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CHAPTER 7.12

Liver/Kidney/Pancreas Transplantation

Stephan Busque, MD, MSc, FRCSC[1]
Marc L. Melcher, MD, PhD[1]
Dev M. Desai, MD, PhD[2]
Carlos O. Esquivel, MD, PhD[2]
Timothy Angelotti, MD, PhD[3]
Hendrikus J.M. Lemmens, MD, PhD[2]

[1]Kidney, pancreas transplantation

[2]Liver transplantation

[3]Kidney, pancreas transplantation, multiorgan procurement

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Kidney Transplantation—Cadaveric and Live-Donor

Surgical Considerations

Description: Kidney transplantation offers patients with end-stage renal disease (ESRD) freedom from dialysis. The source of the renal graft may be a cadaveric donor, a relative (e.g., parent, sibling) or a genetically unrelated, but emotionally related individual (e.g., spouse).

After induction of anesthesia, a 3-way Foley catheter is placed into the bladder. The kidney allograft is placed in the extraperitoneal iliac fossa. A curvilinear incision is made in the right or left lower quadrant. The retroperitoneal space is developed by retracting the peritoneum medially and cephalad exposing the iliac vessels. A self-retaining retractor is usually placed to maintain exposure. The external iliac artery and vein are identified and surrounding lymphatics are ligated and divided. Several centimeters of the vessels are mobilized. The external iliac vein is clamped first and the renal-vein-to-iliac-vein anastomosis is performed. Then the external iliac-artery-to-renal-artery anastomosis is performed and the clamps are released (Fig. 7.12-1). The patient should be euvolemic at this point; mannitol and/or furosemide can be given. The bladder is filled with an antibiotic irrigation solution to facilitate the implantation of the ureter. The spatulated ureter is anastomosed to the mucosa of the bladder. The detrusor muscle is then re-approximated over 3–4 cm of ureter to create an anti-reflux valve. The wound is closed, normally leaving native kidneys intact.

Variant procedure or approaches: Cadaveric or live-donor transplantation

Usual preop diagnosis: ESRD

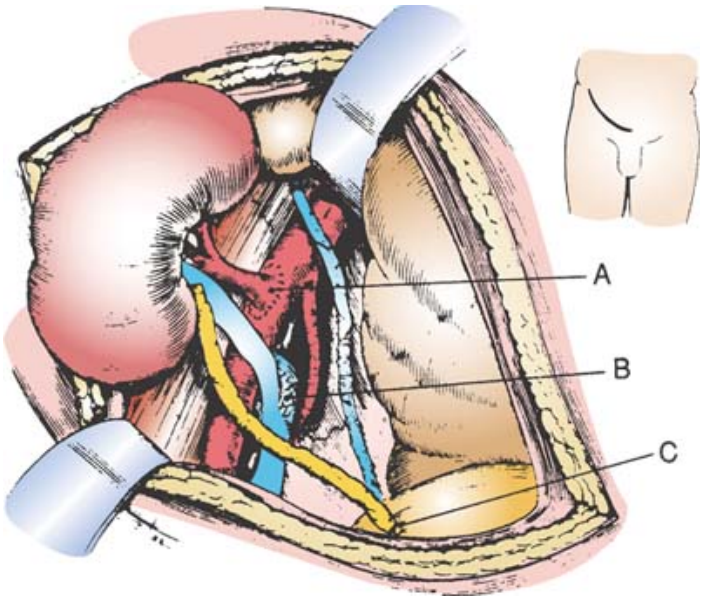

Figure 7.12-1. 1. Kidney transplantation, showing anastomoses of: (A) renal artery to external iliac artery; (B) renal vein to iliac vein; and (C) ureter to bladder. To increase exposure of bladder for a ureteroneocystostomy, antibiotic solution is used to fill the bladder. Lower quadrant curvilinear incision is shown in inset. (Reproduced with permission from Hardy JD: *Hardy's Textbook of Surgery,* 2nd edition. JB Lippincott, Philadelphia: 1988.)

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Summary of Procedures

	Cadaveric Kidney	Live-Donor Kidney
Position	Supine	
Incision	Lower quadrant curvilinear (Fig. 7.12-1 inset)	
Special instrumentation	Self-retaining retractor; vascular instruments; CVP; Foley catheter (3-way)	
Unique considerations	CVP 10–12 mmHg; mannitol 12.5–25 g; intraop immunosuppression before reperfusion (steroids, antilymphocyte preparation); potassium-free iv fluid; protection of shunt or fistula important.	
Antibiotics	Cefazolin 1 g iv, 1 h preop	Cefazolin 1 g iv, 1 h preop
Surgical time	1.5–3 h	2–3 h
EBL	250 mL	
Postop care	Replace UO mL/mL with i.v. fluids; may have delayed graft function 2° prolonged cold storage; ICU selectively	Fluid replacement; delayed graft function unlikely; ICU selectively
Mortality	1–2%	
Morbidity	Lymph or serous leak/stenosis: 3–5% Postop bleeding: 3–5% MI: 2–3% Ureteral leak/stenosis: 2–3% Wound infection: 2–3% Arterial thrombosis: 1–2% Venous thrombosis: 1–2% Wound hematoma: 1–2% Other infectious complications: 15–40%	1–2%
Pain score	5	5

Patient Population Characteristics

Age range	3–70 yr
Male:Female	1:1
Incidence	60/1,000,000
Etiology	Glomerulonephritis (15%); HTN (20%); diabetes mellitus (45%); polycystic disease and others (20%)
Associated conditions	CAD (40%); HTN (25%); uremic and/or diabetic neuropathy (25%); hyperparathyroidism (15–20%)

Anesthetic Considerations

See Anesthetic Considerations following Cadaveric Kidney/Pancreas Transplantation, p. 684.

Suggested Readings

1. Flye MW, ed: *Atlas of Organ Transplantation.* WB Saunders, Philadelphia: 1995.

2. Kahan BD, Ponticelli C: *Principles and Practice of Renal Transplantation.* Martin Dunitz, Ltd., London: 2000.

3. Morris PJ: *Kidney Transplantation.* WB Saunders, Philadelphia: 2001.

4. Morris PJ: Renal transplantation: a quarter century. *Semin Nephrol* 1997; 17:188–95.

5. Odorico JS, Sollinger HW: Technical and immunosuppressive advances in transplantation for insulin-dependent diabetes mellitus. *World J Surg* 2002; 26:194–211.

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Cadaveric Kidney/Pancreas Transplantation

Surgical Considerations

Description: Pancreas transplantation: Combined kidney and pancreas transplantation not only provides renal replacement for the Type I diabetes patient with end-stage renal disease (ESRD), but also controls diabetes. Over eighty percent or more of pancreas transplants are performed in combination with kidney transplantation from the same donor (**simultaneous kidney/pancreas transplant [SPK]**). Pancreas transplantation also can be performed for patients who have already received a kidney transplant (**pancreas after kidney [PAK]**). Less commonly, pancreas transplantation is done for patients with brittle diabetes or with impending complications while they still enjoy normal or near-normal kidney function. Immunosuppression regimen for pancreas transplantation is generally more aggressive than that used for kidney transplantation, and induction therapy with antilymphocyte preparation (ATG, IL-2 blockers, OKT3) is commonly used. The pancreas transplant is placed in the right iliac fossa and the kidney transplant is placed in the left iliac fossa. This can be done through a transperitoneal lower midline incision or through two separate extraperitoneal lower-quadrant incisions in the same manner as kidney transplantation. The graft is first prepared on the back table. For arterial in-flow, a Y-graft is fashioned using the donor iliac artery bifurcation. The portal vein coming off the pancreatic graft is anastomosed to the external iliac vein. The Y extension vascular graft is then anastomosed to the recipient external or common iliac artery. The donor duodenum is anastomosed to a loop of small bowel or to the urinary bladder to drain the exocrine secretions (Fig. 7.12-2 A). With pancreas transplantation, there may be significant blood loss if the graft mesenteric vessels are not occluded properly. After the pancreas is implanted, the kidney transplant is placed into the opposite iliac fossa (as described in Kidney Transplantation, p. 680).

Variant procedure or approaches: The pancreas may be placed in the upper abdomen with the portal vein anastomosed to the superior mesenteric vein. The exocrine secretions are bowel-drained. This more physiologic approach, however, is associated with a higher technical failure rate and requires a long upper midline incision (Fig. 7.12-10). Pancreatic islet cells may be infused via a radiological **portal vein approach,** a procedure that is usually performed in the radiology/angio suite.

Usual preop diagnosis: ESRD 2° diabetes mellitus (DM)

Summary of Procedures

Position	Supine; cushion heels
Incision	Midline or bilateral lower quadrant
Special instrumentation	Thompson retractor; vascular instruments; Foley catheter; NG tube; CVP; arterial line
Unique considerations	Do not correct hyperglycemia < 300 mg/dl. Maintain adequate hydration, CVP 10–12 mmHg; mannitol 0.25–0.5 g/kg on unclamping; intraop immunosuppression before reperfusion (125–250 mg methylprednisolone, monoclonal or polyclonal preparation [e.g., ATG, Zenepax, OKT3, Simulect]).
Antibiotics	Piperacillin (Zosyn) 3.375 g iv q 6 h × 5 d; fluconazole 200–400 mg (nl creatine clearance)
Surgical time	4–6 h
EBL	250–500 mL
Postop care	ICU × 1–2 d, hourly monitoring of glucose; serum glucose should decline by 50 mg/dl each h and remain < 200 mg/dl.
Mortality	2%
Morbidity	Thrombosis of graft: 5–10% Postop bleeding: 5–10% Wound infection: 2–6%
Pain score	7

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Patient Population Characteristics

Age range	15–55 yr
Male:Female	1:1
Incidence	15–20% of all patients with ESRD
Etiology	Type I diabetes
Associated conditions	Retinopathy (100%); uremic and/or diabetic neuropathy (50%); CAD (25–50%); gastropathy (25%); hyperparathyroidism (15–20%)

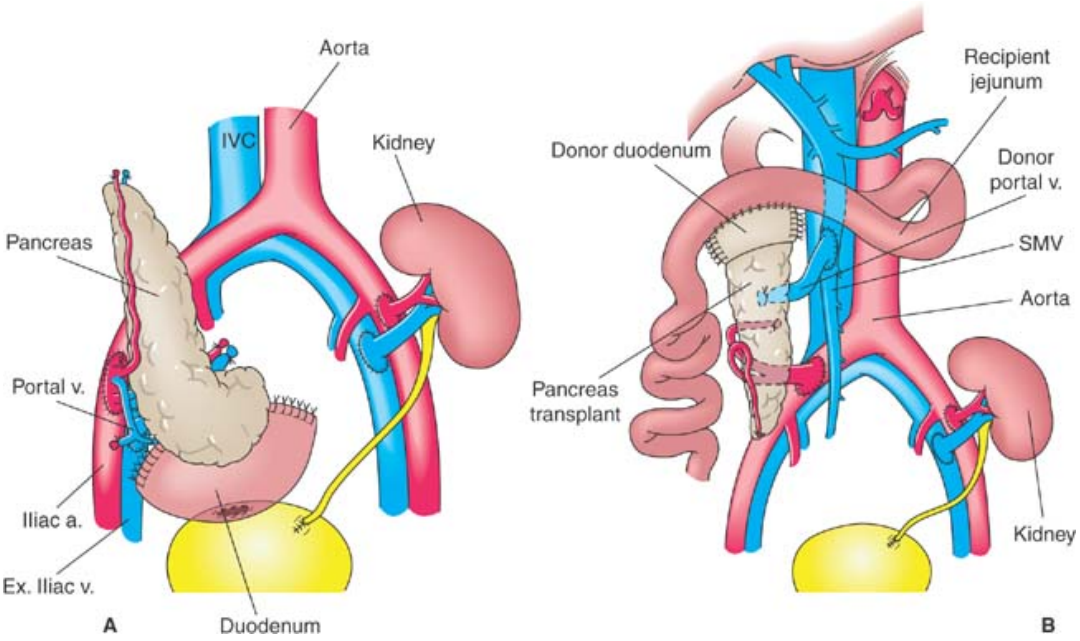

Figure 7.12-2. 2. (A) SPK transplantation, with drainage of pancreatic exocrine secretions into the bladder. Note that portal vein drains into iliac vein (systemic venous [SV] drainage). In normal individuals, 50% of secreted insulin is extracted from the circulation in the first pass through the liver. Transplant recipients with SV have peripheral insulin levels 2-2 × higher than normal. **(B)** SPK transplantation with drainage of pancreatic exocrine secretions into the proximal jejunum (enteric drainage [ED]). This technique has been adopted for SPK by most transplant centers in the U.S. For solitary pancreas transplantation, most centers still utilize ED to allow monitoring of the urinary amylase. Note that donor portal vein drains into the recipient superior mesenteric vein (portal venous [PV] drainage) preventing peripheral hyperinsulinemia. Most centers continue to place the pancreas in the pelvis, combining ED and SV, which requires enteric anastomosis to a more distal segment of jejunum or ilium. (Reproduced with permission from Greenfield LJ, et al, eds: *Surgery: Scientific Principles and Practice,* 3rd edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

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Anesthetic Considerations for Kidney and Kidney/Pancreas Transplantation

Preoperative

Typically, patients presenting for renal transplantation fall into two patient populations: (1) the young and relatively healthy (following dialysis), or (2) an older, more chronically ill group. Rarely, patients will present for transplant surgery without adequate preparation (e.g., ↑K^+, ↓pH, hypervolemia). Patients presenting for pancreas transplantation are usually severe diabetics with many of the associated problems, such as CAD, autonomic neuropathy, gastroparesis, and stiff-joint syndrome (difficult intubation).

