the children's hospital at Westmead

PAEDIATRIC LIVER TRANSPLANT ANAESTHESIA - CHW

PRACTICE GUIDELINE ©

DOCUMENT SUMMARY/KEY POINTS

- This document is a practice guideline and is designed to inform the practice of clinicians while they employ their expertise to provide best patient care.
- In addition to the standard care of a patient requiring general anaesthesia, liver transplantation requires careful attention to fluid and blood product management given the potential for significant fluid shifts and blood losses.
- In those with hepatic disease, blood testing alone does not reveal the full picture relating to the risk of bleeding or tendency to thrombosis as both procoagulant and anticoagulant factors may be altered.
- Guidance on the planned immunosuppression regime will be provided so that the anaesthetics team can commence this at the earliest appropriate moment when there is not too much bleeding.
- Communication through all stages of the procedure remains vital.

Approved by:	SCHN Policy, Procedure and Guideline Committee	
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NSW GOVERNMENT

CHANGE SUMMARY

The current edition (7th) represents a scheduled revision and changes include:

- Updated background information with more recent epidemiology
- Addition of the operating room orientation.
- Revisions on haematological and bleeding consideration, renal protective measures, the surgical procedures especially in anhepatic and implantation stage, Point-of-Care utilisation with more detailed guide on interpretation and treatment.
- Addition of new sections on the operating room set up with a diagram and post-reperfusion syndrome.
- Addition of appendices of Check list, CVL guideline and Table summary of intraoperative stages and test.

READ ACKNOWLEDGEMENT

This document is available to be read by anyone involved in the liver transplantation service at The Children's Hospital at Westmead.

All consultant anaesthetists involved in liver transplantation and anaesthetics fellows rotating through the department are expected to read this document (Read Only).

Anaesthetics and recovery nursing staff involved in liver transplantation should also read this document (Read Only).

Perfusion and physiology staff, transplant surgeons, transplant medicine staff and intensive care staff may review this document on a Discretionary basis.

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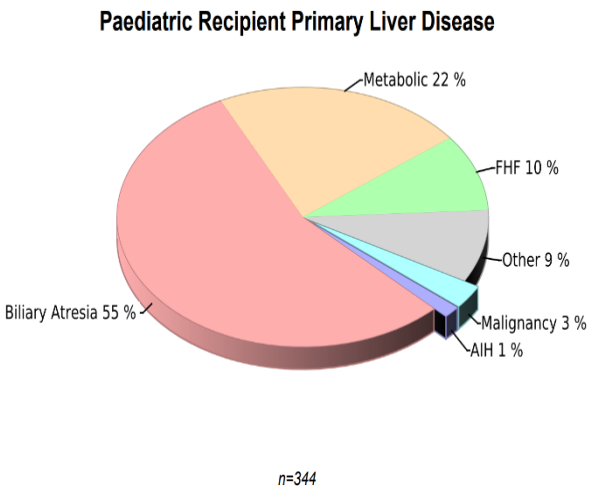
1 Background



2016 marks the start of the fourth decade of paediatric liver transplantation in Sydney. It started at RPA then transferred to The Children's Hospital at Westmead in 1998. By the end of 2019, 344 hepatic transplants had been performed, 3 were redo transplants.

For the 2015 -2019 period, the median age at transplant was 1.8 years old. Using figures extending back to 1986, 88% of patients survived 1 year and 83% of patients have survived to 10 years.

This has improved recently though: the 2015 - 2019 review showed 100% survival to 18 months and 88% to 5 years.

Biliary atresia is the most common primary disease of transplant recipients since 1990s, followed by metabolic disorders. Fulminant Hepatic Failure (FHF) is the next common cause and it increased during the COVID-19 pandemic in early 2020s.

2 Initial Anaesthetic Review During the Work-Up

During transplant work-up, patients are reviewed by consultant anaesthetist on-call for liver transplant for the day. The anaesthetic review is focused to:

- Assess current status of primary liver disease.
- Assess general medical and surgical histories and examination.
- Assess and optimize potential anaesthetic-specific risks may encounter during the surgery.
- Explain the role of the anaesthetics team in the context of the liver transplant operation.

3 Implications of Liver Disease on Anaesthetic Management

The liver is an organ with key roles underpinning almost every other organ system. Most patients having a liver transplant have liver dysfunction, although this is often not the case for patients with metabolic disorders requiring transplant. Some syndromes may be associated with their own associated pathophysiology. Following is a quick refresher on key things to consider when assessing these patients.

3.1 Respiratory Considerations

In addition to a standard review of respiratory status, patients with end-stage liver disease can have particular respiratory issues:

- ***Mechanical:*** Any combination of organomegaly, ascites or pleural effusions can significantly decrease the lung volume and hence functional residual capacity (FRC). This can put patients into the zone where there is closure of small airways in normal respiration and increased work of breathing. That increase in work is further exacerbated by changes in position of the diaphragm, increased abdominal pressures (ascites etc) and the muscle wasting and deconditioning that may be associated with liver disease.
- ***Portopulmonary hypertension:*** This looks histologically like idiopathic pulmonary arterial hypertension but occurs in the setting of portal hypertension. The mechanism is unclear (maybe due to imbalance of vascular mediators, remodelling of pulmonary vessels triggered by increased shear stress). Conventional medications such as calcium channel blockers may cause splanchnic vasodilatation and cannot be used. It is possible that patients may therefore be on other agents such as bosentan or sildenafil though no pharmacological agent is presently standard of care.
- ***Hepatopulmonary syndrome (HPS):*** A small percentage of patients with end-stage liver disease get this triad (portal hypertension, widened alveolar-arterial (A-a) gradient and intrapulmonary vascular dilation (IPVDs)). The areas of vascular dilation tend to be more prominent in lower zones of the lungs so these patients, in a seeming paradox, oxygenate better lying flat as blood is more distributed to areas with less IPVDs. An increase from low saturations by > 4% when lying flat is suggestive. IPVDs can be demonstrated by delayed appearance of agitated fluid in the left atrium on echocardiography, or with nuclear medicine scanning. HPS may be helped a little by oxygen but can only be fixed with a liver transplant.

Note that most patients will be easier to manage from a respiratory point of view sitting up, with the exception of those with hepatopulmonary syndrome, though this pathology is very rare in children.

3.2 Cardiovascular Status

Patients with end-stage liver disease may end up with a high output low resistance systemic circulation. As a result, hypovolaemia and/or any co-existing cardiovascular pathology is poorly tolerated. The reason for the lower systemic vascular resistance (SVR) is not entirely clear but may be a result of enhanced endogenous production or diminished hepatic clearance of vasodilators (like NO, CO, endogenous cannabinoids, TNF-α, adrenomedullin, and hydrogen sulfide) and on some occasions the inflammatory response to bacterial translocation causing splanchnic arterial vasodilation. [1]

Part of the response to low SVR is retention of sodium and water which increases plasma volume. There is also increased venous capacitance where there are any portosystemic shunts.

