**Cholestasis and Biliary Colic**

**History of Presenting Illness:**
- **YELLOW EYES**
- **DARK URINE**
- **YELLOW SKIN**
- **ITCHY ALL OVER**
- **PALE STOOLS**
- **FAT MALABSORPTION**
- **High Cholesterol**
- **Xanthomae**
- **Nausea, Anorexia, Vomiting**

**PAIN:**
- **RIGHT UPPER QUADRANT**
- **Radiating to the back**
- **Severe + Constant**
- **Dull, “boring” pain**
- **Pleuritic-sounding**
- **Worst with fatty foods**
- **Onset in 1-2 hrs after meals**
- **Lasting 1 to 6 hours per episode:**

**RULES OF THUMB:**
- The eyes are the **FIRST** thing to go yellow.
- Bilirubin over 30 = **yellow eyes**
- Bilirubin over 50 = **yellow skin**
- The severity of itching does not correlate well with the bilirubin or bile salt levels.
- **The elderly will itch more.**

**EXAMINATION**

Try to find something to support a gallbladder source for this pain:

**MURPHY’S SIGN:** patient inspires while you have your hand deep in their RUQ. This causes the diseased gall bladder to ride into your fingers as the liver slips downwards. If there is any gall bladder inflammation, it will cause a sudden stop to the inspiration, due to extreme pain.

**CORVOISIER’S LAW** states that **IF YOU CAN FEEL THEIR GALL BLADDER AND THERE IS JAUNDICE, then the patient is NOT JAUNDED BECAUSE OF STONES.** Basically this means they have a cancer in the biliary tree. Why? A fibrotic gall bladder, chronically ridden with stones, is not able to distend to a large enough size for you to feel it. If you can feel it, its probably a soft non-fibrosed gall bladder, dilated because a tumour is obstructing the outflow.

**STIGMA OF CHRONIC LIVER DISEASE** will probably be absent but if there is anything to suggest chronicity, ask yourself: is it due to the chronic bile outflow obstruction, or is there some other pathology which is causing it?

**DIFFERENTIALS:** why could they be yellow...

- **Biliary colic** (small stone)
- **Choledocholithiasis** (big stone)
- **Cholecystitis**
- **Cholangitis**
- **… and Pancreatitis (?)**
- **… and SEPSIS??**

- **Cancer of the Bile Duct, Gall Bladder or Head of Pancreas**
- **Recent transfusion** can make your eyes go yellow (blood in those bags is very old)
- **Chronic or acute liver disease** can cause this sort of presentation
- **Surgery** (prolonged bed rest, TPN, gall bladder hypomotility, thus bile stasis, etc)
- **Are drugs responsible** (eg. asymptomatic anaesthesia jaundice, fluoxacillin hepatitis)
- “**Gut claudication**” can cause transient pain right after meals (vascular disease in the gut results in exercise-induced lactic acidosis and thus PAIN)
INVESTIGATIONS

The USUALS:
- **FBC** – maybe leukocytosis, largely neutrophilic. Maybe nothing.
- **EUC** – maybe hypokalemic, if patient has been vomiting. Probably nothing.
- **LFT** – Alk Phos and GGT – will be elevated: it’s the classical “obstructive pattern”
  - Bilirubin will also be up if they are jaundiced
- **URINE DIPSTICK** – will probably do nothing for you except exclude hematuria as the cause of the dark urine.
- **URINALYSIS** bilirubin and urobilinogen will be present if the jaundice is due to poor bile flow (i.e. conjugated bilirubin is in the blood stream)

The SPECIALS:
- **AMYLASE + LIPASE** may be elevated if the pancreatic duct is also blocked (i.e. the stone is at Ampulla of Vater level)
  - Also important because a head of pancreas cancer is one of the differentials for obstructive jaundice.
- **BLOOD CULTURES**
  - Especially if the patient is febrile and acutely unwell; must rule out sepsis of an ascending biliary origin
  - Prothrombin Time (PT)
    - May be elevated, as you start losing your synthetic liver functions when there is a back-log of bile, and the fat-soluble vitamins aren’t getting absorbed.
- **Fat-Soluble Vitamin Levels:** if your bile is not making it out of the duct, you are malabsorbing fat and everything that comes along with fat.
  - Can test for vitamins A, D, E, and K if the problem is long-standing

IMAGING to CONFIRM your DIAGNOSIS

**UPPER ABDO ULTRASOUND:**
- Ultrasoundography provides greater than 95% sensitivity and specificity for the diagnosis of gallstones more than 2 mm in diameter. Ultrasonography is 90-95% sensitive for cholecystitis and is 78-80% specific. Studies indicate that emergency physicians require minimal training in order to use right upper quadrant ultrasonography in their practice.

