**Bug Schema:** Gram + = 2 cocci (streptococcus, staphylococcus) and 4 rods (2 spore formers = bacillus and clostridium) (2 non-spore formers = corynbacteria and listeria).
Gram - = only 1 coccus (diplococci = neiserria gonorrhea, rests = gram – rods or pleomorphic, with the exception being spirochetes like treponema.

<table>
<thead>
<tr>
<th>Presumed Condition</th>
<th>Common Pathogens</th>
<th>Drugs of Choice</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>URIs/ENT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacterial Pharyngitis</td>
<td>Often Group A Strep -H: nasopharynx/skin of humans -Vi: M protein ~ adherence, anti-phagocytic but antigenic, Streptolysin O and S ~ B-hemolysis (also consider viruses in differential ~ EBV, cocksackie, HSV, adenovirus)</td>
<td>1. Penicillin V x 10 days or benzanthine penicillin G (IM injection) 2. If allergic to Penicillin = clindamycin or macrolide</td>
<td>-Normals -Normals and ~ sporadic and ~ huddling (resp. secretions) -toxic + sore, swollen throat Complications: -Suppurative = scarlet fever -Non-suppurative = RF ~ post-pharyngitis, GN</td>
</tr>
<tr>
<td>Common Cold</td>
<td>1. Rhinovirus -Acid-labile ~ no GI and prefers cold temp ~ nasal mucosa -ICAM → resp mucosa – multiply → cytokine release ~ host symptoms 2. Coronaviruses (SARS) 3. Adenoviruses 4. Influenza 5. Parainfluenza</td>
<td>Deongestant (e.g. Sudafed) +/- acetaminophen</td>
<td>-short incubation time and low infective dose -CONGESTION is a huge clue re: differential (teary eyes, congested nose)</td>
</tr>
<tr>
<td>Influenza</td>
<td>Influenza A, B, and C -ssRNA virus, unique b/c it is the only virus besides retroviruses that replicate in the nucleus -vi: Hemagluttinin (HA) glycoprotein—binds to RBS and cells of the upper resp.</td>
<td>Vaccine: contraindicated in egg allergies Amanditidne and Rimantidine: prevent viral uncoating of influenza A (M2 channel)</td>
<td>Flu = fever, runny nose, cough, myalgias, arthralgias Complications = secondary bacterial pneumonias in elderly -Reye’s syndrome in children who use</td>
</tr>
</tbody>
</table>
tract. HA then cleaved by host cell proteases which allows HA to activate fusion. Viral RNA then dumped into cells
-Neuraminidase glycoprotein: breaks down neuramic acid, an important component of mucin (important in virion release)
-M2 ion channel allows for acidification of endosome so that the entering virus can be uncoated (drug target)

blockers)
Zanamivir (inhaled) and oseltamivir (oral) are neuraminidase inhibitors that can shorten the course of influenza A and B.

aspirin, can get liver and brain disease
-increased mortality in elderly and those with underlying pulmonary and cardiac disease
-antigenic drift ~ minor mutations change antigenicity of HA and NA = epidemics of common flu
-antigenic shift = only with influenza A = reassortment = major changes in HA and/or NA = devastating pandemics

| Otitis Media                      | 1. Strep Pneumo  
- alpha hemolytic  
- capsule  
- pneumolysin  
- multiple serotypes  
2. Moraxella Cattarhalis  
3. H. Flu  
- obligate human dweller  
- capsule (6 types, B = more virulent)  
- attachment pilli  
- IgA1 protease | Decongestant (e.g. Sudafed) ~ underlying cause +/- amoxacillin | -Normal respiratory flora → middle ear  
- 6 mo. – 2 yo, adults ~ structural abnormality +/- IC  
- patho often ~ nasal congestion ~ virus, compress aud. tube, fluid stasis and bug growth  
- red ear drum, bulging, immovable to typanometer  
- complications = hearing loss, meningitis

| Sinusitis                      | 1. Strep Pneumo  
- alpha hemolytic  
- capsule | Decongestant (e.g. Sudafed) ~ underlying cause +/- amoxacillin | - clues = headaches, tooth pain. Also ~ congestion =
| **Epiglottitis** | 1. Strep Pneumo  
- alpha hemolytic  
- capsule  
- pneumolysin  
- multiple serotypes  
2. Group A strep  
- H: nasopharynx/skin of humans  
- Vi: M protein ~ adherence, anti-phagocytic but antigenic, Streptolysin O and S ~ B-hemolysis  
3. H. Flu (less now ~ vaccination)  
- obligate human dweller  
- capsule (6 types, B = more virulent)  
- attachment pilli  
- IgA1 protease | 2nd or 3rd generation cephalosporin is preferred (e.g. cefuroxime or ceftriaxone) | - Pharyngitis \(\rightarrow\) epiglottis, can block airway  
- drooling, cyanosis, inspiratory stridor, etc.  
- clinical \(\sim\) x-ray +/- bacteremia +/- intubate |
| **Diptheria** | Corynbacterium diptheriae  
- transmitted from respiratory droplets from carrier  
- vi: forms pseudo membrane in pharynx; base for toxin secretion, toxins= classic AB, B ~ binding for entry into cardiac and neural tissue,  | 1. Anti-toxin  
2. Penicillin or macrolides  
3. surgery to clear airways  
DPT vaccine | Rare, signs are generally acute \(\sim\) phaygneal growth of organism \(\rightarrow\) sore throat, breathing difficulties. Toxin causes heart and nerve issues, with nerve issues being weeks |
A~ blocks protein synthesis by inactivating EF2 (human antibiotic)
(note: other corynebacteria = normal flora of skin and pharynx associated with foreign bodies and intravenous catheters)
after pharyngeal symptoms. Complication=aspiration of membrane.

<table>
<thead>
<tr>
<th>Croup/Laryngitis</th>
<th>Parainfluenza</th>
<th>Supportive (steam (shower may help)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Glycoproteins with combined HA and NA activity</td>
<td>raspy cough ~ subglottal swelling</td>
</tr>
<tr>
<td></td>
<td>- F-protein (Fusion protein)</td>
<td>-in adults → whisper voice ~ involvement of true cords</td>
</tr>
<tr>
<td></td>
<td>→ multinucleated giant cells</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mucormycosis</th>
<th>Rhizopus, Rhizomucor, mucor, saprophytic molds</th>
<th>Amb and surgery, disease rapidly fatal</th>
</tr>
</thead>
</table>

Opportunistic, associated with Ic patients
1. rhinocerebral: **associated with diabetes**, starts on nasal mucosa and invades sinuses and orbit
   2. pulmonary mucormycosis

<table>
<thead>
<tr>
<th>Community Acquired Bacterial Pneumonia: Typical</th>
<th>1. Strep pneumo -alpha hemolytic -capsule -pneumolysin -multiple serotypes -gram positive diplococci</th>
<th>Ceftriaxone + Macrolide (azithromycin) +/- Vanco (high penicillin resistance of strep pneumo)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2. Group A strep -H: nasopharynx/skin of humans -Vi: M protein ~ adherence,</td>
<td>Vaccine for older adults and children</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pneumococcal pneumonia: -hosts are “normal”- children and elderly ~ decreased Ab, low systemic immunity (aggamglobulinemia, MM, spleenectomy) and local immunity (COPD, mucociliary</td>
</tr>
<tr>
<td>Community Acquired Bacterial Pneumonia: Atypical (&quot;Walking&quot;)</td>
<td></td>
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<td>---</td>
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<td></td>
</tr>
<tr>
<td>1. Mycoplasma</td>
<td>Macrolide (Azithromycin preferred) or Quinolones (intracellular parasites and no cell wall in the differential prevent pencillin and</td>
<td></td>
</tr>
<tr>
<td>-<strong>No cell wall</strong>, smallest bacteria capable of growth and repro outside a living cell</td>
<td>unlike typicals:</td>
<td></td>
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<tr>
<td>-Requires <strong>Cholesterol</strong> for membrane function</td>
<td>-well-tolerated in healthy hosts, worse in IC with decreased CMI and local response</td>
<td></td>
</tr>
<tr>
<td>-vi: Protein P1 adheres to</td>
<td>(think of the smoking, drinking Legionnaires)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-much more benign</td>
<td></td>
</tr>
<tr>
<td>2. Pneumococcus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-virulent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-vi: Protein A (binds IgG preventing opsonization), coagulase =allows fibrin formation around organism, cell killers (e.g. homolysin, leukocidin) and tissue destroyers (hyaluronidase, staphylokinase), <strong>penicillinase</strong></td>
<td></td>
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<tr>
<td>-assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1</td>
<td></td>
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<tr>
<td>-beat hemolytic grapes</td>
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<tr>
<td>4. H-flu</td>
<td></td>
<td></td>
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<tr>
<td>-obligate human dweller</td>
<td></td>
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<tr>
<td>-capsule (6 types, B = more virulent)</td>
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<td></td>
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<tr>
<td>-attachment pilli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-IgA1 protease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Aspirated Mouth Flora</td>
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<td></td>
</tr>
<tr>
<td>(~dental procedures, bad teeth=mixture of bugs above)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>damage ~ cigs, alcohol, prior influenza, CHF ~ edema</td>
<td></td>
<td></td>
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<tr>
<td>-synergy with influenza</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-thick green/yellow/blood tinged sputum, plueritic chest pain, crackles, split, rub, consolidation ~ egophony + pectoriloquoy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-leucocytosis with left shift</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-complications= local (empyema, pericarditis) and distant (sepsis)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Pertussis (whooping cough) | Bordatella Pertusis  
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td></td>
<td>human resevoir, highly contagious via resp. secretions</td>
</tr>
</tbody>
</table>

