CHAPTER 41

Obstetric Anesthesia

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KEY CONCEPTS

1. The most common morbidities encountered in obstetrics are severe hemorrhage and severe preeclampsia.
2. Regardless of the time of last oral intake, all obstetric patients are considered to have a full stomach and to be at risk for pulmonary aspiration.
3. Nearly all parenteral opioid analgesics and sedatives readily cross the placenta and can affect the fetus. Regional anesthetic techniques are preferred for management of labor pain.
4. Using a local anesthetic–opioid mixture for lumbar epidural analgesia during labor significantly reduces drug requirements, compared with using either agent alone.
5. Pain relief during labor requires neural blockade at the T10–L1 sensory level in the first stage of labor and at T10–S4 in the second stage.
6. Continuous lumbar epidural analgesia is the most versatile and most commonly employed technique, because it can be used for pain relief for the first stage of labor as well as analgesia/anesthesia for subsequent vaginal delivery or cesarean section, if necessary.
7. When dilute mixtures of a local anesthetic and an opioid are used, epidural analgesia has little if any effect on the progress of labor.
8. Even when aspiration does not yield blood or cerebrospinal fluid, unintentional intravascular or intrathecal placement of an epidural needle or catheter is possible.
9. Hypotension is a common side effect of regional anesthetic techniques and must be treated aggressively with phenylephrine or ephedrine, supplemental oxygen, left uterine displacement, and intravenous fluid boluses to prevent fetal compromise.
10. Techniques using combined spinal–epidural analgesia and anesthesia may particularly benefit patients with severe pain early in labor and those who receive analgesia/anesthesia just prior to delivery.
11. Spinal or epidural anesthesia is preferred to general anesthesia for cesarean section because regional anesthesia is associated with lower maternal mortality.
12. Continuous epidural anesthesia allows better control over the sensory level than "single-shot" techniques. Conversely, spinal anesthesia has a more rapid, predictable onset; may produce a more dense (complete) block; and lacks the potential for serious systemic drug toxicity because of the smaller dose of local anesthetic employed.
13. Risk of systemic local anesthetic toxicity during epidural analgesia and anesthesia is minimized by slowly administering dilute solutions for labor pain and by fractionating the total dose administered for cesarean section into 5-mL increments.

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14 Maternal hemorrhage is one of the most common severe morbidities complicating obstetric anesthesia. Causes include placenta previa, abruptio placentae, and uterine rupture.

15 Common causes of postpartum hemorrhage include uterine atony, a retained placenta, obstetric lacerations, uterine inversion, and use of tocolytic agents prior to delivery.

16 Intrauterine asphyxia during labor is the most common cause of neonatal depression. Fetal monitoring throughout labor is helpful in identifying which babies may be at risk, detecting fetal distress, and evaluating the effect of acute interventions.

This chapter focuses on the practice of obstetric anesthesia. Techniques for analgesia and anesthesia during labor, vaginal delivery, and cesarean section are presented. The chapter ends with a review of neonatal resuscitation.

ANESTHETIC RISK IN OBSTETRIC PATIENTS

Although the majority of women of childbearing age are healthy and would be considered to be at minimal operative risk, pregnancy, certain maternal–fetal factors, and preexisting medical conditions significantly increase surgical and obstetric risks.

Maternal Mortality

Maternal mortality is usually presented as the number of women who die while pregnant (or within 42 days of pregnancy termination) after excluding accidents and unrelated causes. This number is often indexed to the total number of live births. The maternal mortality index has decreased nearly 100-fold since 1900. Likely due to better reporting, it rose slightly in the United States to 21 deaths per 100,000 live births in 2010. The world average is 400 deaths per 100,000 live births. Of all maternal deaths worldwide, 99% occur in Africa, Asia, Latin America, and the Caribbean.

In the United States, overall mortality risk is greater for women older than 35 years of age, black women, and women who do not receive prenatal care. The leading causes of death associated with a live birth in 2010 were cardiovascular diseases (13.5%), cardiomyopathy (12.6%), hemorrhage (11.9%), noncardiovascular diseases (11.8%), hypertensive disorders of pregnancy (11.1%), infection/sepsis (11.1%), thrombotic pulmonary embolism (5.6%), amniotic fluid embolism (5.6%), cerebrovascular accidents (5.3%) and anesthesia complications (0.6%) Of all maternal deaths, only 34% of patients died within 24 h of delivery, whereas 55% died between 1 and 42 days, and another 11% died between 43 days and 1 year. Direct causes of maternal deaths are more clearly detailed from Canadian data, which show that, in addition to pulmonary embolism and preeclampsia/pregnancy-induced hypertension (PIH), amniotic fluid embolism and intracranial hemorrhage emerge as important additional causes of death.

Severe obstetric morbidity may be a more sensitive measure of outcome than maternal mortality. Data from the United Kingdom suggest that incidence of severe obstetric morbidity is 12 per 1000 deliveries, 100 times more common than mortality. Risk factors include age greater than 34 years, nonwhite ethnic group, multiple pregnancy, history of hypertension, previous postpartum hemorrhage, and emergency cesarean delivery. **Table 41–1** lists the estimated incidence of the most common causes of severe morbidity; thromboembolic disease was deliberately excluded because of the difficulty in making the diagnosis in nonfatal cases. By far the most common morbidities encountered in obstetrics are severe hemorrhage and severe preeclampsia.

TABLE 41–1 Incidence of severe obstetric morbidity.[1,3]

Morbidity	Incidence per 1000
Severe hemorrhage	6.7
Severe preeclampsia	3.9
HELLP syndrome[2]	0.5
Severe sepsis	0.4
Eclampsia	0.2
Uterine rupture	0.2

[1]Note thromboembolic disease was excluded.

[2]HELLP syndrome consists of hemolysis, elevated liver enzymes, and low platelet count.

[3]Data from Waterstone M, Bewley S, Wolfe C: Incidence and predictors of severe obstetric morbidity: Case-control study. BMJ 2001;322:1089.

Anesthetic Mortality

Anesthesia accidents and mishaps account for approximately 2–3% of maternal deaths. Data collected between 1985 and 1990 suggested a maternal mortality of 32 deaths per 1,000,000 live births due to general anesthesia and 1.9 deaths per 1,000,000 live births due to regional anesthesia. More recent data between 1998 and 2005 suggest a lower overall maternal mortality from anesthesia (about 1.2 % of live births), possibly due to greater use of regional anesthesia for labor and cesarean delivery. Most deaths occur during or after cesarean section. Moreover, the risk of an adverse outcome appears to be much greater with emergent than with elective cesarean sections.

Obstetric Anesthesia Closed Claims

Obstetric anesthesia care accounts for approximately 12% of the American Society of Anesthesiologists (ASA) Closed Claims database claims. A comparison of obstetric anesthesia claims from 1990 to 2003 or with pre-1990 claims shows a decrease in maternal deaths, as well as a decrease in respiratory-damaging events (aspiration, difficult intubation, esophageal intubation, and inadequate oxygenation/ventilation). Although newborn deaths and brain damage also decreased over this period, they remained a leading cause of obstetric anesthesia malpractice claims. Maternal nerve injury was more common in claims reported after 1990 compared with earlier years.

General Approach to the Obstetric Patient

All patients entering the obstetric suite potentially require anesthesia services, whether planned or emergent. Patients requiring anesthetic care for labor or cesarean section should undergo a focused preanesthetic evaluation as early as possible. This should consist of a maternal health history, anesthesia and anesthesia-related obstetric history, blood pressure measurement, airway assessment, and back examination for regional anesthesia.

2 Regardless of the time of last oral intake, all patients are considered to have a full stomach and to be at risk for pulmonary aspiration. Because the duration of labor is often prolonged, guidelines usually allow small amounts of oral clear liquid for uncomplicated labor. The minimum fasting period for elective cesarean section remains controversial, but is recommended to be 6 h for light meals and 8 h for heavy meals. Prophylactic administration of a clear antacid (15–30 mL of 0.3 M sodium citrate orally) every 30 min prior to a cesarean section can help maintain gastric pH greater than 2.5 and may decrease the likelihood of severe aspiration pneumonitis. An H_2-blocking drug (ranitidine, 100–150 mg orally or 50 mg intravenously) or metoclopramide, 10 mg orally or intravenously, should also be considered in high-risk patients and in those expected to receive general anesthesia. H_2 blockers reduce both gastric volume and pH but have no effect on the gastric contents already present. Metoclopramide accelerates gastric emptying, decreases gastric volume, and increases lower esophageal sphincter tone. The supine position should be avoided unless a left uterine displacement device (>15° wedge) is placed under the right hip.

Anesthesia for Labor & Vaginal Delivery

PAIN PATHWAYS DURING LABOR

The pain of labor arises from contraction of the myometrium against the resistance of the cervix and perineum, progressive dilation of the cervix and lower uterine segment, and stretching and compression of pelvic and perineal structures.

Pain during the first stage of labor is primarily visceral pain resulting from uterine contractions and cervical dilation. It is usually initially confined to the T11–T12 dermatomes during the latent phase, but eventually involves the T10–L1 dermatomes as labor enters the active phase. The visceral afferent fibers responsible for labor pain travel with sympathetic nerve fibers first to the uterine and cervical plexuses, then through the hypogastric and aortic plexuses, before entering the spinal cord with the T10–L1 nerve roots. The pain is initially perceived in the lower abdomen but may increasingly be referred to the lumbosacral area, gluteal region, and thighs as labor progresses. Pain intensity also increases with progressive cervical dilation and with increasing intensity and frequency of uterine contractions. Nulliparous women and those with a history of dysmenorrhea appear to experience greater pain during the first stage of labor.

The onset of perineal pain at the end of the first stage signals the beginning of fetal descent and the second stage of labor. Stretching and compression of pelvic and perineal structures intensifies the pain. Sensory innervation of the perineum is provided by the pudendal nerve (S2–4) so pain during the second stage of labor involves the T10–S4 dermatomes.

PSYCHOLOGICAL & NONPHARMACOLOGICAL TECHNIQUES

Psychological and nonpharmacological techniques are based on the premise that the pain of labor can be suppressed by reorganizing one's thoughts. Patient education and positive conditioning about the birthing process are central to such techniques. Pain during labor tends to be accentuated by fear of the unknown or previous unpleasant experiences. Techniques include those of Bradley, Dick-Read, Lamaze, and LeBoyer. The Lamaze technique, one of the most popular, coaches the parturient to take a deep breath at the beginning of each contraction followed by rapid, shallow breathing for the duration of the contraction. The parturient also concentrates on an object in the room and attempts to focus her thoughts away from the pain. Less common nonpharmacological techniques include hypnosis, transcutaneous electrical nerve stimulation, biofeedback, and acupuncture. The success of all these techniques varies considerably from patient to patient, and many patients require additional forms of analgesia.

PARENTERAL AGENTS

3 Nearly all parenteral opioid analgesics and sedatives readily cross the placenta and can affect the fetus. Concern over fetal depression limits the use of these agents to the early stages of labor or to situations in which regional anesthetic techniques are not available or appropriate. Central nervous system depression in the neonate may be manifested by a prolonged time to sustain respirations, respiratory acidosis, or an abnormal neurobehavioral examination. Moreover, loss of beat-to-beat variability in the fetal heart rate (seen with most central nervous system depressants) and decreased fetal movements (due to sedation of the fetus) complicate the evaluation of fetal well-being during labor. Long-term fetal heart rate variability is affected more than short-term variability. The degree and significance of these effects depend on the specific agent, the dose, the time elapsed between its administration and delivery, and fetal maturity. Premature neonates exhibit the greatest sensitivity. In addition to maternal respiratory depression, opioids can also induce maternal nausea and vomiting and delay gastric emptying. Some clinicians have advocated use of opioids via patient-controlled analgesia (PCA) devices early in labor because this technique appears to reduce total opioid requirements.

Meperidine, a commonly used opioid, can be given in doses of 10–25 mg intravenously or

25–50 mg intramuscularly, usually up to a total of 100 mg. Maximal maternal and fetal respiratory depression is seen in 10–20 min following intravenous administration and in 1–3 h following intramuscular administration. Consequently, meperidine is usually administered early in labor when delivery is not expected for at least 4 h. Intravenous fentanyl, 25–100 mcg/h, has also been used for labor. Fentanyl in 25–100 mcg doses has a 3- to 10-min analgesic onset that initially lasts about 60 min, and lasts longer following multiple doses. However, maternal respiratory depression outlasts the analgesia. Lower doses of fentanyl may be associated with little or no neonatal respiratory depression and are reported to have no effect on Apgar scores. Morphine is not used because in equianalgesic doses it appears to cause greater respiratory depression in the fetus than meperidine and fentanyl. Agents with mixed agonist–antagonist activity (butorphanol, 1–2 mg, and nalbuphine, 10–20 mg intravenously or intramuscularly) are effective and are associated with little or no cumulative respiratory depression, but excessive sedation with repeat doses can be problematic.

Promethazine (25–50 mg intramuscularly) and hydroxyzine (50–100 mg intramuscularly) can be useful alone or in combination with meperidine. Both drugs reduce anxiety, opioid requirements, and the incidence of nausea, but do not add appreciably to neonatal depression. A significant disadvantage of hydroxyzine is pain at the injection site following intramuscular administration. Nonsteroidal antiinflammatory agents, such as ketorolac, are not recommended because they suppress uterine contractions and promote closure of the fetal ductus arteriosus.

Small doses (up to 2 mg) of midazolam (Versed) may be administered in combination with a small dose of fentanyl (up to 100 mcg) in healthy parturients at term to facilitate neuraxial blockade. At this dose, maternal amnesia has not been observed. Chronic administration of the longer-acting benzodiazepine diazepam (Valium) has been associated with fetal depression.

Low-dose intravenous ketamine is a powerful analgesic. In doses of 10–15 mg intravenously, good analgesia can be obtained in 2–5 min without loss of consciousness. Unfortunately, fetal depression with low Apgar scores is associated with doses greater than 1 mg/kg. Large boluses of ketamine (>1 mg/kg) can be associated with hypertonic uterine contractions. Low-dose ketamine is most useful just prior to delivery or as an adjuvant to regional anesthesia. Some clinicians avoid use of ketamine because it may produce unpleasant psychotomimetic effects (see Chapter 9).

In the past, reduced concentrations of volatile anesthetic agents (eg, methoxyflurane) in oxygen were sometimes used for relief of milder labor pain. Inhalation of nitrous oxide–oxygen remains in common use for relief of mild labor pain in many countries. As previously noted, nitrous oxide has minimal effects on uterine blood flow or uterine contractions.

PUDENDAL NERVE BLOCK

Pudendal nerve blocks are often combined with perineal infiltration of local anesthetic to provide perineal anesthesia during the second stage of labor when other forms of anesthesia are not employed or prove to be inadequate. Paracervical plexus blocks are no longer used because of their association with a relatively high rate of fetal bradycardia; the close proximity of the injection site to the uterine artery may result in uterine arterial vasoconstriction, uteroplacental insufficiency, and increased levels of the local anesthetic in the fetal blood.

During a pudendal nerve block, a special needle (Koback) or guide (Iowa trumpet) is used to place the needle transvaginally underneath the ischial spine on each side (see Chapter 48); the needle is advanced 1–1.5 cm through the sacrospinous ligament, and 10 mL of 1% lidocaine or 2% chloroprocaine is injected following aspiration. The needle guide is used to limit the depth of injection and protect the fetus and vagina from the needle. Other potential complications include intravascular injection, retroperitoneal hematoma, and retropsoas or subgluteal abscess.

