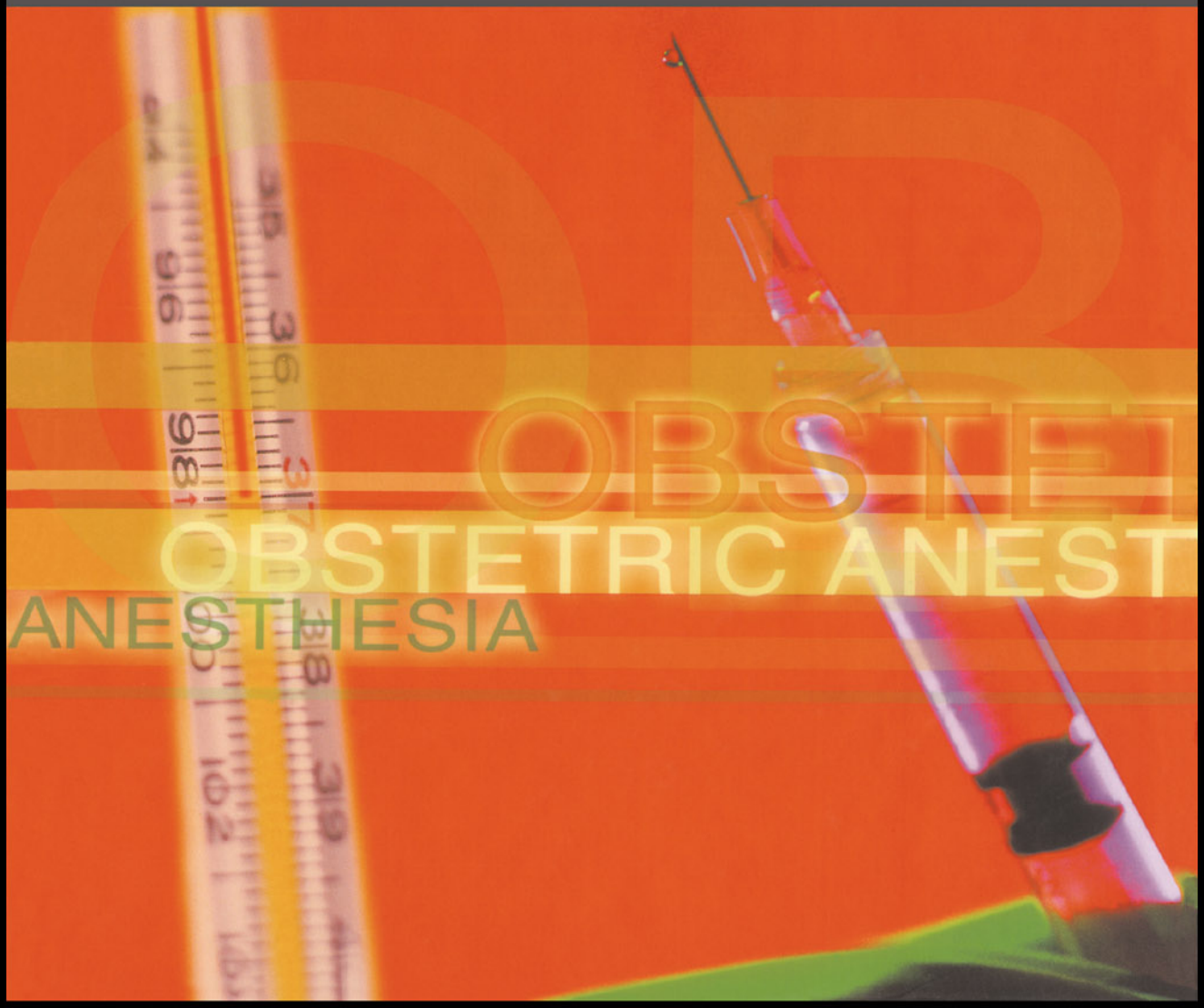
handbook of
OBSTETRIC ANESTHESIA

C. M. PALMER R. D'ANGELO M. J. PAECH



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Contents

Abbreviations

Ach	acetylcholine
AMPA	-amino-3-hydroxy-5-4-isoxazolepropionic acid
BP	blood pressure
bpm	beats per minute
CNS	central nervous system
CPD	cephalopelvic disproportion
CS	cesarean section
CSEA	combined spinal-epidural anesthesia
CSF	cerebrospinal fluid
DIC	disseminated intravascular coagulation
EA	epidural anesthesia
EBP	epidural blood patch
ECG	electrocardiogram
ECV	external cephalic version
EGA	estimated gestational age
FHR	fetal heart rate
FRC	functional residual capacity
GA	general anesthesia
HOCM	hypertrophic obstructive cardiomyopathy
ICP	intracranial pressure
IM	intramuscular
IUGR	intrauterine growth retardation
IV	intravenous
IVC	inferior vena cava
IVH	intraventricular hemorrhage
LA	local anesthetic
LBW	low birth weight
LOR	loss of resistance