2. Legionella  
- ubiquitous in natural water env (air conditioning, cooling towers), not normal flora  
- ~ CMI not Abs like pneumococcus  
- facultative intracellular parasite  
- Vi: pili and flagella  
- attachment and invasion  
- a type IV secretion system prevents phagosome function  
- SOD and catalase-peroxidase protects vs. oxidative burst  
- protein toxins (e.g. phospholipases, RNAase) and cytotoxin

3. Chlamydia Psitacci  
- birds & poulty (pet store owner, bird feces dry out, fecal particles inhaled and infect lungs  
- EB/RB lifestyle  
- Vi: resistant to lysozyme (cw lacks muramic acid), prevents phagolysosome function

4. Chlamydia Pneumoniae (TWAR)  

<table>
<thead>
<tr>
<th>Cephalosporin from being good choice</th>
<th>Macrolides to prevent spread and avoid cops of secondary pneumonia, hypoxemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>presentation (walking) with a non-productive cough, normal WBC and differential, diffuse rather than lobar patches.</td>
<td>sporadic in kids, adults ~ HC workers with 7-10 day incubation</td>
</tr>
<tr>
<td>vi: capsule, filamentous hemagglutinin (FHA) = pilin rod that extends from surface of pertussis that allows it to bind ciliated epi. Cells of bronchi</td>
<td>Pertussis: G protein act $\rightarrow$ increased Camp $\rightarrow$ increased immune response, traps cells by releasing extracytoplasmic AC that weakens neurotrophils, monocytes, lymphocytes, also has tracheal cytotoxin, kills ciliated epis</td>
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</tr>
<tr>
<td>-manifestations $\sim$ age, infants = apnea, kids = whoop, adults = URI $\rightarrow$ LRI, extended cough with no whoop</td>
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</tr>
<tr>
<td>-Catarrhal phase (mild looking URI with dry cough for 1-2 weeks) $\rightarrow$ paroxysmal phase (cough/whoop for 2-6 wks)</td>
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</tr>
<tr>
<td>-kids may have conjunctival bleeds</td>
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</tr>
<tr>
<td>-mostly clinical dx, clue = leukocytosis with lympho predom.</td>
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</table>

**Respiratory Syncytial Virus (RSV)**

<table>
<thead>
<tr>
<th>RSV</th>
<th>RSV</th>
<th>Mostly supportive (fluids + oxygen)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-vi: F-protein (like all paramyxoviruses, parainfluenza, mumps, measles) $\rightarrow$ syncytial cells. No HA or NA glycoproteins</td>
<td>-vi: F-protein (like all paramyxoviruses, parainfluenza, mumps, measles) $\rightarrow$ syncytial cells. No HA or NA glycoproteins</td>
<td>-vi: F-protein (like all paramyxoviruses, parainfluenza, mumps, measles) $\rightarrow$ syncytial cells. No HA or NA glycoproteins</td>
</tr>
<tr>
<td>Most commonly known cause of pneumonia in infants less than 6 months of age, strong comorbidity with acute otitis media.</td>
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</tr>
<tr>
<td>$\sim$ bronchiolitis = necrosis of lower epi cells $\rightarrow$ inflam and cell debris in lower airways (contrast with bronchospasm) $\rightarrow$ rales, wheezes, hypoxemia, infiltrate, lung hyperexpansion</td>
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</tr>
<tr>
<td>-we all get RSV, protection is incomplete $\sim$ shitty antibodies</td>
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</tr>
<tr>
<td>-strong seasonality</td>
<td>-strong seasonality</td>
<td>-strong seasonality</td>
</tr>
</tbody>
</table>

- Monoclonal antibody vs. F protein (palivizumab) possible but controversial $\sim$ price and efficacy
| Tuberculosis (and atypicals) | Mycobacterium TB - found in humans/primates, spread by air-borne droplets - 40% of total cell dry weight is lipid comprised of mycolic acids - red snappers ~ stain acid-fast (w/ nocardia) - have a very slow growth rate - Facultative intracellular growth (can survive and multiply in macrophages) but note that it’s pretty plain in that it is nonmotile, no capsule, no attachment pili. The biggest virulence factor = mycolic acid coat that allows it to survive by preventing the acidification of the phagolysosome (exact mechanism unknown) note: atypical mycobacteria e.g. MAC are environmental (not human to human) and generally produce skin lesions in healthy people can produce pulmonary infection and cervical lymphadenitis (rare) or MAC in AIDS → disseminated infection with fever, wt. loss, hepatitis, | Always treat active disease with multiple drugs to prevent resistance and promote synergy “4 for 2 and 2 for four”. I saw a red pirate burning the liver 1. INH 2. Rifampin 3. Pyrazinamide 4. Ethambutol if going well, only INH, Rifampin INH alone is only given for asymptomatic recent - → + PPD | M. Tb is a true pathogen, can infect anyone but disease tends to show up in young and old (waning immunity) and those with decreased CMI (HIV, transplant patients, corticosteroids, anti-TNF drugs), increased risk ~ crowding 1. **Primary infection:** inhalation → alveoli → ingested by alveolar macrophages (and PMNS) → local spread to lymph nodes (e.g. to hilar nodes) → hematogenous spread that’s silent. Weeks later, some of the macrophages are finally able to ingest the Tb, which they present to Cd4+ Th at lymph nodes. Th activated, head out the look for areas of bug dominance, releases local IFN to activate the macrophages. Macrophages activated and release TNF-a to recruit more |
bone marrow suppression, and chronic watery diarrhea.

macrophages, fibroblasts, T-cells to form granuloma (let’s control this thing by eating and walling). 90% of the time we are good at this= no disease., sometimes = vague viral like illness or rarely very ill, hematogenous can seed =GU, meningeal, bone and joints (also during reactivation)

**Reactivation infection:**
Viable bacilli in granuloma, held at bay by the watchmen, circulating Cd4 cells, bust out (associated with cd4 decline ~ HIV, elderly, other CMI compromisd. Reactivation most common in well-aerated upper lobes. Associated with bad pneumonia + cachexia, seeding? Dissemination can be military (shot gun blast)

PPD test given, ait 48-73 hrs for delayed hypersens. Rxn if
<table>
<thead>
<tr>
<th>Endemic Mycoses (coccidioides, histoplasma, blastomyces)</th>
<th>All of the endemic mycoses have the same pathological pattern: they are true pathogens that are transmitted via the respiratory route. They are almost always symptomatic, but can cause a pneumonia or disseminate to other sites. 1. Coccidioidomycosis - Found in the SW United States, dissemination is primary to skin and bone (SB) but also to meninges 2. Histoplasma - Found in the Mississippi Valley and also present in bird and bat droppings. (blast hits miss valley) Dissemination can occur to almost any organ but especially spleen and liver, a lot like TB in terms of CXR and can even survive inside macrophages.</th>
<th>Mild to moderate disease: Intraconazole Severe diseases = amphotericin B (AMB) Treat for months to years</th>
<th>With dissemination we most commonly think of IC, e.g. AIDS patients (coccidiodes disseminated common in AIDS patients especially from SW U.S.)</th>
</tr>
</thead>
</table>
3. Blastomyces
- found in mississippi valley, soil or rotting wood
- the blast you get and the blast you want, b/c when you get it it is most often dissem = weight loss, night sweats, lung involvement, and skin ulcers

| Aspergillosis | Caused by Aspergillus fumigatus and/or Aspergillus flavus mold
- found all over in environment and on us
- inhaled | If allergic bronchopulmonary aspergillosis, corticosteroids
- Aspergilloma: surgical removal
- Invasive aspergillosis: incredibly high mortality, treat with voriconazole, amph B, possible capsofungin | Opportunistic infection generally effects IC host.
1. allergic bronchopulmonary aspergillosis (Ige mediated): asthma like rxn with shortness of breath and high fever
2. aspergilloma: fungus ball + hemoptysis
3. Invasive aspergillosis: necrotizing pneumonia. May disseminate to other organs in IC hosts |

| PCP | Pneumocystis Jiroveci, technically a fungus - acquired via resp. route, may ben acquired early in life and remain latent until IC?
- flying saucer appearing fungus, previously | TMP/SMX for tx and prophylaxis, good b/c also helps to protect against toxo and to some extent strep pneumo | - most common opportunistic infection in AIDS with cd4 < 200
- cause interstitial pneumonia with fever and a dry non-productive cough. Kind of looks like walking |
<table>
<thead>
<tr>
<th>Protozoan</th>
<th>Protozoan Disease</th>
<th>Antimicrobial Therapy</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Q fever</td>
<td>Coxiella Burnetti - found in cattle, sheep, and goats, member of Rickettsia but <strong>weird</strong> b/c no arthropod transmission (aq. Via direct airborne transmission of endospore from cow hide or dried placenta or consumption of endospore-contaminated unpasteurized milk.</td>
<td>1. Doxy 2.Erythromycin</td>
<td>Fever, headache, + viral-like pneumonia (mild). <strong>No rash!</strong> Only rickettsial disease – skin rash. Complications = hepatitis and endocarditis</td>
</tr>
</tbody>
</table>

**Plague**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Pathogen Characteristics</th>
<th>Antimicrobial Therapy</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
</table>
| Yesinia Pestis - reservoir = wild rodents, rats, squirrels and prarie dogs in SW U.S., transmission = flea bite, contact with infected animal tissues, inhaled aerosolized orgs (human → human transmission in epidemics. | 1. streptomycin or gentamicin 2. doxy | Pneumonic plague: During epidemics pneumonia occurs as bacteria are spread person to person by aerosols  
Bubonic plague: Regional lymph nodes, usually in groin, swell, and become red hot and tender = bubo, + fever + conjunctivitis  
Sepsis: bacteria survive in macrophages and spread to blood and organs |

