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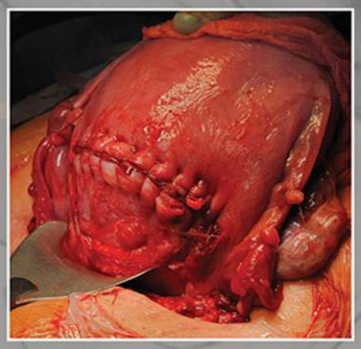

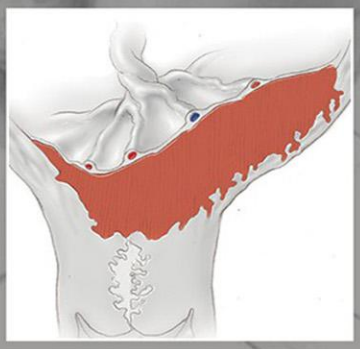

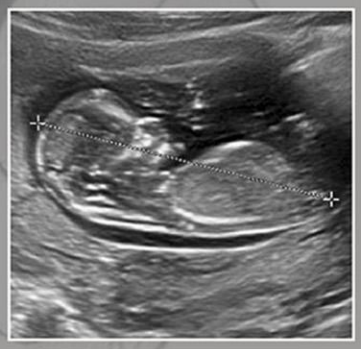

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Williams OBSTETRICS

25TH EDITION

F. Gary Cunningham
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DEDICATION

To our mentors, who inspire us to strive for excellence in obstetrics,
To our colleagues, who are superb role models for obstetricians and gynecologists,
To our students and residents, who challenge us to be better teachers each day,
To our fellows, who dare us to think more boldly,
To our nurses, who encourage us to place patient needs first,
To our support staff, who allow us to respond efficiently in the face of emergencies, and
To our families, whose love and support make our endeavors possible.

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PREFACE

We celebrate this 25th edition of *Williams Obstetrics* with great appreciation for the insight and expertise that the early editors brought to this textbook. To pay tribute to the first author, J. Whitridge Williams, we begin each chapter with a passage from his 1st edition that complements the topic. During this selection process, we were inspired by the strides that modern obstetrics has made since that edition in 1903. Similarly, we were humbled by some of the classic challenges that still persist. Preterm labor, preeclampsia, and infections are some examples. That said, many of these advances were derived from rigorous, evidence-based research. And, we acknowledge and support the power of this academic ideal to further our specialty in the decades to come.

For this 25th edition, we continue to present the detailed staples of basic obstetrics such as maternal anatomy and physiology, preconceptional and prenatal care, labor, delivery, and the puerperium. These accompany detailed discussions of obstetrical complications exemplified by preterm labor, hemorrhage, hypertension, and many more. To emphasize the "M" in Maternal–Fetal Medicine, we continue to iterate the many medical and surgical disorders that can complicate pregnancy. And, our second patient—the fetus—has accrued especial attention with an entire section devoted to diagnosis and treatment of fetal disorders. For all of these, we once again emphasize the science-based underpinnings of clinical obstetrics with special emphasis on biochemical and physiological principles. As was the hallmark of previous editions, these dovetail with descriptions of evidence-based practices. Expert clinical pearls add depth to these discussions and are written for busy practitioners—those "in the trenches."

To accomplish these goals, the text has been updated with more than 3000 new literature citations through 2017. Many of the nearly 900 figures are new, and these graphs, sonograms, magnetic resonance images, photographs, photomicrographs, and data graphs are almost all in vivid color. Much of the original artwork was rendered by our own medical illustrators.

Also, as before, we continue to incorporate contemporaneous guidelines from professional and academic organizations such as the American College of Obstetricians and Gynecologists, the Society for Maternal–Fetal Medicine, the National Institutes of Health and the National Institute for Child Health and Human Development, the Centers for Disease Control and Prevention, and other authoritative sources. Many of these data are distilled into nearly 100 tables, in which information has been arranged in an easy read-and-use format. In addition, several diagnostic and management algorithms are available to quickly guide practitioners. Although we strive to cite numerous sources and provide multiple evidence-based options for such management schemes, we also include our own clinical experiences drawn from the large obstetrical service at Parkland Hospital. We are convinced that these are disciplined examples of evidence-based obstetrics but quickly acknowledge that they do not constitute the sole method of management.

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ACKNOWLEDGMENTS

During the creation and production of this textbook, we were fortunate to have the assistance and support of countless talented professionals both within and outside the Department of Obstetrics and Gynecology. To begin, we acknowledge that an undertaking of this magnitude would not be possible without the unwavering support provided by Dr. Barry Schwarz, Vice-Chairman, whose financial and academic endorsement has been essential.

This 25th edition shows a notable absence of three colleagues who provided valuable editorial assistance for prior editions of *Williams Obstetrics*. Colleagues from the University of Texas Southwestern Medical Center include Dr. George Wendel, Jr.—associate editor for the 22nd and 23rd editions—who has now assumed the important role of Executive Director of the American Board of Obstetrics and Gynecology. Dr. Jeanne Sheffield, with her especial expertise in obstetrical and perinatal infections, has left Dallas and is now the Division Director of Maternal–Fetal Medicine at Johns Hopkins University School of Medicine. From the University of Alabama at Birmingham, Dr. John Hauth, who served as an editor for the 21st through 23rd editions, provided valuable contributions to chapters on chronic hypertension, preterm labor, and labor induction, which have endured in updated forms in this edition.

We are especially grateful for the contributions of our two returning Associate Editors. Dr. Mala Mahendroo is a talented basic scientist who continues to perform a magnificent job of providing a coherent translational version of basic science aspects of human reproduction. Dr. Diane Twickler—the consummate radiologist—has been an invaluable mentor for our residents, fellows, and faculty. She adds her fantastic experiences and extensive knowledge regarding clinical and technological advances related to fetal and maternal imaging to add considerable depth to this textbook. Dr. Seth Hawkins served us well as an Associate Editor in this edition and brought additional strengths to the areas of clinical and academic Maternal–Fetal Medicine. His rigorous analysis of evidence-based data on topics of maternal physiology, fetal-growth disorders, obesity, liver disease, and labor induction has added new perspectives to these chapters.

To add academic breadths to our endeavor, we have enlisted new Contributing Editors—all from UT Southwestern Medical Center—each of whom has expertise in important areas of maternal and perinatal medicine. From the Division of Maternal–Fetal Medicine, Dr. C. Edward Wells adds his extensive clinical experience and his incredible skills with prior cesarean delivery and obstetrical sonography. Dr. April Bailey, with joint appointments in the Departments of Radiology and Obstetrics and Gynecology, shared her tremendous knowledge regarding fetal and maternal imaging with sonography, radiography, computed tomography, and magnetic resonance techniques. Dr. David Nelson brings strong clinical knowledge regarding preterm labor, stillbirth, management of obstetrical hemorrhage, psychiatric disorders in pregnancy, and multifetal gestation. From the Department of Anesthesia, Dr. Weike Tao provided academic insight and clinical mastery in obstetrical anesthesia. Similarly, Dr. Erica Grant graciously and skillfully advanced the discussion of this topic. Dr. Myra Wyckoff, from the Department of Pediatrics, contributed greatly to chapters regarding the term and preterm newborn. Her expertise both in normal care and in treatment for the more vulnerable neonates has greatly strengthened the evidence-based content of these chapters. In toto, the strength of each contributor has added to create the sum total of our academic endeavor.

In constructing such an expansive academic compilation, the expertise of many colleagues was needed to add vital and contemporaneous information. It was indeed fortuitous for us to have access to a pantheon of collaborators from here and from other academic medical centers. From our own Department of Obstetrics and Gynecology, our nationally known pelvic anatomist, Dr. Marlene Corton, prepared graphic masterpieces for the anatomy chapter. Dr. Elysia Moschos contributed a number of sonographic images of early pregnancy and uterine malformations. Drs. Claudia Werner and William Griffith lent valuable insight into the management of cervical dysplasia. Dr. Emily Adhikari was an invaluable source in the construction of the chapters on maternal and perinatal infections. Finally, clinical photographs were contributed by many faculty and fellows, who include Drs. Patricia Santiago-Muñoz, Julie Lo, Elaine Duryea, Jamie Morgan, Judith Head, David Rogers, Kimberly Spoonts, and Emily Adhikari. From the Department of Radiology, Drs. Michael Landy, Jeffrey Pruitt, and Douglas Sims added insights and provided computed tomographic and magnetic resonance images. From the Department of Pathology, Dr. Kelley Carrick generously donated exemplary photomicrographs. Dr. Kathleen Wilson, director of the cytogenomic microarray analysis laboratory, graciously assisted us in updating our cytogenomic nomenclature.

We are also indebted to contributions made by our national and international colleagues. Experts in placental pathology who shared their expertise and images include Drs. Kurt Benirschke, Ona Marie Faye-Petersen, Mandolin Ziadie, Michael Conner, Brian Levenson, Jaya George, and Erika Fong. Input for hypertensive disorders was provided by Drs. John Hauth, Marshall Lindheimer, and Gerda Zeeman; for operative vaginal delivery by Dr. Edward Yeomans; and seminal images were contributed by Drs. Kevin Doody, Timothy Crombleholme, Michael Zaretsky, Togas Tulandi, Edward Lammer, Charles Read, Frederick Elder, April Bleich, Laura Greer, and Roxane Holt.

In addition to these contributors, we relied heavily on our colleagues in the Division of Maternal–Fetal Medicine. These professionals, in addition to providing expert content, graciously assisted us by covering clinical duties when writing and editing were especially time consuming. These include Drs.

Scott Roberts, Oscar Andujo, Vanessa Rogers, Charles Brown, Julie Lo, Robyn Horsager, Patricia Santiago-Muñoz, Shivani Patel, Elaine Duryea, Jamie Morgan, Morris Bryant, Shena Dillon, Denisse Holcomb, Robert Stewart, Stephan Shivvers, Ashley Zink, and Mark Peters. In addition, warm thanks go to our Residency Director, Dr. Vanessa Rogers, and her Associate Program Director, Dr. Stephanie Chang, who have created a nurturing environment for our residents to flourish. Similarly, our Maternal–Fetal Medicine (MFM) Division Associate Fellowship Director, Dr. Charles Brown, has aided our work through his talented mentoring of our MFM fellows.

We also emphasize that production of *Williams Obstetrics* would not be feasible without the help of our Maternal–Fetal Medicine fellows and our residents in Obstetrics and Gynecology. Their insatiable curiosity serves to energize us to find new and effective ways to convey age-old truths, new data, and cutting-edge concepts. Their logical and critical questions lead us to weaknesses in the text, and thereby, always help us to improve our work. In addition, we sincerely thank them for their vigilance in capturing photographs of spectacular examples of both obstetrical pathology and normal findings. For example, included in this edition are photographs contributed by Drs. Devin Macias, Maureen Flowers, Paul Slocum, Jonathan Willms, Stacey Thomas, Kara Ehlers, Nidhi Shah, Abel Moron, Angela Walker, and Elizabeth Mosier.

This edition is heavily populated with seminal examples of sonographic findings. We are grateful for the mentorship and talent of Drs. Diane Twickler and April Bailey; Mary Gibbs, RDMS; Rafael Levy, RDMS; Michael Davidson, RDMS; and the many talented sonographers at Parkland Hospital.

Thanks to generous funding from McGraw-Hill Education, this 25th edition now contains more than 200 color illustrations. Most of these were crafted by several skilled medical illustrators who include Ms. Marie Sena, Ms. Erin Frederickson, Mr. Jordan Pietz, Ms. SangEun Cha, and Ms. Jennifer Hulsey. All of these talented artists trained here at UT Southwestern under the tutelage of Mr. Lewis Calver. Additional artistic support came from Mr. Jason McAlexander and Ms. Suzanne Ghuzzi, of MPS North America LLC, who provided the full-color graphs and line art used to enhance this edition. Their team tirelessly coordinated efforts between author and artist and graciously accommodated our numerous changes and tweaks.

Production of the 5000-page manuscript would not have been possible without a dedicated team to bring these efforts together. Once again, we are deeply indebted to Ms. Dawn Wilson and Ms. Melinda Epstein for their untiring efforts with manuscript production. Ms. Mercedes Salinas also provided excellent, conscientious manuscript assistance. Information technology support was provided by the very knowledgeable and responsive Mr. Charles Richards and Mr. Thomas Ames. For these and many more that go unnamed, we could not have done our job without their expertise.

It again has been a privilege and a pleasure to work with the dedicated professionals from McGraw-Hill Education. Mr. Andrew Moyer has brought his considerable intelligence, unwavering work ethic, and creativity to this edition of *Williams Obstetrics*. His dedication to creating the best textbook possible equaled our efforts, and we are in awe of his productive, gracious style. His assistant, Ms. Jessica Gonzalez, provided professional, timely, and ever-sunny aid. Mr. Richard Ruzycka served as production supervisor for this edition of the textbook. He skillfully kept our project on track through an array of potential hurdles. Last, we have had the pleasure to work with Mr. Armen Ovsepyan in coordinating the artwork for many of our editions. His organization and efficiency are unrivaled.

Our text took its final shape under the watchful care of our compositors at Aptara, Inc. We thank Ms. Indu Jawwad for her talents in graciously and masterfully coordinating and overseeing composition. Her dedicated attention to detail and organization were vital to completion of our project. Also, at Aptara, Mr. Mahender Singh performed a crucial task of quality control. He also assisted, along with Mr. Surendra Mohan Gupta and Mr. Anil Varghese, in creating beautiful chapter layouts to highlight our content aesthetically and informatively. This edition's chapters, for the first time, were posted and available online for use prior to print publication. We thank Mr. Braj Bhushan and Mr. Ashish Kumar Sharma for preparing this content so brilliantly. Special thanks go to Ms. Kristin Landon. As copyeditor for now several editions of both *Williams Obstetrics* and *Williams Gynecology*, Kristin has added precision and clarity to our efforts. Her endurance and pleasant professionalism through many challenging chapters has made our text better.

Finally—but certainly not last—we acknowledge our significant debt to the women who have entrusted themselves and their unborn children to us for obstetrical care. The clinical expertise and many graphic illustrations presented in this text would not have been possible without their collaborative spirit to help us advance obstetrical knowledge. We also offer enthusiastic and heartfelt appreciation to our families and friends. Without their patience, generosity, love, and encouragement, this task would have been impossible.

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SECTION 1

OVERVIEW

CHAPTER 1

Overview of Obstetrics

VITAL STATISTICS 2
PREGNANCY RATES IN THE UNITED STATES 4
MEASURES OF OBSTETRICAL CARE 4
TIMELY TOPICS IN OBSTETRICS 6

> *In the following pages I have attempted to set forth, as briefly as seemed to be consistent with thoroughness, the scientific basis for and the practical application of the obstetrical art. At the same time, I have endeavored to present the more practical aspects of obstetrics in such a manner as to be of direct service to the obstetrician at the bedside.*
>
> —J. Whitridge Williams (1903)

So reads the introduction to Williams' first edition of this textbook, *Obstetrics—A Text-Book for the Use of Students and Practitioners.* In this 25th edition, we strive to follow the tenets described by Williams. And, each chapter begins with a quote from his original textbook.

The science and clinical practice of obstetrics is concerned with human reproduction. Through quality perinatal care, the specialty promotes the health and well-being of the pregnant woman and her fetus. Such care entails appropriate recognition and treatment of complications, supervision of labor and delivery, initial care of the newborn, and management of the puerperium. Postpartum care promotes health and provides family planning options.

The importance of obstetrics is reflected by the use of maternal and neonatal outcomes as an index of the quality of health and life among nations. Intuitively, indices that reflect poor obstetrical and perinatal outcomes would lead to the assumption that medical care for the entire population is lacking. With those thoughts, we now provide a synopsis of the current state of maternal and newborn health in the United States as it relates to obstetrics.

VITAL STATISTICS

The National Vital Statistics System of the United States is the oldest and most successful example of intergovernmental data sharing in public health. This agency collects statistics through vital registration systems that operate in various jurisdictions. These systems are legally responsible for registration of births, fetal deaths, deaths, marriages, and divorces. Legal authority resides individually with the 50 states; two regions—the District of Columbia and New York City; and five territories—American Samoa, Guam, the Northern Mariana Islands, Puerto Rico, and the Virgin Islands.

The standard birth certificate was revised in 1989 to include more information on medical and lifestyle risk factors and obstetrical practices. In 2003, an extensively revised Standard Certificate of Live Birth was implemented in the United States. The enhanced data categories and specific examples of each are summarized in Table 1-1. By 2013, 35 states had implemented the revised birth certificate representing 76 percent of all births (MacDorman, 2015). Importantly, the 2003 version of the population death certificate contains a pregnancy checkbox to eventually be implemented by all states (Joseph, 2017).

Definitions

The uniform use of standard definitions is encouraged by the World Health Organization as well as the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists (2017). Such uniformity allows data comparison not only between states or regions of the country but also between countries. Still, not all definitions are uniformly

TABLE 1-1. General Categories of New Information Added to the 2003 Revision of the Birth Certificate

Risk factors in pregnancy—Examples: prior preterm birth, prior eclampsia
Obstetrical procedures—Examples: tocolysis, cerclage, external cephalic version
Labor—Examples: noncephalic presentation, glucocorticoids for fetal lung maturation, antibiotics during labor
Delivery—Examples: unsuccessful operative vaginal delivery, trial of labor with prior cesarean delivery
Newborn—Examples: assisted ventilation, surfactant therapy, congenital anomalies

applied. For example, the American College of Obstetricians and Gynecologists recommends that reporting include all fetuses and neonates born weighing at minimum 500 g, whether alive or dead. But, not all states follow this recommendation. Specifically, 28 states stipulate that fetal deaths beginning at 20 weeks' gestation should be recorded as such; eight states report all products of conception as fetal deaths; and still others use a minimum birthweight of 350 g, 400 g, or 500 g to define fetal death. To further the confusion, the National Vital Statistics Reports tabulates fetal deaths from gestations that are 20 weeks or older (Centers for Disease Control and Prevention, 2016). This is problematic because the 50th percentile for fetal weight at 20 weeks approximates 325 to 350 g—considerably less than the 500-g definition. Indeed, a birthweight of 500 g corresponds closely with the 50th percentile for 22 weeks' gestation.

Definitions recommended by the National Center for Health Statistics and the Centers for Disease Control and Prevention are as follows:

Perinatal period. The interval between the birth of a neonate born after 20 weeks' gestation and the 28 completed days after that birth. When perinatal rates are based on birthweight, rather than gestational age, it is recommended that the perinatal period be defined as commencing at the birth of a 500-g neonate.

Birth. The complete expulsion or extraction from the mother of a fetus after 20 weeks' gestation. As described above, in the absence of accurate dating criteria, fetuses weighing <500 g are usually not considered as births but rather are termed *abortuses* for purposes of vital statistics.

Birthweight. The weight of a neonate determined immediately after delivery or as soon thereafter as feasible. It should be expressed to the nearest gram.

Birth rate. The number of live births per 1000 population.

Fertility rate. The number of live births per 1000 females aged 15 through 44 years.

Live birth. The term used to record a birth whenever the newborn at or sometime after birth breathes spontaneously or shows any other sign of life such as a heartbeat or definite spontaneous movement of voluntary muscles. Heartbeats are distinguished from transient cardiac contractions, and respirations are differentiated from fleeting respiratory efforts or gasps.

Stillbirth or fetal death. The absence of signs of life at or after birth.

Early neonatal death. Death of a liveborn neonate during the first 7 days after birth.

Late neonatal death. Death after 7 days but before 29 days.

Stillbirth rate or fetal death rate. The number of stillborn neonates per 1000 neonates born, including live births and stillbirths.

Neonatal mortality rate. The number of neonatal deaths per 1000 live births.

