NR511 Midterm Study Guide Worksheet

Disease	Risk	Subjective Finding	Objective Findings	Diagnostics	Treatment	Education
GI DISORDERS						
Appendicitis	-Most common between 10-30yrs; but can occur at any age; rare in infants and older adults -men more at risk - Diets low in fiber, high in fat, refined sugars, & other carbs at increased risk. - Obstruction of appendix is cause of majority of appendicitis - contributing factors: Intra-abdominal tumors, positive family hx - Recent roundworm infection or viral GI infection	-Dx made clinically, based primarily on H&P exam - Classic presentation includes acute onset of mild to severe colicky, epigastric, or periumbilical pain - Pain is vague at first then localizes within 24hrs to RLQ - Pain exacerbated by walking\coughing - Men may feel radiated pain in testes - Abd muscle rigidity, N\V, anorexia - Mildly elevated temp 99-100F common - If RLQ accompanied by shaking chills, perforation should be suspected - Older adults may present with weakness, anorexia, abd distention, mild pain leading to delayed dx and increased morbidity.	-May have HTN\tachy proportional to pain\symptoms -When lying flat, may flex R knee to relieve tension in abd muscle -Pain with palpation in abd, diffuse in early stages. Localized to RLQ later -Positive for rebound pain; ask pt to cough to localize pain location -Sudden cessation of pain means perforation and is ER	-Labs are not diagnostic and nonspecific -Women should have urine human chorionic gonadotrophin to r\o ectopic pregnancy - **+Rovsing's Sign-** deep palpation & release in LLQ causes rebound pain in RLQ - **+Psoas Sign**- lift R leg against gentle pressure causes pain - **+Obturator Sign-** flex R hip & knee and slowly rotate internally causes pain - **+McBurney's Sign**- pain with pressure applied to point between umbilicus & ilium - x-ray\CT helpful when paired with positive H&P findings	-Surgical; preoperative care, NPO, correction of fluid\electrolyte imbalances -Avoid narcotics -Atb with 3rd gen cephalosporin; Ex: ampicillin, gentamycin, flagyl	-F\U with surgeon -Ambulation after surgery -Adv diet when bowel sounds return -Return to hosp with s\s of infection -Avoid heavy lifting for at least 2 wks
Celiac disease ** (autoimmune disorder caused by an immunologic response to gluten)	Mostly diagnosed in adulthood. A family member with celiac disease or dermatitis herpetiformis Type 1 diabetes	Many asymptomatic. May complain of diarrhea, gas, dyspepsia, wt loss. Atypical symptoms: fatigue, bone or joint pain, arthritis, osteoporosis, or	Muscle wasting (anemia), reduces subcutaneous fat, ataxia, & peripheral neuropathy (vitamin B12 deficiencies) osteoporosis or osteopenia (bone loss)	Serologic testing for anti-tTG IgA antibody Total IgA (2% of pts have IgA deficiency and will falsely test negative) duodenal biopsies	lifelong adherence to a strict gluten-free diet. Referral to a dietician to help. Some pts may need treatment with immunomodulating	teaching related to gluten free diet. Some people with celiac disease have vitamin or nutrient deficiencies that do not cause them to feel ill, such as anemia due to iron

	Down syndrome or Turner syndrome Autoimmune thyroid disease Microscopic colitis (lymphocytic or collagenous colitis) Addison's disease	osteopenia (bone loss) liver and biliary tract disorders (transaminitis, fatty liver, primary sclerosing cholangitis, depression or anxiety peripheral neuropathy seizures or migraines missed menstrual periods infertility or recurrent miscarriage canker sores inside the mouth dermatitis herpetiformis (itchy skin rash)	hypothyroidism Pts with dermatitis herpetiformis found to have signs of celiac disease on intestinal biopsy.	 Test for nutritional deficiencies associated with malabsorption of C.D. (hemoglobin, iron, folate, vit B12, Calcium, and Vitamin D.)	agents.	deficiency or bone loss due to vitamin D deficiency. However, these deficiencies can cause problems over the long term. Untreated celiac/developing certain types of gastrointestinal cancer. This risk can be reduced by eating a gluten-free diet.
Cholelithiasis	is the formation of gallstones and is found in 90% of patients with cholecystitis. --Risk factors--2 types of stones (cholesterol and pigmented) a. Cholesterol (most common form): female, obesity, pregnancy, increased age, drug-induced (oral contraceptives and clofibrates: cholesterol lowering agent), cystic fibrosis, rapid weight loss, spinal cord injury, Ileal disease with extensive resection, Diabetes mellitus, sickle cell anemia. b. Pigmented: hemolytic diseases, increasing age, hyperalimentation	Patient complaint of indigestion, nausea, vomiting (after consuming meal high in fat), and pain in RUG or epigastrium that may radiate to the middle of the back, infrascapular area or right shoulder.	Right side involuntary guarding of abdominal muscles, Positive Murphy's sign, possible palpable gallbladder, Low grade fever between 99-101 degrees. Possible jaundice from common bile duct edema and diminished bowel sounds.	Mild elevation of WBC up to 15, 000 Abdominal Xray: Quick, noninvasive, reliable, and cost-effective means of identifying the presence of cholelithiasis.	*a. Initial management--begins with definitive diagnosis. When asymptomatic (normally an incidental finding while exploring another problem) require no further treatment except teaching s/sx of "gallbladder attack". Nonsurgical candidate can be treated with dissolution therapy or lithotripsy. Acute includes hydration (IV fluids), antibiotics, analgesics, GI rest.* *b. Treatment of choice for Acute cholecystitis is early surgical intervention after stabilization. Poor surgical risk may benefit from cholecystectomy operatively or percutaneously.*	Nonsurgical intervention: weight loss, avoidance of fatty foods to decrease attacks, alternative birth control for persons taking oral contraceptives, menopausal women taking estrogen informed about alternative sources of phytoestrogens (soy products).

	(artificial supply of nutrients, typically IV), cirrhosis, biliary stasis, chronic biliary infections.					
Crohn's **	Ages 15-25 of onset and then again at 50-80. Familial tendency, smoker Carcinoma less common in patients with CD due to treatment sometimes colectomy	Mild-Four or fewer loose bowel movements per day, can have small amounts of blood and mucus in the stool, and cramping in the rectum. Moderate-4-6 loose bowel movements per day containing more blood and mucus and other sx such as tachycardia, weight loss, fever, mild edema. Severe-frequent bloody bowel movements (6-10), abd pain and tenderness, sx of anemia, hypovolemia, impaired nutrition. Most common sx are abd cramping/tenderness, fever, anorexia, wt loss, spasm, flatulence, RLQ pain or mass	Tenderness in LLQ or across entire abd with guarding and abd distension. DRE performed to look for anal and perianal inflammation, rectal tenderness, and blood in stool. S/Sx of peritonitis and ileus may be found depending on severity of crohns. Tender mass in RLQ, anal fissure, perianal fissure, edematous pale skin tags. Extra intestinal finding may be episcleritis, erythema nodosum, nondeforming peripheral arthritis, and axial arthropathy	Stool analysis to r/o bacterial, fungal, or parasitic infection for cause of diarrhea. CBC to check for anemia, eval for hypocalcemia, vit D deficiency., hypoalbuminemia, and steatorrhea. LFT to screen for primary sclerosis cholangitis, and other liver problems assoc with IBD. Check fluid and electrolytes. May have elevated WBC count and sed rate and prolonged prothrombin time. Barium upper GI series, colonoscopy, and CT to determine bowel wall thickening or abscess formation	Glucocorticoids, there is no cure for CD and treatment is aimed at suppressing inflammation and symptomatic relief of complications. Initially oral prednisone 40-60 mg/d, tapered over 2-4 months, then can have daily maintenance dose of 5-10mg/d. Sulfasalazine for mild to moderate CD 500 mg BID, increased to 3-4 g/d. Clinical improvement in 3-4 wks, and then tapered to 2-3 g/d for 3-6 months, this medication interferes with folid acid absorption and patient must take supplements. Metronidazole effective in tx perianal disease and in controlling crohns colitis, other ABT's such as Cipro, Ampicillin, and Tetracycline effective in controlling CD ileitis, and ileocolitis. Immunosuppressive meds when unresponsive to other treatments.	Pt educated on disease process, diet and lifestyle changes. Stress reduction, adequate rest to decrease bowel motility and promote healing. Low residue diet when obstructive sx present such as canned fruits, vegetables and white bread
Diverticulitis **	-Uncommon under 40yrs; risk rises after -Rare in pediatric; equal in men\women -More common in	-25% develop symptoms -LLQ abd pain, worsens after eating -Pain sometimes	-LLQ abd tenderness with possible Firm, fixed mass may be identified in area of diverticula	-Abd x-ray can reveal free air, ileus, obstruction -Barium studies show sinus tracts, fistulas,	-Asymptomatic cases managed with high fiber diet or fiber supplement with psyllium -Mild symptoms managed	-Increase fiber in diet to avoid constipation and straining -H2O intake of at

