Gastrointestinal - Clinical Management

Conditions Associated with Increased Gastrin and Secretin secretion

Gastrin
Peptide hormone normally produced in antral portion of stomach in response to peptide fragments & amino acids (esp. tryptophan, phenylalanine) released from ingested foods. Physiologic gastrin production is also stimulated by gastric distention via acetylcholine. Gastrin release is inhibited when intragastric pH falls below 3.0. Gastrin stimulates acid production by oxyntic cells and growth of gastric mucosa.

Supraphysiologic gastrin production as result of gastrinoma is called Zollinger-Ellison Syndrome (incidence 0.1 to 1 per million). ZE syndrome is often manifested by abdominal pain, ulcers of stomach and duodenum (90%), diarrhea (50%), and esophagitis. 75% occur sporadically and 25% are associated with MEN I syndrome (hyperparathyroidism, Pituitary adenomas, & Pancreatic islet cell tumors). Gastrinoma should be suspected in the setting of: ulcers recurrent/unresponsive to medical therapy/recurrent after ulcer surgery; family hx MEN I.

Diagnosis: fasting serum gastrin levels > 200 pg/ml (values > 1000 pg/ml pathognomnic) and Basal gastric acid output > 15mEq/hr. Secretin stimulation test- administer 2u/kg IV measure serum gastrin levels at 5min. intervals for 30 minutes. Increase of > 200pg/ml above baseline is diagnostic.

Treatment: Proton pump inhibitor. Imaging studies to localize the tumor and r/o metastases - most commonly to regional LN and liver, but also lung, bone, skin, spleen. Most gastrinomas are located in the gastrinoma triangle (described by Passaro, et.al)- the area encompassed by the junction of cystic and CBD superiorly, junction of 2nd and 3rd portions of the duodenum inferiorly, and junction of neck and body of the pancreas medially. Somatostatin Receptor Scintigraphy (SRS) - most sensitive imaging modality but limited by tumor size - detects 96% of tumors > 2cm, 64% of tumors 1.1 to 2 cm, 30% of tumors < 1.1cm. SRS has same sensitivity as UTZ, MRI, CT, and selective angiography combined. Can also detect carcinoids/neuroendocrine tumors and bone, lung, liver metastases. Endoscopic ultrasound + SRS can increase detection of duodenal wall gastrinomas. At operation, Intra-op UTZ, duodenectomy w/ bimanual palpation of duodenal wall, Kocher maneuver should be performed. Operative management: Duodenal tumors (71% 1st portion, 21% 2nd portion)- full-thickness excision w/ primary closure; Pancreatic Tumor- enucleation of tumor (distal pancreatectomy/whipple only if necessary); Liver metastases- should be resected if limited and can be done safely.

Prognosis: 49% pts w/ sporadic gastrinoma are disease free 5 yrs s/p surgery, 34% at 10 yrs vs. only 6% patients with gastrinoma assoc. w/ MEN I are disease free at 5 yrs, virtually none at 10 yrs . LN metastases has no impact on survival but liver metastases significantly decreases survival.

Other conditions associated with increased gastrin: Atrophic gastritis, pernicious anemia, vagotomy, renal failure, short gut syndrome, gastric outlet obstruction, retained excluded antrum, antral G cell hyperplasia.

Secretin
Produced by S cells of the duodenum and jejunum in response to acidification of the duodenum which occurs following a meal. Inhibited by release of pancreatic bicarbonate into the duodenal lumen. Stimulates pancreatic production of bicarbonate.

Fistulas are frequent manifestations of the transmural nature of Crohn’s disease. Immune activation triggers the release of a variety of proteases and matrix metalloproteinases that may contribute directly to tissue destruction, sinus tract formation, and, finally, penetration to adjacent tissue planes. Perianal fistulas are common, estimated to occur among 15% to 35% of patients. When the fistula arises from an anal gland, a low-lying perianal fistula develops. Such fistulas are often minimally symptomatic and may resolve with local care alone. Surprisingly, not all perianal fistulas occur in the setting of active rectal inflammation. In some cases, perianal fistulization may be extensive, forming a network of passages and extending to multiple openings that may include not only the perianal region but also the labia or scrotum, buttocks, or thighs.

A fistula that involves a substantial portion of the anal sphincter requires special treatment to avoid incontinence. Transanal advancement flaps are the most common surgical repair for complex fistulas. Success rates vary from 68% to 75%. Rates of incontinence after a flap repair range from 10% to 35%.

Fibrin adhesive made from autologous blood or commercial fibrin sealant has been used to close anal fistulas. Success rates vary from 60% to 80%. The fistulous tract is aggressively curetted, and the fibrin product is injected via the external opening until it is seen to emerge in the anal canal. No complications have been reported with this method, and continence is not affected. Re-treatment for failures can be successful. Setons may be used to drain a fistulous tract before surgical repair to prevent the accumulation of pus. However, they can also be therapeutic if used as a cutting seton. With this approach, after the seton is placed, the skin alone (not the sphincter) is incised over the fistulous tract, and the seton is tightened gradually over several weeks so that it gradually cuts through the muscle. With gradual division of the muscle, the muscle will remain scarred in place and the ends will not spring back as they would when cut during a fistulotomy. This technique allows the fistulous tract to be unroofed gradually as the cut ends of the muscle scar close to their usual location, thereby minimizing the chance of incontinence.

Describe the diagnosis and treatment of duodenal atresia

Duodenal atresia occurs during early intrauterine life and is probably related to a lack of revacuolization of the duodenum from its solid cord stage. Obstruction may be complete (atresia) or incomplete (stenosis). In 85% of cases, the obstruction occurs just distal to the ampulla of Vater and causes bilious vomiting. In 15% of cases, however, the obstruction occurs proximal to the ampulla, and the infant may vomit clear material. Maternal polyhydramnios is noted in 33 to 50% of cases, and the anomaly can often be detected by prenatal ultrasonography examination. Infants with duodenal obstruction are often premature (40 to 50%), 33% have Down's syndrome, and 50 to 75% have associated anomalies, including cardiac, renal, and other gastrointestinal defects (esophageal atresia with TE fistula and imperforate anus). At birth, the infant usually fails to tolerate attempted feeding.