Respiratory	Pleuritis and pleural effusions may occur in this patient population. Increased susceptibility to infection is common in the patient with chronic uremia.
Cardiovascular	Pericarditis (acute or constrictive), HTN, CHF, dysrhythmias, pericardial effusion are common, especially in the undialyzed patient. Diabetes, a common cause of ESRD, is often associated with PVD, CAD, and autonomic neuropathy. **Tests:** ECG (rhythm, CAD, electrolyte abnormalities, pericarditis, LVH). Other tests (ECHO, stress, etc.) as indicated from H&P.
Gastrointestinal	Gastroparesis may occur, especially in diabetic patients with autonomic neuropathy. Consider full stomach precautions. Ranitidine (50 mg iv) and metoclopramide (10 mg iv) should be given 60 min preop to aid gastric emptying and ↓ acidity. Na citrate (30 mL, 0.3 M po) should be given immediately before

	induction.
Renal	Patients usually on dialysis. Postdialysis goals include: K^+ = 4–5 mEq/L, BUN < 60 mg%, creatinine < 10 mg%. Metabolic acidosis, hypocalcemia, and hypermagnesemia often present, and may require preop correction. Patient may be hypovolemic following dialysis; pre- and postdialysis weight (> 2 kg loss is significant). Rapid correction of severe hyperkalemia can be achieved by giving 50 mL of 50% glucose iv, together with 10 U regular insulin and 50 mEq NaHCO. Further correction can be obtained by coadministration of an inhaled β-agonist (e.g., albuterol) (5–10 puffs). **Tests:** Cr; BUN; creatinine clearance; electrolytes
Hematologic	These patients frequently anemic. Preop correction usually not needed. A coagulation disorder may be present with abnormal Plt function (improved by dialysis) as well as thrombocytopenia, resulting in a prolonged bleeding time. There is a high incidence of posttransfusion hepatitis in this patient population. **Tests:** Hct; PT; PTT; Plt count; hepatic screen, consider platelet function assay.
Endocrine	Asses glycemic control preop. Individualize diabetic rx for day of surgery. In general, hold oral agents 12–24 hours, and ↓or hold usual insulins, and consider sliding scale insulin day of surgery. Favor hyperglycemia (≥ 120–200 mg/dl) over tight glucose control. Preop/intraop corticosteroid immunosuppressive Rx will likely → hyperglycemia. Postop glycemic management complex and dynamic. **Tests:** Serial glucose levels
Neurologic	Peripheral neuropathy may occur and specific deficits should be documented. Autonomic neuropathy can → cardiac problems (e.g., orthostatic hypotension, ↑HR, or ↓HR), silent MI, and GI problems.
Premedication	Consider midazolam 1–2 mg iv.

Intraoperative

Anesthetic technique: GETA favored. Spinal, epidural, or combined spinal-epidural anesthesia may be considered for renal transplantation, if coagulation and platelet function acceptable. Avoidance of hypotension after organ(s) transplanted an important consideration which may limit effectiveness of RA techniques.

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Induction	Rapid-sequence induction (see p. B-4). ET intubation is aided by succinylcholine (1 mg/kg), if K^+ < 5.5 mEq/L; otherwise, use cisatracurium (0.2–0.5 mg/kg) or rocuronium (1.2 mg/kg). Fentanyl (2–5 mcg/kg) may be used to suppress the cardiovascular response to intubation.
Maintenance	Standard maintenance (see p. B-2). Maintain muscle relaxation with cisatracurium or rocuronium, titrated to effect using a nerve stimulator. Avoid meperidine (accumulation of normeperidine → CNS toxicity). Anticipate prolonged drug effects, and avoid agents that are primarily excreted by the kidney.
Emergence	Usually extubated in the OR after protective laryngeal reflexes have returned. Ensure adequate reversal of NMB's drugs. Pancreatic transplant patients (e.g., brittle diabetics, hemodynamically unstable) are sent to the ICU for close glycemic management.
Blood and fluid requirements	IV: 14–16 ga × 1 NS/colloid to keep CVP = 10–15 mmHg Warm fluids Preop fluid status is highly variable (hypo →hypervolemia). Give fluids to maintain CVP 10–15 mmHg. Important to maintain adequate vascular volume and BP. Mannitol (0.25–1 g/kg), furosemide (5–20 mg), and low-dose dopamine are often

		given with reperfusion of the kidney.
Monitoring	Standard monitors (see p. B-1). Arterial line CVP/PA line	Arterial pressure is often monitored. Avoid the side of AV fistulae. Axillary artery is a useful alternative. CVP is essential and a PA line is needed occasionally (severe cardiac disease). CVP is kept at 10–15 mmHg, especially after the new kidney is reperfused, to ensure adequate renal blood flow.
	Hct, K+, and glucose	In pancreatic transplant patients, glucose should be checked q 30 min and then q 10 min for the first h following reperfusion. Keep glucose < 300 mg/dl prior to reperfusion, but do not fully correct to < 150 mg/dl.
	Neuromuscular	Monitor neuromuscular block to avoid excessive use of neuromuscular relaxants; anticipate prolonged effects.
Positioning	and pad pressure points eyes	
	Protect/pad AV fistulas	Pressure on AV fistula may lead to thrombosis. Carefully pad and protect.
Complications	Disruption of renal anastomoses Hemorrhage Low UO Reperfusion injury (pancreas transplant)	Bucking or coughing during emergence, due to inadequate neuromuscular blockade, may → forceful tugging on transplanted kidney. This "popping" of kidney may → disruption of venous and arterial anastomoses and possible ischemic damage requiring urgent surgical revision.

Postoperative

Complications	Fluid overload and CHF Femoral neuropathy Hemorrhage Electrolyte abnormalities	Monitor UO. Dialysis may be needed until renal function returns. Sudden cardiac arrest can complicate pancreatic transplantation (due to autonomic neuropathy).
	Hypo/hyperglycemia	Especially dynamic in pancreas tx
	PONV	see p. B-6
	VTE	see p. B-7
Pain management	PCA (see p. C-3) Epidural	Anticipate prolonged effect of some opiates. Strive to avoid hypotension.
Tests	Hct Electrolytes Cr, BUN Amylase Glucose	A rise in amylase and blood glucose may indicate failure of the pancreatic transplant.

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Suggested Readings

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2. Halpern H. Miyoshi E. Kataoka LM. Khouri Fo RA. Miranda SB. Marumo CK. Omati O. Genzini T. Miranda MP. Anesthesia for pancreas transplantation alone or simultaneous with kidney. *Transplant Proc* 2004; 36(10):3105–6.

3. Lemmens, HJM. Kidney transplantation: recent developments and recommendations for anesthetic management. *Anesthesiol Clin North Am* 2004; 22(4):651–62.

Live-Donor Nephrectomy—Laparoscopic and Open

Surgical Considerations

Description: Use of a kidney donated by a healthy genetically or emotionally related donor greatly increases the number and quality of kidneys available for transplantation. Kidney transplantation from living donors is associated with a better patient and graft survival rate. **A laparoscopic (LSC) approach** for kidney donation was introduced in 1995 as an alternative that would reduce postop pain, wound morbidity, and recovery time associated with open nephrectomy. Now the LSC approach is the procedure of choice for more than 75% of the live kidney donations in the United States. Initial concerns regarding ureteral complications and longer warm ischemic time have mostly subsided with the improvement of the surgical technique and greater experience. The left kidney is preferred for the LSC approach, as the renal vein is longer. Some centers use the LSC approach for the right kidney with comparable results.

The patient is positioned in lateral decubitus over a cushioned beanbag, the kidney rest is slightly elevated, and pillows and an axillary roll are used to prevent compression injuries. Three or four ports are used. The pneumoperitoneum is kept < 15 mmHg to avoid decreased perfusion to the kidney. Aggressive hydration and intermittent use of IV mannitol help improve kidney perfusion. On the left side, the descending colon and spleen are mobilized medially; the renal vessels are exposed; the adrenal, lumbar, and gonadal veins are clipped and divided; the ureter is mobilized en bloc, along with the gonadal vein, down to the pelvic inlet. The artery is freed from surrounding lymphatic and neural tissue as it comes off the aorta (Fig. 7.12-3). Gerota's fascia is mobilized to completely free the kidney. The ureter is transected distally. A 6-cm suprapubic incision is made, the peritoneum is exposed in the midline, and an 18-mm port is used to insert a 15-mm Endocatch retrieval bag. The kidney is placed in the bag as it continues to be perfused, avoiding warm ischemia. The patient is fully heparinized. An endo GIA vascular stapler is used to staple and transect the artery close to the aorta and the vein close to the vena cava. An endo TA vascular stapler can also be used. It may add safety and vessel length. The retrieval bag is brought to the suprapubic incision and gently extracted. The kidney is immediately immersed in the cold slush solution, and the staple lines are cut off the renal artery and vein. The kidney is perfused with preservation solution in the usual manner. The heparin is reversed with protamine, the suprapubic incision is closed, and homeostasis is verified before extracting the ports. For a right nephrectomy, the right colon and duodenum are mobilized medially and the liver is retracted upward. The remainder of the operation is as described for the left kidney. The surgeon's hand may be inserted in the abdomen to help with the stapling of the vessels and kidney retrieval.

The **hand-assisted laparoscopic donor nephrectomy** is similar to the pure laparoscopic approach described earlier; however, a midline 8–10-cm incision is made at the level of the umbilicus or infra-umbilical to position a device (e.g., *(Print pagebreak 687)* Gelport, Lapdisc) that allows the surgeon to put one hand inside the abdomen without losing the pneumoperitoneum. The hand is used to help with retraction and exposure to the kidney. The warm ischemia time (clamping of the renal artery to perfusion) is reduced 50%. Operative time also may be reduced. This approach requires a longer abdominal incision and could be associated with slightly more wound complications than the pure laparoscopic approach. The hand-assisted approach has gained popularity over the years and it is now the preferred technique of a majority of Transplant Centers in the United States.

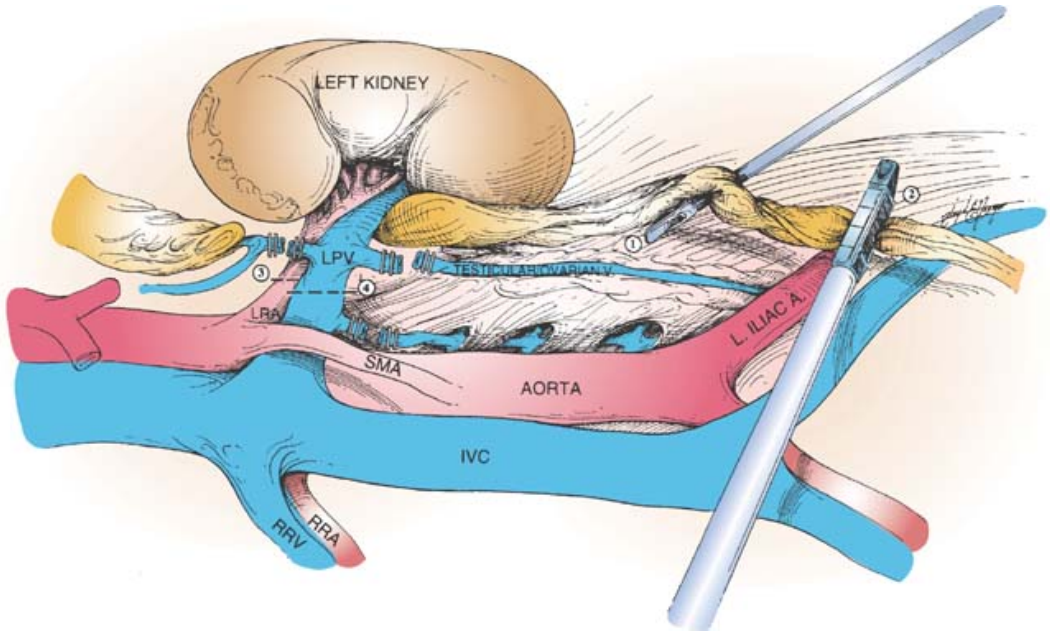

Figure 7.12-3. 3. Anatomy for laparoscopic live-donor nephrectomy. (Reproduced with permission from Cho ES, Flowers JL: Laparoscopic live-donor nephrectomy. In *Surgical Laparoscopy,* 2nd edition. Zuker KA, ed. Lippincott Williams & Wilkins, Philadelphia: 2001.)

In **open nephrectomy,** the **donor/patient** is placed in a lateral decubitus position on a flexible OR table with a kidney rest. A beanbag or sandbags are also helpful for positioning. An incision is made from the rectus muscle, angling slightly cephalic to cross into the flank just below the tip of the 12th rib. The retroperitoneum is exposed using a Thompson retractor. The kidney is then mobilized and the ureter is transected. A clamp is placed across the renal artery at the aorta and the renal vein at the IVC. Just before clamping the renal artery, furosemide and/or mannitol may be given to stimulate diuresis. It is important to keep the vascular volume expanded in these patients before kidney removal. The kidney is removed and taken to the back table where it is flushed with a cold preservation solution. It is then transported into the recipient room for reimplantation. Some surgeons use a full dose of heparin (75 U/kg) before clamping and use protamine afterwards. Smaller incisions and muscle-sparing incisions are now used to improve postop recovery.

Usual preop diagnosis: Donor nephrectomy

Summary of Procedures

	Laparoscopic Nephrectomy	Open Nephrectomy
Position	Lateral decubitus	
Incision	3–4 ports; retrieval incision, Pfannenstiel's or vertical suprapubic	Flank; may require 12th rib resection
Special instrumentation	Foley catheter; flexible OR table with kidney rest; beanbag; SCDs for DVT prophylaxis; harmonic scalpel	Foley catheter; Thompson retractor; flexible OR table with kidney rest; beanbag or sandbags; SCDs for DVT prophylaxis
Unique considerations	Avoid ETT dislodgement when turning patient from supine to flank position; vigorous hydration.	Possible pneumothorax; avoid ETT dislodgement when turning patient from supine to flank position. Vigorous hydration to encourage urine production.
Antibiotics	Cefazolin 1 g iv	Cefazolin 1 g, iv
Surgical time	2.5–4.5 h	2–2.5 h
Closing considerations	–	Delfex table to facilitate closure
EBL	Minimal	100 mL
Postop care	PACU → room; PCA for pain management	CXR to r/o pneumothorax. Epidural or PCA (morphine) is helpful for pain management. PACU → room.