REGIONAL ANESTHETIC TECHNIQUES

Epidural or intrathecal techniques, alone or in combination, are currently the most popular methods of pain relief during labor and delivery. They can provide excellent analgesia while allowing the mother

to be awake and cooperative during labor. Although spinal opioids or local anesthetics alone can provide satisfactory analgesia, techniques that combine the two have proved to be the most satisfactory in most parturients. Moreover, the synergy between opioids and local anesthetics decreases dose requirements and provides excellent analgesia with few maternal side effects and little or no neonatal depression.

1. Spinal Opioids Alone

Opioids may be given intrathecally as a single injection or intermittently via an epidural or intrathecal catheter (Table 41–2). Relatively large doses are required for analgesia during labor when epidural or intrathecal opioids are used alone. For example, the ED_{50} during labor is 124 mcg for epidural fentanyl and 21 mcg for epidural sufentanil. The higher doses may be associated with a high risk of side effects, most importantly respiratory depression. For that reason combinations of local anesthetics and opioids are most commonly used (see below). Pure opioid techniques are most useful for high-risk patients who may not tolerate the functional sympathectomy associated with spinal or epidural anesthesia (see Chapter 45). This group includes patients with hypovolemia or significant cardiovascular disease such as moderate to severe aortic stenosis, tetralogy of Fallot, Eisenmenger's syndrome, or pulmonary hypertension. With the exception of meperidine, which has local anesthetic properties, spinal opioids alone do not produce motor blockade or sympathectomy. Thus, they do not impair the ability of the parturient to "push." Disadvantages include less complete analgesia, lack of perineal relaxation, and side effects such as pruritus, nausea, vomiting, sedation, and respiratory depression. Side effects may be ameliorated with low doses of naloxone (0.1–0.2 mg/h intravenously).

TABLE 41–2 Spinal opioid dosages for labor and delivery.

Agent	Intrathecal	Epidural
Morphine	0.1–0.5 mg	5 mg
Meperidine	10–15 mg	50–100 mg
Fentanyl	10–25 mcg	50–150 mcg
Sufentanil	3–10 mcg	10–20 mcg

Intrathecal Opioids

Intrathecal morphine in doses of 0.1–0.5 mg may produce satisfactory and prolonged (4–6 h) analgesia during the first stage of labor. Unfortunately, the onset of analgesia is slow (45–60 min), and these doses may not be sufficient in many patients. Higher doses are associated with a relatively high incidence of side effects. Morphine is therefore rarely used alone. The combination of morphine, 0.1–0.25 mg, and fentanyl, 12.5 mcg (or sufentanil, 5 mcg), may result in a more rapid onset of analgesia (5 min). Intermittent boluses of 10–15 mg of meperidine, 12.5–25 mcg of fentanyl, or 3–10 mcg of sufentanil via an intrathecal catheter can also provide satisfactory analgesia for labor. Early reports of fetal bradycardia following intrathecal opioid injections (eg, sufentanil) have not been confirmed by subsequent studies. Hypotension following administration of intrathecal opioids for labor is likely related to the resultant analgesia and decreased circulating catecholamine levels.

Epidural Opioids

Relatively large doses (≥7.5 mg) of epidural morphine are required for satisfactory labor analgesia, but doses larger than 5 mg are not recommended because of the increased risk of delayed respiratory depression and because the resultant analgesia is effective only in the early first stage of labor. Onset may take 30–60 min but analgesia lasts up to 12–24 h (as does the risk of delayed respiratory depression). Epidural meperidine, 50–100 mg, provides good, but relatively brief, analgesia (1–3 h). Epidural fentanyl, 50–150 mcg, or sufentanil, 10–20 mcg, usually produces analgesia within 5–10 min with few side effects, but it has a short duration (1–2 h). Although "single-shot" epidural opioids do not appear to cause significant neonatal depression, caution should be exercised following repeated administrations. Combinations of a lower dose of morphine, 2.5 mg, with fentanyl, 25–50 mcg (or

sufentanil, 7.5–10 mcg), may result in a more rapid onset and prolongation of analgesia (4–5 h) with fewer side effects.

2. Local Anesthetic/Local Anesthetic–Opioid Mixtures

Epidural and spinal (intrathecal) analgesia more commonly utilizes local anesthetics either alone or with opioids for labor and delivery. Analgesia during the first stage of labor requires neural blockade at the T10–L1 sensory level, whereas pain relief during the second stage of labor requires neural blockade at T10–S4. Continuous lumbar epidural analgesia is the most versatile and most commonly-employed technique, because it can be used for pain relief for the first stage of labor as well as analgesia/anesthesia for subsequent vaginal delivery or cesarean section, if necessary. "Single-shot" epidural, spinal, or combined spinal epidural analgesia may be appropriate when pain relief is initiated just prior to vaginal delivery (the second stage). Obstetric caudal injections have largely been abandoned because of less versatility; although effective for perineal analgesia/anesthesia they require large volumes of local anesthetic to anesthetize upper lumbar and lower thoracic dermatomes. They have also been associated with early paralysis of the pelvic muscles that may interfere with normal rotation of the fetal head, and with a small risk of accidental puncture of the fetus.

Absolute contraindications to regional anesthesia include patient refusal, infection over the injection site, coagulopathy, marked hypovolemia, and true allergies to local anesthetics. The patient's inability to cooperate may prevent successful regional anesthesia. Neuraxial anesthesia and full anticoagulation is a dangerous combination. Regional anesthesia should generally not be performed within 6–8 h of a subcutaneous minidose of unfractionated heparin or within 12–24 h of administration of low-molecular-weight heparin (LMWH). Thrombocytopenia or concomitant administration of an antiplatelet agent increases the risk of spinal hematoma. A vaginal birth after cesarean (VBAC) delivery is not considered a contraindication to regional anesthesia during labor. Concern that the anesthesia may mask pain associated with uterine rupture during VBAC may not be justified, because dehiscence of a lower segment scar frequently does not cause pain even without epidural anesthesia; moreover, changes in uterine tone and contraction pattern may be more reliable signs.

Before performing any regional block, appropriate equipment and supplies for resuscitation should be checked and made immediately available. Minimum supplies include oxygen, suction, a mask with a positive-pressure device for ventilation, a functioning laryngoscope and blades, endotracheal tubes (6 or 6.5 mm), oral and nasal airways, intravenous fluids, ephedrine, atropine, propofol, and succinylcholine. The ability to frequently monitor blood pressure and heart rate is mandatory. A pulse oximeter and capnograph should be readily available.

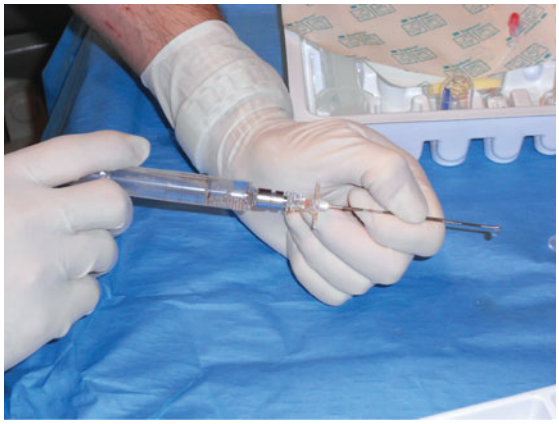
Lumbar Epidural Analgesia

Epidural analgesia for labor may be administered in early labor after the patient has been evaluated by her obstetrician. When dilute mixtures of a local anesthetic and an opioid are used, epidural analgesia has little if any effect on the progress of labor. Concerns that regional analgesia will increase the likelihood of oxytocin augmentation, operative (eg, forceps) delivery, or cesarean section, are unjustified. It is often advantageous to place an epidural catheter early, when the patient is less uncomfortable and can be positioned more easily. Moreover, should an urgent or emergent cesarean section become necessary, the presence of a well-functioning epidural catheter makes it possible to avoid general anesthesia.

A. Technique

Parturients may be positioned on their sides or in the sitting position for the procedure. The sitting position often makes it easier to identify the midline and spine in obese patients. When epidural anesthesia is being given for vaginal delivery (second stage), the sitting position helps ensure good sacral spread.

Because the lumbar epidural space pressure may be positive in some parturients, correct identification of the epidural space may be difficult. Unintentional dural puncture will occur even in experienced hands; the incidence of "wet taps" in obstetric patients is 0.25–9%, depending on clinician



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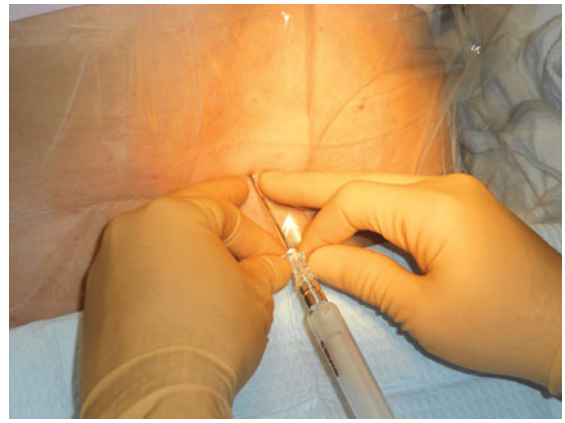

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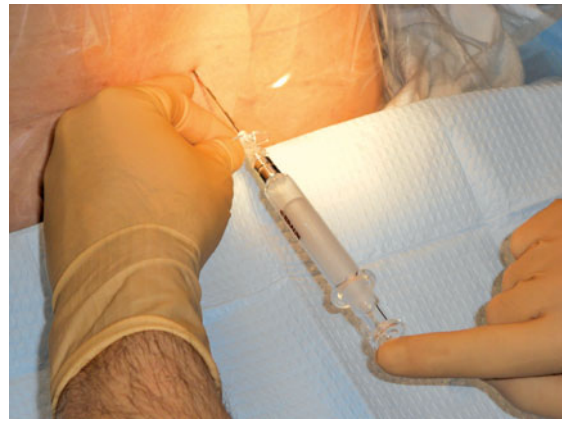

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FIGURE 41–1 **A**: One-handed needle advancement; continuous pressure technique. The operator applies continuous pressure to the plunger of a loss-of-resistance syringe filled with saline and an air bubble while advancing the needle with the left hand braced against the patient's back. **B**: Bimanual needle advancement; intermittent pressure technique. The operator advances the loss-of-resistance syringe with both hands 2–3 mm at a time while appreciating the resistance encountered by the needle. **C**: In between bimanual advancements of the needle, the operator tests the tissue resistance of the needle tip by bouncing the plunger of the air-filled loss-of-resistance syringe. Many practitioners add a compressible air bubble to a saline-filled syringe and bounce the plunger to ensure that the plunger is moving freely and not sticking to the syringe barrel wall.

experience. Many practitioners add a compressible air bubble to the saline syringe and bounce the plunger to ensure that it moves freely and does not stick to the syringe wall (Figure 41–1A and C). Most clinicians advocate the midline approach, whereas a minority favors the paramedian approach. For the placement of a lumbar epidural catheter in the obstetric patient, most anesthesiologists advance the epidural needle with the left hand, which is braced against the patient's back, while applying continuous pressure to a glass syringe filled with sterile saline (Figure 41–1A and C). Alternatively, some make use of the "wings" of the Weiss epidural needle by advancing it with both hands few millimeters at a time (Figure 41–1B). A change of tissue resistance is then tested continuously using tactile feedback when advancing the needle and by intermittently applying pressure to the air-filled loss-of resistance syringe. The later technique allows for precise control of needle advancement and may allow a better distinction of various tissue densities. If air is used for detecting loss of resistance, the amount injected should be limited; injection of larger volumes of air (>2–3 mL) in the epidural space has been associated with patchy or unilateral analgesia and headache. The average depth of the lumbar epidural space in obstetric patients is reported to be 5 cm from the skin. Placement of the epidural catheter at the L3–4 or L4–5 interspace is generally optimal for achieving a T10–S5 neural blockade. Ultrasound guidance has recently been offered as tool in assisting with the placement of an epidural catheter. This technique

allows the practitioner to judge the depth of the epidural space and estimate the best angle of needle insertion. The potential benefit of this technique is most obvious in obese patients with poor anatomic landmarks. However, the technique is highly user-dependent, and few practitioners have adopted it.

If unintentional dural puncture occurs, the anesthetist has two choices: (1) place the epidural catheter in the subarachnoid space for continuous spinal (intrathecal) analgesia and anesthesia (see below), or (2) remove the needle and attempt placement at a higher spinal level. The intrathecally-placed epidural catheter may be used as continuous spinal anesthetic, possibly reducing the incidence of post–dural puncture headache. If used in this fashion, an infusion of 0.0625–0.125% bupivacaine with fentanyl, 2–3 mcg/mL starting at 1–3 mL/h, is a reasonable choice.

B. Choice of Epidural Catheter

Many clinicians advocate use of a multiholed catheter instead of a single-holed catheter for obstetric anesthesia. Use of a multiholed catheter may be associated with fewer unilateral blocks and greatly reduces the incidence of false-negative aspiration when assessing for intravascular or intrathecal catheter placement. Advancing a multiholed catheter 4–6 cm into the epidural space appears to be optimal for obtaining adequate sensory levels. A single-hole catheter need only be advanced 3–5 cm into the epidural space. Shorter insertion depths (<5 cm), however, may favor dislodgment of the catheter out of the epidural space in obese patients following flexion/extension movements of the spine. Spiral wire-reinforced catheters are very resistant to kinking. A spiral or spring tip, particularly when used without a stylet, is associated with fewer, less intense paresthesias and may also be associated with a lower incidence of accidental intravascular insertion.

C. Choice of Local Anesthetic Solutions

The addition of opioids to local anesthetic solutions for epidural anesthesia has dramatically changed the practice of obstetric anesthesia. The synergy between epidural opioids and local anesthetic solutions reflects separate sites of action, namely, opiate receptors and neuronal axons, respectively. When the two are combined, very low concentrations of both local anesthetic and opioid can be used. More importantly, the incidence of adverse side effects, such as hypotension and drug toxicity, is likely reduced. Although local anesthetics can be used alone, there is rarely a reason to do so. Moreover, when an opioid is omitted, the higher concentration of local anesthetic required (eg, bupivacaine, 0.25%, and ropivacaine, 0.2%) for adequate analgesia can impair the parturient's ability to push effectively as labor progresses. Bupivacaine or ropivacaine in concentrations of 0.0625–0.125% with either fentanyl, 2–3 mcg/mL, or sufentanil, 0.3–0.5 mcg/mL, is most often used. In general, the lower the concentration of the local anesthetic the greater the concentration of opioid that is required. Very dilute local anesthetic mixtures (0.0625%) generally do not produce motor blockade and may allow some patients to ambulate ("walking" or "mobile" epidural). The long duration of action of bupivacaine makes it a popular agent for labor. Ropivacaine may be preferable because of its reduced potential for cardiotoxicity (see Chapter 16). At equi-analgesic doses, ropivacaine and bupivacaine appear to produce the same degree of motor block.

The effect of epinephrine-containing solutions on the course of labor is somewhat controversial. Many clinicians use epinephrine-containing solutions only for intravascular test doses because of concern that the solutions may slow the progression of labor or adversely affect the fetus; others use only very dilute concentrations of epinephrine such as 1:800,000 or 1:400,000. Studies comparing these various agents have failed to find any differences in neonatal Apgar scores, acid–base status, or neurobehavioral evaluations.