Another key thing to remember is that some specific diagnoses that cause end-stage liver disease are also associated with cardiovascular dysfunction (e.g. haemochromatosis, Wilson's disease and amyloidosis).

3.3 Haematological and Bleeding Considerations

While patients with end-stage liver disease have a lack of coagulation factors, low platelet counts (sometimes with platelet dysfunction), dysfibrinogenaemia and elevated tPA levels that might predispose to bleeding, they also have more vWf and fVIII as well as decreased levels of proteins C and S, antithrombin III (ATIII), alpha$_2$-macroglobulin, plasminogen and heparin cofactor II. These latter factors all favour thrombosis. This all means that standard coagulation tests alone don't give the full picture as to whether patients will have a bleeding tendency or not. It is important to consider whether patients have evidence of portal hypertension as this contributes significant in bleeding risk; (around 25-35% of patients with cirrhosis will have bleeding from varices). [2]

There has been a trend internationally to a significant reduction in transfusion requirements for a liver transplant surgery due to advances in preoperative medical optimisation, surgical and anaesthetic techniques, use of cell saver and increased awareness of risks associated with transfusion as well as lowered haemostatic targets. This overall movement has resulted in a downward trend of massive transfusion, and in some centres a portion of their paediatric liver transplants were performed without allogenic RBC transfusion.

Several centres have shifted to Point of Care Thromboelastography (POC TEG) to guide transfusion management, often resulting in increased use of cryoprecipitate and platelets, however data on whether this reduces total transfusion requirements is uncertain.

The use of Fresh Frozen Plasma (FFP) generally appears to have followed the trend of a reduction in overall blood product use, however this varies between institutions. Recent 5-year cohort data collected from a tertiary single institution in 2020 reports a significant reduction in volume of FFP use, paralleling the reduction in massive transfusion. In contrast, the other institution in London reported a reduction in RBC transfusion with higher use of FFP.

3.4 The Kidneys

Renal function is one of the main transplant predictors of graft survival and therefore an important consideration in all patients undergoing liver transplantation. [3] Kidney dysfunction in this population is typically secondary to a combination of factors including pre-existing conditions, pre-transplant renal injury, peri-operative events and post-transplant nephrotoxic immunosuppressive therapies.

Acute kidney injury is relatively common in patients with significant liver disease and is most often due to pre-renal insults. This underlines how poorly these patients tolerate hypovolaemia. Pre-renal insults may include gastrointestinal haemorrhage, diuretic use or diarrhea from infection or use of lactulose. With disease progression and systemic vasodilation there can be activation of the renin-angiotensin system and sympathetic nervous system which contributes to ascites, oedema and renal vasoconstriction. Hepatorenal syndrome is a severe extension of this pathophysiological state but is very rare in children. [4]

Post-transplant kidney dysfunction is increased where there is prior kidney failure, hepatorenal syndrome, intraoperative hypotension or hypovolaemia and in those needing more blood products. [5] The incidence of renal dysfunction post-transplantation has been estimated to be between 1-32%. [6] Post-transplant kidney injury is a major contributor to bad outcomes post-transplantation including increased ICU and hospital stay, increased post-operative infection rates, lower graft survival, increased risk of progression to chronic kidney disease as well as higher mortality. [5] [6] [7]

Post-transplantation immunosuppressive therapy with calcineurin inhibitors such as Tacrolimus commonly results in nephrotoxicity and hypertension requiring strict monitoring of renal function and drug levels in order to avoid long-term kidney injury. Basiliximab (an Interleukin2-receptor monoclonal antibody), is commonly used on Day 0 and Day 4 and allows for delayed introduction of calcineurin inhibitors in patients with pre-transplant renal impairment. Nevertheless, the input of a nephrologist is often required in the short term and occasionally long-term.

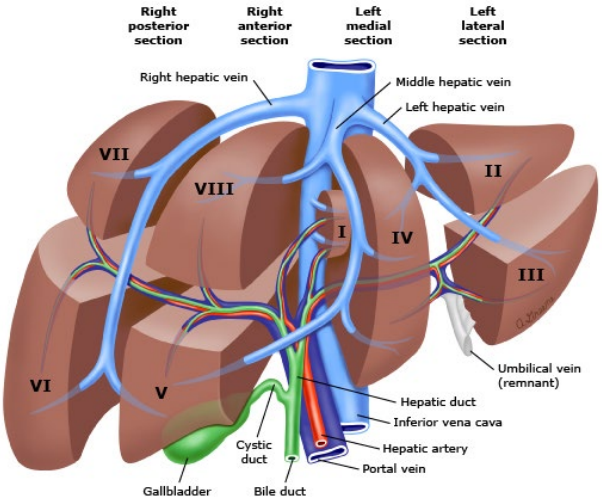
3.5 Neurological Factors

Encephalopathy can be evident in liver disease, particularly with acute decompensation. It is particularly related to hyperammonaemia as the liver's detoxification function fails. Intestinal tract bacteria produce ammonia which, when not cleared, crosses the blood brain barrier and is taken up by astrocytes. In astrocytes this is converted to glutamine and the end result is swollen astrocytes with reduced ability to regulate neurotransmission. [1] It is also worth noting that chronic liver disease has its own neurodevelopmental implications. Patients with early onset liver disease have a lower IQ than those with later onset of the same disease (as measured 8-11 years after diagnosis) and around 54% will have some form of delay in neurological development. [9]

4 Surgical Technique

4.1 The Donor Organ

Following retrieval, the liver is flushed with cold saline and later University of Wisconsin (UW) solution and kept cold. Minimising ischaemia time (Cold Ischaemic Time) is always the aim with the maximal ischaemia time allowed 20 hours.



Anatomically, the liver is divided into 8 segments:

- The large right lobe contains segments 5, 6, 7 and 8.
- The caudate lobe comprises segment 1.
- The left lobe consists of segments 2, 3 and 4. Segments 2 and 3 are also referred to as the left lateral segments. Segment 4 is also known as the medial segment.

In paediatric liver transplantation either a whole or reduced liver graft is implanted. The concept of **split** liver grafting refers to transplantation to 2 recipients while **cutdown** implies liver reduction to fit a recipient with the remaining part discarded.

Sizing the graft to the patient is vital. As a rough guide a **whole graft** is suitable if the ratio of donor body weight to recipient body weight ratio (D/R ratio) is $\leq$ 2. This requirement is rarely met in children so reduced grafts are the norm.