Plain radiograph of the abdomen can identify the classic double-bubble sign (air-filled stomach and proximal duodenum) of duodenal atresia or stenosis (see figure below). If atresia is present, no air is seen distal to the duodenum. However, if stenosis is present, some air is seen in the intestine beyond the duodenum. In instances of complete obstruction, contrast studies are usually not necessary to confirm the diagnosis. An orogastric tube is passed to decompress the stomach and to avoid aspiration. Fluid and electrolyte disturbances are corrected to replete any losses related to earlier vomiting and dehydration. Antibiotics are administered, especially if the infant also has a cardiac anomaly (ampicillin 100 mg per kg per day and gentamicin 5 to 7 mg per kg per day). During preoperative preparation, cardiac and renal ultrasonographic examinations are indicated because of the high incidence of associated anomalies.

When the child's condition is stable (usually within 24 to 48 hours), the infant undergoes laparotomy under general endotracheal anesthesia through a supraumbilical right upper quadrant transverse incision. The operation of choice is a duodenoduodenostomy to bypass the obstruction. This can be performed with either a side-to-side or a proximal transverse-to-distal longitudinal (diamond-shaped) anastomosis. In instances of windsock web, duodenoctomy and excision of the web can be performed. The surgeon can identify the windsock web by using an intraluminal catheter to delineate the position of the obstruction and the site of proximal web attachment. The ampulla should be identified in each case by gently compressing the gallbladder, which results in a jet of bile from the ampulla. The anastomosis is accomplished in either one or two layers using interrupted 5-0 sutures. A side-to-side or diamond-shaped anastomosis is equally effective. At the time of the procedure, a small (8- to 10-French) red-rubber catheter should be passed distally to rule out a second distal mucosal web, which may occur in 2 to 3% of cases. Annular pancreas and/or malrotation is observed in 25 to 30% of cases. A preduodenal portal vein may be observed in rare cases and must be recognized and not divided. When the proximal atretic duodenal pouch is grossly dilated, an antimesenteric tapering duodenoplasty is a useful adjunct. Although concomitant gastrostomy was usually recommended in the past, a gastrostomy tube is rarely used now because of its potential role in causing GERD and because of the risk of aspiration. An orogastric tube effectively decompresses the stomach until the anastomosis functions.

Postoperatively, the infant receives intravenous maintenance support, and antibiotics are given for 24 to 48 hours and then are discontinued. Feedings are started when a bowel movement is observed and the gastric drainage and volume are minimal. Half-strength, low-asmolar, small-curc formulas such as a soybean-based formula or Pregestimil are useful diets. When increases are tolerated, the density and volume can be increased to full strength, 20 calories per ounce, in amounts adequate to deliver 120 calories per kg per day. The current survival is greater than 90%, with most deaths related to associated anomalies, particularly those affecting the cardiovascular system. Some late complications occur, including GERD, delayed gastric emptying, and megaduodenum. Duodenal stenosis presents with a partial obstruction and may not be detected until beyond the newborn period. Poor eating habits, bilious vomiting, and failure to thrive are often observed. Plain abdominal radiographs often show dilatation of the stomach and proximal duodenum, with some air noted distally. Duodenal stenosis may be caused by annular pancreas, malrotation with Ladd bands (incomplete rotation and fixation), an anterior portal vein, or a mucosal web with a small diaphragm. The preparation and operative treatment are similar to those noted earlier for duodenal atresia.
Plain roentgenogram of the abdomen demonstrates a "double-bubble" sign consistent with a diagnosis of duodenal obstruction. Air outlines the stomach and the first part of the duodenum. (From Rescorlia FJ, Grosfeld JL: Intestinal atresia and stenosis
Describe the treatment of a post lap-chole fluid collection

Patients who have persistent abdominal pain bloating, distension, anorexia, nausea, vomiting, fever, or jaundice after laparoscopic cholecystectomy must be worked up for 1) bile leak or 2) biliary obstruction.

Laboratory work up include LFTS, CBC, and amylase/lipase. A chemistry panel is helpful in patients who have had vomiting or limited oral intake. Hyperbilirubinemia and leukocytosis are associated with bile leakage and biliary obstruction.

Imaging should begin with abdominal ultrasound or HIDA scan which can reveal an intraabdominal fluid collection or dilated bile duct. A fluid collection can represent a bile leak, bleeding from hemorrhage, or gastrointestinal fluid from visceral injury. The etiology of most bile leaks is a cystic duct stump leak. Other causes include a partially or completely severed duct. A dilated bile duct may be a consequence of bile duct obstruction.

Treatment of a bile leak includes 1) drainage (which can be performed percutaneously using ultrasound/CT guidance or at the time of surgery if an operation is required) and 2) ERCP (endoscopic retrograde cholangiopancreatography). Failure to drain intraabdominal fluid collections can lead to sepsis and multiorgan failure. Drainage of a bile leak should be performed until the leaking duct seals. ERCP is 1) diagnostic, as it delineates bile duct anatomy in detail and can reveal leaks, obstructions, and retained common bile duct stones, and 2) therapeutic if combined with placement of a biliary stent or sphincterotomy and/or extraction of bile duct stones.

If ERCP demonstrates complete obstruction, percutaneous transhepatic cholangiography will delineate the duct anatomy above the injury, permit insertion of a stent, and allow for drainage until definitive operative treatment is performed.

Hepatocellular CA

Epidemiology
- risk factors:
  - cirrhosis (alcohol, metabolic liver disease)
  - Hepatitis B
    - weak link with hepatitis C
    - no link with hepatitis A
  - toxins (aflatoxin)
- gender:
  - high-prevalence regions: M:F = 8:1
  - low-prevalence regions: M:F = 2:1
- age:
  - high-prevalence regions: 30s-50s
  - low-prevalence regions: 70s-80s
- most common causes in US:
  - alcoholic cirrhosis
  - steroid use
  - hemochromatosis
- most common causes internationally:
  - hepatitis B
  - hepatitis C
  - aflatoxin exposure

Presentation
- high-prevalence (more aggressive tumors): bleeding, hepatic rupture, hemoperitoneum
- low-prevalence (more aggressive tumors): FUO, weight loss, malaise, malaise, hepatomegaly

Diagnosis and Workup
- CT and MRI are good for imaging lesions
  - MRI may be better for identifying smaller lesions
- AFP levels may be elevated

References
Emedicine: http://www.emedicine.com/radio/topic332.htm
Characterize Zenker's diverticulum

*Pharyngoesophageal diverticulum* was first described by Ludlow in 1769; however, in 1878, Zenker became associated with this entity. The pharyngoesophageal diverticulum, the most common esophageal diverticulum, usually presents in patients older than 60 years of age. The diverticulum characteristically arises within the inferior pharyngeal constrictor, between the oblique fibers of the posterior pharyngeal constrictors and the cricopharyngeus muscle or the UES. The transition in direction of these muscle fibers (Killian triangle) represents a point of potential weakness in the posterior pharynx. The Zenker diverticulum is a pulsion diverticulum resulting from a transient incomplete opening in the UES, also referred to as *cricopharyngeal achalasia*. The swallowed bolus exerts pressure within the pharynx above the UES and causes the mucosa and submucosa eventually to herniate through the anatomically weak area proximal to the cricopharyngeus muscle. The diverticulum enlarges, drapes over the cricopharyngeus, and dissect inferiorly in the prevertebral space behind the esophagus, occasionally well into the mediastinum. Manometry of the UES shows incoordination during swallowing, with pharyngeal contraction occurring after cricopharyngeal closure and resting pressures lower than in control subjects.

