

Review Article

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Anesthetic Concerns and Management in Parturient with Cardiac Disease: Congenital Non-Congenital and Structural Pathologies

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ABSTRACT

Maternal cardiovascular disease remains a leading cause of indirect maternal mortality in modern obstetrics. The complex interactions between the normal physiological adaptations of pregnancy and underlying structural, valvular, or ischemic cardiac defects present a significant management challenge. This article evaluates the anesthetic concerns and clinical management strategies for parturients with congenital and non-congenital cardiac diseases. We trace the hemodynamic changes across the peripartum period, examining the physiological stress points of labor and the immediate postpartum window. The pathophysiology of congenital heart disease is analyzed across three distinct categories of cardiac shunts: left-to-right, right-to-left, and complex bidirectional flows (Eisenmenger syndrome). Furthermore, we compare the risks and benefits of general anaesthesia versus regional blocks, focusing on the specific hemodynamic effects of each approach. Clinical decision-making is structured around the Modified World Health Organization (mWHO) risk classification framework, which guides the management of high-risk cases, including the choice between termination of pregnancy (TOP) and carrying to term. Finally, we establish a robust multi-disciplinary protocol governing elective versus emergent lower segment cesarean sections (LSCS) and assisted spontaneous vaginal deliveries (SVD).

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Introduction

Cardiovascular disease complicates approximately 1% to 4% of all pregnancies in developed countries, yet it accounts for an outsized proportion of severe maternal morbidity and indirect mortality. Over the past several decades, the clinical profile of the cardiac parturient has shifted. Advances in pediatric cardiothoracic surgery and intensive care have enabled an unprecedented number of patients with complex congenital heart disease (CHD) to survive into adulthood and achieve pregnancy. Concurrently, advancing maternal age, the prevalence of metabolic syndrome, and lifestyle changes have increased the incidence of acquired, non-congenital cardiac conditions, such as ischemic heart disease, rheumatic valvular defects, and peripartum cardiomyopathy [1].

The peripartum management of these patients represents a significant clinical challenge. The standard cardiovascular adaptations of pregnancy can unmask or worsen latent cardiac dysfunction, precipitating heart failure, arrhythmias, or thromboembolic events. Optimizing maternal and fetal outcomes requires a deep understanding of altered physiology, lesion-specific hemodynamics, and the precise application of anesthetic techniques.

This article provides an in-depth clinical reference manual addressing the anesthetic concerns and management strategies for parturients with congenital and non-congenital cardiac diseases. By examining gestational hemodynamic shifts, analyzing shunt

dynamics, evaluating the choice between general and regional anesthesia, and utilizing the mWHO classification framework to guide delivery planning, this work establishes a robust clinical template for high-risk obstetric anesthesia care.

Cardiovascular Adaptations of Pregnancy: The Stress Test Gestational Hemodynamic Shifts

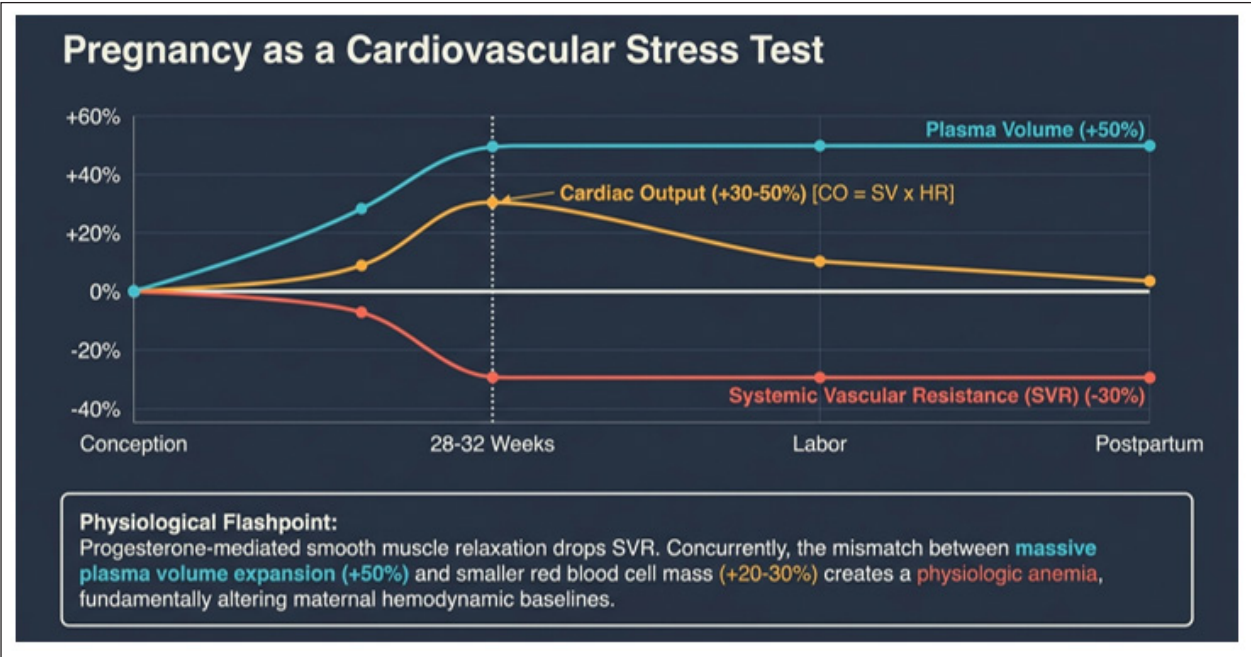
Pregnancy functions as a prolonged hemodynamic stress test that challenges maternal cardiovascular reserves. To support fetal development, the maternal cardiovascular system undergoes extensive adaptations that begin in the first trimester and peak between 28 and 32 weeks' gestation.

