Anesthesia for the parturient with cardiovascular disease

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Introduction

The pregnant patient with heart disease represents a unique challenge to the anesthesiologist. Determination of the appropriate analgesic and anesthetic modalities requires a thorough understanding of the parturient's pathophysiology as well as pharmacological therapy and how these interact with anesthetic care.

Over the past two decades, greater awareness of the physiologic burden that pregnancy places on an already compromised cardiovascular system in this subset of pregnant women has led to more accurate counseling before conception and 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.[1] In general, the incidence of rheumatic heart disease has decreased, but in some regions it has risen again with the recent influx of immigrants from Asia. Also, many more women with congenital heart defects are reaching childbearing age, a consequence of surgical correction.[2] As more women delay childbearing to later reproductive years, ischemic cardiac disease may be expected to become increasingly prevalent.[3,4]

GENERAL CONSIDERATIONS

Pregnancy normally results in dramatic changes in the cardiovascular system. Four principal changes that present unique problems to the patient with cardiac disease have been well delineated[5] and have special anesthetic implications.

First, there is a 50% increase in intravascular volume that generally peaks by the early-to-middle third trimester. This relative volume overload may be poorly tolerated in patients whose cardiac output is limited by myocardial dysfunction from ischemia or intrinsic or valvular lesions.

Second, there is a progressive decrease in systemic vascular resistance (SVR) throughout pregnancy, so that mean arterial pressure (MAP) is preserved at normal values despite a 30-40% increase in cardiac output. This may be of importance in those patients at risk for right-to-left shunting as well as for patients with some types of valvular disease (e.g., aortic stenosis).

Third, the compromised cardiovascular system is further stressed by the marked fluctuations in cardiac output observed during labor. Pain and apprehension may precipitate an increase in cardiac output to as much as 45-50% 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 cardiac output of 10-25%.[7] The Valsalva maneuver results in wide swings in both venous and arterial pressures, which have been associated with acute cardiac decompensation. The increases in cardiac output reach a maximum of 80% higher than antepartum levels immediately following delivery secondary to relief of inferior vena cava obstruction and a final autotransfusion of approximately 500 ml from uterine contraction.

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 or chronic atrial fibrillation). Therapeutic anticoagulation affects the options for anesthetic management, perhaps the location of invasive monitors, and increases the risk of postpartum hemorrhage.

Optimal 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 physiologic changes are likely to affect the specific limitations imposed by the intrinsic disease. Specifically, to determine the most appropriate anesthetic regimen, the anesthesiologist must consider the following:

1. Patient's tolerance to pain during labor or surgery.
2. Impact of uterine contraction-induced autotransfusion.
3. Postpartum changes induced by relief of vena caval obstruction.
4. Potential for postpartum hemorrhage.
5. Use of uterine oxytocic agents.

The most basic principles of obstetric anesthesia management must always apply[8]:

1. Provisions for maintenance of uteroplacental perfusion by avoidance of aortocaval compression.
2. Minimizing sympathetic blockade coupled with intravascular volume maintenance.
3. Standard-of-care monitoring of parturient and fetus.
4. Provision for aspiration prophylaxis.

Analgesia during the first stage of labor is focused on reducing the pain-related rises in catecholamine levels and avoiding aortocaval compression. Intravenous fluid management should be carefully

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monitored to avoid both a lack of and excess of fluids. Arterial, central venous, and/or pulmonary artery monitoring may be required to optimally manage the patient. Although such lines are generally reserved for symptomatic women, patients who have "tight" aortic stenosis, coarctation of the aorta, aortic aneurysm, right-to-left shunts, or primary pulmonary hypertension may benefit from invasive monitoring even with minimal symptoms.

Appropriate analgesia should be supplied. All of the available modalities have application for some patients. Continuous lumbar epidural analgesia with local anesthetics, narcotics, or both is frequently optimal. Limited sympathetic blockade may prove helpful with mitral valve lesions because of the effect on both preload and afterload. For a patient whose condition is so compromised that even the modest changes induced by segmental epidural analgesia are worrisome, the use of intrathecal narcotic analgesia by single injection or continuous catheter may be beneficial because all of 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. 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.

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 the vacuum extractor or forceps. Again, a regional technique is optimal. Epidural analgesia or anesthesia may be continued. Attention must be paid to extension of the sympathetic blockade. 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 satisfactorily employed as an adjunct to regional anesthesia, or used alone.

It is generally thought that cesarean delivery should be reserved for obstetric indications only and that the presence of heart disease should not influence that decision. The overall stresses of labor and vaginal delivery, as measured by alterations in cardiac output, are approximately the same as with cesarean section. However, some circumstances might lead to the decision to perform an elective cesarean section. The choice of anesthesia depends on the lesion and its severity. Epidural anesthesia provides the least amount of alteration in hemodynamics during cesarean section, although general anesthesia can be equally as 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.

