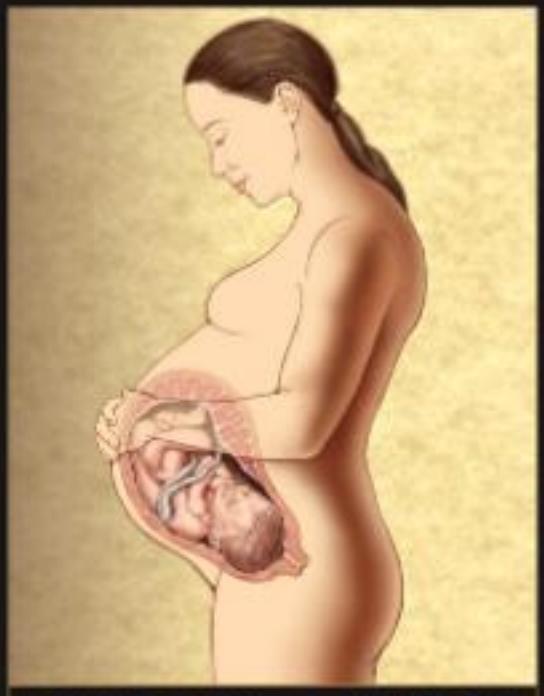
Chestnut's OBSTETRIC ANESTHESIA

PRINCIPLES AND PRACTICE



FOURTH EDITION

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Dedication

To my wife, **Janet**, and our children, **Stephen**, **Annie**, **Mary Beth**, **Michael**, and **John Mark** and **Catherine**

DHC

To my sons, **Tad** and **John**

LSP

To my wife, **Paulita**, our children, **London** and **Hamilton**, and my parents-in-law, **Deirdre** and **Oscar**

LCT

To my husband, **Lawrence**, and our children, **Anna**, **Molly**, **Leah**, and **Sofie**

CAW

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Preface

Three decades ago, I could not decide whether I wanted to pursue a career in anesthesiology or obstetrics. So ... I decided to do both. I obtained training in both specialties largely because of my love of perinatal medicine. This text is the result of my desire to prepare a comprehensive resource for all anesthesia providers (and obstetricians) who provide care for pregnant women.

In the preface to the first edition, I identified two goals: (1) to collate the most important information that anesthesia providers should know about obstetrics, and (2) to prepare a thorough and user-friendly review of anesthesia care for obstetric patients. I asked each contributor to prepare a comprehensive, scholarly discussion of the subject and also to provide clear, practical recommendations for clinical practice. As we unveil the fourth edition, those goals remain intact. Further, the fourth edition has undergone a more extensive revision than either the second or the third edition.

First, consider the **cover**. The maternal-fetal image draws attention to the fact that the anesthesia provider and the obstetrician provide simultaneous care for two (or more) patients – both the mother and her unborn child. As I look at that image, I am awestruck by the miraculous beauty of conception, pregnancy, and childbirth.

Second, consider the **content**. At least 10 chapters – including the chapters on neuraxial labor analgesia, cesarean delivery, the difficult airway, and hypertensive disorders – have been rewritten from start to finish. The three previously published chapters on neuraxial labor analgesia have been consolidated into a single chapter, as have the two previously published chapters on anesthesia for cesarean delivery. All chapters have been updated, and most chapters have undergone extensive revision. Two new chapters address the important subjects of patient safety and maternal mortality. And the fourth edition – for the first time – features a dedicated website that includes the whole content of the book and is fully searchable. The book's interior has been enhanced by the addition of a second color, which allows a better and more attractive visual presentation.

Third, consider the **contributors**. The fourth edition includes an astounding number of 32 new contributors! Of special significance is the addition of three outstanding new editors. I hand-picked these new editors as a result of my personal interaction with them in the preparation of previous editions of this textbook, as well as my familiarity with – and respect for – the depth and breadth of their knowledge and judgment, and the quality of their scholarship. The title page lists their academic affiliations and hospital leadership positions. In addition, **Linda S. Polley, M.D.,** is the current President of the Society for Obstetric Anesthesiology and Perinatology (SOAP). **Lawrence C. Tsen, M.D.,** is the President-elect of SOAP, and he also serves as Editor-in-Chief of *International Journal of Obstetric Anesthesia*. And **Cynthia A. Wong, M.D.,** is the obstetric anesthesia section editor for *Anesthesia and Analgesia*. The addition of these extraordinary obstetric anesthesiologists to the editorial team has resulted not only in the preparation of a better product, but it also reflects the publisher's (and my own) long-term commitment to this text.

My fellow editors and I should like to acknowledge the important roles of four groups of special people. First, we should like to thank the 72 other talented and distinguished contributors to the fourth edition, as well as the contributors to previous editions of this text. Without their commitment to this project, the fourth edition would not have seen the light of day. Second, we should like to gratefully acknowledge the invaluable contributions made by our competent, loyal, and at times long-suffering assistants: Jennifer Lee and Donna Stortz (DHC); Mary Lou Greenfield (LSP); Judy Johnson (LCT); and Allison Ernt and Sean Jones (CAW). Third, we should like to acknowledge the encouragement, expertise, and attention to detail provided by the professional production team at Elsevier Mosby. And finally, we should like to thank *you*, the readers, not only for your support of this text through the years, but also for your ongoing commitment to the provision of safe and compassionate care for pregnant women and their unborn children.

David H. Chestnut, M.D.

Micah 6:8

The History of Obstetric Anesthesia

Donald Caton, M.D.

For I heard a cry as of a woman in travail, anguish as of one bringing forth her first child, the cry of the daughter of Zion gasping for breath, stretching out her hands, "Woe is me!"

—Jeremiah 4:31

"The position of woman in any civilization is an index of the advancement of that civilization; the position of woman is gauged best by the care given her at the birth of her child." So wrote Haggard[1] in 1929. If his thesis is true, Western civilization made a giant leap on January 19, 1847, when James Young Simpson used diethyl ether to anesthetize a woman with a deformed pelvis for delivery. This first use of a modern anesthetic for childbirth occurred a scant 3 months after Morton's historic demonstration of the anesthetic properties of ether at the Massachusetts General Hospital in Boston. Strangely enough, Simpson's innovation evoked strong criticism from contemporary obstetricians, who questioned its safety, and from many segments of the lay public, who questioned its wisdom. The debate over these issues lasted more than 5 years and influenced the future of obstetric anesthesia.[2]

JAMES YOUNG SIMPSON

Few people were better equipped than Simpson to deal with controversy. Just 36 years old, Simpson already had 7 years' tenure as Professor of Midwifery at the University of Edinburgh, one of the most prestigious medical schools of its day (Figure 1-1). By that time, he had established a reputation as one of the foremost obstetricians in Great Britain, if not the world. On the day he first used ether for childbirth, he also received a letter of appointment as Queen's Physician in Scotland. Etherization for childbirth was only one of Simpson's contributions. He also designed obstetric forceps (which still bear his name), discovered the anesthetic properties of chloroform, made important innovations in hospital architecture, and wrote a textbook on the practice of witchcraft in Scotland that was used by several generations of anthropologists.[3]

An imposing man, Simpson had a large head, a massive mane of hair, and the pudgy body of an adolescent. Contemporaries described his voice as "commanding," with a wide range of volume and intonation. Clearly Simpson had "presence" and "charisma." These attributes were indispensable to someone in his profession, because in the mid-nineteenth century, the role of science in the development of medical theory and practice was minimal; rhetoric resolved more issues than facts. The medical climate in Edinburgh was particularly contentious and vituperative. In this milieu, Simpson had trained, competed for advancement and recognition, and succeeded. The rigor of this preparation served him well. Initially, virtually every prominent obstetrician, including Montgomery of Dublin, Ramsbotham of London, Dubois of Paris, and Meigs of Philadelphia, opposed etherization for childbirth. Simpson called on all of his professional and personal finesse to sway opinion in the ensuing controversy.

MEDICAL OBJECTIONS TO THE USE OF ETHER FOR CHILDBIRTH

Shortly after Simpson administered the first obstetric anesthetic, he wrote, "It will be necessary to ascertain anesthesia's precise effect, both upon the action of the uterus and on the assistant abdominal muscles; its influence, if any, upon the child; whether it has a tendency to hemorrhage or other complications."[4] With this statement he identified the issues that would most concern obstetricians who succeeded him and thus shaped the subsequent development of the specialty.

Simpson's most articulate, persistent, and persuasive critic was Charles D. Meigs, Professor of Midwifery at Jefferson Medical College in Philadelphia (Figure 1-2).

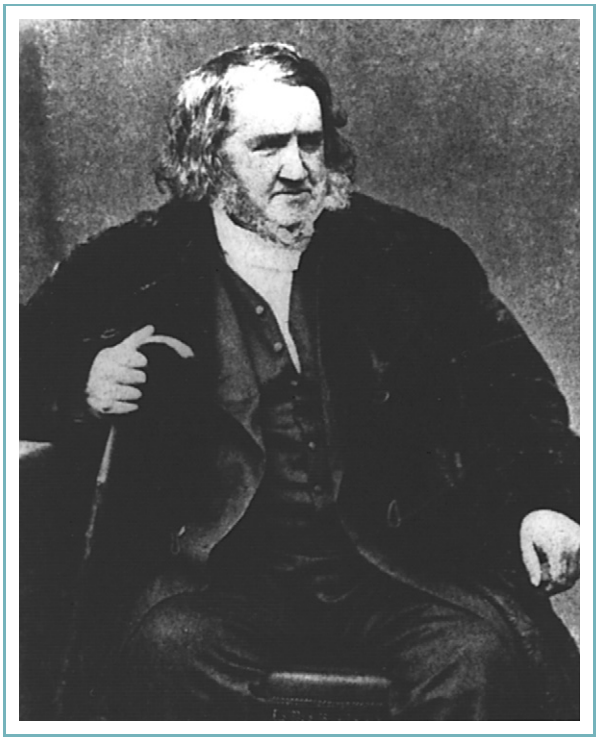


FIGURE 1-1 James Young Simpson, the obstetrician who first administered a modern anesthetic for childbirth. He also discovered the anesthetic properties of chloroform. Many believe that he was the most prominent and influential physician of his day. (Courtesy Yale Medical History Library.)