Mortality	< 0.1%	
Morbidity	Ileus: 2–5% Urinary retention: 2–5% Wound infection: 1–2% Bleeding: 0.1–0.5%	5–10% 5–10% 1–3% Pneumothorax: 1%
Pain score	3–5	7

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Patient Population Characteristics

Age range	18–70 yr
Male:Female	1:1
Incidence	Up to 50% of all kidney transplants at some centers
Etiology	N/A
Associated conditions	Good health is mandatory for renal donation.

Anesthetic Considerations

See Anesthetic Considerations following Kidney Transplant Nephrectomy, p. 690.

Kidney Transplant Nephrectomy

Surgical Considerations

Description: With improvements in graft survival and immunosuppressive therapy, the necessity of removing a kidney transplant graft for uncontrolled rejection has decreased significantly. This operation is divided into categories: **early nephrectomy,** performed during the first month post-transplant, and **late nephrectomy,** thereafter. **Early transplant nephrectomy** may be required for primary nonfunction, vascular thrombosis, and, rarely, refractory rejection. In these cases, an **extracapsular approach,** through the original transplant incision, is used. The *(Print pagebreak 689)* kidney is freed up from the surrounding adhesion to obtain vascular control of the renal artery and renal vein. These structures are clamped and oversewn individually. The ureter is ligated as close as possible to the bladder and excised completely, with primary repair of the bladder. A suction drain is used if minimal oozing or lymph drainage is present. **Late transplant nephrectomy** is performed most commonly for acute, irreversible rejection with failure of the renal allograft. Most of these patients have returned to dialysis and the immunosuppressive medications are stopped. Chronic infection and HTN associated with nonfunctional grafts are also an indication for surgical removal of the kidney allograft. It may be a difficult operation, as intense inflammatory adhesions are present between the renal capsule and the surrounding tissue. In the setting of acute late rejection, the graft is usually swollen and enlarged. Hematuria may be present, and the graft is friable. Spontaneous rupture and hemorrhage have been reported. The surgical approach is through the same incision as the implantation. In contrast to early transplant nephrectomy, extracapsular dissection may not be possible with late nephrectomy. To avoid injury to extrarenal structures, such as the iliac vessels, an **intracapsular approach** may be preferred. The kidney is mobilized gently from within the capsule toward the hilum. The capsule is reopened on the medial side to have access to the renal vessel high in the hilum. When the hilum is sufficiently mobilized, a strong vascular clamp is applied high in the hilum of the kidney away from the iliac vessels. After clamping the hilum en bloc, confirmation of distal pulses is obtained; then the kidney is excised over the vascular clamp. A running suture is used over the clamp, which is then released, and hemostasis is obtained. The ureter is identified and excised as close as possible to the bladder. The intracapsular dissection of the kidney may be associated with significant bleeding, as the kidney may fracture. This step should be done expeditiously to avoid excessive bleeding. The patient must have good vascular access for fluid resuscitation. Blood must be available for transfusion. After hemostasis is obtained, a low-pressure suction drain may be placed before closing.

Usual preop diagnosis: Transplant rejection

Summary of Procedures

Position	Supine
Incision	Previous incision used for kidney transplant
Instrumentation	Self-retaining retractor; vascular instruments
Unique considerations	Large-bore vascular access; PRBCs available; stress dose of steroids, if chronic usage; protection of shunt or fistula
Antibiotics	Cefazolin 1 g iv
Surgical time	1–2.5 hr
EBL	200–1000 mL
Postop care	PCA for pain management → PACU → room
Mortality	1–3%
Morbidity	Overall: 3–5% Wound infection: 3–5% Abscess formation: 1–2% Exsanguinating hemorrhage: < 1%
Pain score	5

Patient Population Characteristics

Age range	3–75 yr
Male:Female	1:1
Incidence	60/1,000,000
Etiology	Failed kidney transplant
Associated conditions	CAD (40%); HTN (25%); uremic and/or diabetic neuropathy (25%); hyperparathyroidism (15–20%)

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Anesthetic Considerations for Live-Donor and Posttransplantation Nephrectomy

Preoperative

In order to be a live donor, one must be in good health with bilaterally functional kidneys. Diabetes, HIV infection, liver disease, and malignancy are all contraindications to kidney donations.

Cardiovascular	Assess for HTN and CAD
Renal	Normal bilateral renal function is required. **Tests:** IVP; Cr, creatinine clearance
Fluid status	Adequate hydration is important and UO should be >1.5 mL/kg/h. Various regimes are used to ensure adequate hydration, usually with iv fluid starting the night before.
Premedication	Consider midazolam 1–2 mg iv. Organ donors are making a great sacrifice and should be treated with special care. Standard premedication (see p. B-1).

Intraoperative

Anesthetic technique: GETA ± epidural for postop pain management. Epidurals are not necessary for most laparoscopic donor nephrectomies.

Induction	Standard induction (see p. B-2).

Maintenance	Standard maintenance (see p. B-2). Avoid long-acting, renally excreted drugs. Ventilate to maintain normocapnia to avoid possible renal artery vasoconstriction. Use of an epidural with local anesthetic and/or narcotic may aid both intraop and postop pain relief, but ↓BP should be avoided.	
Emergence	Routine extubation in OR	
Blood and fluid requirements	IV: 14–16 ga ×2 NS/LR @ 6–8 mL/h Warm fluids UO ≥ 1.5 mL/kg/h	Aim for a minimum of 1.5 mL/kg/h UO. Mannitol (0.25–1 g/kg) given iv once kidney is being manipulated, and if UO decreases. Consider dopamine infusion to ↑ BP as needed. Limit use of direct vasoconstrictors.
Monitoring	Standard monitors (see p. B-1).	CVP or invasive arterial monitoring are rarely required. Lower extremity NIBP measurements on the same side as implant will be inaccurate during period of iliac vessel clamping.
Positioning	and pad pressure points eyes	
Complications	Hemorrhage Pneumothorax	Because the vessels are tied close to the aorta and IVC, the possibility of severe hemorrhage exists. Pneumothorax is always possible, especially when the 12th rib is resected.

Postoperative

Complications	Pneumothorax/pulmonary problems Hemorrhage Infection PONV (see p. B-6) Electrolyte abnormalities VTE (see p. B-7) Ileus
Pain management	Epidural narcotics (see p. C-2). Avoid hypotension. PCA (see p. C-3).
Tests	CXR Hct. electrolytes, BUN/Cr

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Liver Transplantation

Surgical Considerations

Description: Liver transplantation is the treatment of choice for patients with acute and chronic end-stage liver disease (ESLD). Patients with ESLD, besides intrinsic liver dysfunction, also may have other organ system dysfunction, including hepatorenal and hepatopulmonary syndrome resulting in oliguria and hypoxia, respectively. Patients with alcohol-mediated cirrhosis and Wilson's disease are at risk for significant cardiomyopathy, while those with fulminant hepatic failure may have significantly elevated intracranial pressures. These additional comorbidities present an added level of complexity to the anesthetic and surgical management of the liver transplant recipient. The liver transplant operation can be divided into three stages: **(1) hepatectomy; (2) anhepatic phase**, which involves the implantation of the liver; and **(3) postrevascularization,** which includes hemostasis and reconstruction of the hepatic artery and common bile duct.[16] There are many variations in the technical aspects of the liver transplant operation that may result in physiologic changes during anesthesia. The anesthesiologist must be aware of these technical variations to optimize the intraoperative management of the liver transplant recipient. Examples of these variations include: cross-clamping of the vena cava during the implantation of the liver, which results in impairment of the systemic venous return, with possibility of profound hypotension; utilization of the venovenous bypass, which may be associated with thrombus or air embolism, and/or fibrinolysis; and the use of a “cutdown liver,” which may result in significant bleeding from the cut surface following revascularization.

The **hepatectomy** may be a formidable task in patients with severe portal hypertension (HTN), coagulopathy, and previous surgery in the upper abdomen. In such circumstances, blood loss is significant and may be minimized by placing the patient on venovenous bypass or by creating a temporary portocaval shunt to relieve the portal HTN. Table 7.12-1 lists factors that may be associated with significant blood loss during the transplant operation. The hepatectomy is usually much easier in patients with acute fulminant hepatitis, primary biliary cirrhosis, or inborn *(Print pagebreak 692)* errors of metabolism than in patients with shrunken cirrhotic livers, such as in postnecrotic cirrhosis from hepatitis B or C, alpha-1 antitrypsin deficiency, or Wilson's disease, among others. The subcostal incision usually extends from the left midclavicular line across the midline to just medial of the right 12th floating rib, along with a vertical midline extension from the xiphoid process to the transverse incision. This provides wide exposure to the upper abdomen.

Table 7. 12-1. Contributing Factors Associated with Increased Blood Loss in Liver Transplantation

1. Severe coagulopathy
2. Severe portal HTN
3. Portal or splenic vein thrombosis
4. Previous surgery in the RUQ
5. Renal failure
6. Uncontrolled sepsis
7. Retransplantation
8. Transfusion reaction
9. Venous bypass-induced fi brinolysis
10. Primary graft nonfunction
11. Intraop vascular complications

The hepatectomy usually begins with manual exploration of the abdomen to ensure that there are no occult malignancies, abscesses, or other abdominal processes that may contraindicate proceeding with the transplant. The liver is then mobilized by freeing the falciform and left cardinal ligaments, followed by entering the lesser sac through the division of the gastrohepatic ligament. The mobilization of the liver and the subsequent dissection of the portahepatis may be significantly complicated and a tedious process due to large, thin-walled varices that require careful dissection and ligation. The dissection of the portahepatis begins with identification and ligation of the hepatic artery, followed by the common bile duct. The portal vein is carefully dissected from its bifurcation into left and right branches, proximally to its emergence from behind the pancreas. If the degree of portal HTN is severe—such that mobilization of the liver may result in significant blood loss—or the patient is hemodynamically unstable—then portal vein mobilization may be performed early so that a temporary portocaval shunt or venous bypass may be instituted to allow decompression of the varices and enhance venous return to the heart.

After the portal dissection is complete, the right lobe of the liver is mobilized. The infrahepatic vena cava is carefully dissected to prevent injury to the right renal and adrenal veins, followed by mobilization of the suprahepatic vena cava. The liver can be

removed easily by cross-clamping and dividing the supra- or infrahepatic vena cava, with or without the use of venous bypass. Alternatively, the recipient vena cava may be left in situ (piggy-back technique) by further mobilization of the liver with division of the short hepatic veins that run from the anterior surface of the vena cava directly into the posterior aspect of the liver. To gain access to the short hepatic veins, the liver must be lifted and rotated to the left. This maneuver may result in partial occlusion of the inferior vena cava, which may impair venous return causing a temporary drop in blood pressure. The piggyback technique, where the recipient vena cava is left in situ, has the advantage that venous return is not compromised during the anhepatic phase and thus precludes the need for venous bypass.

The **anhepatic phase** may be associated with significant hemodynamic changes, depending on the technique used for vascular control. This stage of the operation consists of implantation of the liver allograft, with or without venous bypass. The use of venous bypass is particularly helpful in coagulopathic patients with severe portal HTN. In these high-risk patients, the goal of the venous bypass system is to relieve the portal HTN by "bypassing" the liver.[13] Cannulas, placed in the femoral and portal veins, draw the blood out of the systemic and splanchnic venous systems into a Biomedicus pump that delivers the blood into the axillary or jugular vein, maintaining the venous return (Fig. 7.12-4). This system allows the interruption of the vena cava with mild-to-moderate hemodynamic changes, depending on the blood flow rate through the system. The benefits and potential complications of the venous bypass system are listed in Table 7.12-2.

Wound complications and nerve injuries may be prevented by introducing the bypass cannulas percutaneously, rather than approaching the vessels through a surgical incision. A subclavian or IJ line may be placed preop, and can be easily and rapidly exchanged during the operation to bypass cannulas using the **Seldinger technique.** If lines are placed preoperatively for the specific purpose of venous bypass, a confirmatory CXR should be performed to ensure that the 15 Fr or larger bypass cannula will lie in the appropriate vessel when placed later in the operation. This also obviates the need for a CXR when the bypass cannula is placed later, when the patient may be unstable. Because of the potential complications, several transplant teams have opted not to use venous bypass. In these cases, vascular control is obtained by placing vascular clamps across the supra- and infrahepatic vena cava or the confluence of the hepatic veins (piggy-back technique) and the portal vein. The splanchnic venous return is interrupted during the anhepatic phase while the systemic venous return is either interrupted in the case of formal cross-clamping of the supra- and infra-hepatic vena cava or mildly diminished in the case of the piggy-back technique which can lead to significant hypotension unless preventive measures, as reviewed in Anesthetic Considerations (p. 699), are taken (Fig. 7.12-5).

In a **standard orthotopic liver transplant,** with or without venous bypass, the recipient's vena cava is removed, leaving two cuffs—one just below the diaphragm and the other above the entry of the renal veins. A cadaveric donor liver comes with the corresponding segment of the vena cava that is used for restoring the continuity of the recipient's vena cava. The first vascular anastomosis consists of an end-to-end anastomosis of the allograft suprahepatic vena cava and the cuff of the recipient's infradiaphragmatic vena cava. This is followed by the reconstruction of the infrahepatic vena *(Print pagebreak 693)* cava with an end-to-end anastomosis. Immediately prior to completion of the infrahepatic vena caval anastomosis, the liver is purged with chilled or room temperature albumin and/or crystalloid solution via the allograft portal vein to remove the University of Wisconsin (UW) preservative solution, which contains 145 mEq/L K^+. Additionally, flushing the liver also removes a significant amount of the air that gets introduced during the procurement and preparation of the allograft for transplantation. Finally, the portal vein reconstruction is completed with an end-to-end anastomosis. At this point, the clamps are removed, ending the anhepatic phase of the operation.

