APPENDIX D

PBL questions and answers

Fluids, Electrolytes and Acid/Base Balance

PROBLEM 1

Severe Hypochloremic, Hypokalemic Metabolic Alkalosis with Intravascular Volume Deficit

A 45 year old man with previously known duodenal ulcer disease presents with complaints of persistent vomiting for the past 36 hours. The vomit is clear-looking and acidic in taste. He has no abdominal pain. Prior to the vomiting, he had difficulty with solid foods causing "fullness" in the stomach and he had been taking only liquids for one week. His heartburn had been aggravated at the time of the "fullness", but antacids did not help and he did not seek medical attention until today. He complains of being dizzy when he stands up. His blood pressure changes from 120/70 when lying to 105/55 when standing, his pulse changes from 100 to 130.

Study Questions:

1. What is the electrolyte composition of gastric contents?

Na+ 20-120 mEq/L; Cl- 130 mEq/L; K+ 10-15 mEq/L; HCO3- (-); H+ 30-100 mEq/L (KEY CONCEPT: high in Cl-, K+, H++; can rationalize the type of acid-base disturbance observed based on this knowledge)

2. What is the most likely acid-base disturbance in the patient?

Metabolic alkalosis (see title)

3. What percentage of intravascular volume has been lost?

Orthostatic--can relate analogously to Class II type shock (15-30% volume loss)--can also see decreased pulse pressure
Class I shock: typically see no physical exam changes.
Class III: (30-40%)-marked tachycardia, decreased systolic blood pressure, and oliguria
Class IV: (>40%)-significantly decreased systolic BP, severe oliguria/anuria; imminently life threatening

4. What intravenous fluid would be most likely to correct the acid/base and volume deficit?

0.9% NaCL (also replete K+ separately)--leads to decreased resorption and increased excretion of HCO3 – Na/HCO3 cotransporter in prox tubules (3:1 ratio) – therefore by replacing sodium with volume you decrease the HCO3 reabsorption here.

5. How much would be needed to replace the intravascular volume deficit?

TBW=60% (body Weight)
TBW=2/3 Intracellular Volume (40%); 1/3 Extracellular Volume (20%) 
Extracellular Volume=3/4 Interstitial Fluid; ¼ Plasma Volume (4-5%) of TBW

In 70 Kg man, the plasma volume is thus about 3 - 3.75 L. (appr. 2L RBC)

A loss of blood volume of 30% corresponds to an approximate 1.5 L loss (assuming 5L blood volume): 14 L is the Extracellular Volume in a 70-kg person.

1.5 L x 14 L/3.75 L = 5.6 L

OR

Volume deficit = blood volume (66mL/kg males, 60mL/kg females) X %loss BV
Resuscitation volume = VD x 4

**6. What would be the sodium, potassium, and hydrogen ion concentration in the urine (normal, high, low for each)?**

Na+ low, K+ high (aldosterone effects); H+ high (paradoxical aciduria)—this latter occurs as hypokalemia worsens and the kidneys try to preserve K+ in exchange for excreting H+. – draw aldosterone pathway

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**PROBLEM 3**

**Excessive Ileostomy Output and Dehydration**

A 36 year old man has undergone a total colectomy and proctectomy for ulcerative colitis two years previously, and has an ileostomy which usually drains about 800 cc per day. Two days before coming to the hospital he developed crampy abdominal pain, bloating, and began draining large quantities of liquid from his ileostomy. Because of nausea and two episodes of vomiting he did not take any food or liquids over the past 24 hours.

**Study Questions:**

1. **What is the usual amount of ileum output into the colon each day?**

   1-3 L

2. **What are the electrolytes of ileostomy output?**

   New ostomy: Na+ 130, K+ 20, Cl- 110, HCO3 30
   Adapted ileostomy: Na 50, K+ 5, Cl- 30, HCO3 25
   LR – Na 130, Cl 109, lactate 28, 4mEq K, 3mEq Ca
   pH = 6.5, but is an alkalizing solution (because lactate is converted to bicarb)

3. **What would be physical examination evidence of dehydration?**
Dry mucous membranes, tachycardia, low urine output, decreased pulse pressure/systolic pressure (more extreme)

4. What laboratory tests would you order to assess the degree of dehydration and what alterations would you expect?

Panel 7 (increased BUN/Cr ratio)
Urine electrolytes (FeNA) −<1%

5. What intravenous fluid would you administer to replace the ileostomy output?

LR or D5/2 NS c 20 K

PROBLEM 5
Measurement of Fluid Balance

A 65 year old man with known congestive heart failure treated with digoxin and a diuretic undergoes an uneventful abdominal aortic aneurysm repair with 1,000 cc of blood loss. During the four hours of surgery he is administered 4,000 cc of lactated Ringer's solution. During his first eight hours after surgery he is administered 1,000 cc of lactated Ringer's, has made 250 cc of urine and has drained 200 cc from a nasogastric tube.

Study Questions:

1. What methods would you use to evaluate the intravascular volume status in this patient eight hours after surgery?

Urine output, CVP, SG catheter, Urine electrolytes (FENA), physical exam findings-edema etc.

2. What methods would you use to evaluate the total body fluid status of this patient?

Physical exam-edema, total body weight, Serum Na

PROBLEM 8
Hyponatremia

A 55 year old woman with alcoholic cirrhosis and ascites is admitted for upper intestinal bleeding. Upper endoscopy reveals gastritis which is not actively bleeding. She is admitted and given an intravenous of 5% dextrose and 0.2% NaCl at 125 cc/hr. Over the next 24 hours her abdomen becomes tense and her urine output is 15 to 20 cc per hour. Her serum sodium has decreased from 132 on admission to 122 and she is less responsive to verbal stimuli.
Study Questions:

1. What are possible etiologies of hyponatremia?

Pseudohyponatremia (protein, lipids)
Dilutional hyponatremia (hyperglycemia, mannitol)
True Hyponatremia—
   Decreased ECF volume: (plasma, GI skin, renal losses)
   Nl ECF volume: (SiADH, psychogenic, hypothyroidism, hypoadrenalism)
   Expanded ECF volume: CHF, cirrhosis, nephritic syndrome, malnutrition

2. What is the most likely etiology in this patient?

Cirrhosis (expanded ECF volume)

3. What happens to urine sodium concentrations with the several etiologies listed above?

Psychogenic (100)
SiADH (>100)
Heart Failure (Low Urine Na+)
Renal Failure (High Urine Na+)

4. What is the treatment of hyponatremia in this patient?

Free water restrict; + Na restrict (loop diuretic)

PROBLEM 12

Acute Renal Failure

A 64 year old man has surgical resection of an abdominal aortic aneurysm with graft interposition. The operation is difficult and six units of packed cells are infused during the surgery. The patient’s blood pressure twice fell to 70 systolic during the four-hour operation. Eight liters of crystalloid were administered, 500 cc of fresh frozen plasma, and 1,000 cc of Hetastarch. The patient came to the intensive care unit with a systolic blood pressure of 60 mmHg. Three additional units of blood were given before his pressure is over 100 mmHg systolic. The patient made 100 cc of urine output during the case but in the ICU is noted to be oliguric, with 5 to 10 cc of urine output during the first four postoperative hours. Mannitol, which was given in the operating room, was repeated with no increase in urine output. During the next twelve hours six liters of crystalloid and 500 cc of packed red cells are administered resulting in 75 cc of dark yellow urine. By this time his hemoglobin and hematocrit have stabilized at 9.6 gms and 27.8%.

