carbamazepine
- Complex partial: phenytoin, carbamazepine, lamotrigine
- Tonic-clonic: phenytoin, carbamazepine, lamotrigine
- Absence: ethosuximide, lamotrigine
- Myoclonic: valproic acid, clonazepam
- Febrile: clonazepam

### Status epilepticus
- Single seizure lasting 30 min. or multiple seizures w/o regaining consciousness
- Tonic-clonic seizures
- EEG: most important
- CMP, LP, CT/MRI, EEG
- Rule out other causes: drug screen, lorazepam or diazepam
- Phenytoin or fosphenytoin
- May need to intubate
- Immediate thiamine & glucose

### Syncope
- Vasodepressor:
  - Caused by excessive vagal tone or impaired reflex control of the peripheral circulation
- Transient loss of consciousness & postural tone for few seconds to few min.
- Examine for orthostatic changes
- Treat underlying cause
“Common faint” most common – often initiated by stressful situations
-Orthostatic:
  - Caused by impaired vasoconstrictive response to assuming upright posture, leading to abrupt decrease in venous return
  - Occurs in advanced age, DM, blood loss or hypovolemia, vasodilator, diuretic or adrenergic-blocker therapy
-Cardiogenic:
  - Caused by rhythm disturbances (sick sinus syndrome, AV block, tachyarrhythmias) or mechanical causes (aortic or pulmonary stenosis, hypertrophic obstructive cardiomyopathy, pulmonary HTN, atrial myxoma)
  - Episodes are often exertional

Tourette disorder
- Inherited disorder w/ onset in childhood – cause is unknown
- Multiple physical tics (eye blinking, facial movements, sniffing, throat clearing) & at least one vocal tic
- Atypical neuroleptics (risperidone, ziprasidone, haloperidol, or clonidine)

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**PANCE/PANRE Blueprint – Musculoskeletal**

<table>
<thead>
<tr>
<th>Shoulder fracture</th>
<th>General Characteristics</th>
<th>Symptoms</th>
<th>Diagnostics</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| -Clavicle:        | - Very common in pediatrics
                  | - D/t FOOSH (fall on outstretched hand) injury |
                  | - Exam: holds ipsilateral arm close to trunk |
|                   | - XR                     | - Check neurovascular status |
|                   | - Figure 8 or cradle sling |

Shoulder dislocation
- Anterior - 90% of all dislocations: fall on abducted/externally rotated arm or forceful throwing motion
- Posterior – direct blow to anterior shoulder when arm is in adduction & internal rotation; s/p seizure
- Squared-off appearance of shoulder
- Acromion is more prominent
- Anterior: arm held externally rotated & internal rotation painful
- Posterior: arm held internal rotation & external rotation painful
- Exam: check for possible axillary nerve injury (numbness in middle of deltoid muscle)
- XR pre- & post-reduction
- Reduction – straight traction or Stimson’s method
- Immobilize
- Follow up 2-3 weeks & begin PT
- Surgery (recurrent dislocations)

Shoulder soft tissue injury
- Rotator cuff disorders:
  - Muscles: supraspinatus, infraspinatus, teres minor, subscapularis
- Rotator cuff disorders:
  - Dull aching in shoulder; difficulty reaching overhead
  - Weakness on external rotation & abduction
- Rotator cuff disorders:
  - MRI or arthrography
  - Shoulder separation:
  - XR: >5 mm of separation between clavicle & acromial process
- Rotator cuff disorders:
  - Avoid aggravating factors/rest
  - NSAIDs, steroid injection, PT, surgical repair
  - Shoulder separation:
<table>
<thead>
<tr>
<th>Forearm/wrist/hand fractures &amp; dislocations</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Boxer’s fracture:</td>
</tr>
<tr>
<td>- Fracture at distal end of 5th metacarpal</td>
</tr>
<tr>
<td>- Result from direct blow of closed fist against another object</td>
</tr>
<tr>
<td>- Increased angulation (&gt;25-30%) may result in malunion (malunion leads to permanent hyperextension deformity)</td>
</tr>
<tr>
<td>- Radial fracture:</td>
</tr>
<tr>
<td>- D/t FOOSH w/ elbow extended</td>
</tr>
<tr>
<td>- Ulnar shaft fracture:</td>
</tr>
<tr>
<td>- Nightstick fracture</td>
</tr>
<tr>
<td>- D/t direct blow to ulna</td>
</tr>
<tr>
<td>- Colles’ fracture:</td>
</tr>
<tr>
<td>- Most common injury of the wrist</td>
</tr>
<tr>
<td>- Fracture of the distal radius</td>
</tr>
<tr>
<td>- D/t FOOSH w/ wrist in extension</td>
</tr>
<tr>
<td>- Transverse fracture of the distal radial metaphysis w/ dorsal displacement of the distal fragment</td>
</tr>
<tr>
<td>- Typically also an injury to the ulnar styloid or ulnar collateral ligament</td>
</tr>
<tr>
<td>- Check for fractures of the elbow</td>
</tr>
<tr>
<td>- Smith’s fracture:</td>
</tr>
<tr>
<td>- Boxer’s fracture:</td>
</tr>
<tr>
<td>- Swelling over fracture site &amp; depression of knuckle</td>
</tr>
<tr>
<td>- Radial fracture:</td>
</tr>
<tr>
<td>- Tenderness over radial head or pain w/ passive rotation or flexion of forearm</td>
</tr>
<tr>
<td>- Ulnar shaft fracture:</td>
</tr>
<tr>
<td>- Point tenderness</td>
</tr>
<tr>
<td>- Colles’ fracture:</td>
</tr>
<tr>
<td>- “Dinner fork” deformity of the wrist</td>
</tr>
<tr>
<td>- Gamekeeper’s thumb:</td>
</tr>
<tr>
<td>- Pain over MCP of the thumb</td>
</tr>
<tr>
<td>- Scaphoid fracture:</td>
</tr>
<tr>
<td>- Point tenderness on anatomical snuffbox</td>
</tr>
<tr>
<td>- Pain w/ hand grip</td>
</tr>
<tr>
<td>- Limited ROM of wrist &amp; thumb</td>
</tr>
<tr>
<td>- Radial fracture:</td>
</tr>
<tr>
<td>- Positive fat pad: anterior or posterior</td>
</tr>
<tr>
<td>- Ulnar shaft fracture:</td>
</tr>
<tr>
<td>- XR</td>
</tr>
<tr>
<td>- Colles’ fracture:</td>
</tr>
<tr>
<td>- Fracture through radial metaphysis</td>
</tr>
<tr>
<td>- Hutchinson’s fracture:</td>
</tr>
<tr>
<td>- Difficulty to see on lateral view, need AP</td>
</tr>
<tr>
<td>- Look for associated scaphoid fracture</td>
</tr>
<tr>
<td>- Monteggia fracture:</td>
</tr>
<tr>
<td>- XR: ulna fracture w/ dislocation of the radial head, in the direction of angulation of the ulnar fracture</td>
</tr>
<tr>
<td>- Gamekeeper’s thumb:</td>
</tr>
<tr>
<td>- Tear diagnosed by measuring angle of joint opening w/ abduction stress: &gt;20% means complete tear</td>
</tr>
<tr>
<td>- Scaphoid fracture:</td>
</tr>
<tr>
<td>- XR: may need to repeat in 2 weeks to detect</td>
</tr>
<tr>
<td>- Ulnar shaft fracture:</td>
</tr>
<tr>
<td>- Long arm cast or posterior splint</td>
</tr>
<tr>
<td>- Colles’ fracture:</td>
</tr>
<tr>
<td>- Reduction &amp; immobilization w/ short arm cast (6 weeks)</td>
</tr>
<tr>
<td>- Smith’s fracture:</td>
</tr>
<tr>
<td>- Casting in supination</td>
</tr>
<tr>
<td>- May require open reduction w/ internal fixation</td>
</tr>
<tr>
<td>- Hutchinson’s fracture:</td>
</tr>
<tr>
<td>- Thumb spica or double sugar-tong splint</td>
</tr>
<tr>
<td>- Internal fixation</td>
</tr>
<tr>
<td>- Monteggia fracture:</td>
</tr>
<tr>
<td>- Closed treatment or open reduction w/ internal fixation</td>
</tr>
<tr>
<td>- Galeazzi fracture:</td>
</tr>
<tr>
<td>- Surgery</td>
</tr>
<tr>
<td>- Gamekeeper’s thumb:</td>
</tr>
<tr>
<td>- Thumb spica (partial disruption)</td>
</tr>
<tr>
<td>- Surgery (complete disruption)</td>
</tr>
</tbody>
</table>

- Boxer’s fracture:
  - Gutter splint
  - Surgery – pinning
- Radial fracture:
  - Sling support
  - Surgery
- Ulnar shaft fracture:
  - Long arm cast or posterior splint
- Colles’ fracture:
  - Reduction & immobilization w/ short arm cast (6 weeks)
- Smith’s fracture:
  - Casting in supination
  - May require open reduction w/ internal fixation
- Hutchinson’s fracture:
  - Thumb spica or double sugar-tong splint
  - Internal fixation
- Monteggia fracture:
  - Closed treatment or open reduction w/ internal fixation
- Galeazzi fracture:
  - Surgery
- Gamekeeper’s thumb:
  - Thumb spica (partial disruption)
  - Surgery (complete disruption)

- Limit ROM of shoulder
- Sling for 1st & 2nd degree
- 3rd degree may require surgery
- 4th & 5th degree will require surgery
- Rest & ice for first 48-72 h
- Fracture of distal radius w/ volar displacement of distal fragments
- D/t FOOSH or direct blow to back of wrist
- Reverse Colles’ fracture
- Radial styloid fracture – Chauffeur’s fracture
- D/t FOOSH or high-energy impact injury
- Can occur w/ radiocarpal dislocations
- Monteggia fracture: Ulna fracture usually in the proximal 1/3 & radial head dislocation
- D/t forced pronation of the forearm or direct blow over the posterior aspect of the ulna
- Galeazzi fracture:
  - Radial fracture usually located at the junction of the middle & distal thirds, & dislocation of the distal radioulnar joint
- D/t direct blow on dorsolateral wrist or from a fall
- Gamekeeper’s thumb:
  - D/t forced radial abduction at the MCP, w/ injury to ulnar collateral ligament of the thumb
- Affects pincer function
- Scaphoid fracture:
  - Most common carpal bone fracture; seen in young adults
  - Think of a pt w/ a “sprained wrist” w/ persistent pain & swelling
- D/t FOOSH injury

<table>
<thead>
<tr>
<th>Fracture</th>
<th>Diagnosis</th>
<th>Immobilization</th>
<th>Treatment Duration</th>
<th>Red Flags</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clavicle</td>
<td>Point tender clavicle XR</td>
<td>Figure 8 or cradle sling</td>
<td>4-6 weeks</td>
<td>Nonunion</td>
</tr>
<tr>
<td>Radial head</td>
<td>Elbow XR Fat pad</td>
<td>Variable</td>
<td>Variable</td>
<td>Displaced or large fracture</td>
</tr>
<tr>
<td>Ulnar shaft (nightstick)</td>
<td>Forearm XR</td>
<td>Long-arm cast Long-arm splint</td>
<td>4-6 weeks</td>
<td>Comminuted or displaced</td>
</tr>
<tr>
<td>Colles</td>
<td>Wrist XR</td>
<td>Short arm cast</td>
<td>6-8 weeks</td>
<td>Articular or comminuted</td>
</tr>
<tr>
<td>Scaphoid</td>
<td>Scaphoid XR</td>
<td>Short-arm thumb spica</td>
<td>8-12 weeks</td>
<td>Displaced, nonunion</td>
</tr>
<tr>
<td>Boxers</td>
<td>Hand XR</td>
<td>Ulnar gutter splint</td>
<td>2-3 weeks</td>
<td>&gt;15 degrees angulated</td>
</tr>
<tr>
<td>Metacarpal shaft</td>
<td>Hand XR</td>
<td>Radial or ulnar gutter splint</td>
<td>4 weeks</td>
<td>&gt;10 degree angulated</td>
</tr>
<tr>
<td>Phalangeal</td>
<td>Hand XR</td>
<td>Cast, splint, tape</td>
<td>3 weeks</td>
<td>Displaced</td>
</tr>
</tbody>
</table>

Forearm/wrist/ hand soft tissue injury

- Carpal tunnel syndrome:
  - Compression of the medical nerve under the transverse carpal ligament
  - Etiology: repetitive overuse, pregnancy, DM, hypothyroidism, RA, scleroderma, SLE
  - De Quervain’s tenosynovitis:
  - Dull ache in wrist, then burning pain, numbness, & tingling in thumb & digits 2, 3, half of 4
  - Relief w/ shaking of wrist
  - Weakness, dropping items
  - Pain which awakens at night
  - Exams: thenar muscle atrophy, sensory in distribution of medical nerve

- Carpal tunnel syndrome:
  - Nerve conduction studies/EMG
  - De Quervain’s tenosynovitis:
    - XR normal
    - Olecranon bursitis:
      - XR to rule out fracture

- Carpal tunnel syndrome:
  - Eliminate the cause
  - Splint in neutral position: cock up splint
  - Anti-inflammatory agents/steroids
  - Surgery: nerve release
  - De Quervain’s tenosynovitis:
    - RICE
    - NSAIDs
- Inflammation of the sheath around the extensor pollicis brevis & abductor pollicis longus of the thumb
- Etiology: overuse/repetitive gripping
- Epicondylitis:
  - Lateral
    - D/t manual labor, sports, or spontaneous event
    - Tennis elbow – caused by racquet sports
  - Medial
    - D/t repetitive motions in golfing, pitching, & racquet sports
- Olecranon bursitis:
  - D/t trauma & repeated irritation (bar stool elbow)
- Positive Phalen’s test & Tinel’s sign
  - Phalen:
  - Tinel:
- De Quervain’s tenosynovitis:
  - Pain (sharp or aching) radiates into hand or forearm; inability to grip
  - Point tenderness over radial styloid process
- Positive Finkelstein test (passive stretching of affected tendons)
- Epicondylitis:
  - Lateral – pain at insertion site on lateral epicondyle; • on pronation of the forearm & dorsiflexion of the wrist; palm down-lifting is painful
  - Medial – pain at insertion site on medial epicondyle; • on pronation & wrist flexion against resistance
- Olecranon bursitis:
  - If acute → painful swelling over the olecranon
  - Chronic disease is typically not painful
  - If fever, think septic bursa
- PE: normal except for pain & swelling (ROM normal)

**Ankylosing spondylitis**
- Inflammation & progressive fusion of the vertebrae
- Affects sacroiliac joint (required for dx), spine & hips (axial skeleton)
- Seronegative spondyloarthropathy
- Possible immune-mediated dz
- Onset ages 15-30, very rare after age 40
- Low aching back pain (improves w/ bend forward)
- Morning stiffness & stiffness after inactivity – improves w/ exercise
- Anterior uveitis, aortic insufficiency, 3rd degree heart block
- Increased sed rate
- Negative RF/ANA
- +HLA-B27
- XR: “bamboo appearance,” squaring of vertebral bodies, bilateral sacroiliitis

- Steroids
- Thumb spica splint
- Epicondylitis:
  - Lateral: rest, NSAIDs, elbow brace, gradual resumption of sports
  - Medial: rest, NSAIDs, gradual resumption of sports
- Olecranon bursitis:
  - Steroid injection
  - RICE
  - NSAIDs

- NSAIDs: indomethacin DOC
- Exercise & PT
- TNF blockers (infliximab, adalimumab, etanercept)
- Men > women 3:1 (different than other autoimmune dz)
- PE: limited spinal motion, loss of chest expansion, + Schober’s test (measures spine flexion)
- Pain in low back w/ radiation down leg; suggests nerve root irritation
- Area of point tenderness suggests MSK cause
- Sciatic pain in buttocks, posterior thigh, & posterolateral aspect of the leg around lateral malleolus

**Back strain/sprain**
- Most common cause of lower back pain is prolapsed disk or low back strain (mechanical)
- Pain in low back w/ radiation down leg; suggests nerve root irritation
- Area of point tenderness suggests MSK cause
- Sciatic pain in buttocks, posterior thigh, & posterolateral aspect of the leg around lateral malleolus

<table>
<thead>
<tr>
<th>Lumbar Radiculopathy</th>
<th>Lumbar Root</th>
<th>Pain</th>
<th>Sensory</th>
<th>Motor</th>
<th>DTR Altered</th>
</tr>
</thead>
<tbody>
<tr>
<td>L3</td>
<td>Medial thigh</td>
<td>Medial thigh</td>
<td>Hip flexors</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>L4</td>
<td>Medial calf</td>
<td>Medial calf</td>
<td>Quads</td>
<td>Knee</td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td>Lateral calf, dorsum foot</td>
<td>Lateral calf, dorsum foot</td>
<td>Tibialis anterior, extensor hallucis longus</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>Posterior calf, heel</td>
<td>Lateral foot &amp; ankle</td>
<td>Plantar flexors</td>
<td>Achilles</td>
<td></td>
</tr>
</tbody>
</table>

**Cauda equina**
- Secondary to spinal trauma or disc protrusion
- Bilateral sciatica
- Saddle anesthesia
- Disturbance in bowel &/or bladder function
- PE: saddle anesthesia, bilateral root dysfunction, • anal sphincter tone

**Herniated nucleus pulposus**
- Nucleus pulposus in a degenerated disk may prolapse & push out the weakened annulus → usually posterolaterally
- D/t bending or heavy loading w/ the back in flexion, usually from DDD in pts btwn 30-50
  - <1% result in a nerve root impingement syndrome
- 95% of clinically significant lumbar herniations involve L4-5 or L5-S1 (90%)
- Can lead to cauda equina if massive midline disk herniation (usually L4-5)
- Sciatica (95% sensitivity) worsens w/ back flexion
- Pain worse w/ activity
- Radicular pain w/ compression of neural structures

**Kyphosis**
- Increased convex curvature of thoracic spine
- Scheuermann’s disease: fixed kyphosis which develops at the time of puberty; cause unknown

**Lumbar Radiculopathy**
- XR not required when exam normal
- MRI/CT scan
- EMG
- Myelogram

**Back strain/sprain**
- Short-term bed rest (2d)
- NSAIDs
- Ice vs. heat
- Fitness program

**Cauda equina**
- Surgery
- Steroids

**Herniated nucleus pulposus**
- Resolves over a period of weeks
- Bed rest for up to 48h
- Modified activity, NSAIDs, PT → 1st line
- Steroid injections
  - <10% require surgical decompression

**Kyphosis**
- Curves 45-60 degrees: observe
- Curves >60 degree: use Milwaukee brace
- Surgery if no improvement
- Scheuermann’s“ brace, postural exercise
**Low back pain**
- Most common MSK complaint and a leading cause of work disability
- 80% of people experience it during their lifetime
- Degenerative change in the lumbar spine is the most commonly identified cause of low back pain
- Risk factors: heavy lifting, driving motor vehicles, jogging, weaker trunk strength, obesity, pregnancy, psychosocial factors, and cigarette smoking
- Other causes: disk herniation, spinal stenosis, spondylolisthesis
- Pattern of pain:
  - Radiation down the buttock & below knee → herniated disc
  - Pain that worsens w/ rest & improves w/ activity → ankylosing spondylitis
  - Most degenerative back dz produces opposite patterns (rest alleviating & activity aggravating)
  - Low back pain at night unrelieved by rest or supine position → possible malignancy
  - Rapidly evolving neuro deficits → urgent for possible cauda equina tumor, epidural abscess or massive disk herniation
- Testing rarely indicated in the absence of significant neurologic involvement unless sx persist beyond 4 wks.
  - XR, CT, MRI
  - Positive straight leg raising test → nerve root irritation → positive if radicular pain is produced w/ leg raised 60 degrees or less
  - Crossed straight leg sign – 90% specific for disk herniation → positive test when raising contralateral leg reproduces sciatica
- Radiographs – for suspected infxn, cancer, fx, or inflammation
  - MRI – urgent for suspected epidural mass or cauda equina tumor
- Pt education – don’t lift heavy objects, use legs rather than back when lifting, use a chair w/ arm rests, rise from bed by first rolling to one side then use arms to push to an upright position
- NSAIDs for analgesia, but severe pain may need opioids
- Epidural corticosteroid injections (not effective for chronic low back pain & only short-term relief)
- Massage therapy for chronic back pain
- Limited evidence for muscle relaxants (diazepam, cyclobenzaprine, carisoprodol, methocarbamol) → should be reserved for those who do not respond to NSAIDs
- Surgical consultation for the following:
  - Large or evolving neuro deficit
  - Sciatica caused by disk herniation

**Scoliosis**
- Lateral curvature of the spine
  - Causes:
    - 2/2 underlying causes (motor neuron dz, myopathies)
    - Idiopathic most common
  - Greatest risk in women btwn puberty growth spurt & cessation of spinal growth
  - Ages 10-13
  - Right thoracic curve most common
- Asymmetry in shoulders, iliac height & scapular prominence
  - Flank crease w/ forward bending
  - Gait & neuro exam normal
  - Screen w/ Adams test (forward bending)
- XR
  - Vertebral levels: measure greatest tilt anterior by Cobb method
- Treatment based on curvature
  - 10-15 degrees: observe & follow w/ forward bending test
  - 15-20 degrees: follow w/ AP XR
  - >20-45 degrees: brace
  - >40 degrees: refer to surgeon for internal fixation rods

**Spinal stenosis**
- Nerve compression d/t narrowing of the spinal canal or neural foramina
- Most cases result from degenerative changes
- Age 60 is avg. age
- Low back pain & bilateral extremity pain in buttocks, legs & thighs
  - Walking makes pain worse
  - Neurogenic claudication pain relieved by 15-30 min. in supine position w/ pillow btwn knees
  - Relief of sx when leaning forward while standing
  - Exam: may be normal; • pain w/ spinal extension suggests stenosis
- XR: extensive vertebral osteophytes & DDD
  - CT/MRI
- NSAIDs
  - Epidural steroid injections
  - Surgical decompression
  - Avoid aggravating activities

<table>
<thead>
<tr>
<th>Claudication</th>
<th>Feature</th>
<th>Neurogenic</th>
<th>Vascular</th>
</tr>
</thead>
</table>
| Avascular necrosis | -Complication of corticosteroid use, alcoholism, trauma, SLE, pancreatitis, gout, sickle cell, & infiltrative diseases  
-Most commonly affects proximal & distal femoral heads  
-Other common sites are ankle, shoulder & elbow  
-Jaw necrosis is associated w/ bisphosphonate therapy  
-If untreated → cortical collapse → significant joint dysfunction | -Hip or knee pain | -XR: can be normal initially  
-MRI, CT, bone scan more sensitive | -Avoidance of wt. bearing on affected joint for several weeks  
-Bone grafting for necrosis of the hip  
-Total hip replacement |
| Developmental dysplasia | -Congenital or acquired deformation or misalignment of the hip joint  
-Ranges from barely detectable to severely dislocated | -PE: click or clunk noted w/ Ortolani or Barlow maneuver | -Confirmed by U/S or XR | -Early dysplasia often treated w/ Pavlik harness  
-Surgery |
| Hip fracture | -Common in elderly w/ osteoporosis  
-Types:  
- Intracapsular: femoral head & neck: may damage blood supply  
- Extracapsular: inter or subtrochanteric | -Pain in hip that radiates to groin & inner thigh  
-Leg is short & held in external rotation | -Hip XR: PA, lateral, frog leg view | -Immobilization  
-Surgical:  
- Intracapsular: prosthetic replacement  
- Intertrochanteric: open reduction & internal fixation  
-Watch for avascular necrosis  
-Mortality high d/t DVT & PE |
| Hip dislocation | -D/t high impact trauma: knee is struck w/ hip & knee flexed, femoral head displaced from acetabulum | -PE:  
- Posterior (90%): limb short, adducted & internally rotated  
- Anterior: flexion, abduction, & external rotation | -XR, CT | -Prompt reduction, watch for sciatic nerve injury w/ posterior & avascular necrosis |
| Slipped capital femoral epiphysis | -Displacement of the femoral epiphysis  
- Femoral head slips posteriorly & inferiorly  
-Pathogenesis:  
- Abnormal stress on normal physical plate  
- Process which weakens the plate  
-If this occurs before puberty, evaluate for hormonal abnormality or systemic disorders  
-Epidemiology: | -Gait changes:  
- Limp if unilateral  
- Waddling gait if bilateral  
- Tenderness of hip, thigh atrophy  
- Lack of full internal rotation of hip  
- Decreased motion in all planes | -XR: first widening of physis w/o slippage, as slippage occurs, femoral neck rotates anteriorly while head remains in acetabulum | -Goals:  
- Prevent further slippage  
- Stabilize until physis closes  
- Do no further harm by the treatment  
- Avascular necrosis  
- Chondrolysis  
- Epiphyseal fixation  
- In-situ fixation  
- Risk of damage to articular surface or growth plate |
- Men > women 3:2
- Age of onset: men ages 14-16 & women ages 11-13
  - Associated w/:
    - Obesity or thin w/ recent growth spurt
    - Genital underdevelopment
    - Pituitary tumors
  - Hx: knee or hip pain, occasional hx of trauma

| Knee fracture | - Includes fx of patella, femoral condyles, tibial eminence, tibial tuberosity & tibial plateau
|              | - Patella: d/t direct blow
|              | - Femoral condyle: d/t axial loading w/ valgus/varus stress
|              | - Tibial eminence: d/t direct blow to proximal tibia w/ knee flex or hyperextension w/ varus/valgus stress
|              | - Tibial tubercle: d/t jumping activities, more common in males & adolescents
|              | - Tibial plateau: d/t axial loading w/ varus or valgus forces

| Knee dislocation | - May be seen w/ high or low velocity injuries
|                  | - High incidence of popliteal artery injury
|                  | - Tibiofemoral joint dislocations are an orthopedic emergency & may be limb-threatening
|                  | - May dislocations have associated fx
|                  | - Anterior: caused by severe knee hyperextension
|                  | - Posterior: occurs w/ anterior-to-posterior force to the proximal tibia such as high-energy fall on a flexed knee
|                  | - Tenderness & joint effusion w/ tibiofemoral joint dislocation
|                  | - Gross deformity

- AP, lateral & oblique XR views
- CT/MRI
- Ottawa rules for obtaining knee radiographs:
  - Age 55 years or older
  - Tenderness at head of fibula
  - Isolated tenderness of patella
  - Inability to flex knee to 90 degrees
  - Inability to bear wt. (4 steps) immediately after injury & in ED
  - Arthrocentesis if effusion present

- Patellar:
  - Knee immobilizer, crutches & restriction to partial wt. bearing
  - 6 weeks of immobilization
- Femoral condyle:
  - Surgery for open, displaced or neovascular injury
- Tibial spine:
  - Nondisplaced → immobilize
  - Surgery for unstable cases
- Tibial tubercle:
  - Nondisplaced → immobilize
  - Open reduction & internal fixation if displaced
- Tibial plateau:
  - Nonweightbearing
  - Open reduction & internal fixation for displaced fx
  - Goal is to stabilize, align, mobilize, & reduce pain of knee joint to minimize risk of posttraumatic OA
- XR
- ABI (ankle-brachial index)
- Duplex U/S for vascular injury assessment

- Many (50%) spontaneously reduce
- Reduction – sedation & longitudinal traction relocates majority
- Posterolateral dislocations often require operative reduction
| **Osgood-Schlatter disease** | Over-use injury of the apophysis of the anterior tibial tubercle at insertion of the patellar tendon. Microtrauma & inflammation cause apophysitis. Typically at insertion sites of large tendons. Often secondary to microtrauma w/ overuse-use injury. Epidemiology:
- Most common of apophyseal injuries
- Most common cause of chronic knee pain in young active adolescents
- Results from repeated extension of the knee
- Boys > girls
- Peak onset is early puberty: boys age 13 & girls age 11 | Young adolescent complaining of pain just below the knee
- Usually begins after sports or running activities
- Relieved by rest
- Bilateral in 50%
- **Tenderness of anterior tibial tubercle**
- Prominence of affected tubercle
- Pain at site elicited by knee extension against resistance or by full passive flexion
- With **intermittent pain**, avoid activities which cause pain; apply ice compress to area for 15 min. after playing
- With **severe/persistent pain**, complete joint rest for several weeks & gradual return to physical activity; knee immobilizer → includes hamstrings & quads stretching
- NSAIDs usually not recommended |
| **Knee soft tissue injury** | Knee bursitis:
- Inflammatory periarticular disorder of the bursa
- Anseria bursa: located medially — Tenderness noted below the joint line
- Recurrent bursitis here is noted w/ OA of the knee
- Prepatellar bursa (housemaid’s knee): noted between skin & patella
- Semimembranous bursa: noted posterior knee → site of Baker’s cysts
- Caused by repetitive friction, trauma, or systemic dz (RA, gout, infxn)
- Baker’s cyst:
  - Fluid-filled semimembranous bursa behind the knee
  - Located medial aspect in popliteal space
  - Develops at any age
- Knee bursitis:
  - Pain & tenderness, swelling, • in fluid
- Baker’s cyst:
  - Bulge behind knee – lateral to medial hamstrings in popliteal fossa
- Meniscal/ligament injuries:
  - Present w/ pain & effusion
  - Hemarthroses are common
  - Meniscal injury will see joint line pain, effusion & locking/popping
  - Ligament injury will see popping of knee, inability to bear wt., & swelling
  - Drawer test: cruciate ligaments
  - Lachman & pivot-shift test (ACL)
  - Bulge test for effusion
  - McMurray’s: pt supine, knee flexed & externally (medial meniscus) & internally (lateral meniscus) rotated, then extended → **PAIN = TEAR**
  - Apley’s: pt prone, knee to 90 degrees, axial loading w/ rotation causes pain w/ meniscal disease
- Baker’s cyst:
  - US/MRI
  - Meniscal/ligament injuries:
  - MRI
  - Physical exam
  - Knee bursitis:
  - Prevention
  - Rest
  - NSAIDs, steroid injections
  - Baker’s cyst:
  - Drainage
  - Surgical removal
  - Meniscal/ligament injuries:
  - Knee immobilization
  - Crutches
  - Analgesics
  - Ortho consult

**Meniscal/Ligament Injuries**
- Etiology: in adults, d/t pathology in the knee such as medial meniscus, RA or OA
  - Meniscal/ligament injuries:
    - D/t trauma
    - Ligament involved depends on mechanism of injury
    - Meniscal injuries commonly associated w/ ligament injuries

<table>
<thead>
<tr>
<th>Meniscus/Ligament</th>
<th>Mechanism of Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meniscus</td>
<td>Twisting or hyperflexion</td>
</tr>
<tr>
<td>Medial collateral ligament</td>
<td>Blow to lateral aspect of leg or lower thigh</td>
</tr>
<tr>
<td>Lateral collateral ligament</td>
<td>Blow to medial aspect of leg or lower thigh</td>
</tr>
<tr>
<td>Anterior cruciate ligament</td>
<td>Sudden deceleration or rotation</td>
</tr>
</tbody>
</table>
| Posterior cruciate ligament | External force on anterior aspect w/ knee flexed
  OR Forced hyperflexion or hyperextension w/ a varus/valgus force |

### Acute osteomyelitis

- Inflammatory/infectious process of the bone
- Acute: develops over 4-6 weeks
- Chronic: develops over months to yrs.
- Etiology: *S. aureus*, *E. coli*, *Pseudomonas*, *Salmonella*, anaerobes
- 2 pathways of infxn:
  - Hematogenous: spread from distant infectious foci
  - Contiguous: secondary to infxn close to bone → DM a risk factor for chronic dz

