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CHAPTER 6.1

Cardiac Surgery

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Cardiopulmonary Bypass

Surgical Considerations

The development of cardiopulmonary bypass (CPB) technology has allowed the repair of many congenital and acquired lesions of the heart and great vessels. Designed to replace cardiac and pulmonary functions, full CPB requires a blood pump and oxygenator. The pump may be of the roller-head or centrifugal variety with the latter producing less trauma to formed blood elements. The oxygenator may bubble gases (O_2 and CO_2) through a blood-filled reservoir (bubble oxygenator), or allow O_2 and CO_2 to diffuse through a thin membrane into the surrounding blood (membrane oxygenator). Utilization of any blood pump requires at least partial heparinization (ACT > 180 sec), and introduction of an oxygenator mandates full heparinization (ACT > 400 sec).

Full CPB typically drains systemic venous return via the right atrium into a venous reservoir, from which the blood is pumped through an oxygenator and then returned to the aorta or femoral artery, completely bypassing the heart and lungs (Figs 6.1-1 and 6.1-2). **Partial CPB** usually supports only a portion of the body—typically the infradiaphragmatic portion—and may use the patient's lungs as an oxygenator (left atrium → femoral artery) or a mechanical oxygenator (femoral vein → femoral artery). Full CPB is utilized during a sternotomy for work on the heart, ascending aorta, and transverse arch. Partial CPB, in which some systemic venous blood returns to the heart and is ejected into the aorta, is normally used for work on the descending or thoracoabdominal aorta. Heparin-coated components, which partially eliminate the necessity for heparin, are available.

After exposure of the relevant organs (heart or descending thoracic aorta), and after heparinization, venous and arterial cannulae must be placed intraluminally. **Cannulation of the heart** usually involves venous drainage from the right atrium, with either two cannulae inserted through the atrium into the SVC and IVC (bicaval), or via a larger, dual-stage cannula draining the right atria and IVC. Bicaval cannulation reduces venous return (and rewarming) to the heart, and allows caval snares to be placed so that the right atrium can be opened without introducing air into the venous return. Occasionally, atrial manipulation for cannulation can depress CO with resultant hypotension. This usually can be reversed with volume replacement. Aortic cannulation usually is not associated with any physiologic perturbation, although HTN must be avoided to minimize aortic complications. After the cannulae are in place and connections are made to the bypass circuit, CPB may be instituted electively. Most cardiac operations are conducted under mild hypothermia (28°C), unless profound hypothermic circulatory arrest is to be utilized. In that case, a target temperature of 16–18°C is desirable. For operations on the descending thoracic aorta, normothermia is maintained.

Cessation of CPB is accomplished by gradually decreasing pump flows, allowing for right heart filling, and gradually replenishing the circulating blood volume. Pulmonary and coronary vasodilations are mandatory during this phase, as there appears to be heightened vasoreactivity after periods of ischemia and hypothermia. For periods of cardiac arrest, during which the heart is deprived of its arterial blood supply, the metabolic demands of the myocardium must be minimized. This usually is accomplished by achieving diastolic arrest with a hyperkalemic cardioplegic solution, and also by lowering myocardial temperature to < 15°C. Frequent reinfusions of cardioplegia maintain hypothermia, prevent lactic acid accumulation, and deliver some minimally available dissolved O_2.

The **physiologic response to CPB** is complex and is associated with a massive catecholamine release which resolves after its cessation. Subsequent changes include abnormal bleeding tendencies, increased capillary permeability, leukocytosis, renal dysfunction, and impairment of the immune response. Hemodilution, nonpulsatile flow, hypothermia, exposure of formed elements to nonendothelial surfaces, complement activation, protein denaturation, cascading effects within the coagulation and fibrinolytic system, and activation of the kallikrein-bradykinin cascade, all contribute to this unphysiologic state, and account for much of the morbidity and mortality after CPB.

Many physiologic variables are now controlled by the anesthesiologist, perfusionist, and surgeon, including systemic flow and

perfusion pressure, arterial O_2 and CO_2, temperature, and Hct. Other physiologic parameters follow either directly or indirectly. Thus, physiologic monitoring for the anesthesiologist and perfusionist include, at a minimum, arterial pressure, CVP, ABG determination (preferably on-line during CPB), CO, UO, and ECG. Constant communication among surgeons, perfusionist, and anesthesiologist is mandatory for a smooth operation. Transesophageal echocardiography (TEE) is rapidly becoming standard practice for cardiac surgery.

Secondary effects of CPB demand some special considerations during the final stages of the procedure and chest closure. Adverse effects on coagulation have already been mentioned, and vigorous attention to maintenance and replacement of coagulation factors is essential. The capillary leak phenomenon results in interstitial myocardial and pulmonary edema. Decreased myocardial performance and compliance mandate an increased preload, especially *(Print pagebreak 337) (Print pagebreak 338)* during the physical act of chest closure, where a transient rise in intramediastinal pressure may depress systemic venous return. Similarly, decreased pulmonary compliance and gas exchange mandate vigilance over inspiratory pressures and lung volumes during chest closure, because mediastinal volume is physically decreased.

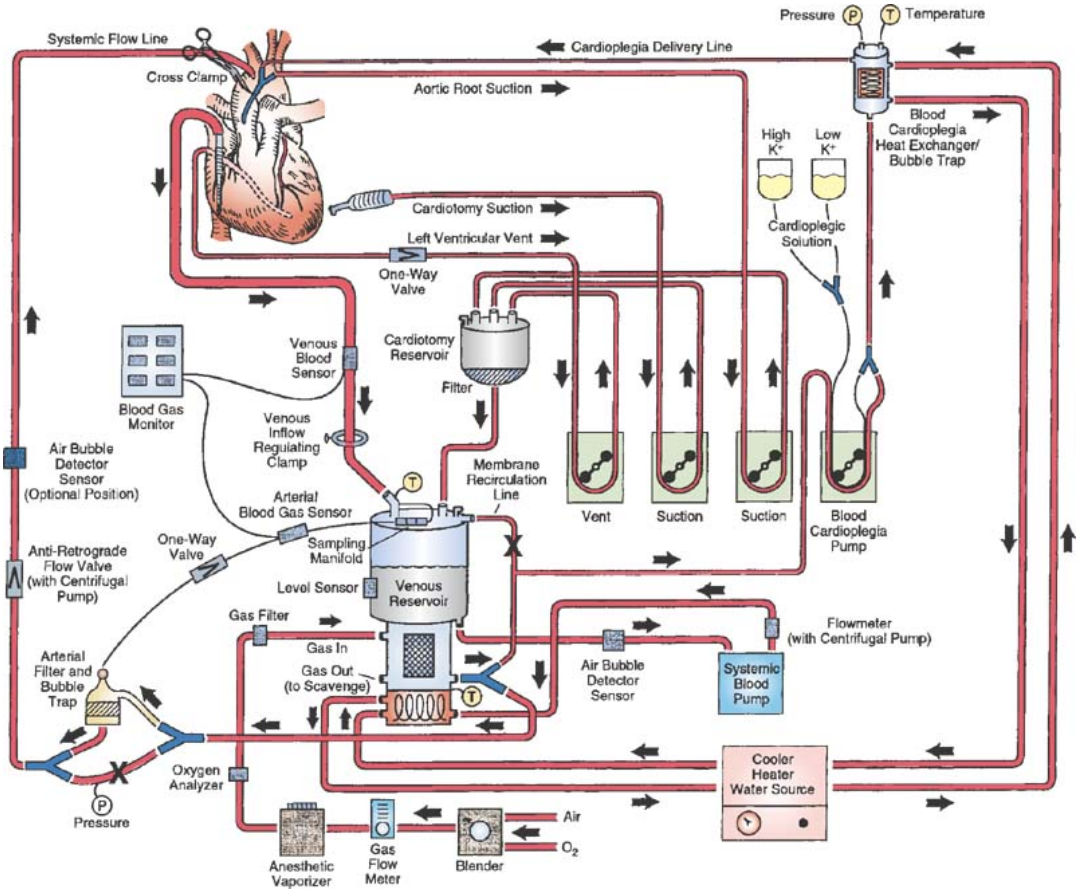

Figure 6.1-1. 1. Detailed schematic diagram of the arrangement of a typical cardiopulmonary bypass circuit using a membrane oxygenator with integral hard-shell venous reservoir (**lower center**) and external cardiotomy reservoir. Venous cannulation is by a cavoatrial cannula and arterial cannulation is in the ascending aorta. Some circuits do not incorporate a membrane recirculation line; in these cases, the cardioplegia blood source is a separate outlet connector built into the oxygenator near the arterial outlet. The systemic blood pump may be either a roller or centrifugal type. The cardioplegia delivery system (**right**) is a one-pass combination blood/crystalloid type. The cooler-heater water source may be operated to supply water to both the oxygenator heat exchanger and cardioplegia delivery system. The air bubble detector sensor may be placed on the line between the venous reservoir and systemic pump, between the pump and membrane oxygenator inlet, or between the oxygenator outlet and arterial filter (neither shown), or on the line after the arterial filter (optional position on drawing). One-way valves prevent retrograde flow (some circuits with a centrifugal pump also incorporate a one-way valve after the pump and within the systemic flow line). Other safety devices include an oxygen analyzer placed between the anesthetic vaporizer (if used) and the oxygenator gas inlet and a reservoir level sensor attached to the housing of the hard-shell venous reservoir (on the **left center**). *Arrows*, directions of flow; *X*, placement of tubing clamps; *P* and *T* (within circles), pressure and temperature sensors. Hemoconcentrator (described in text) not shown.

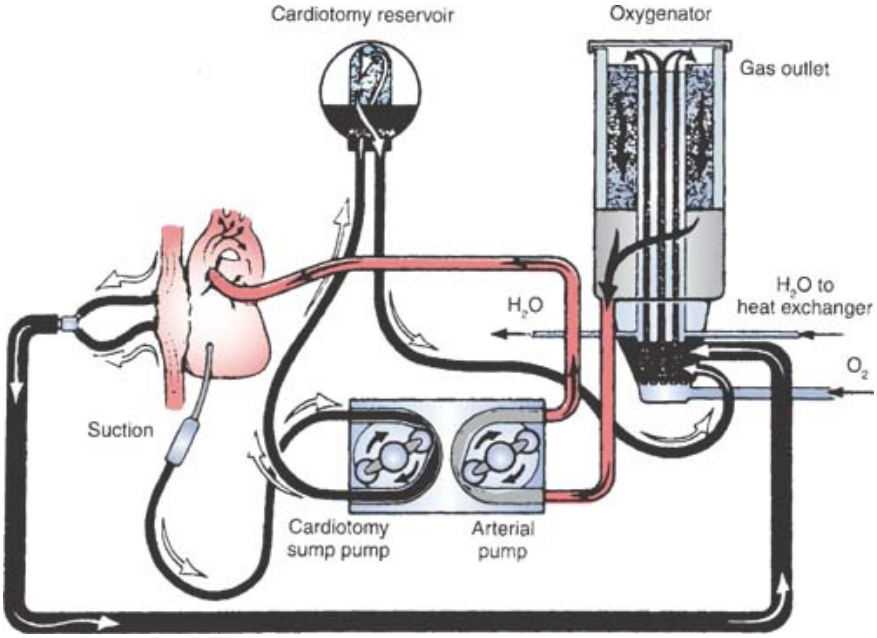

Figure 6.1-2. 2. Schematic representation of the CPB circuit. (Reproduced with permission from Hardy JD: *Hardy's Textbook of Surgery,* 2nd edition. JB Lippincott, Philadelphia: 1988.)

Anesthetic Considerations for Cardiopulmonary Bypass (CPB)

This segment is not meant to be a definitive text on CPB, but rather a guide to the anesthetic management of bypass. Communication among surgeons, anesthesiologists, and pump technicians is of vital importance in carrying out this procedure.

Preparation for Bypass

Prebypass/anticoagulation	baseline ACT (normal = 90–130 sec). Heparin (3 mg [300 U]/kg) is administered via a central vein (3 back-bleeding to verify intravascular position) or by the surgeon directly into the atrium (preferred). 3 ACT 3 min after heparin. It is essential to ensure adequate anticoagulation (ACT > 400 sec).
Aortic cannulation	Control MAP to 70 mmHg for cannulation to ensure that the aortotomy does not extend. If needed, vasodilators may be used. The arterial line should be inspected for air bubbles.
Venous cannulation	Venous cannulation may be associated with atrial dysrhythmias. Blood loss can be excessive. Be prepared to infuse volume boluses, if necessary.
Pupils	Assess pupil symmetry for later comparison.

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Transition onto CPB

Stop ventilation	Commencing bypass is a dangerous period for the patient as a result of the many hemodynamic changes that occur. D/C ventilation once there is no pulmonary blood flow.
Withdraw PA catheter	Withdraw PA catheter 4–5 cm.
Stop infusions	Stop iv drugs and reduce iv fluid infusions to TKO.
Anticoagulation	ACT should be ' d after 5 min on CPB to ensure anticoagulation (ACT > 400 sec).