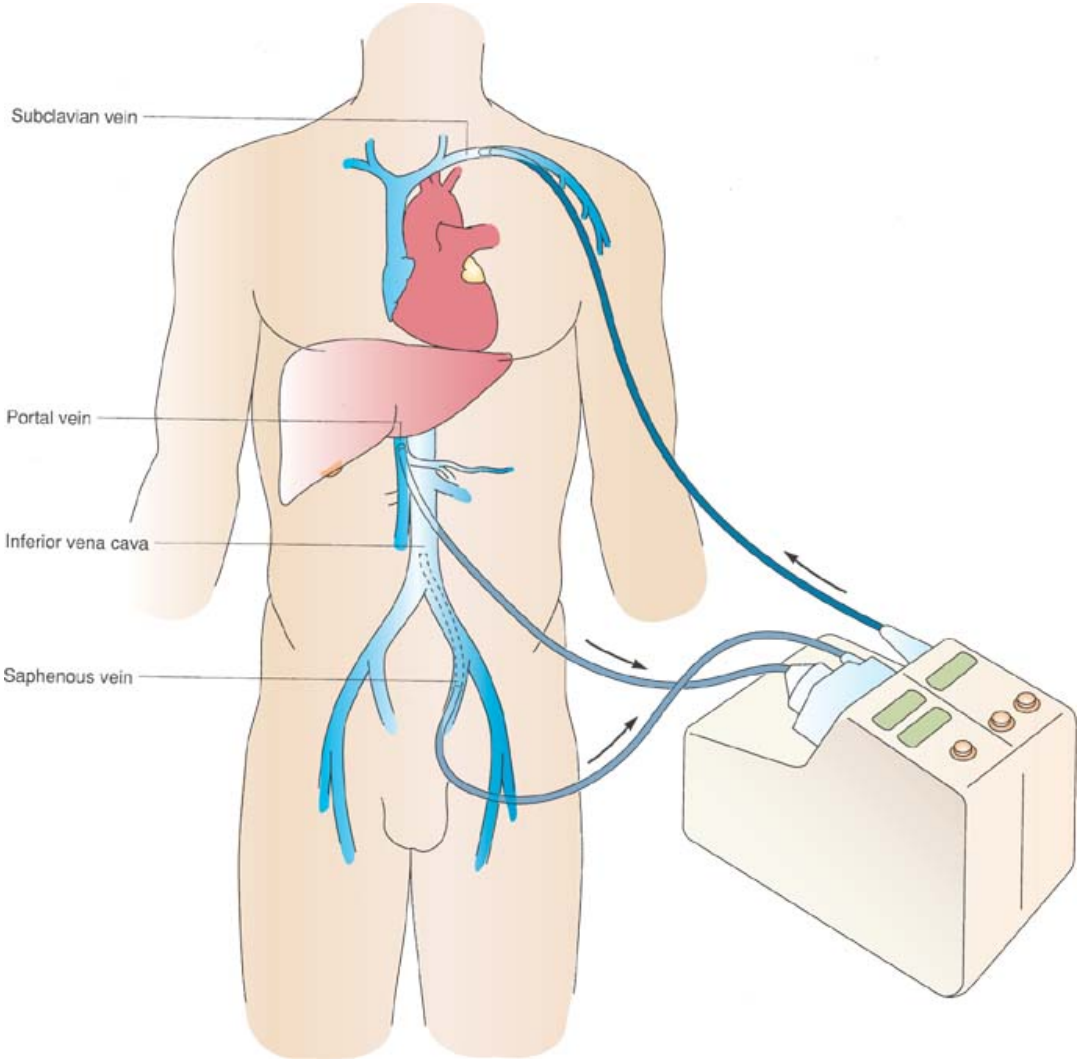

Figure 7.12-4. 4. Setup for venovenous bypass during hepatic transplantation. Cannulas are placed into the portal vein to decompress the splanchnic bed and inferior vena cava (through the greater saphenous vein) to decompress the lower extremities and kidneys during the anhepatic phase of the transplant. A centrifugal pump is used to deliver bypassed blood to the central circulation by means of a cannula passed into the axillary vein. Cannulas also may be placed percutaneously directly into the femoral and subclavian veins. (Reproduced with permission from Greenfield LJ, et al, eds: *Surgery: Scientific Principles and Practice*, 3rd edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

Table 7. 12-2. Benefits and Potential Complications of the Venovenous Bypass System

Benefits	Complication
Improved hemodynamics during anhepatic phase	PE
↓ blood loss	Air embolism
	Brachial plexus injury
May improve perioperative renal function.*	Wound seroma/infection
	Vascular injury

*In a prospective randomized trial comparing venovenous bypass with no bypass, no difference was found in the periop renal function between the two groups.[5]

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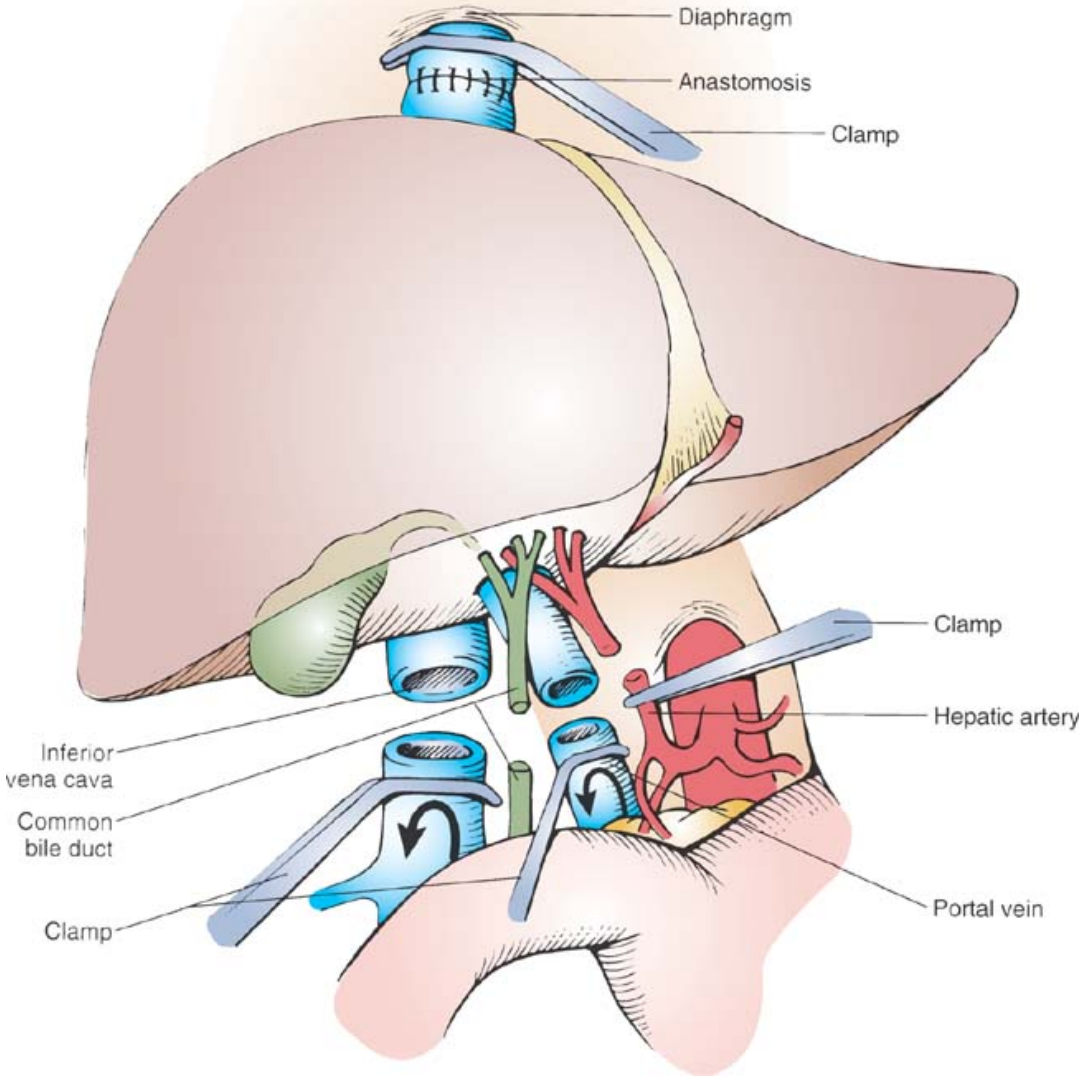

Figure 7.12-5. 5. Standard liver transplantation without venovenous bypass. Venous return is significantly impaired.

Venous bypass is not necessary when the **piggyback technique of liver transplantation** is utilized, because the diseased liver is separated from the vena cava (systemic venous return remains unimpaired), and vascular control is obtained by placing a clamp across the confluence of the hepatic veins as they join the vena cava (Fig. 7.12-6). A temporary portocaval shunt may be created to minimize bleeding in cases with severe portal HTN. The first anastomosis is between the suprahepatic vena cava of the liver allograft and the cuff created from the hepatic veins. The infrahepatic vena cava of the liver allograft is ligated, and the portal vein reconstruction is then completed. The clamps are then removed and the liver is revascularized.

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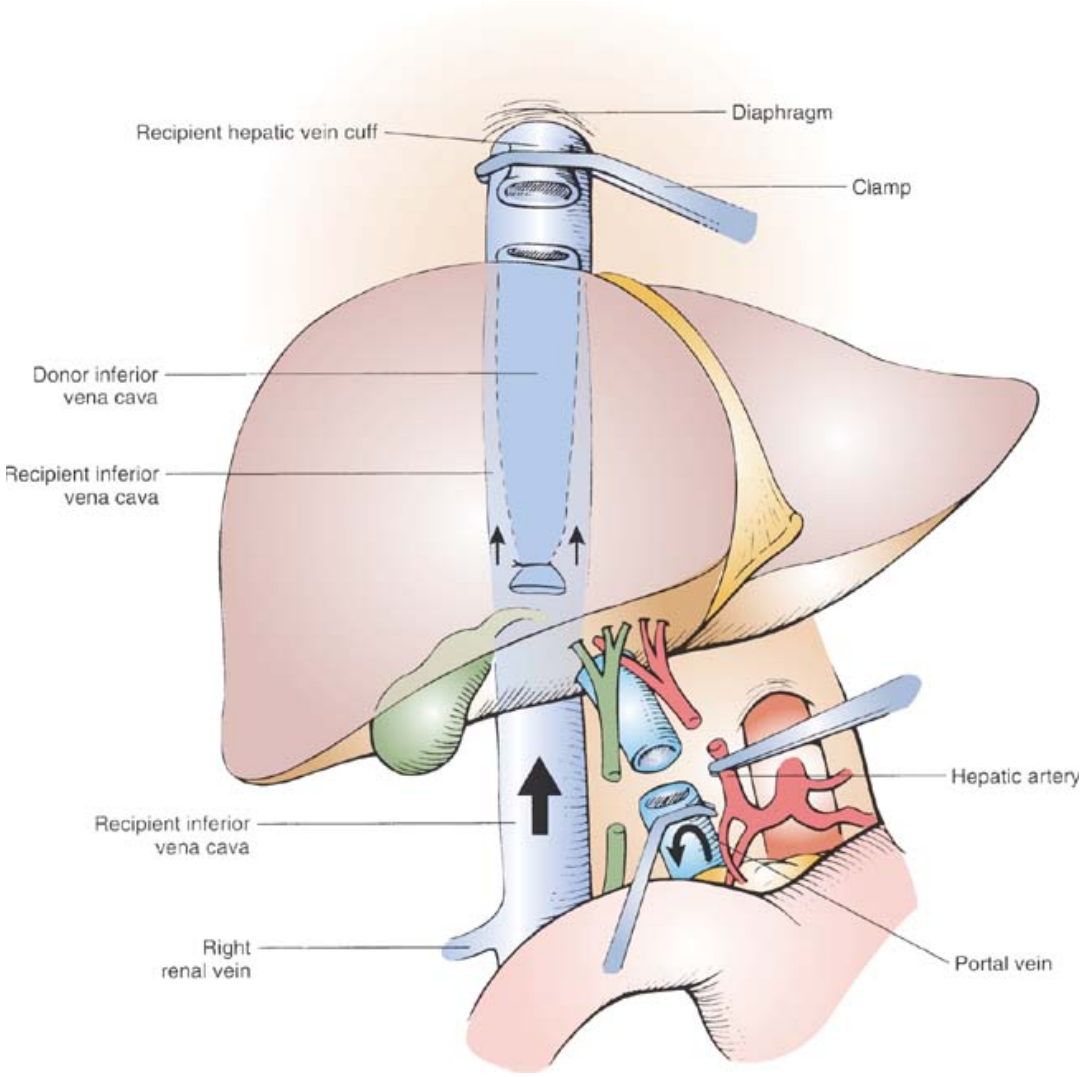

Figure 7.12-6. 6. Piggyback liver transplantation. Note that the recipient's vena cava is left intact and systemic venous return is unimpaired.

The **postrevascularization stage** of the transplant begins with the removal of the vascular clamps. The reperfusion of the liver may be the most critical part of the operation. Despite flushing the liver to remove the high K^+-containing organ preservation solution, hyperkalemia may be troublesome following liver reperfusion, particularly with livers that sustained significant injury during preservation and reperfusion. Additionally, massive air embolism is an immediate concern following revascularization, as it may quickly lead to cardiac arrest. It is also during this stage that the patient may experience pulmonary HTN, which can lead to right heart failure and severe systemic hypotension. Pulmonary hypertension and right heart failure must be treated aggressively with inotropic agents; otherwise, the liver is subjected to high outflow resistance resulting in congestion and worsening of the allograft preservation injury. The cause of this phenomenon is not well understood; fortunately, it is seen in very few patients. Another reperfusion phenomenon is that of systemic hypotension secondary to peripheral vasodilation. This may be due to the release of systemic inflammatory mediators, which include kinins, cytokines, and free radicals from the liver allograft. Reperfusion of the liver also can have dramatic effects on coagulation, such as fibrinolysis resulting in severe hemorrhage or hypercoagulation that can result in venous thrombosis and massive pulmonary embolism with cardiovascular collapse.