**Endoscopic Retrograde Cholangio-Pancreatography (ERCP)**
- allows visualization of the anatomy and may be therapeutic by removing stones from the common bile duct. In a major teaching hospital, with two on-call gastroenterologists, this is the diagnostic and management measure of choice after a small stone has been visualised with ultrasound.

**Magnetic Resonance Cholangio-Pancreatography**
- Non-invasive, but therefore also not a management option.
- Findings suggestive of cholecystitis include:
  - wall thickening (>4 mm),
  - pericholecystic fluid,
  - subserosal edema (in the absence of ascites),
  - intramural gas (gas gangrene of the gall bladder),
  - sloughed mucosa.

**Abdo X-ray** will only pick up ~10% of the stones. Sometimes you may see:
- A calcified “porcelain” Gall Bladder
- An obvious huge radio-opaque stone
- Free air in the biliary tree and gall bladder wall: the “emphysematous” gall bladder, like gas gangrene: *E.Coli* and *Clostridium*. Commonly seen in acalculous cholecystitis.
- **SINISTER SIGN:** Free air under the diaphragm; means its perforated!!
  - *Unspeakable Nightmare*

**What if the imaging shows no stones?**
- About 5 to 10% of acute cholecystitis is acalculous
  - The Gall Bladder is distended, there’s fluid around and in its walls, the patient is febrile BUT NO STONE TO BE SEEN.
  - What’s happened? BILE STASIS due to some critical illness
  - Prolonged TPN feeding
  - Dehydration
  - Heart failure
- All these lead to hypomotility, increasing bile viscosity, microscopic stone formation and infiltration with gut flora, superimposed on a possibly ischaemic gall-bladder which makes an excellent host for bugs.
- **MORTALITY OF ACALCULOUS CHOLECYSTITIS IS FAR GREATER THAN OF THE CALCULOUS TYPE:**
  - Already the patient is very ill, PLUS this is the sort of cholecystitis which can erode the wall quickly

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**Speaking of LFTs...**

### LIVER DEATH ENZYMES (transaminases)
- Indicate liver parenchyma is involved.
- **LOOK AT WHICH IS THE HIGHEST!**

#### AST = everywhere
- Aspartate aminotransferase
- = when AST is the highest, its **ALCOHOLIC LIVER DISEASE**

#### ALT = LIVER ONLY
- Alanine aminotransferase
- = when ALT is the highest, its **VIRAL HEPATITIS**
- also glandular fever,

#### AP = everywhere (bone, kidney, intestines)
- Alkaline Phosphatase
- **THE MARKER OF POOR BILE FLOW**
- You may justifiably ask for AP fractionation (and this will tell you if it came from bone or liver).

#### GGT = LIVER ONLY;
- Gamma Glutamyl Transpeptidase
- Gets elevated in practically all types of liver disease

### CHOLESTASIS ENZYMES
- Indicate that the bile duct cells are involved.
- Will elevate to ~10x the normal in cholestasis.

### LDH = in every tissue
- Increased levels are found in myocardial infarction, liver disease, haemolysis, ineffective erythropoiesis, some malignancies (esp non-Hodgkin’s lymphoma), muscle disease etc...