Oxygenation	Verify oxygenation by checking arterial inflow color and inline sensors and by ABG within 5 min of beginning CPB. for adequate venous drainage (CVP falls to a low level).
Flow	Ensure adequate arterial inflow. Initial pressures may be very low, but usually will increase. View the descending thoracic aorta on TEE to verify flow (no aortic dissection).
Anesthesia	Anesthetics (e.g., fentanyl and midazolam) may be needed. A repeat dose (e.g., 10 mg pancuronium or 50 mg of rocuronium) should be given to prevent movement or shivering (increases O_2requirements).
Pupils	Assess pupils. Unilateral dilation may indicate arterial inflow into the innominate artery (unilateral carotid perfusion).

Bypass Period

Anticoagulation	ACT levels should be checked regularly (q 20–30 min) and kept at > 400. Add heparin (5000–10,000 U), if needed.
Pressure/flow	There is controversy about safe flows and pressures. Generally, flows of 1.2–3 L/m²/min are used, with pressures of 30–80 mmHg. A MAP of 50–60 mmHg is probably best for cerebral perfusion and does not result in excessive noncoronary blood flow.
Acid-base status	αstat (ABG measured and interpreted at 37°, regardless of actual patient temperature) regulation of acid-base status is preferred because of maintenance of normal cerebral flow and autoregulation on CPB.
Hct/Electrolytes	Generally, Hct will fall to 20, which may be acceptable (however, Hct ≤ 25 is preferable) in most patients. Hypokalemia is common and should be corrected. A K^+> 4.5 mEq/L is desirable. Blood glucose levels should be monitored q 1 h. Treat blood glucose ↑ 125 mg/dl with iv regular insulin. (See Table 6.1-2)
UO	Keep UO >1 mL/kg/h. If needed, mannitol and/or furosemide should be given (assuming pump flow is adequate).
Temperature	During bypass, T is usually maintained at 28°C.

Table 6. 1-1. Difference Between αStat and pH-Stat Management During CPB

αStat (Temperature-uncorrected)	pH Stat (Temperature-corrected)
Plasma pH maintained at 7.4 (when measured at 37°C).	Plasma pH maintained at 7.4 at actual patient temperature.
Arterial PCO_2maintained at 40 mmHg (when measured at 37°C).	Arterial PCO_2maintained at 40 mmHg at actual patient temperature. Requires addition of $CO_2$to inspired gases
Relative respiratory alkalosis during hypothermia	Relative normocapnia during hypothermia
Cerebral autoregulation preserved—CBF maintained.	Results in loss of cerebral autoregulation.

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Table 6. 1-2. Continuous Insulin Infusion in Intraoperative Adult Cardiac Surgery Patients

1. Bolus and start infusion pump as follows:

Blood Glucose	Insulin Bolus units	Insulin Drip units/h
< 125	0	0
125–175	5	1

175–225	10	2
> 225	15	3

2. Frequency of blood glucose determination: every 30 min intraoperatively!
3. Insulin titration:

Blood Glucose	Action
< 75	Stop insulin; give D50w and recheck blood glucose in 30 min. When blood glucose > 150, restart with rate 50% of previous rate.
75–100	Stop insulin; recheck blood glucose in 30 min. When blood glucose > 150, restart with rate 50% of previous rate, unless the dose is < 0.25 u/h.
101–125	If < 10% lower than last test, decrease rate by 0.5 U/h. If > 10% lower than last test, decrease rate by 50%. If neither continue current rate.
126–175	Same rate
176–225	If lower than last test—same rate. If higher than last test—increase rate by 0.5 U/h.
> 225	If > 19% lower than last test— same rate. If < 10% lower than last test OR if higher than last test increase rate by 1 U/h. If blood glucose > 225 and has not dcreased after 3 hourly increases in insulin, then double the insulin rate.

Termination of Bypass

Rewarming	Prior to discontinuing CPB, the patient should have a core temperature of at least 36°C.
Anesthesia/relaxation	Patient awareness may be a problem during rewarming. Volatile agents ± benzodiazepine (e.g., midazolam 3–5 mg) ± a narcotic to prevent awareness. In addition, a muscle relaxant also should be given.
Acid-base/electrolytes/Hct	electrolytes, acid-base status, and Hct. Correct acidosis. K^+ 4.5–5.5, normal ionized Ca^{++} and Hct ≤ 20 should be assured.
Air maneuvers	Air maneuvers (to remove intracardiac and intraaortic air) are carried out when the heart is opened. Ventilation is commenced after there is pulmonary blood flow and will aid in the evacuation of air. Pleural fluid should be removed.

Weaning from Bypass

Prior to weaning	Prior to weaning from CPB, the aortic cross-clamp should have been off for 30 min to allow rewarming and reperfusion of the heart. Defibrillation is often needed and pacing may be required. NSR or AV pacing is preferred. Vasoactive drugs should be available. After normal T, ventilation, cardiac rhythm, and reperfusion are established, bypass may be terminated. The heart is gradually volume-loaded (transfused from oxygenator) to adequate filling pressures and bypass flow slowly decreased over 15–45 sec until it is off. Return to bypass in the event of progressive cardiac distension or dysfunction. Assess CO and BP, and adjust vascular resistance as necessary. Inotropic agents often are required at this stage.
Reversal of anticoagulation	After the patient is off CPB, anticoagulation must be reversed with protamine (1–1.3 mg/100 U heparin), administered slowly over 10–20 min, because rapid administration can be associated with ↓ BP (treat with volume, calcium, αagonists as necessary). Other reactions include pulmonary HTN and true allergic reactions. 3 ACT to ensure that it has returned to

	control (90–130 sec).
BP management	Vasoactive support often is needed in the postbypass period; the need varies with surgical procedure, disease process, and underlying cardiac function. In general, SBP should be limited to 120 mmHg to avoid stress on the aortotomy site.

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Coagulation and CPB

General	Bleeding is common post-CPB and may be considerable. Both preop and intraop factors contribute to this. Knowledge of these factors and the tests involved will aid with the management of these patients (Table 6.1-3).
Preop factors	Hx of previous bleeding during surgery is important. Many drugs may contribute to bleeding: aspirin/NSAIDs/clopidogrel (Plt dysfunction), anticoagulants (heparin, Coumadin) and fibrinolytic agents. These should be stopped preop, if possible, or their action reversed. Other pathological processes (e.g., liver failure/congestion, renal failure, or hemophilia) also play a role. Preop testing is important and should include PT, PTT, Plt count and bleeding time, as a minimum.
Intraop factors	CPB is associated with ↓ Plt count and function. Circulating clotting factors are decreased and fibrinolysis occurs. Many surgical teams routinely use either Amicar (5–10 g loading dose; 1 g/h, maintenance) or Aprotinin (1 mL, test dose; 1–2 million KIU, loading; 0.25–0.5 million KIU/h, maintenance) prophylactically during cardiac surgery to inhibit the fibrinolytic process. Aprotinin also has anti-inflammatory properties that may offset the inflammatory response to CPB. Recent studies have associated aprotinin (and not aminocaproic acid or tranexamic acid) with increased risk of CV events, including MI and heart failure, cerebrovascular events and renal dysfunction/failure in patients undergoing CABG surgery.
Post-CPB bleeding	The common causes of post-CPB bleeding are: surgical causes, inadequate heparin reversal, ↓ number of Plt and Plt dysfunction, ↓ clotting factors, fibrinolysis, DIC, excessive BP, and hypothermia. Surgical causes and inadequate heparin reversal (3 ACT) should be ruled out. If no surgical cause is found and ACT is normal, Plt (1–2 plateletpheresis units) should be infused. PT, PTT, Plt count, TT, reptilase time, fibrinogen, and FSP should be checked. TEG may prove very useful. (See Fig 7.12-9 and Table 7.12-3 in Liver/Kidney Transplantation, p. 703.)

Table 6. 1-3 A Treatment Plan for Excessive Bleeding After Cardiac Surgery

Action	Dosage	Indication
Rule out surgical cause	—	Absence of oozing at puncture sites and incision
More protamine	0.5–1 mg/kg	ACT > 150 sec or aPTT > 1.5 times control
Warm the patient	—	“Core” temperature < 35°C
Apply positive end-expiratory pressure (PEEP)*	5–10 cm H_2O	
	0.3 mcg/kg IV	Prolonged bleeding time

Desmopressin		
Aminocaproic acid	50 mg/kg, then 25 mg/kg/h	Elevated D-dimer or teardrop shaped TEG tracing
Tranexamic acid	10 mg/kg, then 1 mg/kg/h	Elevated D-dimer or teardrop shaped TEG tracing
Platelet transfusion	1 U/10 kg	Platelet count < 100,000/mm^3
Fresh frozen plasma	15 mL/kg	PT or aPTT > 1.5 times control
Cryoprecipitate	1 U/4 kg	Fibrinogen < 1 g/L or 100 mg/dL

* PEEP is contraindicated in hypovolemia.

Suggested Readings

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Coronary Artery Bypass Graft Surgery

Surgical Considerations

Description: Coronary artery bypass grafting (CABG) is the most frequently performed cardiac operation. Since the discovery that coronary thrombosis is the causative event for MI, many schemes have been devised to augment the restricted coronary blood flow, including collateral pericardial blood flow to epicardial arteries and implantation of the internal mammary artery (IMA) with unligated side branches into the LV muscle. With the discovery by Favalaro that saphenous veins can be anastomosed to the epicardial coronary arteries, a new era of myocardial revascularization began. Basically, the technique involves bypass to a narrowed or occluded epicardial coronary > 1 mm in diameter with a small-diameter conduit (usually reversed saphenous vein or IMA) distal to the narrowed segment, with the proximal arterial inflow source being the ascending aorta. The IMA may be mobilized from the chest wall, leaving its proximal origin with the subclavian artery intact (pedicled graft), or the IMA may be transected and its proximal end anastomosed to the aorta or saphenous vein as a "free mammary graft." An **"all-arterial revascularization,"** using the right IMA and nondominant RA, is being utilized with increasing frequency.

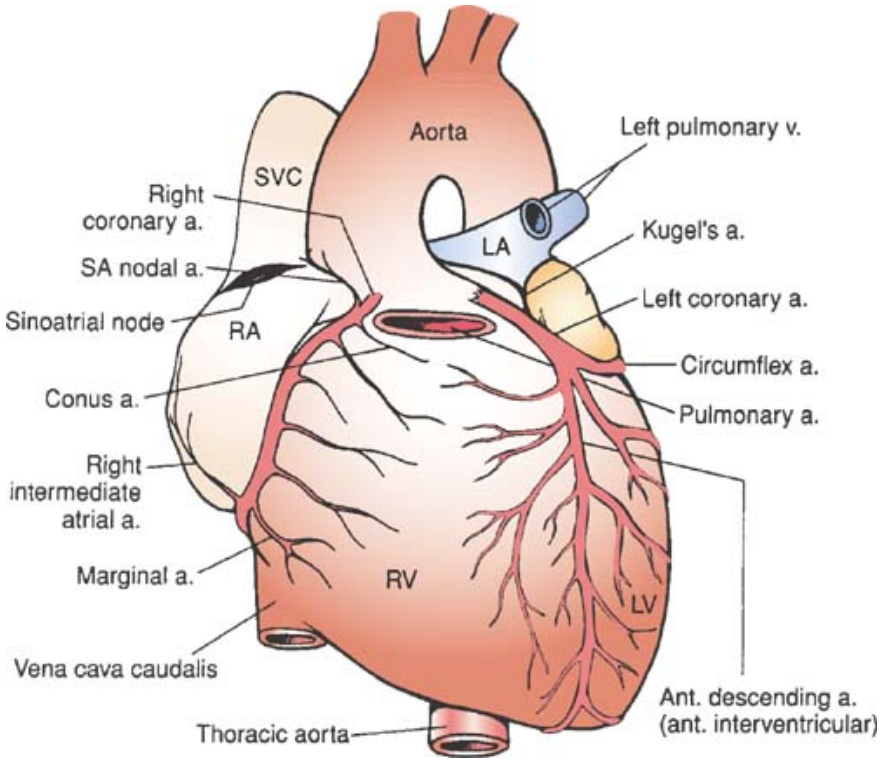

Figure 6.1-3. 3. Coronary artery circulation—anterior view. (Reproduced with permission from Edwards EA, Malone PD, Collins JJ Jr: *Operative Anatomy of the Thorax.* Lea & Febiger, Philadelphia: 1972.)

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The heart is approached through a median sternotomy, with the patient supported on full **CPB** (see separate section on CPB, p. 336). Although various operative strategies may be used, the most common regimen is for all distal (epicardial) anastomoses to be performed during a single period of aortic cross-clamping and cardiac arrest. During that period of induced asystole, myocardial protection is achieved by hypothermia and occasional reperfusion via antegrade or retrograde cardioplegia. Cardiac standstill and a bloodless field are mandatory to allow these very demanding small-diameter anastomoses to be constructed, with no obstruction to flow, in a minimal amount of time. The cross-clamp is then removed and the heart allowed to resume beating. A partially occluding aortic cross-clamp can then be applied to allow construction of the proximal aortic anastomoses. After a sufficient period of resuscitation, the patient is weaned from CPB, and decannulation, heparin reversal, and chest closure are allowed to proceed as previously noted.

