

CASE REPORT

Hemodynamic Stability Under Graded Epidural Anesthesia for Emergency Cesarean Delivery in a Parturient with Large Atrial Septal Defect and Pulmonary Hypertension: An Observational Analytic Study

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ARTICLE INFO

Article history

Received: August 22, 2025
Revised: September 19, 2025
Accepted: October 15, 2025
Available online: October 25, 2025
Published: October 29, 2025

Keywords

Atrial septal defect
Cesarean delivery
Graded epidural anesthesia
Levobupivacaine
Pulmonary hypertension

DOI

<https://doi.org/10.37275/jacr.v6i2.990>

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ABSTRACT

Background. Pregnancy with a large uncorrected secundum atrial septal defect (ASD) and pulmonary hypertension carries the highest maternal risk (modified WHO class IV). A sudden fall in systemic vascular resistance can reverse the shunt and precipitate right ventricular failure, which makes the anesthetic technique for cesarean delivery critical.

Objective. To describe the hemodynamic course of a graded lumbar epidural with invasive monitoring in such a patient.

Methods. A 28-year-old primigravida at 37+2 weeks with a 4.8 cm secundum ASD, severe right-heart dilatation, and an echocardiographic systolic pulmonary artery pressure of 50.2 mmHg needed emergency cesarean delivery for premature rupture of membranes and intrauterine fetal demise. After radial arterial and central venous cannulation, 0.5% levobupivacaine (85 mg in four aliquots) was given epidurally to a T6 level. Mean arterial pressure (MAP), heart rate, and central venous pressure (CVP) were recorded at five time points. The report follows the CARE guideline.

Results. The T6 level was reached at 25 minutes. MAP was 83 mmHg at baseline, 72 mmHg after induction (–13.3%), and 71 mmHg at closure (–14.5%). Heart rate stayed at 72–82 beats/min and CVP at 8–10 mmHg. No vasopressor, inotrope, or pulmonary vasodilator was needed. She was discharged on day 4 without complications.

Conclusion. In this patient, slow epidural titration of levobupivacaine was followed by stable MAP, heart rate, and CVP without vasoactive support. One case cannot prove a benefit, and systemic vascular resistance and shunt direction were not measured.

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1. Introduction

Women with complex structural heart disease now reach childbearing age in growing numbers. They form a rising share of the high-risk obstetric population.^{1–3} Advances in pediatric cardiology, corrective surgery, and catheter-based intervention over recent decades mean that more than 85% of females born with significant congenital cardiac anomalies survive to reproductive maturity.^{4–6} Cardiac disease has therefore become the leading cause of indirect maternal death in developed and emerging nations alike, accounting for approximately 15% to 25% of all maternal deaths.^{7–9} Among the structural defects seen in women of reproductive age, the atrial septal defect (ASD) is one of the most frequently diagnosed.^{10,11} A small, restrictive defect is usually well tolerated in pregnancy. A large, uncorrected secundum defect is not. It drives progressive cardiopulmonary derangement.^{11,12}

An unrestrictive interatrial communication creates a persistent left-to-right shunt, driven by the difference in compliance between the high-pressure left-sided chambers and the low-resistance right heart.¹⁰ The shunt

never lets up. It places a continuous volume load on the right atrium, the right ventricle (RV), and the pulmonary arterial circulation.¹⁰ Over years or decades, this chronic right-sided overload causes first adaptive and then maladaptive remodeling: the chambers dilate, the myocardium undergoes eccentric hypertrophy, and the tricuspid annulus stretches.^{10,13} High pulmonary blood flow also exposes the pulmonary vessels to sustained shear stress. Endothelial injury, medial muscular hypertrophy, and plexogenic arteriopathy follow, and secondary precapillary pulmonary hypertension (PH) results.^{13,14} When a large uncorrected shunt coexists with secondary pulmonary hypertension, maternal risk reaches its highest tier. The modified World Health Organization (WHO) classification places such patients in Class IV, in which pregnancy is contraindicated because rates of maternal cardiac decompensation and death are prohibitive.^{1,15}

Pregnancy itself stresses the cardiovascular system. It pushes maternal circulatory reserve toward its limits.¹⁶ Plasma volume expands by 40% to 50%, baseline cardiac output rises by 30% to 50%, systemic vascular resistance (SVR) falls by 20% to 30%, and resting heart rate increases by 10 to 15 beats/min.^{1,17} A healthy heart copes through

biventricular remodeling. A heart already burdened by a large left-to-right shunt and raised pulmonary pressure has no reserve left for extra volume or pressure.

The peripartum period is the most dangerous phase of gestation for a cardiac patient. Large hemodynamic swings are the rule. They come from labor pain, uterine contractions, autotransfusion, and abrupt changes in vascular tone.¹⁷ Each contraction pushes roughly 300 to 500 mL of blood from the uteroplacental bed into the central circulation, and that volume arrives suddenly at a chronically distended, poorly compliant right heart. Pain, anxiety, and autonomic stress also release endogenous catecholamines. These raise systemic afterload, heart rate, and pulmonary vascular resistance (PVR). In a patient with chronic RV pressure and volume overload, ventricular interdependence becomes the main determinant of circulatory collapse.¹⁸

The two ventricles share a septum, reciprocal myofibers, and a fibrous pericardial envelope that cannot expand. A change in the volume or pressure of one chamber therefore acts mechanically on the other.¹⁸ In a parturient with chronic RV overload, the dilated, hypertensive RV displaces the interventricular septum toward the left ventricular (LV) cavity in both systole and diastole. The resulting septal flattening produces the characteristic 'D-shaped' left ventricle.^{18,19} LV diastolic filling is restricted, end-diastolic volume falls, and forward stroke volume suffers. Systemic output now hangs on RV forward performance. Any sudden change in RV loading conditions can cause severe systemic hypoperfusion.

Choosing the anesthetic technique for cesarean delivery in such a patient is hard. The margin for error is almost nil. Single-shot spinal anesthesia is widely considered relatively contraindicated in parturients with pulmonary hypertension and large intracardiac communications, because a rapid subarachnoid injection causes abrupt, uncontrolled sympathetic denervation.^{20,21} This sympathectomy produces profound vasodilation that can lower SVR by more than 40% within minutes. Coronary perfusion pressure to the hypertrophied RV then drops. The interatrial pressure gradient may reverse and produce a right-to-left shunt with refractory hypoxemia (an Eisenmenger crisis), and venous return to a preload-dependent RV collapses.^{11,22}

General endotracheal anesthesia has its own hazards. Laryngoscopy and intubation provoke sympathetic surges. Volatile agents depress the myocardium directly, and positive-pressure ventilation compresses the pulmonary alveolar capillaries, which raises pulmonary vascular resistance and impedes RV ejection.^{21,23} Extubation adds the risk of coughing, hypertension, and hypoventilation with hypercarbia and acidosis, both of which are potent pulmonary vasoconstrictors.²¹ In published parturients with pulmonary arterial hypertension, general anesthesia was associated with higher maternal mortality.²⁴

Graded epidural anesthesia may offer a better alternative in theory. The technique uses small, incremental doses of local anesthetic through an indwelling epidural catheter. This gives temporal control over the sympathetic blockade. The block ascends segment by segment, so the cardiovascular system has time to compensate, systemic vascular resistance is preserved, abrupt venous pooling is avoided, and dense surgical anesthesia is still achieved.²⁰ The rationale is sound. Yet detailed reports remain scarce in the international literature, and few describe beat-to-beat hemodynamic trajectories, RV-pulmonary arterial

interaction, and longitudinal recovery in pregnant patients with a large, unclosed secundum ASD and secondary pulmonary hypertension.^{25,26} Most existing publications are brief anecdotal descriptions. They lack quantitative hemodynamics, rigorous methodology, and trajectory data.

This report describes the longitudinal hemodynamic course, the invasive arterial and central venous trajectories, and the recovery of a parturient with a 4.8 cm secundum ASD and moderate pulmonary hypertension. She underwent emergency cesarean delivery under a titrated 0.5% levobupivacaine graded epidural protocol. Continuous beat-to-beat invasive arterial pressure, serial central venous pressure (CVP) trends, and transthoracic echocardiography were available. The objective is to give a verifiable clinical account of maternal cardiovascular stability that may inform anesthetic planning in high-acuity obstetric cardiology.

2. Methods

2.1. Report design and reporting guideline

This is a single case report from a tertiary university teaching hospital, written in accordance with the CARE guideline.^{27,28} The aim was to describe maternal cardiovascular adaptation, biventricular pressure-volume interaction, and autonomic responses in a parturient with an uncorrected large secundum atrial septal defect and secondary precapillary pulmonary hypertension. She underwent emergency cesarean delivery under a titrated graded lumbar epidural regimen. The report combines real-time continuous physiological data acquisition with a retrospective review of the record. Observation covered five epochs. These were pre-induction baseline evaluation, incremental epidural titration, the surgical intraoperative epoch (skin incision, uterine delivery, and peritoneal closure), the immediate postpartum resuscitation epoch, and longitudinal recovery in the Cardiovascular Intensive Care Unit (CVICU) from postoperative day 0 through day 4. The CARE checklist is provided as supplementary material.

