ESTIMATES suggest that 1% or 2% of pregnant women undergo anesthesia for surgical procedures unrelated to delivery in the United States, but pregnancy may be unrecognized at the time of surgery, and there are no formal reporting mechanisms for data collection. Cerclage procedures for cervical incompetence typically are performed at the end of the first trimester. Most nonobstetric procedures result from circumstances common for the maternal age group, such as appendicitis, cholelithiasis, ovarian cysts or ovarian torsion, breast tumors, trauma, and more rarely for life-threatening cardiac or neurosurgical conditions or organ transplantation.

Anesthetic considerations for surgery during pregnancy include concern for the safety of two patients, the mother and fetus. Alterations in maternal anatomy and physiology induced by pregnancy have clinical anesthetic implications and present potential hazards for the mother and fetus undergoing anesthesia. The fetus may be subjected to hazard by (1) the risk of intraoperative hypoxemia or asphyxia caused by reduced uterine blood flow, maternal hypotension, excessive maternal mechanical ventilation or maternal hypoxia, depression of the fetal cardiovascular system or central nervous system from placental passage of anesthetic agents; (2) exposure to teratogenic drugs; and (3) the risk for preterm delivery as a consequence of the surgical procedure or drugs administered. In most circumstances, the fetus is a passive recipient of anesthesia administered to the mother, suffers no blood loss, and undergoes passive changes rather than direct stress or hemodynamic alterations caused by surgery (fig. 1).

Maternal Safety

During pregnancy, maternal anatomic and physiologic changes with implications for anesthetic management cause pregnant women to differ from those who are not pregnant. Although there are increased metabolic demands, these do not account for the magnitude of the increases in maternal respiratory and cardiovascular function.

Minute ventilation and oxygen consumption increase and residual volume and functional residual capacity decrease; therefore, oxygen reserve decreases and pregnant women develop hypoxia and hypercapnia more rapidly with hypoventilation or apnea. Airway management by face mask, laryngeal mask, or tracheal intubation can be technically difficult in pregnant women because of increased anteroposterior chest wall diameter, breast enlargement, laryngeal edema, and weight gain that affects the soft tissues of the neck.

During gestation, plasma volume and cardiac output increase, and peripheral vascular resistance decreases. From about mid-gestation, women in the supine position are at risk for aortic and venal caval compression by the
gravid uterus. Physiologic compensation for aortocaval compression can be compromised by anesthetic techniques (spinal, epidural, or general) that interfere with sympathetic tone, and can result in profound hypotension. For these women, avoiding the supine position by displacing the uterus laterally is important.

Pregnancy is associated with decreased anesthetic requirements: The minimum alveolar concentration decreases, and pregnant women may be more susceptible to axonal block by local anesthetics for reasons that remain unclear.

Because of mechanical and hormonal changes, pregnant women are at increased risk for gastric acid aspiration with anesthetic induction or unconscious sedation. Gastroesophageal sphincter tone is reduced, and although gastric motility remains normal during gestation, it is significantly impaired by opioid administration, onset of labor, pain, trauma, and so forth. For fasted pregnant women in the second or third trimester, or those with a history of reflux esophagitis, I advocate induction of general anesthesia using techniques for “full stomach precautions.”

Avoiding Fetal Asphyxia

To ensure fetal well-being, decreases in uterine blood flow or its oxygen content must be avoided. In addition,
excessive maternal mechanical hyperventilation can reduce venous return and thereby cardiac output, which reduces uterine blood flow. The asphyxiated fetus cannot increase oxygen extraction; rather, compensation is by redistribution of blood flow to vital organs. The uterine circulation is not autoregulated; it represents approximately 10% of cardiac output by full-term gestation and remains sensitive to vasopressors. Vasoactive medication that reduces uterine blood flow, such as α-adrenergic agents, dopamine, or epinephrine, are not ideal agents for treating maternal hypotension; although blood pressure may increase, uterine blood flow may remain depressed. However, small doses of phenylephrine have been used safely in several studies and reported cases; I consider it an agent of second choice when ephedrine is inappropriate or when the mother is tachycardic, has a stenotic valvular cardiac lesion, or receives ineffective or when the mother is tachycardic, has a stenotic valvular cardiac lesion, or receives β-agonist therapy. Besides uterine displacement, fluid bolus, Trendelenburg position, leg elevation, the use of compression stockings, or any combination of these, ephedrine remains the agent of choice in the initial pharmacologic management of maternal hypotension.