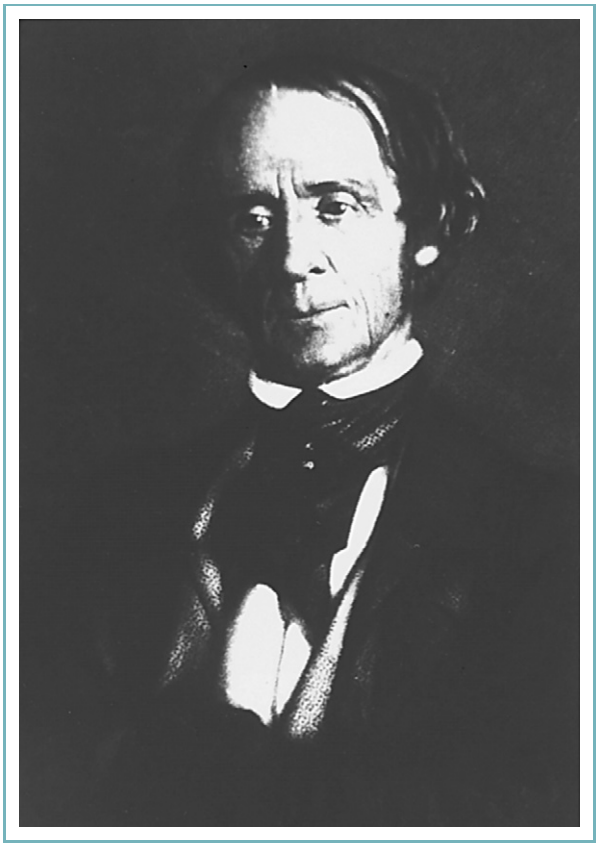


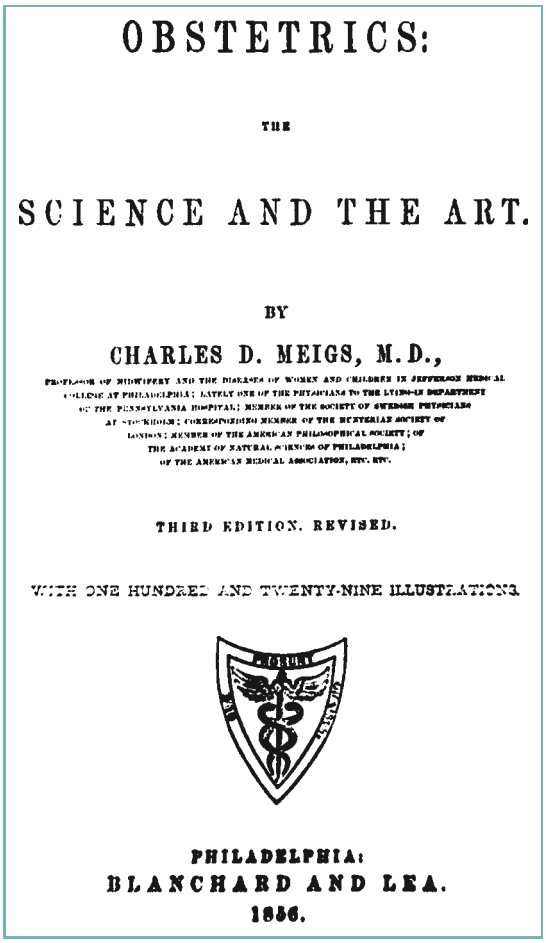
FIGURE 1-2 Charles D. Meigs, the American obstetrician who opposed the use of anesthesia for obstetrics. He questioned the safety of anesthesia and said that there was no demonstrated need for it during a normal delivery. (Courtesy Wood Library Museum.)

In character and stature, Meigs equaled Simpson. Born to a prominent New England family, Meigs' forebears included heroes of the American revolutionary war, the first governor of the state of Ohio, and the founder of the University of Georgia. His descendants included a prominent pediatrician, an obstetrician, and one son who served the Union Army as Quartermaster General during the Civil War.[5]

At the heart of the dispute between Meigs and Simpson was a difference in their interpretation of the nature of labor and the significance of labor pain. Simpson maintained that all pain, labor pain included, is without physiologic value. He said that pain only degrades and destroys those who experience it. In contrast, Meigs argued that labor pain has purpose, that uterine pain is inseparable from contractions, and that any drug that abolishes pain will alter contractions. Meigs also believed that pregnancy and labor are normal processes that usually end quite well. He said that physicians should therefore not intervene with powerful, potentially disruptive drugs (Figure 1-3). We must accept the statements of both men as expressions of natural philosophy, because neither had facts to buttress his position. Indeed, in 1847, physicians had little information of any sort about uterine function, pain, or the relationship between them. Studies of the anatomy and physiology of pain had just begun. It was only during the preceding 20 years that investigators had recognized that specific nerves and areas of the brain have different functions and that specialized peripheral receptors for painful stimuli exist.[2]

In 1850, more physicians expressed support for Meigs's views than for Simpson's. For example, Baron Paul Dubois[6] of the Faculty of Paris wondered whether ether, "after having exerted a stupefying action over the cerebro-spinal nerves, could not induce paralysis of the muscular element of the uterus?" Similarly, Ramsbotham[7] of London Hospital said that he believed the "treatment of rendering a patient in labor completely insensible through the agency of anesthetic remedies . . . is fraught with extreme danger." These physicians' fears gained credence from the report by a special committee of the Royal Medical and Chirurgical Society documenting 123 deaths that "could be positively assigned to the inhalation of chloroform."[8] Although none involved obstetric patients, safety was on the minds of obstetricians.

The reaction to the delivery of Queen Victoria's eighth child in 1853 illustrated the aversion of the medical community to obstetric anesthesia. According to private records, John Snow anesthetized the Queen for the delivery of Prince Leopold at the request of her personal physicians. Although no one made a formal announcement of this fact, rumors surfaced and provoked strong public criticism. Thomas Wakley, the irascible founding editor of *The Lancet*, was particularly incensed. He "could not imagine that anyone had incurred the awful responsibility of advising the administration of chloroform to her Majesty during a perfectly natural labour with a seventh child."[9] (It was her


OBSTETRICS:

THE

SCIENCE AND THE ART.

BY

CHARLES D. MEIGS, M.D.,

THIRD EDITION. REVISED.

WITH ONE HUNDRED AND TWENTY-NINE ILLUSTRATIONS.

PHILADELPHIA:
BLANCHARD AND LEA.
1856.

FIGURE 1-3 Frontispiece from Meigs's textbook of obstetrics.

eighth child, but Wakley had apparently lost count—a forgivable error considering the propensity of the Queen to bear children.) Court physicians did not defend their decision to use ether. Perhaps not wanting a public confrontation, they simply denied that the Queen had received an anesthetic. In fact, they first acknowledged a royal anesthetic 4 years later when the Queen delivered her ninth and last child, Princess Beatrice. By that time, however, the issue was no longer controversial.[9]

PUBLIC REACTION TO ETHERIZATION FOR CHILDBIRTH

The controversy surrounding obstetric anesthesia was not resolved by the medical community. Physicians remained skeptical, but public opinion changed. Women lost their reservations, decided they wanted anesthesia, and virtually forced physicians to offer it to them. The change in the public's attitude in favor of obstetric anesthesia marked the culmination of a more general change in social attitudes that had been developing over several centuries.

Before the nineteenth century, pain meant something quite different from what it does today. Since antiquity, people had believed that all manner of calamities—disease, drought, poverty, and pain—signified divine retribution inflicted as punishment for sin. According to Scripture, childbirth pain originated when God punished Eve and her descendants for Eve's disobedience in the Garden of Eden. Many believed that it was wrong to avoid the pain of divine punishment. This belief was sufficiently prevalent and strong to retard acceptance of even the idea of anesthesia, especially for obstetric patients. Only when this tradition weakened did people seek ways to free themselves from disease and pain. In most Western countries, the transition occurred during the nineteenth century. Disease and pain lost their theologic connotations for many people and became biologic processes subject to study and control by new methods of science and technology. This evolution of thought facilitated the development of modern medicine and stimulated public acceptance of obstetric anesthesia.[10]

The reluctance that physicians felt about the administration of anesthesia for childbirth pain stands in stark contrast to the enthusiasm expressed by early obstetric patients. In 1847, Fanny Longfellow, wife of the American poet Henry Wadsworth Longfellow and the first woman in the United States anesthetized for childbirth, wrote:

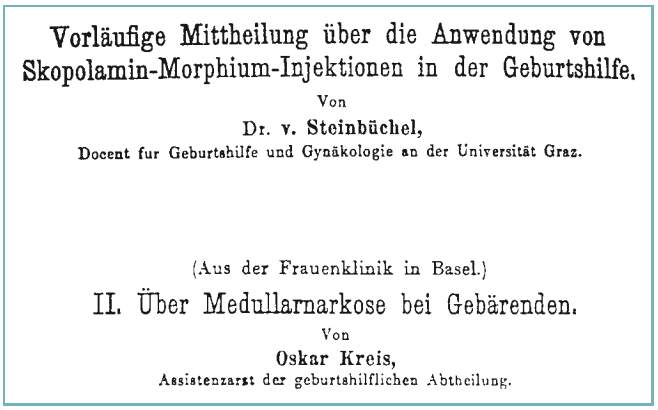
> *I am very sorry you all thought me so rash and naughty in trying the ether. Henry's faith gave me courage, and I had heard such a thing had succeeded abroad, where the surgeons extend this great blessing more boldly and universally than our timid doctors. . . . This is certainly the greatest blessing of this age.*[11]

Queen Victoria, responding to news of the birth of her first grandchild in 1860 and perhaps remembering her own recent confinement, wrote, "What a blessing she [Victoria, her oldest daughter] had chloroform. Perhaps without it her strength would have suffered very much."[9] The new understanding of pain as a controllable biologic process left no room for Meigs's idea that pain might have physiologic value. The eminent nineteenth-century social philosopher John Stuart Mill stated that the "hurtful agencies of nature" promote good only by "inciting rational creatures to rise up and struggle against them."[12]

Simpson prophesied the role of public opinion in the acceptance of obstetric anesthesia, a fact not lost on his adversaries. Early in the controversy he predicted, "Medical men may oppose for a time the superinduction of anaesthesia in parturition but they will oppose it in vain; for certainly our patients themselves will force use of it upon the profession. The whole question is, even now, one merely of time."[13] By 1860, Simpson's prophecy came true; anesthesia for childbirth became part of medical practice by public acclaim, in large part in response to the demands of women.

OPIOIDS AND OBSTETRICS

The next major innovation in obstetric anesthesia came approximately 50 years later. *Dämmerschlaff*, which means "twilight sleep," was a technique developed by von Steinbüchel[14] of Graz and popularized by Gauss[15] of Freiberg. It combined opioids with scopolamine to make women amnestic and somewhat comfortable during labor (Figure 1-4). Until that time, opioids had been used sparingly for obstetrics. Although opium had been part of the medical armamentarium since the Roman Empire, it was



Vorläufige Mittheilung über die Anwendung von Skopolamin-Morphium-Injektionen in der Geburtshilfe.

Von

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(Aus der Frauenklinik in Basel.)

II. Über Medullarnarkose bei Gebärenden.

Von

Oskar Kreis,

Assistenzarst der geburtshilflichen Abtheilung.