Laboratory values are:

WBC 11,800
BUN 55 mg/dl

Urinalysis=

sp. gr. 1.010
Creatinine 2.1 mg/dl
Na++134 mEq/l
K+5.8mEq/l

urine osmolarity 300 mOsm/L
0 rbc/hpf
0 wbc/hpf
0 pigmented granular casts
protein 2+
urine Na++45 mEq/l

Study Questions:

1. What is the differential diagnosis of the oliguria? The most likely diagnosis?

Pre-renal; Intra-renal; Post-renal

2. What is the most likely etiology?

ATN

3. What is the natural history of this disease?

The disease typically lasts 1-3 weeks as tubular cells regenerate after ischemic insult, but course may be quite variable depending upon extent of initial insult.

4. What are the principles of management of this disease?

IV hydration
Cease nephrotoxic drugs
Optimize oxygen delivery to organ systems
Potentially Dialysis
(Sometimes diuretics are used to maintain urine output in the setting of renal failure, but studies have not shown a benefit to ultimate kidney(s) or patient survival with this approach.)
Biliary Tract

PROBLEM 50

Gallstones
A 19 year old woman presents with history of intermittent epigastric and right upper quadrant pain which occurs about 15 minutes after eating and lasts for one to two hours. This pain has been happening for about one month, two to three times a week, especially after eating french fries. She is 5 ft. 4 inches tall and weighs 130 pounds. She has never been pregnant and is currently taking birth control pills. Physical examination is normal. An ultrasound of the gallbladder demonstrates multiple small stones.

Background info:
Approximately 8% males and 17-20% females have gallstones
80% of those are asymptomatic and 20% are symptomatic
2% of the asymptomatic patients will become symptomatic
75% of those have biliary colic. 30% of pts with biliary colic develop acute cholecystitis.

Bile contains bile salts, phospholipids (in the form of lecithin), and cholesterol. Any imbalance of the three will cause lithogenic bile. Excess cholesterol relative to the bile salts and lecithin will cause gallstone formation.

DDX of RUQ pain: PUD, pancreatitis, appendicitis, RLL PNA, cecal volvulus, MI, hepatic congestion, Fitz-Hugh-Curtis, hepatitis, Mirizzi syndrome.

Study Questions:

1. What types of gallstones can be formed? Which type is most likely in this patient?

3 Types of gallstones:
   - Mixed (80% - most common type): approx 70% cholesterol
   - Pure Cholesterol (10%): often solitary, large, non-calcified
   - Pigment (10%): composed of unconjugated bilirubin, calcium, and organic material. Black stones associated with cirrhosis and chronic hemolytic states. Usually sterile. Brown stones are associated with biliary stasis and more frequently found in CBD. Usually infected.

2. What factors predispose to the formation of gallstones?

Elevated estrogen and progesterone levels (fertile female/pregnant females), older age, DM, obesity, IBD, starvation/NPO, TPN, hemolytic states, rapid weight loss, malabsorptive states, certain race/ethnic backgrounds.

3. What is the pathophysiology of chronic and acute cholecystitis? What are the presenting symptoms, physical examination, and laboratory findings for each? Which one does this patient have?

95% of acute cholecystitis cases are due to obstruction of cystic duct by stones, which leads to biliary
stasis and bacterial overgrowth and infected bile. The difference between acute and chronic cholecystitis is that acute is severe enough to cause patients to seek medical attention right away whereas chronic patients develop either multiple mild acute symptoms or smoldering infections for long periods.

**Sx:** RUQ pain, N/V, fever, anorexia

**Signs:** mod elevation of WBC, alk phosphatase, and possible elevation of LFTs, temp, murphy’s sign, peritonitis/sepsis, palpable gallbladder.

4. **What are the treatment options for chronic and acute cholecystitis?**

Admit, NPO/IVF, IV abx, Lap vs open cholecystectomy 24-48hrs upon admission. Perc. cholecystostomy tube for unstable or patients that would not tolerate an operation.

5. **What complications of gallstones might develop in this young female if the gallbladder is not removed?**

Hydrops, gangrene or perforation of gallbladder, suppurative cholecystitis, cholecystenteric fistula, gallstone ileus, sepsis, death. Most common microbes: E. Coli, Klebsiella, enterococcus, enterobacter

**PROBLEM 51**

**Choledocholithiasis**

A 36 year old woman presents to the emergency room with a history of right upper quadrant pain, shaking chills, and jaundice. This pain came on suddenly six hours earlier and has been progressing. She took her temperature at home and it was 102. She vomited once at the onset of the pain. She has had intermittent episodes of epigastric and right upper quadrant pain after eating for the past six months. The pain always abated after thirty to sixty minutes. Her blood pressure is 110/60, her pulse 110, and her temperature 39 in the emergency room.

**Background info:**
Approx. 8-16% of pts w/ stones will have stones found in the CBD.
Most arise from the gallbladder thus secondary CBD stones
Stones that form de novo in the CBD are referred to as primary stones and almost always associated with bile stasis.

**Study Questions:**

1. **How is extra-hepatic obstructive jaundice differentiated from other etiologies?**

Lab values, radiographs (U/S, CT, ERCP/MRCP)

2. **What are the etiologies of obstructive jaundice?**

Stone, stricture, cancer (head of pancreas/bile ducts) or intrinsic or extrinsic tumors, primary sclerosing cholangitis, acute and chronic cholangitis, parasitic infections (liver flukes or ascaris lumbricoides),
choledochal cysts, and AIDS cholangiopathy.

3. **What tests would you use to differentiate etiologies of obstructive jaundice?**

Lab values (elevated T. bili., D-bili., GGT, AP, AST/ALT [normal in early stage] + urine bilirubin), Signs (clay-colored stools, dark urine, jaundice [90%], scleral icterus, hepatomegaly[20-40%], RUQ mass), Sxs (pruritis [66%), abd pain[30-50%], wt loss[30-50%], fever[20%]). Courvoisier’s sign (palpable gallbladder)

RUQ U/S: best to evaluate bile ducts
ERCP/MRCP: diagnostic and therapeutic
CT of abdomen/pelvis
PTC +/- Bx

4. **Which etiology is most likely in this case? What are the treatment priorities and management options for this case?**

Pt’s w/ Charcots triad (RUQ pain, fever, jaundice) or Reynolds pentad (charcot + hypotension/MS change) is an emergency and rapid decompression of the biliary tree is necessary. O/W make pt NPO/IVF, IV abx, ERCP/sphincterotomy and stone retrieval, then urgent lap vs open cholecystectomy +/- IOC (intraoperative cholangiography). If no ERCP, or if it is unsuccessful, then open surgical stone extraction with T-tube placement or choledocho-enterostomy.

Complications of untreated choledocholithiasis: cholangitis, pancreatitis, biliary cirrhosis, biliary stricture

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### PROBLEM 52

**Obstructive Jaundice**

A 74 year old woman presents with a complaint of jaundice which her husband noticed two days before. She has had no specific pain but has noted post prandial epigastric discomfort which has not responded to antacids. She has lost 20 pounds over the past three months, but is on a diet. Physical exam reveals an obviously icteric woman with a non-tender globular mass in the right upper quadrant.