2.2. Setting

The patient was managed at a tertiary university teaching hospital (Dr. Moewardi Regional General Hospital, Surakarta, affiliated with the Faculty of Medicine, Universitas Sebelas Maret). Care involved the emergency obstetric unit, the operating room, and the Cardiovascular Intensive Care Unit. Attending anesthesiologists, obstetric surgeons, adult congenital cardiologists, neonatologists, and specialized CVICU nursing staff took part. Time is reported relative to the start of observation (T = 0) and in postoperative days.

2.3. Patient information and case selection

The patient was a 28-year-old primigravida at 37 weeks and 2 days of gestation who presented acutely to the emergency obstetric unit. Her cardiology records documented an isolated secundum atrial septal defect diagnosed at age 19. She received detailed multidisciplinary counseling on transcatheter device closure versus conservative surveillance. She chose medical follow-up. She had remained compensated in New York Heart Association (NYHA) functional class II throughout adult life, with no prior admission for cardiac failure.

The case was selected for reporting because it combined an uncorrected congenital interatrial communication

confirmed by two-dimensional Doppler echocardiography, documented secondary pulmonary hypertension, a term pregnancy requiring surgical delivery, and complete invasive hemodynamic monitoring data. No patient name, initials, or hospital number are given.

2.4. Case timeline

The chronology from the original diagnosis to discharge is set out in Table 1 and Figure 1, using relative time. Day 0 is the day of admission and cesarean delivery.

Table 1. Clinical timeline of the case from the original diagnosis to discharge, using relative time points.

Time point	Clinical event	Investigation and result	Intervention
Age 19 years	Diagnosis of large secundum ASD; NYHA class II thereafter, no admission for cardiac failure	Echocardiographic diagnosis (details not reported)	Counseling on transcatheter device closure versus surveillance; medical follow-up chosen
Third trimester	High-risk pregnancy co-managed by obstetrics and cardiology	TTE: ASD 3.2–4.8 cm, sPAP 50.2 mmHg, RA and RV dilatation, LVEF 60.0% (repeated on admission; see Table 3)	Cardiology and obstetric co-management
Day 0, admission	PROM at 37+2 weeks; IUFD confirmed by CTG and ultrasonography; RR 20 breaths/min	BP 118/65 mmHg, HR 78/min, SaO ₂ 98% (room air); pH 7.41, PaCO ₂ 31.8 mmHg, lactate 1.1 mmol/L	Multidisciplinary briefing; decision for emergency cesarean delivery
Day 0, T = 0–25 min	Invasive monitoring; epidural placement; graded blockade to T6	MAP 83.0 → 72.0 mmHg; HR 78 → 72/min; CVP 8.0 → 9.0 mmHg	Radial arterial line; IJ central line; test dose; 0.5% levobupivacaine 5.0, 5.0, 4.0, 3.0 mL at T = 5, 10, 15, 20 min (85 mg)
Day 0, T = 35–90 min	Skin incision (T = 35); delivery of the demised infant (T = 45); end of surgery (T = 90)	MAP 73, 75, 71 mmHg; HR 79, 82, 74/min; CVP 9, 10, 8 mmHg; EBL 350 mL	Oxytocin 0.03 IU/min; no vasopressor, inotrope, or iNO
Day 0, after surgery	Transfer to CVICU	MAP 72–80 mmHg; CVP 7–9 mmHg; urine output 1.2–1.5 mL/kg/h	PCEA: 0.1% levobupivacaine + fentanyl 2 µg/mL, 4 mL/h
Day 1	Mobilized to a chair; oral fluids advanced	ABG: pH 7.40, PaCO ₂ 34.5 mmHg, PaO ₂ 98.4 mmHg, lactate 1.0 mmol/L	—
Day 2	Care de-escalation	Coagulation tests normal	Epidural catheter and invasive lines removed; oral paracetamol and tramadol
Day 3	Independent ambulation	VAS pain < 2/10	—
Day 4	Discharged home	BP 115/70 mmHg, HR 72/min, SaO ₂ 99%; Hb 10.5 g/dL; pH 7.42	Cardiology follow-up scheduled for transcatheter ASD closure

Notes: ABG, arterial blood gas; ASD, atrial septal defect; BP, blood pressure; CTG, cardiotocography; CVICU, cardiovascular intensive care unit; CVP, central venous pressure; EBL, estimated blood loss; HR, heart rate; IJ, internal jugular; iNO, inhaled nitric oxide; IUFD, intrauterine fetal demise; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; NYHA, New York Heart Association; PCEA, patient-controlled epidural analgesia; PROM, premature rupture of membranes; RA, right atrium; RR, respiratory rate; RV, right ventricle; sPAP, systolic pulmonary artery pressure; T, minutes from the start of observation; TTE, transthoracic echocardiography; VAS, visual analog scale.



Figure 1. Longitudinal timeline of maternal clinical milestones, obstetric emergency presentation, anesthetic intervention, and postoperative recovery. Chronological evolution spanning historical baseline diagnosis at age 19, antepartum multidisciplinary monitoring, acute presentation with premature rupture of membranes and intrauterine fetal demise, emergency cesarean delivery under graded epidural blockade, and intensive care recovery. ASD, atrial septal defect; CVICU, cardiovascular intensive care unit; IUFD, intrauterine fetal demise; NYHA, New York Heart Association; PROM, premature rupture of membranes; TTE, transthoracic echocardiography.

2.5. Clinical findings and diagnostic assessment

On admission she was examined by the emergency obstetric and anesthesia teams. Non-stress cardiotocography and bedside obstetric ultrasonography were used to assess the fetus. Arterial blood gas analysis was performed on ambient air. The airway was assessed by Mallampati class, thyromental distance, inter-incisor opening, and cervical extension. Transthoracic echocardiography (TTE) was performed during third-trimester cardiology co-management and repeated on admission. It used two-dimensional imaging with color-flow Doppler and continuous-wave Doppler of the

tricuspid regurgitation jet to estimate systolic pulmonary artery pressure (sPAP), with right atrial pressure estimated from the inferior vena cava diameter and its inspiratory collapse.¹⁹ Right heart catheterization was not performed because of the late-gestation emergency presentation and its procedural risks.¹⁴ The diagnosis was therefore an echocardiographic one: a large secundum ASD with moderate secondary pulmonary hypertension, right-heart dilatation, and biventricular interdependence. The numerical findings are reported in Section 3.

2.6. Therapeutic intervention

Once emergency cesarean section was confirmed, a formal multidisciplinary briefing was held in the preoperative holding suite. A designated team leader ran a closed-loop communication framework. The cardiovascular substrate was fragile. Quantitative hemodynamic targets were therefore set in advance. Mean arterial pressure (MAP) was to stay within $\pm 20\%$ of baseline, with an absolute target above 65 mmHg. Heart rate was to remain between 60 and 90 beats/min to protect diastolic ventricular filling. Central venous pressure (CVP) was to be held at 8 to 12 mmHg. That range gives adequate RV preload without congestive distension.

Emergency pharmacological protocols were prepared in advance. A phenylephrine infusion (50 $\mu\text{g}/\text{mL}$) was primed on an automated syringe pump for rapid titration of systemic vascular resistance.²⁹ Dobutamine (2.5–10 $\mu\text{g}/\text{kg}/\text{min}$) and milrinone (0.25–0.75 $\mu\text{g}/\text{kg}/\text{min}$) were placed on immediate standby, and inhaled nitric oxide (iNO) equipment was checked for rescue pulmonary vasodilation. The uterotonic protocol excluded carboprost tromethamine (PGF 2α) and methylergonovine maleate, because both can precipitate acute bronchoconstriction and sharp rises in pulmonary vascular resistance.^{9,30} Oxytocin was limited to a dilute intravenous infusion (10 IU in 500 mL normal saline at 0.02–0.04 IU/min). Rapid boluses were deliberately avoided. They cause peripheral vasodilation and tachycardia.³⁰

The patient was positioned in the operating room in left lateral decubitus with a 15-degree pelvic tilt to limit aortocaval compression. Standard monitoring comprised 5-lead electrocardiography with continuous ST-segment analysis, dual-channel pulse oximetry, and non-invasive blood pressure cycling. Invasive monitoring was established before neuroaxial blockade, under local infiltration (2% lidocaine, 3 mL). A 20-gauge arterial catheter was placed in the left radial artery after a modified Allen's test. It was connected to a pressure transducer at the phlebostatic axis (fourth intercostal space, mid-axillary line), which gave beat-to-beat arterial waveforms and continuous digital MAP recording.³¹ A triple-lumen 7-French, 15-cm central venous catheter was inserted into the right internal jugular vein under real-time ultrasound guidance with an aseptic Seldinger technique. Its distal port went to a calibrated transducer for continuous CVP tracking. Baseline values at T = 0 were systolic blood pressure 118 mmHg, diastolic blood pressure 65 mmHg, MAP 83.0 mmHg, heart rate 78.0 beats/min, and CVP 8.0 mmHg.