Plasma volume expands by up to 50%, while total red blood cell mass increases by 20% to 30%. This mismatch results in a physiologic anemia of pregnancy, which reduces blood viscosity and optimizes uterine perfusion. To maintain systemic oxygen delivery, resting cardiac output (CO) rises by 30% to 50% above baseline. This increase is initially driven by an elevated stroke volume (SV) and later sustained by a 15% to 20% increase in maternal heart rate (HR), following the equation $CO = SV \times HR$. Concurrently, systemic vascular resistance (SVR) declines by 20% to 30%, driven by progesterone-mediated smooth muscle relaxation and the low-resistance placental circulation [2-5].

Peripartum Flashpoints and Volumetric Shifting

The greatest risk of cardiovascular decompensation occurs during labor, delivery, and the immediate postpartum period, where hemodynamic parameters undergo rapid fluctuations.

During labor, each uterine contraction auto transfuses approximately 300 to 500 mL of blood back into the central circulation, causing temporary spikes in venous return (preload) and systemic blood pressure (afterload). Pain, anxiety, and sympathetic activation further elevate maternal heart rate and myocardial oxygen demand. Immediately following delivery, the relief of aortocaval compression combined with rapid uterine involution shifts a massive volume of fluid into the intravascular space, causing cardiac output to spike up to 80% above baseline within hours of birth. This immediate postpartum window is the most common time for acute pulmonary edema and cardiovascular collapse in vulnerable cardiac patients.



The mWHO Classification and Risk Stratification Matrix

To standardize risk assessment and clinical pathways, international guidelines endorse the Modified World Health Organization (mWHO) classification framework. This system categorizes specific cardiovascular conditions based on maternal risk, guiding monitoring intensity and delivery location.

The mWHO Risk Stratification Framework			
Class	Risk Level	Pathology	Protocol
Class I	Low. No detectable mortality increase.	Mild pulmonic stenosis, successfully repaired simple defects (ASD, VSD).	Normal pregnancy management at regional centers.
Class II	Mild-Moderate.	Unoperated simple shunts, corrected Tetralogy of Fallot.	Trimester cardiology follow-ups; delivery at a cardiac-aware center.
Class III	Severe. Significantly increased risk.	Mechanical prosthetic valves, systemic RV, Fontan circulation.	Monthly multidisciplinary reviews; tertiary cardio-obstetric center delivery mandatory.
Class IV	Extremely High. (10-30% mortality). Pregnancy is contraindicated.	Pulmonary Arterial Hypertension (PAH), severe systemic dysfunction (LVEF <30%), severe symptomatic aortic stenosis.	Immediate counseling for Termination of Pregnancy (TOP).

Examples of specific conditions within these categories include: Class I covers mild pulmonic stenosis or successfully repaired simple defects (ASD, VSD); Class II involves unoperated simple shunts or corrected Tetralogy of Fallot; Class III includes mechanical prosthetic valves, a systemic right ventricle, or Fontan circulation; and Class IV includes pulmonary arterial hypertension (PAH) of any etiology, severe systemic ventricular dysfunction (LVEF <30%), severe symptomatic aortic stenosis, or prior peripartum cardiomyopathy with residual dysfunction.