Immediately prior to revascularization, the patient is usually given methylprednisolone (250–1000 mg) as part of the immunosuppressive regimen, as well as an adjunct to counteract the systemic effects of ischemia-reperfusion injury of the liver. At this point, all of the vascular anastomoses, the retroperitoneum, and the liver (especially the cut surface in segmental or reduced-size grafts) are inspected for surgical bleeding.

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The hepatic artery reconstruction is performed after stabilization of the patient following revascularization of the liver. After the hepatic arterial anastomosis is completed, it is important to maintain adequate arterial BP (MAP > 65 mmHg) to prevent hepatic artery thrombosis. This is especially critical in pediatric transplant recipients, where the hepatic artery diameter ranges from 1–3 mm. The last part of the procedure involves hemostasis, removal of the gallbladder, and reconstruction of the bile duct (Fig. 7.12-7).

There are two basic methods for the **bile duct reconstruction:** an end-to-end anastomosis, with or without a T tube (in patients with normal common bile ducts), or a choledochojejunostomy to a Roux-en-Y limb of jejunum (Fig. 7.12-8) (in patients with biliary atresia, primary sclerosing cholangitis, or diseased common bile ducts, or when there is a size discrepancy between the donor and recipient common bile duct). In cadaveric or live-donor segmental transplantation, the technique for the recipient's hepatectomy and the implantation of the allograft is not different from that of full-size liver transplantation; however, the technique of piggyback liver transplantation must be used with live donors, because the allograft segment does not include the vena cava. The anesthesiologist must be alert during the reperfusion of a segmental graft, because significant bleeding may ensue from the raw surface of the liver.

After the biliary reconstruction is completed and hemostasis has been achieved, a feeding jejunostomy tube and 2–3 closed-suction drains may be placed. The position of an OG or NG tube (placed at the beginning of the case) is confirmed and the abdomen is closed.

Usual preop diagnosis: ESLD

Summary of Procedures

Position	Supine; arms tucked. Left arm and left groin area out for access to the axillary and femoral veins if venous bypass is anticipated.
Incision	Bilateral subcostal, in children; in adults, incision must extend cephalad to the xiphoid process.
Special instrumentation	Upper hand or Thompson retractor; venous bypass pump; rapid-infusion system; Cell Saver; argon beam coagulator, ultrasound.
Unique considerations	Thrombus or air embolism may occur during removal of clamps from vena cava or with the use of venovenous bypass. Right heart failure, ↓BP, and ↓SVR may be observed after revascularization. Continuous AV hemofiltration may be required if renal failure is present. Head and extremities should be covered with plastic to maintain core body temperature, particularly in children. OG tube required.
Antibiotics/drugs	Ampicillin (1 g q 8 h) and ceftriaxone (1 g q 24 h) prior to making incision. Methylprednisolone and antilymphocyte antibody preparations for immunosuppression. Aprotinin had been used occasionally during the hepatectomy and anhepatic phase in patients with severe coagulopathy and fibrinolysis (venous thrombus formation and pulmonary embolism can be seen with aprotinin use); however, its use is now contraindicated due to multiple reports of increased renal failure and mortality in cardiac surgery patients, although this has not been reported in liver transplant recipients.[18,20] Just recently, the manufacturer, Bayer, has removed aprotinin from clinical use.[21]
Surgical time	4–12 h
EBL	6 U average blood loss (range 0–100 U)
Postop care	ICU: 1–2 d. HTN commonly seen.
Mortality	10% at 1 yr
Rejection	20–50% during first yr
Morbidity	Infectious complications: 20–50% Biliary stenosis or leaks: 5–15% Retransplantation: 6–14% Primary graft nonfunction: 2–5% Hepatic artery thrombosis: 0–6% Portal vein thrombosis: 1–4%
Pain score	7–8

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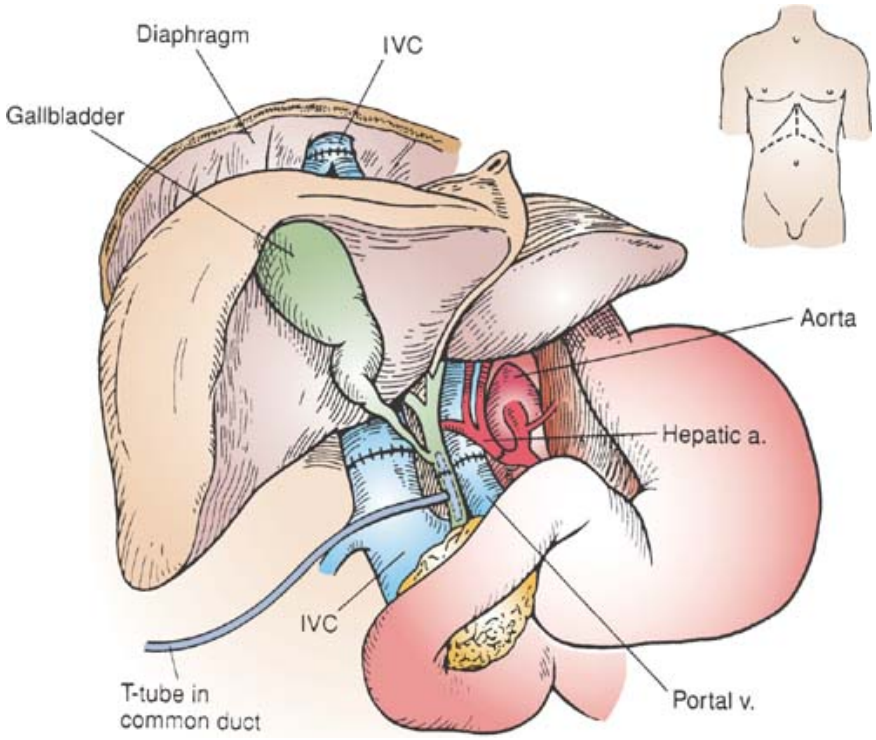

Figure 7.12-7. 7. Liver transplantation. Anastomoses—including suprahepatic and infrahepatic IVC, portal vein, hepatic artery, and common bile duct—are complete as shown here. Roux-en-Y loop of small intestine is an alternative biliary drainage conduit. Inset shows a chevron incision with midline extension. (Reproduced with permission from Hardy JD: *Hardy's Textbook of Surgery*, 2nd edition. JB Lippincott, Philadelphia: 1988.)

Patient Population Characteristics

Age range	Neonate-70 yr
Male:Female	1:1
Incidence	10/million/yr (15% pediatrics)
Etiology	Adult: hepatitis C cirrhosis; alcoholic cirrhosis; primary biliary cirrhosis; primary sclerosing cholangitis; hepatitis B cirrhosis; hepatocellular carcinoma. Pediatric: biliary atresia; inborn errors of metabolism, hepatoblastoma.
Associated conditions	Coagulopathy; hypoalbuminemia; ascites; cardiomyopathy (in alcoholic patients, hemochromatosis, and Wilson's disease); hepatorenal syndrome; hepatopulmonary syndrome, GI bleed, hepatic encephalopathy, and hypoglycemia in acute fulminant hepatitis.

Anesthetic Considerations

Preoperative

Patients needing liver transplantation represent a formidable challenge to the anesthesiologist. Frequently, these patients present for surgery with multiorgan system failure.

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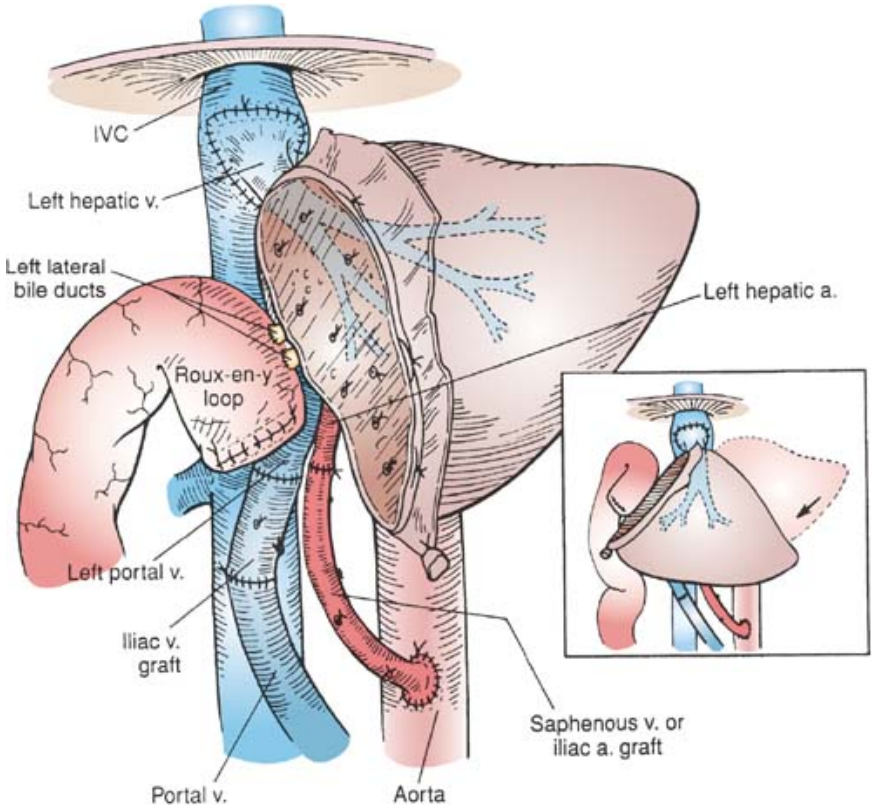

Figure 7.12-8. 8. Liver transplantation (child) using left lateral segment from an adult liver. The hepatic artery and portal vein are extended with donor iliac artery and vein, respectively. The final position of the graft is shown (inset). A Roux-en-Y loop of small intestine is used to drain the bile duct(s). The IVC is left intact. The cut surface of the liver can bleed excessively if the central venous pressure is too high. (Reproduced with permission from Broelsch CE, et al: Liver transplantation in children from living related donors: surgical techniques and results. Ann Surg 1991; 2l4 (4):432.)

Respiratory

Patients are often hypoxic because of ascites, pleural effusions, atelectasis, V/Q mismatch, pulmonary AV shunting, or hepatopulmonary syndrome. As a result, they are usually tachypneic and have a respiratory alkalosis. Evidence of pulmonary infection is usually a contraindication to surgery, but ARDS that may occur with hepatic failure is not.
Tests: ABG; PFT, as indicated. CXR: infection, effusions, atelectasis.

Cardiovascular

These patients demonstrate a hyperdynamic state with ↑plasma volume, ↑CO, and ↓SVR 2° arteriolar vasodilation in the splanchnic circulation, with intense vasoconstriction in other vascular territories (e.g., renal, brain, muscle, spleen). The SVR usually is not responsive to α-agents. AV fistulae may occur across the pulmonary circulation, so that precautions to prevent air embolism are important. Ejection fraction (EF) is usually high (> 60%), but some patients have cirrhotic cardiomyopathy → ↓ contractility 2° ↓β-receptors, alterations in myocardial cell membrane properties, and ↑myocardial depressant substances. This cardiac dysfunction, however, usually is masked by a reduction in afterload. Pericardial effusions may be present, and should be drained at surgery. Many of these patients will have dysrhythmias, HTN, pulmonary HTN (very high risk), valvular disease, cardiomyopathy (alcoholic disease, hemochromatosis, Wilson's disease), and CAD. These patients will require appropriate preop consultation and workup.
Tests: ECG; stress ECHO: EF, contractility, pulmonary HTN, wall motion abnormalities, valve problems. If abnormal, right-

	and left-heart catheterization with coronary artery angiography should be performed.
Neurological	Patients are often encephalopathic and may be in hepatic coma; however, other organic causes of coma should be ruled out. In fulminant hepatic failure, ↑ICP is common, accounting for 40% of mortality (herniation), and may require prompt treatment (mannitol, hyperventilation, etc.). **Tests:** Continuous ICP monitoring in fulminant hepatic failure
Hepatic	Hepatitis serology and the cause of hepatic failure should be determined. Vascular abnormalities, previous RUQ surgery or portal-vein decompressive surgery places the patient in a high-risk group. Albumin is usually low, with consequent low plasma oncotic pressure → edema, ascites. The magnitude and duration of drug effects may be unpredictable, but, generally, these patients have ↑sensitivity to all drugs and their actions are prolonged. **Tests:** Bilirubin; PT; ammonia level; SGOT; SGPT; albumin
Gastrointestinal	Portal HTN, esophageal varices, and coagulopathies ↑ risk of GI hemorrhage. Gastric emptying is often slow and, together with the emergent nature of this surgery, warrants rapid-sequence induction (see p. B-4). H_2-antagonists are indicated preop.
Renal	Renal function ↓, especially in fulminant hepatic failure (hepatorenal syndrome). The kidneys often recover after transplantation, but simultaneous kidney transplantation may be justified. These patients are often hypervolemic, hyponatremic, and possibly hypokalemic. Ca^{++} is usually normal. Metabolic alkalosis may be present. Consider preop dialysis and intraop continuous AV hemofiltration. Mannitol (0.5–1 g/kg) may be used intraop to maintain renal function. **Tests:** BUN; Cr, creatinine clearance; electrolytes; ABG
Endocrine	Patients often glucose-intolerant or frankly diabetic, although acute hypoglycemia may be seen in acute hepatic failure. Hyperaldosteronism may be present. **Tests:** Glucose; electrolytes
Hematologic	These patients are often anemic 2° either blood loss or malabsorption. Coagulation is impaired because of ↓ hepatic synthetic function (all factors except VIII and fibrinogen are ↓), abnormal fibrinogen production,↓/impaired Plt, fibrinolysis, and low-grade DIC. **Tests:** PT; PTT; Plt count; bleeding time; fibrinogen; fibrin-split products (FSP); TEG
Premedication	Low doses of benzodiazepines may be used judiciously, but often nothing is given prior to surgery. Usually good preop evaluation and discussion suffice. Intramuscular injection should be avoided. Full-stomach precautions are justified. Metoclopramide 10 mg iv, ranitidine 50 mg iv and Na citrate 0.3 M 30 mL po should be given prior to surgery.