The choice of conduit depends on availability and durability. Historically, the saphenous vein was the first small vessel conduit with acceptable patencies; but with prolonged experience it appears that 50% of vein grafts will be significantly diseased or occluded at 10 yr. The IMA appears to have superior long-term performance with 90% 10-yr patency rates. Other arterial conduits, such as the left gastroepiploic artery, superficial epigastric artery, and the radial artery, are being investigated as to their long- and short-term durability. Typical target arteries include the distal right coronary and its major terminal branch, the posterior descending artery. From the left circulation, the left anterior descending (LAD), with its diagonal and septal branches, is the most important, having been estimated to supply blood to 60% of the left ventricle.

The left circumflex coronary artery courses in the posterior atrioventricular groove, and is not easily accessible for bypass, which is usually performed to its obtuse marginal or posterolateral branches.

In a randomized study on coronary artery surgery (CASS), coronary bypass was noted to be superior to medical management for relief of angina, and to prolong life in patients with left main CAD and in those with three-vessel disease and impaired LV function. Other patients may receive bypass for intractable angina refractory to medical management.

Variant procedure or approaches: port access coronary artery revascularization (see p. 378); **off-pump and minimally invasive coronary artery bypass** (see p. 378).

Usual preop diagnosis: CAD with Class 3 or 4 angina (angina with minimal exertion or at rest)

Summary of Procedures

Position	Supine
Incision	Median sternotomy with legs prepped for saphenous vein harvest
Special instrumentation	Complete hemodynamic monitoring; TEE
Unique considerations	CPB
Antibiotics	Cefazolin 2 g iv prior to incision, then 1 g q 4 h
Surgical time	3.5–4.5 h
Closing considerations	Prevention of coagulopathy; maintenance of preload
EBL	500–600 mL
Postop care	ICU: 1–3 d, intubated 6–24 h.
Mortality	2–4%
Morbidity	Cognitive decline in patients > 60 yr: 26% MI: 3–6% Pneumonia: 5% CVA/stroke: 2–4% ↓ renal function
Pain score	7–8

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Patient Population Characteristics

Age range	60–80 yr (mean = 74 yr)
Male:Female	2:1
Incidence	Common
Etiology	Coronary atherosclerosis
Associated conditions	LV failure; pulmonary HTN; ischemic mitral regurgitation; diabetes mellitus; obstructive pulmonary disease

Anesthetic Considerations

(Procedures covered: CABG; LV aneurysmectomy)

Preoperative

H&P and tests will divide these patients broadly into two groups: (a) high-risk, characterized by poor LV function (cardiac failure; EF < 40%; LVEDP > 18 mmHg; CI < 2.0 L/min/m^2; ventricular dyskinesia; three-vessel disease; occlusion of left main or left main equivalent; valvular disease; recent MI; ventricular aneurysm; VSD; MI in progress; and old age); and (b) low-risk, characterized by good LV function. The type of monitoring chosen will depend on the patient's group.

Respiratory	Hx of smoking or COPD—patient should be encouraged to stop smoking at least 2 wk before surgery. Treat COPD and optimize therapy before surgery. **Tests:** CXR; PFTs; as indicated by H&P. In the preop assessment, the following factors will affect patient management and surgical outcome: • Hx of angina (stable, unstable at rest, and precipitating factors) Patient's exercise tolerance will provide a clue to LV

functions and surgical outcome.

Cardiovascular

- The presence of CHF (Sx: SOB, PND, orthopnea, DOE, pulmonary edema, JVD, 3rd-heart sound).
- Recent (< 6 mo) MI, dysrhythmias, HTN, vascular disease (particularly carotid stenosis, aortic disease).
- Valvular disease (particularly MR or AS) or the presence of a VSD or LV aneurysm may portend increased risk of periop complications.

Tests: 12-lead ECG: ischemia (area involved), LVH, previous MI, dysrhythmias. Exercise stress testing: exercise tolerance, area of ischemia, maximal HR and BP before ischemia occurs, dysrhythmias. Thallium scan: component of reversible ischemia. ECHO (may be combined with stress ECHO): LV function, wall motion abnormalities, valvular disease, VSD. Cardiac catheterization: extent and location of disease, LV function, valvular pathology, VSD, LVEDP, LV aneurysm.

Postinfarction VSD

The development of a postinfarction VSD is associated with high operative morbidity and mortality because of the difficulty in repairing the lesion due to friable tissue, difficulty in obtaining hemostasis, emergent nature of the condition, and possible pulmonary edema. These patients effectively have poor LV function and should be considered as high-risk patients. They often require support, including IABP, during induction, prebypass, and postbypass.

LV aneurysm

LV aneurysm is usually a late complication of infarction; however, it can occur early, when it usually is associated with cardiac rupture (and high mortality). These patients usually have poor myocardial function and should be anesthetized with full monitoring, including PA catheterization. Postbypass, the LV cavity is reduced in size and compliance. To ensure an adequate CO, maintain adequate preload, a higher than normal HR (A-V pacing if needed), and sinus rhythm, and consider the use of inotropes. LV aneurysms are often associated with dysrhythmias and may require cardiac mapping. Hemostasis is often difficult to obtain, and adequate iv access is a necessity. Treatment usually entails the use of blood and blood products.

Emergency revascularization

Emergency revascularization occurs in the setting of acute MI, often with acute LV failure or after failed PTCA, where the patient may be stable, suffering from acute ischemia and hemodynamically unstable, or even in full cardiac arrest. Factors to consider in these cases are: full stomach (and the need for rapid-sequence induction [p. B-4] in the face of ischemia); the prior use of fibrinolytic agents (with increased risk of hemorrhage); need for inotropes; antianginals; IABP; and dysrhythmias. These patients have a higher morbidity and mortality. In patients who have received fibrinolytic agents, consider postbypass use of antifibrinolytic agents (e.g., aminocaproic acid).

Neurological

Previous stroke or Hx/Sx of carotid artery disease should be documented and evaluated.

Endocrine

Diabetes is common and periop control of blood glucose is important.

Tests: Blood glucose

Renal

baseline renal function, as CPB places these patients at risk for renal failure.

Tests: Cr; BUN; electrolytes (particularly K+)

Patients are often on aspirin or other antiplatelet therapy, which may lead to increased intraop hemorrhage. These agents should

Hematologic	be stopped 7–10 d before surgery, if possible. Some patients may be on anticoagulants (usually heparin in the immediate preop period). Heparin should be stopped 6–8 h preop; however, in some patients, heparin infusion is continued into the OR. Other patients may have received thrombolytic agents that put them at increased risk for intraop hemorrhage.
Laboratory	**Tests:** Consider Plt count, PTT, if indicated Hb/Hct; other tests as indicated from H&P. T&C 2–4 U PRBCs.
Premedication	Patients should be instructed to continue all medications (e.g., nitrates, β-blockers; Ca^{++}antagonists, antidysrhythmics, and antihypertensives) before surgery, with the exception of diuretics on the day of surgery. Allaying anxiety may decrease the incidence of periop ischemia and help with the preinduction placement of lines. If necessary, preop sedation may include diazepam (10 mg po) or lorazepam (1–2 mg po) the night before and again 1–2 h before arrival in the OR with the addition of morphine (0.1 mg/kg im) and scopolamine (0.3 mg im). Most often midazolam (1–5 mg) iv immediately prior to OR is used. Severely compromised patients will require less premedication.

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Intraoperative

Anesthetic technique: GETA. An arterial line should be inserted, using liberal amounts of local anesthetic, before induction. The presence of real-time BP monitoring can be critical in the care of these patients, especially during induction. Although it is helpful to have a CVP line before induction for preload monitoring and drug infusion, it is not essential. This line usually is inserted after the patient is intubated. If infused drugs are necessary before the CVP catheter is in place, they can be administered through a separate peripheral iv.

Induction	Generally, a moderate- to high-dose narcotic technique (e.g., fentanyl 10–100 mcg/kg or sufentanil 2.5–20 mcg/kg), supplemented by etomidate (0.1–0.3 mg/kg) or midazolam (50–350 mcg/kg), is appropriate. As with all cardiac cases, the speed of induction and total drug dose depend on the patient's cardiac function and pathology. Muscle relaxation may be obtained using pancuronium (0.1 mg/kg), given slowly to avoid tachycardia, or vecuronium (possibility of bradycardia, especially if the patient is β-blocked). It is important to avoid the sympathetic response to laryngoscopy. The use of high-dose narcotics (see above), esmolol (100–500 mcg/kg over 1 min, followed by 40–100 mcg/kg/min infusion), SNP (0.5–3 mcg/kg/min), lidocaine (1–2 mg/kg), or a combination of these agents, may decrease or ablate this response. NTG (0.5–2 mcg/kg/min) also may be used during induction if evidence of ischemia occurs.
Maintenance	Usually narcotic (total: fentanyl 10–100 mcg/kg or sufentanil 5–20 mcg/kg) with midazolam (50–350 mcg/kg) for amnesia. Patients with good LV function may benefit from the decreased myocardial O_2

	demand associated with the use of volatile agents (2° ↓ contractility). N_2O is generally avoided. Propofol infusion may be used while rewarming and postbypass.
Emergence	Transported to ICU, sedated, intubated, and ventilated. Extubate when able—often < 6 h if lower-dose narcotic technique used (fast-track).
Blood and fluid requirements	IV: 14 ga × 1–2 NS/LR @ 6–8 mL/kg/h UO 0.5–1 mL/kg/h Warm all fluids. Humidify gases.
Monitoring	
Standard monitors (p. B-1)	Standard monitors and an A-line are placed before induction.
Arterial line	BP in both arms. Right radial preferred if left IMA graft, because retraction of the sternum may compress the left subclavian.
CVP or PA catheter	CVP (and/or PA line, if indicated), usually is placed after intubation. In the low-risk/good LV function group, CVP is adequate; in high-risk/poor LV function, a PA catheter is useful for hemodynamic monitoring, weaning from bypass, and vasoactive therapy. Some groups use routine PA catheterization for all patients undergoing CABG surgery.
ECG	Five-lead monitoring II and V_5 (or area most at risk for ischemia).
TEE	Will reflect regional wall motion abnormalities, papillary muscle dysfunction, and MR. The balance of myocardial O_2 supply vs demand is important in the management of these patients. The goal of

		anesthesia is to ensure that this balance remains in equilibrium and that no ischemia occurs, or, if it does, that it is treated promptly. Those patients with poor LV function or complicated disease will benefit from maintenance of contractility (avoid volatile agents) and a high FiO_2 whereas those with good LV function may benefit from mild cardiac depression (↓ demand associated with the addition of low-dose volatile agents). Certain events are associated with **increased risk of intraop ischemia:** intubation, incision, sternotomy, cannulation, tachycardia, ↑ BP or ↓ BP, ventricular fibrillation or distension, inadequate cardioplegia, emboli, spasm, or inadequate revascularization. Care should be taken to avoid these complications and to ablate responses to stimuli.
Myocardial O_2 Balance	Supply – Coronary blood flow: Perfusion pressure (DBP-LVEDP) Diastolic filling time (HR) Blood viscosity (optimal Hct = 30) Coronary vasoconstriction: Spasm $PaCO_2$(hypocapnia →constriction) a-sympathetic activity Supply – O_2delivery: O_2sat Hct Oxyhemoglobin dissociation curve Demand – O_2 consumption: BP (afterload) Ventricular volume (preload) Wall thickness (↓ subendocardial perfusion) HR Contractility	
Detection of ischemia	ECG	ST segment depression or elevation or a new T-wave alteration may suggest ischemia. Monitoring two leads—one lateral (e.g. V_5) and one inferior (e.g., II)—gives the best detection rate.
	PA catheter	Elevations of PCWP may be indicative of ischemia. A new V-wave on the PCWP trace is a better sign of possible ischemia

		(papillary muscle dysfunction).
	TEE	The appearance of a new regional wall motion abnormality is the most sensitive indicator of ischemia, but it requires constant monitoring.
Treatment of ischemia	Caused by tachycardia: Esmolol (100–500 mcg/kg) ↑ anesthesia Verapamil (2.5–10 mg iv) Caused by ↑ BP: NTG (0.5–4 mcg/kg/min) ↑ anesthesia Caused by ↓ BP: Phenylephrine (0.2–0.75 mcg/kg/min) ↑ preload Caused by ↓ ↓ HR: A-V pacing	While avoidance of ischemia is the goal, when it does occur it should be treated aggressively. Treatment may include inotropic support (e.g., dopamine 1–5 mcg/kg/min, epinephrine 25–150 ng/kg/min or milrinone 0.375–0.75 mcg/kg/min). If LV failure persists despite other therapy, an IABP or VAD may be inserted.
Positioning	and pad pressure points. eyes.	

Postoperative

Complications	Infarction Ischemia Tamponade Dysrhythmias Cardiac failure Coagulopathy Hemorrhage	Postop control of ischemia is important since hemodynamic instability may be associated with inadequate pain relief, awakening and ventilation.
Pain management	Parenteral opioids	Supplement with benzodiazepine for sedation.
Tests	ECG CPK CXR Electrolytes ABG Coag profiles	

(Print pagebreak 346)(Print pagebreak 347)

Suggested Readings

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2. DiNardo JA, Zvara DA: Anesthesia for myocardial revascularization. In: *Anesthesia for Cardiac Surgery*, 3rd edition. Blackwell

Science, Boston: 2008, Ch 4.