FIGURE 1-4 Title pages from two important papers published in the first years of the twentieth century. The paper by von Steinbüchel introduced twilight sleep. The paper by Kreis described the first use of spinal anesthesia for obstetrics.

not used extensively, in part because of the difficulty of obtaining consistent results with the crude extracts available at that time. Therapeutics made a substantial advance in 1809 when Sertürner, a German pharmacologist, isolated codeine and morphine from a crude extract of the poppy seed. Methods for administering the drugs remained unsophisticated. Physicians gave morphine orally or by a method resembling vaccination, in which they placed a drop of solution on the skin and then made multiple small puncture holes with a sharp instrument to facilitate absorption. In 1853, the year Queen Victoria delivered her eighth child, the syringe and hollow metal needle were developed. This technical advance simplified the administration of opioids and facilitated the development of twilight sleep approximately 50 years later.[16]

Although reports of labor pain relief with hypodermic morphine appeared as early as 1868, few physicians favored its use. For example, in an article published in *Transactions of the Obstetrical Society of London*, Sansom[17] listed the following four agents for relief of labor pain: (1) carbon tetrachloride, the use of which he favored; (2) bichloride of methylene, which was under evaluation; (3) nitrous oxide, which had been introduced recently by Klikgowich of Russia; and (4) chloroform. He did not mention opioids, but neither did he mention diethyl ether, which many physicians still favored. Similarly, Gusserow,[18] a prominent German obstetrician, described using salicylic acid but not morphine for labor pain. (Von Baeyer did not introduce acetylsalicylic acid to medical practice until 1899.) In retrospect, von Steinbüchel's and Gauss's descriptions of twilight sleep in the first decade of the century may have been important more for popularizing morphine than for suggesting that scopolamine be given with morphine.

Physicians reacted to twilight sleep as they had reacted to diethyl ether several years earlier. They resisted it, questioning whether the benefits justified the risks. Patients also reacted as they had before. Not aware of, or perhaps not concerned with, the technical considerations that confronted physicians, patients harbored few doubts and persuaded physicians to use it, sometimes against the physicians' better judgment. The confrontation between physicians and patients was particularly strident in the United States. Champions of twilight sleep lectured throughout the country and published articles in popular magazines. Public enthusiasm for the therapy subsided slightly after 1920, when a prominent advocate of the method died during childbirth. She was given twilight sleep, but her physicians said that her death was unrelated to any complication from its use. Whatever anxiety this incident may have created in the minds of patients, it did not seriously diminish their resolve. Confronted by such firm insistence, physicians acquiesced and used twilight sleep with increasing frequency.[19,20]

Although the reaction of physicians to twilight sleep resembled their reaction to etherization, the medical milieu in which the debate over twilight sleep developed was quite different from that in which etherization was deliberated. Between 1850 and 1900, medicine had changed, particularly in Europe. Physiology, chemistry, anatomy, and bacteriology became part of medical theory and practice. Bright students from America traveled to leading clinics in Germany, England, and France. They returned with new facts and methods that they used to examine problems and critique ideas. These developments became the basis for the revolution in American medical education and practice launched by the Flexner report published in 1914.[21]

Obstetrics also changed. During the years preceding World War I, it had earned a reputation as one of the most exciting and scientifically advanced specialties. Obstetricians experimented with new drugs and techniques. They recognized that change entails risk, and they examined each innovation more critically. In addition, they turned to science for information and methods to help them solve problems of medical management. Developments in obstetric anesthesia reflected this change in strategy. New methods introduced during this time stimulated physicians to reexamine two important but unresolved issues, the effects of drugs on the child, and the relationship between pain and labor.

THE EFFECTS OF ANESTHESIA ON THE NEWBORN

Many physicians, Simpson included, worried that anesthetic drugs might cross the placenta and harm the newborn. Available information justified their concern. The idea that gases cross the placenta appeared long before the discovery of oxygen and carbon dioxide. In the sixteenth century, English physiologist John Mayow[22] suggested that "nitro aerial" particles from the mother nourish the fetus. By 1847, physiologists had corroborative evidence. Clinical experience gave more support. John Snow[23] observed depressed neonatal breathing and motor activity and smelled ether on the breath of neonates delivered from mothers who had been given ether. In an early paper, he surmised that anesthetic gases cross the placenta. Regardless, some advocates of obstetric anesthesia discounted the possibility. For example, Harvard professor Walter Channing denied that ether crossed the placenta because he could not detect its odor in the cut ends of the umbilical cord. Oddly enough, he did not attempt to smell ether on the child's exhalations as John Snow had done.[24]

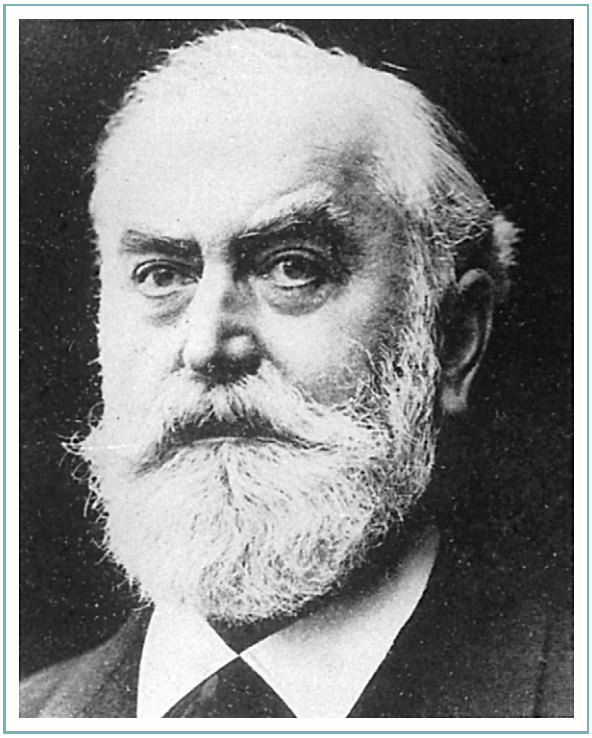


FIGURE 1-5 Paul Zweifel, the Swiss-born obstetrician who performed the first experiments that chemically demonstrated the presence of chloroform in the umbilical blood and urine of infants delivered by women who had been anesthetized during labor. (Courtesy J.F. Bergmann-Verlag, München, Germany.)

In 1874, Swiss obstetrician Paul Zweifel[25] published an account of work that finally resolved the debate about the placental transfer of drugs (Figure 1-5). He used a chemical reaction to demonstrate the presence of chloroform in the umbilical blood of neonates. In a separate paper, Zweifel[26] used a light-absorption technique to demonstrate a difference in oxygen content between umbilical arterial and venous blood, thereby establishing the placental transfer of oxygen. Although clinicians recognized the importance of these data, they accepted the implications slowly. Some clinicians pointed to several decades of clinical use "without problems." For example, Otto Spiegelberg,[27] Professor of Obstetrics at the University of Breslau, wrote in 1887, "As far as the fetus is concerned, no unimpeachable clinical observation has yet been published in which a fetus was injured by chloroform administered to its mother." Experience lulled them into complacency, which may explain their failure to appreciate the threat posed by twilight sleep.

Dangers from twilight sleep probably developed insidiously. The originators of the method, von Steinbüchel and Gauss, recommended conservative doses of drugs. They suggested that 0.3 mg of scopolamine be given every 2 to 3 hours to induce amnesia and that no more than 10 mg of morphine be administered subcutaneously for the whole labor. Gauss, who was especially meticulous, even advised physicians to administer a "memory test" to women in labor to evaluate the need for additional scopolamine. However, as other physicians used the technique, they changed it. Some gave larger doses of opioid—as much as 40 or 50 mg of morphine during labor. Others gave additional drugs (e.g., as much as 600 mg of pentobarbital during labor as well as inhalation agents for delivery). Despite administering these large doses to their patients, some physicians said they had seen no adverse effects on the infants.[28] They probably spoke the truth, but this probability says more about their powers of observation than the safety of the method.

Two situations eventually made physicians confront problems associated with placental transmission of anesthetic drugs. The first was the changing use of morphine.[29] In the latter part of the nineteenth century (before the enactment of laws governing the use of addictive drugs), morphine was a popular ingredient of patent medicines and a drug frequently prescribed by physicians. As addiction became more common, obstetricians saw many pregnant women who were taking large amounts of morphine daily. When they tried to decrease their patients' opioid use, several obstetricians noted unexpected problems (e.g., violent fetal movements, sudden fetal death), which they correctly identified as signs of withdrawal. Second, physiologists and anatomists began extensive studies of placental structure and function. By the turn of the century, they had identified many of the physical and chemical factors that affect rates of drug transfer. Thus, even before twilight sleep became popular, physicians had clinical and laboratory evidence to justify caution. As early as 1877, Gillette[30] described 15 instances of neonatal depression that he attributed to morphine given during labor. Similarly, in a review article published in 1914, Knipe[31] identified stillbirths and neonatal oligopnea and asphyxia as complications of twilight sleep and gave the incidence of each problem as reported by other writers.

When the studies of obstetric anesthesia published between 1880 and 1950 are considered, four characteristics stand out. First, few of them described effects of anesthesia on the newborn. Second, those that did report newborn apnea, oligopnea, or asphyxia seldom defined these words. Third, few used controls or compared one mode of treatment with another. Finally, few writers used their data to evaluate the safety of the practice that they described. In other words, by today's standards, even the best of these papers lacked substance. They did, however, demonstrate a growing concern among physicians about the effects of anesthetic drugs on neonates. Perhaps even more important, their work prepared clinicians for the work of Virginia Apgar (Figure 1-6).

Apgar became an anesthesiologist when the chairman of the Department of Surgery at the Columbia University College of Physicians and Surgeons dissuaded her from becoming a surgeon. After training in anesthesia with Ralph Waters at the University of Wisconsin and with E. A. Rovenstine at Bellevue Hospital, she returned to Columbia Presbyterian Hospital as Director of the Division of Anesthesia. In 1949, she was appointed professor, the first woman to attain that rank at Columbia University.[32]

In 1953, Apgar[33] described a simple, reliable system for evaluating newborns and showed that it was sufficiently sensitive to detect differences among neonates whose mothers had been anesthetized for cesarean delivery by different techniques (Figure 1-7). Infants delivered of women with spinal anesthesia had higher scores than

FIGURE 1-6 Virginia Apgar, whose scoring system revolutionized the practice of obstetrics and anesthesia. Her work made the well-being of the infant the major criterion for the evaluation of medical management of pregnant women. (Courtesy Wood Library Museum.)

those delivered with general anesthesia. The Apgar score had three important effects. First, it replaced simple observation of neonates with a reproducible measurement—that is, it substituted a numerical score for the ambiguities of words such as oligopnea and asphyxia. Thus it established the possibility of the systematic comparison of different treatments. Second, it provided objective criteria for the initiation of neonatal resuscitation. Third, and most important, it helped change the focus of obstetric care. Until that time the primary criterion for success or failure had been the survival and well-being of the mother, a natural goal considering the maternal risks of childbirth until that time. After 1900, as maternal risks diminished, the well-being of the mother no longer served as a sensitive measure of outcome. The Apgar score called attention to the child and made its condition the new standard for evaluating obstetric management.