MAC	minimum alveolar concentration
MLK	myosin light-chain kinase
NMDA	N-methyl-D-aspartate
PCEA	patient-controlled epidural anesthesia
PCIA	patient-controlled intravenous analgesia
PDPH	post-dural puncture headache
PIH	pregnancy-induced hypertension
p.p.m.	parts per million
PPV	positive-pressure ventilation
PTL	preterm labor
PVR	pulmonary vascular resistance
RDS	respiratory distress syndrome
SA	spinal anesthesia
SVC	superior vena cava
SVR	systemic vascular resistance
UPP	uterine perfusion pressure
WDR	wide dynamic range

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Preface

Obstetric anesthesia is a rapidly expanding and evolving field, and this is certainly not the first book to address the subspecialty. When the idea of a completely new text was first raised, our first questions were whether there was a need for a book, and how it would differ from previous textbooks.

We saw a need for a practical guide to patient care and management, one that could be consulted quickly (and often) to outline a concrete course of clinical management: how to manage specific patients in specific situations, what drug to give, how to give it, how much to give, and when to stop and try something different. Our goal in this book has been to provide anethesiologists and anesthetists, both in training and in practice, with that practical reference.

We have worked diligently to make the information easy to use—when possible, we have tried to distill essential information down into tables, charts, diagrams, and flowcharts which can be quickly accessed and applied. We have tried to limit the discussion to the essential background, rationale, and science behind clinical decision-making. This is not intended to be an exhaustive reference textbook, though the readings and references at the end of each chapter do provide additional background and the basis for further study.

Of course, as the saying goes, "…there is more than one way to skin a cat." Where our experience has shown one approach works best, we have advocated it; where there are equally viable options, we have tried to present each, with the advantages and disadvantages of alternate approaches.

All three of us are full-time clinical obstetric anethesiologists, as are most of our contributors. We care for these patients and deal with these problems everyday. The *Handbook of Obstetric Anesthesia* draws on our experience and study to tell you what we do, how we do it and why.

Craig M.Palmer, M.D.
Robert D'Angelo, M.D.
Michael J.Paech, FANZA

Chapter 1
Overview of obstetric anesthesia

Craig M.Palmer, MD

Contents

1.1
Introduction

The field of obstetric anesthesia has a long and proud tradition, very nearly as long as the specialty of anesthesiology itself. The way obstetric anesthesia is practiced today has been shaped by the social pressures of the last century. Much more than other subspecialties within anesthesiology, and more than most other fields of medicine in general, the practice of obstetric anesthesia has been shaped by the desires of the patients it serves—laboring women. The demands and desires of the parturient remain the strongest influence on the field today.

1.2
A brief history of obstetric anesthesia

The year 1997 marked the 150th anniversary of the birth of obstetric anesthesia. On 19 January 1847, James Young Simpson *(Figure 1.1)* administered diethyl ether to a young woman with a deformed pelvis to aid in the vaginal delivery of her stillborn infant. Simpson was Professor and Chair of Midwifery and Diseases of Women and Children at the University of Edinburgh in Scotland. It was only 5 years earlier that Crawford Long, a physician in Georgia, was the first to use ether for a surgical procedure, the painless removal of a neck tumor.

Simpson immediately became a great advocate of the use of ether to relieve the pain accompanying labor and delivery, but most of the established institutions of the day failed to exhibit his enthusiasm. The Church of Scotland condemned its use as contrary to God's will, citing the book of Genesis in the Bible: 'In sorrow thou shalt bring forth children.' Many of the most eminent physicians of the day also opposed its use. Charles Meigs, Professor of Obstetrics at Jefferson Medical College in Philadelphia, was representative of many of the opponents of ether, arguing that pain was a necessary and useful part of childbirth, and

Figure 1.1 James Young Simpson. Simpson was Professor and Chair of Midwifery and Diseases of Women and Children at the University of Edinburgh in Scotland. In January of 1847 he used ether to assist a patient during a difficult delivery. He is considered the 'Father of Obstetric Anesthesia'. Courtesy of The Wood Library-Museum of Anesthesiology, Park Ridge, IL

relieving pain might interfere with the normal progress of labor. Some of this opposition was well founded—little or nothing was known about ether's effects on the infant, mother, or progress of labor. As neither side had much in the way of objective evidence on their side, the debate continued for years. The controversy about anesthetic effects on labor continues to this day (see Section 5.8).

The first use of ether for childbirth in the USA also occurred in 1847. Fanny Longfellow, wife of the poet Henry Wadsworth Longfellow, had heard of the use of this new drug for analgesia during labor, and enlisted the aid of a dentist, Nathan Cooley Keep, to administer it when she could not find a physician willing to do so. Also in 1847, Walter Channing, Dean of the Harvard Medical School, first used ether for a forceps delivery.

