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Anesthesia for pregnant women with valvular heart disease: the state-of-the-art

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Abstract
Pregnancy results in dramatic changes in the cardiovascular system. Maternal heart disease complicates 0.2%–3% of pregnancies. Valvular heart disease in women of reproductive age is most commonly due to rheumatic heart disease, endocarditis, or congenital abnormalities. In general, regurgitant lesions are well tolerated during pregnancy because the increased plasma volume and lowered systemic vascular resistance result in increased cardiac output. In contrast, stenotic valvular disease is poorly tolerated with advancing pregnancy, owing to the inability to increase cardiac output in relation to the increased plasma volume preload. The choice of anesthesia depends on the lesion and its severity. Usually, regional anesthesia provides the least amount of alteration in hemodynamics, although general anesthesia for cesarean section can be equally safe when the abrupt changes associated with laryngoscopy, intubation, and extubation are blunted by the appropriate choice of pharmacological agents and anesthetic techniques.

Key words Pregnancy · Heart disease · Rheumatic · Valvular · Aortic stenosis

Introduction

Pregnancy results in dramatic changes in the cardiovascular system. Maternal heart disease complicates 0.2%–3% of pregnancies [1]. The incidence of heart disease during pregnancy is steadily declining as a result of better medical and surgical care [2]. The pregnant woman with heart disease represents a unique challenge to the obstetrician and the obstetric anesthesiologist. Determination of the appropriate anesthetic modalities requires an understanding of the parturient's pathophysiology, as well as an understanding of pharmacological therapy and the potential for their interaction with peripartum anesthetic care.

Over the past 25 years, greater awareness of the physiological burden that pregnancy places on an already compromised cardiovascular system in this subset of pregnant women has led to better counseling before conception, and to major advances in treatment. Formerly, rheumatic heart disease was the most common cardiac disorder in pregnancy, with mitral stenosis the single most prevalent resulting lesion. Today, in general, the incidence of rheumatic heart disease has decreased dramatically in the United States and Western Europe, but in many other regions of the world it still remains an important cause of maternal morbidity and mortality [1,3–5].

The goal of this article is to review the current recommendations for the management of labor pain in women with valvular heart disease.

Cardiovascular changes during pregnancy

Anesthesiologists providing care to pregnant women should remember that pregnancy maximally dilates the uterine vasculature; so that autoregulation is absent, and uterine blood flow is entirely dependent on maternal mean arterial blood pressure (MAP). The compensatory cardiovascular changes during pregnancy may complicate the evaluation of intravascular blood volume and the assessment of peripartum blood loss [6]. Maternal hemodynamic measurements may not always accurately reflect the status of the uteroplacental circulation. Pregnancy represents a state of accelerated but

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compensated intravascular coagulation, which has both advantages and disadvantages for the parturient [7]. Increased levels of coagulation factors may improve hemostasis following obstetric bleeding; however, at the same time, parturients remain at increased risk for thromboembolic complications during periods of immobilization. Because buffering capacity during pregnancy is diminished, pregnant women rapidly develop metabolic acidosis during periods or hypoperfusion and hypoxia [1].

Four principal changes in the cardiovascular system during pregnancy that present unique problems to the parturient with underlying heart disease have been well delineated [1,8] and pose special anesthetic implications. First, there is a 50% increase in intravascular volume that generally peaks by the early-to-middle third trimester. Second, there is a progressive decrease in systemic vascular resistance (SVR) throughout pregnancy, so that MAP is preserved at normal values, despite a 30%–40% increase in cardiac output (CO). Systolic blood pressure (SBP) is normally decreased [6–8]. Third, the compromised cardiovascular system is further stressed by the marked fluctuations in CO observed during labor. Pain and apprehension may precipitate an increase in CO to as much as 40%–50% over those levels seen in the late second stage of labor [6]. Further, each uterine contraction serves, in effect, as an autotransfusion to the central blood volume, resulting in an increase in CO of 10%–25% [1]. The fourth consideration is the hypercoagulability associated with pregnancy and the possible need for appropriate anticoagulation, especially in those patients at increased risk for arterial thrombosis and embolization (prosthetic heart valve) [9–11].