D. Epidural Activation for the First Stage of Labor

Initial epidural injections may be done either before or after the catheter is placed. Administration through the needle can facilitate catheter placement, whereas administration through the catheter ensures proper function of the catheter. The following sequence is suggested for epidural activation:

1. Test for unintentional subarachnoid or intravascular placement of the needle or catheter with a 3-mL test dose of a local

anesthetic with 1:200,000 epinephrine (controversial; see the section on Prevention of Unintentional Intravascular and Intrathecal Injections). Many clinicians test with lidocaine 1.5% because of less toxicity following unintentional intravascular injection and a more rapid onset of spinal anesthesia than with bupivacaine and ropivacaine. The test dose should be injected between contractions to help reduce false positive signs of an intravascular injection (ie, tachycardia due to a painful contraction).

2. If after 5 min signs of intravascular or intrathecal injection are absent, with the patient supine and left uterine displacement, administer 10 mL of the local anesthetic–opioid mixture in 5-mL increments, waiting 1–2 min between doses, to achieve a T10–L1 sensory level. The initial bolus is usually composed of 0.1–0.2% ropivacaine or 0.0625–0.125% bupivacaine combined with either 50–100 mcg of fentanyl or 10–20 mcg of sufentanil.
3. Monitor with frequent blood pressure measurements for 20–30 min or until the patient is stable. Pulse oximetry should also be used. Oxygen is administered via face mask if there are any significant decreases in blood pressure or oxygen saturation readings.
4. Repeat steps 2 and 3 when pain recurs until the first stage of labor is completed. Alternatively, a continuous epidural infusion technique may be employed using bupivacaine or ropivacaine in concentrations of 0.0625–0.125% with either fentanyl, 1–5 mcg/mL, or sufentanil, 0.2–0.5 mcg/mL at a rate of 10 mL/h, which subsequently is adjusted to the patient's analgesic requirements (range: 5–15 mL/h). A third choice would be to use patient-controlled epidural analgesia (PCEA). Some studies suggest that total drug requirements may be less and patient satisfaction is greater with PCEA compared with other epidural techniques. PCEA settings are typically a 5-mL bolus dose with a 5–10 min lockout and 0–12 mL/h basal rate; a 1-h limit of 15–25 mL may used. Migration of the epidural catheter into a blood vessel during a continuous infusion technique may be heralded by loss of effective analgesia; a high index of suspicion is required because overt signs of systemic toxicity may be absent. Erosion of the catheter through the dura results in a slowly progressive motor blockade of the lower extremities and a rising sensory level.

E. Epidural Administration During the Second Stage of Labor

Administration for the second stage of labor extends the block to include the S2–4 dermatomes. Whether a catheter is already in place or epidural anesthesia is just being initiated, the following steps should be undertaken:

1. If the patient does not already have a catheter in place, identify the epidural space while the patient is in a sitting position. A patient who already has an epidural catheter in place should be placed in a semiupright or sitting position prior to injection.
2. Give a 3-mL test dose of local anesthetic (eg, lidocaine 1.5%) with 1:200,000 epinephrine. Again, the injection should be completed between contractions.
3. If after 5 min signs of an intravascular or intrathecal injection are absent, give 10–15 mL of additional local anesthetic–opioid mixture at a rate not faster than 5 mL every 1–2 min.
4. Administer oxygen by face mask, lay the patient supine with left uterine displacement, and monitor blood pressure every 1–2 min for the first 15 min, then every 5 min thereafter.

F. Prevention of Unintentional Intravascular and Intrathecal Injections

Safe administration of epidural anesthesia is critically dependent on avoiding unintentional intrathecal or intravascular injection. Unintentional intravascular or intrathecal placement of an epidural needle or catheter is possible even when aspiration fails to yield blood or cerebrospinal fluid (CSF). The incidence of unintentional intravascular or intrathecal placement of an epidural catheter is 5–15% and 0.5–2.5%, respectively. Even a properly placed catheter can subsequently erode into an

epidural vein or an intrathecal position. This possibility should be considered each time local anesthetic is injected through an epidural catheter.

Test doses of lidocaine, 45–60 mg, bupivacaine, 7.5–10 mg, ropivacaine, 6–8 mg, or chloroprocaine, 100 mg, can be given to exclude unintentional intrathecal placement. Signs of sensory and motor blockade usually become apparent within 2–3 min and 3–5 min, respectively, if the injection is intrathecal.

In patients not receiving β-adrenergic antagonists, the intravascular injection of a local anesthetic solution with 15–20 mcg of epinephrine consistently increases the heart rate by 20–30 beats/min within 30–60 s if the catheter (or epidural needle) is intravascular. This technique is not always reliable in parturients because they often have marked spontaneous baseline variations in heart rate with contractions. In fact, bradycardia has been reported in a parturient following intravenous injection of 15 mcg of epinephrine. Moreover, in animal studies, 15 mcg of epinephrine intravenously reduces uterine blood flow. Alternative methods of detecting unintentional intravascular catheter placement include eliciting tinnitus or perioral numbness following a 100-mg test dose of lidocaine or eliciting a chronotropic effect following injection of 5 mcg of isoproterenol. The use of dilute local anesthetic solutions and slow injection rates of no more than 5 mL at a time may also enhance detection of unintentional intravascular injections before catastrophic complications develop.

G. Management of Complications

9 **1. Hypotension**—Generally defined as a greater than 20% decrease in the patient's baseline blood pressure, or a systolic blood pressure less than 100 mm Hg, hypotension is a common side effect of neuraxial anesthesia. It is primarily due to decreased sympathetic tone and is greatly accentuated by aortocaval compression and an upright or semiupright position. Treatment should be aggressive in obstetric patients and consists of intravenous boluses of ephedrine (5–15 mg) or phenylephrine (25–50 mcg), supplemental oxygen, left uterine displacement, and an intravenous fluid bolus. Although the routine use of a crystalloid fluid bolus prior to dosing an epidural catheter is not effective in the prevention of hypotension, ensuring proper intravenous hydration of the pregnant patient is important. Use of the head-down (Trendelenburg) position is controversial because of its potentially detrimental effects on pulmonary gas exchange.

2. Unintentional intravascular injection—Early recognition of intravascular injection, facilitated by the use of small, repeated doses of local anesthetic instead of a large bolus, may prevent more serious local anesthetic toxicity, such as seizures or cardiovascular collapse. Intravascular injections of toxic doses of lidocaine or chloroprocaine usually present as seizures. Propofol, 20–50 mg, will terminate seizure activity. Maintenance of a patent airway and adequate oxygenation are critical; however, immediate endotracheal intubation with succinylcholine and cricoid pressure is rarely necessary. Intravascular injections of bupivacaine can cause rapid and profound cardiovascular collapse as well as seizure activity. Cardiac resuscitation may be exceedingly difficult and is aggravated by acidosis and hypoxia. An immediate infusion of 20% Intralipid has shown efficacy in reversing bupivacaine-induced cardiac toxicity. Amiodarone is the agent of choice for treating local anesthetic–induced ventricular arrhythmias.

3. Unintentional intrathecal injection—Even when dural puncture is recognized immediately after injection of local anesthetic, attempted aspiration of the local anesthetic will usually be unsuccessful. The patient should be placed supine with left uterine displacement. Head elevation accentuates the adverse cerebral effects of hypotension and should be avoided. Hypotension should be treated with phenylephrine and intravenous fluids. A high spinal level can also result in diaphragmatic paralysis, which necessitates intubation and ventilation with 100% oxygen. Delayed onset of a very high and often patchy or unilateral block may be due to unrecognized subdural injection (see Chapter 45), which is managed similarly.

4. Postdural puncture headache (PDPH)—Headache frequently follows unintentional dural puncture in parturients. A self-limited headache may occur without dural puncture; in such instances, injection of significant amounts of air into the epidural space during a loss-of-resistance technique may be responsible. PDPH is due to decreased intracranial pressure with compensatory cerebral vasodilation (see Chapter 45). Bed rest, hydration, oral analgesics, and caffeine sodium benzoate (500 mg

added to 1000 mL intravenous fluids administered at 200 mL/h) may be effective in patients with mild headaches and as temporary treatment. Patients with moderate to severe headaches usually require an epidural blood patch (10–20 mL) (see Chapter 45). Prophylactic epidural blood patches are not recommended; 25–50% of patients may not require a blood patch following dural puncture. Delaying a blood patch for 24 h increases its efficacy. Intracranial subdural hematoma has been reported as a rare complication 1–6 weeks following unintentional dural puncture in obstetric patients.

5. Maternal fever—Maternal fever is often interpreted as chorioamnionitis and may trigger an invasive evaluation for neonatal sepsis. There is no evidence that epidural anesthesia affects maternal temperature or that neonatal sepsis is increased with epidural analgesia. An elevation in maternal temperature is associated with a high body mass index and with nulliparity in women and prolonged labor.

Combined Spinal & Epidural (CSE) Analgesia

10 Techniques using CSE analgesia and anesthesia may particularly benefit patients with severe pain early in labor and those who receive analgesia/anesthesia just prior to delivery. Intrathecal opioid and local anesthetic are injected after which an epidural catheter is left in place. The intrathecal drugs provide nearly immediate pain control and have minimal effects on the early progress of labor, whereas the epidural catheter provides a route for subsequent analgesia for labor and delivery or anesthesia for cesarean section. Addition of small doses of local anesthetic agents to intrathecal opioid injection greatly potentiates their analgesia and can significantly reduce opioid requirements. Thus, many clinicians will inject 2.5 mg of preservative-free bupivacaine or 3–4 mg of ropivacaine with intrathecal opioids for analgesia in the first stage of labor. Intrathecal doses for CSE are fentanyl, 5–10 mcg, or sufentanil, 5 mcg. Some studies suggest that CSE techniques may be associated with greater patient satisfaction and lower incidence of PDPH than epidural analgesia alone. A 24- to 27-gauge pencil-point spinal needle (Whitacre, Sprotte, or Gertie Marx) is used to minimize the incidence of PDPH.

The spinal and epidural needles may be placed at separate interspaces, but most clinicians use a needle-through-needle technique at the same interspace. Use of saline for identification of the epidural space may potentially cause confusion of saline for CSF. With the needle-through-needle technique, the epidural needle is placed in the epidural space and a long spinal needle is then introduced through it and advanced farther into the subarachnoid space. A distinct pop is felt as the needle penetrates the dura. The needle-beside-needle technique typically employs a specially designed epidural needle that has a channel for the spinal needle. After the intrathecal injection and withdrawal of the spinal needle, the epidural catheter is threaded into position and the epidural needle is withdrawn. The risk of advancing the epidural catheter through the dural hole created by the spinal needle appears to be negligible when a 25-gauge or smaller needle is used. The epidural catheter, however, should be aspirated carefully and local anesthetic should always be given slowly and in small increments to avoid unintentional intrathecal injections. Moreover, epidural drugs should be titrated carefully because the dural hole may facilitate entry of epidural drugs into CSF and enhance their effects.

Spinal Anesthesia

Spinal anesthesia given just prior to delivery—also known as saddle block—provides profound anesthesia for operative vaginal delivery. Use of a 22-gauge or smaller, pencil-point spinal needle (Whitacre, Sprotte, or Gertie Marx) decreases the likelihood of PDPH. Hyperbaric tetracaine (3–4 mg), bupivacaine (2.5–5 mg), or lidocaine (20–40 mg) usually provides excellent perineal anesthesia. Addition of fentanyl (12.5–25 mcg) or sufentanil (5–7.5 mcg) significantly potentiates the block. A T10 sensory level can be obtained with slightly larger amounts of local anesthetic. Three minutes after injection, the patient is placed in the lithotomy position with left uterine displacement.

GENERAL ANESTHESIA

Because of the increased risk of aspiration, general anesthesia for vaginal delivery is avoided except for a true emergency. If an epidural catheter is already in

TABLE 41–3 Possible indications for general anesthesia during vaginal delivery.

Fetal distress during the second stage
Tetanic uterine contractions
Breech extraction
Version and extraction
Manual removal of a retained placenta
Replacement of an inverted uterus

place and time permits, rapid-onset regional anesthesia can be obtained with alkalinized lidocaine 2% or chloroprocaine 3%. Table 41–3 lists indications for general anesthesia during vaginal delivery. These indications are rare, and most share the need for uterine relaxation.

Anesthesia for Cesarean Section

Common indications for cesarean section are listed in Table 41–4. The choice of anesthesia for cesarean section is determined by multiple factors, including the indication for operative delivery, its urgency, patient and obstetrician preferences, and the skills of the anesthetist. In a given country, cesarean section rates may vary as much as two-fold between institutions. In some countries, cesarean delivery is seen as preferable to labor and rates are much greater than those in the United States (which generally vary between 15% and 35% from hospital to hospital). In the United States most elective cesarean sections are performed under spinal anesthesia. Regional anesthesia has become the preferred technique because general anesthesia has been associated with a greater risk of maternal morbidity and mortality. Deaths associated with general anesthesia are generally related to airway problems, such as inability to intubate, inability to ventilate, or aspiration pneumonitis, whereas deaths associated with regional anesthesia are generally related to excessive dermatomal spread of blockade or to local anesthetic toxicity.

Other advantages of regional anesthesia include (1) less neonatal exposure to potentially depressant drugs, (2) a decreased risk of maternal pulmonary aspiration, (3) an awake mother at the birth of her child, and (4) the option of using spinal opioids for postoperative pain relief. Continuous epidural anesthesia allows better continuing control over the sensory level than "single-shot" techniques. Conversely, spinal anesthesia has a more rapid, predictable onset; may produce a more dense (complete) block; and lacks the potential for serious systemic drug toxicity because of the smaller dose of local anesthetic employed. Regardless of the regional technique chosen, one must be prepared to administer a general anesthetic at any time during the procedure. Moreover, administration of a nonparticulate antacid within 30 min of surgery should be considered.

General anesthesia offers (1) a very rapid and reliable onset, (2) control over the airway and ventilation, (3) greater comfort for parturients who have morbid fears of needles or surgery, and (4) potentially less hypotension than regional anesthesia. General anesthesia also facilitates management in the event of severe hemorrhagic complications such as placenta

TABLE 41–4 Major indications for cesarean section.

Labor unsafe for mother and fetus
Increased risk of uterine rupture
Previous classic cesarean section
Previous extensive myomectomy or uterine reconstruction
Increased risk of maternal hemorrhage
Central or partial placenta previa
Abruptio placentae
Previous vaginal reconstruction
Dystocia
Abnormal fetopelvic relations
Fetopelvic disproportion
Abnormal fetal presentation
Transverse or oblique lie
Breech presentation
Dysfunctional uterine activity
Immediate or emergent delivery necessary
Fetal distress
Umbilical cord prolapse with fetal bradycardia
Maternal hemorrhage
Genital herpes with ruptured membranes
Impending maternal death

accreta. Its principal disadvantages are the risk of pulmonary aspiration, the potential inability to intubate or ventilate the patient, and drug-induced fetal depression. Present anesthetic techniques, however, limit the dose of intravenous agents such that fetal depression is usually not clinically significant with general anesthesia when delivery occurs within 10 min of induction of anesthesia. Regardless of the type of anesthesia, neonates delivered more than 3 min after uterine incision have lower Apgar scores and pH values.