In practice, the transplant team will calculate whether the graft will fit by looking at the weight of the graft vs the weight of the patient receiving the graft with a range of 1-5% of recipient body weight being acceptable. As an example:

Adult donor Total Body Weight (TBW)	Liver weight = ~2% TBW	Left Segment = 30% total liver	Left lateral segment = 50% Left Segment
80 kg	1.6kg	480g	240g

Therefore, the graft could be as little as 240g, making it feasible even for a 5kg recipient.

4.2 The Operation

It is vital during the operation for the anaesthetists to keep in constant communication with the surgeons and to be aware of the operative phase. This ensures all team members are aware when surgical manoeuvres such as application or release of clamps are about to take place and if there are any new issues arising.

The operative procedure can be considered in 5 stages:

I. Hepatectomy Stage

This is from skin incision until isolation of the native liver from its blood supply. This can be prolonged in patients with prior abdominal surgery. A key consideration for the surgeons during this phase is to avoid any irreversible steps until it is confirmed that there are no issues with the donor organ. Most commonly the biliary system will also be disconnected during this phase both to prepare for the later biliary anastomoses and provide better surgical access to the hepatic vessels.

II. Anhepatic & Implantation Stage

This starts when both the arterial and portal venous supply are clamped. The inferior vena cava (IVC) is cross-clamped below and above the liver (infra-hepatic IVC and supra-hepatic IVC respectively). A trial cross-clamping of the IVC is done before committing to the anhepatic stage to determine the effect on haemodynamics. Any hypotension can usually be corrected with a fluid bolus. Sometimes titrated vasopressors or inotrope infusions are often required.

The donor hepatic vein is joined to the recipient's IVC with an end-to-end anastomosis (the piggy-back technique). A small portion of this is left open in order to be able to flush out residual preservative solution in the liver which has a very high potassium content. Glycine solution (2mmol/L) in plasmalyte which has cytoprotective effects on hepatocytes; is used in this process. Failure to flush out this preservative solution could result in sudden dangerous hyperkalemia when the liver is re-perfused. Potassium levels of graft flush solution are checked prior to reperfusion to ensure they are below 10mmol/L.

In children with Biliary Atresia with Splenic Malformation (BASM), the infrahepatic cava may be absent in which case the donor hepatic drainage vein is joined end to end to the

suprahepatic cava. In rare cases with hepatic tumours, naïve IVC may be replaced with a donor vein and there will be a suprahepatic as well as an infrahepatic caval anastomosis.

Following this, the portal vein is anastomosed. Often, a vein graft is used to reconstruct the native portal vein. The donor liver temperature is raised by running blood from the portal vein into the graft prior to release of the portal vein and hepatic vein clamps. The surgeons will take particular care to avoid air emboli with release of the clamps. Methylprednisolone 10 mg/kg is generally given in the 15 minutes prior to reperfusion. This timing is not critical and should be delayed if there is significant bleeding.

III. Reperfusion Stage

The first stage of reperfusion is release of the clamps on the portal vein and hepatic vein. The hepatic arterial anastomosis is then performed. It is after this anastomosis that the surgical team will usually take a break to refresh.

When getting on with the next stage of the operation, blood flow through the portal vein and hepatic artery is measured using a Doppler flow probe with the aid of the perfusion and/or physiology teams. An assumption is made that hepatic blood flow is around 25-30% of cardiac output (estimated as 3.0 L/min/m^2) with 75% via the portal vein and 25% via the hepatic artery.

IV. Biliary Connection

Once flows are felt to be acceptable and haemostasis is reasonable, the biliary anastomosis is completed. This usually involves a Roux-en-Y biliary-enteric anastomosis as more than 50% of recipients have biliary atresia. A duct-to-duct anastomosis is preferable where is possible regardless the size of patients. When the new liver starts functioning bile flow will generally commence.

V. Abdominal Closure

In paediatric liver transplants, secondary closure is most common a few days after the initial transplant operation to allow swelling to settle. This is done in preference to risking intra-abdominal pressure elevation which might compromise graft flow. The intervening temporary closure involves coverage of the defect with a plastic bag. Primary closure may be attempted if the graft function is unlikely to be compromised and there is a strong desire for early weaning off ventilation (e.g. cystic fibrosis patients).

5 The Anaesthetic

5.1 Immediate pre-operative assessment

Most recipients on the liver transplant list are seen by on-call anaesthetist for liver transplant when they are registered on the waiting list as discussed earlier on this protocol. The purpose of the immediate pre-operative visit is to establish current clinical status; and check final preparation steps and safety for the transplant.

The key questions of assessment are:

- What is the patient's current health status?
  - Do they have stable liver disease?
  - Do they have worsening chronic disease?
  - Do they have acute end-organ dysfunction on top of the chronic disease?

The development of new pathology since initial transplant work-up (e.g. portal hypertension, respiratory dysfunction, worsened ascites) is important to pick up.

Next you need to check if tests have been updated and preparations made:

- Have preoperative blood tests been done?
- Are blood products available? (Baseline is 3 units PRBCs and check availability of other products.)
- Are there additional medication requirements?
  - Medications for patients with metabolic disorders.
  - Premedication where appropriate and patient not encephalopathic.
  - Are there specific immunosuppression requirements?
  - Are there particular requirements for antibiotic prophylaxis?

5.2 Pre-Operative Preparation

The anaesthetics team generally starts preparation 1 hour before scheduled anaesthetic induction time (the perfusionist generally arrives 30 minutes prior to this). Note this section is summarized with extra guidance in Appedix 1 and 2. It is anaesthetist's responsibility to contact all essential personnels (Appendix 3).

Checks and Equipment

- Prepare for a massive transfusion:
  - 3 units red blood cells in the blood fridge in operating theatre.
  - Check with blood bank if supply of FFP, platelets and cryoprecipitate is adequate.
  - Check there is an adequate supply of 4% albumin in the anaesthetic workroom.
  - Early involvement of a haematologist on-call if recipient's coagulation profile is markedly deranged or known haematological disorder.
- The room checks:
  - Anaesthetic machine in the bay and inside the room.
  - Temperature management – room temperature, warming devices
  - Operating table – brackets, arm supports if needed, anaesthetic screen, pressure area check and positioning plan.
  - The trauma/cardiac induction trolley for induction to allow CXR.

Preparation of medications

	Medication	Guide on preparation and dosage
Vasoactive	Metaraminol	10 microg/kg/dose
	Adrenaline* Noradrenaline*	0.05 – 0.5 microg/kg/min 1mg in 50mL for up to 10kg 0.3 mg/kg in 50mL for > 10kg Max 3mg in 50mL
	Dopamine*	15 mg/kg in 50mL 1 – 5 microg/kg/min
	Atropine	20 microg/kg/dose
Ion/glucose supplement	Calcium chloride 10%	0.15 mmol/kg/dose (= 0.2mL/kg/dose)
	Potassium chloride	25 mmol in 50mL 0.1 – 0.3 mmol/kg/hour (Max 20mmol/hr)
	Glucose 10% (PIVC) or 50% (CVL)	0.2g/kg/dose (= 2mL/kg/dose of 10%D or 0.4 mL/kg/dose of 50%D)
	Insulin neutral (Actrapid)	50units/50mL NaCl 0.9% 0.02 – 0.06 units/kg/hr
	Sodium bicarbonate 8.4%	1mmol/kg/dose slow infusion
Antibiotics	Piperacillin with Tazobactam	100/12.5mg/kg max 4/0.5g/kg 8 hourly. Renal adjustment required.
Immunosuppressant	Methylprednisolone	10mg/kg/dose

*The infusion rate is a guide only. Clinical judgment is required in each circumstance.