### Risk Factor

<table>
<thead>
<tr>
<th>Organism</th>
<th>First Choice</th>
<th>Second Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>S. aureus</em> (methicillin-sensitive)</td>
<td>Nafcillin, Oxacillin</td>
<td>2nd generation cephalosporin</td>
</tr>
<tr>
<td><em>S. aureus</em> (methicillin-resistant)</td>
<td>Vancomycin</td>
<td>Gentamicin or TMP/SXT plus rifampin, linezolid</td>
</tr>
<tr>
<td><em>Enterococcus</em></td>
<td>Ampicillin</td>
<td>Vancomycin</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>Ciprofloxacin</td>
<td>3rd generation cephalosporin</td>
</tr>
<tr>
<td><em>Pseudomonas</em></td>
<td>Ciprofloxacin</td>
<td>Piperacillin &amp; gentamicin</td>
</tr>
<tr>
<td>Anaerobes</td>
<td>Clindamycin</td>
<td>Ampicillin, metronidazole</td>
</tr>
<tr>
<td>Mixed</td>
<td>Ampicillin-sulbactam</td>
<td>Imipenem</td>
</tr>
</tbody>
</table>

### Acute osteomyelitis

- Fever, chills, malaise, anorexia, night sweats
- Erythema, drainage, swelling, pain at site of infxn, warmth
- Increased WBC, sedimentation rate (if normal NOT osteomyelitis)
- Blood cultures
- XR: soft tissue swelling, periosteal rxn, cortical irregularities (may not be noted for 10-14 d)
- MRI, white blood cell scan (indium 111 white cell scan)

### Septic arthritis

- Underlying joint dz predisposes to septic arthritis
- Hx of joint trauma &/or hematogenous spread
- May be 2/2 UTI, IVDA, IV lines
- Microbiology:
  - *S. aureus* most common
  - IVDA – think *pseudomonas*
  - Young adults, think *N. gonorrheae*
  - Chronic: TB, fungi, Lyme dz

- Fever, shaking chills
- Swollen, tender, erythematous joint, limitation of motion
- Synovial fluid: • WBC w/ neutrophils (positive gram stain & cultures)
- Drainage & abx
- Staphylococcus: nafcillin, vancomycin, 1st generation cephalosporin
- GC: ceftriaxone
- Pseudomonas: ceftazidime, aminoglycoside
**Bone cysts/tumors**

- **Osteosarcoma:**
  - Osteogenic sarcoma is most common primary malignant tumor of bone
  - Common in male children/young adults & common around the knee
  - Other common locations – proximal humerus & proximal femur

- **Chondrosarcoma:**
  - Results from malignant cartilaginous cells
  - Peak 50–60 yrs. old
  - Common in the knee, shoulder, pelvis & spine

- **Ewing sarcoma:**
  - Small blue cell tumor w/ characteristic t(11:22) chromosomal translocation
  - Common in children >5 & in young adults → if <5 yrs. leukemia, & metastatic neuroblastoma must be excluded
  - Pelvis, knee, proximal humerus & femur diaphysis are most common locations

- **Osteoid osteoma:**
  - Benign bone lesion producing pain in pts 5-30 yrs. old

- **Enchondroma:**
  - Benign cartilaginous tumors in the metaphyses of long bones & the hand
  - Ollier dz – multiple enchondromas w/ 30% risk of chondrosarcoma

- **Osteochondroma:**
  - Benign surface lesion of bone characterized by a cartilaginous cap connected to the medullary cavity of the underlying bone
  - Can be pedunculated or sessile
  - Multiple hereditary exostoses – autosomal dominant condition where pts have multiple osteochondromas

- **Giant cell tumor:**
  - Benign but can be locally aggressive

---

- **Bone cysts/tumors**

<table>
<thead>
<tr>
<th>Bone Cyst/Tumor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteosarcoma</td>
<td>Histology: osteoid production w/ malignant stromal cells</td>
</tr>
<tr>
<td></td>
<td>XR: destructive lesion w/ some bone formation</td>
</tr>
<tr>
<td></td>
<td>Chondrosarcoma:</td>
</tr>
<tr>
<td></td>
<td>XR: cortical thickening &amp; stippling consistent w/ cartilage deposition</td>
</tr>
<tr>
<td></td>
<td>Ewing sarcoma:</td>
</tr>
<tr>
<td></td>
<td>Elevated inflammatory markers &amp; leukocytosis</td>
</tr>
<tr>
<td></td>
<td>XR: destructive, frequently diametaphyseal lesion</td>
</tr>
<tr>
<td></td>
<td>Classic “onionskin” appearance of multiple layers of reactive periosteum is uncommon</td>
</tr>
<tr>
<td></td>
<td>Osteoid osteoma:</td>
</tr>
<tr>
<td></td>
<td>XR: radiolucent nidus w/ sclerotic, reactive rim</td>
</tr>
<tr>
<td></td>
<td>Bone scan always positive</td>
</tr>
<tr>
<td></td>
<td>Enchondroma:</td>
</tr>
<tr>
<td></td>
<td>XR: lytic lesion w/ stippled appearance</td>
</tr>
<tr>
<td></td>
<td>Giant cell tumor:</td>
</tr>
<tr>
<td></td>
<td>XR: metaphyseal lytic lesions extending to the epiphysis</td>
</tr>
<tr>
<td></td>
<td>Aneurysmal bone cyst:</td>
</tr>
<tr>
<td></td>
<td>XR: expansile lesion w/ thin rim of cortical bone</td>
</tr>
<tr>
<td></td>
<td>Blood-filled interior w/o endothelial lining</td>
</tr>
<tr>
<td></td>
<td>Osteoid osteoma:</td>
</tr>
<tr>
<td></td>
<td>NSAIDs for pain</td>
</tr>
<tr>
<td></td>
<td>50% of lesions will “burn out” w/ conservative management</td>
</tr>
<tr>
<td></td>
<td>Percutaneous radiofrequency ablation of nidus for persistent pain</td>
</tr>
<tr>
<td></td>
<td>Enchondroma:</td>
</tr>
<tr>
<td></td>
<td>Observe &amp; follow w/ serial radiographs at 3 months &amp; 1 yr. after presentation</td>
</tr>
<tr>
<td></td>
<td>Curettage &amp; bone grafting if needed</td>
</tr>
<tr>
<td></td>
<td>Osteochondroma:</td>
</tr>
<tr>
<td></td>
<td>If asx → observation</td>
</tr>
<tr>
<td></td>
<td>Painful → resection</td>
</tr>
<tr>
<td></td>
<td>Giant cell tumor:</td>
</tr>
<tr>
<td></td>
<td>Cortical windowing, aggressive curettage, chemical cauterization w/ phenol &amp; bone grafting</td>
</tr>
<tr>
<td></td>
<td>Radiation if inoperable</td>
</tr>
<tr>
<td></td>
<td>Aneurysmal bone cyst:</td>
</tr>
<tr>
<td></td>
<td>Curettage w/ bone grafting</td>
</tr>
<tr>
<td></td>
<td>Recurrence common if physes are open</td>
</tr>
</tbody>
</table>

---

- **Osteosarcoma:**
  - Neoadjuvant chemo followed by resection & maintenance chemo
  - Chondrosarcoma: Surgical resection w/ wide margins is the tx of choice
  - Ewing sarcoma: Chemo, radiation, surgical intervention → 70% long-term survival
  - Osteoid osteoma: NSAIDs for pain
  - 50% of lesions will “burn out” w/ conservative management
  - Percutaneous radiofrequency ablation of nidus for persistent pain
  - Enchondroma: Curettage & bone grafting if needed
  - Osteochondroma: If asx → observation
  - Painful → resection
  - Giant cell tumor: Cortical windowing, aggressive curettage, chemical cauterization w/ phenol & bone grafting
  - Radiation if inoperable
  - Aneurysmal bone cyst: Curettage w/ bone grafting
  - Recurrence common if physes are open
### Aneurysmal Bone Cyst
- Benign but can be associated with other tumors (giant cell tumor, chondroblastoma, fibrous dysplasia)
- May also be found within a malignant tumor
- 75% are <20 yrs. old
- Pain & swelling months to years

### Ganglion Cysts
- A cystic collection of synovial fluid within a joint or tendon sheath
- Arise from herniation of synovial tissue from a joint capsule or tendon sheath
- Presents with tender cystic swelling over or near a tendon sheath
- Common locations include dorsal or volar wrist, flexor surface of MCP joint, or base of nail
- Many resolve spontaneously

### Osteoarthritis
- Most common form of joint disease
- Increased incidence w/ age, obesity, & joint wear & tear
- Non-inflammatory
- Primary & secondary causes:
  - Idiopathic: no underlying factor
  - Secondary: have underlying factor (trauma, avascular necrosis, hip dysplasia, metabolic disorders, gout)
- Decreased ROM, deep achy pain, crepitus, tenderness
- Pain in AM <60 min., relieved by rest (early in dz)
- Heberden’s nodes (DIP)
- Bouchard’s nodes (PIP)

### Osteoporosis
- Metabolic dz of bone
- Decreased bone mass; susceptibility to fracture
- Primary:
  - Type I: loss of estrogens, seen in postmenopausal women only; fx: wrist & vertebrae
  - Type II: age-related, both men & women; fx: hip
- Secondary:
  - D/t Cushing’s, steroids, thyrotoxicosis, multiple myeloma, hyperparathyroidism, anticonvulsants, alcohol
- Asx; pain w/ fracture

<table>
<thead>
<tr>
<th>Classification</th>
<th>Density Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; -1 SD below normal</td>
</tr>
<tr>
<td>Osteopenia</td>
<td>&gt; -1 SD but &lt; -2.5 below normal</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>&gt; -2.5 SD below normal</td>
</tr>
<tr>
<td>Severe osteoporosis</td>
<td>&gt; -2.5 SD below normal &amp; presence of fracture</td>
</tr>
</tbody>
</table>

- Labs: normal
- Bone density: dual energy XR absorptiometry
- XR: asymmetric narrowing of joint space, osteophytes, subchondral sclerosing, & bone cysts
- ESR normal
- Synovial fluid: mild inflammation, no crystals

- Acetaminophen, NSAIDs, steroid injections, exercise
- Capsaicin cream
- Joint replacement
- PT

### Prevention
- Activity, calcium, vitamin D, estrogen-progesterone therapy
- Estrogen therapy contraindicated in pts at high-risk of endometrial or breast cancer
- Anti-resorptive:
  - Bisphosphonates: slow resorption & density; S/Es → “frozen bones,” jaw osteonecrosis
  - Calcitonin: inhibits bone resorption; S/Es → nasal stuffiness, flushing
- USPSTF recommendations for bone-mineral density testing in women:
  - Screen all women age 65+
  - Screen women younger than age 65 if their 10-yr fx risk is similar to...
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Management</th>
</tr>
</thead>
</table>
| **Compartment syndrome**        | Predominant in lower extremities but can occur anywhere on the body  
- Caused by compromised blood flow  
- Pallor, pulselessness (late & ominous sign), pain, paresthesias & poikilothermia  
- Pain on passive stretching of the muscle groups  
- Pain out of proportion to physical findings | Measure intracompartamental pressures w/ Stryker pressure monitor or w/ needle connected to an arterial line pressure monitor  
- > 30 mmHg are abnormal & lead to necrosis of nerve & muscle  
- Immobilization  
- Removal of any constricting bandages or splints  
- Fasciotomy if pressure > 30 mmHg |
| **Fibromyalgia**                | Etiology unknown: women > men  
- Non-articular musculoskeletal aches  
- Chronic pain & stiffness  
- Fatigue  
- Poor sleep  
- Exam: multiple tender points (> 11 of 18 tender points positive) | No lab markers  
- TCAs  
- Physical activity  
- Pt education |
| **Gout**                        | D/t altered purine metabolism  
- Sodium urate crystals in synovial fluid  
- Uric acid in the urine (kidney stones)  
- Men, peak age 45 | Abrupt onset, throbbing pain, single joint (great toe)  
- First attack at night  
- Pain, redness, swelling of joint – mimics cellulitis  
- Increased serum uric acid (> 6.0 mg/dl)  
- Monosodium urate crystals in synovial fluid – needle-shaped, negatively birefringent (the refraction of light in an anisotropic material (as calcite) in two slightly different directions to form two rays)  
- High-dose NSAIDs, colchicine, steroids (acute)  
- Allopurinol (overproducers, or under secretors)  
- Probenecid (under secretors)  
- Avoid thiazide, furosemide, cyclosporine, ASA, alcohol (will elevate uric acid levels) |
| **Pseudogout**                  | AKA calcium pyrophosphate dihydrate deposition disease (CPPD)  
- Etiology: calcium-containing deposits in pericellular matrix of cartilage | Affects large joints in lower extremities – knee, wrist, shoulder, ankle  
- Asx or arthritis-like pain  
- Calcium pyrophosphate crystal in synovial fluid – rhomboid shaped, positive birefringent  
- XR: chondrocalcinosis  
- NSAIDs, intraarticular steroids  
- No role for uric-acid lowering drugs |
| **Juvenile rheumatoid arthritis** | Chronic arthritis in one or more joints for at least 6-12 weeks  
- Cause unknown, possible autoimmune  
- 4 main subtypes:  
  - Oligoarticular, polyarticular, systemic & enthesitis-associated  
  - Oligoarticular – most common, medium to large joints, leg length discrepancy  
  - Polyarticular – large & small joints, symmetrical  
  - Systemic – known as Still disease, high fever  
- Pain, swelling, warmth, tenderness, morning stiffness, & • ROM | No diagnostic tests  
- Elevated ESR, CRP, WBC & platelet count  
- Positive anti-cyclic citrullinated peptide (CCP)  
- NSAIDs  
- Methotrexate  
- Leflunomide – antipyrimidine medication  
- TNF medications – etanercept, infliximab  
- Corticosteroids  
- Rehab |
| **Polyarteritis nodosa**         | Vasculitic disease involving medium-sized arteries | Fever, malaise, wt. loss  
- Positive anti-neutrophilic cytoplasmic antibody (P-ANCA)  
- Steroids  
- Immunosuppressive therapy |
<table>
<thead>
<tr>
<th><strong>Polymyositis</strong></th>
<th><strong>Polymyalgia rheumatica</strong></th>
<th><strong>Reactive arthritis (Reiter’s syndrome)</strong></th>
<th><strong>Rheumatoid arthritis</strong></th>
</tr>
</thead>
</table>
| - Mainly middle-aged men  
- Affects the skin, kidneys, peripheral nerves & GI tract | - An idiopathic inflammatory myopathy  
- Women > men; age 40-50 yrs. | - Reactive arthritis in response to an infectious process elsewhere in the body  
- Chlamydia, gastroenteritis (Yersinia, Campylobacter, Shigella, Salmonella)  
- Associated w/ HLA B-27 | - Etiology:  
- Chronic, systemic disease – immune complex formation leading to immune rxn & pannus formation –thickening of the synovium  
- Symmetric synovial inflammation of peripheral joints – PIP, MCP, wrists, elbows, knees & ankles  
- Epidemiology:  
- Women > men 3:1 |
| - Renal involvement, peripheral neuropathy  
- Skin: palpable purpura  
- Post-prandial abdominal pain | - Present w/ gradual, progressive bilateral proximal muscle weakness  
- Leg weakness presents first – difficulty in rising from a chair  
- No facial or ocular muscle weakness | - Asymmetrical oligoarthritis which favors the lower extremities  
- Conjunctivitis  
- Urethritis  
- Exam:  
  - “Sausage toes or fingers”  
  - Mucocutaneous lesions – small, shallow, painless ulcers on glans penis (circinate balanitis)  
  - Painless, papuloquamous eruptions on palms & soles – keratoderma blennorrhagicum | - Criteria:  
- Morning stiffness (>1h) for 6 weeks  
- Swelling (soft tissue) of 3+ joints – DIP & lower back not involved  
- Swelling (soft tissue) of hand joints (PIP, MCP or wrist)  
- Symmetrical swelling (soft tissue)  
- Subcutaneous rheumatoid nodules  
- Serum rheumatoid factor positive |
| - Elevated ESR  
- Anemia, leukocytosis  
- UA: positive protein & blood  
- Dx by tissue biopsy  
- Angiogram reveals micro-aneurysms | - Elevated CK & aldolase  
- ESR & CRP are often normal  
- Dx by muscly biopsy | - Negative ANA, RF  
- Increased sedimentation rate (>50)  
- Normocytic, normochromic anemia | - RF positive in 80%  
- Anti-CCP (cyclic citrullinated peptide) positive  
- Elevated ESR  
- N/N anemia  
- XR: erosions, bony decalcifications, osteopenia |
| - Watch for occlusion & aneurysms | | | - NSAIDs, ASA, PT, rest → first line  
- Disease-modifying anti-rheumatic drugs (DMARDs):  
  - Hydroxychloroquine: monitor retinas  
  - Methotrexate: monitor CBC, LFTs  
  - Corticosteroids (oral or intra-articular) – long-term use → cataracts, osteoporosis |
| | | | - NSAIDs (relief in 24-48 h)  
- Corticosteroids |

**Notes:**
- Polymyalgia rheumatica:
  - Aching/stiffness in proximal muscles & inflammation of synovial joints shoulder/hip
  - Sx occur for >1 month
  - Tends to be self-limited
  - Linked to giant cell arteritis
  - Age >50; women > men 2:1

- Reactive arthritis (Reiter’s syndrome):
  - Associated with HLA B-27
  - Asymmetrical oligoarthritis which favors the lower extremities
  - Conjunctivitis
  - Urethritis
  - Exam: “Sausage toes or fingers”
  - Mucocutaneous lesions – small, shallow, painless ulcers on glans penis (circinate balanitis)
  - Painless, papuloquamous eruptions on palms & soles – keratoderma blennorrhagicum

- Rheumatoid arthritis:
  - Chronic, systemic disease – immune complex formation leading to immune response & pannus formation
  - Symmetric synovial inflammation of peripheral joints – PIP, MCP, wrists, elbows, knees & ankles
  - Epidemiology: Women > men 3:1
  - Criteria:
    - Morning stiffness (>1h) for 6 weeks
    - Swelling (soft tissue) of 3+ joints – DIP & lower back not involved
    - Swelling (soft tissue) of hand joints (PIP, MCP or wrist)
    - Symmetrical swelling (soft tissue)
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    - Serum rheumatoid factor positive

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  - Hydroxychloroquine: monitor retinas
  - Methotrexate: monitor CBC, LFTs
  - Corticosteroids (oral or intra-articular) – long-term use → cataracts, osteoporosis
### Systemic lupus erythematosus
- Chronic, multisystem inflammatory disease (autoimmune)
- Linked to HLA-DR2 & HLA-DR3
- Active dz interspersed w/ remissions
- Mainly women (90%), ages 15-40
- Etiology unknown
- Drug-induced lupus – procainamide, hydralazine, isoniazid, quinidine
- In pregnancy anti-Ro can cross the placenta, causing neonatal lupus & heart block
- Fever
- Malar/discoid rash
- Arthritic pain
- Photosensitivity
- Oral ulcers
- Pericarditis
- Proteinuria
- Seizure/psychosis
- Anemia
- Leukopenia
- Positive ANA
- Positive anti-ds DNA & anti-Sm antibodies
- Positive anti-histone in drug-induced SLE
- Elevated ESR
- Antiphospholipid antibodies (hypercoagulable states)
- Corticosteroids
- Chloroquine
- Hydroxychloroquine
- Methotrexate
- NSAIDs

### Scleroderma (systemic sclerosis)
- Disorder of connective tissue
- Fibrosis/thickening of skin, blood vessels, & visceral organs
- Overproduction & accumulation of collagen
- 2 subtypes:
  - Diffuse – changes on extremities, face & trunk, risk of visceral dz early on
  - Limited cutaneous (CREST) – changes to distal extremities & face
- Women > men, ages 20-40
- Joint stiffness, arthralgia, myalgia
- Skin thickening, “hide-bound”
- Raynaud phenomenon
- Systemic: carpal tunnel, pulmonary fibrosis, dysphagia, pericarditis, renal failure
- Positive ANA
- Positive anti-Scl-70 in diffuse; anti-centromere in limited
- Symptomatic – ACEi for renal failure, CCB for Raynaud, D-penicillamine for skin changes
- Immunosuppressive therapy – cyclophosphamide, methotrexate

### Sjogren syndrome
- Chronic, immune-mediated inflammatory disease
- Lymphocytic invasion of exocrine glands (lacrimal/salivary glands)
- Mainly women (90%); avg. ages 30-50
- Linked to B-cell lymphoma
- Dry eyes, dry mouth, oral sores, dry skin
- Polyarthralgia, polyarthritis, dysphagia
- Low-grade fever, malaise
- Schirmer’s test – measures the wetting of standardized tear test strips, which are applied btwn eyeball & lateral inferior lid
- Autoantibodies – ANA, RF, anti-Ro/La
- Salivary gland bx: lymphocyte infiltration
- Artificial tears & saliva
- NSADIs, chloroquine, glucocorticoids (to treat extraglandular manifestations)
<table>
<thead>
<tr>
<th>General Characteristics</th>
<th>Symptoms</th>
<th>Diagnostics</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anemia of chronic disease</strong></td>
<td>- Feeling weak or tired</td>
<td>- Labs (anemia of chronic disease vs. iron deficiency):</td>
<td>- Treat underlying cause</td>
</tr>
<tr>
<td>- Seen in infxn, inflammatory disease, malignancy, renal disease</td>
<td>- Headache</td>
<td></td>
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<tr>
<td></td>
<td>- Paleness</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Shortness of breath</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td><strong>ACD</strong> <strong>Fe def</strong></td>
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<tr>
<td></td>
<td>Serum Fe</td>
<td>-</td>
<td></td>
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<tr>
<td></td>
<td>Transferrin</td>
<td>NL/•</td>
<td></td>
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<tr>
<td></td>
<td>% sat</td>
<td>•</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ferritin</td>
<td>NL/•</td>
<td></td>
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<tr>
<td></td>
<td>BM Fe stores</td>
<td>•</td>
<td></td>
</tr>
<tr>
<td><strong>Aplastic anemia</strong></td>
<td>- Weakness &amp; fatigue</td>
<td>- Pancytopenia</td>
<td>- Bone marrow transplant</td>
</tr>
<tr>
<td>- Acquired or genetic – toxin, radiation, immunologic reason, NSAIDs, chemo, chloramphenicol, EBV, CMV, &amp; parvovirus B19</td>
<td>- Physical exam:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- D/t damage to stem cells</td>
<td>•</td>
<td>Pallor</td>
<td></td>
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<td></td>
<td>•</td>
<td>Purpura</td>
<td></td>
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<tr>
<td></td>
<td>•</td>
<td>Petechiae</td>
<td></td>
</tr>
<tr>
<td><strong>Folate deficiency</strong></td>
<td>- Same as B12 deficiency, however there are <strong>no neurologic abnormalities</strong></td>
<td>- Megaloblastic blood smear – macroovalocytes &amp; hypersegmented neutrophils</td>
<td>- Daily oral folic acid 1mg</td>
</tr>
<tr>
<td>- Macrocytic anemia</td>
<td>- Glossitis, angular cheilosis</td>
<td>- Reduced folic acid levels</td>
<td></td>
</tr>
<tr>
<td>- Most common cause is inadequate dietary intake</td>
<td></td>
<td>- Normal serum B12</td>
<td></td>
</tr>
<tr>
<td>- Alcoholics, persons who do not eat fresh fruits &amp; veggies &amp; those who overcook their food are at risk</td>
<td></td>
<td>- Normal methylmalonic acid</td>
<td></td>
</tr>
<tr>
<td>- Phenytoin, trim-sulf a or sulfasalazine may interfere w/ absorption</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>- Folic acid requirements are • in pregnancy, hemolytic anemia &amp; exfoliative skin disease</td>
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<td></td>
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</tr>
<tr>
<td>- Body stores only 4 mos.</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>- Absorbed in proximal jejunum</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>G6PD deficiency</strong></td>
<td>- Asx until times of stress</td>
<td>- Crisis:</td>
<td></td>
</tr>
<tr>
<td>- Sex-linked</td>
<td></td>
<td>- Smear: spherocytes, blister cells, burr cells</td>
<td></td>
</tr>
<tr>
<td>- Populations include Mediterranean, West African, Middle Eastern, &amp; SE Asian</td>
<td></td>
<td>- G6PD levels</td>
<td></td>
</tr>
<tr>
<td>- Oxidant compounds (stress) lead to hemolysis</td>
<td></td>
<td>- Heinz bodies</td>
<td></td>
</tr>
<tr>
<td>- Stressors of G6PD system: antimalarials, sulfonamides, nitrofurans, Fava beans, infxn, DKA</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
- Hemolytic anemia
  - 3 elements of the RBC:
    - Metabolic machinery
    - Hemoglobin
    - Red cell membrane
  - Coombs negative:
    - Hypersplenism: remove of cellular elements by the spleen
    - Primary cause – idiopathic
    - Secondary causes – acute/chronic infection, chronic inflammatory disease, congestive splenomegaly, myeloproliferative disorders, leukemia/lymphoma
    - Microangiopathic: mechanical disruption of RBC, schistocytes present
    - Etiology: DIC, TTP, HUS, prosthetic heart valves
    - Chemical – dapsone, fresh-water drowning
    - Physical – burns, snake bites
    - Infection – malaria, Babesia, Clostridium perfringens, EBV
  - Coombs positive:
    - Drug-induced - 3 types:
    - Hapten (PCN) – drug on RBC then Ab

- May have Raynaud’s phenomenon w/cold antibody
- Coombs Test (DAT):
  - Test for autoimmune hemolytic anemias
  - Detects significant amounts of IgG & C3 on RBCs

- Summary of drug induced hemolytic anemia:

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Hapten</th>
<th>Immune Complex</th>
<th>Autoantibody</th>
</tr>
</thead>
<tbody>
<tr>
<td>Example</td>
<td>PCN</td>
<td>Quinidine</td>
<td>Methyl dopa</td>
</tr>
<tr>
<td>DAT</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Anti-IgG</td>
<td>+</td>
<td>Rarely +</td>
<td>+</td>
</tr>
<tr>
<td>Anti-C3d</td>
<td>Rarely +</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Indirect Coombs (drug not in system)</td>
<td>–</td>
<td>–</td>
<td>Either</td>
</tr>
<tr>
<td>Indirect Coombs (drug in system)</td>
<td>+</td>
<td>+</td>
<td>No change</td>
</tr>
<tr>
<td>Examples</td>
<td>Cephalothin</td>
<td>HCTZ</td>
<td>L-dopa</td>
</tr>
<tr>
<td></td>
<td>Ampicillin</td>
<td>Antihistamines</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td></td>
<td>Methicillin</td>
<td>Rifampin/INH</td>
<td>Diclofenac</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sulfonamides</td>
<td>α-interferon</td>
</tr>
</tbody>
</table>
- Immune complex (quinine) – Ab bind to drug then RBC, anti-C3 type
- Autoantibody (Aldomet) – autoantibodies against RBC by a drug, hemolysis even after drug discontinued
- Warm antibodies – IgG
  - IgG type – direct Coombs + for IgG, • haptoglobin
  - Causes – primary idiopathic or secondary (lymphoma, CLL, SLE or RA)
- Cold antibodies – IgM
  - Usually IgM – direct Coombs + for C3, IgM triggers complement
  - Causes: viral (EBV) or mycoplasma, lymphoproliferative diseases (lymphoma)
- Congenital:
  - Membrane abnormalities – hereditary spherocytosis or elliptocytosis
  - Enzyme deficiency – G6PD deficiency or pyruvate kinase deficiency
  - Hemoglobinopathies – sickle cell & Hgb C & SC disease

<table>
<thead>
<tr>
<th>Iron deficiency</th>
<th>Sickle cell anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Microcytic, hypochromic</td>
<td>-Frequency 1:400-500 African Americans</td>
</tr>
<tr>
<td>-Causes: Gi blood loss, vaginal blood loss, urinary blood loss, malabsorption, • demand, poor dietary intake</td>
<td>-D/T substitution of valine for glutamic acid at position 6 on β chain</td>
</tr>
<tr>
<td>-Easy fatigability</td>
<td>-Bone, chest &amp; abdominal pain in crisis</td>
</tr>
<tr>
<td>-Fainting</td>
<td>-Splenomegaly</td>
</tr>
<tr>
<td>-Lightheadedness</td>
<td>-Leg ulcers</td>
</tr>
<tr>
<td>-Pallor</td>
<td>-Pulmonary infxn &amp; infarctions</td>
</tr>
<tr>
<td>-Tachycardia</td>
<td>-Strokes</td>
</tr>
<tr>
<td>-Anemia</td>
<td>-CBC w/ diff</td>
</tr>
<tr>
<td>-Leukopenia</td>
<td>-Sickledex</td>
</tr>
<tr>
<td>-Iron deficiency</td>
<td>-Hgb electrophoresis: 25-45% Hgb S, remainder Hgb A, F &amp; A2</td>
</tr>
</tbody>
</table>

- Elemental iron 60mg per day
- Response to therapy:
  - reticulocyte count in 7-10 days
  - hemoglobin in 2-3 weeks
  - Normal hemoglobin in 2 months

- CBC w/ diff
  - Sickledex
  - Hgb electrophoresis: 25-45% Hgb S, remainder Hgb A, F & A2
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Description</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thalassemia</strong></td>
<td>- Defect in globin polypeptide chain</td>
<td>- Genetic counseling, avoid iron</td>
</tr>
<tr>
<td></td>
<td>- Causes absent or * synthesis of the affected globin chain</td>
<td>- Transfusions</td>
</tr>
<tr>
<td></td>
<td>- 2 types: *α &amp; *β</td>
<td>- Splenectomy</td>
</tr>
<tr>
<td></td>
<td>- *β thalassemia – 2 types:</td>
<td>- Iron chelation</td>
</tr>
<tr>
<td></td>
<td>- Minor</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Major (Cooley's anemia) – severe</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- *α thalassemia:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Silent carrier, 1 gene inactive → no sx</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Trait, 2 genes inactive → no sx</td>
<td></td>
</tr>
</tbody>
</table>
|                   | - 
|                   |   - Hemoglobin H disease, 3 genes inactive → splenomegany                                                |                                              |
|                   |   - Hydrops fetalis, 4 genes inactive, death in utero or neonatally                                      |                                              |
|                   | - \*O thalassemia:                                                                                    |                                              |
|                   |   - Cerebral palsy, mental retardity, growth retardation                                                  |                                              |
|                   |   - Bone marrow hypertrophy, iron deficiency                                                             |                                              |
|                   |   - Hgb = 6 g/dL, \*MCV                                                                                   |                                              |
|                   | - Moderate anemia, level Hgb F                                                                                 |                                              |
| **B12 deficiency**| - Macrocytic anemia                                                                                     | - Oral replacement once initial correction has occurred                                      |
|                   | - Rare but seen in vegans, persons \*w/ \*hx of abdominal surgery                                       |                                              |
|                   | - Gastrectomy, resection                                                                               |                                              |
|                   | - Essential for normal nuclear maturation                                                              |                                              |
|                   | - Diet is only source of intake                                                                        |                                              |
|                   | - Total body content 2-5 mg                                                                              |                                              |
|                   | - Body stores last years                                                                               |                                              |
|                   | - Absorption in terminal ileum                                                                          |                                              |
|                   | - Neurologic (tingling in feet, gait abnormalities)                                                     |                                              |
|                   | - Affects pyramidal tracts & posterior column                                                           |                                              |
|                   | - Glossitis                                                                                             |                                              |
|                   | - Anorexia                                                                                             |                                              |
|                   | - Diarrhea                                                                                             |                                              |
|                   | - Loss of vibratory sensation                                                                          |                                              |
|                   | - Megaloblastic blood smear – macrocytic/ovalocytes & hypersegmented neutrophils                       |                                              |
| **Clotting factor disorders** | - Factor VIII deficiency:                                                                               |                                              |
|                   |   - Hemophilia A, X-linked disorder                                                                     |                                              |
|                   |   - Categorized by factor activity level                                                               |                                              |
|                   |   - Bleeding into muscles, joints, \& soft tissue; microscopic hematuria & cranial bleed                |                                              |
|                   |   - Seen primarily in men \*1:10,000                                                                   |                                              |
|                   | - Factor IX deficiency:                                                                                |                                              |
|                   |   - Hemophilia B, X-linked disorder                                                                    |                                              |
|                   |   - Bleeding into muscles, joints, \& soft tissue; microscopic hematuria & cranial bleed                |                                              |
|                   |   - Seen primarily in men \*1:10,000                                                                   |                                              |
|                   | - Factor XI deficiency:                                                                               |                                              |
|                   |   - Autosomal recessive                                                                                 |                                              |
|                   | - Normal PT, \*PTT, normal thrombin time                                                                |                                              |
|                   | - Normal platelets                                                                                      |                                              |
|                   | - Reduced Factor VIII activity                                                                         |                                              |
|                   |   - Factor IX deficiency:                                                                               |                                              |
|                   |     - Normal PT, \*PTT, normal thrombin time                                                            |                                              |
|                   |     - Normal platelets                                                                                   |                                              |
|                   |     - Reduced levels factor XI                                                                          |                                              |
|                   |     - Factor XI activity                                                                                 |                                              |
|                   |     - Prolonged PTT                                                                                      |                                              |
|                   | - Factor XI disorder                                                                                     |                                              |
|                   |   - FFp or cryoprecipitates                                                                              |                                              |
|                   |   - \*e-aminocaproic acid (EACA) w/ or without DDAVP                                                     |                                              |
|                   | - Prolonged bleeding time                                                                               |                                              |
|                   | - Prolonged PTT d/t \*level factor VIII                                                                   |                                              |
- Ashkenazi Jews
- Mild bleeding (post-op bleeding)
  - Von Willebrand’s disease:
  - Most common severe congenital bleeding disorder – affects 1% of the population
  - Autosomal dominant pattern
  - Problem w/ platelet adhesion – defect in vWF
  - 3 types:
    - Type 1 (1-2%): 80% of all vWF cases, partial deficiency, bleeding time may be normal
    - Type 2: multiple subtypes – 2A, 2B, 2N, & 2M
    - Type 3 (1:250,000): severe deficiency, typically dx in childhood

