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Shnider and Levinson's Anesthesia ostetrics

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Fifth Edition



Williams & Wilk

Shnider and Levinson's

Anesthesia for Obstetrics

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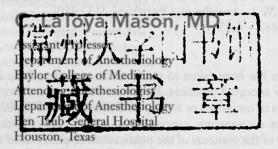
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FOREWORD =

It has been ten years since the publication of the last edition of Shnider and Levinson's Anesthesia for Obstetrics. I am very pleased, as I am sure Dr. Shnider would be, that Dr. Suresh has undertaken the formidable task of updating and

completely revising this textbook.

Reviewing the contents of the book's first edition, published in 1979, and each of the subsequent editions, provides an interesting review of the progress obstetric anesthesiologists have made in providing safe analgesia and anesthesia for women having babies. For example, when the book was first published, anesthesia was the third leading cause of maternal deaths, 45% of cesarean sections were performed under general anesthesia, less than 20% of women in the United States received epidural analgesia for labor, and epidural infusions and neuraxial opioids were not available.

Currently the majority of women having babies in the United States receive epidural anesthesia and many institutions report labor epidural rates between 80 and 90% of vaginal deliveries. The current practice of administering continuous epidural infusions with dilute concentrations of local anesthetics and low-dose opioids has made for much safer anesthesia with significantly greater patient satisfaction. General anesthesia for cesarean section is now a rarity,

to cutting edge modern an sthesia practice.

Moternal mortality has emerged as on the most challenging healthcare issues in the last decade, in addition

in many hospitals less than 5% of all cesarean sections, and typically is limited to patients with one of a few uncommon medical conditions or those requiring extremely emergent delivery. Despite increasing maternal age, with the inevitable increase in pre-existing maternal disease, the marked increase in maternal obesity, and the increase in cesarean section rates, anesthetic-related maternal mortality has fallen dramatically

and is no longer one of the major culprits.

Anesthesia for Obstetrics was intended to be both a basic clinical guide and a reference source for students and practitioners. To accomplish this, great emphasis was placed on presenting in a lucid and concise fashion the various aspects of the pregnant women's modified response to anesthetic drugs, the fetal effects of both maternal physiologic alterations and placental transfer of these drugs, as well as understanding the unique perinatal and obstetric issues. In the fifth edition, Dr. Suresh has continued this approach and has produced an authoritative and comprehensive textbook of obstetric anesthesia. Those who practice and those who receive obstetric anesthesia should benefit greatly.

GERSHON LEVINSON, MD San Francisco, California Shnider and Levinson's Anesthesia for Obstetrics, Fifth Edition is the result of the contribution of several dedicated national and international experts in obstetric anesthesia, who have conceptualized the current evidence-based practice of modern obstetrical anesthesia in this textbook.

Dr. Sol M. Shnider, the first editor of this book, was born in Yorktown, Saskatchewan, Canada. Dr. Shnider received his medical degree from the University of Manitoba and underwent his residency training at the Columbia University in New York. He was the founding member of the Society for Obstetric Anesthesia and Perinatology and the recipient of numerous awards and honors. Indeed, he was one of the pioneers of modern obstetrical anesthesia. The first three editions were edited by Dr. Sol M. Shnider and Dr. Gershon Levinson. The fourth edition, published in 2002, was edited by the late Dr. Samuel C. Hughes, Dr. Gershon Levinson, and Dr. Mark Rosen. I am grateful to both Dr. Levinson and Dr. Mark Rosen for giving us approval to proceed with the

publication of the fifth edition.

The fifth edition is unique due to the contributions of both national and international editors: Dr. Scott Segal (USA), Dr. Roanne Preston (Canada), Dr. Roshan Fernando (UK), and Dr. LaToya Mason (USA), who with their editing style have provided a global perspective to the practice of obstetric anesthesia. Since the first edition in 1979, this book has become the international standard in the field of obstetric anesthesia, with translations in Spanish, French, Portuguese, German, and Japanese; we hope to add other languages including Chinese translation and an electronic version. This textbook will continue, as in the past, to serve as a valuable guide and reference source for the present and next generation of anesthesia trainees, academic and private anesthesia practitioners, and other clinicians. The fifth edition is divided into eleven sections and comprises 50 chapters and 4 appendices. There are other textbooks on obstetric anesthesia that are complete and well written, whereas the focus and organization of the current Sol M. Shnider Anesthesia for Obstetrics fifth edition is in keeping with the vision of Dr. Sol M. Shnider; it reflects evidence-based, best practice approach and complete care of the obstetric patient. This book provides a comprehensive view of the role of the anesthesiologist as a physician responsible for sound judgment and for optimal and best outcomes for mother and baby, a view more in keeping with the approach to cutting-edge modern anesthesia practice.