CARDIAC SURGERY DURING PREGNANCY

Surgery of any type is usually avoided during pregnancy because of potential compromise to the unborn infant. The developing fetus may experience teratogenic effects from drugs administered during the course of anesthesia. This has been demonstrated in animals with a multitude of anesthetic adjuvants but has never been clearly documented in humans.[9-11] Teratogenic changes may also be induced by hypoxia during the procedure or by decreased uteroplacental perfusion. Premature labor is often associated with surgery during pregnancy, particularly abdominal procedures.[12,13] Thus, if surgery is indicated during the course of pregnancy, it is usually performed during the second trimester whenever possible. This avoids the period of organogenesis and premature labor is said to be less likely. Many centers utilize tocolytic therapy as part of their routine for the surgical patient who is pregnant. While this may be useful for patients without cardiovascular disease, most of these drugs have potent cardiovascular side effects and would prove less desirable or the patient with heart disease.[14]

The pregnant patient with heart disease is often managed with medical therapy including long periods of bed rest if necessary. If a surgical lesion is present, every attempt is made to delay the definitive procedure until after delivery of the infant. However, there are patients who decompensate so severely from the cardiovascular stress imposed by pregnancy that their chance of survival is very small unless surgical correction is attempted. These patients usually suffer from rheumatic valvular disease, most often mitral stenosis.

The first cardiac operations performed during pregnancy were closed mitral commissurotomies for severe congestive heart failure due to mitral stenosis in 1952. A review of 514 cases by Ueland, revealed a maternal mortality of 1.75% and a fetal loss of 8.6%. This was quite favorable when compared to a maternal mortality of 4.2 - 18.7% in pregnant patients with NYSHA Class III and IV cardiac disease managed with medical therapy. The fetal loss in this group of patients was approximately 50%. The extremely good surgical survival figures are probably due to the fact that these patients are young with relatively healthy hearts, which have been overburdened by the circulatory changes of pregnancy and did not involve cardiopulmonary bypass. The use of cardiopulmonary bypass for open heart procedures in the pregnant patient soon followed. The risk for both mother and fetus increases with this more complex procedure. A multi-institutional study by Zitnik et al revealed a 5% maternal mortality and a fetal loss of 33%. More current data indicate a maternal mortality of 0 - 2.9% and a fetal loss of 20%.[15] The high fetal loss has been attributed to several factors, which could affect fetal oxygen delivery during cardiopulmonary bypass. These include non-pulsatile perfusion, inadequate perfusion pressure, embolic events to the uteroplacental circulation, and disturbance of uterplacental blood flow by cannulae, release of catecholamines and renin, and hypothermia.[16] Some of these potential hazards can be avoided by the use of fetal monitoring during the operation.

Several reports of the benefit of fetal monitoring during cardiopulmonary bypass have appeared in the literature.[17,18] In one instance a fetal heart rate of 60 was restored to 100 by an increase in pump flow from 3100 to 3600 ml/minute.[18] In addition to identifying potentially threatening events, all have reported a sustained fetal bradycardia to between 80 and 100 beats per minute during hypothermic cardiopulmonary bypass, which resolves with the restoration of maternal temperature and normal circulation. A recent metaanalysis revealed that fetal mortality was reduced to 0% when normotheric cardiopulmonary bypass was utilized.[15]

Based on the available knowledge of the physiologic changes of pregnancy, the pharmacology of drugs employed during cardiopulmonary bypass, the physiology of extracorporeal circulation, and experiences reported the following recommendations for cardiopulmonary bypass during pregnancy could be made. Although it would be desirable to avoid such surgery until after pregnancy, no pregnant patient should be denied a definitive operation because of gestation. Whenever possible the period of organogenesis should be avoided and the second trimester favored. If cardiopulmonary bypass is required after 28 weeks gestation, it has been suggested that cesarean section immediately prior to the cardiac surgery is a reasonable and safe procedure.[16] We believe that hemostasis must be meticulous in this case as full anticoagulation is necessary.