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Intraoperative

Anesthetic technique: GETA. These patients are extremely complex to manage because of the hemodynamic instability, massive blood loss, coagulopathy, and metabolic problems. It is convenient to divide the operation into three stages: preanhepatic, anhepatic and neohepatic (discussed later).

Induction	Often, a narcotic (e.g., fentanyl 2–5 mcg/kg) is given just before induction; and rapidsequence induction is preferred. STP (3–5 mg/kg) or etomidate (0.3 mg/kg) with

succinylcholine (1–2 mg/kg), together with cricoid pressure.

Maintenance

Standard maintenance (see p. B-2) with fentanyl 10–50 mcg/kg. A benzodiazepine (e.g., midazolam 0.1–0.3 mcg/kg or scopolamine) often is given to ensure amnesia during periods of hemodynamic instability when the volatile agent may need to be off. N_2O is avoided because of bowel distention and possible air embolism. Ventilation with $FiO_2 > 0.5$ and $PaCO_2 = 35$ mmHg. Occasionally, PEEP (5 cm H_2O) is added. Antibiotics and immunosuppressants should be given per surgeon's direction. Muscle relaxation usually is maintained with vecuronium.

Preanhepatic phase

The **preanhepatic phase** starts at skin incision and ends with removal of the recipient liver. Pleural and pericardial effusions are drained, which may improve oxygenation. Hyperglycemia is common during this period. A drop in filling pressures may be 2° hemorrhage or

vascular compression. Hemorrhage can be severe 2° portal HTN. Coagulation problems usually increase during this stage, although fibrinolysis is not usually a problem. Blood loss replacement is accomplished with blood (PRBC) and FFP. Cryoprecipitate and Plts are given as needed, but a hypercoagulable state should be avoided, particularly if venovenous bypass is contemplated. Hemodynamic instability is not uncommon during the hepatic vascular dissection 2° manipulation of the liver and ↓ venous return. Venovenous bypass relieves most of the complications of portal and IVC cross-clamping (↓ venous return, low CO, tachycardia, acidosis, ↓ renal function, intestinal swelling.) Blood is pumped from the femoral vein and the portal system (either portal vein or inferior mesenteric) via a centrifugal pump to the left axillary or subclavian vein. Generally, no heparin is used, but heparin-bonded cannulas and tubing are used. Bypass flow need to be at least 1 L/min to avoid possible thromboembolism. Bypass flow depends on venous inflow and is drawn into the pump by negative pressure. Low flows may be caused by hypovolemia or obstructed cannulae. Complications include unexpected decannulation, thromboembolism, and air embolism, all of which may need rapid termination of bypass and treatment of ↓ BP; fibrinolysis is seen with prolonged venovenous bypass.

Massive blood transfusion is associated with ↓ Ca^{++} and replacement is usually needed (± 500 mg/1000 mL of blood/FFP/plasmalyte mixture). If hyperkalemia occurs, it should be treated aggressively. Metabolic acidosis > 5 mEq/L should be treated with bicarbonate or THAM acetate (tris-hydroxymethyl aminomethane) to avoid a rapid increase in sodium. Typical loading dose: mL of 0.3M THAM = lean body weight [kg] x base deficit [mmol/L]. Occasionally, inotropic support is needed, but α-adrenergic agents should be avoided because of ↓ renal and peripheral perfusion. UO needs to be maintained by ensuring adequate intravascular volume; occasionally mannitol may be needed.

Anhepatic phase

The **anhepatic phase** begins with clamping of the hepatic vessels and vena cava and removal of the liver; it ends with the reperfusion of the donor liver. Problems during this period include hemorrhage, increasing coagulopathy and fibrinolysis, acidosis, hypothermia, and ↓ renal function. The hemodynamic instability associated with clamping of the hepatic vessels and the congestion of the bowel that occurs can be decreased by venovenous bypass (see the previous text). Care should be taken to maintain intravascular volume, while avoiding volume overload, because this would worsen fluid overloading on reperfusion. At the completion of vena caval anastomoses, the liver is flushed via the portal vein to remove air, preservation fluid, and metabolites. Reperfusion may take place after completion of the portal vein anastomosis or after both portal vein and hepatic artery anastomoses are completed. As in the preanhepatic phase, acidosis, ↓ Ca^{++}, glucose, coagulation, and other electrolyte abnormalities should be treated. Fibrinolysis usually starts in this period, but is not usually treated unless severe, because of the potential for embolism during venovenous bypass.

Neohepatic phase

The **neohepatic phase** begins with the unclamping of the portal vein, hepatic artery, and vena cava and reperfusion of the donor liver. Preparation for this phase is important because this may be a period of great hemodynamic instability. Before removal of the clamps, acidosis should be corrected, ionized Ca^{++} should be normal, and K^+ should be < 4.5 mEq/L. $CaCl_2$ $NaHCO_3$ and epinephrine should be readily available. Fluid overload prior to declamping should be avoided. High venous filling pressures decrease hepatic perfusion, especially prior to hepatic artery anastomosis. Declamping can be attended

	by ↓BP, ↓HR, dysrhythmias, hypothermia, lactic acidosis, coagulopathy, hyperglycemia, and thromboembolism.
Reperfusion syndrome	**The "reperfusion syndrome"** (which can occur in this phase) is characterized by ↓ HR, ↓BP (30% of patients develop MAP < 70% of baseline), conduction defects, and ↓ SVR in the face of acutely ↑RV filling pressures. Cause unknown. A rapid ↑K^+can → cardiac arrest. (Rx: ensure normal pH and electrolytes prior to unclamping; rapid therapy when it occurs.) ↓BP and ↓ HR are treated with epinephrine (10 g increments), whereas $CaCl_2$and $NaHCO_3$are used to correct hyperkalemia and acidosis. Pulmonary edema may occur as a result of fluid overload and can be treated with diuretics, inotropes, and phlebotomy. A high venous pressure will cause graft congestion and should be avoided. Reperfusion is associated with severe coagulopathy due to fibrinolysis (usually primary), release of heparin, and hypothermia. As liver function returns, there should be an improvement in coagulation, acid-base status (metabolic alkalosis may occur), ↓lactic acidosis, return of glucose to normal, and bile production. Hypokalemia may occur 2° uptake by the liver. Graft failure is associated with coagulopathy, ↑lactic acid, citrate intoxication, hyperglycemia, and ↓ bile formation.
Emergence	Extubation is deferred. These patients are generally ventilated postop in ICU until stable and able to be weaned from ventilatory support. Apart from the usual tests, monitor hepatic function. Also ensure immunosuppression provided, infection controlled, analgesia adequate (usually fentanyl), and peptic ulcer prophylaxis given (ranitidine preferred).
Blood and fluid requirements	Massive blood loss IV: 10 Fr × 2 Plasmalyte A or Normosol UO > 1 mL/kg/h Warm all fluids Humidify gases Rapid-infusion system Cell Saver 20 U PRBC 20 U FFP 20 U PLT Generally, iv's are placed in the right antecubital fossa, left or right IJ or EJ. The left arm is avoided because the axillary or left subclavian vein may be used for venovenous bypass. Plasmalyte A or Normosol are preferred (absence of glucose, $Ca^{++}$and lower Na^+content) over NS or LR. Hypernatremia may occur due to administration of $NaHCO_3$ The ability to infuse up to 1.5 L/min of blood should be available. Usually a mixture of Normosol (250 mL), PRBC (1 U), and FFP (1 U) is used, yielding Hct = 26–30%. Actual blood loss estimation is extremely difficult, and usually replacement is judged by hemodynamic status, UO, and $S\bar{v}O_2$ Cell Savers are used to conserve blood. Anticoagulation is achieved with citrate solution to avoid heparin contamination, and cells are washed with Normosol/Plasmalyte A. Discontinue use before biliary reconstruction (infection) or in neoplasms, hepatitis B, or spontaneous bacterial peritonitis.
Monitoring	Standard monitors (see p. B-1). ECG (5-lead) Temp-bladder ETN_2 Include 5-lead ECG and bladder T. (These patients sustain significant heat loss.) A full-time anesthesia technologist and lab/blood bank runner are useful. Lab and blood bank should be notified of the expected transplant. An automated data acquisition system also is useful, because there are times during the case when record keeping may not be kept up to date in favor of providing patient care. One or two arterial lines are placed at the outset—one in the right radial, for ongoing

	Arterial line(s)	lab and blood gas sampling; another line, in the right femoral artery is utilized for continuous pressure measurement.
	PA catheter/$S\bar{v}O_2$/CO	A PA catheter is essential for management of hemodynamics in these patients, because of the rapid changes in VS. A catheter capable of measuring mixed-venous O_2sat is very useful, because it gives early clues to impending decompensation. Coagulopathy complicates the placement of central lines, and the use of ultrasound-guidance is recommended.
	TEE	TEE is useful to monitor cardiac filling and function and to diagnose problems such as PE or air embolism. Care needs to be taken in placing the TEE because many of these patients have esophageal varices.
	ICP	ICP should be measured in patients with fulminant hepatic failure if ↑ICP is a concern.
	Laboratory	ABG, acid base status, electrolyte, lactate, osmolality, Ca++, PT, PTT, Plt, Hct—all should be monitored on a regular basis (hourly or half-hourly; occasionally, more frequently).
	Thromboelastograph (TEG)	TEG is useful for monitoring coagulation (see the subsequent discussion of coagulation management).
Coagulation management	PT PTT Plt counts Fibrinogen FSP	Patients are prone to a variety of coagulopathies (↓Plt, ↓coagulation factors, DIC, fibrinolysis, etc.) because of preop factors, massive hemorrhage, anhepatic period, and reperfusion of the new liver; therefore, monitoring and treatment are necessary. Also, states of hypercoagulopathy need to be avoided because of unheparinized venovenous bypass.
	TEG	While PT, PTT, Plt counts, fibrinogen, and FSP may provide relevant information, they may not reflect the true coagulability of patient's blood, and tend to take considerable time to perform. Thus, in some centers, TEG has gained in popularity. It measures whole blood coagulability, not specific factors. TEG works by measuring viscoelastic properties of blood as it forms clot (fibrin connections) between a rotating cuvette and a spindle. Characteristic patterns are formed by the various coagulopathies with the common types shown in Fig. 7.12-9. Evaluation of the TEG leads to more rational transfusion therapy, reducing the number of U of blood/blood products used. Comparing specimens of native whole blood vs blood mixed with EACA or protamine can guide pharmacologic therapy

		of coagulopathies. Table 7.12-3 gives specific recommendations.
Positioning	and pad pressure points eyes	Table and arm boards should be very well padded. Head should be placed on a foam rest. Particular care should be taken to pad the retractor supports where they may impinge on the arms and on the radial nerve as it curls around the humerus. Patient's arms, head, and legs should be wrapped in plastic to protect against heat loss. Plastic drapes and the use of a cesarian section-type drape to protect the ECG electrodes and direct fluid flow off the table are useful to prevent the patient from lying in a pool of fluid. A warming blanket under the patient and over the lower legs is very useful.
Temperature control	Warming blanket Humidifier	
Complications	Coagulopathy Hemorrhage Air embolism RV failure Metabolic acidosis	

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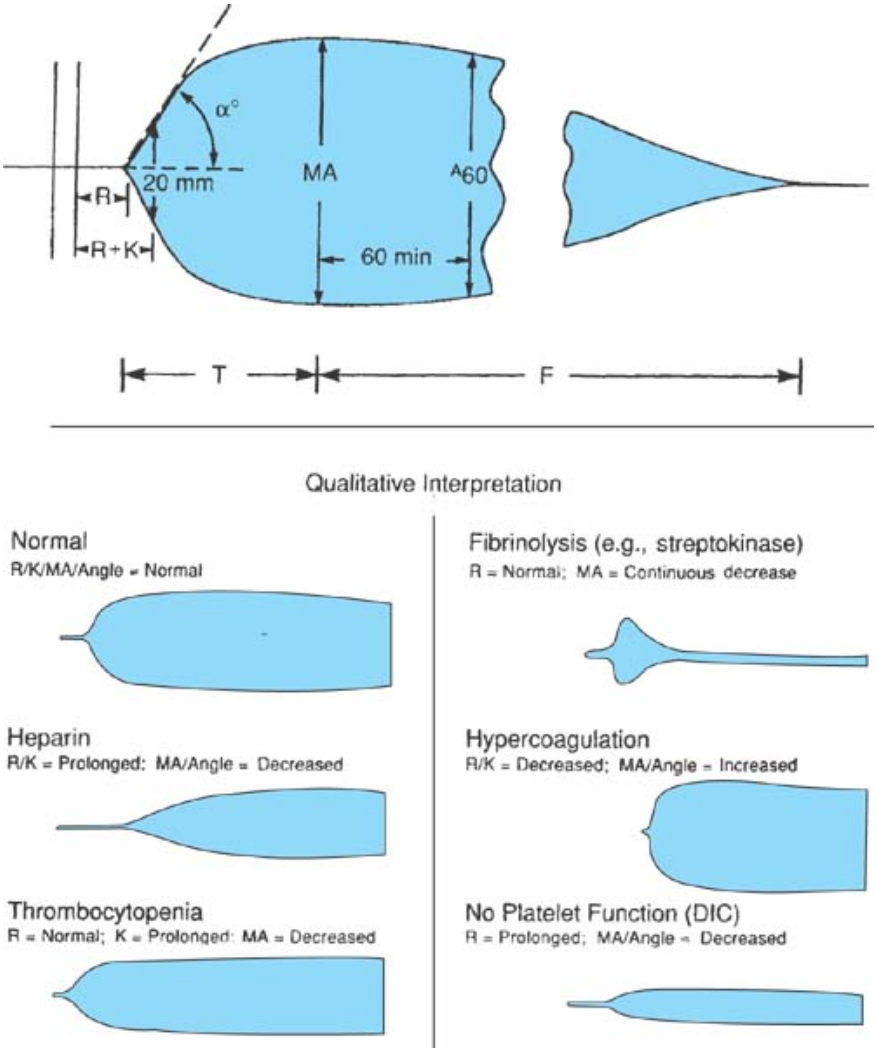

Figure 7.12-9. 9. Variables and normal values measured by TEG: R = reaction time, 6–8 min R + k = coagulation time, 10–12 min α = clot formation rate, > 50° MA = maximum amplitude, 50–70 mm A_{60} = amplitude 60 min after MA A_{60}

/MA-100 = whole blood clot lysis index, > 85% F = whole blood clot lysis time, > 300 min (Reproduced with permission from Kang YG, et al: Intraoperative changes in blood coagulation and thromboelastographic monitoring in liver transplantation. *Anesth Analg* 1985; 64:891.)