With the patient sitting, the lumbar spine was palpated and prepared with 2% chlorhexidine gluconate in 70% isopropyl alcohol. After local infiltration with 1% lidocaine (4 mL), the L3–L4 interspace was entered by a midline approach with an 18-gauge Tuohy needle. The epidural space was identified at a depth of 4.5 cm by loss of resistance to saline. A 19-gauge multi-orifice nylon catheter was advanced 4.0 cm into the epidural space and secured at the skin at 8.5 cm. Aspiration for blood and cerebrospinal fluid was negative. A test dose of 3.0 mL of 1.5% lidocaine with 1:200,000 epinephrine (15 μg) was given. Over 4 minutes there was no chronotropic surge (>10 beats/min) and no neurosensory symptoms. Intravascular or intrathecal placement was excluded.

Surgical blockade was established with a slow, fractional dosing protocol of 0.5% isobaric levobupivacaine.³² At T = 5 minutes an initial aliquot of 5.0 mL (25 mg) was given.

Sensory levels were tracked by cold ice-pack dermatomal mapping and loss of pinprick discrimination. At T = 10 minutes, hemodynamics were stable and the sensory block had reached T10, so a second aliquot of 5.0 mL (25 mg) was injected. At T = 15 minutes the sensory level was T8 and hemodynamics were still within the target corridor, and a third aliquot of 4.0 mL (20 mg) followed. A final aliquot of 3.0 mL (15 mg) was given at T = 20 minutes. The cumulative induction dose was 17.0 mL (85 mg) of 0.5% levobupivacaine over 15 minutes. At T = 25 minutes the bilateral sensory block was confirmed at the T6 dermatome. Sharp pinprick sensation was completely lost, and diaphragmatic breathing was unlabored. Motor block on the modified Bromage scale was grade 1 (partial knee and foot flexion preserved), so impairment of the lower-limb venous muscle pump was minimal. Skin incision began at T = 35 minutes. Hemodynamics were stable.

After surgery the patient went directly to the Cardiovascular Intensive Care Unit with the arterial and central venous catheters in place and the epidural catheter connected to a patient-controlled epidural analgesia infusion of 0.1% levobupivacaine with fentanyl 2 $\mu\text{g}/\text{mL}$ at a basal rate of 4 mL/h.

2.7. Outcome measures and follow-up

Hemodynamic parameters (MAP, HR, and CVP) were sampled continuously and extracted at five predetermined milestones: baseline (T = 0), post-induction nadir at confirmation of the T6 block (T = 25 min), skin incision (T = 35 min), uterine delivery (T = 45 min), and end of surgery (T = 90 min). Changes were expressed as percentage delta relative to the pre-induction baseline: $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Here T₀ is the baseline value and T_n is the value at milestone n. Stability corridors were defined a priori. MAP had to stay within $\pm 20\%$ of baseline (66.4 to 99.6 mmHg), and HR within $\pm 20\%$ of baseline (62.4 to 93.6 beats/min). Arterial blood gases, metabolic parameters, hemoglobin, hematocrit, and urine output were recorded from the preoperative baseline through CVICU surveillance to discharge, and pain was scored on a visual analog scale. This is a single-participant report (n = 1). Inferential tests such as the t-test, analysis of variance, chi-square tests, or p-values were therefore avoided on purpose. Hypothesis testing on one subject violates the assumptions of sampling distributions and variance homogeneity. The analysis was descriptive and longitudinal.

2.8. Ethics and consent

The work followed the ethical principles of the Declaration of Helsinki (64th WMA General Assembly, Fortaleza, Brazil, 2013),³³ the International Council for Harmonisation (ICH) Good Clinical Practice guidelines, and institutional research policies. The Central Medical & Health Committee (CMHC) Ethics Committee, Indonesia, granted formal ethical review and expedited approval (Protocol Approval Number: 2025/0842). Delivery was urgent. Premature rupture of membranes and confirmed intrauterine fetal demise demanded emergency surgery. Even so, a comprehensive written informed consent procedure was completed. The patient and her legal surrogate were briefed on the high-risk hemodynamic setting, the reasons for invasive arterial and central venous monitoring, the indications for graded epidural anesthesia compared with alternative modalities, the collection of serial hemodynamic and laboratory parameters, and the scientific dissemination of anonymized clinical data, echocardiographic images, and trajectory models.

Permission was documented for permanent de-identification and anonymization of all medical records. No identifying biometric features or private identifiers were disclosed.

3. Results

3.1. Presentation and baseline findings

The baseline demographic, anthropometric, clinical presentation, and airway risk profile of the patient is detailed in Table 2. The clinical chronology is shown in Table 1 and Figure 1. The 28-year-old primigravida presented at 37 weeks and 2 days of gestation with a documented body mass index of 24.2 kg/m² and body surface area of 1.62 m². Her medical history was notable for an uncorrected 4.8 cm secundum atrial septal defect diagnosed in early adulthood (age 19). Functional compensation in NYHA class II was preserved until acute presentation with spontaneous premature rupture of membranes. Non-stress cardiotocography and bedside obstetric ultrasonography confirmed intrauterine fetal

demise. She was anxious and mildly tachypneic (respiratory rate 20 breaths/min). Preoperative baseline resting blood pressure was 118/65 mmHg, with a calculated mean arterial pressure of 83.0 mmHg. Resting heart rate was 78.0 beats/min, central venous pressure 8.0 mmHg, and peripheral arterial oxygen saturation (SaO₂) 98% on ambient air. Preoperative laboratory investigations revealed stable hemoglobin (11.2 g/dL), hematocrit (33.8%), and a normal platelet count (215 × 10³ /μL). Renal function was preserved (blood urea nitrogen 12.4 mg/dL, serum creatinine 0.68 mg/dL). On ambient air, arterial blood gas analysis showed pH 7.41, partial pressure of arterial carbon dioxide (PaCO₂) 31.8 mmHg, partial pressure of arterial oxygen (PaO₂) 94.5 mmHg, bicarbonate (HCO₃⁻) 20.2 mmol/L, base excess -3.2 mmol/L, and blood lactate 1.1 mmol/L. These values indicate the compensated respiratory alkalosis expected in late gestation, without tissue hypoperfusion. Airway examination revealed Mallampati class II with a thyromental distance of 6.0 cm, inter-incisor distance of 4.5 cm, and unrestricted cervical mobility.

Table 2. Baseline demographic, clinical, cardiopulmonary, and obstetric risk stratification profile.

Clinical Domain	Baseline Metric / Clinical Finding	Physiological & Diagnostic Significance
Maternal Age and Parity	28 years / Primigravida (G1P0A0)	Peak reproductive age; first cardiovascular challenge of full-term gestation
Gestational Age	37 weeks + 2 days (Term pregnancy)	Maximum physiological plasma volume expansion (+45%) and cardiac output (+40%)
Presenting Diagnosis	Premature rupture of membranes (PROM) with intrauterine fetal demise (IUFD)	Urgent obstetric indication for surgical delivery; high emotional and autonomic stress
Primary Cardiac Defect	Large secundum atrial septal defect (3.2–4.8 cm diameter)	Low-resistance interatrial communication driving chronic left-to-right shunt
Pulmonary Hemodynamics	Estimated systolic PAP 50.2 mmHg (TTE TR jet)	Secondary moderate precapillary pulmonary hypertension (normal sPAP <30 mmHg)
Ventricular Morphology	Severe right atrial and right ventricular dilatation	Eccentric RV remodeling from chronic volume overload; reduced diastolic reserve
Ventricular Interdependence	Interventricular septal flattening ('D-shaped' left ventricle)	Mechanical compression of LV cavity by high-pressure/distended RV, impairing LV filling
Valvular Function	Moderate functional tricuspid regurgitation (annular dilatation)	Secondary to severe RV chamber remodeling; preserved intrinsic valve leaflets
Left Ventricular Function	Preserved LV ejection fraction (LVEF 60%)	Maintained intrinsic LV contractility; cardiac output limited primarily by preload
Baseline Hemodynamics	Blood pressure 118/65 mmHg (MAP ~83 mmHg), HR 78 beats/min	Normotensive baseline; preserved systemic vascular resistance prior to block
Baseline Filling Pressure	Central venous pressure 8.0 mmHg	Optimal resting preload for compliant dilated RV without systemic congestion
Preoperative Blood Gas	pH 7.41, PaCO ₂ 31.8 mmHg, PaO ₂ 94.5 mmHg, HCO ₃ ⁻ 20.2 mmol/L, SaO ₂ 98%	Mild compensated chronic gestational respiratory alkalosis; adequate oxygenation
Preoperative Hemoglobin	11.2 g/dL (Hematocrit 33.8%)	Normal physiological dilutional anemia of late pregnancy; preserved O ₂ delivery
WHO Maternal Risk Class	Modified WHO Class IV (Prohibitive pregnancy risk)	Extremely high risk of maternal cardiac decompensation, RV failure, and mortality

Notes: ASD, atrial septal defect; IUFD, intrauterine fetal demise; LV, left ventricle; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; PAP, pulmonary artery pressure; PROM, premature rupture of membranes; RV, right ventricle; sPAP, systolic pulmonary artery pressure; TR, tricuspid regurgitation; TTE, transthoracic echocardiography; WHO, World Health Organization.