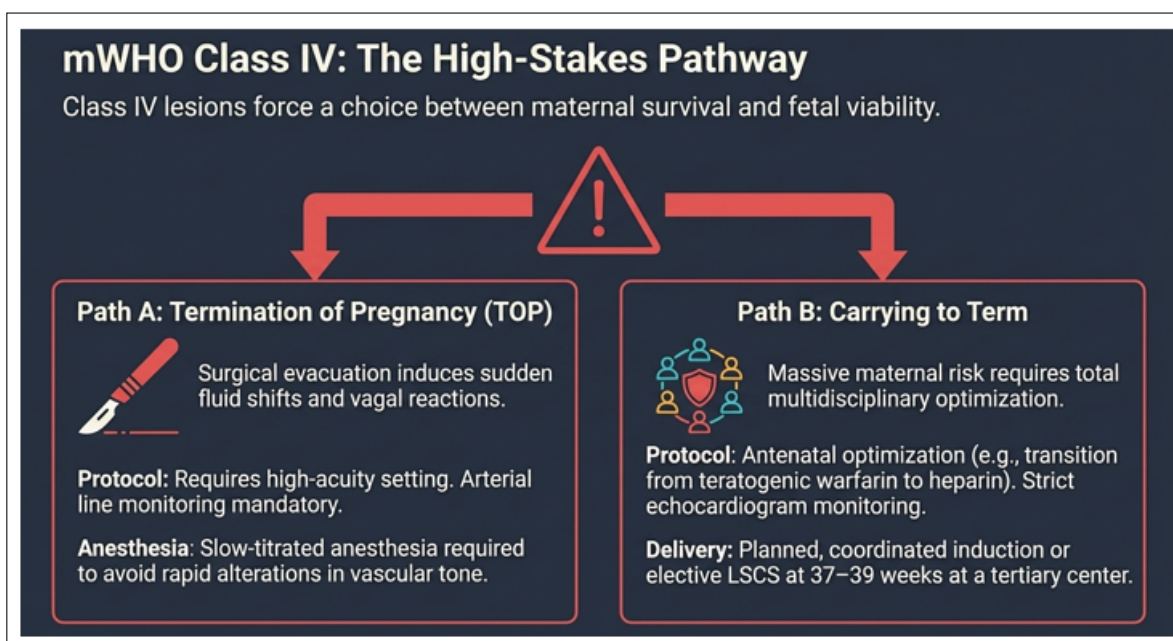
Clinical Dilemma: Termination of Pregnancy (TOP) vs. Carrying to Term

When a patient presents with an mWHO Class IV condition, the medical team faces a critical clinical decision. Conditions such as severe pulmonary hypertension carry maternal mortality rates historically ranging from 10% to 30%, even with modern advanced therapies, making pregnancy a direct threat to maternal life.

If a patient with an mWHO Class IV lesion presents early in the first trimester, current international guidelines recommend comprehensive counseling regarding Termination of Pregnancy (TOP). This consultation must be managed by a multidisciplinary team, providing clear, objective data regarding maternal risks while respecting the patient's autonomy and personal choices.

If the patient chooses to undergo a TOP, the anesthetic management remains highly complex. Surgical evacuation (such as dilation and evacuation) can induce sudden fluid shifts, systemic inflammatory responses, and vagal reactions that can destabilize conditions like severe aortic stenosis or pulmonary hypertension. Anesthesia for first- or second-trimester TOP in these patients must be performed in a high-acuity setting with advanced hemodynamic monitoring (e.g., an arterial line) and careful titration of low-dose sedatives or regional blocks to avoid rapid alterations in systemic or pulmonary vascular tone.

If the patient declines TOP or presents too late in gestation to safely undergo termination, the clinical focus shifts to carrying to term with intensive risk-mitigation strategies. Care must be centralized at a tertiary center featuring a dedicated cardio-obstetric team. Antenatal optimization includes adjusting teratogenic medications (e.g., switching warfarin to low-molecular-weight heparin or unfractionated heparin in patients with mechanical valves), managing heart failure with pregnancy-safe agents, and scheduling regular echocardiograms to track ventricular function. While spontaneous labor is preferred for stable patients, high-risk mWHO III/IV lesions frequently warrant a planned, highly coordinated induction of labor or elective cesarean delivery between 37 and 39 weeks' gestation to ensure full team availability [6-10].



Corrected vs. Uncorrected Cardiac Lesions

Surgically Corrected Lesions and Residual Risks

Surgical correction of a congenital defect does not necessarily restore normal cardiac physiology. Anesthesia providers must carefully evaluate patients for residual anatomical or physiological defects.

In patients with a repaired Tetralogy of Fallot, although the primary ventricular septal defect is closed and the right ventricular outflow tract obstruction relieved, patients frequently develop severe, chronic pulmonary regurgitation. Over time, this leads to right ventricular dilation, dysfunction, and a high risk for malignant ventricular arrhythmias under the hemodynamic stress of pregnancy. Similarly, patients who underwent physiological repairs for d-Transposition of the Great Arteries (such as the historical Mustard or Senning procedures) rely on the morphologic right ventricle to support the systemic circulation. The right ventricle is unsuited for long-term systemic pressures; by the time these patients reach childbearing age, they are at high risk for systemic ventricular failure, tricuspid regurgitation, and sinus node dysfunction. Furthermore, patients with single-ventricle pathways (Fontan circulation) represent a unique challenge, as passive systemic venous return relies entirely on elevated central venous pressure (CVP) and low pulmonary vascular resistance (PVR). Any drop in preload or increase in PVR can interrupt this delicate balance, resulting in immediate cardiovascular collapse.