Zenker's diverticula are usually asymptomatic initially and are only discovered during a routine radiographic evaluation. Symptomatic patients may complain of a vague sensation or sticking in the throat, intermittent cough, excessive salivation, and intermittent dysphagia (particularly with solid foods). When the sac enlarges, especially in elderly persons, more severe symptoms develop, including cervical dysphagia, gurgling sounds during swallowing, regurgitation of food ingested several hours earlier, halitosis, voice change, retrosternal pain, and respiratory obstruction. To aid in swallowing, patients often develop various "maneuvers" including clearing the throat, coughing, or placing pressure on the neck. In rare cases, the pouch may be so large it obstructs the esophagus. The most serious complication associated with the Zenker diverticulum is aspiration, especially nocturnal, which can lead to pneumonia or a lung abscess. Other complications include perforation, bleeding, and carcinoma. Weight loss and dysphagia suggest esophageal malignant disease when the pouch is large and obstruction becomes severe.

An air-fluid level in the diverticulum can be detected on a plain film during chest or cervical studies. A barium esophagogram establishes the diagnosis. Because the origin of these diverticula is posterior, lateral views are essential. Anterior views are helpful to confirm the side of displacement. A persistent cricopharyngeal bar represents the incompletely relaxed or hypertrophied cricopharyngeal muscle. Manometric tracings of patients with a Zenker diverticulum compared with control patients (matched for age) show no difference, a finding casting doubt on the concept of incomplete UES relaxation in Zenker's diverticulum. Histologic studies support the concept of diminished UES opening by showing degenerative changes. Manometric testing of the cricopharyngeal area is generally not necessary in evaluating patients with a Zenker diverticulum. CT and MRI scanning are generally not necessary for diagnosis.

Endoscopic assessment and biopsy are necessary when mass defects or ulcers are seen on a barium esophagram. Safe endoscopy requires prior recognition of the diverticulum. When the endoscope tip enters a diverticulum, the endoscopist may feel a typical "pop" suggestive of passage by the cricopharyngeus and entry into the proximal esophagus; however, within a few centimeters, no lumen is found distally. An association exists between the Zenker diverticula-cricopharyngeal bar complex and GERD. Assessment of associated GERD with esophagoscopy and manometry may be best performed after surgical correction of the diverticula because the risk/benefit ratio of Zenker diverticulectomy myotomy is so favorable.

Surgical therapy in symptomatic patients is indicated, in most cases, regardless of size of the pouch. The degree of cricopharyngeal muscle dysfunction, not the absolute size of the diverticulum, determines the severity of cervical dysphagia. Therefore, the proper surgical treatment of pharyngoesophageal diverticulum, like that of every pulsion diverticulum, is directed toward the underlying motor abnormality responsible for formation of the pouch.

The most popular current surgical approach to the incoordinated UES is cervical esophagomyotomy and resection of the diverticulum performed through an oblique left cervical incision that parallels the anterior border of the sternocleidomastoid muscle or a transverse cervical incision centered over the cricoid cartilage. The sternocleidomastoid muscle and carotid sheath and its contents are retracted laterally, and the thyroid and
the trachea are retracted medially. The inferior thyroid artery is identified and is divided. The diverticulum is located beneath this vessel. With a 40-French bougie within the esophagus, the pouch is dissected to its base, and an extramucosal esophagomyotomy is performed in both directions from the base of the pouch (7 to 10 cm) to ensure that all cricopharyngeal muscle fibers are divided. Most pouches between 1 and 2 cm in diameter blend into the exposed mucosa and submucosa after the cervical esophagomyotomy. Some surgeons terminate the operation at this point without resecting the diverticulum, regardless of its size. Most surgeons advocate excising larger pouches by using a surgical stapler. Diverticulopexy (mobilizing the pouch, inverting it, and suspending it from adjacent tissues so the mouth is dependent) combined with a cricopharyngeal myotomy is an alternative. Endoscopic division of the common wall between the diverticulum and the esophagus (internal pharyngoesophagotomy, the Dohlman procedure) has also been used with success. Patients with known incompetence of the lower sphincter must be postured upright postoperatively after a cricopharyngeal myotomy is performed that renders the upper sphincter incompetent. Regardless of the surgical approach, recurrence is rare, and results are excellent.

List the contraindications to lap chole

Laparoscopic cholecystectomy can be used to treat just about anybody that is a candidate for open cholecystectomy. The few contraindications for a laparoscopic approach are:

1. Any contraindication to open cholecystectomy should also be considered a contraindication to the laparoscopic approach. These contraindications include recent MI, inability to tolerate general anesthesia, any uncorrectable coagulopathy.

2. Inability of the patient to tolerate pneumoperitoneum

3. Pregnancy, other than in the second trimester, is a relative contraindication. It is undesirable during the first trimester because of the possibility of induced abortion and exposure of the embryo to drugs and anesthetic agents. It is also undesirable during the third trimester because of the possibility of inducing early labor.

4. Known gallbladder cancer

Gallstone ileus

Gallstone ileus is a rare complication of cholelithiasis, and it represents a composite of gallstones and intestinal obstruction. Although it accounts for 3% of intestinal obstruction, gallstone ileus accounts for 25% of cases of non-strangulated intestinal obstruction in patients over age 65. A prerequisite for the development of gallstone ileus is a biliary-enteric fistula, enabling the passage of a gallstone into the intestinal lumen. Such cholecystenteric fistulae usually result from the inflammation of cholecystitis between the affected gallbladder and adjacent duodenum. Once within the intestinal lumen, gallstones can produce intestinal obstruction; this classically occurs at the narrower portions of the small intestine (i.e., terminal ileum). The obstruction is classically termed “tumbling ileus,” as obstructive symptoms are often intermittent while the stone passes distally through the small bowel. Patients are classically dehydrated elderly females, and they usually present with abdominal pain, distension, nausea, and vomiting, and they usually have a long history of biliary complaints (RUQ pain).