The popularity of anesthesia for labor and delivery was greatly accelerated in 1853, when John Snow *(Figure 1.2)* administered chloroform to Queen Victoria for the birth of her eighth child, Prince Leopold. This event quickly gave the use of inhaled anesthetics during labor a measure of validity in the public eye. Snow was also among the first to begin to look at the use of anesthesia during childbirth in a careful, rational way. That same year, 1853, he observed that infants born to mothers who had received chloroform during delivery were less vigorous at birth than those born without anesthesia; he concluded that the chloroform must also affect the infant, though to a lesser extent than the mother.

Use of inhaled anesthesia for labor continued to gain momentum through the latter half of the 19th century. Ether was usually the anesthetic of choice. While gaining in popularity, inhaled anesthesia for labor still presented problems—the risk of aspiration became apparent, and the potential for asphyxia of both mother and infant in untrained hands. The vast majority of births during this period occurred at home, attended by lay midwives; since only physicians could use anesthetics, they were actually available to very

Figure 1.2 John Snow. In 1853, he administered chloroform to Queen Victoria of England for the birth of her eighth child, Prince Leopold. The event catalyzed the use of inhaled anesthetics for obstetric anesthesia. Courtesy of The Wood Library-Museum of Anesthesiology, Park Ridge, IL

few women. Further, as labor was often prolonged for several hours, even in those cases where anesthesia was used, its actual administration was often left to the husband or some other untrained bystander who happened to be available.

By the turn of the century, a new approach was being developed. Morphine had first been isolated in the early 1800s, but was not used for labor analgesia until the 1900s. By 1907, Richard von Steinbuchel, an Austrian physician, described a method for labor analgesia that had been used by surgeons for several years: *Dämmerschlef,* or 'Twilight Sleep'. Twilight sleep entailed subcutaneous injections of morphine and scopolamine; used alone, neither provided a satisfactory result and could even be dangerous, but two German obstetricians, Carl Gauss and Bernhardt Krönig, believed that by combining the two in the correct amounts, the benefits of each could be gained without the drawbacks. The parturient was given an initial injection of both drugs at the beginning of labor, then intermittent injections of scopolamine throughout the duration of labor. The initial morphine given, about 10 mg, provided a modest degree of analgesia, while the scopolamine was an amnestic; pain may not have been eliminated, but it was rarely remembered. In addition to amnesia, however, scopolamine disinhibited many patients, causing them to become disoriented and thrash about; because of this, parturients required constant attendance.

Despite these drawbacks, twilight sleep gained a popular following. A popular US magazine of the pre-war period, *McClure's,* sent two journalists, Mary Boyd and Marguerite Tracy to the Freiburg, Germany clinic of Gauss and Krönig to report on the technique firsthand. In 1914, *McClure's* published an article detailing Boyd's own experience giving birth at the clinic; the article presented the technique in a very

favorable light, characterizing twilight sleep as a safe and effective method of pain relief during labor. Upon their return, the two women were instrumental in the founding of the National Twilight Sleep Association. The Association was a 'grassroots' lay organization dedicated to expanding the use of this form of analgesia in the US. It was formed as a reaction to the lack of enthusiasm the medical establishment of the day showed for the technique. The association was part of a larger social feminist movement of the time, aimed at improving the health and welfare of women and children in a wide array of areas.

Several years later, in 1927, a similar organization was founded in Great Britain, the National Birthday Trust Fund. The Fund was initiated by two upper class women, Ladies Rhys-Williams and Cholmondelay, with the aim of extending to all classes of the British public the same quality of maternal and obstetric care enjoyed at the time only by the aristocracy and royalty. While providing maternal labor analgesia was a major part of the drive, the Fund had broader goals than the Twilight Sleep Association; it aimed to improve all aspects of obstetric care, not just obstetric anesthesia. Rather than just championing a single method, they encouraged the involved professionals—obstetricians, midwives, and anesthetists—to actively innovate and develop better methods for health care delivery. The Fund provided monies to research new techniques and agents for the provision of labor analgesia. Over the succeeding 20 years, the Fund was able to dramatically improve the delivery of health care to British women and children.

Together, both these movements had an important secondary effect—moving labor and delivery from the home to hospitals and nursing homes. Because of the maternal supervision which twilight sleep entailed, it was ill suited to being administered in parturients' homes, even if individuals who understood the technique would have been available. Likewise, many of the inhalation techniques that were developed with the help of the National Birthday Trust Fund required apparatus that was not easily moved to individual homes. Movement of deliveries to nursing homes and hospitals also had the benefit of providing cleaner surroundings, and increased the use of aseptic techniques during the delivery process. From the anesthetic perspective, this movement from home to hospital had a dramatic effect—the flow of patients through these facilities allowed physicians to specialize in the provision of labor analgesia, and develop the regional anesthetic techniques that have become the mainstay of labor analgesic practice today.

Despite the ever-increasing enthusiasm of the lay public for labor analgesia, not all professionals were convinced of its merits. Grantly Dick-Read published his first book, *Natural Childbirth,* in 1933, though he had written his first draft over 10 years earlier while still in training to be an obstetrician *(Figure 1.3).* Based partly on his observations of childbirth in 'primitive' societies, Dick-Read believed that it was society that was responsible for labor pain, by propagating misconceptions, superstitions, and misinformation about childbirth which caused women to fear childbirth. Fear produces tension, which in turn produces pain; eliminate the fear, and the pain of labor will be eliminated also. The key to the elimination of fear, according to Dick-Read, is education about the delivery process, and training in breathing exercises to attain a state of relaxation.