Assessment of the parturient with heart disease

Antepartum and peripartum care of pregnant women with heart disease should include a joint obstetric, cardiological, and anesthetic evaluation of each parturient [12]. Optimal obstetric and anesthetic management requires a thorough assessment of the anatomic and functional capacity of the diseased heart, along with an analysis of how the described major physiological changes are likely to affect the specific limitations imposed by the intrinsic disease [1,6]. Specifically, to determine the most appropriate anesthetic regimen, the obstetric anesthesiologist must consider the following; first, the patient's tolerance to pain during labor, delivery, and/or surgery; second, the impact of uterine contraction–induced autotransfusion; third, the postpartum changes induced by the relief of vena cava obstruction; fourth, the potential for postpartum hemorrhage; and fifth, the use of uterine oxytocic agents.

The basic principles of obstetric anesthesia management must always apply [13–15]; first, provisions for the maintenance of uteroplacental perfusion by the avoidance of aortocaval compression; second, minimizing sympathetic blockade coupled with intravascular volume maintenance; third, monitoring of the parturient and the fetus; and fourth, provision for aspiration prophylaxis.

Peripartum considerations

Labor analgesia during the *first stage of labor* is focused on reducing the pain-related increases in catecholamine levels and avoiding aortocaval compression. Intravenous fluid management should be carefully monitored to avoid both a lack of and an excess of fluids [1]. Arterial, central venous, and/or pulmonary artery monitoring may be required to optimally manage the patient. However, such lines are generally reserved for symptomatic women and patients who have severe (narrowing to less than $1\,cm^2$) aortic stenosis or other hemodynamically significant valvular lesions.

Appropriate labor analgesia should be provided. Continuous lumbar epidural analgesia with local anesthetics, opioids, or both, is frequently optimal. Limited sympathetic blockade may prove helpful with mitral valve lesions because of the effect on both preload and afterload [1,6]. For a patient whose condition is so compromised that even the modest changes induced by segmental epidural analgesia are worrisome, the use of subarachnoid opioid analgesia by single injection or continuous catheter may be beneficial because the hemodynamic alterations of sympathetic blockade are avoided.

Once the patient with significant cardiac disease has entered the *second stage of labor*, it is prudent for her to avoid pushing (to avoid increase in CO). The lithotomy position may need to be avoided for patients with lesions such as mitral stenosis, inasmuch as this position results in an acute increase in central blood volume [1,6,16].

For second-stage management, analgesia for uterine contractions and anesthesia of the perineum are the objectives. Uterine contractions spontaneously bring the infant's head to a deliverable position, and delivery may then be assisted by the application of a vacuum extractor or forceps. Again, a regional technique is optimal. Epidural analgesia or anesthesia may be continued. If an epidural block is not used, a low spinal anesthetic may be appropriate. Pudendal nerve block, while not providing as complete analgesia as an epidural, may be employed as an adjunct to regional anesthesia, or used alone.

It is generally thought that cesarean section should be reserved for obstetric indications only, and that the presence of heart disease should not influence that decision. The choice of anesthesia depends on the lesion and its severity. Although spinal anesthesia may be appropriate for some patients with well-compensated lesions, adequate intravascular volume management to maintain maternal preload, SVR, and hemoglobin-oxygen saturation is necessary. Epidural anesthesia provides the least amount of alteration in hemodynamics during cesarean section, although general anesthesia can be equally safe when the abrupt changes associated with laryngoscopy and intubation, as well as suction and extubation, are blunted by the appropriate choice of pharmacological agents and anesthetic technique [1,6,17].

Valvular heart disease: general considerations

The presence of valvular heart disease due to either acquired or congenital etiology in a pregnant woman poses a clinical challenge to physicians involved in her antepartum and peripartum care [1,6,18–24]. Rheumatic fever continues to be the predominant etiology of valvular heart disease in pregnancy [1,15,18,20,22]. Complications during pregnancy include atrial dysrhythmias, univentricular or biventricular failure, systemic or pulmonary embolism, and infective endocarditis, with an overall incidence of complications estimated at 15% of all patients with valvular disease [1].