Perinatal mortality rate. The number of stillbirths plus neonatal deaths per 1000 total births.

Infant death. All deaths of liveborn infants from birth through 12 months of age.

Infant mortality rate. The number of infant deaths per 1000 live births.

Low birthweight. A newborn whose weight is <2500 g.

Very low birthweight. A newborn whose weight is <1500 g.

Extremely low birthweight. A newborn whose weight is <1000 g.

Term neonate. A neonate born any time after 37 completed weeks of gestation and up until 42 completed weeks of gestation (260 to 294 days). The American College of Obstetricians and Gynecologists (2016b) and Society for Maternal–Fetal Medicine endorse and encourage specific gestational age designations. *Early term* refers to neonates born at 37 completed weeks up to $38^{6/7}$ weeks. *Full term* denotes those born at 39 completed weeks up to $40^{6/7}$ weeks. Last, *late term* describes neonates born at 41 completed weeks up to $41^{6/7}$ weeks.

Preterm neonate. A neonate born before 37 completed weeks (the 259th day). A neonate born before 34 completed weeks is early preterm, whereas a neonate born between 34 and 36 completed weeks is late preterm.

Postterm neonate. A neonate born anytime after completion of the 42nd week, beginning with day 295.

Abortus. A fetus or embryo removed or expelled from the uterus during the first half of gestation—20 weeks or less, or in the absence of accurate dating criteria, born weighing <500 g.

Induced termination of pregnancy. The purposeful interruption of an intrauterine pregnancy that has the intention other than to produce a liveborn neonate and that does not result in a live birth. This definition excludes retention of products of conception following fetal death.

Direct maternal death. The death of the mother that results from obstetrical complications of pregnancy, labor, or the puerperium and from interventions, omissions, incorrect treatment, or a chain of events resulting from any of these factors. An example is maternal death from exsanguination after uterine rupture.

Indirect maternal death. A maternal death that is not directly due to an obstetrical cause. Death results from previously existing disease or a disease developing during pregnancy, labor, or the puerperium that was aggravated by maternal physiological adaptation to pregnancy. An example is maternal death from complications of mitral valve stenosis.

Nonmaternal death. Death of the mother that results from accidental or incidental causes not related to pregnancy. An example is death from an automobile accident or concurrent malignancy.

Maternal mortality ratio. The number of maternal deaths that result from the reproductive process per 100,000 live births. Used more commonly, but less accurately, are the terms *maternal mortality rate* or *maternal death rate*. The term *ratio* is more accurate because it includes in the numerator the number of deaths regardless of pregnancy outcome—for example, live births, stillbirths, and ectopic pregnancies—whereas the denominator includes the number of live births.

Pregnancy-associated death. The death of a woman, from any cause, while pregnant or within 1 calendar year of termination of pregnancy, regardless of the duration and the site of pregnancy.

Pregnancy-related death. A pregnancy-associated death that results from: (1) complications of pregnancy itself, (2) the chain of events initiated by pregnancy that led to death, or (3) aggravation of an unrelated condition by the physiological or pharmacological effects of pregnancy and that subsequently caused death.

PREGNANCY RATES IN THE UNITED STATES

According to the Centers for Disease Control and Prevention (CDC), the fertility rate of women aged 15 to 44 years in the United States in 2015 was 62.5 live births per 1000 women (Martin, 2017). This rate began slowly trending downward in 1990 and has now dropped below that for replacement births. This indicates a population decline (Hamilton, 2012). There were 3.98 million births in 2015, and this constituted the lowest birth rate ever recorded for the United States—12.3 per 1000 population. The birth rate decreased for all major ethnic and racial groups, for adolescents and unmarried women, and for those aged 20 to 24 years. For women older than 30 years, the birth rate rose slightly. Almost half of newborns in 2010 in the United States were minorities: Hispanic—25 percent, African-American—14 percent, and Asian—4 percent (Frey, 2011).

The total number of pregnancies and their outcomes in 2015 are shown in Table 1-2. According to the Guttmacher Institute (2016b), 45 percent of births in the United States are unintended at the time of conception. Importantly, the overall proportion of unintended births has declined only slightly since 2001. Unmarried women, black women, and women with less education or income are more likely to have unplanned pregnancies.

In Table 1-2, induced abortion information derives from CDC abortion surveillance data from 45 states combined with Guttmacher Institute data on induced abortion. These data have been collected beginning in 1976. Since *Roe v. Wade* legalization of abortion, more than 46 million American women have chosen legalized abortions. As discussed later, this provides a compelling argument for easily accessible family planning.

TABLE 1-2. Total Pregnancies and Outcomes in the United States in 2015

Outcome	Number or Percent
Births	3,988,076
Cesarean deliveries	32.2%
Preterm births (<37 weeks)	9.5%
Low birthweight (<2500 g)	8.0%
Induced abortions	664,435
Total pregnancies[a]	4,652,511

[a]Excludes spontaneous abortions and ectopic pregnancies.
Data from Martin, 2017.

MEASURES OF OBSTETRICAL CARE

Perinatal Mortality

Several indices are used to assess obstetrical and perinatal outcomes as measures of medical care quality. As noted, the perinatal mortality rate includes the numbers of stillbirths and neonatal deaths per 1000 total births. In 2013, the perinatal mortality rate was 9.98 per 1000 births (Fig. 1-1) (MacDorman, 2015). There were 25,972 fetal deaths at gestational ages of 20 weeks or older. Fetal deaths at 28 weeks or more have been declining since 1990, whereas rates for those between 20 and 27 weeks are static (Fig. 1-2). By way of comparison, there were a total of 19,041 neonatal deaths in 2006—meaning that nearly 60 percent of the perinatal deaths in the United States were fetal.

Infant Deaths

There were 6.1 infant deaths per 1000 live births in 2013 compared with 6.8 in 2001 (MacDorman, 2015). The three leading causes of infant death—congenital malformations, low birthweight, and sudden infant death syndrome—accounted for almost half of all deaths (Heron, 2015). Infants born at the lowest gestational ages and birthweights add substantively to these mortality rates. For example, more than half of all infant deaths in 2005 were in the 2 percent of infants born before

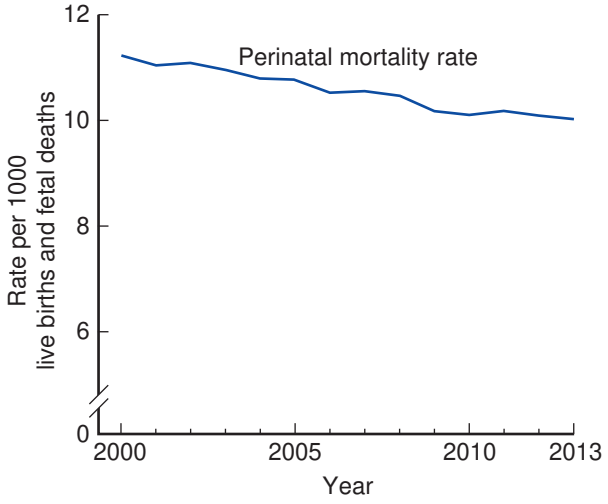

FIGURE 1-1 Perinatal mortality rates: United States, 2000–2013. (Reproduced with permission from MacDorman MF, Gregory EC: Fetal and perinatal mortality: United States, 2013. Natl Vital Stat Rep. 2015 Jul 23;64(8):1–24.)

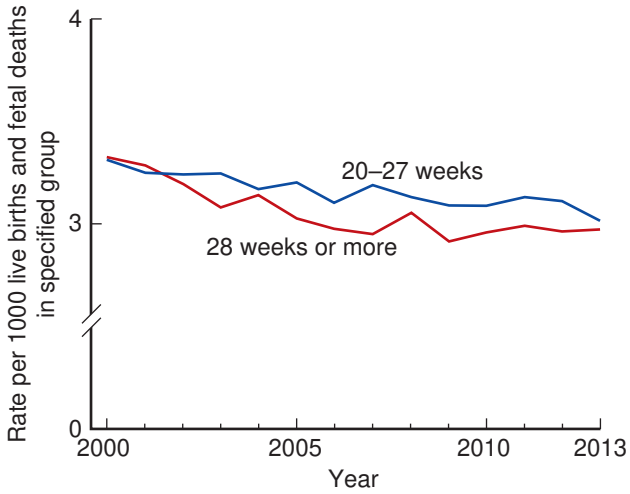

FIGURE 1-2 Fetal and neonatal deaths: United States, 2000–2013. (Modified with permission from MacDorman MF, Gregory EC: Fetal and perinatal mortality: United States, 2013. Natl Vital Stat Rep. 2015 Jul 23;64(8):1–24.)

32 weeks' gestation. Indeed, the percentage of infant deaths related to preterm birth increased from 34.6 percent in 2000 to 36.5 percent in 2005. When analyzed by birthweight, two thirds of infant deaths were in low-birthweight neonates. Of particular interest are infants with birthweights <500 g, for whom neonatal intensive care can now be offered.

Maternal Mortality

As shown in Figure 1-3, maternal mortality rates dropped precipitously in the United States during the 20th century. Pregnancy-related deaths are so uncommon as to be measured per 100,000 births. The CDC (2017a) has maintained data on pregnancy-related deaths since 1986 in its Pregnancy Mortality Surveillance System. In the latest report, Creanga and coworkers (2017) described 2009 pregnancy-related deaths during the period from 2011 to 2013. Approximately 5 percent were early-pregnancy deaths due to ectopic gestation or abortive outcomes. The deadly obstetrical triad of hemorrhage, preeclampsia, and infection has accounted for a third of all deaths (Fig. 1-4). Thromboembolism, cardiomyopathy, and other cardiovascular

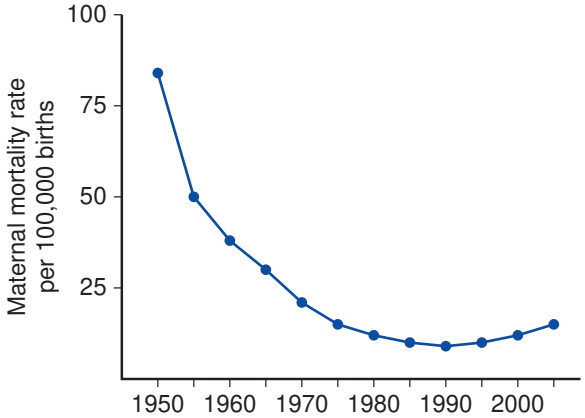

FIGURE 1-3 Maternal mortality rates for the United States, 1950–2003. (Data from Berg, 2010; Hoyert, 2007.)

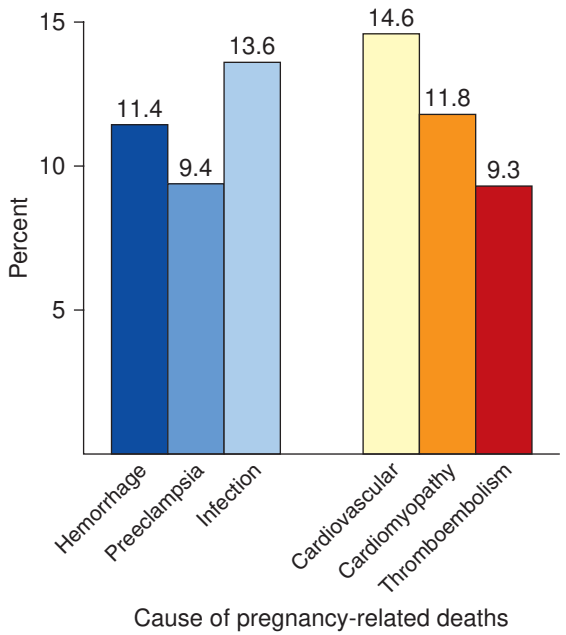

FIGURE 1-4 Six common causes of pregnancy–related deaths for the United States, 2006–2010. (Data from Creanga, 2015.)

disease together accounted for another third. Other significant contributors were amniotic fluid embolism (5.3 percent) and cerebrovascular accidents (6.2 percent). Anesthesia-related deaths were at an all-time low—only 0.7 percent. Similar causes were reported for selected cohorts for years 2008 to 2009 and 2013 to 2014 (MacDorman, 2017).

Shown in Figure 1-5, the pregnancy-related mortality ratio of 23.8 per 100,000 live births in 2014 is the highest during the previous 40 years. And, according to the Institute of Health Metrics, it was 28 per 100,000 in 2013 (Tavernise, 2016). This rise simply may be that more women are dying, however, other factors explain this doubling of the rate from 1990 to 2013 (Joseph, 2017). The first is an artificial elevation caused by the International Statistical Classification of Diseases, 10th Revision (ICD-10), implemented in 1999. Second, improved

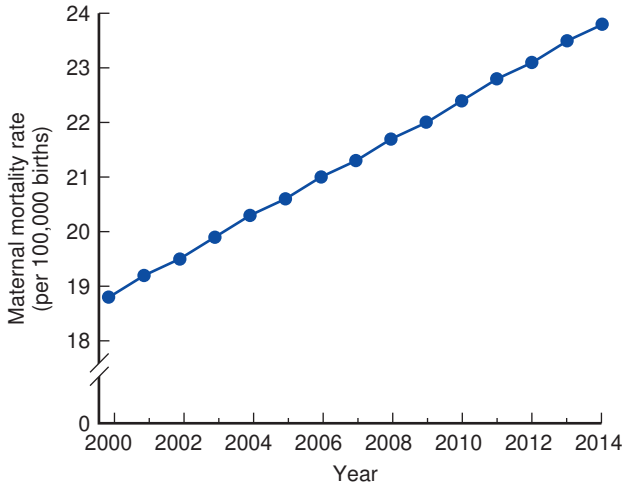

FIGURE 1-5 Estimated maternal mortality rates in 48 states and the District of Columbia. (Data from MacDorman, 2016.)

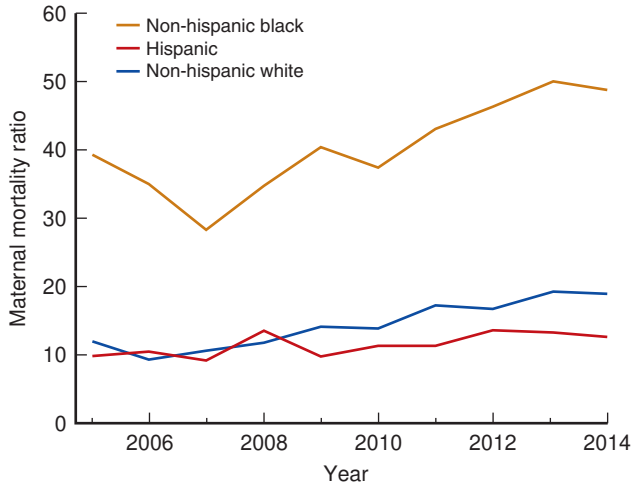

FIGURE 1-6 Trends in maternal mortality ratio (per 100,000 live births) by race: United States, 2005–2014. (Data from Moaddab, 2016.)

reporting definitely contributes to the rise (MacDorman, 2016b, 2017). In the past, maternal deaths were notoriously underreported (Koonin, 1997). Third, and related to the second explanation, the rate of rise is at least partially due to the revised death certificate and its pregnancy checkbox described earlier (Main, 2015). Fourth, the number of pregnant women with severe chronic health conditions, which place women at higher risk, is greater (Centers for Disease Control and Prevention, 2017a). Finally, the increased proportion of births to women older than 40 years contribute to higher mortality rates (MacDorman, 2017).

Whatever the cause, the apparent sharp rise of the maternal mortality rates has galvanized the obstetrical community to action (Chescheir, 2015). According to Barbieri (2015), the Joint Commission has recommended that birthing centers establish standardized protocols and implement simulation efforts. D'Alton and colleagues (2016) described efforts of a working group to lower morbidity and mortality rates.

Another consideration is the obvious disparity of higher mortality rates among black, Hispanic, and white women as shown in Figure 1-6. Racial disparities translate to health care availability, access, or utilization (Howell, 2016; Moaddab, 2016). And, maternal mortality is disparately high in rural compared with metropolitan areas (Maron, 2017).

Importantly, many of the reported maternal deaths are considered preventable. Berg and colleagues (2005) estimated that this may be up to a third of pregnancy-related deaths in white women and up to half of those in black women. In one evaluation of an insured cohort, 28 percent of 98 maternal deaths were judged preventable (Clark, 2008). Thus, although significant progress has been made, further efforts are imperative for obstetrics in the 21st century.

■ Severe Maternal Morbidity

This serves as another measure to guide prevention efforts. Lowering medical error rates serves to diminish risks for maternal mortality or severe maternal morbidity. The terms *near misses* or *close calls* were introduced and defined as unplanned events caused by error that do not result in patient injury but have the potential to do so (Institute for Safe Medication Practices, 2009). These are much more common than injury events, but for obvious reasons, they are more difficult to identify and quantify. Systems designed to encourage reporting have been installed in various institutions and allow focused safety efforts (Clark, 2012; Main, 2017; Shields, 2017). The American College of Obstetricians and Gynecologists and the Society for Maternal–Fetal Medicine (2016f) have provided lists of suggested screening topics for this purpose.

Several data systems now measure indicators of unplanned events caused by errors that have injurious potential. This evolution followed inadequacies in the ability of hospitalization coding to reflect the severity of maternal complications. Thus, coding indicators or modifiers are used to allow analysis of serious adverse clinical events (Clark, 2012; King, 2012). Such a system was implemented by the World Health Organization. It has been validated in Brazil and accurately reflects maternal death rates (Souza, 2012). Similar systems are in use in Britain as the *UK Obstetric Surveillance System—UKOSS* (Knight, 2005, 2008). In the United States, one example is the National Partnership for Maternal Safety (D'Alton, 2016; Main, 2015).

To study severe morbidity, the CDC analyzed more than 50 million maternity records from the Nationwide Inpatient Sample from 1998 to 2009 (Callaghan, 2012). They used ICD-9-CM codes and reported that 129 per 10,000 of these gravidas had at least one indicator for severe morbidity (Table 1-3). Thus, for every maternal death, approximately 200 women experience severe morbidity. The CDC (2017b) estimates that 65,000 women per year have such maternal morbidity. These numbers are greatest in smaller hospitals with <1000 deliveries annually (Hehir, 2017). Finally, as with mortality rates, there are serious racial and ethnic disparities for severe maternal morbidity, and black women are disproportionately affected (Creanga, 2014).

TIMELY TOPICS IN OBSTETRICS

Various topics have been in the forefront for obstetrical providers in the 4 years since the last edition of this textbook. In the following, we discuss several of these topics.

■ U.S. Health Care in Crisis

Obamacare and Medicaid

In a 2016 issue of the *Journal of the American Medical Association (JAMA),* then-President Barack Obama presented a summary of the Affordable Care Act (ACA), so-called Obamacare. He described the successes, the challenges ahead, and the policy implications of the policy (Bauchner, 2016). He summarized three lessons from his experiences with the ACA. First, change is especially difficult in the face of hyperpartisanship. Second, special interests pose a continued obstacle to change. Third, he stressed the importance of pragmatism. Here, he was referring to the pragmatism necessary when the ACA did not work effectively on day 1 of implementation.

TABLE 1-3. Severe Maternal Morbidity Indicators

Acute myocardial infarction
Acute renal failure
Adult respiratory distress syndrome
Amnionic fluid embolism
Cardiac arrest/ventricular fibrillation
Disseminated intravascular coagulation
Eclampsia
Heart failure during procedure
Injuries of thorax, abdomen, and pelvis
Intracranial injuries
Puerperal cerebrovascular disorders
Pulmonary edema
Severe anesthesia complications
Sepsis
Shock
Sickle-cell crisis
Thrombotic embolism
Cardiac monitoring
Conversion of cardiac rhythm
Hysterectomy
Cardiac surgery
Tracheostomy
Ventilation

Summarized from the Centers for Disease Control and Prevention, 2017b.

