# Anesthesia for Cardiac Valvular Surgery

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**Introduction**

Approximately 71,000 patients undergo cardiac valvular surgery each year in the United States. The most common problem presenting for valvular surgery is calcific aortic stenosis in the elderly, and the incidence of this problem is increasing for reasons that are not fully understood.[1] In previous years, cardiologists delayed referral for valvular surgery as long as possible believing that the inherent risks of surgery justified its use only as a last therapeutic resort. Although the ideal timing of valvular surgery remains controversial, advancements in surgical and anesthetic techniques, especially mitral valve repair, have lead most authorities to recommend valvular surgery before the onset of ventricular dilation and, in some cases, even before the onset of symptoms.[2]

In this review lecture, I will summarize the principals of perioperative anesthetic management for patients requiring aortic and mitral valve surgery. In addition, I will briefly cover two controversial issues in the field: minimally invasive techniques and the Ross procedure.

**Aortic Stenosis**

Symptoms (angina, syncope, and congestive heart failure) of aortic stenosis (AS) begin late in the course of the disease when the gradient across the valve exceeds 50 mm Hg and the valve area has narrowed to less than 1 $cm^2$ (normal 2-3 $cm^2$). Doppler echocardiography accurately estimates the gradient across the valve and its cross sectional area, and therefore is the diagnostic test of choice. Cardiac catheterization is required when coronary artery disease is strongly suspected (patients > 55 years with one or more risk factors).[3] The aortic valve gradient is underestimated by 5-20 mm Hg by catheterization, because at catheterization the left ventricular and aortic pressures are not measured simultaneously.

When moderate or severe (<0.7 $cm^2$) AS is present, the left ventricle is invariably hypertrophied and noncompliant. Therefore, coordinated atrial and ventricular contraction (normal sinus rhythm) and a high filling pressure are needed to adequately distend the left ventricle during diastole. Similarly, bradycardia is very poorly tolerated because the stiff left ventricle cannot increase stroke volume to compensate for the reduced heart rate. System vascular resistance and left ventricular work are uncoupled, that is, vasodilatation does not reduce the work of the heart with severe AS, because of the fixed resistance at the aortic valve. However, vasodilatation does reduce coronary perfusion pressure and will likely provoke myocardial ischemia. Modest decreases in myocardial contractility are generally well tolerated in patients with AS, even when there is a history of congestive heart failure. In contrast, augmentation of contractility or tachycardia may induce myocardial ischemia in patients with AS even in the absence of coronary artery disease. Thus, regardless of the specific anesthetic agents used, the following are the hemodynamic goals during anesthetic management for patients with:

**Aortic Stenosis**

| HR | Rhythm | Preload | SVR | Inotropy |
|---|---|---|---|---|
| 70-85 | Sinus | ↑ | No △ or ↑ | No △ or ↓ |

**Aortic Regurgitation**

Aortic regurgitation (AR) is usually caused by congenital defect of the valve, dissection of the annulus, or bacterial endocarditis. Acute AR causes a sudden increase in filling pressures and pulmonary edema. Chronic AR causes a progressive dilation of the left ventricle which is initially well tolerated, but eventually symptoms of congestive heart failure ensue. Patients with chronic AR have a characteristic diastolic murmur, an enlarged heart, and a widened pulse pressure. If the pulse pressure isn't at least 50% of the systolic blood pressure, or the diastolic pressure is greater than 70 mm Hg, significant AR is unlikely. Doppler echocardiography is the diagnostic method of choice.