Items for Other Team Members but not limited to:

- Perfusion – cell saver, TEG, rapid infusion system, Near-InfraRed Spectroscopy (NIRS) monitor, Doppler flow checks (very rarely venovenous bypass).

5.3 Induction of anaesthesia

There is no particular evidence for one induction technique over another specific to liver transplantation. Rapid sequence, intravenous or inhalational induction may be chosen based on patient age, condition and clinician judgment.

Monitors

Two pulse oximeters (wrap around), non-invasive blood pressure at the start and ECG once feasible. Patients will need temperature monitoring, IDC, the largest feasible NG and NIRS monitoring to the forehead (severe jaundice may invalidate these readings). Monitoring to minimise awareness is not generally used.

Airway and Breathing

A cuffed endotracheal tube, most likely nasally placed and controlled ventilation using an oxygen-air mixture. There is no evidence to recommend one inhalational agent over another for maintenance. Isoflurane may maintain more physiological hepatic arterial and portal venous blood flow. There are some evidence patients with end-stage liver disease have lower requirements of volatile agents to avoid awareness. [10]

Circulation and Lines

All lines must be placed with strict aseptic precautions to minimise infection risk. Standard lines are:

- 2 x large peripheral cannulae for intravenous volume (must be upper limb, preferably not antecubital fossae).
- 1 x long saphenous line for pressure monitoring (alternate is a femoral venous cannula).
- 2 x arterial lines (one for monitoring and the other one for blood sampling)
- 1 x CVC (Refer Appendix 2)
- Once lines are placed and prior to entry of the operating room, chest X-ray is performed to check placement of the ETT/NG and lines (and to help exclude complications).

5.4 Inside the Operating Room

The Operating Room Orientation

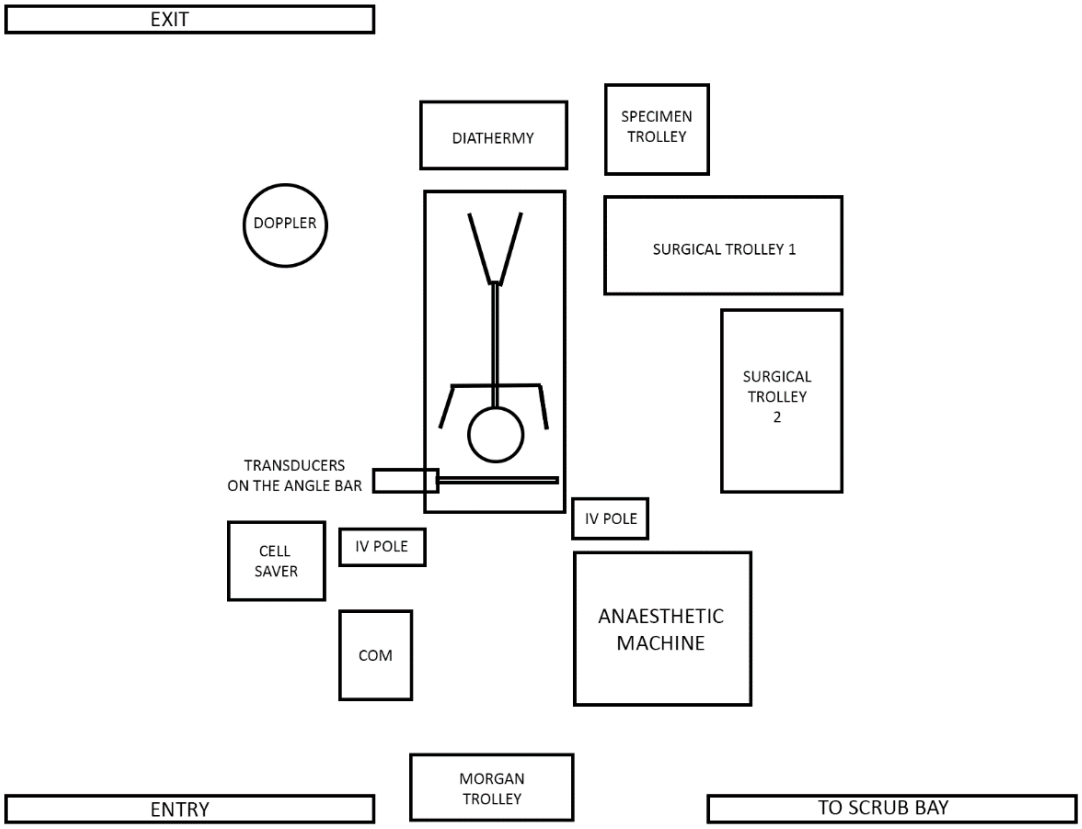


Fig. The operating room and patient orientation. The patient is positioned head towards the entry of the room and arms bent up with appropriate paddings to protect nerves. COM: computer

Positioning and Protection Considerations

The surgeons may place a roll under the lower thoracic spine to produce better surgical access. Pad all pressure areas, with gel pads recommended if appropriate for the size of patient. Consider Velband wrapping of limbs as a way of providing padding and managing temperature status. All lines and monitors must be shielded from direct contact with the skin.

Standard eye protection for a longer case is required. Extra patient identification band with extension strap should be prepared for all patients.

Airway, Breathing and Circulation

Standard ventilation to physiological targets of oxygenation and ventilation are appropriate. Nitrous oxide is not recommended to avoid gaseous distention of the bowel loops. While there is evidence from adult practice that postoperative pulmonary complications are a significant factor in patient morbidity and mortality, risk factors for development of this seem to be mostly related to excessive fluid and blood product administration, and preoperative presence of severe ascites. [11]

Patients with end-stage liver disease often have a high cardiac output and low systemic vascular resistance system. Excessive fluid administration may be associated with complications in the post-operative period and maintenance of a high CVP (previously a measure used to prevent air embolus) is no longer standard care. Maintaining CVP in the range of 6-10 mmHg is reasonable to minimise this risk, along with PEEP. Have a high index of suspicion for air embolus if there is sudden cardiovascular collapse.

