# Combined Spinal-Epidural Analgesia for Laboring Parturient with Mitral Stenosis

C K Choi, G Torres, O Bogatyryova, W Bethune, N Younger, K Tyagaraj

**Citation**

C K Choi, G Torres, O Bogatyryova, W Bethune, N Younger, K Tyagaraj. *Combined Spinal-Epidural Analgesia for Laboring Parturient with Mitral Stenosis*. The Internet Journal of Anesthesiology. 2013 Volume 32 Number 1.

**Abstract**

Understanding the changes in cardiovascular physiology that occur during pregnancy is important in order to optimize anesthetic management and to avoid adverse maternal and fetal outcomes. The effects of a normal gestation on the cardiovascular system are particularly significant in parturients with cardiac valvular pathology. In this paper, we discuss the anesthetic management of a laboring parturient with mitral stenosis using combined spinal-epidural labor analgesia. The patient received minimal intravenous hydration (5-10 mL/h) to avoid fluid overload and was encouraged to drink clear liquids during labor. High dose of fentanyl (20-25 µg) was injected intrathecally for initial pain control. Preservative-free morphine (0.2-0.3 mg) was then used to manage contraction-related pain while maintaining stable maternal hemodynamics on an epidural infusion. The patient had adequate analgesia and tolerated labor and vaginal delivery without complications. Due to the severity of her mitral stenosis, intrapartum fluid management required guidance by invasive monitoring. A brief literature review of the anesthetic management of parturients with mitral stenosis is also presented to compare and contrast the different combined spinal-epidural techniques and outcomes.

## 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 vulvular lesions, these changes are omnious. Incidence of cardiac disease in pregnancy (0.1-4%) has remained stable over the years.1 Valvular disease and New York Heart Association (NYHA) functional class are both important predictors for adverse outcomes.2-4 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.5

## 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 × 109/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 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

| Class | Functional Description |
|---|---|
| I | Asymptomatic except during severe activity |
| II | Symptomatic with moderate activity |
| III | Symptomatic with minimal activity |
| IV | Symptomatic at rest |

**Table 2**

Intrapartum and postpartum hemodynamic measurements

| | First Stage of Labor | | | | | | | | Second Stage of Labor | Postpartum | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Before induction of labor | After CSE placement | During first painful contraction 45 min after CSE | After 10 mL of bupivacaine 0.0625% and fentanyl 100 μg | During second painful contraction 2 h after CSE | After 5 mL of bupivacaine 0.125% | During third painful contraction 15 min before delivery | After 10 mL of bupivacaine 0.125% | During contraction bearing down | After 15 min | After 2 h |
| Blood pressure (mmHg) | 96/55 | 101/62 | 97/52 | 92/57 | 92/57 | 99/53 | 110/62 | 108/62 | 110/65 | 109/62 | 100/68 |
| Heart rate (beats/min) | 88 | 70 | 61 | 63 | 62 | 64 | 72 | 70 | 72 | 74 | 58 |
| $SpO_2$ (%) | 98 | 98 | 98 | 98 | 98 | 98 | 99 | 99 | 98 | 98 | 99 |
| EKG | SR | SR | SR | SR | SR | SR | SR | SR | SR | SR | SR |
| CVP (mmHg) | 10 | 10 | 9 | 9 | 9 | 9 | 9 | 9 | 13 | 13 | 10 |
| FHR (beats/min) | 135 | 135 | 140 | 140 | 140 | 140 | 140 | 130 | — | — | — |
| FHR tracing category | I | I | I | I | II | I | I | II | — | — | — |

EKG: electrocardiogram; SR: sinus rhythm; FHR: fetal heart rate; CSE: combined spinal-epidural; CVP: central venous pressure

**Table 3**

Cardiovascular changes of maternal hemodynamics during term pregnancy

| Parameter | % Change |
|---|---|
| Cardiac output | +50 |
| Stroke volume | +25 |
| Heart rate | +25 |
| Blood volume | +45 |
| Plasma volume | +55 |
| Contractility | Variable |
| Systemic blood pressure | -5 |
| Systemic vascular resistance | -20 |
| Central venous pressure | Unchanged |
| Pulmonary capillary wedge pressure | Unchanged |