REGIONAL ANESTHESIA

Cesarean section requires that dermatomes up to and including T4 be anesthetized. Because of the associated sympathetic blockade, patients should receive an appropriate intravenous bolus of crystalloid such as lactated Ringer's (typically 1000–1500 mL) or colloid (typically 250–500 mL) solution at the time of neural blockade. Such boluses will not consistently prevent hypotension but can virtually eliminate preexisting hypovolemia. After the local anesthetic injection, phenylephrine may be titrated to maintain blood pressure within 20% of baseline. An approximate 10% decrease in blood pressure is expected. Administration of ephedrine (5–10 mg) may be necessary in the hypotensive patient with reduced heart rate. Some studies suggest that phenylephrine produces less neonatal acidosis compared with ephedrine.

After spinal anesthetic injection, the patient is placed supine with left uterine displacement; supplemental oxygen (40–50%) is given; and blood pressure is measured every 1–2 min until it stabilizes. Hypotension following epidural anesthesia typically has a slower onset. Slight Trendelenburg positioning facilitates achieving a T4 sensory level and may also help prevent severe hypotension. Extreme degrees of Trendelenburg may interfere with pulmonary gas exchange.

Spinal Anesthesia

The patient is usually placed in the lateral decubitus or sitting position, and a hyperbaric solution of lidocaine (50–60 mg) or bupivacaine (10–15 mg) is injected. Bupivacaine should be chosen if the obstetrician will not likely complete the surgery in 45 minutes. Use of a 22-gauge or smaller, pencil-point spinal needle (Whitacre, Sprotte, or Gertie Marx) decreases the incidence of PDPH. Adding 10–25 mcg of fentanyl or 5–10 mcg of sufentanil to the local anesthetic solution enhances the intensity of the block and prolongs its duration without adversely affecting neonatal outcome. Addition of preservative-free morphine (0.1–0.3 mg) can prolong postoperative analgesia up to 24 h, but requires monitoring for delayed postoperative respiratory depression. Regardless of the anesthetic agents used, considerable variability in the maximal dermatomal extent of anesthesia should be expected (see Chapter 45). In obese patients, a standard 3.5-in. (9-cm) spinal needle may not be long enough to reach the subarachnoid space. In these cases, longer spinal needles of 4.75 in. (12 cm) to 6 in. (15.2 cm) may be required. To prevent these longer needles from bending, some anesthesiologists prefer larger diameter needles, such as the 22-gauge Sprotte needle. Alternatively, a 2.5-in. (6.3-cm) 20-gauge Quincke type spinal needle can be used as a long introducer and guide for a 25-gauge pencil-point spinal needle.

Continuous spinal anesthesia is also a reasonable option, especially for obese patients, following unintentional dural puncture sustained while attempting to place an epidural catheter for cesarean section. After the catheter is advanced 2–2.5 cm into the lumbar subarachnoid space and secured, it can be used to inject anesthetic agents; moreover, it allows later supplementation of anesthesia if necessary.

Epidural Anesthesia

Epidural anesthesia for cesarean section is typically performed using a catheter, which allows supplementation of anesthesia if necessary and provides an excellent route for postoperative opioid administration. After negative aspiration and a negative test dose, a total of 15–25 mL of local anesthetic is injected slowly in 5-mL increments in order to minimize the risk of systemic local anesthetic toxicity. Lidocaine 2% (typically with 1:200,000 epinephrine) or chloroprocaine 3% are most commonly used in the United States. The addition of fentanyl,

50–100 mcg, or sufentanil, 10–20 mcg, greatly enhances the intensity of the analgesia and prolongs its duration without adversely affecting neonatal outcome. Some practitioners also add sodium bicarbonate (7.5% or 8.4% solution) to local anesthetic solutions (1 mEq/10 mL of lidocaine) to increase the concentration of the nonionized free base and produce a faster onset and more rapid spread of epidural anesthesia. If pain develops as the sensory level recedes, additional local anesthetic is administered in 5-mL increments to maintain a T4 sensory level. "Patchy" anesthesia prior to delivery of the baby can be treated with 10–20 mg of intravenous ketamine in combination with 1–2 mg of midazolam or 30% nitrous oxide. After delivery, intravenous opioid supplementation may also be used, provided excessive sedation and loss of consciousness are avoided. Pain that remains intolerable in spite of a seemingly adequate sensory level and that proves unresponsive to these measures necessitates general anesthesia with endotracheal intubation. Nausea can be treated intravenously with a 5-HT3-receptor antagonist such as ondansetron, 4 mg.

Epidural morphine (5 mg) at the end of surgery provides good to excellent pain relief postoperatively for 6–24 h. An increased incidence (3.5–30%) of recurrent herpes simplex labialis infection has been reported 2–5 days following epidural morphine administration in some studies. Postoperative analgesia can also be provided by continuous epidural infusions of fentanyl, 25–75 mcg/h, or sufentanil, 5–10 mcg/h, at a volume rate of approximately 10 mL/h. Epidural butorphanol, 2 mg, can also provide effective postoperative pain relief, but marked somnolence is often a side effect.

CSE Anesthesia

The technique for CSE is described in the earlier section on Combined Spinal & Epidural Analgesia for labor and vaginal delivery. For cesarean section, it combines the benefit of rapid, reliable, intense blockade of spinal anesthesia with the flexibility of an epidural catheter. The catheter also allows supplementation of anesthesia and can be used for postoperative analgesia. As mentioned previously, drugs given epidurally should be administered and titrated carefully because the dural hole created by the spinal needle may facilitate movement of epidural drugs into CSF and enhance their effects.

GENERAL ANESTHESIA

Pulmonary aspiration of gastric contents and failed endotracheal intubation are the major causes of maternal morbidity and mortality associated with general anesthesia. All patients should receive prophylaxis against aspiration pneumonia with 30 mL of 0.3 M sodium citrate 30–45 min prior to induction. Patients with additional risk factors predisposing them to aspiration should also receive intravenous ranitidine, 50 mg, or metoclopramide, 10 mg, or both, 1–2 h prior to induction; such factors include morbid obesity, symptoms of gastroesophageal reflux, a potentially difficult airway, or emergent surgical delivery without an elective fasting period. Premedication with oral omeprazole, 40 mg, at night and in the morning also appears to be highly effective in high-risk patients undergoing elective cesarean section. Although anticholinergics theoretically may reduce lower esophageal sphincter tone, premedication with glycopyrrolate (0.1 mg) helps reduce airway secretions and should be considered in patients with a potentially difficult airway.

Anticipation of a difficult endotracheal intubation may help reduce the incidence of failed intubations. Examination of the neck, mandible, dentition, and oropharynx often helps predict which patients may have problems. Useful predictors of a difficult intubation include Mallampati classification, short neck, receding mandible, prominent maxillary incisors, and history of difficult intubation (see Chapter 19). The higher incidence of failed intubations in pregnant patients compared with nonpregnant surgical patients may be due to airway edema, a full dentition, or large breasts that can obstruct the handle of the laryngoscope in patients with short necks. Proper positioning of the head and neck may facilitate endotracheal intubation in obese patients: elevation of the shoulders, flexion of the cervical spine, and extension of the atlantooccipital joint (**Figure 41–2**). A variety of laryngoscope blades, a short laryngoscope handle, at least one extra stiletted endotracheal tube (6 mm), Magill forceps (for nasal intubation), a laryngeal mask airway (LMA), an intubating LMA (Fastrach), a fiberoptic bronchoscope,

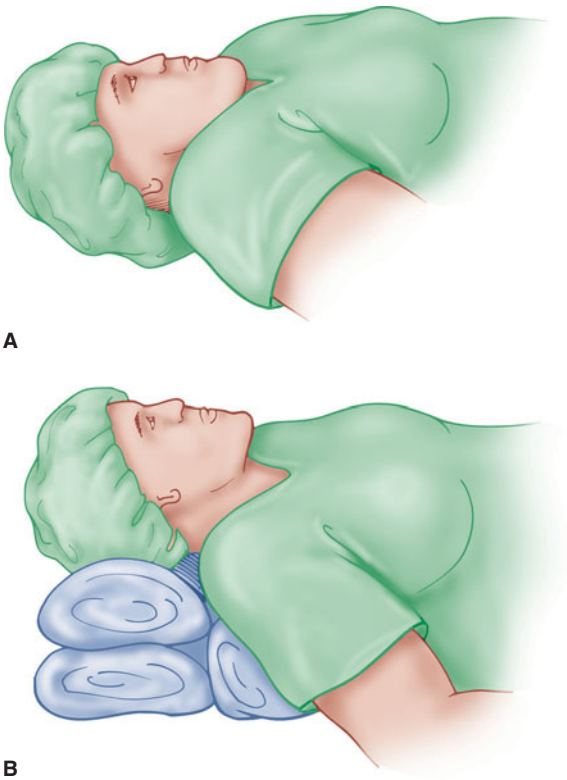

FIGURE 41–2 Optimal positioning for obese patients with a short neck. **A**: The normal supine position often prevents extension of the head and makes endotracheal intubation difficult. **B**: Elevation of the shoulder allows some neck flexion with more optimal extension of the head at the atlantooccipital joint, facilitating intubation.

a video-assisted laryngoscope (GlideScope or Stortz CMAC), the capability for transtracheal jet ventilation, and possibly an esophageal–tracheal Combitube should be readily available (see Chapter 19).

When potential difficulty in securing the airway is suspected, alternatives to the standard rapid-sequence induction with conventional laryngoscopy, such as regional anesthesia or awake fiberoptic techniques, should be considered. We have found that video-assisted laryngoscopy has greatly reduced the incidence of difficult or failed tracheal intubation at our institutions. Moreover, a clear plan should be formulated for a failed endotracheal intubation following induction of anesthesia (Figure 41–3). In the absence of fetal distress, the patient should be awakened, and an awake intubation, with regional or local (infiltration) anesthesia, may be tried. In the presence of fetal distress, if spontaneous or positive-pressure ventilation (by mask or LMA) with cricoid pressure is possible, delivery of the fetus may be attempted. In such instances, a potent volatile agent with oxygen is employed for anesthesia, but once the fetus is delivered, nitrous oxide may be added to reduce the concentration of the volatile agent; sevoflurane may be the best volatile agent because it may be least likely to depress ventilation. The inability to ventilate the patient at any time may require immediate cricothyrotomy or tracheostomy.

Suggested Technique for Cesarean Section

1. The patient is placed supine with a wedge under the right hip for left uterine displacement.
2. Denitrogenation is accomplished with 100% oxygen for 3–5 min while monitors are applied.
3. The patient is prepared and draped for surgery.
4. When the surgeons are ready, a rapid-sequence induction with cricoid pressure is performed using propofol, 2 mg/kg, or ketamine, 1–2 mg/kg, and succinylcholine, 1.5 mg/kg. Ketamine is used instead of propofol in hypovolemic patients. Other agents, including methohexital and etomidate, offer little benefit in obstetric patients.
5. With few exceptions, surgery is begun only after proper placement of the endotracheal tube is confirmed. Excessive hyperventilation ($Paco_2 < 25$ mm Hg) should be avoided because it can reduce uterine blood flow and has been associated with fetal acidosis.
6. Fifty percent nitrous oxide in oxygen with up to 0.75 MAC of a low concentration of volatile agent (eg, 1% sevoflurane, 0.75% isoflurane, or 3% desflurane) is used for maintenance of anesthesia. The low dose of volatile agent helps ensure amnesia but is generally not enough to cause excessive uterine relaxation or prevent uterine contraction following oxytocin. A muscle relaxant of intermediate duration (atracurium, cisatracurium, or rocuronium) is used for relaxation, but may exhibit prolonged neuromuscular blockade in patients who are receiving magnesium sulfate.

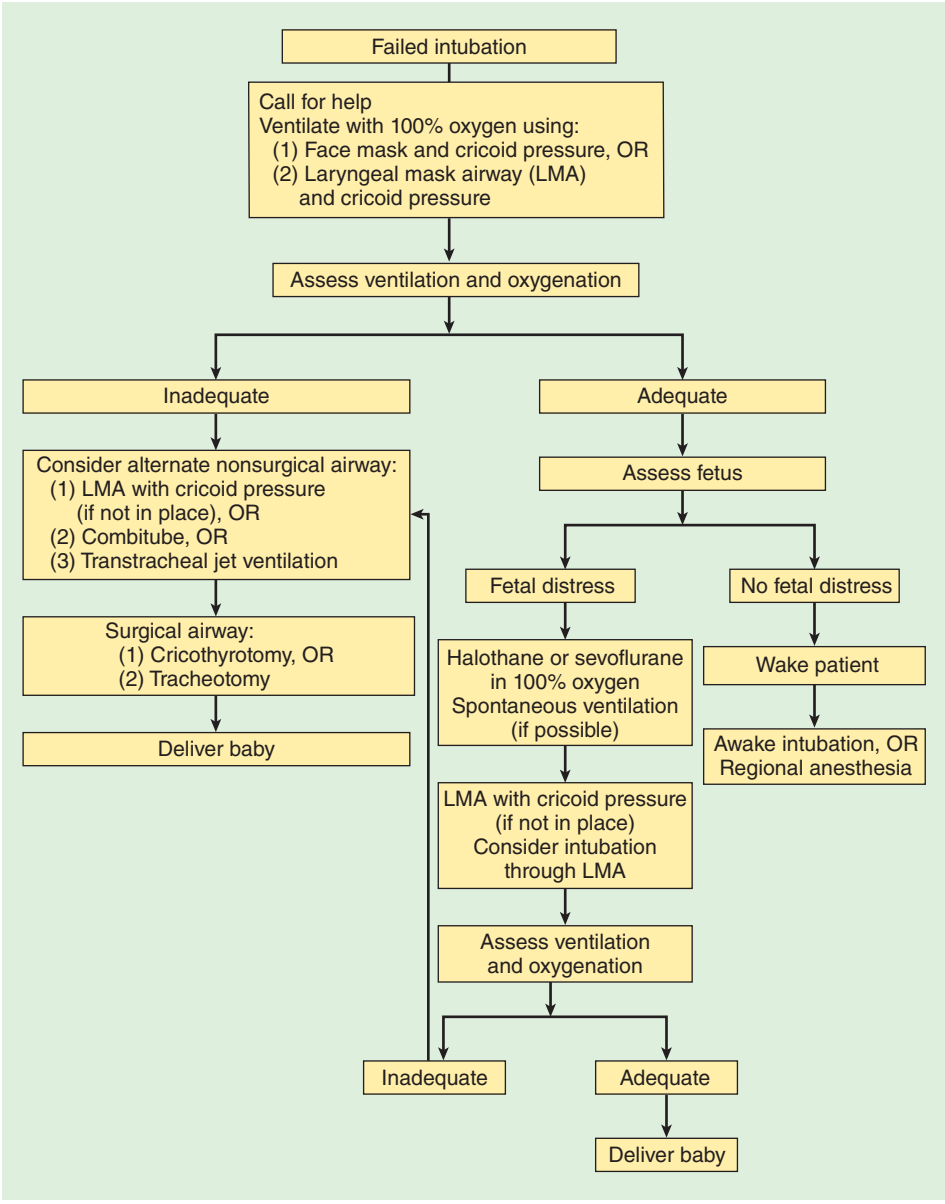

FIGURE 41–3 An algorithm for a difficult intubation in obstetric patients.

7. After the neonate and placenta are delivered, 20–80 units of oxytocin are added to the first liter of intravenous fluid, and another 20 units to the next. Additional intravenous agents, such as propofol, opioid, or benzodiazepine, can be given to ensure amnesia.

