Combined Spinal-Epidural Analgesia for Laboring Parturient with Mitral Stenosis
C K Choi, G Torres, O Bogatyryova, W Bethune, N Younger, K Tyagaraj

INTRODUCTION
Anesthetic management of parturients with cardiac valvular pathology, such as rheumatic heart disease (RHD), can be challenging. The cardiovascular changes of normal pregnancy are well tolerated in healthy parturients but in those with valvular lesions, these changes are ominous. Incidence of cardiac disease in pregnancy (0.1-4%) has remained stable over the years. Valvular disease and New York Heart Association (NYHA) functional class are both important predictors for adverse outcomes. The maternal mortality for parturients with mitral stenosis and NYHA functional class III and IV is 6.8% as compared to 0.4% for those in the NYHA functional class I and II (Table 1). In this article, we present the anesthetic management of a parturient with symptomatic mitral stenosis who underwent labor and vaginal delivery using combined spinal-epidural (CSE) analgesia. We combine an evidence-based approach with our own clinical experience, patient and provider preferences, and a detailed knowledge of pathophysiology to guide individualized anesthetic management.

CASE REPORT
A 28 year-old female, G5P2, with history of childhood rheumatic fever and tonsillectomy was admitted to a community hospital at 28-week gestation complaining of dyspnea with exertion and orthopnea. The previous two vaginal deliveries were uncomplicated. She was diagnosed with RHD after transferring to our facility for management of acute pulmonary edema. On admission, physical examination showed a 82 kg woman afebrile, in mild distress with a blood pressure of 101/66 mmHg and a respiratory rate of 21. Electrocardiogram showed sinus tachycardia of 111/min with left atrial enlargement. Bibasilar crackles and scattered rhonchi were heard on chest auscultation. A mild pansystolic, early diastolic murmur was heard at the apex of the heart. Chest radiograph revealed pulmonary vascular congestion with bibasilar opacity. B-type natriuretic peptide (BNP) was elevated to 482 pg/mL.

An echocardiogram revealed moderate mitral stenosis (valve area of 1.2 cm² and mean gradient of 9-10 mmHg), left ventricular ejection fraction of 60%, severe mitral regurgitation, and mild to moderate pulmonary hypertension. Furosemide was initiated for aggressive diuresis and propranolol for heart rate control. BNP decreased to 166 pg/mL after treatment. Due to severe mitral regurgitation, the patient was not a candidate for valvuloplasty. She was re-admitted for dyspnea at 37-week gestation for non-compliance with medications. After
optimization for nine days, the multidisciplinary team decided for a trial of vaginal delivery. Baseline laboratory values were: hemoglobin 11.0 g/dL, platelets 186 × 10^9/L, prothrombin time 9.6 sec, and partial prothrombin time 21.9 sec. Vital signs: blood pressure 105/54 mmHg, heart rate 56/min, and pulse oximetry 97% at 2 L of oxygen on nasal cannula. A 20-gauge radial arterial line was placed on the left hand and a 7.0-French triple lumen catheter on the right internal jugular vein for central venous pressure (CVP) monitoring.

CSE analgesia was achieved using a 17-gauge Tuohy needle and 5-inch Whitacre spinal needle into the L5-S1 interspinous space without prehydration. Analgesia was obtained with fentanyl 20 µg and preservative-free morphine 0.2 mg. Aspiration of the epidural catheter was negative. A test dose (3 mL of lidocaine 1.5% with 1:200,000 epinephrine) was not performed because inadvertent intravascular injection of epinephrine (15 µg) can potentially produce life-threatening tachyarrhythmia, or lidocaine (45 mg) into the subarachnoid space can cause profound sympathectomy with sudden vasodilation. Oxytocin 0.004% infusion at rate of 1 mU/min was started.

An epidural infusion of bupivacaine 0.1% and fentanyl 0.0002% was initiated at the rate of 10 mL/h. Diphenhydramine 25 mg was administered for intense pruritus after 2 h of infusion. Left lateral tilt was performed to avoid aortocaval compression. Fetal heart rate (FHR) and uterine contractions were continuously monitored by an external cardiotocograph. Category I FHR tracing was noted throughout the first stage of labor. Bolus of bupivacaine 0.0625% and fentanyl 100 µg were injected epidurally 45 min after placement of CSE and subsequently augmented with another bolus of bupivacaine 0.125% when oxytocin was increased to 2 mU/min to provide further analgesia and prevent undue tachycardia. Dermatomal analgesia was achieved at the T10 level.

### Table 1
New York Heart Association functional classification of heart failure

<table>
<thead>
<tr>
<th>Class</th>
<th>Functional Description</th>
</tr>
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<tbody>
<tr>
<td>I</td>
<td>Asymptomatic except during severe activity</td>
</tr>
<tr>
<td>II</td>
<td>Symptomatic with moderate activity</td>
</tr>
<tr>
<td>III</td>
<td>Symptomatic with minimal activity</td>
</tr>
<tr>
<td>IV</td>
<td>Symptomatic at rest</td>
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</table>

### Table 2
Intrapartum and postpartum hemodynamic measurements

<table>
<thead>
<tr>
<th>Parameter</th>
<th>% Change</th>
</tr>
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<tbody>
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<td>Cardiac output</td>
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</tr>
<tr>
<td>Stroke volume</td>
<td>+25</td>
</tr>
<tr>
<td>Heart rate</td>
<td>+25</td>
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<tr>
<td>Blood volume</td>
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<tr>
<td>Plasma volume</td>
<td>+55</td>
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<tr>
<td>Contractility</td>
<td>Variable</td>
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<tr>
<td>Systemic blood pressure</td>
<td>-5</td>
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<tr>
<td>Systemic vascular resistance</td>
<td>-20</td>
</tr>
<tr>
<td>Central venous pressure</td>
<td>Unchanged</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>Unchanged</td>
</tr>
</tbody>
</table>