At this same time, draconian cuts to Medicaid were being proposed, and President Obama ended his JAMA report with a quotation from John Kasich, the Republican governor of Ohio. "For those that live in the shadows of life, those who are the least among us, I will not accept the fact that the most vulnerable in our state should be ignored. We can help them."

These potential effects to Medicaid ripple into the specialty of obstetrics. In 2010, it was estimated that Medicaid insured 48 percent of the births in the United States (Markus, 2013). Importantly, Medicaid covered a disproportionate number of complicated births. Specifically, Medicaid insured more than half of all hospital stays for preterm and low-birthweight infants and approximately 45 percent of infant hospital stays due to birth defects.

Repeal and Replace

The young, healthy Americans who were expected to financially bolster the ACA ultimately enrolled in insufficient numbers to ensure long-term ACA sustainability. Thus, long-term options included repair or repeal of the ACA. Throughout Donald Trump's campaign for the presidency of the United States, he made repeal of the ACA a focus of his candidacy. As of this writing, both the United States House of Representatives and the Senate have grappled with "repeal and replace" for 6 months. According to the Congressional Budget Office, this action would result in 23 million Americans losing health care insurance and cuts in Medicaid dollars (Fiedler, 2017). The latter was to be accomplished by transferring funding of Medicaid from the Federal government to the states. These potential outcomes have prompted considerable debate among voters, and "repeal and replace" has become politically charged. Currently, the Senate has been unable to recruit sufficient Republican votes for Senate passage of such a bill. We suggest that the health care crisis should be reframed and redirected instead to a critical analysis of health care costs and resource utilization.

Maternal and Infant Health Care Costs

The Centers for Medicare and Medicaid Services estimated that spending on health care in the United States in 2015 accounted for 17.8 percent of the gross domestic product—GDP (Voelker, 2010). The total amount of health-care spending—$3.2 trillion—equated to an estimated $10,000 per person. Moreover, compared with 12 other high-income countries, health-care spending in the United States as a proportion of GDP was approximately 50 percent more than the next highest country. Yet, health-care outcomes, which included infant mortality rates, were worse in the United States. And, approximately two thirds of U.S. infant deaths result from complications stemming from preterm births (Matthews, 2015). Indeed, in its 2010 annual global Premature Birth Report Card, the United States garnered a grade of "D" from the March of Dimes for its recognition and prevention of preterm labor in the more than 540,000 neonates born annually before 37 weeks' gestation.

Causes for the excessive health care costs in the United States are attributed, in part, to greater use of medical technology and excessive prices (Squires, 2017). Two recent studies demonstrate the detrimental effect of obstetrics on health care costs. The first report by Nelson and coworkers (2017) described the ineffectiveness of 17-alpha hydroxyprogesterone caproate (17-OHP-C) to prevent recurrent preterm birth. Methodology for this trial is presented in Chapter 42 (p. 817). Several lessons can be learned from this investigation. First, use of 17-OHP-C was legitimized in the United States by a national consensus committee using expert opinion. These opinions were promulgated, despite FDA reservations that the evidence was lacking in several important respects. However, once approved, 17-OHP-C was sold by one pharmaceutical company for $1500 for a single, 250-mg injectable dose. Remarkably, this same dose could be compounded and purchased for $25 from local pharmacies. In the subsequent price-gouging controversy, members of the United States Congress intervened to permit continued use of the less expensive 17-OHP-C.

The second study is a multisite prospective trial of the effectiveness of transvaginal sonography to screen for cervical-length shortening to predict preterm birth (Esplin, 2017). A total of 9410 nulliparous women were studied. The Society for Maternal-Fetal Medicine and the American College of Obstetricians and Gynecologists (2016d) both legitimized *universal* cervical-length screening in their joint Committee Opinion (Bloom, 2017). And, by 2015, one survey of 78 Maternal-Fetal Medicine fellowship programs showed that 68 percent were using universal cervical-length screening to predict preterm birth (Khalifeh, 2017). It was estimated that a modest Medicaid rate of $237 per cervical-length ultrasound would result in approximately $350 million in added health care costs. But, Esplin and

associates (2017) found that *routine* screening for a short cervix was not beneficial. That is, a widely used intervention was actually ineffective. This is a clear example of how unproven technology can seep into widespread practice.

These two reports highlight a substantial problem in U.S. health care, namely, ineffective yet expensive interventions introduced into broad use without robust evidence. These two reports also speak to a demand for robust scientific evidence. Scrutiny of other ingredients in the health-care paradigm such as prices for hospitalization, prices for surgical procedures, and prices charged by health insurance companies may illuminate similar contributions to the health care fiscal crisis.

Cesarean Delivery Rate

In past editions of this textbook, the rising cesarean delivery rate was considered problematic. This rate has leveled, but there are still imperatives in progress to help lower this rate. One collateral source of cesarean delivery morbidity is from the growing incidence of morbidly adherent placentas encountered in women with a prior hysterotomy incision, discussed in Chapters 31 and 41.

Genomic Technology

Breakthroughs in fetal testing and diagnosis continue to stun. By 2012, prenatal gene microarray techniques were used for clinical management (Dugoff, 2012). The advantages of these techniques are outlined in Chapters 13 and 14. Wapner and coworkers (2012) compared chromosomal microarray analysis of maternal blood with karyotyping for chromosomal anomalies. Reddy and associates (2012) applied this technology to stillbirth evaluation and reported it to be superior to karyotyping. Another report by Talkowski and colleagues (2012) described whole-genome sequencing of a fetus using maternal blood.

Screening for fetal aneuploidy using cell-free DNA (cfDNA) was first introduced in 2011. The technique is described in Chapter 14 (p. 284), and it is based on isolation of free fetal (placental) DNA in maternal blood. In a landmark study, Norton and associates (2015) found that cfDNA had a higher sensitivity and specificity compared with standard prenatal screening for trisomy 21 fetuses. Still, invasive testing is currently necessary to confirm a positive cfDNA test result (Chitty, 2015; Snyder, 2015).

The Ob/Gyn Hospitalist

The term "hospitalist" was coined in the 1990s and referred to physicians whose primary professional focus was generalized care of hospitalized patients. From this concept came the obstetrical and gynecological hospitalist whose primary role was to care for hospitalized obstetrical patients and to help manage their emergencies. These physicians could also provide urgent gynecological care and emergency department consultation. Alternative terms include "obstetrical hospitalist" or "laborist," but the preferred standardized term by the American College of Obstetricians and Gynecologists (2016e) is "Ob/Gyn hospitalist."

Although not a recognized subspecialty of obstetrics and gynecology, the Ob/Gyn hospitalist movement has gained momentum. The Society of Ob-Gyn Hospitalists had 528 members in 2017 (Burkard, 2017). Various practice models are described to fit the needs of a wide spectrum of obstetrical volumes (McCue, 2016). In addition to providing lifestyle modifications, Ob/Gyn hospitalists are used by some hospitals to improve the quality and safety of their women's services and to reduce adverse events. Aside from a possible lowering of the labor induction rate, studies are needed to demonstrate improved outcomes with these providers (American College of Obstetricians and Gynecologists, 2016e; Srinivas, 2016).

Medical Liability

The American College of Obstetricians and Gynecologists periodically surveys its fellows concerning the effect of liability on their practice. The 2015 Survey on Professional Liability is the 12th such report since 1983 (Carpentieri, 2015). From this survey, it appears that there is still a "liability crisis," and the reasons for it are complex. Because it is largely driven by money and politics, a consensus seems unlikely. Although some interests are diametrically opposite, other factors contribute to the problem's complexity. For example, each state has its own laws and opinions on tort reform. In some states, annual premiums for obstetricians approach $300,000—expenses that at least partially are borne by the patient and certainly by the entire health-care system. In 2011, all tort costs in the United States totaled nearly $265 billion. This is an astounding 1.8 percent of the gross domestic product and averages to a cost of $838 per citizen (Towers Watson, 2015).

The American College of Obstetricians and Gynecologists (2016a,c) has taken a lead in adopting a fair system for malpractice litigation—or *maloccurrence litigation*. And nationally, there is the possibility of federal tort reform under the Trump administration (Lockwood, 2017; Mello, 2017).

Home Births

Following a slight decline from 1990 through 2004, the percentage of out-of-hospital births in the United States increased from 0.86 to 1.5 percent—almost 75 percent—through 2014 (MacDorman, 2016a). Of these home births, only a third are attended by nurse midwives certified by the American Midwife Certification Board (Grünebaum, 2015; Snowden, 2015).

Proponents of home births cite successes derived from laudatory observational data from England and The Netherlands (de Jonge, 2015; Van der Kooy, 2011). Data from the United States, however, are less convincing and indicate a higher incidence of perinatal morbidity and mortality (Grünebaum, 2014, 2015; Snowden, 2015; Wasden, 2014; Wax, 2010). These latter findings have led Chervenak and coworkers (2013, 2015) to question the ethics of participation in planned home births. Greene and Ecker (2015) take a broader view. Given data from these more recently cited studies, they are of the view that these data empower women to make a rational decision regarding home delivery. The American College of Obstetricians and Gynecologists (2017b) believes that hospitals and accredited birth centers offer the safest settings, but that each woman has the right to make a medically informed decision regarding delivery.

Family Planning Services

Politics and religion over the years have led to various governmental interferences with the reproductive rights of women. These intrusions have disparately affected indigent women and adolescents. This is despite all reports of the overwhelming success of such programs. One example is the exclusion of Planned Parenthood affiliates from the Texas Medicaid fee-for-service family planning program. In some groups of women served, there was discontinuation of contraception and an increased rate of Medicaid births (Stevenson, 2016).

According to the Guttmacher Institute (2016a), publicly funded family planning services are needed by 20 million American women. In 2014, such services prevented nearly 2 million unintended pregnancies and 700,000 abortions in the United States. The fate of family planning services is not fully determined, while waiting for decisions regarding provisions within the 2017 American Health Care Act (AHCA), or "Trumpcare." In his response to news that the AHCA may dismantle contraceptive coverage, American College of Obstetricians and Gynecologists President Dr. Haywood Brown (2017) called this a deep disregard for women's health.

Opioid Abuse in Pregnancy

According to the CDC (2014), there were 259 million prescriptions written in 2012 for opioid medications. In 2013, more than a third of American adults reported prescription opioid use (Han, 2017). These freely available—albeit requiring a prescription—addictive drugs are associated with *opioid use disorders*. It remains uncertain if opioid use is teratogenic (Lind, 2017). Still, their abuse by pregnant women has caused an unprecedented rise in the *neonatal abstinence syndrome,* described further in Chapters 12 (p. 248) and 33 (p. 625). Treatment of opioid abuse in pregnancy and its sequelae result in $1.5 billion annually in hospital charges.

For obstetrical providers to better deal with opioid-addicted pregnant women and their fetus–newborns, the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development convened a workshop in 2016 to study many aspects of the problem (Reddy, 2017). The Workshop was cosponsored by the American College of Obstetricians and Gynecologists, the American Academy of Pediatrics, the Society for Maternal–Fetal Medicine, the CDC, and the March of Dimes. Several topics were addressed, and hopefully implementation of these findings will help improve maternal treatment and neonatal outcomes (American College of Obstetricians and Gynecologists, 2017a).

Brave New World

The bold new concept of in-vitro fertilization (IVF) produced the first IVF baby in Britain in 1978. This was soon followed in 1981 with an American success. After four decades, the Society for Assisted Reproductive Technology (SART) reports that more than 1 million babies have been born in the United States using assisted reproductive technologies (ART) offered by 440 clinics (Fox, 2017).

After 15 years of experimental preparation, the promise of a successful human uterine transplant was finally realized with an IVF-conceived liveborn neonate in Sweden (Brännström, 2015). During pregnancy, the mother was treated with tacrolimus, azathioprine, and corticosteroids and underwent cesarean delivery at 32 weeks for preeclampsia and abnormal fetal heart rate testing. This was followed by uterine transplantation programs at the Cleveland Clinic and Baylor Medical Center in Dallas (Flyckt, 2016, 2017; Testa, 2017). In 2017, the Swedish team had completed a nine-patient trial, in which seven women had become pregnant and five had successful deliveries (Kuehn, 2017). Also, in Dallas, the first such newborn in the United States was born (Rice, 2017).

Meanwhile, researchers at Children's Hospital of Philadelphia pursued a 20-year goal in search of an artificial womb (Yuko, 2017). Using incubator technology, the team devised an artificial amnionic sac. Through this, the umbilical vessels were perfused and drained, and the blood was returned to systems that performed extracorporeal membrane oxygenation and dialysis. To date, lamb fetuses have been kept alive for as long as 1 month. Adverse effects of cerebrovascular hypotension and hypoxemia are conjectural but highly worrisome.

The ethical and legal challenges of these new technologies are daunting. Of those that arose from IVF, most are settled. For the other two endeavors, there are likely many years of ethical and legal milestones ahead.

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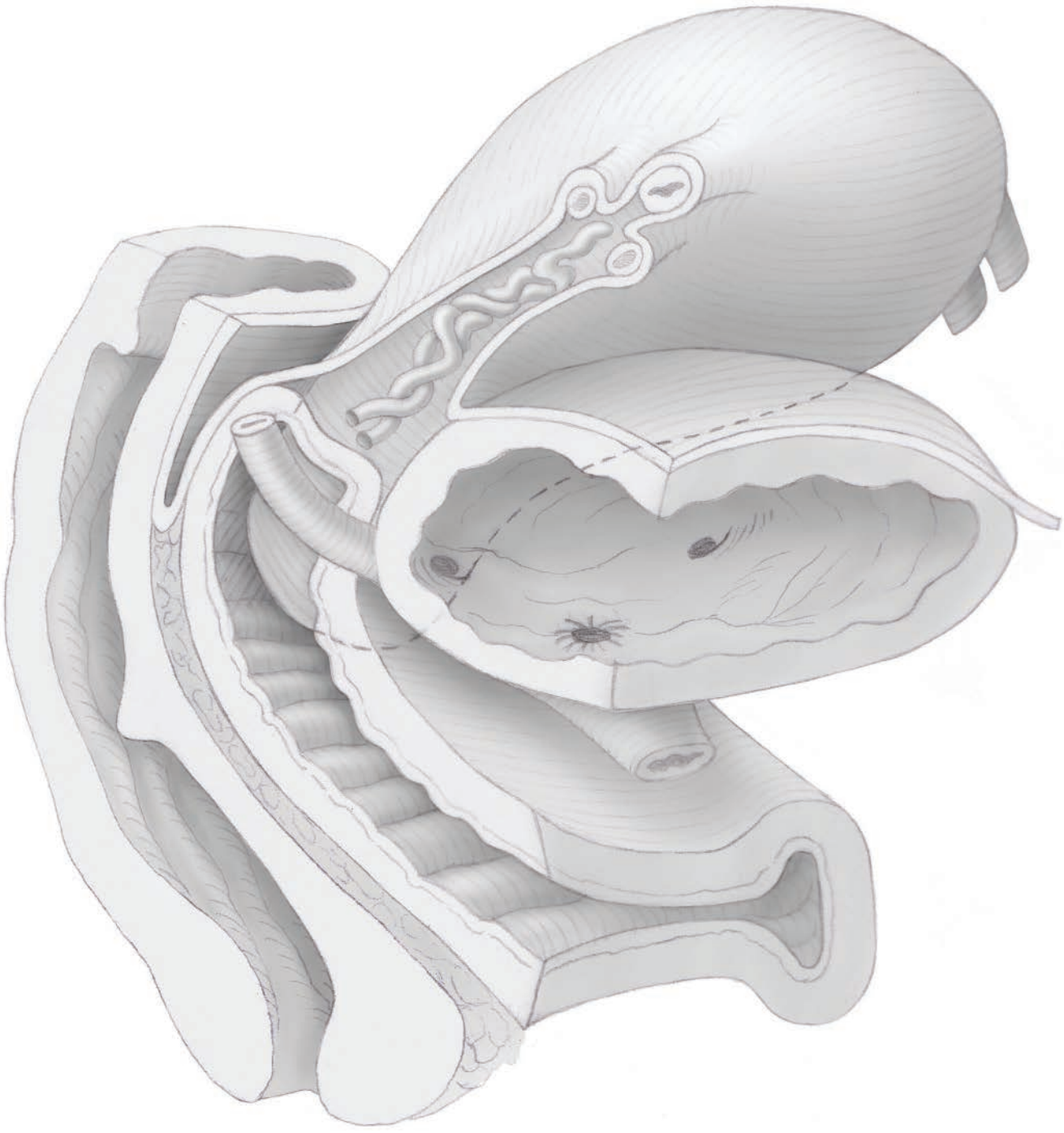
SECTION 2

MATERNAL ANATOMY AND PHYSIOLOGY



CHAPTER 2

Maternal Anatomy

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MUSCULOSKELETAL PELVIC ANATOMY 29

As the mechanism of labour is essentially a process of accommodation between the foetus and the passage through which it must pass, it is apparent that obstetrics lacked a scientific foundation until the anatomy of the bony pelvis and of the soft parts connected with it was clearly understood.

—J. Whitridge Williams (1903)

ANTERIOR ABDOMINAL WALL

Skin, Subcutaneous Layer, and Fascia

The anterior abdominal wall confines abdominal viscera, stretches to accommodate the expanding uterus, and provides surgical access to the internal reproductive organs. Thus, a comprehensive knowledge of its layered structure is required to surgically enter the peritoneal cavity.

Langer lines describe the orientation of dermal fibers within the skin. In the anterior abdominal wall, they are arranged transversely. As a result, vertical skin incisions sustain greater lateral tension and thus, in general, develop wider scars. In contrast, low transverse incisions, such as the Pfannenstiel, follow Langer lines and lead to superior cosmetic results.

The subcutaneous layer can be separated into a superficial, predominantly fatty layer—Camper fascia, and a deeper membranous layer—Scarpa fascia. Camper fascia continues onto the perineum to provide fatty substance to the mons pubis and labia majora and then to blend with the fat of the ischioanal fossa. Scarpa fascia continues inferiorly onto the perineum as Colles fascia, described on page 19.

Beneath the subcutaneous layer, the anterior abdominal wall muscles consist of the midline rectus abdominis and pyramidalis muscles as well as the external oblique, internal oblique, and transversus abdominis muscles, which extend across the entire wall (Fig. 2-1). The fibrous aponeuroses of these three latter muscles form the primary fascia of the anterior abdominal wall. These fuse in the midline at the linea alba, which normally measures 10 to 15 mm wide below the umbilicus (Beer, 2009). An abnormally wide separation may reflect diastasis recti or hernia.

These three aponeuroses also invest the rectus abdominis muscle as the rectus sheath. The construction of this sheath varies above and below a boundary, termed the arcuate line (see Fig. 2-1). Cephalad to this border, the aponeuroses invest the rectus abdominis bellies on both dorsal and ventral surfaces. Caudal to this line, all aponeuroses lie ventral or superficial to the rectus abdominis muscle, and only the thin transversalis fascia and peritoneum lie beneath the rectus (Loukas, 2008). This transition of rectus sheath composition can be seen best in the upper third of a midline vertical abdominal incision.

The paired small triangular pyramidalis muscles originate from the pubic crest and insert into the linea alba. These muscles lie atop the rectus abdominis muscle but beneath the anterior rectus sheath.