8. If the uterus does not contract readily, an opioid should be given, and the halogenated agent should be discontinued. Methylergonovine (Methergine), 0.2 mg intramuscularly or in 100-mL normal saline as slow intravenous infusion, may also be

given but can increase arterial blood pressure. 15-Methylprostaglandin $F_{2\alpha}$ (Hemabate), 0.25 mg intramuscularly, may also be used.

9. An attempt to aspirate gastric contents may be made via an oral gastric tube to decrease the likelihood of pulmonary aspiration on emergence.
10. At the end of surgery, muscle relaxants are completely reversed, the gastric tube (if placed) is removed, and the patient is extubated while awake to reduce the risk of aspiration.

ANESTHESIA FOR EMERGENCY CESAREAN SECTION

Indications for emergency cesarean section include massive bleeding (placenta previa or accreta, abruptio placentae, or uterine rupture), umbilical cord prolapse, and severe fetal distress. A distinction must be made between a true emergency requiring immediate delivery (previously referred to as "crash") and one in which some delay is possible. Close communication with the obstetrician is necessary to determine whether fetus, mother, or both are in immediate jeopardy.

The choice of anesthetic technique is determined by consideration for maternal safety (airway evaluation), technical issues, and the anesthesiologist's personal expertise. Criteria leading to the diagnosis of nonreassuring fetal status should be reviewed as the fetal evaluation may be based on criteria with poor predictive accuracy and the fetal status may change. This information is required to choose the anesthetic technique that will produce the best outcome for both mother and fetus. Rapid institution of regional anesthesia is an option in selected cases but is problematic in severely hypovolemic or hypotensive patients. If general anesthesia is chosen, adequate denitrogenation may be achieved rapidly with four maximal breaths of 100% oxygen while monitors are being applied. Ketamine, 1 mg/kg, may be substituted for propofol in hypotensive or hypovolemic patients.

Table 41–5 lists commonly accepted signs of fetal distress, an imprecise and poorly defined term. In most instances the diagnosis is primarily based on monitoring of fetal heart rate. Because worrisome fetal heart rate patterns have a relatively high incidence of false-positive results, careful interpretation of other parameters, such as fetal scalp pH or fetal pulse oximetry, may also be necessary. Moreover, continuation of fetal monitoring in the operating room may help avoid unnecessary induction of general anesthesia for fetal distress when additional time for use of regional anesthesia is possible. In selected instances where immediate delivery is not absolutely mandatory, epidural anesthesia (with 3% chloroprocaine or alkalinized 2% lidocaine) or spinal anesthesia may be appropriate.

TABLE 41–5 Signs of fetal distress.

Nonreassuring fetal heart rate pattern
Repetitive late decelerations
Loss of fetal beat-to-beat variability associated with late or deep decelerations
Sustained fetal heart rate <80 beats/min
Fetal scalp pH <7.20
Meconium-stained amniotic fluid
Intrauterine growth restriction

Anesthesia for the Complicated Pregnancy

UMBILICAL CORD PROLAPSE

Prolapse of the umbilical cord complicates 0.2–0.6% of deliveries. Umbilical cord compression following prolapse can rapidly lead to fetal asphyxia. Predisposing factors include excessive cord length, malpresentation, low birth weight, grand parity (more than five pregnancies), multiple gestations, and artificial rupture of membranes. The diagnosis is suspected after sudden fetal bradycardia or profound decelerations and is confirmed by physical examination. Treatment includes immediate steep Trendelenburg or knee–chest position and manual pushing of the presenting fetal part back up into the pelvis until immediate cesarean section under general anesthesia can be performed. If the fetus is not viable, vaginal delivery is allowed to continue.

DYSTOCIA & ABNORMAL FETAL PRESENTATIONS & POSITIONS

Primary Dysfunctional Labor

A prolonged latent phase by definition exceeds 20 h in a nulliparous parturient and 14 h in a multiparous patient. The cervix usually remains at 4 cm or less but is completely effaced. The etiology is likely ineffective contractions without a dominant myometrial pacemaker. Arrest of dilation is present when the cervix undergoes no further change after 2 h in the active phase of labor. A protracted active phase refers to slower than normal cervical dilation, defined as less than 1.2 cm/h in a nulliparous patient and less than 1.5 cm/h in a multiparous parturient. A prolonged deceleration phase occurs when cervical dilation slows markedly after 8 cm. The cervix becomes very edematous and appears to lose effacement. A prolonged second stage (disorder of descent) is defined as a descent of less than 1 cm/h and 2 cm/h in nulliparous and multiparous parturients, respectively. Failure of the head to descend 1 cm in station after adequate pushing is referred to as arrest of descent.

Oxytocin is usually the treatment of choice for uterine contractile abnormalities. The drug is given intravenously at 1–6 mU/min and increased in increments of 1–6 mU/min every 15–40 min, depending on the protocol. Use of amniotomy is controversial. Treatment is usually expectant management, as long as the fetus and mother are tolerating the prolonged labor. When a trial of oxytocin is unsuccessful or when malpresentation or cephalopelvic disproportion is also present, operative vaginal delivery or cesarean section is indicated.

Breech Presentation

Breech presentations complicate 3–4% of deliveries and significantly increase both maternal and fetal morbidity and mortality rates. Breech presentations increase neonatal mortality and the incidence of cord prolapse more than 10-fold. External cephalic version may be attempted after 34 weeks of gestation and prior to the onset of labor; however, the fetus may spontaneously return to the breech presentation before the onset of labor. Some obstetricians may administer a tocolytic agent at the same time. External version can be facilitated, and its success rate improved, by providing epidural analgesia with 2% lidocaine and fentanyl. Although an external version is successful in 75% of patients, it can cause placental abruption and umbilical cord compression necessitating immediate cesarean section.

Because the shoulders or head can become trapped after vaginal delivery of the body, some obstetricians employ cesarean section for all breech presentations. Manual or forceps-assisted partial breech extraction is usually necessary during these vaginal deliveries. The need for breech extraction does not appear to be increased when epidural anesthesia is used for labor—if labor is well established prior to epidural activation. Moreover, epidural anesthesia may decrease the likelihood of a trapped head, because the former relaxes the perineum. Nonetheless, the fetal head can become trapped in the uterus even during cesarean section under regional anesthesia; rapid induction of general endotracheal anesthesia and administration of a volatile agent may be attempted in such instances to relax the uterus. Alternatively, nitroglycerin, 50–100 mcg intravenously, can be administered.

Abnormal Vertex Presentations

When the fetal occiput fails to spontaneously rotate anteriorly, a persistent occiput posterior presentation results in a more prolonged and painful labor. Manual, vacuum, or forceps rotation is usually necessary but increases the likelihood of maternal and fetal injuries. Regional anesthesia can be used to provide perineal analgesia and pelvic relaxation, allowing manual or forceps rotation followed by forceps delivery.

A face presentation occurs when the fetal head is hyperextended and generally requires cesarean section. A compound presentation occurs when an extremity enters the pelvis along with either the head or the buttocks. Vaginal delivery is usually still possible because the extremity often withdraws as the labor progresses.

Shoulder dystocia, or impaction of a shoulder against the pubic symphysis, complicates 0.2–2% of deliveries and is one of the major causes of birth injuries. The most important risk factor is fetal

macrosomia. Shoulder dystocias are often difficult to predict. Several obstetric maneuvers can be used to relieve it, but a prolonged delay in the delivery could result in fetal asphyxia. Induction of general anesthesia may be necessary if an epidural catheter is not already in place.

MULTIPLE GESTATIONS

Multiple gestations account for approximately 1 in 150 births and are commonly associated with two complications: breech presentation and prematurity. Anesthesia may be necessary for version, extraction, or cesarean section. The second baby (and any subsequent ones) is often more depressed and asphyxiated than the first. Regional anesthesia provides effective pain relief during labor, minimizes the need for central nervous system depressants, and may shorten the interval between the birth of the first and second baby. Some studies suggest that the acid–base status of the second twin is better when epidural anesthesia is used. Patients with multiple gestations, however, are more prone to develop hypotension from aortocaval compression, particularly after regional anesthesia.

ANTEPARTUM HEMORRHAGE

14 Maternal hemorrhage is one of the most common severe morbidities complicating obstetric anesthesia. Causes include uterine atony, placenta previa, abruptio placentae, and uterine rupture.

Placenta Previa

A placenta previa is present if the placenta implants in advance of the fetal presenting part. The incidence of placenta previa is 0.5% of pregnancies. Placenta previa often occurs in patients who have had a previous cesarean section or uterine myomectomy; other risk factors include multiparity, advanced maternal age, and a large placenta. An anterior-lying placenta previa increases the risk of excessive bleeding for cesarean section.

Placenta previa usually presents as painless vaginal bleeding. Although the bleeding often stops spontaneously, severe hemorrhage can occur at any time. When the gestation is less than 37 weeks in duration and the bleeding is mild to moderate, the patient is usually treated with bed rest and observation. After 37 weeks of gestation, delivery is usually accomplished via cesarean section. Patients with low-lying placenta may rarely be allowed to deliver vaginally if the bleeding is mild.

Active bleeding or an unstable patient requires immediate cesarean section under general anesthesia. The patient should have two large-bore intravenous catheters in place; intravascular volume deficits must be replaced, and blood must be available for transfusion. The bleeding can continue after delivery because the placental implantation site in the lower uterine segment often does not contract well (as does the rest of the uterus).

A history of a previous placenta previa or cesarean section increases the risk of abnormal placentation.

Abruptio Placentae

Premature separation of a normal placenta complicates approximately 1–2% of pregnancies. Most abruptions are mild (grade I), but up to 25% are severe (grade III). Risk factors include hypertension, trauma, a short umbilical cord, multiparity, prolonged premature rupture of membranes, alcohol abuse, cocaine use, and an abnormal uterus. Patients usually experience painful vaginal bleeding with uterine contraction and tenderness. An abdominal ultrasound can help in the diagnosis. The choice between regional and general anesthesia must factor in the urgency for delivery, maternal hemodynamic stability, and any coagulopathy. The bleeding may remain concealed inside the uterus and cause underestimation of blood loss. Severe abruptio placentae can cause coagulopathy, particularly following fetal demise. Fibrinogen levels are mildly reduced (150–250 mg/dL) with moderate abruptions but are typically less than 150 mg/dL with fetal demise. The coagulopathy is thought to be due to activation of circulating plasminogen (fibrinolysis) and the release of tissue thromboplastins that precipitate disseminated intravascular coagulation (DIC). Platelet count and factors V and VIII are low, and fibrin split products are elevated. Severe abruption is a life-threatening emergency that necessitates an emergency cesarean section. Massive blood transfusion, including replacement of coagulation factors and platelets, may be anticipated.

Uterine Rupture

Uterine rupture is relatively uncommon (1:1000–3000 deliveries) but can occur during labor as a result of (1) dehiscence of a scar from a previous (usually classic) cesarean section (VBAC), extensive myomectomy, or uterine reconstruction; (2) intrauterine manipulations or use of forceps (iatrogenic); or (3) spontaneous rupture following prolonged labor in patients with hypertonic contractions (particularly with oxytocin infusions), fetopelvic disproportion, or a very large, thin, and weakened uterus. Uterine rupture can present as frank hemorrhage, fetal distress, loss of uterine tone, or hypotension with occult bleeding into the abdomen. Even when epidural anesthesia is employed for labor, uterine rupture is often heralded by the abrupt onset of continuous abdominal pain and hypotension. Treatment requires volume resuscitation and immediate laparotomy, typically under general anesthesia. Ligation of the internal iliac (hypogastric) arteries, with or without hysterectomy, may be necessary to control intraoperative bleeding.

PREMATURE RUPTURE OF MEMBRANES & CHORIOAMNIONITIS

Premature rupture of membranes (PROM) is present when leakage of amniotic fluid occurs before the onset of labor. The pH of amniotic fluid causes Nitrazine paper to change color from blue to yellow. PROM complicates 10% of all pregnancies and up to 35% of premature deliveries. Predisposing factors include a short cervix, prior history of PROM or preterm delivery, infection, multiple gestations, polyhydramnios, and smoking. Spontaneous labor commences within 24 h of ruptured membranes in 90% of patients. Management of PROM balances the risk of infection with the risk of fetal prematurity. Delivery is usually indicated sometime after 34 weeks of gestation. Patients with a gestation of less than 34 weeks can be managed expectantly with prophylactic antibiotics and tocolytics for 5–7 days to allow some additional maturation of fetal organs. The longer the interval between rupture and the onset of labor, the higher the incidence of chorioamnionitis. PROM also predisposes to placental abruption and postpartum endometritis.

Chorioamnionitis represents infection of the chorionic and amnionic membranes, and may involve the placenta, uterus, umbilical cord, and fetus. It complicates up to 1–2% of pregnancies and is usually but not always associated with ruptured membranes. The contents of the amniotic cavity are normally sterile but become vulnerable to ascending bacterial infection from the vagina when the cervix dilates or the membranes rupture. Intraamniotic infections are less commonly caused by hematogenous spread of bacteria or retrograde seeding through the fallopian tubes. The principal maternal complications of chorioamnionitis are premature or dysfunctional labor, often leading to cesarean section, intraabdominal infection, septicemia, and postpartum hemorrhage. Fetal complications include acidosis, hypoxia, and septicemia.

Clinical signs of chorioamnionitis include fever (>38°C), maternal and fetal tachycardia, uterine tenderness, and foul-smelling or purulent amniotic fluid. Blood leukocyte count is useful only if markedly elevated because it normally increases during labor (normal average 15,000/μL). C-reactive protein levels are usually elevated (>2 mg/dL). Gram stain of amniotic fluid obtained by amniocentesis is helpful in ruling out infection.

The use of regional anesthesia in patients with chorioamnionitis is controversial because of the theoretical risk of promoting the development of meningitis or an epidural abscess. Available evidence suggests that this risk is very low and that concerns may be unjustified. Moreover, antepartum antibiotic therapy appears to reduce maternal and fetal morbidity. Nonetheless, concerns over hemodynamic stability following sympathectomy are justified, particularly in patients with chills, high fever, tachypnea, changes in mental status, or borderline hypotension. In the absence of overt signs of septicemia, thrombocytopenia, or coagulopathy, most clinicians offer regional anesthesia to those patients with chorioamnionitis who have received antibiotic therapy.

PRETERM LABOR

Preterm labor by definition occurs between 20 and 37 weeks of gestation and is the most common complication of the third trimester. Approximately 8% of live-born infants in the United States are delivered before term. Important contributory maternal

factors include extremes of age, inadequate prenatal care, unusual body habitus, increased physical activity, infections, prior preterm labor, multiple gestation, and other medical illnesses or complications during pregnancy.

Because of their small size and incomplete development, preterm infants—particularly those less than 30 weeks of gestational age or weighing less than 1500 g—experience a greater number of complications than term infants. Premature rupture of membranes complicates one third of premature deliveries; the combination of premature rupture of membranes and premature labor increases the likelihood of umbilical cord compression resulting in fetal hypoxemia and asphyxia. Preterm infants with a breech presentation are particularly prone to prolapse of the umbilical cord during labor. Moreover, inadequate production of pulmonary surfactant frequently leads to the idiopathic respiratory distress syndrome (hyaline membrane disease) after delivery. Surfactant levels are generally adequate only after week 35 of gestation. Lastly, a soft, poorly calcified cranium predisposes these neonates to intracranial hemorrhage during vaginal delivery.