A second vocal opponent of the use of medication to relieve labor pain was Fernand Lamaze of France *(Figure 1.4).* Lamaze ran a clinic in Paris, and built upon the work of the Russian physiologist Velvoski who used 'psychoprophylaxis' for pain relief during labor. Lamaze and Velvoski believed that positive conditioned reflexes could be used to eliminate labor pain, and pregnant women could learn to use this method with training in breathing exercises and relaxation techniques. Such psychoprophylactic techniques are the direct application of Pavlovian conditioning to labor. Lamaze wrote a book, *Painless Childbirth,* that was widely read and has become the basis of most 'natural childbirth' classes today.

Regional anesthesia for labor and delivery was slowly advancing during this period. In 1900, Oskar Kreis first described the use of spinal anesthesia in parturients. He injected 10 mg of cocaine into the lumbar spine of six parturients, and observed pain relief that lasted about 2 hours. Despite such early successes, regional

Figure 1.3 Grantly Dick-Read. In 1933 he published his first book, *Natural Childbirth.* Dick-Read, a British obstetrician, did not believe labor and delivery were inherently painful; he believed society was responsible for labor pain, causing women to fear childbirth. This fear produced tension, which in turn produced pain. Courtesy of The Wellcome Trust, London, UK.

anesthesia took many years to gain momentum. Part of the reason for this slow development was the relative paucity of equipment and medication, but part was also due to the fact that obstetricians, who were providing the vast majority of anesthesia during labor, reserved spinal anesthesia for delivery.

In the early 1930s, a Romanian anesthesiologist, Eugene Aburel, described the sensory innervation of the uterus and perineum. He also pioneered early sacral epidural techniques and caudal analgesia for the second stage of labor. Unfortunately, due to the fact that he published his findings in the German and French literature, the disruption of scientific literature that surrounded World War II prevented his work from appearing in the West for almost 40 years.

About the same time, in 1933, John Cleland published a report on the use of paravertebral block for labor analgesia and also outlined the relevant nerve pathways associated with sensation and pain during labor. In 1942, Robert Hingson and Waldo Edwards published a report detailing the use of caudal anesthesia for labor and delivery, in which they used a small malleable needle that remained taped in place during labor. Later, the development of flexible catheters that could be threaded into the epidural space sparked a shift to lumbar epidural anesthesia that remains the dominant method of regional anesthesia for labor today.

It took more than just the development of regional techniques to supplant the use of twilight sleep and inhalational anesthesia for labor, however. Though pain was reduced with these systemic techniques, the amnesia induced often left the mother feeling disconnected from the birth experience, and somehow 'unfulfilled'. By the 1960s, the desire to be actively involved in the birth process was overtaking the desire for analgesia in the minds of parturients, and enhanced the popularity of Dick-Read's and Lamaze's 'natural childbirth' methods. The rapid development in the last two decades of regional anesthetic techniques that

Figure 1.4 Fernand Lamaze. Lamaze was a French obstetrician who popularized the use of 'psychoprophylaxis' for pain relief during labor. Lamaze believed that positive conditioned reflexes, based on breathing exercises and relaxation techniques, could be used to eliminate labor pain. Lamaze's book, *Painless Childbirth,* has become the basis of most 'natural childbirth' classes today. Courtesy of The Wood Library-Museum of Anesthesiology, Park Ridge, IL

lack significant motor block, and even allow ambulation, has eliminated most of the concerns associated with systemic analgesics, and (much to their surprise) has fulfilled the expectations of 'natural childbirth' advocates.

It is interesting to note that the goals of both the natural childbirth movement and the obstetric anesthesia community have been the same: an awake, fully-functional, and participating mother during delivery, without neonatal side effects or interference with the natural course of labor. Despite occasional tensions between the two camps, only the paths they have taken to this common goal differ.

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Chapter 2
Anatomic and physiologic changes of pregnancy

Laura S.Dean, MD and Robert D'Angelo, MD

Contents

2.1 Introduction

Profound physiologic and mechanical changes occur during pregnancy. Unique alterations allow for the development of a growing fetus and prepare the parturient for the demands of labor and delivery. Anesthesia providers must recognize the anesthetic implications of this altered physiology in order to care for patients throughout the puerperium as well as during non-obstetric surgery. Many of the physiologic

adaptations occur during the first trimester making recognition of pregnancy in women of childbearing age imperative.

2.2 Cardiovascular

2.2.1 Hemodynamics

Cardiovascular parameters are altered progressively throughout pregnancy and are accentuated in pregnancies subsequent to the first *(Table 2.1)*. Beginning as early as 4–8 weeks gestation and plateauing between 16 and 24 weeks, these alterations become further dependent on changes in the parturient's position as aortocaval compression worsens during the third trimester. In general, these alterations return to pre-pregnancy baseline during the first 6 months to 1 year postpartum.

