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Anesthesia for severe hypertensive disease of pregnancy and ischemic heart disease

Alison MacArthur, M.D.*

* Department of Anesthesia, Mount Sinai Hospital, Toronto, Ontario, Canada.

INTRODUCTION

Severe hypertensive disorders during pregnancy and ischemic heart disease are two unique cardiovascular diseases that complicate maternal health during pregnancy and particularly during delivery. These women may be unable to face the physiological changes of pregnancy that often necessitate additional energy expenditure (increased oxygen consumption).

The cardiovascular system's changes begin in the first three months of pregnancy (first trimester). Increases in maternal heart rate begin between 5 - 8 weeks gestation with heart rates reaching 17 - 29% above pre-pregnancy values[1,2]. The volume of blood pumped with every heart beat (stroke volume) begins to increase at 8 weeks gestation, eventually to levels 22 - 32% above pre-pregnancy values by the end of the pregnancy. Thus, both parameters which determine cardiac output (stroke volume and heart rate) contribute to an increasing cardiac output of pregnancy (cardiac output = heart rate multiplied by stroke volume in milliliters of blood pumped per minute). Cardiac output increases approximately 35-40% during the first trimester, and continues increasing until approximately 33 - 36 weeks gestation (53 - 65% overall increase)[3].

The amount of energy expended by the heart is determined by its cardiac output, which increases to provide sufficient metabolic substrates for maternal physiological changes and fetal development. There are physiological adaptations, which reduce the amount of work of the heart during pregnancy, including reductions in systemic peripheral resistance that falls by 30% from preconception levels at 8 weeks gestation[]. In addition, the heart's contractility improves during pregnancy due to increased left ventricular wall thickness and mass. Anatomical changes which accompany physiological changes include, increased valve areas, and movement of the heart in a cephalad, anterior direction due to elevation of the diaphragm.

During labor and delivery, further demands are placed upon the cardiovascular system. With each uterine contraction cardiac output increases from an average of 7.23 litres/minute to 8.65 litres/minute. The blood delivered into the circulation by the contraction of the uterine smooth muscle is on average 500 ml/uterine contraction. These initial findings from 1950's studies using dye dilution techniques have been confirmed recently using Doppler echocardiography techniques. Robson et al demonstrated that cardiac output not only increased during contractions, but also depended upon the stage of labor[5]. As the cervix progressively dilated, the change in cardiac output associated with each contraction increased from 1.14 L/min ($\leq$ 3 cm dilated) to 1.75 l/min (4-7 cm dilated) and finally to 2.69 L/min at dilation $\geq$ 8 cm.

Immediately following delivery hemodynamic changes begin to return maternal physiology to non-pregnant conditions[6]. Maternal heart rate falls within the first hour postpartum, followed by diminishing stroke volume, mean arterial pressure and cardiac output. By two weeks postpartum many cardiovascular changes have almost returned to pre-pregnancy values. However, left ventricular thickness and mass are significantly increased above pre-conception values at 24 weeks postpartum. Despite increased left ventricular size, myocardial contractility appears to diminish in the postpartum period[6].

The Anesthesiologist's role in care of these patients ranges from optimizing patients' hemodynamics prior to elective delivery to provision of acute resuscitative care around

imminent delivery and unstable cardiovascular presentations. In this discussion I will review the following information for each disorder:

1. Epidemiology of disorder – incidence, risk factors
2. Pathophysiology of disease and relevance to anesthesia
3. Obstetric goals for mothers with this disorder
4. Anesthetic goals for labor and delivery

PREGNANCY INDUCED HYPERTENSION

1. Epidemiology

Pregnancy induced hypertension (PIH) is a maternal disease which spares no country, socioeconomic class or age group. It is defined by the development of blood pressure greater than 140 mmHg (systolic) or 90 mmHg (diastolic) on at least 2 measurements more than 4 hours apart after 20 weeks gestation. The presence of proteinuria (> 300 mg per 24 hours) is common, however mandatory for its diagnosis. One of the common causes of maternal morbidity and mortality PIH incidence is estimated at 2-7% of all pregnancies. The disease occurs more frequently in women with chronic hypertension, type I & II diabetes mellitus, previous preeclampsia, multiple gestation, thrombophilias, maternal obesity, and women at extremes of maternal ages.

