Case Scenario: Cesarean Section Complicated by Rheumatic Mitral Stenosis

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CARDIAC disease in pregnancy remains an important etiology of maternal and fetal morbidity and mortality. Estimates of the incidence of cardiac disease in pregnant patients in developed countries range from 0.2 to 3%. Although the incidence of rheumatic heart disease (RHD) has decreased in developed countries, it still accounts for most of the cardiac disease–related maternal mortality in developing countries—as well as in immigrants to the United States from these nations.

Mitral stenosis is the most commonly acquired valve lesion encountered in pregnant women and is almost invariably caused by RHD. Pregnancy and the peripartum period represent a physiologic burden that may worsen symptoms in even moderate degrees of cardiac disease. Consequently, many women are first diagnosed with cardiac disease during pregnancy. The need to provide labor analgesia or anesthesia for a Cesarean section to a woman with cardiac disease is not infrequent and can be challenging. In this case scenario, we discuss the peripartum management of a patient with severe mitral valve disease.

Case Report

A 37-yr-old woman (gravida, 2; para, 1) with a history of rheumatic mitral stenosis presented at 28 weeks gestational age with complete placenta previa, possible placenta accreta, and worsening dyspnea. Although the patient complained of dyspnea with moderate exertion before pregnancy (class II symptoms, New York Heart Association Functional Classification system; table 1), she was now unable to lie flat and even became dyspneic when speaking (class IV). Her symptoms improved with heart rate control using 25 mg metoprolol orally twice daily. Physical examination revealed an arterial blood pressure of 101/59 mmHg, a regular heart rate of 107 beats/min, and a respiratory rate of 22 breaths/min. There were diastolic and holosystolic apical murmurs, 2:6 and 3:6, respectively. The patient’s lungs were clear to auscultation bilaterally, and she had mild pedal edema. Electrocardiogram showed sinus tachycardia and left atrial enlargement. Preoperative transthoracic echocardiogram revealed severe left ventricular systolic dysfunction and pulmonary hypertension with estimated pulmonary artery systolic pressure of 54 mmHg. There was moderate tricuspid regurgitation. Left and right ventricular systolic function were normal.

The patient was admitted to the hospital for observation and management. Multidisciplinary meetings were held to discuss her management and included cardiology, high-risk obstetrics, and cardiac surgery, as well as cardiac and obstetric anesthesiology. Multiple studies have shown that vaginal delivery is well tolerated in most patients with valvular heart disease. Cesarean section is usually performed for obstetrical indications only. Because our patient had placenta previa, an elective repeat Cesarean section was planned at 36 weeks gestational age in a cardiac operating room with cardiac surgery and cardiopulmonary bypass capabilities on standby.

As prophylaxis against acid aspiration, 30 ml sodium bicarbonate was administered orally in the holding area. While American Society of Anesthesiologists standard monitors were placed, the patient was positioned in the left uterine displacement position. Fetal heart rate monitoring was performed by one of the obstetricians from the time of entry into the operating room until surgical site preparation. A left radial arterial catheter was placed. Because of the patient’s pre-existing pulmonary hypertension, the pulmonary artery was catheterized via the right internal jugular vein. The patient’s initial systemic arterial blood pressure was 125/65 (mean 76) mmHg. Her heart rate was 105 beats/min. Initial pulmonary
pressures were 90/50 (mean 62) mmHg. Central venous pressure was 14 mmHg with a prominent V wave. Although thermodilution cardiac output was not measured because of tricuspid regurgitation, initial calculated mixed venous oxygen saturation was 34%. Remifentanil (0.2 µg · kg⁻¹ · min⁻¹) was started to attenuate the sympathetic response to laryngoscopy and intubation. General anesthesia was induced with etomidate, and succinylcholine was used for paralysis, which was administered in a rapid sequence fashion. Maintenance of anesthesia consisted of a remifentanil infusion (0.05–0.1 µg · kg⁻¹ · min⁻¹), a low level of isoflurane (less than 0.5 minimum alveolar concentration), and vecuronium for muscle paralysis. Higher doses of inhalational agents were avoided to prevent uterine atony. Depth of anesthesia was monitored using bispectral index (Aspect Medical Systems, Norwood, MA) and values from 40 – 60 were maintained during the intraoperative period. The patient was ventilated using 100% oxygen. To optimize pulmonary vascular resistance, vigilant attention was paid to maintaining normocarbia.