Abdominal radiographs reveal distended loops of small bowel with air fluid levels, and classically, pneumobilia (see above, blue arrow). Treatment usually requires ivf resuscitation, electrolyte replacement, bowel rest, and NGT decompression; with no response to these measures, the intestinal obstruction requires operative management. At operation, the obstructing gallstone is usually encountered in the terminal ileum; if small, it can be milked past the ileocecal valve. Most of the time, however, the stone is retrieved through an enterotomy, performed with care to avoid gross contamination of the peritoneal cavity with stagnant small bowel contents. Any non-viable intestine at the site of obstruction should be resected. Attempt at taking down the biliary-enteric fistula is generally discouraged, as it lengthens the case, adds significant morbidity, and most close spontaneously.

References:
Cameron JL. Current Surgical Therapy, 7th ed. pp 472-474.
THE DIAGNOSIS OF VIPoma

Clinical presentation – VIP (vasoactive intestinal peptide) is a 28 amino-acid peptide that functions as a neuropeptide. VIP is a potent vasodilator, stimulates pancreatic and intestinal secretion, and inhibits gastric acid secretion.

VIPomas produce the WDHA syndrome (a.k.a. Verner-Morrison syndrome), which stands for watery diarrhea, hypokalemia, and achlorhydria. They can also cause metabolic acidosis and flushing. Stool volume is >3L/day, and a non-gap acidosis results from loss of bicarbonate. Diarrhea is secretory, and persists despite bowel rest. Some patients may develop hyperglycemia which results secondary to hypokalemia - as well as VIP-induced glucogenolysis in the liver.

VIPomas are rare, accounting for 6% of familial pancreatic endocrine tumors (PETs) (MEN1), and <3% in sporadic PETs. Like all PETs except insulinomas (<10%), VIPomas are usually (>60%) malignant. Whereas gastrinomas occur in the gastrinomas triangle, VIPomas mostly occur in the body and tail of the pancreas (90%). However other tumors such as ganglioneuroblastomas, mastocytomas, small cell carcinomas of lung, pheochromocytomas, and GI carcinoids can also secrete VIP, and account for about 10% of tumors that produce VIP.

VIPomas tend to grow to large sizes secondary to delayed diagnosis. On average, the size of VIPomas at diagnosis is second only to non-functional PETs, and most have metastasized to liver at the time of diagnosis.

Differential diagnosis may include – infectious (bacterial or parasitic), post-surgical (short-gut, cholerrheic after ileal resection, bacterial over-growth), medical (AIDS, ischemic bowel, IBD, celiac disease, pancreatic insufficiency, lactase insufficiency), carcinoid syndrome (which shows high 5-HIAA level in the urine), and Zollinger-Ellison syndrome (elevated serum gastrin level), villous adenoma of colon/rectum, and laxative abuse or other drug induced diarrhea.

a. Laboratory tests – One can measure directly for VIP levels. Normal levels are <200pg/ml, whereas in tumor samples levels range from 225-2000pg/ml. Also as mentioned above, one must distinguish between the types of diarrhea. Stool studies, measurement of urine 5-HIAA (hydroxyindole acetic acid) and fasting and/or secretin-stimulated serum gastrin levels may be useful. Lower GI endoscopy may be helpful in diagnosis of villous adenoma of colon/rectum, and laxative abuse or other drug induced diarrhea.

b. Radiologic studies – CT, MRI, and arteriography may be used to localize VIPomas. 10% of VIP secreting tumors are extra-pancreatic (see above) and a CT of the chest may be helpful. Somatostatin receptor scintigraphy (OctreoScan) may detect both intra- and extra-abdominal VIPomas. EUS may also be helpful. However, because these tumors tend to be quite large at diagnosis, EUS and arteriography are usually not required.

c. Treatment (surgical) – Distal pancreatectomy is the operation for most VIPomas. These tumors are slow growing and debulking surgery with resection/ablation of liver mets may be indicated in select patients. If no tumor is found, one should explore the retroperitoneum and both adrenals.

d. Treatment (medical) – octreotide should be given as soon as the diagnosis is made to decrease metabolic derangement. Long acting formulations octreotide LAR and lanreotide are monthly or twice monthly injections. Streptozocin (STZ)- based chemotherapy regimens have only generated modest response.

References:
Treatment of gallstone pancreatitis

Initial therapy of acute pancreatitis consists of stopping oral intake, IV rehydration, and pain relief. Nasogastric intubation is useful in the presence of an ileus. Prophylactic antibiotic for severe pancreatitis has been shown to reduce infectious complications. Mild pancreatitis is usually self-limiting disease. Cholecystectomy should be undertaken during the initial hospitalization. If preoperative ultrasound documents choledocholithiasis or if choledocholithiasis is suspected, ERCP is undertaken before laparoscopic cholecystectomy to obviate laparoscopic common bile duct exploration. Regardless of whether ERCP is done preoperatively, operative cholangiography is recommended if there is suspected or documented choledocholithiasis.

Severe pancreatitis usually requires mechanical ventilation, hemodynamic support, and early nutritional support. Dynamic contrast-enhanced CT scan delineates anatomy and complications. The findings of peripancreatic fluid collections, pseudocysts or peripancreatic necrosis is not an absolute indications for surgical treatment. Instead, in the absence of deterioration and sepsis, observation is in order. Persistent abnormalities may require elective surgical treatment after the acute illness has passed. Pancreatic necrosis with signs of sepsis should prompt aspiration sampling. Pancreatic necrosectomy is considered when septic physiology accompanies pancreatic necrosis. Differentiated between inflammation and infection is critical. Without documented infection, aggressive nonoperative support is indicated. The risk of death for patients with very severe peripancreatic necrosis can be reduced from 40% to 10% by aggressive surgical treatment. There is no role for percutaneous drainage of infected peripancreatic necrosis. Results with open and closed drainage after operative debridement is comparable. Pancreatectomy is rarely if ever, necessary in the treatment of severe pancreatitis.

Norton et.al., Surgical decision making. 2000

Treatment of a hemangioma of the right lobe of the liver

First, a brief review:
In adults, hemangiomas of the liver are composed of large, cavernous vascular channels. They are the most common benign liver lesions, and are seen in 2%-7% of autopsies (mostly young women). Patients present with pain (50% will have chronic abdominal pain and vague RUQ pain), but this pain is usually due to coexistent cholelithiasis, ulcer disease, gastritis, esophagitis, or biliary cysts. Kasabach-Merritt syndrome is a coagulopathy characterized by low fibrinogen, increased fibrin degradation products, and thrombocytopenia, and is more common in children because their hemangiomas are comprised of smaller, capillary-like vessels. Rarely, there will be biliary obstruction, duodenal obstruction, or Budd-Chiari syndrome.