**Table 4**

Summary of anesthetic management of patients with mitral stenosis

| Authors and references | Gravida and para | Gestation age (weeks) | NYHA Class | Delivery method | Apgar at 1 min, 5 min | Mitral valve area ($cm^2$) | Neuraxial method | IV fluid preloading | Intrathecal drug | Epidural drug | Arterial line | CVP | PAC |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Hyun Kee et al.[10] | G4P1 | 40 | III* | NSVD | 8, 9 | 1.5 | CSE | No | Fentanyl | Fentanyl, bupivacaine | Yes | Yes | No |
| | G6P2 | 40 | III* | NSVD | 9, 10 | 1.1 | CSE | No | Fentanyl | Fentanyl, bupivacaine | Yes | Yes | No |
| | G2P1 | Not reported | I* | NSVD | 9, 10 | 1.4 | CSE | Not reported | Fentanyl | Fentanyl, bupivacaine | Yes | Yes | No |
| Hemmings et al.[13] | G1P0 | 37 | II | NSVD | 6, 9 | Not reported | Epidural | Not reported | Not reported | Lidocaine, bupivacaine | Yes | Yes | Yes |
| Shin et al.[14] | G1P0 | Not reported | Not reported | NSVD | Not reported | 1.2** | Epidural | Not reported | Not reported | Fentanyl, bupivacaine | Yes | Yes | Yes |
| Sharma et al.[15] | G3P2 | 38.5 | II* | NSVD | 9, 9 | 2.0 | Epidural | Not reported | Not reported | Fentanyl, bupivacaine | Yes | Yes | Yes |
| Kocum et al.[16] | Not reported | 32 | Not reported | CS | Not reported, 10 | 0.67 | Epidural | Not reported | Not reported | Fentanyl, bupivacaine | Yes | Yes | No |
| Afrangui et al.[17] | G3P1 | 34 | I* | CS | 6, 8 | 1.3 | Epidural | Not reported | Not reported | Fentanyl, morphine, bupivacaine | Yes | Yes | Yes |
| VanHelder et al.[9] | G1P0 | 38 | I* | CS | 7, 8 | 1.6** | CSE | Not reported | Sufentanil, bupivacaine | Bupivacaine | Yes | Yes | No |
| Pan et al.[8] | G1P0 | 35 | I* | CS | Not reported | 2.0 | CSE | Yes | Fentanyl, morphine, bupivacaine | Fentanyl, bupivacaine | No | No | No |
| | G1P0 | Term | IV* | CS | 3, 8 | 1.2 | Epidural | Not reported | Not reported | No | Yes | Yes | Yes |
| Ziskind et al.[12] | Not reported | 35 ± 2.2*** | III-IV | CS | Not reported | 0.9 ± 0.2*** | Epidural | No | Not reported | Bupivacaine | Yes | Yes | Yes |
| Our patient | G5P2 | 37 | III | NSVD | 9, 9 | 1.2 | CSE | No | Fentanyl, morphine | Fentanyl, bupivacaine | Yes | Yes | No |

CS: cesarean section; NSVD: normal spontaneous vaginal delivery; CSE: combined spinal-epidural; NYHA: New York Heart Association; CVP: central venous pressure; PAC: pulmonary arterial catheter; IV: intravenous
* Interpreted from the functional description of NYHA classification
** Patient has also severe aortic stenosis (valve area < 1 $cm^2$)
*** Expressed as mean ± standard deviation for seven patients

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 valvular 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.

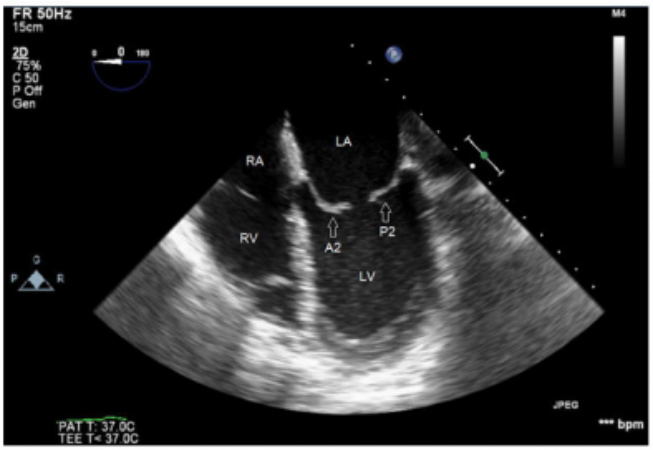
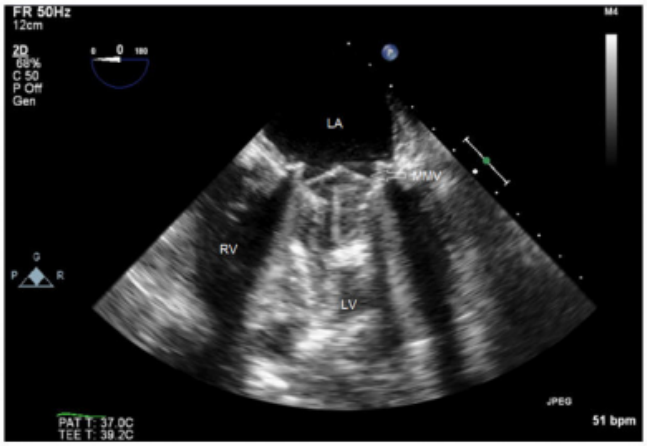



