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Obstetric Anesthesia Handbook

Fifth Edition

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ISBN 978-0-387-88601-5 e-ISBN 978-0-387-88602-2
DOI 10.1007/978-0-387-88602-2
Springer New York Dordrecht Heidelberg London

Library of Congress Control Number: 2009938708

Printed on acid-free paper

Springer is part of Springer Science+Business Media
(www.springer.com)

Preface to the Fifth Edition

It is a great honor for us to join Dr. Sanjay Datta in presenting the fifth edition of *The Obstetric Anesthesia Handbook*. As former students and now colleagues of our mentor, we have attempted to update this classic and widely read book to reflect the constantly evolving face of obstetric anesthesiology. It is astounding to envision the initial writing of this text, nearly 20 years ago, in an era before the ubiquitous availability of online search tools, downloadable papers, and searchable textbooks. The early editions were formed predominantly out of Dr. Datta's personal command of the field, its literature, and his personal teaching files. Many benefited from the depth and breadth of his wisdom presented in the original *Handbook*, and it is now our privilege to help pass on this work to the next generation of residents, fellows, and obstetric anesthesia practitioners around the world. We have attempted to retain the compact style of the original single-author version, while adding some newer material, reorganizing some chapters to enhance their utility, updating references, and revising some figures and appendices. We hope you will find it as helpful in your practice as it is in ours.

Scott Segal, MD
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Preface to the First Edition

One of the major "perks" of an academic anesthesiologist is the opportunity to interact with residents and fellows. Most of them are bright, energetic, and hardworking individuals. During my professional life, I enjoyed my dealings with this special group, and their enthusiasm in obstetric anesthesia is the basis for the germination of this project.

Parturients are different from their nonpregnant counterparts in various ways. Their expectations, demands, and needs make obstetric anesthesia more challenging and also gratifying. This book basically deals with these aspects at a level that I found stimulating to the residents as well as fellows.

There are 19 chapters in this book that address the various aspects of maternal physiology, perinatal pharmacology, and, ultimately, anesthetic techniques for different procedures; my hope is that this is done in a concise manner. Every effort has been made to discuss the controversial issues of anesthetic techniques covering the majority of problems that might arise.

It is my deepest desire that this book be both helpful and stimulating to residents, fellows, and my contemporaries. To this end, periodic updates of this manual will be made to keep its contents current and to address topics of interest and controversy.

I want to express my gratitude to a few individuals without whom this project would remain incomplete. My thanks are directed to Dr. Knapp for his very eloquently expressed views regarding medicolegal aspects of obstetric anesthesia. My special thanks go to Ms. Vehring, whose editorial assistance was extremely necessary. Finally, I must also express my gratitude to Ms. Racke for her graphic illustrations and Ms. Russo and Ms. Spelling for secretarial help.

Sanjay Datta, MD, F.F.A.R.C.S. (Eng)

Contents

1
Maternal Physiological Changes During Pregnancy, Labor, and the Postpartum Period

Parturients undergo remarkable changes during pregnancy, labor, and the immediate postpartum period that can directly affect anesthetic techniques; hence a broad knowledge of these changes is essential for optimum management of these women.

Changes in the Hematological System

Maternal blood volume increases during pregnancy, and this involves an increase in plasma volume as well as in red cell and white cell volumes.[1] *The plasma volume increases by 40–50%, whereas the red cell volume increases by only 15–20%, which causes a "physiological anemia of pregnancy"* (normal hemoglobin 12 g/dL; hematocrit 35).[2] Because of this hemodilution, blood viscosity decreases by approximately 20%. The exact mechanism of this increase in plasma volume is unknown. However, several mediators such as

S. Datta et al., *Obstetric Anesthesia Handbook*,
DOI 10.1007/978-0-387-88602-2_1,

renin–angiotensin–aldosterone, atrial natriuretic peptide, estrogen, progesterone, and nitric oxide may be involved. The most likely hypothesis attributes the increase to an "underfill" state caused by initial vasodilation, which stimulates hormones such as renin, angiotensin, and aldosterone to cause fluid retention.[3] Alternatively, some have proposed an "overfill" state characterized by an early increase in sodium retention (due to an increase in mineralcorticoids) that leads to fluid retention, causing an increase in blood volume, followed subsequently by vasodilation.

Blood volume increases further during labor, as uterine contractions squeeze blood out of the intervillious space and into the central circulation. After delivery, involution of the uterus and termination of placental circulation causes an autotransfusion of approximately 500 mL of blood.