<table>
<thead>
<tr>
<th>Factor VII or XI level (U/dL)</th>
<th>Clinical picture</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>Severe, spontaneous bleeding</td>
<td>70% 50%</td>
</tr>
<tr>
<td>1-5</td>
<td>Moderate bleeding w/ minimal trauma or surgery</td>
<td>15% 30%</td>
</tr>
<tr>
<td>5-30</td>
<td>Mild bleeding w/ minimal trauma or surgery</td>
<td>15% 20%</td>
</tr>
</tbody>
</table>

Hypercoagulable states
- Abnormality of blood coagulation that • the risk of thrombosis
- Causes:
  - Congenital: factor V Leiden & prothrombin G20210A
  - Acquired: SLE (antiphospholipid syndrome), heparin-induced, paroxysmal nocturnal hemoglobinuria, sickle cell dz, cancer, nephrotic syndrome, pregnancy, obesity
- DVT – usually in the legs, characterized by pain, swelling & redness of limb
- PE – SOB, chest pain, palpitations
- CBC, PT, PTT, lupus anticoagulant
- No specific treatment unless caused by underlying medical illness
- Warfarin – risks vs. benefits
- LMWH for pregnant women at risk

Idiopathic thrombocytopenic purpura
- Most common consumptive thrombocytopenia in adults
- Both adult & childhood ITP
- More common in women 3:1 ages 20-30
- Can be associated w/ other autoimmune diseases – recent viral infxn in children
- Pathogenesis:
  - Results from platelet sensitization by auto-reactive antibodies → react to glycoprotein IIb/IIIa complex
  - • amounts of platelet-associated IgG, leading to shortened survival d/t phagocytosis by splenic macrophages
- Petechiae, purpura, gingival bleeding & menorrhagia
- No splenomegaly
- Thrombocytopenia – may be <10,000
  - megakaryocytes in bone marrow & large platelets in peripheral blood
- Prednisone
- Splenectomy
- Danazol
- High dose IV immunoglobulin – will • platelet count in 1-5 d but lasts only 1-2 weeks

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Childhood (Acute)</th>
<th>Adult (Chronic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Childhood</td>
<td>Women ages 20-50</td>
</tr>
<tr>
<td>Previous infxn</td>
<td>Common</td>
<td>Usually not</td>
</tr>
<tr>
<td>Platelet count</td>
<td>&lt;20,000</td>
<td>30,000-80,000</td>
</tr>
<tr>
<td>Duration of thrombocytopenia</td>
<td>Few weeks</td>
<td>Months to years</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Abrupt</td>
<td>Insidious</td>
</tr>
<tr>
<td>Spontaneous remission</td>
<td>Occurs in most</td>
<td>Rare</td>
</tr>
<tr>
<td>Condition</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>----------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Thrombotic thrombocytopenic purpura</td>
<td>- Seen in previously healthy young women; often fatal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- A platelet consumption disorder</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Five classic findings:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Thrombocytopenia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Microangiopathic hemolytic anemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Fever</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Neuro signs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Renal involvement: mild</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- insufficiency, hematuria</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Labs: schistocytes, thrombocytopenia, elevated LDH, Coombs negative</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Plasma exchange w/ FFP</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Steroids &amp; ASA</td>
<td></td>
</tr>
<tr>
<td>Acute lymphocytic leukemia</td>
<td>- Most common in children (peak age 4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Over 70% are cured</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Anemia, bruising, infxn</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- CNS involvement</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Lymphadenopathy, splenomegaly</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Labs: blasts &gt;30%, elevated WBC, anemia, bone marrow for dx</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Chemo – daunorubicin, vincristine, prednisone</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- CNS tx – methotrexate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Transplant</td>
<td></td>
</tr>
<tr>
<td>Chronic lymphocytic leukemia</td>
<td>- Cause unknown</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- w/ age &gt;50</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Men &gt; women</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Mainly B-cell lymphocytes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- risk of bacterial infxn</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Usually ask</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Splenomegaly, lymphadenopathy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Slow, indolent course</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Labs: lymphocytosis, smudge cells</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Chlorambucil to reduce WBC count</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Chemo - fludarabine</td>
<td></td>
</tr>
<tr>
<td>Acute myelogenous leukemia</td>
<td>- More common in adults</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Risk factors: radiation, chemicals, prior chemo</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Low survival rate even w/ tx</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Classification: FAB classes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Fatigue, anorexia, dyspnea</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Lymphadenopathy, splenomegaly</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Labs: blasts &gt;30%, Auer rods, elevated WBC, anemia, bone marrow for dx</td>
<td></td>
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<tr>
<td></td>
<td>- Chemo – cytosine arabinoside &amp; daunorubcin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- No CNS tx</td>
<td></td>
</tr>
<tr>
<td>Chronic myelogenous leukemia</td>
<td>- A myeloproliferative disorder</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Typical patient is middle-age</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Phases:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Chronic – lasts 3-5 yrs., elevated WBC but few blasts</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Blastic – conversion to acute leukemia, blasts fill marrow</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Fatigue, lethargy, SOB, wt.-loss, easy bruising</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Splenomegaly</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Labs: Philadelphia chromosome positive, anemia, thrombocytosis,</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- elevated LDH, low LAP score, differential</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- reveals entire WBC cell line</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Imatinib</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- α interferon (rarely used)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Etiology: EBV?</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Non-Hodgkin’s:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Most commonly B lymphocytes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- More common than Hodgkin’s</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Incidence   w/ age</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Moderate predominance in men</td>
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<tr>
<td></td>
<td>- Perifollicular B lymphocytes – small lymphocytic leukemia &amp;</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- chronic lymphocytic leukemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Hodgkin’s:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Painless, enlarged lymph nodes, lymphadenopathy, splenomegaly,</td>
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<tr>
<td></td>
<td>- hepatomegaly</td>
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</tr>
<tr>
<td></td>
<td>- B sx: fever, night sweats, wt. loss</td>
<td></td>
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<tr>
<td></td>
<td>- Non-Hodgkin’s:</td>
<td></td>
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<tr>
<td></td>
<td>- Present w/ lymphadenopathy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Can involve skin, GI tract &amp; CNS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Splenomegaly common</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- B sx: fever, night sweats, wt. loss</td>
<td></td>
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<tr>
<td></td>
<td>- Staging for both:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Hodgkin’s:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Labs: classic Reed-Sternberg cells, CT chest/abd/pelvis, bone marrow,</td>
<td></td>
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<tr>
<td></td>
<td>- lymph node biopsy</td>
<td></td>
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<td></td>
<td>- Non-Hodgkin’s:</td>
<td></td>
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<tr>
<td></td>
<td>- CT chest/abd/pelvis, bone marrow, lymph node biopsy</td>
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<td></td>
<td>- Hodgkin’s:</td>
<td></td>
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<tr>
<td></td>
<td>- Depends on stage</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Chemotherapy – Adriamycin, bleomycin, vincristine</td>
<td></td>
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<tr>
<td></td>
<td>- Radiation therapy</td>
<td></td>
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<td></td>
<td>- Non-Hodgkin’s:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Chemotherapy – responds well but relapse is common</td>
<td></td>
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<tr>
<td></td>
<td>- Radiation therapy</td>
<td></td>
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<tr>
<td></td>
<td>- Stem cell transplant</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Monoclonal antibodies – rituximab</td>
<td></td>
</tr>
<tr>
<td>Germinal center of lymphoid follicle – follicular lymphoma, large cell lymphoma, Burkitt’s lymphoma</td>
<td>Stage 1: single lymph node region or structure</td>
<td></td>
</tr>
<tr>
<td>Mantle zone of lymphoid follicle – mantle cell lymphoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marginal zone of lymphoid follicle – monocytoïd lymphoma &amp; mucosa-associated lymphoid tissue (MALT) lymphoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2: 2 or more lymph node regions on same side of diaphragm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 3: lymph node regions or structures on both side of diaphragm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 4: involvement of other organs (liver, bone marrow, CNS, etc.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interferon</td>
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</tr>
</tbody>
</table>

### Multiple myeloma

- **Plasma cell dyscrasia**
- **Monoclonal production of IgG, IgA, IgD, IgE or monoclonal light chains** (Bence Jones proteins)
- **-** risk w/ age, African Americans
- **-** Men = women
- **-** Risk factors: radiation exposure & chemical exposure (asbestos)
- **-** Infxn w/ encapsulated organisms is common – *pneumococcus, Haemophilus*

- Early: fatigue, weakness, wt. loss
- Later: bone pain, anemia, renal function changes, neuro abnormalities
- Exam: skeletal pain on palpation, bruising, wasting, neuro deficits

- **Anemia – Rouleaux on smear**
- **-** sedimentation rate
- **-** plasma cells (>10% in bone marrow)
- **-** serum total protein – β2-microglobulin
- **Monoclonal spike on SPEP**
- Bence Jones protein on UPEP
- Renal insufficiency
- Calcium level elevated
- **XR: classic punched-out lesions, skull/ribs/spine/pelvis, compression fx later**

- Supportive: transfusions, pain control
- Chemo: melphalan & prednisone
<table>
<thead>
<tr>
<th><strong>General Characteristics</strong></th>
<th><strong>Symptoms</strong></th>
<th><strong>Diagnosis</strong></th>
<th><strong>Treatment</strong></th>
</tr>
</thead>
</table>
| **Benign prostatic hyperplasia** | -Noncancerous abnormal hyperplasia of the prostate gland  
-Onset 4th decade of life  
-Sx can start around age 50; significant problems onset age 60  
-Etiology – related to testosterone conversion to dihydrotestosterone by 5α-reductase  
-Rapid onset manifestations before age 50 – filling sx w/o voiding sx are red flags & suggest other dx | -Blockage in passage of urine,  
-frequency, urgency, nocturia, hesitancy, hematuria, dripping or dribbling, weak stream  
-Exam: rubbery, enlarged, firm prostate | -Cystoscopy  
-Pressure-flow studies | -5-α-reductase inhibitors (finasteride)  
-SEs: sexual or ejaculatory dysfunction  
-α-1-adrenergic inhibitors (doxazosin, terazosin)  
-SEs: orthostatic hypotension, dizziness, retrograde ejaculation  
-Surgery: transurethral prostatectomy (TURP) |
| **Congenital abnormalities** | -Renal dysgenesis:  
- D/t immature, undifferentiated renal tissue  
-Some cases the normal # of nephrons is not compatible w/ life once the child reaches a critical body size  
-Oligomeganephronia – presence of only a few large glomeruli  
-Cystic dysplasias – presence of renal cysts  
-Polycystic kidney disease:  
- Autosomal recessive  
-Kidneys nonfunctional in utero  
-ADPKD1/ADPKD2 are for 80% & 10% of cases respectively  
-Medullary cystic disease:  
- Cysts of varying sizes in the renal medulla w/ tubular & interstitial nephritis  
-Reflex nephropathy:  
- Retrograde flow of urine from the bladder into the ureter  
-Complications: renal insufficiency & HTN | -PCKS:  
- Potter facies  
- HTN  
-Medullary cystic disease:  
- Renal failure  
- Signs of tubular dysfunction -  
- concentration, Fanconi syndrome | -U/S  
-Reflex nephropathy:  
- Ultrasound  
- Confirmed by a voiding cystourethrogram | -Surgery for chronic reflux, but most spontaneously resolve |
| **Cryptorchidism** | -A testis which does not remain at the bottom of scrotum after cremasteric muscle has been fatigued by overstretching  
-Can cause infertility  
-Thermal effects – predominates after 2 yrs. old, risk of testicular cancer  
-Risk of testicular torsion | -Undescended testis | -U/S, CT, MRI (50% accurate in demonstrating intra-abdominal testes) | -Hormonal therapy – GnRH & hCG  
-30-50% success rate, most helpful in low undescended testes, 25% relapse rate  
-Orchidopexy  
-Goals:  
- Prevent thermal damage  
- Repair associated inguinal hernia |
**Majority** will descend by age 3 mos. – associated w/ gonadotropin surge responsible for germ cell maturation. History – maternal steroid use, family hx

**Erectile dysfunction**
- The consistent inability to attain or maintain a sufficiently rigid penile erection for sexual performance
- Typically d/t organic causes – CV dz, DM, androgen deficiency or 2/2 meds (HCTZ, nifedipine, propranolol, fluoxetine, amoxapine, clorazepate, phenytoin, dimenhydrinate, flutamide, alcohol, marijuana, & cocaine)

**Hydrocele**
- Fluid btwn 2 layers of tunica vaginalis
- Must rule out testicular cancer

**Varicocele**
- Engorgement of internal spermatic veins above the testis – abnormal dilatation of the pampiniform plexus
- Common cause of subfertility in men
- For age <10 yrs. or sudden onset right-side varicocele think retroperitoneal malignancy
- For sudden-onset left side varicocele think renal cell carcinoma

**Incontinence**

<table>
<thead>
<tr>
<th></th>
<th>Stress</th>
<th>Urge</th>
<th>Overflow</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amount of loss</strong></td>
<td>Small</td>
<td>Large</td>
<td>Small</td>
</tr>
<tr>
<td><strong>Duration of loss</strong></td>
<td>Brief</td>
<td>Moderate</td>
<td>Continuous</td>
</tr>
<tr>
<td><strong>Associated eve</strong></td>
<td>Cough</td>
<td>None</td>
<td>Change in position</td>
</tr>
<tr>
<td></td>
<td>Laugh</td>
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<tr>
<td></td>
<td>Sneeze</td>
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</tr>
<tr>
<td><strong>Associated symptoms</strong></td>
<td>None</td>
<td>Urgency</td>
<td>Fullness</td>
</tr>
<tr>
<td></td>
<td>No urine loss at night</td>
<td>Nocturia</td>
<td>Pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Frequency</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>Cystocele</td>
<td>Loss of bladder inhibition</td>
<td>Obstruction</td>
</tr>
<tr>
<td></td>
<td>Uretherocele</td>
<td>Loss of neurologic control</td>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Kegel exercises</td>
<td>Anticholinergic</td>
<td>Self-catheterization</td>
</tr>
<tr>
<td></td>
<td>Estrogen replacement</td>
<td></td>
<td>Cholinergic meds</td>
</tr>
<tr>
<td></td>
<td>Surgery</td>
<td></td>
<td>α-blockers</td>
</tr>
</tbody>
</table>

**Inhibit voiding**
Bladder relaxants
- Antispasmodics: oxybutynin

**Promote voiding**
Bladder contractions
- Cholinergics: bethanechol
| Nephro/ urolithiasis          | -Peak incidence ages 30-50; men > women  
|                              | -Formed from calcium oxalate, calcium phosphate, uric acid & cysteine  
|                              | -Size:  
|                              | • <5 mm: pass w/o difficulty  
|                              | • 5-10 mm: pass 50% of time  
|                              | • >10 mm: obstruction  
|                              | -Risk – calcium intake, purine-containing foods, oxalate, UTI, gout, fluid status  
|                              | -Calcium oxalate – most common, from hypercalcemia, sarcoidosis, hyperparathyroidism, hypercalcuria, idiopathic  
|                              | -Struvite – UTI, alkaline urine  
|                              | -Uric acid – gout, high purine diets  
|                              | -Cystine - genetic  
| Paraphimosis                  | -Retracted foreskin develops a fixed constriction proximal to the glans  
| Phimosis                      | -Fibrous constriction of the foreskin preventing retraction  
| Cystitis                      | -Incidence • w/ sexual activity, DM, self-catheterization  
|                              | -Cystitis: infxn of bladder  
|                              | -Etiology:  
|                              | • *E. coli* >80% of cases  
|                              | • Enterobacter  
|                              | • Klebsiella  
|                              | • Proteus  
|                              | • Pseudomonas  
|                              | • *Staphylococcus*  
| Epididymitis                  | -Inflammation of the epididymis  
|                              | -Seen typically in sexually active adults  
|                              | -D/t retrograde infxn from urethra  
|                              | -Etiology:  
|                              | • Young boys – anatomic abnormalities, *H. influenza* type B  
| | Anticholinergics: Pro-Banthine  
| | Tricyclics: imipramine  
| Vesical neck contractions     | -Sudden onset flank pain, awakens pt at night, pain waxes & wanes, radiation to groin, scrotum/vulva  
|                              | -Stone in bladder develops frequency, urgency, dysuria  
|                              | -Hematuria, N/V  
|                              | -Exam: CVA tenderness, afebrile, soft abdomen  
| | Vesical neck relaxants       | -Hematuria, pyuria  
| | Alpha adrenergic: ephedrine   | -XR: KUB – cystine & uric acid stones not visible on KUB  
| | | -Helical CT test of choice  
| Progestrone                   | -Pain control – narcotics  
|                              | -Hydration  
|                              | -Diet: • protein & sodium intake  
|                              | -Metabolic evaluation  
| | Nephro/ urolithiasis          | -Penis distal to constricting foreskin may become swollen & painful  
| | | -Manual reduction  
| | | -Circumcision  
| | Paraphimosis                  |  
| | | -Circumcision  
| | Phimosis                      |  
| | Cystitis                      | -Dysuria, frequency, urgency, suprapubic discomfort, foul-smelling, cloudy urine, hematuria  
| | | -Fever & chills more common in pyelonephritis  
| | | -May be asx in DM & elderly  
| | Epididymitis                  | -Nitrites, leukocyte esterase & bacteria +  
| | | -WBC >5/HPF, RBCs  
| | | -Culture  
| | | -7 d plan for pregnant, DM, elderly, & recurrent infxn  
| | | -Fluoroquinolones  
| | | -Trim/sulfa  
| | | -Nitrofurantoin – safe in pregnancy  
| | | -Cephalexin  
| | | -Pyridium – turns urine orange (analgesic)  
| | Cystitis                      | -Urethral smear  
| | | -UA/UC  
| | Epididymitis                  | -Chlamydia/GC: doxycycline, ceftriaxone  
| | | -TMP/sulfa  
| | | -Fluoroquinolones  
<p>|</p>
<table>
<thead>
<tr>
<th>Males &lt;35 – chlamydia, &gt;35 - coliforms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammation of the testicle</td>
</tr>
<tr>
<td>Viral etiology most common – mumps</td>
</tr>
<tr>
<td>presents about 5 d after the parotitis</td>
</tr>
<tr>
<td>May result in atrophy</td>
</tr>
</tbody>
</table>

**Orchitis**

- Acute bacterial prostatitis type I:
  - Etiology: *E. coli*, *pseudomonas*, *seratia*, *klebsiella*, *proteus*
- Chronic bacterial prostatitis type II:
  - The elderly & those w/ recurrent UTI
  - Chronic inflammation 2/2 bacteria
- Chronic non-bacterial prostatitis (type III):
  - Autoimmune disease or viral cause – chlamydia or Ureaplasma
- Prostatodynia type IV:
  - Chronic prostatitis sx w/o signs of prostatic inflammation

**Prostatitis**

- Acute bacterial prostatitis type I:
  - Fever, dysuria, suprapubic or perineal pain, malaise
  - Exam: febrile, prostate tenderness, enlarged
- Chronic bacterial prostatitis type II:
  - Dysuria, irritative voiding discomfort, not as much pain
  - Exam: prostate findings will vary
- Chronic non-bacterial prostatitis (type III):
  - Pelvic pain, urinary sx, pain during or after ejaculation
  - Exam: prostate findings vary

**Pyelonephritis**

- Infection of the parenchyma of the kidney, often follows UTI
- Etiology: *E. coli*, *proteus*, *klebsiella*, *enterobacter*, *pseudomonas*

**Urethritis**

- Gonococcal Urethritis
  - Venereal disease – *Neisseria gonorrhoea*
  - Gram – diplococci
  - Incubation period 2-8 d
  - Complications – epididymitis/salpingitis/PID, disseminated
- Chlamydial Urethritis
  - Cause: Chlamydia trachomatis

**Bladder carcinoma**

- Men > women 3:1
- Mean age 70 yrs., rare before 40
- Risk factors: smoking, occupational exposure (rubber), chronic infxn

**Sx relief w/ recumbency & analgesics**

**Prostatitis**

- Acute bacterial prostatitis type I:
  - UA – pyuria, + culture, prostate massage not recommended
  - PSA • but returns to normal post tx
- Chronic bacterial prostatitis type II:
  - UA negative, prostate massage w/ secretions showing • WBC & culture +
- Chronic non-bacterial prostatitis (type III):
  - UA negative, prostate secretions • WBC but culture is negative
  - Prostatodynia type IV:
  - Prostatic secretion note WBC, culture is negative

**Pyelonephritis**

- Fever, chills, flank/back pain, N/V, anorexia, dysuria, urgency
- Exam: CVA tenderness, fever, toxic appearing
- UA w/ culture
- WBC casts
- Proteinuria
- Nitrite leukocyte esterase +
- Blood culture
- CBC w/ diff

**Urethritis**

- Gonorrhea:
  - Milky to yellow purulent discharge
  - Painful/burning urination
- Chlamydia:
  - May have clear to cloudy urethral discharge
  - Painful, burning urination
  - Men – dysuria, urinary frequency, mental itching, urethral discharge
  - Women – vaginal discharge, lower abdominal pain, dyspareunia, cervical ectopy

**Bladder carcinoma**

- Cystoscopy w/ biopsy

**Chlamydial Urethritis**

- Culture of discharge = GOLD STANDARD
- Nucleic acid amplification

**Bladder carcinoma**

- Depends on staging
- Radical cystectomy w/ pelvic lymphadenectomy
- Chemotherapy combined w/ surgery

**Prostatitis**

- Acute bacterial prostatitis type I:
  - Fluoroquinolones, ampicillin/gentamicin, TMP/sulfa
  - Abx for 1 mo. to avoid chronic dz
- Chronic bacterial prostatitis type II:
  - TMP/sulfa, fluoroquinolones – treat for 6-12 wks.
- Chronic non-bacterial prostatitis (type III):
  - Doxycycline or erythromycin
  - α-blocker therapy
- Prostatodynia type IV:
  - Pain control, no abx

**Pyelonephritis**

- Inpatient: children, pregnant, septic pts
- Abx: fluoroquinolones, ceftriaxone

**Urethritis**

- Gonorrhea:
  - Ceftriaxone/cefixime/cefpodoxime
  - Treat also for chlamydia (doxycycline/ azithromycin)
<table>
<thead>
<tr>
<th>Prostate carcinoma</th>
<th></th>
<th></th>
<th>RT in early-stage disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most commonly ages &gt;65</td>
<td>Urinary obstruction</td>
<td>PSA elevated &gt;4 ng/mL</td>
<td>Transurethral resection</td>
</tr>
<tr>
<td>Mets are common to the bone</td>
<td>Bone pain &amp; LE edema in advanced disease</td>
<td>-CT/MRI for staging</td>
<td>Radical prostatectomy</td>
</tr>
<tr>
<td>Histology: adenocarcinoma</td>
<td>Impotence, hematuria, nocturia</td>
<td>Transrectal U/S w/ biopsy for dx</td>
<td>-RT</td>
</tr>
<tr>
<td>Risk factors: advancing age, + family hx, African American</td>
<td>PE: DRE prostate enlarged, nodules hard</td>
<td></td>
<td>Hormone therapy (androgen deprivation) – flutamide (Eulexin)</td>
</tr>
<tr>
<td>Complications: pathologic fx &amp; spinal cord compression</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Renal cell carcinoma</th>
<th>-Men &gt; women 3:1, • incidence in African Americans</th>
<th>-Flank pain, abdominal mass, hematuria (classic triad)</th>
<th>-U/S, CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ages 50-70</td>
<td>-Wt. loss, fatigue, anorexia</td>
<td></td>
<td>-Surgery</td>
</tr>
<tr>
<td>Risk factors: obesity, HTN, smoking, diuretic use</td>
<td>-Associated paraneoplastic syndrome is common – hypercalcemia, HTN</td>
<td></td>
<td>-Immunomodulatory w/ interleukin-2 in metastatic dz</td>
</tr>
<tr>
<td>Complications: pathologic fx &amp; spinal cord compression</td>
<td></td>
<td></td>
<td>-Resistant to radiation &amp; chemo</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Testicular carcinoma</th>
<th>-Painless, solid testicular swelling</th>
<th>-Testicular U/S: hypoechoic mass</th>
<th>USPSTF recommends against screening for testicular cancer in adolescent or adult males</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young men ages 20-40</td>
<td>Asx</td>
<td>Elevated beta-HCG</td>
<td>-Orchectomy – treatment of choice0</td>
</tr>
<tr>
<td>Risk factors: cryptorchidism</td>
<td>Associated w/ hydrocele (10%)</td>
<td>Elevated alpha fetoprotein</td>
<td>-Chemo/radiation</td>
</tr>
<tr>
<td>Complications: pathologic fx &amp; spinal cord compression</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Wilms tumor</th>
<th>-Nephroblastoma</th>
<th>-Abdominal CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignant tumor of kidney occurring in children</td>
<td>Asx abdominal mass, may have fever</td>
<td></td>
</tr>
<tr>
<td>Peak age 2-5</td>
<td>Hematuria in 20-30%</td>
<td></td>
</tr>
<tr>
<td>May be associated w/ cryptorchidism</td>
<td>HTN: d/t obstruction of renal artery</td>
<td></td>
</tr>
<tr>
<td>Complications: pathologic fx &amp; spinal cord compression</td>
<td>PE:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Asx abdominal mass extending from flank toward midline (most common presentation)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anemia d/t hemorrhage in tumor</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acute renal failure</th>
<th>-Abrupt • in creatinine – • by 0.3 mg/dl or 50%</th>
<th>-Anuria &lt;100 mL = sign of obstruction</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>3 major classes:</td>
<td>-Based on severity of disease</td>
<td>-BUN, creatinine elevated, urine sediment</td>
<td>-Treat cause, remove toxic drugs, fluid balance</td>
<td></td>
</tr>
<tr>
<td>Prerenal (perfusion problem)</td>
<td>-Fatigue, dizziness, swelling, • urine output</td>
<td>-Renal U/S</td>
<td>-Low sodium, low potassium diet</td>
<td></td>
</tr>
<tr>
<td>Shock, dehydration, • cardiac output, renal arterial obstruction</td>
<td>-Exam: orthostatic hypotension, tachycardia, rales, JVD, edema, bladder distention</td>
<td></td>
<td>-Dialysis</td>
<td></td>
</tr>
<tr>
<td>Renal (glomerular, tubular or interstitial problem)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ishemic/inflammation, nephritis, vasculitis, HUS (E. coli), toxins/drugs (aminoglycosides, NSAIDs, contrast material)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postrenal (obstruction)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasms, stones, prostate disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Index</th>
<th>Prerenal</th>
<th>Renal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine specific gravity</td>
<td>&gt;1.020</td>
<td>&lt;1.010</td>
</tr>
<tr>
<td>Plasma BUN/creatinine</td>
<td>&gt;20</td>
<td>10-15</td>
</tr>
<tr>
<td>Urine osmolarity</td>
<td>&gt;500</td>
<td>&lt;350</td>
</tr>
<tr>
<td>Sodium fraction</td>
<td>&lt;1%</td>
<td>&gt;2%</td>
</tr>
</tbody>
</table>
### Chronic kidney disease

- Etiologies: diabetes #1, HTN #2

- Multiple system failure:
  - Metabolic – hyperkalemia, hyperphosphatemia, hyperuricemia, hyperglycemia, hypocalcemia, metabolic acidosis
  - CV – HTN, CHF, pericarditis
  - Hematology – anemia
  - Dermatology – pruritus
  - GI – anorexia, N/V, diarrhea
  - Neurologic – encephalopathy, neuropathy
  - Skeletal – osteomalacia, osteoporosis

- FE \( \text{Na} \) = \((\text{U} \text{Na} / \text{P} \text{Na})/(\text{U} \text{Cr} / \text{P} \text{Cr}) \times 100\)

### Stage Reduction GFR

<table>
<thead>
<tr>
<th>Stage</th>
<th>Reduction</th>
<th>GFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Slight</td>
<td>&gt;90 mL/min but presence of markers of kidney disease</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>60-89 mL/min w/ presence of markers of kidney damage</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>30-59 mL/min</td>
</tr>
<tr>
<td>4</td>
<td>Severe</td>
<td>15-29 mL/min</td>
</tr>
<tr>
<td>5</td>
<td>Established</td>
<td>&lt;15 mL/min or permanent replacement therapy</td>
</tr>
</tbody>
</table>

### Glomerulonephritis

- Damage of the renal glomeruli
- D/t deposits of inflammatory protein
- 2/2 an autoimmune event, circulation antibodies or vasculitis
- Etiologies:

<table>
<thead>
<tr>
<th>Vascular dz</th>
<th>Glomerular dz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wegener granulomatosis</td>
<td>Goodpasture syndrome</td>
</tr>
<tr>
<td>Churg-Strauss syndrome</td>
<td>Postinfectious glomerulonephritis</td>
</tr>
<tr>
<td>Henoch-Schonlein purpura</td>
<td>IgA nephropathy</td>
</tr>
<tr>
<td>Polyarteritis nodosa</td>
<td>SLE</td>
</tr>
<tr>
<td>TTP</td>
<td>Idiopathic rapidly progressive glomerulonephritis</td>
</tr>
<tr>
<td>Goodpasture's syndrome</td>
<td>Alport syndrome</td>
</tr>
<tr>
<td>Lupus nephritis</td>
<td></td>
</tr>
</tbody>
</table>

### Disease S&S Serology Treatment Notes

<table>
<thead>
<tr>
<th>Disease</th>
<th>S&amp;S</th>
<th>Serology</th>
<th>Treatment</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poststreptococcal</td>
<td>Children Oliguric Edema</td>
<td>ASO titer + complement</td>
<td>Symptomatic</td>
<td>Occurs after pharyngitis &amp; impetigo</td>
</tr>
<tr>
<td>IgA nephropathy</td>
<td>Microscopic or gross hematuria</td>
<td>IgA levels elevated NL complement</td>
<td>Corticosteroids</td>
<td>Associated w/ URI or flu-like illness</td>
</tr>
<tr>
<td>Wegener's granulomatosis</td>
<td>Fever Malaise Wt. loss</td>
<td>ANCA + NL complement</td>
<td>Corticosteroids Cyclophosphamide</td>
<td>Respiratory tract sx common</td>
</tr>
<tr>
<td>Goodpasture's syndrome</td>
<td>Hemoptyis</td>
<td>Anti-GMB +</td>
<td>Plasma exchange Corticosteroids</td>
<td></td>
</tr>
<tr>
<td>Lupus nephritis</td>
<td></td>
<td>ANA +</td>
<td>Corticosteroids</td>
<td></td>
</tr>
<tr>
<td>Cryoglobulinemia</td>
<td>DM &amp; HTN</td>
<td>Amyloid</td>
<td>• complement</td>
<td>Cyclophosphamide</td>
</tr>
<tr>
<td>------------------</td>
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</tr>
</tbody>
</table>