Additional monitoring consisted of transesophageal echocardiogram. In the midesophageal four-chamber view, characteristic features of RHD are seen (fig. 1A). Mitral valve leaflets were thickened and calcified with decreased mobility. Some calcification of the mitral valve annulus was seen as well. The addition of color flow doppler showed turbulence of antegrade flow through the stenotic valve in addition to severe regurgitation (fig. 1B). The midesophageal mitral commissural view demonstrated commissural fusion (fig. 2). Transmitral blood flow velocity using continuous-wave Doppler is presented in figure 3. The mean gradient across the mitral valve was 16 mmHg. Pressure half-time measurement was 154 ms, which corresponded to an estimated mitral valve area of 1.4 cm². Three-dimensional ultrasound reconstruction of the mitral valve, viewed from the atrial side (“surgeon’s perspective”), is presented in figure 4. The mitral valve is severely retracted with significant commissural fusion.

Cesarean section was performed without complications. Apgar scores of the neonate were 4 at 1 min and 9 at 5 min. The baby required assisted ventilation for approximately 1 min. After delivery, 40 U oxytocin was administered intravenously in 2 h. No significant changes in arterial or pulmo-
nary pressures, or in transesophageal echocardiographic examination, occurred during the procedure.

At the conclusion of the procedure, the patient was extubated. She was initially monitored in the operating room after extubation to ensure normocarbia and stable pulmonary artery pressures. She was then transferred to the cardiac care unit for postoperative monitoring. Mixed venous oxygen saturation improved to 54% within 4 h after Cesarean section. The patient was discharged from the cardiac care unit on postoperative day 1 and from the hospital on postoperative day 5. She underwent mitral valve repair and tricuspid valve annuloplasty 4 months later.

![Fig. 3](image1.png)

Fig. 3. (A) Transmitral blood flow velocity using continuous-wave Doppler. Mean gradient across the patient’s mitral valve was 16 mmHg, which was calculated by integrating the velocity versus time spectrum. Pressure half-time (P1⁄2t) measurement, 154 ms, was used to calculate the mitral valve area (MVA) as 1.4 cm². (B) Alternatively, normal transmitral spectrum consists of an early diastolic phase (E wave) and a late diastolic component associated with atrial contraction (A wave). E wave velocity is usually larger than A wave velocity. LA = left atrium; LV = left ventricle; MV = mitral valve; PG = peak gradient; Vmax = maximum velocity; Vmean = mean velocity; VTI = velocity time integral.

![Fig. 4](image2.png)

Fig. 4. Three-dimensional ultrasound reconstruction of the mitral valve, viewed from the atrial side (“surgeon’s perspective”). The anterior (AL) and posterior (PL) mitral valve leaflets are visible with the anterolateral (left) and posteromedial (right) commissures. The mitral valve is severely retracted with significant commissural fusion. AV = aortic valve; LA = left atrium; TV = tricuspid valve.

### Discussion

Normal pregnancy results in dramatic changes to the cardiovascular system (table 2). Pregnancy produces a 30–50% increase in blood volume and cardiac output with physiologic anemia as a result of a greater increase in blood volume than red cell mass.6–8 The increase in cardiac output is primarily the result of an increase in stroke volume with a smaller contribution from an increase in heart rate. Pregnancy reduces systemic vascular impedance. Anemia decreases blood viscosity with resultant decrease in systemic vascular resistance.

At the time of labor and delivery, pain and anxiety increase catecholamine release with resultant increases in heart rate, arterial blood pressure, and cardiac output.6–8 Autotransfusion of up to 500 ml with each contraction increases preload and, hence, cardiac output.3 After delivery, there is an additional increase in venous return as a result of autotransfusion from the contracting uterus as well as from the loss of fetal compression of the inferior vena cava.9

### Table 2. Cardiovascular Changes in Pregnancy at Early Third Trimester4,32

<table>
<thead>
<tr>
<th>Variable</th>
<th>Peak Change, %</th>
</tr>
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<tbody>
<tr>
<td>Blood volume</td>
<td>+35</td>
</tr>
<tr>
<td>Plasma volume</td>
<td>+45</td>
</tr>
<tr>
<td>Heart rate</td>
<td>+20</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>+50</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>+40</td>
</tr>
<tr>
<td>Contractility</td>
<td>Variable</td>
</tr>
<tr>
<td>Central venous pressure</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Systemic vascular resistance</td>
<td>−15</td>
</tr>
<tr>
<td>Systemic blood pressure</td>
<td>−5</td>
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</tbody>
</table>
Rheumatic Mitral Stenosis