	developed countries -High in low fiber, high fat\red meat diets -Obesity, chronic constipation, h\o diverticulitis, & number of diverticula which occur in sigmoid colon.	relieved with BM or flatus -BM may alternate between diarrhea\ constipation -May present with bleeding w\o pain or discomfort -Fever, chills, tachy; LLQ with anorexia, N\V -Fistula may form causing dysuria, pneumaturia, fecaluria	-May have rebound tenderness with guarding\rigidity -Tender rectal exam; stool usually + for occult blood	obstruction -Colonoscopy to r\o Ca, but less sensitive than barium for diverticula -CT with contrast	outpatient with clear liquid diet and rest -Atb should not be routinely used but can be with diverticula abscess culture -Amoxicillin\clavulanate K (or) flagyl with bactrim -Symptoms usually subside quickly and diet can be advanced slowly -Pain managed with antispasmotics Ex; Levsin, Bentyl, BuSpar -Avoid morphine -NG for ileus or intractable N\V -Pt can be D\C'd from hosp once able to maintain adequate nutrition\ hydration if acute phase resolved -Colon resection may be necessary if no improvement or deterioration after 72hrs of treatment	least 8\8oz glasses to promote bowel regularity -Bulk-forming laxative may be needed Ex: psyllium, FiberCon, Metamucil
GERD **	-Can occur at any age -Risk increases with age, then decreases after 69yrs -Prevalence equal across gender, ethnic, cultural -Obesity, alcohol, caffeinated beverages, chocolate, fruit, decaf coffee, fatty foods, onions, peppermint\ spearmint, tomato products Anticholinergics, beta-	-Heartburn; mild to severe -Regurgitation, water brash, dysphagia, sour taste in AM, belching, coughing, odynophagia (painful swallow), hoarseness or wheezing at night -Substernal\ retrosternal pain -Worsens if reclined after eating, eating large meals, constrictive clothing	-H&P usually normal -May be + for occult blood in stool	-Usually Hx alone diagnoses -May manifest with atypical symptoms such as adult-onset asthma, chronic cough, chronic laryngitis, sore throat, noncardiac chest pain -If pt fails to respond to 4-8wks PPI, EGD is ordered -EGD warranted over empiric treatment when heartburn &	-8wk trial of PPI; weight loss, avoiding triggers -If unresponsive to once daily dosing; can increase to twice daily; if no relief EGD needed -PPI and H2-RA should not be taken together -Pt's on long term therapy should be re-eval'd q6mos	-Weight loss, med compliance and avoidance of triggers -Small frequent meals; main meal mid-day, avoid eating 4hrs before bed, avoid straining, sleep with HOB elevated, smoking cessation, stress mgmt

	adrenergics, CaChannel blockers, diazepam, Estrogen\ progesterone, Nicotine, Theophylline	-May present with dysphagia; dysphagia should only occur with first bite		dysphagia, bleeding, anemia, weight loss, or recurrent vomiting -EGD with Barrett's esophagus q3-5yrs		
Giardia	Can harbor in intestine, protozoan attaches to mucosa of small bowel. In US, risk in adults is oral-anal intercourse, children in daycare. In third world countries, risk of contamination through water sources.	Bloating, flatulence, nausea, watery diarrhea, weight loss, anorexia,	Malabsorption	Stool testing positive for trophozoites 50% of the time. Duodenal aspirate or small bowel biopsy	Quinacrine Hydrochloride (Atabrine) 100 mg TID after meals for 5-7 days or Metronidazole (Flagyl) 250 mg TID for 5-7 days	Teach good hand washing technique, sanitize surfaces, and avoid swimming in all types of water sources to avoid further contamination.
H. Pylori Infection	Risks: Increased age, living in crowded conditions, no clean water source (nonfiltered water), smoking	Ache or burning pain in abdomen. Abdominal pain that is worse when stomach is empty. Nausea/loss of appetite/unintentional weight loss. Frequent burping/bloating	Objective Findings RUQ/LUQ tenderness	-Fecal antigen assay -Urea breath Test -Biopsy with histological examination -Serological antibody	Standard triple drug therapy is clarithromycin and either amoxicillin or metronidazole with a PPI BID for 14 days. Amoxicillin preferred over metronidazole b/c there are some resistant strands of metronidazole.	-Complications (PUD) -Medication side effects
Irritable bowel syndrome **	Women more than men, rate 3:1; starts in late adolescence and early adulthood; rare in pts >50	-2 kinds of patients- those with abdominal pain and altered bowel habits, and those with painless diarrhea. -Left lower quadrant pain, sharp and burning with cramping or a diffuse, dull ache, precipitated by eating,	The physical exam tenderness in LLQ and over the umbilicus or epigastric area in those with small bowel involvement. Digital rectal exam may reveal tenderness and may exacerbate	CBC, ESR, CMP (electrolytes, serum amylase), urinalysis, stools for occult blood, ova and parasites, and cultures. Labs mostly normal and any diagnostic clue as to the cause is	Producing IBS include caffeine, legumes (and other fermentable carbohydrates), and artificial sweeteners. alleviate symptoms by eating a lower-fat diet that contains more protein. High fiber diet is good, introduced slowly to avoid	Recognize triggers and avoid them. Patients must understand that the goal of treatment is to improve their symptoms, not cure the disease, and that improvement in symptoms can be

		stress and relieved with a bm or flatus. -The pain does not interfere with sleeping, frequent complaints of abdominal distention, gas, and belching, urgency to defecate, passage of large volumes of mucus within the stool. -frequently associated with psych dg, which presents in the form of anxiety, depression, and somatoform disorders (marital discord, death, or abuse)	symptoms. -No weight loss or deterioration in health. -Key to diagnosis is the lack of fever, leukocytosis, or bloody stools. pg579 advanced assessment	helpful. If WBC found in the stool = infectious or inflammatory process and not IBS. Rule out food intolerance, lactase deficiency (hydrogen breath test or lactose tolerance test). IBS is often confused with lactose intolerance and can be evaluated by removing lactose from the diet for 2 weeks and monitoring the symptoms.	the sensation of bloating, 8 glasses of water per day, probiotic VSL#3 one packet bid, Antidiarrheal medications only temporary. -If diarrhea is severe, episodic use of loperamide (Imodium) 2 mg or diphenoxylate (Lomotil) 2.5–5.0 mg every 6 hours can be used as needed. -Constipation- lactulose or magnesium hydroxide. -Postprandial pain- dicyclomine 10 to 20 mg 3-4x a day by mouth or hyoscyamine 0.125 to 0.75 mg twice a day. Anticholinergics avoid in glaucoma and bph. Tricyclic antidepressants and ssri in some pt	a time-consuming process. Dietary education- fiber intake increase
Peptic ulcer disease ** (includes gastric ulcers and duodenal ulcers)	3 major causes: (1) Infection w/ H.Pylori, (2) chronic ingestion of ASA and other NSAIDs, (3) acid hypersecretion such as in Zollinger-Ellison syndrome. Genetics, blood type, personality type, and cigarette smoking may also play a role in the development of PUD. Pts w/ COPD, cirrhosis, renal failure, and renal transplant have higher incidence.	Hallmark: c/o burning or gnawing (hunger) sensation or pain (dyspepsia) in epigastrium, often relieved by food or antacids. Pts describe pain episodic pattern of c/o in which the pain tends to cluster and last for minutes, w/ episodes separated by periods of no sx. Almost half w/ NSAID-induced ulcers are asymptomatic. Nocturnal pain: in 2/3 of pts w/ duodenal	Pts w/ duodenal ulcers often demonstrate epigastric tenderness 2.5cm to right of midline, but this may also be present in cholecystitis, pancreatitis, non-ulcer dyspepsia, and other GI disorders. Reports of melena or coffee-ground-like emesis usually indicate bleeding ulcer, and perforated ulcer may present w/ abdominal rigidity.	Routine lab tests: normal unless significant bleeding or vomiting. Pt actively bleeding à CBC w/ diff. to eval HGB levels is paramount. Most pts w/ upper GI bleeding should have restrictive strategy, defined as transfusing when HGB levels fall below 7 g/dL. Diagnostic standard à upper GI endoscopy. Serology test or direct bacteriological analysis via an esophagogastroduode	Aim to relieve pain, heal ulcer, & prevent complication and recurrences. -PPIs: drugs of choice & includes omeprazole, raveprazole, lansoprazole, esomeprazole, dexlansoprazole, pantoprazole. PPIs heal duodenal ulcers in 4 wks therapy and gastric ulcers after 8 wks. -H2-R eceptor Antagonists: Used for mild symptoms with no complication or serious	Smoking cessation; avoid foods that precipitate dyspepsia. MUST follow treatment regimen. Educate about side effects such as change in stool color to black with bismuth preparations. If sucralfate with antacid, PPI, H2RA being taken, stress that sucralfate cannot be taken with other meds or

		ulcers and 1/3 of those w/ gastric ulcers. -Nausea & anorexia sometimes occur in pts w/ gastric ulcers. Vomiting and weight loss indicate more serious complications like gastric malignancy or pyloric obstruction. Pts w/ duodenal ulcers may report a reduction in pain after eating; pts w/ gastric ulcers tend to experience more intense pain after eating.		noscopy (EGD) Bx à to check for H. Pylori. EGD is ordered for pts who have failed the standard triple-drug therapy for H. Pylori. A serological antibody (enzyme-linked immunosorbent assay) test can be used detect infection w/ H. Pylori, doesn't distinguish between active or past (treated) infection and is expensive. Urease is plentiful in pts w/ H.Pylori infection. Breath tests for H. Pylori are based on the production of ammonia from the metabolism of urea by urease à indicate active infection and are noninvasive way of dx H. Pylori. In pts w/ increase in gastric acid secretion is suspected, a fasting serum gastrin level should be drawn. Levels higher than 200 pg/mL should be confirmed on repeat testing and followed by basal and peak acid-output measurements. Zollinger-Ellison syndrome should be suspected in pts	disease; treatment for 2 wks. If symptoms persist past 2 weeks, EGD considered. If used for peptic ulcer tx, standard therapy is daily x 6 wks or half the dose bid x 8 weeks (cimetidine, ranitidine, nizatidine, famotidine) -Other agents: antacids were mainstay of ulcer treatment. Do not use antacids with calcium in PUD because calcium causes rebound acid secretion. Sucralfate 1g QID heals duodenal ulcers, bismuth (also has antimicrobial action against H. Pylori), misoprostol (Cytotec) used for prophylactic measure to prevent gastric ulcer formation in pts who use NSAIDs. Triple therapy for H. Pylori is a combination of 2 antibiotics (clarithromycin and either amoxicillin or metronidazole) w/ a PPI BID x 14 days. Amoxicillin preferred over metronidazole due to resistant h. pylori strains. Bismuth subsalicylate & 2 antibiotics is also effective but dosing is QID.	with digoxin, ciprofloxacin, phenytoin due to it binding with these meds.