Every effort to ensure adequate fetal oxygenation and perfusion during the procedure should be exercised. Maternal inspired oxygen

concentration should be maintained as high as possible and arterial blood gases checked frequently. Maternal ventilation should be adjusted to avoid respiratory alkalosis as this causes a shift in the oxyhemoglobin dissociation curve to the left thus potentially decreasing oxygen transport to the fetus. Aortocaval compression must be minimized by utilizing a wedge to provide left uterine displacement if the patient is at 20 weeks of gestation or greater. Calculation of pump flows should include a 30 - 50% increase over normal to compensate for the increase in cardiac output that occurs with pregnancy. Pomini recommends maintaining flows at 2.7 L/m2/min or greater.[15] Perfusion pressures of 60 mm Hg or greater appear optimal for maintaining uteroplacental perfusion. Perfusion times should be kept to a minimum and normothermic bypass utilized whenever possible. Electrolyte balance should be maintained and the impact of vasopressors on the uteroplacental circulation considered before their institution. Electronic fetal monitoring should be used with a member of the health care team experienced in its interpretation available. Monitoring of uterine activity may also be desirable as increased uterine activity has been associated with cardiopulmonary bypass.[16]

VALVULAR HEART DISEASE

Rheumatic fever continues to be the predominant etiology of valvular heart disease in pregnancy. Complications during pregnancy include univentricular or biventricular failure, atrial dysrhythmias, systemic or pulmonary embolism, and infective endocarditis, with an overall incidence of complications estimated at 15% of all patients with valvular disease. In general, regurgitant lesions are well tolerated during pregnancy because the increased plasma volume and lowered systemic vascular resistance results in increased cardiac output. In contrast, stenotic valvular disease is poorly tolerated with advancing pregnancy, owing to the inability to increase cardiac output sufficient to accommodate the augmented plasma volume; this situation leads to pulmonary venous congestion and, possibly, frank pulmonary edema.

Mitral Stenosis

Mitral stenosis may occur as an isolated lesion or in conjunction with right-sided or aortic valvular disease. It accounts for nearly 90% of rheumatic heart disease in pregnancy, with 25% of patients first experiencing symptoms during pregnancy.[19] The principal pathophysiologic derangement is a decrease in mitral valve orifice, resulting in obstruction to left ventricular filling. This hemodynamic aberration leads to a relatively fixed cardiac output. Although initially the left atrium may overcome this obstruction, with progression of disease left atrial volume and pressure ultimately increase and lead to a progressive and chronic rise in pulmonary capillary wedge pressure and pulmonary venous pressure; pulmonary hypertension and right ventricular hypertrophy and failure may ensue. An anatomically moderate lesion may become functionally severe with the marked increase in cardiac output that accompanies normal pregnancy, labor, and delivery.

Anesthetic management is oriented toward the avoidance of tachycardia, as the time required for left ventricular diastolic filling is prolonged. 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 arterial and pulmonary artery catheter monitoring continuing through a minimum of 24 hours post partum.[20] 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 for patients with a fixed cardiac output.[6,21]

Anesthesia for labor and vaginal delivery may be best accomplished with segmental lumbar epidural anesthesia to avoid changes in monitored hemodynamic parameters. A sudden decrease in SVR may be tolerated poorly following the development of reflex tachycardia. Although other analgesic modalities may be employed, segmental epidural analgesia allows for careful titration to the desired result while minimizing undesirable changes. The addition of opioids, such as fentanyl, to the dilute local anesthetic mixture enhances the quality of analgesia yet does not add to the sympathetic blockade. Opioids alone may be administered by the epidural or intrathecal 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. When epidural anesthesia has not been used, a low spinal anesthetic may be administered to allow for a controlled second stage and delivery.

Caudal anesthesia is another reasonable option. Pudendal nerve block can provide adequate, although not ideal, pain relief for some patients.

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. Either regional or general anesthesia may be used. Epidural anesthesia is preferred over spinal anesthesia because the former results in slower onset of blockade and thus more controllable hemodynamic alterations. Prophylactic 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 hypotension with epidural anesthesia, secondary to a combination of venous pooling and prior beta-adrenergic blockade and diuretic therapy.[22] The usual vasopressor choice of ephedrine should be avoided, as it may result in tachycardia. Instead, judicious use of metaraminol or low-dose (20-40 mcg) phenylephrine assists in restoration of maternal blood pressure with little or no unwanted effect on uteroplacental perfusion.

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. Depending on the severity of the disease, the need to blunt the hemodynamic response to endotracheal intubation may necessitate the use of a high-dose narcotic-based technique. This also serves to avoid myocardial depression and the decreases in SVR that may occur with commonly employed short-acting barbiturates. Anesthesia is maintained with narcotics, muscle relaxants, nitrous oxide, and oxygen. Emergence must be carefully controlled to ensure return of protective reflexes and avoidance of tachycardia.

Aortic Stenosis

Aortic stenosis is a rare complication of pregnancy, primarily because the natural history of this lesion occurring secondary to rheumatic heart disease typically requires three to four decades to yield severity adequate to produce symptoms. However, women with congenitally bicuspid aortic valves and patients with a history of bacterial endocarditis may present in pregnancy with severe aortic stenosis. Unlike mitral stenosis, symptoms of congestive heart failure, an-

gina, and syncope develop relatively late in the course of the disease. The pathophysiology of severe aortic stenosis entails narrowing of the valve orifice to less than 1 cm^2, associated with a transvalvular gradient of 50 mm Hg, resulting in significant increases in afterload to left ventricular ejection. 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.[23]

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 cardiac output across the stenotic valve.
3. Maintaining hemodynamic parameters within a narrow therapeutic window.