**Skin and Soft Tissue Infections**

- mostly caused by strep and staph (commonly found in throat and skin) as well as other environmental bacteria. The hosts can be normal but often have underlying diseases (DM, neuropathy, IVDU, burns, trauma, lymphedema, surgery.  
-these condition predispose for increased bacterial colonization ~ entry (via breaking of the skin, medical devices, and attractive culture (e.g. sugar, fluid buildup, ~edema, maceration, with low removal ~ low blood supply).  
**in general if it spreads lean more toward strep, if it’s localized, lean more toward staph**
| Impetigo | 1. Group A Strep  
-H: nasopharynx/skin of humans  
-Vi: M protein ~ adherence, anti-phagocytic but antigenic, Streptolysin O and S ~ B-hemolysis  
-chains  
2. Staph Aureus  
-**coagulase** +  
-Vi: Protein A (binds IgG preventing opsoniazation), coagulase =allows fibrin formation around organism, cell killers (e.g. homolysin, leukocyidin) and tissue destroyers (hyaluronidase, staphylokinase), **penicillinase**  
-assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1 ~ beat hemolytic grapes | Penicillin or 1st generation cephalosporin to cover staph (keflex), oxacillin/dicloxacilin | -superficial infection of the skin leaving vesicular/crusted lesions.  
-the child wants to get back out and play with their friends  
-complications= GN post-group A strep skin infection |
| --- | --- | --- | --- |
| Cellulitis | -often combined infection  
1. Group A Strep  
-H: nasopharynx/skin of humans  
-Vi: M protein ~ adherence, anti-phagocytic but antigenic, Streptolysin O and S ~ B-hemolysis  
-chains  
2. Group B strep (agalactiae)  
3. Staph Aureus  
-**coagulase** + | Oxacillin (IV covers both strep and staph) | -spreading infection of the dermis, deeper and more serious  
-looks inflamed, systemic effects ~ bacteremia |
<table>
<thead>
<tr>
<th></th>
<th>Staph Aureus</th>
<th>For boils/faruncles, no abx just drain</th>
<th>For carbuncles drain + IV abx vs. staph (keflex, first generation cephalosporin?)</th>
<th>-mini-abscess in dermis and subQ tissue -boils/faruncles are localized collections of pus, carbuncles are more complex compartments of pus (combined boils) +/- systemic effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boil (faruncles), carbuncles</td>
<td>coagulase +</td>
<td>-Vi: Protein A (binds IgG preventing opsoniazation), coagulase = allows fibrin formation around organism, cell killers (e.g. homolysin, leukocidin) and tissue destroyers (hyaluronidase, staphylokinase), <strong>penicillinase</strong> -assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1 -beat hemolytic grapes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abscess</td>
<td>coagulase +</td>
<td>-Vi: Protein A (binds IgG preventing opsoniazation), coagulase = allows fibrin formation around organism, cell killers (e.g. homolysin, leukocidin) and tissue destroyers (hyaluronidase, staphylokinase), <strong>penicillinase</strong> -assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1 -beat hemolytic grapes</td>
<td>Come back</td>
<td>-walled off collection of pus bigger than a boil -systemic symptoms likely</td>
</tr>
<tr>
<td>Pathology</td>
<td>Description</td>
<td>Treatment</td>
<td>Notes</td>
<td></td>
</tr>
<tr>
<td>-----------</td>
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<tr>
<td>Necrotizing Fasciitis</td>
<td>Can be a variety of organisms but often - often combined infection 1. Group A Strep - H: nasopharynx/skin of humans - Vi: M protein ~ adherence, anti-phagocytic but antigenic, Streptolysin O and S ~ B-hemolysis - chains 2. Group B strep (agalactiae) 3. Clostridium perfringens (anaerobics) - anaerobic, non-motile organism - Vi: alpha toxin (lecithinase) and other tissue destructive enzymes → gas gangrene + others</td>
<td>Surgery, broad spectrum IV antibiotics (Imepenem, Penicillin + AG + clindamycin (+, -, toxin))</td>
<td>- spreading infection of fascial tissue - infection moves rapidly with little immune response, very serious can → amputation (flesh-eating bacteria)</td>
<td></td>
</tr>
<tr>
<td>Myonecrosis</td>
<td>Can be a variety of organisms 1. Group A Strep - H: nasopharynx/skin of humans</td>
<td>Surgery, broad spectrum IV antibiotics (Imepenem, Penicillin + AG + clindamycin (+, -, toxin))</td>
<td>- deepest of infections → muscle death (think of farm accident)</td>
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<tr>
<td>Toxin shock syndrome</td>
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<tr>
<td>1. Group A Strep</td>
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<td>-H: nasopharynx/skin of humans</td>
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<tr>
<td>-Vi: M protein ~ adherence, anti-phagocytic but antigenic, Streptolysin O and S ~ B-hemolysis -chains</td>
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<td>2. Staph Aureus</td>
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<tr>
<td>-<strong>coagulase</strong> +</td>
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<tr>
<td>-Vi: Protein A (binds IgG preventing opsoniazation), coagulase =allows fibrin formation around organism, cell killers (e.g. homolysin, leukocyidin) and tissue destroyers (hyaluronidase, staphylokinase), <strong>penicillinase</strong></td>
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<tr>
<td>-assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1 -beat hemolytic grapes</td>
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<td>Maintain with fluids and remove focal point (e.g. tampons, wound complications)</td>
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<tr>
<td>-fever, rash, desquamation, diarrhea, hypotension</td>
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</tbody>
</table>
| Scladed skin syndrome | Staph Aureus  
*coagulase +*  
*Vi: Protein A (binds IgG preventing opsoniazation), coagulase = allows fibrin formation around organism, cell killers (e.g. homolysin, leukocyidin) and tissue destroyers (hyaluronidase, staphylokinase), penicillinase*  
-assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1  
-beat hemolytic grapes | Supportive | Peeling skin in infants  
Toxin acts on desmosomes |
|----------------------|-------------------------------------------------|-----------------|----------------------|
| Osteomyelitis         | 90% of time, Staph Aureus  
*coagulase +*  
*Vi: Protein A (binds IgG preventing opsoniazation), coagulase = allows fibrin formation around organism, cell killers (e.g. homolysin, leukocyidin) and tissue destroyers (hyaluronidase, staphylokinase), penicillinase*  
-assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1  
-beat hemolytic grapes | Tx needs to be cidal ~ decreased WBC efficacy in bone, = anti-staph IV drugs like nafcillin (met a nasty ox) | -can be found in healthy kids and adults but often some underlying disease (e.g. peripheral diabetic neuropathy ~ contiguous spread, or sickle-cell ~ microinfarcts = fluid buildup and no drainage = culture medium.  
-ors enter bone either ~ trauma, hematogenous (kids ~ metaphysis of long bones, adults ~ from vert. disk space. Local invasion eg. ~ chronic
ulcers  
- growth decreases pH 
which decreases PMN potencv 
- pain +/ visual signs of inflam, ESR

<table>
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<tr>
<th>Septic Arthritis</th>
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</table>
| 1. 50-60% = Staph aureus  
  - coagulase +  
  - Vi: Protein A (binds IgG  
    preventing opsoniaiztion),  
  coagulase = allows fibrin 
  formation around organism,  
  cell killers (e.g. homolysin, 
  leuokcyidin) and tissue  
  destroyers (hyaluronidase, 
  staphylokinase),  
  **penicillinase**  
  - assualt weaponry ~ toxins: 
    exfoliatin ~ scalded skin 
    syndrome, enterotoxin ~ 
    food poisoning, TSST-1  
  - beat hemolytic grapes  
  2. Neisseria gonorrhoea (in 
  sexually active people)  
  - human reservoir (no 
    immunity to repeated 
    infections) ferments only 
    glucose  
  - vi: pili ~ adherence,  
    antigenic variation, anti- 
    phagocytic (~ binds tightly 
    to host cell)  
  - IgA1 protease  
  - Outer membrane proteins  
    including porins and opa (~ 
    opacity)  
  - unique proteins that can  |
| Tx needs to be cidal  
  Ceftriaxone for 
  gonorrhea (+)  
  Nafcillin  
  Drain fluid, wash out  |
| - found in healthy kids 
  and adults but also in 
  patients with underlying "joint 
  риск" — IVDU 
  (injections near joints) 
  chronic joint diseases 
  (RA, gout).  
  - entrance into joint is 
    most likely 
    hematogenious but can 
    be inoculated, 
    contiguous (younger), 
    or traumatic 
  \( \rightarrow \) growth of bugs and 
  destruction of cartilage 
  - pain and inflam (only 
    pain in hip)  
  - **gonorrhea = most 
    common cause in 
    younger, sexually 
    active**  
  - in case of prosthetic 
    joints, or chronic 
    osteomyelitis, 
    suspect coag – staph 
    (e.g. S. Epidermidis) 
    and need to remove 
    prosthesis |
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<tbody>
<tr>
<td>Measles (10 days)</td>
<td>Rubeola -ssRNA, has F protein for fusion → multinucleated giant cells (paramyxovirus with parainfluenza, RSV, mumps) -along with chicken pox, probably the most contagious disease</td>
<td>MMR vaccine 1. Prodrome of high fever, hacking cough, conjunctivitis 2. Koplik’s spots: small red based blue-white centered lesions in the mouth 3. Rash: from head, then to neck and torso, then to feet. As rash spreads it coalesces Complications: pneumonia, eye damage, myocardiitis, encephalitis. Subacute sclerosing panencephalitis = slow form of encephalitis that occurs many years after measles infection ~ mutated virus</td>
</tr>
<tr>
<td>Mumps</td>
<td>Mumps virus,</td>
<td>MMR vaccine 1. parotid gland</td>
</tr>
</tbody>
</table>
| Rubella | Aka german measles of 3 day measles | Very concerned about congenital transmission (TORCHES) with inverse relationship of likelihood of transmission and severity of outcomes as pregnancy continues  
MMR (given post-natally in non-immune mothers, to protect against next time) |  
• Many asymptomatic  
• Low grade fever  
• Malaise  
• Mild pharyngitis  
• Rash lasts for three days (hence, “3-day measles”)  
• Maculopapular  
• Blotchy  
• Not as bright  
• Textured  
• Raised  
• Lymphadenopathy (e.g. lumps in back of head)  
• Arthritis (particularly in adults) |
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<tbody>
<tr>
<td>Fifth disease</td>
<td>Parvo B19</td>
<td>No tx just try to avoid exposure, IVIG for ab deficient, also RBC transfusion for anemia</td>
<td></td>
</tr>
</tbody>
</table>
Respiratory tract infection that infects young RBS  
• Symptoms  
  • Usually none  
  • Mild malaise  
  • Mild fever  
• Signs  
  • Rash  
  • Fine, lacy, over |
| Leprosy (Hansen’s disease) | M. Leprae -like TB has facultative intracellular growth | 1. rifampin  
2. Dapsone | Disease ranges from tuberculoid form (granulomas destroy nerves and cause local raised ulcers) to lepromatous form (associated with almost no host response where macrophages are stuffed with bacilli, perfuse |
|---------------------------|--------------------------------------------------------|-----------------------------------------------|