				whose fasting serum gastrin level is > 600 pg/mL and who have a basal acid output > 15 mmol/hr.		
Pancreatitis **ACUTE\ CHRONIC**	**ACUTE**: About 80% of hospital admissions are a result of biliary tract disease (passing gallstones) or alcoholism. --Risk: Infection (mumps), Hyperlipidemia, Metabolic disorders (hyperparathyroidism, hypercalcemia), Drugs (furosemide, valproic acid, sulfonamides, thiazides), Endoscopic retrograde cholangiopancreatography (ERCP), Abn pancreatic duct (stricture, carcinoma, pancreas divisum), Abn Common bile duct and ampullary region, Surgery of stomach and biliary tract, vascular disease (artherosclerosis, severe hypotension), trauma. **CHRONIC:** *Slow progressive process* *--Risk: alcoholism, diets high in protein combined with high/low fat can further predispose to pancreatic injury from*	**ACUTE:** Pain that is intense, abrupt onset deep epigastric pain that last for hours to days. Radiates straight through the back. Pain is often refractory to narcotics. Aggravated by vigorous activity (coughing) and lying supine. Alleviated when seated and leaning forward. Intractable nausea/vomiting. Depending on severity may present with seating, weakness and anxiety. May report ingestion of alcohol or big meal before onset of symptoms. **CHRONIC:** Patient presents with intractable abdominal pain, weight loss, diarrhea but can be mild (dyspepsia, nausea, vomiting). Abdominal pain normally epigastric/LUQ that may radiate to back or left lumbar region that is described as dull and constant. Pain is aggravated by food or	**ACUTE:** Severe abdominal tenderness over epigastric area accompanied by guarding. Abdominal distension presents in about 20% of patients. Bowel sounds hypoactive or absent if paralytic ileus present. Tachycardia (100-140 b/min) with rapid, shallow respirations. Increased blood pressure due to pain. Temp initially normal but increases to 100.4-102.2. **CHRONIC:** Mild to Moderate epigastric tenderness without rebound tenderness or guarding.	**ACUTE:** Abdominal Pain Elevated Serum Amylase/Lipase that return to normal after 3-7 days WBC between 12-20, 000 CT of abdomen: provides fast and accurate for definitive diagnosis **CHRONIC:** CT and /or US of the abdomen to show abnormal size or consistency of pancreas. Evaluation of pancreatic function: Bentiromide Test--collections of normal volume and low in bicarbonate suggest chronic pancreatitis.	**ACUTE:** Management is aimed at limiting severity of pancreatic inflammation, preventing further complications and managing symptoms. Mild symptoms can resolve on its own and managed outpatient conservatively. Fasting is necessary until symptoms have subsided. Maintain fluid status with parenteral fluids Pain medication other than opiates (to prevent pressure within sphincter of Oddi). Introduction of clear fluids implemented once pain free, amylase/lipase levels returned to normal, bowel sounds have returned, Low fat diet as patient tolerates. **CHRONIC:** Aimed at preventing further pancreatic damage, managing pain and supplementing exocrine and endocrine function. Sustaining from alcohol use. Relief of pain by pancreatic enzymes in some patients and others may need narcotic pain management. Operative	ACUTE: Informed the cause of pancreatitis Reduction of dietary intake of fat Abstain from alcohol abuse Drug induced--avoid causing agent Hyperlipidemia--diet instruction and information on avoidance of factors such as alcohol, estrogens. **CHRONIC:** Patho of disease and long-term outlook Decrease in frequency in attacks after 5-10 years Medication regimen/Rational for medications (control diarrhea and gain body weight) Pain management if long term narcotic is needed.

	alcohol, *autoimmune disease*, *genetic mutations*, *hereditary predisposition*, *high triglycerides*, *severe malnutrition*, *tropical pancreatitis*, *obstruction caused by stenosis*, *stones*, *tumor*, *cystic fibrosis*.	alcohol.			treatment considered in patients that fail pain management with pancreatic enzymes or analgesics. Malabsorption managed with low-fat diet and oral pancreatic enzymes (Viokase/Cotazym/Pancrease/Creon/Donnazyme).	
Salmonella **	One of the major causes of diarrhea worldwide. Three species: S. typhi, S. choleraesuis, and S. enteritidis. Found primarily in chicken, eggs, and livestock, causing 85% of community-acquired Sal monella outbreaks. Individuals must ingest 10,000–1 million organisms to become infected. Duration is 2–5 days; onset is 8–48 hours after ingestion. Patients may become "chronic carriers," defined as individuals with positive stool cultures 1 year after initial disease. Peak incidence is in summer and fall. Symptoms begin with nausea and vomiting, followed by colicky abdominal pain and bloody or mucoid diarrhea. Enteric fever results from organisms entering the bloodstream via the	Present with varying degrees of nausea, vomiting, diarrhea, fever, and abdominal pain and cramping. Symptoms depend on the underlying cause but can also include fatigue, malaise, anorexia, tenesmus, and borborygmus. Individuals with profuse diarrhea may complain of rectal burning and hematochezia from rectal abrasion and bleeding. Patients may complain of symptoms that suggest dysentery, including passage of numerous small-volume stools containing blood and mucus. Reports of voluminous stools are suggestive of a source in the small bowel or proximal colon; small stools accompanied by a sense of urgency suggest a source in the	Present with varying degrees of nausea, vomiting, diarrhea, fever, and abdominal pain and cramping. Symptoms depend on the underlying cause but can also include fatigue, malaise, anorexia, tenesmus, and borborygmus. Individuals with profuse diarrhea may complain of rectal burning and hematochezia from rectal abrasion and bleeding. Patients may complain of symptoms that suggest dysentery, including passage of numerous small-volume stools containing blood and mucus. Reports of voluminous stools are suggestive of a source in the small bowel or proximal colon; small stools accompanied by a sense of urgency	The physical exam is usually normal except for the aforementioned GI problems. Depending on the degree of dehydration, the skin turgor may be poor, and mucous membranes may be dry. -Vital signs may reflect dehydration, such as a fever with an increased heart rate. Older and very young patients with gastroenteritis m ay show signs of severe dehydration such as orthostatic hypotension and dizziness. Patients who have had prolonged illness and are malnourished may present with edema resulting from hypoalbuminemia. Diagnosis is made by isolation of organism in stool. No treatment	Treatment includes trimethoprim-sulfamethoxazole (Bactrim DS) or a quinoline, norfloxacin 400 mg or ofloxacin 400 mg PO twice daily for 7–10 days.	Stress proper handling of food, thorough cooking, and good hand washing.

	bowel lymphatics, causing bacteremia, headache, and myalgias. Tissue abscesses may develop. Stools may be foul smelling.	left colon or rectum. Bloody stools suggest mucosal damage and an inflammatory process secondary to invasive pathogens. Frothy stools and flatus suggest a malabsorption problem.	suggest a source in the left colon or rectum. Bloody stools suggest mucosal damage and an inflammatory process secondary to invasive pathogens. Frothy stools and flatus suggest a malabsorption problem.	is necessary unless associated with fever and systemic disease.		
Shigella	One of the most common causes of bacillary dysentery. Several species: S. sonnei is isolated in 75% of cases in the United States. Because of poor hygiene and overcrowding, it is spread via the fecal–oral route and requires only a small number of organisms to produce disease. Organism causes epithelial invasion of intestinal mucosa. Duration usually 4–7 days and is self-limiting. Incubation period of 1–2 days after exposure or ingestion of pathogen.	See Salmonella	See Salmonella	Diagnosis is made by isolation of organism in stool or rectal swab. In severe cases sigmoidoscopy shows mucosal hyperemia, friability, and ulceration. Initially patients present with watery diarrhea and high fever. Later colitis-type symptoms develop: Abdominal cramps, tenesmus, urgency, frequent small stools with blood and mucus. Low-grade fever may persist for 2–20 days. Complications can include hemolytic-uremic syndrome and colitis.	Treat with Bactrim DS twice daily for 3 days if infection was acquired in the United States.	Stress proper handling of food, thorough cooking, and good hand washing.
Ulcerative Colitis **	Peak age of onset: 15 to 30 y/o, but may occur at any age. More common	Mild: 4 or fewer loose BMs per day associated w/	Tenderness in LLQ or across the entire abdomen, often	Digital Rectal Exam: to assess for anal and perianal	Initial: nutrition counseling. Parenteral nutrition may be	Colonoscopy should be avoided w/