Patients with transvalvular gradients greater than 50 mm Hg and patients with symptomatic aortic stenosis warrant invasive monitoring with arterial and pulmonary artery catheters in the peripartum period.[24]

Provision of labor analgesia with segmental epidural anesthesia remains a controversial issue because these patients may not be able to tolerate the decreases in preload and afterload that may attend epidural analgesia and its associated sympathetic blockade.[19] Easterling et al. described a series of four patients with moderate to severe aortic stenosis who were successfully managed with epidural anesthesia without untoward sequelae; adequate time was allowed to carefully titrate the level of block and initiate appropriate compensatory actions to correct hemodynamic alterations associated with the anesthetic agent. Intrathecal or epidural opioids, whether alone or in combination with an epidural segmental anesthetic, are other appropriate choices. Spinal opioids have no cardiovascular effects. In particular, myocardial contractility is unaltered, preload is preserved, and, most importantly, SVR is not diminished by this technique.[25] Local anesthetics and opioids are believed to act synergistically, allowing for a decrease in concentration of both drugs when used together. Effective analgesia can prevent the tachycardia associated with labor pain.

For cesarean section, either judiciously titrated epidural anesthesia or general endotracheal anesthesia may be utilized. General anesthesia may be accomplished with the same caution that applies for parturients with mitral stenosis; myocardial depression associated with halogenated volatile anesthetics should be avoided.

Mitral Insufficiency

Mitral valve insufficiency and regurgitation is the second most prevalent valvular lesion in pregnancy. Chronic left ventricular volume overload and work are usually well tolerated, with symptoms developing relatively late in life after childbearing age; thus, most patients with mitral regurgitation tolerate pregnancy well. Complications include an increased risk of atrial fibrillation during pregnancy, bacterial endocarditis requiring antibiotic prophylaxis, systemic embolization, and pulmonary congestion during pregnancy.

In one review of maternal deaths associated with rheumatic valvular lesions, no patient died from complications of mitral regurgitation without the presence of coexisting mitral stenosis.[26] Congenital mitral valve prolapse is much more common during pregnancy than mitral regurgitation and may be present in 10-17% of pregnancies. It is a well-tolerated and generally benign form of mitral regurgitation, and therapeutic interventions are thus rarely necessary.[27,28]

The pathophysiology of regurgitation through an incompetent mitral valve results in chronic volume overload of the left ventricle and dilatation. If left ventricular compromise is sufficiently long-standing 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, which occur with labor pain, uterine contractions, or surgical stimulation, may result in a rise in the proportion of regurgitant blood flow, perhaps leading to acute left ventricular failure.

Anesthetic management of labor and delivery can be safely provided via any of the available techniques, including segmental lumbar epidural anesthesia. Adequate analgesia and anesthesia minimize the peripheral vasoconstriction and thus attenuate the increase in left ventricular afterload associated with labor pain and thereby augment the forward flow of blood. 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 with intravenous fluid infusion to maintain left ventricular filling volume.

Asymptomatic patients at term are unlikely to require invasive monitoring. Continuous electrocardiographic (ECG) monitoring is a reasonable addition to basic standards of peripartum monitoring. In symptomatic patients, invasive monitoring with arterial and pulmonary arterial catheter should be utilized.

Aortic Insufficiency

Aortic insufficiency 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. 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 end-diastolic pressure.

Anesthetic considerations thus center on:
1. Minimizing pain and, therefore, catecholamine-induced increases in SVR.
2. Avoiding bradycardia, which serves to increase time for regurgitant flow.
3. Avoiding myocardial depressants, which may exacerbate failure.

Because the anesthetic concerns are similar to those for patients with mitral regurgitation, epidural anesthesia for labor and delivery is desirable in order to prevent increases in peripheral vasoconstriction. Epidural anesthesia is also appropriate as a surgical anesthetic agent, as is general anesthesia with judicious avoidance of direct myocardial depressants. Invasive monitoring is a requirement in any patient with symptoms of congestive heart failure.