Maternal mortality has emerged as one of the most challenging healthcare issues in the last decade; in addition, incidence of obesity has reached epidemic proportions in the USA and globally increasing the challenges confronted by the practitioner caring for obstetrical patients. Obstetric anesthesia practice has had an important albeit positive influence on maternal mortality. The contributing authors have made a conscious effort to address new technologies such as ultrasound-guided approach to regional anesthesia, new airway devices, and technologies in advanced airway management. Since the last edition, significant changes and advances have occurred; therefore, almost all the chapters have been

rewritten. There are new chapters that address the challenges confronting the anesthesia practitioner in the United States and globally. These chapters include: "Global Perspective on Obstetric Anesthesia," "Near Misses and Mortality," "Utilization of Crisis Resource Management in Maternal and Neonatal Safety," "Jehovah's Witness: Ethical and Anesthetic-related Issue," "Anesthesia for Vaginal Birth after Cesarean Delivery," "Difficult and Failed Intubation: Strategies, Prevention and Management of Airway-related Catastrophes," and much more. The authors have also focused on postoperative pain management, "Postoperative Multimodal Acute Pain Management: Cesarean and Vaginal Delivery," and "Chronic Pain Issues in the Postpartum Period." Chapters on amniotic fluid embolism, thromboembolism, and hemorrhage have new information. The exciting field of in utero fetal surgery and EXIT procedure has been highlighted in this book. The authors and editors have attempted to present the information with key points at the end of every section in order to facilitate learning; it also makes it easy for the reader to understand, retain, and discuss the information cogently. The book also serves as a useful reference guide to the practicing anesthesiologists in academic centers, tertiary referral centers, and community hospitals.

At the outset, a major investment was made by the publisher of the textbook who recognized that the computer savvy as well as the millennial reader is accustomed to creative graphs and figures in color and therefore opted for enhanced visual aesthetics by having full color figures and graphical presentations throughout the book. We also hope that this book with the color illustrations will not only make it interesting for the reader, but it will also help the reader use the reference and illustrations to prepare lectures, slides, and other creative

illustrative media.

We trust that this textbook will continue the tradition of high quality as in the previous editions. A comprehensive textbook of this depth and scope is not possible without the support and assistance of the family members, colleagues, friends, and support staff who have assisted the authors and editors in preparation of this textbook. I personally wish to thank all the authors and I am very grateful for their dedication and contribution to this illustrious textbook that bears the name of Sol M. Shnider, an Obstetric Anesthesiologist icon. I want to acknowledge our editors and express my utmost gratitude for their valuable time and dedication to the book; they have put in an enormous amount of time to enhance the quality of the contributions. I would like to acknowledge and thank Brian Brown for giving me the opportunity to be the lead senior editor. I also wish to acknowledge the masterful assistance of Tom Conville, Nicole Dernoski, and Ruchira Gupta for their skilled efficiency in organizing and managing the manuscripts, the illustrations, and obtaining permissions and trying to keep everyone on a tight timeline. Finally, I would like to express my sincere gratitude to my husband, my grandson, and my administrative secretary Annette Brieno for their continued support.

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PHYSIOLOGY AND PHARMACOLOGY

CHAPTER

1

Physiologic Changes of Pregnancy

Brenda A. Bucklin Andrea J. Fuller

Unique anatomic and physiologic modifications occur during pregnancy, labor, delivery, and the postpartum period. Every organ system undergoes changes—from the substantial increase in cardiac output observed throughout pregnancy and the peripartum period to the brain's increased sensitivity to anesthetic agents during pregnancy. The increased production of hormones from the ovaries and placenta and release of endorphins further impacts the physiologic changes. A thorough understanding of the anatomical and physiologic changes is a requirement for an anesthesia practitioner caring for women during this period in order to ensure safe and optimal outcomes for mother and baby.

CARDIOVASCULAR CHANGES OF PREGNANCY (TABLE 1-1)

The physiologic alterations of the cardiovascular system function to support fetal growth and metabolism, by significantly increasing uterine perfusion and also to prepare the parturient for blood loss at delivery.