Blood Supply

The superficial epigastric, superficial circumflex iliac, and superficial external pudendal arteries arise from the femoral artery just below the inguinal ligament within the femoral triangle

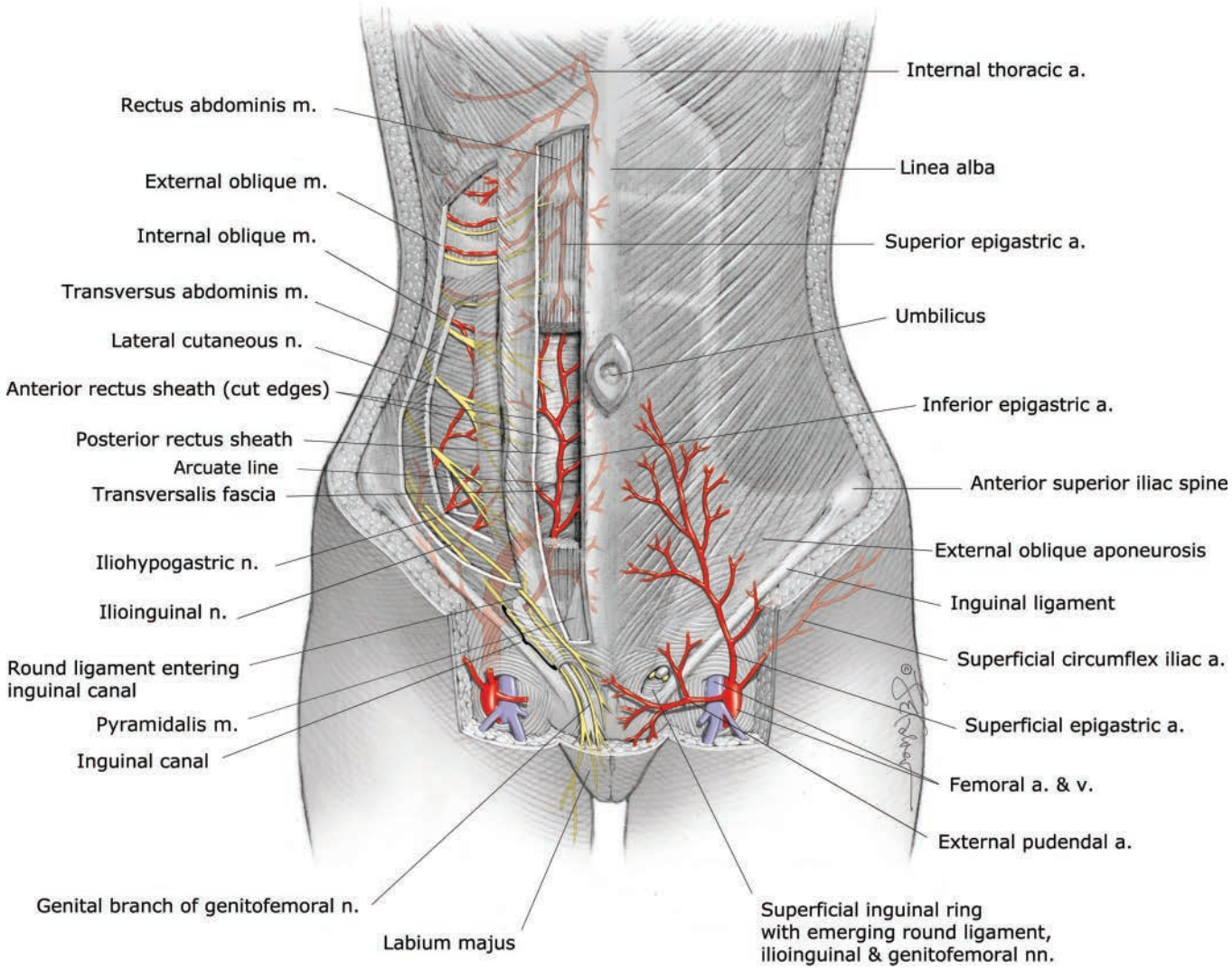

FIGURE 2-1 Anterior abdominal wall anatomy. (Modified with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

(see Fig. 2-1). These vessels supply the skin and subcutaneous layers of the anterior abdominal wall and mons pubis. Of these three, the superficial epigastric vessels are surgically important to the obstetrician and course diagonally from their origin toward the umbilicus. With a low transverse skin incision, these vessels can usually be identified at a depth halfway between the skin and the anterior rectus sheath. They lie above Scarpa fascia and several centimeters from the midline. Ideally, these vessels are identified and surgically occluded.

In contrast, the inferior "deep" epigastric vessels are branches of the external iliac vessels and supply anterior abdominal wall muscles and fascia. Of surgical relevance, the inferior epigastric vessels initially course lateral to, then posterior to the rectus abdominis muscles, which they supply. Above the arcuate line, these vessels course ventral to the posterior rectus sheath and lie between this sheath and the posterior surface of the rectus muscles. Near the umbilicus, the inferior epigastric vessels anastomose with the superior epigastric artery and vein, which are branches of the internal thoracic vessels. Clinically, when a Maylard incision is used for cesarean delivery, the inferior epigastric vessels may be lacerated lateral to the rectus belly during muscle transection. Preventively, identification and surgical occlusion are preferable. These vessels rarely may rupture following abdominal trauma and create a rectus sheath hematoma (Tolcher, 2010; Wai, 2015).

On each side of the lower anterior abdominal wall, Hesselbach triangle is the region bounded laterally by the inferior epigastric vessels, inferiorly by the inguinal ligament, and medially by the lateral border of the rectus abdominis muscle. Hernias that protrude through the abdominal wall in Hesselbach triangle are termed direct inguinal hernias. In contrast, indirect inguinal hernias do so through the deep inguinal ring, which lies lateral to this triangle, and then may exit out the superficial inguinal ring.

Innervation

The entire anterior abdominal wall is innervated by intercostal nerves ($T_{7–11}$), the subcostal nerve (T_{12}), and the iliohypogastric and the ilioinguinal nerves (L_1). Of these, the

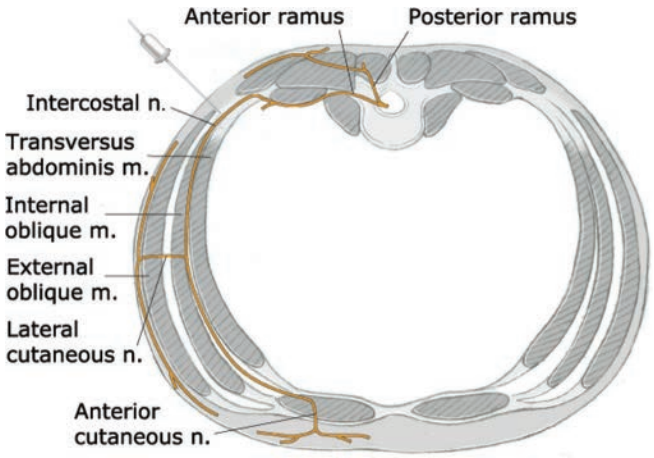

FIGURE 2-2 Intercostal and subcostal nerves are the anterior rami of spinal nerves. In this figure, an intercostal nerve extends ventrally between the transversus abdominis and internal oblique muscles. During this path, the nerve gives rise to lateral and anterior cutaneous branches, which innervate the anterior abdominal wall. As shown by the inserted needle, the transversus abdominis plane (TAP) block takes advantage of this anatomy. (Modified with permission from Hawkins JL: Anesthesia for the pregnant woman. In Yeomans ER, Hoffman BL, Gilstrap LC III, et al: Cunningham and Gilstraps's Operative Obstetrics, 3rd ed. New York, McGraw Hill Education, 2017.)

intercostal and subcostal nerves are anterior rami of the thoracic spinal nerves and run along the lateral and then anterior abdominal wall between the transversus abdominis and internal oblique muscles (Fig. 2-2). This space, termed the transversus abdominis plane, can be used for postcesarean analgesia blockade (Chap. 25, p. 500) (Fusco, 2015; Tawfik, 2017). Others report rectus sheath or ilioinguinal-iliohypogastric nerve blocks to decrease postoperative pain (Mei, 2011; Wolfson, 2012).

Near the rectus abdominis lateral borders, anterior branches of the intercostal and subcostal nerves pierce the posterior sheath, rectus muscle, and then anterior sheath to reach the skin. Thus, these nerve branches may be severed during a Pfannenstiel incision creation during the step in which the overlying anterior rectus sheath is separated from the rectus abdominis muscle.

In contrast, the iliohypogastric and ilioinguinal nerves originate from the anterior ramus of the first lumbar spinal nerve. They emerge lateral to the psoas muscle and travel retroperitoneally across the quadratus lumborum inferomedially toward the iliac crest. Near this crest, both nerves pierce the transversus abdominis muscle and course ventromedially. At a site 2 to 3 cm medial to the anterior superior iliac spine, the nerves then pierce the internal oblique muscle and course superficial to it toward the midline (Whiteside, 2003). The iliohypogastric nerve perforates the external oblique aponeurosis near the lateral rectus border to provide sensation to the skin over the suprapubic area (see Fig. 2-1). The ilioinguinal nerve in its course medially travels through the inguinal canal and exits through the superficial inguinal ring, which forms by splitting of external abdominal oblique aponeurosis fibers. This nerve supplies the skin of the mons pubis, upper labia majora, and medial upper thigh.

The ilioinguinal and iliohypogastric nerves can be severed during a low transverse incision or entrapped during closure, especially if incisions extend beyond the lateral borders of the rectus abdominis muscle (Rahn, 2010). These nerves carry sensory information only, and injury leads to loss of sensation within the areas supplied. Rarely, chronic pain may develop (Whiteside, 2005).

The T_{10} dermatome approximates the level of the umbilicus. Analgesia to this level is suitable for labor and vaginal birth. Regional analgesia for cesarean delivery or for puerperal sterilization ideally extends to T_4.

EXTERNAL GENERATIVE ORGANS

Vulva

Mons Pubis, Labia, and Clitoris

The pudenda—commonly designated the vulva—includes all structures visible externally from the symphysis pubis to the perineal body. This includes the mons pubis, labia majora and minora, clitoris, hymen, vestibule, urethral opening, greater vestibular or Bartholin glands, minor vestibular glands, and paraurethral glands (Fig. 2-3). The vulva receives innervations and vascular support from the pudendal nerve (p. 22).

The mons pubis is a fat-filled cushion overlying the symphysis pubis. After puberty, the mons pubis skin is covered by curly hair that forms the triangular escutcheon, whose base aligns with the upper margin of the symphysis pubis. In men and some hirsute women, the escutcheon extends farther onto the anterior abdominal wall toward the umbilicus.

Labia majora usually are 7 to 8 cm long, 2 to 3 cm wide, and 1 to 1.5 cm thick. They are continuous directly with the mons pubis superiorly, and the round ligaments terminate at their upper borders. Hair covers the labia majora, and apocrine, eccrine, and sebaceous glands are abundant. Beneath the skin, a dense connective tissue layer is nearly void of muscular elements but is rich in elastic fibers and fat. This fat mass provides bulk to the labia majora and is supplied with a rich venous plexus. During pregnancy, this vasculature may develop varicosities, especially in multiparas, from increased venous pressure created by the enlarging uterus. They appear as engorged tortuous veins or as small grapelike clusters, but they are typically asymptomatic and require no treatment.

Each labium minus is a thin tissue fold that lies medial to each labium majus. The labia minora extend superiorly, where each divides into two lamellae. From each side, the lower lamellae fuse to form the frenulum of the clitoris, and the upper lamellae merge to form the prepuce (see Fig. 2-3). Inferiorly, the labia minora extend to approach the midline as low ridges of tissue that join to form the fourchette. The labia minora dimensions vary greatly among individuals, with lengths from 2 to 10 cm and widths from 1 to 5 cm (Lloyd, 2005).

Structurally, the labia minora are composed of connective tissue with numerous vessels, elastin fibers, and very few smooth muscle fibers. They are supplied with many nerve endings and are extremely sensitive (Ginger, 2011a; Schober, 2015). The epithelia of the labia minora differ with location. Thinly keratinized stratified squamous epithelium covers the

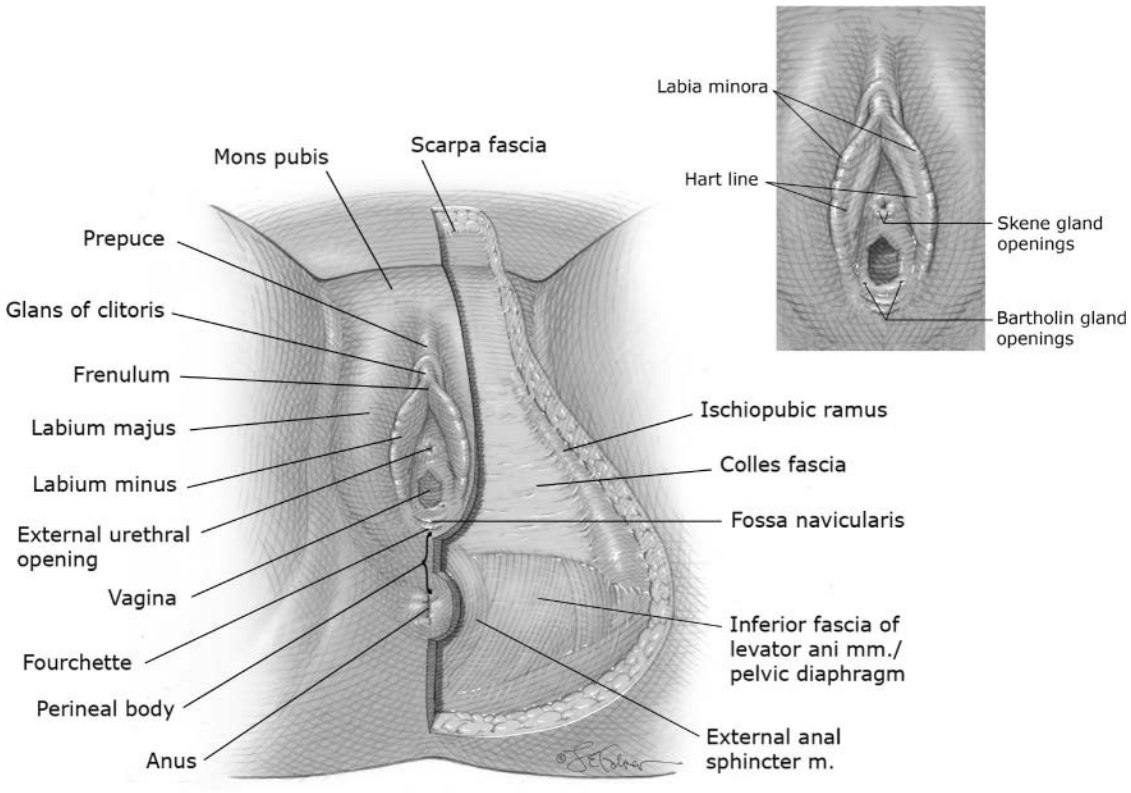

FIGURE 2-3 Vulvar structures and subcutaneous layer of the anterior perineal triangle. Note the continuity of Colles and Scarpa fasciae. Inset: Vestibule boundaries and openings onto vestibule. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

outer surface of each labium. On their inner surface, the lateral portion is covered by this same epithelium up to a demarcating line, termed Hart line. Medial to this line, each labium is covered by squamous epithelium that is nonkeratinized. The labia minora lack hair follicles, eccrine glands, and apocrine glands. However, sebaceous glands are numerous (Wilkinson, 2011).

The clitoris is the principal female erogenous organ. It is located beneath the prepuce, above the frenulum and urethra, and projects downward and inward toward the vaginal opening. The clitoris rarely exceeds 2 cm in length and is composed of a glans, a corpus or body, and two crura (Verkauf, 1992). The glans is usually less than 0.5 cm in diameter, is covered by stratified squamous epithelium, and is richly innervated. The clitoral body contains two corpora cavernosa. Extending from the clitoral body, each corpus cavernosum diverges laterally to form a long, narrow crus. Each crus lies along the inferior surface of its respective ischiopubic ramus and deep to the ischiocavernosus muscle. The clitoral blood supply stems from branches of the internal pudendal artery. Specifically, the deep artery of the clitoris supplies the clitoral body, whereas the dorsal artery of the clitoris supplies the glans and prepuce.

Vestibule

In adult women, the vestibule is an almond-shaped area that is enclosed by Hart line laterally, the external surface of the hymen medially, the clitoral frenulum anteriorly, and the fourchette posteriorly (see Fig. 2-3). The vestibule is usually perforated by six openings: the urethra, the vagina, two Bartholin gland ducts, and two ducts of the largest paraurethral glands—the Skene glands. The posterior portion of the vestibule between the fourchette and the vaginal opening is called the fossa navicularis. It is usually observed only in nulliparas.

The bilateral Bartholin glands, also termed greater vestibular glands, measure 0.5 to 1 cm in diameter. On their respective side, each lies inferior to the vestibular bulb and deep to the inferior end of the bulbospongiosus muscle (former bulbocavernosus muscle). A duct extends medially from each gland, measures 1.5 to 2 cm long, and opens distal to the hymeneal ring—one at 5 and the other at 7 o'clock on the vestibule. Following trauma or infection, either duct may swell and obstruct to form a cyst or, if infected, an abscess. In contrast, the minor vestibular glands are shallow glands lined by simple mucin-secreting epithelium and open along Hart line.

The paraurethral glands are a collective arborization of glands whose numerous small ducts open predominantly along the entire inferior aspect of the urethra. The two largest are called Skene glands, and their ducts typically lie distally and near the urethral meatus. Clinically, inflammation and duct obstruction of any of the paraurethral glands can lead to urethral diverticulum formation. The urethral opening or meatus is in the midline of the vestibule, 1 to 1.5 cm below the pubic arch, and a short distance above the vaginal opening.

■ Vagina and Hymen

In adult women, the hymen is a membrane of varying thickness that surrounds the vaginal opening more or less completely.

It is composed mainly of elastic and collagenous connective tissue, and both outer and inner surfaces are covered by nonkeratinized stratified squamous epithelium. The aperture of the intact hymen ranges in diameter from pinpoint to one that admits one or even two fingertips. As a rule, the hymen is torn at several sites during first coitus. However, identical tears may form by other penetration, for example, by tampons used during menstruation. The edges of the torn tissue soon reepithelialize. In pregnant women, the hymeneal epithelium is thick and rich in glycogen. Changes produced in the hymen by childbirth are usually readily recognizable. For example, over time, the hymen transforms into several nodules of various sizes, termed hymeneal or myrtiform caruncles.

Proximal to the hymen, the vagina is a musculomembranous tube that extends to the uterus and is interposed lengthwise between the bladder and the rectum (Fig. 2-4). Anteriorly, the vagina is separated from the bladder and urethra by connective tissue—the vesicovaginal septum. Posteriorly, between the lower portion of the vagina and the rectum, similar tissues together form the rectovaginal septum. The upper fourth of the vagina is separated from the rectum by the rectouterine pouch, also called the cul-de-sac or pouch of Douglas.

Normally, the anterior and posterior walls of the vaginal lumen lie in contact, with only a slight space intervening at the lateral margins. Vaginal length varies considerably, but commonly, the anterior wall measures 6 to 8 cm, whereas the posterior vaginal wall is 7 to 10 cm. The upper end of the vaginal vault is subdivided by the cervix into anterior, posterior, and two lateral fornices. Clinically, the internal pelvic organs usually can be palpated through the thin walls of these fornices.

The vaginal lining is composed of nonkeratinized stratified squamous epithelium and underlying lamina propria. In premenopausal women, this lining is thrown into numerous thin transverse ridges, known as rugae, which line the anterior and posterior vaginal walls along their length. Deep to this, a muscular layer contains smooth muscle, collagen, and elastin. Beneath this muscularis lies an adventitial layer consisting of collagen and elastin (Weber, 1997).

The vagina lacks glands. Instead, it is lubricated by a transudate that originates from the vaginal subepithelial capillary plexus and crosses the permeable epithelium (Kim, 2011). Due to increased vascularity during pregnancy, vaginal secretions are notably increased. At times, this may be confused with amniotic fluid leakage, and clinical differentiation of these two is described in Chapter 22 (p. 435).