Current Researches in Anesthesia and Analgesia—July-August, 1953

A Proposal for a New Method of Evaluation of the Newborn Infant.*

Virginia Apgar, M.D., New York, N. Y.

Department of Anesthesiology, Columbia University, College of Physicians and Surgeons and the Anesthesia Service, The Presbyterian Hospital

FIGURE 1-7 Title page from the paper in which Virginia Apgar described her new scoring system for evaluating the well-being of a newborn.

THE EFFECTS OF ANESTHESIA ON LABOR

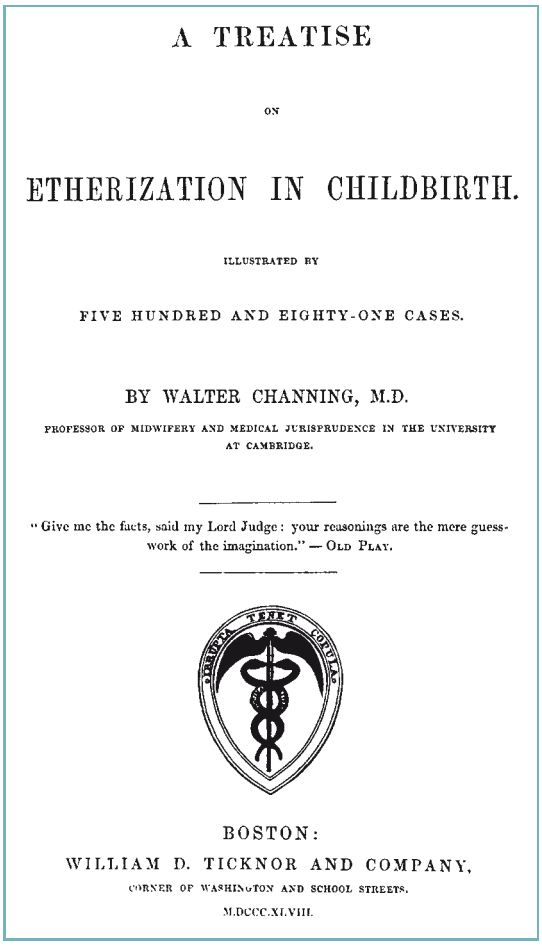
The effects of anesthesia on labor also worried physicians. Again, their fears were well-founded. Diethyl ether and chloroform depress uterine contractions. If given in sufficient amounts, they also abolish reflex pushing with the abdominal muscles during the second stage of labor. These effects are not difficult to detect, even with moderate doses of either inhalation agent.

Simpson's method of obstetric anesthesia used significant amounts of drugs. He started the anesthetic early, and sometimes he rendered patients unconscious during the first stage of labor. In addition, he increased the depth of anesthesia for the delivery.[34] As many people copied his technique, they presumably had ample opportunity to observe uterine atony and postpartum hemorrhage.

Some physicians noticed the effects of anesthetics on uterine function. For example, Meigs[35] said unequivocally that etherization suppressed uterine function, and he described occasions in which he had had to suspend etherization to allow labor to resume. Other physicians waffled, however. For example, Walter Channing,[36] Professor of Midwifery and Medical Jurisprudence at Harvard (seemingly a strange combination of disciplines, but at that time neither of the two was thought sufficiently important to warrant a separate chair), published a book about the use of ether for obstetrics (Figure 1-8). He endorsed etherization and influenced many others to use it. However, his book contained blatant contradictions. On different pages Channing contended that ether had no effect, that it increased uterine contractility, and that it suspended contractions entirely. Then, in a pronouncement smacking more of panache than reason, Channing swept aside his inconsistencies and said that whatever effect ether may have on the uterus he "welcomes it." Noting similar contradictions among other writers, W. F. H. Montgomery,[37] Professor of Midwifery at the King and Queen's College of Physicians in Ireland, wrote, "By one writer we are told that, if uterine action is excessive, chloroform will abate it; by another that if feeble, it will strengthen it and add new vigor to each parturient effort."

John Snow[23] gave a more balanced review of the effects of anesthesia on labor. Originally a surgeon, Snow became the first physician to restrict his practice to anesthesia. He experimented with ether and chloroform and wrote many insightful papers and books describing his work (Figure 1-9). Snow's technique differed from Simpson's. Snow withheld anesthesia until the second stage of labor, limited administration to brief periods during contractions, and attempted to keep his patients comfortable but responsive. To achieve better control of the depth of anesthesia, he recommended using the vaporizing apparatus that he had developed for surgical cases. Snow[23] spoke disparagingly of Simpson's technique and the tendency of people to use it simply because of Simpson's reputation:

> *The high position of Dr. Simpson and his previous services in this department, more particularly in being the first to administer ether in labour, gave his recommendations very great influence; the consequence of which is*



A TREATISE

ON

ETHERIZATION IN CHILDBIRTH.

ILLUSTRATED BY

FIVE HUNDRED AND EIGHTY-ONE CASES.

BY WALTER CHANNING, M.D.

PROFESSOR OF MIDWIFERY AND MEDICAL JURISPRUDENCE IN THE UNIVERSITY AT CAMBRIDGE.

"Give me the facts, said my Lord Judge: your reasonings are the mere guess-work of the imagination." — OLD PLAY.

BOSTON:

WILLIAM D. TICKNOR AND COMPANY,

CORNER OF WASHINGTON AND SCHOOL STREETS.

M.DCCC.XLVIII.

FIGURE 1-8 Frontispiece from Walter Channing's book on the use of etherization for childbirth. Channing favored the use of etherization, and he persuaded others to use it, although evidence ensuring its safety was scant.

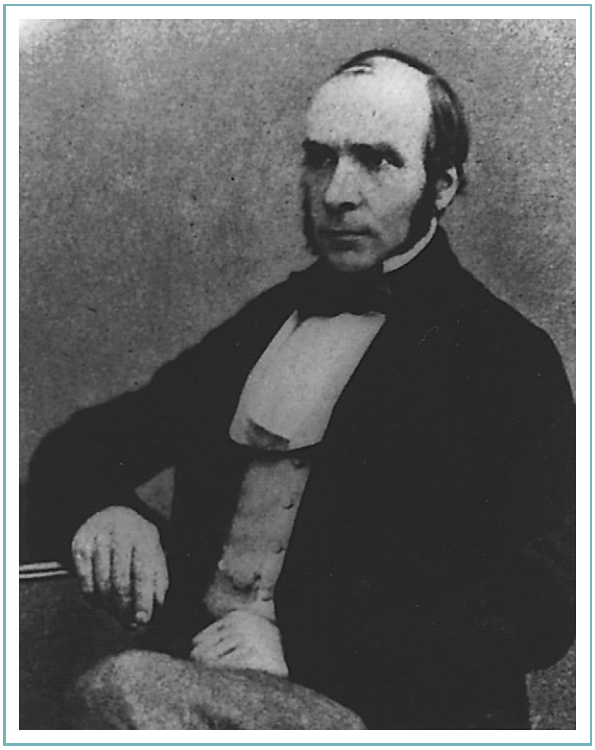


FIGURE 1-9 John Snow, a London surgeon who gave up his surgical practice to become the first physician to devote all his time to anesthesia. He wrote many monographs and papers, some of which accurately describe the effects of anesthesia on infant and mother. (Courtesy Wood Library Museum.)

that the practice of anesthesia is presently probably in a much less satisfactory state than it would have been if chloroform had never been introduced.

Snow's method, which was the same one he had used to anesthetize Queen Victoria, eventually prevailed over Simpson's. Physicians became more cautious with anesthesia, reserving it for special problems such as cephalic version, the application of forceps, abnormal presentation, and eclampsia. They also became more conservative with dosage, often giving anesthesia only during the second stage of labor. Snow's methods were applied to each new inhalation agent—including nitrous oxide, ethylene, cyclopropane, trichloroethylene, and methoxyflurane—as it was introduced to obstetric anesthesia.

Early physicians modified their use of anesthesia from experience, not from study of normal labor or from learning more about the pharmacology of the drugs. Moreover, they had not yet defined the relationship between uterine pain and contractions. As physicians turned more to science during the latter part of the century, however, their strategies began to change. For example, in 1893 the English physiologist Henry Head[38] published his classic studies of the innervation of abdominal viscera. His work stimulated others to investigate the role of the nervous system in the control of labor. Subsequently, clinical and laboratory studies of pregnancy after spinal cord transection established the independence of labor from nervous control.[39] When regional anesthesia appeared during the first decades of the twentieth century, physicians therefore had a conceptual basis from which to explore its effects on labor.

Carl Koller[40] introduced regional anesthesia when he used cocaine for eye surgery in 1884. Recognizing the potential of Koller's innovation, surgeons developed techniques for other procedures. Obstetricians quickly adopted many of these techniques for their own use. The first papers describing obstetric applications of spinal, lumbar epidural, caudal, paravertebral, parasacral, and pudendal nerve blocks appeared between 1900 and 1930 (see Figure 1-4).[41-43] Recognition of the potential effects of regional anesthesia on labor developed more slowly, primarily because obstetricians seldom used it. They continued to rely on inhalation agents and opioids, partly because few drugs and materials were available for regional anesthesia at that time, but also because obstetricians did not appreciate the chief advantage of regional over general anesthesia—the relative absence of drug effects on the infant. Moreover, they rarely used regional anesthesia except for delivery, and then they often used elective forceps anyway. This set of circumstances limited their opportunity and motivation to study the effects of regional anesthesia on labor.

Among early papers dealing with regional anesthesia, one written by Cleland[44] stands out. He described his experience with paravertebral anesthesia, but he also wrote a thoughtful analysis of the nerve pathways mediating labor pain, an analysis he based on information he had gleaned from clinical and laboratory studies. Few investigators were as meticulous or insightful as Cleland. Most of those who studied the effects of anesthesia simply timed the length of the first and second stages of labor. Some timed the duration of individual contractions or estimated changes in the strength of contractions by palpation. None of the investigators measured the intrauterine pressures, even though a German physician had described such a method in 1898 and had used it to evaluate the effects of morphine and ether on the contractions of laboring women.[45]

More detailed and accurate studies of the effects of anesthesia started to appear after 1944. Part of the stimulus was a method for continuous caudal anesthesia introduced by Hingson and Edwards,[46] in which a malleable needle remained in the sacral canal throughout labor. Small, flexible plastic catheters eventually replaced malleable needles and made continuous epidural anesthesia even more popular. With the help of these innovations, obstetricians began using anesthesia earlier in labor. Ensuing problems, real and imagined, stimulated more studies. Although good studies were scarce, the strong interest in the problem represented a marked change from the early days of obstetric anesthesia.

