Acute Care SINS: Surgical Insights for the Non-surgeon
Chapter 7: Liver and Portal Venous SINS

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Summary
“Surgical Insights for the Non-surgeon,” or SINS, is composed of several short chapters intended to cover fundamental surgical knowledge for non-surgeons. The authors focus on surgical pearls, operative insights, and applied anatomy. In Chapter 7 of this series, the authors address the liver and portal veins; their discussion includes anatomy, surgeries, and transplantation.

Résumé
L’ouvrage « Surgical Insights for the Non-surgeon » ou SINS (aperçu de chirurgie pour le non-chirurgien) se compose de plusieurs courts chapitres conçus pour couvrir les connaissances fondamentales en matière de chirurgie chez ceux qui ne sont pas chirurgiens. Les auteurs se concentrent sur des « trésors de sagesse » tirés de leur expérience personnelle en chirurgie, certaines idées en matière d’interventions, et sur l’anatomie appliquée. Le chapitre 7 traite des veines hépatiques et de la veine porte; l’analyse porte notamment sur l’anatomie et sur les aspects liés à la chirurgie et à la transplantation.

In surgery, eyes first and most; fingers next and little; tongue last and least.
— Sir Lancelot Spratt, Doctor in the House, 1954

Hepatic Anatomy
Fortunately, external liver anatomy is relatively constant; unfortunately, internal liver anatomy is not. The liver is divided into right and left lobes and further subdivided by vascular supply. What follows is our long-winded attempt to describe what takes just a second to look at … so refer to the diagram frequently (Figure 1).

Cantlie’s line runs from the gallbladder fossa to the inferior vena cava (IVC). This divides the liver into right and left lobes but does no more than highlight where the middle hepatic vein leaves the liver parenchyma. A French surgeon (Couinaud, for those who care) further divided the liver into four vertical segments based on the three hepatic veins. Because the portal vein divides the liver in the horizontal plane, each vertical segment could be divided into two more segments. Overall, this means the liver can be divided into eight segments.

Those who have peered at a liver (whether in the operating room, anatomy laboratory … or abattoir) may recall a small area
posterior to — and wrapped around — the IVC. This is the caudate lobe, which has been awarded “segment 1.” The remaining segments are numbered clockwise starting with the left upper segment (2) and ending with the right medial upper segment (8). Those still awake (!) will realize that this should mean nine segments, not eight. However, Couinaud divided the medial segment of the left lobe (segment 4: located to the right of the falciform ligament) into two parts: 4a and 4b. Voilà, eight segments!

Liver Resections

**Hepatic Lobectomies, Segmentectomies, and Trisegmentectomies**

Based upon this “straightforward” nomenclature, hepatectomies becomes “easy” to describe (Table 1):

- Right lobectomy involves removing segments 5–8: about 60–70% of liver parenchyma
- Left lobectomy removes segments 2–4: about 30–40%
- Lateral segmentectomy

**Liver Resections**

- Left lateral resection that removes segments 2 and 3
- However, we rarely talk of right lateral segmentectomy; rather, it’s a 6/7 resection
- The trisegmentectomies are also less intuitive
  - A left “triseg” involves segments 2–4, 5, and 8
  - A right triseg involves segments 4–8
- Solitary segmentectomies are occasionally performed
- Non-anatomical resections are also performed
  - These do not follow the usual vascular divisions
  - They are done in an effort to preserve liver mass
  - Typically performed for cancer; performed less commonly in hemorrhage/trauma

**Hepatic Transplantation**

Liver transplantation is difficult technically but easy to understand anatomically (we mean it this time):

- The old liver is “disconnected”
  - Ligate the artery, bile duct, and the portal vein supply
  - Separate from the vena cava
- New liver is sewed into the old liver’s place
  - Each anastomosis can leak or become blocked
  - Most problematic are the bile duct and the hepatic artery
  - If the common bile duct is of good length, or is not diseased, then primary duct-to-duct anastomosis occurs (choledochocholedostomy); however, frequently, a roux-en-Y hepatojejunostomy is needed (particularly in retransplantation or primary sclerosing cholangitis)
- Whether for transplantation, cancer, or trauma, procedure is more difficult technically when the proximal biliary tree is close to the liver

**Post-operative Complications**

Liver and biliary surgeries are challenging; therefore, complications are not uncommon. Bile and bugs predispose to peritonitis and cholangitis. Leaks should be suspected in a patient who is uncomfortable, jaundiced, or septic. Also watch for biosynthetic hepatic failure (especially when excess liver lands in the pathologist’s bucket). Follow the serum glucose, international normalized ratio (INR) and partial thromboplastin time (PTT), and electrolyte levels (hypophosphatemia is common as this electrolyte is required to regenerate liver cells).