- Arthritis
Can → chronic hemolytic anemia,
In patients with hemolytic anemia, parvovirus B19 supresses bone marrow erythropoietic activity, leading to transient aplastic crisis.

- Not very raised (just macule, not papule)
- Red cheek ("slappe d cheek" appearance)
| **Candidiasis** | **Candida albicans**  
- Normal inhabitant (yeast)  
  of skin, mouth and GI tract,  
  **not found in blood** | **Tx depends on site of infection:**  
**Oral thrush:**  
1. oral fluconazole  
2. nystatin swish and spit  
3. clotrimazole candies  
**Cutaneous infection:**  
1. Topical imidazole or oral fluconazole  
**Esophageal (most common in HIV)**  
1. Fluconazole  
2. capsofungin  
**Systemic:**  
1. Intravenous AMB  
2. Fluconazole  
3. Capsofungin | **Can effect normal and IC hosts with diff disease manifestations.**  
**In normal host:**  
1. oral thrush  
2. vulvovaginal candidiasis (yeast infection)  
3. cutaneous (rash in skin folds of obese, diaper rash, under breasts)  
**In immunocompromised, all the above +:**  
1. esophagitis (retrosternal chest pain, + fever + dysphagia)  
2. Disseminated (generally very sick hospitalized patients  \(\rightarrow\) multi-organ failure)  
3. Chronic mucocutaneous candidiasis |

<p>| <strong>Superficial fungal infection</strong> | <strong>Malassezia furfur, looks like</strong> | <strong>Dandruff shampoo like</strong> | <strong>Pityriasis versicolor:</strong> |</p>
<table>
<thead>
<tr>
<th><strong>Cutaneous mycoses</strong></th>
<th><strong>Subcutaenous mycoses</strong></th>
<th><strong>Hypo or hyperpigmented patches on the skin, surrounding skin darkens with sunlight while the patches remain white</strong></th>
</tr>
</thead>
</table>
| Microsporum species, trichophyton species (hair), epidermophyton (skin) | Sporotrich schenckii -found on rose thorns | Seborrheic dermatitis  
Itchy, flaky, erythematous patches along the eyebrow line  
Tinea nigra (exophiala weneckii ~ brown/black patches on soles of hands or feet) |
| Local creams and lotions work for most of these (e.g. topical imidazole) but need oral (systemic) tx for onychomycosis b/c at nail bed = oral grisofulvin or oral terbinafine | 1. Itraconazole  
2. Fluconazole  
3. KOH (pot flowers = potassium iodide) | Dermatophytosis:  
Tinea corporis = ringworm  
Tinea cruris = jock itch  
Tinea pedia = athlete's foot  
Tinea capitis = scalp  
tinea unguium = onychomycosis = nail |
| **Dermatophytosis:** | 1. subQ nodule gradually appears at site of thorn prick  
2. nodule becomes necrotic and ulcerates  
3. ulcer heals but new nodules pop up | Sprotrichosis:  
1. **Sp**rotrichosis  
2. nodule becomes necrotic and ulcerates  
3. ulcer heals but new nodules pop up |
<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Key Points</th>
</tr>
</thead>
</table>
| Rocky Mountain Spotted Fever | Rickettsia rickettsii (reservoir: dogs, rabbits, and wild rodents; vector: tick) | 1. Doxy  
2. Chloramphenicol  
3. fever  
4. conjunctival infection (redness)  
5. severe headache  
6. rash on wrists, ankles, soles & palms initially, becomes more generalized later (Rickettsia has a tropism for endothelial cells which explains the eye and skin findings) |
| Ehrlichiae | Ehrlichia chaffeensis (HME) (I’m a man, I get the real deal) and *Anaplasma phagocytophilum* (HGA) (reservoir: dogs; transmission: ticks) | 1. Doxy  
Similar to RMSF, only that the accompanying rash is less common |
| Bartonellosis | Bartonella Henselae | Unique in that 1st choice = azithromycin followed by doxy  
Cat-scratch diseases = cat bite or scratch → enlargement of regional lymph node or nodes + low-grade fever and malaise. Usually self-limiting w/in a few months. May cause bacteremia and endocarditis |
In AIDS patients → bacillary angiomatosis = proliferation of small blood vessels in skin and organs of AIDS patients

<table>
<thead>
<tr>
<th>Condition</th>
<th>Organism/Description</th>
<th>Early Treatments</th>
<th>Late Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lyme Disease</td>
<td>Borrelia Burgdorferi&lt;br&gt;-spirochete, large&lt;br&gt;-reservoir = white-footed mouse, white-tailed deer&lt;br&gt;-transmission: vector = nymph stage of small Ixodes tick&lt;br&gt;-found in NE, MW, and Pacific West</td>
<td>1. Doxycycline&lt;br&gt;2. Amoxicillin&lt;br&gt;3. Ceftriaxone for neurologic disease (~ CSF pen)&lt;br&gt;-prevent with DEET, tick checks</td>
<td>1. Early Localized Stage&lt;br&gt;Erythema chronicum migrans (bullseye) ECM&lt;br&gt;2. Early disseminated stage (stage 2)&lt;br&gt;-multiple smaller ECM&lt;br&gt;-neurologic—aseptic meningitis, CN palsies (Bell’s), peripheral neuropathy&lt;br&gt;-cardiac: transient heart block, myocarditis&lt;br&gt;-brief attacks of arthritis of large joints&lt;br&gt;3. Late Stage (stage 3)&lt;br&gt;-Chronic arthritis&lt;br&gt;-encephalopathy</td>
</tr>
</tbody>
</table>
| Relapsing fever            | Borrelia Recurrentis<br>-only Borrelia species transmitted by louse, often in developing world | 1. Doxy<br>2. Erythromycin<br>3. Penicillin G<br>-recurring fever every 8 days<br>-fevers break w/ drenching sweats<br>-rash and splenomegaly<br>-occasional meningeal involvement | 1. Acute = Staph aureus <br>coagulase +<br>-Vi: Protein A (binds IgG<br>Use Cidal drugs: always use aminoglycoside (gent), if acute (staph) | 1. Pathogenesis: turbulent blood flow (~ previous valve

Cardiac

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<thead>
<tr>
<th>Condition</th>
<th>Cause/Description</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Infective Endocarditis</td>
<td>1. Acute = Staph aureus coagulase +&lt;br&gt;-Vi: Protein A (binds IgG&lt;br&gt;Use Cidal drugs: always use aminoglycoside (gent), if acute (staph)</td>
<td>-Pathogenesis: turbulent blood flow (~ previous valve</td>
</tr>
</tbody>
</table>
| Preventing opsonization), coagulase = allows fibrin formation around organism, cell killers (e.g., homolysin, leukocidin) and tissue destroyers (hyaluronidase, staphylokinase), **penicillinase** - assault weaponry ~ toxins: exfoliatin ~ scalded skin syndrome, enterotoxin ~ food poisoning, TSST-1 - beat hemolytic grapes 2. More gradual onset - Strep Viridans and Group D strep (specifically enterococci faecalis and faecium) - both have extracellular dextran that allows them to bind heart valves - prey on hospitalized patients - viridans is from the moth (V for valves) and Enterococci are from GI and GU 3. Rare causes = gram –s, not as sticky, maybe pseudomonas aeruginosa - obligate aerobe member of the enterics - vi: polar flagellum, ECM busters (collagenase, elastase, fibrinolysin), and fancy stuff DNAase and some = anti-phagocytic combine with vanc or Penicillinase-resistant penicillin (met na ox) If subacute (D strep, viridans) combine with penicillin If pseudomonas suspected, consider pipe car tick compromise e.g. insufficiency = fibrin-platelet aggregate on low pressure side of valve (regurg. egenrall L but can be right ~ IVDU, CVC) → bacteria sticks (~ gram + are sticky, ~ bacteremia) → vegetation ~ avascular nature of valves - vegetation can break off, form Ag-AB complexes → GN, other forms of vasculitis (osler’s nodes, janeway’s lesions, conjunctival petechiae, roth’s spots. Rf ~ ab to ag:ab complexes - but Abs ineffective in handling gram positives, need PMNs - classic signs of HF, dyspnea, orthopnea rales~ edema, palpations, Jugular distension, Central hemorrhagic necrosis, arthmia - embolism = huge concern, dx with persistently pos. blood cultures (needs to be hematogenous
### Capsule
Has exotoxin ~ diptheria toxin inhibits protein synthesis by blocking EF2
- blue/green growth on some media
- always consider a possible agent in **BE PSEUDO:**
- burns, endocarditis, pneumonia, sepsis, external malignant otitis media, UTI, diabetic osteomyelitis
4. coag negative staph for prosthesis