**Hydronephrosis**
- Distention of renal calyces & pelvis of one or both kidneys by urine
- Result of urinary blockage that occurs at the level of the kidney, ureters, bladder or urethra
- Occurs in up to 80% of pregnant women
- Ureteropelvic junction obstruction is a common congenital abnormality leading to this condition
- Severe pain that radiates to lower abdomen, testicles or labia
- Flank pain w/ urination is pathognomonic for vesicoureteral reflux
- Exam: distention of kidney or bladder
- Ultrasound
- Azotemia may be present – impaired excretory function of sodium, urea, & water
- Bladder catheterization – if diuresis occurs then obstruction is below bladder neck
- UA – pyuria, hematuria, proteinuria or bacteruria
- BUN/creatinine
- Spontaneous resolution in most cases
- Frequent voiding or catheterization
- Anticholinergics for neurogenic bladder
- Hydronephrosis + infxn = EMERGENCY → prompt drainage w/ retrograde stent or percutaneous nephrostomy

**Nephrotic syndrome**
- Comprised of glomerular proteinuria (> 3.5 g/day), hypoalbuminemia, hyperlipidemia, & edema
- Causes:
<table>
<thead>
<tr>
<th>Primary renal dz</th>
<th>Secondary renal dz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal glomeruloneph.</td>
<td>Post-strep glomeruloneph.</td>
</tr>
<tr>
<td>Focal glomerulosclerosis</td>
<td>SLE</td>
</tr>
<tr>
<td>IgA nephropathy</td>
<td>Malignancy</td>
</tr>
<tr>
<td>Membrano-proliferative glomeruloneph.</td>
<td>Toxemia of pregnancy</td>
</tr>
<tr>
<td>Membranous glomerulopathy</td>
<td>Drugs &amp; nephrotoxins</td>
</tr>
<tr>
<td>Congenital nephrotic syndrome</td>
<td>Lymphoma/leukemia</td>
</tr>
</tbody>
</table>
- Abdominal distention, anorexia, oliguria, puffy eyes, SOB, wt. gain
- Ascites, edema, HTN, hematuria
- UA: protein, oval fat bodies (maltese cross appearance in polarized light), hematuria
- RBC casts, fatty casts
- Serum: • albumin, azotemia
- Steroid, cyclophosphamide
- Dietary management
- Avoid nephrotoxic drugs

**Polycystic kidney disease**
- Autosomal dominant - + family hx
- Age at onset 20s-30s
- Cysts form in multiple organs – kidney, liver, cerebral aneurysms
- Abdominal or flank pain
- Hematuria
- Frequent UTIs
- Ultrasound/CT
- Treat HTN
- • fluid intake
- Transplant
## Renal vascular disease

- **Renal artery stenosis:**
  - Blockage of an artery to the kidneys that may result in kidney failure and HTN (also a cause)
  - Higher risk is smokers and more common in men between 50-70
  - May be caused by **atherosclerosis** or fibromuscular dysplasia
- **Renal artery thrombosis:**
  - May cause kidney failure
  - May be a result of trauma, infection, inflammatory dz, renal artery aneurysm, renal cell cancer of fibromuscular dysplasia
- **Renal artery aneurysm:**
  - 4 main types:
    - Saccular – bulge or balloon out only one side of the artery, congenital
    - Fusiform – bulge or balloon out on all sides of the artery, associated w/ fibromuscular dysplasia
    - Dissecting – weakened artery wall d/t a tear in the inner layer of the artery wall
    - Intrarenal – occur on an artery inside the kidney, congenital or from trauma
- **Atheroembolic renal disease – d/t atherosclerosis, mainly in older pts**

## Fluid/electrolyte disorders

- **Hyponatremia:**
  - Sodium <135 mEq/L
  - D/t • free water retention or urinary sodium loss
- **Hypernatremia:**
  - Nausea, HA, weakness, mental confusion
  - Seizures, lethargy, coma, death

## Treatment

<table>
<thead>
<tr>
<th>Renal artery stenosis</th>
<th>Renal artery thrombosis</th>
<th>Renal artery aneurysm</th>
<th>Atheroembolic renal disease</th>
<th>Renal vein thrombosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>HTN medications (other than ACEi)</td>
<td>Thrombolytics</td>
<td>Surgery to remove clot or bypass the artery</td>
<td>Medications to reduce cholesterol, BP, and other related conditions</td>
<td>Anticoagulants – may be given IV for several days, then orally for several weeks</td>
</tr>
<tr>
<td>Medications to lower cholesterol</td>
<td>Surgery to remove clot or bypass the artery</td>
<td>Surgical – angioplasty, bypass</td>
<td>Surgical – angioplasty, bypass</td>
<td>Anticoagulants – may be given IV for several days, then orally for several weeks</td>
</tr>
<tr>
<td>Treat underlying medical conditions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypervolemia</td>
<td>Hyperkalemia</td>
<td>Hypokalemia</td>
<td>Hypocalcemia</td>
<td>Hypercalcemia</td>
</tr>
<tr>
<td>--------------</td>
<td>--------------</td>
<td>-------------</td>
<td>--------------</td>
<td>---------------</td>
</tr>
<tr>
<td>Excessive sodium retention in the setting of low arterial underfilling (CHF or cirrhosis)</td>
<td>Weakness, paralysis, abdominal distension, diarrhea</td>
<td>Usually severe thirst unless mental confusion</td>
<td>Abdominal &amp; muscle cramps, tetany, seizures</td>
<td>Polyuria, constipation, abdominal pain</td>
</tr>
<tr>
<td>- Usually severe thirst unless mental confusion</td>
<td>- Serum potassium &gt; 5.5 mEq/L</td>
<td>- Weakness, paralysis, abdominal distension, diarrhea</td>
<td>- Abdominal &amp; muscle cramps, tetany, seizures</td>
<td></td>
</tr>
<tr>
<td>- Etiologies: normovolemia (diabetes insipidus), hypovolemia (dehydration, osmotic diuresis, loop diuretics)</td>
<td>- Etiologies: normovolemia (diabetes insipidus), hypovolemia (dehydration, osmotic diuresis, loop diuretics), acidosis, burns/hemolysis, spurious (+ platelets or WBC)</td>
<td>- Etiologies: normovolemia (diabetes insipidus), hypovolemia (dehydration, osmotic diuresis, loop diuretics)</td>
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<td>- Etiologies: normovolemia (diabetes insipidus), hypovolemia (dehydration, osmotic diuresis, loop diuretics)</td>
</tr>
<tr>
<td>- D/t • free water retention or urinary sodium loss</td>
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<td>- D/t • free water retention or urinary sodium loss</td>
</tr>
<tr>
<td>- Hypernatremia:</td>
<td>- Hypertension:</td>
<td>- Hypertension:</td>
<td>- Hypertension:</td>
<td>- Hypertension:</td>
</tr>
<tr>
<td>- Do not • sodium by more than 12-24 mEq/L over 24 h</td>
<td>- Do not • sodium by more than 12-24 mEq/L over 24 h</td>
<td>- Do not • sodium by more than 12-24 mEq/L over 24 h</td>
<td>- Do not • sodium by more than 12-24 mEq/L over 24 h</td>
<td>- Do not • sodium by more than 12-24 mEq/L over 24 h</td>
</tr>
<tr>
<td>- Hypervolemia:</td>
<td>- Edema</td>
<td>- Urine sodium &gt; 20: acute &amp; chronic renal failure</td>
<td>- Diuretics</td>
<td>- Restriction of water</td>
</tr>
</tbody>
</table>
# Hyponatremia

- Hyponatremia from water retention in edematous states is associated with sodium retention
- Hallmark of volume overload is sodium retention

## Hypovolemia

- Renal losses: diuretic excess, mineralocorticoid deficiency
- Extrarenal losses: vomiting, diarrhea, dehydration, sweating
  - In ECF volume (ECG volume declines when losses exceed input)
  - Hx of blood loss, GI losses (one of the most common causes of hypovolemia is diarrhea) or excessive sweating or diuretic use

## Acid/base disorders

- Renal tubular acidosis:
  - Type I: Distal H+ secretion defect
  - Sporadic, autoimmune disorders (Sjogren’s), hypercalcemia, drugs (lithium, ifosfamide, amphotericin B)
    - Kidney stones develop
  - Type II: Proximal HCO3 reabsorption defect
    - Associated with multiple myeloma, Fanconi syndrome, Wilson disease
  - Type III: Rare, associated with renal insufficiency
  - Type IV: Hyporeninemic hypaldosteronism
    - Associated with DM, drugs (NSAIDs), sickle cell dz & Addison’s dz

- Metabolic alkalosis:
  - Etiologies:
    - Chloride-responsive – vomiting, NG drainage, laxative abuse, diuretics, posthypercapnic states
    - Chloride-resistant – severe Mg or K deficiency, diuretics, Cushing’s syndrome, primary

## Normal ranges:

- pH $\rightarrow$ 7.40
- pCO2 $\rightarrow$ 35-45 mmHg
- HCO3- $\rightarrow$ 24 mEq/L

## ABG interpretation:
aldosteronism, renal artery stenosis, Bartter syndrome

- Respiratory acidosis:
  - DDX: COPD, CNS depressants, structural disorders of the thorax, neurologic disorders (Guillain-Barre), myxedema
- Respiratory alkalosis:
  - DDX: restrictive lung dz/hypoxia, CNS lesion, PE, salicylate toxicity, anxiety, sepsis, pregnancy

\[ \text{pH} = 6.1 + \log \left( \frac{[\text{HCO}_3^-]}{0.03 \times \text{pCO}_2} \right) \]

**Step 1: pH**
- Look at pH
  - Whichever side of 7.40 the pH is on, the process that caused it to shift to that side is the **primary abnormality** (>7.40 = alkalosis, <7.40 is acidosis)
  - Remember the body does not fully compensate for primary acid-base disorders

<table>
<thead>
<tr>
<th>Variable</th>
<th>Primary disorder</th>
<th>Normal range</th>
<th>Primary disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>Acidemia</td>
<td>&lt;7.40</td>
<td>Alkalemia</td>
</tr>
<tr>
<td>pCO2</td>
<td>Respiratory alkalosis</td>
<td>&lt;35-45</td>
<td>Respiratory acidosis</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>Metabolic acidosis</td>
<td>&lt;22-26</td>
<td>Metabolic alkalosis</td>
</tr>
</tbody>
</table>

**Step 2: Anion gap**
- Calculate the anion gap: \( AG = \text{Na} – (\text{Cl} + \text{HCO}_3^-) \)
  - If anion gap is \( \geq 20 \) mmol/L there is a primary metabolic acidosis regardless of the pH or serum bicarbonate
  - Remember, the body does not generate a large anion gap to compensate for a primary disorder

**Step 3: Excess anion gap**
- Calculate the excess anion gap: \( \text{Excess} = \text{total anion gap of 12 + measured bicarbonate} \)
  - If >30 mmol/L there is metabolic alkalosis, if <23 mmol/L there is non-anion gap metabolic acidosis
  - Normal bicarbonate is 23-30 mmol/L
### General Characteristics

<table>
<thead>
<tr>
<th>Esophagitis</th>
<th>Symptoms</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Infectious esophagitis usually seen in immunocompromised pts</td>
<td>- Progressive odynophagia, dysphagia</td>
<td>- EGD</td>
<td>- Nystatin, clotrimazole (candida)</td>
</tr>
<tr>
<td>- Causes: candida, herpes, CMV</td>
<td></td>
<td>- Barium swallow</td>
<td>- Fluconazole (AIDS)</td>
</tr>
<tr>
<td>- Other etiologies:</td>
<td></td>
<td>- Biopsy</td>
<td>- Acyclovir (herpes)</td>
</tr>
<tr>
<td>- Pill induced:</td>
<td></td>
<td></td>
<td>- Ganciclovir (CMV)</td>
</tr>
<tr>
<td>- Common in the elderly</td>
<td></td>
<td></td>
<td>- Sucralfate for pill-induced</td>
</tr>
<tr>
<td>- Drugs include: alendronate, quinine, risedronate, vitamin C, KCl, doxycycline, NSAIDs, iron sulfate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Radiation – if over 3000 rad</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Caustic – ingestion of drain cleaners, bleach, etc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motility disorders</td>
<td>- Arise from diseases of smooth muscle or intrinsic nervous system</td>
<td>- Achalasia:</td>
<td>- Achalasia:</td>
</tr>
<tr>
<td>- Three common causes:</td>
<td></td>
<td>- Dysphagia: liquids &amp; solids</td>
<td>- Muscle relaxant (nifedipine)</td>
</tr>
<tr>
<td>- Achalasia (most common)</td>
<td></td>
<td>- Regurgitation hrs. after eating of non-acidic material</td>
<td>- Pneumatic dilatation</td>
</tr>
<tr>
<td>- Etiology: loss of ganglion cells in Auerbach’s plexus</td>
<td></td>
<td>- Diffuse esophageal spasm:</td>
<td>- Botox injection</td>
</tr>
<tr>
<td>- tone &amp; impaired LES relaxation, absent peristalsis</td>
<td></td>
<td>- Chest pain &amp; dysphagia</td>
<td></td>
</tr>
<tr>
<td>- Diffuse esophageal spasm (uncommon)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Scleroderma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mallory-Weiss tear</td>
<td>- Non-penetrating mucosal tear at the GE junction</td>
<td>- Painless hematemesis (self-limiting)</td>
<td>- Endoscopy</td>
</tr>
<tr>
<td>- D/t rise in transabdominal pressure</td>
<td></td>
<td></td>
<td>- May stop spontaneously</td>
</tr>
<tr>
<td>- Alcoholism strong predisposing factor</td>
<td></td>
<td></td>
<td>- Epinephrine</td>
</tr>
<tr>
<td>- Prior hx of vomiting, retching</td>
<td></td>
<td></td>
<td>- Cauterization</td>
</tr>
<tr>
<td>Esophageal neoplasms</td>
<td>- D/t chronic irritation &amp; inflammation</td>
<td>- Presents as a mechanical obstruction</td>
<td>- Barium swallow</td>
</tr>
<tr>
<td>- Both squamous cell carcinoma &amp; adenocarcinoma</td>
<td></td>
<td>- Progressive dysphagia w/ solids first, then liquids</td>
<td>- EGD w/ biopsy</td>
</tr>
<tr>
<td>- Adenocarcinoma linked to Barrett’s esophagus (a complication of GERD)</td>
<td>- Odynophagia</td>
<td></td>
<td>- Esophagectomy</td>
</tr>
<tr>
<td>- Risk factors: smoking, alcohol, achalasia, RT</td>
<td></td>
<td>- Anemia, wt. loss, enlarged lymph nodes</td>
<td>- RT/chemo (5-FU)</td>
</tr>
<tr>
<td>Esophageal stricture</td>
<td>- Lower esophageal ring (Schatzki ring):</td>
<td>- Lower esophageal ring (Schatzki ring):</td>
<td>- Lower esophageal ring (Schatzki ring):</td>
</tr>
<tr>
<td>- Circumferential, lower esophageal ring</td>
<td></td>
<td>- Intermittent solid dysphagia</td>
<td>- Barium swallow</td>
</tr>
<tr>
<td>- Zenker’s diverticulum:</td>
<td></td>
<td>- Zenker’s diverticulum:</td>
<td>- Zenker’s diverticulum:</td>
</tr>
<tr>
<td>- Protrusion of pharyngeal mucosa at proximal esophagus</td>
<td></td>
<td>- Dysphagia, halitosis, regurgitation</td>
<td>- Surgery</td>
</tr>
<tr>
<td>- Esophageal web:</td>
<td></td>
<td>- Esophageal web:</td>
<td>- Esophagectomy</td>
</tr>
<tr>
<td>- Typically asx</td>
<td></td>
<td>- Typically asx</td>
<td>- Esophageal web</td>
</tr>
<tr>
<td>- May cause dysphagia to solids only</td>
<td></td>
<td>- May cause dysphagia to solids only</td>
<td>- Esophageal bougie</td>
</tr>
</tbody>
</table>
- **Non-circumferential, thin, squamous, mucosal membrane in mid or upper portion of esophagus**
  - Associated w/ severe iron deficiency & dysphagia – Plummer-Vinson syndrome

- **Esophageal varices**
  - Dilated submucosal veins
  - 2/2 portal HTN d/t cirrhosis
  - Same sx as acute upper GI bleed

- **GERD**
  - Chronic & reoccurring
  - Etiology:
    - Loss or lack of resting LES tone, allows reflux of gastric contents into the esophagus
    - Persistent irritation of squamous epithelium leads to metaplastic columnar epithelium (Barrett’s esophagus)
    - Precipitating factors: nicotine, alcohol, caffeine, peppermint, chocolate, & anticholinergics
  - Complications – esophageal stricture & Barrett’s esophagus

  - Recurrent heartburn (worse w/ bending over or lying down), belching, regurgitation, sore throat
  - Red flags: progressive dysphagia, recurrent pneumonia, persistent cough, bleeding → endoscopy exam required
  - Exam: normal

  - 24 h pH monitoring
  - EGD
  - XR: barium swallow: injury, ulcer, stricture, hernia

  - If response to medication → + dx

  - Lifestyle modification
    - Elevate head of bed 6 in.
    - Stop smoking & alcohol intake
    - Reduce dietary fat & meal size, avoid bedtime snacks
    - Lose wt.
    - Avoid chocolate, coffee, tea, cola, juice
    - H2 blockers
    - PPI
    - Surgery to tighten sphincter:
      - Indicated if refractory side effects w/ PPIs – HA, diarrhea
      - Alternative to long-term/lifelong PPIs

**Abdominal Pain Differentials:**

<table>
<thead>
<tr>
<th>Right Upper Quadrant</th>
<th>Right Lower Quadrant</th>
<th>Epigastic</th>
<th>Periumbilical</th>
<th>Left Upper Quadrant</th>
<th>Left Lower Quadrant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholecystitis</td>
<td>Appendicitis</td>
<td>Gastric ulcer</td>
<td>Early appendicitis</td>
<td>Gastritis</td>
<td>Diverticulitis</td>
</tr>
<tr>
<td>Choledocholithiasis</td>
<td>Crohn’s dz</td>
<td>Gastritis</td>
<td>SBO</td>
<td>Peptic ulcer dz</td>
<td>IBD</td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>Ileitis</td>
<td>Pancreatitis</td>
<td>Pancreatitis</td>
<td>Splenic infarct</td>
<td>Colonic ischemia</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Ovarian cyst</td>
<td>Esophagitis</td>
<td>Gastroenteritis</td>
<td>Colonic ischemia</td>
<td>Ovarian cyst</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
<td>Tubal pregnancy</td>
<td>Myocardial infarct</td>
<td>AAA</td>
<td>Pneumonia</td>
<td>Tubal pregnancy</td>
</tr>
<tr>
<td>Fitz-Hugh-Curtis</td>
<td></td>
<td>Cholecystitis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Inflammation, erosion, or damage of the gastric mucosa
- Common causes:
  - Stress – CNS injury, burns, sepsis, surgery
  - H. pylori – gram-negative flagellated rod
  - NSAIDs – cause injury by • local prostaglandin production in the stomach or cause direct injury to the cells by the pill

- Dyspepsia, wt. loss, occult bleeding, progressive dysphagia, postprandial vomiting, early satiety
- Zollinger-Ellison: abdominal pain, diarrhea, heartburn

- Anemia
- LFTs • w/ metastases
- Low albumin w/ malnourishment
- EGD w/ bx
- Barium swallow
- Abdominal CT

- Chemo
- Surgery – only chance for cure
- Radiation therapy – for lymphoma w/ chemo
- Zollinger-Ellison: PPI, surgical resection
### Peptic ulcer disease

- **Risk factors:** H. pylori infxn, dietary (excess salt & nitrates/nitrites), smoking, pernicious anemia, chronic peptic ulcer, gastritis
- **Zollinger-Ellison syndrome:**
  - Gastrin-producing endocrine tumor (duodenal or pancreatic) leading to gastric acid hypersecretion
  - Ages 35-65, men > women 3:2

- **Recurrence**
  - Recurrent episodes of deep, gnawing or burning pain in midepigastric region
  - Pain w/ eating (gastric ulcer), awakening at night
  - Relief w/ food (duodenal ulcer)
  - Nausea w/ or w/o vomiting
  - PE unrevealing, tenderness in RUQ/epigastrium is unusual, possible peritoneal signs if rupture

- **Zollinger-Ellison:**
  - Elevated gastrin level (off PPI & H blockers), gastric pH <2.0
  - EGD: multiple ulcers

- **Special tests**
  - H. pylori testing: bx, serology, stool antigen test, breath test
  - Breath test or stool antigen helpful w/ follow-up
  - EGD w/ bx

- **Treatment**

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Drug</th>
<th>Triple Therapy</th>
<th>Quadruple Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acid suppression</td>
<td>PPI</td>
<td>20-40 mg BID</td>
<td>20-40 mg BID</td>
</tr>
<tr>
<td>Standard antimicrobials</td>
<td>Bismuth compound</td>
<td>2 tablets BID</td>
<td>2 tablets BID</td>
</tr>
<tr>
<td></td>
<td>Amoxicillin</td>
<td>1 g BID</td>
<td>500 mg TID</td>
</tr>
<tr>
<td></td>
<td>Metronidazole</td>
<td>500 mg BID</td>
<td>500 mg BID</td>
</tr>
<tr>
<td></td>
<td>Clarithromycin</td>
<td>500 mg QID</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tetracycline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salvage antimicrobials</td>
<td>Levofloxacin</td>
<td>300 mg BID</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rifabutin</td>
<td>150 mg BID</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Furazolidone</td>
<td>100 mg BID</td>
<td></td>
</tr>
</tbody>
</table>

### Pyloric stenosis

- **Diffuse hypertrophy & hyperplasia of pyloric sphincter muscle**
- **Men > women; onset ages 2-4 weeks**
- **First born males**

- **Projectile, non-bilious vomiting, child still hungry**
- **Movable olive size 2 cm mass in midepigastrium**
- **Dehydration**

- **Hypochloremic hypokalemic metabolic alkalosis**
- **Barium swallow: delayed gastric emptying, string sign**
- **Ultrasound: elongation & thickening of pylorus**

- **Rehydration**
- **Surgery (pylorotomy)**

### Acute cholecystitis

- **Inflammation of the gallbladder**
- **Etiology:** obstruction by a stone in cystic duct; distention & inflammation; infxn by bowel flora
- **Risk factors:** age 40s, women, obesity, parity, elevated TG, meds (estrogen, clofibrate, ceftriaxone, Sandostatin)

- **RUQ pain, fever, leukocytosis**
- **Pain is steady, unremitting, may radiate to right scapula (15-30 min. after a meal)**
- **N/V, dehydration**
- **Exam:**
  - RUQ tenderness, + bowel sounds, guarding, tachycardia

- **Increased WBC w/ a left shift**
- **Increased bilirubin & alkaline phosphatase**
- **U/S: look for thick gallbladder wall, sludge & stones**
- **HIDA scan: gallbladder fails to fill**

- **Abx: ampicillin + aminoglycoside**
- **Surgery - cholecystectomy**
### Chronic cholecystitis
- Positive Murphy’s sign
  - Results from repeated episodes of acute cholecystitis or chronic irritation by gallstone
  - May be associated w/ fistulization to the bowel, pancreatitis & rarely carcinoma of the gallbladder
  - Jaundice – Mirizzi syndrome (stone in neck of gallbladder that compresses common hepatic duct)
  - “Strawberry gallbladder” – villi of gallbladder undergo polypoid enlargement d/t cholesterol deposition
- Elective surgical cholecystectomy

### Cholangitis
- Infection of the bile duct
- Caused by bacteria (E. coli, Klebsiella, Enterococcus) ascending from the duodenum
- D/t gallstones, strictures, or tumors
- Jaundice, fever, RUQ pain
- Charcot’s triad – abdominal pain, jaundice & fever
- Increased WBC & LFTs
- U/S, ERCP
- IV abx – PCN/aminoglycosides
- ERCP

### Cholelithiasis
- Develop slowly
- Cholesterol stones = 80-90%
- Pigment stones – most common pigment is bilirubin
- Risk factors:
  - 4 Fs: fat, female, fertile & 40
  - 5 Fs: fair, fat, female, fertile & 40
- Other risk factors include:
  - Rapid weight loss → due to stasis
  - Crohn’s dz (+ bile salts)
  - Hyperlipidemia
  - Meds: clofibrate, ceftriaxone, octreotide, HRT (hormone replacement therapy)
  - Insulin resistance, metabolic syndrome & diabetes
- 2/3 are asymptomatic
- Biliary colic:
  - Highly predictive of gallstone dz
  - “Colic” is a MISNOMER b/c it indicates severe, fluctuating pain but in biliary colic the pain is actually dull, steady, sudden onset, & builds to max w/in 1 hr. & lasts 2-4 hrs.
  - Localized to RUQ or epigastrium; sometimes presents as chest pain
  - Can radiate to left or right shoulder or scapular region
  - Often associated w/ N/V
  - Atypical: may be associated w/ gallstones but not specific:
    - Flatulence
    - Heartburn
    - Acid regurgitation
    - Bloating
    - Belching
- U/S
- Watch & wait if asx
- Low-carb diet, physical activity, & cardio fitness may help prevent gallstones
- Medical tx w/ bile acids to dissolve cholesterol stones only
  - Ursodeoxycholic acid
  - Chenodeoxycholic acid
  - Tx can be for up to 2 years
  - Complication: gallstones recur once meds are stopped
  - Laparoscopic cholecystectomy only for symptomatic cases

### Hepatitis
- Inflammation of the hepatocytes
- Caused by toxin & viruses:
  - 5 hepatitis viruses: A, B, C, D, E
  - Toxins/drugs include alcohol, acetaminophen, isoniazid, lovastatin
  - Autoimmune – women, positive ANA, treat w/ steroids
- Fatigue, malaise, nausea, anorexia
- Skin & scleral icterus
- Hepatomegaly
- Dark urine & light stools
- Elevated AST & ALT 10-20x normal
- Prothrombin time
- Serology testing

### Hepatitis Summary

<table>
<thead>
<tr>
<th>Virus</th>
<th>Spread</th>
<th>Incubation Period</th>
<th>Chronic Disease</th>
<th>Diagnosis</th>
<th>Antibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Fecal-oral</td>
<td>15-45 d</td>
<td>No</td>
<td>IgM anti-HAV</td>
<td>Anti-HAV</td>
</tr>
<tr>
<td>B</td>
<td>Parenteral</td>
<td>30-180 d</td>
<td>5%</td>
<td>HbsAg</td>
<td>Anti-HBs, Anti-HBc, Anti-HBe</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>Parenteral</td>
<td>15-150 d</td>
<td>50-80%</td>
<td>HCV RNA</td>
<td>Anti-HCV</td>
</tr>
<tr>
<td>------------</td>
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<td>--------</td>
<td>---------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Transmitted by fecal-oral route, shellfish</strong></td>
<td>Parenteral</td>
<td>30-150 d</td>
<td>5%</td>
<td>Presence of hepatitis B</td>
<td>Anti-HDV</td>
</tr>
<tr>
<td><strong>No chronic hepatitis</strong></td>
<td>Sexual</td>
<td>30-60 d</td>
<td>No</td>
<td>IgM anti-HEV</td>
<td>Anti-HEV</td>
</tr>
<tr>
<td><strong>Incubation 20-40 d</strong></td>
<td>Fecal-oral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Hepatitis A**

- Transmitted by fecal-oral route, shellfish
- No chronic hepatitis
- Incubation 20-40 d

**Hepatitis B**

- Transmitted by direct contact w/ blood or body fluids, sexual contact
- Acute & chronic dz
- Incubation 60-100 d

**Hepatitis C**

- Transmitted by blood & body fluids
- Time to seroconversion: 6 weeks
- 50-80% develop chronic hepatitis
- Link btwn hepatitis C & hepatocellular carcinoma
- Serology: hepatitis C antibody

**Hepatitis D**

- Delta hepatitis
- Transmitted parenterally
- Seen only in conjunction w/ hepatitis B
- Serology: hepatitis D antibody

**Hepatitis E**

- Fecal-oral
- 30-60 d
- No
- IgM anti-HEV
- Anti-HEV

**Hepatitis A**

- Vaccine
- Post-exposure immunoglobulin to contacts

**Hepatitis B**

- Lamivudine (Epivir-HBV) (nucleoside reverse transcriptase inhibitor) – interferon not helpful
- Prophylaxis: hepatitis B immunoglobulin used in newborns of infected mothers, percutaneous, or sexual exposure
- Vaccine at 0, 1 & 6 months

**Hepatitis C**

- Peginterferon alfa
- Ribavirin – chronic

**Hepatitis D**

- Hepatitis B vaccine
| Hepatitis E | -Transmitted by fecal-oral route  
-Incubation 15-60 d  
-Contaminated food & water  
-No chronic dz |
| Cirrhosis | -End-stage of chronic liver dz  
-Involves fibrosis & is irreversible  
-Causes:  
- Alcohol abuse  
- Viral hepatitis: B, D, C  
- Metabolic disorders  
- Autoimmune hepatitis  
- Biliary disorders  
- Drugs & toxins  
- Nonalcoholic fatty liver  
-Complications: portal HTN, varices & hemorrhage, ascites, hepatorenal syndrome, spontaneous bacterial peritonitis, encephalopathy, jaundice  
-Weakness/fatigue  
-Anorexia  
-Wt. loss  
-Abdominal pain  
-Jaundice  
-Edema  
-Spider angiomas  
-Telangiectasis  
-Palmar erythema  
-Purpura  
-Signs of feminization  
-Muscle atrophy  
-Small right liver lobe  
-Determine underlying cause  
-Abdominal U/S  
-CT scan w/ liver bx  
-Avoid alcohol  
-Treat underlying cause, sx, complications  
-Liver transplant  
-Varices – beta-blockers to • portal pressure  
-Ascites – salt restriction (2g/day) & spironolactone, paracentesis  
-Hepatorenal syndrome – transplant  
-SBP – cefotaxime, ceftriaxone, amoxicillin-clavulanic acid  
-Hepatic encephalopathy - lactulose |
| Liver neoplasms | -Malignant neoplasm of liver that arises from parenchymal cells  
-Cholangiocarcinoma – originates in ductular cells  
-Usually a complication of cirrhosis  
-Risk factors: cirrhosis, hepatitis B/C/D, hemochromatosis  
-In pts w/ cirrhosis, additional risk factors:  
- Male, >55 yrs., Asian/Hispanic, family hx, DM, hypothyroidism, overweight, EtOH, hepatic C, elevated transferrin, prolonged PT, low platelet  
-Fibrolamellaer variant – young women, absence of risk factors, indolent course  
- BCLC staging system is preferred method of staging  
- Unsuspected until deterioration in a cirrhotic pt who was formerly stable  
- Cachexia, weakness & wt. loss  
- Sudden appearance of ascites (may be bloody)  
- Portal/hepatic vein thrombosis  
- Enlargement of liver w/ palpable mass  
- Leukocytosis  
- Anemia  
- Hematocrit normal or elevated  
- Sudden sustained elevation of alk. phosphatase  
- Hepatitis panels  
- Alpha-Fetoprotein levels  
- Elevated des-gamma-carboxy prothrombin  
- Multiphasic helical CT scan & MRI enhancement are preferred for location & vascularity  
- Liver bx: if lesion <1 cm then U/S may repeated every 3 months, for lesions >1 cm bx can be deferred  
- Chemo, hormonal therapy w/ tamoxifen & long-acting octreotide do not prolong life  
- Adaptive immunotherapy, adjuvant chemo & tx of underlying dz may lower recurrence  
- Sorafenib is standard of care for advanced dz  
- Liver transplant  
- Chemoembolization – may be palliative  
- Small tumors  
- Inject absolute ethanol, radiofrequency ablation, cryotherapy may prolong survival |
| Acute pancreatitis | -Inflammatory dz of the pancreas  
-Results in edema, vascular injury, tissue loss & necrosis  
-Constant mid-epigastric/LUQ deep, boring pain  
-Radiation to back  
-Increased amylase & lipase  
-Amylase • in 2-12 h & • over 3-5 d  
-Lipase best test & elevated longer  
-Alcohol abstinence  
-Pain control  
-NPO |
Pancreas returns to normal after the episode.