Blood Volume

Both the intravascular and extravascular fluid volumes increase substantially during pregnancy. Much of the average 12.5 kg weight gain during pregnancy is attributed to the increase in the intravascular and extravascular fluid volumes. Significant increases in maternal blood volume occur, with plasma volume increasing 55% from 40 mL/kg to 70 mL/ kg and red blood cell volume increasing approximately 17% from 25 mL/kg to 30 mL/kg (1,2) (Fig. 1-1). This increase in volume begins in the first few weeks of gestation, rises sharply in the second trimester, peaks early in the third trimester and decreases slightly by term (1). The rise in plasma volume is likely achieved by a decreased osmotic threshold for thirst and alterations in arginine vasopressin metabolism (3). A large portion of the increased blood volume perfuses the gravid uterus and 300 to 500 mL of blood may be forced back into the maternal circulation with contractions during labor (2,4). Blood volume returns to prepregnancy values at approximately 7 to 14 days postpartum (2).

Increased red blood cell production is stimulated by a rise in erythropoietin by the second month of gestation (5). The disproportionate increase in plasma volume to red blood cell volume results in the "physiologic anemia of pregnancy" and a normal hemoglobin concentration of 11.6 gm/dL (6). Maternal anemia is present when the hemoglobin and hematocrit fall to less than 11 g/dL or 33% respectively, the most likely cause of which is iron deficiency.

The increase in blood volume during pregnancy prepares the parturient for normal blood loss at delivery. Blood loss is usually less than 500 mL for vaginal delivery and 1,000 mL for cesarean delivery. Hemodynamic changes due to blood loss are usually not observed until the blood loss is greater than 1,500 mL and transfusion is rarely required unless blood loss exceeds this amount. Blood volume decreases to 125% of prepregnancy levels in the first postpartum week and by the sixth to ninth postpartum week there is a more gradual decline in the blood volume to 110% of the prepregnancy level. The hemoglobin and hematocrit also decrease during the initial postpartum period and then gradually increase to prepregnancy levels by the sixth postpartum week.

Central Hemodynamics (Fig. 1-2)

Cardiac output begins to increase around 10 weeks' gestation (7). Serial assessment of maternal cardiac output by impedance cardiography and echocardiography demonstrates that changes in cardiac output start early in gestation with an increase of 35% to 40% by the end of first trimester. The cardiac output continues to increase during pregnancy until 34 weeks when it reaches 50% above prepregnant values and remains stable until term (8,9) (Fig. 1-3). During this time, the percentage of cardiac output devoted to uterine blood flow increases from 5% to 11% (8).

The increase in cardiac output is due to increases in heart rate and stroke volume. The initial increase in cardiac output is due to an increase in the heart rate which starts to occur as early as the fifth week of gestation. The heart rate rises steadily during pregnancy and is elevated approximately 10 to 20 bpm above baseline at term (Fig. 1-4). The hormonal changes and release of estrogens results in an early increase in stroke volume of approximately 20% as early as the fifth to eighth week of gestation. The stroke volume continues to increase by 25% to 30% from the first to third trimester of gestation.

During parturition, further demands are placed on the heart. Additional increases in cardiac output occur during labor and delivery as a result of elevated heart rate and stroke volume

TABLE 1-1 Changes in Cardiovascular System

Variable	Direction of Change	Average Change
Blood volume	1	+35–40%
Plasma volume	1	+50%
Red blood cell volume	1	+20%
Cardiac output	1	+40-50%
Stroke volume	1	+30%
Heart rate	1	+15–20%
Femoral venous pressure	1	+15 mm Hg
Total peripheral resistance	1	–15 mm Hg
Mean arterial blood pressure	\	–15 mm Hg
Systolic blood pressure		–0–15 mm Hg
Diastolic blood pressure	\	–10–20 mm Hg
Central venous pressure	None	No change