The parturient who has undergone mitral or aortic valve replacement faces several potential problems, such as thromboembolism, valvular outflow obstruction, endocarditis, and hemolysis. These patients have received anticoagulation, usually with coumarin derivatives, to prevent the thrombotic problems mentioned. Heparin is usually substituted for coumarin anticoagulants during pregnancy to avoid potential congenital anomalies.[29] Full anticoagulation is a direct con-

traindication to the use of regional anesthesia because of the risk of causing epidural or spinal hematoma. 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.[30] This is probably due to its longer half-life than unfractionated heparin. It is therefore recommended that regional anesthesia not be administered unless the drug has been discontinued for at least 12 - 24 hours depending upon the dosage. One alternative is to continue heparinization throughout labor and delivery and 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 results, use regional anesthesia, and restart heparin 12 hours later. This may not be practical in an obstetric setting. The choice depends on the severity of the patient's hemodynamic derangement and the optimal analgesic management.

CONGENITAL HEART DISEASE

Congenital heart disease is becoming the most common cardiac problem encountered in the pregnant patient.[28] Patients are increasingly likely to survive to childbearing age with the advent of palliative surgery or total correction of their defects. Many of these patients can be expected to have an uneventful pregnancy and delivery.

Left-to-Right Shunts

Ventricular septal defect (VSD) occurs in 7% of adults with congenital heart disease. Patients with uncorrected lesions in the absence of pulmonary hypertension do well during pregnancy. In the small percentage of patients with large VSDs and coexisting pulmonary hypertension, maternal mortality ranges from 7-40%. Severe right ventricular failure with shunt reversal (Eisenmenger's syndrome) is the major ensuing complication. During pregnancy, elevation of plasma volume, cardiac output, and heart rate may increase left-to-right shunt and may further worsen the degree of pulmonary hypertension.

The major goals in peripartum management include awareness that marked increases in peripheral vascular resistance and heart rate may be poorly tolerated, with ventricular failure a distinct possibility. Conversely, acute increases in pulmonary vascular resistance (PVR) and right ventricular compromise may lead to shunt reversal and hypoxia.

Anesthesia for labor and vaginal delivery is optimally achieved with segmental epidural anesthesia consisting of local anesthetics, opioids, or their combination to permit control of painful stimuli, thus minimizing changes in heart rate and SVR. Anesthesia for cesarean section may be accomplished either with slow titration of an epidural anesthetic agent to allow time for correction of pressure changes or with a general anesthetic agent that combines opioid and inhalation technique to depress the adrenergic response to endotracheal intubation and minimize myocardial depression.

Atrial septal defect is one of the most common congenital cardiac lesions in women of childbearing age, and pregnancy is generally well tolerated even when pulmonary blood flow is increased. However, the risk of left ventricular failure is increased during pregnancy. Increases in atrial volume result in biatrial enlargement, and thus supraventricular dysrhythmias are likely.

Pregnancy-associated increases in plasma volume and cardiac output serve to accentuate the left-to-right shunt, right ventricular volume work, and pulmonary blood flow, with the possible development of pulmonary hypertension and left and right ventricular failure. Peripartum management centers on avoiding vascular resistance changes that may increase the degree of shunt. Increases in SVR or decreases in PVR may not be well tolerated.

Although all of the common methods of providing labor analgesia are useful, lumbar epidural analgesia for labor, vaginal delivery, or cesarean section attenuates the hazards of increased SVR. General anesthesia for cesarean section is also well tolerated, provided that increases in SVR are avoided and sinus rhythm is maintained.

Patent ductus arteriosus (PDA) accounts for 15% of all cases of congenital heart disease; today most patients with a large PDA (>1 cm) receive early surgical intervention. Patients with a small PDA have typically normal pregnancies, but in those pregnant women with superimposed pulmonary hypertension, maternal mortality may reach 5-6% from ventricular failure. The progressive decrease in SVR development throughout pregnancy can be associated with shunt reversal and peripheral cyanosis.

Anesthetic considerations include avoidance of increases in SVR and hypervolemia. Conversely, acute decreases in SVR may result in reversal of shunt in patients with preexisting pulmonary hypertension and right ventricular compromise. Again, all modalities may be used, depending on the severity of the disease. Continuous lumbar epidural analgesia for labor and delivery diminishes the increase in SVR associated with pain. Epidural or general anesthesia is appropriate for cesarean section if increases in SVR associated with endotracheal intubation and surgical stimulation are adequately addressed.

Right-to-Left Shunts

Eisenmenger's syndrome consists of pulmonary hypertension, a right-to-left intracardiac shunt resulting from pulmonary hypertension superimposed on a previously left-to-right shunt, and arterial hypoxemia. Pregnancy is not well tolerated by patients in this condition. The maternal mortality rate is estimated at 30-50%. This entity is responsible for approximately 50% of the maternal mortality in parturients with congenital heart disease.[2]

Anesthetic considerations center on avoidance of any decrease in SVR and, therefore, hypotension or myocardial depression. Hypotension from any cause, including conduction block or hemorrhage, can progress to insufficient right ventricular pressures to perfuse the hypertensive pulmonary arterial bed and may result in sudden death.

