Cardiac Anesthesia for the Occasional Cardiac Anesthesiologist

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Introduction

For more than 20 yr, the vast majority of my practice has been in patients undergoing heart surgery. However, outside university practice, a full-time cardiac anesthesia practice is the exception to the rule. Because of this and because cardiac surgery is performed in thousands of nonuniversity hospitals, many anesthesiologists are called on, occasionally, to provide cardiac anesthesia. This can be an anxiety-provoking experience, especially when the other members of the cardiac surgery team are full-time cardiac specialists and when the literature documents that the anesthesiologist is an independent predictor of outcome in cardiac surgery (1).

The purpose of this article is to summarize the practical points of providing cardiac anesthesia that make it safer and more efficient. This article cannot be a definitive treatise on the subject, but it will provide many of the essential points I have learned over the past 20 yr in my role as a provider and teacher of cardiac anesthesia.

Perioperative Assessment and Management

A few special issues should be considered in patients scheduled for heart surgery. The most obvious are cardiac anatomy and function. Specifically, you want to know whether the patient has good or bad (ejection fraction of <40%) ventricular function or any significant valvular disease. This information will assist you in defining your hemodynamic goals and monitoring strategies (see below). Also, you will want to note whether the patient has a history of hypertension, peripheral vascular disease, diabetes, or chronic lung or renal disease. As you will see, each of these problems may affect your management during cardiac surgery.

In general, most chronic medications should be continued until surgery, especially β-blockers, nitrates, and IV heparin. The three exceptions are warfarin, digoxin, and subcutaneous insulin. Warfarin should be discontinued several days before surgery to allow its effects to resolve, and a short-acting anticoagulant should be started in its place (2,3). Digoxin need not be given on the day of surgery unless it is required for control of heart rate in patients with atrial fibrillation. Insulin requirements vary markedly during heart surgery. Therefore, I recommend a continuous infusion of insulin in patients with diabetes when their blood sugar exceeds 250 mg/dL. The starting infusion rate is easy to estimate: divide the blood sugar level by 100, and that gives you the infusion rate of regular insulin in units per hour. Measure the blood sugar every 30 min, and adjust the infusion rate to keep the blood sugar <250 mg/dL.

Patients should be informed that they may require transfusions during surgery and that they will likely awaken with an endotracheal tube in place. I do not discuss the risk of awareness with my patients, because it has been so rare in my practice (once). Most important, at the conclusion of the preoperative assessment, clearly formulate the hemodynamic goals and overall management plan for the patient. In the next section, I've outlined an approach to the determination for hemodynamic goals. These are the guides that will keep you on track when the going gets tough.

Determination of Hemodynamic Goals

Determine your goals for the major hemodynamic variables affected by anesthesia: preload, heart rate, systemic vascular resistance, pulmonary vascular resistance, contractility, and rhythm. These goals depend on the cardiovascular pathology of the patient. For instance, when ventricular filling is impaired because of hypertrophy, our goals for preload are high filling pressures, adequate volume administration, and avoidance of factors that decrease venous return. Note that the hemodynamic goals during anesthesia may be quite different from the goals during chronic care of the patient. In the same patient with ventricular hypertrophy, a primary care physician may have spent months reducing preload ("pruning") to relieve symptoms of pulmonary congestion. However, this state of relative dehydration may be disastrous in some patients during the induction of anesthesia.

Similarly, heart rate must be maintained at low normal levels in patients with dynamic ventricular outflow obstructions (hypertrophic obstructive cardiomyopathy) to allow for adequate ventricular filling and ejection. Systemic vascular resistance should be reduced in patients with mitral regurgitation to promote forward ejection of blood, and it should be maintained, or even augmented, in patients with aortic stenosis to provide adequate coronary artery blood flow. In anesthesia dogma, too much emphasis has been placed on maintaining cardiac contractility. Indeed, the most successful cardiac drugs of our time have been myocardial depressants. Most patients tolerate modest decreases in contractility, and some—those with coronary artery disease and those with hypertrophic obstructive cardiomyopathies—benefit. In contrast, no patient is improved by the loss of sinus rhythm, and some—those with impaired ventricular filling, who need their atrial "kick" to maintain an adequate stroke volume (i.e., aortic stenosis)—tolerate it quite poorly.