When preterm labor occurs before 35 weeks of gestation, bed rest and tocolytic therapy are usually initiated. Treatment is successful in 75% of patients. Labor is inhibited until the lungs mature and sufficient pulmonary surfactant is produced, as judged by amniocentesis. The risk of respiratory distress syndrome is markedly reduced when the amniotic fluid lecithin/sphingomyelin ratio is greater than 2. Glucocorticoid (betamethasone) may be given to induce production of pulmonary surfactant, which requires a minimum of 24–48 h. The most commonly used tocolytics are β_2-adrenergic agonists (ritodrine or terbutaline) and magnesium (6 g intravenously over 30 min followed by 2–4 g/h). Ritodrine (given intravenously as 100–350 mcg/min) and terbutaline (given orally as 2.5–5 mg every 4–6 h) also have some β_1-adrenergic receptor activity, which accounts for some of their side effects. Maternal side effects include tachycardia, arrhythmias, myocardial ischemia, mild hypotension, hyperglycemia, hypokalemia, and, rarely, pulmonary edema. Other tocolytic agents include calcium channel blockers (nifedipine), prostaglandin synthetase inhibitors, oxytocin antagonists (atosiban), and possibly nitric oxide. Fetal ductal constriction can occur after 32 weeks of gestation with nonsteroidal antiinflammatory drugs such as indomethacin, but it is usually transient and resolves after discontinuation of the drug; renal impairment in the fetus may also cause oligohydramnios.

When tocolytic therapy fails to arrest labor, anesthesia often becomes necessary. The goal during vaginal delivery of a preterm fetus is a slow controlled delivery with minimal pushing by the mother. An episiotomy and low forceps are often employed. Spinal or epidural anesthesia allows complete pelvic relaxation. Cesarean section is performed for fetal distress, breech presentation, intrauterine growth retardation, or failure of labor to progress. Residual effects from β-adrenergic agonists may complicate general anesthesia. The half-life of ritodrine may be as long as 3 h. Ketamine and ephedrine (and halothane) should be used cautiously due to interaction with tocolytics. Hypokalemia is usually due to an intracellular uptake of potassium and rarely requires treatment; however, it may increase sensitivity to muscle relaxants. Magnesium therapy potentiates muscle relaxants and may predispose to hypotension (secondary to vasodilation). Residual effects from tocolytics interfere with uterine contraction following delivery. Lastly, preterm newborns are often depressed at delivery and frequently need resuscitation. Preparations for resuscitation should be completed prior to delivery.

HYPERTENSIVE DISORDERS

Hypertension during pregnancy can be classified as pregnancy-induced hypertension (PIH, often also referred to as preeclampsia), chronic hypertension that preceded pregnancy, or chronic hypertension with superimposed preeclampsia. **Preeclampsia is usually defined as a systolic blood pressure greater than 140 mm Hg or diastolic pressure greater than 90 mm Hg after the 20th week of gestation, accompanied by proteinuria (>300 mg/d) and resolving within 48 h after delivery.** When seizures occur, the syndrome is termed eclampsia. The HELLP syndrome describes preeclampsia associated with *h*emolysis, *e*levated *l*iver enzymes, and a *l*ow

platelet count. In the United States, preeclampsia complicates approximately 7–10% of pregnancies; eclampsia is much less common, occurring in one of 10,000–15,000 pregnancies. Severe preeclampsia causes or contributes to 20–40% of maternal deaths and 20% of perinatal deaths. Maternal deaths are usually due to stroke, pulmonary edema, and hepatic necrosis or rupture.

Pathophysiology & Manifestations

The pathophysiology of preeclampsia is probably related to a vascular dysfunction of the placenta that results in abnormal prostaglandin metabolism. Patients with preeclampsia have elevated production of thromboxane A_2 (TXA_2) and decreased production of prostacyclin (PGI_2). TXA_2 is a potent vasoconstrictor and promoter of platelet aggregation, whereas PGI_2 is a potent vasodilator and inhibitor of platelet aggregation. Endothelial dysfunction may reduce production of nitric oxide and increase production of endothelin-1. The latter is also a potent vasoconstrictor and activator of platelets. Marked vascular reactivity and endothelial injury reduce placental perfusion and can lead to widespread systemic manifestations.

Severe preeclampsia substantially increases both maternal and fetal morbidity and mortality, and is defined by a blood pressure greater than 160/110 mm Hg, proteinuria in excess of 5 g/d, oliguria (<500 mL/d), elevated serum creatinine, intrauterine growth restriction, pulmonary edema, central nervous system manifestations (headache, visual disturbances, seizures, or stroke), hepatic tenderness, or the HELLP syndrome (Table 41-6). Hepatic rupture may also occur in patients with the HELLP syndrome.

Patients with severe preeclampsia or eclampsia have widely differing hemodynamic profiles. Most patients have low-normal cardiac filling pressures with high systemic vascular resistance, but cardiac output may be low, normal, or high.

TABLE 41–6 Complications of preeclampsia.

Neurological
Headache
Visual disturbances
Hyperexcitability
Seizures
Intracranial hemorrhage
Cerebral edema
Pulmonary
Upper airway edema
Pulmonary edema
Cardiovascular
Decreased intravascular volume
Increased arteriolar resistance
Hypertension
Heart failure
Hepatic
Impaired function
Elevated enzymes
Hematoma
Rupture
Renal
Proteinuria
Sodium retention
Decreased glomerular filtration
Renal failure
Hematological
Coagulopathy
Thrombocytopenia
Platelet dysfunction
Prolonged partial thromboplastin time
Microangiopathic hemolysis

Treatment

Treatment of preeclampsia consists of bed rest, sedation, repeated doses of antihypertensive drugs (usually labetalol, 5–10 mg, or hydralazine, 5 mg intravenously), and magnesium sulfate (4 g intravenous loading, followed by 1–3 g/h) to treat hyperreflexia and prevent convulsions. Therapeutic magnesium levels are 4–6 mEq/L.

Invasive arterial and central venous monitoring are indicated in patients with severe hypertension, pulmonary edema, or refractory oliguria; an intravenous vasodilator infusion may be necessary. Definitive treatment of preeclampsia is delivery of the fetus and placenta.

Anesthetic Management

Patients with mild preeclampsia generally require only extra caution during anesthesia; standard anesthetic practices may be used. Spinal and epidural anesthesia are associated with similar decreases in

arterial blood pressure in these patients. Patients with severe disease, however, are critically ill and require stabilization prior to administration of any anesthetic. Hypertension should be controlled and hypovolemia corrected before administration of anesthesia. In the absence of coagulopathy, continuous epidural anesthesia is the first choice for most patients with preeclampsia during labor, vaginal delivery, and cesarean section. Moreover, continuous epidural anesthesia avoids the increased risk of a failed intubation due to severe edema of the upper airway.

A platelet count and coagulation profile should be checked prior to the institution of regional anesthesia in patients with severe preeclampsia. It has been recommended that regional anesthesia be avoided if the platelet count is less than 100,000/μL, but a platelet count as low as 70,000/μL may be acceptable in selected cases, particularly when the count has been stable. Although some patients have a qualitative platelet defect, the usefulness of a bleeding time determination is questionable. Continuous epidural anesthesia has been shown to decrease catecholamine secretion and improve uteroplacental perfusion up to 75% in these patients, provided hypotension is avoided. Judicious fluid boluses with epidural activation may be required to correct the disease-related hypovolemia. Goal-directed hemodynamic and fluid therapy utilizing arterial pulse wave contour analysis (Virgileo/Flotrac, LiDCOrapid) or echocardiography may be employed to guide fluid replacement. Use of an epinephrine-containing test dose for epidural anesthesia is controversial because of questionable reliability (see earlier section Prevention of Unintentional Intravascular and Intrathecal Injection) and the risk of exacerbating hypertension. Hypotension should be treated with small doses of vasopressors because patients tend to be very sensitive to these agents. Recent evidence suggests that spinal anesthesia does not, as previously thought, result in a more severe reduction of maternal blood pressure. Therefore, this technique is a reasonable anesthetic choice for cesarean section in a preeclamptic patient.

Intraarterial blood pressure monitoring is indicated in patients with severe hypertension during both general and regional anesthesia. Intravenous vasodilator infusions may be necessary to control blood pressure during general anesthesia. Intravenous labetalol (5–10 mg increments) can also be effective in controlling the hypertensive response to intubation and does not appear to alter placental blood flow. Because magnesium potentiates muscle relaxants, doses of nondepolarizing muscle relaxants should be reduced in patients receiving magnesium therapy and should be guided by a peripheral nerve stimulator. The patient with suspected magnesium toxicity, manifested by hyporeflexia, excessive sedation, blurred vision, respiratory compromise and cardiac depression, can be treated with intravenous administration of calcium gluconate (1 g over 10 minutes).

HEART DISEASE

The marked cardiovascular changes associated with pregnancy, labor, and delivery often cause pregnant patients with heart disease (2% of parturients) to decompensate during this period. Although most pregnant patients with cardiac disease have rheumatic heart disease, an increasing number of parturients are presenting with corrected or palliated congenital lesions. Anesthetic management is directed toward employing techniques that minimize the added stresses of labor and delivery. Specific management of the various lesions is discussed elsewhere. Most patients can be divided into one of two groups. Patients in the first group benefit from the falls in systemic vascular resistance caused by neuraxial analgesia techniques, but usually not from overzealous fluid administration. These patients include those with mitral insufficiency, aortic insufficiency, chronic heart failure, or congenital lesions with left-to-right shunting. The induced sympathectomy from spinal or epidural techniques reduces both preload and afterload, relieves pulmonary congestion, and in some cases increases forward flow (cardiac output).

Patients in the second group do not benefit from a decrease in systemic vascular resistance. These patients include those with aortic stenosis, congenital lesions with right-to-left or bidirectional shunting, or primary pulmonary hypertension. Reductions in venous return (preload) or afterload are usually poorly tolerated. These patients are better managed with intraspinal opioids alone, systemic

medications, pudendal nerve blocks, and, if necessary, general anesthesia.

AMNIOTIC FLUID EMBOLISM

Amniotic fluid embolism is a rare (1:20,000 deliveries) but often lethal complication (86% mortality rate in some series) that can occur during labor, delivery, cesarean section, or postpartum. Mortality may exceed 50% in the first hour. Entry of amniotic fluid into the maternal circulation can occur through any break in the uteroplacental membranes. Such breaks may occur during normal delivery or cesarean section or following placental abruption, placenta previa, or uterine rupture. In addition to fetal debris, amniotic fluid contains various prostaglandins and leukotrienes, which appear to play an important role in the genesis of this syndrome. The alternate term anaphylactoid syndrome of pregnancy has been suggested to emphasize the role of chemical mediators in this syndrome.

Patients typically present with sudden tachypnea, cyanosis, shock, and generalized bleeding. Three major pathophysiological manifestations are responsible: (1) acute pulmonary embolism, (2) disseminated intravascular coagulation (DIC), and (3) uterine atony. Mental status changes, including seizures, and pulmonary edema may develop; the latter has both cardiogenic and noncardiogenic components. Acute left ventricular dysfunction is common. Although the diagnosis can be firmly established only by demonstrating fetal elements in the maternal circulation (usually at autopsy or less commonly by aspirating amniotic fluid from a central venous catheter), amniotic fluid embolism should always be suggested by sudden respiratory distress and circulatory collapse. The presentation may initially mimic acute pulmonary thromboembolism, venous air embolism, overwhelming septicemia, or hepatic rupture or cerebral hemorrhage in a patient with toxemia.

Treatment consists of cardiopulmonary resuscitation and supportive care. When cardiac arrest occurs prior to delivery of the fetus, the efficacy of closed-chest compressions may be marginal at best. Aortocaval compression impairs resuscitation in the supine position, whereas chest compressions are less effective in a lateral tilt position. Moreover, expeditious delivery appears to improve maternal and fetal outcome; immediate (cesarean) delivery should therefore be carried out. Once the patient is resuscitated, mechanical ventilation, fluid resuscitation, and inotropes are best provided under the guidance of invasive hemodynamic monitoring. Uterine atony is treated with oxytocin, methylergonovine, and prostaglandin $F_{2\alpha}$, whereas significant coagulopathies are treated with platelets and coagulation factors based on laboratory findings.

POSTPARTUM HEMORRHAGE

Postpartum hemorrhage is the leading cause of maternal mortality in developing countries. It is diagnosed when the postpartum blood loss exceeds 500 mL. Up to 4% of parturients may experience postpartum hemorrhage, which is often associated with a prolonged third stage of labor, preeclampsia, multiple gestations, and forceps delivery. Common causes include uterine atony, a retained placenta, obstetric lacerations, uterine inversion, and use of tocolytic agents prior to delivery. Atony is often associated with uterine overdistention (multiple gestation and polyhydramnios). Less commonly, a clotting defect may be responsible.

The anesthesiologist may be consulted to assist in venous access or fluid (and blood) resuscitation, as well as to provide anesthesia for careful examination of the vagina, cervix, and uterus. Perineal lacerations can usually be repaired with local anesthetic infiltration or pudendal nerve blocks. Residual anesthesia from prior epidural or spinal anesthesia facilitates examination of the patient; however, supplementation with an opioid, nitrous oxide, or both may be required. Induction of spinal or epidural anesthesia in the presence of hypovolemia is problematic. **General anesthesia is usually required for manual extraction of a retained placenta, reversion of an inverted uterus, or repair of a major laceration.** Uterine atony should be treated with oxytocin (20–30 units/L of intravenous fluid), methylergonovine (0.2 mg intramuscularly or in 100 mL of normal saline administered over 10 min intravenously), and prostaglandin $F_{2\alpha}$ (0.25 mg intramuscularly). Emergency laparotomy and hysterectomy may be necessary in rare instances. Early ligation of the

internal iliac (hypogastric) arteries may help avoid hysterectomy or reduce blood loss.

Fetal & Neonatal Resuscitation

FETAL RESUSCITATION

Resuscitation of the neonate starts during labor. Any compromise of the uteroplacental circulation readily produces fetal asphyxia. Intrauterine asphyxia during labor is the most common cause of neonatal depression. Fetal monitoring throughout labor is helpful in identifying which babies may be at risk, detecting fetal distress, and evaluating the effect of acute interventions. These include correcting maternal hypotension with fluids or vasopressors, supplemental oxygen, and decreasing uterine contraction (stopping oxytocin or administering tocolytics). Some studies suggest that the normal fetus can compensate for up to 45 min of relative hypoxia, a period termed fetal stress; the latter is associated with a marked redistribution of blood flow primarily to the heart, brain, and adrenal glands. With time, however, progressive lactic acidosis and asphyxia produce increasing fetal distress that necessitates immediate delivery.

1. Fetal Heart Rate Monitoring

Monitoring of fetal heart rate (FHR) is presently the most useful technique in assessing fetal well-being, although alone it has a 35–50% false-positive rate of predicting fetal compromise. Because of this, the term *fetal distress* in the context of FHR monitoring has been largely replaced with *nonreassuring* FHR. Correct interpretation of heart rate patterns is crucial. Three parameters are evaluated: baseline heart rate, baseline variability, and the relationship to uterine contractions (deceleration patterns). Monitoring of heart rate is most accurate when fetal scalp electrodes are used, but this may require rupture of the membranes and is not without complications (eg, amnionitis or fetal injury).