Most are less than 2cm. If they are larger than 4cm, they are giant hemangiomas.

If a suspected liver hemangioma is found incidentally in an asymptomatic patient during surgery, gross inspection makes the diagnosis. Follow-up CT-scan is necessary if a hemangioma is found with a radiographic study. If the hemangioma is discovered during a work-up for abdominal pain, you should first look for other sources of pain, and try analgesics if all other sources of pain have been eliminated. Resection of a presumed symptomatic hemangioma only helps the pain in 50% of cases.

Operate if:
1. The lesion is large and the patient is involved in high risk sports
2. Possibly before pregnancy (since it may enlarge)
3. There is a significant likelihood of cancer (other GI cancers, rising CEA) since the suspected benign hemangioma may be a vascular hepatocellular carcinoma or secondary metastasis.

Small peripheral lesions may be removed by enucleation or wedge resection. Large, deep-seated lesions require a hepatic lobectomy. Pre-operative percutaneous embolization may make the lesion easier to resect, and will be definitive treatment in elderly or high risk patients. Some centers use XRT or interferon-alpha.
Describe the treatment management of choledochal cysts

Bile duct cysts are associated w/ cholangiocarcinoma and gallbladder cancer.
The underlying pathogenesis is unknown, but possibly related to bile stasis, superinfections, repeated episodes of inflammation, or conversion of the bile salts to carcinogens.

Types

I- Choledochal Cysts-Cystic, fusiform sacular extrahepatic biliary dilation (50-60%)
   - Excision w/ hepaticojejunostomy
II- Extrahepatic Biliary Diverticulum
   - Simple cyst excision
III- Choledochoceles – dilation of extrahepatic, intraduodenal biliary tree
   - Excision via a lateral duodenostomy w/ simple cyst excision
IVA- Intrahepatic and extrahepatic secular dilation
   - Resection w/ hepaticojejunostomy
IVB- Multiple extrahepatic cysts
V- Caroli’s disease – multiple intrahepatic cysts
   If single lobe hepatic resection
   If entire liver – liver transplantation
Describe the diagnosis of malabsorption bile salts

Bile salts play an important role in the digestion and absorption of fat. They are synthesized from cholesterol in the liver (200 – 600mg/day) as conjugates of cholic and chenodeoxycholic acid. They are conjugated for excretion in the bile in the form of glycine or taurine conjugates. During digestion, the luminal concentration of conjugated bile salts causes aggregation in the form of micelles. Fatty acids and monoglycerides enter these micelles, forming mixed micelles. These mixed micelles more effectively achieve the solubilization of fatty acids and monoglycerides at the pH normally present in the intestinal lumen, and are absorbed via a passive process in the jejunum.

Ninety percent of conjugated bile salts are absorbed in the ileum entering into the portal vein – forming the enterohepatic circulation. As a consequence, only about 200 to 600 mg of bile salts are excreted in the feces per day. If the ileum is diseased or removed, absorption of bile salts is impaired leading to impairment of fat absorption. A similar situation occurs if bile salt reabsorption is prevented by chelating agents.

Malabsorption syndromes secondary to reduced bile salt concentration (with impaired micelle formation):
- Liver dz – parenchymal dz, cholestasis (intrahepatic, extrahepatic)
- Abnormal bacterial proliferation in small bowel – afferent loop stasis, strictures, fistulas, blind loops, hypomotility
- Interrupted enterohepatic circulation of bile salts – ileal resection, Crohn’s dz
- Drugs (sequestration or precipitation of bile salts) – Neomycin, Calcium Carbonate, Cholestyramine

Impaired excretion of conjugated bile salts results in impaired micellar lipid and frequently produces steatorrhea. In addition to steatorrhea, patients may have impaired absorption of fat-soluble vitamins (A, D, E or K) and calcium.

Tests useful in Malabsorption:
1) Quantitative determination of Stool fat (>6g/24h) – establishes presence of steatorrhea
2) Serum Calcium – frequently decreased in malabsorption
3) Serum Vitamin A
4) Prothrombin time – associated w/ Vitamin K deficiency

Reference: Harrison’s Principles of Internal Medicine. Greenberger, Isselbacher, 14th edition; Chapter 302
PRIMARY PIGMENT OF HUMAN BILE

Bile: Bile is made up of bile salts, bile pigments, and other substances dissolved in an alkaline electrolyte solution that resembles pancreatic juice. About 500ml of bile is secreted per day. Some of the components of bile are reabsorbed in the intestine and then excreted again by the liver (enterohepatic circulation).

Bile Salts: These are sodium and potassium salts of the bile acids (primary bile acids cholic and chenodeoxycholic acid and secondary bile acids deoxycholic and lithocholic acid), which are conjugated to either glycine or taurine and secreted into intestines. They serve many important functions that aid in fat emulsification, digestion, and absorption.

Bile Pigments: The glucuronides of the bile pigments, bilirubin and biliverdin, are responsible for the golden-yellow color of bile. Bilirubin, the primary pigment of human bile, is the end-product of the metabolic degradation of heme, the prosthetic group of hemoglobin, myoglobin, the cytochrome P450s, and various other hemoproteins. 70-90% of bilirubin is derived from the degradation of hemoglobin of senescent or injured circulating red blood cells.

The conversion of heme to bilirubin first entails the oxidative opening of the heme molecule by the microsomal enzyme heme oxygenase, resulting in the formation of the green tetrapyrrole biliverdin. Biliverdin is then reduced by a second enzyme, biliverdin reductase, to bilirubin. Bilirubin produced in the extrahepatic reticuloendothelial system (e.g. spleen) is transported to the liver within the plasma where it is tightly bound to albumin.

In the liver hepatocytes, the free (unconjugated) bilirubin is conjugated to glucoronic acid in a reaction catalyzed by glucoronyl transferase. This bilirubin diglucuronide (conjugated bilirubin) is much more water-soluble than free (unconjugated) bilirubin. Most of the conjugated bilirubin is secreted into the bile and into the intestine; however, a small amount of conjugated bilirubin can escape into the blood and is filtered and excreted in the urine, unlike the free (unconjugated) bilirubin that is never excreted in the urine regardless of how high the concentration gets because it is tightly bound by albumin and not filtered across the glomeruli. Normally, the gut does not “see” unconjugated (free) bilirubin because all of it would have been conjugated before it was excreted.