When moderate or severe AR is present (3+ or 4+, that is, roughly greater than 6 L/min of regurgitation), the primary compensatory mechanism for the regurgitant flow is an increase in stroke volume. This mechanism can only be maintained if preload is adequate and systemic vascular resistance is normal or decreased. Because AR occurs during diastole, its severity increases as the heart rate slows. Thus, bradycardia is to be avoided. Modest decreases in myocardial contractility are generally well tolerated in patients with AR, but provide no special advantage. Vasodilators are beneficial in some patients with AR, but because the aortic diastolic pressure (coronary driving pressure) is already low (typically 30-50 mm Hg), they cannot be administered without the risk of inducing myocardial ischemia. Therefore, the following are the hemodynamic goals during anesthetic management for patients with:

**Aortic Regurgitation**

| HR | Rhythm | Preload | SVR | Inotropy |
|---|---|---|---|---|
| 85-100 | Sinus | No △ or ↑ | No △ or ↓ | No △ |

**Mitral Stenosis**

Mitral stenosis (MS) is usually caused by rheumatic heart disease. Moderate (1-1.5 $cm^2$ valve area) or severe (<1 $cm^2$) MS is invariably associated with symptoms of congestive heart failure. Left atrial pressure is chronically elevated, accounting for the signs and symptoms of pulmonary congestion and pulmonary hypertension. Doppler echocardiography will document the presence and severity of MS and is the usual diagnostic test of choice. Often patients with MS have chronic atrial fibrillation (due to marked left atrial enlargement) and are receiving digoxin to control their heart rate. Cardioversion is not likely to restore sinus rhythm and may be dangerous, because left atrial thrombus (commonly present in MS) may be dislodged into the systemic circulation.

In patients with MS, the gradient across the mitral valve is critically dependent on heart rate. For instance, doubling the heart rate from 60 to 120 decreases the duration of diastole by more than three fold. Consequently, the gradient across the valve (and left atrial pressure) must more than triple to maintain the same trans-mitral flow. Even with normal heart rates, patients with MS need a high filling pressure to provide adequate left ventricular preload. Thus, with the onset of tachycardia, very high left atrial pressures and pulmonary edema are to be expected. Vasodilators usually decrease systemic more than pulmonary blood pressure, and as a result, may induce right ventricular ischemia. In patients with severe MS and pulmonary hypertension, high systemic vascular resistance may be needed to provide the aortic diastolic pressure necessary for adequate coronary perfusion of the right ventricle. Modest decreases in contractility are usually well tolerated, but factors which aggravate pulmonary hypertension (hypoxia, hypercarbia, acidosis, and inadequate anesthesia) should be avoided, or the right ventricle can acutely fail. Therefore, the following are the hemodynamic goals during anesthetic management for patients with:

**Mitral Stenosis**

| HR | Rhythm | Preload | SVR | Inotropy |
|---|---|---|---|---|
| 65-80 | Stable | No △ or ↑ | No △or ↑ | No △ |

**Mitral Regurgitation**

Usually, acute mitral regurgitation (MR) is caused by ischemic damage to the papillary muscles, rupture of their chordae, or bacterial infection of the leaflets. Usually, chronic MR is caused by progressive mitral prolapse or rheumatic heart disease. In either case, left atrial pressure is elevated, resulting in pulmonary hypertension and accounting for the usual symptoms of congestive heart failure. On examination, a systolic murmur radiating into the axilla can be heard, but Doppler echocardiography or angiography is needed to ascertain the severity of MR. Moderate or severe MR is present when 50% or more of the left ventricular ejection is regurgitated into the left atrium. However, in acute MR, a smaller regurgitant fraction can cause severe pulmonary congestion because sufficient left atrial enlargement/compliance hasn't had time to develop. Often patients with MR have chronic atrial fibrillation (due to marked left atrial enlargement) and are receiving digoxin to control their heart rate.