**Etiology:**
- **Gallstones, alcohol,** meds, trauma, HLD
- Meds – immunosuppressents, trim-sulfa, pentamidine, didanosine, furosemide, thiazide, diuretics, ACEi, & estrogens
- Hypertriglyceridemia levels typically >1000 mg/dL

**Complications:** infxn, pseudocysts, renal/respiratory failure

- Severe nausea & vomiting
- Pt should avoid lying supine
- Decreased bowel sounds
- Grey-Turner’s sign: flank ecchymosis
- Cullen’s sign: periumbilical ecchymosis
- Pleur effusion

- Increased AST/ALT & alkaline phosphatase
- Increased glucose & calcium
- Leukocytosis
- U/S to r/o gallbladder dz
- CT scan to evaluate for complications

**Ranson’s Criteria**

<table>
<thead>
<tr>
<th>On admission</th>
<th>Within 48 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;55 yrs.</td>
<td>Glucose &gt;200 mg/dL</td>
</tr>
<tr>
<td>WBC &gt;16,000/uL</td>
<td>Hct by &gt;10%</td>
</tr>
<tr>
<td>AST &gt;250 U/L</td>
<td>BUN by &gt;5 mg/dL</td>
</tr>
<tr>
<td>LDH &gt;350 U/L</td>
<td>Ca++ &lt;8 mg/dL</td>
</tr>
<tr>
<td>Art. pO2 &lt;60 mmHg</td>
<td>Base deficit &gt;4 mEq/L</td>
</tr>
<tr>
<td>Fluid sequestration &gt;6 L</td>
<td></td>
</tr>
</tbody>
</table>

**Chronic pancreatitis**
- Inflammatory dz w/ progressive & permanent damage to the pancreas
- Alcohol abuse 90% of cases
- Gallstones DO NOT cause chronic pancreatitis
- Intermittent or chronic abdominal pain
- Diarrhea & **steatorrhea**
- Wt. loss, jaundice
- Exam: thin pt, mild jaundice, diffuse abdominal tenderness

- Amylase & lipase – normal to mild elevation
- Bilirubin •, hyperglycemia
- 72 h fecal fat (measures pancreatic exocrine function)
- XR: pancreatic calcifications
- U/S – ERCP – best test

- Abstinence from alcohol
- Pain control
- Enzyme replacement for malabsorption
- Surgery

**Pancreatic neoplasms**
- Ductal adenocarcinoma most common – 5 yr. survival rate of <5%
- Risk factors: smoking, hx of chronic pancreatitis, obesity, high-fat diet
- Early:
  - Nonspecific abdominal pain, N/V, anorexia & malaise
- Late:
  - Wt. loss, obstructive jaundice
- Classic painless jaundice is not very common
- Exam: large, non-tender, palpable gallbladder (Courvoisier’s sign)
- Jaundice

- Anemia, elevated alk. phosphatase, bilirubin, AST/ALT
- ERCP w/ bx
- Tumor marker CA-19-9 positive
- CT scan

- Palliative care
- Surgery: RT & chemotherapy (5-FU or gemcitabine)

**Appendicitis**
- D/t obstruction of appendiceal lumen by a fecalith
- Leads to inflammation & infxn
- Ages 10-30

- Initial: intermittent periumbilical pain
- 12 h later: pain RLQ (McBurney’s pont), worse w/ movement
- Nausea, low-grade fever, • appetite
- Exam:
  - Guarding RLQ, cutaneous hypersensitivity
  - Positive Rovsing’s sign

- Increased WBC w/ left shift
- U/S, CT scan

- Appendectomy
<table>
<thead>
<tr>
<th>Celiac disease</th>
<th>Positive psoas/obturator sign w/ peritonitis</th>
<th>IgA tissue transglutaminase (tTG) antibody screening test</th>
<th>Gluten-free diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Inflammatory rxn in upper small intestine</td>
<td>-Watery diarrhea, abdominal pain</td>
<td>-Small bowel bx</td>
<td></td>
</tr>
<tr>
<td>-Present w/ iron deficiency anemia</td>
<td>-Malabsorption sx</td>
<td>-Endomysial IgA antibodies</td>
<td></td>
</tr>
<tr>
<td><strong>Autoimmune disorder:</strong></td>
<td>-Dermatitis herpetiform – vesicular rash on extensor surfaces</td>
<td>-Response to gluten-free diet</td>
<td></td>
</tr>
<tr>
<td>• 1% of population</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Women &gt; men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Breastfeeding may be protective</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• European ancestry, Middle Eastern, Asian, North African</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Precipitated by ingestion of gluten</strong> – found in wheat, barley, rye</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Complications: osteoporosis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Constipation</th>
<th>Abdominal pain or bloating</th>
<th>CBC, CMP, TSH</th>
<th>Lifestyle modification:</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Occurs in 10-15% of adults</td>
<td></td>
<td>Colonoscopy or sigmoidoscopy</td>
<td>Toileting habits – timing, positioning &amp; abdominal pressure</td>
</tr>
<tr>
<td>-Risk factors: elderly, meds, mobility, bed-bound, poor eating habits</td>
<td></td>
<td></td>
<td>Fluids &amp; fiber (most beneficial for those w/ normal colonic transit time)</td>
</tr>
<tr>
<td>-Etiology:</td>
<td></td>
<td></td>
<td>Laxatives – be careful of laxative dependence especially in younger populations</td>
</tr>
<tr>
<td>• Primary – slow colonic transit time, dyssynergic defecation (impaired relaxation or paradoxical contraction of the anal sphincter &amp;/or pelvic floor muscles during defecation)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Secondary – systemic (neurologic, myopathy, endocrine or electrolyte abnormalities (hypercalcemia or hypokalemia)), meds (anti-cholinergic or opioids), neoplasms</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diverticular disease</th>
<th>Diverticulosis: presence of diverticula</th>
<th>Diverticulitis: inflammation &amp; infxn of diverticulum</th>
<th>Diverticulitis: high fiber diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Onset &gt; 40 yrs.</td>
<td>-Diverticulosis: 80% asx, crampy LLQ pain (rare) &amp;/or painless rectal bleeding</td>
<td>-Diverticulitis: acute LLQ pain, anorexia, fever, chills, N/V, + peritoneal signs</td>
<td></td>
</tr>
<tr>
<td>-Diverticulitis: inflammation &amp; infxn of diverticulum</td>
<td>-Rectal bleeding is rare</td>
<td>-CT scan: abscess, perforations, colonic wall thickening</td>
<td>-Diverticulitis:</td>
</tr>
<tr>
<td></td>
<td>-Exam:</td>
<td></td>
<td>• Abx – broad spectrum: ciprofloxacin &amp; metronidazole</td>
</tr>
<tr>
<td></td>
<td>• Diverticulosis: firm, tender mass LLQ, rectal bleeding</td>
<td></td>
<td>• Clear liquid diet &amp; IVF</td>
</tr>
<tr>
<td></td>
<td>• Diverticulitis: bowel sounds, distention, rebound tenderness &amp; guarding, fever</td>
<td></td>
<td>• Surgery – young pts ages &lt;40</td>
</tr>
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<td></td>
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</tr>
</tbody>
</table>
## Inflammatory bowel disease

### Ulcerative colitis:
- Men = women, ages 15-25 & 55-65
- Complications: 1/3 develop colon cancer
- Involves only mucosa & submucosa of the colon
- **Starts in rectum & is continuous**
- Fistulas & fissures are rare
- Complications: toxic megacolon

**Crohn’s dz:**
- Peak ages 15-25 & 55-65
- Can affect any GI mucosa
- Is transmural – fistulas & fissures are common
- “Skip lesion,” cobblestone appearance
- Complications: abscess, fistula, obstruction, perianal dz

### Ulcerative colitis vs. Crohn’s

<table>
<thead>
<tr>
<th>Pathologic:</th>
<th>Ulcerative colitis</th>
<th>Crohn’s dz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectal involvement</td>
<td>Always</td>
<td>Common</td>
</tr>
<tr>
<td>Fissures/fistulas</td>
<td>Never</td>
<td>Never</td>
</tr>
<tr>
<td>Skip lesions</td>
<td>Never</td>
<td>Always</td>
</tr>
<tr>
<td>Perianal dz</td>
<td>Never</td>
<td>Common</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical:</th>
<th>Ulcerative colitis</th>
<th>Crohn’s dz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectal bleeding</td>
<td>Always</td>
<td>Occasional</td>
</tr>
<tr>
<td>Malaise, fever</td>
<td>Occasional</td>
<td>Common</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>Occasional</td>
<td>Common</td>
</tr>
<tr>
<td>Abdominal mass</td>
<td>Never</td>
<td>Common</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Endoscopic:</th>
<th>Ulcerative colitis</th>
<th>Crohn’s dz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobblestoning</td>
<td>Rare</td>
<td>Common</td>
</tr>
</tbody>
</table>

### Intussusception
- Telescoping of proximal portion of intestine into a distal portion – mostly ileal-colic
- Can lead to necrosis
- Cause is unknown – frequently a hx of adenovirus or rotavirus infxn, URI or OM
- Common ages 5 mo. to 2 yrs.

- Sudden onset, recurrent, paroxysmal, sharp abdominal pain
- Vomiting
- Exam:
  - Lethargy
  - **Sausage-like mass in upper abdomen**
  - Heme-positive stool
  - “Currant jelly” stool

- U/S: loss of bowel gas
- Barium or air enema: meniscus & coiled spring signs

- Reduction by BE
- IV fluids
| Irritable bowel syndrome | -D/t abnormal motor function of the GI tract & gut visceral sensitivity  
-Women > men; onset ages 30-50 | -Crampy, lower abdominal pain that is relieved by defecation  
-Irregular disturbances in defecation (constipation & diarrhea)  
-Abdominal bloating  
-PE: abdominal tenderness | -Rome criteria (2 or more):  
- Pain or discomfort relieved by defecation  
- Pain associated w/ or stool frequency  
- Pain associated w/ harder or looser stools  
-Watch for red flags of cancer – wt. loss, bleeding, etc.  
-Increase fiber, avoid milk products & high-fat diet  
-Antispasmodics: hyoscyamine (Levsin), belladonna  
-MiraLAX (constipation) & loperamide (diarrhea) |  |
| Ischemic bowel disease | -Etiology: drugs, trauma, connective tissue dz | -Acute abdominal pain, melena  
-Clinical findings out of portion to PE  
-Exam: abdomen distention, shock, minimal exam findings | -Elevated WBC, amylase, CPK  
-Angiogram  
-Surgery  
-Heparin |  |
| Lactose intolerance | -Lactase is a brush border enzyme that hydrolyzes lactose into glucose & galactose  
-Diarrhea, bloating, flatulence, & abdominal pain after ingestion of milk products | -Improvement on lactose-free diet  
-Hydrogen breath test | -Lactase enzyme replacement |  |
| Intestinal neoplasms | -Most common in 50s-60s  
-Risk factors: red meat, ingestion of smoked or cured foods, Crohn’s disease, celiac sprue, hereditary nonpolyposis colorectal cancer, familial adenomatous polyposis and Peutz-Jeghers syndrome  
-Adenomas are most common benign neoplasm of small intestine  
-Other benign neoplasms include lipomas, fibromas, hemangiomas, lymphangiomas, and neurofibromas  
-Adenocarcinomas comprise 35-50% of all cancerous lesions of the small intestine  
-Asx unless they become large  
-Crampy abdominal pain and distention  
-N/V  
-Palpable abdominal mass in 25%  
-Serum 5-hydroxyindole acetic acid – may be elevated in carcinoid syndrome  
-CEA elevated only when liver mets are present  
-Enteroclysis is the test of choice – 90% sensitivity  
-EGD/biopsy |  |
| Intestinal obstruction | -Small intestine:  
-Adhesions are the most common cause  
-Also d/t hernia, neoplasma, intussusception, volvulus  
-Types:  
- Mechanical – d/t a physical barrier  
- Paralytic ileus – neurogenic failure of peristalsis  
- Simple – occludes the lumen only  
- Strangulation – impairs the blood flow & leads to necrosis  
-Large intestine:  
- Vomiting & abdominal pain  
- Increased bowel sounds  
-Deep, visceral, cramping pain referred to the hypogastrium  
-High pitched bowel sounds  
-Constipation or obstipation  
-Abdominal tenderness  
-XR: abdominal reveals ladderlike pattern of dilated small bowel loops w/ air-fluid levels, colon is devoid of gas |  |
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-Abdominal tenderness  
-XR: abdominal reveals ladderlike pattern of dilated small bowel loops w/ air-fluid levels, colon is devoid of gas |  |
| Polyps                          | -Discrete mass lesion that protrude into intestinal lumen  
|                               | -Most commonly sporadic but can be inherited as familial polyposis syndrome  
|                               | -95% of adenocarcinoma of the colon arise from adenomas & serrated polyps  
|                               | -4 types:  
|                               | -Mucosal adenomatous (70%) – tubular, tubulovillous, & villous  
|                               | -Mucosal serrated polyps (25-30%) – hyperplastic, sessile serrated & traditional serrated  
|                               | -Mucosal nonneoplastic – juvenile, hamartomas, inflammatory  
|                               | -Submucosal – lipomas, lymphoid aggregates, carcinoids, pneumatosis cystoides intestinalis  
|                               | -Most adenomas are small & have a low risk of malignancy  
| -Asx                          | -Large polyps may ulcerate resulting in intermittent hematochezia  
| -BE                           | -CT colonography  
| -CT colonography              | -Colonoscopy – best means of detecting & removing adenomatous & serrated polyps  
| -Colonoscopy                  | -Colonoscopic polypectomy → repeat colonoscopy in 3-5 yrs.  
|                               | -1-2 small (<1 cm) tubular adenomas → colonoscopy in 5-10 yrs.  
|                               | -3-10 adenomas, adenoma >1 cm, or adenoma w/ villous features or high-grade dysplasia → colonoscopy at 3 yrs.  
|                               | ->10 adenomas → colonoscopy at 1-2 yrs. & possible evaluation for familial polyposis syndrome  
| Toxic megacolon               | -Noted in pts w/ ulcerative colitis  
|                               | -Signs of toxicity  
|                               | -Colonic diameter >6 cm on plain films  
|                               | -NG suction  
|                               | -If pt worsens or fails to improve then surgery to prevent perforation  
| Anal fissure                  | -Linear shaped ulcers, typically <5 mm in length  
|                               | -Most commonly in the posterior midline  
|                               | -If off midline think Crohn’s dz  
|                               | -Chronic fissures result in skin tags at outermost edge (sentinel pile)  
|                               | -Typically arise from trauma  
|                               | -Sever, tearing pain during defecation followed by throbbing pain  
|                               | -Hematochezia  
|                               | -Fiber supplements & sitz baths  
|                               | -Topical anesthetics  
| Abscess/fistula                | -Infected anal glands at base of anal crypts at the dentate line  
|                               | -Throbbing, continuous perianal pain  
|                               | -Erythema, fluctuance, & swelling  
|                               | -Local I&D  
| Fecal impaction               | -Severe impaction may lead to partial or complete bowel obstruction  
|                               | -Risk factors: meds (opioids), severe psych dz, prolonged bed rest, neurogenic d/o of the colon, & spinal cord d/o  
|                               | -Decreased appetite  
|                               | -N/V  
|                               | -Abdominal pain/distention  
|                               | -May be paradoxical "diarrhea" as liquid stool leaks around impacted feces  
|                               | -PE: firm feces palpable on DRE  
|                               | -Enemas – saline, mineral oil, diatrizoate  
|                               | -Digital disruption of impacted material  
|                               | -Long-term care aimed at maintaining soft stool & regular BMs  
| Hemorrhoids                    | -Symptomatic as a result of activities that • venous pressure – straining, constipation, prolonged sitting, pregnancy, obesity  
|                               | -Rectal bleeding  
|                               | -May enlarge & protrude from anal opening  
|                               | -Pain noted w/ external hemorrhoids, rare w/ internal  
|                               | -High-fiber diet, fluid intake  
|                               | -Sclerotherapy, rubber band ligation, electrocoagulation  
|                               | -Surgical excision (hemorrhoidectomy)  

- External hemorrhoids are noted on perianal inspection.

**Rectal neoplasms**

- 95% adenocarcinoma (lymphomas, malignant carcinoid, leiomyoma, Kaposi’s sarcoma)
- M:F = 9:1
- Lifetime risk is 1 in 18, most have no risk factors
- Risk factors:
  - Age > 60
  - Family hx
  - Smoking, low fiber diet high in red meats, obesity
  - IBD
  - Physical activity
  - Hereditary syndromes (15%)

-African Americans should begin screening at 45 due to incidence & death:

- Less likely than Caucasians to get screening tests for CRC, more likely than Hispanics
- Less likely to have colorectal polyps detected at a time when they can easily be removed
- More likely to be dx w/ CRC in advanced stages, less likely to live 5+ yrs. after being dx w/ CRC than other populations.
- Genetic factors may contribute to incidence
- Hereditary colon cancer syndromes:
  - Familial polyposis – young age, 35 yrs., numerous polyps
  - Hereditary nonpolyposis colorectal cancer – Lynch’s syndrome, most common, autosomal dominant, onset age 40, no polyps
  - Gardner’s syndrome – numerous polyps but also extraintestinal manifestations
  - Peutz-Jeghers syndrome – autosomal dominant, age 20s, polyps, mucocutaneous pigmentation

**CRC screening for AVERAGE risk male/female pts 50+ (negative family hx):**

- Tests that find polyps & cancer:
  - Flexible sigmoidoscopy every 5 yrs.*
  - Colonoscopy every 10 yrs.
  - Double-contrast barium enema every 5 yrs.*
  - CT colonography (virtual colonoscopy) every 5 yrs.*
- Tests that mainly find cancer:
  - Fecal occult blood test (FOBT) every year*, **
  - Fecal immunochemical test (FIT) every year*, **

*Colonoscopy should be done if test results are +
**For FOBT or FIT used as a screening test, the take-home multiple sample method should be used. FOBT or FIT done during a digital rectal exam in office is not adequate for screening.

### Risk Factor | Age to Begin | Recommended Tests | Frequency
--- | --- | --- | ---
-Colorectal cancer or adenomatous polyps in any first-degree relative before age 60, or in 2 or more first-degree relatives at any age (if not a hereditary syndrome) | -Age 40 or 10 yrs. before the youngest case in the immediate family, whichever is earlier | -Colonoscopy | -Every 5 yrs.
-Colorectal cancer or adenomatous polyps in any first-degree relative 60 or older, or in at least two 2nd degree relatives at any age | -Age 40 | -Same options as for those at AVERAGE risk | -Same schedule as for those at AVERAGE risk

### Screening for colorectal cancer: USPSTF recommendations:

<table>
<thead>
<tr>
<th>Population</th>
<th>Ages 50-75</th>
<th>Ages 76-85</th>
<th>Older than 85</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Recommendation</strong></td>
<td>Screen w/ high-sensitivity FOBT, sigmoidoscopy, or colonoscopy</td>
<td>Do not screen routinely</td>
<td>Do not screen</td>
</tr>
</tbody>
</table>

**Screening test**

- High-sensitivity FOBT, sigmoidoscopy w/ FOBT, & colonoscopy are effective

**Screening test intervals**

- Annual screening w/ high-sensitive FOBT
- Sigmoidoscopy every 5 yrs. w/ high sensitivity FOBT every 3 yrs.
- Screening colonoscopy every 10 yrs.

**Other**

- USPSTF does not recommend the use of ASA or NSAIDs for primary prevention of colorectal cancer
### Hernias

- **Indirect:**
  - Passes from internal inguinal ring obliquely toward the external inguinal ring & into scrotum
- **Direct:**
  - Protrudes outward & forward & is medial to the internal inguinal ring

- Bulge in the inguinal region
- Pain or vague discomfort in the region
- Extreme pain w/ strangulation

- **PE:**
  - Bulge in inguinal region
  - Cough or Valsalva maneuver can facilitate identification
  - Bulge moving lateral to medial suggests indirect
  - Bulge progresses from deep to superficial through inguinal floor suggests direct
  - Bulge identified below the inguinal ligament suggests femoral hernia

- **U/S may help**

- **Surgery**

### Diarrhea

- Increased volume & liquidity of stool
- Acute vs. chronic:
  - Acute: duration 2-3 weeks
  - Chronic: >4 weeks

- **Types:**
  - Secretory: *E. coli* toxin, *Salmonella*, *Clostridium perfringens* toxin
  - Osmotic: lactose deficiency, laxative
  - Inflammatory: IBD, radiation colitis, ischemic colitis, pseudomembranous
  - Must separate infectious from non-infectious

- Post abx or chemotherapy
- Inflammatory
- Many fecal WBC, positive C. difficile toxins

- **Other:**
  - Ciguatera toxin:
    - Found in barracuda, red snapper, grouper

<table>
<thead>
<tr>
<th>Feature</th>
<th>Osmotic</th>
<th>Secretory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect of fasting</td>
<td>Diarrhea stops</td>
<td>Diarrhea usually continues</td>
</tr>
<tr>
<td>Fecal pH</td>
<td>Frequently  •</td>
<td>Normal</td>
</tr>
<tr>
<td>Fecal osmolality</td>
<td>330</td>
<td>290</td>
</tr>
<tr>
<td>Fecal electrolytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium (Na)</td>
<td>30</td>
<td>100</td>
</tr>
<tr>
<td>Potassium (K)</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>(Na + K) x2</td>
<td>120</td>
<td>280</td>
</tr>
<tr>
<td>Osmotic gap</td>
<td>210</td>
<td>10</td>
</tr>
</tbody>
</table>

- **Osmotic vs. secretory diarrhea**
- Pseudomembranous colitis – metronidazole or vancomycin
- Ciguatera – no specific tx
- Scombroid - antihistamines

**Treatment:** colectomy, chemo, radiation for rectal cancer, monitor response w/ CEA
- Sx w/ 2-6 h of ingestion – paresthesias, numbness, N/V, abdominal cramps
- Scombroid:
  - Ingestion of scombroid fish – tuna, mackerel, mahi mahi
  - Contain large amount of histamine & cause – rash, diarrhea, vomiting, wheezing, dizziness

<table>
<thead>
<tr>
<th>Infectious diarrhea</th>
<th>-80% of acute diarrheas</th>
<th>-Bloody diarrhea, abdominal pain, dysentery, fecal WBC</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Epidemiology: food, abx, sexual activity, daycare, outbreaks, hospitalization</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Etiology:</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Viral</strong></td>
<td><strong>Bacterial</strong></td>
<td><strong>Protozoal</strong></td>
</tr>
<tr>
<td>Rotavirus</td>
<td>Shigella</td>
<td>Giardia lambia</td>
</tr>
<tr>
<td>Norovirus</td>
<td>Salmonella</td>
<td>Entamoeba</td>
</tr>
<tr>
<td>Norovirus-like agent</td>
<td>Campylobacter</td>
<td>Cryptosporidium</td>
</tr>
<tr>
<td>Enteric adenovirus</td>
<td>E. coli</td>
<td></td>
</tr>
<tr>
<td>Calicivirus</td>
<td>Yersinia</td>
<td></td>
</tr>
<tr>
<td>Astrovirus</td>
<td>C. difficile</td>
<td></td>
</tr>
<tr>
<td>Small round viruses</td>
<td>C. perfringens</td>
<td></td>
</tr>
<tr>
<td>Coronavirus</td>
<td>S. aureus</td>
<td></td>
</tr>
<tr>
<td>HSV</td>
<td>Bacillus creus</td>
<td></td>
</tr>
<tr>
<td>CMV</td>
<td>Vibrio</td>
<td></td>
</tr>
<tr>
<td>Chlamydia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N. gonorrhoeae</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Organism</th>
<th>Spread</th>
<th>Comments</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. coli (not 0157:H7)</td>
<td>Fecal contamination of food &amp; water</td>
<td>Traveler’s diarrhea Secretory</td>
<td>TMP-SMX Fluroquinolone</td>
</tr>
<tr>
<td>Campylobacter</td>
<td>Contaminated water or milk</td>
<td>Self-limited Short duration</td>
<td>Fluroquinolone Azithromycin</td>
</tr>
<tr>
<td>Salmonella</td>
<td>Contamination of eggs &amp; milk</td>
<td>Acute, self-limited crampy abdominal pain</td>
<td>None</td>
</tr>
<tr>
<td>Shigella</td>
<td>Fecal/oral</td>
<td>Day cares Bloody diarrhea</td>
<td>Ampicillin or TMP-SMX</td>
</tr>
<tr>
<td>Giardia</td>
<td>Fecal contamination of water</td>
<td>Water-borne infxn</td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Norovirus/ Rotavirus</td>
<td>Person-to-person</td>
<td>Self-limited dz</td>
<td>None</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Non-infectious diarrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>-20% of acute diarrheas</td>
</tr>
<tr>
<td>-Epidemiology: meds, ingestion of osmotic particles, fecal impaction</td>
</tr>
<tr>
<td>-Watery diarrhea, crampy abdominal pain, no fecal WBC</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamin &amp; nutritional deficiencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Vitamin C (scurvy) – d/t poor diet, smoking</td>
</tr>
<tr>
<td>-Vitamin A: night blindness, xerosis, poor wound healing</td>
</tr>
<tr>
<td>-Riboflavin: neovascularization of cornea, dermatitis, glossitis, cheliosis, angular stomatitis</td>
</tr>
<tr>
<td>-Vitamin C (scurvy): bleeding perifollicular &amp; bruising upper thighs, bleeding gums (2/2 gingivitis)</td>
</tr>
<tr>
<td>-Vitamin D: rickets, osteomalacia, osteoporosis, muscle weakness</td>
</tr>
<tr>
<td>-Vitamin K: bruising &amp; bleeding</td>
</tr>
</tbody>
</table>

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>-Treat underlying cause</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-Replace vitamins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-Vitamin K is SQ administration</td>
</tr>
</tbody>
</table>
| Phenylketonuria | Hereditary (recessive) familial dz  
|                | Deficiency of phenylalanine hydroxylase  
|                | Children often blonde & blue-eyed | After birth, develop vomiting, irritability, convulsion, mental retardation  
|                | Urine has musty odor | Screen 24 h after birth | Diet low in phenylalanine |
## General Characteristics

### Hyperparathyroidism

- Excess secretion of PTH
  - Absorption of calcium from bones, kidneys & GI systems
  - Primary:
    - Middle-aged to older adults, women > men
    - Etiology: hyperfunctioning benign parathyroid adenoma (80-85%); multiple endocrine neoplasia (MEN) 1 & 2a
  - Secondary:
    - D/t chronic renal disease
    - Metastatic bone disease
    - Osteomalacia
    - Multiple myeloma

### Hypoparathyroidism

- Follows parathyroid or thyroid surgery
- Autoimmune, congenital

### Hyperthyroidism

- Graves disease (90% of causes)
  - Women > men, ages 30-60
  - Autoimmune disease – may have +ANA, familial tendency – + incidence of other autoimmune diseases (PA, DM)
  - Toxic nodular goiter – elderly, no eye or skin changes
  - Thyroiditis
  - Drugs – amiodarone (hypothyroidism more common)
  - Thyroid storm:
    - Life threatening

## Symptoms

### Hyperparathyroidism

- Sx vary w/ calcium level
  - Asx to anorexia, N/V, constipation, fatigue, weakness, confusion
  - Polyuria, polydipsia, bone pain & kidney stones
  - “Bones, stones, abdominal groans, psychiatric moans w/ fatigue overtones”

### Hypoparathyroidism

- Acute: circumoral tingling, tetany, muscle cramps, irritability
- Chronic: lethargy, personality changes, blurry vision, mental retardation
- Exam:
  - + Chvostek’s sign (facial muscle spasm)
  - Trousseau’s test (carpal spasm)
  - Hyperactive DTRs

### Hyperthyroidism

- Graves – exophthalmos, pretibial myxedema, goiter (w/ a bruit), lid lag
  - Appetite change, diarrhea, weakness, sweating, wt. loss
  - Exertional SOB, palpitations
  - Fatigue, HA, heat intolerance, hyperactivity, irritability, menstrual disturbances, nervousness, tremor
  - Thyroid storm – high fever, tachycardia, vomiting, diarrhea, dehydration, delirium, CHF

## Diagnostics

### Hyperparathyroidism

- Must rule out other causes of hypercalcemia: **malignancy, multiple myeloma, sarcoidosis**
  - + calcium (>10.5)
  - PTH (essential for dx)
  - + phosphate (<2.5)
  - + urine calcium
  - + urine phosphate (seen in secondary causes d/t renal disease)
  - XR: demineralization, cysts

### Hypoparathyroidism

- + calcium
- + PTH
- + magnesium
- + phosphate
- Normal renal function
- EKG: prolonged QT interval, T wave changes

### Hyperthyroidism

- Graves – • T4, • TSH, positive thyroid-stimulating immunoglobulin (TSI)

## Treatment

### Hyperparathyroidism

- Surgical removal of gland
- Medical treatment of hypercalcemia
- Complications – pathologic fractures, UTI, renal failure

### Hypoparathyroidism

- Maintenance therapy: calcium, vitamin D
- Monitor serum calcium levels
  - Tetany → IV calcium gluconate

