This article provides an in-depth review of the anatomic and physiologic changes of pregnancy. The author discusses how these changes impact on the anesthetic management of the obstetrical patient.

Pregnancy, in most situations, is a time of anxious anticipation. Throughout the "evolution" of pregnancy, the body of the expectant mother undergoes major anatomic and physiologic changes. Perhaps the most significant changes of pregnancy are noted to occur in the cardiovascular, respiratory and gastrointestinal systems. This article will focus primarily on these areas.

**Cardiovascular system**

One of the major alterations to the cardiovascular system seen in the pregnant patient is that of blood volume. During pregnancy, an increase is seen in plasma volume (up to 50%), red blood cell volume (up to 20%) and total blood volume (up to 35%). Healthy women in a normal first pregnancy increase their plasma volume from a nonpregnant level by approximately 1250 ml. In subsequent pregnancies, the increase is greater and may be as high as 1500 ml. Most of the rise takes place before 32-34 weeks gestation; thereafter, there is relatively little change. The increase is related to the size of the fetus, and there are particularly large increases in plasma volume associated with a multiple pregnancy.¹

An increase in red blood cell volume is also noted; the amount of increase seen may be attributed to the use or lack of iron supplementation during pregnancy.

With regard to blood volume, it is first noted to expand at approximately 12 weeks gestation, increasing rapidly during the second trimester and more slowly during the third trimester. Earlier data obtained with the parturient supine had suggested that blood volume decreased after 34 weeks gestation. However, more recent measurements performed with the gravid uterus positioned to avoid aortocaval compression (left lateral position) show no such diminution after delivery.

Blood loss at vaginal delivery ranges from 250-500 ml, with cesarean section, it ranges from 500-1000 ml. At delivery, autotransfusion of as much as 500 ml of blood into the maternal circulation from the uterus and placenta minimizes the impact of maternal hemorrhage. Blood volume returns to nonpregnant levels by the second postpartum week.² Blood volume near term ranges between 73-96 ml/kg.³

The above mentioned changes in blood volume result in hemodilution with a subsequent decrease in red blood cell volume, hematocrit, hemoglobin and serum protein levels.

An increase in cardiac output is the most impressive hemodynamic change during pregnancy. This increase begins at the eighth week of gestation and reaches a maximum of 30-50% above normal near term.⁴ During early labor, cardiac output increases a further 15% in response to catecholamine
secretion associated with pain. Also, augmented venous return occurs during each contraction, when 300-500 ml of blood are expelled into circulation. In the second stage of labor, cardiac output increases to 45% above prelabor values. This rises to 80% above prelabor values during the third stage.2

The increased output of the heart during labor is achieved by an increase in heart rate and an increase in stroke volume.1

Maternal position and its effect on cardiac output must also be discussed. The uterus occludes the inferior vena cava, in addition to displacing and partly obstructing the aorta and its branches. This is a usual and not exceptional finding in women in the supine position late in pregnancy; uterine contraction and maternal hypotension enhance markedly the obstruction effects. The vascular region exposed to uterine compression is the most protruding part of the vertebral column at the level of lumbar lordosis, L4-L5. The force of the contracting myometrium added to uterine gravitation may exert a pressure 2-3 times greater than the same force distributed over the large surface area of the amniotic cavity. It is sufficient to completely occlude the artery traversing this region. Although a direct mechanical effect could hardly be expected as high as L1, flow through the right renal artery is frequently seen to be obstructed. Retrograde flow through the left ovarian vein suggests occlusion of the left renal vein at the same level.5

To continue, a decreased peripheral vascular resistance (PVR) results in a small decrease in systolic blood pressure and, in midpregnancy, a more marked decrease in diastolic blood pressure. An elevated blood pressure is normal, except in labor, when factors which modify cardiac output temporarily increase blood pressure as previously described.

Central venous pressure is found to remain normal during pregnancy, but is elevated 4-5 centimeters of H2O with each contraction due to transient blood volume increases. Maternal bearing down and oxytocin infusion also increase central venous pressure.

Cardiac wall thickness and chamber volume increase during pregnancy, and upward displacement of the diaphragm causes the heart to become elevated and dextroverted. This can result in left axis deviation changes on the electrocardiogram. Arrhythmias occur more commonly than usual in pregnancy but are usually not significant.2

Anesthetic considerations: Due to the relative state of "hypervolemia," blood loss at the time of delivery is generally well tolerated. One must constantly reassess the parameters of oxygen availability, that is, hemoglobin, oxygen saturation, cardiac output, and so forth, when deciding if transfusion is necessary.

Induction of spinal or epidural anesthesia or other procedures that entail vasomotor block deprive the pregnant patient of compensatory vasoconstriction and usually result in a much greater decrease in arterial pressure than in the nonpregnant state. Patients with severe supine hypotensive syndrome (aortocaval syndrome) are particularly vulnerable and unless proper precautions are taken, severe hypotension may develop such as to threaten the life of the mother and the fetus. The anesthetist should ascertain the degree of supine hypotension prior to the induction of the block by measuring the blood pressure first with the patient on her left side, and then again after she has been supine for 5-15 minutes.