### CNS Infections

<table>
<thead>
<tr>
<th>Bacterial Meningitis (note Abs especially important for Gram –s ~ repeating LPS and encapsulated orgs ~ requisite oponization)</th>
<th><strong>In healthy children and adults:</strong></th>
</tr>
</thead>
</table>
| 1. Strep pneumo-alpha hemolytic capsule-pneumolysin-multiple serotypes 2. Neisseria Meningitidis-found in nasopharynx of humans only-ferments maltose and glucose-vi: same as gonorrhea (pili, IgA1 protease, iron-abtracting proteins) but unique b/c **has capsule** 3. Type B H-flu (rare now because of vaccine)-obligate human dweller capsule (6 types, B = more) | Need Cidal drugs ~ low immune penetration in CSF
- Generally use broad spectrum 3rd generation cephalosporins + vanc to cover resistant strep pneumo
- Vaccines can be good prophylaxis, HI B for kids, meningococcal (college dorms), pneumococcal. |
| -bacteria in subarachnoid space → inflammation of meninges-hematologic spread to subarachnoid space requires intracellular or intercellular breech of BBB → colonization with minimal initial immune resistance (sacred area)-cytokines eventually releaed to prolif bacteria → increased BBB permeability, accounts for some labs findings (LP = WBCs with PMNs, low gluc, increased protein). |
In newborns and immunosuppressed: picture a crying “listerecol baby”!

1. Listeria monocytogenes
   - neonatal meningitis, transmitted vaginally or transplacentally from bacteremic mother, in IS adults ~ raw cheese and milk from infected cows
   -vi: flagella + hemolysin, but most importantly = toxins
   **listeriolysin O and phospholipases** (allows escape from phagolysosomes of macrophages)

2. E.coli
   -O, K, and H antigens; enteric
   -vi: fimbriae (pili) called colonization factor, siderophore (~ iron) and adhesins
   -spec. strains have spec. toxins

3. Group B Strep (most common cause of neonatal meningitis)
   -part of normal flora (25% of pregnant women carry B strep in birth canal)

-consequences we are most worried about = inflammation → compression of brain and specifically intracranial bf → hypoxemia/ischemia/necrosis → Helen Keller
-clinically look for buldging soft spot in infants, nuchal rigidity (meningismus) + Kernig’s/Brudzinski’s
-in general bacterial > viral ~ severity
<table>
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<tr>
<th>Viral Meningitis (aseptic meningitis)</th>
<th>Most commonly (70%) enteroviruses (Poliovirus, coxsackie A and B, echoviruses)</th>
<th>Supportive Pleconaril for compassionate use Vaccine for polio IVIG for antibody deficient as prophylaxis</th>
<th>GI/Resp tract → peyer's patches for replication → CNS entry via BBB or retrograde neuronal transport -<strong>enteroviruses unique because they</strong> don't need T cells to be cleared, largely antibodies, therefore normals but also Ab deficient (young, old, IC) at risk -summer to fall -huge difference from bacterial meningitis in that its usually not that severe, self-limiting w/in a week, and panel looks different with <strong>lympho predom, normal glucose (viruses don't use), and neg. gram stain</strong> -headache and stiff neck with acute onset generally normal mental status + pain/photophobia</th>
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<tr>
<td>Crytococcal meningitis</td>
<td>Crytococcus neoformans, yeast -respiratory transmission, often found in pigeon droppings (like histo) -has a polysaccharide capsule</td>
<td>Amp B and Flucytosine (synergy)</td>
<td>Opportunistic, seen in IC patients with AIDS often Causes subacute or chronic meningitis (headache, fever,</td>
</tr>
<tr>
<td>Viral Encephalitis</td>
<td>Most common form of sporadic viral encephalitis = 1. HSV - alpha herpes virus = primary infection ~ primary cutaneous exposure $\rightarrow$ retrograde transport to sensory ganglia where they can be latent. Stress $\rightarrow$ anxiety, fever, sunlight, or weakening of CMI $\rightarrow$ reactivation $\rightarrow$ direct neuronal spread to either periphery or CNS $\rightarrow$ cell damage from both direct lysis and immune response 2. Arborviruses (WEE, EEE, Venzuelan EE, WNV) - these viruses are carried by mosquitoes and ticks, generally cycled among the small animals and then transferred epi-zootically to larger mammals (humans, horses $\rightarrow$ disease). Bite $\rightarrow$ viremia $\rightarrow$ BBB, or resp transmission via olfactory bulb - besides encephalitis, Empirically meningitis antibacterials are started (ceftriaxone + vanc) and discontinued in favor of acyclovir upon HSV confirmation - arborviruses = supportive, preventative vaccine for travelers for JEV</td>
<td>With encephalitis you get fever, headache, progressive neurological symptoms (general mental status $\rightarrow$ focal) - key diagnostic tool with HSV is presence of very high RBCs in the LP (HSV-1 leads to uniquely pronounced BBB permeability increases $\rightarrow$ RBCs in tap) - Arborvirus encephalitis = also summer months (mosquito season) $\rightarrow$ fairly non-specific symptoms and in general, look for an unexpected onset in a healthy young person</td>
<td></td>
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</tbody>
</table>
associated with systemic febrile illness (WNV) and/or hemorrhagic fever
3. Rabies virus encephalitis (extremely rare ~ vaccine)

| Leptospirosis | Leptospira interrogans -zoonotic reservoir (dogs, cats, livestock, wild animals), we get the disease by **direct contact with infected urine or animal tissue through damaged mucous membranes or swallow** (contam water) | 1. penicillin G  
2. doxy | 1. first phase =leptospiremia = orgs in blood and csf → high spiking temps, headaches, severe muscle aches in thighs and lower back  
2. second phase = immune ~ IgM emergence and involves recurrence of above symptoms + meningismus often **Weil’s disease**: severe case of leptospirosis with renal failure, hepatitis, mental status changes, and hemorrhage in many patients |

| Urethritis/Cervicitis associated with STD risk | 1. *Neisseria Gonorrheae* (GC)  
*Neisseria gonorrhoea* (in sexually active people) -human reservoir (no immunity to repeated infections) ferments only glucose  
-fastidious, do not survive in | GC: Ceftriaxone (b/c high resistance to penicillins, quinolones)  
Chlamydia: need to use something that will kill that intracellular big= one dose of azithromycin) | GC and Chlamydia are both much more common in tends than adults.  
The biggest differences are:  
1. GC generally acquired from |
the env.
v: pili ~ adherence, antigenic variation, anti-phagocytic (~ binds tightly to host cell)
-IgA1 protease
-Outer membrane proteins including porins and opa (~ opacity)
-unique proteins that can abstract iron from human iron sources

2. Chlamydia Trachomatis
-most common bacterial STD, obligate intracellular bacteria
-rest like psittaci: -EB/RB lifestyle
-v: resistant to lysozyme (cw lacks muramic acid), prevents phagolysosome function

In practice, you treat for both (ceftriaxone + azithromycin)
For PID ~ to initial STD: Ampicillin/subactam (Unasyn) for Gram -s and anerobes + doxy for Chlamydia
(or ceftriaxone for GC, metronidazole for anaerobes, and doxy for chlamydia)

recent contact (2-3 days → pus) whereas Chlamydia is often acquired from remote past (makes sense ~ more asymptomatics)

2. While around 75% of women are asymptomatic with GC and chlamydia, only 5% of men as asymp with GC versus 50% in Chlamydia.

3. Discharge generally a little thicker for GC

4. GC can go systemic with some strains, more common in females than males → flu-like symptoms + possible septic arthritis + skin lesions on extremities

5. GC can see with gram strain, Chlamydia b/c intracellular can’t see

Complications: for both → lower GU scarring, and can be
transmitted to fetus. PID in females ~ damage to lower GU tract makes it more likely for normal flora bugs to ascend into uterus, fallopions, ovaries, and even systemically via peritoneum. Can → fertility problems, ectopic preg.

Genital ulcerative infections

From most common to least common:
1. HSV alpha herpes virus= primary infection ~ primary cutaneous exposure → retrograde transport to sensory ganglia where they can be latent. Stress ~ anxiety, fever, sunlight, or weakening of CMI → reactivation → direct neuronal spread to either periphery or CNS → cell damage from both direct lysis and immune response. HSV-1 ~ cold sores, HSV-1 ~ genital herpes
2. Treponema Pallidum (syphilis)
   - only found in humans, thick rigid spirochete that cannot be cultured
3. Haemophilus ducreyi

Acyclovir is used for HSV
Azithromycin or ceftriaxone are used for chancroid
Syphilis = penicillin (along with Group A strep the last two bugs exquisitely sensitive to penicillin)