In general, regurgitant lesions are well tolerated during pregnancy because the increased plasma volume and lowered systemic vascular resistance result in increased CO [1,6,20]. In contrast; stenotic valvular disease is poorly tolerated with advancing pregnancy, owing to the inability to increase CO in relation to the increased plasma volume preload [1,6,20].

Specific valvular lesions

Mitral stenosis

Mitral stenosis accounts for nearly 90% of rheumatic heart lesions in pregnancy, with 25% of patients first experiencing symptoms during pregnancy [1,6,15]. Mitral stenosis may occur as an isolated lesion or in conjunction with other valvular disease. The principal pathophysiological derangement is a decrease in the area of the mitral valve, resulting in obstruction to left ventricular filling. This hemodynamic aberration leads to a relatively fixed CO. Although initially the left atrium may overcome this obstruction, with progression of the disease left atrial volume and pressure increase and lead to a progressive increase in pulmonary capillary wedge pressure and pulmonary venous pressure; pulmonary hypertension and right ventricular hypertrophy and failure may then occur [1,6,21–28]. Fetal mortality rates increase with deteriorating maternal condition and reach 25% in pregnant women with severe mitral stenosis.

Peripartum anesthetic management is oriented toward the avoidance of tachycardia, as the time required for left ventricular diastolic filling is prolonged in mitral stenosis. Patients who are asymptomatic at term generally require increased vigilance, but should not require invasive monitoring. Patients with marked symptoms are at significant risk in the peripartum period and should receive peripheral and pulmonary artery catheter monitoring continuing through a minimum of 24 h postpartum [1,6,15]. An increase in central circulating blood volume may occur suddenly in the immediate postpartum period, and tolerance of this intravascular load may be poor, especially by patients with a fixed CO [1,6,15].

Vaginal delivery is best accomplished with segmental lumbar epidural analgesia to minimize hemodynamic changes. Combined spinal-epidural analgesia (CSEA) is an attractive alternative to the conventional epidural block. A sudden decrease in SVR may be tolerated poorly following the development of reflex tachycardia [1,6,15]. The addition of opioids to the dilute local anesthetic mixture enhances the quality of labor analgesia, yet does not add to the sympathetic blockade. Opioids alone may be administered by the epidural or subarachnoid route for the critically ill patient. Adequate segmental and perineal anesthesia reduces catecholamine-induced increases in heart rate as well as the urge to push, allowing fetal descent to be accomplished by uterine contractions and avoiding the deleterious effects of the Valsalva maneuver during the second stage of labor [1,6,15]. When epidural anesthesia has not been used, a low spinal anesthetic may be administered to allow for a controlled second stage and delivery. Pudendal nerve block can provide adequate pain relief for some patients, and it may be suitable in selected situations where neuraxial anesthesia is contraindicated.

Cesarean section should be considered for obstetric indications. Anesthetic options for cesarean delivery must take into account the additional potential hazards of marked fluid shifts secondary to anesthetic technique, operative blood loss, and the mobilization of fluid in the postpartum period. Epidural anesthesia is preferred over spinal anesthesia because the former results in slower onset of blockade and, thus, more controllable hemodynamic alterations [1,6,15]. Prophylactic administration of ephedrine and arbitrary intravascular volume loading are best avoided; instead, a careful titration of anesthetic level allows judicious and appropriate

intravenous fluid administration, which should be guided by hemodynamic monitoring in the symptomatic patient. These patients may be prone to develop hypotension with epidural anesthesia secondary to a combination of venous pooling and prior beta-adrenergic blockade and diuretic therapy [1,15]. The usual vasopressor choice of ephedrine should be avoided, as it may result in tachycardia. Instead, judicious use of low-dose phenylephrine assists in restoration of the mother's blood pressure with little or no unwanted effect on uteroplacental perfusion.