The etiology of PIH is complex, and results in a heterogenous disorder with significant maternal dysfunction, or significant fetal dysfunction, near term gestation or presenting as a second trimester disease. The pathogenesis of pregnancy induced hypertension is likely the result of immune maladaptation which results in both pathological inflammatory and ischemic states. The ultimate result is development of a hypertensive maternal inflammatory response and/or a state of fetal intrauterine growth restriction[7].

The following table illustrates some of the associated complications of PIH and their frequency of occurrence:

Panel 1: Maternal and fetal complications in severe pre-eclampsia

Maternal complications

- **Abruptio placentae (1-4%)**
- **Disseminated coagulopathy/HELLP syndrome (10-20%)**
- **Pulmonary oedema/aspiration (2-5%)**
- **Acute renal failure (1-5%)**
- **Eclampsia (<1%)**
- **Liver failure of haemorrhage (<1%)**
- **Stroke (rare)**
- **Death (rare)**
- **Long-term cardiovascular morbidity**

Neonatal complications

- **Preterm delivery (15-67%)**
- **Fetal growth restriction (10-25%)**
- **Hypoxia-neurologic injury (<1%)**
- **Perinatal death (1-2%)**
- **Long-term cardiovascular morbidity associated with low birthweight (fetal origin of adult disease)**

Magnitude of risk depends on gestational age at time of diagnosis, delivery, severity of disease process, and presence of associated medical disorders.

2. Pathophysiology

The pathological features of pre-eclampsia that concern to anesthetists include changes to the respiratory, cardiovascular, hematological, neurological and hepato-renal systems. The changes that can increase anesthesia-related morbidity or mortality include:

a. Reductions in plasma colloid oncotic pressures are more common in pre-eclampsia than normal pregnancy, and coupled with increased vascular permeability can result in fluid third-spacing. Increased extra-vascular fluid within the lungs can predispose the parturient to impaired oxygen exchange or pulmonary edema, and soft tissue swelling can increase the incidence of airway difficulties at time of intubation.

b. Increased mean arterial pressures arise from either increased cardiac output (primarily stroke volume increases), increased systemic vascular resistance or combination of the two. The end result is an increase in myocardial oxygen demand, which in combination with the pain of labor may exceed the delivery of oxygen to the myocardium. Women with this disease are at an increased incidence for high-output cardiovascular disease, including myocardial ischemia and cerebrovascular hemorrhage.

c. The potential coagulopathies that can accompany the disease may interfere with the ability to provide central axial anesthesia. Disturbances of platelet counts, platelet function or disseminated intravascular coagulation can increase the risk for excessive bleeding. In the presence of a coagulopathy, anesthetists are concerned over an increased risk of epidural hematoma formation leading to spinal cord compression and permanent paralysis. There are two periods of risk; the first at the time of epidural or spinal anesthetic insertion and the second during the removal of epidural catheters.

d. Significant end organ dysfunction involving kidneys and liver can occur in cases of severe preeclampsia. The development of oliguria warrants immediate assessment of intravascular volume. The usual etiology of reduced urine output is intravascular hypovolemia which responds to mild fluid challenges (250 – 500 ml intravenous isotonic fluid

bolus over one hour). However, renal failure can occur in the absence of hypovolemia, and continued re-hydration attempts may precipitate pulmonary edema. Diminished renal clearance can unexpectedly prolong serum levels of anesthetic agents that are dependent upon renal excretion, predominantly muscle relaxants used during general anesthesia. Liver dysfunction is uncommon with preeclampsia unless the HELLP syndrome develops. In this case, the parturient who develops subcapsular hematomas or hepatic rupture can present with acute hypovolemic shock.

c. Massive placental abruption can accompany this disease leading to diminished fetal reserve or demise requiring urgent delivery. As well, the normal coagulation cascade can be exaggerated leading to secondary disseminated intravascular coagulation (DIC).

f. Central nervous system dysfunction associated with preeclampsia include CNS irritability (headaches, hyperreflexia, visual disturbances), seizures, and intracranial hemorrhage. Any of these dysfunctions indicate severe maternal disease and necessitate modification of anesthetic management. The parturient may require evaluation of adequacy in protection of her airway from aspiration (appropriate level of consciousness), reduction of paralyzing agents used during general anesthesia due to interactions with magnesium sulphate therapy, or evaluation and documentation of normal intracranial function/intracranil pressure prior to the administration of central-axial anesthesia (spinal or epidural).