### Table 3
Cardiovascular changes of maternal hemodynamics during term pregnancy
Labor progressed rapidly and lasted 6 h. The patient gave birth to a 2,825 g healthy girl. Apgar scores at 1 and 5 min were both 9. CVP (10-13 mmHg) and maternal heart rate (58-74/min) were stable from the second stage of labor to postpartum (Table 2). She received intravenous fluid at the rate of 5-10 mL/h and sips of water orally intrapartum. Estimated blood loss was 200 mL. Intramuscular oxytocin 10 mg and rectal misoprostol 1 mg were given to prevent postpartum hemorrhage. The patient recovered uneventfully and was discharged five days later. Thereafter, patient successfully underwent open mitral valve repair three months later (Fig. 1 and 2).

**Figure 1**
Transesophageal echocardiogram before mitral valve replacement. Arrows indicate the classic rheumatic mitral valvar thickening involving both the anterior leaflet (A2) and posterior leaflet (P2) in the midesophageal four-chamber view. RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle.

**Figure 2**
Transesophageal echocardiogram after mitral valve replacement. MMV, mechanical mitral valve prosthesis, LA, left atrium; RV, right ventricle; LV, left ventricle. Note the shadowing artifact within the LV chamber caused by the metallic bileaflet prosthesis.

**DISCUSSION**
Cardiovascular changes in healthy parturients begin in the first trimester and progress to its maximum at term (Table 3). Pregnancy produces a 30-50% increase in blood volume, with red cell mass lagging behind plasma volume resulting in a relative anemia. Both heart rate and stroke volume increase as a result of cardiac muscle hypertrophy, leading to increased cardiac output. Nonetheless, maternal hemodynamic stability is dependent on intact vasomotor
responses. These observations, along with the understanding that uterine contractions increase both CVP and blood pressure, underscore the importance of fluid and pain management during pregnancy.

Tachycardia worsens mitral stenosis by decreasing diastolic filling time, in turn decreasing cardiac output through a fixed stenotic valvular orifice. The normal atrial contribution to stroke volume is approximately 20%. However, this can increase to as much as 35% in mitral stenosis and is an important compensatory mechanism that preserves adequate cardiac output and blood pressure. The increase of intravascular volume in pregnancy results in further left atrial enlargement, predisposing the parturient for potential arrhythmias as well as increased pulmonary venous filling pressure. The endpoint of pulmonary vascular congestion, if severe and left untreated, is right ventricular failure secondary to pulmonary hypertension.

Previous publications describing the use of CSE or epidural analgesia for laboring parturients with mitral stenosis were limited to case reports (Table 4). CSE was used to achieve labor analgesia in our patient. Neuraxial blockade is beneficial but can pose challenges to control the hemodynamics. With an arterial line, slow segmental boluses through the epidural can preserve the hemodynamic integrity of both the mother and fetus. We advocate high intrathecal fentanyl (20-25 µg) for immediate pain control with addition of preservative-free morphine (0.2-0.3 mg) for longer effect. Morphine can decrease the local anesthetic requirement during the later stages of labor and can minimize the sympathetic response from contraction pain.7 While dual intrathecal opioids were proposed, local anesthetic was not administered due to her low blood pressure.8-10 The heart rate decreased from 88 to 70/min after intrathecal opioids without significant changes to blood pressure.

The greatest risks posed to parturients with mitral stenosis are acute pulmonary edema and atrial tachyarrhythmias during the first several hours postpartum since cardiac output can increase as much as 75-80% and return to prelabor value two days later. Measure to prevent fluid overload included implementing minimal intravenous and moderate oral hydration. This strategy reduced the total amount of hydration and also offered increased comfort and patient satisfaction.11 With aortocaval compression released after delivery of the fetus, the involution of the uterus, and autotransfusion of placental blood, there is a surge of blood back into the pulmonary circuit. With preemptive epidural bolus of bupivacaine gradually titrated 15 min before the patient beared down to deliver, the expansion of the venous circuit allowed blood volume to accumulate without causing much fluid shift into the lungs.

The use of invasive monitors during labor and delivery is controversial especially for placement of pulmonary arterial catheter (PAC).12 Many propose the minimum of an arterial line and CVP monitoring for symptomatic mitral stenosis for strict blood pressure control and fluid management, respectively. While others have used PAC for mitral stenosis parturients with multiple valvular abnormalities, cardiac arrhythmia, maternal hypertension, severe pulmonary hypertension, and even without a clear indication.13-17 CVP monitoring was used to guide our management on her fluid status. Pulmonary arterial pressure was not monitored because the risks outweighed the benefits.

Although there are no evidence-based guidelines except for recommendations as to which technique is optimal for labor analgesia and anesthesia for Cesarean section (CS), perioperative management should be goal-directed towards the pathophysiology of the cardiac disease and tailored to the needs of the mother and fetus to avoid complications.18,19 Spontaneous vaginal birth with labor analgesia is the preferred mode of delivery over CS, provided there are no obstetrical indications, since studies have shown that vaginal deliveries are well tolerated in most patients with valvular diseases.20 In summary, the case report describes the successful anesthetic management of laboring patients with mitral stenosis using CSE. Early planning, multidisciplinary care, cardiac screening, fluid management, selective usage of invasive monitoring, and timely implementation of CSE analgesia with appropriate choice of opioids and local anesthetics were essential in producing good maternal and fetal outcomes.

References
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