Acute rheumatic fever is an immune-mediated, multisystem inflammatory disease that is a sequela of group A streptococcal infection. The pathogenesis of acute rheumatic fever is believed to involve the triad of a genetically susceptible individual, infection with a rheumatogenic strain of group A streptococcus, and an aberrant host immune response. Rheumatic fever is characterized by carditis, valvulitis, arthritis, chorea, erythema marginatum, and subcutaneous nodules. Although the long-term effects of acute rheumatic fever on most tissues are minimal, its effects on the heart may be devastating. Inflammation leads to neovascularization, which allows further recruitment of T cells, leading to granulomatous inflammation and the establishment of chronic RHD. Carditis occurs in 30–80% of patients with acute rheumatic fever, and at least 60% of untreated patients develop chronic RHD.

Due to its association with RHD, mitral stenosis is highly prevalent in developing countries. In India, the prevalence of RHD is 6:1,000 among school-aged children, which is in contrast to that seen in the United States, 0.05:1,000. In Asia proper, the prevalence of RHD has been reported as being as high as 12:1,000. If one extrapolates these data, there are currently 1.9–2.2 million cases of RHD in children aged 5–14 yr in Asia proper, with a total of 11–16 million cases. Others have estimated approximately 15.6 million cases of RHD worldwide, with 80% of these cases in developing nations. More than 200,000 new cases of RHD and 180,000 RHD-related deaths occur each year in the developing world.

In India, the mean ± SD age of presentation is 15.1 ± 4.4 yr old with approximately two-thirds of patients presenting with mitral stenosis. Although mitral stenosis frequently follows rheumatic fever, fewer than half of patients with RHD remember contracting rheumatic fever. The persistent inflammatory and hemodynamic valvular injury from rheumatic fever contributes to the progression of the disease. The valve area usually narrows 0.1–0.3 cm² per year; however, the rate of progression may be accelerated with repeated episodes of rheumatic fever. In a retrospective cross-sectional study of 714 patients presenting for valvular surgery for RHD in Africa, 31% had pure mitral regurgitation, 38% had pure mitral stenosis, and 31% had mixed presentation. Ongoing rheumatic activity was diagnosed in 47% of patients with pure mitral regurgitation and in 2% of patients with pure stenosis. Although regurgitation was the most common lesion in the first and second decades, the prevalence of stenosis increased with age.

Mitral Stenosis in Pregnancy

In general, mitral regurgitation is well tolerated by pregnant patients because the reduction in systemic vascular resistance reduces regurgitant flow. Mitral stenosis, however, is generally less well tolerated. In the normal adult, the mitral valve has an area of 4–6 cm². Among patients who do not have mitral valvular stenosis, there are no clinically significant increases in transmural valvular gradient due to pregnancy-induced increases in cardiac output. With worsening mitral stenosis, however, increases in blood volume and cardiac output associated with pregnancy are less well tolerated. When mitral valve area is reduced to less than 2.0 cm², a pressure gradient develops across the mitral valve. The magnitude of this gradient depends on stenosis severity and the amount of blood flow across the valve. Thus, as cardiac output increases during pregnancy, the gradient across the diseased mitral valve increases. This increase in left atrial pressure is reflected back into the pulmonary venous circulation and increases the risk of pulmonary edema. Untreated, this progression results in pulmonary arterial hypertension that may lead to increases in right ventricular pressures and, possibly, to right ventricular failure.

The etiology of the pulmonary hypertension is usually passive and hence reversible after intervention; however, endothelial changes and vascular remodeling may affect its course. Pulmonary hypertension is associated with extremely high maternal and fetal mortality.

A mitral valve area greater than 1.5 cm² usually does not cause symptoms at rest. However, as the severity of stenosis increases, patients develop decreased exercise tolerance, orthopnea, cough, paroxysmal nocturnal dyspnea, or pulmonary edema. Furthermore, atrial arrhythmia associated with ventricular rate acceleration is a common cause of worsening symptoms. Although the clinical presentation of rheumatic mitral stenosis is usually associated with symptoms of congestive heart failure, patients may also present with hemoptysis and chest pain from pulmonary hypertension.