Ironically, "natural childbirth" appeared just as regional anesthesia started to become popular and as clinicians began to understand how to use it without disrupting labor. Dick-Read,[47] the originator of the natural method, recognized "no physiological function in the body which gives rise to pain in the normal course of health." He attributed pain in an otherwise uncomplicated labor to an "activation of the sympathetic nervous system by the emotion of fear." He argued that fear made the uterus contract and become ischemic and therefore painful. He said that women could avoid the pain if they simply learned to abolish their fear of labor. Dick-Read never explained why uterine ischemia that results from fear causes pain, whereas ischemia that results from a normal contraction does not. In other words, Dick-Read, like Simpson a century earlier, claimed no necessary or physiologic relationship between labor pain and contractions. Dick-Read's book, written more for the public than for the medical profession, represented a regression of almost a century in medical thought and practice. It is important to note that contemporary methods of childbirth preparation do not maintain that fear alone causes labor pain. However, they do attempt to reduce fear by education and to help patients manage pain by teaching techniques of self-control. This represents a significant difference from and an important advance over Dick-Read's original theory.

SOME LESSONS

History is important in proportion to the lessons it teaches. With respect to obstetric anesthesia, four lessons stand out. First, every new drug and method entails risks. Physicians who first used obstetric anesthesia seemed reluctant to accept this fact, perhaps because of their inexperience with potent drugs (pharmacology was in its infancy) or because they acceded too quickly to patients, who wanted relief from pain and had little understanding of the technical issues confronting physicians. Whatever the reason, this period of denial lasted almost half a century, until 1900. Almost another half-century passed before obstetricians learned to modify their practice to limit the effects of anesthetics on the child and the labor process.

Second, new drugs or therapies often cause problems in completely unexpected ways. For example, in 1900, physicians noted a rising rate of puerperal fever.[48] The timing was odd. Several decades had passed since Robert Koch had suggested the germ theory of disease and since Semmelweis had recognized that physicians often transmit infection from one woman to the next with their unclean hands. With the adoption of aseptic methods, deaths from puerperal fever had diminished dramatically. During the waning years of the nineteenth century, however, they increased again. Some physicians attributed this resurgence of puerperal fever to anesthesia. In a presidential address to the Obstetrical Society of Edinburgh in 1900, Murray[49] stated the following:

> *I feel sure that an explanation of much of the increase of maternal mortality from 1847 onwards will be found in, first the misuse of anaesthesia and second in the ridiculous parody which, in many hands, stands for the use of antiseptics. . . . Before the days of anaesthesia, interference was limited and obstetric operations were at a minimum because interference of all kinds increased the conscious suffering of the patient. . . . When anaesthesia became possible, and interference became more frequent because it involved no additional suffering, operations were undertaken when really unnecessary . . . and so complications arose and the dangers of the labor increased.*

Although it was not a direct complication of the use of anesthesia in obstetric practice, puerperal fever appeared to be an indirect consequence of it.

Changes in obstetric practice also had unexpected effects on anesthetic complications. During the first decades of the twentieth century, when cesarean deliveries were rare and obstetricians used only inhalation analgesia for delivery, few women were exposed to the risk of aspiration during deep anesthesia. As obstetric practice changed and cesarean deliveries became more common, this risk rose. The syndrome of aspiration was not identified and labeled until 1946, when obstetrician Curtis Mendelson[50] described and named it. The pathophysiology of the syndrome had already been described by Winternitz et al.,[51] who instilled hydrochloric acid into the lungs of dogs to simulate the lesions found in veterans poisoned by gas during the trench warfare of World War I. Unfortunately, the reports of these studies, although excellent, did not initiate any change in practice. Change occurred only after several deaths of obstetric patients were highly publicized in lay, legal, and medical publications. Of course, rapid-sequence induction, currently recommended to reduce the risk of aspiration, creates another set of risks—those associated with a failed intubation.

The third lesson offered by the history of obstetric anesthesia concerns the role of basic science. Modern medicine developed during the nineteenth century after physicians learned to apply principles of anatomy, physiology, and

chemistry to the study and treatment of disease. Obstetric anesthesia underwent a similar pattern of development. Studies of placental structure and function called physicians' attention to the transmission of drugs and the potential effects of drugs on the infant. Similarly, studies of the physiology and anatomy of the uterus helped elucidate potential effects of anesthesia on labor. In each instance, lessons from basic science helped improve patient care.

The fourth and perhaps the most important lesson is the role that patients have played in the use of anesthesia for obstetrics. During the nineteenth century it was women who pressured cautious physicians to incorporate routine use of anesthesia into their obstetric practice. A century later, it was women again who altered patterns of practice, this time questioning the overuse of anesthesia for routine deliveries. In both instances the pressure on physicians emanated from prevailing social values regarding pain. In 1900 the public believed that pain, and in particular obstetric pain, was destructive and something that should be avoided. Half a century later, with the advent of the natural childbirth movement, many people began to suggest that the experience of pain during childbirth, perhaps even in other situations, might have some physiologic if not social value. Physicians must recognize and acknowledge the extent to which social values may shape medical "science" and practice.[52,53]

During the past 60 years, scientists have accumulated a wealth of information about many processes integral to normal labor: the processes that initiate and control lactation; neuroendocrine events that initiate and maintain labor; the biochemical maturation of the fetal lung and liver; the metabolic requirements of the normal fetus and the protective mechanisms that it may invoke in times of stress; and the normal mechanisms that regulate the amount and distribution of blood flow to the uterus and placenta. At this point, we have only the most rudimentary understanding of the interaction of anesthesia with any of these processes. Only a fraction of the information available from basic science has been used to improve obstetric anesthesia care. Realizing the rewards from the clinical use of such information may be the most important lesson from the past and the greatest challenge for the future of obstetric anesthesia.

KEY POINTS

- Physicians have debated the safety of obstetric anesthesia since 1847, when James Young Simpson first administered anesthesia for delivery. Two issues have dominated the debate: the effects of anesthesia on labor and the effects of anesthesia on the newborn.
- Despite controversy, physicians quickly incorporated anesthesia into clinical practice, largely because of their patients' desire to avoid childbirth pain.
- Only after obstetric anesthesia was in use for many years did problems become apparent.
- Important milestones in obstetric anesthesia are the introduction of inhalation agents in 1847, the expanded use of opioids in the early decades of the twentieth century, and the refinement of regional anesthesia starting in the mid-twentieth century.
- Outstanding conceptual developments are (1) Zweifel's idea that drugs given to the mother cross the placenta and affect the fetus and (2) Apgar's idea that the condition of the newborn is the most sensitive assay of the quality of anesthetic care of the mother.
- The history of obstetric anesthesia suggests that the major improvements in patient care have followed the application of principles of basic science.

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Part II

Maternal and Fetal Physiology

Metabolism was among the first areas of physiology to influence clinical practice. By the beginning of the twentieth century, physiologists had established many of the principles that we recognize today, including normal rates of oxygen consumption and carbon dioxide production, the relationship between oxygen consumption and heat production, and the relationship between metabolic rate and body weight and surface area among individuals and species. Almost simultaneously, clinicians began to apply these principles to their studies of patients in different states of health and disease.

In one early study, physiologist Magnus-Levy[1] found an exception to the rule that basal metabolic rate varied in proportion to body surface area. As he measured a woman's oxygen consumption during pregnancy, he observed that her metabolic rate increased out of proportion to increments in her body weight and surface area. Subsequent studies by other investigators established the basis of this phenomenon. Per unit of weight, the fetus, placenta, and uterus together consumed oxygen (and released carbon dioxide and heat) at a higher rate than the mother. In effect, the metabolism of a pregnant woman represented the sum of two independent organisms, each metabolizing at its own rate in proportion to its own surface area. Thus, each kilogram of maternal tissue consumed oxygen at a rate of approximately 4 mL/min, whereas the average rate for the fetus, placenta, and uterus was approximately 12 mL/min, although it could rise as high as 20 mL/min. Therefore, during pregnancy, the mother's metabolism was the sum of her metabolic rate plus that of the fetus, placenta, and uterus.[1-4] Subsequent studies established that the highest rates of fetal metabolism occurred during the periods of most rapid growth, thereby reaffirming another physiologic principle—the high metabolic cost of synthesizing new tissue.[5]

The aforementioned studies gave clinicians estimates of the stress imposed by pregnancy. To maintain homeostasis during pregnancy, a pregnant woman had to make an appropriate adjustment in each of the physiologic mechanisms involved in the delivery of substrates to the fetal placental unit and in the excretion of metabolic wastes. Thus, for every increment in fetal weight, clinicians could expect to find a proportional change in all the mechanisms involved in the delivery of substrate to the fetus and in the excretion of all byproducts. In fact, subsequent clinical studies established predictable changes in uterine blood flow, cardiac output, blood volume, minute ventilation, the dissipation of body heat, and the renal excretion of nitrogenous waste and other materials.

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Physiologic Changes of Pregnancy

Robert Gaiser, M.D.

Marked anatomic and physiologic changes occur in women during pregnancy. The pregnant woman must adapt to the developing fetus and provide for its increased metabolic demands. The enlarging uterus places mechanical strain on the woman's body. The greater production of various hormones by the ovaries and the placenta further alters maternal physiology. The hallmark of successful anesthetic management of the pregnant woman is recognition of these anatomic and physiologic changes and appropriate adaptation of anesthetic techniques to these changes. This chapter reviews the physiologic alterations of normal pregnancy and their anesthetic implications.

BODY WEIGHT AND COMPOSITION

The mean maternal weight increase during pregnancy is 17% of the prepregnancy weight, or approximately 12 kg.[1] Weight gain results from an increase in the size of the uterus and its contents (uterus, 1 kg; amniotic fluid, 1 kg; fetus and placenta, 4 kg); increases in blood volume and interstitial fluid (approximately 2 kg each); and deposition of new fat and protein (approximately 4 kg). Normal weight gain during the first trimester is 1 to 2 kg, and there is a 5- to 6-kg gain in each of the last two trimesters.