In patients who manifest supine hypotension, it may be desirable to avoid techniques which produce vasomotor block. If they are used, prophylactic measures against severe arterial hypotension must be taken, such as infusion of fluids prior to block and lateral displacement of the uterus, or having the patient lie on her side after induction of the block.4 The above "precautions" should be noted on all parturients, and the above mentioned anesthetic steps of management should be employed on all patients undergoing regional block for labor and delivery.

During pregnancy, the peridural venous plexus is found to be distended. This distension leads to: (1) increased epidural space pressure, with less reliability on the "hanging drop" technique, (2) decreased epidural capacity, mandating use of smaller volumes of local anesthetics and (3) increased risk of intravascular placement of the epidural needle and catheter, with greater potential for local anesthetic toxicity.2 It should be noted that the dosage for spinal anesthetics must also be reduced.

To continue, with regard to spinal anesthesia, the fluctuation in cerebrospinal fluid pressure produced by uterine contractions, bearing-down efforts or straining promotes turbulent currents in the fluid compartment. Consequently, injection of a hyperbaric local anesthetic solution into the subarachnoid space during such conditions is likely to result in an abnormally high level of spinal anesthesia. This effect may be accentuated by the abnormal curvature of the spine associated with pregnancy.4

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Respiratory system

During pregnancy, the respiratory system is noted to undergo several dramatic anatomic and physiologic changes involving the patient's airway, lung volumes and ventilatory mechanics. During labor and delivery, these changes are further modified as a result of anxiety, pain and the use of analgesic drugs.

In the majority of pregnant women, capillary engorgement takes place throughout the respiratory tract so that the nasopharynx, larynx, trachea and bronchi become swollen and reddened. These changes simulate inflammation often causing changes in the voice and make breathing through the nose difficult for women at term. The growing uterus causes the diaphragm to rest 4 centimeters higher than in the nonpregnant state but does not impair its excursions. The abdominal muscles have much less tone and are less active in the pregnant than in the nonpregnant state. A reduction in the total lung capacity (TLC) due to a rise in the diaphragm might reasonably be expected, but it is compensated to a large extent by the increased anterior-posterior and transverse diameters of the rib cage as illustrated in Figure 1.

Significant ventilatory changes are not seen until the fifth or sixth month of pregnancy; Figure 2 demonstrates the alterations in lung volumes seen in pregnancy. After the fifth or sixth month of pregnancy, there is a progressive decrease of
both the expiratory reserve volume (ERV) and residual volume (RV). At term, the sum of these two volumes, known as the functional residual capacity (FRC), is decreased some 17%. However, there is a concomitant increase in the inspiratory capacity (IC) with the result that the vital capacity (VC) and total lung capacity (TLC) remain unaltered. Due to the progressive decrease of the residual volume in the face of an unchanging total lung capacity, the ratio of the residual volume to total lung capacity diminishes during the last trimester.7

In addition, minute ventilation (Ve) usually increases by 50% at term. The relatively greater increase in tidal volume (40%) than respiratory rate (15%) decreases the dead space component of ventilation, so that alveolar ventilation reaches a level 70% above that in the nonpregnant state. During labor and delivery, pain acts as a potent ventilatory stimulus. Oxygen consumption increases by 10-20% during pregnancy and is further elevated as much as 100% over normal during labor. This occurs in response to demand by the growing fetus, placenta and uterus and to increased cardiac and respiratory work, particularly during labor.8

Hyperventilation during pregnancy, due mainly to an increase in tidal volume, has been ascribed to the action of progesterone; results of one study of maternal PACO2 found that levels decline to a mean of 30-32 mmHg in the last 12 weeks of pregnancy.

In a study of PACO2 conducted by Reed, it was found that in early labor, with infrequent, slight uterine activity, little change in ventilation or PACO2 occurred. As contractions became stronger and more frequent, hyperventilation tended to occur usually with a slight fall in PACO2. When contractions became more frequent or hyperventilation nearly constant, the PACO2 fell even between contractions; the lowest levels being noted at the end of the first stage; during the second stage with the associated breath holding and less hyperventilation, resulted in some recovery of PACO2.8

Anesthetic considerations: Increased alveolar ventilation augments the transfer of gases between mother and fetus. Because of these changes, the parturient is much more susceptible to rapid changes in respiratory blood gas levels during respiratory complications than the nonpregnant woman. Consequently, hypoxia, hypercarbia and respiratory acidosis as a result of hypoventilation, breath-holding or respiratory obstruction develop more readily in the parturient than in the nonpregnant woman.4