Following secretion into bile, conjugated bilirubin reaches the duodenum and passes down the gastrointestinal tract without reabsorption by intestinal mucosa. Although some reaches the feces unaltered, an appreciable fraction is converted to urobilinogen (a colorless compound), urobilin and stercobilin by bacterial metabolism within the ileum and colon. Urobilinogen is reabsorbed from these sites, reaches the liver via the portal circulation, and re-excreted into bile. Urobilinogen not taken up by the liver reaches the systemic circulation from which it is cleared by the kidneys. Urobilin and stercobilin are excreted in the feces with the stool and are primarily responsible for the brownish pigmentation of stool.

Harrison’s On-Line, Chapter 294: Bilirubin Metabolism and the Hyperbilirubinemias
**List the site of intrinsic factor secretion**

Intrinsic Factor is secreted by the **gastric parietal cells**. Cobalamin (vitamin B12), after being released from food in the stomach binds with R binder, a glycoprotein found in saliva and gastric juice. Cobalamin is released from the R binder in the duodenum where it binds to intrinsic factor. The IF-cobalamin complex is resistant to proteolytic digestion. The complex is transported into the mucosa of the terminal ileum. The IF is then removed and the cobalamin is bound to transcobalamin, which is released into circulation.

Both autoimmune destruction of parietal cells and atrophy of the gastric mucosa can lead to deficiency of IF. The result is inability to absorb cobalamin, and signs and symptoms of vitamin B12 deficiency including: megaloblastic anemia, gastrointestinal (smooth, beefy red tongue, anorexia, and diarrhea) and neurologic findings (numbness, paresthesia, weakness, and ataxia). Pernicious Anemia applies to Vitamin B12 deficiency which is secondary to chronic atrophic gastritis.

**Sources**


**Describe the inhibition of gastric mucous / bicarbonate secretion**

Gastric and duodenal mucous cells are located on the surface of the gastroduodenal mucosa facing the lumen and are responsible for secreting gastric mucous and bicarbonate. Their function has been postulated to protect the gastroduodenal mucosa from intraluminal acid. The high concentration of bicarbonate ion in the immediate space near the gastroduodenal mucosa neutralizes the acidic pH in the central lumen; there is a pH gradient. It also serves to protect gastroduodenal epithelium from intraluminal pepsin—the agent responsible for its degradation. **Cholinergic agonists, vagal nerve stimulation, and sham feeding** have all been shown to increase mucous cell bicarbonate. Inhibition with atropine results in acidification of the mucosal surface, thus predisposing it to ulceration and gastritis. **Adrenergic input** (stressful situations) also inhibit secretion. Further, cholinergic inhibition of bicarbonate secretion, thus resulting in a lower pH adjacent to gastroduodenal mucosal cells, has been shown to result in higher production of Prostaglandin E2. Prostaglandin E2 further induces mucous cells to secrete mucous and bicarbonate. **Inhibition of Prostaglandin E2 by Aspirin or NSAIDs** and thus the production of a mucous / bicarbonate layer to protect the gastroduodenal mucosa is the proposed mechanism of their role in formation of ulcer disease.

**Surgery: Scientific Principles and Practice.** Greenfield, 3rd edition; Chapter 18

List the activated pancreatic proenzymes

Pancreas has a major **exocrine** function in the production of digestive enzymes.

Amylase-breakdown starch
Lipase-hydrolyze fatty acids
Trypsin and chymotrypsin-degrade proteins in the meal
Nucleases: deoxyribonuclease and ribonuclease (DNAse and RNAse)-breakdown DNA and RNA.

Pancreatic enzymes are made and stored in an inactive form, and then activated after secretion by the duodenal epithelial brush border enzyme, enterokinase. Enterokinase hydrolyzes trypsinogen to its active form, trypsin; the latter molecule in turn activates other proenzymes. To prevent damage from inadvertent intrapancreatic activation of digestive enzymes, a trypsin inhibitor is also secreted by the pancreas.

### Pancreatic Digestive Enzymes

<table>
<thead>
<tr>
<th>Proteases</th>
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<tbody>
<tr>
<td>Trypsin</td>
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<tr>
<td>Chymotrypsin</td>
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<td>Carboxypeptidases</td>
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<tr>
<th>Amylolytics</th>
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<td>Amylase</td>
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<tr>
<th>Lipases</th>
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<tr>
<td>Lipase</td>
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<td>Phospholipase A2</td>
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<tr>
<th>Nucleases</th>
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<td>DNAse</td>
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<td>RNAse</td>
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**Endocrine Function:** second major function of the pancreas is to regulate blood glucose levels. Islets of Langerhans has 3 cell types:
1) alpha-glucagon
2) beta-insulin
3) delta –somatostatin

Discuss the etiology of gastroschisis

Gastroschisis (Greek for belly cleft) is a defect of the anterior abdominal wall just lateral to the umbilicus. The exact cause is unknown; however, most investigators believe that it is the result of a defect at the site where the second umbilical vein involutes. Other investigators suggest that it results from a disruption of the omphalomesenteric artery. Still others have implicated use of maternal medications (the cyclooxygenase inhibitors aspirin and ibuprofen), and environmental exposures (solvents, colorants, and x-ray exposure) as risk factors. The defect is almost always to the right of an intact umbilical cord. Unlike in omphalocele, no peritoneal sac is present, so evisceration of the bowel occurs through the defect during intrauterine life. The irritating effect of amniotic fluid (pH 7.0) on the exposed bowel wall results in a chemical form of peritonitis characterized by a thick, edematous membrane that is occasionally exudative. The exposed viscera may be congested, and the bowel appears foreshortened. Nonrotation always accompanies this condition.

In contrast to omphalocele, the incidence of associated anomalies in gastroschisis is relatively infrequent. The exception to this general observation is the occurrence of intestinal atresia, which may complicate gastroschisis in 10 to 15% of patients. Atresia of the bowel is often related to intrauterine volvulus or an interruption of the blood supply to a segment of exposed intestine by compression in an extremely tight abdominal wall defect. Intestinal necrosis, perforation, and consequent short bowel length may occasionally complicate these instances of gastroschisis. The liver is rarely eviscerated; however, the ovaries and fallopian tubes or abdominal testes are occasionally found outside the defect. The sexes are equally affected, and 40% of the patients are either premature or small for gestational age. Twenty to 25% of the patients are born to unwed teenage mothers.