	in males. Familial tendency.	abdominal cramps relieved w/ defecation, small amounts of blood and mucus in the stool, and sometimes tenesmus Moderate: 4-6 loose BMs per day w/ more blood and mucus. Systemic Sx: tachycardia, mild fever, weight loss and mild edema depending on serum albumin levels Severe: more frequent blood BMs (6-10 per day, abdominal pain and tenderness, Sx of anemia, hypovolemia, and impaired nutrition If Ulcerative Colitis (UC) confined to rectal or sigmoid area, stools can be normal or hard and dry; however, the rectum will continue to dispel mucus containing both RBCs and WBCs. As disease process moves proximally, the stools become looser. Pts may report eating less to decrease BM frequency, which leads to further nutritional deficiencies.	accompanied by guarding and abdominal distention. Depending on severity: S/S of ileus and peritonitis may be found. Serological: + for antineutrophil cytoplasmic antibodies (pANCA). Fever & malaise w/ severe disease. Early disease: mucous membrane is granular, friable, and edematous w/ loss of normal vascular pattern. May be scattered areas of hemorrhage that bleed w/ minor trauma. Resulting ulcerations develop after mucosa breaks down, leaving the mucous membranes dotted w/ numerous bleeding and pus-oozing ulcers. Severe disease: Copious amounts of purulent exudate. Periods of remission, sigmoidoscopy always shows some friability and granulation present	inflammation, rectal tenderness, and blood in the stool. Dx made by correlating sx w/ hx and physical exam. Stool analysis and Cx are obtained to r/o bacterial, fungal, or parasitic infection (ova & parasites) as cause for diarrhea. Stool is examined for mucus and blood. Contrast radiography and endoscopy primary diagnositic tool to confirm IBD (Irritable Bowel Disease). Sigmoidoscopy, defines the actual extent of the mucosal inflammation. Bx results à chronic inflammation. Colonoscopy to determine the extent of the disease, to avoid perforation, usually reserved for pts who have started tx.	necessary w/ severe anorexia or uncontrollable diarrhea. Pts w/ mild-mod diarrhea may benefit from diphenoxylate w/ atropine (Lomotil) 2.5 to 5.0 mg PO BID up to 4x daily, loperamide (Imodium) 2 mg after each BM, or codeine 15 to 30 mg PO Q4-6H. Disease limited to rectosigmoid area: topical steroids or mesalamine. Steroid enemas and foams (hydrocortisone [Cortifoam] 100 mg) nightly x 2 wks. PO formulation of Asacol (5-ASA) med help maintain remission after enemas have been d/c'd More advanced disease: Systemic glucocorticoid in combo w/ sulfasalazine or 5-ASA therapy. Glucocorticoids esp. helpful in controlling extracolonic manifestations à peripheral arthritis, ankylosing spondylitis, erythema nodosum, anterior uveitis, and pyoderma gangrenosum: Oral prednisone (Prelone), up to 40 to 60 mg in single or divided doses, tapered and not d/c'd abruptly. Severe or fulminant: (10 or > bloody stools per day):	severe colitis or deep ulcerations because of risk of perforation or development of toxic megacolon. Pts should avoid caffeine, raw fruits, vegetables, and other foods high in fiber à can cause trauma to the already inflamed mucosal surface. Some pts may benefit from lactose-free diet, but not recommended unless a trial produces symptomatic relief. Bland diet high in calories and protein yet low in fat can help to control diarrhea and flatulence and maintain nutrition and weight. Antidiarrheal meds should be avoided in acute phase but can be helpful for pts w/ mild sx. All pts should be informed of disease process, tx options, and expected outcomes. Education about diet and lifestyle

					abdominal tenderness, fever, colon dilation and tachycardia à require hospitalization, monitor closely for development of toxic megacolon and colonic perforation. If no improvement after 7-10 days; consider surgical intervention. Surgery: Subtotal or total colectomy à prevent perforation of bowel and its complications. Some pts may need fluid/electrolyte management and/or blood transfusions. Most common procedure protocolectomy: Brooke ileostomy, curative and functional procedure. Immunosuppressive agents: azathioprine (Imuran), cyclosporine, and metabolit 6-mercaptopurine (6MP) à used in cases unresponsive to other medical management and in pts who are not surgical candidates. For disease unresponsive to other therapies: anti-tumor necrosis factor (anti-TNF) agents can be used à infliximab (Remicade) 5 mg/kg and adalimumab (Humira) administered SubQ 160 mg @ wk 1, 80 mg @ wk 2, then maintenance of 40	changes. Importance of adequate rest and stress reduction to decrease bowel motility and promote healing. Stress management techniques: guided imagery, referred for counseling if necessary. Provided information and addresses for national organizations à Crohn's and Colitis Foundation of America: up-to-date info and local support groups. If no S/S of acute attack, they can eat whatever they want or can tolerate. About possibility of parenteral nutrition or oral supplementation during acute attacks. Foods that can cause diarrhea and gas-producing foods should be avoided during acute attacks. Female pts require special guidance and counseling before attempting pregnancy. If pregnancy occurs,

					mg Q other Wk beginning @ wk 4. Pts w/ toxic megacolon: NG tube placement for intermittent suction, NPO, antidiarrheal meds should be d/c'd. F/E imbalances need corrected: hypokalemia. Total parenteral nutrition may be necessary short term. Daily abdominal x-rays.	pt must be followed closely by gastroenterologist
Viral gastroenteritis **	Causes of gastroenteritis are numerous; however, bacterial, viral, and parasitic infections are among the most common. Almost all forms of enteric infection manifest with diarrhea. Several different viruses including rotavirus, norovirus, adenovirus, and astroviruses account for most cases of acute viral gastroenteritis. Most are transmitted via the fecal-oral route, including contaminated food and water. Transmission has also been shown to occur via fomites, vomitus, and possibly airborne methods. Peak viral load within the stool is anywhere between 24 to 48 hours after symptomatology.	See Salmonella	See Salmonella	Viral gastroenteritis is a known cause of nausea, vomiting, diarrhea, anorexia, weight loss, and dehydration. Clinical manifestations for viral gastroenteritis are due to the effects that the viruses, along with specific cytotoxins, have on the enterocytes of the intestine. The virus uses the enterocyte to replicate, leading to interference with brush border enzyme production, which in turn leads to malabsorption and osmotic diarrhea [8]. Additionally, viral toxins lead to direct damage and cell lysis of enterocytes and intestinal villa, causing a transudative loss of fluid into the intestine [15]. The	Most important goal of treatment is to maintain hydration status and effectively counter fluid and electrolyte losses. Antimotility drugs are the most frequently prescribed and most effective drugs for the treatment of symptomatic gastroenteritis. These agents work by slowing intraluminal peristalsis, thereby slowing the passage of fluids through the lumen, which facilitates absorption. Patients with febrile dysentery should not receive antimotility medications because slowing the intraluminal time may prolong the duration of the disease	Prevention of the spread of disease from patients with infectious diarrhea to other individuals. Teaching includes good hand washing and safe disposal of waste products. Any infant or child with infectious diarrhea should not attend day care until the diarrhea has stopped or the child has completed the prescribed course of antibiotics. Good hand washing technique is imperative to prevent household outbreaks of the disease.Patients traveling in high-risk areas should be instructed to consume only safe foods and beverages there and

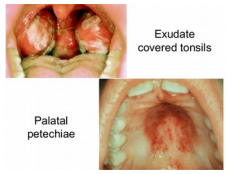
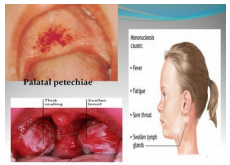
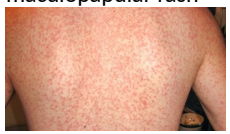
	Some studies show viral shedding lasting for several weeks past symptomatology			loss of cell function can lead to electrolyte abnormalities which are caused by the loss of transporter functionality. That can lead to acid-base disturbances as well. The virus is then shed through feces, and occasionally in the vomitus. Complete blood counts may reveal a mild leukocytosis in a patient with viral gastroenteritis. Other serum inflammatory markers may also show mild elevation. Patients who are suffering from significant dehydration may demonstrate hemoconcentration on complete blood count testing as well as electrolyte disturbances on chemistry panels. Dehydration may also present as acute kidney injury, evidenced by changes in the BUN and creatinine on chemistry panel.		on the airplane leaving the area.
HEENT DISORDERS						
Bacterial conjunctivitis (viral is most contagious)	Occurs in fall and winter. More common in children than adults.	Discharge is purulent, thick with crusted eyelids shut in the	Normal visual acuity. No pupillary abnormalities. No	Usually none. Consider culture of exudates for	Consider fluorescein staining is corneal abrasion suspected.	-Good hand hygiene and eye hygiene. -Use clean