Should the patient “not look right,” further imaging such as Doppler sonography may be indicated. This can identify both vascular and biliary complications. A hepatobiliary iminodiacetic acid (HIDA) scan (see below) can identify smaller bilious leaks if a drain is not present to show an obvious leak. Regardless of
the surgery, keep your surgeon up to date on any concerns and keep the cross-match current: your patient’s proclivity toward bleeding means you may need to act fast.

**Radiological and Other Assessments of Liver Function**

Liver enzyme tests lack sensitivity and specificity, and a thorough review is outside of our scope. Instead, we discuss here the radiological investigations.

**Sonography**
- Assesses the biliary system and gallbladder
- Can identify stones and obstruction (computed tomography [CT] can easily miss these)
- Can identify liver parenchymal lesions (i.e., collections, tumours, or metastases)
- Addition of Doppler allows for vascular assessment

**HIDA Scan**
- Nuclear medicine (aka “unclear medicine”) test
- Involves intravenous injection of radioactive chemical
- Chemical is removed from blood by liver, with excretion in the bile (therefore, you can see a bile leak with this test)
- Offers clues regarding hepatic function, biliary obstruction, and gallbladder emptying

**CT Scan**
- Assesses liver parenchyma; not as good at defining the biliary system
- Allows a simultaneous look at the entire peritoneal cavity

**Endoscopic Retrograde Cholangio-pancreatography**
- Also known as ERCP
- Upper gastrointestinal endoscopy–guided visualization of duodenum and papilla
- Injection of contrast highlights bile ducts, pancreatic duct, and gallbladder
- Can reveal obstruction or lesions
- Can be both diagnostic and therapeutic
- Facilitates stone removal, stent placement, sphincterotomy, and biopsies

**Liver Biopsy**
- Performed either percutaneously or intra-operatively
- Provides liver histology/pathology

**Magnetic Resonance Cholangio-pancreatography**
- Also known as MRCP
- Modification of magnetic resonance imaging (MRI)
- Provides very detailed view of the biliary tree, pancreatic ducts, and liver/pancreas

**Portal Venous Anatomy**

The liver receives two sources of arterial blood: the portal vein (yes, vein) and the hepatic artery. Approximately half of the liver’s oxygen comes from the hepatic artery, and half from the portal vein. Two thirds of the liver’s blood volume comes from the portal vein; one third is from the hepatic artery. Following portal vein occlusion (i.e., portal vein thrombus in cirrhosis) the hepatic artery can rapidly increase flow. In contrast, following hepatic artery occlusion, the portal vein cannot rapidly compensate: portal vein collaterals develop over months. This is why patients can tolerate portal vein thrombosis, but transplanted livers fail following hepatic artery occlusion. Total arterial hepatic flow constitutes about 25% of cardiac output, whereas the liver represents only 2.5% of body weight. As such, this mighty organ can bleed like a “stuck pig.”

The superior mesenteric vein meets the splenic vein at the portal vein (with the inferior mesenteric vein draining into the splenic vein). It runs for 8–10 cm to the liver hilum, and then divides into lobar branches. The left gastric vein (aka coronary vein) usually inserts into the portal vein just above the pancreas. In 25% of cases, the left gastric vein joins the splenic vein (rather than draining directly into the portal vein). The anatomy of other small pancreatic and duodenal veins is even more variable and must therefore be determined during portal vein surgery.

In short, the portal vein collects venous blood from the majority of intra-abdominal structures but also supplies the liver with nutrients. (Hence, it really is also an arterial supply to the liver – we didn’t make that bit up!) Liver drainage occurs via the hepatic vein (a true vein; no more tricks), which then enters the IVC. Once again, anatomical variants abound.