Distinguishing factors between HSV, syphilis, and chancroid
1. Number: syphilis and chancroid present as 1-2 lesions on genitals while HSV = clusters of tiny vesicles
2. Pain: both HSV (blisters!) and Chancroid are painful but syphilis is non-painful
3. Appearance: HSV = flat, syphilis = indurated, and chancroid = dirty base
4. Incubation: both herpes and chancroid present after a couple days
<table>
<thead>
<tr>
<th>Post-primary syphilis</th>
<th>Treponema Pallidum (syphilis)</th>
<th>Penicillin! (dose ~ stage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-most common in southern U.S.</td>
<td>-only found in humans, thick rigid spirochete that cannot be cultured</td>
<td>Ceftriaxone if rash, AG or doxy if anaphylactic</td>
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<td>Warn about common Jarisch-Herxheimer rxn (tons of bugs lysing → cytokine release → flu-like symptoms), therefore, often given with acetaminophen</td>
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<td>For time course think rules of 6: 6 week from incubation until chancre dissapeering with chancre coming up in the middle, 6 weeks after chancre heals → secondary syphilis. 6 weeks for secondary syphilis to resolve. 66% of latent stage patients have resolution, it takes at least 6 years (maybe 40) → tertiary syphilis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-primary ~ chancre and disappear</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-secondary ~ systemic spread → flu-like symptoms, ulcers/skin dudes on various sites: rash on palms and soles, white mucosy mouth ulcers, condyloma lata</td>
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<td>-tertiary ~ CNS/PNS damage (neurosyphilis, senses messed up, gate probs) CV damage ~ obliterative arteriolitis (vasa vasorum) ~ CNS and ascending aortic aneurysm, gummas</td>
</tr>
</tbody>
</table>
### UTIs

#### Cystitis (general UTIs risk factors listed here)

<table>
<thead>
<tr>
<th>Uncomplicated</th>
<th>Young woman:</th>
<th>Complicated</th>
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</table>
| 1. E. Coli (most common in women)  
-O, K, and H antigens; 
-enteric  
-vi: fimbriae (pili) called colonization factor,  
siderophore (~ iron) and adhesins  
spec. strains have spec. toxins  
2. Staph Saprophyticus  
(second most common in young women)  
-coagulase negative staph  |
| 1. TMP/SMX x 3 days + pyridium (anesthetic for urination)  
2. Quinolone (cipro) is alternative  
Older women:  
Try TMP/SMX may need to add  
All other non-E. Coli UTIs ~ tx for longer 10-14 days ~ culture/susceptibility |
| 1. Klebsiella pneumoniae  
-capssule, nonmotile,  
-common cause of hospital acquired UTIs and sepsis  
2. Proteus  
-unique b/c has urease which splits urea into NH3 and CO2 (gives urine high ph)  
-“swarming” motility  
3. Psudomonas  
--obligate aerobe member of the enterics  |
| Prophylaxis in young women (> 3 UTIs/year)  
= single low dose  
bactrim daily, older women try same thing  
but during menopause,  
estriol cream can work to restore protective vaginal mucous membrane  
Try to avoid prophylaxis in others ~ resistance  |
| -UTIs most common in young women, risk increases with onset of sexual activity (urethra closer to GI tract flora)  
-underlying risk factors for UTIs in anyone = urine stasis (obstruction ~ prostate, strictures, stones), pregnancy (uterus pushes on bladder → reflex into higher GU tract), Neuro issues (Dm ~ autonomic neuropathy, stroke, MS, SC lesions)  
-infections can start in urethra bladder and ascend to kidneys → bloodstream,  
hematogenous infection rare (think endocarditis)  
-cystitis: suprapubic tendernis, frequent/painful voiding, rare to have systemic symptoms  |
| Dx ~ VDRL and RPR (nonspecific) and FTA-abs (specific) to confirm  |

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hematogenous infection rare (think endocarditis)  
-cystitis: suprapubic tendernis, frequent/painful voiding, rare to have systemic symptoms  
-Dx ~ clean catch mid
- vi: polar flagellum, ECM busters (collagenase, elastase, fibrinolysin), and fancy stuff DNAase and some = anti-phagocytic capsule. Has exotoxin α ~ diptheria toxin= inhibits protein synthesis by blocking EF2
- blue/green growth on some media
- always consider a possible agent in **BE PSEUDO:** *burns, endocarditis, pneumonia, sepsis, external malignant otitis media, UTI, diabetic osteomyelitis*

**Elderly men**
Enterococcus faecalis and faecium
-- both have extracellular dextran that allows them to bind heart valves
- prey on hospitalized patients

**Pyelonephritis**

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<tr>
<th>Same as above</th>
<th>IV drugs if patient looks toxic</th>
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<tbody>
<tr>
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<td>Otherwise for young women:</td>
</tr>
<tr>
<td></td>
<td>1. Quinilone (Cipro)</td>
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<td>2. Bactrim is now second choice</td>
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</table>

stream technically > 10000, but symptoms can present earlier so we generally don’t even culture young women (playing percentages = ecoli)
<table>
<thead>
<tr>
<th>Sepsis</th>
<th>Bacterial Sepsis</th>
<th>Physiologic support</th>
<th>-bacteremia does not = sepsis (only bacteria in blood vs. systemic illness ~ hemodynamic derangements and organ malfunction)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A variety of bugs + fungi + viruses + parasites can causes</td>
<td>-maintain BP</td>
<td>-generally = severe immune response ~ microbial products (LPS, lipoteichoic acid) vs. host reticuloendothelial cells</td>
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<td>Most common bacterial causes from intrinsic flora = staph aureus amd Beta-hemolytic strep, but again can be any bug → bloodstream</td>
<td>-correct acidosis (buildup of CO2 ~ decreased removal of wastes)</td>
<td>-high risk ~ disruption of barriers/mechanical obstruction (cholecystitis, UTIs, perf. Gut, burn wounds) and specific immune defects (aplenia/Ig def. _ decreased Abs ~ encapsulated bacteria, neutropenia ~ gram –s from gut, first line of defense), sporadic occurance ~ post-op,</td>
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<td>-don’t even necessarily need bugs → sepsis, e.g. pancreatitis can cause it</td>
<td>-Increase oxygenation</td>
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<td>Attack microbes responsible,= braod-spectrum high does IV antibiotics until spec. etiology known = Vanco + ceftriaxone or piperacillin/tazobactam or imipenem.</td>
<td>-maintain cardiac function</td>
<td></td>
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<tr>
<td>Catheters, etc.</td>
<td>LPS/LTA → cytokines → endothelia damage → leak = hypoperfusion of vital organs (organ failure) + hypotension ---DIC (decrease platelets, fibrinogen, increase in clotting markers)</td>
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**GI infections**

- General: Young (high exposures, more severe illness ~ faster dehydration) and elderly most at risk (decreased immunity, hypochlorhydria, comorbidities), decreased mucus, motility, normal flora (anaerobic colonization resistance) at highest risk
- Most spread fecal—oral
- Response ~ expel bug = cramps (increased motility), nausea/vomiting, diarrhea
- **Bacterial:** upper = secretory (in general acid sensitive), lower = secretory (in general acid resistant but there are exceptions), not true for viruses
- Careful history important, note signs of dehydration = orthostatic, tachy, poor skin turgor, dry mouth/axillary, lethargy/confusion
- Culture for bacteria and/or toxin, also look for parasites

| Bacterial Gastroenteritis | These bugs are generally acid sensitive and do their damage via toxin release 1. ETEC/EAEC ~ traveler’s diarrhea -ETEC ~ pili (colonization factor) binds intestinal epis → release exotoxins similar to cholera toxin (LT and ST) (G protein → camp → increased NaCl bicarb K+ secretion and decreased NaCl reabsorption. Water follows and → rice water diarrhea. | Fluoroquinolone x 3 days + fluid therapy with electrolytes  Prophylaxis currently not recommended ~ resistance | -With the bacterial gastroenteritis you get rice water diarrhea with generally not a ton of nausea and vomiting  Associated with contaminated food and water  Note for differential: nausea and vomiting are much more likely with viral |
### Viral Gastroenteritis

| -like cholera toxin | -any foreign travel with foreign bugs can cause
| 2. EPEC | 3. Vibrio Cholerae |
| -causes same thing as ETEC/EAEC, not common in U.S., associated with epidemics during catastrophes (e.g. wars, natural disasters) |

| Both of the viral gastroenteritis bugs are acid resistant (read: low infectious dose. |
| -short incubation period and high attack rate for both  |
| 1. Norovirus -most common cause of infectious diarrhea in adults and older children |
| -this virus can exist for a while in environment (think of cruise ships, nursing homes) |
| -fecal → oral, but spread person → vomitus, stool, via env. |
| -best way to get rid of virus would be to blow the ship up b/c the virus itself is heat labile |
| 2. Rotavirus -most common cause of infectious diarrhea in youngins, 6-24 months |
| -also starts with fecal oral |

| Supportive |
| Rotarix or RotaTeq available to prevent rotavirus |

| Both noro and rota virus are associated with secretory diarrhea + nausea/vomiting, they may or may not be associated with fever |
| -they are both self-limited and last a few days |
| -the biggest differences between the two = age group most commonly effected (noro ~ adults and older children on the cruise, rota ~ youngings and elderly), noro sticks around on the cruise ship, and vaccine is only for rota |
transmission but does not stick around in environment, primarily effects infant and elderly ~ a few serotypes, therefore exposure = lifetime protection for everyone else