3. Obstetric goals for women with severe preeclampsia

Obstetric management of this disease depends upon the severity of the maternal disease and the gestational age at which the disease occurs. The development of PIH late in pregnancy (> 38 weeks) or > 34 weeks gestation with severe disease dictates delivery of fetus. Most obstetricians will opt for trial of labor as over 45% of inductions will be successful with a vaginal delivery[8]. For women < 34 weeks gestation the course of management depends upon whether the mother's medical condition can allow continuation of the pregnancy. Delivery is indicated if imminent eclampsia, multiorgan dysfunction, severe intrauterine growth restriction, suspected abruption or non-reassuring fetal assessment. However, if maternal condition is stable and fetal assessment is reassuring then expectant management of pregnancy may be indicated. This continuation of the pregnancy is controversial and awaits further study to decide whether the potential risk for the mother is balanced by an obvious benefit to the fetus[9].

The most recent research projects of therapeutic interventions for women with PIH have raised the question of benefit in treating mothers with antihypertensives and the benefit of magnesium sulfate infusions in prevention of eclampsia[10]. Antihypertensives appear to reduce the incidence of maternal cerebrovascular and myocardial complications, but do not alter the course of PIH nor improve the fetal well being[11].

4. Anesthetic goals for labor and delivery

Anesthesia for Labor

The goal of labor pain relief for preeclamptic parturients is to provide the most effective analgesia to reduce the stimulation of the sympathetic nervous system. Prior to epidural analgesia being proven safe for use in this population in the 1980's, other pain-relieving modalities were used. Concern for the use of epidurals for labor arose over the potential for maternal hypotension, an event that would be deleterious in women with reduced uteroplacental reserve[12]. Although women with preeclampsia have exaggerated hypertensive responses to vasopressor medications and pain, they do not appear to have the same lability in hypotensive responses to regional anesthesia as compared to normal pregnant women[13-16]. However, alternative techniques for labor pain relief have largely failed because they are inadequate analgesics compared to the "gold standard" of epidural analgesia. However, alternative analgesic techniques may have to be employed when a contraindication exists for epidural or spinal labor analgesia.

Nitrous oxide & systemic narcotics

Methods for labor pain relief that do not require particular technical expertise include the use of nitrous oxide with supplemental oxygen and systemic narcotics. The nitrous oxide (N_2O) – oxygen mixture contains 50% N_2O gas with 50% O_2 gas. Therefore the mother receives increased oxygen during the periods of inhalation. If this is timed to coincide with uterine contractions, the mother is receiving increasing her oxygen delivery during the period of increased oxygen demand. Most nitrous oxide delivery systems for maternity units require a close fitting of a mask to the mother's face during inhalation. Otherwise, the one-way valve in the system (designed to reduce occupational exposure to nitrous oxide gases) does not open to allow the flow of gases. The pulmonary elimination of nitrous oxide from the maternal and fetal systems is rapid due to its low insolubility in the blood. Therefore, once the parturient ceases to use the gas, it is quickly exhaled in the breaths following. Nitrous oxide, however, is a poor analgesic as judged by studies assessing the pain scores of laboring women. It appears to provide a mood-altering quality that allows parturients to tolerate pain. The time span of its effectiveness is quite short, in that parturients often will abandon the technique after an hour. Nitrous oxide would likely be most useful in a multip-

arous preeclamptic parturient with previous rapid labor histories. As well, this modality could be used as an adjunct to other pain-relieving techniques such as a pudendal nerve block or local infiltration of perineum.