Overall maternal mortality associated with mitral stenosis has been reported at 1%, but this number increases to 7% with worsening symptoms. Desai et al. found the most common complication to be pulmonary edema. The most significant risk factors for the prediction of maternal pulmonary edema were the severity of mitral stenosis, late antenatal presentation, moderate to severe symptoms outside of pregnancy, and cardiac disease diagnosed for the first time in the index pregnancy. Silversides et al. reported that pulmonary edema and arrhythmia were the most common maternal complications; prematurity and intrauterine growth retardation were the most common fetal complications. Both maternal and fetal complications are directly related to stenosis severity and prepregnancy New York Heart Association Functional Classification.

Echocardiographic Evaluation. The most common etiology of mitral stenosis is RHD. Other rare (less than 1%) causes are congenital valvular stenosis, vegetations and calcifications of the leaflets, parachute mitral valve, and annular calcification. Generally, mitral stenosis is characterized by restricted leaflet movement, a reduced orifice, and diastolic doming. In RHD, calcification of the valvular and subvalvular apparatus, as well as thickening, deformation, and fusion of the valvular leaflets at the commissures produce a characteristic
fish mouth–shaped orifice. Chordal fusion and shortening may result in significant subvalvular stenosis. Other characteristics that may be associated with chronic mitral stenosis include an enlarged left atrium, spontaneous echo contrast or smoke (related to low velocity blood flow with subsequent rouleaux formation by red blood cells), thrombus formation, and right ventricular dilatation.

Because planimetry of the mitral valve orifice is not influenced by assumptions of flow conditions, ventricular compliance, or associated valvular lesions, it is used as the reference standard for the evaluation of mitral valve area in mitral stenosis.26 This orifice opening is best visualized in the trans-gastric basal short axis view and is best measured mid-diastole; however, three-dimensional reconstruction may improve accuracy. Severe calcification of the mitral valve may interfere with mitral valve area determination and, in patients with significant subvalvular stenosis, underestimation of the degree of hemodynamic compromise may occur when determining mitral valve area by planimetry.26

A transmitral Doppler spectrum is measured along the axis of transmittral blood flow, most often obtained in a midesophageal four- or two-chamber view.26 Transmitral valve flow is characterized by two peaked waves of flow away from the transducer (fig. 3). The first wave represents early diastolic filling (E wave) and the second wave represents atrial systole (A wave). The transvalvular gradient may be estimated using the modified Bernoulli equation: pressure gradient = 4 × velocity². Because peak gradient is heavily influenced by left atrial compliance and ventricular diastolic function, the mean gradient is the relevant clinical measurement.26 The values obtained through this method have high correlation with those obtained using a transseptal puncture during cardiac catheterization.27

Normally, with mitral valve opening during early diastole, there is a torrential increase in transmitial flow, which rapidly decreases to zero during diastasis, when the left atrial and ventricular pressures equilibrate. With mitral stenosis, a gradient between the left atrium and ventricle may be maintained for a longer amount of time (fig. 3). This sustained pressure differential maintains flow between the left atrium and ventricle, decreasing the slope of this early transmitral flow. The rate of decline in E-wave velocity may be described by its pressure half time, which is the time interval from the peak E-wave velocity to the time when the E-wave velocity has declined to half of its corresponding peak pressure value. The pressure half time is inversely proportional to the mitral valve area,26 mitral valve area = 220/pressure half time.

The advantage of this technique is that it is independent of valvular geometry. The formula assumes that the mitral valve is at least mildly stenotic. The presence of either mitral regurgitation or aortic regurgitation will decrease the accuracy of pressure half-time measurements for the determination of mitral stenosis.26 Echocardiographic assessment of mitral stenosis is summarized in table 3.