	Direct contact with secretions or with contaminated objects and surface.	morning. Sandy, gritty feeling in eye. Unilateral but usually becomes bilateral due to contamination.	photophobia. Lymph nodes NOT palpable. Reddened conjunctiva (both over the eyeball and inside lid) and eyelid swelling. Hallmark symptom of bacterial conjunctivitis is purulent discharge.	recurrent conjunctivitis but rarely indicated.	Bacterial form is also self-limited. Treatment shortness course if initiated early. Self-limiting in 5-7 days; can delay treatment until third day -Eyedrops or ointment: trimethoprim/polymyxin B (Polytrim), erythromycin, tobramycin, gentamicin, sodium sulfacetamide, or ciprofloxacin, levofloxacin -Contact lens wearers: fluoroquinolones are first line Tobramycin (These medicines include ciprofloxacin (Cipro), gemifloxacin (Factive), levofloxacin (Levaquin), moxifloxacin (Avelox), norfloxacin (Noroxin), and ofloxacin (Floxin) Children: ointment preferred over drops One exception to the rule in regards to the effectiveness of antibiotic drops for all bacterial conjunctivitis cases is gonococcal infections. **Gonococcal conjunctivitis is sight threatening because it can affect the cornea**, so patients should be sent to the ER immediately. Gonococcal conjunctivitis is associated	washcloth each time face is washed. -Change pillowcases daily. -Warm compresses for infectious origin. -do not wear contact lenses until inflammation resolved (1 week); discard current contact lenses. -Discard makeup used. -Symptoms should improve in 2-4 days -Instruct patients to treat the eye that is affected but to start treatment in the other eye if symptoms develop -Bacterial conjunctivitis very contagious; stay home from work or school until 24 hours of antibiotic treatment or as soon as clinical improvement (decreased redness and discharge)

					with hyper-purulent discharge	
Corneal abrasion	Mechanical or chemical means; Trauma induced by contact lenses, damaged contact lenses, or foreign body. Spontaneous induced and often known as recurrent erosions that stems from a previous injury. More common in young, active patient. Uncommon in older adults.	**Excessive tearing**, severe eye pain and inability to open eye due to foreign body sensation, photophobia, conjunctival hyperemia. Hx of scratching the eye, contact lens irritation, or actual trauma. Patients with recurrent corneal erosion syndrome experience searing pain in the middle of the night. It awakens them, or they feel pain on awakening	-Constricted pupil, foreign body, lacrimation. Profuse tearing. -Invert eyelid to r/o foreign body underneath.	Stain the eye with fluorescein and use a cobalt blue filter light or slit lamp to inspect the eye for foreign objects or scratches. Areas of epithelial disruption fluoresce green when exposed to a Wood's lamp. Access visual acuity: should be normal unless abrasion is large.	Treatment includes antibiotic eye drops or **ointment** for 5 – 7 days to prevent bacterial infection. **Traumatic/foreign body/recurrent abrasions**: Erythromycin ointment OR sulfacetamide **Contact lens abrasion:** ofloxacin, ciprofloxacin OR tobramycin drops/ointment Oral analgesics for pain Only ophthalmologists should prescribe topical anesthetics due to delayed wound healing and risk of ulceration, scarring, perforation and blindness Tetanus prophylaxis Normal saline to irrigate eye.	Patching is not usually necessary. The patient should avoid wearing contact lenses until the abrasion heals. f/u in 24-48 hours if no improvement f/u in 24 hours to assess healing f/u by eye doctor
Epiglotittis	Common in young children 2-4 years; most common >7 years; may occur in older children and adults. Men > women. Infection with Haemophilus influenzae B (Hib) (most common); streptococci now major pathogen of cause.	Odynophagia (pain on swallowing), dyspnea, drooling, stridor.	Never use tongue blade or light due to laryngospasm and airway obstruction may occur.	Transport to OR for fiberoptic laryngoscope visualization showing that epiglottis is swollen and erythematous (cherry red). Endotracheal tube should be inserted.	ER care for adequate airway control. Needs hospitalization for IV antibiotics such cefuroxime (Ceftin), ceftriaxone (Rocephin), or ampicillin/sulbactam (Unasyn). Dexamethasone (Decadron) should also be administered IV and tapered as signs and symptoms resolve.	

					Continuous pulse oximetry and careful monitoring of the patient's airway are critical. Patients who develop hypoxemia and respiratory distress will require intubation.	
Eustachian tube disorder	Some of the most common causes include conditions causing nasal congestion as is seen with allergic rhinitis, sinusitis, URIs, enlarged adenoids, and pregnancy. Additionally, those who have recently traveled in an airplane or who have been scuba-diving are at risk for ETD.	Often people complain of decreased hearing or a fullness in the ears. Hearing may be muffled or diminished. May report an inability to "pop" or "clear" their ears, which normally occurs with changes of barometric pressure. They may have accompanying tinnitus or disequilibrium. Patients may come to you thinking that they have an ear infection due to pain or pressure. They may also be concerned of cerumen impaction if they are experiencing hearing loss.	Physical exam findings with ETD depend on precipitating event. Nasopharyngeal examination may reveal findings consistent with allergic rhinitis, sinusitis, or URI. On the affected side, typically you will see a TM that appears retracted or "sucked back."	Diagnosis of ETD is based on the history and physical exam. If pneumatic otoscopy is performed, the affected TM will be immobile. A Weber and Rinne hearing test will reveal conductive hearing loss on the affected side.	Key it to treat underlying problem. -If a cold, then nasal saline drops or a neti pot may help. -AOM and sinus infections are treated with antibiotics. -Allergic rhinitis should be treated with nasal steroids and decongestants; however, decongestants are contraindicated in children under 6 years of age. Comfort measures can include acetaminophen or ibuprofen. Patients can be instructed to attempt to relieve pressure by yawning, chewing, or sucking. Holding the nose and blowing out is not recommended due to risk of TM performation. For chronic ETD unresponsive to tx, refer to ENT. Tympanostomy tubes may be placed to equalize pressure.	
Hyphema (a layer of RBCs - hemorrhage)	Usually a result of blunt or penetrating trauma.	Vision loss and eye pain; may be	Conjunctival injection noted; blood in	Based on physical findings but may	Possible evacuation of blood by ophthalmologist.	Immediate referral to ophthalmologist.

	May be spontaneous. Spontaneous hyphema is a result of DM, iris melanoma, retinoblastoma, eye tumors, juvenile xanthogranuloma, clotting disorders, sickle cell disease or trait; anticoagulant medications. 70% of cases occur in children; peak between ages 10 and 20. Boys > girls	accompanied by nausea/vomiting	anterior chamber of eye, visible fluid line in pupil, photophobia, decreased visual acuity	include CT, orbital ultrasonography, or US biomicroscopy. Consider hematology studies (like clotting factors) based on history and exam	Eye shield; head elevated 30-40 degrees; complete bedrest and dim lighting Pain management, treat n/v; correct coagulopathy; avoid aspirin products, miotics, mydriatics in acute setting	Protective eye devices. Control of diabetes and hemophilia. Med education on what to avoid at home.
Meniere's disease	Age of onset 30-60; most cases develop in 50's. Rare in young children and adults >70 years. White Americans of European descent at increased risk. Equally affects men/women. Stress, allergies; high sodium, caffeine, alcohol intake; hormonal changes; changes in barometric pressure; exposure to high noise levels for many years. Inflammatory response of inner ear from insults (blunt trauma, viral infections, allergies, reduced or negative middle ear pressure).	Recurrent tinnitus, vertigo, and progressive low-frequency hearing loss or complete hearing loss in severe cases. Acute episode last anywhere from 20min-3hrs. Attacks rarely last > 4 hrs. Characterized as sudden attacks of nausea, emesis, pallor, diaphoresis, dizziness (spatial disorientation), vertigo, roaring tinnitus, increased pressure, fullness, and hearing loss in affected ear. Rapid movement aggravates symptoms, and possible report of falls or accidents	No apparent abnormalities on otoscopic exam unless otitis media present. Dilation of inner ear endolymphatic system present on autopsy. Spontaneous nystagmus is observed after preventing eye fixation by having pt wear 40 diopter glasses (Frenzel Lenses). Is a diagnosis of exclusion; numerous disorders mimic this disease.	Careful history, neurologic assessment, and response to empiric therapy. Weber and RInne show sensorineural hearing loss; diagnostic criteria involves 2 episodes of vertigo lasting 20 minutes along with sensorineural hearing loss and either tinnitus or a perception of aural fullness. Audiometry shows low-frequency hearing loss and impaired speech discrimintation.	1st, r/o other cause of symptoms. No proven cure; palliative tx given for reducing symptoms. Acute attacks treated with rest by closing eyes and protection from falling. Vestibular rehab reduces symptoms of unilateral peripheral vestibular dysfunction. Meds: Vestibular sedatives like prochlorperazine for severe n/v; a antihistamine like betahistine to reduce frequency and severity of vertigo attacks. Intratympanic	Education on reducing sodium 1g/day, caffeine, and alcohol intake; stop smoking. Manage stress levels. Avoid all ototoxic drugs and polypharmacy. Return if further symptoms worsen or acute episodes increase in frequency. Acute attacks best managed by quiet bedrest and prevention of falls -

	Familial history. History of migraines, autoimmune conditions (systemic lupus erythematosus), RA, certain thyroid disorders.	during episodes. Frequency and severity may decrease over time with hearing improvement post attack, but some episodes may last 24 hours.		Cold and warm caloric responses are typically reduced in the affected ear, as demonstrated by electronystagmography or direct patient observation (while wearing 40-diopter Frenzel lenses); the direction of the fast phase of nystagmus is variable. These findings are not diagnostic for Ménière's disease.	dexamethasone used in pts refractory to lifestyle changes. Last resort: Aminoglycosides like streptomycin or gentamicin ablation therapy to reduce unbearable vestibular symptoms.	not medicine. Reduce food intake during episodes to avoid n/v.
Mononucleosis: primarily caused by EBV Angie	**RISK** -Common: Adolescents, young adults (especially college students) -Rarely: Elderly -Immunocompromised persons.	**SUBJECTIVE** -Gradual onset -Mild to Severe throat pain, but mono tends to be the most painful. -low grade fever -Marked fatigue -Tickle sensation -Head and body aches -swollen lymph nodes in the neck and armpits -swollen liver/spleen or both (less common). -rash -spread through saliva (kissing, sharing drinks/cups.	**OBJECTIVE** -Exudative tonsillitis (50% of cases). -Palatal petechiae   -exanthem (rash) maculopapular rash 	**DIAGNOSTIC** -VCA (viral capsid antigen Anti-VCA IgM appears early in EBV) -VCA Anti-VCA IgG appears in the acute phase of EBV -Monospot test positive by wk 2-3 (nonspecific heterophile antibody test) and decreases in sensitivity when used at the extremes of age (not recommended for general use). -CBC if viral will show 60% lymphocytes, of which at least 10% show atypical morphology. -fewer normal neutrophils or platelets	**TREATMENT** -No specific treatment for EBV -Relief of symptoms -drink fluids to stay hydrated -get plenty of rest -take OTC medication for pain/fever relief. -No vigorous exercise, sport or heavy lifting -warm salt-water gargle -avoid stress -eat a balanced diet **PHARMACOLOGIC:** -Acetaminophen for fever, aches, pain--Avoid if elevated liver function. -Ibuprofen: **AVOID:** -ampicillin or amoxicillin due to increased susceptibility to reaction	**EDUCATION** Instruct patient: -drink plenty of fluids and get plenty of rest. -No vigorous exercise, sports or heavy lifting for 6 wks to avoid rupture spleen. -symptoms may subside in 1-3 wks, but fatigue may last for several more weeks. -perform good hand washing -avoid stress -eat a balanced diet -Contagious for 3 months after symptoms subside and could last as long as 18 mo. -Can be spread