| Bacterial Colitis | Almost all kill colonic epi cells → inflam diarrhea
Almost all are acid resistant
1. C. diff
   -anaerobic intestinal bug that can also be found as endospores in hospitals and nursing homes
   -motile
   -has toxin A ~ diarrhea (works on TJs) and toxin B ~ cytotoxic to colonic epithelial cells → inflam diarrhea.
Psuedomembranous colitis is almost always associated with Abx usage but there are growing excpetions
2. Camylobacter jejuni
   -most common cause of inflammatory diarrhea in U.S.
   -zoonotic, found in wild and domestic animals, especially chicken (uncooked meat and unpasteurized milk
   -it has an enterotoxin that is like cholera toxin and LT of E. Coli (diarrhea) but alo a cytotoxin that destroys
   Anything but EHEC and non-typhi Salmonella, as long as the patient is showing symptoms, treat with Cipro + supportive care.
   We don’t use Abx for EHEC b/c it’s been shown to arrest the bacteria's DNA synthesis which is a signal to produce more toxin (makes things worse)
   We don’t treat non-typhi salmonella b/c its not that big of a deal usually
   If psuedomembranous colitis, switch from current Abx if possible to metronidazole if moderate or oral vanco if serious
| -the range of inflam diarrhea goes from campylobacter, most common in U.S. often diagnosed and self-limiting to Shigella which can be severe (associated with frequent small volume/bloody stools, tenesmus, and a toxic appearance.
Contaminated water and pools are sites for both shigella and EHEC
The only one of these with a potentially very severe complication (besides dehydration) = EHEC = Hemolytic uremic syndrome (toxin enter destroys endothelium of bv → renal failure, and associated hemolytic anemia (passed jagged bv) + thrombocytopenia
mucosal cells (→ inflam diarrea)
3. Shigella
- found in humans, enters via fecal-oral
- non-motile
- shiga-toxin: inactivates 60s ribosome, inhibiting protein synthesis and killing the epithelial cells → bloody diarrhea with mucus and pus
4. EHEC/EIEC
- wanna be shigella with shigalike toxin, invade colonic epis and can be associated with systemic symptoms
- EHEC found in beef (ground beef) and other foodstuffs linked to cows
5. Salmonella
- the strain that gives diarrhea is not the typhi but rather non typhic groups.
- non-typhi groups are zoonotic (pet turtles, chickens, uncooked eggs)
- bug is motile, produces **H2S** and has a capsule called the VI antgen that protects it from intracellular killing
Entamoeba histolytica as an uncommon parasite

<table>
<thead>
<tr>
<th>Enteric fever</th>
<th>Salmonella Typhi</th>
<th>Always treat:</th>
<th>Both salmonella species are acid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- same virulence factors as</td>
<td>1. Ceftriaxone</td>
<td></td>
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<tr>
<td>Food poisoning</td>
<td>1. Staph aureus</td>
<td>Supportive</td>
<td>Staph ~ potato salad</td>
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<tr>
<td></td>
<td>coagulase +</td>
<td>Practice safe food practices</td>
<td>B. Cereus ~ fried rice</td>
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<td></td>
<td>-Vi: Protein A (binds IgG preventing opsoniazation), coagulase = allows fibrin formation around organism, cell killers (e.g. hemolysin, leukocyidin) and tissue destroyers (hyaluronidase, staphylokinase), penicillinase</td>
<td></td>
<td>Both heat stable toxins</td>
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<td>- Toxins are heat stable</td>
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<td>These bugs have short incubation period and an explosive onset, nausea + vomiting</td>
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<tr>
<td></td>
<td>2. B. Cereus</td>
<td></td>
<td>Diarrhea is actually rare</td>
</tr>
<tr>
<td>non-typhis, motile with Vi antigen (capsule), produces H2S, the biggest difference is that it is only found in humans -carriers can transmit to other humans via fecal → oral route (Typhoid Mary)</td>
<td>2. Cipro</td>
<td>sensitive (need high infectious dose) Enteric fever ~ systemic illness with spread of organism, may or may not be present with diarrhea after the first bit, can localize and cause focal infection (meningitis, arthritis, osteomyelitis) especially in asplenic patients (~ Ab ~ opsonization of encapsulated orgs) - clinical clue = WBC count is often not elevated</td>
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</tbody>
</table>
| -several heat stable toxins  
3. C. Perfringens  
Clostridium perfringens (anaerobics)  
-anaerobic, non-motile organism  
-vi: alpha toxin (lecithinase) and other tissue destructive enzymes → gas gangrene + others  
-unique b/c toxins produced in vivo after ingestion |

Herpes Viruses  
-in general, these viruses have double stranded DNA genomes with a tegument that lies between the nucleocapsid and the envelope.  
-viruses have both a lytic (virus → host nuc, uses DNA rep. machinery to transcribe genes in temporal fashion (immediate early (suppress immune system), early, late (structural proteins and glycoproteins, assembly) and latent phase (alpha n DRG, Beta in minocytes, and gamma in B lymphocytes  
-hepes uses a viral-specific thymidine kinase that can be used as a potential drug target  
-generally rely on PCR for diagnosis |

| HSV-1 and HSV-2  
-alpha herpes virus= primary infection ~ primary cutaneous exposure (sexually transmitted) → retrograde transport to sensory ganglia where they can be latent. Stress ~ anxiety, fever, sunlight, or weakening of CMI → reactivation → direct neuronal spread to either periphery or CNS → cell damage from both direct lysis and immune response  
-viral shedding occurs in | 1. Acyclovir  
2. Valacyclovir  
3. Famcyclovir  
4. Trifuridine eye drops for corneal infections |

Gingivostomatitis  
(cold sores ~ hsv-2) and genital herpes ~ hsv-1, can both be reactivated in times of stress  
-number 1 cause of viral encephalitis in u.s.  
-herpetic keratitis of the eye = most common cause of corneal blindness in the U.S.
<table>
<thead>
<tr>
<th>Virus</th>
<th>Description</th>
<th>Prevalence</th>
<th>Treatment Options</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>VZV</td>
<td>VZV - alpha, therefore latent in DRG - highly contagious! ~ respiratory secretions and/or contact with ruptured vesicles - zoster (shingles) = reactivation from DRG</td>
<td>neonatal herpes (TORCHES) disseminated</td>
<td>1. Acyclovir 2. Valacyclovir 3. Famciclovir</td>
<td>VZV → chicken pox: - 2 wk incubation → fever and headache → vesicles arise on trunk and face and spread to extremities. Pox at all different points. - pneumonia or encephalitis can occur in IC patients Zoster → shingles - painful eruption of vesicles along a dermatome, usually lasts ~ 3 wks. - especially concerned with herpes zoster ophthalmicus (shingles along V1 can involve cornea)</td>
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<tr>
<td>CMV</td>
<td>CMV - B virus, infects monocytes/macrophages virus is ubiquitous, present in milk saliva, urine, tears with transmission ~ prolonged exposures e.g. children in households or day car centers (can also be sexually transmitted) - many of us have</td>
<td>TORCHES - CMV mono - Reactivation in</td>
<td>1. Gancyclovir 2. Foscarnet 3. Cidofovir</td>
<td>- histopathology reveals large (cytomegaloc) cells with intranuclear and cytoplasmic inclusion bodies - aysymptomatic infection (latent) - TORCHES - CMV mono - Reactivation in</td>
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<td>Oncogenic Viruses</td>
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<tr>
<td><strong>EBV</strong></td>
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<td>EBV -gamma virus, infects B cells and possibly transforms them -spread by intimate contact from asymptomatic shredders of EBV (smooches)</td>
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<td>Only supportive -differential white count shows elevated “atypical lymphocytes” -Infectious mono: fever, sore throat, severe lethargy, enlarged lymph nodes and spleen Complication: associated with Burkitt’s B cell lymphoma (virus can be oncogenic)</td>
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<td><strong>HPV</strong></td>
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<td>HPV -small, circular dsDNA virus -transmission ~ skin-skin, skin-fomite contact → oncogenesis through E6 and E7 genes inhibition of p53 and Rb respectively (apoptosis and stop-light) -&gt; 100 genotypes, replication and life cycle linked to keratinocyte differentiation (starting form basal layer) -Genotypes 1, 2, 3, 4, 10 → cutaneous warts -Genotypes 6, 11 = 90% of low malignancy risk anogenital warts -Genotypes 16, 18 = 70% of</td>
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<td>Ablative therapy (must remove all of area) laser, liquid nitrogen, surgery, etc. Subunit vaccine contains only L1 protein shell, recommended for females 9-26, OK for immunosuppressed/lactating</td>
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<tr>
<td><strong>Polyomaviruses</strong></td>
<td>- same family as pappilomavirus</td>
<td>- only speculative link to cancer</td>
<td>- BK virus → hemorrhagic cystitis and polyomavirus nephropathy (common in kidney transplant patients)</td>
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<td>- BK virus → hemorrhagic cystitis and polyomavirus nephropathy</td>
<td>- JC virus → progressive multifocal leuokencephalopathy (PML)</td>
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<tr>
<td><strong>Kaposi's sarcoma-associated herpes virus (KSHV)</strong></td>
<td>- Gamm herpes virus (found in B cells) aka HHV-8</td>
<td>Incurable, but try to minimize underlying IC</td>
<td>- sporadic in IC hosts, associated with red, purple, brown, black popular nodules (look like large moles), can appear on skin but also mouth, lung, Gi tract - its technically a malignancy of the lymphatic endothelium (leaking of these channels → dark welts)</td>
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<td>- linked HIV patients who aquired the virus through sex vs. other means (e.g. MSM)</td>
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<tr>
<td><strong>Hepatitis viruses</strong></td>
<td>- Hepatitis A and E are fecal oral (anal, enteric), BCD are parenteral (fluid → blood, potentially STD, transfusion, needle sticks, sexual, across placenta)</td>
<td>- viral hepatitis associated with acute elevation of AST and Alt ~ hepatocyte necrosis with some destruction of pericicular cells (= little elevation of alk phos and GGT). As infection worsens, liver swells and caniculi narrows → back up of bilirubin into the blood. This narrowing of canaliculi is also associated with further damage to lining cells ~ bilirubin, GGT, and Alk phos are higher later in the course.</td>
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<td><strong>Hepatitis A</strong></td>
<td>- Hep A: ssRNA virus transmitted fecal → oral</td>
<td>Supportive care</td>
<td>Associated with acute viral hepatitis (fever, jaundice, and a painful enlarged liver.</td>
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<td>Pooled immunoglobulin</td>
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<tr>
<td>Hepatitis</td>
<td>Description</td>
<td>Prevention</td>
<td>Complications</td>
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<td><strong>Hepatitis B</strong></td>
<td>Ds DNA virus transmitted parenterally - antigens: HBsurface (HBs) ~ envelope + capsid associated proteins → immunogenic (present = disease, abs = immune) - HBcore (HbC) ~ genetic material and machinery ~ age (present = new, abs = old) - HBe antigen = soluble component of core, only released during active disease (present = high infectivity, abs = low infectivity)</td>
<td>Prevention: Hep B recombinant vaccine Interferons or nucleoside analogs may help</td>
<td>Co-infection with D (D is defective and it needs is Buddy) Complications: Primary HCC, cirrhosis</td>
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<tr>
<td><strong>Hepatitis C</strong></td>
<td>ssRNA virus transmitted parenterally</td>
<td>Combination therapy with interferon and ribavirin</td>
<td>Associated with acute viral hepatitis, differs from hep b in that 85% of patients will develop chronic hepatitis with 20% getting cirrhosis.</td>
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<td>Hep D</td>
<td>Control of HBV, the HBV vaccine protect against HDV</td>
<td>-associated with coinfection with hep D, or part of superinfection where a patient with chronic HBV is infected with HDV (requires lack of ability to make antiHBs antibodies) Complications=fulminant hepatitis, cirrhosis</td>
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<tr>
<td>Hep D</td>
<td>Incomplete virus (defective) that is only active with its buddy (Hep B), transmitted parenterally</td>
<td>-</td>
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<tr>
<td>Hep E</td>
<td>ssRNA transmitted fecal→oral</td>
<td>Like Hep A → acute hepatitis, responsible for epidemics in Asia but very rare in US</td>
<td></td>
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<tr>
<td>Abscesses</td>
<td>Abscesses are often polymicrobial, aerobes first use up O2 then anaerobes: Gram-negative bacilli: 1. Bacteroides Fragilis and melaninogenicus -along with other bacteroides species, member of the normal flora of intestine -has a polysaccharide capsule -no real virulence factors, only really problematic when fragilis enters peritoneal cavity (e.g. after intestinal rupture) or</td>
<td>Surgery is often recommended to drain, debride, aerate the area Anaerobic bugs generally do not respond to usual abx, often because of beta-lactamases 1. Metronidazole (Flagyl) 2. Penicillin/Beta Lactamase inhibitor combo</td>
<td>-abscesses = walled of collection of purulent material -often result from some penetrating trauma that brings bacteria from one environment (gut, skin) to a different environment that is often not vascularized (prime for anaerobic growth) ~ deep to mucosa -aerobes, then anaerobes -non-specific flu-like</td>
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</tr>
</tbody>
</table>
| melaninogenicus → periodontal disease and aspiration pneumonias  
| (found in mouth, vagina, intestine)  
| 2. Prevotella and Porphyromonas  
| Gram-positive cocci: Peptococci and peptostreptococci  
| -mouth, vagina, and intestine associated with abscesses and aspiration pneumonias  
| Gram positive bacilli:  
| 1. Clostridium  
| Gram positive anaerobes that produce toxins  
| 2. Propionibacterium can infect artificial hips and knees  
| symptoms + specific inflammation signs of area  
| -can be localized e.g. in liver, of diffuse e.g. peritonitis (note that peritonitis is indicated with silent bowel sounds—ileus,, rebound tenderness, and involuntary guarding  
| -Clues to presence of anaerobic infections or mixed aerobic/anaerobic infections: near mucosal surface, foul smelling exudate, gas in tissues (e.g. clostridium --.> gas, mixed zoo of bugs on culture, no growth on "routine" aerobic cultures  
| 1. Classically = actinomyces israelli  
| Classic b/c it is anaerobic, members of the normal flora of mouth and GI tract.  
| 2. Nocardia asteroids -more unique in this category b/c it is aerobic, and partially acid-fast ~ mycolic acid in the wall.  
| Also never part of the normal flora, comes from  
| Tx: for actinomyces = Pencillin G and/or surgery  
| Tx for nocardia = TMP/SMX  
| Actinomyces is associated with eroding abscesses of the mouth, lung or GI tract called cervicofacial, thoracic, abdominal actinomycosis, look for “yellow sulfar granules” at infectious site pus. Associated with healthy hosts  