### Table 3. Quantification of Mitral Stenosis

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve area (cm²)</td>
<td>&gt;1.5</td>
<td>1.0–1.5</td>
<td>&lt;1.0</td>
</tr>
<tr>
<td>Mean gradient (mmHg)</td>
<td>≤5</td>
<td>5–10</td>
<td>&gt;10</td>
</tr>
</tbody>
</table>


**Peripartum Management.** For women with a diagnosis of moderate to severe mitral stenosis, class II–IV on the New York Heart Association Functional Classification system, or severe pulmonary hypertension, percutaneous mitral balloon valvuloplasty (PBMV) or mitral valve surgery should be considered before pregnancy to reduce the need for gestational treatment and to improve pregnancy outcomes.2,3,25,28 For pregnant patients who develop cardiac symptoms as a result of mitral stenosis, heart rate reduction with β blockers and restriction of physical activity are the mainstays of medical therapy. To avoid interference with uterine relaxation, β₁-selective antagonists are preferred. However, atenolol should be avoided in the early stages of pregnancy and only given with caution in the later stages because of its association with fetal growth retardation.29 There is no direct evidence from clinical studies that atenolol is teratogenic, but the studies available included only a small number of women and the treatment was often not started in the first trimester. Maternal heart rate and arterial blood pressure should be monitored frequently during therapy with β blockers to avoid hypotension and placental hypoperfusion.28 Diuretics should also be used cautiously because hypovolemia can impair fetal blood supply. If atrial fibrillation develops, digoxin can be used to control the rate of ventricular response and anticoagulation should be initiated to prevent thromboembolic events.

Selected patients with mitral stenosis who develop worsening or recurrent heart failure despite medical therapy can be effectively managed with PBMV in experienced centers.7,28,30 An echocardiographic score developed by Wilkins et al.30 is used to select patients who may be appropriate candidates. Four variables of mitral valve morphology are assessed, as shown in table 4. Each variable is graded on a 5-point (0–4) scale. Total echocardiographic score is then derived as the sum of the points assigned for each variable, yielding a score between 0 and 16. Patients with an echocardiographic score of 8 or less have the best results from PBMV, whereas a score higher than 11 is associated with increased incidence of poor outcomes. Echocardiographic scores of 9–11 are less predictive for good outcomes, but patients with extensive subvalvular disease tend to have poorer outcomes.

Significant (3+ or 4+) mitral regurgitation and left atrial thrombus are contraindications to PBMV. The procedure is
usually performed near the end of the second trimester to reduce the negative effects of ionizing radiation to the fetus. Potential complications include atrial fibrillation, systemic embolism, bleeding, cardiac tamponade, mitral regurgitation, uterine contractions, and premature labor. Currently, it is not clear whether PBMV improves fetal weight and maturity. Considering the potential procedural risks, PBMV during pregnancy should be reserved for symptomatic patients with severe mitral stenosis who do not respond sufficiently to medical therapy. Mitral valve surgery is reserved for those refractory to medical therapy who are not suitable candidates for PBMV because cardiovascular surgery during pregnancy is associated with fetal mortality in about one third of cases. \(^{31,32}\)

Our patient had no prior awareness of RHD. She had baseline dyspnea with moderate exertion until late in the second trimester. In addition to having moderate to severe mitral stenosis, she had moderate to severe mitral regurgitation, a situation not well suited to PBMV. At presentation, she was short of breath with minimal exertion, but exercise tolerance improved with metoprolol treatment. With \(\beta\) blockade and restriction of physical activity, her pregnancy continued without unusual difficulty. She remained in sinus rhythm throughout pregnancy, developing infrequent episodes of symptomatic sinus tachycardia that responded to supplemental intravenous doses of metoprolol.

**Anesthetic Goals.** With the increase in transmirtal gradient associated with clinically significant mitral stenosis, more time for diastolic filling is necessary to ensure adequate preload. Whereas heart rate is inversely proportional to diastolic filling time, low heart rates are recommended. The decrease in diastolic left ventricular filling time associated with pregnancy-induced tachycardia further increases left atrial pressures. \(^{20}\) The main hemodynamic goal in mitral stenosis is to avoid tachycardia to optimize left ventricular diastolic filling time. The increase in transmitral gradient can be further complicated during labor. Tachycardia resulting from pain and \(\beta\)-agonist tocolytic therapy further decreases diastolic left ventricular filling time. Vasodilation from neuroaxial blockade can also lead to reflex tachycardia. Other hemodynamic goals are the maintenance of normal to high preload, afterload, and contractility. Significant decreases in systemic vascular resistance, which result in reflex tachycardia, are poorly tolerated. Accordingly, in case of hemodynamic instability, the vasopressor choice should be tailored to these hemodynamic goals. Epinephrine should be avoided as it may induce tachycardia. Phenylephrine may restore stable hemodynamics with little or no unwanted effect on uteroplacental perfusion. \(^{33}\) The use of norepinephrine may provide additional inotropic support without significant increases in heart rate, while vasopressin relatively spares pulmonary vasculature.