Table 2.1 Cardiovascular adaptations during pregnancy

Parameter	Change
Cardiac output	α 20–40%
Stroke volume	α 30%
Heart rate*	α 16%
Systemic vascular resistance	α 30%
Mean arterial pressure**	α 8%
Plasma volume	α 11–50%
Oxygen consumption †	α 20%

*At 4 weeks gestation there is a 20 bpm increase in heart rate. **Mean arterial blood pressure will fall as a result of a decrease in systemic vascular resistance despite an increase in cardiac output. † Cardiac oxygen consumption increases in conjunction with resting tachycardia.

Initially, systemic vascular resistance decreases as a result of increased circulating estrogen and progesterone. This decrease in systemic tone results in reduced afterload and preload triggering a reflex increase in heart rate. Volume restoring mechanisms including angiotensin and aldosterone are activated to elevate blood volume. Cardiac output elevation thus results from an increase in both stroke volume and heart rate. Increased left ventricular wall thickness and ventricular cavity accommodate the elevated cardiac output. Despite this remodeling, contractility is probably unchanged. It is crucial to recognize the normal changes in the cardiac exam that occur during pregnancy *(Table 2.2)*. While diastolic murmurs and S4 heart sounds are not uncommon, both should be further evaluated for underlying heart disease. The increased blood flow is redistributed to meet the demands of the growing fetus and altered maternal physiology *(Table 2.3)*.

Table 2.2 Cardiac exam findings during pregnancy*

Accentuated and split S_1

Normal S_2

Systolic ejection murmur (90%)

Diastolic flow murmur (20%)

S_3 heart sound (80%)
Occasional S_4

*Flow murmurs correlate with the increase in blood volume rather than the alteration in the cardiac output. A pericardial effusion (9%) may cause isoelectric T waves and ST changes on the electrocardiogram.

Table 2.3 Redistribution of cardiac output during pregnancy

Organ	Increase in blood flow
Uterus	500 ml/min
Skin	300–400 ml/min
Renal	400 ml/min
Breasts	200 ml/min
Overall increase during pregnancy	1.5–2 l/min

2.2.2 Blood volumes

A 40–50% increase in blood volume during pregnancy meets the metabolic demands of the enlarging uterus and growing fetus. This relative hypervolemia is crucial for protecting the mother and fetus from hemorrhage at delivery and from the deleterious effects of decreased venous return in the supine position. The mechanisms accounting for the increase in blood volume are multifactorial. Progesterone relaxes venous smooth muscle thus increasing venous capacitance. The increase in blood volume may be a response to fill this increased vascular capacity. Estrogen and progesterone are also thought to directly mediate an increase in hepatic renin production which triggers enhanced secretion of aldosterone. The resultant retention of sodium allows for the increase in total body water. As early as the first trimester, increased renin and angiotensin diminish the pressor response to angiotensin during pregnancy. In contrast, response to norepinephrine infusions is unchanged. Enhanced renal erythropoietin production simultaneously increases red blood cell volume. However, the plasma volume increase is proportionately greater than the red cell volume increase resulting in a hemodilutional anemia *(Table 2.4)*.

2.2.3 Aortocaval compression

A symptomatic reduction in cardiac output in the supine position occurs in 5–10% of parturients and is referred to as the supine hypotensive syndrome. Manifestations include dizziness, nausea, maternal hypotension, shortness of breath, tachycardia and possibly fetal distress. Compression of the inferior vena cava by the

Table 2.4 Physiologic anemia of pregnancy

Parameter	Level during pregnancy
Blood volume	α 45%
Plasma volume	α 55%
Red cell volume	α 30%
Hemoglobin (average)	11.6 mg/dl

Parameter	Level during pregnancy
Hematocrit (average)	33.5 mg/dl

enlarged uterus reduces venous return and can result in profound hypotension. Abdominal aortic compression further compromises uterine blood flow. Neuraxial or general anesthesia may exaggerate these hemodynamic effects. It is important to recognize that the standard 15 degree lateral tilt that is vital for the obstetric patient may not be adequate to relieve aortocaval compression in all parturients. The lateral decubitus position or even the knee chest position may be necessary to alleviate maternal hypotension or fetal heart rate changes. Although supine hypotension is classically described beyond 20 weeks gestation, partial or complete compression can occur before this time. Anesthesia providers should consider left uterine displacement in pregnant women beyond 12–16 weeks gestation.

If aortocaval compression is suspected in a hypotensive patient, the compressed inferior vena cava may act like two pieces of wet glass that resist separation. Alleviating the compression and restoring adequate venous return are paramount. Treatment consists of ensuring left uterine displacement, ephedrine administration, and elevation of the legs, rather than placing the patient in the Trendelenburg's position.

2.3 Pulmonary

As summarized in *Table 2.5,* there are numerous mechanical and physiologic pulmonary adaptations that occur during pregnancy.

