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CHAPTER 12.9

Pediatric Transplantation

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Pediatric Renal Transplantation

Surgical Considerations

Description: Renal transplantation is the therapy of choice for children with end-stage renal disease providing for freedom from dialysis and improvement in growth. Pre-emptive transplantation is recommended when possible to minimize loss of growth potential, and currently accounts for 25% of transplants. The source of the renal allograft may be a cadaveric (40%), living related, or living unrelated donor. More than 90% of donors are adults.

A mid-line transperitoneal approach is used to transplant an adult-sized kidney (ASK) into a child weighing 20 kg or less. A bilateral nephroureterectomy can be performed through this incision at the same time should it be necessary. A medial visceral rotation is performed mobilizing the right colon and small bowel mesentery to expose the recipient inferior vena cava and abdominal aorta. The donor renal artery and vein are anastomosed directly to the recipient aorta and vena cava respectively. An adult-sized kidney may occupy the majority of the right upper quadrant in a small recipient. Meticulous attention to the positioning of the kidney will prevent kinking or twisting of the donor vasculature. This may require mobilization of the right lobe of the liver or even hepatectomy in some cases. The donor kidney can be temporarily taken out of ice and placed into the recipient to determine the best site for the anastomoses. In this manner the vessel length necessary to fashion straight, yet tension-free anastomoses can be determined. It is important to avoid redundancy in the vessels and ensure a straight lie from the renal hilum to the aorta and vena cava without the hooking of one vessel over another. The venous anastomosis is fashioned first. The vena cava is clamped, and an appropriate size cavotomy is made. The renal vein is sutured to the vena cava in an end-to-side fashion. A small vascular bulldog clamp is then applied to the renal vein above the anastomosis to allow for removal of the vena caval clamp and reconstitution of lower extremity venous return to the heart. Heparin is administered and the aorta is then cross-clamped proximal and distal to the aortotomy. An end-to-side anastomosis is fashioned between the renal artery and aorta, taking care to interrupt the front wall sutures and prevent the purse-string effect of a running suture. Warm ischemia can be minimized during this time by intermittently placing iced slush around the kidney. It is critically important to achieve substantial hypervolemia prior to reperfusion of the ASK because reperfusion will cause an immediate drain of a large portion of a child's relatively small blood volume into the ASK. This necessitates bringing the central venous pressure to approximately 18–20 cm H_2O before reperfusion with a combination of crystalloid and colloid to minimize tissue edema. As prophylaxis against ischemia-reperfusion injury, a single dose of IV mannitol is administered at the time of graft re-vascularization and low-dose dopamine is also initiated. Adequate renal blood flow to an ASK cannot be obtained in children without maintenance of a hypervolemic state, and infants and small children are frequently kept intubated for 24 to 48 hours postop to maintain control of their respiratory function while large volumes of fluid are administered.

The type of ureteral re-implantation depends on the quality of the recipient bladder. An extravesicular ureteral re-implantation can be considered in a healthy bladder of adequate size. This requires distending the bladder with GU irrigant via a three-way Foley catheter. The bladder is reflected medially so as to accomplish the implantation near the postero-lateral portion of the bladder with the ureteral orifice located close to the trigone. The detrusor muscle is divided and a mucosal to mucosal anastomosis is fashioned between the bladder and the donor ureter over a ureteral stent. The detrusor muscle is re-approximated over the ureter for an adequate length to create an anti-reflux valve. If the bladder is of small capacity or defunctionalized, a transvesicular approach to ureteral re-implantation is required. A bladder cystotomy is made at the dome and the transplant ureter is brought into a shallow, mucosa-denuded, rectangular trough extending from a superiorly placed ureteral hiatus distally to the trigone. The ureter is then spatulated and directly sutured to the urinary mucosa over a ureteral stent. The ureteral stent is sutured to a cystotomy tube brought out through a separate incision in the bladder for easy removal of the urethral catheter with its associated discomfort, while ensuring adequate drainage and prevention of clot obstruction. The cystotomy is then closed and the kidney inspected for perfusion and hemostasis. The kidney allograft is then reperitonealized by re-approximating the colon to its lateral attachments and the abdomen is closed in the usual fashion.