With these physiologic changes in mind, one can see that during endotracheal intubation, even by a skilled anesthetist, the oxygen tension may fall precipitously after only a brief period of apnea. Conversely, moderate to severe hyperventilation can quickly lead to severe respiratory alkalosis with arterial carbon dioxide tensions as low as 10-15 mmHg and pH as high as 7.7. The use of excessive positive pressure ventilation in the anesthetized patient rapidly leads to respiratory alkalosis, associated with decreases in cerebral and uterine blood flow, and may have deleterious effects on the fetus.4

It is important to remember that inhalational anesthesia is rapidly induced in the parturient because hyperventilation, particularly during labor, delivers more anesthetic to the alveoli. At the same time, a smaller than usual functional residual capacity results in less dilution of incoming gases, allowing higher alveolar concentrations to be reached more quickly. In sheep and possibly in humans, minimal alveolar concentration (MAC) is decreased in pregnancy (halothane by 25%, methoxyflurane by 32% and isoflurane by 40%) perhaps because of progesterone's sedative effect. Adverse effects of hyperventilation may result from vasoconstriction of cerebral, umbilical, and possibly uterine vessels. Although progressive fetal acidosis does occur during iatrogenic hyperventilation during anesthesia, it is doubtful whether this is solely due to hypocapnic uterine vasoconstriction. Decreased venous return associated with intermittent positive pressure ventilation is more likely to be responsible for impaired uterine perfusion. Also respiratory alkalosis shifts the material hemoglobin dissociation curve to the left, thereby rendering oxygen unloading of fetal hemoglobin more difficult. Analgesic techniques (such as a continuous lumbar epidural) which minimize hyperventilation in labor should prove beneficial to the fetus.2

Gastrointestinal system

Prolonged gastric emptying is noted from at least the 34th week of pregnancy and is most likely hormonal in origin (progesterone). It is well known that anxiety and excitement have a severe depressant effect on gastric motility and that this effect is mediated by the splanchnic nerves. Pain and emotional disturbances which accompany labor also depress gastric motility and delay gastric emptying.9

Heartburn, a symptom of gastroesophageal reflux, is a common and frequently distressing com-
plication of pregnancy. Nagler and Spiro, performed manometric studies on pregnant women and found that in 55% of patients with heartburn, and in 20% without heartburn, the resting inferior esophageal sphincter pressure decreased with advancing pregnancy. During pregnancy there is an increase in intragastric pressure. In pregnant women without heartburn, the gastroesophageal sphincter responded to this increase in pressure by an increase in maximum sphincteric pressure. In the group of pregnant women with heartburn, there was no significant increase in sphincter pressure, and the stomach-to-sphincter pressure gradient was decreased. Under these circumstances, the sphincter does not maintain competence of the gastroesophageal junction and the symptoms of reflux occur.10

Anesthetic considerations: One must remember the above mentioned items when anesthetizing the pregnant patient. With regard to general anesthesia, pregnant women from 32 weeks gestation onward must be considered as having “full stomachs” and when general anesthesia is employed, have a rapid intravenous induction followed by cricoid pressure and tracheal intubation. The patient should be extubated awake when reflexes are intact.

Whenever feasible and appropriate, the use of regional anesthesia should be employed. The use of prophylactic antacids at the present time is a controversial issue due to the untoward effects of aspiration of antacid materials themselves.

Plasma cholinesterase

It has been shown that plasma cholinesterase activity declines during pregnancy and in the immediate postpartum period. Plasma cholinesterase activity in the 2nd and 4th postpartum days are even lower than at time of delivery.

Anesthetic considerations: Since plasma cholinesterase is responsible for the hydrolysis of succinylcholine, a low degree of enzymatic activity may result in an undesirably long duration of paralysis following succinylcholine administration.11 It is strongly recommended that whenever succinylcholine is being administered to a parturient, that the neuromuscular status be monitored by a peripheral nerve stimulator.

Conclusion

A number of significant maternal physiologic changes occur during pregnancy with regard to the respiratory, cardiovascular and gastrointestinal systems. The etiology of these changes is thought due to hormonal changes which are placental in origin, mechanical growth of the gravid uterus, or a combination of both. It is imperative that the anesthesiologist obtain an in-depth understanding of the physiologic changes in the parturient and how they relate to anesthetic management; if not, an error may be made which may prove fatal for the mother, newborn or possibly both.

REFERENCES


AUTHOR

Thomas Ratigan, CRNA, BS, is a graduate of the Ellis Hospital School of Nursing, Schenectady, New York. Mr. Ratigan received his Bachelor of Science Degree from George Washington University, Washington, D.C. and his anesthesia education through the United States Navy Program for Nurse Anesthetists. At the time this article was written, Mr. Ratigan was employed by the University of Texas Health Science Center at Houston as an instructor in the Program in Nurse Anesthesia Education. Currently, Mr. Ratigan is a staff nurse anesthetist at the Veteran's Administration Medical Center in Houston, Texas.

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