2.3.1 Mechanical effects

Venous engorgement and edema of the upper airway involves the pharynx, glottis, trachea and vocal cords making visualization during endotracheal intubation more challenging than in the nonpregnant patient. Enlarged breasts and redundant soft tissue in the neck and chest may inhibit placement of the laryngoscope. Chest circumference increases 5–7 cm as a result of both increased anterior posterior and transverse diameters. The anesthetic implications of these anatomic changes are shown in *Table 2.6.*

Table 2.5 Pulmonary alterations during pregnancy

Parameter	Change during pregnancy
Respiratory rate	α 9%*
Tidal volume	α 19–28%
Expiratory reserve volume	α 17%
Residual volume	No change-α 20%
Functional residual capacity	α 12–25%**
Vital capacity	No change-α 6%***
Total lung capacity	No change-α 5%
O2 consumption	α 20–40%†
Minute ventilation	α 20–50%
FEV_1	No change‡

Parameter	Change during pregnancy
Basal metabolic rate	α 14%

*Early in the first trimester, hyperventilation is stimulated by a progesterone-mediated hypersensitivity of respiratory centers to CO_2.** The decrease in FRC may result in airway closure during tidal breathing in as many as 50% of term parturients in the supine position. *** Despite elevation of the diaphragm by the enlarging uterus there is no change in vital capacity because of the simultaneous increase in chest wall diameter. † The increase in cardiac output is greater than the increase in oxygen consumption so the AVO_2 difference in early pregnancy is decreased. As cardiac output plateaus and the metabolism of the fetus and growing uterus further increases 0_2 consumption the AVO_2 difference approaches pre-pregnancy levels. ‡ There are no documented changes in airway flow or diffusion capacity.

2.3.2
Acid-base physiology in pregnancy

Hyperventilation induces a slight respiratory alkalosis. Renal compensation decreases plasma HCO_3 allowing for a near normal pH. Although respiratory alkalosis shifts the oxyhemoglobin dissociation curve to the left, 2,3-DPG production rises 30% above nonpregnant levels and shifts the curve back to the right. P_{50} is increased from 26.7 to 30.4 aiding delivery of oxygen to the fetus. The increase in minute ventilation, cardiac output and blood volume along with a fall in alveolar dead space contribute to a negligible arterial to end tidal CO_2 difference at term. Arterial blood gas values seen in nonpregnant and pregnant patients are listed in *Table 2.7*.

2.4
Gastrointestinal

Controversy exists over the effects of pregnancy on the rate of gastric emptying. Some investigators have suggested that motility decreases early in pregnancy, some not until the third trimester and others contend there is never any alteration in gastric emptying. This issue is important clinically during pregnancy but also in the days post delivery. *Table 2.8* reviews studies that have examined the changes in gastric physiology. The placenta and fetus produce gastrin which increases the volume and acidity of gastric contents. Progesterone slows gastric motility by inhibiting motilin and diminishes the lower esophageal sphincter tone by relaxing smooth muscle. The gravid uterus increases gastric pressure and elevates the lower esophagus into the thorax in many women. Consequently, 80% of term parturients experience pyrosis. Opioids administered during labor or for non-obstetric surgery will further impair gastric motility. All pregnant women should be considered to have full stomachs regardless of NPO status. Obstetrics provides a unique anesthetic setting during which the likelihood of difficult mask ventilation and failed intubation is higher than normal at a time when aspiration is also more likely to occur. Aspiration prophylaxis to reduce the chance of pneumonitis should be routinely followed. Recommendations are noted in *Table 2.9*.

2.5
Renal

The renal system undergoes anatomical and physiologic changes during pregnancy. Increased vascular and interstitial volumes result in slightly enlarged kidneys. The pelvis, calyces and ureters are dilated by the

effects of progesterone and probably by the mechanical effects of the gravid uterus. Renal plasma flow and glomerular filtration increase early in the first trimester. These changes precede the

Table 2.6 Anesthetic implications of pulmonary manifestations of pregnancy*

Anatomic/physiologic change	Anesthetic implication/recommendations
Redundant soft tissue: neck, chest, breasts	May obstruct laryngoscope placement: Have short handle available.
Mucosal venous engorgement/edema Increased Mallampati scores throughout labor	May result in impaired visualization and a friable bleeding airway with intubation. Avoid nasal instrumentation. Have smaller ETT available. Important to have experienced personnel available.
α FRC, αO_2 consumption	Prone to hypoxia during general anesthesia induction. Maximal preoxygenation is crucial. Rapid sequence induction with cricoid pressure indicated.
Normal $paCO_2$–32 mmHg There is little to no arterial/end-tidal CO_2 gradient.	Maintain normocarbia during general anesthesia. Avoid hyperventilation (α$paCO_2$ will cause uterine vasoconstriction and αplacental blood flow. Alkalosis will shift $HgbO_2$ curve to the left decreasing release of O_2 to the fetus).