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Summary of Procedures

Position	Supine
Incision	Midline laparotomy
Special instrumentation	Self-retaining retractor Vascular instruments Foley catheter (three-way)
Unique considerations	Recipient hypervolemia (CVP 18–20 mm H_2O) Mannitol Dopamine Immunosuppression Potassium free I.V. fluids
Antibiotics	Cephalosporin
Surgical Time	3–5 h
EBL	Minimal
Postop Care	Maintain hypervolemia (CVP > 10 mm Hg) Maintain adequate SBP > 120 mm Hg with dopamine Maintain high UO with a combination of I.V. fluids + Lasix. Goal 150–200 cc/h ICU postop Transplant ultrasound postop
Mortality	< 2%
Morbidity	Primary nonfunction: 2.6% Vascular thrombosis: 10.3% Renal artery stenosis: 0.7% Other technical: 1.3% Bleeding: 2% Wound infection: 5%
Pain Score	7

Anesthetic Considerations for Kidney Transplantation in Infants and Children

Preoperative

Patients presenting for renal transplantation tend to fall into two groups: (a) children in the infant/toddler age group who suffer from congenital syndromes (congenital nephrotic syndrome, severe polycystic kidney disease, obstructive uropathies, FSGS) or (b) the older child who may have a variety of conditions including severe autoimmune nephropathies. These children are often the recipient of a living related transplant and as such, the surgery proceeds as a somewhat elective procedure. Most patients have been on a well-established regimen of peritoneal or hemodialysis.

Airway	Some renal conditions may have associated syndromes and dysmorphia. A thorough airway examination as well as perusal of old anesthetic records should reveal any potential airway problems.
Respiratory	Patients with autoimmune diseases (e.g. lupus) may have pulmonary involvement. Any patient presenting with signs and symptoms of URI should be allowed adequate time for resolution of heightened airway reactivity. Children born with large polycystic kidneys may have pulmonary hypoplasia. **Tests:** Pulmonary Function Tests
	Relatively long-standing hypertension and LVH can be seen in

Cardiovascular	this patient population and may require bilateral nephrectomies prior to or during the transplantation. Adequate control of hypertension should be achieved during the preoperative admission period. **Tests:** EKG, echocardiogram
Gastrointestinal/ Hepatic	Patients with polycystic kidney disease may also have hepatic dysfunction and require future liver transplantation. Patients with congenital nephrotic syndrome tend to be hypoalbuminemic and may be on continuous albumin infusions. **Tests:** Albumin, LFTs, PT, PTT if applicable
Renal	Most patients are well-maintained on hemodialysis or peritoneal dialysis. Other common electrolyte abnormalities are metabolic acidosis, hypocalcemia, and hypermagnesemia. Be aware of current weight relative to dry weight as some patients may present with dehydration in the postdialysis period. **Tests:** Serum electrolytes, BUN and Creatinine
Hematologic	Anemia and platelet dysfunction are commonly seen. **Tests:** CBC, Platelet count
Premedication	Most children will require a premedication to allay separation anxiety. Oral midazolam 0.5–0.75 mg/kg or IV midazolam 0.1 mg/kg (maximum 2 mg) is usually sufficient.

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Intraoperative

Anesthetic technique: GETA. Epidural anesthesia may be a possibility; however, hypotension must be avoided. There are also risks of epidural hematoma due to dysfunctional platelets.

Induction	An inhalational induction with standard monitors will work well in patients without prior IV access. Patients in the infant/toddler age group may have significant venous access problems, and the hemodialysis catheter (if present) may be used provided that sufficient heparinized blood has been aspirated from the dead-space of the catheter lumen. Use of muscle relaxation once IV access has been established is necessary for adequate surgical visualization and mobilization.
Maintenance	Standard balanced anesthetic maintenance. Titration of a long-acting narcotic is recommended although high doses of meperidine should be avoided due to accumulation of normeperidine. Adequate muscle relaxation is especially important in the patient under 20 kg to facilitate surgical exposure. Any coughing or straining can result in vascular injury. Significant hypothermia can be seen when the iced organ is placed in the peritoneum. Tachycardia is frequently seen following reperfusion of the ASK due to this large volume and low-resistance circuit. Urine output should be closely monitored post-reperfusion.
Emergence	Most smaller children will remain intubated in anticipation of large fluid shifts due to volume loading. Older children can be extubated in the OR. All patients are sent to the ICU for monitoring of hemodynamics and urine output.
Management of reperfusion	**Blood and fluids:** Maintenance IV fluids without K Volume loading Albumin loading Blood may be required Warm all fluids **Promoting diuresis**	Preop fluid status is variable. Albumin boluses (10cc/kg) may be needed. Since crystalloid may → visceral edema and complicate surgery. Volume loading for a CVP of 15–20 (or titrated to kidney turgor postperfusion) with albumin, is usually requested by the surgeon in anticipation of reperfusion. PRBC transfusion may be needed if CUP or BP response to albumen is insufficient. Doses of mannitol (0.5 g/kg) and furosemide (1 mg/kg) are also given prior