Systemic opioids are the most common form of labor analgesia in the world and when administered by the best possible delivery system, can be the second most efficacious form of pain relief. Intramuscularly administered narcotics is a poor method of drug delivery for labor pain as relatively slow absorption leads to fluctuating peaks and troughs in serum levels. As well, this method of administration is painful and is contraindicated when the patient is thrombocytopenic (< 50,000 – 75,000 platelets). Intravenous administration of narcotics is the best method of delivering narcotics for the pain of labor. Ideally, the patient controlled analgesia (PCA) system would be available for these patients. This system contains a computerized delivery system to connect to the intravenous line that allows patient demand to access the supplied narcotic. Safety features include barriers to access the narcotic supply and limits to the amount of narcotic administered. Particular narcotics used in this system for obstetrics patients include morphine, fentanyl[17], meperidine[18], nalbuphine, remifentanil[19] and alfentanil[20]. The ideal choice of agent is a short-acting narcotic that does not accumulate over time and has a large therapeutic to toxic plasma ratio. Regardless of the drug chosen, particular monitoring requirements must be in place to avoid potential maternal and neonatal complications. Therefore, the minimum monitoring for the mother during administration is hourly respiratory rate and sedation score. Respiratory rates below 10 breaths per minute between contractions or women who are only rousable with stimulation require further oxygen saturation monitoring and prophylactic oxygen administration. When magnesium sulfate prophylaxis is concurrently administered, the preeclamptic parturient is at greater risk for excessive respiratory depression with the use of systemic narcotics. All narcotics cross the placenta and therefore will be present in the neonatal bloodstream depending upon concentration and timing of the narcotic used. At the time of delivery, personnel responsible for neonatal resuscitation should be informed of the use of maternal narcotics and should be familiar with reversing narcotic sedation in the newborn. Monitoring guidelines for nurses, preprinted physician order sheets, and pharmacy-provided narcotic mixtures are useful to reduce human errors.

Paracervical & pudendal blocks

Analgesic techniques that require specific training and whose success depends upon the frequent use include: paracervical block, pudendal nerve block, epidural block (including lumbar and caudal epidural blocks) and spinal block. The paracervical block is the administration of local anesthesia between the 4 to 5 o'clock and 7 to 8 o'clock position of the lateral cervical surface to block the pain of cervical dilatation. It is not recommended for patients delivering a viable fetus as local anesthesia uptake can compromise uteroplacental blood flow. This technique is recommended as a first stage analgesia for mothers with nonviable fetus, for postpartum dilatation & curettage, or repair of cervical lacerations. Pudendal nerve blocks are useful for the pain of vaginal and perineal distension during second stage, however the block must be successfully placed bilaterally, and have sufficient time for the local anesthetic effect prior to delivery. It is ideally placed when the fetal head is at or above the ischial spines, to allow proper deposition of the local anesthetic agent. The pudendal block's length of action is time-limited (lidocaine duration of action 60 - 120 minutes; bupivicaine 90 - 200 minutes). With proper effect, the pudendal block can provide excellent analgesia for operative vaginal deliveries.

Epidurals & CSE

Central axial blocks for labor analgesia (epidurals, combined spinal epidurals) in preeclamptics have become the standard of practice over the past twenty years. Provided there are no contraindications to these blocks, such as coagulation abnormalities, overwhelming maternal septicemia, or ongoing acute blood loss, continuous lumbar epidural analgesia has many advantages. It is the most effective and reliable method of pain relief of labor with a low incidence of complications. During labor, epidural analgesia decreases maternal oxygen requirements and prevents maternal hyperventilation. Often in pre-eclampsia, maternal levels of catecholamines are elevated above normal, which can impair uteroplacental perfusion. As well, effective analgesia blunts hypertensive and neuroendocrine responses to labor pain[21], which may reduce the risk of eclampsia[22]. Another benefit of this technique is that the level of anesthesia can quickly be altered for operative vaginal delivery or for cesarean delivery. Lastly, randomized clinical trial evidence has demonstrated that epidural analgesia does not increase the incidence of cesarean delivery in women with preeclampsia[23-25].