*The leading causes of anesthesia related morbidity and mortality in pregnancy are failure to intubate or ventilate and aspiration.

Table 2.7 Arterial blood gas results in normal pregnancy

Parameter	Pregnant	Nonpregnant
$PaCO_2$	30–32 mmHg	40 mmHg
PaO_2	92–106 mmHg	100 mmHg
Supine paO_2	101–94 mmHg	100 mmHg
HCO_2	16–21 meq/l	24 meq/l
PH	7.405–7.44	7.40

increase in plasma volume suggesting hormonal mechanisms. BUN and creatinine are lowered as GFR and renal plasma flow increase. *(Table 2.10)* Even mild elevations in plasma levels should be investigated for evidence of renal disease.

Glucosuria, independent of blood sugar concentration, is noted soon after conception. The renal tubules are likely unable to accept the

Table 2.8 Summary of studies investigating gastric emptying during pregnancy

Study	Findings
1. Davison JS *et al.*	Positive delayed emptying
2. O'Sullivan GM *et al.*	No difference in rate of emptying during pregnancy
3. Simpson KH *et al.*	Positive delay at 12–14 weeks gestation compared to controls
4. Macfie AG *et al.*	No delay during pregnancy compared to controls

Study	Findings
5. Sandhar BK *et al*.	No statistically significant change in gastric emptying as a result of pregnancy.
6. Carp H *et al.*	Emptying of the stomach is delayed for many hours after the onset of labor.
7. Whitehead EM *et al*.	Significant delay in gastric emptying 2 hours post partum, but no differences in emptying times during three trimesters of pregnancy when compared to 18 hours after delivery.
8. Levy DM *et al*.	Positive delay at 8–12 wks
9. Gin T	Rapid emptying post-partum

1. *J. Obst. Gyn. Br. Com.* 1970; **77:**37. 2. *Anaesth. Analg.* 1987; **66:**505. 3. *Br.J. Anaesth.* 1988; **60:**24. 4. *Br.J.Anaesth.* 1991; **67:**54. 5. *Anaesthesia* 1992; **47:**196. 6. *Anesth. Analg.* 1992; **74:**683. *7. Anaesthesia* 1993; **48:**53. 8. *Br.J.Anaesth.* 1994; **73:** 237.9. *Anaesthesia.* 1993; **48:**821.

Table 2.9 Recommended aspiration precautions

- Nonparticulate antacid—30 ml, 30–45 minutes before surgery
- Metoclopramide—Consider in high risk patients 30–90 minutes before surgery
- Consider all pregnant patients to have full stomachs
- Use rapid sequence induction with cricoid pressure
- Avoid positive pressure mask ventilation ifpossible
- Extubate patients when fully awake

Table 2.10 Renal hemodynamics

Parameter	First trimester	Third trimester
RPF (renal plasma flow)	α 75–85%	α 50%
GFR (glomerular filtration rate)	α 50%	α 50%
Filtration fraction—RPF/GFR	α	α
Creatinine	WNL	α 0.4–0.6 mg/dl
BUN	8–9 mg/dl	α 6–8 mg/dl

increased filtered load of glucose accompanying the increase in GFR. Uric acid tubular reabsorption declines so that plasma uric acid concentration increases in the third trimester. Plasma osmolality decreases early in pregnancy as water is retained in excess of sodium. The osmotic threshold for thirst declines further increasing fluid intake to contribute to the decline in osmolality.

2.6 Hepatic

The diagnosis of liver disease in pregnancy is confounded by the normal changes that occur in liver function studies during gestation *(Table 2.11)*. Plasma cholinesterase levels decline 20–30% by term pregnancy.

However, the simultaneous increase in volume of distribution likely counters any clinically significant prolongation of neuromuscular blockade from succinylcholine.

Table 2.11 Diagnostic signs of liver disease vs. normal pregnancy

Parameter	Liver disease	Pregnancy
Clinical signs	Spider angiomata/palmar erythema	Spider angiomata/palmar erythema
Protein synthesis	Normal 444	α 20–30%
Alkaline phosphatase	Normal to ααα secondary to obstructive disease	α 200–400% by placental production
GGT, LDH, AST, ALT	Normal to ααα	High normal to α

2.7 Gallbladder

Decreased cholecystokinin release and a reduced contractile response of the gallbladder to cholecystokinin is mediated by increased progesterone levels. This combined effect yields a sluggish milieu with propensity for gallstone formation.

2.8 Glucose metabolism

Although insulin secretion rises during gestation, pregnancy is considered to be a 'diabetogenic' state. There is a relative insulin resistance mediated by decreased peripheral sensitivity to insulin evoked by circulating placental hormones (particularly human placental lactogen (HPL)). Carbohydrate loads result in higher plasma glucose levels than in nonpregnant patients allowing for placental transfer of glucose to the fetus. These alterations in glucose metabolism likely contribute to the propensity for gestational diabetes. Shortly after delivery of the fetus and placenta, insulin sensitivity returns to baseline. Insulin should be administered cautiously prior to delivery because of the possibility of hypoglycemia immediately postpartum. Insulin does not cross the placenta so the fetus is responsible for secreting its own insulin in response to glucose loads. The fetus of a hyperglycemic mother may become profoundly hypoglycemic after delivery when it no longer receives a glucose load but still has elevated circulating levels of insulin.