		to reperfusion.
	Pressor support	Dopamine 3–5 mcg/kg/min is started.
Monitoring	Standard Monitors (see p. D-1) Arterial line CVP (2–3 lumen)	Adequate blood pressure to perfuse the ASK is crucial as is sufficient preload (CVP 10–15) prior to reperfusion.
	Hct/electrolytes	Goal Hct after reperfusion is about 25. (higher Hct can lead to occlusion of the renal artery). Electrolytes must be closely followed especially in the setting of blood transfusion or large volume albumin infusion. Hyperkalemia and hypocalcemia must be corrected.
Urine Output	Goal UOP after reperfusion is about 5–10 mL/kg/h. Urine output must be continually monitored after ureteral implantation.	
PIP	Follow ventilation pressures as patients can go into pulmonary edema from the large volume challenge.	
Temperature control	Hypothermia must be avoided in all children. Placement of the iced donor kidney can drop the temperature by as much as 1.5°C.	
Complications	Ventilatory difficulties Electrolyte abnormalities Hypotension/inadequate renal perfusion Low urine output	

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Postoperative

Complications	Electrolyte abnormalities Ventilatory difficulties Inadequate pain control in the face of hypotension Hemorrhage Clot retention given small catheter size	
Pain Management	Fentanyl 1 mcg/kg iv Small doses of ketamine (eg 1–5 mg iv) can be used for analgesia if patient is intubated	Care must be taken in the administration of iv opioids to prevent hypotension.

Suggested Readings

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Pediatric Liver Transplantation

Surgical Considerations

Description: Liver transplantation was first pioneered in the 1960s as a life-saving experimental procedure for children with end-stage liver disease (ESLD). Since then, it has developed into the accepted treatment modality for pediatric patients with ESLD or fulminant hepatic failure. The indications for liver transplantation in children are broad and range from cholestatic cirrhosis secondary to biliary atresia to inborn errors of metabolism that, if untreated, result in devastating neurological injury.

The current liver allocation system prioritizes pediatric recipients based on a calculated numerical value known as the Pediatric End-Stage Liver Disease (PELD) score. The PELD score was validated as a model for predicting the 3-month wait-list mortality. Although exemption points can be petitioned for on a case-by-case basis, a patient's degree of illness generally correlates with his PELD. Absolute contraindications for liver transplantation include irreversible encephalopathy, uncontrollable infection, and untreatable extrahepatic malignancy. A general contraindication is poor quality of life postoperatively.

The main constraint to pediatric liver transplantation compounding the pre-existing organ shortage involves the donor-to-recipient size ratio. The minimum acceptable graft-to-body weight ratio to provide adequate postop liver function is 1%. However, the suitability of a donor is more often determined by the maximum amount of donor liver that a recipient can accommodate in his abdominal cavity. This results in the utilization of several different types of grafts in pediatric liver transplantation.

Type of Graft	Artery	Portal Vein	Venous Outflow	Biliary Reconstrution
Cadaveric Full Size	Celiac Trunk	Main PV	Full VC	Main HD
Cadaveric Reduced Size	Celiac Trunk	Main PV	Full VC	Main HD
Cadaveric Left Lobe	Common HA	Left PV	Left HV	Left HD
Cadaveric Left Lateral Segment	Common HA	Left PV	Left HV	Left HD
Live Donor Left Lateral Segment	Left HA	Left PV	Left HV	Left HD

Although the type of graft used determines certain technical aspects of the hepatectomy and implantation, the general sequence of events consists of:

Recipient hepatectomy

Anhepatic phase (during which portal venous inflow and hepatic venous outflow are reconstituted)