Uncorrected Lesions and Active Pathophysiology

Uncorrected lesions present active structural abnormalities that must directly guide hemodynamic management goals. An uncorrected simple shunt, such as an isolated atrial or ventricular septal defect, is generally well tolerated during pregnancy provided pulmonary arterial pressures remain normal. In contrast, an uncorrected stenotic valvular lesion, such as severe aortic or mitral stenosis, restricts forward blood flow and leaves the patient unable to increase cardiac output to meet gestational demands, frequently culminating in acute pulmonary edema or cardiogenic shock under labor conditions [11].

Pathophysiology and Anesthetic Management of Cardiac Shunts

Left-to-Right Shunts

Simple left-to-right shunts including Atrial Septal Defects (ASD), Ventricular Septal Defects (VSD), and Patent Ductus Arteriosus (PDA) are typically well tolerated during normal pregnancy. The standard gestational decrease in systemic vascular resistance (SVR) reduces the net left-to-right shunting, balancing the systemic and pulmonary circulations.

The primary hemodynamic goal is to maintain a stable or slightly decreased SVR and avoid acute elevations in SVR, which can worsen left-to-right flow and strain the right heart. Controlled, titrated epidural analgesia is beneficial during labor because it lowers SVR mildly and minimizes labor-induced spikes in systemic blood pressure. However, care must be taken to prevent sudden drops in blood pressure that could reverse the shunt direction.

Right-to-Left Shunts

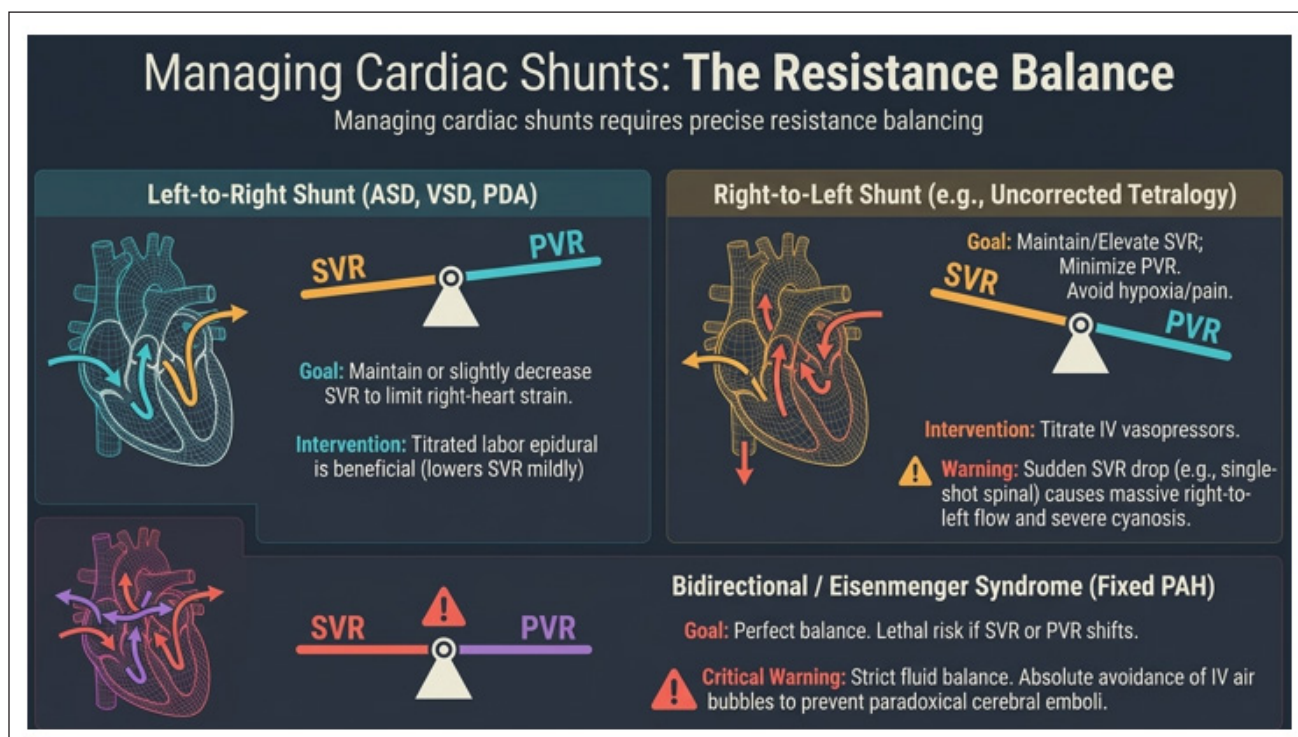
Right-to-left shunts occur when right-sided chamber or pulmonary pressures exceed systemic pressures, causing deoxygenated blood to bypass the lungs and enter the systemic arterial circulation. This leads to chronic hypoxemia, cyanosis, and compensatory polycythemia, as seen in uncorrected Tetralogy of Fallot.