Institution of epidural analgesia requires assessment for coagulopathies due to the concern for epidural hematoma. Epidural anesthesia and to a lesser extent spinal anesthesia have been associated with the development of epidural hematomas and permanent neurological deficits in the presence of coagulopathic conditions. This is an extremely rare event, and therefore difficult to define the exact risk for each anti-coagulant. There are reported cases in the obstetric population, but the relative risk in this population compared to other patients receiving anticoagulation is not known at this time[26]. The period of greatest risk are the institution of epidural or spinal anes-

thesia and on removal of epidural catheters. Future methods for screening may include thromboelastography, the results of which can be immediately processed and available with blood sampling. Parturients who have been on anticoagulants prior to labor require a window of time between the last dose and administration of central axial anesthesia. The recommendation for pregnant patient care is based upon current anesthesia guidelines established for all patients[27]. For patients receiving normal heparin (unfractionated heparin) subcutaneous prophylactic doses do not contraindicate the use of epidural or spinal anesthesia, however normal platelet count must be checked for due to the occasional development of heparin-induced thrombocytopenia (HIT). Therapeutic anticoagulation with normal heparin requires cessation of therapy and normalization of laboratory PTT findings prior to insertion of central axial anesthesia (usually within 6 hours of stopping heparin infusion). The use of low molecular weight heparin (LMWH) prophyllaxis or therapy requires the longest waiting period from last dose (12 hours following low dose prophylactic therapy or 24 hours from higher dose therapy such as enoxaparin 1.5 mg/kg daily or dalteparin 200 U/kg daily or tinzaparin 175 U/kg daily). The initiation of postpartum low molecular weight heparin also requires manipulation of the epidural catheter to reduce risk on removal. The use of twice daily dosing of LMWH has an increased risk of epidural hematoma and therefore the catheter should be removed at least two hours prior to the first postpartum dose. Single daily dosing of LMWH allows for the epidural catheter to remain inplace for postpartum analgesia continuation while starting the anticoagulation, however the catheter can only be removed following a 10 – 12 hour window from the last dose.

The optimal time to administer epidural analgesia during a woman's labor must be individualised. The delivering physician with the patient must consider the potential urgency for delivery, the certainty that induction of labor will continue to delivery and not be stopped, and the history of the fetal and placental well being. There is no evidence to suggest an optimal point in labor to administer epidural analgesia.

Operative vaginal delivery

Epidural or spinal analgesia can prevent the sometimes-uncontrollable urge to bear down which is associated with sudden rises in venous pressures in the cardiac and central nervous system. The raised venous pressures may reduce perfusion through these capillary beds, as well in other susceptible organs (placenta). This method of analgesia also minimizes the likelihood of a precipitous delivery of a preterm or small neonate. If vaginal delivery is imminent, spinal block can safely provide rapid and complete analgesia for vaginal delivery while maintaining motor strength and response. This is achieved by the use of small spinal doses of local anesthesia mixed with narcotics. Again, as with every institution of central axial anesthesia, appropriate left uterine displacement is mandatory, intravenous access to the patient, and monitoring on the patient to identify hypotension and heart rate. The combined spinal epidural technique allows prolongation or extension of the initial block if necessary. Often with preeclamptic parturients epidural analgesia has been instituted earlier in labor, and thus a top-up is required for perineal analgesia prior to the operative vaginal delivery. Epidural top-ups require 5 to 20 minutes to achieve an effective response, depending upon the volume and specific local anesthestic administered.

Anesthesia for cesarean delivery

There are essentially three methods of anesthesia available for a cesarean delivery in the preeclamptic patient: epidural, spinal and general anesthesia. The most common method provided for this population is epidural anesthesia, as most patients have received an epidural during their trial of labor and have progressed onto the cesarean delivery. In this scenario, the epidural catheter in place is topped up with a more concentrated local anesthetic and requires additional time (15 - 30 minutes) to achieve maximal anesthetic effect. The anesthetist can speed up the onset of local anesthetic effect with specific agents such as lidocaine and chlorprocaine, or by the alterations of the agent's pH (lidocaine + CO_2 is faster in onset than plain lidocaine). In the case of an emergent cesarean delivery, where waiting until full effect is impossible, the anesthetist must choose between two options: providing intravenous sedation until the epidural dose is effective or administering general anesthesia. Other limitations of the epidural technique include increased unpredictability of the anesthetic when compared to spinal anesthesia or general anesthesia, and requirement for large doses of local anesthesia, which may approach toxic levels.