Fungi-like bacteria associated with abscesses: Actinomyces and Nocardia (both really gram + rods)
| Toxin-producing anaerobic infections | Clostridium species, all produce gas  
1. C. botulinum -found in soil but also stored vegetables (home-canned, Ziploc storage bags), smoked fish, fresh honey -releases neurotoxin produced in vitro and in vivo upon death that inhibits release of ACH from peripheral nerves. The toxin is heat labile, so this disease normally effects mom and grandma who are cooking and tasting while they are  | Botulism and tetanus give anti-toxin + penicillin + supportive care  
Perfringens = surgery + penicillin  
C diff = switch to metro or oral vanco  | Botulism ~ descending flaccid paralysis (start with dilated pupils, dry mouth, then cranial nerve palsy, and eventually respiratory muscles)  
Tetanus ~ spastic paralysis (opisthotonos and trismus)  
C. perfringens (think of the farm accident, traumatic dirty wound)  |

often recently having undergone surgery → abscess that tears through fascial planes e.g. lumpy jaw (non-healing abscesses break through and drain at surface)  
Nocardia is differentiated from actinomycetes based on its bug x-tics (e.g. aerobic, acid-fast, never part of normal flora) but also b/c it, unlike actinomycetes, generally occurs in Ic patients. → pneumonia, abscesses in CNS, kidney, skin

Toxin-producing anaerobic infections

- C. botulinum: found in soil but also stored in vegetables (home-canned, Ziploc storage bags), smoked fish, fresh honey. It releases neurotoxin produced in vitro and in vivo upon death that inhibits the release of ACh from peripheral nerves. The toxin is heat labile, so this disease normally affects mom and grandma who are cooking and tasting while they are.

- C. perfringens: causes botulism and tetanus.

- C. difficile: causes botulism.

- Nocardia: is differentiated from actinomycetes based on its bug x-tics (e.g. aerobic, acid-fast, never part of normal flora) but also because it generally occurs in Ic patients. It can cause pneumonia, abscesses in CNS, kidney, skin.

- Toxin-producing anaerobic infections: produce gas. 1. C. botulinum - found in soil but also stored in vegetables (home-canned, Ziploc storage bags), smoked fish, fresh honey. It releases neurotoxin produced in vitro and in vivo upon death that inhibits the release of ACh from peripheral nerves. The toxin is heat labile, so this disease normally affects mom and grandma who are cooking and tasting while they are.

- Botulism and tetanus: give anti-toxin + penicillin + supportive care.

- Perfringens: surgery + penicillin.

- C. difficile: switch to metro or oral vanco.

- Botulism: descends flaccid paralysis (start with dilated pupils, dry mouth, then cranial nerve palsy, and eventually respiratory muscles).

- Tetanus: spastic paralysis (opisthotonos and trismus).

- C. perfringens: think of the farm accident, traumatic dirty wound.

- Nocardia: is differentiated from actinomycetes based on its bug x-tics (e.g. aerobic, acid-fast, never part of normal flora) but also because it generally occurs in Ic patients. It can cause pneumonia, abscesses in CNS, kidney, skin.
2. C. Tetani
- found in the soil (step on a nail), toxin produced \textbf{in vivo} blocks inhibitory interneurons from doing their job (releasing GABA and glycine) thereby resulting in sustained muscle contraction.

3. C. perfringens
- anaerobic, non-motile organism
- vi: alpha toxin (lecithinase) and other tissue destructive enzymes $\rightarrow$ gas gangrene + others
- toxin produced \textbf{in vivo}

4. C. diff
- anaerobic intestinal bug that can also be found as endospores in hospitals and nursing homes
- motile
- has toxins \textbf{made in vivo}, A $\sim$ diarrhea (works on TJs) and toxin B $\sim$ cytotoxic to colonic epithelial cells $\rightarrow$ inflam diarrhea.
Psuedomembranous colitis is almost always associated with Abx usage but there are growing exceptions

Fungi Generalities (see above for specific differentials)
- fungi = non-motile, eukaryotic orgs ($\sim$ organelles) with CW that reproduce sexually by spores. Ergosterols in \textbf{cell membrane} (not CW). In general they are usually aerobic, live off of consuming dead material or parasitizing living
- Mold = characteristic hyphae (multicellular branching filaments) with conidia—a sexual reproductive structure at the tips of the hyphae that contains the spores spread by wind, masses of hyphae = mycelium (fuzzy growth). Examples = aspergillus, mucor, rhizopus
Yeast = round or oval single cells that are often found in the normal flora. They reproduce by budding and sometimes form pseudohyphae = string of attached cells produced by incomplete budding. Examples = Candida, Cryptococcus
- Dimorphic fungi: mold (infectious form) in environment and yeast (pathogenic form) in the body.
- Fungi are broken up into two large groups called the endemic mycoses and the opportunistic mycoses
- Endemic ~ histo, blasto, coccidioides = true pathogens that are dimorphic, environmental, and largely depend on CMI (largely like TB ~ eaten by macros → CD4 ~ destruction) vs. opportunistic that cause serious disease in Ic host, normal flora, serologies not helpful (b/c they are part of normal flora), PMS are main defense (first line protection against these mucosal agents that spread) although CMI is also important in crytocccus. Anchor: cryto → CNS, not going to rely on just polys, PCP → massive lung infection, don’t fuck around

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<thead>
<tr>
<th>Miscellaneous</th>
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<tbody>
<tr>
<td>Brucellosis</td>
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<tr>
<td>Different brucella species found in goats, cattle, pigs, and dogs, we get it primarily from direct contact with live-stock or aborted placentas or ingestion of infected milk products (it has a virulence factor that has tropism for a sugar found in animal placentas)</td>
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