### BILIRUBIN: is there too much of it or is it not being disposed of?
- i.e **HAEMOLYSIS** or LIVER DISEASE / CHOLESTASIS
- Bilirubin, GGT and AP = cholestasis
- Levels greater than 3 mg/dL are usually noticeable as jaundice.
- Because only conjugated bilirubin appears in urine, the finding of bilirubinuria also implies liver disease.
- **THUS:**
  - **ELEVATED CONJUGATED** = look for liver disorder
  - in absence of liver disease, its got to be a weird congenital defect
  - eg. Dubin-Johnson syndrome
  - **ELEVATED UNCONJUGATED** = look at the blood smear
  - Normal everything? Its probably ANOTHER weird syndrome, probably Gilbert’s Syndrome which causes failure of hepatic bilirubin uptake

### ALBUMIN:
- A true indicator of liver function: a gauge of its failure, a herald of impending complications.
- **The half-life of serum albumin normally is 19–21 days,**
- Thus **shows up CHRONIC PROBLEMS**
- Albumin levels may be diminished due to poor nutritional status, severe illness with protein catabolism, nephrosis, and malabsorption

- **Prothrombin Time:** vitamin K factors (2, 7, 9, 10)
- May be diminished due to malabsorption
- Half life of factors is 1-2 days,
- Thus **show up ACUTE PROBLEMS**

### WEIRDO TESTS
- **Ceruloplasmin** for Wilson’s disease (presents as psychiatric problem)
- **Blood Ammonia tests** for hepatic encephalopathy (but EEG is diagnostic!)
- **Alpha-Fetoprotein** is a sensitive marker for hepatocellular carcinoma
- **Alpha-1 Antytripsin** deficiency of which causes hepatitis and cirrhosis
- **Anti-mitochondrial Antibody:** if you suspect PRIMARY BILIARY CIRRHOSIS
  - (only other elevated enzyme = AP)
In elective laparoscopic cholecystectomy, the rate of conversion from a laparoscopic procedure to an open surgical procedure is approximately 5%. The conversion rate for emergency cholecystectomy where perforation or gangrene is present may be as high as 30%.

**MANAGEMENT: SYMPTOMATIC (at the emergency department)**
- Place patient on **Nil-by-mouth** (They may be having surgery soon)
- **Rehydrate** intravenously (patient may have been not eating and vomiting)
- Control itching with **opiates**! They will also help the RUQ pain.
  - Swine may tell you that morphine increases sphincter of Oddi spasm, and that therefore you should avoid it in this setting. Practically speaking, often the NSAIDs these swine would give do nothing for the patient’s pain, and one may justifiably ignore their feeble complaints and opt in favour of more powerful and thus humanitarian opiate analgesia. Go Mighty Opium!
- Control nausea + vomiting with metoclopramide or ondansetron
- IF FEBRILE, OR YOURE CERTAIN ITS CHOLECYSTITIS OR CHOLANGITIS:
  - Give **Triple Antibiotic Therapy**, favoured by the GI surgeons

| Ampicillin, Gentamicin, Metronidazole |

**MANAGEMENT: DEFINITIVE; …meaning surgical.**

**ACUTE BILIARY COLIC:**
Analgesia is all that is required in most cases. Uncomplicated colic resolves by itself without surgical intervention. Repeated rapidly recurring episodes herald the onset of complications (eg. cholecystitis). **BOOK an OUT-PATIENT ELECTIVE ERCP for later…**
→ ERCP: if there is a common bile duct stone, you should go after it. Otherwise, **Wait 6 hours; let it develop into**

**ACUTE CHOLECYSTITIS:**
Admit the pt.;

Now that your patient has a fever and physical signs, the surgeons will be much more interested. They may be persuaded to perform a

- **LAPAROSCOPIC CHOLECYSTECTOMY** which may upgrade to **OPEN CHOLECYSTECTOMY** if there is empyema, gangrene, abscess etc.

Either way: the gall bladder is now uselessly damaged and needs to come out.
If the patient is particularly elderly and feeble, you may want to merely decompress the gall bladder with a **percutaneous draining tube** which is less dangerous to install

**CONTROVERSY: do you wait for complications?**
Studies show that patients who have elective cholecystectomy after their episode of colic have shorter hospital stay and a less problematic recovery than those who patiently wait for a stone to obstruct their common bile duct.

**ACUTE ACALCULOUS CHOLECYSTITIS**
The patient is in great danger, **MUST OPERATE:**
This is an emergency open cholecystectomy, with drainage and debridement of any gangrenous tissue and debris.