Allograft reperfusion

Reconstitution of hepatic arterial inflow

Biliary reconstruction

A bilateral subcostal incision is used with a midline subxiphoid extension as needed. The abdomen is explored and adhesions are lysed taking care to suture-ligate varices in patients with portal hypertension. This portion of the procedure may be tedious and bloody in patients with prior liver surgery. The falciform ligament is divided down to the suprahepatic vena cava. The left coronary ligament is divided and the left lateral segment is mobilized from the diaphragm. The lesser omentum is divided and the lesser sac entered. The peritoneum of the hepatoduodenal ligament is divided and a hilar dissection is performed. The connective and vascular tissue of the hepatoduodenal ligament is carefully divided taking care to suture-ligate any varices en masse until the common bile duct is identified. This portion of the procedure may result in significant blood loss in patients with severe portal hypertension. The common bile duct is then suture-ligated, and divided high in the hilum of the liver. The hepatic artery is similarly identified, suture ligated, and divided. The portal vein is then completely skeletonized. The right lobe of the liver is then mobilized. The infra and suprahepatic vena cava are encircled taking care not to injure the right adrenal vein, right renal vein, or inferior phrenic veins. High central-venous pressures during this portion of the procedure can exacerbate portal hypertension and make variceal bleeding difficult to control. However, the patient must have adequate circulatory volume to support the interruption of subdiaphragmatic venous return to the heart without developing vasopressor refractory hypotension. The portal inflow is then occluded with a vascular clamp followed by occlusion of the infrahepatic and suprahepatic *(Print pagebreak 1433)* vena cava. The recipient liver and retrohepatic vena cava are excised, and donor liver implantation begins. In the piggyback technique the liver is completely mobilized from the retrohepatic vena cava by individually ligating the short hepatic veins draining directly from the liver to the cava. The liver is excised with preservation of the retrohepatic vena cava and venous return can be restored prior to implantation by moving the vascular clamp to the junction of the vena cava with the hepatic veins.

The goal of the surgical team during the anhepatic phase is to minimize the duration of caval disruption with its associated intestinal edema and variceal congestion. The hepatic vein outflow is first reconstructed by fashioning the suprahepatic caval anastomosis. In the **piggyback technique,** there is no infrahepatic caval anastomosis and an end-to-end portal vein anastomosis is fashioned next. The liver is flushed with albumin prior to reperfusion to minimize the risk of cardiac arrest 2° ↑ K^+ and ↓ pH. Venous outflow is then re-established by removing the suprahepatic caval or hepatic venous clamp prior to opening the portal venous anastomosis. In a standard procedure the infrahepatic caval clamp is also removed. Reperfusion is the portion of the procedure associated with significant hemodynamic lability, as large volume shifts may occur → pulmonary HTN and RV failure. Alternatively, the release of systemic inflammatory mediators may result in vasodilatation and significant ↓ BP. The anesthesia team needs to be ready to pharmacologically intervene. Hemostasis is achieved prior to arterial reconstruction. Patients who receive cut down or split livers performed ex-vivo may have significant bleeding from the cut surface of the liver.

The donor hepatic artery is typically anastomosed in an end-to-end fashion to the recipient common hepatic artery. In situations of large size discrepancy or poor recipient hepatic artery quality, an anastomosis may be fashioned directly to the supraceliac aorta. This requires aortic cross-clamping with its associated risk of ischemia/reperfusion injury and renal failure. The surgical team should notice the immediate production of bile and resolution of coagulopathy with clot formation in patients with a well functioning graft. The biliary reconstruction can then be performed in an end-to-end fashion if possible but frequently requires a Roux-en-Y reconstruction. If a Roux-en-Y is performed the bowel is divided distal to the ligament of Treitz and the distal limb is brought up to the bile duct. Intestinal continuity is restored with an end-to-side enteroenterostomy and a hepaticojejunostomy is fashioned to the Roux limb. Abdominal drains are typically left along the cut surface in split or cut down livers due to the increased incidence of bile leaks.

The pediatric patient is then taken to the intensive care unit. In patients with end-to-end arterial anastomosis, the patient should be maintained in hypervolemic state to ensure adequate hepatic perfusion in the immediate postop period.

Summary of Procedures

Position	Supine with arms tucked
Incision	Bilateral subcostal with midline subxiphoid extension as needed
Special instrumentation	Thompson liver retractor; Vascular instruments; Argon beam coagulator; Arterial and central venous access (above and below diaphragm); Foley catheter
	Hypothermia requiring covering extremities with plastic to