Some patients with mitral stenosis may require general anesthesia. General anesthesia may also provide a very stable hemodynamic course if the sympathetic stimulation associated with laryngoscopy and intubation as well as with suction and extubation is minimized. This may be accomplished with anesthetic agents and/or beta-adrenergic blockade. Induction of general anesthesia should be carefully accomplished without drugs that commonly produce tachycardia [1,6,15]. Anesthesia is maintained with opioids, neuromuscular blocking drugs, nitrous oxide, and oxygen. Emergence must be carefully controlled to ensure avoidance of tachycardia.

Aortic stenosis

Congenital aortic defects are the usual causes of aortic stenosis in women of reproductive age [18]. Rheumatic disease-related aortic stenosis rarely complicates pregnancy, primarily because the natural history of this lesion occurring secondary to rheumatic heart disease typically requires three to four decades to achieve severity adequate to produce symptoms [1,15,18]. The pathophysiology of severe aortic stenosis entails narrowing of the valve area to less than $1\,cm^2$, associated with a transvalvular gradient of 50 mmHg, resulting in significant increases in valvular afterload to left ventricular ejection [18]. The left ventricle appropriately and concentrically hypertrophies and becomes markedly less compliant, although contractility is usually well preserved. The transvalvular gradient increases progressively throughout pregnancy as a result of increasing blood volume and decreasing SVR [29–31].

Peripartum anesthetic management encompasses the following goals: (1) avoiding both tachycardia and bradycardia; (2) maintaining adequate preload in order that the left ventricle may generate an adequate CO across the stenotic valve; and (3) maintaining hemodynamic parameters within a narrow therapeutic window. Patients with transvalvular gradients greater than 50 mmHg and patients with symptomatic aortic stenosis warrant invasive monitoring with peripheral and pulmonary artery catheters [1,6,15].

Labor analgesia with segmental epidural anesthesia or CSEA remains a controversial issue, because these patients may not be able to tolerate the decreases in preload and afterload due to sympathetic blockade [1,15,16]. Kuczkowski and Chow [16] described a case of a parturient with severe aortic stenosis who received uneventful CSEA with levobupivacaine and fentanyl for the first stage of labor; however, in the second stage of labor, cesarean section under general anesthesia was required for fetal indications (fetal distress). The authors concluded that the anesthetic management of a parturient with aortic stenosis must be based on individual assessment of cardiac function and reserve, and anticipation of the impact of the selected anesthetic technique (regional or general for vaginal or abdominal delivery) on cardiac performance [16].

Subarachnoid or epidural opioids, whether alone or in combination with an epidural segmental anesthetic, are other appropriate choices. Spinal opioids have no cardiovascular effects; myocardial contractility is unaltered, preload is preserved, and, most importantly, SVR is not diminished by this technique [1,15,16]. For cesarean section, either judiciously titrated epidural anesthesia or general anesthesia may be utilized. General anesthesia should be accomplished with the same caution that applies for parturients with mitral stenosis; myocardial depression associated with volatile anesthetics should be avoided [1,15,16].

Mitral regurgitation

Mitral regurgitation is the second most prevalent valvular lesion in pregnancy [1]. In young women, mitral regurgitation is most commonly due to mitral valve prolapse [18]. Chronic left ventricular volume overload and workload are usually well tolerated, with symptoms developing relatively late in life after childbearing age; thus, most patients with mitral regurgitation tolerate pregnancy well [15]. Complications include an increased risk of atrial fibrillation, bacterial endocarditis requiring antibiotic prophylaxis, systemic embolization, and pulmonary congestion during pregnancy. Congenital mitral valve prolapse is much more common among pregnant women than mitral regurgitation, and may be present in 10%–15% of pregnancies. It is a well-tolerated and generally benign form of mitral regurgitation [1,16].