3. Moller JT, Cluitmans P, Rasmussen LS, et al: Long-term postoperative cognitive dysfunction in the elderly ISPOCD1 study. International Study of Post-Operative Cognitive Dysfunction. *Lancet* 1998; 351(9119):1888–9.

4. Munsch C: What cardiology trainees should know about coronary artery surgery–and coronary artery surgeons: ischaemic heart disease? *Heart* 2008; 94(2):230–6.

5. Myles PS, McIlroy D: Fast-track cardiac anesthesia: choice of anesthetic agents and techniques. *Semin Cardiothorac Vasc Anesth* 2005; 9(1):5–16.

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7. Roach GW, Kanchuger M, Mangano CM, et al: Adverse cerebral outcomes after coronary bypass surgery. Multicenter Study of Perioperative Ischemia Research Group and Ischemia Research and Education Foundation Investigators. *N Engl J Med* 1996; 335 (25):1857–63.

8. Rogers WJ, Coggin CJ, Green B, et al: 10-year followup of quality of life in patients randomized to receive medical treatment or coronary artery bypass graft surgery. *Circulation* 1990; 82(5):1647–58.

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Left Ventricular Aneurysmectomy

Surgical Considerations

Description: Extensive MI may → large areas of myocardial necrosis, with subsequent aneurysm formation. LV failure may ensue as a result of continuous LV dilatation or mitral insufficiency 2° annular dilatation or involvement of papillary muscles. Indications for this surgery include worsening CHF and increased dysrhythmias.

Typically, apical dilatation with maintenance of basilar myocardial contractility allows for aneurysm resection and preservation of both myocardial contractility and chamber size to produce an adequate CO. Operation is commenced in the usual manner—by establishing CPB (see p. 336), cross-clamping the aorta, establishing myocardial protection, and then assessing the left ventricle. A thinned, dilated ventricular segment with full-thickness scar formation can be resected and ventricular continuity restored with improvement of ventricular geometry and myocardial energy demands. Coronary bypass can be performed during this same period. Then air is removed from the left side of the heart, the cross-clamp is removed, and coronary perfusion is reestablished. After a sufficient period of resuscitation and return of vigorous contractility, bypass is D/C'd, not infrequently with the assistance of intraaortic balloon pump (IABP) to augment forward output. Decannulation, protamine administration, and closure proceed as described in CPB, p. 336.

Usual preop diagnosis: LV aneurysm with CHF

Summary of Procedures

Position	Supine
Incision	Median sternotomy, ± leg incision for saphenous vein harvest if coronary bypass is planned.
Unique considerations	Preparations (ECG leads) should be made for pre- or postop IABP or LV assist device (LVAD)
Antibiotics	Cefazolin 2 g iv prior to incision, then 1 g q 4 h

Surgical time	Aortic cross-clamp: 40–100 min CPB: 70–130 min Total: 3–4 h
EBL	300–400 mL
Postop care	ICU × 1–3 d, intubated 6–24 h; usually requires inotropic support ± mechanical support (LVAD, IABP).
Mortality	5–7%
Morbidity	Overall: 8–10% Requirement for IABP: 10% Respiratory insufficiency: 5% CVA: 2–3%
Pain score	7–10

Patient Population Characteristics

Age range	50–70 yr
Male:Female	3:1
Incidence	Uncommon
Etiology	Usually the end result of MI 2° CAD
Associated conditions	Mitral insufficiency; pulmonary HTN; CAD

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Anesthetic Considerations

See Anesthetic Considerations following Coronary Artery Bypass Graft Surgery, p. 344.

Suggested Readings

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Aortic Valve Replacement

Surgical Considerations

Description: Disease of the aortic valve may present as valvular stenosis, insufficiency, or a combination of the two. Valvular disease most commonly occurs as a result of rheumatic disease, but also may occur 2° calcific degeneration (aortic sclerosis) in the elderly. Congenitally bicuspid valves and endocarditis account for most of the remainder. Repair of the aortic valve is rarely possible, and most conditions require valve replacement. The three most commonly used **prostheses** are: porcine bioprostheses, especially in the older patient; mechanical prostheses with the necessity for lifelong anticoagulation; and cryopreserved homografts, which, unfortunately, are expensive and in short supply. The operation, on full CPB, usually is performed through a median sternotomy. After routine bicaval and aortic cannulation, the patient is taken onto full CPB. Left heart drainage is through a pulmonary artery vent, and a left atrial vent usually is inserted through the right superior pulmonary vein. Because of the LV

hypertrophy, myocardial protection is of utmost importance. Most centers favor hyperkalemic, hypothermic cardioplegic arrest, augmented by topical cooling, using either a continuous infusion of cold saline into the pericardial well or a cooling jacket. Cardioplegic administration can be achieved either antegrade into the coronary ostia or retrograde via the coronary sinus. Myocardial temperature is monitored continuously.

After the heart is arrested, the aorta is opened to expose the aortic valve. Continuous insufflation of the operative field with CO_2 reduces the amount of dislodged or trapped air. The rheumatic, stenotic valve, including aortic annulus, is frequently heavily calcified, and all Ca^{++} must be débrided to allow the prosthetic valve to be securely seated. This is frequently a tedious and time-consuming procedure, but one which then allows the remainder of the procedure to proceed in a timely fashion. After excision of the valve leaflets and debridement of the annulus, assuring that no particulate debris embolizes into the ventricle or coronary arteries, the annulus is measured to assure a proper match between prosthetic valve and annulus, and an appropriate valve prosthesis is selected. Interrupted sutures are placed through the annulus for its entire circumference and then passed through the sewing ring of the prosthesis. The prosthesis is lowered into the annulus and securely tied in place. Proper sizing and positioning are mandatory to prevent perivalve leaks or impingement on the coronary ostia. Systemic rewarming is initiated during the final stages of the valve implantation and the LV is allowed to fill during aortic closure. With the patient in the head-down position, all remaining air is vented from the left heart and aorta, and the cross-clamp is removed to allow myocardial perfusion. The heart is allowed to recover from this period of ischemia and, after sufficient resuscitation, with continuous venting of air from the aorta, the patient is weaned from CPB. Vasodilators are almost always utilized, as there appears to be excessive vasospasm present in both the coronary and pulmonary circulations after hypothermia. Decannulation and heparin reversal with protamine are then accomplished in the routine manner.

Usual preop diagnosis: Severe AS with syncope, chest pain or CHF; aortic insufficiency with CHF

Summary of Procedures

	Valve Replacement with Prosthesis	Homograft Valve Replacement
Position	Supine	
Incision	Median sternotomy	
Special instrumentation	TEE; hemodynamic monitoring; CPB	TEE or surface ECHO
Unique considerations	ECHO assessment of valve function and regional wall motion intraop	ECHO assessment of annular size and intraop evaluation of valve function after implantation
Antibiotics	Cefazolin 2 g iv prior to incision and 1 g q 4 h	
Surgical time	Aortic cross-clamp: 45 min CPB: 90 min Total: 3 h	60 min 105 min
EBL	300–400 mL	
Postop care	ICU: 1–3 d, intubated 6–24 h; hypertrophied, noncompliant LV requiring high preload.	
Mortality	5–8%	
Morbidity	Pneumonia: 5–10% Neurological sequela: Transient: 3–7% CVA: 1–2% Permanent: 1–2% Infection: 1%	
Pain score	7–10	7–10

Patient Population Characteristics

Age range	Bicuspid valves – 50–60 yr; rheumatic – 55–80 yr (mean = 58 ± 13 yr)

Male:Female	3:1
Incidence	70–100 cases/yr in tertiary care center
Etiology	Postrheumatic (majority of patients); aortic sclerosis; progressive stenosis of a bicuspid aortic valve; endocarditis
Associated conditions	Poststenotic dilatation of the ascending aorta (may require separate surgical attention); rheumatic mitral valvular involvement; CAD; CHF

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Anesthetic Considerations

Preoperative

Respiratory

Respiratory compromise may occur 2° pulmonary congestion (LV failure) and pleural effusion. An effusion, if significant, should be drained prior to surgery, as it may impair oxygenation and, with IPPV, may impair venous return →↓ CO + ↓ myocardial perfusion.
Tests: CXR
Aortic stenosis (AS): Sx are those of angina pectoris (if at rest may indicate concurrent CAD), syncope, and CHF (indicates severe disease with 2-yr life expectancy). The ejection murmur of AS is best heard at the 2nd right interspace. ECG shows LVH. Important points in the preop investigations include:

- Aortic orifice size: moderate AS = 0.7–0.9 cm^2; critical AS = < 0.5 cm^2 (normal = 2.6–3.5 cm^2)
- Aortic valvular gradient: severe = > 70 mmHg
- Ejection fraction (EF): ↓ EF indicates evidence of LV failure (normal = > 0.6).
- Coronary angiography: associated CAD often demonstrated.

AS →↑ LV work (↑ pressure load) →
LV concentric hypertrophy →↑ LV diastolic function + ↑ risk for ischemia (MVO_2 + O_2 supply).

Cardiovascular

- Reliance on atrial "kick:" important to maintain NSR.
- Sensitivity to changes in SVR:↓ SVR →↓↓ BP →↓ myocardial perfusion + ↓ CO →↓↓↓ BP.
- Sensitivity to volume changes: hypovolemia →↓ preload →↓ ↓ CO.
- Sensitivity to rate changes: tachycardia →↓ ejection time →↓ myocardial perfusion.
- LV wall tension + ↑ duration of systole →↑ MVO_2.
- ↑ LVEDP + ↑ wall thickness and tension + ↓ diastolic aortic pressure →↓ O_2 supply.

Aortic regurgitation (AR): Sx include DOE, orthopnea, PND, palpitations, and (less frequently) angina. Exercise tolerance may remain reasonably good, even with severe chronic AR. Acute AR is very poorly tolerated. The pandiastolic murmur of AR is loudest over the sternum and left lower sternal border.
AR → chronic LV volume overload →
LV eccentric hypertrophy →massive cardiomegaly →LV failure (CHF) →↑ LVEDP →↑ PA pressure and pulmonary congestion.

- Possibility of ischemia: ↑ MVO_2 and ↓ supply (↓ diastolic pressure, ↑ HR).
- Sensitivity to rate changes: ↓ HR →↑ AR + ↓ CO.
- Sensitivity to changes in SVR: ↑ SVR →↑ regurgitation + ↓ CO.

Tests: ECG: hypertrophy, LV strain, ischemia, rhythm. ECHO: LV function, valve area, regurgitant fraction. Angiography: LV function, right heart pressure, CAD, valve area, regurgitant fraction.

Hepatic

CHF may result in passive liver congestion with ↓ liver function and possible coagulopathy.
Tests: LFTs; PT; PTT

Neurological

Syncopal episodes may have resulted in neurologic deficits. These should be well documented.

Renal

Prerenal failure often is associated with AR 2° ↓ CO.
Tests: BUN; Cr, creatinine clearance, if indicated; electrolytes

Hematologic

Tests: Hb/Hct; clotting profile to investigate abnormalities. T&C for 8 U PRBCs.

Laboratory

digitalis level and electrolytes; other tests as indicated from H&P.

Premedication

Beware of oversedation in patients with AS where ↓ BP could be detrimental. Light premedication with an anxiolytic is usually sufficient. Digitalis and diuretics should be continued.

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Intraoperative

Anesthetic technique: GETA. An arterial line should be inserted, using liberal amounts of local anesthetic, before induction. The presence of real-time BP monitoring can be critical in the care of these patients, especially during induction. Although it is helpful to have a CVP line before induction for preload monitoring and drug infusion, it is not essential. This line usually is inserted after the patient is intubated. If infused drugs are necessary before the CVP catheter is in place, they can be administered through a separate peripheral iv.

Induction

AS: Typically, $O_2$with moderate- to high-dose narcotic (e.g., fentanyl 10–100 mcg/kg). Avoid sufentanil in AS because of ↓ BP. Etomidate (0.1–0.3 mg/kg), midazolam (50–350 mcg/kg) may be used to supplement the above. Paralysis with vecuronium or pancuronium (0.1 mg/kg, depending on desired HR). Induction is a critical period. CPB and surgeons should be available and ready to proceed. Danger is hypotension with a cycle of ischemia, further hypotension and more ischemia. Hypotension should be treated aggressively with fluid and αadrenergic agonists (phenylephrine 50–100 mcg iv bolus, infusion 0.1–0.75 mcg/kg/min). Critical AS patients may benefit from the use of phenylephrine as an infusion during induction and prebypass. The avoidance of hypotension is even more critical in the presence of CAD. Drugs causing tachycardia should be avoided. Atrial fibrillation (AF) or SVT should be treated with cardioversion. β-blockers and other negative inotropes are generally contraindicated. Ventricular irritability should be treated early as ventricular fibrillation may be refractory to defibrillation. Beware of vasodilators, including NTG.
AR: Again, $O_2$with moderate- to high-dose narcotic (e.g., fentanyl 10–100 mcg/kg or sufentanil 2.5–10 mcg/kg). Etomidate (0.1–0.3 mg/kg) or benzodiazepines (e.g., midazolam 50–350 mcg/kg) may be used to supplement the opiates. Muscle relaxation with pancuronium (0.1 mg/kg). These patients benefit from fluid augmentation, high normal HR (90 bpm) with afterload reduction (e.g., SNP 0.25–2 mcg/kg/min) to improve forward flow.