Normal portal vein pressure is 5 –10 mm Hg. Portal hypertension (PH) occurs at >12 mm Hg. Most PH results from resistance to portal flow. Causes of PH can be divided into pre-hepatic (often portal vein thrombosis or, more proximally, splenic vein thrombosis), hepatic (typically cirrhosis or, in the developing world, schistosomiasis) and post-hepatic (Budd-Chiari syndrome, which involves occlusion of the hepatic vein or IVC). Portal flow becomes more sluggish as portal resistance increases. Eventually portal flow reverses (your friendly Doppler sonography technician can tell you when). If portal vein blood flows backwards, it redirects into the systemic venous circulation (via the aforementioned splenic, gastric, and mesenteric veins). This can result in life-threatening encephalopathy (blood is no longer detoxified) and enlargement of venous plexi (aka varices, which are friable and...
can burst, causing massive hemorrhage). Gastroesophageal varices are caused by backflow in the gastroesophageal veins, hemorrhoids are caused by expansion of the hemorrhoidal veins, and caput medusae are caused by recanalization of the fetal umbilical vein.

Mortality is high following variceal hemorrhage (10–40%). Therapy can be divided into (1) pharmacological (medical), (2) endoscopic, (3) radiological, and (4) surgical routes. The therapeutic goals are to decrease variceal blood volume, obliterate the varices, and decrease portal pressure. In plain language, we try to shift blood from the portal to systemic veins. In (more pompous) medical language, we create a portosystemic shunt.

**Treatment for Bleeding Varices**

**Pharmacotherapy**
- Terlipressin
  - Bolus 1–2 mg intravenous (IV) q4–6h (not available in North America at time of writing!)
  - Standard of care in Europe
  - Potent splanchnic vasoconstrictor
  - Cardiac monitoring recommended initially
- Vasopressin
  - Potent splanchnic vasoconstrictor
  - Rate: 0.02–0.04 IU/min
  - Performed in monitored settings (given via central venous access)
- Somatostatin
  - 250 µg IV bolus, followed by infusion of 250 µg/h × 2–4 days
  - Or octreotide, 50 µg IV bolus, followed by 25–50 µg/h × 2–4 days

**Balloon Tamponade**
- Sengstaken-Blakemore tube (gastric and esophageal balloon [Figure 2]) or Minnesota/Linton tube (no esophageal balloon)
  - 85% success rate
  - Bridge to definitive endoscopy (banding esophageal varices or gluing gastric varices)
  - More often a bridge to transjugular intrahepatic portosystemic shunt (TIPS; see below)

**Esophageal Endoscopy**
- Endoscopic variceal ligation/banding
  - Often performed serially (several sessions after bleeding controlled); oral β-blockers (nadolol/carvedilol) are added to reduce re-bleeding
  - Endoscopic sclerosis (endoscopic injection of venous sclerosing agent); if esophageal varices are scarred or have been banded previously

**Gastric Endoscopy**
- Injection of cyanoacrylate glue
  - Small doses to avoid emboli (patients with a patent foramen ovale are at risk for arterial strokes)

**Radiological: TIPS**
- Decompresses the portal system
  - Performed percutaneously via the internal jugular vein
    - Locate the hepatic vein
    - Puncture the liver (to create a shunt between hepatic vein and intrahepatic portal vein)
    - Insert an expandable metal stent … easy, right?
  - Success defined by a post-TIPS pressure-gradient of <12 mm Hg
  - Used for acute bleeds: uncontrollable by other non-operative means
  - Increasingly used for secondary prophylaxis after initial significant bleed (even if controlled by endoscopy)
  - Short-term bridge to liver transplant
  - 95% success rate
    - Re-bleeding unlikely (3% at 1 year) if done in non–life-threatening setting
    - Follow-up with Doppler sonography or venography
    - Can be adjusted if there are residual varices (upsize) or hepatic encephalopathy (downsize)
  - Absolute contraindications: right-sided heart failure, polycystic liver
  - Relative contraindications: portal vein thrombosis, vascular liver tumours, encephalopathy, decompensated cirrhosis (model for end-stage liver disease [MELD] score > 20)
• **TIPS is the preferred emergency procedure**; if still unsuccessful, more drastic options include surgery (see below)

**Surgery for Variceal Disease**

Surgery for variceal disease is rare nowadays, but the discussion is included here for historical context and to assist in understanding the anatomy and pathophysiology.