**Study Questions:**

1. **How is extra-hepatic obstructive jaundice differentiated from other etiologies?**

As above

2. **What are the etiologies of obstructive jaundice?**

As above

3. **What tests would you use to differentiate etiologies of obstructive jaundice?**

As above + CA 19-9/CEA
4. Which etiology is most likely in this case? What are the treatment priorities and management options for this case?

Gallbladder cancer.
Associated with gallstones >95% of the time. Increased incidence for those with porcelain gallbladder. F>M 2:1, especially elderly and Native Americans. Adenocarcinoma (most common cell type, 80%).
Tx: In situ disease, only cholecystectomy
Thicker lesions (in or through muscularis propria, require cholecystectomy and wedge resection of adjacent liver, and LAD)
Poor prognosis: >90% in 1yr

Cholangiocarcinoma:
Rare but highly lethal. >90% adenocarcinoma. Account for 3% of all GI cancers. Incidence of 1-2/100,000. Avg. 5yr survival rate 5-10%, this also depends on where the lesion is located on the biliary tree. There is no evidence that bile duct stones have a role.
Risk factors: PSC, fibropolycystic liver disease, parasitic infection, cholelithiasis/hepatolithiasis, toxic exposure (thorotrast), Lynch Syndrome and biliary papillomatosis, chronic liver disease, viral hepatitis, DM, and HIV.
Tx: Surgical resection only chance for cure. Palliation can be achieved by surgery, IR, or endoscopic decompression.
Small Intestine - Crohn's Disease

PROBLEM 77

Right Lower Quadrant Pain

A 22 year old woman presents with a two-day history of increasingly severe abdominal pain. The pain began in the periumbilical region, and after 24 hours migrated to the right lower quadrant where it has remained. She has vomited three times and has had a poor appetite. Bowel movements are normal. There are no urinary symptoms. Her last menstrual period was two weeks ago. She is sexually active, and she has noted a foul-smelling vaginal discharge during the past week.
On physical examination, the patient has a temperature of 101°F and is in moderate abdominal stress. There is significant guarding and tenderness in the right lower quadrant, and slight tenderness without guarding in the left lower quadrant. On bimanual vaginal examination there is mild tenderness on motion of cervix; no adnexal masses are palpable.

Study Questions:

1. What is your differential diagnosis?
   Appendicitis, salpingitis, ectopic pregnancy, pregnancy, ruptured ectopic pregnancy, gastroenteritis, inguinal hernia, nephrolithiasis, ruptured ovarian cysts, ovarian/testicular torsion, epididymitis, pelvic inflammatory disease, Crohn’s disease, acute ileitis(Yersinia), UTI, acute mesenteric adenitis(children), intussusception (children), diverticulitis (adults), Henoch-Scholein Purpura, perforated peptic ulcer or bowel.

2. If this patient had never had sexual intercourse, how would that change your differential diagnosis?
   It shouldn’t

3. Which laboratory and imaging studies (if any) would be useful?
   How would each help in ruling in or out the diagnoses in question #1?
   a) Labs: CBC, Panel, b-HCG, urinalysis and culture
   b) X-ray: Obstruction series, CT abd/pelvis, abd/pelvic ultrasound

PROBLEM 78

Abdominal Pain

A five year old boy presents with abdominal pain of four days duration. Three days ago his parents took him to his pediatrician because he had a fever of 102°F, was complaining of a "bellyache," and was
vomiting. The parents were told that the boy probably had gastroenteritis, that he should be given a clear liquid diet and that the symptoms would resolve. Instead, the boy has become increasingly ill with worse abdominal pain. He continues to vomit and his temperature has reached 104°F. On examination, the child appears to be dehydrated and he lies on his side quite still with his knees drawn up. The abdomen is distended, with a diffuse guarding and tenderness. Rectal examination reveals tenderness and fullness anteriorly. A surgeon is consulted and takes the child to the operating room with a preoperative diagnosis of perforated appendicitis.

**Study Questions:**

1. **What is the differential diagnosis of the child's abdominal problem?**

   Perforated appendicitis, incarcerated/strangulated inguinal/umbilical hernia, intussusception, meckel's diverticulum, volvulus, mesenteric lymphadenitis

2. **If it is perforated appendicitis, what will be the definitive treatment for this child's condition?**

   Abx plus lap or open appendectomy with drain placement or abx +/- perc drainage and interval (6wks later) open vs lap appendectomy if pt stable

3. **Why does appendicitis so frequently perforate in this age group?**

   Delay in diagnosis 2/2 differing presentation than in adults.

4. **What is the usual pattern of temperature elevation in patients with non-perforated and perforated appendicitis? If perforated appendicitis is found, what are the more frequent postoperative complications for which this patient is at risk?**

   High spiking fevers for perforated appendix vs low grade fevers in non-perfs
   Complication risk 3% nonperf to 37% perf
   Sepsis, abcess formation, wound infection, wound dehiscence, bowel obstruction 2/2 extensive adhesion formation, fecal fistula, incisional hernia

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**PROBLEM 79**

**Meckel's Diverticulum**

A 14 year old boy presents with a two-day history of suprapubic and right lower quadrant pain. He has localized guarding and tenderness, and surgery is performed for suspected appendicitis. At operation the appendix appears to be normal, but an acutely inflamed Meckel's diverticulum is noted.

**Study Questions:**

1. **If the appendix appears normal during a laparotomy for presumed appendicitis, where should one look for a possible Meckel's diverticulum?**
45-60cm proximal from ileocecal valve on antimesenteric surface of ileum

2. Are there any signs or symptoms of Meckel's diverticulitis that can readily differentiate it preoperatively from appendicitis?

Not really, pt may have bleeding. Pre-op studies not really needed/unnecessary for prior to OR

3. Following the resection for the inflamed Meckel's diverticulum, should one also remove the normal appendix?

Yes

Also discuss the possible role for resection of an asymptomatic Meckel's diverticulum found incidentally at the time of laparotomy for an unrelated condition.

In children studies say “yes” b/c risk of rxn less than risk of complication of diverticulum. In adults, opposite is true.

PROBLEM 80

Small Bowel Obstruction

A 54 year old man is admitted to the hospital because of crampy abdominal pain and bilious vomiting that has lasted for three days. He has not had a bowel movement or flatus for two days. Ten years ago he had an appendectomy for a perforated appendicitis, with no postoperative complications. Physical examination reveals a pulse of 100, blood pressure of 110/60, temperature 37.5°C. His abdomen is distended with hyperactive bowel sounds and tympany throughout. There is no abdominal tenderness to palpation. His hemoglobin is 15.4, WBC 10,000.

Study Questions:

1. What is the most likely diagnosis and what is the likely etiology for this patient's current condition?

SBO. Adhesive disease

2. How would your answer to question #1 be altered if this individual had never had any previous abdominal surgery?

Hernia, Colon Cancer, Diverticular disease, Volvulus

3. Which abdominal signs, if present, would be ominous indications that the patient needed urgent operative treatment?

Peritonitis

4. Which diagnostic laboratory tests are indicated? Which imaging study should be initially obtained
to confirm your clinical diagnosis?