There is still a difficult balance to maintain in managing fluids. While excessive fluids may not be appropriate, maintenance of CVP as described above may still require large amounts of fluid. Minimum losses to consider per hour would be (maintenance + 10 mL/kg/hour for the open abdomen). Careful attention to fluid responsiveness, which may be aided by monitoring techniques such as measurement of pulse pressure variation (PPV aiming to be < 10), and early utilisation of vasoconstrictor agents (such as metaraminol, noradrenaline or dopamine) seems appropriate, though outcome studies are not available. [1] Surgical procedures, in particular manipulation of the liver by the surgeons, can cause substantial changes in blood volume distribution in smaller children, creating haemodynamic instability that is not blood loss related. Close, clear communication between surgeon and anaesthetist during such procedures is crucial to prevent overuse of fluids in these settings. The lower limb or femoral venous pressure line is specifically useful in these settings. Trans-oesophageal echocardiography may be considered in some cases to assess cardiac function.

Maintenance and Analgesia

Maintenance is generally with volatile as mentioned above. Ongoing analgesia is opioid-based, with fentanyl generally preferred. An infusion for ongoing analgesia in PICU should be prepared. In patients having primary closure, consider wound catheters for infusion of local anaesthetic.

Blood Product Management

Rational management of blood products is an area where careful anaesthetic care may meaningfully contribute to patient outcomes. As described earlier, these patients have some deficiencies that predispose to bleeding but others that can potentially promote clotting. Standard coagulation tests do not reliably predict patient bleeding. [2]

Since 2010s, the approach to blood product management at The Children's Hospital at Westmead has shifted, aiming to prevent thrombosis through post-revascularisation administration of antithrombin III and early heparinisation. This has been associated with a lower vessel thrombosis rate and fewer biliary complications in the longer term.

There are further reasons to minimise use of blood products. One-year patient and graft survival is associated with the number of RBC and FFP units transfused during surgery. [12]

Plasma-containing blood products are associated with development of transfusion-associated lung injury (TRALI) while the RBCs are associated with elevated risk of postoperative infection. [13]

Therefore, some basic principles of blood product management are particular to these operations:

- Aim for haematocrit is 25-28% towards the end.
- Once the patient has received more than 20 mL/kg of RBCs, consider utilisation of FFP or possibly cryoprecipitate and platelets.
- Point-of-Care testing such as Thromboelastography (TEG) or Rotational thromboelastometry (ROTEM) may provide useful information but must be interpreted in the context of the amount of bleeding evident clinically.
- Hypothermia and hypocalcaemia may contribute slow coagulation process.
- The metabolic insult of RBC transfusion may be significant, particularly in younger patients. Wash all RBCs utilising the Cell Saver. This reduces the potassium and citrate load and is particularly important in the anhepatic phase.

NB *Cytomegalovirus (CMV) infection is a potentially significant post-transplant infectious complication.[11] CMV status of the donor is always noted:*

- If donor or recipient are CMV positive, post-operative anti-CMV treatment is given;
- If both the donor and recipient are CMV-negative then it is important that CMV negative blood is utilised for all transfusions.

Role of FFP in Liver Transplant

There has been a trend internationally to significant reduction in transfusion requirements for liver transplantation due to advances in medical, surgical and anaesthetic techniques as well as utilisation of point-of-care devices and increased awareness of transfusion risks. The use of FFP generally appears to have followed the trend of a reduction in blood product use in liver transplantation, reflecting the overall reduction in massive transfusion. [14][15][16] However, this varies between institutions.

- 2020 Queensland Children Hospital data reports a significant reduction in volume of FFP use, paralleling the reduction in massive transfusion in a recent 5-year cohort compared to earlier groups.[17]
- 2019 Kings College London review reported a reduction in RBC transfusion, but a slightly higher use of FFP in a recent cohort vs earlier groups.[18]
- Overall, there is a paucity of data on the optimal maintenance fluid (crystalloid vs colloid), and on the use of FFP as the primary maintenance and/or resuscitation fluid in this specialized population.

At CHW, there has been a trend towards more liberal use of FFP for resuscitation and maintenance over last 5 years, however, there is as yet no strong evidence internationally to support this practice. Use of FFP needs to be carefully balanced in an attempt to reduce massive transfusion but not to facilitate pro-coagulant status.

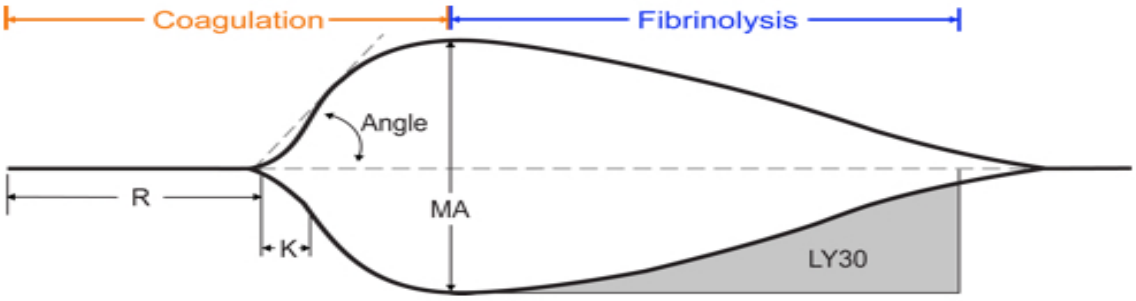
Thromboelastography

Historically blood tests for coagulation were sent regularly throughout the cases. There can be significant delays in these tests returning which makes it hard to use results to guide blood product management. There are some studies suggesting using TEG may be useful and may be associated with transfusion of fewer blood products. [19-23] It should be noted that many of these studies are small and open to issues of bias and there are also studies suggesting no change in blood product use or that results on testing can be equivocal. [24-26]

The operating theatre, with the particular help of the perfusionists, has a TEG 6S (Haemonetics™) as the primary TEG device available. The information from the company states that this can provide useful information within 10 minutes as you can perform tests relying on Kaolin, Functional Fibrinogen, Heparinase testing and a RapidTEG™ although it's not always as clear as that clinically. It is critical to recall that any result on the TEG is only relevant when interpreted with reference to the degree of bleeding seen clinically.

The TEG 6S provides a number of traces, which can be visualised over the top of each other or viewed separately.

Below is the general scheme for a TEG tracing with parameters in example (note that these images come from info provided by Haemonetics, so the traces may be potentially idealized.