Unique considerations	minimize convective heat loss, active rewarming Encephalopathy requiring ICP monitoring Hypoxemia secondary to hepatopulmonary syndrome Hyperkalemic cardiac arrest on reperfusion Air embolism on reperfusion RV failure on reperfusion Pressor refractory hypotension with vena caval disruption DIC/fibrinolysis on reperfusion Severe acidosis Renal failure requiring intraoperative CVVH Coagulopathy and hemorrhage requiring massive transfusion Hypoglycemia
Antibiotics	Zosyn (piperacillin + tazobactam) 80 mg of piperacillin component/kg/8h.
Surgical Time	4–6 h
EBL	300–1,500 mL (1–5 U)
Postop Care	Maintain infusion hypervolemia in end-to-end arterial reconstructions. Heparin infusion, Dextran infusion, and aspirin for end-to-end arterial reconstructions ICU postop Monitor closely for signs of bleeding. Monitor closely for acidosis or worsening coagulopathy suggestive of graft dysfunction - Transplant ultrasound postop.
Mortality	2–5%
Morbidity	Primary nonfunction: <5% Portal vein thrombosis: 2–5% early, 10% late Hepatic artery thrombosis: 5–10% Bile leak: 10–20% Bleeding: 10% Wound infection: 5% Reoperation: 18%
Pain Score	10

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Patient Population Characteristics

Age range	0–18 yr
Male:Female	1:1
Incidence	500–600 transplants annually
Etiology	Biliary atresia 35–40%; TPN related 15%; Metabolic diseases 10%; Acute hepatic necrosis 10–15%; Hepatoblastoma 3%; Autoimmune 3%; Congenital hepatic fibrosis 2–3%

Anesthetic Considerations

Preoperative

Indications for liver transplant (LT) in children include (a) progressive subacute or chronic primary liver disease, such as biliary atresia, (b) metabolic disease of the liver, (c) fulminant hepatic failure, (d) hepatic tumors, and (e) retransplantation for hepatic graft failure. The most common disorder for which LT is performed in children is **biliary atresia,** accounting for more than 50% of patients. It is the most common cause of chronic cholestasis in infants and children. For the majority of these patients, a Kasai portoenterostomy is performed in early infancy. Even in those infants in whom bile flow is achieved, however, progressive liver

failure commonly ensues, resulting in cirrhosis, portal HTN, and malnutrition. Complications include coagulopathy, esophageal varices, hypersplenism with splenomegaly and thrombocytopenia, ascites, and growth failure. Recurrent cholangitis following the Kasai procedure may lead to progressive hepatic injury and repeated hospitalizations for IV antibiotic therapy. Children with metabolic liver disease represent the second largest group of patients presenting for LT. Of these, **alpha-1-antitrypsin deficiency** is most prevalent, followed by tyrosinemia and Wilson's disease. LT may also be indicated in patients with metabolic liver disorders presenting solely with extrahepatic manifestations. Oxalosis, or primary hyperoxaluria, is a metabolic liver disease presenting with renal failure due to deposition of oxalate in the renal tubules. These children generally have normal hepatic synthetic function and do not have portal hypertension. Metabolic liver diseases including protein C, protein S, and anti-thrombin III deficiency may present with portal vein thrombosis, portal hypertension, and hypoxemia due to intrapulmonary shunting, and may be treated with LT. Unlike the patients with biliary atresia or alpha-1-antitrypsin deficiency, these children may have normal synthetic liver function and appear relatively healthy.

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Respiratory	Previous prolonged intubation may have caused subglottic stenosis. Ascites, pleural effusions, and hepatosplenomegaly cause a reduction in lung volumes in children with liver failure. Intrapulmonary R → L shunting through abnormally dilated pulmonary arterioles and impaired hypoxic pulmonary vasoconstriction may cause severe hypoxemia. Pulmonary edema may result from hypoalbuminemia and I.V. fluid administration, impairing oxygenation as well as ventilation. Supplemental oxygen and, in advanced cases, mechanical ventilation may be required preop. **Tests:** CXR, ABG in hepatopulmonary syndrome
Circulatory	Chronic liver failure may be accompanied by a hyperdynamic circulatory state with ↓ SVR + ↑ CO. On the other hand, cardiomyopathy and/or pulmonary HTN may be present and CO may actually be diminished. These changes appear to be less common in children than adults. **Tests:** Echocardiogram
Neurologic	Hepatic encephalopathy is a life-threatening complication of ESLD. Causes include accumulation of toxins such as ammonia, GABA agonists, and other neuroactive substances. Cerebral metabolism and the blood-brain barrier may also be abnormal in advanced liver disease. The clinical manifestations of hepatic encephalopathy range from mild somnolence to coma. **Tests:** Head CT to rule out bleed. Rarely, ICP monitoring may be indicated, especially in cases of fulminant hepatic failure.
Hematologic	In addition to the coagulation problems secondary to poor hepatic synthetic function, patients with ESLD may have anemia and thrombocytopenia. Anemia may arise from malnutrition and bleeding. Thrombocytopenia may be a result of splenic sequestration. Both conditions are worsened by dilutional effects from increased plasma volume. **Tests:** CBC, PT/INR/PTT, fibrinogen
Hepatic	Decreased concentrations of the vitamin K-dependent clotting factors II, VII, IX and X may lead to severe bleeding. Deficiency of clotting factors may be caused by hepatic synthetic dysfunction as well as malabsorption of vitamin K 2° ↓ bile salts in the gastrointestinal tract or antibiotic therapy. Hypoalbuminemia contributes to low serum oncotic pressure which predisposes to intravascular hypovolemia, interstitial edema, ascites, and pleural effusions. ESLD results in diminished hepatic drug clearance reduced hepatic blood flow and hepatic extraction ratio. **Tests:** LFTs, albumin, bilirubin, PT/INR/PTT, fibrinogen, NH_3
	Gastrointestinal variceal hemorrhage in patients with portal hypertension is common. Aspiration of blood during an acute