**Total or Non-selective Shunts**

- Diverts all portal flow into the systemic system, but at the expense of increased encephalopathy (because the liver is no longer detoxifying venous blood)
- Pre-existing sluggish portal flow can precipitate portal vein thrombosis; this precludes surgical shunting directly from portal vein
- Shunt surgery can alter anatomy and impact transplantation
Options
- End-to-side portacaval shunt (Figure 3)
- Side-to-side portacaval shunt (Figure 4)
- Large-diameter interposition shunt
- Conventional splenorenal shunt (a proximal ligation – not as common as the Warren shunt [below])

Partial or Selective Shunts
- Maintains some portal perfusion (benefit is less encephalopathy; cost is residual portal hypertension)
- Options
  - Distal splenorenal shunt (Warren shunt) (Figure 5)
  - Left gastric vena caval shunt
  - Mesocaval shunt (Figure 6); small-diameter interposition graft

Non-shunt Operations
- Goal is to block flow through collateral vessels
- Options
  - Transection and re-anastomosis of the distal esophagus; performed with a stapler
- Sugira procedure – extensive esophago-gastric devascularization; two parts: (1) thoracotomy (transect varices and re-anastomose esophagus) and (2) laparotomy (gastric devascularization and splenectomy)

Bacterial Peritonitis
- Classification
  - Spontaneous bacterial peritonitis (SBP): usually associated with cirrhosis and infected ascitic fluid (due to bacterial translocation)
  - Secondary: usually due to trauma or a perforated viscus
  - Most commonly from gram-negative pathogens (Escherichia coli, Klebsiella, Enterobacteriaceae) and enterococci
  - Diagnosis of SBP
    - Ascitic fluid has >250 neutrophils and/or known pathogen
    - Classified as “sterile” if elevated white blood cell count but no bacteria are isolated
  - Diagnosis of secondary bacterial peritonitis
    - High neutrophil count (usually >1,000 polymorphonuclear leukocytes)
    - Polymicrobial culture
- Treatment
  - SBP: ceftriaxone 1 g IV 24h and albumin 20–40 g/d
  - Secondary bacterial peritonitis: extended spectrum β-lactamase or carbapenem; consult the surgery department regarding a laparotomy to identify the source

Predicting Surgical Mortality in Cirrhotic Patients
- MELD score
  - 10*(0.957*log[creatinine]) + 0.378*log[bilirubin] + 1.12*log[INR])
  - MELD score >15 associated with <50% survival at 1 year
  - MELD >20 associated with 50% mortality following non-cardiac surgery at 30 days
- Child-Turcotte-Pugh score (CTP score; Table 2)
  - CTP A (5–6): 1-year survival >90%
    - CTP B (7–9): 1-year survival approximately 80%
    - CTP C (10–15): 1-year survival <40%
  - Mortality in abdominal surgery for a CTP C cirrhotic is >50%

Dedication
This chapter is dedicated to our friend and colleague Dr. J. Drew Sutherland.
Surgical Pearl: How to Perform a Paracentesis

Note: Remember to maintain sterility (to prevent further infection)

1. Place the patient supine; head of the bed slightly elevated
2. Typically performed through the abdominal wall in the left lower quadrant (also in the right lower quadrant, but there is theoretical concern about “dinging” a distended cecum)
3. Beware: in the midline, there can be abdominal wall collateral vessels (both cephalad and caudad to the umbilicus) ... so avoid those too
4. Clean the selected needle entry site with iodine or chlorhexidine using widening circular motions starting at the “X”
5. Draw up 1% lidocaine solution into a sterile 3 mL syringe
6. Anesthetize the skin using a 3.8 cm (1.5 inch), 25-gauge needle; this needle can confirm the presence of fluid and the depth of penetration needed to reach the ascites
7. Next, pass a 16-gauge needle/angiocatheter (attached to a 10–20 cc syringe) through the abdominal wall (while aspirating the syringe)
8. Once fluid is obtained, send the initial 20 cc for cell count and differential (SBP = neutrophil count >250). Also send for Gram stain, culture, and albumin level
9. Remove the needle; leave the angiocatheter in. This may be connected to a vacuum bottle or urinary drainage bag
10. Replace every litre of ascitic fluid with 8 g of albumin (simple rule: every 3 L, one 100 cc bottle of 25% albumin)

Contraindications
• Uncorrected coagulopathy
• Distended bowel (e.g., bowel obstruction)
• Severe intra-abdominal adhesions (risk of damage to the bowel)
• Surgical scars should also be avoided (? bowel tethered to the abdominal wall)

Complications
• Persistent leak from the puncture site
• Bowel perforation
• Hypotension after a large-volume paracentesis
• Major bleed (Usually from the inferior epigastric, found within the six-pack/rectus sheath)
• Infection (we did warn you!)