Symbol	Title	Measures	Normal	What changes mean	Suggestion
R	Reaction time	Minutes to first measurable clot formation (to 2mm amplitude)	5 – 10 min	↑ : clotting factor deficiencies, anti-coagulants e.g. Heparin	FFP/Protamin if R > 10 min
				↓ : Hypercoagulation	Consider Anticoagulant
A	α angle	Angle of tangent line from 2mm to 20mm representing fibrinogen function	45 – 75°	↑ : Platelet hypercoagulability	No treatment needed
				↓ : Hypofibrinogenemia or Factor VIII deficiency	Cryoprecipitate, FFP if α < 53°
K	Clot formation time	Minutes taken from 2mm to 20mm amplitude, representing fibrin cross-linkage and function	1 – 5 min	↑ : Hypofibrinogenmia	Cryoprecipitate
				↓ : Hypercoagulation	
MA	Maximum Amplitude	Amplitude measured at peak clot strength; most reflects platelet	50 – 75 min	↑ : Platelet hypercoagulability	May need anti-platelet therapy
				↓ : Platelet dysfunction	Platelets, DDAVP
LY30	Lysis Index	Percent loss of amplitude at 30 min after MA reached	0 – 10%	↑ : Enzymatic or mechanical hyperfibrinolysis	Tranexamic acid if LY30 > 3% and bleeding

Fig: Illustration of thromboelastographical analysis Table: Parameters measured in thromboelastography, interpretation and suggestion of treatment [27][28] which should be used as a guide only.

Below is a table of products that should be discussed with surgeon prior to administration.

	Indication	Dosage
Antithrombin III	May be given once all anastomoses are complete and clinical evidence of bleeding is not present.	< 30 kg: 1000 units IV 30 – 60 kg: 2000units IV > 60 kg: 3000 units IV
Anti-fibrinolytics e.g. Tranexamic acid	Should only be considered in patient with a significantly elevated risk of bleeding.	Loading dose 20mg/kg/dose IV over 30 minutes, then infusion at 10mg/kg/hr. Dose limit 100mg/kg up to 2g in total.
Recombinant Factor VIIa	Should only be considered in a case of life-threatening exsanguination where product replacement and surgical attempts to control bleeding are proving unsuccessful.	90microg/kg IV

Anti-Rejection Therapy

Optimally anti-rejection medications are given prior to revascularisation. If there is significant bleeding at that time, confirm with the surgeons that therapy should be given. It is best if the anti-rejection medications are not immediately lost due to bleeding.

- 5-10 mins prior to revascularization give methylprednisolone 10 mg/kg/dose intravenously.

On some occasions, the hepatology team may request basiliximab, an IL-2 receptor monoclonal antibody (used when calcineurin inhibitors will be utilised later). It is particularly used when there is pre-transplant renal impairment. This is also given on day 0, but again should not be administered until haemorrhage control is reasonable and is more often given once the patient is in the intensive care unit.

If used, the intraoperative dose of basiliximab (given over 20-30 mins) is:

- 10 mg IV if < 35 kg.
- 20 mg IV if ≥ 35 kg.

Metabolic Management

Blood Glucose Level

Some patients may have minimal hepatic glucose storage. Patients requiring glucose pre-operatively should have this continued. During the anhepatic phase, the absence of the liver's glucose stores usually causes a fall in blood sugar. As a separate indication, those with primary metabolic disorders should have glucose maintained at normal levels to ensure minimal protein catabolism.

Therefore, if blood glucose falls below 5 mmol/L, commence 10% dextrose at 1 mL/kg/hr (or adjust for concentration used). Aim to keep BSL at 6-10 mmol/L as monitored on regular arterial blood gas testing.

Potassium

Serum potassium is often low preoperatively. A pause in any potassium infusion around revascularisation should be considered as there may be a brief spike in potassium when the clamps are released. Once the graft starts working, potassium may fall rapidly, and potassium infusion may be required again.

- If K^+ is < 3.5 mmol/L, start an infusion at 0.25 mmol/kg/hour (0.5 mL/kg/hr of the 0.5 mmol/mL solution suggested in this guideline) with titration of the infusion aided by the testing schedule in section 6.

If potassium is being replaced, consider checking serum magnesium and replacing to keep it > 0.7 mmol/L.

In the event of hyperkalaemia with revascularisation (as evidenced by peaked T waves or QRS changes):

- Calcium chloride 10% 0.2 mL/kg IV.
- Consider sodium bicarbonate 1 mmol/Kg IV.
- Consider 0.1 U/kg actrapid insulin with 50% dextrose 0.5 mL/kg IV.

Acid-Base Balance

Metabolic acidosis is common intraoperatively due to the effects of major vessel clamping, reduced renal function and fluid infusion. Patients who have had chemotherapy for hepatoblastoma may present with metabolic acidosis. Base deficit up to 15 mEq/L can be well tolerated. These derangements generally recover once the new liver is functioning. Using Hartmann's avoids the additive issue of hyperchloraemic metabolic acidosis with use of 0.9% sodium chloride.

A functioning graft in good haemodynamic conditions will generally allow a rapid normalisation of acid-base status. If this is not the case, sodium bicarbonate up to 1 mmol/kg IV may be considered. If acidosis is worse or stays the same once the graft is in, this may be a sign of dysfunction and should be mentioned to the surgical team.

Renal Protection

This relies on maintenance of good haemodynamics. Note that oliguria is expected during IVC cross clamping and is probably due to back pressure transmitted into the renal veins and an associated fall in glomerular filtration. True hepatorenal syndrome is rare, and intraoperative management still depends on management of haemodynamics and fluid management. There is no evidence supporting the use of diuretic agents to optimise urine output measurements intraoperatively.

Bleeding and coagulopathy can result in haemodynamic instability and decreased intraoperative renal perfusion. Surgical techniques are vital in minimising blood loss however good blood management practices are also important in minimizing coagulopathy and secondary bleeding. Unfortunately, no data currently exists to suggest a perioperative intervention that reliably demonstrates renal protection. [8]

Post-Reperfusion Syndrome

Post-reperfusion syndrome (PRS) is a well-documented complication in adult liver transplantation, however information regarding PRS in paediatric liver transplantation remains scarce.[29] The overall incidence of PRS varies amongst reports, ranging between 8% and 30%. [30] PRS was originally described by Aggarwal et al. in 1987 as cardiovascular collapse after reperfusion of the newly transplanted liver. They further defined PRS as a 30% decline of mean arterial pressure relative to the value in the anhepatic phase that lasts for at least 1 minute, occurring within 5 minutes of reperfusion of the donor liver. [31] After the release of the portal vein clamp, the right side of the heart and cardiovascular system is

suddenly exposed to cold, acidotic blood resulting in variable decrease in heart rate, contractility, and peripheral vascular tone. [32]

In paediatric liver transplantation intraoperative PRS occurrence seems to be associated with poor liver allograft function and worse patient postoperative outcomes.[29] Risk factors for PRS include advanced age of donor, prolonged ischaemic times (cold and warm) and organ size mismatch. Just prior to reperfusion low haemtocrit and electrolytes abnormalities such as low calcium and high potassium are also risk factors. [33] In general preventative measures should be taken prior to reperfusion to decrease the incidence and severity of PRS. These would include correction of electrolyte abnormalities, correction of low haematocrit, optimisation of volume status and consideration of pretreatment with inotropes.