**CHOLANGITIS:** obstruction of the common bile duct has favoured its colonisation with gut organisms, and now they creep up the hepatic biliary tree, soon to break into the hepatic parenchyma and subsequently into your bloodstream.

**INFECTION LOCALISED TO BILIARY TREE:**
- Endoscopic Decompression + Drainage of bile duct
- Broad-spectrum Antibiotics, INTRAVENOUSLY

**INFECTION SPREAD: SEPTIC, LIVER ABSCESSSES, etc**
- Open Cholecystectomy + Debridement
- Broad-spectrum Antibiotics, INTRAVENOUSLY

**PROGNOSIS**
Asymptomatic individuals with gallstones develop pain at a rate of 1% per year (i.e. 10% chance in 10 years)
The frequency of progression to acute cholecystitis is 10-30%.
Most patients with acute cholecystitis have a complete remission within 1-4 days. However, 25-30% of patients either require surgery or develop some complication
Cholangitis mortality ranges from 7-40%.

**EPIDEMIOLOGY**
Rare in the under-20s
Prevalence increases with age
Women more than men
More common with obesogenic Western McDiet
Acalculous cholecystitis is more common in elderly men.
The gallbladder is a distensible sac and, when not distended, its mucosa is thrown into many folds. The lumen of the gallbladder is lined with a high columnar epithelium. The connective tissue wall contains abundant elastic fibers and layers of smooth muscle which predominantly run obliquely. The whole columnar lining is very uniform and rests on a highly vascular basement membrane. Its duty is to absorb inorganic salts and water, and these get carried off by the veins in the gallbladder wall → they go back to the liver.

**Major Function:**

STORE and CONCENTRATE the BILE
And release it at the behest of Cholecystokinin the Gut Hormone

**Arterial Supply:** Cystic Artery (branch of the Right Hepatic Artery)

**Venous Drainage:** Cystic Veins (go directly from GB wall to hepatic sinusoids)

**Sensory innervation:** Right Phrenic Nerve
BASIC SCIENCES: CHOLESTEROL & BILE

The HEPATOCYTE

25% of cholesterol comes from DE-NOVO BIOSYNTHESIS:
10% in the hepatocytes, 15% in the small intestine.

CHOLESTEROL

BILE ACID SYNTHESIS: this is how you rid yourself of excess cholesterol

CHOLESTEROL

7-alpha-hydroxylase

7-hydroxycholesterol

Numerous complicated steps which we don’t need to know about

Cholic Acid

Chenodeoxycholic Acid

Taurocholic Acid

Glycocholic acid

Glychenodeoxycholic Acid

Glycochenodeoxycholic Acid

SECRETION OF BILE ACIDS: (very soluble, up to $10^{-3}$ mmol/L)- via the bile salt export pump (BSEP)
the conjugate export pump (MRP2) - also exports conjugated Bilirubin
the multidrug export pump (MDR1) - for hydrophobic cationic compounds

Biliary Canaliculus:
Where the organic components of the bile will meet and mingle.
 bile acids (80%) phospholipids (16%), unesterified cholesterol (4.0%).

GALL BLADDER: Bile maturation
The Gall Bladder epithelium actively pumps out much of the Na+, K+, Cl-, HCO3- and therefore also WATER.
BILE CONCENTRATED 10 to 15 times RELEASE STIMULATED by cholecystokinin (when fat hits the gastric mucosa) and by secretin (when the duodenum becomes acidic)

Gut bacteria convert the primary bile acids into the secondary bile acids, DEOXYCHOLATE and LITHOCHOLATE

Un-esterified raw CHOLESTEROL: Exit via two hemitransporters ABCG5 and ABCG8

So how do these three behave in aqueous solution?
- Cholesterol forms cholesterol monohydrate crystals in water.
- Lecithin forms bi-layered micelles, like oil droplets
- Bile salts will form micro-micelles (only a few molecules)

BUT: together they will form small micelles half-lecithin, half-bile salt; and within these the cholesterol can be dissolved as if in fat (though in reality the bile here is very watery). !! IMPORTANT !!
80% are cholesterol stones; 20% are bile pigment stones composed of calcium bilirubinate...pigment stones only form when there is too much bilirubin being formed. Eg. when there is excess erythrocyte destruction. Plus normally bilirubin is conjugated and soluble. **THUS:** pigment stones will only form when:
1. There is too much bilirubin
2. Some bacterium crawls up the common duct and deconjugates it, causing it to precipitate from the solution.