After birth-related epithelial trauma and healing, fragments of stratified epithelium occasionally are embedded beneath the vaginal surface. Similar to its native tissue, this buried epithelium continues to shed degenerated cells and keratin. As a result, epidermal inclusion cysts, which are filled with keratin debris, may form. These are a common vaginal cyst.

The vagina has an abundant vascular supply. The proximal portion is supplied by the cervical branch of the uterine artery and by the vaginal artery. The latter may variably arise from the

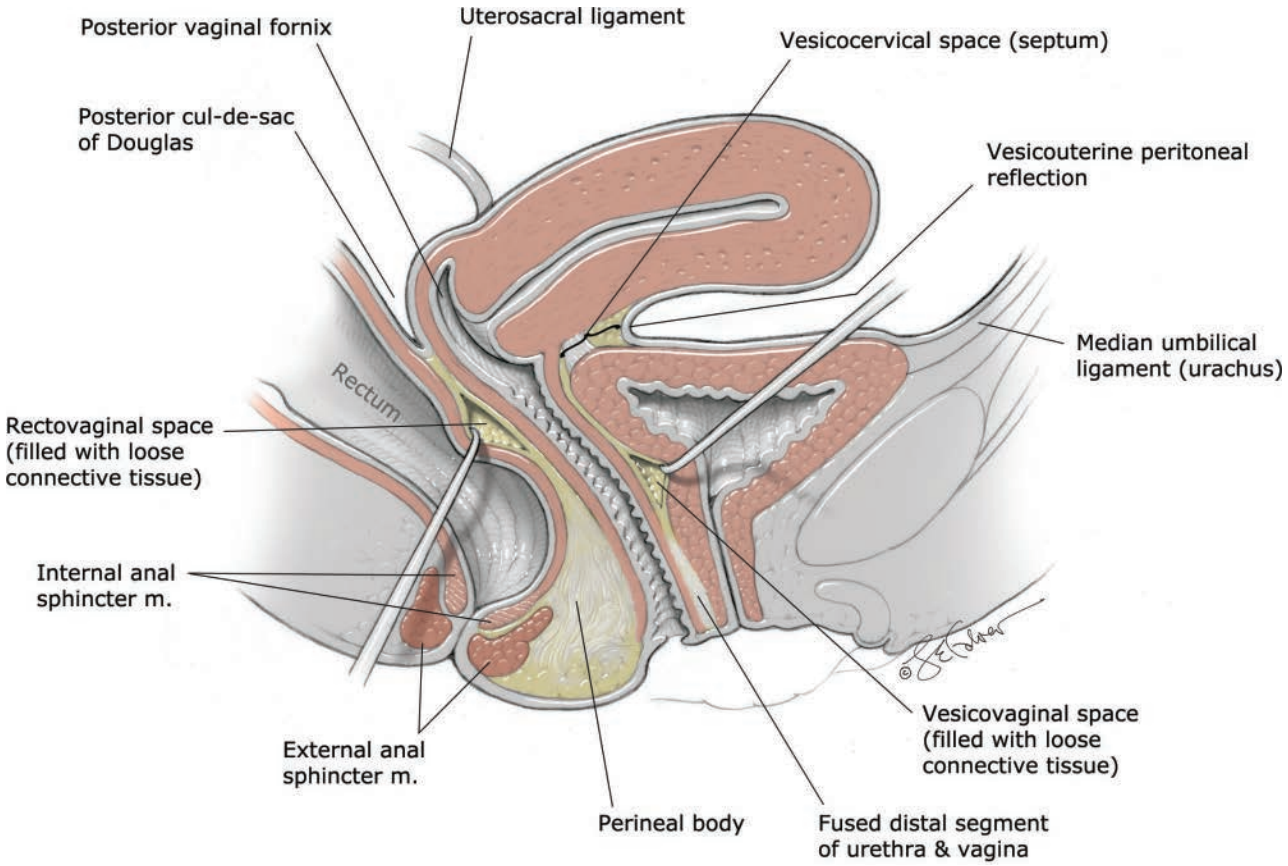

FIGURE 2-4 Vagina and surrounding anatomy. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

uterine or inferior vesical artery or directly from the internal iliac artery. The middle rectal artery contributes supply to the posterior vaginal wall, whereas the distal walls receive contributions from the internal pudendal artery. At each level, vessels supplying each side of the vagina course medially across the anterior or posterior vaginal wall and form midline anastomoses.

An extensive venous plexus also surrounds the vagina and follows the course of the arteries. Lymphatics from the lower third, along with those of the vulva, drain primarily into the inguinal lymph nodes. Those from the middle third drain into the internal iliac nodes, and those from the upper third drain into the external, internal, and common iliac nodes.

Perineum

This diamond-shaped area between the thighs has boundaries that mirror those of the bony pelvic outlet: the pubic symphysis anteriorly, ischiopubic rami and ischial tuberosities anterolaterally, sacrotuberous ligaments posterolaterally, and coccyx posteriorly. An arbitrary line joining the ischial tuberosities divides the perineum into an anterior triangle, also called the urogenital triangle, and a posterior triangle, termed the anal triangle.

The perineal body is a fibromuscular pyramidal mass found in the midline at the junction between these anterior and posterior triangles (Fig. 2-5). Also called the central tendon of the perineum, the perineal body sonographically measures 8 mm tall and 14 mm wide and thick (Santoro, 2016). It serves as the junction for several structures and provides significant perineal support (Shafik, 2007). Superficially, the bulbospongiosus, superficial transverse perineal, and external anal sphincter muscles converge on the perineal body. More deeply, the perineal membrane, portions of the pubococcygeus muscle, and internal anal sphincter contribute (Larson, 2010). The perineal body is incised by an episiotomy incision and is torn with second-, third-, and fourth-degree lacerations.

Superficial Space of the Anterior Triangle

This triangle is bounded by the pubic rami superiorly, the ischial tuberosities laterally, and the superficial transverse perineal muscles posteriorly. It is divided into superficial and deep spaces by the perineal membrane. This membranous partition is a dense fibrous sheet that was previously known as the inferior fascia of the urogenital diaphragm. The perineal membrane attaches laterally to the ischiopubic rami, medially to the distal third of the urethra and vagina, posteriorly to the perineal body, and anteriorly to the arcuate ligament of the pubis (see Fig. 2-5).

The superficial space of the anterior triangle is bounded deeply by the perineal membrane and superficially by Colles fascia. As noted earlier, Colles fascia is the continuation of Scarpa fascia onto the perineum. On the perineum, Colles fascia securely attaches laterally to the pubic rami and fascia lata of the thigh, inferiorly to the superficial transverse perineal muscle and inferior border of the perineal membrane, and medially to the urethra, clitoris, and vagina. As such, the superficial space of the anterior triangle is a relatively closed compartment.

This superficial pouch contains several important structures, which include the Bartholin glands, vestibular bulbs, clitoral body and crura, branches of the pudendal vessels and nerve, and the ischiocavernosus, bulbospongiosus, and superficial transverse perineal muscles. Of these muscles, the ischiocavernosus muscles each attach on their respective side to the medial aspect of the ischial tuberosity inferiorly and the ischiopubic ramus laterally. Anteriorly, each attaches to a clitoral crus and may help maintain clitoral erection by compressing the crus to obstruct venous drainage. The bilateral bulbospongiosus muscles overlie the vestibular bulbs and

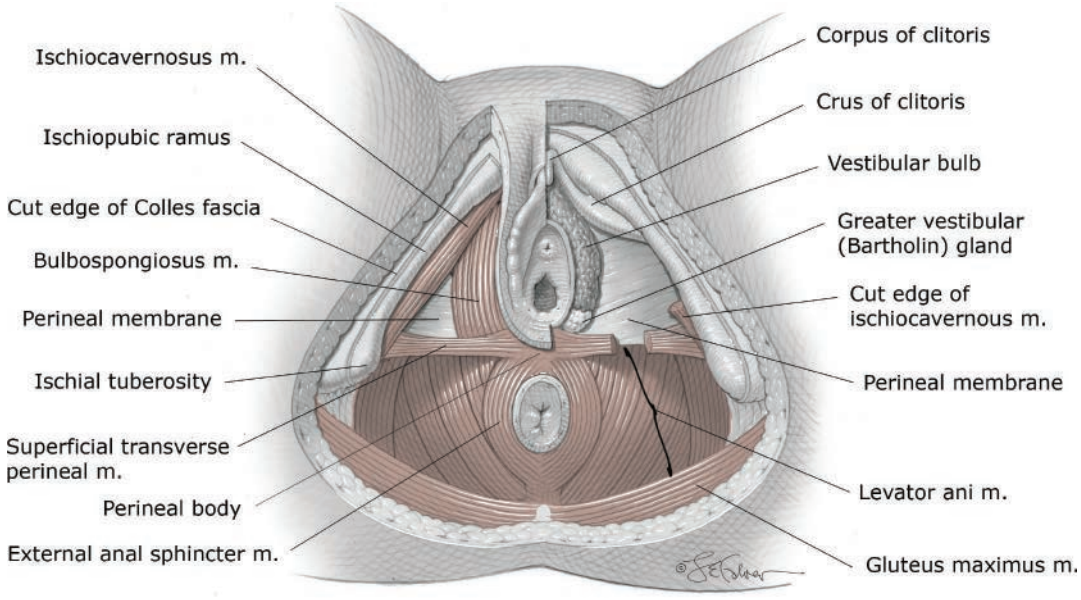

FIGURE 2-5 Superficial space of the anterior perineal triangle and posterior perineal triangle. Structures on the left side of the image can be seen after removal of Colles fascia. Those on the right side are noted after removal of the superficial muscles of the anterior triangle. (Modified with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

Bartholin glands. They attach to the body of the clitoris anteriorly and the perineal body posteriorly. The muscles constrict the vaginal lumen and aid release of secretions from the Bartholin glands. They also may contribute to clitoral erection by compressing the deep dorsal vein of the clitoris. The bulbospongiosus and ischiocavernosus muscles also pull the clitoris downward. Last, the superficial transverse perineal muscles are narrow strips that attach to the ischial tuberosities laterally and the perineal body medially. They may be attenuated or even absent, but when present, they contribute to the perineal body (Corton, 2016).

The vestibular bulbs are almond-shaped aggregations of veins that lie beneath the bulbospongiosus muscle on either side of the vestibule. They measure 3 to 4 cm long, 1 to 2 cm wide, and 0.5 to 1 cm thick. The bulbs terminate inferiorly at approximately the middle of the vaginal opening and extend upward toward the clitoris. Their anterior extensions merge in the midline, below the clitoral body. During childbirth, veins in the vestibular bulbs may be lacerated or even rupture to create a vulvar hematoma enclosed within the superficial space of the anterior triangle (Fig. 41-11, p. 765).

Deep Space of the Anterior Triangle

This space lies deep to the perineal membrane and extends up into the pelvis (Mirilas, 2004). In contrast to the superficial perineal space, the deep space is continuous superiorly with the pelvic cavity (Corton, 2005). It contains portions of urethra and vagina, certain portions of internal pudendal artery branches, and muscles of the striated urogenital sphincter complex (Fig. 2-6).

Urethra. The female urethra measures 3 to 4 cm and originates within the bladder trigone (p. 28). The distal two thirds of the urethra are fused with the anterior vaginal wall. The epithelial lining of the urethra changes from transitional epithelium proximally to nonkeratinized stratified squamous epithelium distally. The walls of the urethra consist of two layers of smooth muscle, an inner longitudinal and an outer circular. This is in turn surrounded by a circular layer of skeletal muscle referred to as the *sphincter urethrae* or *rhabdosphincter* (see Fig. 2-6). Approximately at the junction of the middle and lower third of the urethra, and just above or deep to the perineal membrane, two strap skeletal muscles called the *urethrovaginal sphincter* and *compressor urethrae* are found. Together with the sphincter urethrae, these constitute the *striated urogenital sphincter complex.* This complex supplies constant tonus and provides emergency reflex contraction to sustain continence.

Distal to the level of the perineal membrane, the walls of the urethra consist of fibrous tissue, serving as the nozzle that directs the urine stream. Here, the urethra has a prominent submucosal layer that is lined by hormonally sensitive stratified squamous epithelium. Within the submucosal layer on the dorsal (vaginal) surface of the urethra lie the paraurethral glands, described earlier (p. 17).

The urethra receives its blood supply from branches of the inferior vesical, vaginal, or internal pudendal arteries. Although still controversial, the pudendal nerve is believed to innervate the most distal part of the striated urogenital sphincter complex. Somatic efferent branches from S_2–S_4 that course along the inferior hypogastric plexus variably innervate the sphincter urethrae.

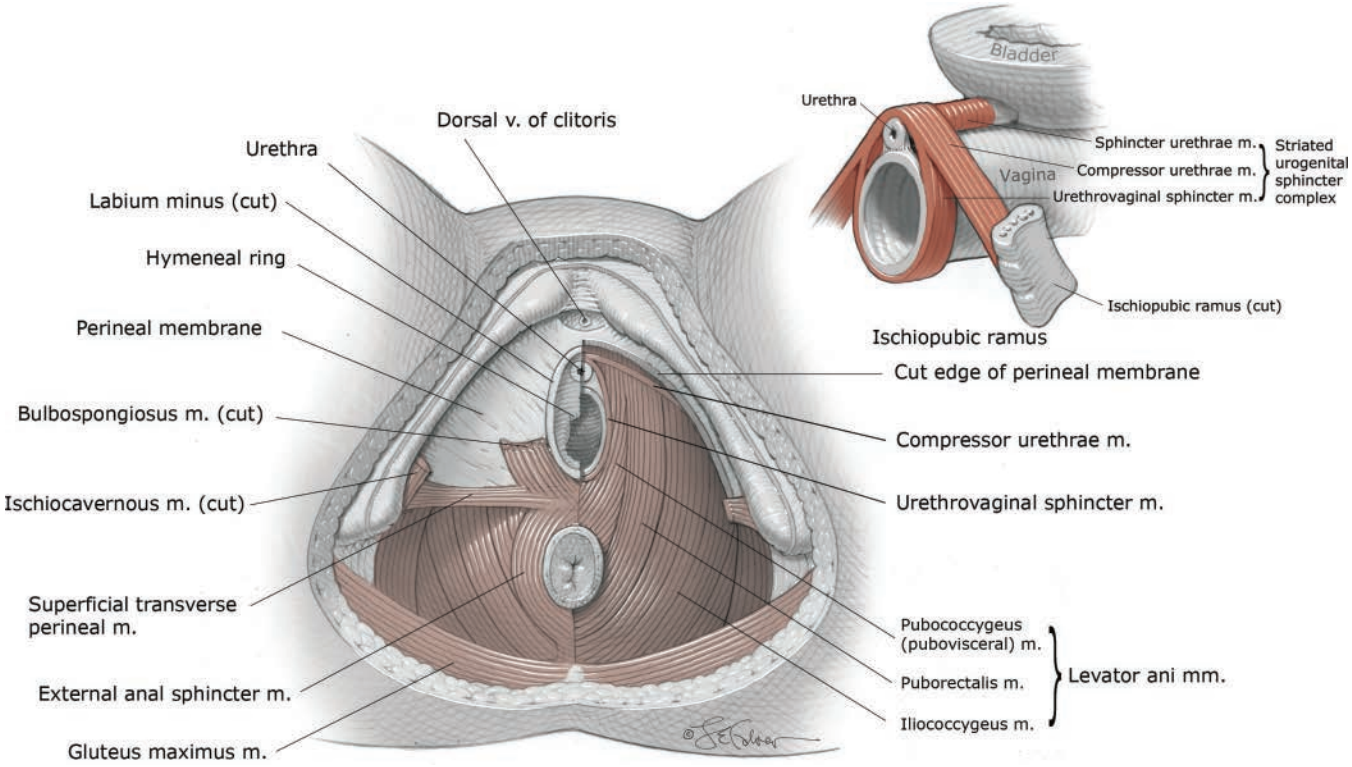

FIGURE 2-6 Deep space of anterior triangle of the perineum. Structures on the right side of the image can be seen after removal of the perineal membrane. Also shown are structures that attach to the perineal body: bulbospongiosus, superficial transverse perineal, external anal sphincter, and puboperinealis muscles as well as perineal membrane. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

Pelvic Diaphragm

Found deep to the anterior and posterior triangles, this broad muscular sling provides substantial support to the pelvic viscera. The pelvic diaphragm is composed of the levator ani and the coccygeus muscles. The levator ani, in turn, contains the pubococcygeus, puborectalis, and iliococcygeus muscles. The pubococcygeus muscle is also termed the pubovisceral muscle and is subdivided based on points of insertion and function. These include the pubovaginalis, puboperinealis, and puboanalis muscles, which insert into the vagina, perineal body, and anus, respectively (Kearney, 2004).

Vaginal birth conveys significant risk for damage to the levator ani or to its innervation (DeLancey, 2003; Weidner, 2006). Evidence supports that levator ani avulsion may predispose women to greater risk of pelvic organ prolapse (Dietz, 2008; Schwertner-Tiepelmann, 2012). For this reason, current research efforts are aimed at minimizing these injuries.

Posterior Triangle

This triangle contains the ischioanal fossae, anal canal, and anal sphincter complex, which consists of the internal anal sphincter, external anal sphincter, and puborectalis muscle. Branches of the pudendal nerve and internal pudendal vessels are also found within this triangle.

Ischioanal Fossae. Also known as ischiorectal fossae, these two fat-filled wedge-shaped spaces are found on either side of the anal canal and comprise the bulk of the posterior triangle (Fig. 2-7). Each fossa has skin as its superficial base, whereas its deep apex is formed by the junction of the levator ani and obturator internus muscles. Other borders include: laterally, the obturator internus muscle fascia and ischial tuberosity; inferomedially, the anal canal and sphincter complex; superomedially, the inferior fascia of the downwardly sloping levator ani; posteriorly, the gluteus maximus muscle and sacrotuberous ligament; and anteriorly, the inferior border of the anterior triangle.

The fat found within each fossa provides support to surrounding organs yet allows rectal distention during defecation and vaginal stretching during delivery. Clinically, injury to vessels in the posterior triangle can lead to hematoma formation in the ischioanal fossa, and the potential for large accumulation in these easily distensible spaces. Moreover, the two fossae communicate dorsally, behind the anal canal. This can be especially important because an episiotomy infection or hematoma may extend from one fossa into the other.

Anal Canal. This distal continuation of the rectum begins at the level of levator ani attachment to the rectum and ends at the anal skin. Along this 4- to 5-cm length, the mucosa consists of columnar epithelium in the uppermost portion. However, at the pectinate line, also termed dentate line, simple stratified squamous epithelium begins and continues to the anal verge. At the verge, keratin and skin adnexa join the squamous epithelium.

The anal canal has several tissue layers (see Fig. 2-7). Inner layers include the anal mucosa, the internal anal sphincter, and an intersphincteric space that contains continuation of the rectum's longitudinal smooth muscle layer. An outer layer contains the puborectalis muscle as its cephalad component and the external anal sphincter caudally.

Within the anal canal, three highly vascularized submucosal arteriovenous plexuses, termed anal cushions, aid complete closure of the canal and fecal continence when apposed. Increasing uterine size, excessive straining, and hard stool create increased pressure that ultimately leads to degeneration and subsequent laxity of the cushion's supportive connective tissue base. These cushions then protrude into and downward through the anal canal. This leads to venous engorgement within the cushions—now termed hemorrhoids. Venous stasis results in inflammation, erosion of the cushion's epithelium, and then bleeding.