Labs: CBC, panel, lactic acid, amylase, lipase, lfts
X-ray: obstruction series, CT abd/pelvis if pt stable for study

5. Discuss your immediate and subsequent management of this patient. Is more than one treatment option available?

Admit, NPO/IVF, NGT decompression, fluid/electrolyte resuscitation, serial abd exams, serial labs. If pt regains bowel function w/in 48hrs no surgical intervention necessary. If by clinical deteriorates or does not regain bowel function, then exploratory laparatomy is warranted.

PROBLEM 86

Ulcerative Colitis and Crohn’s Disease of the Colon

A 45 year old woman has just moved to town and has a 18 year history of "colitis". She comes to your office because she's been told that she needs close follow-up. She does not know if she has Crohn's colitis or ulcerative colitis, but she is fairly sure it is one of the two. She complains of two to three loose bowel movements per day that occasionally contain mucous and/or blood. Her medical history is otherwise negative.

Study Questions:

1. What points in her history might help you distinguish what type of colitis she has? Difficult to dx based on hx. Up to 10% IBD indeterminate

UC: diarrhea (79%), Abd pain (71%), rectal bleeding (55%), pus and mucus in stool, weight loss (20%), tenesmus(15%), vomiting(14%), fever (11%)
Crohn’s: diarrhea(90%), abd pain, fever, malaise, anorectal lesions, weight loss, extraintestinal manifestation(20%)

Why is it important to find out which type she has?

Surgery can be curative for UC but not for Crohn’s

2. Which diagnostic tests would you get to help distinguish which type of colitis she has?

UGI small bowel follow through, Barium enema, CT abd/pelvis, Sigmoidoscopy, colonoscopy

How do the radiographic and pathologic findings differ?

Crohns: x-ray: bowel wall thickening, longitudinal ulcers, transverse fissures, cobblestone formation rectal sparing, abscess, fistulas
Path: rectal sparing, aphthous ulcers, mucosal ulcerations and fissures, cobblestoning, skip areas, focal strictures, bowel wall thickening, transmural involvement, granulomas, mesenteric adenopathy,
anorectal disease

UC: x-ray: colonic distention(toxic megacolon), mucosal irregularity “collar button” ulcers, pseudopolyps, loss of haustations, colonic narrowing/shortening, strictures
Path: friable mucosa, granular irregularity, pseudopolyys continuous disease, “stove pipe” narrowing, mucosa involvement, loss of goblet cells, crypt abscesses, plasma cell infiltrate

3. What is the risk of cancer in ulcerative colitis? In Crohn's Disease?

UC: 1-2%/yr if present over 10yrs
Crohns: slightly increased incidence over average population

4. What is the role of surgery in these two diseases?

UC: surgery can be curative
Crohns: only indicated for fistulas, obstruction, perforation, abscess, growth retardation is children, cancer, perianal disease, hemorrhage

5. Are there any medical therapies for either of these diseases? If so what are they?

Yes

Crohn’s: Acute: steroids and sulfasalazine(Azulfidine), antibiotics (gm-/anaerobic coverage)
Maintenance: azathioprine(imuran), 6-MP, other immunomodulators
UC: Acute: steroids and sulfasalazine(oral/enema:5-ASA)
Maintenance: azathioprine, sulfasalazine
Spleen/Abdominal Wall

PROBLEM 60

Thrombocytopenic Purpura

A 35 year old woman sees her gynecologist for increased menstrual bleeding. She complains of bruises which come following minimal trauma. On physical examination her gynecologist notices petechiae on her legs and arms. Her uterus and ovaries feel normal. She is not pregnant. Her hemoglobin is 11 and her platelet count is 20,000. Her prothrombin time and partial thromboplastic time are normal.

Study Questions:

1. What is the differential diagnosis of thrombocytopenia in this patient?

Nl platelet count range in adult: 150K to 450K /microL

DDX of thrombocytopenia:

- **Decreased production**: 1) Chemotherapy/Radiation, 2) Infectious agents/viral (HIV, mumps, rubella, HepC, EBV), 3) Congenital (Fanconi anemia, TAR syndrome, Alport syndrome), 4) alcohol toxicity, 5) vitamin deficiency (B12)

- **Increased consumption**: ITP, TTP/HUS syndrome, allo-immune (post-transplant, post-transfusion), medications (heparin, valproic acid, quinine), mechanical destruction (after CPB, large aneurysms), HELLP syndrome, DIC, anti-phospholipid antibody syndrome, infectious etiologies (CMV, HIV), other auto-immune disorders (SLE)

- **Sequestration**: splenomegaly

- **Dilutional**: transfusions

- **Pseudothrombocytopenia**: platelet clumping from inadequate anti-coagulation

2. What information from history, physical examination, and other laboratory tests would you use to select a diagnosis?

- **History**: medications, pregnancy, recent viral infections, nutritional/dietary habits, transfusions/transplants, family hx, hematological/autoimmune disorders

- **Physical**: spleen size, LN enlargement, evidence of other congenital abnormalities or evidence of other autoimmune disorders

- **Labs**: CBC with peripheral smear

3. Which is most likely?
4. What is the management of this condition?

Depending on plt count, symptomatology:
Asymptomatic with platelet count over 50 K: typically no intervention

Medical Management:
1) corticosteroids-prednisone (1mg/kg) (initial)
2) IVIG (acute bleeding, preparing for surgery)

Indications for splenectomy:
Failure of medical management (refractory or relapsing cases), unable to take steroids, ITP diagnosis with plt count<30 K and no complete response to medical therapy; ITP dx for 6 weeks and platelet count<10K; pregnant women in 2nd trimester with plt count<10K or <30K and symptomatic unresponsive to IVIG or steroids; success rate of about 2/3 with splenectomy

PROBLEM 61

Hypersplenism

A 65 year old man sees his internist for complaints of fatigue, weakness, and increasing abdominal size. On physical examination his spleen is markedly enlarged, extending to the left iliac crest. His liver is palpated two fingerbreaths below the costal margin. His hemoglobin is 9.9, white blood count 4,500, and his platelets 100,000.

StudyQuestions:

1. What is the differential diagnosis of splenomegaly in this patient?

DDX for splenomegaly:
Congestive (liver failure/cirrhosis, CHF, splenic vein thrombosis), inflammatory (sarcoid, lupus, Felty's syndrome), malignancy (lymphomas, leukemias, myelomas, metastatic lesions, primary tumors), infectious (viral-EBV, CMV, Hepatitis, parasitic-toxoplasmosis, shistosomiasis), infiltrative (Gaucher's, amyloid), hematologic (hemolytic anemias, hereditary spherocytosis, sickle cell (children), thalassemia)

2. What information from history, physical examination, and other laboratory tests would you use to select a diagnosis?

History: alcohol use, hx of hematologic disorders,

Physical: evidence of ascites/liver size; leymphadenopathy, cardiac exam,

Labs: CBC with peripheral smear; reticulocyte count; other radiographic imaging (CT); possible tissue bx

3. Which is most likely?
4. What is the management of this condition?

Depends on extent and nature of liver disease

PROBLEM 62

Groin Hernia

A 25 year old man presents to your office with a complaint of an intermittent bulge in his right groin, which occurs with heavy lifting, but which goes back in easily.