Obesity is a major problem in the United States and also represents a potential problem for the parturient. Obesity increases the risk of adverse pregnancy outcome, including the rate of cesarean delivery. The odds ratios for cesarean delivery are 1.46, 2.05, and 2.89 for overweight, obese, and severely obese women, respectively, compared with pregnant women of normal weight.[2] Excessive weight gain during pregnancy constitutes a major risk factor for long-term increase in body mass index (BMI).[3]

THE HEART AND CIRCULATION

Physical Examination of the Heart

Pregnancy causes cardiac hypertrophy. The hypertrophy is a result of greater blood volume as well as increased stretch and force of contraction.[4] These changes, coupled with the elevation of the diaphragm, lead to several alterations in cardiac findings.

Changes in heart sounds include accentuation of the first heart sound with exaggerated splitting of the mitral and tricuspid components (Box 2-1).[5] The second heart sound changes little, although the aortic-pulmonic interval tends to vary less with respiration during the third trimester. The third heart sound may be heard during the third trimester and does not possess any clinical significance. A fourth heart sound may be heard in 16% of pregnant women, although it typically disappears by term. A grade II systolic ejection murmur is commonly heard at the left sternal border.[6] The murmur is considered a benign flow murmur that is attributed to cardiac enlargement from increased intravascular volume, which causes dilation of the tricuspid annulus and regurgitation.

Elevation of the diaphragm shifts the heart anteriorly and to the left during pregnancy. On physical examination the point of maximal cardiac impulse is displaced cephalad to the fourth intercostal space and leftward to at least the midclavicular line.

The electrocardiogram (ECG) typically changes during pregnancy, especially during the third trimester. There is an increase in heart rate and a shortening of both the PR interval and the uncorrected QT interval. This shortening has implications for the clinical course of women with long QT syndrome.[7] During pregnancy in these women, the risk of cardiac events is reduced (risk ratio [RR] = 0.38) compared with that in periods between pregnancy. However, the initial 9 months after delivery are associated with a markedly higher risk of adverse cardiac events (RR = 2.7), suggesting that the QT interval may be prolonged in the postpartum period. Other ECG changes include an axis shift; the QRS axis shifts to the right during the first trimester but may shift to the left during the third trimester as a result of displacement by the expanding uterus.[8] Depressed ST segments and isoelectric low-voltage T waves in the left-sided precordial and limb leads are commonly observed during pregnancy.[9]

Echocardiography demonstrates left ventricular hypertrophy by 12 weeks' gestation, with a 50% increase in mass at term.[10] This hypertrophy occurs as a result of an increase in the size of the preexisting cardiomyocytes rather than in the number of cells. The hypertrophy is eccentric, resembling that developed during exercise.[1] A significant increase in the annular diameters of the mitral, tricuspid, and pulmonic valves occurs; 94% of term pregnant women exhibit tricuspid and pulmonic regurgitation, and 27% exhibit mitral regurgitation.[11] The aortic annulus is not dilated.

BOX 2-1 Changes in the Cardiac Examination in the Pregnant Patient

- Accentuation of S1 (first heart sound); exaggerated splitting of the mitral and tricuspid components
- Typical systolic ejection murmur
- Possible presence of S3 (third heart sound) and S4 (fourth heart sound) (no clinical significance)
- Leftward displacement of point of maximal cardiac impulse

Central Hemodynamics

Prerequisites for the accurate determination of hemodynamic changes during pregnancy require that measurements be made with subjects in a resting position, which minimizes compression of the aorta and inferior vena cava by the gravid uterus. Further, comparisons must be made with an appropriate control, such as the same group of women before pregnancy or a matched group of nonpregnant women. If control measurements are made during the postpartum period, a sufficient interval must have elapsed so that hemodynamic parameters return to prepregnancy values. For some measurements, this return may take 24 weeks or more.[12]

Cardiac output increases during pregnancy (Table 2-1). This change occurs by 5 weeks' gestation, with a resultant increase of 35% to 40% by the end of the first trimester of pregnancy.[10,13] Cardiac output continues to rise throughout the second trimester until it reaches a level approximately 50% greater than that of nonpregnant women (Figure 2-1).[10,12,14-16] This parameter does not change from this level during the third trimester. In the past it had been postulated that cardiac output declined during

TABLE 2-1 Central Hemodynamics at Term Gestation

Parameter	Change*
Cardiac output	+50%
Stroke volume	+25%
Heart rate	+25%
Left ventricular end-diastolic volume	Increased
Left ventricular end-systolic volume	No change
Ejection fraction	Increased
Left ventricular stroke work index	No change
Pulmonary capillary wedge pressure	No change
Pulmonary artery diastolic pressure	No change
Central venous pressure	No change
Systemic vascular resistance	−20%

*Relative to nonpregnant state.

Adapted from Conklin KA. Maternal physiological adaptations during gestation, labor, and the puerperium. Semin Anesth 1991; 10:221-34.

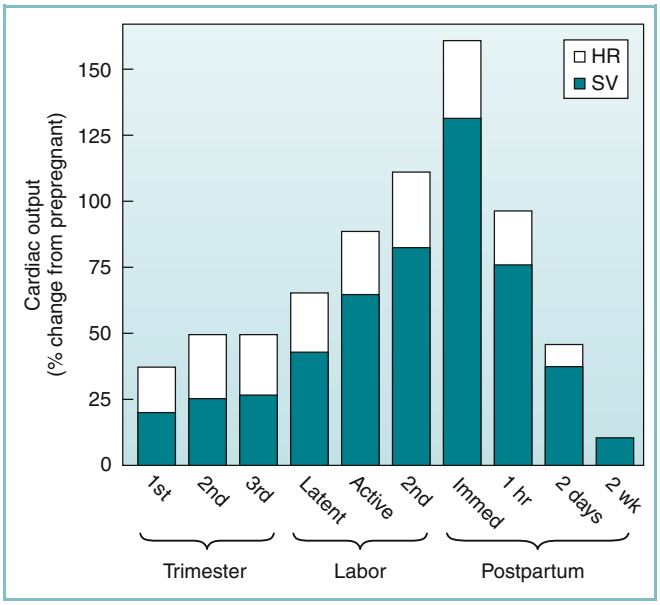

FIGURE 2-1 Cardiac output during pregnancy, labor, and the puerperium. Values during pregnancy are measured at the end of the first, second, and third trimesters. Values during labor are measured between contractions. For each measurement, the relative contributions of heart rate (HR) and stroke volume (SV) to the change in cardiac output are illustrated.

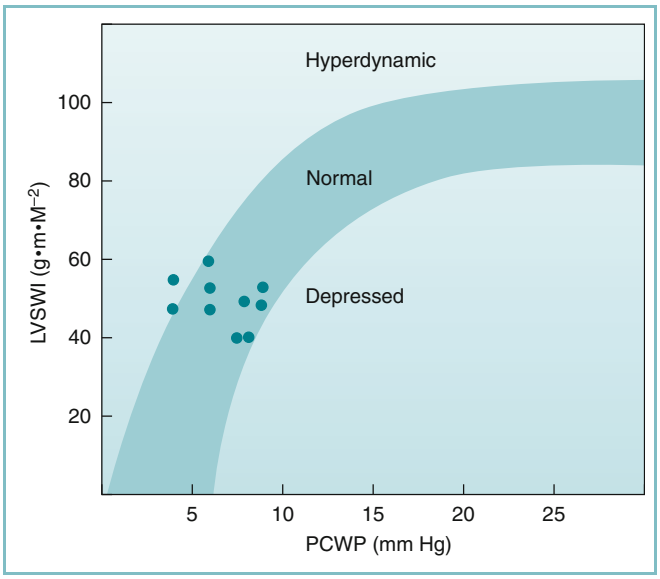

FIGURE 2-2 Left ventricular function in late phase of third-trimester normotensive pregnant patients. LVSWI, left ventricular stroke work index; PCWP, pulmonary capillary wedge pressure. (Modified from Clark SL, Cotton DB, Lee W, et al. Central hemodynamic assessment of cardiac function. Am J Obstet Gynecol 1989; 161:439-42.)

the third trimester. The problem with this supposition was that measurements were obtained with pregnant women in the supine position; thus, the observed decrease in cardiac output was a reflection of aortocaval compression rather than a true physiologic change.

Cardiac output is a reflection of heart rate and stroke volume. The earliest change in cardiac output is attributed to an increase in heart rate, which occurs by the fourth to fifth week of pregnancy.[10] The heart rate increases approximately 15% to 25% over baseline by the end of the first trimester and does not change during the third trimester.[10,12-17] Stroke volume rises by approximately 20% between the fifth and eighth weeks of gestation, and by 25% to 30% by the end of the second trimester, and then remains at this level until term.[10,12,13,17] Left ventricular mass increases by 23% from the first to the third trimester.[18] The increase in stroke volume correlates with rising estrogen levels.[1]

Left ventricular end-diastolic volume increases during gestation, whereas end-systolic volume remains unchanged, resulting in a larger ejection fraction.[10,12-15,17] Central venous, pulmonary artery diastolic, and pulmonary capillary wedge pressures during pregnancy are within the normal range for nonpregnant individuals.[16] The apparent discrepancy between left ventricular filling pressure and end-diastolic volume is explained by hypertrophy and dilation, with the dilated ventricle accommodating a greater volume without an increase in pressure.

Myocardial contractility is also increased, as demonstrated by the higher velocity of left ventricular circumferential fiber shortening (Figure 2-2).[10,14,17] Tissue Doppler imaging has been used to assess diastolic function, as this technique is relatively independent of preload.[19] Investigators found that left ventricular diastolic function was not impaired during pregnancy, whereas systolic function was increased during the second trimester.

The rise in cardiac output during pregnancy results in greater perfusion to the uterus, kidneys, and extremities. Blood flow to the brain and liver do not change. Uterine blood flow increases from a baseline of approximately 50 mL/min to 700 to 900 mL/min at term.[20-24] Approximately 90% of this flow perfuses the intervillous space, with the balance perfusing the myometrium.[22] At term, skin blood flow is approximately three to four times the nonpregnant level, resulting in higher skin temperature.[25] Renal plasma flow is increased by 80% at 16 to 26 weeks' gestation but declines to 50% above the nonpregnant baseline at term.[26]

Blood Pressure

Positioning, gestational age, and parity affect blood pressure measurements. Brachial sphygmomanometry yields the highest measurements in the supine position and the lowest measurements in the lateral position.[15,27] Blood pressure increases with maternal age, and for a given age, nulliparous women have a higher mean pressure than parous women.[28] Systolic, diastolic, and mean arterial pressures decrease during mid-pregnancy and return toward baseline as the pregnancy approaches term.[29] Diastolic blood pressure falls to a greater degree than does systolic blood pressure, with early to mid-gestational decreases of approximately 20%.[30] The changes in blood pressure are consistent with changes in systemic vascular resistance, which falls during early gestation, reaches its nadir (35% decline) at 20 weeks' gestation, and rises during late gestation. Unlike blood pressure, systemic vascular

resistance remains approximately 20% below the nonpregnant level at term.[12,16] The decreased systemic vascular resistance results from the development of a low-resistance vascular bed (the intervillous space) as well as vasodilation caused by prostacyclin, estrogen, and progesterone.