There are no evidence-based guidelines detailing which anesthetic technique is optimal in pregnant patients with mitral stenosis for labor analgesia or anesthesia for Cesarean section. Timely provision of analgesia and anesthesia, however, is essential for reduction of peripartum maternal morbidity and mortality. \(^{33}\) Expert opinion\(^ 6\) is that the choice of anesthetic technique must be individualized. It is dependent on understanding the physiology of pregnancy and its interaction with the individual patient’s pathophysiology, including lesion severity. An understanding of the impact that any pharmacologic therapy will have on peripartum anesthetic care is essential. Because alterations in hemodynamic status continue to occur for the first 24 h after delivery, adequate cardiovascular monitoring must be maintained from the peripartum into the postpartum period to reduce morbidity and mortality. \(^{34}\)

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**Table 4. Wilkin’s Scoring of Mitral Stenosis**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Mobility</th>
<th>Subvalvular Thickening</th>
<th>Leaflet Thickening</th>
<th>Calcification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Highly mobile valve with only leaflet tips restricted</td>
<td>Minimal thickening just below the mitral leaflets</td>
<td>Leaflets near normal in thickness (4–5 mm)</td>
<td>A single area of increased echo brightness</td>
</tr>
<tr>
<td>2</td>
<td>Leaflet mid and base portions have normal mobility</td>
<td>Thickening of chordal structures up to one third of the chordal length</td>
<td>Mid leaflets normal, considerable thickening of margins (5–8 mm)</td>
<td>Scattered areas of brightness confined to leaflet margins</td>
</tr>
<tr>
<td>3</td>
<td>Valve continues to move forward in diastole, mainly from the base</td>
<td>Thickening extending to the distal third of the chords</td>
<td>Thickening extending through the entire leaflet (5–8 mm)</td>
<td>Brightness extending into the mid-portions of the leaflets</td>
</tr>
<tr>
<td>4</td>
<td>No or minimal forward movement of the leaflets in diastole</td>
<td>Extensive thickening and shortening of all chordal structures extending down to the papillary muscles</td>
<td>Considerable thickening of all leaflet tissue (&gt;8–10 mm)</td>
<td>Extensive brightness throughout much of the leaflet tissue</td>
</tr>
</tbody>
</table>

There is uncertainty regarding the use of neuroaxial anesthesia in women with fixed cardiac output. Although neuroaxial blockade has the ability to reduce or even abolish the cardiovascular stress response to pain, it also reduces systemic vascular resistance and cardiac preload, causing a reflex tachycardia. Gomar et al. conducted a literature review on neuroaxial anesthesia for obstetric patients with cardiac disease. In general, single-shot spinal anesthesia is not recommended for the pregnant patient with significant mitral stenosis. In contrast, a well-controlled, individualized neuroaxial block using incremental dosing of local anesthetic with invasive monitoring of arterial and cardiac filling pressures may be beneficial even for the most severe cardiac disease. The presence of atrial fibrillation may necessitate the use of anticoagulation. In these patients, American Society of Regional Anesthesia guidelines for the use of neuroaxial anesthesia should be followed to minimize the risk of spinal hematoma formation.

Kuczkowski et al. published recommendations for peripartum anesthesia for pregnant women with valvular disease. Labor analgesia for vaginal delivery is best accomplished with lumbar epidural anesthesia to minimize hemodynamic changes. Adequate analgesia will reduce catecholamine-induced increase in heart rate. The addition of opioids to dilute local anesthetic solutions enhances the quality of the analgesia without adding to the sympathetic blockade. Neuroaxial opioids alone may be administered to critically ill patients who cannot tolerate any sympathetic blockade. Intravenous fluid needs to be closely monitored to avoid both deleterious increases and decreases in preload. Invasive monitoring may be required to manage patients optimally.