**SUPER-SATURATED BILE:** why??
- Increased biliary cholesterol secretion
  - **AGE**: you lose some of your 7-alpha-hydroxylase, the rate-limiting step in making bile acids out of cholesterol. Thus you excrete more cholesterol and less bile acids, and the CSI rises to over 1.0
  - **OBESITY**: there is a linear relationship between weight and biliary cholesterol secretion. This may be due to increased de-novo synthesis.
  - **FEMALINESS**: estrogen causes more cholesterol to be excreted into bile, partly by increasing hepatic lipoprotein uptake. It’s the downside of estrogen’s lipid-lowering cardiovascular protection.
  - **McDiet**: vegetarians don’t get gall stones. Fat carnivores always do.
  - **GENETICS**: stupidly, the liver of individuals so predisposed does not divert more cholesterol into bile acid synthesis. Instead it allows it all to just leak out into the biliary canaliculus. Seems to be some sort of negative feedback loop dysfunction, dominant trait involving at least 2 genes.

**GALL BLADDER MOTILITY DYSFUNCTION: Why??**
- Exposure to bile
  - Reduced emptying; **GALL BLADDER HYPOMOTILITY**
  - Prolonged **FASTING** and **PREGNANCY** also lead to gall bladder hypomotility
- **Penetration of bile into gall bladder wall**
  - Smooth muscle contractility impaired: poisonous bile!
  - And cholesterol is the culprit; it does something to the smooth muscle cells

!!! **THIS IS AN INFLAMMATORY PROCESS!!!** the bile-soaked epithelium goes overboard with the production of mucous proteins.

**THUS → MORE PROTEIN IN THE BILE**
Plus, IgG secreted from the biliary canaliculi epithelium also contributes.

**PAIN** referring to back + shoulder blades
**PAIN** localising to Murphy’s point

**JAUNDICE**

**INFECTION** ascending the stagnant bile duct

**FEVER**

Penetration of liver **SEPSIS**
ACUTE ABDOMEN: PANCREATITIS

SYMPTOMS:
- Pain which is eventually VERY SEVERE
- Epigastric or LUQ
- MUCH WORSE AFTER EATING!!
- Better by leaning forward
- May be nauseous, light-headed, anaemic

SIGNS:
- EXQUISITE EPIGASTRIC TENDERNESS
- Possibly positive rebound
- Signs of SHOCK!!

PROGNOSTIC FACTORS
Early multiorgan failure from shock = 50% mortality
Mild disease = <5% mortality
Late infected necrosis = WILL KILL YOU
If you're over 55, with WCC over 16 and rising LFTs, your prognosis is very poor

PATHOPHYSIOLOGY OF SHOCK FROM PANCREATITIS

IMMEDIATE MANAGEMENT:
Goal is to arrest progression before the development of systemic symptoms:
- Na+, Cl-, K+ and H+ are lost through vomiting: NEED TO REPLACE
  - Thus give oxygen, normal saline, analgesia.
  - Put in a central line
  - Urinary catheter to watch output: if acutely dropping, WORRY!
  - Replace fluids

RUN TESTS:
- FBC
- LFTs
- EUC + CMP (watch potassium and calcium)
- AMYLASE / LIPASE:
  - not specific or sensitive; 20% of cases have normal results
  - UNLESS: good pancreatitis history and levels elevated to over 1000!!
  - LIPASE MAY BE MORE SENSITIVE...

SURGICAL MANAGEMENT:
NECROSECTOMY: scoop out the abscess, if...
- Clinical deterioration, AND
- Bacteriological proof of infection
High intraoperative mortality (~40%), due to Hemorrhage of splenic artery
  (the one that runs almost through the pancreas)
  = caused by inflammation, which weakens the vessel wall and produces a PSEUDOANEURYSM which often bursts in the surgeon's hands.