Aortocaval Compression

The extent of compression of the aorta and inferior vena cava by the gravid uterus depends on positioning and weeks' gestation. At term there is partial vena caval compression in the lateral position, as documented by angiography.[31] This finding is consistent with the 75% elevation—above nonpregnant levels—of femoral venous and lower inferior vena caval pressures.[32] Despite caval compression, collateral circulation maintains venous return, as reflected by the right ventricular filling pressure, which is unaltered in the lateral position.[16]

In the supine position, there is nearly complete obstruction of the inferior vena cava at term gestation.[33] Blood return from the lower extremities occurs through the intraosseous vertebral veins, paravertebral veins, and the epidural veins.[34] However, venous return via these collaterals is less than would occur through the inferior vena cava, resulting in a decrease in right atrial pressure.[35] Compression of the inferior vena cava occurs as early as 13 to 16 weeks' gestation and is evident from the 50% increase in femoral venous pressure that occurs when a woman assumes the supine position at this stage of pregnancy (Figure 2-3).[36] By term, femoral venous and lower inferior vena cava pressures are approximately 2.5 times the nonpregnant measurements in the supine position.[32,36]

In the supine position, the aorta may be compressed by the term gravid uterus. This compression accounts for the lower femoral artery pressure compared with brachial artery pressure in the supine position.[37,38] These findings are consistent with angiographic studies in supine pregnant women, which show partial obstruction of the aorta at the level of the lumbar lordosis and enhanced compression during periods of maternal hypotension.[39]

At term, the left lateral decubitus position results in less enhancement of cardiac sympathetic nervous system activity and less suppression of cardiac vagal activity than the supine or right lateral decubitus position.[40] Women who assume the supine position at term gestation experience a 10% to 20% decline in stroke volume and cardiac output.[41,42] These effects are consistent with the fall in right atrial filling pressure. Blood flow in the upper extremities is normal, whereas uterine blood flow decreases by 20%, and lower extremity blood flow falls by 50%.[43] Perfusion of the uterus is less affected than that of the lower extremities because vena caval compression does not obstruct venous outflow via the ovarian veins.[34] The adverse hemodynamic effects of aortocaval compression are reduced once the fetal head is engaged.[37,38]

Many term pregnant women exhibit an increase in brachial artery pressure when they assume the supine position. This finding is caused by a higher systemic vascular resistance, which is attributed to compression of the aorta. Up to 15% of women at term experience bradycardia and a substantial drop in blood pressure when supine, the so-called **supine hypotension syndrome**.[44,45] It may take several minutes for the bradycardia and hypotension to develop, and the bradycardia is usually preceded by a period of tachycardia. The syndrome results from a profound drop in venous return for which the cardiovascular system cannot compensate.

Hemodynamic Changes during Labor and the Puerperium

Cardiac output during labor (but between uterine contractions) increases from prelabor measurements by approximately 10% in the early first stage, by 25% in the late first

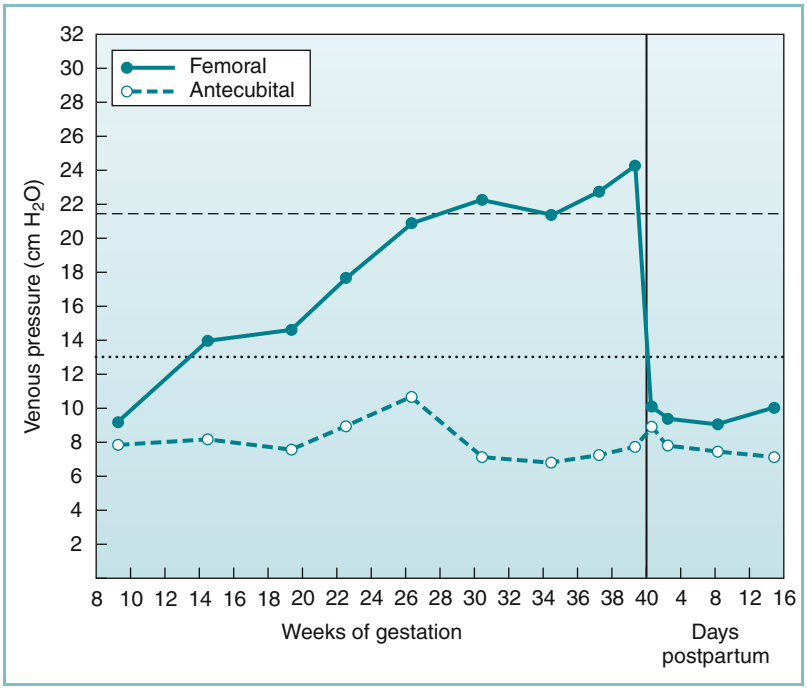

FIGURE 2-3 Femoral and antecubital venous pressures in the supine position throughout normal pregnancy and the puerperium. (Modified from McLennan CE. Antecubital and femoral venous pressure in normal and toxemic pregnancy. Am J Obstet Gynecol 1943; 45:568-91.)

stage, and by 40% in the second stage.[46-48] In the immediate postpartum period, cardiac output may be as much as 75% above predelivery measurements.[47] These changes result from an increase in stroke volume due to greater venous return and alterations in sympathetic nervous system activity. During uterine contractions, 300 to 500 mL of blood is displaced from the intervillous space into the central circulation (i.e., autotransfusion).[49-51] Increased intrauterine pressure forces blood from the intervillous space through the relatively unimpeded ovarian venous outflow system.[34] The postpartum rise in cardiac output results from relief of vena caval compression, diminished lower extremity venous pressure, and a reduction of maternal vascular capacitance.[48] Cardiac output falls to just below prelabor measurements at 24 hours postpartum.[49] It returns to prepregnancy levels between 12 and 24 weeks postpartum.[12] Heart rate falls rapidly after delivery, reaches the prepregnancy rate by 2 weeks postpartum, and is slightly below the prepregnancy rate for the next several months.[12,52] Other anatomic and functional changes of the heart are also fully reversible.[19,53]

TABLE 2-2 Effects of Pregnancy on Respiratory Mechanics

Parameter	Change*
Diaphragm excursion	Increased
Chest wall excursion	Decreased
Pulmonary resistance	Decreased 50%
FEV_1	No change
FEV_1/FVC	No change
Flow-volume loop	No change
Closing capacity	No change

FEV_1, Forced expiratory volume in 1 second; FVC, forced vital capacity.

*Relative to nonpregnant state.

Adapted from Conklin KA. Maternal physiological adaptations during gestation, labor, and the puerperium. Semin Anesth 1991; 10:221-34.

THE RESPIRATORY SYSTEM

Despite the multiple anatomic and physiologic changes that occur during pregnancy, it is remarkable that pregnancy has a relatively minor impact on lung function.

Anatomy

The thoracic cage enlarges in circumference by 5 to 7 cm during pregnancy because of increases in both anteroposterior and transverse diameters.[54,55] An increase in the hormone relaxin causes the rib cage to undergo structural changes, including relaxation of the ligamentous attachments of the ribs.[55,56] The vertical measurement of the chest decreases by as much as 4 cm as a result of the elevated position of the diaphragm.

Capillary engorgement of the larynx and the nasal and oropharyngeal mucosa begins early in the first trimester and increases progressively throughout pregnancy.[54] Voice changes frequently result from involvement of the false vocal cords and the arytenoid region of the larynx. Nasal breathing commonly becomes difficult, and epistaxis may occur. The nasal congestion may contribute to the perceived shortness of breath of pregnancy.[57]

Airflow Mechanics

Inspiration in the term pregnant woman is almost totally attributable to diaphragmatic excursion.[58] This is caused by a greater descent of the diaphragm from its elevated resting position and limitation of thoracic cage expansion because of its expanded resting position (Table 2-2). Both large- and small-airway function are minimally altered during pregnancy. The shape of flow-volume loops, the absolute flow rates at normal lung volumes,[59] forced expiratory volume in 1 second (FEV_1), and the ratio of FEV_1 to forced vital capacity (FVC) are unchanged during pregnancy, as is closing capacity.[60]

The peak expiratory flow rate achieved with a maximal effort following a maximal inspiration is often considered a surrogate for the FEV_1. The test is often used to monitor asthma therapy. Studies are conflicting as to whether peak expiratory flow rates decrease during pregnancy.[61,62] Harirah et al.[61] found that peak expiratory flow rate declined significantly throughout gestation in all positions (Figure 2-4) and that flow rates in the supine position were lower than those during standing and sitting. The mean rate of decline was 0.65 L/min per week, and peak expiratory flow remained below normal at 6 weeks postpartum.

Lung Volumes and Capacities

Tidal volume increases by 45% during pregnancy, with approximately half of the change occurring during the first trimester (Table 2-3; Figure 2-5). The early change in tidal volume is associated with a reduction in inspiratory reserve volume. Total lung capacity usually is preserved or

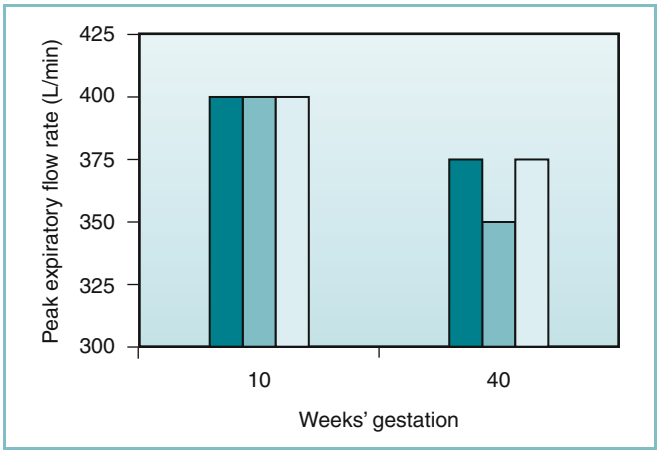

FIGURE 2-4 Peak expiratory flow rates during standing (*dark blue bars*), lying supine (*medium blue bars*), and during sitting (*gray bars*) in pregnant women. Peak expiratory flow rates declined significantly throughout gestation. The rate of decline was greater for the supine position than for the standing position. (Based on data from Harirah HM, Donia SE, Nasrallah FK, et al. Effect of gestational age and position on peak expiratory flow rate: A longitudinal study. Obstet Gynecol 2005; 10:372-6.)