Table 7-12-3. 3. Coagulation Therapy Guided by TEG Monitoring[6]

1. Maintenance fluid
   RBC: FFP: Plasmalyte A = 300:200:250 mL
2. Replacement therapy
   1. FFP (2 U) for prolonged reaction time (R > 15 min)
   2. Plt (10 U) for small MA (MA < 40 mm)
   3. Cryoprecipitate (6–12 U) for persistent slow-clot formation rate (α < 40°) with normal MA
3. Pharmacologic therapy
   1. Compare coagulability of whole blood, blood treated with protamine sulfate, and blood treated with epsilon aminocaproic acid.
   2. Epsilon aminocaproic acid (1 g) for severe fibrinolysis(F < 60 min)
   3. Protamine sulfate (50 mg) for severe heparin effect
   4. Heparin (1000–2,000 U) for hypercoagulable state

(Print pagebreak 704)

Postoperative

Monitoring of hepatic function	Serial LFTs PT, PTT Ammonia level Lactate TEG Bile output	Initial LFTs often show very high liver enzymes, which subside over a period of days. PT generally improves to normal levels, while lactic acidosis usually corrects quickly. Often a metabolic alkalosis follows and may need HCl treatment.
Complications	Bleeding Partial vein thrombosis Hepatic artery thrombosis Biliary tract leaks Primary nonfunction Rejection Infection Pulmonary complication HTN Electrolyte abnormalities (hypokalemia, ↓ Ca++, ↑Na) Alkalosis Renal failure Peptic ulceration Neurologic	This is not a complete list. Feared complications that may → graft loss include portal vein thrombosis, hepatic artery thrombosis, bile leaks, and rejection. These are attended by ↑LFTs, lactic acidosis, coagulopathy, hypoglycemia, ↓ renal function, and poor bile formation.

Suggested Readings

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Living-Donor Liver Transplantation

Surgical Considerations

Description: The success of deceased donor liver transplantation has resulted in an ever-increasing number of patients with end stage liver disease (ESLD) waiting for transplantation; however, the number of deceased donors has remained relatively constant. Consequently, the waiting time to receive an organ has increased significantly, *(Print pagebreak 705)* and 15% of patients will die while waiting. The success of **living-donor renal transplantation,** coupled with the experience in adult-to-pediatric living-donor liver transplantation, as well as advances in surgical and postsurgical care of patients undergoing major liver resections, has lead to the implementation of adult-to-adult living-donor liver transplantation. This provides a potentially larger source of healthy livers for transplantation.

Potential liver donors undergo extensive medical and psychosocial evaluation to ensure psychological as well as physical fitness to undergo a major surgical procedure with no medical benefits to the donor. Donors must have full blood typing to ensure compatibility with the recipient, and then fill out an extensive medical questionnaire, followed by a complete physical exam and screening lab tests. Any evidence of diabetes, HTN, or renal, pulmonary, cardiovascular, or hepatic abnormalities usually is a contraindication to donation. After the potential donor is medically and psychosocially cleared, they undergo a detailed imaging study of the liver; and, if there are no anatomical contraindications, then an elective living-donor transplant is scheduled.

The donor and recipient operations usually are conducted simultaneously to minimize the ischemic injury to the donor liver segment. The donor operation, however, is initiated first, with the recipient operation started only after the donor liver has been directly examined and no barriers to proceeding are found. The donor operation is similar to either a right or left hepatic lobectomy, although there are some differences that can have a significant impact on anesthetic management, as detailed later.

The donor may elect to have an epidural catheter for postop analgesia, and this usually is placed before surgery. A vertical midline incision is made from the xiphoid to just above the umbilicus and extended transversely to the right anterior axillary line. Occasionally, bisubcostal incisions are required. Following exploration of the abdomen, intraoperative ultrasound may be performed to map the hepatic venous anatomy so the plane of dissection can be delineated. Additionally, an intraoperative cholangiogram is performed via the cystic duct (a cholecystectomy is performed in a right or left hepatic lobectomy) or the common bile duct, to define the biliary anatomy. After this is performed, the corresponding portal vein and hepatic artery are isolated. Unlike in a hepatic

lobectomy for tumor, the venous and arterial inflow to the liver segment is not ligated; thus, the transaction of the liver parenchyma may result in significant hemorrhage. Next, the respective lobe of the liver is mobilized from its attachments, and the liver is dissected from the retrohepatic vena cava, with ligation of the short-hepatic veins. This maneuver can cause transient hypotension secondary to torque and compression of the IVC and hepatic veins, as well as potential bleeding from the vena cava itself. Next, the hepatic vein is isolated, and the liver is then divided, which can be a slow and tedious process. After the parenchyma is divided, the liver segment is ready to be removed. Heparin (80–100 U/kg) is given to prevent intrahepatic clot formation. Following heparinization, the hepatic artery and portal vein are ligated and divided, followed by the hepatic vein. The donated hepatic lobe is immediately placed in ice and flushed with Viaspan or other organ preservation solution. After the donated liver segment is flushed, the recipient's hepatic vein stump is oversewn and the abdomen and cut surface of the remaining liver are inspected for hemostasis and bile leak. Closed drains are placed, after which the abdomen is closed. Essentially, the same procedure is followed for a left lateral segmentectomy (adult-to-child), except that the extent of liver resection is about 25%, compared with 40% or 60% for a left or right hepatic lobectomy, respectively.

Usual preop diagnosis: Healthy, living liver donor

Summary of Procedures

Position	Supine; arms tucked
Incision	Vertical midline with a right and/or left subcostal extension, depending on liver segment being utilized
Special instrumentation	Retractor; ultrasonic or hydrojet dissection/aspirator; irrigating bipolar cautery; argon beam coagulator; Cell Saver; rapid-infusion system
Unique considerations	Intraop cholangiogram and/or ultrasound. Hemodilution immediately before surgery, with removal of 1 U whole blood if Hct ≥ 40. Patients also may have donated 1–2 U of autologous blood. Heparin (80–100 U/kg)
Antibiotics	Ampicillin 1 g and ceftriaxone 1 g iv before skin incision
Surgical time	3–5 hr
EBL	250–500 mL
Postop care	ICU overnight or surgical floor. Hospital stay 5–7 d.
Mortality	14 deaths reported world-wide (as of December 2006) following 1,500 donor operations.
Morbidity	Infectious complications: 3% Biliary leak: 1% Reoperation: 3–5% Acute hepatic failure: < 0.1%
Pain score	7–9

(Print pagebreak 706)

Patient Population Characteristics

Age range	18–55 yr
Male:Female	1:1
Incidence	Uncommon

Anesthetic Considerations

See Anesthesia for Hepatic Resection, p. 553.

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Multiorgan Procurement

Surgical Considerations

Description: The families of brain-dead patients may allow donation of the patient's functioning organs—which may include, but is not limited to, heart, lungs, liver, kidneys, pancreas, and small intestine. The process of organ procurement can be chaotic, with multiple operative teams and technicians working simultaneously. Moreover, brain-dead patients tend to be hemodynamically unstable, sometimes requiring multiple pressors, and the possibility of acute decompensation is ever present.

The donor patient's chest and abdomen are opened in the midline from sternal notch to pubis (Fig. 7.12-10). The chest is opened with a sternal saw and generally an extra-large Balfour retractor is used to widely retract the abdomen. The aorta and IVC are dissected first to allow rapid placement of a flush line in the event that the patient experiences a cardiovascular collapse. Following this, the liver vasculature is identified in the hepatoduodenal ligament and is dissected out. This part of the procedure generally takes about 1.5 h. In cases where the pancreas is procured, an additional 45–60 min is required for mobilization of the pancreas. During pancreas procurement, a Betadine/amphotericin B solution is administered through an NG tube into the stomach and duodenum. A total of about 300–500 mL of the Betadine solution is passed in two divided aliquots. After the heart, liver, pancreas, and kidneys have been mobilized, the supraceliac aorta just below the diaphragm is dissected for placement of the aortic cross-clamp. Immediately before cross-clamping, 30,000 U of heparin (300 U/kg) is given systemically, with the α-antagonist phentolamine in some cases. The supraceliac aorta is then clamped and the organs are perfused with *(Print pagebreak 707)* Viaspan, a hyperosmotic and hyperkalemic solution containing insulin, glucose, and reducing agents. At this point, the ventilator can be turned off, except in cases where the lungs are being procured. In this case, the lungs must be inflated with 100% O_2 just before removal. The heart is the first organ to be removed, followed by the lungs.

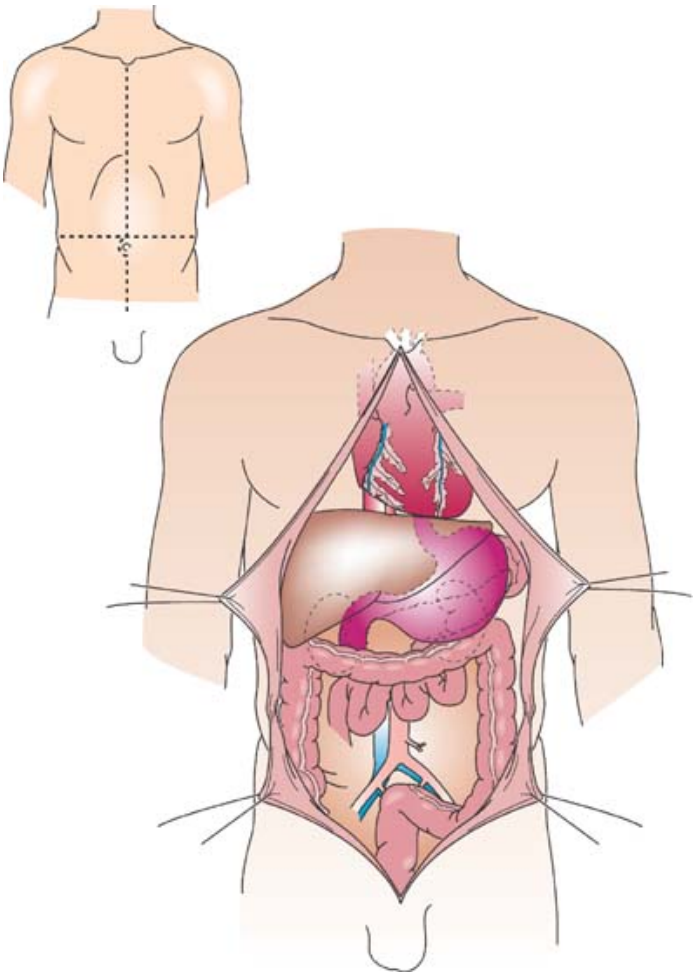

Figure 7.12-10. 10. A complete midline incision, from suprasternal notch to pubis, is made for multiple organ procurement; and the sternum is split. If necessary, cruciate abdominal incisions are added to facilitate exposure of the intraabdominal organs. (Reproduced with permission from Greenfield LJ, et al, eds: *Surgery: Scientific Principles and Practice,* 3rd edition. Lippincott Williams & Wilkins, Philadelphia: 2001.)

If the lungs are procured, an extra 20–30 min of perfusion time is required. After removal of the heart and/or lungs, the liver can be removed, followed by the pancreas, small intestines, and kidneys. After the organs are removed, spleen and lymph nodes for tissue typing are obtained from the abdominal and thoracic cavities; and, because of the possible need for vascular reconstruction in the recipients, bilateral iliac veins and arteries are removed. The total time for multiorgan procurement is 4 h, although the anesthesia time typically ends with aortic cross-clamping.

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Variant procedures: During the "**rapid-flush" technique** used by some procurement teams, the dissection of individual abdominal organs is minimized and an en bloc resection is done after clamping the aorta below the diaphragm and flushing preservation solution through the distal aorta. The en bloc organs are then dissected ex vivo, often at the transplant center, in preparation for transplantation. This technique is more rapid, requiring only about 1.5 h.

A second variant procedure, developed in response to the chronic shortage of transplantable organs, is the use of non-heart-beating donors. Patients, who are not brain-dead, but who have no hope of recovery (due to irreversible brain injury or pulmonary or cardiac failure), may be suitable donors. In this case, the patient is taken to the OR and, under the supervision of a physician who is not part of the transplant team, the life-sustaining treatment (e.g., pressors, ventilator) is D/C'd. When it has been determined that the donor has suffered cardiac death, the body is rapidly cooled with ViaSpan through a cannula that is located in the aorta at the level of the renal arteries. Depending on the regional regulations, this cannula may be placed before cardiac death, or immediately following the declaration of cardiac death. After the preservative flush is initiated, the abdomen is entered as rapidly as possible and the peritoneal cavity is packed with ice and the organs are removed expeditiously. The anesthesiologist's role is ended when cardiac death has been declared. The disadvantage of this approach is that there can be significant warm ischemia from the time the life-sustaining treatment is stopped to the time that cardiac death is reached.