Study Questions:

1. What predisposing medical conditions will you inquire about in the patient's history?

Hx of heavy lifting (although association is not completely clear); other etiologies of increased abdominal pressure (coughing); patient based causes (obesity, smoking);

2. On physical examination, a finger placed through the upper scrotum into the external ring palpates a bulge with Valsalva maneuver. Based on physical examination and the patient's age and sex, what is the likely type of hernia: indirect, direct or femoral?

Indirect inguinal hernia (most common hernia type in males and females, although much more common in males)

3. At the time of surgery the patient is noted to have a bulge through a weakness in Hesselbach's triangle. Define Hesselbach's triangle. Is this a direct or indirect hernia? Compare and contrast the anatomic and developmental differences between direct and indirect hernias.

Direct hernia--through Hesselbach's triangle; formed through wear and tear

Indirect hernia-lateral to Hesselbach's triangle; formed from failure of processus vaginalis to close perinatally

Hesselbach's triangle formed by inguinal ligament (Poupart's) inferiorly, inferior epigatsric vessels (laterally), lateral border of rectus muscle (medially)

4. What are your operative options for repair of this hernia defect?

Open (traditional):
Bassini
McVay
Shouldice (Canadian)
Lichtenstein repair

Laparoscopic:
TAPP (transabdominal preperitoneal repair)
TEPA (totally extraperitoneal approach)

PROBLEM 64

Ventral Hernia

You perform surgery for a perforated duodenal ulcer in a 70 year old man who has a history of steroid-dependent chronic obstructive pulmonary disease. Postoperatively, he develops an infection of his upper midline abdominal incision. After the wound is fully healed, the patient presents back to your office with complaint of a bulge in the incision, with straining. You make a diagnosis of incisional hernia.

StudyQuestions:

1. What risk factors contributed to the development of this incisional hernia? What other risk factors exist for development of incisional hernias?

   Poor technique (inadequate suture, too much tension); Wound infection
   Patient factors (obesity, ascites, diabetes, malnutrition/vitamin deficiencies, advanced age, pregnancy, smoking, coughing)
   Medications (steroids, chemotherapy)

2. What are the indications for repair of the incisional hernia? How can you minimize risk factors for recurrence after the repair?

   Indications
   1) Symptomatic--episode of incarceration; pt. medically fit for surgery
   2) arguably most all should be repaired because of risk of incarceration if pt. medically fit, but the larger the defect, arguably, the more elective the surgery (lower likelihood of incarceration)
   Optimize patient factors (decrease steroids if possible--Vit A, nutrition optimization, weight loss, smoking cessation, control of DM etc.)

   Optimize technical aspects (tension free repair/use of mesh, minimize likelihood of infection)
Colon and Rectum

PROBLEM 82

Diverticular Disease

A 55 year old obese man with known diverticular disease discovered at a colonoscopy for hemetest positive stool, comes to the emergency room complaining of severe left lower quadrant pain of four hours' duration. Prior to the severe pain he had crampy, poorly localized lower abdominal pain and nausea which progressed to severe, well localized left lower quadrant pain. His temperature is 38.5, his pulse 100, his blood pressure 120/70. He is distended with decreased bowel sounds and tympany. He demonstrates percussion tenderness and involuntary guarding in the left lower quadrant. Rectal exam reveals hemetest negative stool and tenderness on the left. His WBC is 15,000.

Study Questions:

1. Which complication(s) of diverticular disease is most likely in this patient?

Acute Diverticulitis—with or without abscess; the localization of the clinical symptoms argues against free perforation with diffuse contamination of peritoneal cavity.

2. What test(s) would allow you to differentiate the possibilities?

CT scan abdomen/pelvis radiographic study of choice for diagnosis of suspected diverticulitis (can get abdominal flat plate first to r/o free air) (other laboratory studies should include CBC, which often demonstrates an elevated WBC--this can be followed during treatment with IV abx to assess response; Panel 7; LFTs, amylase, lipase to assess for other GI pathology e.g. pancreatitis)

3. Which complication(s) are usually well treated without operation?

Diverticulitis with contained perforation and small abscess usually responds well to conservative therapy with IV abx, bowel rest (NPO), +/- drain placement (by IR) of abscess (depending upon size of abscess--very large abscesses may require surgical exploration) via trans-rectal, trans-vaginal, trans-peritoneal route.

Other complications include free perforation, obstruction, fistula formation (colo-vesicular, colo-vaginal, colo-enteric), which typically require surgical intervention.

4. When surgery is indicated what are the surgical options?

For acute diverticulitis:

1) Partial colectomy, closure of rectal stump, and formation of colostomy (Hartmann's procedure)--this can be reversed at a later date.

2) In general, if surgery is indicated, conservative approach is chosen, i.e. no primary anastomoses. If
contamination localized (no free perforation), one can consider anastamosis (should be made at level of rectum not sistal sigmoid colon), + pelvic drain, + diverting loop ileostomy.

PROBLEM 83

Rectal Bleeding

A 60 year old woman presents in the emergency room with a complaint of sudden onset of painless bright red blood per rectum which has occurred three times in two hours and is associated with feeling faint when standing. She has no previous significant medical or surgical history and is not taking aspirin or anticoagulants. Physical exam reveals a pulse of 110 supine with a blood pressure of 100/60. These change to a pulse of 130 and a blood pressure of 90/50 when sitting. Her temperature is 37. Her abdomen is not distended nor tympanic. There is no abdominal tenderness. Rectal exam demonstrates frank blood with clots with no obvious hemorrhoidal disease.

Study Questions:

1. What are the management priorities for this patient?

Physical exam findings c/w orthostasis, suggesting potential blood loss of up to 20-40% blood volume.

1. start with ABCs (airway, breathing, circulation)
2. two large bore IVs--2 liters of normal saline while awaiting blood products.
3. Type&Cross; Chem 7, CBC and coags (any abnormalities should be corrected with FFP, croprecipitate, platelets)
4. NG tube/NG lavage to evaluate for upper GI source
5. PRBCs as needed; uncrossed blood if hemodynamic stability persists with fluids resuscitation and crossed blood not available
6. Thorough Hx important to be taken concurrent with resuscitation efforts—e.g. hx of allergies; prior GI hx, hx of colonoscopy, medication hx (hx of anti-platelet agents like ASA and plavix)

2. What is the differential diagnosis for massive rectal bleeding?

Massive Upper GI bleed (e.g. peptic ulcer)
Diverticular disease
Angiodysplasia
Malignancy
Inflammatory bowel disease
Ischemic colitis
Meckel's diverticulum

Anal fissure
Hemorrhoids
Polyps

Note: The three conditions above can lead to rectal bleeding but seldom cause massive rectal bleeding.
3. What is the most likely etiology in this patient?
Diverticulosis

4. What diagnostic procedures would you recommend and in which order?

1) NG lavage--r/o upper GI source
2) anorectal exam
3) bleeding scan, CT angiography (former is more sensitive, but latter can give more specific vessel localization)
4) +/- colonoscopy (in the setting of massive lower GI bleed, it may be difficult to assess the source of colonic bleeding)

The key for lower GI bleed is to try to localize bleed--otherwise may need to commit to subtotal colectomy.