GI	bleed can lead to pulmonary decompensation. Previous need for a Blakemore tube should be noted and one should be available for intraoperative use. Large protuberant abdomens usually warrant rapid sequence or modified rapid sequence induction with rocuronium. Most patients are already on proton pump inhibitors and some will be on an octreotide drip. Octreotide (a somatostatin mimic) is incompatible with most TPN solutions and usually requires a separate IV for infusion.
Renal	Most children with relative long-term hepatic insufficiency coming to transplant have chronically been on diuretic therapy for management of their ascites This can result in severe alteration of electrolytes. Hepatorenal syndrome requiring hemodialysis or CVVH is not uncommon in fulminant hepatic failure. Preop discussion MUST occur with the nephrology team as to the feasibility of intraop CVVH or an intraop dialysis run. For small children with limited vascular access, the dialysis catheter may be needed for fluid resuscitation purposes. Communicate with the blood bank to request both washed units of blood (which take about 1 h to prepare) or freshest units of RBCs. Correction of metabolic acidosis while on dialysis is also preferred. **Tests:** Serum electrolytes, BUN, Cr
Metabolic	Many children with liver failure have severely impaired glycogen metabolism. Preop dextrose infusion (TPN) must be continued intraop. Either the TPN solution or a D25 mixture run at equivalent grams of dextrose per h can be used. Preop consultation with the geneticist is invaluable in the management of complex metabolic disorders. **Tests:** Serum glucose
Premedication	IV midazolam is usually required to facilitate separation from parents. Ketamine 0.5–1 mg/ kg iv is also effective in those children with paradoxical reactions to benzodiazepines.
Preop Prep	Preparation of the room including iv fluids and medications may take 1 h. Underbody forced air warmers are used since the infant is prepped from sternum to pubis. The following drips are routinely prepared: dextrose containing carrier, epinephrine, dopamine, and calcium chloride. Rocuronium and fentanyl drips may be used. The following resuscitation drugs must be readily available at a variety of concentrations; epinephrine, phenylephrine, ephedrine, and atropine. Drugs to treat hyperkalemia should be unit dosed and drawn up, including sodium bicarbonate 1 mEq/kg, calcium chloride 10–20 mg/kg, dextrose with insulin (0.5 g/kg dextrose and 0.2 U/kg insulin). A variety of ET tubes should be ready. Extreme changes in chest wall and pulmonary compliance can occur during the surgery resulting from large volumes of fluids given and changes in chest wall compliance from surgical retraction and placement of a sometimes large organ in the abdomen. A cuffed tube may be invaluable in providing the ability to alter ETT leak to allow for sufficient ventilation at different stages of the operation. Other equipment includes TEE, ultrasound to aid in vascular access, and rapid infusion systems (for larger children and teens).

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Intraoperative

Anesthetic technique: GETA with postop ventilation.

Induction

A modified rapid-sequence induction with rocuronium (0.6 mg/kg) follows placement of standard monitors (see D-1). Provide continuous glucose infusion throughout.

Maintenance

Standard maintenance (see D-3) with volatile agent, muscle relaxation, and fentanyl 10–50 mcg/kg. During periods of instability when vapor concentrations may be low, IV ketamine 1 mg/kg can be used. Nitrous oxide is avoided to prevent bowel distention. PEEP is useful to prevent atelectasis exacerbated by surgical retraction of the diaphragm. Antibiotics are dosed per surgical protocol. ABGs are drawn frequently depending on the degree of blood loss.