3.2. Diagnostic findings

Quantitative echocardiographic morphological and Doppler hemodynamic parameters are compiled in Table 3, with illustrative echocardiographic recordings presented in Figure 2. As detailed in Table 3 and shown in the upper panel of Figure 2, the primary anatomical defect comprised a large secundum atrial septal defect measuring 3.2 to 4.8 cm in maximal diameter, creating an unrestrictive interatrial communication. Color-flow Doppler interrogation confirmed a persistent left-to-right interatrial shunt from the left atrium into the right atrium. Flow across the low-resistance interatrial septum was prominent. The right atrial volume index was severely enlarged at 48.5 mL/m² (normal range as printed: 16.0–

28.0 mL/m², representing a +73.2% expansion above the upper normal limit). Right ventricular basal diameter was markedly dilated at 46.2 mm (normal range as printed: 25.0–35.0 mm, +32.0% above upper normal limit). Right ventricular chamber enlargement resulted in functional tricuspid annular dilatation. Tricuspid regurgitation was moderate (grade II/IV). Continuous-wave Doppler of the tricuspid regurgitant jet demonstrated a peak velocity of 3.25 m/s, which gives a trans-tricuspid systolic pressure gradient of 42.2 mmHg. Incorporating an estimated right atrial pressure of 8.0 mmHg (derived from an inferior vena cava diameter of 18.2 mm with preserved >50% inspiratory collapsibility), the calculated systolic pulmonary artery pressure (sPAP) was 50.2 mmHg,

consistent with moderate secondary pulmonary hypertension.

Table 3. Quantitative echocardiographic morphological and Doppler hemodynamic parameters.

Echocardiographic Parameter	Patient Value	Reference Interval	Analytical Deviation & Significance
ASD Defect Diameter (cm)	3.2 – 4.8	0 (Intact septum)	Markedly uncorrected defect; massive non-restrictive interatrial communication
Interatrial Shunt Direction	Left-to-right (Doppler confirmed)	None	Persistent high-volume systemic-to-pulmonary recirculation
Systolic Pulmonary Artery Pressure (mmHg)	50.2	< 30.0	Moderately elevated sPAP; reflects pulmonary vascular hyperkinetic remodeling
Tricuspid Regurgitation Severity	Moderate (Grade II/IV)	None to trivial	Functional regurgitation due to tricuspid annular stretching
Right Atrial Volume Index (mL/m ²)	48.5	16.0 – 28.0	Severe dilatation resulting from chronic interatrial volume shunting (+73.2% vs upper normal)
Right Ventricular Basal Diameter (mm)	46.2	25.0 – 35.0	Severe basal RV enlargement; eccentric remodeling (+32.0% vs upper normal)
Interventricular Septal Curvature	Septal flattening in diastole/systole	Convex toward RV	Classic 'D-shaped' LV configuration confirming RV pressure and volume overload
LV End-Diastolic Dimension (mm)	39.5	38.0 – 52.0	Low-normal LV cavity size due to septal displacement and preload restriction
Left Ventricular Ejection Fraction (%)	60.0	52.0 – 72.0	Normal intrinsic myocardial contractility; unaffected by left-to-right shunt
Inferior Vena Cava Diameter (mm)	18.2	12.0 – 21.0	Preserved respiratory collapse (>50%); estimated right atrial pressure 5–10 mmHg

Notes: ASD, atrial septal defect; LV, left ventricle; RV, right ventricle; sPAP, systolic pulmonary artery pressure.

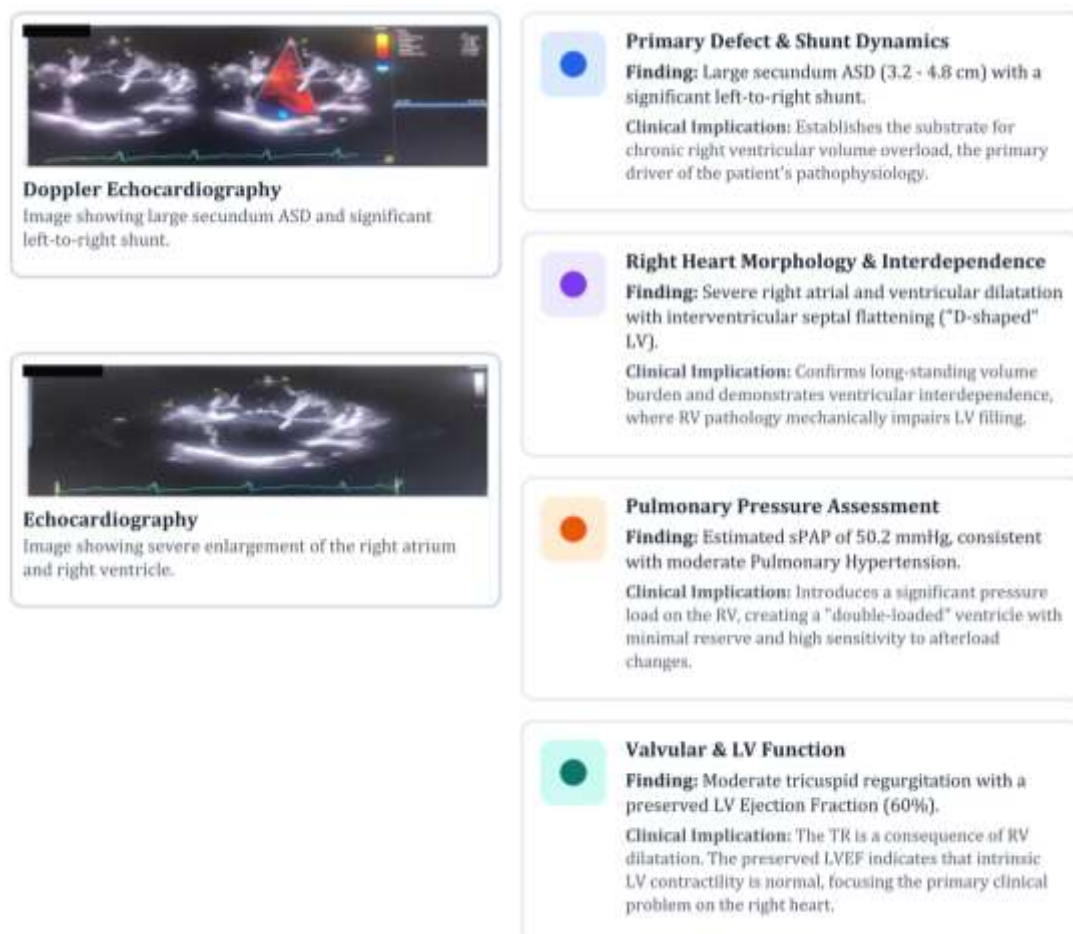


Figure 2. Quantitative preoperative transthoracic echocardiographic and Doppler assessment. Top left: color-flow Doppler imaging demonstrating large secundum atrial septal defect (3.2–4.8 cm diameter) with prominent left-to-right interatrial shunting. Bottom left: two-dimensional echocardiography showing severe right atrial and right ventricular dilatation with marked interventricular septal flattening producing a characteristic 'D-shaped' left ventricle. On-screen date and time stamps were removed from the images. ASD, atrial septal defect; LV, left ventricle; LVEF, left ventricular ejection fraction; RV, right ventricle; sPAP, systolic pulmonary artery pressure; TR, tricuspid regurgitation.