PROBLEM 85

Cancer of the Colon

A 65 year old man has a complaint of crampy lower abdominal pain and constipation. His physician finds hemetest positive stool with no rectal mass and recommends a colonoscopy. At colonoscopy a large, friable mass partially obstructing the sigmoid colon at 35 cm is biopsied and is positive for adenocarcinoma. No other colonic lesions are noted.

Study Questions:

1. What other diagnostic study(ies) besides colonoscopy might have been used to make this diagnosis?
   Barium enema study

2. What preoperative tests would you order specific for evaluation of adenocarcinoma?
   Labs (CBC, Chem 7, coags, LFTs)
   CEA (tumor marker)
   CT scan abdomen/pelvis to assess for metastatic disease (with particular attention to liver)
   Chest Xray

Tests to ensure proper medical candidacy of pt. for surgery (e.g. stress test, echocardiogram etc.

3. What does the Duke's classification refer to? What are the stages of colon cancer and how do these relate to prognosis?

Duke's classification refers to method of staging colorectal cancer--largely supplanted by TNM staging system.

Dukes A, B, C, D roughly correlate with TNM stages I, II, III, IV respectively. Dukes A refers to tumors that
are limited to the bowel wall but do not involve serosa; Dukes B tumors involve serosa or the perirectal fat; Dukes C indicates lymph node involvement; Dukes D indicates presence of distant metastatic disease. The 5 year survival rate for these stages, respectively is about 90%, 70-80%, 40-60%, <20%.

4. **How might this patient’s presentation be different if he had a right colon carcinoma?**

Often present later, but can present with dark stools, abdominal mass on R side of abdomen, microcytic anemia.

5. **How do you follow a patient postoperatively after resection of a colon cancer? What methods are currently available to detect recurrence?**

Follow up with physical exam, CEA levels (if elevated pre-operatively) q 3 months for first two years, then every 6 months, +/- CT scan abdomen/pelvis (depending on initial stage of tumor). If suspicion for recurrence or metastatic disease based on PE or CEA levels, imaging with CT, MRI (if diagnosis of liver mets in question by CT) and can also consider PET scan.

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**PROBLEM 87**

**Colon Obstruction, Volvulus, Intussusception and Impaction**

A 95 year old woman is sent to the emergency room from a nursing home with complaints of abdominal pain and distention. It is unclear, but staff at nursing home think it has been about four days since she had a bowel movement. On examination, the woman has an expressive aphasia and a right hemiparesis from a previous stroke. Her vital signs are pulse 90, blood pressure 120/80, and she is afebrile. Her abdomen is markedly distended with hyperactive bowel sounds and tympany throughout. There is no abdominal tenderness. Rectal exam reveals large quantities of soft, brown, guaiac negative stool. KUB and upright reveal marked dilated colon consistent with obstruction.

**Study Questions:**

1. **What is the differential diagnosis of a large bowel obstruction?**

Colon cancer, diverticular disease, volvulus, hernia, IBD, adhesions, intussusception, pseudo-obstruction (Ogilvie's), other malignancies causing external compression on large bowel

**What test(s) would you employ to determine a specific diagnosis?**

start with abdominal flat plate

Then consider

Gastrografin enema

CT scan of abdomen/pelvis with rectal contrast

Pending results of these studies, one can follow with sigmoidoscopy/colonoscopy if clinically indicated to confirm diagnosis/therapeutic purposes.
2. Where can a volvulus develop and why? What would you see on abdominal x-rays if this patient had a volvulus?
Location: Sigmoid (more common, about 80% of cases) or cecum (less common)

3. Outline your treatment plan for a patient with volvulus. How would this differ from your treatment of a patient with a colon cancer or impaction?

Sigmoid volvulus--proceed with sigmoidoscopy and sigmoid decompression (if no evidence of necrotic bowel)
Should follow with elective sigmoidectomy and colorectal anastomosis to prevent recurrence if pt. operative candidate
Otherwise, if sigmoidoscopic decompression not possible may need to perform sigmoidectomy/Hartmann's procedure (or if necrotic bowel).

For cecal volvulus,
In emergent setting can do R hemicolecotomy, ileocolonic anastomosis.
Can also do cecopexy, but associated with higher recurrence rates
Colonoscopic decompression, as in sigmoid volvulus, also an option for acute setting, but often not successful, and in any case only a temporizing measure, since R hemicolecotomy should also be performed to prevent future recurrence.

4. What are the potential complications of a patient with large bowel obstruction if left untreated?

Large bowel obstruction is an emergency situation requiring early surgical consult. If condition left untreated, can lead to colonic ischemia, perforation and fecal peritonitis, with ensuing sepsis and potentially death.
**Shock**

**PROBLEM 19**

Hemorrhagic Shock

A 22 year old man was driving drunk and without his seatbelt fastened when he was involved in a single-vehicle automobile accident. When attended by EMT personnel, no information was available about the time of the accident. He was found agitated and complaining of abdominal pain. His airway was patent. At the scene, he was breathing at 20 per minute with a blood pressure of 90/60 and a pulse of 130. He was placed in a hard cervical collar and on a back board and transported to your emergency room. Upon arrival his vital signs are the same, with a temperature of 36°C. His abdomen is markedly distended. His hands and feet are cold, his legs mottled. A nasogastric tube reveals green liquid. A urinary catheter reveals dark yellow urine. His hemoglobin is 7. His abdominal lavage reveals gross blood.

**Study Questions:**

1. What type of shock does this patient exhibit?

   Hemorrhagic

2. What alterations in oxygen delivery are present?

   Diminished perfusion leads to diminished oxygen delivery. Oxygen carrying capacity is also decreased given loss of hemoglobin.

3. What acid/base category would be expected?

   Patient will mount a lactic acidosis (anion-gap acidosis). All patients in shock will be anaerobic to some degree and will be producing lactic acid as a result.

4. What is the effect of this kind of shock on the kidneys, the heart, the lungs, the brain, the intestine?

   With diminished perfusion, certain capillary beds will constrict before others in order to maintain perfusion to more “important” organs. Perfusion to the brain will be maintained at the cost of all other organs. Generally, the hierarchy of perfusion is heart/lungs>brain>intestine>kidneys>extremities.

5. What would be the cardiac output (low, normal, high)?

   Low, due to diminished cardiac filling.

6. What would be the systematic resistance (low, normal, high)?
High. The patient’s regional capillary beds would be clamped down in an attempt to maintain systemic pressure.

7. What would be the central venous and/or pulmonary capillary occlusion pressure (low, normal, high)?

Both CVP and PCWP would be low due to diminished volume in the entire vascular system.

8. What therapy would reverse the shock?

Immediate replacement with volume to increase global perfusion pressure is necessary. In true hemorrhagic shock, blood replacement is required, but if blood products are not available, general perfusion can be restored with replacement of volume with crystalloid.

PROBLEM 20

Septic Shock
A 65 year old man with known coronary artery disease (myocardial infarct three years earlier, currently taking a beta blocker) is admitted with acute left lower quadrant pain of six hours duration. His blood pressure is 90/50, pulse 120, respirations 18, temperature 39°C. He is flushed with warm hands and warm feet, his legs are pink. Physical examination reveals findings consistent with peritonitis in the left lower quadrant.

Study Questions:

1. What type of shock does this patient exhibit?

Septic

2. What alterations in oxygen delivery are present?

Diminished perfusion leads to diminished oxygen delivery. Oxygen carrying capacity remains normal.