The essential hemodynamic goals are to maintain or elevate SVR and minimize PVR by avoiding hypoxia, hypercapnia, acidosis, pain, and hypothermia. A sudden drop in SVR (e.g., from a rapid-onset single-shot spinal block) decreases systemic outflow resistance, rapidly increasing right-to-left shunting and precipitating severe hypoxemia and cyanosis. Intravenous vasopressors (such as phenylephrine or noradrenaline) must be titrated to support SVR throughout the perioperative period.

Bidirectional Shunts and Eisenmenger Syndrome

Eisenmenger syndrome represents the advanced endpoint of a long-standing, uncorrected left-to-right shunt. Chronic pulmonary over circulation induces irreversible pulmonary vascular remodeling, leading to fixed pulmonary arterial hypertension (PAH). When PVR exceeds SVR, the shunt reverses to a bidirectional or predominantly right-to-left flow.

This represents an mWHO Class IV scenario associated with a high maternal mortality risk. Management requires maintaining a balance between SVR and PVR, as these patients are sensitive to alterations in either system. Fluid balance must be monitored, as these patients are sensitive to both hypovolemia (which decreases venous return and precipitates a right-to-left shunt) and hypervolemia (which overloads a failing right ventricle). Air bubbles in all intravenous lines must be avoided to prevent paradoxical emboli to the cerebral circulation [12-13].



Anesthetic Techniques: General Anesthesia vs. Regional Anesthesia
Regional Anesthesia (RA) Protocols

The choice between General Anesthesia (GA) and Regional Anesthesia (RA) for operative delivery or labor analgesia must be individualized based on the patient's specific lesion, cardiac reserve, and clinical status. Regional anesthesia includes continuous epidural, single-shot spinal, and combined spinal epidural (CSE) techniques.

RA avoids the sympathetic stimulation associated with endotracheal intubation, maintains spontaneous ventilation (minimizing changes in intrathoracic pressure and pulmonary blood flow), and provides postoperative analgesia. However, the sympathectomy induced by local anesthetics can cause a rapid, unpredictable decrease in SVR. Therefore, a standard single-shot spinal anesthesia is contraindicated in lesions sensitive to sudden drops in afterload (such as severe aortic stenosis, hypertrophic obstructive cardiomyopathy, or Eisenmenger syndrome). Instead, a low-dose, incrementally titrated catheter technique (either a continuous epidural or a sequential CSE) should be utilized. This allows the anesthesia provider to establish a sensory block slowly over 30 to 45 minutes, facilitating controlled compensation for the gradual sympathectomy [14].

General Anesthesia (GA) Protocols

General anesthesia is indicated for emergency deliveries, patients with deep coagulopathies, or specific anatomical lesions where the hemodynamic predictability of controlled mechanical ventilation is preferred. GA allows precise, rapid control over ventilation, oxygenation, and systemic vascular tone.

However, the induction of general anesthesia, direct laryngoscopy, and endotracheal intubation can trigger a large catecholamine surge, causing transient tachycardia and hypertension. Positive-pressure ventilation also increases intrathoracic pressure, which can reduce venous return (preload) and increase PVR. To prevent tachycardia and hypertension during induction, the sympathetic response to intubation must be blunted. This is achieved using short-acting opioids (such as remifentanyl or alfentanil, bearing in mind the risk of neonatal respiratory depression) combined with induction agents that preserve cardiovascular stability, such as etomidate or low-dose ketamine paired with midazolam, followed by rapid-acting neuromuscular blockade (succinylcholine or rocuronium).

The Anesthetic Arsenal: RA vs. GA			
Regional versus general anesthesia profiles dictate physiological impact.			
Regional Anesthesia (RA)		Protocol	
✓ Pros	⚠ Cons	Single-shot spinals are explicitly contraindicated in severe lesions. Mandates low-dose, incrementally titrated Continuous Epidural or CSE established slowly over 30–45 minutes.	
Avoids intubation stress; preserves spontaneous ventilation (maintains normal intrathoracic pressure).	Sympathectomy causes unpredictable SVR drop.		
General Anesthesia (GA)		Protocol	
✓ Pros	⚠ Cons	Requires cardioprotective induction (etomidate/low-dose ketamine + short-acting opioids like remifentanyl) to blunt the sympathetic response.	
Precise, rapid control over ventilation, oxygenation, and systemic tone. Indicated for emergencies or deep coagulopathy.	Direct laryngoscopy/intubation triggers massive catecholamine surge (tachycardia/hypertension).		

Valvular and Non-Congenital Cardiac Pathology
Stenotic Valvular Lesions (Aortic and Mitral Stenosis)

Stenotic lesions restrict forward blood flow, limiting the heart's capacity to scale up cardiac output to meet gestational demands. Severe aortic stenosis (AS) and severe mitral stenosis (MS) are poorly tolerated during pregnancy and require careful hemodynamic management.