LES Pressure during Swallowing

Lower Esophageal sphincter (LES) is a physiologic rather than anatomic sphincter. Normally, the LES is ≥ 2cm in length with at least 1cm or more of the LES being intraabdominal (positive surrounding pressure). Normal resting LES pressure ranges from 10 to 20 mmHg. LES pressure below 6 mmHg, short intraabdominal LES segment (<1cm), and total LES length < 2cm are associated with a defective sphincter and GERD.

When swallowing, the food bolus is propelled into the hypopharynx by positive pressure generated by the tongue, elevation of the soft palate, and contraction of the pharyngeal constrictors (UES). The pressure difference between the positive-pressure hypopharynx and the negative-pressure (-6 mmHg) thoracic esophagus along with peristalsis of the esophageal body propel the food towards the LES. The LES then relaxes so that the LES pressure equals intraabdominal pressure (6 mmHg) to allow passage of the food bolus. LES pressure then transiently increases (to approx. 40 mmHg) prior to returning to its resting pressure (10-20 mmHg). LES pressure ≥ 26 mmHg is considered to be hypertensive and is associated with esophageal dysmotility, as is incomplete relaxation of the LES (achalasia).


DECREASED CL LEVEL/INCREASED PANCREATIC EXOCRINE SECRETION

The most important stimuli for pancreatic secretion comes from three hormones secreted by the enteroendocrine system:

Cholecystokinin: This hormone is synthesized and secreted by enteroendocrine cells located in the duodenum. Its secretion is strongly stimulated by the presence of partially digested proteins and fats in the small intestine. As chyme floods into the small intestine, cholecystokinin is released into blood and binds to receptors on pancreatic acinar cells, ordering them to secrete large quantities of digestive enzymes.

Secretin: This hormone is also a product of endocrinoocytes located in the epithelium of the proximal small intestine. Secretin is secreted (!) in response to acid in the duodenum, which of course occurs when acid-laden chyme from the stomach flows through the pylorus. The predominant effect of secretin on the pancreas is to stimulate duct cells to secrete water and bicarbonate. As soon as this occurs, the enzymes secreted by the acinar cells are flushed out of the pancreas, through the pancreatic duct into the duodenum.

Gastrin: This hormone, which is very similar to cholecystokinin, is secreted in large amounts by the stomach in response to gastric distention and irritation. In addition to stimulating acid secretion by the parietal cell, gastrin stimulates pancreatic acinar cells to secrete digestive enzymes.

Additionally, the concentrations on Chloride and Bicarb are reciprocally linked according to the volume of secretion, which is approximately 2.5 liters of pancreatic juice per day.

Somatostatin Analogues

Somatostatin is released by D cells in the antrum and pancreas, as well as from the hypothalamus. Its stimulants to secretion include vagal cholinergic innervation, interneuron-mediated vagal innervation, luminal pH < 3, bombesin, CCK, gastrin, and secretin. Its acts locally to decrease both acid and gastrin release, and also inhibits GI tract absorption of nutrients and GI smooth muscle contraction.

Somatostatin exerts activity through 5 distinct receptor subtypes (SSTR1 - SSTR5). The only two commercially available somatostatin analogues, octreotide and lanreotide both have similar profiles of activity at these receptor sites. Native somatostatin has a very short plasma half-life and is therefore not useful clinically. Octreotide (Sandostatin) has a plasma half-life of 90 minutes, and 8 hours of activity after IM injection.
Lanreotide (Somatuline) has a longer half-life, approximately 7-14 days. Both products have formulations that allow for dosing every 3-4 weeks.

The effect of somatostatin analogues on portal pressure is marginal and controversial. Some studies have reported decreased hepatic pressure gradient with injection, but these effects are not consistent. Somatostatin analogues are used for treatment of bleeding esophageal varices in cirrhotic patients. A systematic review of the literature, recently performed by the Cochrane group, showed a small beneficial effect, corresponding to one unit of blood saved per patient.

References:
Cochrane Collaboration: http://www.cochranelibrary.com/abs/ab000193.htm

Alimentary Pharmacology & Therapeutics Volume 18 Issue 4 Page 375 - August 2003

Characterize T3 colon CA

The TNM (Tumor – Node Status – Metastasis) classification scheme is commonly used for staging of colon cancer. The T stage is determined by the depth of invasion.

Tx – primary tumor cannot be assessed
T0 – no evidence of primary tumor (carcinoma in situ)
T1 – invades submucosa
T2 – invades muscularis propria
T3 – invades through muscularis propria and into the subserosa or into nonperitonealized pericolic or perirectal tissues.
T4 – tumor perforates the visceral peritoneum or directly invades other organs or structures.

T3 invasion is at minimum Stage II disease, which has 62 to 76% survival. Positive nodal and/or metastasis status can bring T3 lesions to stage III or IV, which has a worse prognosis.

Staging Criteria for Colon Cancer

<table>
<thead>
<tr>
<th>Stage</th>
<th>T</th>
<th>N</th>
<th>M</th>
<th>5-year survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Tis</td>
<td>N0</td>
<td>M0</td>
<td>100%</td>
</tr>
<tr>
<td>Stage I</td>
<td>T1</td>
<td>N0</td>
<td>M0</td>
<td>80-90%</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
<td></td>
</tr>
<tr>
<td>Stage II</td>
<td>T3</td>
<td>N0</td>
<td>M0</td>
<td>62-76%</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>N0</td>
<td>M0</td>
<td></td>
</tr>
<tr>
<td>Stage III</td>
<td>Any T</td>
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<td>M0</td>
<td>30-40%</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>N3</td>
<td>M0</td>
<td></td>
</tr>
<tr>
<td>Stage IV</td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
<td>4-7%</td>
</tr>
</tbody>
</table>