Echocardiographic examination also showed marked mechanical ventricular interdependence, as illustrated in the lower panel of Figure 2. The dilated right ventricle pressed on the left ventricle and flattened the

interventricular septum in both diastole and systole, and the left ventricular cavity took the classic 'D' shape on short-axis views. Left ventricular end-diastolic dimension was low-normal (39.5 mm). Intrinsic left ventricular

myocardial contractility was preserved, with a left ventricular ejection fraction of 60.0% and no segmental wall motion abnormalities.

3.3. Treatment course and outcome

The institutional safety and multidisciplinary briefing framework established prior to anesthesia is detailed in Figure 3. The intraoperative procedural sequence and corresponding longitudinal hemodynamic parameters are tabulated in Table 4 and visualized in Figure 4. Incremental

epidural administration of 0.5% isobaric levobupivacaine (cumulative induction dose 17.0 mL, 85 mg) delivered in fractional aliquots over 15 minutes achieved smooth, segmental ascending dermatomal blockade. Cold ice discrimination mapped bilateral sensory block reaching T10 at 10 minutes and T8 at 15 minutes. The target T6 sensory level was confirmed at 25 minutes. Bilateral motor block was restricted to Bromage grade 1.



Figure 3. Pre-anesthetic multidisciplinary safety governance and emergency contingency framework. Comprehensive operational structure comprising multidisciplinary briefing protocols, quantifiable hemodynamic stability goals, invasive radial arterial and central venous monitoring, and pre-positioned pharmacological contingency pathways for acute right ventricular failure or systemic vasoplegia. CVICU, cardiovascular intensive care unit; CVP, central venous pressure; iNO, inhaled nitric oxide; MAP, mean arterial pressure; RV, right ventricle.

Table 4. Longitudinal intraoperative hemodynamic trajectories and percentage change across surgical epochs.

Surgical Epoch / Milestone	Elapsed Time (min)	MAP (mmHg)	MAP Δ%	Heart Rate (beats/min)	HR Δ%	CVP (mmHg)	CVP Δ%
Baseline (Pre-induction)	T = 0	83.0	Reference (0.0%)	78.0	Reference (0.0%)	8.0	Reference (0.0%)
Post-Induction Nadir (T6 Block)	T = 25	72.0	-13.3%	72.0	-7.7%	9.0	+12.5%
Surgical Incision	T = 35	73.0	-12.0%	79.0	+1.3%	9.0	+12.5%
Uterine Incision & Delivery	T = 45	75.0	-9.6%	82.0	+5.1%	10.0	+25.0%
End of Surgery	T = 90	71.0	-14.5%	74.0	-5.1%	8.0	0.0%

Notes: All parameters derived from continuous indwelling radial arterial line (MAP) and internal jugular central line (CVP). Percentage delta: $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Targeted stability window: MAP within $\pm 20\%$ of baseline (66.4–99.6 mmHg). No systemic vasopressor or inotropic infusions required throughout the procedure. CVP, central venous pressure; HR, heart rate; MAP, mean arterial pressure; T, minutes from the start of observation.



Figure 4. Intraoperative graded epidural titration kinetics and continuous longitudinal hemodynamic stability trajectories. Top: procedural chronology of incremental 0.5% levobupivacaine administration achieving surgical T6 sensory blockade over 25 minutes. Bottom: longitudinal tracking of mean arterial pressure (blue line), heart rate (pink line), and central venous pressure (teal line) across surgical epochs, with values from Table 4 and the pre-defined $\pm 20\%$ MAP stability zone (shaded green band). CVP, central venous pressure; HR, heart rate; MAP, mean arterial pressure.

Continuous beat-to-beat radial arterial waveform tracking documented stable hemodynamics throughout all surgical phases. All parameters remained within the pre-established $\pm 20\%$ target corridor, as shown in Figure 4 and detailed in Table 4. At baseline (T = 0), pre-induction MAP was 83.0 mmHg, heart rate was 78.0 beats/min, and CVP was 8.0 mmHg. Following completion of the graded epidural titration at T = 25 minutes, MAP exhibited a gradual, controlled descent to its nadir of 72.0 mmHg ($\Delta -13.3\%$ relative to baseline). This occurred without sudden hypotension or autonomic instability. Heart rate decreased slightly to 72.0 beats/min ($\Delta -7.7\%$), while CVP rose slightly to 9.0 mmHg ($\Delta +12.5\%$).

At surgical skin incision (T = 35 min), MAP was 73.0 mmHg ($\Delta -12.0\%$), heart rate was 79.0 beats/min ($\Delta +1.3\%$), and CVP remained at 9.0 mmHg ($\Delta +12.5\%$). There was no hemodynamic response to incision. Following hysterotomy, delivery of the demised infant occurred at T = 45 minutes. There were no surgical complications. At the delivery epoch, MAP was 75.0 mmHg ($\Delta -9.6\%$), heart rate was 82.0 beats/min ($\Delta +5.1\%$), and CVP exhibited a modest, transient increase to 10.0 mmHg ($\Delta +25.0\%$). The right ventricle tolerated this change without signs of acute volume overload, pulmonary congestion, or right-sided decompensation. Oxytocin infusion (10 IU in 500 mL normal saline at 0.03 IU/min) achieved firm uterine contraction without requiring second-line uterotonics.

Total estimated intraoperative blood loss was 350 mL. Surgery concluded at T = 90 minutes. At completion of surgery, MAP was 71.0 mmHg ($\Delta -14.5\%$), heart rate was 74.0 beats/min ($\Delta -5.1\%$), and CVP returned to baseline at 8.0 mmHg ($\Delta 0.0\%$). At no point during the operative procedure was vasopressor rescue (phenylephrine), inotropic support (dobutamine/milrinone), or pulmonary vasodilator therapy (inhaled nitric oxide) required. Maximal MAP excursion was -14.5% . Maximal HR excursions were -7.7% and $+5.1\%$. All stayed within the pre-specified $\pm 20\%$ corridor.

Serial arterial blood gas, metabolic parameters, and hematological evolution from preoperative baseline through CVICU surveillance to hospital discharge are compiled in Table 5, and the recovery milestones are outlined in Figure 5. During the first 24 hours (postoperative day 0), continuous invasive monitoring showed sustained hemodynamic stability. MAP stayed between 72 and 80 mmHg and CVP between 7 and 9 mmHg. Serial arterial blood gas analysis at post-delivery intraoperative tracking and CVICU day 1 demonstrated preserved acid-base equilibrium (pH 7.39 to 7.40, PaCO₂ 33.2 to 34.5 mmHg, PaO₂ 112.0 to 98.4 mmHg, HCO₃⁻ 21.0 to 22.1 mmol/L, base excess -2.8 to -1.5 mmol/L, and lactate 1.4 to 1.0 mmol/L). Urine output remained robust at 1.2 to 1.5 mL/kg/h without diuretic therapy.

Table 5. Serial arterial blood gas, metabolic, and hematological evolution from preoperative baseline to discharge.

Physiological Biomarker	Baseline (Preop)	Intraop (Post-Delivery)	ICU Day 1	Discharge (Day 4)	Change (Δ%)	Reference Interval
Arterial pH	7.41	7.39	7.40	7.42	+0.1%	7.35 – 7.45
PaCO ₂ (mmHg)	31.8	33.2	34.5	36.2	+13.8%	35.0 – 45.0 (Pregnancy: 28–32)
PaO ₂ (mmHg)	94.5	112.0	98.4	95.0	+0.5%	80.0 – 100.0
Arterial SaO ₂ (%)	98.0	99.5	98.5	98.0	0.0%	95.0 – 100.0
Serum Bicarbonate (mmol/L)	20.2	21.0	22.1	23.5	+16.3%	22.0 – 26.0 (Pregnancy: 18–22)
Base Excess (mmol/L)	-3.2	-2.8	-1.5	-0.2	+93.8%	-2.0 – +2.0
Blood Lactate (mmol/L)	1.1	1.4	1.0	0.8	-27.3%	0.5 – 1.6
Hemoglobin (g/dL)	11.2	10.4	10.1	10.5	-6.2%	11.0 – 14.5
Hematocrit (%)	33.8	31.5	30.8	32.0	-5.3%	33.0 – 44.0
Estimated Blood Loss (mL)	—	350	—	—	—	< 500 mL for Cesarean Delivery
Urine Output (mL/kg/h)	1.2	1.5	1.3	1.1	-8.3%	> 0.5 mL/kg/h

Notes: ABG, arterial blood gas; PaCO₂, partial pressure of arterial carbon dioxide; PaO₂, partial pressure of arterial oxygen; SaO₂, arterial oxygen saturation.

Postoperative Management & Recovery Milestones



Figure 5. Structured postoperative recovery course and intensive care de-escalation milestones. Sequential daily management pathway from intensive hemodynamic surveillance on postoperative day 0, early chair mobilization on day 1, epidural catheter removal and transition to oral analgesia on day 2, independent functional ambulation on day 3, to safe hospital discharge on day 4. CVICU, cardiovascular intensive care unit.