Levels of clotting factors I, VII, VIII, IX, X, and XII and fibrinogen are elevated during pregnancy as well. Platelet production is increased, thrombopoietin levels are increased,[4] and platelet aggregation measured in vitro is likewise increased; indices of platelet destruction are also increased. The overall effect of these changes is variable, but prospective observations have reported a statistically significant fall in platelet count as pregnancy progresses, with 7.6% of women having a count less than 150,000 and 1% less than 100,000 at term.[5] Endogenous anticoagulants, such as protein S, are decreased in normal pregnancy and there is acquired resistance to activated protein C during pregnancy, adding to the prothrombotic state. Fibrinolysis is impaired in normal pregnancy due to placentally derived plasminogen activator inhibitor (PAI), but returns to normal following delivery of the placenta. Overall indices of coagulation indicate that normal pregnancy is a hypercoagulable state.[6]

Clinical Implications

Increased blood volume and enhanced coagulation serve several important functions: (1) the increased circulatory needs of the enlarging uterus and growing fetus and placenta are met and (2) the parturient is protected from bleeding at the

time of delivery. Anesthesiologists should consider the enlarged blood volume when making decisions on fluid and blood replacement in the peripartum period. Parturients become hypercoagulable as gestation progresses and are at increased risk of thromboembolism. After a rapid mobilization and diuresis of some fluid in the first few postpartum days, blood volume slowly returns to normal over 8 weeks.

Changes in the Cardiovascular System

An increase in cardiac output is one of the most important changes of pregnancy. *Cardiac output increases by 30–40% during pregnancy, and the maximum increase is attained around 24 weeks' gestation.*[7] The increase in heart rate occurs first (by the end of the first month of pregnancy) and plateaus at an increase of 10–15 beats per minute by 28–32 weeks' gestation. Stroke volume increases by mid-first trimester and progressively increases through the second trimester. Echocardiography demonstrates increases in end-diastolic chamber size and total left ventricular wall thickness but no change in end-systolic volume, so ejection fraction is increased. Cardiac output can vary depending on the uterine size and maternal position at the time of measurement. The enlarged gravid uterus can cause aortocaval compression and reduced cardiac filling while the pregnant woman is in the supine position (Fig. 1-1), leading to an underestimation of cardiac function. Normal pregnant women exhibit a marked increase in femoral venous and inferior vena caval pressures. Collateral vessels maintain atrial filling but lead to engorgement of veins, including the epidural venous (Batson's) plexus.

Filling pressures (CVP, pulmonary capillary wedge pressure, LV end-diastolic pressure) do not change despite the increased cardiac dimensions, due to myocardial remodeling during gestation. Systemic vascular resistance is decreased approximately 20%. Blood pressure never increases in normal pregnancy, and systolic and diastolic blood pressures decrease by approximately 8 and 20%, respectively, on average.[9] Pregnancy hormones (estradiol and progesterone), prostacyclin, and nitric

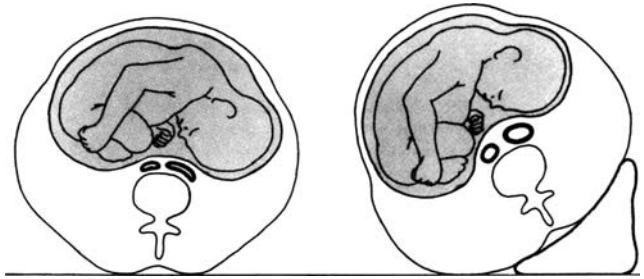

Figure 1–1. Aortocaval compression. (From Chestnut.[8] Used with permission from Elsevier.)

oxide all may play a role in the reduction in blood pressure observed despite an increase in cardiac output.

Cardiac output increases further during labor, up to 50% higher than pre-labor values, although effective analgesia can attenuate some of this increase. In the immediate postpartum period, cardiac output increases maximally and can rise 80% above pre-labor values and approximately 150% above nonpregnant measurements. An increase in stroke volume as well as in heart rate maintains the increased cardiac output.

The heart is displaced to the left and upward during pregnancy because of the progressive elevation of the diaphragm by the gravid uterus. The electrocardiogram of normal parturients may include (1) sinus tachycardia or benign dysrhythmias, (2) depressed ST segments and flattened T waves, (3) left axis deviation, and (4) left ventricular hypertrophy. Auscultation frequently reveals a systolic murmur of tricuspid or mitral regurgitation, and a third or fourth heart sound.

Cardiac output, heart rate, and stroke volume decrease to pre-labor values 24–72 h postpartum and return to nonpregnant levels within 6–8 weeks after delivery.[10]

Clinical Implications

An increased cardiac output might not be well tolerated by pregnant women with valvular heart disease (e.g., aortic or mitral stenosis) or coronary arterial disease. *Decompensation in*

myocardial function can develop at 24 weeks' gestation, during labor, and especially immediately after delivery.

Engorgement of the epidural venous plexus increases the risk of intravascular catheter placement in pregnant women; direct connection of the azygos system to the heart as well as brain also increases the risks of local anesthetic cardiovascular and central nervous system toxicity.