Adapted from: Ueland K. Maternal cardiovascular dynamics. VII. Intrapartum blood volume changes. Am J Obstet Gynecol 1976;126:671-677; Pritchard J. Changes in blood volume during pregnancy and delivery. Anesthesiology 1965;26:393-399; Lindheimer M, Davison J. Osmoregulation, the secretion of arginine vasopressin and its metabolism during pregnancy. Eur J Endocrinol 1995;132:133-143; Hendricks C. Hemodynamics of a uterine contraction. Am J Obstet Gynecol 1958;76:968-982; Cotes P, Canning C, Lind T. Changes in serum immunoreactive erythropoietin during the menstrual cycle and normal pregnancy. Br J Obstet Gynaecol 1983;90:304-311; Clark S, Cotton D, Lee W. Central hemodynamic assessment of normal term pregnancy. Am J Obstet Gynecol 1989;161:1439-1442; Flo K, Wilsgaard T, Vartun A, et al. A longitudinal study of the relationship between maternal cardiac output measured by impedance cardiography and uterine artery blood flow in the second half of pregnancy. BJOG 2010;117:837-844; Mabie W, DiSessa T, Crocker L, et al. A longitudinal study of cardiac output in normal human pregnancy. Am J Obstet Gynecol 1994;174:1061-1064; Warner M, Fairhead A, Rawles J, et al. An investigation of the changes in aortic diameter and an evaluation of their effect on Doppler measurement of cardiac output in pregnancy. Int J Obstet Anesth 1996;5:73-78; Ueland K, Hansen J. Maternal cardiovascular dynamics. III. Labor and delivery under local and caudal analgesia. Am J Obstet Gynecol 1969;103:8-18; Ueland K, Hansen J. Maternal cardiovascular dynamics. II. Posture and uterine contractions. Am J Obstet Gynecol 1969;103:1-7; Seth R, Moss A, McNitt S, et al. Long QT syndrome and pregnancy. J Am Coll Cardiol 2007;49:1009-1018.

(10,11). Cardiac output further increases 15% during the latent phase of labor, 30% during the active phase, and 45% during the expulsive stage of labor compared to prelabor values (11). Every uterine contraction results in an increase in cardiac output by an additional 10% to 25% (12). Immediately following cesarean delivery, cardiac index increases by 40% and systemic vascular resistance index (SVRI) decreases by 39%. However, the mean arterial pressure is maintained. These changes persist for approximately 10 minutes but may be present for up to 30 minutes after delivery, and return to baseline values by 2 to 5 days postpartum (13). Hemodynamic changes at delivery are

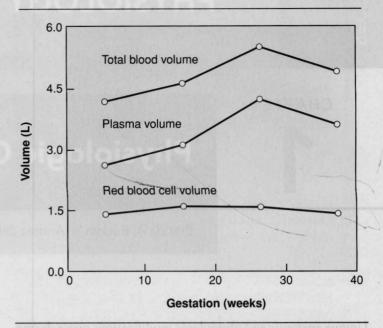


FIGURE 1-1 Changes in intravascular fluid volume (blood volume), plasma volume, and erythrocyte volume during progression of normal pregnancy. The disproportionate increase in plasma volume accounts for the relative anemia of pregnancy. Adapted from: Moir DD, Carty MJ. In: Moir DD, ed. Obstetric Anesthesia and Analgesia. Baltimore, MD: Williams & Wilkins; 1977.

similar regardless of mode of delivery (13,14). While this substantial increase in cardiac work is well tolerated by most parturients, those with cardiac disease who are unable to increase cardiac output by meeting the large demands are often at high-

est risk for complications immediately postpartum.

Systemic vascular resistance decreases from approximately 1,530 dyn s/cm⁵ to 1,210 dyn s/cm⁵ during pregnancy by several mechanisms (7). The production of prostacyclin, a potent vasodilator, is increased during pregnancy (15). Progesterone also has a vasodilator effect on vascular smooth muscle. The low resistance placental circulation is in parallel with the systemic circulation. The sum of two resistances in parallel is less than either alone, which serves to decrease the afterload. The physiologic anemia of pregnancy results in a change in rheology resulting in decreased blood viscosity and improved blood flow, which also decreases afterload (16). Pulmonary vascular resistance (PVR) is also reduced by approximately 30% during pregnancy, presumably by similar mechanisms (7,17). This may have important implications in a patient with a shunt due to a congenital cardiac lesion as the balance between SVR and PVR may be disrupted during pregnancy.

The increase in cardiac output during gestation results in an overall increase in uteroplacental perfusion, renal perfusion, and lower y perfusion. Uterine blood flow increases gradually from 50 mL/min to 700 to 900 mL/min at term with over 90% of the blood flow going to the intervillous space. The remainder of the perfusion goes to the myometrium. At term, the skin blood flow increases by 3- to 4-fold thus resulting in an increase in the skin temperature.

Cardiac Evaluation

During gestation, the diaphragm is shifted upward by the gravid uterus. The result is a leftward shift in the position of the heart that can produce an enlarged appearance of the cardiac silhouette on chest radiograph (Fig. 1-5) as well as axis changes on the ECG. Echocardiographic studies reveal