Patients with moderate or severe MR do not tolerate a large increase in systemic vascular resistance, because this change acutely increases the regurgitant fraction. The cornerstone in the care of these patients is to reduce systemic vascular resistance. In addition, faster than normal heart rates (except in the presence of ischemic heart disease) are sometimes beneficial because they reduce left ventricular filling and thereby mitral annular distension. Patients with MR tolerate modest decreases in contractility, and if ischemic heart disease is present, modest myocardial depression may be beneficial. Over concern about the myocardial depressant effects of the inhaled anesthetics should not prevent their judicious use in patients with valvular heart disease. This is especially true for patients with MR, in whom inadequate anesthesia during surgical stimulation may allow a disastrous increase in systemic vascular. Therefore, the following are the hemodynamic goals during anesthetic management for patients with:

**Mitral Regurgitation**

| HR | Rhythm | Preload | SVR | Inotropy |
|---|---|---|---|---|
| 80-95 | Stable | No △ | ↓ | No △ or↓ |

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**Monitoring**

Direct arterial and central venous pressure determinations are standards of care in most cardiac surgical procedures, while pulmonary artery pressure monitoring and transesophageal echocardiography (TEE) are used more selectively. Currently, few surgical teams would perform valvular repair without the ability to confirm its success with TEE before decannulating the heart. In a study of 154 patients undergoing valve surgery, intraoperative TEE documented unsatisfactory repairs in 10 patients (6%) requiring immediate further surgery.[4] Although six of these 10 patients had abnormal V waves or elevated pulmonary capillary wedge pressures, hemodynamics were normal in the other four patients and only TEE indicated defective repair. This study and subsequent studies confirm that intraoperative TEE assessment of mitral valve function is predictive of postoperative function and patient outcome.[5,6] When successful mitral valve repair is accomplished, patients require no postoperative anticoagulation (unless atrial fibrillation is present), have better ventricular function, fewer perioperative complications and longer life expectancy than when replacement is performed.[7] When mitral valve replacement is necessary, TEE reliably detects prosthetic malfunction (rare) and periprosthetic leaks (surprisingly common).[8] While moderate or severe periprosthetic leaks should almost always undergo immediate repair, the treatment of smaller periprosthetic leaks is controversial.[9] Thus, TEE is viewed by some authorities as clearly indicated during valve repair and potentially useful during valve replacement.[10]

The role of pulmonary artery pressure monitoring is less clear. The literature provides no studies indicating improvement in patient outcome resulting from the use of pulmonary artery catheters. Thus, the individual anesthesiologist must decide whether in his/her hands the inherent risks of this technique are likely to be offset by the information gained. In my patients undergoing valvular surgery, I use 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 (where TEE is not as readily available as it is in the operating room). In contrast, our “fast track” cardiac patients almost never receive a pulmonary artery catheter.

**Anesthetic Agents and Adjuvants**

No anesthetic agent has been proven best for patients with valvular disease. Indeed, provided the hemodynamic goals outlined above are attained, the agents used are unimportant. Achieving those goals may be easier with some agents than with others depending on the pharmacodynamics of the agents and the experience of the anesthesiologist with those agents. Fentanyl and etomidate (induction only) are used frequently in our cardiac operating rooms, but so are inhaled agents including nitrous oxide. Nitrous is not used during or after bypass, because of concern about expanding residual air in the systemic circulation. Either isoflurane, desflurane, or sevoflurane is administered throughout surgery in most patients (including during bypass) at low doses (about 1.0-0.5 MAC) to help insure unconsciousness. Remifentanil is used in some of our patients in whom early extubation is anticipated and neuroaxial analgesia is provided (see below). Midazolam is our premedication of choice and serves as an intraoperative agent as well. After bypass, midazolam administration is limited in patients for whom early extubation is anticipated. In these patients, propofol serves as the sedative of choice after bypass, during transport to the intensive care unit, and in the intensive care unit until extubation. Thus, in the absence of proven superiority of one anesthetic agent over the others, we use a variety of standard agents to achieve the required hemodynamic and overall goals for our patients.