3. What acid/base category would be expected?

Patient will mount a lactic acidosis (anion-gap acidosis). All patients in shock will be anaerobic to some degree and will be producing lactic acid as a result.

4. What is the effect of this kind of shock on the kidneys, the heart, the lungs, the brain, the intestine?

With diminished perfusion, certain capillary beds will constrict before others in order to maintain perfusion to more “important” organs. Perfusion to the brain will be maintained at the cost of all other organs. Generally, the hierarchy of perfusion is heart/lungs>brain>intestine>kidneys>extremities.

5. What would be the cardiac output (low, normal, high)?
Initially high. Systemic vascular resistance will drop as the vasculature dilates, and cardiac output will increase in a compensatory fashion as the heart works harder to maintain perfusion in this setting. In end stage septic shock, however, cardiac output can be low as the heart fails.

6. **What would be the systemic resistance (low, normal, high)?**

Low. The patient’s vascular system is dilated in septic shock, leading to very low peripheral vascular resistance. This is the primary pathologic hemodynamic alteration, and while the exact mechanisms remain unknown, it is generally attributed to a massive release of systemic inflammatory cytokines that have a profound vasodilatory effect.

7. **What would be the central venous and/or pulmonary capillary occlusion pressure (low, normal, high)?**

Both CVP and PCWP would be low or normal due to diminished cardiac filling pressure. The patient’s volume is maintained in the dilated peripheral vasculature and does not make it back to the heart properly. In sum, the heart senses that the patient is low on volume (mimicking hypovolemic shock).

8. **What therapy would reverse the shock?**

Therapy primarily consists of intravenous administration of alpha adrenergic agonists (Phenylephrine, Norepinephrine) which will restore peripheral vascular tone and allow the patient to regain vasoconstrictive ability. Another primary therapy is coincident volume resuscitation.

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**PROBLEM 21**

**Cardiogenic Shock**

A 55 year old man with stable angina which occurs twice a week while walking uphill and is treated with nitroglycerin undergoes an uneventful sigmoid resection for diverticular disease. On postoperative day four he develops severe substernal chest pain, sudden hypotension (85/55), tachycardia (120), and becomes agitated. Physical exam reveals total body mottling, cold hands and feet, distended neck veins and an S3 gallop. ECG demonstrates elevated ST-T wave segments in all of the anterior leads.

**Study Questions:**

1. **What type of shock does this patient exhibit?**

Cardiogenic

2. **What alterations in oxygen delivery are present?**

Diminished perfusion leads to diminished oxygen delivery. Oxygen carrying capacity remains normal.

3. **What acid/base category would be expected?**
Patient will mount a lactic acidosis (anion-gap acidosis). All patients in shock will be anaerobic to some degree and will be producing lactic acid as a result.

4. What is the effect of this kind of shock on the kidneys, the heart, the lungs, the brain, the intestine?

With diminished perfusion, certain capillary beds will constrict before others in order to maintain perfusion to more “important” organs. Perfusion to the brain will be maintained at the cost of all other organs. Generally, the hierarchy of perfusion is heart/lungs>brain>intestine>kidneys>extremities.

5. What would be the cardiac output (low, normal, high)?

Low. In cardiogenic shock, intrinsic inotropy is diminished. This results in decreased cardiac output independent of what is happening in the peripheral vasculature. This is the primary pathologic physiologic disturbance in cardiogenic shock.

6. What would be the systemic resistance (low, normal, high)?

High. Increasing vascular tone is a compensatory mechanism to maintain blood pressure in the setting of diminished “forward flow” or cardiac output.

7. What would be the central venous and/or pulmonary capillary occlusion pressure (low, normal, high)?

CVP and PCWP would both be high, as blood gets backed up from the heart into the lungs and the systemic venous system.

8. What therapy would reverse the shock?

Pharmacologic agents that increase cardiac inotropy (milrinone, epinephrine, dobutamine) are useful. Mechanical devices which augment forward flow (intraaortic balloon pumps; ventricular assist devices) are also useful, particularly when pharmacologic agents fail to restore cardiac performance.

PROBLEM 22

Neurogenic Shock

A 35 year old man dove into three feet of water at a swimming pool, did not emerge and was rescued by friends who performed CPR. When the EMTs arrived they found the patient to have a blood pressure of 80/50, pulse 100, and no spontaneous respirations, although he was opening his eyes. They began ambu bag assistance of respiration and placed a hard cervical collar. He was placed on a back board and transported to your emergency room. Upon arrival he has the same vital signs with warm hands and feet and pink extremities.

Study Questions:
1. What type of shock does this patient exhibit?

Neurogenic

2. What alterations in oxygen delivery are present?

Diminished perfusion leads to diminished oxygen delivery. Oxygen carrying capacity remains normal.

3. What acid/base category would be expected?

Patient will mount a lactic acidosis (anion-gap acidosis). All patients in shock will be anaerobic to some degree and will be producing lactic acid as a result.

4. What is the effect of this kind of shock on the kidneys, the heart, the lungs, the brain, the intestine?

With diminished perfusion, certain capillary beds will constrict before others in order to maintain perfusion to more “important” organs. Perfusion to the brain will be maintained at the cost of all other organs. Generally, the hierarchy of perfusion is heart/lungs>brain>intestine>kidneys>extremities.

5. What would be the cardiac output (low, normal, high)?

High. Similar to septic shock, the primary hemodynamic abnormality in this situation is diminished peripheral vascular tone, although the etiology of this decreased tone is different in the two situations. Cardiac output will increase as the heart rate and inotropy increase to maintain BP in the face of diminished PVR.

6. What would be the systemic resistance (low, normal, high)?

Low. This is the primary hemodynamic disturbance in this type of shock. Low SVR is a consequence of blocked sympathetic output to the vasculature from the spinal cord. This is most common in high cord lesions (cervical).

7. What would be the central venous and/or pulmonary capillary occlusion pressure (low, normal, high)?

CVP and PCWP would both be low, as blood pools in the periphery and does not return to the heart.

8. What therapy would reverse the shock?

Pharmacologic agents that increase PVR (intravenous alpha adrenergic agonists) are useful. Therapy is therefore similar to therapy for septic shock since a similar hemodynamic alteration is present.
Surgical Bleeding Disorders

PROBLEM 17

A 24 year old man is in the operating room for a massive liver injury sustained when his motorcycle hit a truck. After one hour of surgery he has received 15 units of packed cells and has developed diffuse oozing from the surface of his liver. Clots are no longer forming. His body temperature is 34°C.

Massive transfusion: >10uPRBCs/24hr

Study Question:

1. What are the most common coagulation difficulties associated with massive transfusion? What is the management of each?