Analgesia for vaginal delivery may be accomplished with systemic narcotics, intrathecal narcotics, or cautious application of a segmental epidural if SVR is maintained. During the first stage of labor, epidural or intrathecal opioid administration would be a useful adjunct, and its sole administration has been recommended as the safest approach.[31] If an epidural block is employed, epinephrine should be omitted from the test dose because peripheral beta-adrenergic effects may cause a decrease in SVR.

For analgesia and anesthesia in the second stage of labor, a caudal epidural block may be preferable to the lumbar route because dense perineal analgesia can be provided without extensive sympathetic blockade. Delivery by cesarean section is most safely accomplished via general anesthesia, although regional anesthesia for elective cesarean delivery has been reported.[32,33]

Regardless of the anesthetic technique employed, the postpartum period is probably the most likely time for life-threatening complications of hypoxemia, cardiac dysrhythmias, and thromboembolic events to occur[34]; most maternal deaths, in fact, occur in the first postpartum week.[35] The use of invasive monitoring is highly recommended in management of these patients in the peripartum period; pulmonary artery catheters and serial arterial blood gas determinations may allow early detection of changes in cardiac output, pulmonary artery pressures, and shunt fraction. Serial measurements of cardiac output and especially SVR are useful in this regard. It should be recognized

that technical difficulty in passage of a pulmonary artery catheter and obtaining wedge pressures is well documented and a central venous catheter may have to suffice.[36,37]

Tetralogy of Fallot comprises 15% of all congenital heart disease and is the most common etiologic factor in right-to-left shunt in women of childbearing age. Particularly poor prognostic signs include a history of syncope, polycythemia (hematocrit > 60), decreased arterial oxygen saturation (<80%), right ventricular hypertension, and congestive heart failure. Increased right-to-left shunt may accompany pregnancy-induced decreases in SVR. The stress of labor may increase PVR and thus increase the degree of shunt. Most complications occur in the postpartum period, when SVR is lowest, thus exacerbating the right-to-left shunting of blood and worsening the degree of arterial hypoxemia.

Anesthetic considerations must focus on minimizing the hemodynamic changes that would exacerbate the degree of shunt. Strict avoidance of decreased SVR and decreased venous return and myocardial depression are of paramount importance. Analgesia for labor and vaginal delivery in these patients is most safely provided by systemic medication, inhalational nitrous oxide analgesia, or pudendal block. Intrathecal opioids may prove optimal in some circumstances. Regional anesthetic techniques should be used with extreme caution because the decrease in SVR may result in increased shunt. Low-dose ketamine may prove a reasonable option for forceps-assisted deliveries. Anesthesia for cesarean section should be provided by general anesthesia.

Invasive monitoring with arterial and pulmonary artery catheters to evaluate cardiac filling pressures and SVR is warranted in those patients with uncorrected tetralogy or only palliative correction.

Primary pulmonary hypertension predominantly affects women of childbearing age and is associated with a high maternal mortality (>50%). Most deaths occur during labor and the puerperium.[38-40] Signs and symptoms depend on severity of the disease and are caused by a fixed low cardiac output, the degree of pulmonary hypertension, and the degree of right ventricular compromise.

Anesthetic considerations are focused on:

1. Evaluating the severity of the disease and its responsiveness to therapy.
2. Maintaining hemodynamic stability.
3. Administering the appropriate analgesia and anesthesia for labor and delivery.

In selecting an analgesic or anesthetic regimen, the anesthesiologist must primarily consider the prevention of increases in PVR from underventilation, pharmacological agents, pulmonary hyperinflation, and stress. Furthermore, decreases in right ventricular volume from intravascular volume depletion, venodilation, or aortocaval compression are poorly tolerated. Significant decreases in SVR from sympathetic blockade from regional anesthesia or volatile anesthetic agents may produce severe decompensation because the cardiovascular system may be unable to compensate for the decline in afterload.[39] Finally, right ventricular contractility may be marginal, and the addition of negative inotropic anesthetic agents may lead to marked depression in cardiac function. The parturient should be monitored with an ECG, pulse oximetry, radial artery catheter, and a pulmonary artery catheter throughout labor and the postpartum period. The latter allows early detection of changes in PVR or right ventricular function and serves as a guide to fluid and pharmacological therapy.

Labor and vaginal delivery may best be managed by the judicious use of systemic narcotic analgesics and pudendal nerve block. Epidural analgesia with local anesthesia may be provided only if the block is slowly titrated in a limited dermatomal fashion from T10 to L1 to avoid extensive sympathetic blockade. Intrathecal or epidural opioids also provide effective first-stage analgesia. Vaginal delivery can be managed by the addition of a caudal catheter or pudendal block.