The primary hemodynamic goals are to maintain a slow-to-normal heart rate to ensure adequate diastolic filling time (especially critical in mitral stenosis) and coronary perfusion pressure, maintain SVR to preserve myocardial perfusion, and maintain adequate preload without causing volume overload. Pain, anxiety, and tachycardia must be avoided. For labor, early titrated epidural analgesia is effective for blunting the sympathetic response to pain and contractions. If a cesarean section is required, a carefully titrated epidural or sequential CSE is preferred over general anesthesia to avoid the intubation-induced catecholamine surge, provided SVR is supported with a continuous vasopressor infusion [15].

Regurgitant Valvular Lesions (Aortic and Mitral Regurgitation)

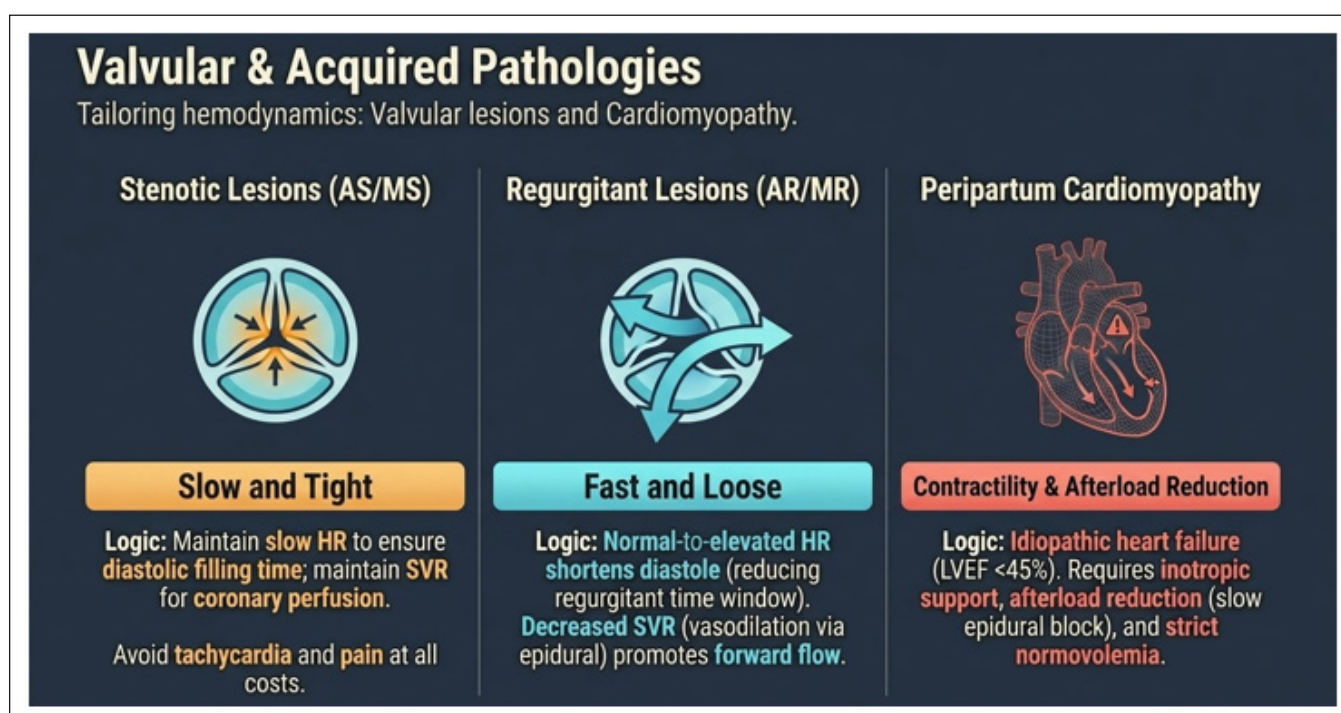
Regurgitant lesions are generally well tolerated during pregnancy. The standard physiological reduction in SVR acts as a natural afterload reducer, promoting forward flow and minimizing the regurgitant fraction. The mild gestational tachycardia also shortens the duration of diastole, reducing the time window for regurgitant flow per minute.

The primary hemodynamic goals are to maintain a normal-to-elevated heart rate ('fast') and a stable-to-decreased SVR ('loose') while avoiding fluid overload. Titrated regional anesthesia is well suited for these patients, as the induced vasodilation supports the primary hemodynamic goal of reducing afterload.

Peripartum Cardiomyopathy (PPCM)

PPCM is an idiopathic form of heart failure presenting with an ejection fraction typically <45%, developing in the final month of pregnancy or within five months postpartum in patients without prior heart disease. Management requires supporting myocardial contractility, optimizing afterload reduction to ease the workload on the failing left ventricle, and maintaining normovolemia.