			-Posterior cervical lymphadenopathy (90%) *OCC. OCCURS -significant tender lymphadenopathy of the draining anterior cervical lymph nodes.	-Liver enzymes: abnormal liver function -US-dx splenomegaly -consider rapid strep/throat culture: pharyngitis is similar to presentation of strep.	(rash). -Steroids unless severe pharyngeal erythema or tonsillar hypertrophy develops, resulting in obstruction (may prolong illness) -Aspirin (risk of Reye's syndrome)	through saliva, bodily fluids such as blood, sexual contact, organ transplant, cough, sneeze, kissing, sharing food/drinks.
Nasal polyps	Caused by poorly controlled rhinitis. increases with age, female>male. Associated with cystic fibrosis, Asthma, bronchiectasis, ASA hypersensitivity, chronic sinusitis, primary ciliary dyskinesia (Kartagener syndrome), and laryngopharyngeal reflux.	Rhinorrhea, nasal congestion, postnasal drainage, hyposmia (inability to smell), inability to breath through nose, dull headache, facial pain/pressure over middle 3rd of face or No symptoms in some cases. Usually bilateral; if unilateral is reported; check for malignancy	Gray-blue to yellow-tan nasal polyps may present with chronic perennial rhinitis If large posterior nasal polyps, examine tympanic membrane for ETD. If unilateral, check for malignancy.	**Flexible/rigid endoscopy (gold standard of diagnosis)** Pale-translucent mass on anterior rhinoscopy. CT scan may help reveal extent of disease and differentiate a polyp from another mass. MRI if neoplasia, mycetoma, or encephalocele suspected.	Goal – reduce size or eliminate polyp. Daily intranasal corticosteroid use with saline irrigation 1st-line therapy. Treat for minimal of 12 weeks. Use budesonide, beclomethasone dipropionate, fluticasone, mometasone furoate. Mometasone furoate preferred for children. Short course of oral corticosteroids (14-21 days) and/or doxycycline (21 days) in symptomatic patients despite initial tx. (prednisone, prednisolone, doxycycline)	
Otitis Externa (AKA swimmer's ear) **	Common in warmer months. No ethnic predisposition. Men/women equally affected. Those at risk: Immunocompromised pts on corticosteroid therapy or with chronic conditions such as DM. Pseudomonas infection common from excess	Acute, often severe otalgia of sudden or gradual onset; may be bilaterally. Pain may be worse at night, more severe when pulling on pinna or earlobes or applying pressure to tragus.	Ear canal may be erythematous and edematous; absence or presence of cerumen or accumulation of purulent drainage. Tenderness on traction of pinna and/or pain with	Rarely needed if symptoms fits classic pic or otitis externa. Fluid from ear may be cultured and antibiotic sensitivity tested if organisms found. Done for those who	Treat pain: local application of heat or ice-pack to outer ear. Nonprescription pain relievers: aspirin or acetaminophen or NSAIDS - 1st line agents. Extreme pain: Acetaminophen/codeine 325mg/5mg 1-2 tabs po	Keep ear dry, avoid swimming or submersion of ear during and after acute episodes for 4-6 wks. Use shower caps and ear plugs to shower. Those susceptible to repeated infections, a 2% acetic acid

	swimming in hot, humid weather, especially in polluted water. Highly chlorinated pool water leads to drying out of ear canal creating potential entry of bacteria and fungi. Inadequate cerumen (a protective barrier). Patients with seborrhea due to excess sebum production. Manual ear picking; forging bodies in auditory canal(like leaving cotton in ear); long use of ear plugs, hearing aids, cotton swabs may lead to local irritation and predispose to infection. Previous ear infections and hx of skin allergies.	Chewing may elicit pain. Initially, ear may feel full or obstructed with temporary conductive hearing loss if edema present. May be pruritic. Purulent drainage. Fever/chills. chronic otitis externa may have dryness and pruritus of ear canal.	pressure over tragus. May be diffused with complete involvement of auditory canal or localized with focal lesions (pustules or furuncles) along auditory or external ear structures. Sebaceous secretions in those with seborrhea. Fluid may be apparent: Pseudomonas -copious green exudate Staphylococcus infection - yellow crusting with purulent exudate. Fungal infections - fluffy white or black malodorous carpet of growth. Allergic reactions - seen as scaly, cracked, and/or weepy tissue. Frank invasive disease - granulation tissue spreading out from primary site of infection and eroding into temporal bone,	do not respond to treatment or those with chronic otitis externa, especially those with purulent exudates indicating bacterial infection. Culture also done for immunocompromised pts. Rule our fungi and mycobacteria in these pts. ESR level may be elevated. CT and MRI used to determine soft tissue or bony involvement in malignant disease. Temporal bone 1st bone affected.	q6h OR Acetaminophen/hydrocodone (Vicodin) 325mg/5mg po q8h for 1st 24-48 hours (risk for abuse). To facilitate healing: Clean ear canal to remove Cerumen, exudate, debris with cotton pledget or gentle irrigation using warm water. **1st line agents:** Acetic acid/aluminum acetate, acetic acid/hydrocortisone, ciprofloxacin/hydrocortisone, **ciprofloxacin/dexamethasone**, **neomycin/polymyxin B/hydrocortisone**, and ofloxacin. Liquid ophthalmic preparations of gentamicin and tobramycin may be used otically to cover both P. aeruginosa and S. aureus. Bacterial otitis externa **Safe with perforated tympanic membrane (TM):** include ciprofoxacin 0.3% and dexamethasone 0.1% (Ciprodex otic); not for 6 months of age. Ofloxacin 0.3% (Floxin otic) 6 months-13 years 5 drops in affected ear daily for 7 days; adults 10 drops in the affected ear for 7 days **Not safe with perforated**	solution may be used prophylactically to acidify ear canal whenever ears get wet. Teach proper method to clean ears using soft cotton pledget NOT swabs, sticks, or agents. Excessive cleaning harmful; small earwax necessary to prevent infection. Cured 7-10 days of treatment. F/U 1 week for uncomplicated pts. If ear wick placed, F/U 2 days for removal and canal cleaning and symptoms should begin to subside in 48h - pt to call if unresolving. F/U daily in hospitalized patients immunocompromised on IV therapy. F/U closely in healthy pts with invasive disease. Gallium scans performed to evaluate efficacy

			outer auricle, or through perforated tympanic membrane. Neck lymphadenopathy not detected.		**TM:** Chloroxylenol 1mg+pramoxine HCL 10 mg + hydrosortisone 10mg/mL (cortone B Aqueous) Colistin 3 mg Neomycin 3.3 mg, hydrocortisone Acetate 10 mg Thonzonium bromide 0.5 mg (Cortisporin-TC Otic) **Refractory cases to initial therapy or involve auricular cellulitis required systemic ABX covering both Staphylococcus and Pseudomonas**. Given for those immunocompromised or with factors such as DM. **1st -gen cephalosporins or penicillins with narrow coverage,** like cephalexin (Keflex) 250 to 500 mg PO four times daily and dicloxacillin 250 to 500 mg PO QID. **2nd-gen cephalosporins with broader-spectrum coverage**, like cefuroxime (Ceftin) 250 to 500 mg PO BID or cefdinir (Omnicef) 300 mg PO BID, or beta-lactamase–resistant penicillins like amoxicillin/clavulanate (Augmentin XR) 1,000 mg PO BID based on the amoxicillin component.	during follow-up (not CT or MRI). Neomycin, an antibiotic commonly found in otic preparations, is known to cause skin reactions and ototoxicity; limit duration of therapy.