Maintenance

Narcotic (e.g., total fentanyl 10–100 mcg/kg). Low–dose volatile agent/air/O_2 Relaxant. (See Anesthetic Considerations for Cardiopulmonary Bypass, pp. 356.) The following table summarizes the goals of intraop management:

Parameter	AS	A
LV preload	↑	Normal-to-↑
HR	Normal-to-slow ↓	Modest ↑
Rhythm	NSR	NSR
Contractility	Maintain	Maintain
SVR	Modest ↑	↓
PVR	Maintain	Maintain

Postbypass

AS: Postbypass, patients may be hyperdynamic and require vasodilators for HTN, although inotropes also may be needed. Because of the hypertrophied, noncompliant ventricle, filling pressure may be higher than normally required.
AR: In the immediate postbypass period, patients with AR may require inotropic support (e.g., dopamine 3–10 mcg/kg/min, dobutamine 5–10 mcg/kg/min; epinephrine 25–100 ng/kg/min) is often needed. Maintain LV filling. Other supportive measures (e.g., IABP) may be necessary.

Emergence

Transport to ICU sedated, intubated (6–24 h), and ventilated. Early extubation may be possible if a lower-dose narcotic technique is used (fast-track).

Blood and fluid requirements

IV: 14 ga × 1–2
NS/LR @ 6–8 mL/kg/h
UO 0.5–1 mL/kg/h
Warm all fluids.
Humidify gases.
T&C 2–4 U blood

Monitoring

Standard monitors (p. B-1)	Standard monitors and arterial line should be placed before induction.
Arterial line	
CVP line PA catheter	CVP (and/or PA line, if indicated), usually is placed after intubation. CVP may underestimate left-side pressures. In acute AR, PCWP may underestimate true LVEDP.
ECG	V_5lead should be monitored for ischemia.
TEE	TEE may be useful to estimate LV filling and regional wall motion abnormalities.
Urinary catheter	

Positioning

and pad pressure points.
eyes.

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Postoperative

Complications

Hemorrhage
Tamponade
Cardiac failure
Dysrhythmias
Ischemia

Inotropic and vasodilator therapy usually is continued into the postop period, and then weaned.

Pain management

Parenteral opioids for pain relief
Benzodiazepine for sedation

Tests

ECG
CXR
Electrolytes
Coag profile
HCT

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Suggested Readings

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4. Murtuza B, Pepper JR, Stanbridge RD, et al: Minimal access aortic valve replacement: is it worth it? *Ann Thorac Surg* 2008; 85 (3):1121–31.

5. Tjang YS, van Hees Y, K´rfer R, et al: Predictors of mortality after aortic valve replacement. *Eur J Cardiothorac Surg* 2007; 32 (3):469–74.

Mitral Valve Repair or Replacement

Surgical Considerations

Description: Mitral valve repair or replacement is utilized typically for the correction of postrheumatic mitral valvular stenosis or insufficiency, as well as mitral valve prolapse, degenerative mitral insufficiency, or repair after endocarditis. For mitral regurgitation (MR) 2° posterior leaflet abnormalities (myxomatous degeneration, torn chordae) or pure annular dilatation, most valves can be repaired. For severe rheumatic calcific mitral stenosis (MS), mitral valve replacement with preservation of subannular structures may be necessary.

The technique of mitral valve repair or replacement is similar regardless of the mitral valve pathology. After aortic and bicaval venous cannulation, CPB (see p. 336) is established, and the left heart is vented through the PA. After cross-clamping of the aorta, diastolic arrest is accomplished with cardioplegia administered via the aortic root, augmented with topical cooling with either continuous pericardial saline infusion or a cooling jacket. Exposure is accomplished via a vertical incision in the left atrium just posterior to atrial septum. If the left atrium is not large enough, access may be gained via an incision in the right atrium and then incising the atrial septum, with caval snares in place to prevent air entry into the venous cannulae.

After suitable exposure, the atrium, atrial appendage, and mitral valve are carefully inspected, and a decision is made to repair or replace the valve. Repair of regurgitant valves caused primarily by posterior leaflet problems is usually possible. Similarly, annular dilatation 2° LV enlargement is also usually possible by means of a ring annuloplasty. Regurgitation 2° anterior leaflet abnormalities are more problematic, requiring significant expertise, and may be less durable. Preop and postop evaluations are greatly facilitated by use of TEE. Valve replacement can be performed after excising the valve leaflets; or, the leaflets may be preserved in an attempt to maintain the benefits of the subvalvar apparatus to global ventricular performance. After appropriate excision and debridement, the annulus is rimmed with interrupted sutures passed through the sewing ring of the valve prosthesis. The prosthesis is carefully positioned, and the sutures are tied. After filling both the LV and atrium with blood, the atrium is closed, air is evacuated from the left heart, and the cross-clamp is removed to allow coronary perfusion. After a satisfactory period of resuscitation, and after all air has been evacuated from the circulation, CPB may be D/C'd, usually with the assistance of vasodilator agents. TEE may be utilized at this stage to assess adequacy of mitral valve repair.

Variant procedure or approaches: Mitral commissurotomy may be done closed (e.g., during pregnancy).

Usual preop diagnosis: Class 3 or 4 CHF 2° mitral insufficiency or mitral stenosis

Summary of Procedures

	Mitral Valve Replacement/Repair	Closed Commissurotomy
Position	Supine	Right lateral decubitus

Incision	Median sternotomy	Left lateral thoracotomy
Special instrumentation	TEE and epicardial ECHO to assess valve function and regional wall motion; CPB.	Performed without CPB.
Unique considerations	Special loading conditions may be used to estimate the amount of MR, which is frequently afterload-dependent.	
Antibiotics	Cefazolin 2 g iv prior to incision, then 1 g q 4 h	
Surgical time	Aortic cross-clamp: 45–150 min CPB: 90–200 min	Total: 2 h Total: 3–4 h
EBL	300–400 mL	200–400 mL
Postop care	ICU × 1–3 d, intubated 6–24 h; vasodilator therapy for reversal of pulmonary HTN and afterload reduction.	
Mortality	5–8%	< 5%
Morbidity	Pneumonia: 10–15% CNS complications: Transient neurologic dysfunction: 5–10% CVA: 1–2% Infection: < 1%	
Pain score	7–10	7–10

Patient Population Characteristics

Age range	40–75 yr	20–40 yr
Male:Female	1:1	Almost exclusively pregnant females
Etiology	Postrheumatic: majority of cases; increased aging; myxomatous degeneration; ischemic etiologies (increasing)	
Associated conditions	Aortic valvular involvement with rheumatic disease, CAD, pulmonary HTN, and tricuspid regurgitation	

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Anesthetic Considerations

Preoperative

Respiratory	Pulmonary congestion, edema, pleural effusions may be present, with an overall restrictive lung pattern. Pleural effusions should be drained before surgery if significant (↓ oxygenation, ↓ venous return with IPPV). ↑ LA volume may compress the left recurrent laryngeal nerve →left vocal cord paralysis (Ortner's syndrome). **Tests:** CXR; PFTs, if indicated **Mitral Stenosis (MS):** Sx include exertional dyspnea and fatigue, progressing to pulmonary edema, atrial fibrillation (AF), and hemoptysis. Embolic events occur in 15% of patients. The opening snap and diastolic murmur of MS are best heard between apex and left sternal border. The ECG may show AF or wide-

notched P-waves. It is important to grade the severity of MS by symptomatology and valve area: mild = 1.5–2 cm^2; moderate = 1–1.5 cm^2; and severe = < 1.0 cm^2 (nl = 4–6 cm^2). AF is often the factor precipitating deterioration in these patients.
Because of the decreased LV filling, these patients are sensitive to:

- Loss of atrial "kick:" maintain NSR.
- Volume changes: keep full.
- Rate changes: avoid ↑ HR →↓diastolic filling time →↓ CO.

Pathophysiology:

- →↓ LVH filling →↓ CO.
- MS →↑ LA pressure →pulmonary edema + ↑ PA pressure →↑ PVR →RV failure + TR + left shift of iv septum →↓ CO →↑ LA pressure →↑ LA volume →AF and thrombi.

Mitral Regurgitation (MR): May be acute (MI/endocarditis) or chronic (often associated with MS). Chronic Sx include: palpitations, DOE, PND, fatigue, and orthopnea. Acute MR may →sudden ↓↓ CO and pulmonary edema. MR can be classified as mild (< 30% RF), moderate (30–60% RF), or severe (> 60% RF). The pansystolic murmur is loudest at the apex. The ECG may show LA and LV overload. MR patients are sensitive to:

Cardiovascular

- Changes in SVR:
  ↑ SVR →↑ RF →↓ CO + ↓ BP.
  ↓ SVR →↓ RF →↑ CO + ↑ BP.
- Change in HR:
  - ↓ HR →acute LA volume overload.
    Mild ↑ HR→↑ CO

Pathophysiology:

- Acute MR
  →↑ ↑ LA pressure →↓ CO + ↓ BP →↑ HR →↑ contractility →↑ O_2demand.
  →↑ LV diastolic volume →↑ LVEDP →↑ O_2demand.
  → Pulmonary edema.
- Chronic MR
  →↑ LA volume + AF → pulmonary edema → RV failure.
  → LV volume overload → LVH →↓ CO + LV failure.

Tests: ECG: LVH, left and right atrial enlargement, rhythm.
ECHO: RF, valve pressure gradients, LV function, valve pressure gradient and area.

- Changes in SVR:
  ↑ SVR →↑ RF →↓ CO + ↓ BP.
  ↓ SVR →↓ RF →↑ CO + ↑ BP.
- Change in HR:
  - ↓ HR →acute LA volume overload.
    Mild ↑ HR→↑ CO
- Acute MR
  →↑ ↑ LA pressure →↓ CO + ↓ BP →↑ HR →↑ contractility →↑ O_2demand.
  →↑ LV diastolic volume →↑ LVEDP →↑ O_2demand.
  → Pulmonary edema.
- Chronic MR
  →↑ LA volume + AF → pulmonary edema → RV failure.
  → LV volume overload → LVH →↓ CO + LV failure.

Neurological	The large left atrium and AF may result in thrombus formation, with the possibility of embolism. Neurological deficits should be documented.
Gastrointestinal	Hepatic congestion may → decreased function, which may be reflected in coagulation problems. **Tests:** Consider LFTs; PT; PTT
Renal	↓ CO may lead to renal failure. **Tests:** BUN; Cr; electrolytes
Hematologic	Because of the potential for thromboembolism, these patients may be on anticoagulants, which may be given up to the day before surgery. In the case of Coumadin, anticoagulant effects can be reversed by the use of FFP and vitamin K. **Tests:** Consider PT; PTT
Laboratory	T&C for 2–4 U PRBCs. Hb/Hct; coagulation profile; other tests as indicated from H&P.
Premedication	Premedication with an anxiolytic (e.g., lorazepam 1–2 mg po, midazolam 0.05–0.2 mg/kg im) or an analgesic (e.g., morphine 0.1–0.2 mg/kg); or a combination of these agents may be used, depending on patient status.

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Intraoperative

Anesthetic technique: GETA. An arterial line should be inserted, using liberal amounts of local anesthetic, before induction. The presence of real-time BP monitoring can be critical in the care of these patients, especially during induction. Although it is helpful to have a CVP line before induction for preload monitoring and drug infusion, it is not essential. This line usually is inserted after the patient is intubated. If infused drugs are necessary before the CVP catheter is in place, they can be administered through a separate peripheral iv.

Induction

Typically, moderate- to high-dose narcotic (fentanyl 10–100 mcg/kg or sufentanil 2.5–20 mcg/kg), supplemental midazolam (50–350 mcg/kg) or etomidate (0.1–0.3 mg/kg). Use pancuronium (MR) or vecuronium (MS) (0.1 mg/kg), depending on the desired HR to facilitate intubation.
MS: ↓ BP should be treated with fluid, but beware of precipitating pulmonary edema. Occasionally, phenylephrine may be needed to maintain SVR. Tachycardia should be avoided; if it occurs, it should be treated (↑ anesthesia, esmolol if ventricular function is preserved). Sinus rhythm should be maintained. New onset atrial flutter/AF should be treated with defibrillation. Avoid factors that may increase PVR (N_2O, acidosis, hypoxia, hypercarbia).
MR: Maintain or augment preload, depending on response to fluid load. Inotropic agents may be useful to maintain co ntractility. Afterload reduction will improve forward flow. Generally SNP (0.5–4 mcg/kg/min) is used, but NTG (0.5–4 mcg/kg/min) may be more appropriate in patients with ischemia-induced regurgitation. Avoid ↑ PVR caused by N_2O, acidosis, hypoxia, or hypercarbia. IABP may be useful periop in patients with acute MR 2° MI (↓ afterload, ↑ coronary perfusion pressure).