If the patient is stable and undergoing a cesarean section, a slow epidural block provides useful afterload reduction. In cases of severe decompensation or cardiogenic shock, invasive monitoring (arterial line, central venous catheter, or advanced non-invasive cardiac output monitoring) is required. General anesthesia with inotropic support (such as dobutamine or milrinone) may be necessary to stabilize hemodynamics [16-18].



Delivery Planning: LSCS vs. SVD in the Cardiac Parturient

Elective Planning: SVD as the Hemodynamic Standard

The decision between LSCS and SVD in a cardiac patient is guided by hemodynamics, with the primary objective of minimizing variations in preload and afterload. Contrary to historical assumptions, vaginal delivery (SVD) remains the safest mode of delivery for most cardiac patients. SVD is associated with significantly less intraoperative blood loss, fewer fluid shifts, lower infection rates, and a lower incidence of postpartum thromboembolism compared to an elective LSCS.

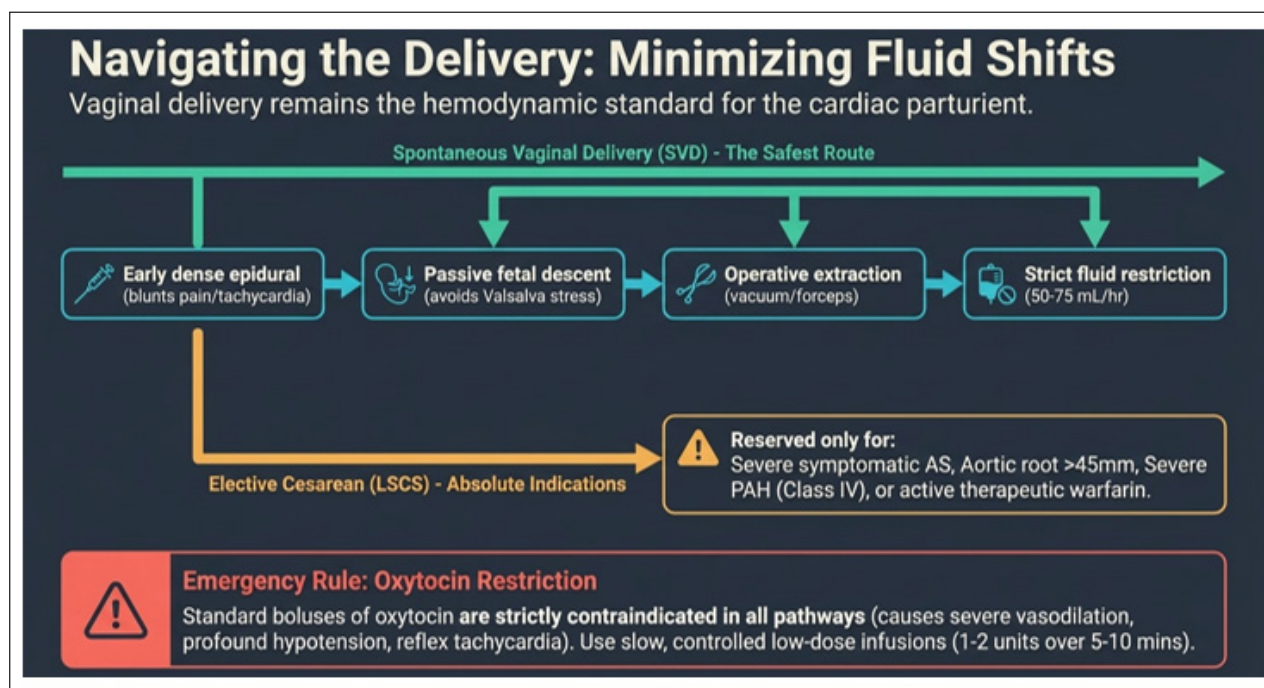
To maximize safety, a 'Cardiac SVD' protocol is implemented:

- **Effective Analgesia:** Early, dense epidural analgesia is mandatory to eliminate labor pain and anxiety, thereby blunting the maternal sympathetic surge (tachycardia and hypertension) during contractions.
- **Passive Descent:** To prevent the hemodynamic stress of the Valsalva maneuver (which alters venous return and afterload), the patient is allowed to let the uterus push the baby down passively during the second stage of labor. The delivery is then completed using operative vacuum or forceps extraction once the fetal head is engaged.
- **Fluid Restrictions:** Intravenous fluids are strictly restricted (often capped at 50–75 mL/hr) using infusion pumps to avoid fluid overload and acute pulmonary edema.