Maternal administration of increased inspired oxygen will increase fetal oxygenation; however, the fetus is never at risk for hyperoxia, because fetal oxygen tension will not exceed approximately 65 mmHg, even with maternal administration of 100% oxygen.

Anesthetic Agent Teratogenicity

Despite the far greater risk to the fetus from maternal hypotension or hypoxia, considerable concern exists about the potential for anesthetic agents or adjuvants to result in abortion or have teratogenic effects. For a defect to be produced, the embryo, fetus, or newborn must be exposed to a teratogenic agent at a given dose during a particular developmental stage in a species or person with a particular genetic susceptibility. Each organ and each system undergoes a critical stage of differentiation during which vulnerability to teratogens is greatest and specific malformations can be produced.

Clinical concentrations of volatile anesthetics have wide-ranging cellular effects, some of which may be potentially harmful to developing cells. Furthermore, it has been shown that nitrous oxide inactivates methionine synthetase, which in turn inhibits the synthesis of thymidine and DNA, inhibits cell division, and potentially disrupts other biochemical pathways in methyltransfer reactions. The concern is whether these known cellular effects of anesthetic agents are teratogenic.

To date, no clinical data link these cellular actions with teratogenic outcomes. Investigations of anesthetic teratogenicity have included studies of anesthetic agents on reproduction in rodents, epidemiologic surveys of chronic occupational exposure to trace concentrations of anesthetic agents, and outcome studies of women who have undergone surgery during pregnancy.

Laboratory investigations of the teratogenicity of inhalation agents in rodents suggest that modern volatile anesthetics in trace and subanesthetic concentrations do not result in adverse reproductive or teratogenic effects. Nitrous oxide is a weak teratogen in rodents when administered for long periods. However, the coadministration of halothane or isoflurane reverses the fetal lethality and teratogenic actions of nitrous oxide without affecting methionine synthetase activity. The reasons for these findings are uncertain, but they may be related to the vasoconstricting properties of nitrous oxide. Nitrous oxide appears to be weakly teratogenic in rodents for biochemical reasons unrelated to its inactivation of methionine synthetase.

Large survey studies that considered outcomes in women who underwent surgery during pregnancy suggest no increase in congenital anomalies among their offspring, but rather an increase in the risk for abortions, growth restriction, and increased frequency of low and very low birth-weight neonates for reasons attributed to the requirement for surgery but not anesthetic administration. Some smaller retrospective studies suggest an association with neural tube defects and first-trimester anesthesia exposure. These do not allow us to conclude categorically that anesthetic agents are not teratogenic in humans. However, the patient’s primary disease, site of surgery, or surgical procedure is more likely to increase the risk for abortion than is exposure to anesthesia.

Although many pregnant women undergo anesthesia and many others are exposed by occupation to anesthetics every year, the teratogenic risk of anesthetic agents in humans must be assessed based on incomplete data. Available studies suggest, for a surgical procedure, that administration of nitrous oxide or volatile, opioid, regional, or local anesthetics to pregnant women will not have deleterious effects on embryonic or fetal development and lack clinical significance for adverse neonatal outcome. The danger of teratogenic effects from currently available anesthetic or sedative drugs remains only a potential risk. No anesthetic, opioid analgesic, sedative-hypnotic, or anxiolytic agent appears to be teratogenic or safer than another agent. The long-standing relative
contraindication and concern about benzodiazepine use, particularly in the first trimester, was recently dispelled.12 I use preoperative medication for women to treat pain or anxiety, as appropriate. Catecholamines increased by pain or anxiety may adversely affect uterine blood flow.13

**Other Agents**

Neuromuscular blocking agents do not cross the placental barrier in clinically significant amounts. Although pregnant women have decreased concentrations of plasma cholinesterase and increased volumes for drug distribution, they may have relatively decreased hepatic blood flow. Therefore, the onset, duration, and clearance for neuromuscular blocking agents may be altered and are best administered with neuromuscular monitoring. Reversal agents should be administered slowly to avoid acute increases in acetylcholine, which might stimulate uterine contractions.