Maintenance

Narcotic (total = fentanyl 10–100 mcg/kg, or sufentanil 5–20 mcg/kg) with benzodiazepine (e.g., midazolam 50–350 mcg/kg) for amnesia; low-dose isoflurane; O_2/air.
The following table summarizes the goals of intraop management:

Parameter	MS	MR
LV preload	Normal-to-↑	Normal →↑
HR	↓	↑
Rhythm	NSR	NSR
Contractility	Maintain	Maintain
SVR	Normal	↓
PVR	Avoid ↑	Avoid ↑

Postbypass

Although patients with MS generally do well after valve replacement, inotropes (e.g., dopamine, dobutamine) occasionally are necessary. This is especially true late in the disease when cardiomyopathy may be present.

Emergence	Transport to ICU, intubated and ventilated 6–24 h. Early extubation is possible if lower-dose narcotic technique is used (fast-track).	
Blood and fluid requirements	IV: 14 ga × 1–2 NS/LR @ 6–8 mL/kg/h Maintain UO 0.5–1 mL/kg/h. Warm all fluids. Humidify gases.	
Monitoring	Standard monitors (p. B-1) Arterial line CVP line PA catheter Urinary catheter TEE	Standard monitors and an A-line are placed before induction. CVP (and/or PA line, if indicated), usually is placed after intubation. Care should be exercised with PA catheter insertion, because the dilated PA may be susceptible to rupture with the catheter. In MS, PCWP may over estimate the LV filling pressure due to stenosis. TEE may help in volume management, regional wall motion abnormalities (postacute MI) and in assessing the success of mitral valve repair. Occasionally, the use of a valve ring during repair may cause systolic anterior motion of the valve leaflet with resultant regurgitation, which may be seen on TEE.
Positioning	and pad pressure points. eyes.	

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Postoperative

Complications	Hemorrhage Tamponade Cardiac failure Dysrhythmias Conduction defects Atrioventricular disruption	Inotropic support or vasodilator therapy may be needed. IABP for mitral incompetence, especially in the presence of acute MR 2° infarction.
Pain management	Parenteral opioids for pain relief Benzodiazepine for sedation	
Tests	ECG CXR ABG Coag profile Electrolytes	

Suggested Readings

1. Calvinho P, Antunes M: Current surgical management of mitral regurgitation. *Expert Rev Cardiovasc Ther* 2008; 6(4):481–90.

2. Cook DJ, Housmans PR, Rehfeldt KH: Valvular heart disease: replacement and repair. In: *Kaplan's Cardiac Anesthesia,* 5th edition. Kaplan JA, ed. WB Saunders, Philadelphia: 2006, Ch 20.

3. Dunning J, Versteegh M, Fabbri A, et al. Guideline on antiplatelet and anticoagulation management in cardiac surgery. *Eur J Cardiothorac Surg* 2008.

4. Fann JI, Miller DC, Moore KA, et al: Twenty-year clinical experience with porcine bioprostheses. *Ann Thorac Surg* 1996; 62:1301–2.

5. Frasco PE, de Bruijn NP: Valvular heart disease. In: *Cardiac Anesthesia: Principles and Clinical Practice,* 2nd edition. Estafanous FG, Barash PG, Reves JG, eds. Lippincott Williams & Wilkins, Philadelphia: 2001, 557–84.

6. Greco E, Zaballos JM, Alvarez L, et al: Video-assisted mitral surgery through a micro-access: a safe and reliable reality in the current era. *J Heart Valve Dis* 2008; 17(1):48–53.

Tricuspid Valve Repair

Surgical Considerations

Description: Insufficiency of the tricuspid valve is almost always 2° left-sided valvular disease, with tricuspid annular dilatation and resultant valvular insufficiency usually 2° pulmonary HTN. Some congenital conditions (e.g., Ebstein's deformity) may persist into early adulthood, when replacement is usually necessary. Tricuspid repair is normally possible in the absence of primary involvement of tricuspid leaflets. The procedure is usually accomplished on CPB (see p. 336), either with the heart fibrillating or during a brief period of aortic cross-clamping and diastolic arrest. After instituting CPB and snaring the venous cannulae to prevent air entry into the pump circuit, the tricuspid valve is exposed through an incision in the right atrium. In the absence of leaflet involvement by the rheumatic process, repair usually can be accomplished by a simple **annuloplasty.** After atrial closure, evacuation of air, and myocardial resuscitation, the patient is weaned from CPB with vasodilator agents. Temporary pacing wires usually are inserted and passed to the anesthesiologist in case inadvertent injury to the AV node or His bundle causes complete heart block.

Variant procedure or approaches: Tricuspid valve replacement

Usual preop diagnosis: Tricuspid regurgitation, 2° annular dilatation 2° pulmonary HTN and left-side failure

Summary of Procedures

Position	Supine
Incision	Median sternotomy
Special instrumentation	Intraop ECHO; hemodynamic monitoring; CPB
Antibiotics	Cefazolin 2 g iv prior to incision, then 1 g q 4 h
Surgical time	Aortic cross-clamp: 30–40 min CPB: 70–80 min Total: 3–4 h
EBL	400–800 mL (Long-standing tricuspid regurgitation may → impaired hepatic production of coagulation factors.)
Postop care	ICU × 1–3 d, intubated 6–24 h; vasodilator therapy for pulmonary HTN
Mortality	2–3%
Morbidity	3rd-degree heart block: 10% RV failure: 2–3%
Pain score	7–10

Patient Population Characteristics

Age range	50–75 yr
Male:Female	1:1
Incidence	Rare
Etiology	Rheumatic; congenital (Ebstein's anomaly)
Associated conditions	Rheumatic involvement of left-side valves with pulmonary HTN

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Anesthetic Considerations

Preoperative

Cardiovascular	**Tricuspid regurgitation (TR)** usually is well tolerated and Sx (↓ CO) may go unnoticed, masked by Sx associated with left-side valvular disease and pulmonary HTN. Occasionally, TR is 2° endocarditis and raises the suspicion of iv drug abuse. TR may be caused by pulmonary HTN → ↑ RV afterload → RV dilation, ↑ RV wall tension → dilation of tricuspid valve annulus and TR. Pathophysiology: • TR →↓ CO 2° ↑ RV size → shift of the intraventricular septum to the left →↓ LV size, ↓ LV compliance → LV underloading due to ↓ LV size and ↓ RV stroke volume. • TR → atrial fibrillation (AF) 2° ↑ right atrial size. In isolated insufficiency, the ↑ in right atrial pressure may result in shunting across a patent foramen ovale (PFO), leading to paradoxical embolization with potentially disastrous consequences. **Tests:** ECG: AF. ECHO: relative chamber size, contractility, PFO, valve lesions. Cardiac catheterization: contractility, CO, pulmonary pressures and their response to vasodilators, other valvular pathology.
Respiratory	Pulmonary HTN (> 25 mmHg mean pressure), pulmonary edema, and effusions may be present. **Tests:** CXR: pulmonary edema, pleural effusion.
Renal	Chronic venous congestion may → prerenal failure. **Tests:** BUN; Cr; electrolytes
Hepatic	Hepatic congestion may → impaired synthetic function, particularly coag factors. **Tests:** Consider PT; PTT
Hematologic	**Tests:** Hb/Hct; other tests as indicated from H&P and Sx. Consider viral testing (HIV, hepatitis in isolated tricuspid endocarditis or in iv drug abusers). T&C 2–4 U PRBCs.
Laboratory	Other tests as indicated from H&P.
Premedication	Standard premedication (p. B-1) is usually appropriate.

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Intraoperative

Anesthetic technique: GETA. An arterial line should be inserted, using liberal amounts of local anesthetic, before induction. The

presence of real-time BP monitoring can be critical in the care of these patients, especially during induction. Although it is helpful to have a CVP line before induction for preload monitoring and drug infusion, it is not essential. This line usually is inserted after the patient is intubated. If infused drugs are necessary before the CVP catheter is in place, they can be administered through a separate peripheral iv.

Induction	Typically, O_2 and moderate- to high-dose narcotic (fentanyl 10–100 mcg/kg or sufentanil 2.5–20 mcg/kg) with etomidate (0.1–0.3 mg/kg) or midazolam (50–150 mcg/kg). Muscle relaxation is obtained with a nondepolarizing agent (e.g., pancuronium 0.1 mg/kg, vecuronium 0.1 mg/kg).	
Maintenance	The choice of narcotic/benzodiazepine/O_2 or volatile agent and O_2 will be determined by the underlying lesion and ventricular function. Avoid N_2O (pulmonary HTN). Low-dose isoflurane may be used. Benzodiazepines (e.g., midazolam 50–300 mcg/kg, diazepam 0.3–0.5 mg/kg) may be used for amnesia. Exact choice of agents for maintenance depends on underlying lesion, ventricular function, and coexisting disease. Management goals of TR are often those of coexisting valvular problems. The general principles, however, are: (1) Adequate preload (↓ preload →↓ RV stroke volume); (2) HR: normal-to-increased; (3) Contractility: normal. May require inotropic support for ↑ PVR, anesthetic myocardial depression or IPPV. (4) PVR: normal or decreased (high PVR →↓ RV stroke volume). (5) SVR: little effect unless it affects left-side pathology.	
Emergence	To ICU intubated and ventilated × 6–24 h. Early extubation may be possible if a lower-dose narcotic technique is used (fast-track).	
Blood and fluid requirements	IV: 14 ga × 1–2 NS/LR @ 6–8 mL/kg/h UO 0.5–1 mL/kg/h Warm all fluids. Humidify gases. T&C 2–4 U PRBC.	Because of the possibility of PFO and R→L shunt, ensure that all venous lines are clear of air.
Monitoring	Standard monitors (p. B-1) Arterial line CVP or PA catheter Urinary catheter TEE	Standard monitors and an A-line are placed before induction. CVP (and/or PA line, if indicated), usually is placed after intubation. CVP may be a poor indicator of RV or LV filling. The PA catheter may be helpful in the management of fluid balance, CO, and management of pulmonary HTN, but may be difficult to place and will require removal while valve or annular ring is placed. Because RV distention may affect LV filling and stroke volume, TEE may be useful in judging filling and relative LV size.
Pulmonary HTN	↑ PVR can result from: ↓ PaO_2 ↑ $PaCO_2$ ↓ pH N_2O αagonists Inadequate anesthesia	In TR, control of PVR is important because ↑ PVR may result in ↓ CO. Hypoxia, hypercarbia, acidosis, N_2O, or αagonists may ↑ PVR. PVR may be reduced with hypocarbia, inotropic support with dobutamine (5–10 mcg/kg/min), isoproterenol (10–50 ng/kg/min), or amrinone (loading 0.75 mg/kg, infusion 2–20 mcg/kg/min), and by using pulmonary vasodilators—NTG (1–4 mcg/kg/min), SNP (0.5–4 mcg/kg/min), prostaglandin E_1 (0.05–0.4 mcg/kg/min), or NO (0.1–100 ppm inhaled).
Positioning	and pad pressure points. eyes.	
	Coagulopathy	Coagulopathy 2° prolonged bypass time for

Complications	Hemorrhage	multiple valve replacements.
RV failure	RV failure 2° ↑ PVR (previously, RV decompressed by tricuspid incompetence). Rx includes inotropes: dobutamine (5–10 mcg/kg/min), isoproterenol (25–100 ng/kg/min), milrinone (loading 50 mcg/kg, infusion 0.3–0.75 mcg/kg/min), epinephrine (25–100 ng/kg/min), avoidance of factors causing ↑ PVR, and the use of pulmonary vasodilators.	

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Postoperative

Complications	Hemorrhage Coagulopathy Dysrhythmias RV failure Infection Renal impairment	Inotropic support and pulmonary vasodilators will need to be continued. Avoid those factors which ↑ PVR.
	Methemoglobinemia	Measure metHb levels during NO therapy.
Pain management	Parenteral opioids	Supplement with benzodiazepine for sedation.
Tests	ECG Coag profile Renal panel: BUN, Cr ABG Electrolytes	

Suggested Readings

1. Cook DJ, Housmans PR, Rehfeldt KH: Valvular heart disease: replacement and repair. In: *Kaplan's Cardiac Anesthesia,* 5th edition. Kaplan JA, ed. WB Saunders, Philadelphia: 2006, Ch 20.

2. Frasco PE, de Bruijn NP: Valvular heart disease. In: *Cardiac Anesthesia: Principles and Clinical Practice,* 2nd edition. Estafanous FG, Barash PG, Reves JG, eds. Lippincott Williams & Wilkins, Philadelphia: 2001, 557–84.

Septal Myectomy/Myotomy

Surgical Considerations

Description: Patients with asymmetric septal hypertrophy usually present with symptoms 2° LV outflow tract obstruction (LVOTO), increased diastolic dysfunction and stiffness, or a combination of the two, manifested as syncope, CHF, or severe chest pain. The systolic hemodynamic abnormality is caused by the anterior leaflet of the mitral valve being drawn into the LV outflow tract (LVOT), and abutting the asymmetrically hypertrophied intraventricular septum, narrowing the LVOT, and producing a large intracavitary gradient. Additionally, severe mitral insufficiency *(Print pagebreak 361)* may result. The most common surgical procedure for asymmetric septal hypertrophy is **septal myectomy/myotomy.** After institution of CPB, aortic cross-clamping, and cardioplegic arrest, the aorta is opened, and visualization of the subvalvar ventricular septum is attained. Bimanual palpation, as well as TEE visualization, can localize the asymmetric hypertrophy. Using the right coronary orifice as a landmark, the ventricular septum is longitudinally incised with two parallel incisions 1 cm apart, with care being taken to avoid injury of the papillary muscle or mitral valve chordae. A trough of the hypertrophied septum is then excised, alleviating the LVOTO. Removal of a portion of the asymmetrically hypertrophied myopathic septum also usually reduces the systolic anterior motion (SAM) of the anterior mitral leaflet, and reduces the intracavitary gradient and mitral regurgitation (MR). Infrequently, mitral valve replacement may be necessary for persistent MR.