Prior to the late 1990's spinal anesthesia for cesarean delivery was largely avoided in preeclamptic patients because of the concern over precipitous maternal hypotension as compared to epidural anesthesia. However, observational and randomized clinical trials have provided sufficient evidence that the benefits of spinal anesthesia are significant and that the technique is safe[28-30]. This anesthetic technique can provide almost instantaneous anesthesia with minimal doses of local anesthesia, and is more appropriate in patients with questionable coagulation problems (the potential for epidural bleeding is less with a small, midline-placed spinal needle than placement of an epidural catheter). Thus one recent advantage of spinal anesthesia has been to reduce the incidence of general anesthesia. Spinal anesthesia has been reported in preeclamptic patients with central nervous system disease (eclampsia and transient blindness) when the presence of raised intracranial pressure has been ruled out[31,32].

The administration of general anesthesia to a preeclamptic parturient is one of the most challenging moments to an obstetric anesthetist. The difficulties faced include the potential for difficult intubation (accompanying mucosal swelling of the pharynx may interfere with clear visualization of the larynx, or prevent normal passage of the endotracheal tube through swollen vocal cords), the potential for severe hypertension during laryngoscopy and at time of surgical stimulation and the impairment of intervillous blood flow by positive pressure ventilation. Anesthetic goals include adequate assessment of the airway prior to induction of general anesthesia, preparation for blunting hemodynamic perturbations, and maximizing oxygen delivery for mother and baby. In the face of a potentially difficult intubation the anesthetist's first obligation is to not place the mother at further risk. Therefore, a more careful airway assessment (direct laryngoscopy following airway topicalisation with local anesthetic sprays) may be required to determine whether general anesthesia can be induced prior to securing the airway or after (awake intubation). These decisions are often required during a period of stress on all caregivers, as this event most commonly occurs with significant fetal bradycardias. Whenever possible, general anesthesia should be avoided in parturients with identified difficult airways.

The consequences of a sudden hypertensive crisis under general anesthesia include intracranial hemorrhage, myocardial ischemia and arrthymias and pulmonary edema. Identification of neurological complications is not detectable under general anesthesia, however cardiac and respiratory changes are usually identified with ECG, oxygen saturation and exhaled gas carbon dioxide monitoring. Induction of general anesthesia is often modified with pharmacologic agents that will attenuate maternal hypertension. The agents should be intravenously administered for quickest onset of action, should have a short duration of action in case of sudden hypotension, and should not negatively affect the fetus if they cross the placenta. Suitable agents include nitroglycerine, trimethaphan, labetolol, and narcotics such as fentanyl, sufentanil and remifentanyl. In the case of narcotics, the fetus may need respiratory assistance at birth and reversal of prolonged opiate effect with naloxone. The anesthetist, as with all cesarean deliveries, will be unable to provide primary neonatal resuscitation measures while responsible for the mother.

ISCHEMIC HEART DISEASE

1. Epidemiology

Ischemic heart disease during pregnancy is rare disorder, however, with aging maternal populations and the increasing prevalence of other associated medical disorders in the pregnant population, most anesthesiologists will care for at least one patient during their career. The incidence of myocardial ischemia during pregnancy has been estimated between 0.6 – 2.8 events per 100,000 women delivering[33-36].

Risk factors for development of this disorder are more difficult to ascertain, as individual factors have only been evaluated in two population-based studies[24,25]. The majority of women suffering peripartum myocardial ischemic events are over the age of 30 years, and have greater likelihood of accompanying medical diseases, including type I & II diabetes, pregnancy-induced hypertension, pre-existing hypertension, hyperlipidemia and coronary artery disease. Although the prevalence of these conditions is increasing amongst the pregnant population we were unable to demonstrate a statistical increase over time in the incidence of ischemic heart disease (between the years 1970 and 1998)[24] .Survival of mother and fetus are adversely affected by ischemic events, with approximately 2 – 19% of women and 3 – 10% of fetuses dying.