Baseline Heart Rate

The mature fetus normally has a baseline heart rate of 110–160 beats/min. An increased baseline heart rate may be due to prematurity, mild fetal hypoxia, chorioamnionitis, maternal fever, maternally administered drugs (anticholinergics or β agonists), or, rarely, hyperthyroidism. A decreased baseline heart rate may be due to a postterm pregnancy, fetal heart block, or fetal asphyxia.

Baseline Variability

The healthy mature fetus normally displays a baseline beat-to-beat (R wave to R wave) variability that can be classified as minimal (<5 beats/min), moderate (6–25 beats/min), or marked (>25 beats/min). Baseline variability, which is best assessed with scalp electrodes, has become an important sign of fetal well-being and represents a normally functioning autonomic system. **Sustained decreased baseline variability is a prominent sign of fetal asphyxia.** Central nervous system depressants (opioids, barbiturates, volatile anesthetics, benzodiazepines, or magnesium sulfate) and parasympatholytics (atropine) also decrease baseline variability, as do prematurity, fetal arrhythmias, and anencephaly. A sinusoidal pattern that resembles a smooth sine wave is associated with fetal depression (hypoxia, drugs, and anemia secondary to Rh isoimmunization).

Accelerations

Accelerations of FHR are defined as increases of 15 beats/min or more lasting for more than 15 s. Periodic accelerations in FHR reflect normal oxygenation and are usually related to fetal movements and to responses to uterine pressure. Such accelerations are generally considered reassuring. By 32 weeks, fetuses display periodic increases in baseline heart rate that are associated with fetal movements. Normal fetuses have 15–40 accelerations/h. The mechanism is thought to involve increases in catecholamine secretion with decreases in vagal tone. Accelerations diminish with fetal sleep, some drugs (opioids, magnesium, and atropine), as well as fetal hypoxia. Accelerations to fetal scalp or vibroacoustic stimulation are considered a reassuring sign of fetal well-being. The absence of both baseline variability and accelerations is nonreassuring and may be an important sign of fetal compromise.

Deceleration Patterns

A. Early (Type I) Decelerations

Early deceleration (usually 10–40 beats/min) (Figure 41–4A) is thought to be a vagal response

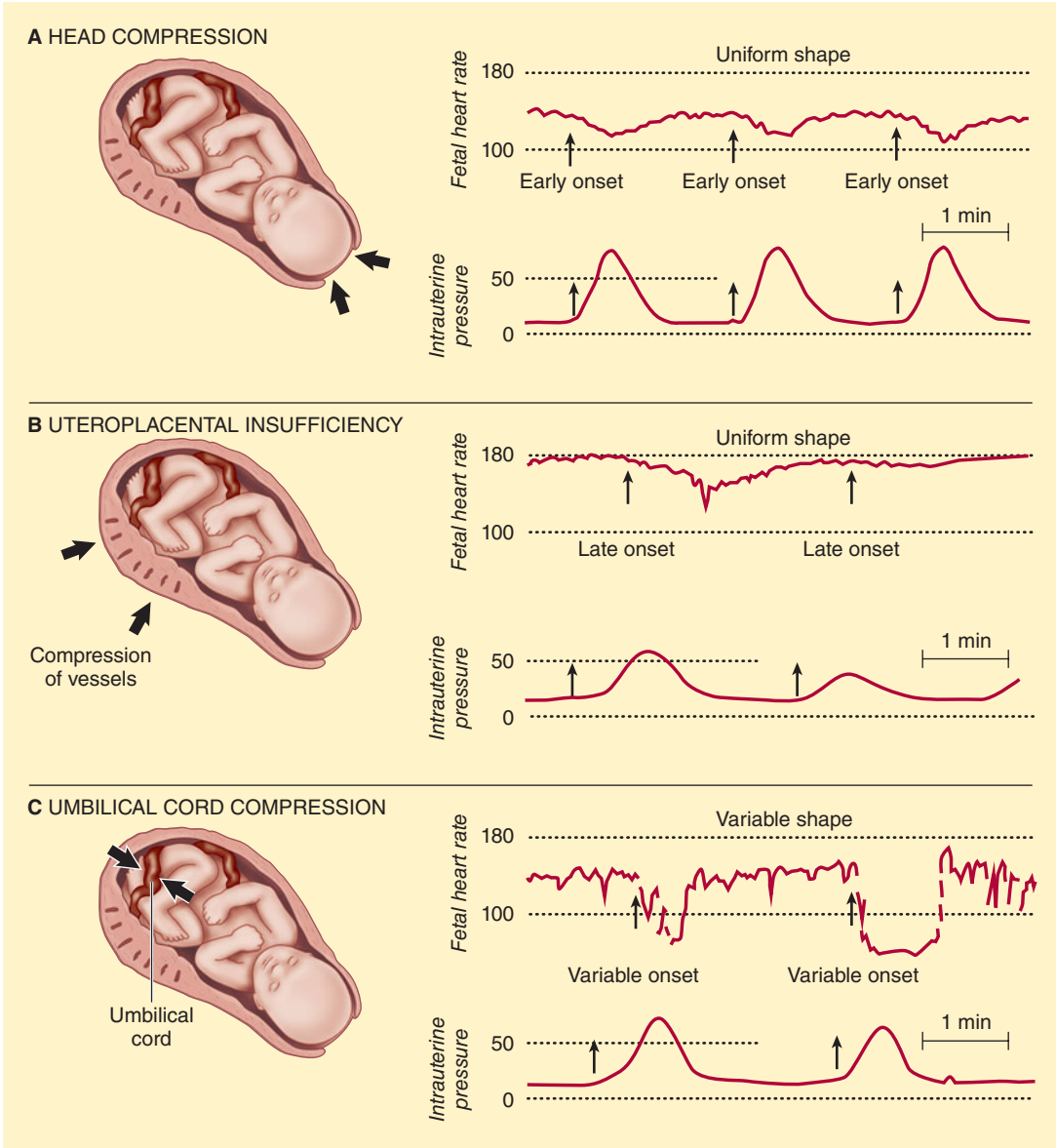

FIGURE 41–4 Periodic changes in fetal heart rate related to uterine contraction. **A:** Early (type I) decelerations. **B:** Late (type II) decelerations. **C:** Variable (type III) decelerations. (Reproduced, with permission, from Danforth DN, Scott JR: *Obstetrics and Gynecology*, 5th ed. Lippincott, 1986.)

to compression of the fetal head or stretching of the neck during uterine contractions. The heart rate forms a smooth mirror image of the contraction. Early decelerations are generally not associated with fetal distress and occur during descent of the head.

B. Late (Type II) Decelerations

Late decelerations (Figure 41–4B) are associated with fetal compromise and are characterized by a decrease in heart rate at or following the peak of uterine contractions. Late decelerations may be

subtle (as few as 5 beats/min). They are thought to represent decreased arterial oxygen tension on atrial chemoreceptors. Late decelerations with normal variability may be observed following acute insults (maternal hypotension or hypoxemia) and are usually reversible with treatment. Late decelerations with decreased variability are associated with prolonged asphyxia and may be an indication for fetal scalp sampling (see Other Monitoring section below). Complete abolition of variability in this setting is an ominous sign signifying severe decompensation and the need for immediate delivery.

C. Variable (Type III) Decelerations

The most common type of decelerations are variable (**Figure 41–4C**). These decelerations are variable in onset, duration, and magnitude (often >30 beats/min). They are typically abrupt in onset and are thought to be related to umbilical cord compression and acute intermittent decreases in umbilical blood flow. Variable decelerations are typically associated with fetal asphyxia when fetal heart rate declines to less than 60 beats/min, last more than 60 s, or occur in a pattern that persists for more than 30 min.

2. Other Monitoring

Other less commonly used monitors include fetal scalp pH measurements, scalp lactate concentration, fetal pulse oximetry, and fetal ST-segment analysis. Clinical experience is limited with all except fetal scalp pH measurements. Unfortunately the latter is associated with a small but significant incidence of false negatives and false positives. Fetal blood can be obtained and analyzed via a small scalp puncture once the membranes are ruptured. A fetal scalp pH higher than 7.20 is usually associated with a vigorous neonate, whereas a pH less than 7.20 is often, but not always, associated with a depressed neonate and necessitates prompt (typically operative) delivery. Because of wide overlap, fetal blood sampling can be interpreted correctly only in conjunction with heart rate monitoring.

3. Treatment of the Fetus

Treatment of intrauterine fetal asphyxia is aimed at preventing fetal demise or permanent neurological damage. All interventions are directed at restoring an adequate uteroplacental circulation. Aortocaval compression, maternal hypoxemia or hypotension, or excessive uterine activity (during oxytocin infusions) must be corrected. Changes in maternal position, supplemental oxygen, and intravenous ephedrine or fluid, or adjustments in an oxytocin infusion often correct the problem. Failure to relieve fetal stress, as well as progressive fetal acidosis and asphyxia, necessitate immediate delivery.

NEONATAL RESUSCITATION

1. General Care of the Neonate

One healthcare provider whose sole responsibility is to care for the neonate and who is capable of providing resuscitation should attend every delivery. As the head is delivered, the nose, mouth, and pharynx are suctioned with a bulb syringe. After the remainder of the body is delivered, the skin is dried with a sterile towel. Once the umbilical cord stops pulsating or neonatal breathing is initiated, the cord is clamped and the neonate is placed in a radiant warmer with the bed tilted in a slight Trendelenburg position.

Neonatal evaluation and treatment are carried out simultaneously (**Figure 41–5**). If the neonate is obviously depressed, the cord is clamped early and resuscitation is initiated immediately. Breathing normally begins within 30 s and is sustained within 90 s. Respirations should be 30–60 breaths/min and the heart rate 120–160 beats/min. Respirations are assessed by auscultation of the chest, whereas heart rate is determined by palpation of the pulse at the base of the umbilical cord or auscultation of the precordium. It is critically important to keep the neonate warm.

In addition to respirations and heart rate, color, tone, and reflex irritability should be evaluated. The Apgar score (**Table 41–7**), recorded at 1 min and again at 5 min after delivery, remains the most valuable assessment of the neonate. The 1-min score correlates with survival, whereas the 5-min score has limited relationship to neurological outcome.

Neonates with Apgar scores of 8–10 are vigorous and may require only gentle stimulation (flicking the foot, rubbing the back, and additional drying). A catheter should first be gently passed

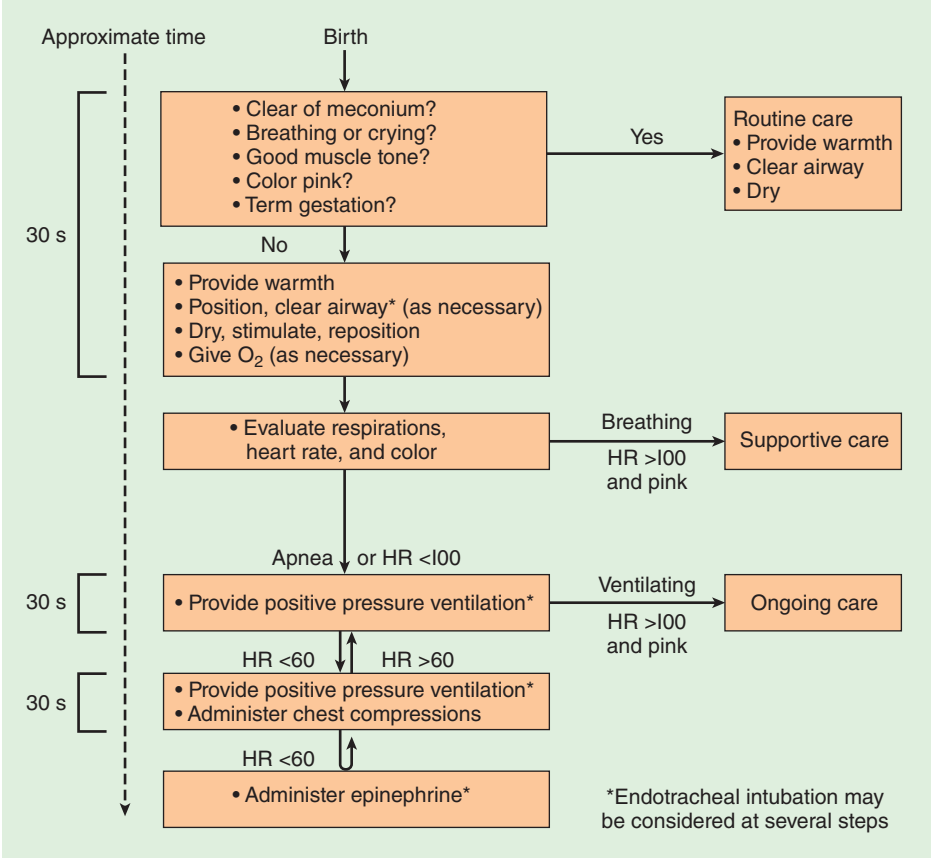

FIGURE 41–5 An algorithm for resuscitation of the newly born infant. (Reproduced, with permission, from ECC Committee, Subcommittees and Task Forces of the American Heart Association: 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2005 Dec 13;112(24 Suppl):IV1-203.)

TABLE 41–7 Apgar score.

	Points		
Sign	***0***	***1***	***2***
Heart rate (beats/min)	Absent	<100	>100
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Flaccid	Some flexion	Active motion
Reflex irritability	No response	Grimace	Crying
Color	Blue or pale	Body pink, extremities blue	All pink

through each nostril to rule out choanal atresia, and then through the mouth to suction the stomach and rule out esophageal atresia.

2. Meconium-Stained Neonates

The presence or absence of meconium in the amniotic fluid (approximately 10–12% of deliveries) changes the immediate management of the neonate at birth. Fetal distress, particularly after 42 weeks of gestation, is often associated with release of thick meconium into the fluid. Fetal gasping during stress results in entry of a large amount of meconium-tainted amniotic fluid into the lungs. When the neonate initiates respiration at birth, the meconium moves from the trachea and large airways down

toward the periphery of the lung. Thick or particulate meconium may obstruct small airways and cause severe respiratory distress in 15% of meconium-stained neonates. Moreover, these infants can develop persistent fetal circulation.

Unless the neonate has absent or depressed respirations, thin watery meconium does not require suctioning beyond careful bulb suctioning of the oropharynx when the head emerges from the perineum (or from the uterus at cesarean section). When thick "pea soup" meconium is present in the amniotic fluid, however, some clinicians intubate and suction the trachea immediately after delivery but before the first breath is taken. If the baby is not vigorous, tracheal suctioning is recommended when meconium is present. Tracheal suctioning of the thick meconium is accomplished by a special suctioning device attached to the endotracheal tube as the tube is withdrawn. If meconium is aspirated from the trachea, the procedure should be repeated until no meconium is obtained—but no more than three times, after which it is usually of no further benefit. The infant should then be given supplemental oxygen by face mask and observed closely. The stomach should also be suctioned to prevent passive regurgitation of any meconium. Newborns with meconium aspiration have an increased incidence of pneumothorax (10% compared with 1% for all vaginal deliveries).

3. Care of the Depressed Neonate

Approximately 6% of newborns, most of whom weigh less than 1500 g, require some form of advanced life support. Resuscitation of the depressed neonate requires two or more persons—one to manage the airway and ventilation and another to perform chest compressions, if necessary. A third person greatly facilitates the placement of intravascular catheters and the administration of fluids or drugs. The anesthesiologist caring for the mother can render only brief assistance and only when it does not jeopardize the mother; other personnel are, therefore, usually responsible for neonatal resuscitation.