Changes in the Respiratory System

Changes in respiratory parameters start as early as the fourth week of gestation. Minute ventilation is increased at term by about 50% above nonpregnant values. The increase in minute ventilation is mainly due to an increase in tidal volume (40%) and, to a lesser extent, an increase in the respiratory rate (15%).[11] Alveolar ventilation is greatly increased as the tidal volume increases without any change in the ratio of dead space to tidal volume (V_D/V_T). At term PCO_2 is decreased to 32–35 mmHg, although renal excretion of bicarbonate keeps arterial pH normal. Increased progesterone concentrations during pregnancy likely stimulate increased respiration, even before an increase in metabolic rate.[12] Oxygen consumption and carbon dioxide production increase by approximately 60% over prepregnant values. PaO_2 is increased in early pregnancy due to a decrease in PCO_2.

Functional residual capacity, expiratory reserve volume, and residual volume are decreased at term (Fig. 1-2). These changes are related to the cephalad displacement of the diaphragm by the large gravid uterus. Inspiratory capacity increases somewhat because of increase in tidal volume and inspiratory reserve volume. Vital capacity is unchanged. Total lung capacity is only slightly reduced because chest circumference increases. Closing capacity (CC) does not change, but the reduction in FRC contributes to a tendency toward earlier desaturation, as lung volume more easily falls below CC.

Anatomic changes also accompany pregnancy. The respiratory mucous membranes become vascular, edematous, and friable. The voice may deepen and there is a progressive increase in the Mallampati score during gestation and labor.[13]

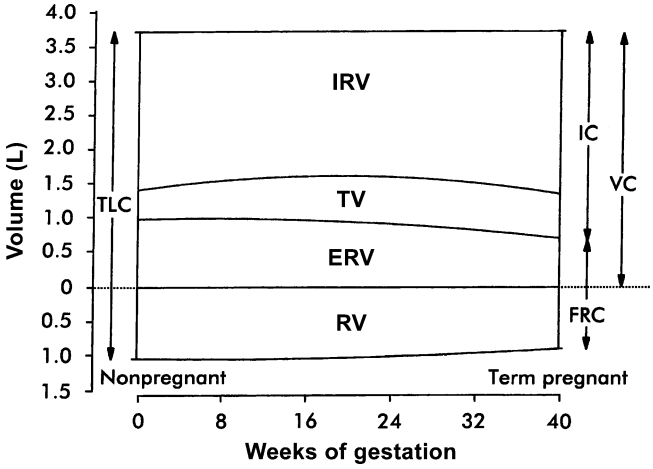

Figure 1–2. Pulmonary volume and capacity changes in pregnancy. (From Chestnut.[8] Used with permission from Elsevier.)

In labor, minute volume further increases in the absence of pain relief, and PCO_2 may decrease to 17 mmHg. Opioids somewhat attenuate this change, but epidural analgesia does so more completely. In the second stage, maternal expulsive efforts increase ventilation, even in the presence of effective regional analgesia.[14,15]

FRC changes return to normal 1–2 weeks postpartum, accompanying the reduction in uterine size. All other respiratory parameters return to nonpregnant values within 6–12 weeks postpartum.

Clinical Implications

Decreased FRC as well as increased oxygen consumption can cause a rapid development of maternal hypoxemia during apnea. Decreased FRC decreases the time for denitrogenation and speeds the uptake of inhaled anesthetics.

Because of the increased edema, vascularity, and friability of the mucous membrane, one should try to avoid nasal

intubation in pregnant women, and smaller endotracheal tubes should be used for oral intubation.

Maternal alkalosis associated with decreased $PaCO_2$ values due to hyperventilation as a result of labor pain can cause fetal acidosis because of (1) decreased uteroplacental perfusion due to uterine vasoconstriction and (2) shifting of the maternal oxygen dissociation curve to the left.

Changes in the Gastrointestinal System

The enlarging uterus displaces and disrupts the lower esophageal sphincter, and progesterone relaxes this high-pressure zone, causing a progressive increase in the incidence of heartburn (up to 80% at term). An increase in gastric pressure due to mechanical compression also contributes to heartburn. Despite the prevalence of this symptom, total acid production is decreased (although placental production of gastrin increases the total concentration of this hormone).

Gastric emptying is normal throughout pregnancy, as measured by acetaminophen absorption, ultrasound, dye-dilution, and radiographic techniques. Intestinal transit time is increased, leading to frequent complaints of constipation in pregnant women. Studies of gastric pH and volume in pregnant and nonpregnant women show no differences in the proportion of women meeting "at risk" criteria (pH <2.5, volume >25 ml[16]) for pulmonary aspiration of gastric contents.[8]

Labor fundamentally alters this pattern. Gastric emptying time is significantly slower during labor and hence gastric volume is increased. Opioids administered by any route will further increase the gastric emptying time. Studies demonstrate solid food in the stomachs of laboring women even after 18 h of fasting.[17] Gastric emptying remains abnormal on the first postpartum day but returns to normal on the second day.

Hepatic transaminases, bilirubin, and LDH are increased slightly in pregnancy. Alkaline phosphatase is markedly increased (2–4 fold), but due to placental production, not hepatic changes. *Serum cholinesterase activity is reduced 24% before delivery and reaches a nadir (33% reduction) on the third postpartum day*[14] (Fig. 1-3). Approximately 11% of post-