On postoperative day 1, early mobilization was initiated, transitioning the patient to a bedside chair, and oral fluid intake was advanced. On postoperative day 2, care de-escalation was achieved: the epidural catheter was removed following normal coagulation testing, analgesia was transitioned to oral paracetamol and tramadol, and invasive arterial and central venous catheters were discontinued. On postoperative day 3, the patient walked independently. Pain scores were below 2/10 on the visual analog scale, and cardiovascular stability was intact. Discharge evaluation on postoperative day 4 documented normal physical findings, blood pressure 115/70 mmHg, resting heart rate 72 beats/min, SaO₂ 99% on ambient air, hemoglobin 10.5 g/dL, hematocrit 32.0%, and normal metabolic panels (pH 7.42, PaCO₂ 36.2 mmHg, PaO₂ 95.0 mmHg, HCO₃⁻ 23.5 mmol/L, base excess -0.2 mmol/L, lactate 0.8 mmol/L). The patient was discharged home in stable clinical condition with outpatient cardiology follow-up scheduled for definitive transcatheter atrial septal defect closure.

4. Discussion

4.1. Principal lesson of the case

In this parturient with a large uncorrected atrial septal defect and echocardiographic pulmonary hypertension, slow incremental titration of epidural levobupivacaine to a T6 block was accompanied by stable MAP, heart rate, and CVP without vasoactive support, and by an uneventful recovery to discharge on day 4. The favorable outcome is compatible with a goal-directed anesthetic strategy built on a detailed analysis of maternal-fetal cardiovascular pathophysiology.²⁰

4.2. Comparison with the published literature

Published experience with pregnancy and an unrepaired ASD consists mainly of single-center series, case reports, and conference abstracts.^{12,34} Pregnancy is generally tolerated in the absence of pulmonary hypertension, while severe pulmonary hypertension carries high maternal and fetal morbidity and mortality.¹¹ Case reports describe the same range of problems that our patient faced: sudden heart failure with pulmonary hypertension,²⁶ pulmonary hypertension with advanced pregnancy,³⁵ hemoptysis,³⁶ supraventricular tachycardia,³⁷ and percutaneous closure of a very large defect during pregnancy.^{38,39} Hemodynamic features of the third trimester in women with ASD have been reviewed,⁴⁰ and the diagnosis and management of ASD with pulmonary hypertension in pregnancy have been summarized.⁴¹ For anesthesia, published reports describe epidural anesthesia for cesarean section in ASD with pulmonary hypertension²⁵ and neuraxial or general techniques in pulmonary hypertension of other causes.^{42–44} In parturients with pulmonary arterial hypertension, general anesthesia has been associated with higher maternal mortality,²⁴ and registry data show high maternal and fetal risk in pulmonary hypertension.⁴⁵ Our case adds quantitative beat-to-beat arterial and central venous data across five time points and a full recovery pathway for a titrated epidural in this setting.

To place the observed trajectories in context, the technique was compared with the main alternatives, single-shot spinal anesthesia and general endotracheal anesthesia, across six physiological domains (Table 6 and Figure 6). That comparison is drawn from published physiology and was not observed in this patient; neither alternative was used.

Table 6. Comparison of the technique used with the main alternatives: observed course (graded epidural) versus expected effects drawn from published physiology (spinal and general anesthesia).

Domain	Graded Epidural Blockade (performed; observed in this patient)	Single-Shot Spinal Anesthesia (not used; expected from the literature)	General Endotracheal Anesthesia (not used; expected from the literature)
Onset kinetics and titratability	Gradual segmental onset over 25 min; titratable aliquot dosing	Rapid onset (< 5 min) expected; fixed single bolus that cannot be titrated	Rapid onset; induction-related vasodilation within 1–2 min expected
SVR and vascular capacitance	MAP kept within 14.5% of baseline; SVR not measured	Larger and faster fall in SVR expected (by 40%–50% in the authors' estimate); marked venodilation	Myocardial depression (propofol, volatiles) and vasodilation expected
ASD shunt dynamics	No desaturation (SaO ₂ 98–99.5%); shunt direction not measured	Fall in SVR expected to increase the left-to-right shunt or, if PVR is high, to reverse it	Hypoxia, hypercarbia, and PEEP may raise PVR and favor right-to-left shunting
Pulmonary vascular resistance	No hemodynamic response to incision; PVR not measured	Systemic hypotension may impair RV coronary perfusion	Positive-pressure ventilation and PEEP compress capillaries and may raise PVR
Right ventricular preload	CVP stable at 8–10 mmHg	Sudden fall in venous return may cause acute RV ischemia	Positive intrathoracic pressure may reduce venous return
Overall course and expected risk	Uneventful surgery, no vasopressors, discharge on day 4	Higher risk of acute RV failure and cardiac arrest expected	Higher risk of post-induction arrest, instability, and prolonged ICU stay expected

Notes: The spinal and general anesthesia columns describe expected effects based on the cited literature^{20,21,23,24} and the authors' reasoning; neither technique was used in this patient. ASD, atrial septal defect; CVP, central venous pressure; MAP, mean arterial pressure; PEEP, positive end-expiratory pressure; PVR, pulmonary vascular resistance; RV, right ventricle; SVR, systemic vascular resistance.

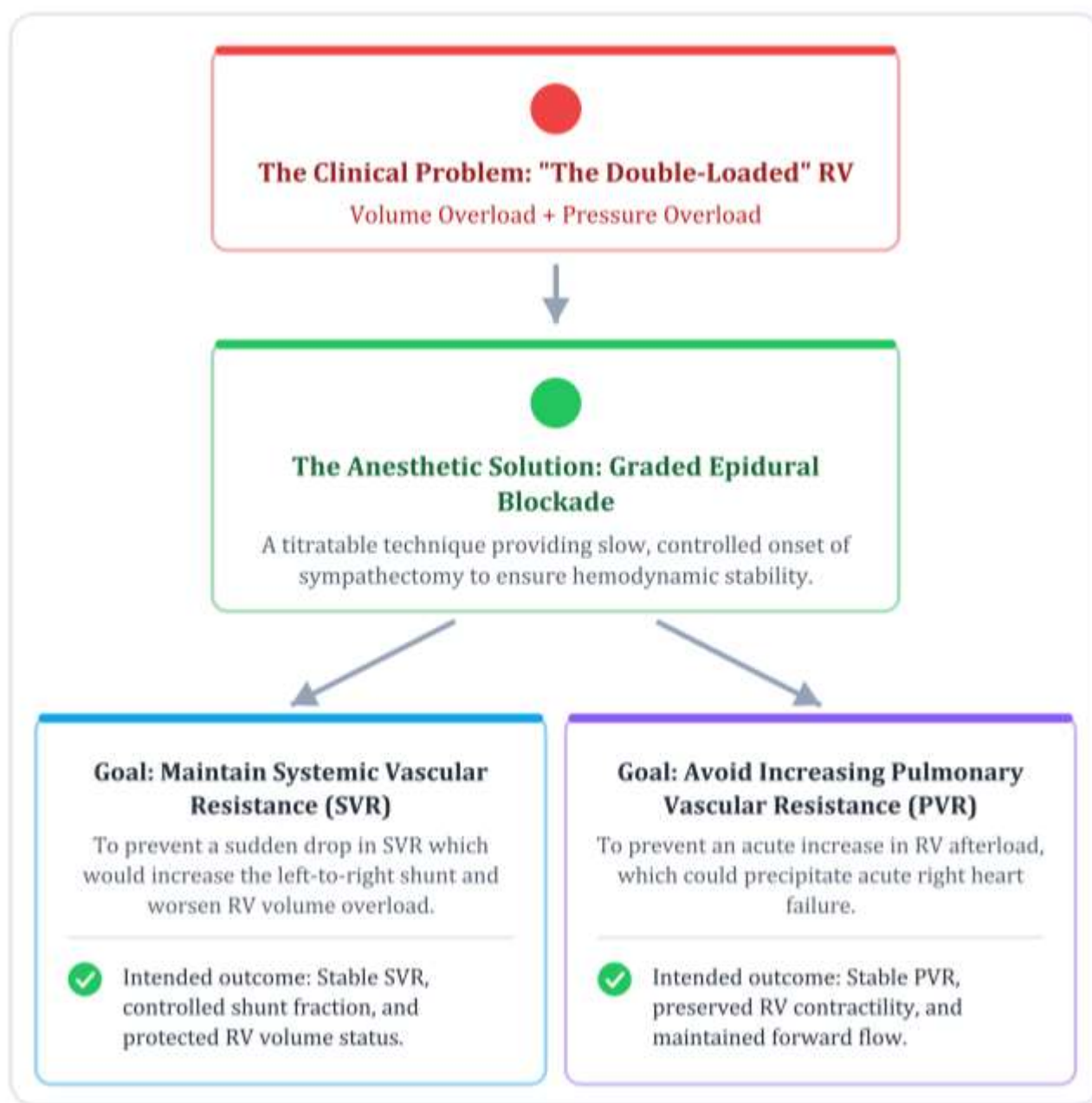


Figure 6. Pathophysiology-based anesthetic management framework for the double-loaded right ventricle. Schematic rationale connecting the dual challenges of volume overload and pressure overload to the therapeutic goal of graded epidural blockade, which aims to maintain systemic vascular resistance (SVR) and prevent elevations in pulmonary vascular resistance (PVR). The outcomes shown are the intended physiological goals; SVR and PVR were not measured in this patient. RV, right ventricle.

4.3. Pathophysiological or mechanistic interpretation

The baseline substrate was an uncorrected 4.8 cm secundum atrial septal defect with moderate secondary pulmonary hypertension (estimated sPAP 50.2 mmHg). The right ventricle was 'double-loaded'. It faced chronic

volume overload and progressive pressure overload at once.^{10,18} Managing peripartum anesthesia depends on understanding how these two loading states differ in their cellular, structural, and mechanical consequences.¹⁴ The proposed cascade from the anatomical lesion to the observed hemodynamic course is summarized in Figure 7.

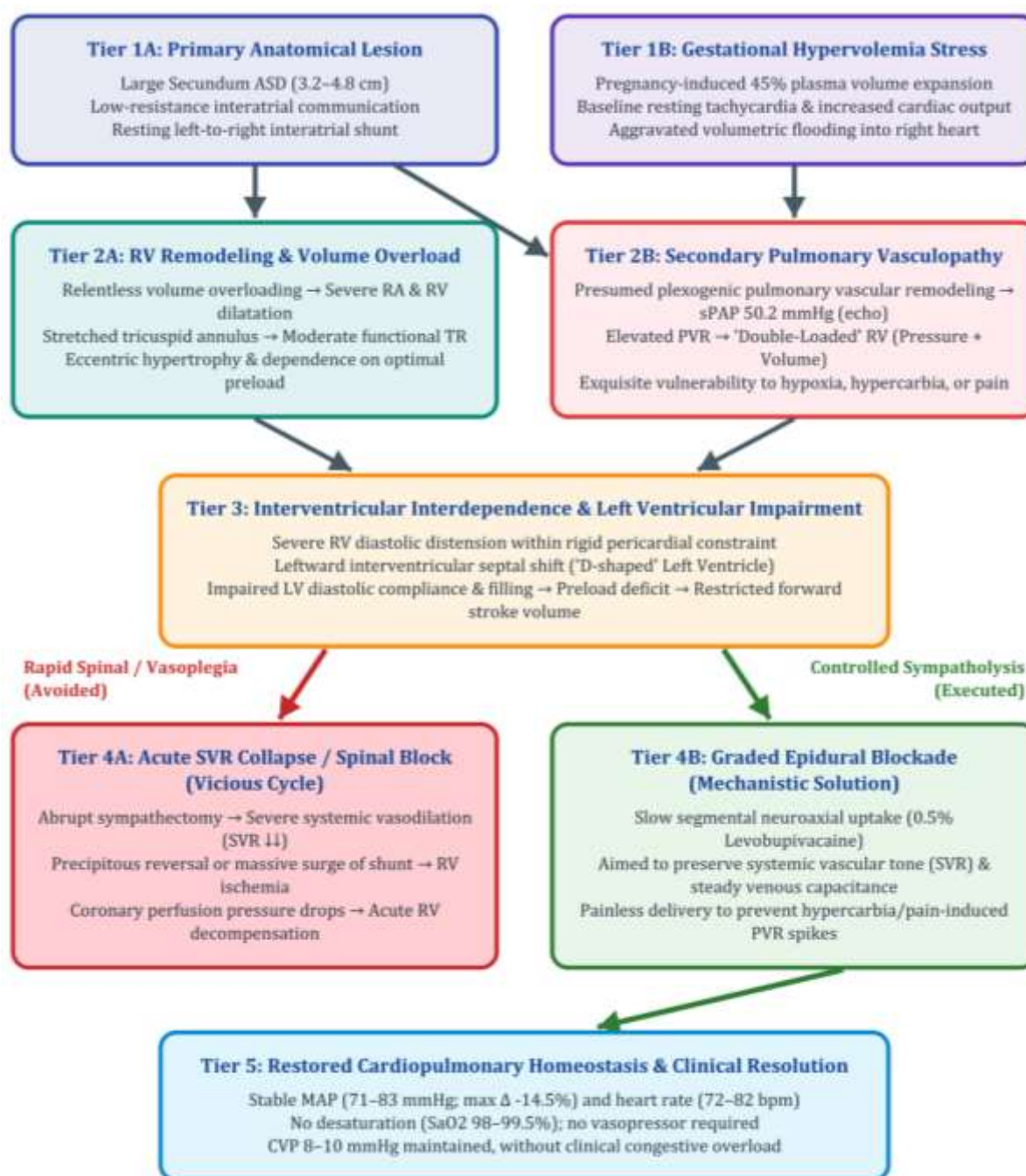


Figure 7. Multi-tier pathophysiological and hemodynamic cascade of biventricular interdependence. Tier 1 primary anatomical defect and gestational hypervolemia, Tier 2 right ventricular remodeling and pulmonary vasculopathy, Tier 3 mechanical ventricular interdependence and left ventricular preload restriction, Tier 4 acute peripartum vasoplegic threat versus graded epidural, Tier 5 observed hemodynamic and clinical resolution. Mechanisms are inferred from the literature and from the echocardiographic findings; they were not measured directly. ASD, atrial septal defect; CVP, central venous pressure; MAP, mean arterial pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricle; SVR, systemic vascular resistance; TR, tricuspid regurgitation.

For many years, left-to-right interatrial shunting, driven by the low compliance of the right heart relative to the left, recirculated large volumes of oxygenated blood through the right atrium and right ventricle, with eccentric hypertrophy and chamber dilatation as the result (right atrial volume index 48.5 mL/m^2 and right ventricular basal diameter 46.2 mm).¹⁰ Eccentric remodeling at first preserves forward stroke volume by shifting the Frank-Starling relationship. The price is dependence on a narrow optimal preload range for ejection.¹⁸ Over the same years, high-volume shear stress on the pulmonary endothelium is thought to lead to intimal proliferation, medial hypertrophy, and plexogenic vasculopathy, which produce precapillary pulmonary hypertension.^{13,14} The added

pressure overload imposes concentric remodeling stress on an already dilated chamber, so RV compliance falls and diastolic filling reserve is reduced.¹⁸ The ventricle was caught in a hemodynamic trap. A sudden fall in venous return would cause ischemic collapse through inadequate preload. A sudden rise in pulmonary vascular resistance, or excessive fluid loading, would raise wall tension and trigger acute right ventricular decompensation.^{21,46}

Echocardiography showed marked septal flattening and a classic 'D-shaped' left ventricular cavity. Ventricular interdependence was therefore the likely determinant of systemic hemodynamic stability in this patient.¹⁸ The ventricles share a septum, reciprocal myofibers, and a non-compliant fibrous pericardial sac, so a change in the

volume or pressure of one chamber acts at once on the other. In our patient, chronic RV volume and pressure overload raised right ventricular end-diastolic pressure and shifted the septum leftward in both diastole and systole. That shift restricted left ventricular diastolic filling and lowered left ventricular end-diastolic dimension (39.5 mm), although intrinsic contractility was preserved (ejection fraction 60.0%). Filling was the problem. Pump function was not.¹⁸

Left ventricular stroke volume and systemic arterial pressure were therefore tied to right ventricular forward performance. Any acute event that dilates the right ventricle, for example a surge in pulmonary vascular resistance from hypoxia, hypercarbia, or surgical pain, pushes the septum further left. This chokes off left ventricular filling and can cause sudden systemic hypotension.^{18,21} The opposite problem arises if systemic vascular resistance falls abruptly. The interatrial pressure gradient widens and the left-to-right shunt increases, which delivers an unmanageable volume load to the right heart. If pulmonary vascular resistance is high, the shunt may reverse from right to left instead. The result is arterial desaturation and, in the worst case, refractory cardiac arrest.^{13,22} Preserving systemic vascular resistance and preventing any rise in pulmonary vascular resistance were therefore the main hemodynamic priorities. In this patient, SVR, shunt direction, pulmonary vascular resistance, and the volume of any autotransfusion were not measured; they are inferred from MAP, heart rate, CVP, and SaO₂, which stayed at 98% to 99.5% throughout.

4.4. Implications for practice

The choice of anesthetic technique for cesarean delivery required a careful comparison of risks and benefits against the alternatives, as set out in Table 6 and illustrated in Figure 6. Single-shot spinal anesthesia was judged unacceptable. It causes rapid, dense, and uncontrollable sympathetic blockade.^{20,31} Subarachnoid local anesthetic produces arterial and venous vasodilation within minutes. Systemic vascular resistance falls by 40% to 50%, and venodilation sharply reduces central venous return. In a patient with an uncorrected atrial septal defect and pulmonary hypertension, this vasoplegia is dangerous in two ways. First, the abrupt fall in systemic resistance increases the left-to-right shunt and floods the strained pulmonary vascular bed. Second, aortic root pressure drops below RV intracavitary systolic pressure. RV coronary hypoperfusion, myocardial ischemia, and rapid circulatory collapse follow.^{21,46}

General endotracheal anesthesia gives secure airway control. It also carries serious hemodynamic hazards, which made it unsuitable as the primary technique.^{21,24} Induction can cause myocardial depression and vasodilation, and direct laryngoscopy and intubation trigger sympathetic surges that raise pulmonary arterial pressure. Positive-pressure ventilation raises mean intrathoracic pressure and compresses the pulmonary alveolar microvessels, which increases pulmonary vascular resistance, impedes RV ejection, and also impedes venous return.²³ Extubation at the end of surgery adds more risk. Coughing, hypertension, and hypoventilation with hypercarbia and acidosis are all possible, and the last two are potent pulmonary vasoconstrictors.

Graded epidural anesthesia, by contrast, gave temporal control over the sympathetic blockade. We titrated small aliquots of 0.5% isobaric levobupivacaine (cumulative induction dose 17.0 mL, 85 mg) through an indwelling

multi-orifice lumbar catheter over 15 minutes. The result was a slow, segmental ascending block that reached the target T6 sensory level over 25 minutes. This gradual onset allowed compensatory autoregulation to maintain systemic vascular resistance and venous return, although this was not measured. Levobupivacaine was chosen for its safety profile as the pure S(-)-enantiomer of bupivacaine. It causes less myocardial depression and has lower arrhythmogenic potential.^{32,47} It also gives dense sensory block with little motor impairment, so the venous muscle pump of the legs is preserved. Mean arterial pressure stayed in a narrow corridor (nadir 72.0 mmHg, Δ -13.3% relative to baseline), and heart rate remained stable (72 to 82 beats/min) without vasopressor infusions.

Invasive radial arterial and central venous monitoring established before induction was valuable for real-time management. Beat-to-beat arterial waveforms gave immediate feedback during neuroaxial titration, skin incision, and delivery of the infant, and they removed the measurement lag of non-invasive oscillometric cuffs.^{20,31} CVP has limits as an absolute volume measure.⁴⁸ It still served as a useful directional indicator of RV filling. A stable CVP trajectory (8.0 to 10.0 mmHg) showed that venous return was maintained without congestive right-sided heart failure. The small rise to 10.0 mmHg at delivery (Δ +25.0%) was compatible with the expected autotransfusion of 300 to 500 mL from the contracting uterus, which the right heart tolerated without decompensation.¹⁷

The pharmacological strategy was equally deliberate. A phenylephrine infusion (50 μ g/mL) was ready as an immediate countermeasure for peripheral vasodilation. Phenylephrine is a pure α -1 adrenergic agonist. It restores systemic vascular resistance and aortic root pressure without increasing heart rate, thereby preserving RV coronary perfusion and stabilizing shunt dynamics.^{29,49,50} Our protocol also excluded carboprost tromethamine and methylergonovine maleate because of their recognized tendency to cause acute pulmonary vasoconstriction and bronchospasm.³⁰ We relied instead on a slow, dilute oxytocin infusion (0.03 IU/min), which secured effective uterine hemostasis (estimated blood loss 350 mL) without cardiovascular perturbation.

The period from 48 to 72 hours after delivery is a time of acute cardiovascular vulnerability in cardiac parturients.^{51,52} Relief of caval compression and mobilization of third-space gestational fluid into the vascular compartment can raise effective circulating volume by 1,000 mL. This may precipitate delayed RV decompensation and pulmonary edema. Our patient went directly to the Cardiovascular Intensive Care Unit with invasive monitoring maintained. Patient-controlled epidural analgesia (0.1% levobupivacaine with fentanyl 2 μ g/mL) helped to limit the postpartum neurohumoral stress response. Careful fluid management kept her euvolemic to slightly negative in balance, which prevented congestive RV volume overload. Early ambulation with pneumatic compression limited venous stasis. Catheter removal on postoperative day 2 was uneventful, and discharge on postoperative day 4 shows that this multidisciplinary protocol worked in this patient.

4.5. Strengths

This report has several strengths. Invasive hemodynamic data were captured continuously across all surgical milestones. Transthoracic echocardiography documented biventricular morphology and shunt dynamics

quantitatively. The timeline, dosing steps, sensory levels, and monitoring targets are documented in enough detail for another team to reproduce the approach, and a descriptive analysis suited to a single patient was used in place of inferential statistics.

4.6. Limitations

Several limitations need to be acknowledged. First, this is a single patient ($n = 1$). The findings reflect individual patient biology and cannot be generalized to all variants of congenital heart disease without caution. A single uncontrolled case cannot establish causation, prevalence, or comparative efficacy, and no alternative technique was applied. The fetus had died, so the report says nothing about fetal effects. Second, preoperative right heart catheterization was deferred because of the late-gestation emergency presentation and its procedural risks. We relied on Doppler echocardiographic estimates of pulmonary artery pressure, which may over- or underestimate true pulmonary vascular resistance compared with invasive thermodilution,^{14,53} and the type of pulmonary hypertension could not be defined hemodynamically. Third, continuous cardiac output monitoring (transesophageal echocardiography or pulmonary artery catheterization) was not used intraoperatively. We wished to keep invasiveness low in an awake patient under epidural anesthesia. SVR, shunt direction, and autotransfusion volume were therefore inferred and not measured. Fourth, the authors applied the Naranjo probability scale (score +8, 'probable' on the standard scale) and the Bradford Hill criteria to the relation between the technique and the stable course. These instruments were designed for adverse drug reactions and for population-level evidence, so they cannot establish causation in one patient and are reported here for transparency only.^{54,55} The follow-up after day 4 is not reported. Even so, beat-to-beat arterial and central venous tracking offers a reproducible framework for managing complex cardiac parturients undergoing cesarean delivery.

4.7. Recommendations for future work

A prospective multicenter case series or registry of parturients with large intracardiac shunts and pulmonary hypertension, with standardized hemodynamic monitoring (including cardiac output and echocardiography during the procedure) and documented neuraxial technique, would be needed to test whether graded epidural anesthesia gives better hemodynamic stability than single-shot spinal or general anesthesia.

5. Conclusion

In this parturient with a large uncorrected secundum atrial septal defect and echocardiographic pulmonary hypertension, a carefully titrated lumbar epidural with 0.5% levobupivacaine was accompanied by stable beat-to-beat mean arterial pressure, heart rate, and central venous pressure for emergency cesarean delivery. The maximum MAP change was -14.5% , within the $\pm 20\%$ corridor. No vasopressor infusion was needed. Arterial and central venous lines placed before induction, close multidisciplinary coordination, and vigilant postoperative intensive care surveillance were part of this course. The case suggests that graded epidural anesthesia can be considered for similar high-risk cardiac parturients where invasive monitoring and intensive care are available. It calls for confirmation in larger series.

6. Declarations

Ethical approval and consent to participate. The study protocol was reviewed and approved by CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2025/0842). Written informed consent was obtained from the patient and her legal surrogate for clinical participation and research data utilization.

Consent for publication. Written informed consent was obtained from the patient and her legal surrogate for the publication of this case report, including all associated anonymized clinical records, laboratory data, and echocardiographic images. A copy of the signed consent form is available for review by the editor on request.

Availability of data and materials. The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable academic request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. V.W. conceptualized the report, performed the anesthetic procedure, collected the clinical data, and wrote the original draft (Conceptualization, Investigation, Data curation, Writing – original draft). S.A.P. and B.N.P. supervised the anesthetic management, took part in critical data interpretation, and reviewed and edited the manuscript (Supervision, Formal analysis, Writing – review & editing). All authors read and approved the final manuscript.

Funding statement. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest (competing interests) statement. The authors declare that they have no competing financial or non-financial interests.

Acknowledgements. The authors thank the clinical and nursing staff of the Department of Anesthesiology and Intensive Therapy, the Department of Obstetrics and Gynecology, the Department of Cardiology and Vascular Medicine, and the Cardiovascular Intensive Care Unit at Dr. Moewardi Regional General Hospital, Surakarta, Indonesia, for their dedicated multidisciplinary care.

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