### Hyperthyroidism

- Antithyroid drugs (methimazole, propylthiouracil):
  - Inhibit hormone synthesis
  - Watch for low WBC w/ treatment
  - Propylthiouracil preferred in pregnancy
- Radioactive iodine – • thyroid activity – monitor for hypothyroidism
- Beta blockers (propranolol) – tachycardia, tremor, diaphoresis, anxiety & palpitations
- Thyroid storm:
<table>
<thead>
<tr>
<th>Hypothyroidism</th>
<th>- Etiologies:</th>
<th>- Hashimoto’s – painless goiter - Arthralgias, muscle cramps, paresthesias, lethargy, fatigue, perspiration - Cold intolerance, constipation, appetite, memory, depression, dry skin, menstrual disturbances, sleepiness, weight gain</th>
<th>- +Thyroid peroxidase/antimicrosomal/antithyroglobulin antibodies • TSH, • T4</th>
<th>- Thyroid replacement – synthroid → start low w/ elderly or CAD - Monitor TSH – check every 6 wks until stable</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Autoimmune thyroiditis (Hashimoto’s) – most common cause, women &gt; men, middle aged</td>
<td></td>
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<tr>
<td></td>
<td>- Post-ablative hypothyroidism</td>
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<td></td>
<td>- Drug-induced: lithium, sulfonamide, amiodarone</td>
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<tr>
<td></td>
<td>- Iodine deficiency</td>
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<tr>
<td>Thyroid cancer</td>
<td>- History of RT to the neck (papillary or follicular) - Link to MEN 2a &amp; 2b (medullary) - Thyroid function usually normal</td>
<td>- Painless, single, hard mass</td>
<td>- Thyroid scan – cold nodule (non-functioning nodules) - FNA is diagnostic</td>
<td>- Surgery - Radioiodine ablation – not effective in medullary</td>
</tr>
<tr>
<td>Thyroiditis</td>
<td>- Subacute:</td>
<td>- Subacute:</td>
<td>- Subacute:</td>
<td>- Subacute:</td>
</tr>
<tr>
<td></td>
<td>- Most common in women 4th–5th decade</td>
<td>- Acute, painful glandular enlargement w/ dysphagia</td>
<td>- Treat w/ ASA for pain &amp; inflammation - Suppurative (acute):</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Question viral infxn as etiology - Suppurative (acute):</td>
<td>- Gland is woody, hard &amp; tender - Suppurative (acute):</td>
<td>- Antibiotics (acute):</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Rare, caused by pyogenic bacteria</td>
<td>- Very painful, tender, red asymmetrical swelling of the thyroid gland</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>- Treat w/ ASA for pain &amp; inflammation - Suppurative (acute):</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>- Antibiotics &amp; surgical drainage</td>
<td></td>
</tr>
<tr>
<td><strong>Corticoadrenal insufficiency</strong></td>
<td><strong>Weakness, easy fatigability, orthostatic hypotension, anorexia, N/V, diarrhea, wt. loss, hyperpigmentation (only in primary disease)</strong></td>
<td><strong>Hyperkalemia</strong> (primary disease), hyponatremia, hypoglycemia</td>
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<td>---------------------------------------------------------------------</td>
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</tr>
<tr>
<td>- Primary = Addison’s disease</td>
<td>- Causes – autoimmune inflammation of adrenal cortex (most common), TB/fungal infxn, hemorrhage, trauma, metastatic disease</td>
<td>- ACTH (cosyntropin) stimulation test:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Women &gt; men</td>
<td></td>
<td>• Cortisol &lt;20 μg/dL in Addison’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Causes – autoimmune inflammation of adrenal cortex (most common), TB/fungal infxn, hemorrhage, trauma, metastatic disease</td>
<td></td>
<td>• ACTH &gt;200 mg/dL (primary disease)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Causes – autoimmune inflammation of adrenal cortex (most common), TB/fungal infxn, hemorrhage, trauma, metastatic disease</td>
<td></td>
<td>- Low AM plasma cortisol (&lt;3 mg/dL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cushing syndrome</strong></td>
<td><strong>Obesity (centripetal), extremities appear waster, buffalo hump</strong></td>
<td><strong>Dexamethasone suppression test – plasma cortisol &gt;10 μg/dL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Exogenous:</td>
<td><strong>Think skin, acne, hirsutism, amenorrhea</strong></td>
<td><strong>free cortisol in urine (&gt;125 μg/dL in 24 h)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Excess steroid medications</td>
<td><strong>Fatigue, proximal muscle weakness, pigmented striae</strong></td>
<td><strong>ACTH level – elevated in pituitary source of disease &amp; in adrenal source</strong></td>
<td></td>
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</tr>
<tr>
<td>• Endogenous:</td>
<td><strong>libido</strong></td>
<td><strong>Hyperglycemia &amp; hypokalemia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Adrenal hyperplasia – pituitary-hypothalamus dysfunction or adenomas (Cushing disease)</td>
<td><strong>HTN</strong></td>
<td><strong>MRI for pituitary tumor</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Nonendocrine tumors</td>
<td></td>
<td><strong>Transsphenoidal resection &amp; replacement therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Adrenal neoplasia</td>
<td></td>
<td><strong>Radiation therapy</strong></td>
<td></td>
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</tr>
<tr>
<td><strong>Adrenal cancer</strong></td>
<td><strong>Sx caused by the hormones made by the tumor or d/t size</strong></td>
<td><strong>Adrenal inhibitors (this alone is not appropriate):</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Uncommon – 5-15%</td>
<td><strong>CXR</strong></td>
<td>• Metyrapone - inhibits 11β-hydroxylase to • cortisol; may also cause HTN &amp; hypokalemia &amp; hirsutism</td>
<td></td>
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</tr>
<tr>
<td></td>
<td><strong>U/S</strong></td>
<td>• Aminoglutethimide – often caused together w/ metyrapone to • SEs, may cause hypothyroidism by interfering w/ iodine incorporation into thyroid hormone</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>• Ketoconazole – blocks cholesterol side-chain cleavage to • cortisol</td>
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<td></td>
<td></td>
<td>- Oral cortisone &amp; mineralocorticoid</td>
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<tr>
<td></td>
<td></td>
<td>- IV saline, glucose, glucocorticoids (if in crisis)</td>
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<td></td>
</tr>
<tr>
<td>Condition</td>
<td>Description</td>
<td>Symptoms</td>
<td>Treatments</td>
<td></td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------</td>
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</tr>
</tbody>
</table>
| Acromegaly/gigantism | Excess of growth hormone from anterior pituitary (acromegaly in adults, gigantism in children) | - D/t a GH-secreting pituitary macroadenoma (90%) – ectopic production w/ pancreatic, breast or lung tumors is rare  
- Effects bone growth & metabolism  
- Men > women, 3rd-5th decade of life | - Enlargement hands, feet, skull – • in glove & shoe size  
- Space btwn teeth  
- Metabolic: DM, wt. gain  
- Kidney stones  
- HA, visual field defects  
- • insulin-like growth factor (IGF-I) screening test  
- Confirm w/ oral glucose suppression test: if GH level high confirms acromegaly  
- MRI for pituitary tumor | - Surgical removal of tumor is primary treatment  
- Dopamine agonists – cabergoline, bromocriptine  
- Somatostatin analogues – octreotide (inhibits GH secretion)  
→ SEs: diarrhea & cholecystitis  
- GH analogue – pegvisomant, blocks GH binding to its receptor |
| Diabetes insipidus   | Causes • ADH  
- Central: • production of vasopressin from posterior pituitary – d/t tumor, pituitary surgery, sarcoid granulomas, trauma (basilar skull fx)  
- Nephrogenic: • renal response to vasopressin – d/t chronic renal disease, sickle cell, lithium, colchicine | - Polyuria (20L/day)  
- Polydipsia  
- Dehydration, hypotension  
- Low urine-specific gravity (<1.005)  
- Hypernatremia  
- Hyperosmolality  
- Vasopressin challenge test for central DI (will • thirst & polyuria) | - Treat underlying disease  
- Central: desmopressin (DDAVP) intranasal or oral (a vasopressin agonist)  
- Nephrogenic: treat underlying process, give thiazide diuretics, limit renal water loss  
- Monitor sodium levels | |
| Dwarfism             | Prototype is achondroplasia                                                                   | - Short limbs, long narrow trunk, large head w/ midface hypoplasia, prominent brows  
- Delayed motor milestones  
- Normal intelligence  
- Neuro complications: bowing of legs, obesity, dental problems, frequent ear infxs | - Mutation in the FGFR3 gene | - Surgical correction of orthopedic problems  
- GH controversial | |
| Pituitary cancer     | Extremely rare - <200 cases reported  
- Most present as hormone-producing invasive macroadenomas w/ sx of mass effect  
- ACTH & PRL-secreting tumors are most common | - Paralysis of eye muscles causing double or blurred vision  
- Loss of peripheral vision  
- Sudden blindness  
- Facial numbness or pain  
- HA, dizziness | - CT or MRI  
- Test for diabetes insipidus | - Surgery  
- Radiotherapy  
- Chemotherapy |
Most often spreads to brain, spinal cord, meninges or bone around the pituitary

**Pituitary adenoma**
- Benign & slow-growing
- Types:
  - Secreting, functioning or endocrine-active tumors – 50% of adenomas produce too much of one of the hormones; some produce more than one type of hormone leading to hyperprolactinemia, acromegaly or Cushing’s
    - Prolactinomas are most common – 30%
    - Microadenoma (<10 mm)
    - Macroadenoma (>10 mm)
  - Non-functioning or endocrine-inactive
- HA
- Vision problems
- Menstrual cycle changes in women
- Mood swings or behavior changes
- Erectile dysfunction
- Wt. change
- Galactorrhea – PRL secreting tumors
- Blood & urine test for PRL, GH, IGF-1, free thyroxine, LH/FSH, cortisol & testosterone in males
- MRI

**Diabetes mellitus type 1**
- Insulin-dependent, autoimmune disease
- Occurs in young people age <30
- Little or no endogenous insulin

**Complications:**
- DKA – ketones a result of fat metabolism, precipitating factors (infln, inadequate insulin tx, MI (maybe silent), other = trauma, stress, PE, drugs, alcohol)
- **Polyuria, polydipsia, polyphagia**
  - Blurry vision, fatigue, wt. loss
  - DKA – abdominal pain, N/V, anorexia, thirst, tachycardia, fruity odor breath
  - Honeymoon period
  - Seen in IDDM (insulin-dependent) patients
  - Ketoadidosis followed by sx-free period, no tx required
- Fasting blood glucose >126 mg/dL
- Random blood glucose >200 mg/dL
- Glycosylated hemoglobin •
- DKA:
  - glucose & ketones
  - Metabolic acidosis: pH & HCO3
  - Electrolyte abnormalities – potassium, sodium

**Insulin:**

<table>
<thead>
<tr>
<th>Type</th>
<th>Onset (hr)</th>
<th>Peak (hr)</th>
<th>Duration (hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspart Glulisine Lispro</td>
<td>Rapid-acting</td>
<td>0.2-0.5</td>
<td>0.5-2</td>
</tr>
<tr>
<td>Regular</td>
<td>Short-acting</td>
<td>0.5-1</td>
<td>2-3</td>
</tr>
<tr>
<td>NPH</td>
<td>Intermediate</td>
<td>1.5</td>
<td>4-10</td>
</tr>
<tr>
<td>Lente</td>
<td>Intermediate</td>
<td>1.5-3</td>
<td>7-15</td>
</tr>
<tr>
<td>Ultralente</td>
<td>Long-acting</td>
<td>3-4</td>
<td>9-15</td>
</tr>
<tr>
<td>Glargine</td>
<td>Long-acting</td>
<td>No peak</td>
<td>24-36</td>
</tr>
</tbody>
</table>
| **Diabetes mellitus type 2** | - Tissue resistant to insulin  
- Middle-aged & older pts; overweight  
- Complications:  
  - Hyperosmolar hyperglycemic state:  
    - Used to be called hyperosmolar non-ketotic hyperglycemia  
    - Common in the elderly  
    - Glucose markedly elevated >600  
    - Ketone negative  
    - Non-acidotic (pH rarely <7.30, HCO₃ rarely <18)  
    - Signs of dehydration (osmolarity >320)  
  - Polyuria, polydipsia, polyphagia  
  - Fatigue, blurry vision  
  - Somogyi effect  
    - Rebound hyperglycemia in AM in response to counterregulatory hormone release after episode of hypoglycemia in middle of the night  
  - Dawn phenomenon  
    - Early morning rise in plasma glucose requiring amounts of insulin to maintain euglycemia  
  - Fasting blood glucose >126 mg/dL  
  - Random blood glucose >200 mg/dL  
  - Hgb A1c  
| **Complications:**  
- Hyperosmolar hyperglycemic state:  
  - Used to be called hyperosmolar non-ketotic hyperglycemia  
  - Common in the elderly  
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- Somogyi effect  
  - Rebound hyperglycemia in AM in response to counterregulatory hormone release after episode of hypoglycemia in middle of the night  
- Dawn phenomenon  
  - Early morning rise in plasma glucose requiring amounts of insulin to maintain euglycemia  
- Fasting blood glucose >126 mg/dL  
- Random blood glucose >200 mg/dL  
- Hgb A1c  
- HHS: fluids, insulin  
  - Sulfonylureas (glyburide (Micronase), glipizide (Glucotrol))  
  - Pancreas insulin secretion  
  - SEs: wt. gain, hypoglycemia  
  - Biguanides (metformin (Glucophage))  
  - Initial pharmacologic tx after lifestyle modifications  
  - Hepatic glucose production, body wt., improve lipids  
  - SEs: lactic acidosis, diarrhea, nausea  
- α-glucosidase inhibitors (acarbose (Precose))  
  - Delays postprandial digestion of sugars  
  - SEs: nausea & diarrhea  
- Thiazolidinediones (rosiglitazone (Avandia))  
  - Peripheral insulin resistance  
  - SEs: cardiovascular risk  
  - Glucagon-like peptide-1 receptor agonist (exenatide (Byetta))  
  - Enhances glucose-dependent insulin secretion by the pancreatic beta-cell  
  - SEs: thyroid cancer, pancreatitis, renal impairment  
- Insulin  
  - Diet – same as in type I  
  - Diabetic Monitoring  
  - Hgb A1c: target <7%  
  - Estimated average glucose (eAG)  
    - Hgb A1c = 135 mg/dL  
    - Add 35 mg/dL for every 1% change in A1c  
    - eAG = 28.7 x A1c – 46.7  
  - Proteinuria: microalbuminuria  
  - BP: target <130/85 mm Hg  
  - Lifestyle modification: diet & exercise (aerobic & resistance training) |
Hyperlipidemia

- Linked to CAD & CVA – 20% of American adults have hyperlipidemia
- Major lipids: cholesterol, TG, & phospholipids
- Causes:
  - Primary – familial hyperlipoproteinemia
  - Secondary – DM, hypothyroidism, hepatic disease, obesity, drugs
- LDL – risk of atherosclerotic heart dz
- HDL – risk of atherosclerotic heart dz
- TG – associated w/ risk of atherosclerotic heart dz in women & diabetics. levels may lead to pancreatitis
- Major risk factors which modify LDL goals:
  - Cigarette smoking
  - HTN: BP > 140/90 or on antihypertensive meds
  - Low HDL < 40
  - Family hx of premature CHD – men w/ 1st degree relative < 55 & women 1st degree relative < 65
  - > 45 in men or > 55 in women

- No sx until signs of ASVD
- Xanthomas, lipemic blood sample, abdominal pain, hepatomegaly, arcus senilis

- Normal labs:
  - TC: < 200 mg/dL
  - HDL: > 60 mg/dL
  - LDL: < 100 mg/dL
  - TG: < 200 mg/dL

Risk factor modification
- Smoking cessation, wt. loss, exercise
- Diet:
  - Cholesterol intake < 200 mg/dL
  - Carbohydrates 50–60% of total calories
  - Fiber 20–30 g/day
  - Total dietary fat to 30%; saturated fat < 7% of total calories
- ASA daily unless contraindicated
- Bile acid-binding resins: form compounds w/ bile acids in intestine
- SEs: GI distress, constipation
- Contraindication: bowel obstruction
- HMG-CoA reductase inhibitors:
  - Statins: Rhabdomyolysis, Myolysis
  - Cholesterol synthesis
  - SEs: myositis, elevated LFTs
  - Contraindications: pregnancy, active liver disease
- Fibric acids:
  - Gallstones
  - Myopathy
  - Severe renal dz

Drug class | Lipid effects | Side effects | Contraindications
--- | --- | --- | ---
Statins | LDL • HDL • TG | Rhabdomyolysis • Myolysis • LFTs | Acute liver dz
Bile acids binding resins | LDL • HDL • TG | GI distress • Constipation • drug absorption | TG > 400 mg/dL
Niacin & nicotinic acids | LDL • HDL • TG | Flushing • Hyperglycemia • uric acid levels | Chronic liver dz • Severe gout
Fibric acids | LDL • HDL • TG | Gallstones • Myopathy | Severe renal dz • Severe liver dz

PANCE/PANRE Blueprint - EENT

Blepharitis
- Inflammation of the eyelids
- Etiology:
  - Anterior: staphylococcus, viral, or seborrheic infxn
  - Posterior: dysfunction of meibomian glands
- Burning & itching of the eyes
- Eye maybe red
- No change in vision
- Eyelids show scaling & crusting – seborrheic blepharitis scales are greasy
- Supportive – lid scrubs (baby shampoo), warm compresses
- Abx – oral tetracycline or topical E-myacin/bacitracin
### Blowout fracture
- Direct trauma to zygomatic prominence or soft tissue of the orbit – pressure blows out weak orbital floor
- **Double vision** – d/t entrapment of inferior rectus
- Anesthesia anteromedial cheek & upper lip, d/t stretch on infraorbital nerve
- PE: double vision, limited upward gaze
- Plain films
- CT scan
- Surgery may be required

### Cataract
- Lens opacity, usually bilateral
- **Etiology:**
  - Congenital (rubella, CMV)
  - Traumatic
  - Systemic dz (diabetes)
  - Medications (corticosteroids)
  - Senile (most common ages >60)
- **Visual loss, contrast sensitivity, glare**
- Halo around lights
- Surgery

### Chalazion
- Granulomatous inflammation of a meibomian gland
- Chronic disease
- PE: painless, red, hard non-tender swelling of upper of lower lid, can distort vision
- Warm compresses
- Topical abx (erythromycin)
- I&D

### Conjunctivitis
- **Bacterial:**
  - Adults > children
  - Etiology:
    - S. pneumoniae, S. aureus, H. influenzae – transmitted via direct contact, autoinoculation
    - Chlamydia, N. gonorrhoeae (ocular emergency) – contact w/ infected genital secretions (neonate)
  - Children > adults; midsummer to early fall
  - Virus: adenovirus type 3
  - Highly contagious, transmitted by direct contact
  - **Pruritus (severe), bilateral sx**
  - PE: injected, mucoid discharge
- **Viral:**
  - URI, sore throat, fever, malaise (early)
  - Starts unilateral but becomes bilateral in 3-5 days
  - Copious watery discharge, erythema, periauricular lymphadenopathy
  - Allergic:
    - Pruritus (severe), bilateral sx
    - PE: injected, mucoid discharge
- Bacterial:
  - Culture & gram-stain of eye
- Viral:
  - Cool compresses, artificial tears
  - Vasoconstrictor; antihistamine if severe itching
- Allergic:
  - Topical vasoconstrictors or antihistamines
  - Topical mast cell stabilizer
  - Cool compresses

<table>
<thead>
<tr>
<th></th>
<th>Bacterial</th>
<th>Viral</th>
<th>Allergic</th>
</tr>
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<tbody>
<tr>
<td>Itching</td>
<td>Minimal</td>
<td>Minimal</td>
<td>Severe</td>
</tr>
<tr>
<td>Hyperemia</td>
<td>Generalized: bright red</td>
<td>Generalized</td>
<td>Generalized: milky</td>
</tr>
<tr>
<td>Tearing</td>
<td>Moderate</td>
<td>Profuse</td>
<td>Moderate</td>
</tr>
<tr>
<td>Discharge</td>
<td>Perfuse</td>
<td>Minimal</td>
<td>Minimal</td>
</tr>
<tr>
<td>Adenopathy (periauricular)</td>
<td>Rare</td>
<td>Common</td>
<td>None</td>
</tr>
<tr>
<td>Sore throat &amp; fever</td>
<td>Occasionally</td>
<td>Occasionally</td>
<td>None</td>
</tr>
</tbody>
</table>

### Corneal abrasion
- Superficial irregularities of the cornea
- Caused by foreign bodies – injury, welder’s arc, contact lens
- Foreign body sensation, pain, photophobia, redness, blurry vision, small pupil
- PE: check visual acuity first, red eye, fluorescein stain
- Cycloplegics (dilate pupil & relieve pain)
- Topical abx:
  - Erythromycin
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Corneal ulcer</strong></td>
<td>Most commonly due to infection by bacteria, viruses, fungi or amoebas. Noninfectious causes—neurotrophic keratitis, exposure keratitis, severe dry eyes, severe allergic eye disease.</td>
</tr>
<tr>
<td></td>
<td>Pain, photophobia, tearing &amp; reduced vision. Red eye with circumcorneal injection. May have purulent or watery discharge.</td>
</tr>
<tr>
<td></td>
<td>REFER to ophthalmology.</td>
</tr>
<tr>
<td><strong>Dacryoadenitis</strong></td>
<td>Infection of lacrimal gland.</td>
</tr>
<tr>
<td></td>
<td>Acute → swelling, erythema &amp; pain at the lacrimal gland located at the temporal aspect of the upper eyelid. Chronic → madarosis (lash loss), trichiasis (misdirection of lashes), tylosis (thickening &amp; distortion of the lid margin), poliosis (loss of lash pigment), punctual misdirection, scarring.</td>
</tr>
<tr>
<td></td>
<td>I&amp;D, abs (topical bacitracin/erythromycin or systemic tetracycline), warm compresses, systemic analgesics. Lid hygiene.</td>
</tr>
<tr>
<td><strong>Ectropion</strong></td>
<td>Outward turning of the lower lid (Basset-hound look). Elderly.</td>
</tr>
<tr>
<td></td>
<td>Lower lid margin droops outwards, away from the globe. May be excessive tearing but eyes are typically dry.</td>
</tr>
<tr>
<td></td>
<td>Surgery.</td>
</tr>
<tr>
<td><strong>Entropion</strong></td>
<td>Inward turning of the lower lid toward the eye. Elderly.</td>
</tr>
<tr>
<td></td>
<td>Eyelashes seen against the eyeball—foreign body insult, can cause scarring. Redness &amp; tearing.</td>
</tr>
<tr>
<td></td>
<td>Surgery.</td>
</tr>
<tr>
<td><strong>Foreign body</strong></td>
<td>Test visual acuity first. May be on cornea or under upper lid.</td>
</tr>
<tr>
<td></td>
<td>Pain &amp; irritation noted w/ eye movement. Foreign body sensation, red eye, tearing &amp; blepharospasm.</td>
</tr>
<tr>
<td></td>
<td>Topical anesthetic &amp; fluorescein to examine.</td>
</tr>
<tr>
<td></td>
<td>Removal:</td>
</tr>
<tr>
<td></td>
<td>Use sterile saline irrigation first. Remove w/ fine-gauge needle – avoid wet cotton-tip applicator. Polymyxin-bacitracin or erythromycin ointment – avoid steroids. Steel foreign bodies, look for rust ring → remove rust ring at 24 h. Close follow-up, referral for intraocular foreign body.</td>
</tr>
<tr>
<td><strong>Glaucoma</strong></td>
<td>D/t an increased intraocular pressure, which results in optic nerve damage &amp; loss of vision. Types: Primary angle-closure → has a narrow anterior chamber angle.</td>
</tr>
<tr>
<td></td>
<td>Primary open-angle:</td>
</tr>
<tr>
<td></td>
<td>None (early stages).</td>
</tr>
<tr>
<td></td>
<td>Gradual loss of peripheral vision (over a period of yrs.). Results in tunnel vision. Chronic, ax (no halos around lights). Slight cupping of optic disc.</td>
</tr>
<tr>
<td></td>
<td>Primary open-angle:</td>
</tr>
<tr>
<td></td>
<td>Beta-adrenergic blocking agents (timolol or betaxolol) – respiratory or cardiac S/Es. Prostaglandin analogs → no systemic S/Es, lower pressure the greatest.</td>
</tr>
</tbody>
</table>
| Hordeolum                  | -Staph infxn of the meibomian gland (internal) or glands of Zeis or Moll (external)  
|                          | -Stye = external                                                                 |
|                          | -Red, swollen, tender area on upper or lower lid                                   |
|                           | -Warm compresses                                                                  |
|                           | -Abx ointment (E-myacin, bacitracin)                                               |
|                           | -If no improvement in 2 d may have to I&D                                          |
|                           | -Fox shield, place pt at 45 degrees (keeps red cells from staining cornea)         |
|                           | -Avoid ASA & NSAIDs                                                                |
| Hyphema                   | -Hemorrhage into the anterior chamber                                              |
|                           | -Increased risk in sickle cell anemia                                              |
|                           | -Pain, photophobia, decreased visual acuity                                         |
|                           | -Fox shield, place pt at 45 degrees (keeps red cells from staining cornea)         |
|                           | -Avoid ASA & NSAIDs                                                                |
| Macular degeneration      | -Age-related, etiology unknown                                                    |
|                           | -Leading cause of permanent vision loss in the elderly                             |
|                           | -2 groups:                                                                      |
|                           | - Atrophic (dry) – gradual progressive vision loss of moderate severity, retinal pigment atrophy, yellow deposits (drusen)  
|                           | - Exudative (wet) – more rapid onset & greater severity of vision loss, hemorrhages, neovascularization |
|                           | -Central vision loss – test w/ Amsler grid chart                                  |
|                           | -Visual acuity                                                                    |
|                           | -Fundoscopic exam                                                                 |
|                           | -Laser photocoagulation                                                           |
| Nystagmus                 | -May be associated w/ esotropia or w/ ocular lesions                               |
|                           | -Rhythmic oscillation or jiggling of the eyes                                      |
|                           | -Unilater or bilateral, more pronounced in one eye or gaze-dependent               |
|                           | -Electroretinogram to rule out retinal pathology if neuroimaging is normal        |
|                           | -Manage underlying disease                                                         |
| **Optic neuritis** | - Inflammation of the optic nerve  
- Most common etiology is MS:  
  - Occurs in ½ of pts w/ MS  
  - Other etiologies include infxn, autoimmune disorders, IBD, & drug induced (chloramphenicol, ethambutol)  
- Present w/ sudden loss of vision or blurry vision, pain w/ eye movement  
- Fundoscopic exam may be normal or show swelling | - Spasmus nutans → rapid, shimmering, disconjugate nystagmus occurs w/ head bobbing & torticollis (seen w/ glioma) | - Corticosteroids |
| **Orbital cellulitis** | - Periorbital (preseptal):  
  - Infxn of the eyelids & periorcular tissues – anterior to orbital septum  
  - Associated w/ URI  
  - Organisms are S. aureus, S. epidermis, & Strep  
- Orbital (postseptal):  
  - Infxn of the orbital soft tissue – posterior to orbital septum  
  - Spread from paranasal sinusitis  
  - Organisms are S. aureus, S. pneumonia, & anaerobes  
- Periorbital:  
  - Tearing, fever, erythema, warmth, tenderness  
  - Visual acuity, pupillary rxn & EOM are normal  
- Orbital:  
  - Tearing, fever, erythema, warmth, tenderness  
  - Pain w/ eye movement, decreased visual acuity, proptosis | - CT to diagnose both orbital & periorbital | - Periorbital:  
  - Amoxicillin/clavulanic acid  
  - 1st generation cephalosporin  
- Orbital:  
  - Hospitalization, IV abx  
  - 2nd or 3rd generation cephalosporins, ampicillin-sulbactam, carbapenems, clindamycin |
| **Papilledema** | - Optic disk swelling d/t increased intracranial pressure  
- Usually bilateral & most commonly produces enlargement of the blind spot w/o loss of acuity  
- Chronic or severe cases may be associated w/ visual field loss & occasionally profound loss of acuity | - Monitor visual fields carefully | - Acetazolamide & weight loss  
- Consider CSF shunt or optic nerve sheath fenestration if progressive visual failure not controlled by medical therapy |
| **Pterygium** | - Fleshy, triangular encroachment of the conjunctive onto the nasal side of the cornea  
- Tropical climates | | - Excision if vision is threatened |
| **Retinal detachment** | - Tear of the retina that is usually spontaneous – most common location is superior temporal area  
- Mainly ages >50  
- Predisposing conditions: cataract extraction, myopia  
- Blurred vision in one eye becoming progressively worse (“curtain came down over my eye”)  
- Flashers & floaters  
- No pain or redness  
- Exam: retina seen hanging in the vitreous | Retinal artery occlusion:  
- Typical onset ages 50-70  
- Many have hx of CAD, not related to HTN  
- Sudden, painless, profound visual loss (central artery) & marked decrease in visual fields (branch artery) | Retinal artery occlusion:  
- Digital global massage  
- Lower IOP  
- Immediate referral |
| **Retinal vascular occlusion** | - Retinal artery occlusion:  
- Blurred vision in one eye becoming progressively worse (“curtain came down over my eye”)  
- Flashers & floaters  
- No pain or redness  
- Exam: retina seen hanging in the vitreous | Retinal artery occlusion:  
- Typical onset ages 50-70  
- Many have hx of CAD, not related to HTN  
- Sudden, painless, profound visual loss (central artery) & marked decrease in visual fields (branch artery) | Retinal artery occlusion:  
- Digital global massage  
- Lower IOP  
- Immediate referral |
| Retinopathy | -Diabetic:  
- Non-proliferative (early) – dilation of veins, microaneurysms, retinal hemorrhage, hard exudates  
- Proliferative (late) – neovascularization, vitreous hemorrhage, cotton-wool spots  
- Leading cause of blindness in the US  
- Hypertensive:  
  - Affects both retinal & choroidal circulation  
  - Retinal arterioles become tortuous & narrow, abnormal light reflexes (silver-wiring, copper-wiring)  
  - AV nicking  
  - Flame-shaped hemorrhages in the nerve layer of the retina | -Diabetic:  
  - Yearly eye exams & control blood sugar  
- Hypertensive:  
  - Control HTN |
|---|---|---|
| Strabismus | - Misalignment of visual axes of the 2 eyes  
- Ocular muscle weakness or imbalance  
- Should be well aligned by age 2-3 mos.  
- May occur in one eye or both  
- Prevalence in childhood 2-3% | - Diplopia, scotoma, or amblyopia  
- Exam: corneal light reflex & cover/uncover test  
- Exercise, surgery |
| Acute/chronic otitis media | - Acute:  
  - Infxn of the middle ear btwn the Eustachian tube & TM  
  - Usually precipitated by a viral URI  
  - Pathogens – *S. pneumoniae, H. influenzae, M. catarrhalis*, viral → these are the same as bronchitis & sinusitis  
  - More common in infants & children:  
    - Peak age 6-18 mos.  
    - Risk factors – daycare attendance, sibling w/ AOM, parental smoking, bottle drinking  
- Chronic:  
  - Purulent aural discharge  
  - Conductive hearing loss  
  - Tympanocentesis (rarely performed) | - Acute:  
  - 1st line – amoxicillin (if PCN allergic use azithromycin)  
  - 2nd line – amoxicillin/clavulanate, cefaclor, cefixime, erythromycin, TMP/sulf (poor activity against *S. pneumoniae*)  
- Chronic:  
  - Sulfamethoxazole  
  - Amoxicillin  
  - Remove debris, avoid water, use abx drops (Cipro)  
  - Possible surgery |
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Prevention/Management</th>
</tr>
</thead>
</table>
| Breast feeding, pneumococcal     | - Chronic:                                     | - D/t recurrent AOM  
- Perforation of TM is usually present  
- Pathogens – *P. aeruginosa, S. aureus*                                                                                                                                 |
| vaccine  → preventative          |                                                                 |                                                                                                                                              |
| Chronic:                         | - Same clinical picture as Meniere’s disease:  
  - Episodic vertigo lasting 1-8 h, rarely longer than 24 h (horizontal rotational)  
  - Low-frequency sensorineural hearing loss  
  - Tinnitus > blowing quality                                                                                                                  |
| Acoustic neuroma                 | - Benign tumor of the myelin forming cells of the vestibulocochlear nerve (CN VIII)                                                                                                                        |
| Barotrauma                       | - D/t inability to equalize barometric stress exerted on middle ear by air travel, rapid altitudinal change, or underwater diving  
- Most acute during airplane descent  
- TM perforation is an absolute contraindication to diving                                                                                   |
| Cholesteatoma                    | - Variety of chronic otitis media  
- Most common cause is prolonged Eustachian tube dysfunction  
leads to a squamous-epithelium lined sac that can fill w/ desquamated keratin & become chronically infected  
- Typically erode bone w/ early penetration of the mastoid & destruction of the ossicular chain                                                                 |
| Dysfunction of Eustachian tube   | - Air trapped in the middle ear becomes absorbed & creates negative pressure  
- Most common causes are dz associated w/ edema of the tubal lining (URI/allergies)                                                                 |
| Foreign body ear                 | - More frequent in children than adults                                                                                                                                                |
|                                 | - Swallow, yawn & autoinflate frequently during descent  
- Oral decongestants several hours before arrival time  
- Descend slowly into water when diving – avoid diving during URI or nasal allergies                                                                 |
|                                 | - Exam: eptympanic retraction pocker                                                                                                                                                |
|                                 | - Otoscope exam                                                                                                                                             |
|                                 | - Surgical marsupilization of the sac or its complete removal  
→ creation of a “mastoid bowl”                                                                                                                                 |
|                                 | - Aural fullness  
- Fluctuating hearing  
- Discomfort w/ barometric pressure change                                                                                                                                   |
|                                 | - Otoscope – retraction of the TM & decreased mobility on pneumatic otoscopy                                                                                       |
|                                 | - Systemic/intranasal decongestants combined w/ autoinflation  
- Allergic pts may benefit from desensitization or intranasal corticosteroids  
- Air travel, rapid altitude change & underwater diving should be avoided during active phase of disease                                                                          |
|                                 | - Firm materials removed w/ loop or a hook  
- Aqueous irrigation should NOT be performed on organic foreign bodies (beans, insects) because it can cause them to swell                                                                            |
| Hearing impairment | Conductive (middle ear):  
- Problem w/ mechanical reception of amplification of sound  
- Disease in the auditory canal m TM or ossicles  
- Etiology: otosclerosis (fixed stapes, women > men), cerumen impaction, middle ear fluid  
- Sensorineural (inner ear):  
  - Degeneration/destruction of hair cells or CN VIII  
  - Disease in the cochlea, semicircular canal, neuron  
  - Etiology: presbycusis (aging, hair cell loss), noise-induced (chronic exposure), drug-induced (aminoglycosides, Lasix, cisplatin), acoustic tumor, Meniere disease, viral | Conductive:  
- Decreased perception of sound  
- Low frequency tones  
- Weber: sound heard in ear w/ loss  
- Rinne: BC > AC  
- Sensorineural:  
  - Difficulty deciphering words  
  - Tinnitus common  
  - Rinne: AC > BC | Weber & Rinne tests | - To remove bugs fill the ear w/ lidocaine first |

| Hematoma of external ear | - Very important to recognize this to avoid cauliflower ear or canal blockage or chondritis | - I&D – do not drain if >7 days |

| Labrynthitis | - Inflammation of the vestibular labyrinth  
- May take weeks to recover  
- Cause is unknown  
- Frequently follows a URI | - Acute onset of continuous, usually severe vertigo  
- Accompanied by hearing loss & tinnitus | - Typically self-limiting but may require diazepam, meclizine or dimenhydrinate |

| Mastoiditis | - Follow inadequately treated AOM (most common complication of AOM)  
- Complications – hearing loss (monitor hearing), labrynthitis, vertigo, facial nerve paralysis | - Postauricular pain & erythema, fever, bulging TM  
- XR: coalescence of mastoid air cells d/t destruction of bony septa | - IV abx – ampicillin, cefuroxime  
- Myringotomy  
- Possible surgical drainage – mastoidectomy  
- Complications – hearing loss (monitor hearing), labrynthitis, vertigo, facial nerve paralysis |

| Meniere disease | - D/t distention of the endolymphatic compartment of inner ear – cause unknown  
- Peak onset ages 40-60 | - Episodic vertigo lasting 1-8 h, rarely longer than 24 h (horizontal rotational)  
- Low-frequency sensorineural hearing loss  
- Tinnitus > blowing quality  
- ^ These 3 above are the classic triad | |

| Otitis externa | - Infxn of external auditory canal  
- Hx of water exposure (swimmer’s ear) or mechanical trauma | - Otolgia, pruritus, purulent drainage  
- If brown/yellow discharge w/ strong odor think cholesteatoma | - Otic aminoglycosides – neomycin sulfta, polymyxin B sulfate  
- Corticosteroids |
Tinnitus