Although some data suggest that sodium nitroprusside may cause cyanide toxicity in laboratory animals, the doses used in experiments far exceeded safe clinical doses. Nitroprusside has been used safely in pregnant women, as has been nitroglycerin. Acute administration of β-adrenergic blocking agents is also safe, although long-term use has been associated with intrauterine growth restriction. High-dose administration of esmolol or other beta-blocking agents has been associated with fetal bradycardia, but these agents are not contraindicated during pregnancy.

**Specific Techniques**

Data from the Swedish Health Registry confirms the safety of laparoscopy during the first half of gestation,14 and there are several case reports of success in the late second and early third trimesters. Recommended precautions include use of pneumatic stockings to promote venous return, use of ultrasound rather than cholangiograms for cholecystectomy, use of nitrous oxide instead of carbon dioxide for pneumoperitoneum, avoidance of fetal respiratory acidosis, and use of the lowest-pressure pneumoperitoneum possible to avoid exacerbating vena caval compression.

When intentional manipulation of the maternal cardiovascular system is anticipated, such as deliberate hypotensive techniques or cardiopulmonary bypass, or when extraordinary procedures are undertaken, such as fetal surgery, it is wise to consult directly with those who have had more experience. In these cases, fetal monitoring is essential to ensure the adequacy of placental perfusion and optimization of the intrauterine environment.

Whenever possible, the fetus should be shielded from radiographic exposure (American College of Obstetricians and Gynecologists, Technical Bulletin #158, 1995).

**Fetal Monitoring**

Surgery and anesthesia can affect uterine activity and placental perfusion, and therefore fetal oxygenation and fetal heart rate. Fetal heart rate can also be affected directly by medications that readily cross the placenta or indirectly by their influence on maternal hemodynamics. Maternal anesthesia, and thus fetal anesthesia, and maternal (and hence fetal) hypothermia may decrease baseline fetal heart rate and beat-to-beat variability, but it will not cause spontaneous decelerations or those in response to a uterine contraction. The latter would be signs of fetal stress (hypoxemia, asphyxia).

Fetal and uterine monitoring during surgery is often possible, but in some circumstances access may be difficult. During surgery, steps can be taken to improve uterine perfusion and fetal oxygenation if they are compromised. However, such monitoring may be impractical in emergent or urgent situations, has not been documented to improve fetal outcome, and requires expertise often not possessed by regular intraoperative personnel. Misinterpretations could lead to unsafe interventions. When used, appropriate personnel trained in basic fetal heart rate interpretation should be immediately available. Although not considered a necessity for the intraoperative management of most pregnant surgical patients, preoperative and postoperative monitoring of uterine activity and fetal heart rate is advocated.

I recommend fetal monitoring whenever possible, particularly for the viable-age fetus and when the surgical procedure is major (e.g., laparotomy), rather than minor (e.g., carpal tunnel release). When monitoring by external abdominal ultrasound is not logistically feasible, I have used a sterile sleeve on a transabdominal ultrasound transducer, and once a transesophageal echocardiography probe placed directly on the uterus. Another alternative is a transvaginal ultrasound probe, particularly in early gestation.

**Conclusions**

Elective procedures, other than postpartum tubal ligations, should be deferred until approximately 6 weeks
after delivery, when the physiologic changes of pregnancy have passed and fetal well-being is no longer a concern (fig. 2). Women of child-bearing age should be asked about their last menstrual period, informed of potential risks, and pregnancy testing offered if their menstrual history is uncertain or they request it to avoid elective procedures during early gestation. Despite the lack of clinical evidence, delaying surgery until the second trimester, when possible, may reduce the risks for teratogenicity and spontaneous abortion. Whenever major surgery is undertaken in the pregnant patient, a perinatologist or obstetrician should be consulted to assist in perioperative management, diagnose and manage possible preterm labor, and to try to avoid preterm delivery. Informing the obstetrician or perinatologist of any surgical procedure may be in the best interest of the patient.

References


Fig. 2. Summary recommendations for management of the pregnant surgical patient.