Characterize enterohepatic circulation

The body thereby reduces the energy cost of bile acid production by recycling the bile salt pool within the enterohepatic circulation. [2] [13] Bile salts flow from the biliary system into the intestine, where they are efficiently absorbed and cycled from the portal system to the liver--constituting the enterohepatic circulation (Fig. 48-12) (Figure Not Available). The total bile acid pool size in humans is 2 to 5 g, and the bile acids recirculate 6 to 15 times per day, depending on dietary habits. Therefore, 0.2 to 0.5 g of bile acid are lost in the stool per day, this quantity replaced by de novo synthesis. Another way to look at this physiology is that the recirculating bile cells are 20 to 40 times more than the amount newly synthesized, underscoring the importance of this circulation. Serum bile acid levels are normally low (approximately 5 mumol) because of the remarkable 95% extraction efficiency of the liver. The liver can remove 80% of the bile acids in a single pass. The liver's roles in the enterohepatic circulation include synthesis, extraction, and secretion of bile acids. The active secretion of bile acids across the canalicular membrane can be considered the primary metabolic pump of the enterohepatic circulation. Newly synthesized bile acids are considered primary bile acids. Bacteria in the intestine produce dehydroxylation, oxidation, isomerization, or deconjugation and the formation of secondary bile acids. Secondary bile acids are more hydrophobic than primary bile acids and promote biliary stasis and gallstone formation. Essentially all primary and secondary bile acids are conjugated with the amino acids glycine and taurine. Conjugation with the amino acids increases the pKa and renders the molecule more neutrally charged, allowing for rapid diffusion into enterocytes. There, the bile acids associate with proteins [15] and enter the splanchnic blood, returning to the liver. Signals from returning bile acids inhibit new bile acid production. Efficient intestinal reabsorption and hepatic extraction of bile acids permit a very effective recycling and conservation mechanism that largely restricts bile acids to the intestinal and hepatobiliary compartments. The anatomic components of the enterohepatic circulation are the liver, biliary tract, intestine, portal venous circulation, and, to a lesser extent, the colon, systemic circulation, and kidney (Fig. 54-3). At a fundamental level, the enterohepatic circulation of bile acids can be thought to consist of a series of storage chambers (gallbladder, small intestine), valves (sphincter of Oddi, ileocecal valve), mechanical pumps (canaliculi, biliary tract, small intestine), and chemical pumps (hepatocyte, cholangiocyte, and ileocyte).

Sources:
Townsend: Sabiston Textbook of Surgery, 16th ed., Copyright © 2001 W. B. Saunders Company pgs.1008-1009
Discuss the diagnosis biliary dyskinesia


Biliary dyskinesia is a motility disorder that affects the gallbladder and sphincter of Oddi. The motility disorder of the gallbladder is called gallbladder dyskinesia. Patients with this condition present with biliary-type pain, and investigations show no evidence of gallstones in the gallbladder. Performing a gallbladder ejection fraction, which is a radionuclide investigation, makes the diagnosis. An abnormal gallbladder ejection fraction has a value less than 40%. Patients with an abnormal gallbladder ejection fraction should undergo cholecystectomy. This procedure has been shown to be effective in curing the symptoms in over 90% of patients.

Motility disorder of the sphincter of Oddi is called sphincter of Oddi dysfunction. This disorder is categorized as two distinct types-biliary sphincter of Oddi dysfunction and pancreatic sphincter of Oddi dysfunction. Typically, patients with biliary sphincter of Oddi dysfunction present with biliary-type pain on average 4 to 5 years after having undergone cholecystectomy. Sphincter of Oddi manometry is essential in making a diagnosis of abnormal motility of the sphincter. On manometry, diagnosis of a sphincter of Oddi stenosis should lead to division of the sphincter. Sphincterotomy results in long-term relief of symptoms in more than 80% of patients. Pancreatic sphincter of Oddi dysfunction clinically presents with recurrent episodes of pancreatitis of unknown cause. Having ruled out all of the common causes of pancreatitis, sphincter of Oddi manometry of the pancreatic duct sphincter should be performed. When manometric stenosis is diagnosed, these patients should undergo division of both the biliary and pancreatic duct sphincter. This treatment results in relief of symptoms in more than 80% of patients.

From Current Surgical Therapy, 7th Edition, Cameron. 2001

Biliary dyskinesia can also be a part of the postcholecystectomy syndrome of biliary type abdominal pain after cholecystectomy. Motor abnormalities of the sphincter of Oddi may produce disturbances in biliary function that may cause pain. There are four patterns of abnormal pressures: increased basal pressure, paradoxic response to CCK, retrograde propagation of phasic-wave contractions, and increased frequency of phasic-wave contractions. Treatment with sphincterotomy results in 91% of patients improved vs. 45% in patients undergoing sham sphincterotomy.
ARTERIAL SUPPLY TO PANCREAS

Head of pancreas
- from the anterior and posterior pancreaticoduodenal arteries
  1. superior pancreaticoduodenal arteries
     - arise from celiac a. to common hepatic a. to gastroduodenal a. then to anterior/posterior branches
  2. inferior pancreaticoduodenal arteries
     - arise from superior mesenteric a.

Body and tail of pancreas
- distribution more variable
  1. dorsal pancreatic artery
     - arise from splenic a.
     - right branch supplies the head and usually joins the posterior arcade.
     - 1 or 2 left branches often connects with splenic a. (or its branches) or left gastroepiploic a.
  2. transverse pancreatic a.
     - arise from left gastroepiplic a.
     - gives off superior and inferior pancreatic arteries

Venous drainage
- all ends in portal vein, which arises posterior to the neck by union of the splenic and superior mesenteric veins
- because of the anatomic relationship with splenic vein, inflammatory/neoplastic diseases involving the body and tail can lead to splenic vein occlusion, which will result in retrograde venous drainage toward the splenic hilum and then, by way of flow through the short gastric and left gastroepiploic veins, create gastric varices.

Feldman: Sleisenger & Fordtran's Gastrointestinal and Liver Disease, 7th ed., Copyright © 2002 Elsevier
Townsend: Sabiston Textbook of Surgery, 16th ed., Copyright © 2001 W. B. Saunders Company
Characterize bile salts

Bile salts are conjugated bile acids. Bile acids account for 50% of the organic components of bile and are synthesized in the liver from cholesterol and contain a steroid nucleus with a branches side chain of 3 to 9 carbon atoms ending in a carboxyl group (carboxylic acids). Four different bile acids are present in bile. The liver synthesizes two bile acids: cholic and chenodeoxycholic acid (primary bile acids). In the lumen of the gut each can be dehydroxylated by bacteria to form deoxycholic and lithocholic acids (secondary bile acids). All four are returned to the liver in the portal blood and secreted into bile. The relative amounts of each in bile are approximately 4 cholic to 2 chenodeoxycholic to 1 deoxycholic to small amounts of lithocholic. The liver conjugates these bile acids to the amino acids glycine or taurine and makes bile salts in order to make the molecules more water soluble in the relatively neutral pH of the intestinal contents.

Bile salts are unique in that they are amphipathic molecules (have hydrophilic and hydrophobic portions). This allows them to arrange themselves in droplets of lipid in the lumen of the duodenum. Above a certain concentration (the critical micellar concentration) bile salts for aggregates called micelles which can hold water insoluble molecules and “solubulize” them. In this way bile salts enable fat digestion and absorption.

References: