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Successful Use of Low-Dose Combined Spinal-Epidural Anesthesia for Cesarean Section in a Parturient with Eisenmenger Syndrome: A Case Report

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A B S T R A C T

Introduction: Eisenmenger syndrome (ES) in pregnancy is a catastrophic condition associated with maternal mortality rates of 30-50%. The profound physiological changes of pregnancy, particularly the decrease in systemic vascular resistance (SVR), exacerbate right-to-left (R-L) shunting, leading to severe hypoxemia and right ventricular failure. Anesthetic management is perilous, as both general and neuraxial anesthesia can precipitate hemodynamic collapse. **Case presentation:** We present the case of a 25-year-old G2P101Ab000 parturient at 32-34 weeks of gestation with ES secondary to a large secundum atrial septal defect and severe pulmonary hypertension. She presented for an urgent Cesarean section due to labor. A meticulous anesthetic plan was executed, centered on a low-dose Combined Spinal-Epidural (CSE) technique. This involved an intrathecal injection of 7.5 mg hyperbaric bupivacaine with 50 mcg fentanyl, followed by incremental epidural titration of 0.2% ropivacaine. Hemodynamic stability was proactively managed with inline infusions of phenylephrine and milrinone. The procedure was successful, maintaining stable maternal hemodynamics, SVR, and oxygen saturation. A healthy infant was delivered with APGAR scores of 7 and 8. The patient had an uncomplicated postoperative recovery. **Conclusion:** This case demonstrates that a carefully titrated, low-dose CSE technique, combined with invasive monitoring and proactive pharmacological support, can be a safe and effective strategy for Cesarean section in ES patients. This approach successfully navigates the hemodynamic dilemma by providing excellent analgesia while preventing a clinically significant drop in SVR.

1. Introduction

Eisenmenger syndrome (ES) represents the most severe form of pulmonary hypertension (PH), arising from a large, uncorrected systemic-to-pulmonary shunt that eventually leads to fixed, high pulmonary vascular resistance (PVR). It is a multisystemic disorder characterized by the classic triad of a large intracardiac or great-vessel defect, pulmonary arterial hypertension, and a reversed (right-to-left) or bidirectional shunt, resulting in systemic hypoxemia and cyanosis.[1] The defects most commonly associated with ES include ventricular septal defects (VSD), atrial septal defects (ASD), and patent ductus arteriosus (PDA). While the incidence of ES has decreased in developed nations due to early surgical correction of these defects, it remains a significant challenge in resource-limited settings and in adults with late diagnoses.[2]

The pathophysiology of ES is a progressive journey from a benign left-to-right (L-R) shunt to a life-threatening condition. Initially, the L-R shunt causes volume overload on the pulmonary circulation.[3] Over years, this chronic volume and pressure overload induces endothelial dysfunction, vascular smooth muscle proliferation, and plexiform lesions—a process known as pulmonary vascular remodeling. This remodeling leads to a progressive and ultimately fixed increase in PVR. As PVR approaches and then exceeds the systemic vascular resistance (SVR), the shunt reverses, becoming bidirectional or predominantly right-to-left (R-L). This R-L shunting allows deoxygenated blood to bypass the lungs and enter the systemic circulation, causing chronic hypoxemia, cyanosis, and erythrocytosis.[4]

Pregnancy in a patient with ES is a near-absolute contraindication and is considered one of the highest-risk medical conditions in obstetrics. Maternal mortality is reported to be between 30% and 50%, with some reports as high as 65% for Cesarean sections. Fetal and neonatal prognosis is equally grim, with high rates of spontaneous abortion, intrauterine growth restriction (IUGR), prematurity, and perinatal death.[5] The primary causes of maternal death are right heart failure, thromboembolism, hypovolemia, and sudden cardiovascular collapse.[5]

The catastrophic risk is a direct consequence of the collision between the fixed, high PVR of ES and the normal, profound physiological adaptations of pregnancy. During a normal pregnancy, blood volume increases by 40-50%, and cardiac output (CO) increases by 30-50% to support the uteroplacental unit. Critically, this increase in CO is facilitated by a 25-30% *decrease* in SVR, mediated by progesterone and other vasodilatory molecules.

In a healthy parturient, these changes are well-tolerated. In a patient with ES, they are lethal. The patient's PVR is already fixed at a high level. The normal pregnancy-induced drop in SVR widens the gradient between PVR and SVR (PVR >> SVR), dramatically increasing the R-L shunt.[6] This leads to profound, worsening systemic hypoxemia and cyanosis. The volume-overloaded right ventricle, already struggling to pump against a high PVR, is now faced with this exacerbated shunt and increasing preload (from increased blood volume), pushing it rapidly into decompensated failure. Furthermore, the hypercoagulable state of pregnancy in a patient with low-flow, dilated pulmonary arteries significantly increases the risk of a fatal pulmonary thromboembolism.

The peripartum period is the time of maximal danger. Labor pain, anxiety, and the Valsalva maneuver during pushing all trigger catecholamine release, which can further increase PVR.[7] Delivery of the fetus and placenta results in an autotransfusion of 500-800 mL of blood from the contracting uterus, suddenly increasing preload to an already failing right heart. Conversely, postpartum hemorrhage can lead to a sudden drop in preload and SVR, causing cardiovascular collapse.

This hemodynamic "razor's edge" makes anesthetic management for Cesarean section exceptionally challenging. The primary, non-negotiable anesthetic goals are: (1) Maintain SVR: Any significant drop in SVR will increase the R-L shunt; (2) Avoid increasing PVR: Pain, hypoxemia, hypercarbia, and catecholamine surges must be avoided; (3) Maintain adequate preload and contractility: Both hypovolemia and fluid overload can be fatal.

General anesthesia (GA) has traditionally been favored by some, but it is fraught with peril. Laryngoscopy and intubation can cause a hypertensive, catecholaminergic storm, spiking both PVR and SVR.[8] Positive pressure ventilation can impair venous return, dropping CO, and may also increase PVR. Furthermore, nearly all anesthetic induction agents (such as propofol and thiopental) and volatile agents (such as sevoflurane) are potent vasodilators that can cause a precipitous drop in SVR, worsening the shunt. Neuraxial anesthesia (spinal or epidural) is also traditionally considered relatively contraindicated.[9] A standard spinal anesthetic, with its rapid, dense, and high sympathetic blockade, causes a profound and rapid drop in SVR—the exact hemodynamic event that must be avoided. This can lead to an immediate, catastrophic increase in R-L shunting and cardiac arrest.

However, modern anesthetic practice has explored a "middle way": the use of meticulously titrated, low-dose neuraxial techniques. The aim is to separate the desired effect (profound sensory analgesia) from the undesired effect (sympathetic blockade and vasodilation). Profound analgesia is, in itself, therapeutic, as it blocks the pain-catecholamine-PVR spiral. A Combined Spinal-Epidural (CSE) technique, using a "low-dose" spinal component and a "high-dose" opioid adjuvant, theoretically provides rapid-onset analgesia with minimal hemodynamic penalty. The epidural catheter then provides a mechanism for slow, titratable extension of the block, and a reliable route for postoperative pain control.[10]

The aim of this case report is to describe and discuss the successful anesthetic management of a high-risk parturient with Eisenmenger Syndrome secondary to an ASD who underwent an urgent Cesarean section. We present a case demonstrating that a meticulously titrated, low-dose Combined Spinal-Epidural (CSE) technique, supported by invasive hemodynamic monitoring and proactive, combined vasopressor/inodilator support, can be a safe and effective strategy. This report provides a detailed, practical framework for managing the critical hemodynamic balance (SVR vs. PVR) in this patient population, challenging the traditional contraindication of neuraxial anesthesia.

2. Case Presentation

A 25-year-old female, weighing 45 kg with a height of 145 cm (BMI 21.4 kg/m^2), presented to our hospital at 32-34 weeks of gestation. She was G2P101Ab000, with a history of one prior normal spontaneous vaginal delivery. Her chief complaints were progressively worsening dyspnea over the past week, accompanied by painful uterine contractions. Her cardiac history was significant for a known congenital heart defect, which had been managed conservatively. A recent cardiology evaluation confirmed a diagnosis of Eisenmenger Syndrome. She also reported a known allergy to seafood, manifesting as urticaria.

On physical examination, the patient was in mild respiratory distress; (1) Vitals: Blood pressure (BP) 108/53 mmHg, heart rate (HR) 86 beats per minute (bpm), respiratory rate (RR) 18 breaths/min, SpO_2 90% on 3 L/min nasal cannula (NC); (2) Airway: Patent, Mallampati class 2, thyromental distance 6 cm, 3-finger mouth opening, and good neck mobility; (3) Cardiovascular: Warm extremities with capillary refill < 2 seconds. A grade 3/6 pansystolic murmur was audible at the left parasternal border (ICS 3-4). No gallops were noted; (4) Respiratory: Lungs were clear to auscultation bilaterally; (5) Extremities: Significant clubbing of the fingers was present. No peripheral edema was noted; (6) Obstetric: Uterine fundal height was consistent with gestational age. The fetal heart rate (DJJ) was 133 bpm. An assessment of ASA (American Society of Anesthesiologists) physical status 3 was assigned, with a cardiac classification of Heart Failure Stage C, Functional Class II.

Preoperative investigations revealed electrocardiogram (ECG showed sinus rhythm with a heart rate of 65 bpm and a Right Bundle Branch Block (RBBB) pattern. Chest X-Ray revealed cardiomegaly with right ventricular and left atrial enlargement (RVE, LAE) and increased pulmonary vascular markings, consistent with a congenital heart disease with a significant shunt. Transthoracic Echocardiogram (TTE) showed a large secundum atrial septal defect (ASD) was identified, measuring approximately 2.8 cm. There was a large bidirectional shunt across the ASD, noted as dominant L-R at rest. Severe pulmonic stenosis (PS) was noted, both subvalvar and valvar. There was mild tricuspid regurgitation (TR). The estimated right ventricular systolic pressure (RVSP), derived from the TR jet, was 95 mmHg, confirming a high probability of severe pulmonary hypertension (PH). Left ventricular (LV) systolic function was normal, with a biplane ejection fraction (EF) of 66%. Right ventricular (RV) systolic function was also grossly normal. Grade I diastolic dysfunction was present. Preoperative laboratory results were collected and presented in Table 1. The mild anemia (Hb 10.3) was noted, but the thrombocytopenia (121k) and hypokalemia (3.1) were also key considerations. The ABG confirmed chronic respiratory alkalosis and significant hypoxemia despite supplemental oxygen.

Table 1. Preoperative laboratory results.

PARAMETER	RESULT	REFERENCE RANGE
Complete Blood Count		
Hemoglobin (Hb)	**10.3 g/dL**	12.0–15.5 g/dL
Hematocrit (Hct)	**30.2%**	36.0–46.0%
White Blood Cell (WBC)	5,800 /mm³	4,500–11,000 /mm³
Platelets	**121,000 /mm³**	150,000–450,000 /mm³
Coagulation Profile		
Prothrombin Time (PT)	10.7 s	9.9–11.8 s
INR	1.03	0.9–1.1
Activated PTT (aPTT)	32.6 s	25.0–35.0 s
Fibrinogen	410 mg/dL	200–450 mg/dL
Renal & Liver Function		
BUN / Urea	11.5 mg/dL	7–20 mg/dL
Creatinine	0.38 mg/dL	0.6–1.1 mg/dL
AST (OT)	15 U/L	10–36 U/L
ALT (PT)	9 U/L	9–52 U/L
Albumin	**3.26 g/dL**	3.5–5.0 g/dL
Metabolic		
Glucose (Random)	89 mg/dL	< 140 mg/dL
Sodium (Na+)	**133 mEq/L**	136–145 mEq/L
Potassium (K+)	**3.1 mEq/L**	3.5–5.0 mEq/L
Chloride (Cl-)	105 mEq/L	98–107 mEq/L
Arterial Blood Gas (on 3L NC)		
pH	7.42	7.35–7.45
PaCO2	32 mmHg	35–45 mmHg
PaO2	**60 mmHg**	80–100 mmHg
HCO3-	23 mEq/L	22–26 mEq/L

A multidisciplinary conference involving Anesthesiology, Obstetrics, Cardiology, and Neonatology was held. Given the risks of GA and the patient's labor, the team opted for a meticulously managed CSE. Anesthetic plan was consisted of: (1) Monitoring: Standard ASA monitors plus a left radial arterial line for beat-to-beat BP monitoring. A right internal jugular central venous catheter was placed for

CVP monitoring and secure access; (2) Technique: Combined Spinal-Epidural (CSE); (3) Hemodynamic Support: Prophylactic, inline infusions of phenylephrine and milrinone; (4) Post-op: Transfer to the cardiovascular intensive care unit (ICU).

The patient was brought to the operating room and monitors were applied. After placement of the arterial and central lines, baseline vitals were: ABP 121/76 mmHg, HR 82 bpm, SpO_2 91% (on 3L NC), CVP 8 mmHg. In the sitting position, under strict aseptic technique, an 18-gauge Tuohy needle was used to identify the epidural space at the L3-L4 interspace. An epidural catheter was threaded 5 cm into the space. A needle-through-needle technique was used. A 27-gauge Whitacre spinal needle was advanced into the subarachnoid space. A low-dose spinal was administered, consisting of 7.5 mg (1.5 mL) of 0.5% hyperbaric bupivacaine and 50 mcg of fentanyl. The patient was immediately placed in the supine position with 15-degree left uterine displacement. Concurrently, two pre-prepared infusions were initiated: (1) Phenylephrine: Started at 40 mcg/min and titrated to maintain mean arterial pressure (MAP) > 65 mmHg and SBP > 100 mmHg; (2) Milrinone: Started at a low-dose infusion of 0.25 mcg/kg/min, per cardiology recommendation. A test dose via the epidural catheter was negative. An incremental epidural regimen was started, consisting of 0.2% ropivacaine with 50 mcg fentanyl, given in 2-3 mL aliquots to a total volume of 8 cc. Within 5 minutes, a sensory block to the T6 level was achieved. Surgery for Seksio Sesarea Transperitoneal Profunda (SCTP) commenced. The total surgical duration was 1 hour and 30 minutes. A male infant was delivered, weighing 2,800 grams. The APGAR scores were 7 at 1 minute and 8 at 5 minutes. Following delivery, a low-dose oxytocin infusion (20 Units in 1 L Ringer's Lactate, run slowly at 125 mL/hr, no bolus) was started. An IUD was also placed as planned. Throughout the procedure, the patient remained conscious, calm, and reported no pain. Hemodynamics were remarkably stable. The SBP was maintained between 105-130 mmHg, and the MAP between 65-80 mmHg. Heart rate remained 65-85 bpm. SpO_2 was stable at 88-91% on 3L NC. Estimated blood loss was 400 mL. The patient received 1000 mL of crystalloid.

Table 2. Intraoperative hemodynamic trends.

TIME (MIN)	EVENT	SBP (MMHG)	DBP (MMHG)	HR (BPM)	SPO2 (%)	NOTES
00:00	Baseline	121	76	82	91	Arterial line in.
00:05	CSE Start	120	75	80	90	---
00:10	**Spinal Done**	118	72	84	91	Supine, LUD. Phenylephrine + Milrinone infusions ON.
00:15	Epidural	110	68	85	90	Epidural titration started.
00:20	**Incision**	108	65	80	89	T6 level. Patient comfortable.
00:25	---	112	66	78	90	---
00:30	**Delivery**	115	70	82	88	Baby out. APGAR 7/8.
00:35	**Oxytocin**	110	65	85	89	*Slow* oxytocin infusion.
00:45	---	118	70	80	90	Hemodynamically stable.
01:00	Closure	122	74	75	91	---
01:15	---	120	72	72	91	---
01:30	End Surgery	118	70	74	91	Transfer to ICU.

Event Key:

Anesthetic Intervention Surgical Stimulus Key Milestone High-Risk Medication

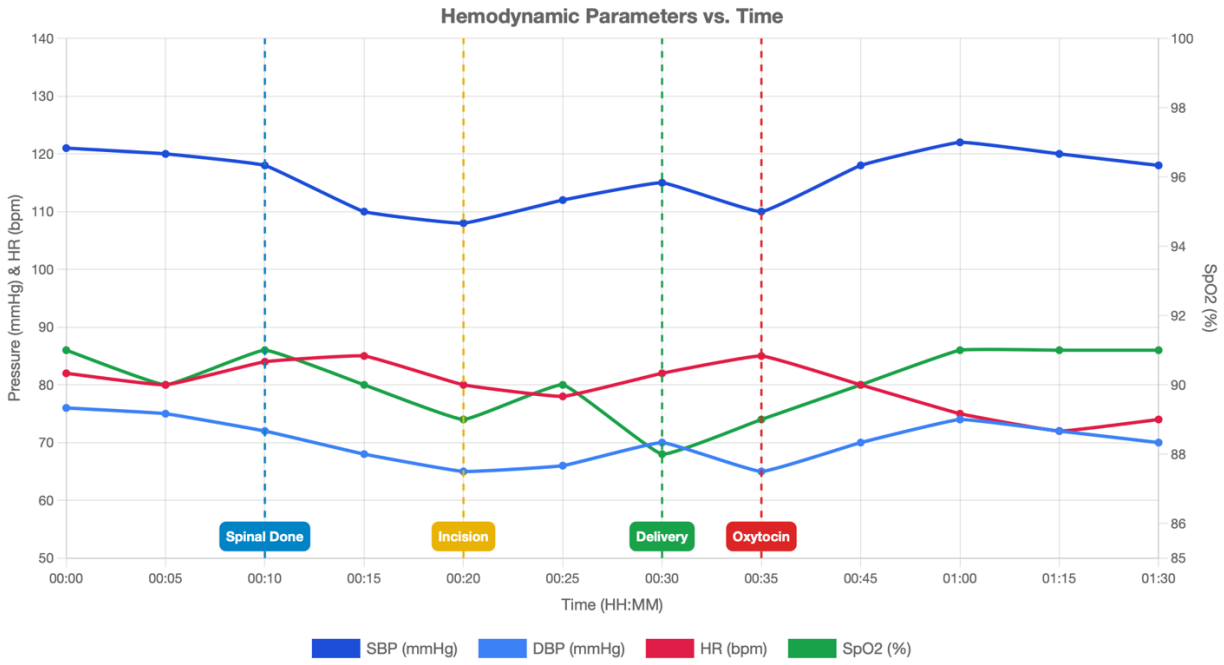

Figure 1. Intraoperative hemodynamic trends.

Postoperatively, the patient was transferred, awake and stable, to the cardiovascular ICU. The epidural catheter was used for postoperative analgesia, managed with an intermittent bolus regimen of Ropivacaine 0.1875% with 50 mcg fentanyl, 8 cc total volume as needed. The patient's reported Numeric Rating Scale (NRS) for pain was consistently 1-2. Follow up on ICU Day 0 (Post-Op); Vital signs: BP 108/63 mmHg, HR 67 bpm, RR 18/min, SpO_2 91% on 3L NC; Cardiovascular: Warm, CRT < 2 sec. Murmur still present; Neurology: GCS 15 (E4V5M6); Renal: Urine output 3800 cc over 14 hours; Medications: Continued propranolol 3x10 mg, injection cefazoline 1g/12h, injection omeprazole 40mg/24h. Post-op laboratory panel is shown in Table 3. Follow-up on ICU Day 1 (H+1) revealed the patient remained stable; Vital signs: BP 128/63 mmHg, HR 67 bpm, RR 18/min, SpO_2 90-92% on 3L NC; Status: GCS 15. Good urine output (2800 cc/24h); Plan: Given her hemodynamic stability, she was transferred from the ICU to the regular inpatient ward on the evening of H+1. The remainder of her hospital stay was uncomplicated.

3. Discussion

The successful outcome of this high-stakes case was not a matter of chance; it was the direct result of a plan that was as much an exercise in physiological reverence as it was in pharmacological science. The patient, a 25-year-old woman with Eisenmenger syndrome, represented one of the most feared scenarios in obstetric anesthesia. To manage her Cesarean section was to stand at the precipice of cardiovascular collapse, where a single misstep—a 10 mmHg drop in pressure, a moment of pain, a breath held in anxiety—could initiate an irreversible cascade. The entire anesthetic endeavor hinged on a singular, profound understanding of this patient's unique pathophysiology. This was not a routine case where one could simply "treat" hypotension as it occurred. This was a case that demanded preemptive management, a plan designed to anticipate and neutralize the catastrophic hemodynamic shifts that define Eisenmenger Syndrome in pregnancy.[11] Our success was born from this philosophy: we would not be reacting to a crisis; we would be architecting a physiological state in which a crisis could not occur.

Table 3. Postoperative laboratory results (H+0).

PARAMETER	RESULT	REFERENCE RANGE	STATUS
Complete Blood Count			
Hemoglobin (Hb)	**11.1 g/dL**	12.0–15.5 g/dL	Stable
Hematocrit (Hct)	**32.7%**	36.0–46.0%	Low
White Blood Cell (WBC)	**8,080 /mm³**	4,500–11,000 /mm³	Normal
Platelets	**143,000 /mm³**	150,000–450,000 /mm³	Borderline Low
Metabolic Panel			
Creatinine	**0.40 mg/dL**	0.6–1.1 mg/dL	Normal
Potassium (K+)	**3.4 mEq/L**	3.5–5.0 mEq/L	Borderline Low

Clinical Key:

Normal / Stable Borderline / Monitor Abnormal / Low

The anesthetic management of an Eisenmenger patient is, at its core, a "battle of two resistances." It is a zero-sum game played between the systemic vascular resistance (SVR) and the pulmonary vascular resistance (PVR). In this patient, the deck was profoundly stacked against us. Our "antagonist," as we saw it, was the pulmonary vascular resistance (PVR).[12] In Eisenmenger syndrome, the PVR is not a dynamic variable; it is a fixed, high, and non-reactive entity. Years of left-to-right shunting from her atrial septal defect had inflicted a relentless, high-pressure flow on the delicate pulmonary vasculature. This chronic assault had forced the vessels to remodel themselves in a desperate act of self-preservation. Endothelial dysfunction, smooth muscle proliferation, and the development of classic plexiform lesions had transformed a low-resistance, high-compliance circuit into a scarred, rigid, and narrow set of pipes.[13] Her PVR was, for all intents and purposes, locked at a level far exceeding that of a healthy individual.

While we could not lower this fixed resistance in any meaningful, acute way, we were acutely aware that we could very easily make it worse. The pulmonary vasculature, though scarred, retains a dangerous reactivity to certain stimuli, which we termed "PVR-spikers." These are the mortal enemies of the Eisenmenger patient: (1) Pain & Anxiety: These are not mere comfort issues; they are hemodynamic events. The sympathetic-adrenal-medullary axis, when activated by pain or fear, floods the body with catecholamines. These catecholamines, particularly norepinephrine, are potent pulmonary vasoconstrictors, and any surge would slam the door shut on an already-narrow pulmonary circuit; (2) Hypoxemia: The patient's baseline SpO_2 was already a precarious 90% due to her shunt. Any further drop would trigger the "hypoxic pulmonary vasoconstriction" (HPV) reflex. In a healthy lung, HPV is a useful mechanism to shunt blood away from non-ventilated alveoli. In this patient, it would be a global, catastrophic vasoconstriction, further spiking her PVR; (3) Hypercarbia & Acidosis: An

elevated $PaCO_2$ or a drop in pH (from hypoventilation or metabolic stress) are both powerful, direct stimuli for pulmonary vasoconstriction.[14]

Opposing this fixed antagonist was the Systemic Vascular Resistance (SVR). This, we knew, was the only major variable that was truly under our anesthetic control. The SVR, the "afterload" of the left ventricle, dictates the pressure against which the heart must pump to supply the body. In this patient, the SVR was our only leverage, the only "dial" we could turn. The entire case—and the patient's life—was governed by a simple, brutal equation: Right-to-Left Shunt $\propto$ (PVR – SVR). This formula dictates that the amount of deoxygenated, "blue" blood that bypasses the lungs and shunts directly into the systemic circulation (the R-L shunt) is directly proportional to the gradient between these two resistances. Because her PVR was already fixed at a high level (95 mmHg RVSP), her SVR (a diastolic of 53 mmHg) was the only thing preventing a total circulatory collapse.[15]

If her SVR were to drop—the very thing that all anesthetics are designed to do—the (PVR – SVR) gradient would widen instantly and dramatically. A torrent of deoxygenated blood from the right heart would bypass the lungs and pour into the systemic circulation, destined for the brain, the coronary arteries, and the kidneys. The patient's SpO_2 would plummet. This profound hypoxemia would, in turn, trigger a massive HPV reflex, spiking PVR even higher.[16] This would widen the gradient further, increasing the shunt, which would cause more hypoxemia. This is the "spiral of death" in Eisenmenger Syndrome: a precipitous drop in SVR leads to a self-propagating cycle of shunting, hypoxemia, and right ventricular failure, culminating in cardiac arrest.

Therefore, our primary, non-negotiable goal was twofold, clear, and absolute; maintain SVR at or above its baseline level at all times and simultaneously avoid, at all costs, any stimulus that could increase PVR. This case report, and the strategy it details, stands as a direct challenge to the long-held dogma that neuraxial anesthesia is absolutely contraindicated in these patients (Figure 2). Textbooks and historical case series are filled with reports of catastrophic arrests in Eisenmenger patients who received a "standard" spinal anesthetic. We argue that it is not neuraxial anesthesia itself that is dangerous, but rather the technique of its application. A standard "single-shot" spinal anesthetic, aiming for a rapid, dense T4 block with 12-15mg of bupivacaine, is a hemodynamic sledgehammer. It induces a rapid, profound, and uncontrolled sympathetic blockade—the very SVR collapse we sought to avoid. Our approach was different. We hypothesized that a meticulously titrated neuraxial technique—a "scalpel" rather than a sledgehammer—could allow us to achieve our goals. We could use it to simultaneously address both sides of the SVR/PVR conflict: we could provide profound analgesia (to prevent PVR spikes from pain) while using a combination of low-dose local anesthetics and proactive vasopressors to maintain SVR.[17]

Our strategy was built upon three fundamental pillars, each designed to work in concert with the others (Figure 2). This was not a simple checklist, but a synergistic system of hemodynamic control. The CSE was chosen to leverage the best of both worlds: the swift, reliable, and profound sensory block of a spinal anesthetic, combined with the unparalleled control and titrability of an epidural catheter. First, we addressed the spinal component. A standard spinal dose was out of the question. We selected a "low-dose" spinal of 7.5 mg hyperbaric bupivacaine. This dose was carefully calculated to be just enough to establish a sensory block, but not enough to cause a catastrophic, widespread sympathetic blockade. Bupivacaine works by blocking voltage-gated sodium channels, thereby inhibiting nerve conduction. This effect is, of course, dose-dependent. A standard 12.5 mg dose would rapidly anesthetize the sympathetic chain efferents up to the mid-thoracic level, causing massive, uncontrolled vasodilation. Our 7.5 mg dose, by contrast, was a targeted strike. We were willing to accept a lower block height from the spinal component alone, knowing we could supplement it later, in exchange for preserving the patient's SVR.[18]

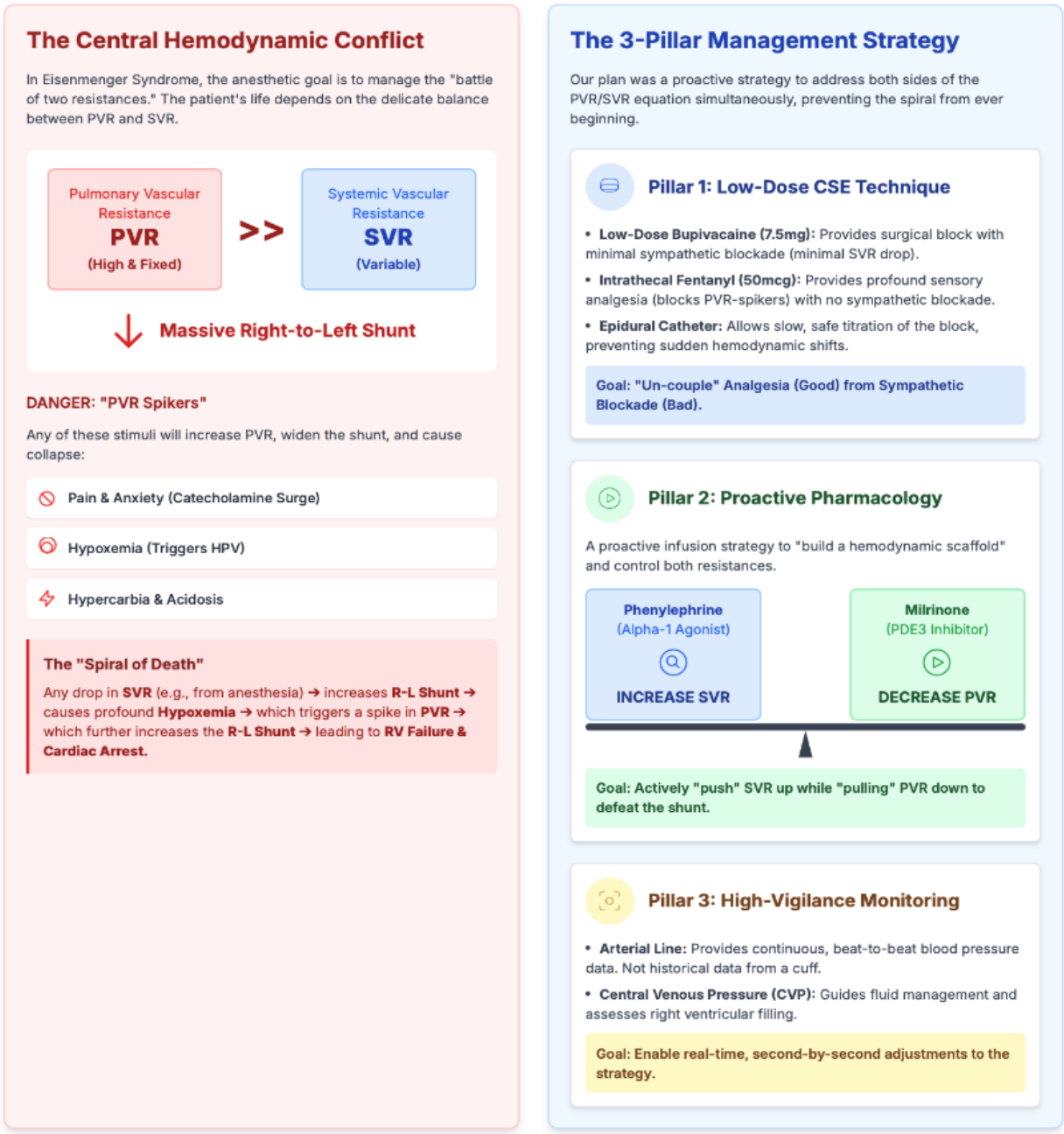

Figure 2. Anesthetic strategy: managing the high-stakes hemodynamics of Eisenmenger syndrome in pregnancy.

The second—and arguably more critical—component of the intrathecal injection was 50 mcg of fentanyl. This was the key to "un-coupling" analgesia from sympathetic blockade. While bupivacaine blocks all nerve traffic (sensory, motor, and sympathetic), intrathecal fentanyl has a much more elegant and specific mechanism. As a potent lipophilic opioid, it diffuses into the dorsal horn of the spinal cord (specifically, the substantia gelatinosa) and binds to μ-opioid receptors. This selectively blocks the transmission of nociceptive (pain) signals from C-fibers and A-delta fibers before they can even ascend to the brain. The result? We achieved a profound, dense sensory block—the patient would feel no pain from the incision—with minimal contribution to vasodilation or hypotension. The fentanyl provided the analgesia, while the low-dose bupivacaine provided the "anesthesia," allowing us to achieve our primary goal of eliminating pain—a key "PVR-spiker"—without paying the hemodynamic price. Finally, the epidural

catheter was our "control knob" and our "throttle." The CSE technique allowed us to place this catheter in the epidural space before initiating the spinal block. This catheter was our lifeline. Once the limited spinal block was established, we began a slow, incremental titration of a dilute solution (0.2% ropivacaine) and more fentanyl. This incremental approach transformed the anesthesia from an "all-or-nothing" event into a controlled, meticulous process. We would inject 2-3 mL of the solution, wait 3-5 minutes, observe the arterial line waveform in real-time, and then—only when we were satisfied that the SVR was stable—would we inject another small aliquot. This allowed us to "walk" the block height up to the required T6 level for a Cesarean section slowly. This careful, controlled onset is the physiological opposite of the rapid, shocking hemodynamic insult of a single-shot spinal. The epidural catheter was our tool of titration.[19]

This pillar represents a complete paradigm shift from traditional anesthetic management. In this high-risk case, we did not wait for hypotension to occur. We did not have "rescue" vasopressors drawn up; we had "support" infusions running from the very beginning. We anticipated, with 100% certainty, that our neuraxial technique would cause some vasodilation. Therefore, we built a "pharmacological scaffold" to maintain the SVR-PVR balance before the first drop in pressure could even be measured. The first component of this scaffold was a prophylactic phenylephrine infusion. Phenylephrine is a pure alpha-1 adrenergic agonist. It is the ideal agent for this scenario: it potently increases SVR by vasoconstricting peripheral arteries, with minimal to no effect on heart rate (it may, in fact, cause a therapeutic reflex bradycardia) or cardiac contractility. By starting this infusion inline as the spinal was being performed, we were not "chasing" hypotension; we were "setting the SVR." We were establishing a new, higher baseline SVR, creating a "hemodynamic floor" before the vasodilatory effects of the spinal took hold. This proactive defense of SVR was the cornerstone of our entire strategy. The second component was the most sophisticated: a prophylactic milrinone infusion. This was the masterstroke of the plan, a testament to a deep understanding of the central hemodynamic conflict. Milrinone is a phosphodiesterase-3 (PDE3) inhibitor, and it provided two crucial, simultaneous benefits for this specific patient. First, milrinone is a potent pulmonary vasodilator. While phenylephrine was "squeezing" the systemic arteries, milrinone was "relaxing" the pulmonary arteries. Second, milrinone is a "non-catecholamine inotrope." It increases cardiac contractility (in-otropy) by a mechanism different from adrenaline, supporting the heart's pumping function.[20]

This combined, simultaneous infusion was the ultimate expression of managing gradient. We were actively and aggressively manipulating both sides of the equation in our favor: phenylephrine was "pushing" SVR up, and milrinone was "pulling" PVR down. The net effect was to maximally decrease the (PVR – SVR) gradient, thereby minimizing the right-to-left shunt. Furthermore, the inotropic support from the milrinone was giving the patient's chronically-overloaded, struggling right ventricle the strength it needed to pump against the high PVR. This was not just anesthesia; this was applied, real-time-systems physiology.

High-vigilance monitoring, a delicate strategy would be impossible—and lethally dangerous—without high-fidelity, continuous, real-time monitoring. This pillar was the "eyes" of the entire operation, providing the data feedback-loop that made the first two pillars possible. The arterial line was essential. In a patient this fragile, a standard, non-invasive blood pressure (NIBP) cuff that cycles every 3-5 minutes is not just inadequate; it is a "historical curiosity." A patient with Eisenmenger Syndrome can suffer a fatal cardiovascular collapse in the 90 seconds between one "normal" cuff reading and the next. The arterial line provided a continuous, beat-to-beat waveform on our monitor. We could see the instant hemodynamic consequence of a 2cc epidural bolus, the instant effect of a 10 mcg/min increase in the phenylephrine infusion. It allowed us to react not in minutes, but in seconds. This "second-by-second" feedback loop is what allowed us to titrate our interventions with such precision, keeping the patient on the hemodynamic razor's edge without falling. The central venous pressure (CVP) line provided another crucial piece of data. It gave us a window into the patient's volume status and, more importantly, the filling pressure of the right ventricle. In this patient,

both hypovolemia (loss of preload) and fluid overload (which would distend the RV and push it into failure) were equally dangerous. The CVP helped us navigate the treacherous path of fluid management.[17,18]

Our choice of a titrated neuraxial technique was a deliberate, strategic one, made after weighing the equally significant, but arguably more volatile, risks of general anesthesia (GA). We had not chosen a "safe" path; we had simply chosen our battle. We felt the risks of our neuraxial approach, while significant, were controllable. The risks of GA, by contrast, are explosive and far less predictable. A rapid sequence induction would have required a bolus of a potent induction agent, such as propofol. Propofol is a powerful vasodilator and myocardial depressant. In a patient already vasodilated by pregnancy and with a failing RV, this induction bolus would be akin to "pulling the floor out" from under her SVR, risking an immediate, profound crash. The very act of laryngoscopy is a profound noxious stimulus, one of the most intense a patient can experience. In this patient, the "sympathetic explosion" from the intubation—the surge of catecholamines—would have been a dagger aimed at her pulmonary artery, causing a massive, acute spike in her PVR. Once intubated, the patient would require PPV. The "bellows" of the ventilator, by increasing intrathoracic pressure, would physically impede the return of blood from the body to the right heart, dropping her preload. In a patient critically dependent on that preload, this could have been disastrous. Maintaining anesthesia with volatile agents like sevoflurane would have introduced another SVR-lowering variable, as these agents are all known vasodilators and myocardial depressants. By choosing our titrated neuraxial technique, we completely avoided the "Catch-22" of laryngoscopy. We avoided the hemodynamic trespass of positive pressure ventilation. And we avoided the cardiodepressant effects of volatile agents. We focused all our efforts on managing the single, controllable variable of SVR.

The high-risk period for this patient did not end with the closing stitch. In many ways, the "battle" had just begun. The postoperative period, with its inflammatory responses, fluid shifts, and—most dangerously—pain, remained a time of high alert. Postoperative pain is a potent "PVR-spiker". Every time the patient would have winced from the incision, every time she "splinted" her breathing from pain, a micro-burst of catecholamines would have been released. This is the exactphysiological insult we had worked so meticulously to avoid for the entire 90-minute surgery. Inadequate analgesia would lead to shallow breathing (atelectasis), hypoventilation (hypercarbia), and hypoxemia—a triad of potent PVR-spikers.

This is why the epidural catheter was a critical component of the entire 72-hour plan. Its utility extended far beyond the operating room. By providing continuous, reliable analgesia (NRS 1-2), we ensured the patient had a stress-free recovery. This was not merely a matter of comfort; it was a life-sustaining-therapy. The epidural analgesia kept her calm, breathing deeply, and hemodynamically stable, preventing the catecholamine surges that could have precipitated a postoperative crisis. It was the bridge that carried her safely from the high-intensity OR environment to a stable recovery, and ultimately, to a successful outcome. However, this is a single case report, and its conclusions cannot be generalized. The success of this technique was dependent on a high-functioning multidisciplinary team, the availability of advanced monitoring and pharmacology, and a patient who, despite her ES, had preserved LV and RV function. The specific underlying defect (ASD) may also be a factor, as ES secondary to ASD is sometimes better tolerated than that from a large VSD.[19,20]

4. Conclusion

This case report demonstrates that Cesarean section in a parturient with Eisenmenger Syndrome, one of the highest-risk scenarios in obstetric anesthesia, can be managed successfully. We have shown that a low-dose, titrated Combined Spinal-Epidural (CSE) technique is a viable and potentially superior strategy. The success of this approach is not in a single intervention, but in a comprehensive, integrated strategy. This includes: (1) A titrated neuraxial technique (low-dose spinal, opioid-heavy, slow epidural titration) to provide profound analgesia (decreasing PVR) while minimizing sympathetic blockade (preserving SVR); (2) A proactive pharmacological plan (inline phenylephrine) to

aggressively defend SVR; (3) The sophisticated use of adjunctive therapy (milrinone) to simultaneously support the RV and decrease PVR; (4) A high-vigilance monitoring environment (arterial line) to allow for real-time, beat-to-beat management; (5) A continuation of care into the postoperative period with excellent epidural analgesia to prevent PVR-spiking. In conclusion, this case demonstrates that by meticulously managing the delicate balance between SVR and PVR, the traditionally feared neuraxial technique can be transformed from a contraindication into a powerful, life-saving tool for the high-risk obstetric patient with Eisenmenger Syndrome.

5. References

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