90% caused by alcohol and gallstones
the other 10%:
- Hypercalcemia
- Drugs:
  - Sodium valproate
  - Salicylates
  - ACE inhibitors
  - Azathioprine
- Tumour
- Mumps or coxsackie virus
- Vascular anomaly
- ERCP complication
- Scorpion bite

GALLSTONE PANCREATITIS
Have to do ERCP within 48 hrs; STILL PROGRESSING? →
do a contrast CT; may have become necrotic
COMPLICATIONS:
- Necrosis and infection in the remains of the pancreas
- Fluid at the operating site → increased intrabdominal pressure → abdo compartment syndrome
- Colonic necrosis, inflammation and subsequent colonic artery thrombosis
- GI haemorrhage
- Respiratory failure
- Renal failure:
  - PRE due to hypovolemia
  - INTRA due to ischaemic tubular necrosis
  - POST due to pressure obstruction by abdominal compartment syndrome
- Hyperglycaemia (effectively, diabettis mellitus type 1)
- Hypocalcemia

PSEUDOCYST: (fake cyst - wall is not lined with epithelium)
Not cyst- instead a pocket formed by fibrin sheaths and adjacent organ walls; filled with necrotic filth.
- 35% of cases will go on to develop pseudocysts after pancreatitis
- Takes about a month to develop.
- 50% resolve in 3 months

SYMPTOMS:
pain (radiates to back) + gastric outlet obstruction

SIGNS: epigastric tenderness
Investigate with ultrasound and CT scan.
Management: drain it before it becomes infected!
  - Endoscopy or US-guided

Cancers of the Bile Duct, Gall Bladder and Liver Tissue
- Most will be secondaries. Even if you can’t find the primary, it’s probably still secondaries.
  Commonest mets are from colo-rectal, lung, breast, pancreas, and stomach.
  IN ABSENCE OF CIRRHOSIS, HEPATITIS or HAEMOCHROMATOSIS, PRIMARY LIVER CANCER IS ALMOST UNHEARD OF. Likewise cancer of the gallbladder, biliary tree and common bile duct. Rare as hens teeth, they are. But…
  You have to start thinking along the CANCER lines if your patient has
  - PAINLESS INSIDIOUS JAUNDICE
  - ACALCULOUS CHOLECYSTITIS
  - WEIGHT LOSS
  - FAT MALABSORPTION
  - Employ Courvoisier’s Law: if the gall bladder is distended in painless jaundice, there must be a tumour constricting the common bile duct
  - These people will have a raised PT due to Vit. K malabsorption
  - Bilirubin, Alk Phos and GGT will be elevated.

Gall Bladder Cancer:
associated with gallstone disease, estrogens, cigarette smoking, alcohol consumption, obesity, and female sex. The epicenter of the tumor usually is the fundus, or neck, of the gallbladder. Local spread through the organ wall leads to direct liver invasion, or, if in the opposite direction, leads to transperitoneal spread (20% of patients at presentation).

Cholangiocarcinoma:
More than 90% are adenocarcinomas, and the remainder are squamous cell tumors. Arise from the intrahepatic or extrahepatic biliary epithelium

Complete surgical resection is the only therapy to assure a chance of cure.
Unfortunately, only 10% of patients present with early stage disease and are considered for curative resection.

Barrier Key-word for Hepatocellular carcinoma: Alpha-Foeto-Protein
**The Acutely Painful Abdomen: Where does it hurt?**

**Rules of thumb:**
- Midline structure pain will radiate to the back
- If the viscera are inflamed, pain is diffuse
- If the abdominal wall is inflamed, the pain is localised to a discrete area

**Characteristic locations:**

* oesophagus pain mimics cardiac pain – same referral

**Obstruction**
Most often caused by post-operative adhesion (may take years)
- Acute constipation, distension, nausea + vomiting, pain
- If constipation + distension is long-standing and progressive, consider partial narrowing or chronic process
- ASK how often the pain pulses are felt

**ASK ABOUT:**
- Previous episodes of obstruction
- Previous abdo / pelvic operations
- History of abdo cancer
- History of abdominal inflammatory disease: Eg.
  - Inflammatory bowel disease
  - Cholecystitis
  - Pancreatitis
  - Pelvic inflammatory disease
  - Abdominal trauma