Establishment of vascular access can be very difficult and may take 1–2 h. Use of ultrasonic guidance can be helpful especially in the profoundly coagulopathic patient. Care should be taken to keep the patient warm during this period of vascular access. Major vascular access should be restricted to the SVC distribution since the IVC will be clamped. In cases of "piggy-back" transplantation, the caval clamp time is usually reduced and if vascular access is severely limited, IVC distribution veins may be used. This should be discussed with the surgical team.

This phase of surgery begins with surgical incision and concludes when the hepatic/portal vessels are clamped. Opening of the abdomen and drainage of ascites will often improve ventilation. Blood loss during mobilization of the liver may be copious in the presence of portal HTN or adhesions from previous surgeries. "Maintenance fluids" may include FFP and blood. Use of platelets and cryoprecipitate should be discussed with the surgical team as a hypercoagulable state is undesirable when the graft hepatic artery and/or portal vein are small. Hypercoagulability should also be avoided if venovenous bypass is planned. (not commonly used in children <30–40 kg).

Large variations in blood pressure and cardiac filling pressures are common during this phase of surgery due to the manipulation and rotation of the liver. Intermittent kinking of the vena cava can occur. The anesthesiologist must be constantly aware of the progress of surgery and communicate any protracted periods of hypotension due to surgical manipulation.

Preanhepatic phase

Transfusion of blood/products may result in hyperkalemia, hypocalcemia and hypothermia. All products need to be warmed. A calcium chloride infusion (10 mg/kg/h) can be used to maintain the serum ionized calcium >1.0, and helps avoid BP associated with bolus administration of calcium. Hyperkalemia is treated with furosemide (1 mg/kg), correcting acidosis and infusing dextrose and insulin as required. Goals of this stage in preparation for the anhepatic phase include: (a) serum potassium < 4, (b) correction of metabolic acidosis, (c) ionized calcium > 1.0–1.2, (d) adequate urine output (0.5 – 1 mL/kg/h), (e) adequate blood pressure at the lowest CVP possible, (f) hematocrit in the 30 range, and (g) euthermia. Mild hyperthermia may help mitigate the hypothermia created by the iced donor liver during the anhepatic phase. Frequent arterial blood-gas sampling is necessary. Hourly sampling is a minimum, and extreme blood loss may necessitate sampling every 15 min.

Blood loss and third space losses are considerable in this phase of surgery. Edema of internal organs and eventually the transplanted liver is undesirable. Closure of the small abdominal cavity with an edematous liver can cause pressure necrosis as well as abdominal compartment syndrome.

Anhepatic phase

The anhepatic phase begins with clamping of the hepatic vessels and ends with reperfusion of the donor liver. Problems during this phase include fibrinolysis, acidosis, and hypothermia.

Communication must exist between surgical and anesthetic teams regarding the amount of bleeding present and which products should be used to treat it. Respiratory alkalosis may be desirable to compensate for existing or projected metabolic acidosis. Correct any metabolic acidosis with bicarbonate. Placement of the iced donor liver usually decreases the core temperature by 1.5°C. This coupled with pre-existing hypothermia can increase coagulopathic bleeding.

The anhepatic phase can be as short as 15 min or longer than 1 h depending on surgical difficulty. In the case of short anhepatic times, the metabolic goals should have been achieved in the preanhepatic phase.

Anticipation and preparation for reperfusion should be ongoing. A split liver transplant usually entails more bleeding upon reperfusion, so the appropriate blood products must be present in the room. Anticipation and preparation for reperfusion should be ongoing.

Reperfusion takes place after the portal vein and bicaval anastomoses are complete. In the piggy back liver transplant, anhepatic time is reduced since only the hepatic vein(s) and portal vein require anastomosis. Prior to tying the anastomotic suture, the liver is flushed to remove any air from the vessels. A 10-min warning to unclamp is usually given to the anesthesia team. Last minute electrolyte corrections can be made. The patient is then placed on 100% O_2 low dose epinephrine infusion may be started (0.03–0.05 mcg/kg/min), and inhalational agent should be decreased or turned off. IV midazolam or ketamine may be given to ensure amnesia.