2. Pathophysiology

Information on the individual pathophysiology of this disease is not available on a population level, however reviews of case reports in the literature can be used to obtain some information on the anatomy of the disease[37,38]. A substantial proportion (42.6%) of the women in Badui's study had none of the traditionally described risk factors for myocardial infarction[26] .Within our study, 67/114 women lacked identifiable risk factors for ischemic heart disease[24]. The location of the myocardial infarction was primarily in the anterior wall (alone or in combination with other anatomic regions).

Comparison of two literature reviews of peripartum myocardial infarctions

Variable	Roth et al [60] N = 125 women	Badui et al [58] N = 136 women
Age – years (range)	32 ± 6 (16 – 45)	32 ± 8 (16 – 45)
Multiparous patients	93/111 (84%)	97/136 (72%)
MI Location – anterior	89/122 (73%)	95/136 (70%)
Coronary angiogram performed:	n = 68	n = 55
Normal	20 (29%)	26 (47%)
Stenosis	29 (43%)	11 (20%)
Thrombus	14 (21%)	9 (16%)
Coronary artery dissection	11 (16%)	6 (11%)

The timing of ischemic events during the pregnancy is distributed between the antepartum (38%), intrapartum (21%) and postpartum (41%) periods[25]. Over 60% of events occur during the peak cardiovascular demands of labor and

delivery, or during the period of autotransfusion and fluid loading in the postpartum period.

The etiology of the peripartum myocardial ischemia leading to infarction appeared to be different than ischemic heart disease occurring in older age groups. Less than half of the women had a coronary angiography performed in conjunction to the infarct event, and in a significant proportion, the results were normal. Typical angiographic findings were narrowing of arteries (11/55 women), and thrombosis (9/55 women). Peripartum myocardial ischemic disease may be a different entity compared to ischemic disease in older populations, and may not have the associated abnormal coronary artery pathology or significant mortality rates. Reports of normal coronary arteries ischemic heart disease is a increasingly frequent topic in the literature[39,40].

Postulated mechanisms of action for the development of myocardial ischemia include coronary artery spasm (induced with exposure to ingested agents such as cocaine, alcohol, or nicotine), or spontaneous coronary artery dissection. The survival rates for patients with normal coronary arteries following myocardial infarction is favorable, reported between 85 – 96%[41,42].

3. Obstetric management of women with ischemic heart disease

Obstetric management for women with ischemic heart disease will depend upon the severity of the disease, the gestational period in which the ischemic event occurs, and the fetal response to the insult. The literature is replete with case reports of women who have significant disease requiring invasive therapy (including angioplasty[43], stent placement[44], cardiopulmonary bypass surgery[45] and thrombolysis with tissue plasminogen activator[46], and in these cases, the health of the mother takes precedence over the pregnancy. With development of acute left ventricular dysfunction, rapid delivery of the fetus may improve cardiopulmonary performance. There are two case reports of women with symptoms of pulmonary edema secondary to LV dysfunction who improved following urgent cesaresan delivery[47,48].

However, for many women with antenatal events, the obstetric management does not deviate from usual practice, and cesarean delivery is reserved for obstetric indications or where there is an immediate threat to maternal well-being. Elective cesarean delivery may allow for better organized management of anesthesia and obstetric practice, however the patient must recover from more significant post-operative morbidity.

4. Anesthetic Management of parturients with ischemic heart disease

The same goals that guide anesthetic management of non-obstetric patients are use for the pregnant patient. Oxygen delivery to the myocardium is maximized with optimal heart rates controlled with the use of beta blocker therapy, including labetelol; by supplementing maternal oxygenation with inspired oxygen therapy during labor; and maintaining coronary artery perfusion pressure by avoiding maternal hypotension (adequate aortocaval decompression at all times, and adequate vasopressor therapy if sudden vasodilation with regional anesthesia). Oxygen consumption is minimized by preventing excessive maternal catecholamines associated with labor pain, so all parturients laboring with ischemic heart disease should receive early epidural analgesia. This analgesic will also reduce inadvertent early expulsive efforts of second stage, and will facilitate vacuum or forceps-assisted vaginal deliveries.