**EXAMINATION:**
- How does the patient look?
  - If they are lying quite still, it looks like peritonism (bad sign !)
- What was aspirated through the NG tube?
  - CLEAR +/- food = gastric outlet obstruction
  - FECULENT = distal small bowel …or colonic obstruction with incompetent iliocaecal valve
  - BILIOUS but NON-FECULENT = either
    - A medial or proximal small bowel obstruction, OR
    - a colonic obstruction with a competent ileocaecal valve (or else the faeces would be regurgitating into the stomach out of the incompetent iliocaecal valve)
- PUT YOUR FINGER IN IT!! ➔ hematomae / abscesses get forgotten
- ABSENT BOWEL SOUNDS? – sinister; maybe ileus
- PICTURE OF ILEUS: mild diffuse pain, not the severe increasing localised pain of obstruction

**APPENDIX PAIN:**
Embarrassing to miss this diagnosis, its bread-and-butter stuff.
- Diffuse and Periumbilical pain at first;
- Moved to Right Iliac Fossa
- Now sharper
- The patient can not hop on their right leg ( psoas contraction pushes the appendix into the abdominal wall, causing a lot of pain)

Obstructive symptoms which come and go suddenly for several days in an elderly patient (over 65) should make you suspicious of a GALLSTONE ILEUS

**Diagnosis:**
- Proximal obstruction = pain pulses every 3-4 minutes;
- Distal obstruction = Pain pulses every 10-15 minutes

**illustration:**
- Duodenum, head of pancreas
- Gallbladder
- Liver
- Appendix
- Caecum and ascending colon
- Stomach
- Spleen
- Small Intestine
- Sigmoid colon
- Kidney and ureter

**Imitation of the Diaphragm, THUS**
- Liver
- Gallbladder
- Duodenum

**Colic is distension of a hollow viscus.**
INVESTIGATIONS for bowel obstruction:

**Bloods:**
- **EUC** (Hypokalemic? Hyponatremic? Hypochloremic?)
- **FBC** for Hb and white cells
- **LFT** for cholestatic issues or portal HT
- **Amylase + Lipase** for pancreatitis

**Abdo Xray**
- May see characteristic water vs. gas levels in distended loops of small or large bowel.
- ? is there gas distal to the obstruction (if yes, then it is only a partial obstruction)
- So there’s no gas BELOW the obstructed section of colon; BUT: is there gas in the small bowel?
- Is it regurgitating into the small bowel from the blocked colon?
- **Barium enema** for bird-beak sign: demonstrates sigmoid volvulus
- **CT with oral / rectal contrast** for apple-core sign: demonstrates colonic carcinoma

**Management**

**Resuscitation**
- **IV fluids** (crystalloids, NS with K+ is good)
- **Urine output should be at least 0.5 ml/hr** (i.e. halve the patient’s weight and expect it in mls of urine per hour)
- **Monitor fluid response**: within 10-15 minutes the urine output should change
- **The need for surgery must be assessed**: most of these things resolve on their own, but if the bowel wall is so distended that its ischaemic, then there is risk of fecal peritonitis from which there is a 50% chance of death – so don’t let it get that far.
- **!! NEVER LET THE SUN GO DOWN TWICE ON A BOWEL OBSTRUCTION!!**
Induced how: WITH INTRAVENOUS DRUGS:
WHICH ARE RAPIDLY ACTING AND WILL PUT YOU OUT VERY QUICKLY.
BUT the IV drugs will only last a short while, as their circulating volume will decrease (with them being taken up into the tissue and metabolised)

**PROPOFOL, the Milky Intravenous Beverage of Blissful Absence**
Its lipid soluble, so it comes in an opaque white emulsion. Nobody knows exactly what it does, but it does it within 30 seconds; and within 5 minutes you’re awake again.

MAINTAINED HOW?
WITH GAS.
The gas acts slowly (and smells bad) and therefore is useless for inducing the unconsciousness. However, it works well as maintenance. Mix with O2 for maximum effect. Serve chilled.