In each patient, some goals are more important than others, and this allows the clinician to prioritize management and interventions. For instance, a relatively slow heart rate (<80 bpm) and high systemic vascular resistance (>1500 dynes $\cdot$ s $\cdot$ cm^{-5}) are most important in patients with severe mitral stenosis. If such a patient is tachycardic, hypotensive, and overtly in congestive heart failure, the correct interventions may include a β-blocker and a vasoconstrictor. Clearly, these interventions would be grossly inappropriate in many patients with congestive heart failure, but they will be effective in this patient because they will restore the appropriate hemodynamic goals.

Monitoring

Invasive arterial and central venous pressure determinations are standards of care, whereas pulmonary artery pressure monitoring is used more selectively. We use ultrasound guidance to cannulate the internal jugular vein, because it increases the success rate and reduces the incidence of carotid puncture (4–6). The literature provides no studies indicating improvement in patient outcome resulting from the routine use of pulmonary artery catheters. As a rule, I place a pulmonary artery catheter when I anticipate hypotensive episodes whose etiologies will not be readily deciphered with the other monitors available. Typically, this is the patient with severe ventricular or pulmonary dysfunction who is expected to require a few days of intensive care support. In contrast, our "fast track" cardiac patients almost never receive a pulmonary artery catheter. However, our standard central venous pressure device is an introducer sheath so that a pulmonary artery catheter could be placed quickly if needed.

The electrocardiogram is the principle tool used for the diagnosis of myocardial ischemia. Therefore, appropriate calibration and lead selection are essential. Standard calibration is 1 cm/mV. At this calibration, 1 mm of ST depression equals 0.1 mV. However, a 1-mm change is very difficult to see on a monitor, and therefore, I recommend that you double the standard calibration to 2 cm/mV. Lead V_5 (fifth intercostal space, anterior axillary line) is the most sensitive single lead for detection of ischemia (7). Place it exactly in its correct location. A few centimeters out of position can dramatically reduce its sensitivity for ischemia. The second lead to monitor is lead II, because it significantly improves the sensitivity for ischemia and usually reveals the P wave for dysrhythmia diagnosis. A few extra seconds to carefully apply the electrodes to the skin securely will reward you with a more stable wave form to interpret.

Rectal or bladder and posterior nasopharyngeal (not esophageal) temperatures are measured routinely. Rectal or bladder temperature provides a good estimate of the core temperature, whereas nasopharyngeal temperature provides a good estimate of central and brain temperature. Usually during bypass, the esophageal temperature is the same as the arterial blood temperature of the bypass machine and is already known.

In our practice, transesophageal echocardiography (TEE) is performed in all patients undergoing heart surgery. We use a simplified examination designed to fulfill the requirements for basic TEE delineated in the American Society of Anesthesiologists/Society of Cardiovascular Anesthesiologists guidelines (8).

Choice of Anesthetics and Adjuvants

Keep your plan as simple as possible. The only special "cardiac" drug that I routinely have ready to go before the induction is phenylephrine (100 μg/mL). In addition, I verify that esmolol, nitroglycerin, epinephrine, and heparin are available on my cart, but rarely will I draw them up before the induction. I have plenty of time to make dopamine, epinephrine, and other infusions during bypass, if it looks as if they will be needed. Occasionally, I have etomidate ready for the induction, but it burns on injection and is expensive, so I rarely use it. Similarly, our usual surgical duration makes it appropriate for me to use pancuronium in most patients, rather than a shorter acting and more expensive alternative.

Most patients undergoing first-time cardiac surgery at the University of California-San Francisco will receive ϵ amino caproic acid starting before bypass (5 g over 10–20 min, then 1 g/hr thereafter). In patients undergoing reoperation, aprotinin is used instead. These drugs decrease blood loss and may limit the need for transfusions. In addition, we administer 1 g

of a cephalosporin antibiotic before skin incision and another during bypass. Failure to give the antibiotic before skin incision results in a doubling of the major wound infection rates in cardiac surgical patients. Perhaps the ideal time to give this antibiotic is just after establishing invasive blood pressure monitoring.