Because the most common cause of neonatal depression is intrauterine asphyxia, the emphasis in resuscitation is on respiration. Hypovolemia is also a contributing factor in a significant number of neonates. Factors associated with hypovolemia include early clamping of the umbilical cord, holding the neonate above the introitus prior to clamping, prematurity, maternal hemorrhage, placental transection during cesarean section, sepsis, and twin-to-twin transfusion.

Failure of the neonate to quickly respond to respiratory resuscitative efforts mandates vascular access and blood gas analysis; pneumothorax (1% incidence) and congenital anomalies of the airway, including tracheoesophageal fistula (1:3000–5000 live births), and congenital diaphragmatic hernia (1:2000–4000) should also be considered.

Grouping by the 1-min Apgar score greatly facilitates resuscitation: (1) mildly asphyxiated neonates (Apgar score of 5–7) usually need only stimulation while 100% oxygen is blown across the face; (2) moderately asphyxiated neonates (Apgar score of 3–4) require temporary assisted positive-pressure ventilation with mask and bag; and (3) severely depressed neonates (Apgar score of 0–2) should be immediately intubated, and chest compressions may be required.

Guidelines for Ventilation

Indications for positive-pressure ventilation include (1) apnea, (2) gasping respirations, (3) persistent central cyanosis with 100% oxygen, and (4) a persistent heart rate less than 100 beats/min. Excessive flexion or extension of the neck can cause airway obstruction. A 1-in.-high towel under the shoulders may be helpful in maintaining proper head position. Assisted ventilation by bag and mask should be at a rate of 30–60 breaths/min with 100% oxygen. Initial breaths may require peak pressures of up to 40 cm H_2O, but pressures should not exceed 30 cm H_2O thereafter. Adequacy of ventilation should be checked by auscultation and chest excursions. Gastric decompression with an 8F tube often facilitates ventilation. If after 30 s the heart rate is over 100 beats/min and spontaneous ventilations become adequate, assisted ventilation is no longer necessary. If the heart rate remains less than 60 beats/min or 60–80 beats/min without an increase in response to resuscitation, the neonate is intubated and chest compressions are started.

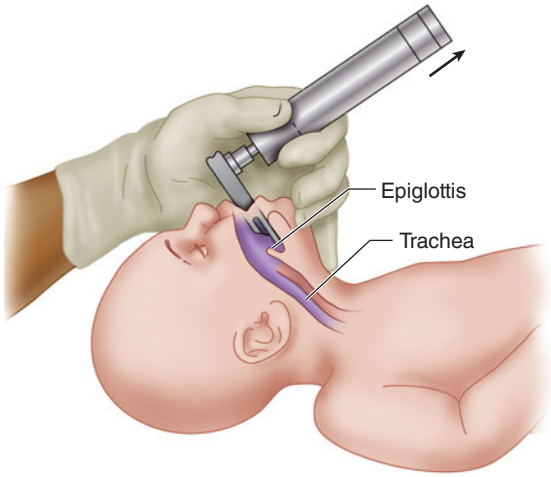

FIGURE 41–6 Intubation of the neonate. The head is placed in a neutral position, and the laryngoscope handle is held with the thumb and index finger as the chin is supported with the remaining fingers. Pressure applied over the hyoid bone with the little finger will bring the larynx into view. A straight blade such as a Miller 0 or 1 usually provides the best view.

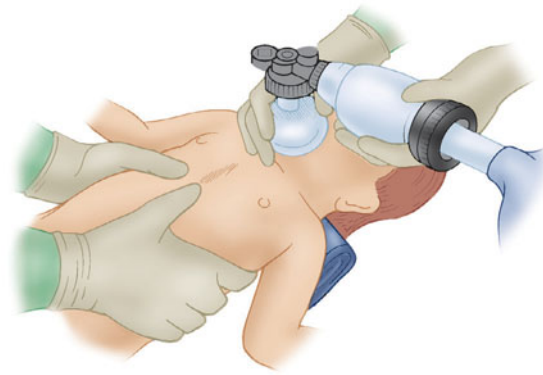

FIGURE 41–7 Chest compressions in the neonate. The neonate is held with both hands as each thumb is placed just beneath a line connecting the nipples and the remaining fingers encircle the chest. The sternum is compressed ⅓ to ¾ in. (1 cm) at a rate of 120/min. (Reproduced with permission from Rudolph CD, et al. *Rudolph's Pediatrics*. 22nd ed. McGraw-Hill; 2011.)

If the heart rate is 60–80 beats/min and increasing, assisted ventilation is continued and the neonate is observed. Failure of the heart rate to rise above 80 beats/min is an indication for chest compressions. Indications for endotracheal intubation include ineffective or prolonged mask ventilation and the need to administer medications.

Intubation (Figure 41–6) is performed with a Miller 00, 0, or 1 laryngoscope blade, using a 2.5-, 3-, or 3.5-mm endotracheal tube (for neonates <1 kg, 1–2 kg, and >2 kg, respectively). Correct endotracheal tube size is indicated by a small leak with 20 cm H_2O pressure. Right endobronchial intubation should be excluded by chest auscultation. The correct depth of the endotracheal tube ("tip to lip") is usually 6 cm plus the weight in kilograms. Oxygen saturation can usually be measured by a pulse oximeter probe applied to the palm. Capnography is also very useful in confirming endotracheal intubation. Transcutaneous oxygen sensors are useful for measuring tissue oxygenation but require time for initial equilibration. Use of a laryngeal mask airway (LMA#1) has been reported in neonates weighing more than 2.5 kg and may be useful if endotracheal intubation is difficult (eg, Pierre Robin syndrome).

Guidelines for Chest Compressions

Indications for chest compressions are a heart rate that is less than 60 beats/min or 60–80 beats/min and not rising after 30 s of adequate ventilation with 100% oxygen.

Cardiac compressions should be provided at a rate of 120/min. The two thumb/encircling hands technique (Figure 41–7) is generally preferred because it appears to generate higher peak systolic and coronary perfusion pressures. Alternatively, the two-finger technique can be used (Figure 41–8). The depth of compressions should be approximately one third of the anterior–posterior diameter of the chest and enough to generate a palpable pulse.

Compressions should be interposed with ventilation in a 3:1 ratio, such that 90 compressions and 30 ventilations are given per minute. The heart rate should be checked periodically. Chest compressions should be stopped when the spontaneous heart rate exceeds 80 beats/min.

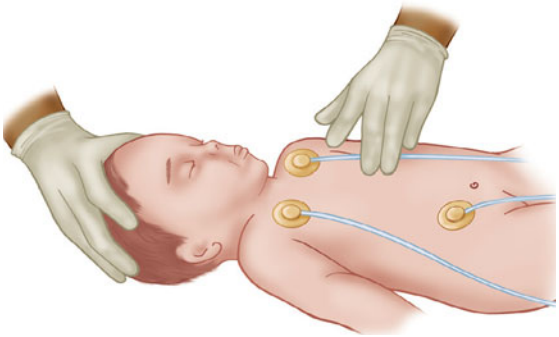

FIGURE 41–8 The alternative technique for neonatal chest compressions: two fingers are placed on the lower third of the sternum at right angles to the chest. The chest is compressed approximately 1 cm at a rate of 120/min.

Vascular Access

Cannulation of the umbilical vein with a 3.5F or 5F umbilical catheter is easiest and the preferred technique. The tip of the catheter should be just below skin level and allow free backflow of blood; further advancement may result in infusion of hypertonic solutions directly into the liver. A peripheral vein or even the endotracheal tube can be used as an alternate route for drug administration.

Cannulation of one of the two umbilical arteries allows measurement of blood pressure and facilitates blood gas measurements but may be more difficult. Specially designed umbilical artery catheters allow continuous Pao_2 or oxygen saturation monitoring as well as blood pressure. Care must be taken not to introduce any air into either the artery or the vein.

Volume Resuscitation

Some neonates at term and nearly two thirds of premature infants requiring resuscitation are hypovolemic at birth. Diagnosis is based on physical examination (low blood pressure and pallor) and a poor response to resuscitation. Neonatal blood pressure generally correlates with intravascular volume and should therefore routinely be measured. Normal blood pressure depends on birth weight and varies from 50/25 mm Hg for neonates weighing 1–2 kg to 70/40 mm Hg for those weighing over 3 kg. A low blood pressure suggests hypovolemia. Volume expansion may be accomplished with 10 mL/kg of lactated Ringer's injection, normal saline, or type O-negative blood cross-matched with maternal blood. Less common causes of hypotension include hypocalcemia, hypermagnesemia, and hypoglycemia.

Drug Therapy

A. Epinephrine

Epinephrine, 0.01–0.03 mg/kg (0.1–0.3 mL/kg of a 1:10,000 solution), should be given for asystole or a spontaneous heart rate of less than 60 beats/min in spite of adequate ventilation and chest compressions. It may be repeated every 3–5 min. Epinephrine may be given in 1 mL of saline via the endotracheal tube when venous access is not available.

B. Naloxone

Naloxone, 0.1 mg/kg intravenously or 0.2 mg/kg intramuscularly, is given to reverse the respiratory depressant effect of opioids given to the mother in the last 4 h of labor. Withdrawal symptoms may be precipitated in babies of mothers who chronically consume prescribed or illicit opioids.

C. Other Drugs

Other drugs may be indicated only in specific settings. Sodium bicarbonate (2 mEq/kg of a 0.5 mEq/mL 4.2% solution) should usually be given only for a severe metabolic acidosis documented by blood gas measurements and when ventilation is adequate. It may also be administered during prolonged resuscitation (>5 min)—particularly if blood gas measurements are not readily available. The infusion rate should not exceed 1 mEq/kg/min to avoid hypertonicity and intracranial hemorrhage. As noted above, in order to prevent hypertonicity-induced hepatic injury, the umbilical vein catheter tip should not be in the liver. Calcium gluconate 100 mg/kg ($CaCl_2$, 30 mg/kg) should be given only to neonates with documented hypocalcemia or those with suspected magnesium intoxication (from maternal magnesium therapy); these neonates are usually hypotensive, hypotonic, and appear vasodilated. Glucose (8 mg/kg/min of a 10% solution) is given only for documented hypoglycemia because hyperglycemia worsens hypoxic neurological deficits. Blood glucose should be measured because up to 10% of neonates may have hypoglycemia (glucose <35 mg/dL), particularly those delivered by cesarean

section. Dopamine may be started at 5 mcg/kg/min to support arterial blood pressure. Lastly, surfactant may be given through the endotracheal tube to premature neonates with respiratory distress syndrome.

CASE DISCUSSION

Appendicitis in a Pregnant Woman

A 31-year-old woman with a 24-week gestation presents for an appendectomy.

How does pregnancy complicate the management of this patient?

Nearly 1–2% of pregnant patients require surgery during their pregnancy. The most common procedure during the first trimester is laparoscopy; appendectomy (1:1500 pregnancies) and cholecystectomy (1:2000–10,000 pregnancies) are the most commonly performed general surgical procedures. Cervical cerclage may be necessary in some patients for cervical incompetence. The physiological effects of pregnancy can alter the manifestations of disease process and make diagnosis difficult. Patients may therefore present with advanced or complicated disease. The physiological changes associated with pregnancy (see Chapter 40) further predispose the patient to increased morbidity and mortality. Moreover, both the operation and the anesthesia can adversely affect the fetus.

What are the potentially detrimental effects of surgery and anesthesia on the fetus?

The procedure can have both immediate and long-term undesirable effects on the fetus. Maternal hypotension, hypovolemia, severe anemia, hypoxemia, and marked increases in sympathetic tone can seriously compromise the transfer of oxygen and other nutrients across the uteroplacental circulation and promote intrauterine fetal asphyxia. The stress of the operative procedure and the underlying process may also precipitate preterm labor, which often follows intraabdominal surgery near the uterus. Laparoscopy may be safely performed although CO_2 insufflation has the potential to cause fetal respiratory acidosis. Mild to moderate maternal hyperventilation and limiting both insufflation pressure and duration of the procedure limit the degree of acidosis. Long-term detrimental effects relate to possible teratogenic effects on the developing fetus.

When is the fetus most sensitive to teratogenic influences?

Three stages of susceptibility are generally recognized. In the first 2 weeks of intrauterine life, teratogens have either a lethal effect or no effect on the embryo. The third to eighth weeks are the most critical period, when organogenesis takes place; drug exposure during this period can produce major developmental abnormalities. From the eighth week onward, organogenesis is complete, and organ growth takes place. Teratogen exposure during this last period usually results in only minor morphological abnormalities but can produce significant physiological abnormalities and growth retardation. Although the teratogenic influences of anesthetic agents have been extensively studied in animals, retrospective human studies have been inconclusive. Past concerns about possible teratogenic effects of nitrous oxide and benzodiazepines do not appear to be justified. Nonetheless, exposure to all anesthetic agents should be kept to a minimum in terms of the total number of agents, dosage, and duration of exposure. We tend to administer only those agents that are required—and in our practice, nitrous oxide is never required and benzodiazepines are only rarely needed—in a pregnant patient.

What would be the ideal anesthetic technique in this patient?

Toward the end of the second trimester (after 20–24 weeks of gestation), most of the major physiological changes associated with pregnancy have taken place. Regional anesthesia, when feasible, is preferable to general anesthesia in order to decrease the risks of pulmonary aspiration and failed intubation and to minimize drug exposure to the fetus. The patient should be maintained with left lateral uterine displacement when supine. Total drug exposure is least with spinal anesthesia. Moreover, spinal anesthesia may be preferable to epidural anesthesia because it is not associated

with unintentional intravascular injection or the potential for accidental intrathecal injection of large epidural doses of local anesthetic. On the other hand, general anesthesia guarantees patient comfort and, when a volatile agent is used, may even suppress preterm labor (see Chapter 40). Nitrous oxide without concomitant administration of a halogenated anesthetic is reported to reduce uterine blood flow.

Although regional anesthesia is preferable in most instances, the choice between regional and general anesthesia must be individualized according to the patient, the anesthesiologist, and the type of surgery. Spinal anesthesia is usually satisfactory for open appendectomies, whereas general anesthesia is appropriate for laparoscopic procedures.

Are any special monitors indicated perioperatively?

In addition to standard monitors, fetal heart rate and uterine activity should be monitored with a Doppler and tocodynamometer immediately prior to surgery and during anesthesia recovery in a woman who is 24 weeks or more pregnant. When regular organized uterine activity is detected, early treatment with a β-adrenergic agonist such as ritodrine usually aborts the preterm labor. Magnesium sulfate and oral or rectal indomethacin may also be used as tocolytics.

When should elective operations be performed during pregnancy?

All elective operations should be postponed until 6 weeks after delivery. Only emergency procedures that pose an immediate threat to the mother or fetus should be routinely performed. The timing of semielective procedures, such as those for cancer, valvular heart disease, or intracranial aneurysms, must be individualized and must balance the threat to maternal health versus fetal well-being. Controlled (deliberate) hypotensive anesthesia has been utilized to reduce blood loss during extensive cancer operations; nitroprusside, nitroglycerin, and hydralazine have been used during pregnancy without apparent fetal compromise. Nonetheless, large doses and prolonged infusions of nitroprusside should be avoided because the immature liver of the fetus may have a limited ability to metabolize the cyanide breakdown product. Cardiopulmonary bypass has been employed in pregnant patients successfully without adverse fetal outcome. Elective use of circulatory arrest during pregnancy is not recommended.

GUIDELINES

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