**ANAESTHETIC GASES:** isofluorane, sevofluorane, enflurane, desflurane.
Once again, nobody knows how they do what they do. Strangely, the noble gas Xenon has excellent anaesthetic properties, but its shamelessly expensive and the only people to use it routinely are Russians (who have cubic miles of it left over after their Cold war uranium enrichment program). Of course, there is the much maligned 'Critical Volume Hypothesis'. It states that the absorption of anaesthetic molecules could expand the volume of a hydrophobic region within the cell membrane and subsequently distort channels necessary for sodium ion flux and the development of action potentials necessary for synaptic transmission. There is limited support for this theory.

REVERSIBLE BY WHAT MEANS?
when the gas is turned off, it will diffuse out of the patient along a concentration gradient, just the way it entered. This means the patient will wake up (the initial IV drugs having worn off hours ago)

UNCONSCIOUSNESS is useful.
It dissociates the higher processing centres from the physical sensation of injury, which is pain.
HOWEVER because there are lower and more primitive processing bodies, pain stimulus will still provoke a response: a totally autonomic and animal response, namely:
- the signs of shock (peripheral vasoconstriction, tachycardia, increased BP, RAAS activation)
and also the WITHDRAWAL REFLEX: the spinal cord will command the limbs to jerk away from the injury.

THAT’S WHY WE SOMETIMES NEED PARALYSIS
This must be controlled with MUSCULAR RELAXANTS (paralysis toxins, eg. curare)
There are short acting ones eg. suxamethonium = acetylcholine receptor agonist
- causes prolonged depolarisation of skeletal muscles to a membrane potential above which an action potential can be triggered. The onset of muscle relaxation will be rapid after intravenous injection (30-60 seconds), and lasts 5-10 minutes. The muscle paralysis can be continued with intermittent intravenous boluses, using about 25% of the initial dose. The total dose should not exceed 6-8 mg/kg.)

There are long acting ones eg. rocuronium = competitive acetylcholine receptor blocker
These work when they outnumber the concentration of acetylcholine at the neuromuscular junction.
TO COMBAT AND REVERSE THIS you need to give an acetylcholinesterase inhibitor eg. sarin gas (too permanent for clinical use but the concept is the same)- which will restore the balance in favour of acetylcholine (by inhibiting its breakdown).
BUT!! Its fine at the NICOTINIC neuromuscular junction receptors, but it will also happen at the MUSCARINIC receptors eg, the parasympathetic M3 receptors at the end of the autonomic, in the heart.
THIS CAUSES A PROFOUND BRADYCARDIA. To protect against this one must also give some atropine (or equivalent anticholinergic) to restore the heart rate.
What you will see on an anaesthetic monitor:

- ECG lead II (the one in the direction of heart propagation)
- SaO2 saturation
- Capnograph (measuring expired CO2)

   (this means during expiration the trace falls to zero)

   **NEED TO KEEP THIS NUMBER ABOVE 30**
   - or else respiratory drive fails
   - Over 40 will probably trigger hyperventilation
   - Opiated patient breathing on their own can have a CO2 of 50 and still breathe: opiate drugs increase the respiratory drive threshold

**ANAESTHETIC ASSESSMENT: pre-operative evaluation of fitness**

**Question One:** can this patient get better before surgery?

Can we optimise their chances of surviving surgery by waiting for any other problems to be fixed first?

**AIRWAY: can this patient be intubated?**

- Need to check **thyro-mental distance** (from tip of thyroid cartilage to chin)
- Should be at least 3 fingers of t-m distance
- How much of the **UVULA** can you see?

**Mallampati score of laryngoscopability:**

- **Grade 1:** whole uvula can be seen
- **Grade 2:** partially blocked
- **Grade 3:** no uvula but soft palate
- **Grade 4:** can’t see anything except tongue

- Can you fit your finger into the open TMJ?
- Can you fit 2 fingers into the mouth? (width of laryngoscope blade)
- Can you hyper-extend their neck?…

Then ask about medical background, eg.

- previous anaesthetic reactions
- exercise tolerance (2 flights of stairs MINIMUM!!)
- functional impairment due to respiratory or CVS disease
- can they lie down in the way which their procedure requires?
- Then, talk about liver + kidney disease