- Perception of abnormal ear or head noises
- Persistent tinnitus often indicates the presence of sensory hearing loss
- Mild, high-pitched tinnitus lasting seconds-minutes is common in normal hearing persons
- Pulsatile may indicate a vascular abnormality
- Clicking may result from middle ear muscle spasm

- Erythema, edema of ear canal skin, pain w/ movement of auricle
- Periauricular lymphadenopathy

- MRA or MRV if pulsatile
- Avoid exposure to excessive noise, ototoxic agents & other factors that may cause cochlear damage
- Masking tinnitus w/ music & a hearing aid may bring relief
- Oral antidepressants are very effective

Vertigo

- BPPV:
  - Very common cause of dizziness – 20% of all cases, common in pts >50, common cause is head trauma
  - D/t debris in inner ear

- Dizziness, vertigo, lightheadedness, imbalance, nausea
- 5x brought on by change in position of the head (tipping head back)
- Episodes last less than 1 minute
- Dizziness triggered by lying down or rolling over

- Dix-Hallpike test positive:
  - Move patient from sitting to supine w/ head turned 45 degrees to one side & 20 degrees backward
  - Positive is burst of nystagmus

- Epley maneuver:
  - Called particle repositioning
  - Involves moving the head into 4 positions & staying at each for 30 seconds
  - May cause weakness, numbness & visual changes

Vertigo Summary

<table>
<thead>
<tr>
<th>Sign/symptoms</th>
<th>Central</th>
<th>Peripheral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
<td>Mild, gradual</td>
<td>Marked, sudden</td>
</tr>
<tr>
<td>Tinnitus/deafness</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

Nystagmus

<table>
<thead>
<tr>
<th>Vertical</th>
<th>Occasional</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Duration</td>
<td>Variable/chronic</td>
<td>Finite/intermittent</td>
</tr>
<tr>
<td>CNS signs</td>
<td>Common</td>
<td>None</td>
</tr>
<tr>
<td>Etiologies</td>
<td>Vascular</td>
<td>Infxn</td>
</tr>
<tr>
<td></td>
<td>Neoplasm</td>
<td>Meniere's</td>
</tr>
<tr>
<td></td>
<td>Multiple sclerosis</td>
<td>Trauma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Toxin</td>
</tr>
</tbody>
</table>

Acute/chronic sinusitis

- Acute:
  - D/t impaired mucociliary clearance & obstruction of the ostiomeatal complex
  - Accumulation of mucous secretions & edema
  - Sx for less than 4 weeks

- Acute/chronic:
  - Sx > 10 days
  - Pain & pressure over sinus
  - Discolored, purulent nasal discharge
  - Fever, malaise, headache & tooth pain

- Acute/chronic:
  - XR: opacification, air-fluid levels, thick mucosa – not routinely indicated
  - CT: bone destruction, air-fluid levels, thick mucosa, test of choice

- Acute/chronic:
  - Oral decongestants – pseudoephedrine
  - Nasal decongestants – oxymetazoline
  - Abx: Augmentin or doxycycline first choice, levofloxacin,
- Maxillary most common, followed by ethmoid, frontal & sphenoid (frontal develops as young child & sphenoid early 20s)
- Pathogens – viral (rhinovirus, parainfluenzae, influenzae, RSV), bacterial (*S. pneumoniae*, *H. influenzae*, & *M. catarrhalis*), fungal (*Rhizopus, Mucor, Aspergillus* (immunocompromised))
- Complications – osteomyelitis, cavernous sinus thrombosis, orbital cellulitis
  - Chronic:
  - Chronic nasal congestion, cough, postnasal drip, fever is rare
  - Lasts >12 weeks
  - Bacteria: *S. aureus*, anaerobes, *H. influenzae*, fungi
  
  **PE:** TTP over sinus, opacification of the sinus w/ transillumination
  
  **candamycin 2nd choice**
  - Macrolides & TMP/sulfa not indicated as empirical therapy d/t increasing antimicrobial resistance
  - Treat for at least 10-14 d; 2-3 weeks for chronic
  - Analgesics
  - Drain sinus
  - Consider foreign body in chronic cases

### Allergic rhinitis

- D/t airborne allergenic particles which initiate & IgE-mediated response
- Seasonal (= hayfever → ragweed, grass, tree pollen) or perennial (house dust mites, animal dander, mold)

- Sneezing, nasal secretions, nasal congestions
- Itching eyes, postnasal drip, cough
- Worse in AM
- Exam:
  - Edematous mucosa that is pale or violaceous
  - Clear secretions
  - Nasal polyps
  - Watery eyes
  - Allergic shiners
  - Allergic salute

- Eosinophils on nasal smear
  - Allergen avoidance
  - Immunotherapy
  - Antihistamines:
    - 1st generation H1 blockers (sedating) → diphenhydramine, hydroxyzine
    - 2nd generation H1 (non-sedating) → loratidine, fexofenadine, cetirizine
  - Sympathomimetics → ephedrine, pseudoephedrine
  - Topical steroids → persistent sx
  - Mast cell stabilizer → cromolyn sodium

### Epistaxis

- Causes: trauma, dry nasal mucosa, rhinitis, medications (cocaine), allergic conditions
- Anterior:
  - Arises from anterior nasal septum (venous blood)
  - Site of bleeding easily seen
  - Unilateral, continuous, recurrent
- Posterior:
  - Intermittent, very brisk
  - Requires endoscopy instruments for localization
  - Bleeding occurs from both nares (lateral wall)

- Trauma, bleeding disorder, nasal discharge

- Direct pressure, ice packs
- Sitting & leaning forward
- Nasal decongestants for vasoconstriction
- Cauterization – silver nitrate
- Nasal packing 24 h:
  - Anterior: petroleum packing
  - Posterior: sponge pack, balloon tamponade
  - Abx prophylaxis: cephalexin, Augmentin, clindamycin, Bactrim
<table>
<thead>
<tr>
<th>Nasal foreign body</th>
<th>Nasal polyps</th>
<th>Acute pharyngitis</th>
<th>Aphthous ulcers</th>
<th>Diseases of the teeth/gums</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding seen in posterior pharynx</td>
<td>Most common locations for objects to lodge are just anterior to the middle turbinate or below the inferior turbinate</td>
<td>Types: GABHS, viral (herpes, coxsackievirus A), Epstein-Barr, <em>Corynebacterium diphtheriae</em>, <em>N. gonorrhoeae</em>, <em>mycoplasma</em></td>
<td>Canker sore</td>
<td>Most common chronic childhood disease</td>
</tr>
<tr>
<td>- Right side more common than left</td>
<td>-Polyloid masses from mucous membranes of nose &amp; paranasal sinuses</td>
<td>-Types: GABHS, viral (herpes, coxsackievirus A), Epstein-Barr, <em>Corynebacterium diphtheriae</em>, <em>N. gonorrhoeae</em>, <em>mycoplasma</em></td>
<td>-Cause unknown but trauma most common trigger</td>
<td>-Most common chronic childhood disease</td>
</tr>
<tr>
<td>- Complications:</td>
<td>-Frequently accompany allergic rhinitis</td>
<td>-Most commonly 5-18 yrs. old</td>
<td>-Initial tingling or burning sensation at site</td>
<td>-Most common chronic childhood disease</td>
</tr>
<tr>
<td>- Bleeding</td>
<td>-Freely movable, non-tender</td>
<td>-Types: GABHS, viral (herpes, coxsackievirus A), Epstein-Barr, <em>Corynebacterium diphtheriae</em>, <em>N. gonorrhoeae</em>, <em>mycoplasma</em></td>
<td>-Progresses to form red spot or bump, followed by an open ulcer</td>
<td>-Simple or marginal cases can be treated w/ tooth brushing &amp; flossing</td>
</tr>
<tr>
<td>- Sinusitis</td>
<td>-Associated conditions: chronic rhinosinusitis, asthma, ASA intolerance, CF, Kartagener's syndrome</td>
<td>-Streptococcal → acute onset fever, exudates posterior pharynx, odynophagia, rhinitis, cervical adenopathy</td>
<td>-Appears as white or yellow oval w/ inflamed red border</td>
<td>-Fluoride for prevention</td>
</tr>
<tr>
<td>- AOM</td>
<td>-Viral → more insidious onset, coryza, no exudate, low-grade fever</td>
<td>-Throat culture</td>
<td>-Analgesic, anesthetic agents, antiseptics, anti-inflammatory agents, sucralfate, silver nitrate</td>
<td>-Proper oral hygiene</td>
</tr>
<tr>
<td>- Nasal septal perforation</td>
<td>-Epstein-Barr → malaise, tender adenopathy, enlarged spleen, jaundice, exudative pharyngitis</td>
<td>-Rapid strep screen</td>
<td>-Gingivitis:</td>
<td>-Dental sealants</td>
</tr>
<tr>
<td>- Pressure necrosis</td>
<td>-Streptococcal → acute onset fever, exudates posterior pharynx, odynophagia, rhinitis, cervical adenopathy</td>
<td>-Monospot/reactive lymphs for mono</td>
<td>-Simple or marginal cases can be treated w/ tooth brushing &amp; flossing</td>
<td>-Gingivitis:</td>
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<tr>
<td>Nasal speculum exam</td>
<td>Nasal block, sinusitis, loss of smell</td>
<td>PCN, erythromycin, cephalexin, azithromycin, clarithromycin</td>
<td>-Avoid amoxicillin &amp; contact sports in mono</td>
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<td>Removal w/ adequate sedation if necessary</td>
<td>Steroids</td>
<td>Supportive if viral</td>
<td>OTC mouth rinses such as chlorhexidine gluconate BID – caution can cause staining of the</td>
<td>Avoid amoxicillin &amp; contact sports in mono</td>
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<td>Balloon catheter removal for poorly visualized objects</td>
<td>Surgery</td>
<td>Positive pressure techniques for occlusive foreign bodies</td>
<td>Diseases of the teeth/gums</td>
<td>Fluoride for prevention</td>
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<td>-Dental caries:</td>
<td>Proper oral hygiene</td>
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<td><strong>Periodontal disease:</strong></td>
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<td>- Life threatening supraglottitis/epiglottic infxn</td>
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<td>- May result in acute airway obstruction</td>
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<td>- Fever</td>
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<td>- Dysphagia</td>
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<td>- Respiratory distress/stridor</td>
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<td>- Cherry-red epiglottis on laryngoscopy</td>
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<td>- Secure airway – child should not be disturbed until personnel is present &amp; ready to perform intubation or tracheotomy</td>
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**cariogenic bacteria** – *Streptococcus mutans*

- Periodontal disease:
  - Most common oral dz in adults
  - Risk factors: poor oral hygiene, crowded teeth, mouth breathing, steroids, smoking, diabetes, weakened immune status, low income
  - 3 types: gingivitis, chronic periodontitis & aggressive periodontitis
- Gingivitis:
  - Reversible inflammatory process from prolonged exposure to plaque
  - ANUG – acute necrotizing ulcerative gingivitis (Vincent dz or trench mouth) – associated w/ anaerobic fusiform bacteria & spirochetes
- Periodontitis:
  - Chronic cases are caused by chronic inflammation of gingival soft tissue & supporting structure by plaque microorganisms (gram-negative)
  - Found in 50% of the population
  - Reversible if treated in early stages (minimal pockets btwn the tooth & periodontal attachment
  - Severe gum disease = 6 mm loss of attachment of the tooth to adjacent gum tissue
  - In kids think about underlying DM, Down syndrome, hypophosphatasia, neutropenia, leukemia, leukocyte adhesion deficiency or histiocytosis
  - Localized juvenile periodontitis & localized prepubertal periodontitis are d/t *Actinobacillus actinomycetemcomitans*

- ANUG – halitosis, bleeding gingival tissue, edema, ulcers, pain
- Periodontal disease:
  - Severe – tartar, gum recession & loose teeth
  - Children <4 = loss of primary teeth, usually first sign & the systemic manifestation of hypophosphatasia

Teeth in pts who drink coffee, tea or red wine

- Periodontitis:
  - Professional care to remove tartar & may require periodontal surgery
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<tr>
<th>Condition</th>
<th>Description</th>
<th>Diagnosis/Management</th>
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| **HIB**                                        | Caused by Strep pyogenes or Staph aureus or Mycoplasma  
- HIB is uncommon in North America as a result of immunization                                                                                 | - Symptoms may overlap croup but toxicity suggest epiglottitis  
- Severe sore throat, drooling, absence of hoarseness, child's insistence on sitting forward w/ neck hyperextended  
- Tongue depressor or exam of oropharynx may cause acute airway obstruction  
- Lateral neck XR: thumb sign – enlarged epiglottis  
- Once airway secure obtain blood cultures, CBC & culture of epiglottis | - Abx therapy: IV ceftriaxone or cefotaxime for 7-10 d  
- Prevention: HIB vaccine, rifampin to eliminate carriers & treat close contacts                                                                 |
| **Laryngitis**                                 | Most common cause of hoarseness  
- May persist for a week or so after other sx clear  
- Viral in origin, but M catarrhalis & H influenzae may be isolated                                                                             | - Avoid vigorous use of the voice since persistent use can lead to formation of traumatic vocal fold hemorrhage, polyps, & cysts  
- Erythromycin, cefuroxime, or Augmentin may reduce severity of hoarseness & cough                                                                 |
| **Oral candidiasis**                           | Also called thrush  
- Etiology: Candida albicans  
- Seen in infants, denture wearers, those w/ diabetes & HIV, & those taking chemo or abx                                                                 | - Wet prep/KOH prep  
- Nystatin  
- Clotrimazole troches                                                                                                                        |
| **Oral herpes simplex**                        | Transmitted by direct contact  
- Etiology: HSV 1 & 2 – 1 is most common  
- After infxn, the virus migrates to the sensory or autonomic ganglia & becomes dormant  
- Reactive w/ stress, UV radiation & foods                                                                                       | - Viral culture  
- Tzanck smear  
- Acyclovir, valacyclovir, famciclovir  
- Suppression therapy  
- Avoid contact                                                                                                                  |
| **Oral leukoplakia**                           | White striated or corrugated mouth lesion  
- Most commonly ages >40  
- Caused by EBV or human papillomavirus  
- Risk factors: tobacco, alcohol, oral infxn  
- Check HIV status & rule out malignancy                                                                                              | - Asx  
- Exam: white corrugated lesion on tongue  
- Sides of tongue, soft palate, floor of mouth  
- Not able to be scraped away (bleeds)  
- Biopsy  
- Hairy leukoplakia → acyclovir  
- Leukoplakia → isotretinoin                                                                                                              |
| **Peritonsillar abscess**                      | Abscess formation btwn anterior & posterior tonsillar pillars & the superior pharyngeal constrictor muscles  
- Complication of tonsillitis, peritonsillar cellulitis, & mononucleosis  
- Infxn extend into tonsil through capsule  
- Avg. ages >30  
- Etiology: polymicrobial (anaerobic)                                                                                         | - Severe sore throat, fever, odynophagia  
- Exam: anterior chain adenopathy, drooling, trismus, “hot potato” voice, asymmetry of oropharynx w/ uvula deviation  
- Leukocytosis  
- CT: abscess & edema  
- Fluid for aspiration, culture & Gram stain  
- Surgical drainage/tonsillectomy  
- Abx: PCN or erythromycin/cephalexin if allergic, metronidazole or clindamycin                                                                 |
| **Parotitis** | **Viral in origin** – leading cause was mumps but since vaccination it is parainfluenza & EBV  
-Suppurative cases in newborns & debilitated elderly | **Suppurative** – swollen, tender & often erythematous gland unilaterally | **Suppurative** – dx made after expression of purulent material from Stensen’s duct  
-Culture | -If suppurative → IV abx |
| **Sialadenitis** | -Infxn, of salivary gland – parotid, submandibular  
-Acute swelling & pain w/ meals, tender & erythema at duct opening  
-Usually d/t mucus plug w/ secondary infxn → mumps, S. aureus, post 131-I therapy for thyroid cancer | **Biopsy** | -IV abx – nafcillin  
-Increased salivary flow (sialogogues – lemon drops)  
-Massage of the gland |
| **Benign & malignant neoplasms** | -Retinoblastoma:  
- Inherited or sporadic  
- Congenital malignancy: lack of tumor suppressor gene  
- Absent red reflex  
- "White pupil"  
- Life-threatening – refer to ophthalmology  
-Oral/oropharyngeal cancer:  
- 3% of cancers in men & 2% in women  
- Overall survival is 51%  
- Most are squamous cell  
- Leukoplakia is a precancerous lesion  
- Commonly occur on the tongue (most common), lips (lower lip vermilion carcinoma) & the floor of the mouth  
- Tobacco & ETOH are responsible for 75% of these cancers | -Retinoblastoma:  
- Leukocoria – loss of normal red reflex is most common sign (50% of cases)  
- Strabismus  
- Decreased visual acuity  
- Unilateral fixed pupil  
- Red, painful eye  
-Oral/oropharyngeal cancer:  
- Leukoplakia – white or red patch that progresses to a superficial ulceration of the mucosal surface  
- Solitary lumps  
- Larger cancers may be painful & erode underlying tissue  
- Actinic cheilosis – precursor to lip vermilion carcinoma – dry, scaly changes progress to healing ulcer  
- Dysphagia, painful swallowing & referred pain to the ear | -Oral/oropharyngeal cancer:  
- Scalpel or small biopsy forceps to biopsy any nonhealing white or red lesion that persists for >2 wks.  
- CT for staging | -Retinoblastoma:  
- Vision-sparing modalities – cryotherapy, brachytherapy, laser therapy &/or chemo  
-Oral/oropharyngeal cancer:  
- Avoid all forms of tobacco (cigarette, pipe, chewing, smokeless) & ETOH  
- Primary screening includes inspection & palpation of extraoral & intraoral tissues  
- Chemo/surgery/radiation |
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<th><strong>PANCE/PANRE Blueprint - Cardiology</strong></th>
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| Atrial fibrillation | IV – pts confined to bed or chair; any physical activity brings on discomfort & sx at rest | -Common sustained arrhythmia in adults  
- risk w/ age  
- risk of intra-atrial clot formation | -Rate control – beta blockers, CCBs  
-Rhythm control – amiodarone, flecainide (+ SEs)  
-Cardioversion – as long as there are no clots!  
-Anticoagulation  
-Catheter ablation |
|-------------------|--------------------------------------------------------------------------------|---------------------------------------------------------------------------------|--------------------------------------------------------------------------------|
| Atrial flutter    | -Associated w/ COPD, PE, thyrotoxicosis, mitral valve disease, alcohol  
-Symptoms: dizziness, palpitations, chest pain, dyspnea | -Rapid atrial rate 250-300 bpm  
-Baseline between QRS contained jagged waves (sawtooth pattern) | -Cardioversion – if unstable  
-Rate control:  
- Acute – BB, CCBs  
-Chronic – amiodarone, sotalol, quinidine, procainamide  
-Ablation of foci |
| AV block          | -First degree heart block:  
- PR interval >0.20 s & constant  
- Etiology – aging, digitalis, ischemia, inflammation, cardiomyopathies  
-Mobitz I/Wenckebach:  
- Second-degree block  
- Progressive lengthening of the PR interval until a QRS complex fails to appear after a P wave  
- Repetitive  
- Think acute inferior wall infarction  
-Mobitz II:  
- Second-degree heart block  
- An AV block w/ failed conduction of a beat w/ constant PR intervals  
- Think acute anterior or anteroseptal MI  
-Third degree heart block:  
- Complete absence of conduction of the electrical impulses through the AV node, Bundle of His, or bundle branches  
- Characterized by independent beating of the atria & ventricles | First degree heart block:  
Mobitz I/Wenckebach:  
Mobitz II:  
Third degree heart block: | -Third degree heart block: pacemaker |
- Etiology: aging, inferior or posterior infarction, digitalis

**Bundle branch block**
- Right bundle branch block:
  - QRS > 0.12 s
  - QRS predominantly positive in V1 or rSR' or rsR' in lead V1
  - Wide S in lead I & V6
- Left bundle branch block:
  - QRS > 0.12 s
  - QRS predominantly negative in V1
  - Upright QRS in leads I & V6 → QRS may be notched

**Paroxysmal supraventricular tachycardia**
- Reentry AV tachycardia
- Common in the elderly w/ underlying heart dz
- Symptoms: palpitations, anxiety
- Rate: 150-180 bpm
- Atrial activity typically not noted

**Premature beats**
- PVC:
  - May originate from anywhere in the ventricles
  - QRS is 0.12 seconds or longer & resembles a LBBB or RBBB
- PAC:

**Vagal maneuver**
- Drugs:
  - Adenosine (DOC) or verapamil
  - Beta-blockers
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<td>Sick sinus syndrome</td>
<td>- Also called sinus node dysfunction&lt;br&gt;- Relatively uncommon syndrome&lt;br&gt;- Can results in sinus arrest, sinus node exit block, sinus bradycardia&lt;br&gt;- Also associated w/ tachycardias such as PSVT &amp; a. fib&lt;br&gt;- Often caused or worsened by meds – digitalis, CCBs, BB, sympatheticic meds, &amp; anti-arrhythmics</td>
<td>- Stokes-Adams attacks&lt;br&gt;- Dizziness&lt;br&gt;- Palpitations&lt;br&gt;- Chest pain&lt;br&gt;- SOB</td>
<td>- EKG w/ a variety of results&lt;br&gt;- Pacemakers for bradycardia &amp; meds for tachycardias</td>
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<tr>
<td>Ventricular tachycardia</td>
<td>- Originates from below the bundle of His&lt;br&gt;- Precipitating causes:&lt;br&gt;  - Electrolytes imbalance&lt;br&gt;  - Acid-base changes&lt;br&gt;  - Hypoxemia&lt;br&gt;  - MI&lt;br&gt;- Symptoms:&lt;br&gt;  - Stable – none w/ short runs of VT&lt;br&gt;  - Unstable – long runs present w/ syncope, CP, dyspnea</td>
<td>- QRS complex loses sharp peak &amp; becomes wide &amp; bizarre in appearance, though somewhat uniform&lt;br&gt;- All other waves absent</td>
<td>- Amiodarone, lidocaine, procainamide if unstable&lt;br&gt;- Cardioversion may be required</td>
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<tr>
<td>Ventricular fibrillation</td>
<td>- Life-threatening&lt;br&gt;- Uncordinated electrical activity&lt;br&gt;- Disorganized electrical activity noted in pts w/ ischemic heart dz &amp; ventricular dysfunction</td>
<td>- Defibrillation</td>
<td>- Amiodarone, lidocaine, procainamide if unstable&lt;br&gt;- Cardioversion may be required</td>
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<td>Torsades de pointes</td>
<td>- Etiology:&lt;br&gt;  - Antiarrhythmic drugs (quinidine, procainamide)&lt;br&gt;  - Psychotropic drugs (phenothiazine, TCA, lithium)&lt;br&gt;  - Electrolyte imbalance&lt;br&gt;  - Subarachnoid hemorrhage</td>
<td>- Polymorphic ventricular tachycardia&lt;br&gt;- Long QT interval</td>
<td>- Lidocaine&lt;br&gt;- Phenytoin&lt;br&gt;- Correct underlying cause</td>
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<td>Atrial septal defect</td>
<td>- Defect in atrial septum (in region of fossa ovalis)&lt;br&gt;- Shunting of blood</td>
<td>- Typically no sx&lt;br&gt;- If severe possible DOE&lt;br&gt;- Physical exam:</td>
<td>- CXR – cardiomegaly&lt;br&gt;- EKG – right ventricular hypertrophy, RAD, RBBB&lt;br&gt;- Spontaneous closure likely in first year of life</td>
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<td>Condition</td>
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<td>Radiological Findings</td>
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| **Coarctation of aorta**        | -Narrowing in descending aorta – most at site of ductus arteriosus  
-Men > women **if patient is a woman think Turner’s syndrome**                                                                                       | -Physical exam:  
- **Weak or absent femoral pulses**  
- Femoral pulse delayed when compared to radial pulse  
- Systolic pressure higher in upper extremities (diastolic pressure the same) | -CXR – enlarged aortic knob, rib notching  
-EKG – RVH in neonate & LVH in older children  
-Echo – aortic obstruction | -Prostaglandin E1  
-Balloon angioplasty or surgery |
| **Patent ductus arteriosus**    | -Ductus arteriosus remains open  
- Connects aorta to left pulmonary artery  
- Typically closes spontaneously by 4 days of age  
- W/ drop in pulmonary resistance, left to right shunting of blood occurs  
- Increased risk in premature infants | -Symptoms vary w/ degree of shunting:  
- Small shunts – no sx  
- Large shunts – signs of CHF, slow growth  
- Continuous, machine-like murmur in the left infraclavicular area (radiated to left back)  
- Wide pulse pressure  
- Bounding arterial pulses | -CXR – large shunt shows cardiomegaly, LAH, LVH  
-EKG – presence of LVH  
-Echo – presence of PDA | -Indomethacin – prostaglandin E1 levels – works best in preterm infants  
-Surgery |
| **Tetralogy of Fallot**         | -Most common congenital heart disease  
- Due to left to right shunting & pulmonary flow  
- Four defects:  
- Ventricular septal defect  
- Right ventricular outflow obstruction  
- Right ventricular hypertrophy  
- Overriding large ascending aorta | -Degree of cyanosis is based on degree of obstruction (another cyanotic disorder is Transposition of Great Vessels)  
- Neonates – cyanosis & agitation  
- Physical examination:  
- RV heave  
- Loud systolic ejection murmur left sternal border | -CXR – boot-shaped heart d/t hypertrophy of right ventricle  
- EKG – right axis deviation, RVH  
- Echo: thick RV wall, overriding of the aorta, ventricular septal defect | -Surgical repair at 3-12 months  
- PGE, infusion to prevent ductal closure (if cyanotic at birth) |
| **Ventricular septal defect**   | -Most common congenital defect  
- Communication between right & left ventricles  
- May develop pulmonary HTN  
- No sx until 6-8 weeks old d/t pulmonary vascular resistance | -Sx vary w/ the size of defect & severity of pulmonary vascular resistance – tachypnea, tachycardia, poor weight gain, trouble feeding  
- Physical examination:  
- Harsh pansystolic murmur lower left sternal border, grade 2-4/6; systolic thrill (louder w/ smaller defects (diamond shaped murmur)) | -Echo – presence of defect  
- Cath – only way to measure pulmonary vascular resistance | -Small defects (shunts): defer until late childhood since many will spontaneously close  
- Large defects require surgery  
- Abx prophylaxis (mandatory) |
### Essential Hypertension

- Incidence in adults is 20-25%
- End organ damage is common
  - Cardiac: LVH, angina/prior MI, heart failure, aortic aneurysm
  - Cerebrovascular: stroke/TIA
  - Renal proteinurin (nephropathy)
  - Retinopathy

### Secondary Hypertension

- Due to an identified cause:
  - Obstructive sleep apnea
  - Drug-induced or related causes
  - Chronic kidney disease
  - Primary aldosteronism
  - Renovascular disease
  - Cushing’s syndrome
  - Pheochromocytoma
  - Coarctation of the aorta
  - Thyroid or parathyroid disease

- When to suspect:
  - Onset ages <30 or >50
  - Sudden worsening of previously controlled HTN
  - Failure to respond to therapy
  - Hypokalemia (w/o diuretics): primary aldosteronism
  - Labile HTN
  - Renal failure after ACEI

### Hypertensive Emergencies

- Severe HTN w/ severe & rapidly worsening sx of end-organ damage

- Neurologic: encephalopathy, HA, confusion, seizures

- CT brain
- EKG
- Electrolytes

- Treat underlying cause
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<th><strong>Cardiogenic shock</strong></th>
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<th><strong>Renal function</strong></th>
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<tr>
<td>- Tissue hypoperfusion d/t acute MI or end-stage heart failure</td>
<td>- SBP &lt;90 mmHg or • from baseline of 30 mmHg</td>
<td>- Echo: • LV function</td>
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<td>- Overall prognosis poor</td>
<td>- Altered mental status, cyanosis, oliguria</td>
<td>- Improve pressure w/ IVF or vasopressor agents</td>
</tr>
<tr>
<td>- Etiologies:</td>
<td>- Physical exam:</td>
<td>• Dopamine: • cardiac output &amp; BP</td>
</tr>
<tr>
<td>- Acute MI</td>
<td>• Cool, clammy extremities, signs of hypovolemia</td>
<td>Dobutamine: • cardiac output but not BP</td>
</tr>
<tr>
<td>- Tachyarrhythmia</td>
<td>Findings of underlying condition</td>
<td></td>
</tr>
<tr>
<td>- Valvular heart disease</td>
<td></td>
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<tr>
<td>- Traumatic cardiac injury</td>
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<tr>
<td>- Myocarditis</td>
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<tr>
<td><strong>Orthostatic hypotension</strong></td>
<td></td>
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<tr>
<td>- Decrease in SBP of 20mmHg or DBP of 10 mmHg when moving from recumbent to standing position</td>
<td>- Change in mental status, confusion</td>
<td></td>
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<tr>
<td>- May result in syncope</td>
<td>- Weak pulse</td>
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</tr>
<tr>
<td>- Etiologies:</td>
<td>• Tachypnea</td>
<td></td>
</tr>
<tr>
<td>- Drugs (antipsychotics, diuretics, alpha blockers, ACEI, alcohol, tranquilizers, vasodilators)</td>
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<tr>
<td>- Polyneuropathies</td>
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<tr>
<td>- Parkinson’s disease</td>
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</tr>
</tbody>
</table>
**STEMI vs. NSTEMI**