TABLE 2-3 Changes in Respiratory Physiology at Term Gestation

Parameter	Change*
Lung volumes:	
Inspiratory reserve volume	+5%
Tidal volume	+45%
Expiratory reserve volume	−25%
Residual volume	−15%
Lung capacities:	
Inspiratory capacity	+15%
Functional residual capacity	−20%
Vital capacity	No change
Total lung capacity	−5%
Dead space	+45%
Respiratory rate	No change
Ventilation:	
Minute ventilation	+45%
Alveolar ventilation	+45%

*Relative to nonpregnant state.

From Conklin KA. Maternal physiological adaptations during gestation, labor, and the puerperium. Semin Anesth 1991; 10:221-34.

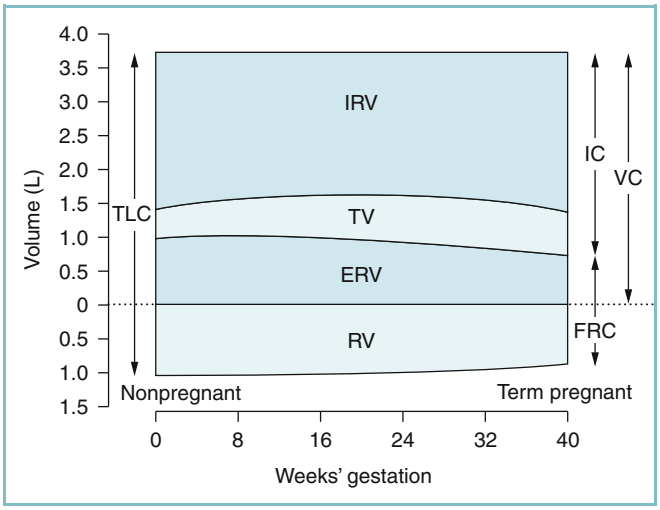

FIGURE 2-5 Lung volumes and capacities during pregnancy. ERV, expiratory reserve volume; FRC, functional residual capacity; IC, inspiratory capacity; IRV, inspiratory reserve volume; RV, residual volume; TLC, total lung capacity; TV, tidal volume; VC, vital capacity.

is decreased minimally. The residual volume tends to drop slightly, a change that maintains vital capacity. Inspiratory capacity increases by 15% during the third trimester because of increases in tidal volume and inspiratory reserve volume.[63,64] There is a corresponding decrease in expiratory reserve volume.[63,64]

Functional residual capacity (FRC) begins to fall by the fifth month of pregnancy and is decreased to 80% of the prepregnancy capacity by term.[60,63,64] This change is caused by elevation of the diaphragm, which occurs as the enlarging uterus enters the abdominal cavity. A 25% reduction in expiratory reserve volume and a 15% reduction in residual volume account for the change. It is important to remember that assumption of the supine position causes FRC to decrease further to 70% of the prepregnancy volume. Total lung capacity is slightly reduced during pregnancy.[63,65]

Ventilation and Blood Gases

Resting minute ventilation rises during pregnancy (see Table 2-3), owing primarily to an increase in tidal volume.[63,66] The inspiratory rate and pattern remain relatively unchanged. The ratio of total dead space to tidal volume remains constant during pregnancy, resulting in an increase in alveolar ventilation of approximately 30% above baseline. The rise in minute ventilation results from hormonal changes and greater carbon dioxide production. Carbon dioxide production at rest rises by about 30% to 300 mL/min[59] and is closely related to the blood level of progesterone.[67] Progesterone acts as a direct respiratory stimulant,[68] and the progesterone-induced increase in chemosensitivity results in a steeper slope and a leftward shift of the CO_2 ventilatory response curve. The greater chemosensitivity occurs early in pregnancy and remains constant until delivery.[59]

The hypoxic ventilatory response is increased during pregnancy to twice the normal level secondary to rises in estrogen and progesterone levels.[69] This increase occurs despite blood and cerebrospinal fluid (CSF) alkalosis.

The arterial pressure of oxygen increases to 100 to 105 mm Hg as a result of greater alveolar ventilation (Table 2-4).[66] The higher Pa_{O_2} results from the decline in Pa_{CO_2} and a lower arteriovenous oxygen difference, which reduces the impact of venous admixture on the Pa_{O_2}.[70,71] As pregnancy progresses, oxygen consumption continues to increase, and cardiac output increases to a lesser extent, resulting in a reduced mixed venous oxygen content and increased arteriovenous oxygen difference. After midgestation, pregnant women in the supine position frequently exhibit a Pa_{O_2} less than 100 mm Hg. This occurs because the FRC may be less than closing capacity, resulting in closure of small airways during normal tidal volume ventilation.[66] Moving a pregnant woman from the supine to the erect or lateral decubitus position improves arterial oxygenation and reduces the alveolar-to-arterial oxygen gradient.

The Pa_{CO_2} declines to approximately 30 mm Hg by 12 weeks' gestation but does not change further during the remainder of pregnancy. Although in nonpregnant individuals a gradient exists between the end-tidal carbon dioxide tension and Pa_{CO_2}, the two measurements are equivalent during early pregnancy,[72] at term gestation,[73]

TABLE 2-4 Blood Gas Measurements during Pregnancy

		Trimester		
Parameter	Nonpregnant	First	Second	Third
Pa_{CO_2} (mm Hg)	40	30	30	30
Pa_{O_2} (mm Hg)	100	107	105	103
pH	7.40	7.44	7.44	7.44
$[HCO_3^-]$ (mEq/L)	24	21	20	20

and in the postpartum period.[74] This finding is attributed to a reduction in alveolar dead space, which results from an increase in cardiac output during pregnancy. The mixed venous P_{CO_2} is 6 to 8 mm Hg below the nonpregnant level from the late first trimester until term.[1]

Metabolic compensation for the respiratory alkalosis of pregnancy reduces the serum bicarbonate concentration to approximately 20 mEq/L, the base excess by 2 to 3 mEq/L, and the total buffer base by approximately 5 mEq/L.[75] This compensation is incomplete, as demonstrated by the elevation of venous,[76] capillary,[77] and arterial[66] blood pH by 0.02 to 0.06 unit.

The rise in minute ventilation that accompanies pregnancy may be interpreted as shortness of breath. By 30 weeks' gestation, 75% of pregnant women have exertional dyspnea.[78] The proposed etiology is the greater drive to breathe and the increased respiratory load. Factors that contribute to the sensation of dyspnea include larger pulmonary blood volume, anemia, and nasal congestion. Exercise has no effect on pregnancy-induced changes in ventilation or alveolar gas exchange.[79]

Sleep

Sleep disturbances are a common occurrence during pregnancy owing to mechanical and hormonal factors. Pregnant women have more complaints of insomnia and daytime sleepiness. The American Academy of Sleep Medicine has established a disease entity, **pregnancy-associated sleep disorder,** defined as the occurrence of insomnia or excessive sleepiness that develops in the course of pregnancy.[80] Progesterone has a strong sedating effect, and cortisol, levels of which are higher in pregnancy, is associated with an increase in rapid eye movement (REM) sleep.[81]

Sleep quality is worsened in the first and third trimesters. Polysomnography reveals reduced slow-wave and REM phases of sleep, decreased total sleep time, and increased rate of wakening after sleep onset.[82] Sleep may be poor for up to 3 months postpartum.[82,83] Snoring is common during pregnancy, occurring in up to 20% of women by the third trimester. Pregnancy is associated with transient restless leg syndrome, a disorder in which the patient experiences the need to move her legs.[84] The incidence ranges from 15% in the first trimester to 23% in the third trimester.[84]

Metabolism and Respiration during Labor and the Puerperium

Minute ventilation of the unmedicated parturient increases by 70% to 140% in the first stage of labor and by 120% to 200% in the second stage of labor compared with prepregnancy measurements.[85] The Pa_{CO_2} may fall to as low as 10 to 15 mm Hg. Oxygen consumption increases above the prelabor value by 40% in the first stage and by 75% in the second stage.[85] The changes in oxygen consumption result from the increased metabolic demands of hyperventilation, uterine activity, and maternal expulsive efforts during the second stage. The maternal aerobic requirement for oxygen exceeds oxygen consumption during labor, as is evident from the progressive rise of blood lactate concentration, an index of anaerobic metabolism.[86-88] Initiation of neuraxial analgesia prevents these changes during the first stage of labor and mitigates the changes during the second stage of labor.[86-89]

FRC increases after delivery but remains below the prepregnancy volume for 1 to 2 weeks. Oxygen consumption, tidal volume, and minute ventilation remain elevated until at least 6 to 8 weeks after delivery. The alveolar and mixed venous P_{CO_2} rise slowly after delivery and are still slightly below prepregnancy levels at 6 to 8 weeks postpartum.[1]

HEMATOLOGY

Blood Volume

Maternal plasma volume expansion begins as early as 6 weeks' gestation and continues until it reaches a net increase of approximately 50% by 34 weeks' gestation (Table 2-5; Figure 2-6).[90-93] After 34 weeks' gestation, the plasma volume stabilizes or falls slightly. Red blood cell volume falls during the first 8 weeks of pregnancy, increases to the prepregnancy level by 16 weeks, and undergoes a further rise to 30% above the prepregnancy volume at term.[91,93,94] The plasma volume expansion increase exceeds that of the red blood cell volume increase, resulting in the **physiologic anemia of pregnancy**. With volumes expressed in milliliters per kilogram (mL/kg), pregnancy results in an increase in plasma volume from 49 to 67 mL/kg, an increase in total blood volume from 76 to 94 mL/kg, and no change in red cell volume (27 mL/kg).[91] Greater increases in blood volume occur with twin than with singleton pregnancies, and blood volume is positively correlated with the size of the fetus in singleton pregnancies.[92] The physiologic hypervolemia facilitates delivery of nutrients to the fetus, protects the mother from hypotension, and reduces the risks associated with hemorrhage at delivery. The decrease in blood viscosity from the lower hematocrit creates lower resistance to blood flow, which may be an essential component of maintaining the patency of the uteroplacental vascular bed.

The rise in plasma volume results from fetal and maternal hormone production, and several hormonal systems may play a role. Additionally, the expansion of plasma volume may be an adaptive physiologic response that helps maintain blood pressure in the presence of decreased vascular tone.[95,96] The maternal concentrations of estrogen and progesterone increase nearly 100-fold during pregnancy.[1] Estrogens increase plasma renin activity, enhancing renal sodium absorption and water retention via the renin-angiotensin-aldosterone system. Fetal adrenal production of the estrogen precursor dehydroepiandrosterone may be the

TABLE 2-5 Hematologic Parameters at Term Gestation

Parameter	Change* or Actual Measurement
Blood volume	+45%*
Plasma volume	+55%*
Red blood cell volume	+30%*
Hemoglobin	11.6 g/dL
Hematocrit	35.5%

*Relative to nonpregnant state.

From Conklin KA. Maternal physiological adaptations during gestation, labor, and the puerperium. Semin Anesth 1991; 10:221-34.