Regional anesthesia is best avoided for cesarean delivery, and a slow induction with either high-dose narcotics or an inhalation agent is recommended. This is necessary to avoid marked increases in PVR with laryngoscopy. Cricoid pressure must be maintained throughout the induction to prevent the aspiration of gastric contents. Ventilation must be adequate, but pulmonary hyperinflation must be avoided. Uterine stimulants may best be omitted because they can be associated with significant elevations in PVR.

CORONARY ARTERY DISEASE

Coronary artery disease is uncommon in women of childbearing age, with a reported incidence of 1 in 10,000 pregnancies. In one review, it was determined that only 13% of gravidas who had a myocardial infarction (MI) had a known history of coronary artery disease; overall maternal mortality was 37%, increasing to 45% if the infarction occurred in the third trimester.[41,42]

The pathophysiology and clinical manifestations are identical to those in the nonpregnant patient.[43] It should be noted that the hemodynamic demands that pregnancy places on the myocardium represent a stress to the coronary circulation. General management guidelines currently include efforts to reduce the cardiac work load with measures such as bed rest, nitrate therapy for preload reduction, and conduction anesthesia during delivery. Cardiac medications, such as beta blockers and nitrates, should be continued throughout the pregnancy, labor, delivery, and puerperium. Effort must be directed toward optimizing myocardial oxygen supply; supplemental oxygen should be provided, anemia treated, and respiratory depression secondary to sedation meticulously avoided.

Although reasonable pain relief can be achieved with systemic narcotic analgesia, the early institution of continuous regional anesthesia for labor and delivery is recommended to minimize the pain and stress that have the potential to precipitate ischemia and angina.[44] Beneficial effects associated with regional anesthesia may also include decreased preload and afterload so that myocardial work is diminished. Marked and sudden decreases in afterload must be avoided because coronary artery perfusion is dependent on diastolic pressure. Also, significant decreases in SVR can precipitate reflex tachycardia, which may increase cardiac workload sufficiently to produce ischemia. Epidural anesthesia effectively attenuates the progressive rise in central venous pressure and cardiac output that occurs during labor in the unanesthetized parturient. Multiple-lead ECG monitoring should be instituted early in labor so that ischemia can be promptly detected and treated.

When one is establishing epidural blockade, it is recommended that epinephrine be omitted from the test dose to avoid potential tachycardia and that the block be established by administration of slower-onset local anesthetic agents, such as bupivacaine or ropivacaine. Additionally, supplementation of a dilute local anesthetic solution with an epidural opioid has been advocated.[45] Fetal descent during the second stage of labor should be by force of uterine contraction, with avoidance of the Valsalva maneuver, according to the patient's baseline ejection fraction and analysis of the hemodynamic response to contractions. When epidural analgesia for the first stage of labor is not employed, a low spinal anesthetic (saddle block) provides excellent conditions for an assisted delivery with minimal hemodynamic trespass.

Elective cesarean section can be safely performed with a slowly titrated level of epidural anesthesia, allowing judicious intravenous fluid infusion to maintain pulmonary capillary wedge pressure and blood pressure.[46] Spinal anesthesia is much less desirable, given the rapid onset of sympathetic block with great potential for hypotension and reflex tachycardia. When administering general anesthesia for cesarean section, the anesthesiologist must take into consideration the importance of minimizing the cardiovascular response to the stress of endotracheal intubation and surgery. In the absence of congestive heart failure, an inhalation technique is recommended.

A history of recent (especially third-trimester) MI of less than 6 weeks, congestive heart failure, or unstable or crescendo angina warrants invasive monitoring with arterial and pulmonary artery catheters.[43] Monitoring should be continued for a minimum of 24 hours into the postpartum period to assess increases in pulmonary capillary wedge pressure as intravascular volume increases following delivery and as anesthesia subsides.

IDIOPATHIC HYPERTROPHIC SUBAORTIC STENOSIS

Idiopathic hypertrophic subaortic stenosis (IHSS) or asymmetric septal hypertrophy (ASH) or is a disease without a defined etiology, but at least one third of the subjects have a familial history and it appears to be inherited as an autosomal dominant trait. The primary features of this cardiomyopathy include marked hypertrophy of the left ventricle and interventricular septum and obstruction of the left ventricular outflow tract during systole by the hypertrophied muscle. The anterior leaflet of the mitral valve may be displaced by the hypertrophied muscle and thus contribute to the obstruction in some patients.

The disease commonly presents during the second to fourth decades of life. Common symptoms include angina pectoris, dizziness, and exertional dyspnea. Physical findings include signs of left ventricular hypertrophy, a systolic ejection murmur, and a third heart sound. The ECG will indicate left ventricular hypertrophy and, in many cases, evidence of Wolff-Parkinson-White syndrome and evidence of abnormal Q waves. There is wide variability in both the findings and the symptoms of the disease.