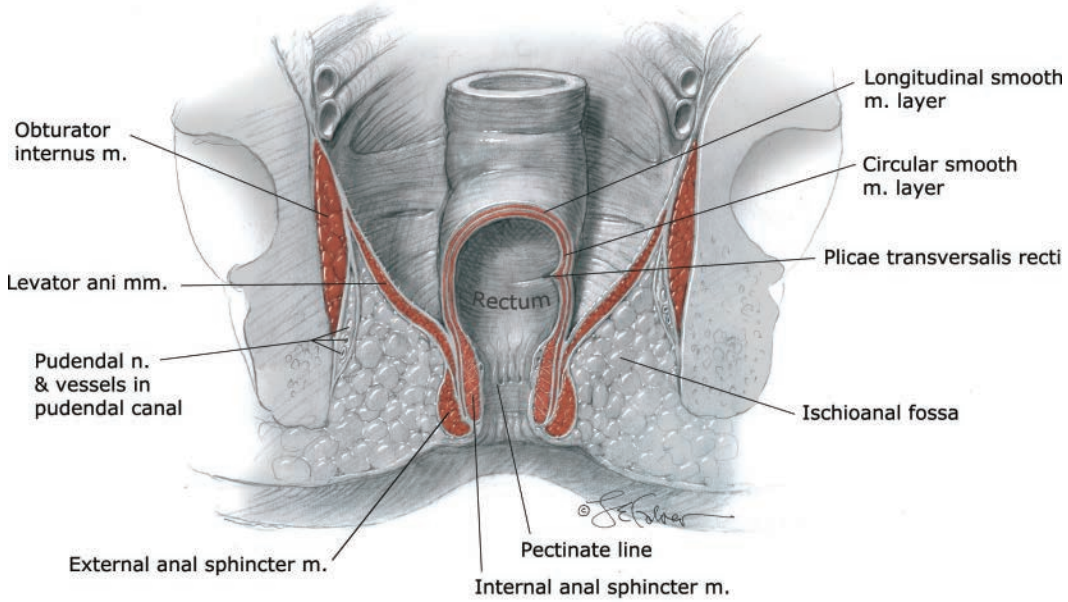

FIGURE 2-7 Anal canal and ischioanal fossa. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

External hemorrhoids are those that arise distal to the pectinate line. They are covered by stratified squamous epithelium and receive sensory innervation from the inferior rectal nerve. Accordingly, pain and a palpable mass are typical complaints. Following resolution, a hemorrhoidal tag may remain and is composed of redundant anal skin and fibrotic tissue. In contrast, internal hemorrhoids are those that form above the pectinate line and are covered by insensitive anorectal mucosa. These may prolapse or bleed but rarely become painful unless they undergo thrombosis or necrosis.

Anal Sphincter Complex. Two sphincters surround the anal canal to provide fecal continence—the external and internal anal sphincters. Both lie near the vagina and may be torn during vaginal delivery. The internal anal sphincter (IAS) is a distal continuation of the rectal circular smooth muscle layer. It receives predominantly parasympathetic fibers, which pass through the pelvic splanchnic nerves. Along its length, this sphincter is supplied by the superior, middle, and inferior rectal arteries. The IAS contributes the bulk of anal canal resting pressure for fecal continence and relaxes prior to defecation. The IAS measures 3 to 4 cm in length, and at its distal margin, it overlaps the external sphincter for 1 to 2 cm (DeLancey, 1997). The distal site at which this overlap ends, called the intersphincteric groove, is palpable on digital examination.

In contrast, the external anal sphincter (EAS) is a striated muscle ring that anteriorly attaches to the perineal body and posteriorly connects to the coccyx via the anococcygeal ligament. The EAS maintains a constant resting contraction to aid continence, provides additional squeeze pressure when continence is threatened, yet relaxes for defecation. The external sphincter receives blood supply from the inferior rectal artery, which is a branch of the internal pudendal artery. Somatic motor fibers from the inferior rectal branch of the pudendal nerve supply innervation. Clinically, the IAS and EAS may be involved in third- and fourth-degree lacerations during vaginal delivery, and reunion of these rings is integral to defect repair (Chap. 27, p. 532).

Pudendal Nerve

This is formed from the anterior rami of S_{2-4} spinal nerves. It courses between the piriformis and coccygeus muscles and exits through the greater sciatic foramen at a location posterior to the sacrospinous ligament and just medial to the ischial spine (Barber, 2002; Maldonado, 2015). Thus, when injecting local anesthetic for a pudendal nerve block, the ischial spine serves an identifiable landmark (Chap. 25, p. 489). The pudendal nerve then runs beneath the sacrospinous ligament and above the sacrotuberous ligament as it reenters the lesser sciatic foramen to course along the obturator internus muscle. Atop this muscle, the nerve lies within the pudendal canal, also known as Alcock canal, which is formed by splitting of the obturator internus investing fascia (Shafik, 1999). In general, the pudendal nerve is relatively fixed as it courses behind the sacrospinous ligament and within the pudendal canal. Accordingly, it may be at risk of stretch injury during downward displacement of the pelvic floor during childbirth (Lien, 2005).

The pudendal nerve leaves this canal to enter the perineum and divides into three terminal branches (Fig. 2-8). The first of these, the dorsal nerve of the clitoris, runs between the ischiocavernosus muscle and perineal membrane to supply the clitoral glans (Ginger, 2011b). Second, the perineal nerve runs superficial to the perineal membrane (Montoya, 2011). It divides into

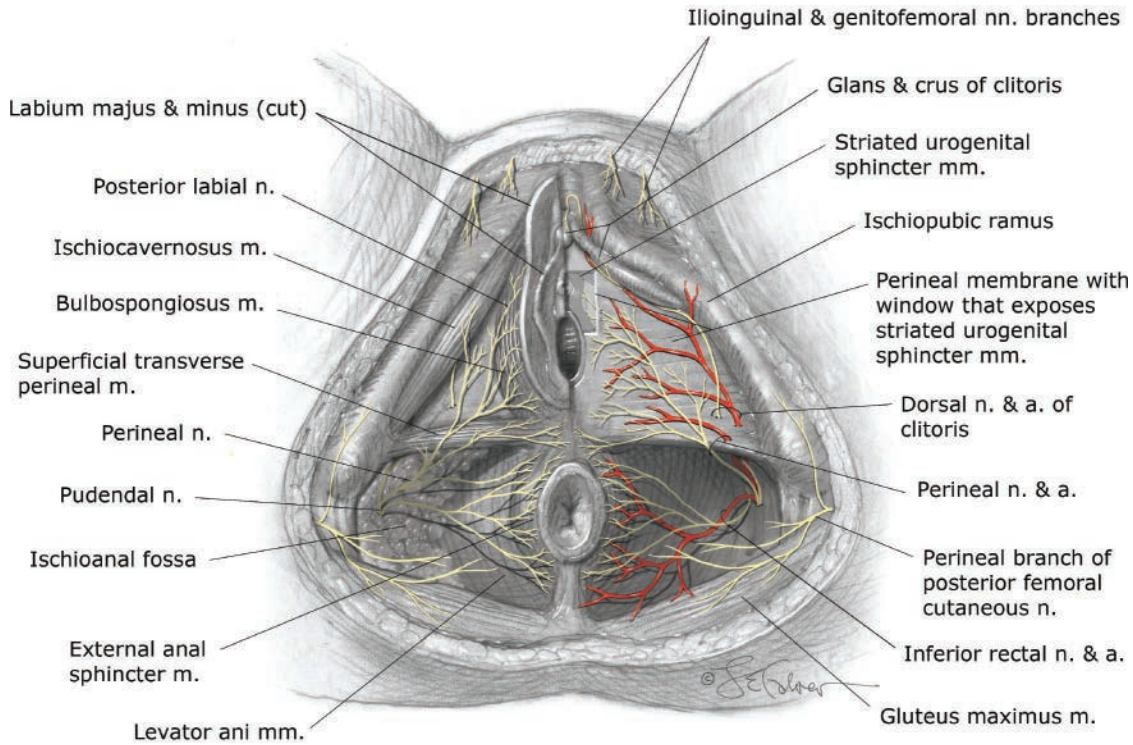

FIGURE 2-8 Pudendal nerve and vessels. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

posterior labial branches and muscular branches, which serve the labial skin and the anterior perineal triangle muscles, respectively. Last, the inferior rectal branch runs through the ischioanal fossa to supply the external anal sphincter, the anal mucosa, and the perianal skin (Mahakkanukrauh, 2005). The major blood supply to the perineum is via the internal pudendal artery, and its branches mirror the divisions of the pudendal nerve.

INTERNAL GENERATIVE ORGANS

Uterus

The nonpregnant uterus lies in the pelvic cavity between the bladder anteriorly and the rectum posteriorly. Almost the entire posterior wall of the uterus is covered by serosa, that is, visceral peritoneum (Fig. 2-9). The lower portion of this peritoneum forms the anterior boundary of the rectouterine cul-de-sac, or pouch of Douglas. Only the upper portion of the anterior uterine wall is covered by visceral peritoneum. At the caudal border of this portion, the peritoneum reflects forward onto the bladder dome to create the vesicouterine pouch. As a result, the lower portion of the anterior uterine wall is separated from the posterior wall of the bladder only by a well-defined loose connective tissue layer—the vesicouterine space. Clinically, during cesarean delivery, the peritoneum of the vesicouterine pouch is sharply incised, and the vesicouterine space is entered. Dissection caudally within this space lifts the bladder safely off the lower uterine segment for hysterotomy and delivery (Chap. 30, p. 573).

The uterus is pear shaped and consists of two major but unequal parts. The upper, larger portion is the body or corpus, whereas the lower smaller cervix projects into the vagina. The isthmus is the union site of these two. It is of special obstetrical significance because it forms the lower uterine segment during pregnancy. At each superolateral margin of the body is a uterine cornu, from which a fallopian tube emerges. This area also contains the origins of the round and ovarian ligaments. Between the points of fallopian tube insertion is the convex upper uterine segment termed the fundus.

The bulk of the uterine body, but not the cervix, is muscle. The inner surfaces of the anterior and posterior walls lie almost in contact, and the cavity between these walls forms a mere slit. The nulligravid uterus measures 6 to 8 cm in length compared with 9 to 10 cm in multiparas. The uterus averages 60 g and typically weighs more in parous women (Langlois, 1970; Sheikhazadi, 2010).

Pregnancy stimulates remarkable uterine growth due to muscle fiber hypertrophy. The uterine fundus, a previously flattened convexity between tubal insertions, now becomes dome shaped. Moreover, the round ligaments appear to insert at the junction of the middle and upper thirds of the organ. The fallopian tubes elongate, but the ovaries grossly appear unchanged.

Cervix

This portion of the uterus is cylindrical and has small apertures at each end—the internal and external cervical ora. The endocervical canal runs through the cervix and connects these ora. The cervix is divided into upper and lower portions by the vagina's attachment to its outer surface. The upper portion—the portio supravaginalis—begins at the internal os, which corresponds to the level at which the peritoneum is reflected up onto the bladder (Fig. 2-10). The lower cervical portion protrudes into the vagina as the portio vaginalis.

Before childbirth, the external cervical os is a small, regular, oval opening. After labor, especially vaginal childbirth, the orifice

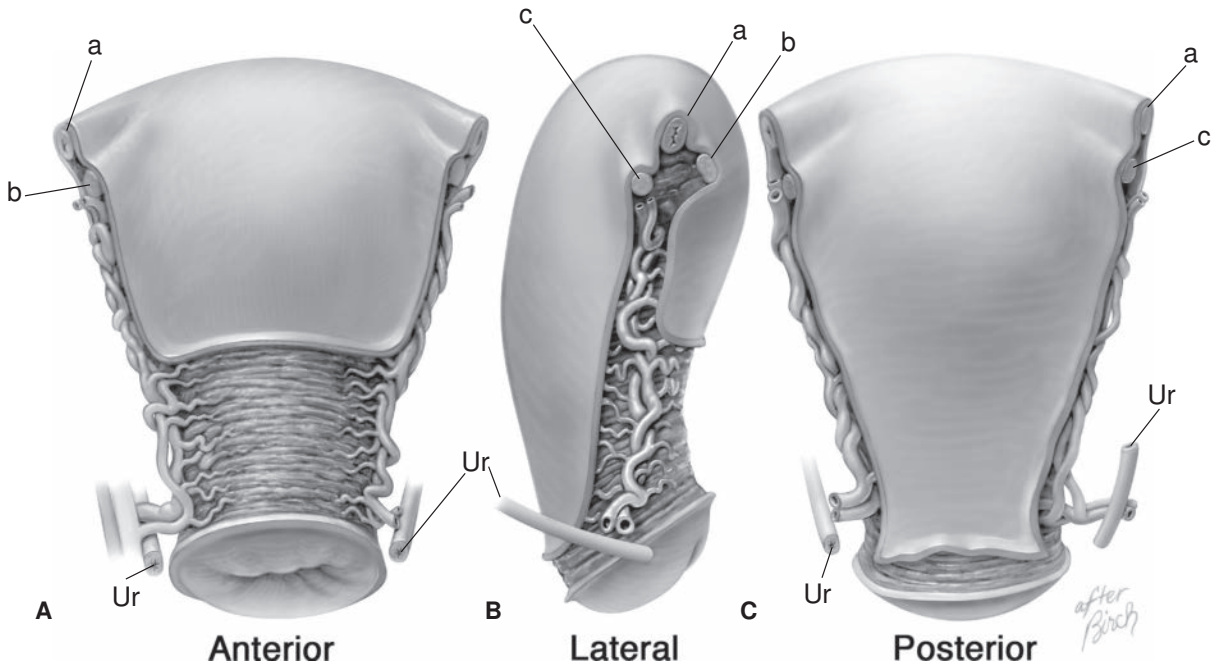

FIGURE 2-9 Anterior **(A)**, right lateral **(B)**, and posterior **(C)** views of the uterus of an adult woman. a = oviduct; b = round ligament; c = ovarian ligament; Ur = ureter.

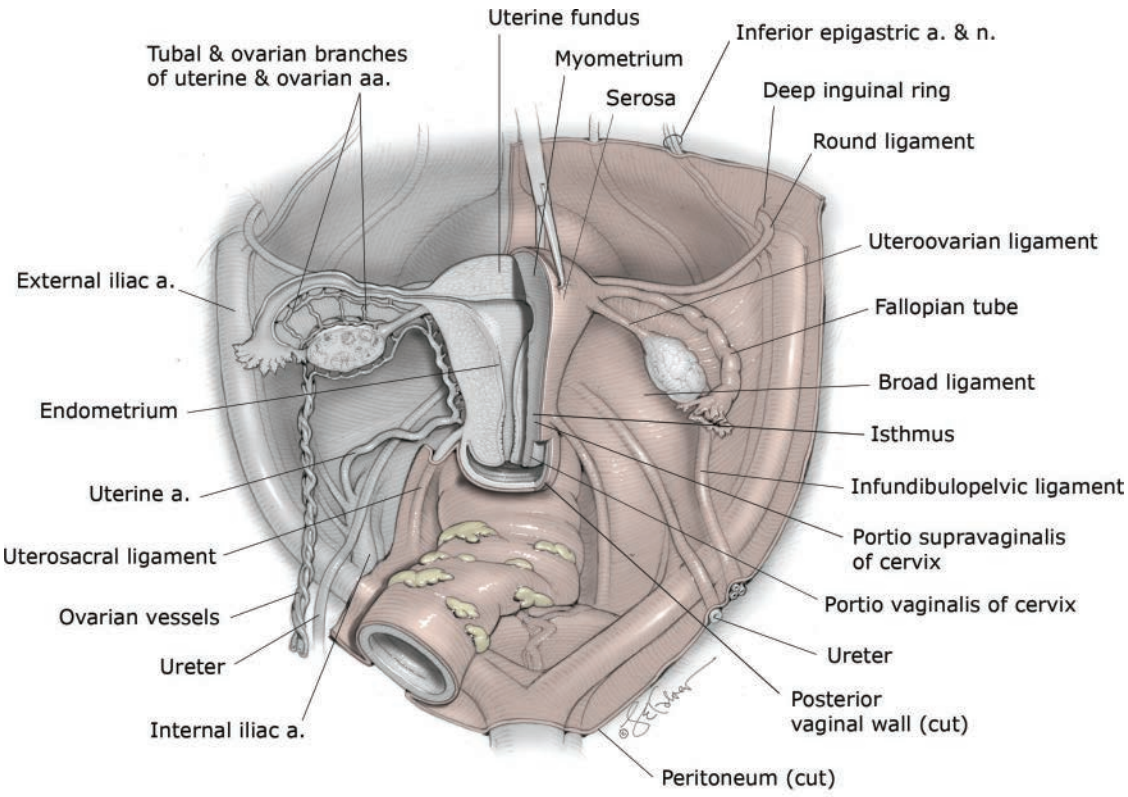

FIGURE 2-10 Uterus, adnexa, and associated anatomy. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

is converted into a transverse slit that is divided such that there are the so-called anterior and posterior cervical lips. If torn deeply during labor or delivery, the cervix may heal in such a manner that it appears irregular, nodular, or stellate (Fig. 36-1, p. 653).

The cervical surface that radially surrounds the external os is called the ectocervix and is lined predominantly by nonkeratinized stratified squamous epithelium. In contrast, the endocervical canal is covered by a single layer of mucin-secreting columnar epithelium, which creates deep cleftlike infoldings or "glands." Commonly during pregnancy, the endocervical epithelium moves out and onto the ectocervix in a physiological process termed eversion (Chap. 4, p. 51).

The cervical stroma is composed mainly of collagen, elastin, and proteoglycans, but very little smooth muscle. As described in Chapter 21 (p. 409), changes in the amount, composition, and orientation of these components lead to cervical ripening prior to labor onset. In early pregnancy, increased vascularity within the cervix stroma beneath the epithelium creates an ectocervical blue tint that is characteristic of Chadwick sign. Cervical edema leads to softening—Goodell sign, whereas isthmic softening is Hegar sign.

Myometrium and Endometrium

Most of the uterus is composed of myometrium, which contains smooth muscle bundles united by connective tissue with many elastic fibers. Interlacing myometrial fibers surround myometrial vessels and contract to compress these. This anatomy allows hemostasis at the placental site during the third stage of labor.

The number of myometrial muscle fibers varies by location (Schwalm, 1966). Levels progressively diminish caudally such that, in the cervix, muscle makes up only 10 percent of the tissue mass. The uterine body's inner wall has relatively more muscle than its outer layers. And, in the anterior and posterior walls, the muscle content is greater than in the lateral walls. During pregnancy, the upper myometrium undergoes marked hypertrophy, but cervical muscle content does not change significantly.

The uterine cavity is lined with endometrium, which is composed of an overlying epithelium, invaginating glands, and a supportive, vascular stroma. As discussed in Chapter 5 (p. 83), the endometrium varies greatly throughout the menstrual cycle. This layer is divided into a functionalis layer, which is sloughed with menses, and a basalis layer, which serves to regenerate the functionalis layer following each menses. During pregnancy, the endometrium is termed decidua and undergoes dramatic hormonally driven alterations.

Ligaments

Several ligaments extend from the uterine surface toward the pelvic sidewalls and include the round, broad, cardinal, and uterosacral ligaments (Figs. 2-10 and 2-11). Despite their appellation, the round and broad ligaments provide no substantial uterine support, which contrasts with the cardinal and uterosacral ligaments.

The round ligament originates somewhat below and anterior to the origin of the fallopian tubes. Clinically, this orientation can aid fallopian tube identification during puerperal sterilization.