Usual preop diagnosis: Brain death

Summary of Procedures

Position	Supine
Incision	Midline only, neck to pubis, ± bilateral transverse extensions
Special instrumentation	Chest and abdominal retractors
Unique considerations	Maintain oxygenation and BP as if live patient. May require pressors and/or blood transfusion. Temporarily deflate lungs for sternal sawing.
Antibiotics	Ampicillin (1 g iv), ceftriaxone (1 g iv); Betadine via NG tube for pancreas (with duodenal segment) procurement. Heparin, relative. (Betadine/Amphotericin in B solution)
Surgical time	4 h; rapid-flush technique: 1.5 h
EBL	200 mL

Patient Population Characteristics

Age range	Neonate-70 yr
Male:Female	N/A
Incidence	Approximately 5000/yr in the United States.
Etiology	Usually head trauma (e.g., motor vehicle accidents, gunshot wounds to the head) or intracranial bleeding
Associated conditions	Vasomotor instability; diabetes insipidus (DI); intracranial HTN

Anesthetic Considerations

Preoperative

In general, organ donors are previously healthy individuals who have suffered catastrophic, irreversible brain injury of known etiology, most commonly due to blunt head trauma, penetrating head injury, or intracranial hemorrhage. A declaration of brain death by physicians not participating in the organ procurement must be documented. This documentation, together with certification of death and familial consent, should be verified by the anesthesiologist before organ procurement. The United Network for Organ Sharing (UNOS) has produced *The Critical Pathway for the Organ Donor* to facilitate the administrative aspects of organ procurement (http://www.unos.org/resources/donorManagement.asp). There should be no evidence of disease or trauma involving the organs targeted for donation *(Print pagebreak 709)* and, in general, the patient should be hemodynamically stable with minimal inotropic requirements. After brain death has been declared, it is important to shift the emphasis away from cerebral resuscitation efforts and to focus instead on the maintenance of adequate tissue perfusion and oxygenation. Brain death is frequently followed by a series of pathophysiological events that may complicate the management of these patients.

Recently, Donation after Cardiac Death (DCD) has been instituted at many medical centers, where a non-brain dead patient is brought to the operating room for withdrawal of life support and subsequent removal of organs for transplant. Although the ethics of such organ donation had been debated, the ASA and other organizations have developed protocols to separate the teams caring for the patient during the withdrawal of life support and the operating room team responsible for organ retrieval. However, in most institutions, an anesthesiologist is present in the operating room as a member of the latter team. After withdrawal of life support has been initiated and the patient meets criteria for cessation of cardio-pulmonary function, organ removal is initiated as quickly as possible to limit warm ischemia time and possible damage to the organs to be removed. DCD patients often do not have as serious pathophysiological alterations as patients who meet brain death criteria.

Pulmonary dysfunction following brain death has many possible etiologies: aspiration, atelectasis, pneumonia, and pulmonary

Respiratory

edema. In addition, trauma may cause pulmonary dysfunction related to contusion, pneumothorax, or hemothorax. Meticulous pulmonary toilet is essential to prevent atelectasis and pneumonia. Maintenance of adequate oxygenation is requisite to ensure preservation of other organs for transplantation. Use mechanical ventilation with TVs of 10–12 mL/kg and a minute ventilation that maintains $PaCO_2$ 30–35 mmHg and pH 7.35–7.45. The FiO_2 should ensure a PaO_2 75–150 mmHg and arterial saturation > 95%. PEEP usually is applied at 3–5 cmH_2O and should not exceed 7.5 cmH_2O because of the deleterious effects on CO and regional blood flow, and possible barotrauma. The FiO_2 generally should be increased to 100% before transport to the OR. An important exception is in the case of heart-lung or lung retrieval, where it is important to maintain FiO_2 < 40% to minimize possible effects of O_2 toxicity. Ideally, PIP should be < 30 cmH_2O to minimize possible barotrauma to the lungs.

Tests: Frequent ABGs, including immediate preop period. Proper position of the ETT should be confirmed preop.

Cardiovascular

Hypotension should be anticipated in all organ donors. This results most commonly from neurogenic shock (derangement of descending vasomotor control → progressive ↓SVR and venous pooling) and hypovolemia. Hypovolemia is usually the result of dehydration therapy for cerebral edema, hemorrhage, DI, or osmotic diuresis 2° hyperglycemia. Hypothermia, LV dysfunction, and endocrine abnormalities also can contribute to ↓ BP. Fluid resuscitation with crystalloid, colloid, and PRBCs to maintain Hct > 30% should be initiated preop. Hemodynamic goals are: (1) CVP 10–12 cmH_2O (6–8 cmH_2O if lungs are to be procured); (2) MAP between 60–100 mmHg; (3) SBP > 100 mmHg; (4) PCWP ≥ 12 mmHg; (5) SVR 800–1200 dynes/second × cm^{-5} (or 800–1200 woods units); and (6) UO > 1 mL/kg/h. Donors are often placed on inotropic therapy to maintain these parameters; however, following adequate volume resuscitation, preop inotropic therapy often may be gradually decreased or D/C'd. If inotropic therapy remains necessary, typically it would consist of dopamine (2–10 mcg/kg/min), followed by dobutamine (3–15 mcg/kg/min) or epinephrine (0.1–1.0 mcg/kg/min), then norepinephrine. The latter 3 agents may be combined with dopamine (2–3 mcg/kg/min) in an attempt to augment or preserve renal, mesenteric, and coronary arterial blood flow. It should be noted that brain death may be accompanied initially by a transient hypertensive crisis that may require short-term treatment with SNP and/or esmolol. ECG abnormalities are common in patients with intracranial injury and are of no pathologic consequence. Atrial and ventricular dysrhythmias and various degrees of conduction block occur frequently in organ donors; the etiology may be electrolyte imbalance, ABG disturbance, ↑ICP, loss of the vagal motor nucleus, inotropic therapy, hypothermia, or myocardial contusions or ischemia. Antidysrhythmic therapy should follow the usual guidelines except for ↓HR, which is resistant to atropine in this setting. Bradycardia, if accompanied by ↓BP, should be treated with isoproterenol, dopamine, epinephrine, or temporary cardiac pacing.

Tests: ECG; ECHO (to assess wall motion abnormalities) and possibly coronary angiography (if CAD is suspected).

Diabetes insipidus (DI) frequently occurs in brain-dead donors; it is likely the result of destruction of the hypothalamic-pituitary axis. Untreated, it may cause marked hypovolemia and electrolyte disturbances (↑Na^+, ↑Mg^{++}, ↓K^+, ↓PO_4^-, ↓Ca^{++}). Therapy with iv

Neurological	vasopressin (titrated from 2 mcg/kg/min) or DDAVP (titrated from 0.3 mcg/kg/min) often is initiated to maintain UO < 1.5–3 mL/kg/h. Many believe that the benefits of minimizing electrolyte imbalance, fluid shifts, and reduction of core T outweigh the risks of vasopressin or desmopressin therapy, including coronary and renal vasoconstriction and possible organ ischemia or uneven distribution of the preservation solutions during flushing. It may be prudent, however, to D/C vasopressin or DDAVP infusions for at least 1 h prior to aortic cross-clamping and infusion of preservation solutions. Thermoregulation is abnormal in brain-dead donors due to hypothalamic dysfunction; and core T should be monitored (bladder, or esophageal). Aggressive warming techniques may have to be employed early to maintain a core T > 34–35°C, as there are numerous undesirable consequences of significant hypothermia (< 32°C) in the organ donor (e.g., cardiac dysrhythmia, cardiac instability, ↓GFR and cold diuresis, a left shift in the oxyhemoglobin dissociation curve, and pancreatitis). While other endocrine or metabolic disturbances may exist as a result of destruction of the hypothalamic-pituitary axis, currently there is no consistent recommendation for any other hormonal replacement therapy. **Tests:** Serum electrolytes and osmolality every 4 h
Hematologic	Donors may be anemic from hemodilution and/or hemorrhage. To ensure adequate tissue O_2delivery, PRBCs are transfused to maintain Hct > 30. Some donors may exhibit a coagulopathy; clinically significant bleeding should be treated with clotting factors and Plts. Persistent or severe primary fibrinolysis or DIC may require rapid transfer of the donor to the OR for organ retrieval. Administration of epsilon-aminocaproic acid to treat fibrinolysis is avoided for fear of microvascular thrombosis in the donor organs. **Tests:** Hb/Hct; PT; PTT; Plt count; DIC screen as clinically indicated.
Other	The role of oxygen-free radicals, with regard to reperfusion injury, has prompted the suggested use of mannitol and steroids (and other compounds) as scavengers.

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Intraoperative

Anesthetic technique: Although anesthesia is unnecessary in brain-dead organ donors, both visceral and somatic reflexes can lead to physiologic responses during the procedure. The goals of intraop management with regard to respiratory, cardiovascular, hematologic, and neurologic status are identical to those discussed under preop considerations earlier.

Induction	Settings for mechanical ventilation parallel those of the ICU, although it may be advisable to begin with an $FiO_2$of 100% until the first ABG result is obtained. The exception is when procurement of the lungs or heart-lungs is anticipated; then FiO_2 should not exceed 40%. To eliminate reflex neuromuscular activity and to facilitate surgical retraction, a long-acting neuromuscular blocking agent, such as pancuronium or pipecuronium (0.15 mg/kg), should be given at the beginning of the procedure and supplemented as necessary.
Maintenance	Reflex hypertensive responses to surgical stimulation occur frequently and may → excessive intraop blood loss and damage to donor kidneys; management should include the weaning of vasopressors and the initiation of vasodilator therapy with isoflurane, SNP, or NTG. Anesthetic care continues until the proximal aortic cross-clamp is applied. D/C all monitoring and supportive therapy at this point. The notable exception is the case of heart-lung or lung procurement; in this situation, all monitoring except

		FiO_2 should cease with proximal aortic cross-clamping. All supportive care is terminated, with the exception of mechanical ventilation of the lungs at 4 breaths/min or as directed by the transplant team, and suctioning of the ETT after cessation of mechanical ventilation just prior to removal of the tube. Extubation marks the termination of anesthetic care of the heart-lung or lung donor.
Blood and fluid requirements	IV: 14–16 ga × 1–2 NS/LR @ 2–4 mL/kg/h	Significant 3rd-space losses may require large volumes of crystalloid, colloid (up to 1 L) and PRBCs (not uncommon to transfuse 2 or more U to maintain Hct > 30). Central venous access is necessary for monitoring and for vasoactive drug delivery.
Monitoring	Standard monitors (see p. B-1). Art line CVP line UO	If a PA catheter is in place, it may be used or removed based on concerns of catheter-related, right-side endocardial lesions. Rarely is insertion of a PA catheter warranted in these operations. ABG, Hb/Hct, serum electrolytes, and glucose should be monitored hourly; for operations involving procurement of lungs or heart-lungs, ABGs should be obtained at least every 30 min.
Complications	Hypotension	Most commonly 2° hypovolemia and neurogenic shock (loss of descending vasomotor control). Ensure adequate volume repletion as described previously, then institute or increase inotropic/vasopressor therapy as previously outlined.
	Dysrhythmias	Multiple possible etiologies as described previously. Standard treatment and diagnosis should be employed, with the exception of bradycardia, which is atropine-resistant and should be treated with isoproterenol, dopamine, epinephrine, or transvenous pacing.
	Cardiac arrest	CPR should be instituted in an effort to maintain the viability of the liver, kidneys, and other abdominal viscera intended for transplantation. Procurement of the liver and kidneys should proceed rapidly to aortic cross-clamping at the diaphragm and administration of cold preservation fluid into the aorta and portal vein. This series of events will undoubtedly preclude use of the heart and lungs for transplantation.
	Oliguria	Ensure adequate volume replacement and BP as outlined, then add dopamine (2–3 mcg/kg/min), if not previously instituted to promote renal vasodilation and to increase renal blood flow, glomerular filtration rate, and UO. If these measures are ineffective at restoring adequate UO (> 1 mL/kg/h), then furosemide or mannitol may be used, after consultation with the transplant team. Fluid and electrolyte therapy as determined by filling pressures and hourly serum electrolyte values. Adjustment of vasopressin or DDAVP infusion to

	Diabetes insipidus (DI)	maintain UO < 1.5–3.0 mL/kg/h; initiation of this infusion should be done in consultation with the transplant team. As previously discussed, it may be advisable to D/C vasopressin or DDAVP at least 1 h before aortic cross-clamping.
	Coagulopathy	Transfuse Plt, FFP, and cryoprecipitate as necessary for clinical bleeding in the setting of abnormal coagulation studies. Avoid EACA due to risk of microvascular thrombosis in the donor organs.
	Hyperglycemia	Avoid dextrose-containing solutions, which may aggravate existing hyperglycemia and contribute to osmotic diuresis and electrolyte abnormalities.
	Hypothermia	Early aggressive attempts to minimize intraop heat loss are essential and include warming the OR, use of a forced-air warming blanket, insulating exposed areas warming all fluids, and using heated, humidified inspired gases.
Special considerations	Heart-lung procurement	Division of the mediastinal pleura and tracheal dissection with manipulation of each lung outside the mediastinum may result in ↓↓BP and may cause problems with oxygenation and ventilation. Adequate intravascular volume is essential, and inotropic therapy may be required during this period. Problems with ventilation and oxygenation must be communicated immediately to the transplant team. Following aortic cross-clamping and infusion of cardioplegia solution, the lung preservation fluid will be infused via the right and left PAs. During this period, the lungs should be ventilated manually with 4 bpm, or as otherwise directed by the transplant team. It is prudent early on in the procurement procedure to verify the position of the ETT with the transplant surgeon to ensure that the tube does not contribute to mucosal injury at the site of the anticipated suture line.
	Organ preservation	Therapy aimed at improving organ preservation may require several pharmacologic manipulations, as directed by the transplant team. Agents commonly used during organ procurement include dopamine (2–3 mcg/kg/min), furosemide, mannitol, allopurinol (free-radical scavenger), chlorpromazine and phentolamine (vasodilators), heparin (prevents microvascular thrombosis and promotes reperfusion), and PGE_1 (vasodilator, membrane stabilizer, antiplatelet effect). Systemic infusion of PGE_1 prior to aortic cross-clamping (commonly used in heart-lung or lung procurement) will lead to predictable and

profound ↓BP; efforts at volume resuscitation toward optimal CVP should continue until the aortic cross-clamp is applied. If heparin is to be administered iv, a catheter should be used after verifying the ability to freely aspirate blood. Methylprednisolone (30 mg/kg) is commonly administered at least 2 h before organ retrieval in an effort to protect the heart and kidneys from ischemic injury.

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Suggested Readings

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5. Robertson KM, Cook DR: Perioperative management of the multiorgan donor. *Anesth Analg* 1990; 70(5):546–56.

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