Usual preop diagnosis: Asymmetric septal hypertrophy with CHF, chest pain, and/or syncope

Summary of Procedures

Position	Supine
Incision	Median sternotomy
Special instrumentation	TEE; hemodynamic monitoring; CPB
Unique considerations	Because of LV hypertrophy, maintenance of adequate preload is essential. Afterload also must be maintained to prevent LVOTO. Temporary pacing wires usually are placed at the start of the procedure.
Antibiotics	Cefazolin 2 g iv prior to incision, then 1 g q 4 h
Surgical time	2.5 h
EBL	150 mL
Postop care	ICU, intubated on ventilator 6–24 h
Mortality	3–4%
Morbidity	Complete heart block: 5% Persistent MR: 5% New aortic insufficiency: 2–5% CVA: < 1% VSD: < 1%
Pain score	6–10

Patient Population Characteristics

Age range	20–80 yr (mean = 45 yr)
Male:Female	1:1
Incidence	10/yr at Stanford University Medical Center
Etiology	Asymmetric septal hypertrophy

Anesthetic Considerations

Preoperative

Respiratory	Affected only 2° to cardiac failure.
Cardiovascular	Patients with idiopathic hypertrophic subaortic stenosis (IHSS) often present with Sx of syncope, angina pectoris (CAD), CHF, and palpitations. The main feature of IHSS is dynamic LVOTO 2° to septal hypertrophy and a possible venturi effect that draws the anterior mitral valve leaflet into the outflow tract. LVOTO →↑ LV hypertrophy →↓ compliance and ↓ diastolic function →↑ LVEDP, making diastolic filling dependent on preload and atrial contraction. Obstruction is worsened by decreased preload or afterload, increased contractility or HR. IHSS also results in ↑ MVO_2 and ↓ coronary perfusion, especially to the septum and subendocardium, which increases the risk of ischemia. Hypertrophy occurs throughout the whole myocardium and may lead to further dysfunction. MR, caused by a venturi suction effect, is often present and is exacerbated by the same conditions that increase LVOTO. (Note that ↓ afterload →↑ MR, unlike the usual [nonventuri-suction] form of MR.) Patients are often on β-blockers or Ca^{+} antagonists. These should be continued to day of surgery.

	Tests: ECG: Q-waves (indicative of septal hypertrophy); short PR interval with slurred QRS complex; supraventricular tachycardia; LV hypertrophy. ECHO: septal hypertrophy; MR; LV hypertrophy, myocardial dysfunction. Cardiac catheterization: LVOT pressure gradient, which may increase with provocation (e.g., Valsalva maneuver); obliteration of the LV cavity; MR; CAD.
Neurological	Document syncope and any neurological deficits.
Laboratory	Hb/Hct; electrolytes; other tests as indicated from H&P.
Premedication	Avoid activation of the sympathetic nervous system since this will cause ↑ HR and ↓ inotropy →↓ CO + ↓ BP. Thus, adequate anxiolysis is essential and can be obtained by premedication with a benzodiazepine (e.g., midazolam 0.05–0.2 mg/kg im, lorazepam 1–2 mg po, or diazepam 5–10 mg po). Avoid ↓ SVR, and maintain β-blockade and Ca^{+}channel blocker therapy.

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Intraoperative

Anesthetic technique: GETA. An arterial line should be inserted, using liberal amounts of local anesthetic, before induction. The presence of real-time BP monitoring can be critical in the care of these patients, especially during induction. Although it is helpful to have a CVP line before induction for preload monitoring and drug infusion, it is not essential. This line usually is inserted after the patient is intubated. If infused drugs are necessary before the CVP catheter is in place, they can be administered through a separate peripheral iv.

Induction	Typically, moderate- to high-dose narcotic (fentanyl 10–100 mcg/kg; avoid sufentanil due to vasodilation). Supplement with etomidate (0.1–0.3 mg/kg), midazolam (50–350 mcg/kg), or STP (2–4 mg/kg). Ketamine should be avoided due to the activation of the sympathetic nervous system. Introduction of a volatile agent prior to intubation may be helpful. Vecuronium (0.1 mg/kg) for muscle relaxation. Use pancuronium with caution (tachycardia) and avoid d-tubocurarine (↓ BP).	
Maintenance	$O_2$and narcotic (total fentanyl 10–100 mcg/kg) + volatile agent. Midazolam (50–350 mcg/kg) can be used for amnesia. The cornerstone of anesthesia for IHSS is to avoid factors which will increase LVOTO: (1) ↓ preload, (2) ↓ afterload, (3) ↑ contractility, (4) loss of NSR, and (5) ↑ HR.	
Emergence	To ICU intubated and ventilated × 6–24 h. Early extubation is possible if a lower-dose narcotic technique is used (fast-track).	
Blood and fluid requirements	IV: 14 ga × 1–2 NS/LR @ 6–8 mL/kg/h Maintain UO 0.5–1 mL/h Warm fluids. Humidify gases.	
Monitoring	Standard monitors (p. B-1)	Standard monitors and an A-line are placed before induction.
	Arterial line	
	PA catheter Urinary catheter	CVP (and/or PA line, if indicated), usually is placed after intubation. A PA catheter is useful for assessment of LV filling (keep high normal PCWP) and for monitoring SVR (normal-to-high). A pacing or pace port PA catheter may be useful for conduction problems occurring postop.
	TEE	TEE is useful to judge LV filling, LV contractility, LVOTO, MR, and VSD.
	↓ Preload	Rx: volume, phenylephrine Rx: phenylephrine

Intraoperative problems	↓ Afterload ↑ Contractility ↑ HR Loss of NSR Complete heart block VSD	Rx: esmolol, halothane Rx: esmolol Rx: cardioversion, verapamil Should have reliable means of pacing available, including temporary ventricular leads. Septum may be damaged, → VSD (diagnosed by TEE). If present, it should be repaired.
Position	and pad pressure points. eyes.	

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Postoperative

Complications	Hemorrhage Complete heart block Late VSD Tamponade	Dx: from chest tube drainage. Rx with blood and factors as needed. coag status. May require reexploration. Pacing should be available. Occasionally, inotropes of vasodilators required. β-blockers and Ca++antagonists usually D/C'd. Late VSD, indicated by development of a murmur, will require repair. Dx: by raising filling pressure, equalization of CVP, and PADP, ↓ CO, ↓ UO, and by ECHO. Rx: re-exploration and drainage.
Pain management	Parenteral opioids	Supplement with benzodiazepine for sedation.
Tests	ECG: conduction problems. ABG ECHO: LVOTO; VSD. Coag profile Electrolytes	

Suggested Readings

1. Cook DJ, Housmans PR, Rehfeldt KH : Valvular heart disease: replacement and repair. In: *Kaplan's Cardiac Anesthesia,* 5th edition. Kaplan JA, ed. WB Saunders, Philadelphia: 2006, Ch 20.

2. Frasco PE, de Bruijn NP: Valvular heart disease. In: *Cardiac Anesthesia: Principles and Clinical Practice,* 2nd edition. Estafanous FG, Barash PG, Reves JG, eds. Lippincott Williams & Wilkins, Philadelphia: 2001, 557–84.

3. Maron BJ: Hypertrophic cardiomyopathy: a systematic review. *JAMA* 2002; 287(10):1308–20.

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Pacemaker Insertion

Surgical Considerations

Description: Pacemaker insertion may be required for relief of abnormalities of the conduction system. A transvenous pacemaker

lead may be placed via the subclavian vein, through the tricuspid valve into the RV (single-chamber *(Print pagebreak 364)* pacing); or two leads may be placed, one into the right atrium and the other into the RV (dual-chamber pacing). Typically, 2nd- or 3rd-degree heart block is the diagnostic indication, although sick sinus syndrome (SSS) and other abnormalities also may be found. Access to the subclavian veins usually is attained percutaneously, although a cut-down may be used to expose the cephalic vein in the deltopectoral groove. Passage of a guide wire into the RV may cause frequent premature ventricular beats, which usually subside spontaneously with repositioning of the guide wire or lead. After ventricular and/or atrial lead placement, the pacing lead will have to be tested for sensing threshold, pacing threshold, depolarization amplitude, and lead resistance. After satisfactory placement of the pacing leads, the actual pacemaker generator unit is connected and then placed in a subcutaneous pocket at the site of percutaneous lead placement.

Usual preop diagnosis: Abnormalities of S-A nodal function (SSS) or A-V nodal function (heart block)

Summary of Procedures

Position	Supine
Incision	Left subclavicular; mostly percutaneous
Special instrumentation	Fluoroscopy with fluoro-table
Unique considerations	Patient usually awake with mild sedation
Antibiotics	Cefazolin 1 g iv prior to incision
Surgical time	1 h
EBL	Minimal
Postop care	PACU → CCU; ECG monitoring × 2–4 h; chest radiograph to document lead configuration.
Mortality	0–1%
Morbidity	Lead displacement: 1–2% Pneumothorax: 1–2%
Pain score	2

Patient Population Characteristics

Age range	50–80 yr
Male:Female	2:1
Incidence	Infrequent
Etiology	CAD; cardiac valve repair/replacement
Associated conditions	Complete heart block with syncope; SSS; paroxysmal tachycardia; SVT

Table 6. 1-4. Five-Position Pacemaker Code (ICHD)

I	II	III	IV	V
Chamber(s) paced	**Chamber(s) sensed**	**Response(s) to sensing**	**Programmability, rate modulation**	**Special tachyarrhythmia functions**
V - Ventricle	V - Ventricle	T - Triggered	P - Simple Programmable (rate and/or output)	B-Bursts
A - Atrium	A - Atrium	I - Inhibited	M -Multiprogrammable	N - Normal rate competition
D - Dual	D - Dual	D - Dual (T + I)	C - Communicating	S - Scanning
O - None	O - None	O - None	R - Rate modulation	
			O - None	E - External

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Anesthetic Considerations

Preoperative

The indications for permanent pacemaker insertion are usually bradydysrhythmias (e.g., 3rd-degree heart block, sinus node dysfunction, etc.) or, less commonly, tachycardia (e.g., atrial flutter not responsive to medical therapy). There are many different types of pacemakers, which are classified according to the chamber paced, chamber sensed, response to sensing, programmability, and antitachyrhythmia functions. The anesthesiologist should be aware of the type of pacemaker to be implanted and the means for external control.

Cardiovascular	Evaluate patient for associated disease, including CAD (50%), HTN (20%), cardiomyopathy, CHF, valvular defect, and for any symptoms or recent changes related to the conduction problems (syncope, CHF). It is important to know the reason for pacemaker implantation and the patient's escape rhythm. These patients are often on antidysrhythmics, diuretics, cardiac glycosides, and a variety of other cardiovascular agents, which should be continued up to the day of surgery. **Tests:** Exercise tolerance by Hx. ECG: rate, ischemic changes, rhythm. Other tests as indicated by H&P. serum digoxin and other antidysrhythmic levels.
Pacemaker	If a permanent pacemaker is already in place, its type and present functional state should be assessed. (Sx of problems include chest pain, palpitations, syncope, and weakness.) If no permanent pacemaker, a temporary transvenous pacemaker should be placed before surgery, except in unusual circumstances. **Tests:** ECG: Valsalva maneuver or carotid sinus massage—may slow the heart sufficiently to allow the permanent pacemaker to fire and allow assessment of function. A rate lower than the set rate may indicate battery failure. CXR: lead continuity.
Hematologic	Hct or Hb
Laboratory	K^+ level, which can affect pacing threshold, → loss of pacemaker capture, if low, or ventricular tachycardia, if high. Other tests as indicated from H&P.
Premedication	Standard premedication (p. B-1)

Intraoperative

Anesthetic technique: Permanent pacemakers commonly are placed via the transvenous route, requiring only local anesthesia and MAC with sedation. For epicardial pacemaker placement, GETA is required. These patients should be monitored during transport to OR. Care is taken to avoid dislodging temporary pacing electrodes. The function of the temporary pacemaker should be checked prior to induction.