					Ceftazidime (Ceftaz, Fortaz) 2 g IV q 8-12h or combination of tobramycin (1–1.5 mg/kg IV eq8h, with dosage adjusted by monitoring serum levels and renal function) and ticarcillin (3 g IV Q4H). These regimens carry significant risk of nephrotoxicity, ototoxicity, and bleeding diatheses.	
Otitis Media	More common in infants/young children. Increases in winter months. Native Americans (Navajos and native Alaskans) higher prevalence rate. Americans of European descent. Equal in men/women. Dysfunction in eustachian tube. Genetic conditions such as Down syndrome at risk. Active/passive smoking, crowded or unsanitary living conditions, exposure to wood-burning stoves, family history of OM. AOM risk factors: child in daycare, presence of tobacco smoke in home, and residing in communities where antibiotic-resistance	Acute OM: Otalgia, Otorrhea, and fever. Unilateral hearing loss, recent hx of URI. Dizziness, vertigo, tinnitus, vomiting, or nausea possible. Pain subsides with TM rupture and then complain of otic drainage. Recurrent OM: clearance of middle ear effusions between acute episodes of inflammation. Chronic OM: Presents with history of repeated bouts of AOM followed by a period of continuous or intermittent otorrhea lasting for more than 3 months. Pain is rare; hearing	AOM: Auralgan otic solution (combination analgesic and anesthetic agent; contra in ruptured TM) may be needed to assess ear. TM may be amber or yellow-orange, or may be infected and pinkish gray to fiery red. T M usually full or bulging in acute cases; absent/ obscured bony landmarks and cone light reflex. Chronic OM: perforated, draining tympanic membrane and possibly granulation tissue. Chronic, foul-smellling otorrhea typical of anaeroobic bacterial infection; a chronic grayish-yellow suppuration may be a cholesteotoma at site	Rarely needs if symptoms fit picture of OM. If confirmation is desired, pneumatic otoscopy will demonstrate decreased or absent tympanic membrane mobility in serous, acute, or chronic OME. Tympanometry may be useful if fluid buildup behind middle ear suspected in absence of other clinical signs; a flat tympanogram is consistent with restrictive disease of the middle ear cavity In subacute, recurrent, or chronic cases of OM, cultures and antibiotic	Uncomplicated OM may not require specific intervention other than pain and symptomatic relief: Acetaminophen, Ibuprofen. If signs and symptoms of AOM persist for 48 to 72 hours in spite of using systemic analgesics, the child should be reassessed, and antibiotic treatment should be considered. No-day care; no ABX in past 90 days: amoxicillin standard dose Day-care or ABX in past 90 days: amoxicillin high dose. **mild PCN allergy**: use 2nd generation cephalosporin: cefuroxime axetil (Ceftin), or cefprozil (Cefzil). **Severe PCN allergy or cephalosporin allergy**:	Teach preventive measures: avoid tobacco exposure, exclusive breastfeeding for 1st 6 mths of life or longer, annual influenza 6 months and older, pneumococcal 13 6 weeks of age and older, pneumococcal 23 for high risk children 2 years and older. Demonstrate proper cleaning technique. Bedrest or reduced activity may be suggested in severe cases until fever and pain subside, and the importance of completing the full regimen of all antibiotic therapies should be emphasized.

	forms of S. pneumoniae are endemic.	loss primary concern. OM w/ effusion: absence of s/s of infection. Typically complain of stuffiness, fullness, and loss of auditory acuity in affected ear. May hv pooping, crackling, gurgling sounds when chewing, yawning, blowing the nose. Pain rare. Vertigo rare. Usually afebrile and may have hs of recent viral URI or either allergic or vasomotor rhinitis.	of infection. Bullae in rare cased forms between tympanic membrane from M. Pneumoniae or certain viruses. Acute infection: lymphadenopathy of preauricular and posterior cervical nodes common. OME: examination of external ear unremarkable; mucous membranes of nasal and oral cavities may be infected or edematous from recent URI. Decrease tympanic mobility of pneumatic otoscopy.	sensitivity testing helpful in guiding alternative treatment. If cultures are obtained, fungi and mycobacteria should be specifically ruled out. sinus x-rays and CT scan to reveal mucosal thickening in middle ear in those with recurrent infection. Weber (sound lateralizes to affected ear) and negative Rinne test (bone conduction superior in duration and volume to air conduction).	extended spectrum ABX: Clarithromycin (Biaxin) or a sulfonomide like TMP/SMX; Bactrim Failure of initial AOM tx in Pedi pts with amoxicillin: give Amoxicillin-Clavulanate (Augmentin) Complicated and recurrent OM require tx	Avoid swimming until OM clears; immersion in water may lead to otitis externa, complicating the middle ear infection. Keep ear canal dry. T M perforation can be avoided by not using cotton swabs or sharp objects to clean ears. Traumatic injuries to the middle ear should be avoided as well to prevent perforation. All cases, especially with ruptured T M **blowing of the nose should be avoided**; do gently as possible if needed. Nasal saline used to liquefy nasal secretions and facilitate drainage.
Presbycusis (*sensorineural hearing loss; not reversible*)	Affects older adults 50 and >; tinnitus may be associated with presbycusis. Some contributing factors include environmental noise, loss of hair cells, hereditary factors, aging, health, and side effects of medication	“difficulty hearing,” hears mumbled or slurred speech, difficult hearing made worse with background noise, men’s voices easier to hear, some sounds seem overly loud, possible tinnitis in one	Bilateral hearing loss to high frequencies. Patient unable to hear you clearly at high frequencies. Perform Weber, RInne, and Schwabach tests. For sensorineural hearing loss: Weber (**sound in less**	Revealed by audiometric testing including pure tone and speech testing.	Hearing loss irreversible. Education should be provided to avoid further damage such as avoiding loud noises, wearing ear plugs, and hearing aids for hearing.	In cases of presbycusis it is important to educate and support the pt so that no further damage will occur; for example, exposure to

		or both ears	**affected/unaffected ear is louder**). Rinne (air should be 2 as long as bone conduction; **but in sensorineural loss, the ratio is equal**).			excessive noise and ototoxic drugs should be avoided.
Rhinosinusitis - viral	URI, airplane travel, smoking, air pollution, sneezing with mouth closed, chronic use of decongestants, cold damp weather, dry indoor heat, dental abscesses, swimming in contaminated water, nasal trauma	URI, airplane travel, smoking, air pollution, sneezing with mouth closed, chronic use of decongestants, cold damp weather, dry indoor heat, dental abscesses, swimming, nasal trauma. All sinusitis – present with nasal congestion, mucopurulent rhinorrhea, head pressure, maybe cough, maybe sore throat, eye pain, malaise, fatigue. Pain exacerbated by sudden head movements. Frontal sinus pain worsen when lying down; maxillary sinus pain worsen when erect; ethmoid sinusitis associated with retro-orbital pain. Subacute or chronic sinusitis – painless as with some cases of acute sinusitis.	Tender sinuses on palpation, nasal congestion, opacification of sinuses on transillumination, red/swollen nasal turbinates Acute sinusitis: total opacification on transillumination On palpation, the affected sinuses may be tender to palpation. Sphenoid sinusitis presents as tenderness over the vertex or mastoids, ethmoid sinusitis as retro-orbital or nasal bridge tenderness, maxillary sinusitis as cheek or dental tenderness, and frontal sinusitis as tenderness of the forehead. In the event of maxillary sinusitis related to a dental abscess, percussion over the affected sinus will produce	Noncontrast head CT recommended in more complicated cases, will show sinus opacification, air-fluid level or mucosal thickening	Saline nasal flushes, cool-mist humidifier, increase fluid intake, hot shower or compress for facial pain; ibuprofen, tylenol for pain, OTC decongestant (not longer than 4 days r/t rebound congestion); expectorants such as guaifenesin. Prescription drugs: fluticasone (Flonase), mometesone (Nasonex), triamcinocole (Nasocort). Oral antihistamines not indicated unless allergic component is evident. They dry the mucosa, thicken purulent sinus fluid, & slow mucosal drainage. Majority of acute rhinosinusitis cases are caused by viruses rather than bacteria, antibiotics are largely unhelpful	Increase fluids to thin nasal secretions, avoid aggravating factors such as smoke, air pollution. Report complications such as peri-orbital swelling, visual impairments, AMS, visual impairments, facial palsy. Avoid OTC decongestants with antihistamine.

			marked tenderness in the teeth and gums			
Rhinosinusitis - bacterial	Similar to above + persistent blockage of nasociliary sinus drainage, deviated septum, adenoidal hypertrophy nasal polyps, nasal neoplasms, Dx such as immunoglobulin A deficiency, immobile cilia syndrome (Kartagener's syndrome), cystic fibrosis, HIV, diabetes	Similar to above, postnasal and nasal drainage tends to be mucopurulent, yellow/green and pt reports symptoms longer than 7-10 days	Similar to above + more mucopurulent drainage	Anteroposterior, lateral, and particularly occipitomental sinus x-ray examinations can be done if symptoms show no improvement after 4 to 5 days of pharmacotherapy; Air–fluid levels, mucosal thickening beyond 4 mm, or complete opacification of the sinuses on any of these views is strongly suggestive of sinusitis. Presence of at least 10,000 organisms per mL on Gram stain of sinus aspirates may confirm local sinus infection. No routinely done since nairs have a diverse array of organisms. If allergic disease suspected perform allergy testing. Eosinophilia and elevated total or allergen specific IgE levels.	If symptoms last longer than 7-10, antibiotic may be warranted. First-line is Amoxicillin alone or Augmentin 1000 mg/125mg PO BID. May use Bactrim or doxycycline	Same as above, in addition to report no signs of improvement with antibiotic
Streptococcus						