1. Dilutional:
   a. Clotting Factors- Tx: Fresh Frozen Plasma(FFP)/4-6u PRBCs
   b. Platelets-Tx: PTLs/4-6u PRBCs. 1 pk=4-6u raises plts 50k
   c. Fibrinogen-Tx: Cryoprecipitate(Cryo). Replaces fibrinogen and some of the clotting factors(VIII, VWF, XIII)

2. Hypothermia:
   a. T< 32º(<90ºF) causes platelet sequestration and inhibits release of platelet factors
   b. Tx: Warming environment and transfusing warm fluids/products

3. Hypocalcemia:
   a. Caused by binding of ionized calcium to citrate (blood product preservative which occurs when blood transfusion rate exceeds 100cc/min. Decreased serum levels of ionized calcium depress myocardial function before impairing coagulation.
   b. Tx: CaCl2 or Ca gluconate

4. Metabolic acidosis:
   a. PH<7.2 (normal pH 7.4) impairs coagulation, myocardial contractility, and oxidative metabolism.
   b. Tx: Use of bicarbonate in the tx of systemic acidosis is controversial. Therefore should be limited to persons with protracted shock despite fluid resuscitation efforts

Wounds and Wound Healing

PROBLEM 23

A 55 year old man who is steroid dependent, asthmatic, and diabetic underwent a sigmoid resection and end sigmoid colostomy for perforated sigmoid diverticulitis. There was gross contamination of the entire abdominal cavity with large intestine contents. During the operation his blood pressure fell to 80 systolic. After irrigation of the abdomen to clear debris,
the abdominal fascia was closed with running #2 nylon suture material and the skin and subcutaneous tissue left open and packed with saline-moistened gauze.

Study Questions:

What type of wound closure was used in this patient?

Types of Wound Closure:

a. Primary Intention- healing of reapproximated tissues
b. Secondary Intention- healing of tissues by granulation, contraction, and epithelialization
c. Delayed Primary Intention- wound is closed after some healing by secondary intention. Usually done for contaminated wounds

What will be the sequence of wound healing events in this patient compared to the patient who had the skin closed?

Sequence of Wound Healing:

a. Inflammatory Phase (0-4d)- tissue damage at the site of injury stimulates the inflammatory process which occurs during the first 24hrs. Signs of inflammation (erythema, edema, heat, pain). Causes activation of coagulation, complement, cytokines (Prostaglandins, thromboxanes, PDGF, TGF-b/a, FGF-2, PD-EGF, PD-ECGF), Sympathetic Nervous System (Epi/NE), cells (platelets, monocytes/macrophages, mast cells, neutrophils.

b. Migratory Phase(>12hrs) - migration of fibroblasts and other mesenchymal cells into the wound during the migratory phase occur under the influence of cytokines. Migration through the provisional matrix is facilitated by the synthesis of metloproteinases(MMPs), which help cleave a path for the cells. Epithelization and angiogenesis also occurs in this phase.

c. Proliferative Phase(>5days)- fibroblasts synthesize proteoglycans and collagen and the wound gains strength. The rate of collagen synthesis increases greatly after the 5th day and collagen content continually increases for 3 wks and then plateaus. Collagen type I 80-90% in skin and collagen type III makes up the remainder

d. Wound Contraction/Scar Remodeling- myofibroblasts responsible for for wound contraction. Wound edges close at a rate of 0.6-0.75mm/day. Scar remodeling occurs >3wks. Collagen synthesis is downregulated and the wound becomes less cellular as apoptosis occurs.

What factors about this patient's illness and illnesses will influence wound healing?

Factors that influence wound healing:
-age, anemia, blood supply, immunosuppression, diabetes, malnutrition, obesity, radiation, sepsis/infection, steroids, wound tension, trauma, uremia, foreign body

On day seven after surgery, this man accumulated a large amount of serosanguinous fluid in the base of the wound. What is the most likely etiology of this fluid? What are the
management options for this condition?

Etiology of Wound Fluid:

- wound dehiscence. tx: OR for reclosure if pt eviscerated or cont dressing changes and later repair of resultant hernia.

Surgical Infections/Antibiotics

PROBLEM 24

A 55 year old woman undergoes an emergency cholecystectomy and common duct exploration for acute cholecystitis and cholangitis. *E. coli*, Klebsiella, and enterococcus grow out of the intraoperative bile cultures. She receives ampicillin and gentamicin preoperatively and this is continued in the postoperative period through day four. On day five she complains of increased pain in the stapled skin closure site. She has an increase in temperature from 38°C to 39°C, and erythema and warmth are noted in the mid portion of the wound.

Study Questions:

What type of wound (clean, clean-contaminated, contaminated, dirty) was present in this case? What is the relative risk of wound infection in these categories?

Definition of Types of Wound (Rates of expected wound infection rates):

a. Clean (1.5-5%)- vascular, skin, hernia
b. Clean-contaminated (>7%)- GI, GU, GYN, Respiratory tract surgery
c. Contaminated (10-15%)- penetrating trauma, bowel spillage
d. Dirty/Infected (15-40%)- gross pus, gangrene, bowel perforation

How do you make a definitive diagnosis of wound infection in this patient?

Definition of Wound Infection:

a. Superficial Wound Infection- approx 75% of wound infections
- Superficial to fascia and muscle
- S/S: fever, erythema, drainage w/wo bacteria, tenderness, fluctuance, non healing
- Tx: open wound widely, drain, explore the wound for deeper infection, loculation, fascial integrity, dressing changes. +/- Abx, culture

b. Deep Wound Infections-
- Involves muscles, fascia, and structures deep to them
- S/S: as above for superficial, fascial dehiscence, drainage bet fascial sutures, and evisceration, ileus
- Tx: explore in OR, abx
What other risk factors can contribute to increased infection rates?

Other Risk Factors that contribute to Increased Infection Rates: breaks in aseptic technique, hair removal, skin prep, avoid strangulation of tissues, avoid too much cautery, minimize suture, avoid post-op hypoxia, prophylactic abx –

What organism(s) would you expect to culture from this wound.

How would you treat a wound infection?

**How to find ID recs for preop and postop abx

Preop –

http://www.uphs.upenn.edu/bugdrug/antibiotic_manual/Perioperative%20Antibiotic%20Prophylaxis%20Guidelines.htm

Postop –


PROBLEM 26

A 35 year old woman who was involved in a motor vehicle accident undergoes a splenectomy for a severely injured spleen. On postoperative day one she has a temperature to 38.5°C, with physical exam demonstrating tubulo-vesicular breath sounds and egophony at her left base. Preoperatively administered cephazolin is discontinued after two doses. Her temperature over the next four days never goes below 38°C and on day five increases to 39°C. Her physical exam is unchanged. Her abdomen is as distended as it was immediately postoperatively, and she has passed little flatus. Her wound looks normal. She has little appetite and still requires intravenous fluid. A urinary catheter is still in place. Her WBC fell from 15,000 immediately postoperatively to 10,000 on day three. On day five it is 18,000.

Study Questions:

What were the likely etiologies of fever during the immediate postoperative period? What was most likely?

Etiologies of fever during the immediate post-operative period: atelectasis most commonly, necrotizing soft tissue infections with Clostridia or group A Streptococcus, anastomotic leaks, and aspiration PNA.

What test(s) besides physical exam would confirm this diagnosis?
What etiologies of fever are likely on day five. What is most likely? What test(s) would discern a diagnosis?

Etiologies of fever are likely on day five: infections become an increasingly likely source. UTI (u/a and culture), Wound infection (open wound, culture, +/- abx), IV line infection (remove catheter, check blood cultures, abx if necessary), intraabdominal abscess (CT, +/- drainage, OR evacuation, abx), DVT (duplex u/s, anticoagulation), cholecystitis (RUQ U/S, HIDA, npo/abx, IR drain, cholecystectomy), PE (CTA, anticoagulation), sinusitis, parotiditis.