6 Intraoperative Testing

Biochemistry and haematology can change quickly over the course of a liver transplant. Regular blood testing can be helpful but must be interpreted in clinical context and must be available rapidly to usefully guide management. Additionally, some tests can take a reasonable amount of time to come back. For labelling purposes:

1. Pre-induction phase.
2. From induction to the blood supply to the native liver being interrupted.
3. The anhepatic and reimplantation phase.
4. Revascularisation.
5. Start of biliary connection (through to end of all anastomoses).

Simple nomenclature is used to indicate which phase of the operation the bloods were taken in, and in which order. So for phase 2, induction phase to anhepatic, the first tests sent would be test 2.1, the next is 2.2, the next is 2.3 and so on.

PHASE	TIMING	TESTS REQUIRED
P1	**Pre-Induction**	Preop bloods on Powerchart.
P2	**INDUCTION/HEPATECTOMY PHASE**	
P2.1	While in bay	ABG / BSL and Electrolytes/ Baseline TEG
P2.2, 2.3 etc.	on hour	ABG / BSL and Electrolytes / Coags once during P2) / TEG (minimum once per phase)/ FBC (with 2.2 then as indicated by blood loss)
P2.(last)	10 minutes before P3	ABG / BSL and Electrolytes
P3	**ANHEPATIC AND REIMPLANTATION PHASE**	
P3.1	on ½ hour	ABG / BSL and Electrolytes
P3.2, 3.3 etc	on hour	ABG / BSL and Electrolytes / Coags (once in phase 3)/ TEG / FBC with 3.2 then as indicated by blood loss.
P3.x	10 minutes before Revascularisation	ABG / BSL and Electrolytes/ TEG
P4	**REVASCULARISATION**	

P4.1	5 minute post revascularisation	ABG / BSL and Electrolytes
P4.2	15 minutes post revascularisation	ABG / BSL and Electrolytes/ TEG
P4.3	on ½ hour	ABG / BSL and Electrolytes/ FBC (then as indicated by blood loss)
P4.4&+	on hour	ABG / BSL and Electrolytes / Coags /TEG Consider one set of formal EUCs with LFTs during P4.

- Suggestions for checking coagulations and TEG are not rigid and should be guided by bleeding state - one TEG should be checked in each phase as a minimum, then repeated as suggested by bleeding or extent of use of blood products. Note that formal coagulation takes up to 45 minutes to be processed in pathology lab.

7 Record Keeping

In addition to the standard anaesthetic record-keeping, the perfusionists record key events during the operative period. Documentation may be aided by annotating key surgical moments, particularly those commencing the anhepatic phase and heralding revascularisation. Most of key-events are pre-populated under Macro 'Liver Transplantation' on SA Anaesthesia Program.

Standard abbreviations may help:

HA: hepatic artery
PV: portal vein
SHIVC: supra-hepatic inferior vena cava
IHIVC: infra-hepatic IVC
CI: closed
Op: opened

8 PICU Transfer and Handover

This is a standard PICU transfer requiring:

- A call to PICU around 30 minutes prior to expected departure then again as you are leaving the theatre with the patient.
- Consider a full set of blood tests (FBC, EUC, CMP, LFT, COAGS and TEG) at the time of the call so results are available around the time of your arrival.
- Full monitoring for critical care transport - end-tidal carbon dioxide monitoring is mandatory.
- Medications to take with you during transfer.
- Continue all infusions, including electrolyte replacement and maintenance glucose.
- Continue IV fluid replacement – patients are likely to have significant third space losses and drain output. Continuous replacement is necessary.

- Take any syringes for analgesic infusions you have arranged or commence them while still in theatres; this would include wound catheter infusions.

A comprehensive handover is vital. It may be useful to consider the information that needs to be conveyed with a framework like the following:

I	**Intro**	Patient ID, underlying condition, type of donor, CMV.
P	**Preop**	The patient's clinical state before today's operation.
I	**Induction**	What you did at the start of the anaesthetic.
K	**Key events**	Key challenging moments or issues through the case.
S	**Systems**	Each system with issues on the way and current status.
I	**In/Out**	Cover fluids and blood products used plus urine output.
N	**Now/Next**	What's running, analgesia/sedation plan, predicted post-op. issues.

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10 Acknowledgement

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Contributors to the current edition: Dr Jenny Chien, Dr Alice Goldsmith, Dr Ji Young Heo, Dr Jaroslaw Latanik, Dr Albert Shun, Dr Gordon Thomas and Dr Andrew Weatherall.

Appendix 1: Set up and Checklists

Checklist 1: Pre-hospital

☐	Note transplant coordinator's contact number – often they call via switch
☐	Confirm the anaesthetic start time
☐	Confirm the start time with theatre-in-charge
☐	Notify on-call anaesthetic fellow
☐	Notify on-call perfusionist, confirm physiologist been notified
☐	Call 6008 if further review required prior to OT
☐	Notify blood bank to crossmatch 3 units PRBCs, check availability of other products

Checklist 2: Pre-operative Set Up

☐	OT room orientation	☐	Notify on-call radiographer if afterhours
☐	Trauma bed	☐	PRBCs in the theatre fridge
☐	External warming devices	☐	IVC x 3, Arterial lines x 2
☐	Airway setup – nasal ETT, adjuncts	☐	Central venous catheter
☐	Nasogastric tube	☐	Blood giving set and fluid warmer
☐	Indwelling urinary catheter	☐	Cell Saver/Rapid Infuser
☐	Temperature probe	☐	Syringe drivers x 4 +
☐	NIRS or other cerebral oximetry	☐	Ultrasound for vascular access
☐	Medications – see appendix 3		

Appendix 2: CVL size and length guideline

Simple Starting Points for CVC Insertion (RIJ)

Age	Weight [P50th] (kg)	Height [P50th] (cm)	CVC depth guide (cm)#	Line choice
Term to 3 months	3-5	***	6-6.5	4 Fr 3-lumen 8 cm
3 months to < 12 months	5-10	***	6.5-7	4 Fr 3-lumen 8 cm
1 yo	10	75	7	4 Fr 3-lumen 8 cm
2 yo	12	89	8	4 Fr 3-lumen 13 cm
3 yo	14	95	8-8.5	4 Fr 3-lumen 13 cm
4 yo	16	100	8.5	4 Fr 3-lumen 13 cm
5 yo	18	107	9	4 Fr 3-lumen 13 cm
6 yo-9 yo	20-29	115-133	9-11	5.5 Fr 3-lumen 13 cm
10 yo- 12 yo	33-42	138-151	11-12.5	5.5 Fr 3-lumen 13 cm
12 yo-14 yo	42-49	151-160	12.5-14	5.5 Fr 3-lumen 13 cm
14+	50+	160+	14-15 cm	5.5 Fr 3-lumen 13 cm