2.9 Thyroid

Although there is an elevation of total serum thyroxin during gestation and the thyroid gland is noted to be enlarged, a euthyroid clinical state is maintained throughout pregnancy.

2.10 Hematologic

The hemodilutional anemia of pregnancy was discussed earlier in this chapter. Platelets remain unchanged or fall slightly with no apparent clinical effect. Clotting mechanisms are activated with an elevated serum concentration of fibrinogen and all clotting factors except XI and XIII. Although this hypercoagulable state may be a protective mechanism to allow hemostasis after delivery, embolic complications remain a leading

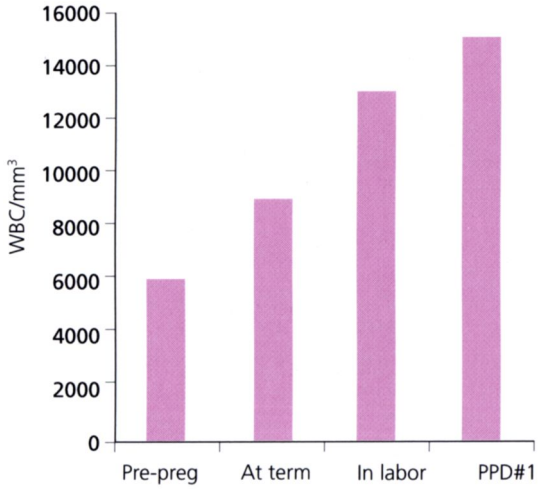

Figure 2.1 WBC count during pregnancy. WBC count increases slightly during pregnancy, acutely increases during labor, and remains elevated for at least the first week postpartum.

cause of morbidity and mortality during pregnancy. Leukocytosis peaks postpartum complicating the clinical diagnosis of infection and response to antibiotics *(Figure 2.1)*.

2.11 Neurologic

Pregnant patients require approximately one third less local anesthetic for regional anesthesia. Theoretical explanations for this increased sensitivity to local anesthetics are listed in *Table 2.12*. Although there may be some contribution of mechanical effects, most experts favor the elevated progesterone theory. Pregnant women have elevated CSF progesterone levels that are speculated to alter neuronal structure and allow for increased local anesthetic effect.

2.12 Uterus

The uterus undergoes massive change during pregnancy. An organ that is 5 cm×6 cm

Table 2.12 Theories for increased local anesthetic sensitivity during pregnancy

1. A decreased epidural space secondary to epidural venous engorgement
2. Increased abdominal pressure enhancing transdural spread of local anesthetics
3. Exaggerated lumbar lordosis allowing increased cephalad spread of local anesthetic.
4. Progesterone enhanced sensitivity of nerves to local anesthetics.

increases in size to 25 cm×30 cm at term. Uterine blood flow in nonpregnant females increases from 50 ml/min to 500–800 ml/min. The uterine vessels are maximally vasodilated. The fraction of cardiac output to the uterus increases from 3–4% up to 12% at term. Perfusion is not autoregulated so that a decrease in systemic

blood pressure will result in impaired uteroplacental blood flow. Uterine contrac-tions result in decreased placental perfusion and can lead to fetal compromise. Indirect acting agents, such as ephedrine, are preferred over peripheral α-1 agonists for maintaining arterial pressure and uterine blood flow.

2.13
Skeletal

Corpus luteal production of relaxin increases joint laxity. Exaggerated lumbar lordosis may contribute to the back and joint pain seen in some parturients.

2.14
Summary

The anatomic and physiologic changes that occur during pregnancy place the parturient at greater risk for complications during anesthesia than the nonpregnant patient. In particular, pregnant women are at an increased risk of complications during general anesthesia. A thorough understanding of these changes allows the anesthesia care provider to alter their anesthetic management to maximize benefits while minimizing risks.

Furthe reading

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Chapter 3
Fetal assessment

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Contents

3.1 Introduction

Obstetric anesthesia is unique among the anesthetic subspecialties in its responsibility to not one, but two patients: the parturient and the fetus. Not only must anesthetic interventions be in the best interests of the mother, but they must also, as far as possible, be in the best interests of the unborn infant. Often, the 'best' interests of these two patients are at odds with each other, and there is no single 'best' course of action. The appropriate anesthetic interventions will depend upon the clinical circumstances and the judgment and experience of the individual anesthesiologist.

Obstetric anesthesiologists need to be familiar with available means of fetal assessment for two reasons. First, all maternal anesthetic interventions have at least the potential to impact fetal well being. Second, in many (most?) emergent situations, anesthetic interventions are undertaken on the mother because of the fetus: understanding the role and limitations of fetal assessment is the knowledge that allows the anesthesiologist to exercise appropriate judgment.