- 50% of pts have identifiable factor – very heavy exercise, severe mental stress
- Often preceded by periods of unstable angina
- Most deaths occur w/in 1h of onset d/t v. fib
- ST elevation & Q waves are the 2 most characteristic features of AMI → seen in only 50% of pts at presentation
- If EKG normal → <10% chance of MI
- ST elevation:
  - Injury pattern
  - Occurs w/ transmural ischemia
  - DDX: pericarditis, aneurysm, LVH, early repolarization
- ST depression:
  - Occurs w/ subendocardial ischemia
  - More often seen in NQMI
  - DDX: hypertrophy, conduction/ electrolyte abnormality, drug effects

- STEMI vs. NSTEMI:
  - STEMI
    - Occulsive thrombus
    - Complete & prolonged occlusion of an epicardial coronary blood vessel
    - Causes full thickness (transmural) damage of heart muscle
    - Defined based on ECG criteria
  - NSTEMI
    - Non-occlusive thrombus
    - Results from severe coronary artery narrowing, transient occlusion or microembolization of thrombus &/or atheromatous material
    - Elevation of cardiac biomarkers w/o ST elevation
    - Causes partial thickness damage of heart muscle

- Pain:
  - Usually severe & intolerable; retrosternal (may radiate to arm, neck & jaw)
  - Prolonged: 20 min. to hours
  - Quality: crushing, constricting, compressing, oppressing
  - Caused by ischemia, not infarction
  - Other sx: N/V in 50% (usually inferior MI), weakness, dizziness, palpitations, cold sweat, sense of impending doom
- May have elevated BP, tachycardia & presence of S4

- Labs: leukocytosis (12-15K), lipid profile, serum cardiac markers
- Creatinine kinase:
  - Exceeds normal range w/in 3-6 h, normalizes in 2-4 d, & peaks at 24 h
  - Check serially every 8-24 h
  - May elevate 10-20x normal limit in AMI
- Isoenzymes: MM (skeletal muscle), BB (brain, kidney), MB (myocardium, small intestine, tongue, diaphragm, uterus, prostate)
- Ratio of CPK-MB to total CPK >4% is diagnostic
- Troponin:
  - Troponin (I or T) has nearly absolute myocardial tissue specificity/sensitivity (T • in renal dz, polymyositis, dermatomyositis)
  - May elevate >20x normal range
  - Begins to rise 2-4 h post-AMI, peaks at 10-24 h & may persist for 5-12 d
  - Aids in picking up AMI in pts who present late
- Myoglobin:
  - Non-specific
  - Detectable in 1-2 h after AMI & lasts <1 d
  - Found in skeletal & cardiac muscle
- Treatment is similar in STEMI vs. NSTEMI except as mentioned below
  - Antiplatelets: ASA, thienopyridine, GP IIb/IIIa antagonists
  - Clopidrogel – rapid onset of action
  - Anticoagulants: heparin, LMWH
  - BB, ACEi (start in all pts w/ AMI), nitrates, statins, O2

<table>
<thead>
<tr>
<th>Affected Cardiac Wall</th>
<th>EKG Leads</th>
<th>Artery Involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
<td>RCA</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
<td>Circumflex</td>
</tr>
<tr>
<td>Anterior</td>
<td>V2-V4, I, aVL</td>
<td>LCA</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1, V2 (ST depression)</td>
<td>RCA or Circumflex</td>
</tr>
<tr>
<td>Apical</td>
<td>V3-V6</td>
<td>LAD</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, aVL, V4-V6</td>
<td>LAD or Circumflex</td>
</tr>
<tr>
<td>Antero septal</td>
<td>V1-V3</td>
<td>LAD</td>
</tr>
</tbody>
</table>
| Stable angina pectoris | -Related to a fixed stenosis of coronary artery  
-Pain that builds up rapidly in 30 seconds & disappears w/in 5-15 min.  
-Precipitated by activity & relieved by rest  
-Aching or dull midsternal discomfort w/ radiation to the neck, L shoulder, or arm  
-Profound weakness & breathlessness may be an “angina equivalent”  
-Physical exam: may be normal or note S4 (stiff ventricle d/t ischemia) | -Labs: enzymes negative  
-EKG: may show ST depression & T wave changes during episode of pain, otherwise normal or arrhythmia  
-Diagnosis:  
- Positive stress test  
- Exercise test: may not detect low grade stenosis (<50%), positive if >2mm ST depression &/or hypotension; contraindicated in unstable angina, MI, aortic dissection, aortic stenosis, uncontrolled HTN, & ventricular arrhythmias  
- Nuclear stress test  
- Dobutamine or adenosine stress test  
- Stress echo  
- Perfusion scintigraphy testing  
- D/c smoking  
- Control HTN/diabetes  
- Exercise  
- Reduce progression → manage lipids, anti-platelet meds, BB, ACEI  
- Revascularization: PTCA, CABG | 
| Unstable angina pectoris | -Diagnosed clinically:  
- New-onset angina  
- Increasing angina  
- Angina occurring at rest  
-Dyspnea, palpitations, fatigue  
-Pain is retrosternal or epigastric (described as pressure, burning or squeezing)  
-Possible nausea, SOB, diaphoresis  
-Physical exam: normal or S4 may be present  
-EKG: normal or nonspecific changes  
-Cardiac enzymes normal  
-Reduce progression to MI  
-Anti-platelets (ASA), BB, ACEI  
-Revascularization | 
| Prinzmetal angina | -Chest pain occurs w/o usual precipitating factors & is associated w/ ST elevation rather than depression  
-Women <50  
-Occurs in the early morning, awakening pts from sleep & is associated w/ arrhythmias or conduction deficits  
- Coronary arteriography to determine if there are fixed or stenotic lesions  
-Ultrasound – tool of choice  
-CT scan – monitors progression  
-CXR: thoracic aneurysm reveals widened mediastinum & enlarged aortic knob  
-Untreated mortality about 1% per hour  
-Monitor BP, rhythm & urine output  
-Central venous line (pts w/ hypotension or CHF)  
-Reduce systolic BP to 100-120 mmHg  
-BB to reduce contractility | 
| Aortic aneurysm | -Pathologic dilatation of the aorta  
-Abdominal aorta most common site  
-Atherosclerosis is the most common underlying causes  
-Risk factors: smoking, HTN, age, hyperlipidemia  
-Most cases are asx  
-May have hypogastric or low back pain  
-Steady gnawing pain  
-W/ rupture, • BP & • pain  
-PE: pulsatile abdominal mass  
-Ultrasound – tool of choice  
-CT scan – monitors progression  
-CXR: thoracic aneurysm reveals widened mediastinum & enlarged aortic knob  
-Screen every 6 months for aneurysms >4 cm  
-Surgery for >5cm | 
| Aortic dissection | -Tear in intimal layer w/ blood entering the media  
-Etiology:  
- Peak in 6th-7th decades  
- Men > women  
- H/o HTN in 80%  
- Bicuspid aortic valve  
-Severe chest pain 75-90% - sudden onset, severe at inception, ripping/tearing/stabbing pain  
-Migration of the pain 70% of cases  
-Location – anterior pain more common ascending involvement, posterior pain more common descending involvement  
-CXR: widened mediastinum or silhouette (80-90%)  
-Contrast-enhanced CT  
-MRI: current gold-standard  
-TEE/TTE | 
<p>| | |
| | |</p>
<table>
<thead>
<tr>
<th>Arterial embolism/thrombosis</th>
<th>Cause of arterial insufficiency</th>
<th>Severe pain</th>
<th>Echo, arteriogram</th>
<th>Morphine for chest pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Secondary to:</td>
<td>- A-fib/flutter</td>
<td>- Diminished pulses</td>
<td>- Embolectomy</td>
<td>- Surgical consult</td>
</tr>
<tr>
<td>- Mitral stenosis</td>
<td>- Transmural infarct</td>
<td>- PE: 5 Ps</td>
<td>- Complications: limb loss, compartment syndrome (treated w/ fasciotomy)</td>
<td></td>
</tr>
<tr>
<td>- Trauma</td>
<td>- Hypercoagulable state</td>
<td>- Pain, pallor, pulseless, paresthesia, paralysis</td>
<td>- Heparin</td>
<td></td>
</tr>
<tr>
<td>- Post-arterial procedures</td>
<td></td>
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<tr>
<td>Giant cell arteritis</td>
<td>Granulomatous vasculitis of the temporal artery</td>
<td>Headache, jaw claudication, vision loss</td>
<td>Labs: elevated sed. rate (&gt;50 mm/hr.), anemia, leukocytosis</td>
<td>Corticosteroids</td>
</tr>
<tr>
<td>- Etiology unknown</td>
<td>- Typically ages &gt;50 – may coexist w/ polymyalgia rheumatic</td>
<td>- Fever, fatigue, weight loss</td>
<td>- Artery biopsy</td>
<td></td>
</tr>
<tr>
<td>- Physical exam: temporal artery tender, enlarged, &amp; erythema</td>
<td>- Peripheral arterial disease</td>
<td>General: Claudication (cramping or tiredness in calf, thigh, or hip while walking) → pain must resolve w/ rest for dx (pain at rest indicates advanced dz)</td>
<td>- Handheld Doppler</td>
<td>General:</td>
</tr>
<tr>
<td>- Affects 5% of US population &gt;55 yrs. &amp; 20% &gt;75 yrs.</td>
<td>- 80% w/ only claudication remain stable &amp; 5% progress to limb loss</td>
<td>- Diminished femoral pulses</td>
<td>- Ankle/brachial Index: segmental BP at arm, upper thigh, above &amp; below knee &amp; above ankle in supine pos. using Doppler to find systolic press. at each place → in pressure</td>
<td>- Control risk factors (smoking, exercise, foot care, hyperlipidemia &amp; HTN, wt. reduction, diabetic control)</td>
</tr>
<tr>
<td>- 80% w/ only claudication remain stable &amp; 5% progress to limb loss</td>
<td>- High mortality d/t other underlying atherosclerotic dz</td>
<td>- General:</td>
<td>- Medical therapy: antiplatelet &amp; anticoagulants</td>
<td></td>
</tr>
<tr>
<td>- Handheld Doppler</td>
<td>- Ankle/brachial Index: segmental BP at arm, upper thigh, above &amp; below knee &amp; above ankle in supine pos. using Doppler to find systolic press. at each place → in pressure</td>
<td>- General:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Aortoiliac dz:
- Most common in smokers & pts w/ hyperlipidemia
- Femoropopliteal dz:
  - 2/3 of cases of peripheral vascular dz
- Tibioperoneal occlusion:
  - Commonly in diabetics & older pts
- Acute arterial occlusion of a limb:
  - Can be caused by an embolus or thrombosis
  - A. fib is the most common cause of cardiac thrombus formation
  - Other causes: valvular or ischemic heart dz
  - Pt w/ acute primary thrombus has prior sx of claudication

Tissue loss d/t ulceration &/or gangrene or rest pain.
- PE: palpate pulses, auscultate aortic, iliac, femoral & popliteal pulses for bruits; look for discolored skin, wounds or ulcers (if pallor occurs on leg elevation & doesn’t return until leg hanging off table → test is +); atrophic skin (thin); prolonged capillary filling time (>5 seconds w/ compression)
- Aortoiliac dz:
  - Claudication in buttocks or thighs
  - Femoral pulses absent or diminished but pedal pulse may be intact
  - First manifestation of systemic dz
- Femoropopliteal dz:
  - Calf pain on exertion
  - Femoral pulses may be preserved but popliteal & pedal absent/diminished
- Tibioperoneal dz:
  - Skin ulcers & atrophic skin changes common in skin distal to occlusions
- Acute arterial occlusion of a limb:
  - Sudden pain in an extremity
  - Associated w/ neurologic dysfunction (numbness or paralysis)
  - Absent pulses of extremities
  - Five Ps
    - Pain
    - Pallor
    - Pulselessness
    - Parasthesias
    - Paralysis
  - AND
    - Poikilothermia → inability to regulate body temp - affected area is cold
- Clinical findings:
  - “Heart attack” of an extremity
  - Poor/lack of blood flow → Pt. w/ long-standing claudication can have extensive collateral circulation
  - W/o prompt tx, limb proceeds quickly to tissue necrosis & possibly gangrene

Suggests arterial occlusion (index <0.8 moderate dz, index <0.6 multilevel dz, <0.4 severe dz & risk)
- Can’t do this test in pts w/ calcified vessels (diabetics)
- Acute arterial occlusion of a limb:
  - Doppler – little or no flow to distal vessels
  - Labs: systemic acidosis d/t cell death
  - Imaging: abrupt cutoff of contrast w/ occlusion → Imaging should be done ASAP, in the OR if necessary since every moment counts!

Cilostazol: phosphodiesterase inhibitor improves walking distance (many SEs, contraindicated in CHF & long term use could be causative of CV injury)
- DO NOT USE vasodilators, vasoconstrictors, alpha or beta-blockers
- Interventions: percutaneous transluminal angioplasty & stents; surgical therapy (bigger vessels are easier to treat)
- Acute arterial occlusion of a limb:
  - 3 hrs. to re-vascularize - immediate revascularization required
  - Neurologic injury, including loss of light touch, indicates collateral flow is inadequate → need revascularization w/in 3 hrs.
  - Risk of irreversible tissue damage about 100% at 6 hrs.
  - At dx, give unfractionated heparin IV → revascularization is still needed (bypass or take clot out)
  - If intact neuro exam → catheter-directed chemical thrombolysis can be done
  - Surgery under general anesthesia is usually indicated → Local anesthesia for extremely high risk pts
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Phlebitis/thrombophlebitis</strong></td>
<td>Inflammatory thrombosis involving superficial veins of the lower extremities</td>
<td>- Rule out DVT</td>
</tr>
<tr>
<td></td>
<td>- Associated w/ varicose veins, pregnancy, catheter placement</td>
<td>- Based on clinical findings</td>
</tr>
<tr>
<td></td>
<td>- Septic thrombophlebitis seen in IVDA</td>
<td>- Warm, moist compresses</td>
</tr>
<tr>
<td></td>
<td>- Vein is palpable &amp; tender – induration, redness, &amp; tenderness along course of vein</td>
<td>- NSAIDs for pain</td>
</tr>
<tr>
<td></td>
<td>- Cord is palpable</td>
<td>- Low molecular weight heparin</td>
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<tr>
<td></td>
<td>- Septic thrombophlebitis seen in IVDA</td>
<td>- Abx if septic thrombophlebitis</td>
</tr>
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<td>- Abx if septic thrombophlebitis</td>
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<tr>
<td><strong>Varicose veins</strong></td>
<td>Due to incompetence of saphenous vein – from intravascular pressure or defective valves</td>
<td>- Conservative management</td>
</tr>
<tr>
<td></td>
<td>- Primarily in superficial veins of thigh, calf, &amp; ankle</td>
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<td></td>
<td>- Factors: prolonged standing, pregnancy, obesity</td>
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<td></td>
<td>- Asx – may have local aching &amp; fatigue</td>
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<td></td>
<td>- Physical exam: tortuous veins easily compressed</td>
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<tr>
<td><strong>Venous insufficiency</strong></td>
<td>Development of clot in deep veins of the extremities or pelvis</td>
<td>- Based on clinical findings</td>
</tr>
<tr>
<td></td>
<td>- Predisposing factors (Virchow’s triad):</td>
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<tr>
<td></td>
<td>- Venous stasis</td>
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<td></td>
<td>- Vascular damage</td>
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<td></td>
<td>- Hypercoagulability</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Risk factors: pregnancy, smoking, obesity, cancer, BCP, post-op, trauma</td>
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<tr>
<td></td>
<td>- May be normal</td>
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</tr>
<tr>
<td></td>
<td>- Palpable cord, skin discoloration</td>
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<tr>
<td></td>
<td>- Positive Homan’s sign</td>
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<td></td>
<td>- Pain swelling at site &amp; distal to site</td>
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<td>- Physical exam:</td>
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<tr>
<td><strong>Aortic stenosis</strong></td>
<td>Sx d/t LV outflow obstruction leading to LV pressure, hypertrophy &amp; EF</td>
<td>- US &amp; venogram</td>
</tr>
<tr>
<td></td>
<td>- Typically age 60-80</td>
<td>- Positive D-dimer</td>
</tr>
<tr>
<td></td>
<td>- Progression leads to calcification &amp; fibrosis which leads to leaflet stiffness &amp; reduced systolic opening</td>
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<tr>
<td></td>
<td>- Area must be reduced to ¼ its original size before clinically significant obstruction occurs</td>
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<tr>
<td></td>
<td>- Etiologies:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Congenital - bicuspid aortic valve (2% of population)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Non-congenital</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Degenerative calcification (most common acquired valvular stenosis, mostly men (80%)</td>
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</tr>
<tr>
<td></td>
<td>- Angina, syncope &amp; exertional dyspnea</td>
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<tr>
<td></td>
<td>- Physical examination:</td>
<td></td>
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<tr>
<td></td>
<td>- Systolic ejection murmur radiating to the neck</td>
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<tr>
<td></td>
<td>- As severity increases, murmur peaks later in systole &amp; can become softer as CO</td>
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<td></td>
<td>- Diminished carotid upstroke</td>
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<td></td>
<td>- Loss of A2 component of second heart sound</td>
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<tr>
<td></td>
<td>- EKG – LVH</td>
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<tr>
<td></td>
<td>- CXR – dilatation of ascending aorta &amp; pulmonary congestions</td>
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<td></td>
<td>- Echo – thick LV wall &amp; valvular calcifications</td>
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<tr>
<td></td>
<td>- Stress testing is contraindicated in sx pts &amp; should be used w/ caution in asx pts</td>
<td></td>
</tr>
<tr>
<td><strong>Aortic stenosis</strong></td>
<td>Sx d/t LV outflow obstruction leading to LV pressure, hypertrophy &amp; EF</td>
<td>- No standard medical therapy proven to improve outcome</td>
</tr>
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<td>- Typically age 60-80</td>
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<td>Sx d/t LV outflow obstruction leading to LV pressure, hypertrophy &amp; EF</td>
<td>- Aortic valve replacement only effective tx</td>
</tr>
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<td></td>
<td>- Typically age 60-80</td>
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</tr>
</tbody>
</table>
### Aortic Regurgitation

- Due to abnormal leaflets or proximal aortic root
- Results in end-diastolic volume & ventricular dilatation, leading to regurgitation
- **Etiologies:**
  - Acute: infectious endocarditis, dissection, valve rupture
  - Chronic: HTN, idiopathic dilation, bicuspid valve, rheumatic heart dz, Marfan syndrome

  **Acute:**
  - No time to mount adaptive response to larger end diastolic volumes
  - End diastolic pressures cause inadequate contractile response, causing stroke volume

  **Chronic:**
  - LV dilation (produces a larger stroke volume)
    - Increased pulse pressure, causing systolic HTN

- **Physical exam:**
  - Widening pulse pressure
  - **Bounding “water hammer” peripheral pulses**
  - Hyperdynamic apical pulse displaced to the left
  - **Diastolic blowing murmur best heard along LSB**
  - Systolic &/or diastolic thrill or murmur head over the femoral arteries (Duroziez sign)

- **EKG:** LVH
- **CXR:** LVH w/ or without CHF, enlargement of aortic knob
- **Echo:** determines severity of regurgitation

### Mitral Stenosis

- Leads to atrial pressure & atrial enlargement, pulmonary congestion, pulmonary hypertension, & right side heart failure
- Most cases secondary to rheumatic heart disease or rare congenital defect
  - Women > men, ages 40-50

- **Typical for those of left-sided heart failure**
- **Progression of symptoms is often subtle & gradual**
  - As severity increases:
    - Fatigue, dyspnea
    - Hemoptyisis
    - RUQ pain 2° venous congestion
    - Palpitations/A-Fib (>50% of patients w/ severe MS have A-fib)

- **Physical exam:**
  - Opening snap w/ low-pitched diastolic rumble
  - Murmur louder w/ isometric handgrips
  - Loud S1 & loud P2
  - JVD – prominent jugular A wave

- **CXR:** LAH, congestion
- **EKG:** LAH, A-fib
- **Echo:** diagnostic

### Mitral Regurgitation

- Most frequently related to ischemic or degenerative process
- Acute or chronic

- **Acute:**
  - Typically symptomatic
  - Left heart failure

- **EKG:** normal or LVH
- **CXR:** normal or LA enlargement
- **Echo:** diagnostic

- **Diuretics, digoxin, ACEI, salt restriction (if CHF)**
- Surgery – sx pts (more than mild), EF <55%, or end diastolic dimension >55 mm
- Mild dz: no therapy, no restriction in activity
- Moderate dz: no therapy, avoid heavy physical exertion, vasodilator therapy may be helpful
- **HR control “mainstay of therapy”**
  - BB
  - CCBs
  - Digitalis if LV/RV dysfunction
  - Diuretics if pulmonary congestion
  - Warfarin if in A-fib
  - Surgery: closed or open commissurotomy, percutaneous mitral balloon valvotomy

- **ACEI** – reduces afterload
- Surgery is definitive therapy
### Mitral valve prolapse
- Most common form of valvular heart disease
- Primary valve disorder:
  - Idiopathic
  - Redundant mitral apparatus
  - Myxomatous degeneration of the valve
- Secondary:
  - Marfan syndrome
  - Rheumatic heart disease
  - Ischemic heart disease
  - Rupture chordae tendineae

### Tricuspid stenosis
- Female predominance
- Right heart failure after rheumatic heart disease, tricuspid valve repair or replacement, & carcinoid disease are the most common causes
- Frequently accompanied by tricuspid regurgitation
- Right heart failure w/ hepatomegaly, ascites & dependent edema
- Diastolic rumble along the LLB – mimics mitral stenosis & w/ inspiration
- Presystolic liver pulsation may be found
- Elevated JVP w/ prominent a wave
- ECG: right atrial enlargement
- Echo/Doppler is diagnostic; mean valve gradient >5 mmHg indicates severe tricuspid stenosis
- CXR:
  - Marked cardiomegaly w/ normal pulmonary artery
  - Dilated superior vena cava &azygous vein

### Tricuspid regurgitation
- Most common cause is RV dilatation:
  - Pulmonary HTN from COPD
  - Infectious endocarditis
  - Thyrotoxicosis
  - Congenital
- Signs of R-sided heart failure: ascites, edema, RUQ pain
- Physical exam:
  - JVD +
  - Pansystolic murmur at left sternal border

### Causes:
- **Acute**
  - Ruptured chordae tendineae
  - Papillary muscle rupture
  - Endocarditis
  - Trauma
- **Chronic**
  - MVP most common cause
  - Ischemic heart disease
  - Cardiomyopathy
  - RHD
  - Infective endocarditis
  - Myocardial disease or tumors
  - Connective tissue disorders (SLE)

### Congenital defect
- Dyspnea on exertion
- PND
- Pulmonary congestion
- Even severe chronic MR is more often diagnosed by murmur – Holosystolic murmur at apex w/radiation to base or left axilla
- Fatigue & mild DOE the most common presentation
- As disease progresses, more severe DOE, PND, pulmonary edema, or hemoptysis may present

### Physical exam:
- Midsystolic click
- Late systolic murmur
- JVD +
- Pansystolic murmur at left sternal border

### Echo
- Determine LV hypertrophy & left atrial abnormality
- Also determine presence of pulmonary HTN

- Echo
- Not needed

- Diuretics for fluid congestion
- Torsemide may be better than furosemide b/c better absorbed from the gut
- Aldosterone inhibitors (spironolactone) if there is liver engorgement or ascites
- If surgery indicated → tricuspid valve replacement is preferred

- Treat underlying cause
### Pulmonary stenosis
- **Congenital etiology is most common cause – rheumatic cause uncommon**
- Angina, syncope
- Physical exam:
  - Early systolic ejection click followed by systolic ejection murmur w/ radiation to base of the heart
- Echo
  - CXR – enlarged RV & pulmonary artery
- Does not need tx

### Pulmonary regurgitation
- Most cases are d/t pulmonary HTN (high pressure cause)
- **Low pressure causes**: dilated pulmonary annulus, congenitally abnormal bicuspid or dysplastic pulmonary valve, following surgical repair or plaque from carcinoid dz
- Most are asx
- Loud diastolic (Graham Steell) murmur in high-pressure PR → w/ inspiration & w/ Valsalva
- Soft or no murmur in low-pressure PR
- PE: hyperdynamic RV van be palpated (RV lift)
- Early onset:
  - S. aureus, Viridans eus, S. epidermidis, enterococci, fungal (Aspergillus, Candida)
- Prosthetic valve
- Congenital abnormalities
- Most commonly involves aortic & mitral valves
- Acute endocarditis:
  - Most commonly associated w/ staph aureus; rapidly destructive; can be fatal <6 weeks if untreated
- Subacute endocarditis:
  - Most commonly associated w/ viridans streptococci; takes from >6 weeks to 1 year to become fatal
- Microorganisms:
  - Community – S. aureus, Viridans strep, enterococci
  - Nosocomial – S. aureus, S. epidermidis, enterococci, fungal (Aspergillus, Candida)
  - Prosthetic valve – S. epidermidis, S. aureus, enterococci
- Prosthetic valve endocarditis:
  - Early onset:
    - Onset of sx <60 days post-op

### Acute/subacute bacterial endocarditis
- Infection which produces vegetations on the valve leaflet or endocardium
- Predisposing conditions:
  - MVP
  - Degenerative valvular dz
  - IV drug abuse: 30x • risk
  - Prosthetic valve
  - Congenital abnormalities
  - Most commonly involves aortic & mitral valves
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    - Prosthetic valve – S. epidermidis, S. aureus, enterococci
  - Prosthetic valve endocarditis:
    - Early onset:
      - Onset of sx <60 days post-op
- Fever: 80-85%
- Chills, weakness: 45-75%
- Anorexia/weight loss: 25-40%
- Myalgia/arthralgia: 25%
- Angina, syncope
- Early systolic ejection click followed by systolic ejection murmur w/ radiation to base of the heart
- Echo
  - Bacteremia or fungemia
    - Blood cultures positive in +95% of cases
    - Subacute cases: 3 sets of cultures over 3-6 hrs.
    - Acute cases: do not delay tx for 3 hrs.
  - Negative cultures, consider: H. parainfluenza, Candida, Aspergillus, Coxieila, Histoplasma
  - Anemia, leukocytosis, elevated sed rate & CRP
  - Transthoracic echo:
    - Rapid, non-invasive, & specificity for vegetations is 98%
    - Sensitivity is <60%
    - Transeosophageal echo:
      - Procedure of choice w/ high index of clinical suspicion
      - Increases sensitivity for vegetations to 75-95% yet maintains specificity of 85-98%
      - Negative transeosophageal echo has negative predictive value of over 92%
- Based on clinical suspicion, labs, & echo
- Labs:
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- Requires rapid eradication of all microorganisms from the vegetations
- Use bactericidal vs. static agent
- Treat for minimum 4-6 weeks
- Viridans streptococci:
  - PCN G or ampicillin plus gentamicin or
  - Ceftriaxone plus gentamicin or
  - Vancomycin
- Enterococci:
  - Ampicillin or PCN G plus gentamicin
  - Staph aureus:
  - Nafcinill or oxacillin plus gentamicin
- MRSA:
  - Vancomycin
  - Staph epidermidis:
  - Vancomycin plus gentamicin plus rifampin
- SBE prophylaxis:
  - New guidelines in 2007
  - Required for certain high-risk pts & certain invasive procedures
  - Potential AEs of antimicrobial agent
    - Allergic rxn complicated by rapid eradication of all microorganisms from the vegetations
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  - MRSA:
    - Vancomycin
    - Staph epidermidis:
    - Vancomycin plus gentamicin plus rifampin
  - SBE prophylaxis:
- Valve contamination during the procedure or bacteremia post-op
- Staphylococci most common type of organism
- Valve dysfunction/dehiscence: fulminant course
  - Late onset:
    - Onset of sx >60 days post-op
    - Streptococci most common type of organism
    - Course similar to that of NVE

- Staphylococci most common type of organism

- Particularly useful in prosthetic valves & for the evaluation of myocardial invasion

- Late onset:
  - Onset of sx >60 days post-op
  - Streptococci most common type of organism
  - Course similar to that of NVE

- Anaphylactic rxn occurs between 0.004-0.004%
- Death occurs 0.001%, approximately 300/yr.

- High risk:
  - Hx of prosthetic valve
  - Previous endocarditis
  - Unrepaired congenital heart dz
  - Repaired congenital heart defect w/ prosthetic material or device – but only during first 6 months after the procedure
  - Repaired congenital heart defect w/ residual defects
  - Cardiac transplant recipients who develop cardiac valvulopathy

- Invasive procedures:
  - Dental procedures
  - Tonsillectomy & adenoidectomy
  - Surgery involving intestinal or respiratory mucosa
  - Surgery on infected soft tissue
  - Established GU or other intra-abdominal infxn

- Oral – amoxicillin
- NPO – ampicillin or cefazolin or ceftriaxone
- Allergic PCN: oral – cepalexin or clindamycin or azithromycin or clarithromycin
- Allergic PCN: NPO – cefazolin or ceftriaxone or clindamycin

- Acute pericarditis
  - Due to inflammation of the pericardial lining of the heart
  - Etiologies:
    - Idiopathic
    - Viral: coxsackie B virus
    - Malignancy: lung, breast CA & lymphoma are most common
    - Metabolic: uremia
    - Infection: TB, SBE, fungal
    - Drug-induced: procainamide, hydralazine

- Characterized by chest pain (most common), pericardial friction rub & serial electrocardiographic abnormalities
  - Coughing or deep breathing • pain, leaning forward may give relief
  - SOB/DOE
  - Fever
  - Cough, weight loss
  - Physical exam: friction rub d/t inflamed pericardial surfaces rubbing together;

- Labs: CBC, electrolytes, BUN/Cr, possible ANA, RF, PPD r/o TB
- EKG: diffuse ST elevation w/ upright T wave, PR interval depression, low voltage, a.fib/flutter

- NSAIDs & ASA for pain
  - Steroids if no response in 48 hrs.
  - The following are self-limiting w/in 2-6 weeks:
    - Viral pericarditis
    - Idiopathic pericarditis
    - Post MI pericarditis
    - Post-pericardiotomy syndrome
- **Idiopathic inflammatory disease:** SLE, RA
  - Post MI injury (1-3 weeks post)
- pathognomonic for acute pericarditis), relative tachycardia, hypotension

| Cardiac tamponade | - Accumulation of fluid in pericardial sac & inability to fill cardiac chambers in diastole lead to reduction stroke volume & cardiac output
| | - Leads to hypotension
| | - Dyspnea on exertion, orthopnea, hepatic engorgement
| | - Hypotension
| | - JVD (elevation in systemic venous pressure)
| | - Muffled heart sounds
| | - **Pulsus paradoxus:**
| | - Exaggerated response from the normal physiologic drop in BP w/ inspiration
| | - Up to 10 mmHg drop in systolic BP occurs normally w/ inspiration, however >10 mmHg drop is tamponade
| | - Total paradox = complete disappearance of palpable pulse (severe)
| | - Echo:
| | - RA & ventricular collapse during diastole
| | - As little as 15cc of fluid can be detected by 2D echo
| | - Differentiate from other causes: tumor compression or hematoma, RV infarct
| | - Drain the fluid: cath or echo guided
| | - Treat sx as for pericarditis
| | - Pericardiectomy or window placement may be required

| Pericardial effusion | - Prolonged & severe inflammation leading to fluid accumulation around the heart
| | - Small effusions – no sx
| | - Large effusions – signs of tamponade
| | - Physical exam: diminished heart sounds, possible friction rub
| | - CXR
| | - EKG – low voltage QRS
| | - Echo – fluid between pericardium layers
| | - Pericardiocentesis
| | - Maintain BP