No anesthetic has been proved best for patients with heart disease. Indeed, provided the appropriate hemodynamic goals and overall clinical requirements are attained, the drugs used are unimportant. Achieving those goals may be easier with some drugs than with others, depending on the pharmacodynamics of the drugs and the experience of the anesthesiologist with those drugs. Fentanyl, midazolam, and etomidate (induction only) are used in our cardiac operating rooms, but so are thiopental and inhaled anesthetics, including nitrous oxide. Nitrous oxide is not used after bypass, because of concern about expanding residual air in the systemic circulation. Isoflurane, desflurane, or sevoflurane is administered throughout surgery in most patients (including during bypass) at low doses (about 0.5–1.0 minimum alveolar anesthetic concentration [MAC]) to help ensure unconsciousness. Higher doses of the inhaled drug are used sometimes to control hypertension during bypass. Midazolam is our premedication of choice and serves as an intraoperative drug as well. After bypass, midazolam administration is limited in patients for whom early extubation is anticipated. Propofol serves as the sedative of choice during transport to the intensive care unit and in the intensive care unit until extubation.

Most centers require early extubation after routine heart surgery. Therefore, the traditional cardiac anesthetic that uses high doses of opioids is inappropriate. However, hemodynamic stability during and for a few hours after cardiac surgery remains a very desirable goal that is difficult to attain without narcotics. One easy approach to this dilemma is to administer the bulk of the opioid (in my practice, fentanyl) during the induction and skin closure, and rely on inhaled anesthetics otherwise. In fact, I rarely turn the inhaled anesthetic <0.5 MAC to minimize the chances of awareness.

Cardiopulmonary Bypass

When a major disaster occurs during bypass, I've noted that at least one of four errors has usually taken place. One, the anesthesiologist has mismanaged anticoagulation and the oxygenator has clotted. Two, the surgeon has mismanaged cannulation and vital organs are not perfused. Three, the perfusionist has mismanaged the blood reservoir and pumped air into the systemic circulation. And four, the surgeon, anesthesiologist, perfusionists, or all three have miscommunicated some vital information, resulting in one of the three problems above. Be aware of these four great mistakes because when they occur, the patient is seriously injured or killed.

The usual dose of heparin is 3–4 mg/kg or 300–400 U/kg. The usual concentration of heparin is 10 mg/mL or 1000 U/mL. Therefore, most patients required 20–40 mL of heparin. Make sure you have verified the identity of the heparin vial and concentration. Give the heparin through a central line whenever possible with draw-back of blood to confirm its delivery. Whenever possible, the adequacy of its effect should be confirmed before starting bypass (activated clotting time [ACT] $>$ 450 s). During bypass, the ACT should be $>$500 s. The ACT should be measured with kaolin-containing, not celite-containing, test vials, because the latter are not accurate in the presence of aprotinin. Because hypothermia decreases heparin degradation, ACT can be monitored less frequently during hypothermia (every 30–60 min) than during normothermia (every 15 min). Whenever there is any doubt about the adequacy of heparinization, give more heparin immediately.

Normally, patients become acutely hypotensive at the onset of bypass because of a marked decrease in blood viscosity secondary to acute hemodilution. Within a few minutes, blood pressure recovers because of the marked release of catecholamines caused by bypass. Although some controversy remains, most centers titrate mean arterial blood pressure during bypass to 50–70 mm Hg. Patients with a history of hypertension or carotid disease are usually managed in the higher end of that range. Phenylephrine is used to raise pressure and inhaled anesthetics (via the oxygenate), or nitroprusside is used to lower it.

With the help of the perfusionists, the anesthesiologist is responsible for ensuring that the patient is adequately oxygenated, perfused, and anesthetized during bypass. I use the same variables to make these assessments during bypass as I do at other times during anesthesia, with a few special additions. When hypothermia is used, a rapid decrease in nasopharyngeal temperature suggests adequate cerebral perfusion. I view the bypass lines as they enter and leave the pump to see that the arterial line is always bright red and that the venous line is appropriately dark (less so during hypothermia than normothermia).