Postperfusion phase

This phase begins with slow unclamping of the portal vein and vena cavae with reperfusion of the donor liver. Unclamping may be associated with hemodynamic instability and even cardiac arrest. Hypotension, tachycardia, dysrhythmias, hypothermia, severe acidosis, coagulopathy and air- or thromboembolism can occur. During the unclamping the anesthesiologist must simultaneously watch the uniform reperfusion of the liver as well as the blood pressure and ECG waveform. Increased T waves or widening of the QRS complex should be assumed to represent hyperkalemia. IV fluids in the form of RBCs or albumin should be ready in the event of anastomotic bleeding or bleeding from the cut edge of a split liver graft. A small amount of epinephrine (1–2 mcg) as well as a bolus of calcium chloride (10 mg/kg) may help maintain hemodynamic stability during unclamping. When giving IV fluids watch the liver for swelling. The hepatic artery anastomosis is then completed, prior to which the surgeon may request that a dose of heparin (10 U/kg) be given.

Once the graft is well-perfused and bleeding is controlled, methylprednisolone (15 mg/kg) is given. Care should be taken at this point to avoid hyperglycemia as it can lead to osmotic diuresis and hemoconcentration. Volatile agent can be restarted and the FiO_2 can be reduced. Blood gas tensions and coagulation parameters should be checked. Care should be taken to maintain the hematocrit between 25 and 30, because relative polycythemia may lead to hepatic artery thrombosis.

The biliary system will be constructed either with a duct-to-duct reconstruction, or, in cases of the small child or the patient with biliary atresia, with a roux-en-Y choledocho-jejunostomy. Patients with previous Kasai procedures will have an existing roux limb and biliary reconstruction takes places quickly. Construction of a new roux limb may take 1–2 h. During this time, it is important to keep up with third space losses and avoid hemoconcentration while avoiding congestion of the liver. Vasopressors may be required.

Emergence

The patient is taken to the ICU intubated and usually paralyzed to facilitate ventilation. Extubation is deferred to the ICU team.

Blood and fluid requirements

Massive blood loss expected. IV × 2 as large as tolerated based on age. Arterial line in radial and femoral 2–3 lumen CVP line.	IV access restricted to SVC destribution. Avoid hand veins for large volume fluid administration, because infiltration is difficult to detect. Two arterial lines may be necessary since extreme vasoconstriction may dampen peripheral arterial line; femoral line interrupted if aortic clamping occurs. Avoid IV solutions containing lactate. Normosol preferred over NS due to lower sodium content. Administer blood based on hemodynamics and ABG since EBL is difficult to measure.
Warm all fluids	**Fluids to have available:** Normosol, or normal saline, PRBC, FFP, Cryo and platelets; 5% albumin
UO 0.5–1 mL/kg/h	Rapid infusion system if child is large
Standard Monitors	Five-lead EKG. Bladder temp if child is large enough.
ETN_2 Arterial line × 2	Carefully insert nasopharyngeal temp probe.
I-STAT	I-STAT (bedside arterial blood gas

Monitoring	CVP	sampling) system is useful.
	TEE	Take care during insertion of TEE due to varices. May be difficult to use, because continuous orogastric decompression is needed.
Coagulation management	PT PTT plt count fibrinogen FSP	Pre-existing coagulopathy is very common. Coagulopathy may necessitate administration of products during line placement. Discuss use of cryo and platelets with surgical team prior to administration. Postreperfusion, treat clinical bleeding not lab values.
Positioning	Pad and protect lines Pad pressure points Retraction devices	Wrap all stopcocks near patient. Use gel headrest to protect occiput. Check for pressure points due to surgical retraction devices.
Temperature	Warming blanket Control fluid warmers Wrap patient	Underbody forced air warming. Hotline, Ranger, or rapid infusion system. The patient is wrapped with Webril and saran wrap for warmth and to keep patient dry.
Complications	Coagulopathy Hemorrhage Air embolism Cardiac arrest Metabolic acidosis IV infiltration	

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Postoperative

Monitoring	Serial LFTs PT, PTT Lactate Glucose Bilirubin	Initial LFTs are quite high depending on level of preservation injury. Bile production and normal glucose level are good signs.
Complications	Bleeding Hepatic artery thrombosis Portal vein thrombosis Biliary leak Primary graft nonfunction Rejection Infection Intracranial bleed Electrolyte abnormalities Alkalosis Renal Failure	Most care is deferred to the ICU team. Postop vascular ultrasounds are followed. Hepatic artery thrombosis is ominous and patients are often relisted for transplant if this complication occurs. Hypertensive swings in the coagulopathic patient can lead to intracranial hemorrhage.

Suggested Readings

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