Anesthetic options for Cesarean section include incrementally dosed lumbar epidural or general anesthesia. In general, an incrementally dosed lumbar epidural will provide the least amount of hemodynamic alteration. General anesthesia may provide very stable hemodynamics if the sympathetic stimulation associated with laryngoscopy and intubation are attenuated either by use of anesthetic agents or β blockade. During the intraoperative period as well, adequate depth of anesthesia is required to avoid tachycardia and hypotension. General anesthesia provides the advantages of definitive airway control and the ability to use transesophageal echocardiographic monitoring throughout the procedure. A number of case reports have described the use of general anesthesia with good maternal and fetal outcomes. Opioid-based techniques are often recommended for anesthesia in patients with severe valvular disease because they have a minimally depressive action on the cardiovascular system and provide excellent analgesia. In the case of Cesarean section, however, this technique could result in prolonged neonatal respiratory depression.

Remifentanil is a synthetic opioid that provides intense analgesia of rapid onset and short duration. Remifentanil crosses the placenta but appears to be rapidly metabolized and redistributed in mother and fetus. Thus, remifentanil has the ability to provide intraoperative hemodynamic stability and rapid maternal emergence and recovery from general anesthesia while preventing prolonged neonatal sedation and respiratory depression.

Because of the need to avoid uterine atony, large doses of inhalation anesthetics have to be avoided. In the present case, low doses of isoflurane were used. Alternative inhalation agents could have been used in the present case. However, with these low doses, we felt that the use of less soluble agents would not have had a significant effect on emergence characteristics.

Knowledge Gap
Despite improvements in medical care and treatments, such as PBMV, mitral stenosis remains a dangerous condition for the pregnant woman and her fetus. Current clinical recommendations regarding the management of mitral stenosis in pregnancy are largely based on the understanding of the underlying pathophysiology and observational studies. Although it has been demonstrated that PBMV can be performed safely in pregnant patients, there are no data from randomized controlled trials comparing PBMV with medical treatment. Fetal growth retardation remains an important concern with medical therapy, particularly with β blockers. Hypotension resulting from rate control with β blockers needs to be avoided, but future clinical studies may better define the minimum blood pressure required to maintain optimal placental perfusion. It also remains unclear whether a more aggressive interventional approach could improve low birth weights. Furthermore, optimal timing of interventional approaches is not defined. PBMV has risks that need to be considered; procedural risks are weighed against the benefits of improved hemodynamics for the remainder of the pregnancy and a potential decrease in the risk of the peripartum anesthesia.

Perioperative use of pulmonary artery catheters is controversial. The most recent practice guidelines for pulmonary artery catheterizations published by the American Society of Anesthesiologists concluded that pulmonary artery catheterization should be reserved for patients and procedures (1) where there is a high risk of significant hemodynamic disturbances, and (2) in practice settings where there are competent and experienced practitioners to insert the catheter and interpret the data derived from it. Although many practitioners continue to use catheterization routinely or in specific situations, its use has been called into question for failure to show benefit—and even a potential for increased mortality. Various explanations have been offered ranging from poor patient selection to misinterpretation of data and unreliable or inaccurate data. Additional studies are needed to define specific patient populations and circumstances where pulmonary artery catheterization can be used to improve outcomes.

Medical and anesthetic care must be individualized and a multidisciplinary approach is warranted. Care for these pa-
tients requires a thorough understanding of the specific cardiac disease and the functional physiologic changes induced by pregnancy and labor. Both management during pregnancy as well the timing and mode of delivery should be discussed and decided upon jointly by the obstetrician, cardiologist, anesthesiologist, and, possibly, cardiac surgeon. With this approach, it is possible to have good outcomes for mother and infant.

References

13. Padmavati S: Rheumatic fever and rheumatic heart disease in India at the turn of the century. Indian Heart J 2001; 53:35–7
“Old Ironsides” through the Holmes Stereoscope

Most anesthesiologists know that physician and writer Oliver Wendell Holmes, Sr. (1809–1894, portrayed right), suggested the word “anaesthesia” to celebrated etherizer W. T. G. Morton. This occurred 16 yr after Holmes immortalized in verse as “Old Ironsides” the 44-gun warship Constitution. Holmes’ poetry prevented “the harpies of the shore” (naval scrapyards) from plucking that “eagle of the sea.” About 29 yr after this intervention, Holmes invented his American hand-held stereoscope for viewing stereographs, which are paired photographs showing the same subjects or scenes from slightly staggered perspectives. To the left is a stereograph of the veteran ship from the War of 1812, the frigate that Holmes rescued—“Old Ironsides.” (Copyright © the American Society of Anesthesiologists, Inc. This image also appears in the Anesthesiology Reflections online collection available at www.anesthesiology.org.)

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