Antifibrinolytics reduce blood loss and transfusions in patients undergoing valvular surgery.[11-18] Aprotinin is more expensive and may be more effective than aminocaproic acid or transexamic acid, but this latter point is under debate. Because of its expense, we reserve aprotinin for patients who are at high risk for excessive bleeding, for example, patients undergoing cardiac reoperations. Aminocaproic acid is the least expensive agent among the antifibrinolytics and is our choice for all other patients requiring valvular surgery (5 grams over 20 minutes prior to bypass and 1 gram per hour thereafter until skin closure). When excessive bleeding persists after bypass despite correction of the activated clotting time, desmopressin may be beneficial especially if the patient has renal insufficiency or a history of recent aspirin ingestion.[19]

**Postoperative Analgesia**

In order to reduce postoperative pain and increase early mobilization of our patients, we administer intrathecally 7-10 µg/kg of preservative-free morphine to most patients undergoing valvular surgery provided they have no contraindication to subarachnoid puncture and are expected to be extubated within eight hours after surgery. The morphine is administered immediately prior to induction of anesthesia. The literature indicates this techniques provides excellent pain relief, but may prolong extubation time.[20] We are completing a randomized study to determine whether the use of intraoperative remifentanil instead of fentanyl will facilitate early extubation in patients receiving intrathecal morphine.

**Controversies**

Minimally invasive cardiac surgery is an issue of major concern for some heart surgery patients. Commercially

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marketed systems are available to assist the cardiac surgeon gain entrance into the chest through smaller incisions than the traditional full sternotomy. Valvular repair can be done successfully with these techniques,[21,22] and some evidence indicates that a minimally invasive approach decreases postoperative pain when compared with traditional techniques.[23] However, not all authorities believe that minimally invasive approaches will prove to be as safe or cost effective as the traditional approach.[24] Usually, minimally invasive techniques take longer, require more equipment and skill, and are targeted for lower risk patients than traditional techniques. Catheter based techniques have had an especially rocky course once they left the hands of their developers for wide spread use and investment.[25] One important point seems clear, when valvular surgery is performed with limited surgical exposure, the view of the heart provided by TEE becomes more important, indeed, some would argue essential.[26]

Sir Donald Ross is credited with developing the Ross procedure in which a diseased aortic valve is replaced with the patient's own pulmonary valve (autograft) and the pulmonary valve with a cryopreserved homograft.[27] Although not new, the Ross procedure has only newly become a widely acceptable alternative to conventional aortic valve replacement, because only recently has its associated complication rate been reduced sufficiently in some experienced centers. The primary advantages of the procedure are that the patient does not require postoperative anticoagulation, the aortic autograft may last indefinitely, and in children, the aortic autograft may grow with the patient. The disadvantages are the technical difficulty of the procedure and the eventual need to replace the pulmonary homograft. The later disadvantage is only relative, because a pulmonary homograft is easier to replace than an aortic homograft or aortic tissue prosthesis - the alternatives to the Ross procedure which free the patient from the need for chronic anticoagulation. Most authorities reserve the Ross procedure for infants or children with aortic stenosis or in adults who want to avoid anticoagulation and aortic valve reoperation. In my experience, this procedure has a significant surgical learning curve. Too few patients have received the Ross procedure to allow a final determination of who should receive it and who should perform it.

**Summary**

Recent improvements in surgical and anesthetic techniques, especially mitral valve repair, have resulted in earlier referral of patients for valvular surgery, shorter hospital stays, and better outcomes than in prior years. TEE has become a key tool for the immediate assessment of surgical results so that flawed operations can be corrected before the patient is separated from cardiopulmonary bypass. No one anesthetic agent or technique has proven best for patients requiring valvular surgery, in fact, almost any anesthetic agent can be used successfully provided the anesthesiologist achieves the hemodynamic goals appropriate for each patient's valvular pathology. Antifibrinolytic therapy is effective and safe for limiting blood loss and transfusion. Clinical innovations, such as minimally invasive techniques, neuroaxial analgesia, and valve repair options present special challenges for the surgeon and anesthesiologist, but have the potential to reduce patient suffering, shorten the length of hospitalization, and enhance the quality of life.

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