The hemodynamic limitations of IHSS are produced as the ventricle contracts. The hypertrophied walls narrow the outflow region during systole. The determinants of the degree of obstruction are the volume of the left ventricle at systole, the force of left ventricular contraction, and the degree of left ventricular distention during systolic contraction. The patient therefore requires a high preload in order to maintain a full left ventricle, a reduced contractile force in order to minimize outflow tract narrowing, and a high systemic vascular resistance to maintain distention of the left ventricle during systole.

Therapy is primarily focused on the administration of beta-adrenergic blocking agents to reduce myocardial contractility and heart rate. Some patients may receive calcium channel-blocking drugs as well. Patients with IHSS do not tolerate hypovolemia, decreased SVR, or increases in myocardial contractility very well. The cardiovascular and hemodynamic changes of pregnancy have a variable effect on patients with IHSS, depending on both the severity and nature of the disorder. The increase in blood volume associated with pregnancy should yield a beneficial effect as it increases preload. The usually observed increase in heart rate and stroke volume during pregnancy can have a negative effect, and the decrease in SVR, which begins during the second trimester, may also have a negative impact on cardiac performance. Although the potential for left ventricular failure and cardiac arrhythmias during pregnancy exists, the outcome of patients with IHSS has been reasonably good.[47]

The therapeutic objectives during parturition should be as follows:

1. Minimizing pain-associated increases in catecholamine levels.
2. Maintaining preload by adequate intravenous fluid administration.
3. Avoiding the Valsalva maneuver, which abruptly decreases preload.

Invasive monitoring with both an arterial line and a pulmonary artery catheter will yield the information necessary to provide precise management. Recommendations for analgesia during the first stage of labor have been to utilize systemic narcotics, inhaled analgesics, or paracervical block. Regional analgesia has been considered a substantial risk because of the potential for both venodilation (decreased preload) and arterial dilation resulting in decreased SVR.[47] This can possibly be avoided, however, if careful incremental titration of continuous lumbar epidural analgesia is carried out. A limited segmental level of analgesia from T10 to L2 provides adequate analgesia with minimal sympathetic blockade, thus preserving preload. Dilute solutions of a local anesthetic agent with the addition of a narcotic, such as fentanyl, provide optimal analgesia.[48] Intrathecal narcotics may also be used, thereby eliminating the risk of sympathetic blockade but adding the potential side effects of respiratory depression, pruritus, and nausea, all of which are easily treated.

A combined spinal and epidural analgesic approach has been used successfully for a patient with IHSS.[49] Vaginal delivery may be accomplished with pudendal block, carefully extended epidural analgesia, or low spinal anesthesia (saddle block). The saddle block involves the spinal segments from L2 to S5 and thus avoids the majority of sympathetic nerve elements. Regional anesthesia is effective at blocking the uncontrollable urge to bear down. If hypotension necessitating a vasopressor does occur, the use of ephedrine is relatively contraindicated because it causes tachycardia and increased myocardial contractility. Metaraminol or a pure vasoconstricting drug, such as phenylephrine (20-40 mcg), should be employed in the lowest effective doses to minimize its effect on the uterine arteries.

Anesthesia for cesarean delivery offers additional challenges. Left uterine displacement must be maintained and volume requirements carefully assessed in view of the greater blood loss. Invasive monitoring will be needed. Regional anesthesia is usually avoided for the aforementioned reasons, and clearly the level of anesthesia required is likely to produce extensive sympathetic blockade with undesirable consequences.[47,50] Nonetheless, a carefully titrated epidural anesthetic with ongoing compensation for the induced hemodynamic changes may prove acceptable. General anesthesia is preferred by many, although the ideal technique is yet to be established and experience is limited.[51] Although the use of volatile anesthetic agents is advantageous because they reduce myocardial contractility, they also decrease uterine contractility and SVR. Modest doses should have a minimal effect on both.

As with the preeclamptic patient, the stimulating effects of laryngoscopy and intubation need to be pharmacologically blunted. Oxytocin must be administered cautiously because it tends to decrease SVR and results in tachycardia when administered rapidly. The parturient with IHSS requires careful attention by means of appropriate monitoring and the immediate availability of the necessary vasopressors, beta-blockers, and intravenous volume expanders.

CONCLUSION

The successful management of the pregnant cardiac patient depends on the cooperative efforts of the obstetrician, the cardiologist and the anesthesiologist involved in peripartum care. A comprehensive understanding of physiology of pregnancy and pathophysiology of underlying cardiac disease is of primary importance in obstetric and anesthetic management of this high-risk group of parturients.

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