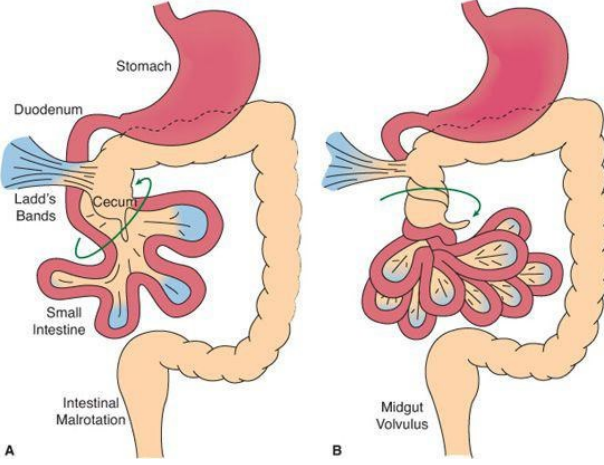
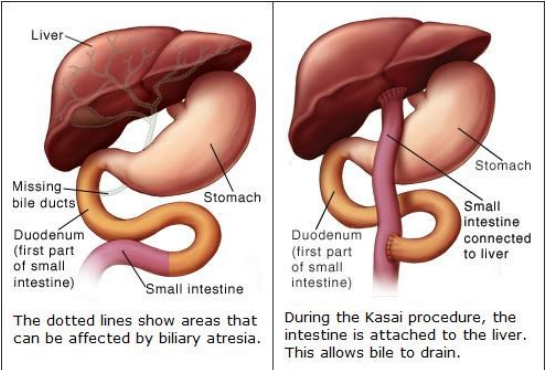


Newborn GI	
Pyloric Stenosis	
Pathophys	Hypertrophy of pylorus. RFs = bottle feeding, maternal smoking
Presentation	Immediate post-prandial projectile vomiting , "hungry vomiter," palpable olive-like mass. Classically presents in 3-6w infants, but can worsen by 2-3 months (rare by 12w), 4:1 male:female
Workup	BMP (hyperchloremic metabolic alkalosis), CBC (should be nml), bili (unconjugated hyperbili), hemoccult stool (should be neg), abdominal ultrasound
Treatment	<ol style="list-style-type: none"> 1. Address dehydration and correct alkalosis 2. Surgical consult for pyloromyotomy (definitive treatment) 3. Post-op refeeding can start within hours
Malrotation/Volvulus	
Pathophys	<ul style="list-style-type: none"> • Malro: arrest in normal rotation in embryonic gut. Misplaced cecum is attached by peritoneal bands (Ladd bands) which cross the duodenum, leading to risk of volvulus. Mostly asymptomatic. • Volvulus: Small bowel twisting around SMA → vascular compromise, ischemia, necrosis. 
Presentation	Bilious vomiting, third spacing, HD instability
Workup	<ul style="list-style-type: none"> • Bilious vomiting + signs of sepsis/hemodynamic compromise + suspicion of volvulus → rapid resuscitation and surgical exploration • If HD stable → KUB, upper GI series (corkscrew appearance), U/S (whirlpool sign), CT in adults. Laproscopy if indeterminate.
Treatment	<ul style="list-style-type: none"> • Ladd procedure: division of Ladd bands, widening mesenteric base, explore duodenum with tube for patency, appendectomy (to avoid future confusion w/abd pain), bowel resection as needed, placement of bowels in nonrotation. • Post-op, address short gut syndrome if relevant
Biliary Atresia	
Pathophys	<ul style="list-style-type: none"> • Grouped into 3 categories • The most common type (70-85%) is perinatal and involves a progressive fibro-proliferative obliteration of the bile ducts → destruction of the extrahepatic biliary tract → direct hyperbili, cirrhosis, liver failure. Etiology unknown. • 2nd type of BA ("Biliary Atresia Splenic Malformation") is associated with laterality malformations - situs inversus, asplenia/ polysplenia, malrotation, interrupted IVC, cardiac anomalies. • 3rd type is associated with other congenital anomalies- intestinal atresia, imperforate anus, kidney and cardiac anomalies.

Biliary Atresia continued on next page →

Newborn GI	
Biliary Atresia	
Presentation	Jaundice, acholic stools, hepatomegaly
Workup	Bilirubin (conjugated hyperbili), liver enzymes(transaminitis, elevated GGTP), abd u/s (inability to visualize gallbladder or small gallbladder), HIDA scan (looks for excretion of bile from liver), liver biopsy, intraoperative cholangiogram
Treatment	<ul style="list-style-type: none"> • 100% mortality by age 2 if untreated. • Kasai procedure (hepatoportoenterostomy) - (best if done before 2 months. Removal of portal tract remnant followed by Roux-en-Y anastomosis of jejunal loop directly to liver capsule to allow bile drainage. • 60-70% of patients undergoing Kasai will eventually need liver transplant <div>  </div>