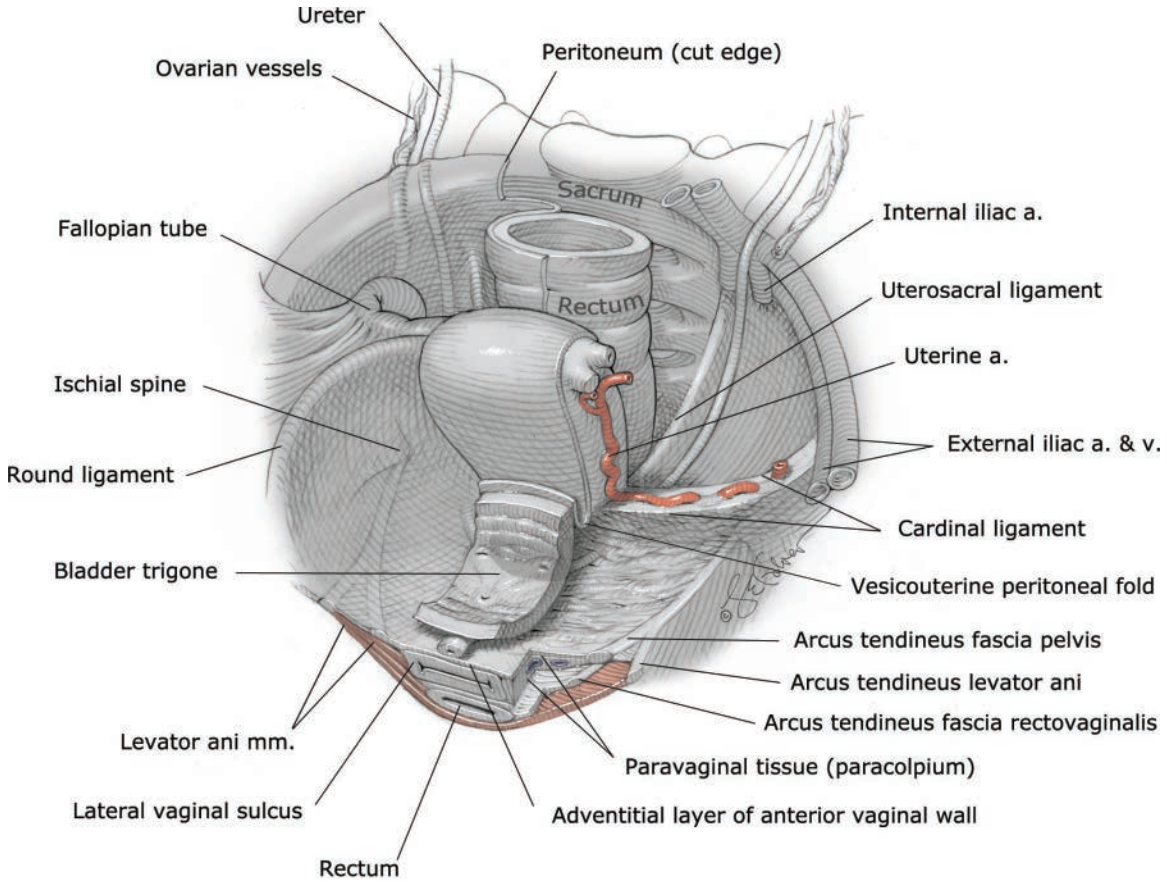

FIGURE 2-11 Pelvic viscera and their connective tissue support. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

This is important if pelvic adhesions limit tubal mobility and thus, hinder fimbria visualization and tubal confirmation prior to ligation. Each round ligament extends laterally and down into the inguinal canal, through which it passes, to terminate in the upper portion of the ipsilateral labium majus. Sampson artery, a branch of the uterine artery, runs within this ligament. In nonpregnant women, the round ligament varies from 3 to 5 mm in diameter and is composed of smooth muscle bundles separated by fibrous tissue septa (Mahran, 1965). During pregnancy, these ligaments undergo considerable hypertrophy and increase appreciably in both length and diameter.

The broad ligaments are two winglike structures that extend from the lateral uterine margins to the pelvic sidewalls. Each broad ligament consists of a double-layer drape of peritoneum. The anterior and posterior layers of this drape are termed the anterior and posterior leaves, respectively. In forming the broad ligament, this peritoneum folds over structures extending from each cornu. Peritoneum that folds over the fallopian tube is termed the mesosalpinx, that around the round ligament is the mesoteres, and that over the ovarian ligament is the mesovarium. Peritoneum that extends beneath the fimbriated end of the fallopian tube toward the pelvic wall forms the suspensory ligament or the infundibulopelvic ligament of the ovary. This contains nerves and the ovarian vessels, and during pregnancy, these vessels, especially the venous plexuses, are dramatically enlarged. Specifically, the diameter of the ovarian vascular pedicle increases from 0.9 cm to reach 2.6 cm at term (Hodgkinson, 1953).

The cardinal ligament—also called the transverse cervical ligament or Mackenrodt ligament—anchors medially to the uterus and upper vagina. The cardinal ligament is the thick base of the broad ligament. As such, during cesarean hysterectomy, sturdy clamps and suture are required for its transection and ligation.

Each uterosacral ligament originates with a posterolateral attachment to the supravaginal portion of the cervix and inserts into the fascia over the sacrum, with some variations (Ramanah, 2012; Umek, 2004). These ligaments are composed of connective tissue, small bundles of vessels and nerves, and some smooth muscle. Covered by peritoneum, these ligaments form the lateral boundaries of the pouch of Douglas.

The term parametrium is used to describe the connective tissues adjacent and lateral to the uterus within the broad ligament. Paracervical tissues are those adjacent to the cervix, whereas paracolpium is that tissue lateral to the vaginal walls.

■ Pelvic Blood Supply

During pregnancy, there is marked hypertrophy of the uterine vasculature, which is supplied principally from the uterine and ovarian arteries (see Fig. 2-10). The uterine artery, a main branch of the internal iliac artery—previously called the hypogastric artery—enters the base of the broad ligament. The uterine artery courses medially to the lateral side of the uterus. Approximately 2 cm lateral to the cervix, the uterine artery crosses over the ureter. This proximity is of great

surgical significance, as the ureter may be injured or ligated during hysterectomy when the uterine vessels are clamped and ligated.

Once the uterine artery has reached the supravaginal portion of the cervix, it divides. The smaller cervicovaginal artery supplies blood to the lower cervix and upper vagina. The main uterine artery branch turns abruptly upward and travels cephalad along the lateral margin of the uterus. Along its path, this main artery provides a branch of considerable size to the upper cervix and then numerous other medial branches serially penetrate the body of the uterus to form the arcuate arteries. As indicated by the name, each branch arches across the organ by coursing within the myometrium just beneath the serosal surface. Arcuate vessels from each side anastomose at the uterine midline. Radial artery branches originate at right angles from the arcuate arteries and travel inward through the myometrium, enter the endometrium/decidua, and branch there to become either basal arteries or coiled spiral arteries. The spiral arteries supply the functionalis layer. Also called the straight arteries, the basal arteries extend only into the basalis layer.

As the uterine artery courses cephalad, it gives rise to Sampson artery of the round ligament. Just before the main uterine artery vessel reaches the fallopian tube, it divides into three terminal branches. The ovarian branch of the uterine artery forms an anastomosis with the terminal branch of the ovarian artery; the tubal branch makes its way through the mesosalpinx and supplies part of the fallopian tube; and the fundal branch penetrates the uppermost uterus.

In addition to the uterine artery, the uterus receives blood supply from the ovarian artery (see Fig. 2-10). This artery is a direct branch of the aorta and enters the broad ligament through the infundibulopelvic ligament. At the ovarian hilum, it divides into smaller branches that enter the ovary. As the ovarian artery runs along the hilum, it also sends several branches through the mesosalpinx to supply the fallopian tubes. Its main stem, however, traverses the entire length of the broad ligament toward the uterine cornu. Here, it forms an anastomosis with the ovarian branch of the uterine artery. This dual uterine blood supply creates a vascular reserve to prevent uterine ischemia if ligation of the uterine or internal iliac artery is performed to control postpartum hemorrhage.

Uterine veins accompany their respective arteries. As such, the arcuate veins unite to form the uterine vein, which empties into the internal iliac vein and then the common iliac vein. Some of the blood from the upper uterus, the ovary, and the upper part of the broad ligament is collected by several veins. Within the broad ligament, these veins form the large pampiniform plexus that terminates in the ovarian vein. From here, the right ovarian vein empties into the vena cava, whereas the left ovarian vein empties into the left renal vein.

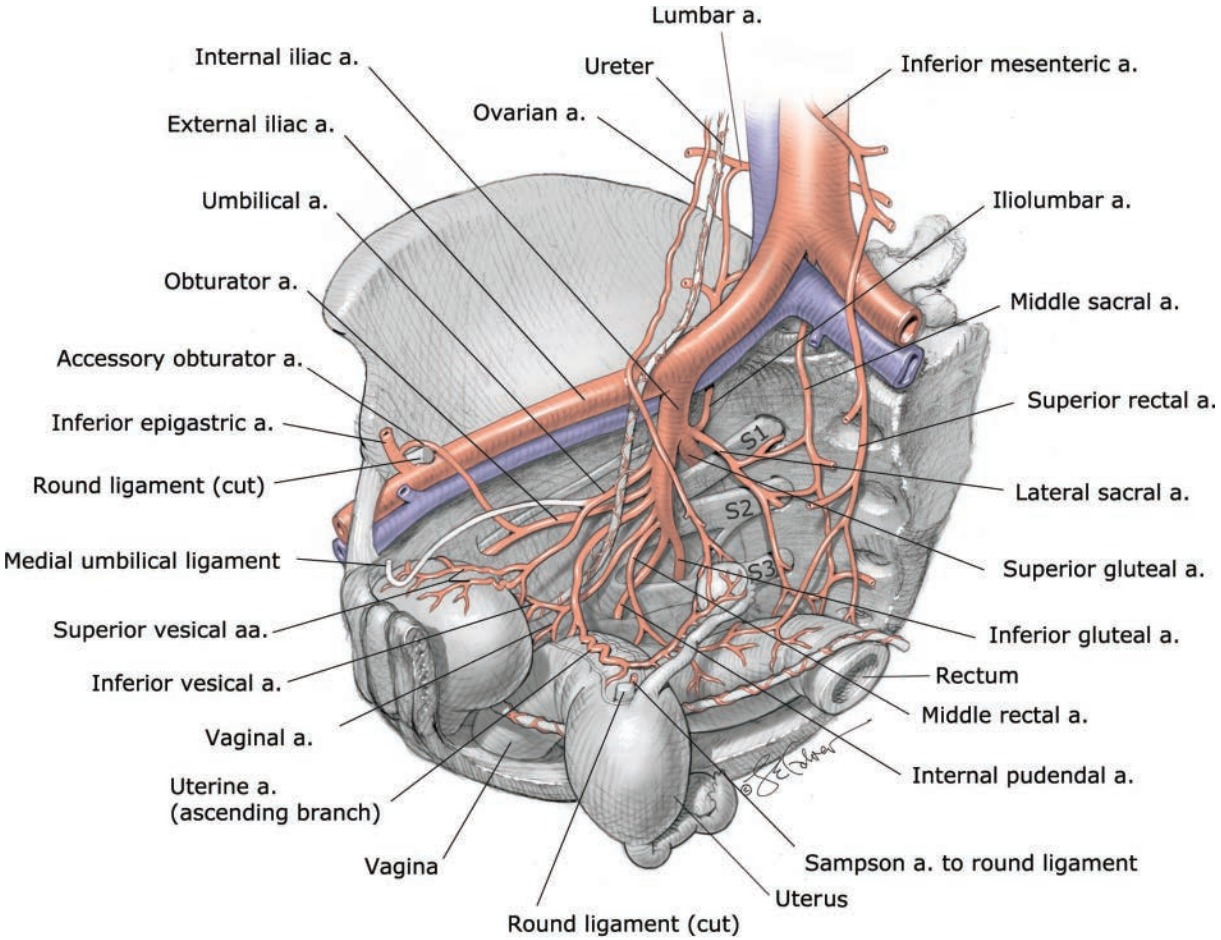

FIGURE 2-12 Pelvic arteries. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

Blood supply to the pelvis is predominantly provided by branches of the internal iliac artery (Fig. 2-12). These branches are organized into anterior and posterior divisions, and subsequent branches are highly variable between individuals. The anterior division provides blood supply to the pelvic organs and perineum and includes the inferior gluteal, internal pudendal, middle rectal, vaginal, uterine, and obturator arteries, as well as the umbilical artery and its continuation as the superior vesical artery. The posterior division branches extend to the buttock and thigh and include the superior gluteal, lateral sacral, and iliolumbar arteries. For this reason, during internal iliac artery ligation, many advocate ligation distal to the posterior division to avoid compromised blood flow to the areas supplied by this division (Bleich, 2007).

Pelvic Lymphatics

The lymphatics from the uterine corpus are distributed to two groups of nodes. One set of vessels drains into the internal iliac nodes. The other set, after joining lymphatics from the ovarian region, terminates in the paraaortic lymph nodes. Lymphatics from the cervix terminate mainly in the internal iliac nodes, which are situated near the bifurcation of the common iliac vessels.

Pelvic Innervation

As a brief review, the peripheral nervous system is divided into a somatic division, which innervates skeletal muscle, and an autonomic division, which innervates smooth muscle, cardiac muscle, and glands. Pelvic visceral innervation is predominantly autonomic, which is further divided into sympathetic and parasympathetic components.

Sympathetic innervation to pelvic viscera begins with the superior hypogastric plexus, also termed the presacral nerve (Fig. 2-13). Beginning below the aortic bifurcation and extending downward retroperitoneally, this plexus is formed by sympathetic fibers arising from spinal levels T_{10} through L_2. At the level of the sacral promontory, this superior hypogastric plexus divides into a right and a left hypogastric nerve, which run downward along the pelvis sidewalls (Ripperda, 2015).

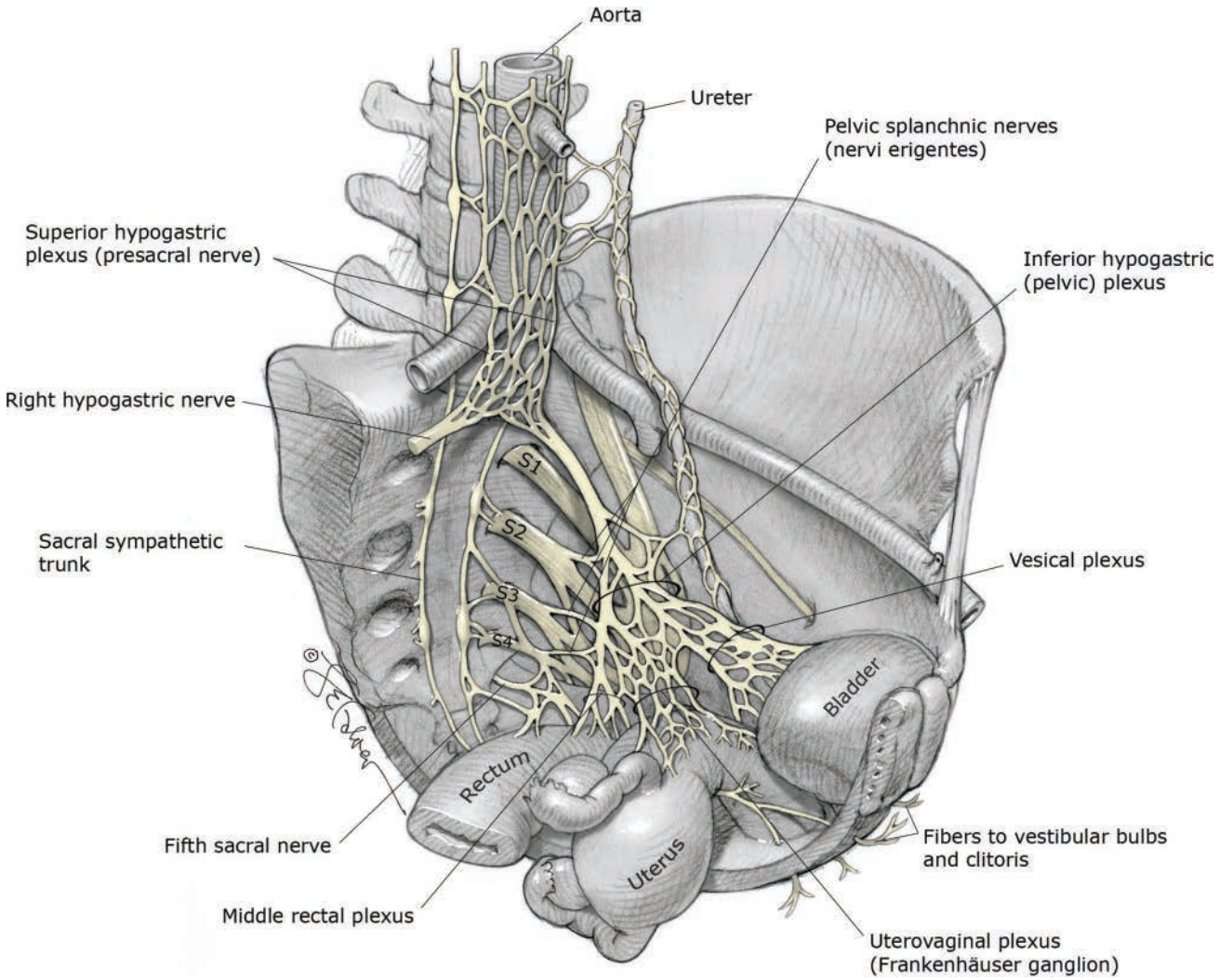

FIGURE 2-13 Pelvic innervation. (Reproduced with permission from Corton MM: Anatomy. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw-Hill Education, 2016.)

In contrast, parasympathetic innervation to the pelvic viscera derives from neurons at spinal levels S_2 through S_4. Their axons exit as part of the anterior rami of the spinal nerves for those levels. These combine on each side to form the pelvic splanchnic nerves, also termed nervi erigentes.

Blending of the two hypogastric nerves (sympathetic) and the two pelvic splanchnic nerves (parasympathetic) gives rise to the inferior hypogastric plexus, also termed the pelvic plexus. This retroperitoneal plaque of nerves lies at the S_4 and S_5 level (Spackman, 2007). From here, fibers of this plexus accompany internal iliac artery branches to their respective pelvic viscera. Thus, the inferior hypogastric plexus divides into three plexuses. The vesical plexus innervates the bladder, and the middle rectal plexus travels to the rectum. The uterovaginal plexus, also termed Frankenhäuser plexus, reaches the proximal fallopian tubes, uterus, and upper vagina. Extensions of the inferior hypogastric plexus also reach the perineum along the vagina and urethra to innervate the clitoris and vestibular bulbs (Montoya, 2011). Of these, the uterovaginal plexus is composed of variably sized ganglia, but particularly of a large ganglionic plate that is situated on either side of the cervix, proximate to the uterosacral and cardinal ligaments (Ramanah, 2012).

For the uterus, most of its afferent sensory fibers ascend through the inferior hypogastric plexus and enter the spinal cord via T_{10} through T_{12} and L_1 spinal nerves. These transmit the painful stimuli of contractions to the central nervous system. For the cervix and upper part of the birth canal, sensory nerves pass through the pelvic splanchnic nerves to the second, third, and fourth sacral nerves. Last, those from the lower portion of the birth canal pass primarily through the pudendal nerve. Anesthetic blocks used during delivery target these levels of innervation.

Ovaries

Along the pelvic sidewall, each ovary usually rests in the ovarian fossa of Waldeyer, which is a slight depression between the external and internal iliac vessels. During childbearing years, ovaries variably measure 2.5 to 5 cm in length, 1.5 to 3 cm in width, and 0.6 to 1.5 cm in thickness.