**Figure 2**

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).6 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 albeit local anesthetic can be given intrathecally in certain patient type with stable hemodynamics.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 vulvular 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

1. van Mook WN, Peeters L. Severe cardiac disease in pregnancy, part I: hemodynamic changes and complaints during pregnancy, and general management of cardiac disease in pregnancy. Curr Opin Crit Care 2005;11:430-4.
2. Siu SC, Colman JM, Sorensen S, et al. Adverse neonatal and cardiac outcomes are more common in pregnant women with cardiac disease. Circulation 2002;105:2179–84.
3. Siu SC, Sermer M, Colman JM, et al. Prospective multicenter study of pregnancy outcomes in women with heart disease. Circulation 2001;104:515–21.
4. Reimold SC, Rutherford JD. Clinical practice. Valvular heart disease in pregnancy. N Engl J Med 2003;349:52-9.
5. Pronovost PJ, Berenholtz SM, Dorman T, Merritt WT,

Martinez EA, Guyatt GH. Evidence-based medicine in anesthesiology. Anesth Analg 2001;92:787-94.
6. Gaiser R. Physiologic changes of pregnancy. In: Chestnut DH, Polley LS, Tsen LC, Wong CA, editors. Chestnut's Obstetric Anesthesia: Principles and Practices. 4th ed. Philadelphia: Elsevier Mosby; 2009. p. 15-36.
7. Vasudevan A, Snowman CE, Sundar S, Sarge TW, Hess PE. Intrathecal morphine reduces breakthrough pain during labour epidural analgesia. Br J Anaesth 2007;98:241-5.
8. Pan PH, D'Angelo R. Anesthetic and analgesic management of mitral stenosis during pregnancy. Reg Anesth Pain Med 2004;29:610-5.
9. VanHelder T, Smedstad KG. Combined spinal epidural anaesthesia in a primigravida with valvular heart disease. Can J Anaesth 1998;45:488-90.
10. Ngan Kee WD, Shen J, Chiu AT, Lok I, Khaw KS. Combined spinal-epidural analgesia in the management of labouring parturients with mitral stenosis. Anaesth Intensive Care 1999;27:523-6.
11. American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Anesthesiology 2007;106:843-63.
12. Hemmings GT, Whalley DG, O'Connor PJ, Benjamin A, Dunn C. Invasive monitoring and anaesthetic management of a parturient with mitral stenosis. Can J Anaesth 1987;34:182-5.
13. Afrangui B, Malinow AM. Severe preeclampsia complicating mitral valve stenosis. Reg Anesth Pain Med 1998;23:204-9.
14. Shin YK, King JC. Combined mitral and aortic stenosis in a parturient: epidural anesthesia for labor and delivery. Anesth Analg 1993;76:682-3.
15. Sharma SK, Gambling DR, Gajraj NM, Truong C, Sidawi EJ. Anesthetic management of a parturient with mixed mitral valve disease and uncontrolled atrial fibrillation. Int J Obstet Anesth 1994;3:157-62.
16. Kocum A, Sener M, Cal?skan E, et al. Epidural anesthesia for cesarean section in a patient with severe mitral stenosis and pulmonary hypertension. J Cardiothorac Vasc Anesth 2010;24:1022-3.
17. Ziskind Z, Etchin A, Frenkel Y, et al. Epidural anesthesia with the Trendelenburg position for cesarean section with or without a cardiac surgical procedure in patients with severe mitral stenosis: a hemodynamic study. J Cardiothorac Anesth 1990;4:354-9.
18. Gomar C, Errando CL. Neuroaxial anaesthesia in obstetrical patients with cardiac disease. Curr Opin Anaesthesiol 2005;18:507-12.
19. Weiner MM, Vahl TP, Kahn RA. Case scenario: Cesarean section complicated by rheumatic mitral stenosis. Anesthesiology 2011;114:949-57.
20. Elkayam U, Bitar F. Valvular heart disease and pregnancy part I: native valves. J Am Coll Cardiol 2005;46:223-30.

## Author Information

**Clark K. Choi**

**Giselle Torres**

**Oksana Bogatyryova**

**Walter Bethune**

**Natalie Younger**

**Kalpana Tyagaraj**