Tinnitus	Hearing loss Labyrinthitis Meniere's Disease Otitis media Otitis externa Otosclerosis Ear canal blockage (from cerumen or foreign body) History of high or low BP Head trauma Anemia Hypothyroidism Hyperthyroidism Allergies Chronic exposure to noise damage cilia & auditory hair cells tinnitus Taking certain medications Reversable tinnitus Salicylates Quinine Alcohol Indomethacin Irreversible tinnitus Kanamycin Streptomycin Gentamicin Vancomycin Me: Hearing loss, labyrinthitis, Meniere's disease, otitis media, otitis externa, otosclerosis, earcanal blockage (from ear wax	Significant subjective findings "Sound of escaping air or running water" "buzzing, ringing, or humming noise" Unilateral or bilateral Often not affected by tinnitus until in an usually quiet environment Me: Sound of escaping air, running water, sound heard in seashell, or as ringing, humming, buzzing sound or roaring or musical sound in one or both ears when no environmental noise is present. **Subjective tinnitus** is more common and heard only by patient. Sound more prevalent and bothersome in quiet environments; less bothersome around noise. May affect sleep, concentration, and cause depression.	Significant objective findings Subjective ringing = will not see objective signs of ringing in ears Do orthostatic BP's Gross hearing tests, Weber & Rinne fork tests Thorough ear exam If unilateral Check for bruit on affected side Palpation of carotid may reveal weak pulse on affected side Consider cardiovascular studies Doppler ultrasound – assess carotids for stenosis EKG – detect changes of atherosclerotic disease Neuro exam Rule out neuro deficits that may suggest a neurologic etiology **Me: Objective tinnitus** heard with a stethoscope placed over head and neck structures near ear. High blood pressure via orthostatic	MRI – diagnostic procedure of choice May reveal ear-related pathology in detail Lab Tests – confirm possible underlying causes of tinnitus CBC – rule out anemia or infection Metabolic studies – rule out thyroid disease, hyperlipidemia, vitamin deficiency, zinc deficiency, electrolyte abnormalities If see drainage in canal culture drainage Me: Lab tests, CBC to r/o anemia or infection. Metabolic studies to r/o thyroid disease, high lipids, vitamin deficiency, zinc deficiency, electrolytes abnormalities. CUlture ear if drainage present. MRI - procedure of choice to evaluate ear pathology or a CT if MRI not possible. Tympanometry to check for presence of	1st line treatment Typically not treated successfully Manage symptoms Treat underlying causative disorder 2nd line treatment Oral antidepressants Effective in reducing symptoms Physical interventions – minimize distress caused by tinnitus Hearing aids Tinnitus-masking devices Eliminate possible offending medications. No successful treatment. No oral meds to help; however, oral antidepressants prove effective in reducing symptoms. Nortriptyline (Elavil), Diazepam (Valium), and Meclizine HCL (Antivert) have been used depending on the reason for tinnitus. If tinnitus due to otitis media, tx with antibiotics or if needed myringotomy. Find caucaustive factor to	Teach coping mechanisms. Avoid excessive noise, wear protective earplugs. Tinnitus-masking devices may help. External white-noise machine. Hearing aids to amplify environmental sounds and suppress tinnitus. Biofeedback for psychological problems may help. Stop smoking, decrease caffeine, chocolate, alcohol, and salt intake. Proper sleep hygiene. Chew gum or swallowing during descent of airplanes.

	or forign body), cardio disorders, hx of high or low BP, head trauma, anemia, hypothyroidism, hyperthyroidism, or allergies. Chronic exposure to noise. Certain meds w/ reversible effects include salicylates, quinine, alcohol, indomethacin [Indocin], and those w/ irreversible effects include kanamycin, streptomycin, gentamicin, and vancomycin. Strongly associated with aging. Men and Whites have a higher risk.		measurements. Perform gross hearing tests, Weber and RInne, otologic exam. Auscultate upper neck proximate to affected ear for possible bruit, and palpate for weak pulse. Cardiovascular studies: carotid doppler to assess for stenosis, EKG to detect atherosclerosis. Neurological exam to r/o other deficits.	middle ear fluid, acoustic reflex measurement, or acoustic reflectometry. Screen psychological disorders due to association with depression or as a somatic symptom to acute anxiety	help the problem from worsening. Supplement with Vit A, Vit C, cyanocobalamin, and nicotinic acid or with mag or copper.	
Viral conjunctivitis (highly contagious; usually caused by adenovirus which are associated with URIs or the common cold. Other viruses include HXV, HZV (zoster), and Molluscum contagiosum. 2 types of herpes viruses. HSV-1 typically occurs above the waist, and HSV-2 typically occurs below the waist. HSV conjunctivitis spread by contact with persons who	Current respiratory infection (common cold); STDs, hx of contact with infected person	Second eye becomes involved within 24-48h; burning/sandy/gritty feeling; initially unilateral, then bilateral s/s irritation, mild light sensitivity, and swollen lids; foreign sensation.	Injected conjunctiva; profuse tearing; mucus discharge; concurrent upper respiratory infections; enlarged or tender preauricular node. **<u>If herpetic HSV1 or HSV2</u>**, recurrences or vesicles on skin; Corneal infection with the hallmark “dendrite” appearance. If severe viral from	Usually none Consider culture of exudates for recurrent conjunctivitis or STD suspected; rarely indicated Immunofluorescence test for herpes simplex or chlamydia Viral swab (10-minute test) for adenovirus is costly, requires 6 passes, and may not be tolerated by children	Self-limiting; resolves in a few days to weeks; therapy reduces symptoms. Artificial tears. Antihistamine/decongestant drops (naphazoline/pheniramine) for severe itching Trifluridine (herpes conjunctivitis); or acyclovir po	Same as bacterial: hand hygiene **Highly contagious; absence from work or school until absence of redness or tearing has resolved**.

have visible, infected lesions and with persons symptomatically shedding the virus. Meaning, the patient may be experiencing a prodrome of ill-related symptoms such as malaise, low grade fever, pain or tingling near site of the lesions (but the lesions are not yet visible).			herpes zoster or simplex: burning sensation, rarely itching; unilateral, herpetic skin vesicles in zoster; palpable preauricular node. Palpable preauricular lymphadenopathy may be present. Hemorrhagic coxsackievirus-related epidemics. **DISCHARGE NOT PURULENT which is the difference between bacterial and viral.**			
Allergic conjunctivitis is usually caused by an environmental allergen such as pollen, grass, trees, and so on. Occurrence can be seasonal and can be isolated to the eyes or include upper respiratory allergy symptoms such as rhinitis.	Allergies.	**The hallmark characteristic symptom is itching**. Tearing; discharge.	There may also be uniquely identifying "bumps" on the conjunctiva which are called follicles and when present are **a Hallmark symptom of allergy.** Other symptoms can include a diffuse, milky, conjunctival hyperemia; swollen conjunctiva; tearing**; and symptoms are almost always bilateral.**		Treatment is symptomatic. Cool artificial tears, anti-allergy eye drops (either OTC or RX) can be helpful, but prescription drops are very expensive, so start with OTC first. Systemic antihistamines are not very helpful for symptoms.	it is important to advise the patient not to scratch as this can result in a corneal abrasion and induces more itching by the inflammatory response.
SKIN DISORDERS						
Acanthosis nigricans: Benign dermatosis	May be a sign of hyperinsulinemia and	Complain of darkening of the skin or itching.	Most often present in posterior neck, flex	Obtain fasting blood glucose or HbA1C;	Diet and weight loss; d/c offending drugs; treat	Encourage diet and exercise.

characterized by velvety, hyperpigmented, hyperkeratotic plaques	insulin resistance but can be a marker to malignancy. A sign of risk of developing metabolic syndrome. Etiologies include obesity, insulin resistance, genetic syndromes, familial AN, malignant AN, and drug reactions. Most common between 11-40 and in those with BMI >30; indicator for risk of DM and subclinical atherosclerosis. .		URL's and inter trig IOU's surfaces (axillae, elbow, inframammary areas, groin and anogenital regions), most often asymptomatic but may cause pruritus. Skin exam: early or mild lesions may appear as a macular discoloration. May have dirty appearance on the affected skin with rough texture. Symmetric hyper-pigmented, hyperkeratotic, velvety to verrucous brown plaques.	fasting lipids, thyroid test, electrolytes to r/o DM or other endocrinopathies. Screen for malignancies. Low testosterone levels may be a predictor if AN in male, obese patients.	malignancy is associated with malignancy. Tx usually not indicated but Metformin has been shown to reducing AN lesions. It also improves insulin levels and promotes weight loss. Gastric bypass for weight reduction.	
Acne	Acne is a condition that is manageable but not curable. A provider must emphasize this to their patients so there are realistic expectations. Adolescent who has already tried self-treatment for several months Females more likely to verbalize emotional distress over their appearance Some patients' c/o pain and tenderness if acne is severe	Acne can occur at any age, and there are different levels of severity. Acne is classified into three categories mild, moderate, and severe. Facial involvement and other locations such as back, chest, and upper outer arms Mild is a patient with a few papules and some pustules. Lesions are primarily noninflammatory comedones with occasional small papules Moderate acne patients have papules,	Adolescent who has already tried self-treatment for several months Females more likely to verbalize emotional distress over their appearance Some patients' c/o pain and tenderness if acne is severe	Diagnosed by its classic location and characteristic lesions. A complete history is crucial to the diagnosis and supplants the importance of most diagnostic tests which are only needed when an underlying predisposing conditions is suspected	Good cleanser: benzoil peroxide or salicylic acid Benzoil peroxide can be drying and does tend to bleach towels or sheets, so make sure you educate your patient and parents on these side effects. (first-line therapy) Treating mild acne is best accomplished with a good cleanser and a retinoid with the possibility of a topical antibiotic. For a moderate case of acne, one would prescribe a retinoid, a topical antibiotic, and oral antibiotics. Adapalene is the lowest potency retinoid and good to use	Education is a vital component of acne treatment because of the long duration of treatment and potential adverse reactions Wait at least 30 minutes after washing the face before applying topical acne medications (topicals should not be used on sunburned or irritated skin) Sunscreen should be used with all acne medications Avoid oily makeup,