The pathophysiology of regurgitation through an incompetent mitral valve results in chronic volume overload and dilatation of the left ventricle. If left ventricular compromise is sufficiently longstanding and severe, the increase in plasma volume with pregnancy progression may result in pulmonary venous congestion. By contrast, the decreasing SVR associated with pregnancy may serve to improve forward flow across the aortic valve at the expense of regurgitant flow. Increases in SVR that occur with labor pain, uterine contractions, or surgical stimulation may result in an increase in the

proportion of regurgitant blood flow, perhaps leading to acute left ventricular failure [1,15].

Provision of adequate labor analgesia should minimize the peripheral vasoconstriction and thus attenuate the increase in left ventricular afterload associated with labor pain. Sympathetic blockade also serves to decrease SVR and is beneficial in this regard; the caveat here is that venous capacitance will increase, and one must be prepared to augment preload cautiously to maintain left ventricular filling volume [15]. Asymptomatic patients at term are unlikely to require invasive monitoring. In symptomatic patients, invasive monitoring should be utilized.

Aortic regurgitation

In young women, aortic regurgitation may be congenital or acquired. If congenital, it is commonly associated with other lesions; if acquired, it may be secondary to rheumatic heart disease or endocarditis in association with aortic root dissection [1,6,15,18]. Symptoms following rheumatic fever usually develop during the fourth or fifth decade of life; thus, most women in whom this is the dominant lesion have uneventful pregnancies.

The basic pathophysiology is of chronic volume overloading of the left ventricle resulting in hypertrophy and dilation associated with increased compliance. Because of hypertrophy, myocardial oxygen requirements are higher than normal, yet perfusion pressure and, thus, oxygen supply may be decreased by a decrease in diastolic pressure and an increased left ventricular enddiastolic pressure [15].

Peripartum anesthetic considerations thus center on minimizing pain and, therefore, catecholamine-induced increases in SVR; avoiding bradycardia, which serves to increase time for regurgitant flow; and avoiding myocardial depressants, which may exacerbate failure. The anesthetic concerns are similar to those for patients with mitral regurgitation, and epidural anesthesia for labor and delivery is desirable in order to prevent increases in peripheral vasoconstriction [1,6,20]. Invasive monitoring is a requirement in any patient with symptoms of congestive heart failure [1,15].

Valve replacement and anticoagulation

Pregnant women who have undergone mitral or aortic valve replacement may present with complications such as thromboembolism, valvular outflow obstruction, endocarditis, and hemolysis [32,33]. Anticoagulation with coumarin derivatives is usually necessary to prevent the thromboembolitic complications. In pregnancy, heparin is usually substituted for coumarin anticoagulants to avoid congenital anomalies in the fetus [34]. Anticoagulation poses the threat of an epidural-spinal hematoma and is a contraindication to the use of regional anesthesia. The use of low-molecular-weight heparin (enoxaparin) has been associated with spinal epidural hematoma when regional anesthesia was used or attempted in Europe and, recently, in the United States [35]. This is probably due to its longer half-life by comparison with unfractionated heparin. It is therefore recommended that regional anesthesia not be administered unless the drug has been discontinued for at least 12–24 h, depending on the dosage. One alternative is to continue heparinization throughout labor and delivery and to use systemic analgesia for labor and general anesthesia for delivery. Another option with unfractionated heparin is to discontinue heparin therapy just prior to labor and delivery, normalize the coagulation parameters, use regional anesthesia, and restart heparin 12 h later.

Summary

Valvular heart disease can affect anyone at any time, and pregnant women are no exceptions. Pregnancy represents a significant physiological burden for women with underlying valvular heart disease [1]. The successful management of pregnant women with valvular heart disease depends on the cooperative efforts of a multidisciplinary team of experts, including the obstetrician, the cardiologist, and the obstetric anesthesiologist among many others involved in the peripartum care of these patients. A comprehensive understanding of the physiology of pregnancy and the pathophysiology of underlying valvular heart defects is of primary importance in the obstetric and anesthetic management of this high-risk group of parturients [6]. Timely provision of labor analgesia and adequate peripartum monitoring of hemodynamic parameters is essential for the reduction of maternal morbidity and mortality.

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