General anesthesia:

Induction	Usually anesthesia can be induced with propofol (1–2 mg/kg) or etomidate (0.2–0.3 mg/kg) in combination with an opiate (e.g., fentanyl 1–2 mcg/kg) to attenuate the response to intubation. Vecuronium (0.1 mg/kg) or rocuronium (1 mg/kg) will provide adequate muscle relaxation.
Maintenance	Standard maintenance (p. B-2). Avoid excessive hyperventilation (→↓ K^+) and use volatile agents with caution, because they may increase A-V conduction time.
Emergence	Generally, patient is extubated at the end of the case.
Blood and fluid requirements	Minimal fluid requirements IV: 16 ga × 1

Monitoring	NS/LR @ 4–6 mL/kg/h Standard monitors (p. B-1) Urinary catheter	Rarely, arterial line if patient disease indicates.
Electrocautery interference	Minimize by: • Using bipolar cautery, if possible; • Grounding pad on leg; • Limiting cautery output; • Limiting cautery use.	The use of electrocautery may interfere with pacemaker function and → dysrhythmias or failure of the pacemaker. Monitor ECG and pulse (since electrical activity does not mean CO) for interference. Have a magnet available to convert the pacemaker to asynchronous mode (check that this is possible and will not change the programming of the pacemaker).
Complications	Dysrhythmias Air embolism Cardiac tamponade Hemo/pneumothorax Hemorrhage	Availability of temporary pacing is important because complete heart block or dislodgement of leads may occur. In addition, pharmacologic agents (atropine, 0.5–2 mg; isoproterenol 10–100 ng/kg/min) to increase HR should be available.

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Postoperative

Complications	Dislodging of electrodes Dysrhythmias Pneumothorax Tamponade
Pain management	Parenteral opioids for pain relief Benzodiazepine for sedation
Tests	ECG CXR Electrolytes

Suggested Readings

1. Luck JC: Arrhythmia, rhythm management devices, and catheter surgical ablation. In: *A Practical Approach to Cardiac Anesthesia,* 4th edition. Hensley FA, Martin DE, Gravlee GP, eds. Lippincott Williams & Wilkins, Philadelphia: 2008, Ch 4.

2. Reynolds DW, Murray CM: New concepts in physiologic cardiac pacing. *Curr Cardiol Rep* 2007; 9(5):351–7.

3. Rosenfeld LE, Elefteriades JA: Surgical and device therapy for cardiac arrhythmias. In: *Cardiac Anesthesia: Principles and Clinical Practice,* 2nd edition. Estafanous FG, Barash PG, Reves JG, eds. Lippincott Williams & Wilkins, Philadelphia: 2001, 617–36.

4. Rozner MA: The patient with a cardiac pacemaker or implanted defibrillator and management during anaesthesia. *Curr Opin Anaesthesiol* 2007; 20(3):261–8.

Pericardiectomy

Surgical Considerations

Description: Constrictive pericarditis, either acute or chronic, interferes with ventricular filling, reducing stroke volume and

depressing the cardiac index (CI). Although there are many possible etiologies (infectious, nephrogenic, postradiation), the cause remains unknown for a majority of patients. Typically, patients present with a progressive Hx of breathlessness, fatigability, or peripheral or abdominal swelling, often months to years after the inciting event. The Dx may be confirmed by cardiac catheterization, with equalization of end diastolic pressures, although volume loading may be necessary to demonstrate this in the patient under medical management. The differentiation between constrictive pericardial disease and restrictive myocardial disease may be difficult, if not impossible, and may coexist in a single patient. After this Dx has been confirmed, surgical **pericardiectomy** should be undertaken, because the outlook without surgical relief is one of gradual, but persistent deterioration. Although surgical mortality remains in the 10–15% range, long-term relief for survivors is good. Because these patients are usually significantly *(Print pagebreak 367)* compromised hemodynamically, intensive monitoring is indicated. Approach may be through a **median sternotomy** or left **anterolateral thoracotomy.** Removal of both visceral and parietal pericardium is essential for relief, but dense adhesions of these layers to underlying muscle may make this dissection very difficult, tedious, and bloody, especially if the visceral pericardium and epicardium are involved in the constrictive process. CPB (see p. 336) may be utilized for hemodynamic instability, but it obviously increases bleeding complications. Complete excision from both ventricular surfaces is mandatory. Most periop difficulties evolve from cardiac failure.

Variant procedure or approaches: A limited **pericardial window,** draining fluid into the left hemithorax, may relieve tamponade, but will be of no benefit for a true constrictive process.

Usual preop diagnosis: Constrictive pericarditis

Summary of Procedures

	Median Sternotomy	Anterolateral Thoracotomy
Position	Supine	Supine, with elevation of left hemithorax
Incision	Midline	5th interspace
Special instrumentation	TEE; full hemodynamic monitoring; CPB standby	
Antibiotics	Cefazolin 2 g iv prior to incision, then 1 g q 4 h	
Surgical time	2–5 h, depending on tenacity of visceral peel	
Closing considerations	Avoid volume overload and cardiac distention. Chronically depressed hearts may require inotropic or mechanical support (i.e., IABP) postop.	
EBL	100–500 mL	
Postop care	ICU; intubated × 4–6 h. Hemodynamic monitoring. Low CO state may persist postop.	
Mortality	5–15%, predominantly from cardiac failure	
Morbidity	Persistent CHF: 5% Transient phrenic nerve dysfunction: < 1%	
Pain score	7–10	7–10

Patient Population Characteristics

Age range	10–80 yr (median = 45 yr)
Male:Female	2:1
Incidence	3–4 patients/yr at tertiary referral center
Etiology	Majority unknown. Infectious, radiation, prior cardiac operation, rheumatic pericarditis, amyloid deposition.

Associated conditions	Restrictive myocardial diseases

Anesthetic Considerations

Preoperative

Pericardiectomy is performed most commonly for patients with constrictive pericarditis, whereas pericardial window procedures are used for patients with cardiac tamponade.

Respiratory	Restrictive disease may be present 2° fibrosis (e.g., post-TB) or pleural effusions. This may impair oxygenation and, if the effusions are significant, may ↓ venous return on institution of IPPV → rapid decompensation (↓ CO). Drain prior to surgery. **Tests:** CXR: active disease, fibrosis, effusions, pericardial calcification. Consider ABG, PFT, as indicated by CXR and Sx and if time permits.
Cardiovascular	Of importance is the presence or absence of a pericardial effusion. A large effusion that develops slowly (chronic pericarditis) may cause minimal Sx. Conversely, a small and rapidly forming effusion may → cardiac tamponade. Although the cardiovascular signs for both tamponade and constriction are similar (pulsus paradoxus, venous HTN, exaggerated venous pulsations, ↓ BP, tachycardia), it is important to differentiate between them because it may affect intraop management. Constrictive pericarditis can be differentiated from cardiac tamponade by ECHO, by pulsus paradoxus (frequent, with tamponade; rare, with constrictive pericarditis). Kussmaul's sign (distention of the jugular veins on inspiration) is rare with tamponade and common with constrictive pericarditis. Electrical alternans is present with tamponade and absent in constrictive pericarditis. Examination of the RV pressure wave form is unchanged in tamponade, but shows a dip and prominent Y descent in constrictive pericarditis. Anesthetic management is influenced by the planned procedure, the underlying process (constriction or tamponade), and its severity. Clues to severity are the physical symptoms and the degree of tachycardia, ↓ BP, and the filling pressure. (Although it is not possible to give exact figures, a HR of >100 bpm, systolic BP < 100 mmHg, and a filling pressure > 15 mmHg are probably significant.) In addition, it is important to assess concurrent cardiac problems: cardiomyopathy, CAD, or valvular disease (especially in constrictive disease associated with TB, radiation therapy, or in rheumatoid diseases, such as lupus). **Tests:** ECG: low-voltage complexes, electrical alternans. ECHO: pericardial effusion, calcification of pericardium, valvular lesions, myocardial function.
Gastrointestinal	Chronic hepatic congestion may →↓ synthetic function (↓ procoagulants). Development of ascites may →↑ intra-abdominal pressure. Because of this and the fact that these are sometimes emergency procedures, consider possible full stomach. **Tests:** Consider LFTs; PT; PTT
Renal	Renal failure may cause pericarditis and, conversely, pericarditis may cause renal failure (2° prerenal factors: ↑ venous pressure, ↓ perfusion pressure). This may affect the choice of drugs used for anesthesia that depend on renal clearance (particularly muscle relaxants). **Tests:** BUN; Cr; consider creatinine clearance; electrolytes.

Hematologic	Some renal or hepatic conditions may be associated with coagulation disorders. These include both procoagulant and Plt problems. If possible, any coagulopathy should be corrected before surgery with FFP, Plt, or both. Consult with a hematologist, if necessary.
Laboratory	**Tests:** Hb/Hct; PT; PTT; Plt count Other tests as indicated from H&P.
Premedication	Little or no premedication may be indicated; otherwise, a benzodiazepine (e.g., midazolam 0.05–0.2 mg/kg im) may be used. Consider full-stomach precautions: H_2antagonists (e.g., ranitidine 50 mg iv), metoclopramide (10 mg iv), antacids (e.g., Na citrate 0.3 M 30 mL po).

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Intraoperative

Anesthetic technique: GETA. An arterial line should be inserted, using liberal amounts of local anesthetic, before induction. The presence of real-time BP monitoring can be critical in the care of these patients, especially during induction. Although it is helpful to have a CVP line before induction for preload monitoring and drug infusion, it is not essential. This line usually is inserted after the patient is intubated. If infused drugs are necessary before the CVP catheter is in place, they can be administered through a separate peripheral iv. Consider pericardiocentesis or *(Print pagebreak 369)* pericardial window under local anesthesia prior to induction, as drainage of even a small amount of fluid may improve the patient's status dramatically. The considerable manipulation of the heart, extensive dissection, blood loss, dysrhythmias, and unrelieved tamponade make pericardiectomy cases a challenge.

Induction	Typically, ketamine (1–2 mg/kg) or etomidate (0.2–0.3 mg/kg) ± narcotic (fentanyl 2–30 mcg/kg), depending on patient status. Consider maintaining spontaneous ventilation in tamponade patients until drained, as institution of IPPV may result in rapid decompensation and cardiac arrest due to ↓ ↓ venous return. Otherwise, succinylcholine (1 mg/kg) with cricoid pressure (full stomach) or vecuronium (0.1 mg/kg) for muscle relaxation. For unstable patients, may prep and drape and have surgeons standing by before induction of anesthesia.
Maintenance	Narcotic (total fentanyl 5–50 mcg/kg), low-dose volatile agent, midazolam (50–350 mcg/kg), or a combination of these agents in 100% O_2 CO is dependent on maintaining a high preload to ensure adequate cardiac filling, avoiding and treating bradycardia and preserving myocardial contractility. The use of inotropes (dopamine, isoproterenol, or epinephrine) may be necessary; αagonists should be avoided but, on occasion, may be needed to increase coronary perfusion. These anesthetic-considerations apply to both tamponade and constrictive disease. In patients with acute tamponade, dramatic increases in BP may occur when the pericardium is opened. Aggressive use of vasodilators and additional anesthetic agents may be necessary.
Emergence	In general, plan for extubation in the OR in the case of pericardial window and transport to ICU for postop ventilation 4–24 h following pericardiectomy.
Blood and fluid requirements	IV: 14 ga (or 7 Fr) × 1–2 UO: 0.5–1 mL/kg/h Warm fluids and humidify gases. T&C 2–4 U for pericardiectomy. During pericardiectomy, anticipate rapid blood loss (a major cause of mortality). For this reason, CPB should be available on standby for all pericardiectomy procedures.
Monitoring	Standard monitors (p. B-1) Arterial line CVP line PA catheter TEE Urinary catheter Standard monitors and an A-line are placed before induction. CVP (and/or PA line, if indicated), usually is placed after intubation. PA catheters aid the management of filling pressures, CO and afterload. TEE may be useful to gauge filling volume and degree of relief of pericardial constriction. Intrathoracic pressure associated with IPPV may produce a ↓ ↓ CO in these patients

		(because of ↓ venous return). Spontaneous ventilation, therefore, is preferred until the tamponade is drained.
Complications	Cardiac tamponade Dysrhythmias Hemorrhage Coagulopathy Heart failure	Hemorrhage is not usually a problem unless penetrating trauma is the cause of the tamponade. Once constriction is relieved, myocardial function does not return to normal quickly. Inotropes are often needed. Due to extensive dissection and hemorrhage, coagulopathies may develop and should be treated aggressively.
Positioning	and pad pressure points. eyes.	

Postoperative

Complications	Hemorrhage Coagulopathy Ventricular hypofunction Dysrhythmias Ischemia	Following pericardial window surgery, patients improve with the relief of the tamponade. Postpericardiectomy patients, however, may have a continuation of intraop dysrhythmias and myocardial depression. Inotropes (e.g., dopamine 5–10 mcg/kg/min) may be needed for 24–48 h. Normal cardiac function can take 4–6 wk to return.
Pain management	Parenteral opioids	Supplement with benzodiazepine for sedation.
Tests	ECG CXR ABG Coag profile Electrolytes	

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Suggested Readings

1. DiNardo JA, Zvara DA: Pericardial disease. In: *Anesthesia for Cardiac Surgery,* 3rd edition. Blackwell Science, Massachusetts: 2008, Ch 8.

2. Hoit BD: Pericardial disease and pericardial tamponade. *Crit Care Med* 2007; 35(8 Suppl):S355–64.

3. Savage RM, Aronson S, Shanewise JS, et al: Intraoperative echocardiography. In: *Cardiac Anesthesia: Principles and Clinical Practice,* 2nd edition. Estafanous FG, Barash PG, Reves JG, eds. Lippincott Williams & Wilkins, Philadelphia: 2001, 237–94.