Elective LSCS: Specific Cardiac Indications

Elective LSCS is reserved for specific, highly unstable cardiovascular pathologies where the mechanical stresses or unpredictable timing of labor could cause structural collapse. These absolute cardiac indications include: severe, symptomatic aortic stenosis; aortic root dilation >45 mm (e.g., in Marfan syndrome or Loeys-Dietz syndrome) due to the risk of aortic dissection during labor; acute, decompensated heart failure; severe pulmonary arterial hypertension (mWHO Class IV), where any acute shift in PVR/SVR

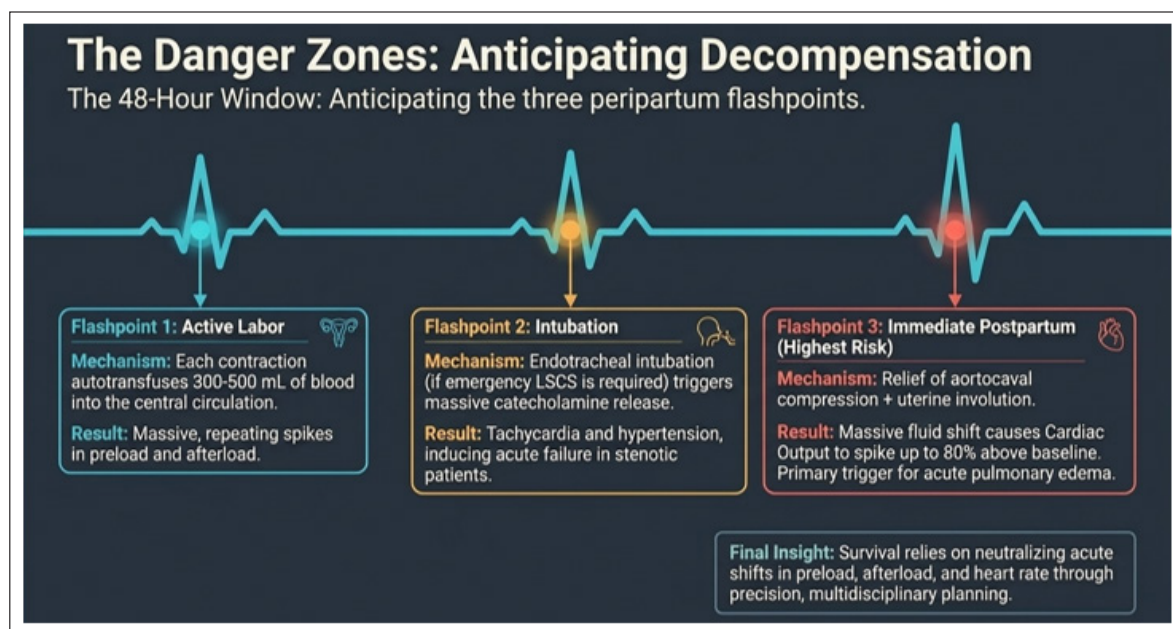
balance can cause right-heart failure; and patients who are actively anticoagulated with therapeutic warfarin and go into labor before converting to heparin, to minimize the risk of fetal intracranial hemorrhage during vaginal transit [19].



Emergency LSCS: Managing Acute Decompensation

An emergency LSCS in a cardiac patient is a critical event that typically occurs due to non-reassuring fetal status or acute maternal cardiac decompensation during labor. If the patient has a working epidural, it should be carefully but rapidly topped up to establish surgical anesthesia. However, if a crash LSCS is required and no neuraxial access exists, general anesthesia is often chosen for speed and airway control.

The anesthesia team must prevent the catecholamine spike associated with intubation. A cardio vascularly stable induction technique using etomidate or tailored doses of propofol, combined with a high-dose short-acting opioid (such as remifentanyl or alfentanil), is used to blunt the hypertensive and tachycardic response. The neonatal team must be notified immediately to manage potential opioid-induced neonatal respiratory depression. The moment the placenta is delivered, a massive autotransfusion occurs. In both emergency and elective LSCS for cardiac patients, standard boluses of oxytocin are strictly contraindicated, as they cause severe peripheral vasodilation, profound hypotension, and reflex tachycardia. Instead, oxytocin must be given as a slow, low-dose controlled infusion (1–2 units over 5–10 minutes) or replaced with carefully titrated second-line uterotonics, alongside immediate administration of diuretics (such as furosemide) if central venous pressures spike [20].



Conclusion

The anesthetic management of the parturient with cardiac disease requires an understanding of underlying cardiovascular structures, lesion-specific hemodynamics, and the physiological demands of pregnancy. Effective management relies on early risk stratification using the mWHO classification, centralizing care within a dedicated cardio-obstetric team, and tailoring anesthetic interventions to preserve hemodynamic stability.

Whether implementing an incrementally titrated epidural for a 'Cardiac SVD' or executing a cardioprotective general anesthesia induction for an emergency LSCS, the primary objective remains the minimization of acute shifts in preload, afterload, and heart rate. Through precise multi-disciplinary planning and vigilant monitoring across the peripartum flashpoints, clinical teams can systematically mitigate risks, manage acute decompensation, and optimize safety outcomes for both mother and child.

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