#: Mean value of the tip reference position between carina and SVC-RA junction from literature search

Note:

- **THIS PROVIDES A GUIDE ONLY**. Additional factors will strongly influence the depth (e.g. pre-term, malnutrition, obesity, phenotypical variation such as long neck or altered thoracic characteristics, clinician's choice of insertion site).
- Final tip position at the level of:
  - Carina: tip is most likely in mid-SVC
  - SVC-RA junction: tip is most likely in low-SVC, small chance that the tip lies within the zone covered by the pericardial reflection (i.e. small risk of tamponade if there is erosion of the vessel wall)
  - Formula to cross-check
    - Patient < 100 cm – depth of insertion = [Height(cm)/10] – 1 cm
    - Patient > 100 cm – depth of insertion = [Height (cm)/10] + 1 cm
- For smaller patients, particularly preterm neonates, there is an additional check that may be helpful:
  - Aspirate the proximal lumen while carefully withdrawing to establish at what point the proximal lumen is no longer within the vessel.
  - If the line is then advanced 1 cm it is likely this will result in a reasonable position of the tip of the line but a relatively low risk of the proximal CVC opening migrating out of the vessel lumen if there is line migration or a change in patient position.

- This guide assumes that the primary use of the CVC is for delivery of critical medications and pressure monitoring.
    - In this case the size of line is adequate for medication delivery and helps reduce risk of line thrombosis.
    - Some clinical scenarios may lead to consideration of choosing a larger diameter line Examples may include difficult IV access preventing placement of adequate peripheral cannulas for delivery of medications or volume resuscitation; or a particularly large number of infusions in the context of metabolic management that leads to consideration of a 4-lumen CVC.

Appendix 3: Liver Transplant Summary Sheet

PATIENT DETAIL
NAME
DOB
MRN
WEIGHT
ALLERGY

ANY SPECIAL REQUIREMENTS
METABOLIC
BEHAVIOURAL +/- PREMED
IMMUNOSUPPRESSION
ANTIBIOTICS

BACKGROUND HISTORY

LAB RESULTS
CONSIDER ORDERING PRODUCTS TO PREPARE FOR NASAL INTUBATION

DONOR DETAIL
START TIME

CONTACT

☐ PERFUSIONIST	☐ THEATRE IN CHARGE	☐ ANAESTHETIC FELLOW
	☐ BLOOD BANK – 3PCS 52284 After hr 6832	☐ RADIOGRAPHER WHEN YOU CALL FOR PT

*Call all of above if there is any change in start time/plan

PREPARATION

ROOM – OT 6 BED – Trauma Bed	Warming – commence in the anaesthetic bay Anaesthetic machine left side, perfusionist right side, operating table head towards the anaesthetic bay, feet towards the exit door	
IV ACCESS	2 X IVC in upper limb – Avoid ACFs 1 in left LSV for venous pressure monitoring CVC – ~1/3 size of Right IJ, at least 3 lumens for CVP, KCL + Glucose, Dopamine	
AIRWAY	Nasal ETT	
Monitoring & Position	Standard monitoring + L art line [monitoring] + R art line [sampling] IDC/TEMP/CXR/NGT/IV warmer + Level 1/NIRS/BSL/KETONE	
Medications	☐ Piperacillin with Tazobactam	100mg/kg/dose q8hr
	☐ Methylprednisone	10mg/kg/dose
	☐ Calcium Chloride 10%	6.8mmol in 10 mL 0.2mL/kg or 0.15mmol/kg/dose
	☐ Anti-thrombin III	1000iu < 30kg, 2000iu 30-60kg, 3000iu > 60kg CALL BLOOD BANK TO ORDER THIS
	☐ Potassium Chloride	20mmols in 40mL 0 – 0.25mmol/kg/hr Commence if K < 3.5mmol/L
	☐ Glucose 50%	Neat: 0.1 – 0.5mL/kg/hr Bolus 0.2mL/kg if BSL < 4.0 CVL only
	☐ Vecuronium	5mg/kg in 50mL: 100microg/kg/hr
	☐ Remifentanil	0.1 – 0.3 microg/kg/min
	☐ Fentanyl	50microg/kg in 50mL: 0 – 10 microg/kg/hr
	☐ Noradrenaline/adrenaline	0.3 mg/kg in 50mL: 0.01 – 0.5 microg/kg/min
	☐ Dopamine	15mg/kg in 50mL: 5microg/kg/min
	☐ Sodium Bicarbonate	1mmol/kg/dose

SURGERY

AIM:
HCT 25 -28 % towards the end, CVP 6 -10mmHg, Euvolaemic, less or controlled bleeding
Normothermic, BSL 6 - 10
FLUIDS: 10ML/KG/HR + Replacement of loss, consider utilising FFP early
POC: TEG (1ml), ABG (0.3ml), DO NOT perform excessive testing if child is stable

PHASE 1 PREPARATION	
☐ Baseline gas/TEG	
☐ Baseline FBC/EUC/LFT/COAGS if not done already, otherwise refer to PowerChart.	
☐ Antibiotics: Piperacillin with Tazobactam 100mg/kg/dose max 4g Repeat every 8 hours	
☐ Connect all infusion and start at low rate	
PHASE 2 INDUCTION AND HEPATECTOMY	
☐ On hour	ABG/BSL/EUC/COAG/TEG
	Repeat if bleeding +, remember calcium
PHASE 3 ANHEPATIC & IMPLANTATION	
☐ Commence Glucose infusion	
☐ 30 min into anhepatic phase	ABG/BSL/EUC
☐ 1 hour	ABG/BSL/EUC/COAG/TEG/FBC
Repeat above if blood loss +	
☐ 10 min before revascularisation	Stop KCL infusion if running & check K level Methylprednisolone - ask surgeon first. Don't give it if blood loss ++
	Basiliximab – only if asked to do so
PHASE 4 REVASCULARISATION	
	Ask surgeon re: Anti-thrombin III
☐ 5 min post	ABG/BSL/EUC
☐ 15 min post	ABG/BSL/EUC/TEG
☐ 30 min post	ABG/BSL/EUC/FBC
☐ 1 hour post	ABG/BSL/EUC/COAGS/TEG/LFT
Repeat above if blood loss +	Remember calcium
SURGICAL BREAK	
☐ Reduce anaesthetic agents	
☐ Call parents	
BILIARY ANASTOMOSIS	
Minor haemodynamic changes	
TRANSFER AND HAND OVER	
Bilateral wound catheter	Use PCA/Regional analgesia paper-chart, order on EMR. Guide for infusion: Bupivacaine 0.125% 0.1 – 0.2mL/kg/hr
Transfer to ICU	Intubated