Cesarean delivery more commonly occurs in women with ischemic heart disease[24]. For women with ischemic heart events prior to delivery, this likely represents a reluctance to allow a prolonged labor in a woman with reduced cardiovascular reserve. As well, women with intrapartum and postpartum ischemic events are more likely to have delivered by cesarean, raising the possibility that the increased physiological adaptations to the surgery and anesthesia may be more poorly tolerated compared to a vaginal delivery. Monitoring during cesarean delivery is often more complex for these patients, and involves multiple lead ECG monitoring during and after the surgery, invasive arterial blood pressure monitoring during and after surgery, and possibly central venous pressure monitoring in the presence of significant ventricular dysfunction.

The choice of general or regional anesthesia should be dictated by the urgency of the delivery, as well as the cardiovascular stability of the mother. Regional anesthesia improves the balance of myocardial work and oxygen delivery by the reduction in systemic vascular resistance and improved rheology of blood flow with systemic vasodilation. However, safe general anesthesia can be provided to these patients with the adherence to safe principles. The generally accepted principle of securing the airway quickly using a rapid sequence induction must be balanced against the need to completely attenuate the cardiovascular response to intubation. Patients with easy airways and expected easy ventilation may be best managed with a modified rapid sequence, where cricoid pressure is maintained while ventilation is allowed during the titration of adequate IV anesthesia. Choices for rapid achievement of adequate anesthetic levels while maintaining hemodynamics include narcotic-based techniques (remifentanyl infusions), or infusions of anesthetic agents such as propofol. In these cases, the patient's blood pressure and heart rate are kept within pre-decided ranges on induction of anesthesia, and require invasive monitor placement prior to induction. Rapidly acting agents to reduce heart rate, such as esmolol, and maintain blood pressure, such as phenylephrine, are ideal. These agents can be quickly stopped when effect is achieved.

Regional anesthesia techniques include the use of epidural and spinal anesthesia. Controversy may exist with the use of spinal anesthesia, however this reliable and quick anesthetic can be used safely with anesthetic attention to cardiovascular parameters. Again, patients receiving spinal anesthesia would ideally have invasive blood pressure monitoring placed prior to administration of local anesthetics. The combined spinal-epidural technique for cesarean delivery is attractive as the spinal effect can be used for the operative delivery, and the epidural component use for administration of excellent postpartum analgesia. Our choice is to administer our usual epidural labor anesthetic solution (bupivicaine 0.0625% with fentanyl 2 µg/ml) with a patient-controlled epidural pump set with both an hourly infusion and bolus capabilities.

The postpartum patient with ischemic heart disease in our institution would be cared for in either the labor and delivery unit with one-to-one nursing or in the coronary care unit (CCU). The best site for these patients requires continuous ECG monitoring for peri-operative arrhythmia detection, and access to nursing personnel capable of responding to cardiovascular collapse and monitoring of usual post-cesarean complications. Often critical care nurses and obstetric nurses co-manage these patients.

Cardiac resuscitation and pregnancy

A recent review article defined succinctly the difficulties in successfully resuscitating a women during cardiopulmonary arrest[49]. "Factors peculiar to pregnancy that weigh the balance against survival include anatomical changes that make it difficult to maintain a clear airway and perform intubation, pathological changes such as laryngeal oedema, physiological factors such as increased oxygen consumption, and an increased likelihood of pulmonary aspiration. In the third trimester the most important factor is compression of the inferior vena cava and impairment of venous return by the gravid uterus when the woman lies supine. These difficulties may be exaggerated by obesity". All cardiac resuscitation medications and procedures are essentially unchanged for pregnancy. The only medication recommendation from this review was the avoidance of lidocaine in the management of tachyarrthymias, if the patient had already received large doses of local anesthesia for a regional block.

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