When it is time to terminate bypass (bleeding is controlled and rectal temperature is $>36°$), resume ventilation. Thereafter, the venous outflow is impeded until the intravascular volume of the patient is restored, and then the arterial inflow is terminated. This can be done quickly ("fill him up, and let's come off") or gradually. The adequacy of filling is judged by filling pressures, TEE, and direct inspection of the heart. If blood pressure is inadequate despite adequate filling, the aorta is palpated to determine whether the hypotension is central as well as peripheral. Often, immediately after bypass (and sometimes during), blood pressure at peripheral sites such as the radial

Table 1. Laboratory Results and Treatments

Variable	ACT	PT	PTT	Platelets	Fibrinogen	Treatment
"Factor 6"	normal	normal	normal	normal	normal	suture
Platelet dysfunction	normal	normal	normal	normal	normal	DDAVP[a] platelets
Dilutional	increased	increased	increased	decreased	decreased	platelets FFP
Primary fibrinolysis	increased	increased	increased	decreased	markedly decreased	cryoprecipitate
DIC	increased	increased	increased	decreased	decreased	platelets FFP
Heparin	increased	increased	increased	normal	normal	protamine

ACT = activated clotting time; PT = prothrombin time; PTT = partial thromboplastin time; FFP = fresh frozen plasma; DIC = disseminated intravascular coagulation.
[a] DDAVP = desmopressin, which may be effective if platelet dysfunction is caused by aspirin therapy or renal failure.

artery is substantially lower than in the aorta. When this occurs after bypass, cuff determinations from the upper arm may be used, or a femoral catheter may be placed. Usually, this problem resolves before chest closure. When the hypotension is central as well as peripheral, then it must be corrected appropriately. No one therapy has proved best, but, in general, I recommend that you focus on correcting the marked abnormalities. For instance, be sure that acid/base status is corrected, hematocrit is reasonable (>25%), preload is adequate, and heart rate and rhythm are acceptable. Thereafter, augment contractility if it is low and normalize systematic vascular resistance. When myocardial ischemia is the problem, consult the surgeon to see if additional grafts might be needed, then begin nitroglycerin or diltiazem (if you think coronary spasm might be the problem). If vasodilators fail, again consult the surgeon for placement of an intraaortic balloon pump. Continue to correct acid/base and maintain anticoagulation status (if you are on partial bypass or using the bypass "pump suckers"). If you are off bypass and the patient is barely surviving, keep plenty of heparin available in case you have to give it to emergently "crash" back on bypass. When hemodynamics are acceptable, administer protamine slowly to reverse the effects of heparin.

Coagulopathies After Bypass

To a certain degree, bleeding after bypass is normal. However, excessive bleeding is not uncommon and usually results from 1) inadequate surgical hemostasis (we call this a "factor 6" deficiency, because it is resolved with 6-0 suture), 2) platelet dysfunction, 3) dilutional coagulopathy, 4) primary fibrinolysis, 5) disseminated intravascular coagulation, 6) residual heparin, or 7) other much less common problems. Table 1 summarizes the usual laboratory results and appropriate treatments.

Transport to the Intensive Care Unit

Most patients will be transported to the intensive care unit while they remain anesthetized and intubated. I use a propofol infusion to maintain anesthesia during this transfer, and I continuously monitor arterial pressure, electrocardiogram, and saturation. Transport is a precarious time for the patient. Be careful not to let down your level of vigilance. Make sure to have with you adequate fluid (at least a liter of something hanging), drugs to raise and lower blood pressure, and a face mask to ventilate the patient in case the endotracheal tube is dislodged. If the intensive care unit is any appreciable distance from the operating room, insist that at least two members of the team accompany you and the patient in the transfer. If cardiac arrest occurs during transport, one of them provides chest compression, and the other pushes the transport bed back to the operating room or intensive care unit (whichever is closer) while you administer drugs and ventilate. If all goes well in transport, the first thing to do on arrival is to make sure that the chest tubes are connected to suction, and then continue with an orderly transfer of monitoring cables and finally a summary of events.

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