The ovarian ligament, also called the uteroovarian ligament, originates from the upper posterolateral portion of the uterus, just beneath the tubal insertion level, and extends to the uterine pole of the ovary (see Fig. 2-10). Measuring a few centimeters long and 3 to 4 mm in diameter, this ligament is made up of muscle and connective tissue and is covered by peritoneum—the mesovarium. Blood supply reaches the ovary through this double-layered mesovarium to enter the ovarian hilum.

The ovary consists of an outer cortex and inner medulla. In young women, the cortex is smooth, has a dull white surface, and is lined by single layer of cuboidal epithelium, the germinal epithelium of Waldeyer. This epithelium is supported by a connective tissue condensation, the tunica albuginea. Beneath this, the ovarian cortex contains oocytes and developing follicles. The medulla is composed of loose connective tissue, numerous arteries and veins, and a small amount of smooth muscle fibers.

The ovaries are supplied with both sympathetic and parasympathetic nerves. The sympathetic nerves are derived primarily from the ovarian plexus that accompanies the ovarian vessels and originates in the renal plexus. Others are derived from the plexus that surrounds the ovarian branch of the uterine artery. Parasympathetic input is from the vagus nerve. Sensory afferents follow the ovarian artery and enter at T_{10} spinal cord level.

Fallopian Tubes

Also called oviducts, these serpentine tubes extend laterally 8 to 14 cm from the uterine cornua. They are anatomically classified along their length as an interstitial portion, isthmus, ampulla, and infundibulum (Fig. 2-14). Most proximal, the interstitial portion is embodied within the uterine muscular wall. Next, the narrow 2- to 3-mm wide isthmus widens gradually into the 5- to 8-mm wide ampulla. Last, the infundibulum is the funnel-shaped fimbriated distal extremity of the tube, which opens into the abdominal cavity. These latter three extrauterine portions are covered by the mesosalpinx at the superior margin of the broad ligament.

In cross section, the extrauterine fallopian tube contains a mesosalpinx, myosalpinx, and endosalpinx. The outer of these, the mesosalpinx, is a single-cell mesothelial layer functioning as visceral peritoneum. In the myosalpinx, smooth muscle is arranged in an inner circular and an outer longitudinal layer. The tubal musculature undergoes rhythmic contractions constantly, the rate of which varies with cyclical ovarian hormonal changes.

The tubal mucosa or endosalpinx is a single layer of columnar epithelium composed of ciliated, secretory, and intercalary cells resting on a sparse lamina propria. Clinically, its close proximity to the underlying myosalpinx contributes to easy invasion by ectopic trophoblast. The tubal mucosa is arranged in longitudinal folds that become progressively more complex toward the fimbria. In the ampulla, the lumen is occupied almost completely by the arborescent mucosa. The current produced by the tubal cilia is such that the direction of flow is toward the uterine cavity. Tubal peristalsis created by cilia and muscular layer contraction is believed to be an important factor in ovum transport (Croxatto, 2002).

The tubes are supplied richly with elastic tissue, blood vessels, and lymphatics. Their sympathetic innervation is extensive, in contrast to their parasympathetic innervation. This nerve supply derives partly from the ovarian plexus and partly from the uterovaginal plexus. Sensory afferent fibers ascend to T_{10} spinal cord levels.

LOWER URINARY TRACT STRUCTURES

Bladder

Anteriorly, the bladder rests against the inner surface of the pubic bones and then, as it fills, also against the anterior abdominal wall. Posteriorly, it rests against the vagina and cervix. The bladder is divided into a dome and a base approximately at the level of the ureteral orifices. The dome is thin walled and distensible, whereas the base is thicker and undergoes less distention during filling. The vesical trigone lies in the bladder base and contains both ureteral orifices and the internal

FIGURE 2-14 The fallopian tube of an adult woman with cross-sectioned illustrations of the gross structure in several portions: **(A)** isthmus, **(B)** ampulla, and **(C)** infundibulum. Below these are photographs of corresponding histological sections. (Used with permission from Dr. Kelley S. Carrick.)

urinary meatus (see Fig. 2-11). The urethral lumen begins at this meatus and then courses through the bladder base for less than 1 cm. This region where the urethral lumen traverses the bladder base is called the bladder neck.

The bladder wall consists of coarse bundles of smooth muscle known as the detrusor muscle, which extends into the proximal part of the urethra. A submucosal layer intervenes between this detrusor muscle and the mucosa. The bladder mucosa consists of transitional epithelium and underlying lamina propria.

The blood supply to the bladder arises from the superior vesical arteries, which are branches of the patent portion of the umbilical artery, and from the middle and inferior vesical arteries, which, when present, often arise from either the internal pudendal or the vaginal arteries (see Fig. 2-12). The nerve supply to the bladder arises from the vesical plexus, a component of the inferior hypogastric plexus (see Fig. 2-13).

Ureter

As the ureter enters the pelvis, it crosses over the bifurcation of the common iliac artery and passes just medial to the ovarian vessels (see Fig. 2-10). As the ureter descends into the pelvis, it lies medial to the internal iliac branches and anterolateral to the uterosacral ligaments. The ureter then traverses through the cardinal ligament approximately 1 to 2 cm lateral to the cervix. Near the level of the uterine isthmus, it courses below the uterine artery and travels anteromedially toward the bladder base. In this path, it runs close to the upper third of the anterior vaginal wall (Rahn, 2007). Finally, the ureter enters the bladder and travels obliquely for approximately 1.5 cm before opening at the ureteral orifices.

The pelvic ureter receives blood supply from the vessels it passes: the common iliac, internal iliac, uterine, and superior vesical vessels. The ureter's course runs medial to these vessels, and thus its blood supply reaches the ureter from lateral sources. This is important during ureteral isolation. Vascular anastomoses on the connective tissue sheath enveloping the ureter form a longitudinal network of vessels.

MUSCULOSKELETAL PELVIC ANATOMY

Pelvic Bones

The pelvis is composed of four bones—the sacrum, coccyx, and two innominate bones. Each innominate bone is formed by the fusion of three bones—the ilium, ischium, and pubis (Fig. 2-15). Both innominate bones are joined to the sacrum at the sacroiliac synchondroses and to one another at the symphysis pubis.

Pelvic Joints

Anteriorly, the pelvic bones are joined together by the symphysis pubis. This structure consists of fibrocartilage and the superior and inferior pubic ligaments. The latter ligament is frequently designated the arcuate ligament of the pubis. Posteriorly, the pelvic bones are joined by articulations between the sacrum and the iliac portion of the innominate bones to form the sacroiliac joints.

The pelvic joints in general have a limited degree of mobility. However, during pregnancy, these joints relax remarkably at term. As one result, upward gliding of the sacroiliac joint, which is greatest in the dorsal lithotomy position, may increase the diameter of the outlet by 1.5 to 2.0 cm for delivery (Borell, 1957). Sacroiliac joint mobility also likely aids the McRoberts maneuver to release an obstructed shoulder in cases of shoulder dystocia (Chap. 27, p. 521). These changes may also contribute to the success of the modified squatting position to hasten second-stage labor (Gardosi, 1989). The squatting position may increase the interspinous diameter and the pelvic outlet diameter (Russell, 1969, 1982).

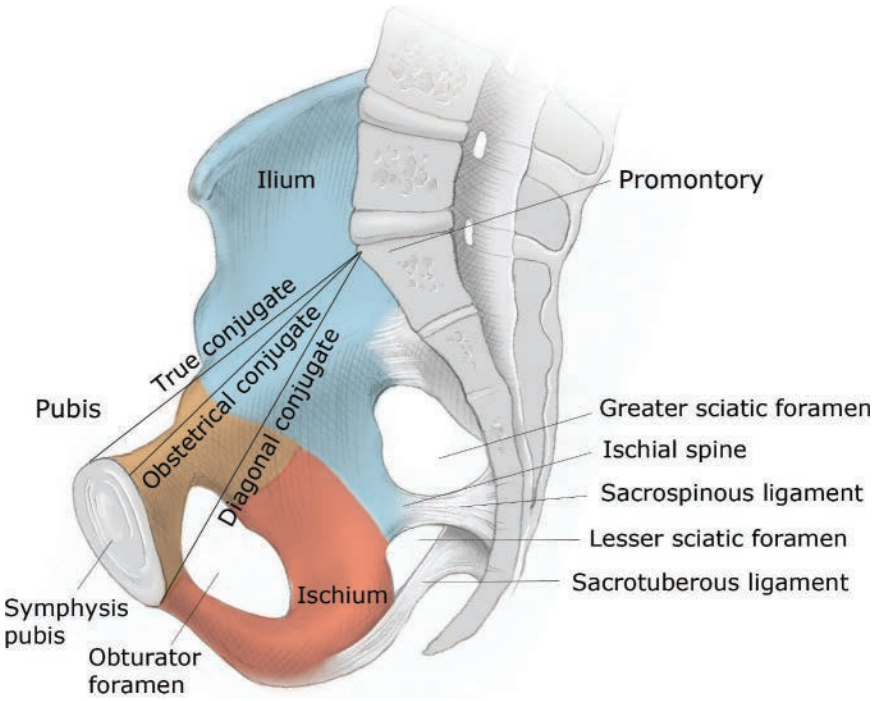

FIGURE 2-15 The innominate bone is composed of the pubis (*brown*), ischium (*red*), and ilium (*blue*). Of the three anteroposterior diameters of the pelvic inlet, only the diagonal conjugate can be measured clinically. The important obstetrical conjugate is derived by subtracting 1.5 cm from the diagonal conjugate.

Planes and Diameters of the Pelvis

The pelvis is conceptually divided into false and true components. The false pelvis lies above the linea terminalis, and the true pelvis is below this boundary (Fig. 2-16). The false pelvis is bounded posteriorly by the lumbar vertebra and laterally by the iliac fossa. In front, the boundary is formed by the lower portion of the anterior abdominal wall.

The pelvis is described as having four imaginary planes:

1. The plane of the pelvic inlet—the superior strait.
2. The plane of the pelvic outlet—the inferior strait.
3. The plane of the midpelvis—the least pelvic dimensions.
4. The plane of greatest pelvic dimension—of no obstetrical significance.

Pelvic Inlet

The pelvic inlet, also called the superior strait, is the superior plane of the true pelvis. It is bounded posteriorly by the promontory and alae of the sacrum, laterally by the linea terminalis, and anteriorly by the horizontal pubic rami and the symphysis pubis. During labor, fetal head engagement is defined by the fetal head's biparietal diameter passing through this plane.

Four diameters of the pelvic inlet are usually described: anteroposterior, transverse, and two oblique diameters. Of these, distinct anteroposterior diameters have been described using specific landmarks. Most cephalad, the anteroposterior diameter, termed the true conjugate, extends from the uppermost margin of the symphysis pubis to the sacral promontory (see Fig. 2-15). The clinically important obstetrical conjugate is the shortest distance between the sacral promontory and the symphysis pubis. Normally, this measures 10 cm or more, but unfortunately, it cannot be measured directly with examining fingers. Thus, the obstetrical conjugate is estimated indirectly by subtracting 1.5 to 2 cm from the diagonal conjugate. To measure the diagonal conjugate, a hand with the palm oriented laterally extends its index finger to the promontory. The distance from the fingertip to the point at which the lowest margin of the symphysis strikes the same finger's base is the diagonal conjugate.

The transverse diameter is constructed at right angles to the obstetrical conjugate and represents the greatest distance between the linea terminalis on either side (see Fig. 2-16). It usually intersects the obstetrical conjugate at a point approximately 5 cm in front of the promontory and measures approximately 13 cm.

Midpelvis and Pelvic Outlet

The midpelvis is measured at the level of the ischial spines, also called the midplane or plane of least pelvic dimensions (see Fig. 2-16). During labor, the degree of fetal head descent into the true pelvis may be described by station, and the midpelvis and ischial spines serve to mark zero station. The interspinous diameter is 10 cm or slightly greater and is usually the smallest pelvic diameter. The anteroposterior diameter through the level of the ischial spines normally measures at least 11.5 cm.

The pelvic outlet consists of two approximately triangular areas whose boundaries mirror those of the perineal triangle described earlier (p. 19). They have a common base, which is a line drawn between the two ischial tuberosities. The apex of the posterior triangle is the tip of the sacrum, and the

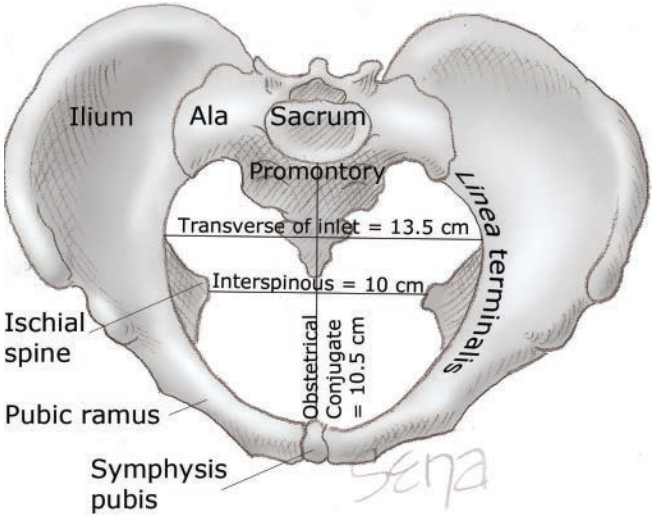

FIGURE 2-16 Axial view of a normal female pelvis. The clinically important obstetrical conjugate and transverse diameter of the pelvic inlet are illustrated. The interspinous diameter of the midpelvis is also marked.

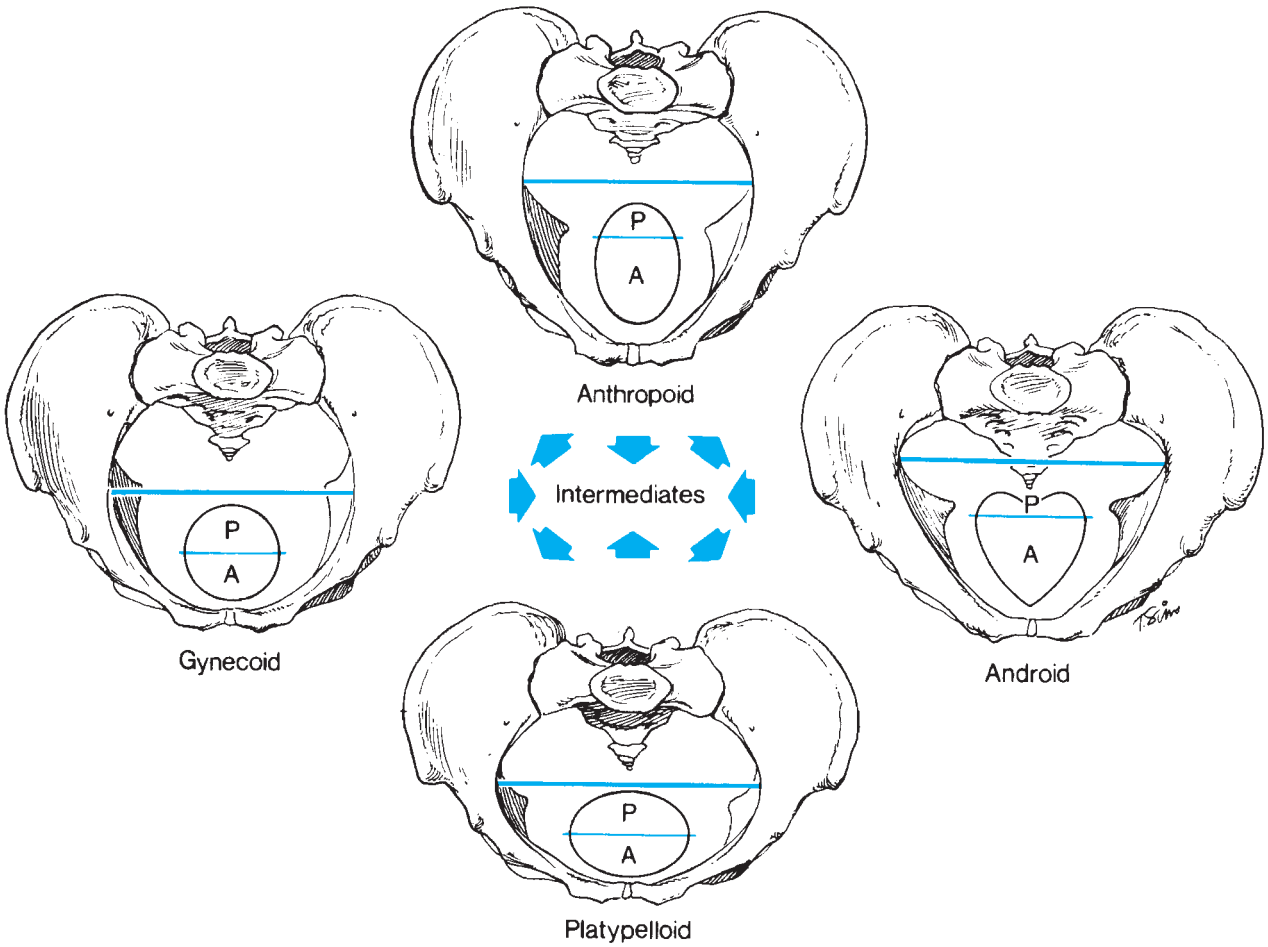

FIGURE 2-17 The four parent pelvic types of the Caldwell–Moloy classification. A line passing through the widest transverse diameter divides the inlets into posterior (P) and anterior (A) segments.

lateral boundaries are the sacrotuberous ligaments and the ischial tuberosities. The anterior triangle is formed by the descending inferior rami of the pubic bones. These rami unite at an angle of 90 to 100 degrees to form a rounded arch under which the fetal head must pass. Unless there is significant pelvic bony disease, the pelvic outlet seldom obstructs vaginal delivery.

Pelvic Shapes

The Caldwell–Moloy (1933, 1934) anatomical classification of the pelvis is based on shape, and its concepts aid an understanding of labor mechanisms. Specifically, the greatest transverse diameter of the inlet and its division into anterior and posterior segments are used to classify the pelvis as gynecoid, anthropoid, android, or platypelloid. The posterior segment determines the type of pelvis, whereas the anterior segment determines the tendency. These are both determined because many pelves are not pure but are mixed types. For example, a gynecoid pelvis with an android tendency means that the posterior pelvis is gynecoid and the anterior pelvis is android shaped.

From viewing the four basic types in Figure 2-17, the configuration of the gynecoid pelvis would intuitively seem suited for delivery of most fetuses. Indeed, Caldwell (1939) reported that the gynecoid pelvis was found in almost half of women.

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CHAPTER 3

Congenital Genitourinary Abnormalities

GENITOURINARY TRACT DEVELOPMENT 33
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Abnormalities in the development or fusion of one or both Müllerian ducts may result in malformations which sometimes possess an obstetrical significance. Pregnancy may be associated with any one of these malformations, provided an ovum be cast off from the ovaries and no serious obstacle be opposed to the upward passage of the spermatozoa and their subsequent union with it.

— J. Whitridge Williams (1903)

GENITOURINARY TRACT DEVELOPMENT

In females, the external genitalia, gonads, and müllerian ducts each derive from different primordia and in close association with the urinary tract and hindgut. Abnormal embryogenesis during this process is thought to be multifactorial and can create sporadic anomalies. Several of these can lead to infertility, subfertility, miscarriage, or preterm delivery. Thus, knowledge of genitourinary system development is essential.

Embryology of the Urinary System

Between the 3rd and 5th gestational weeks, an elevation of intermediate mesoderm on each side of the fetus—the urogenital ridge—begins development into the urogenital tract. Subsequently, the urogenital ridge divides into the genital ridge, destined to become the ovary, and into the nephrogenic ridge (Fig. 3-1). The nephrogenic ridges develop into the mesonephros (mesonephric kidney) and paired mesonephric ducts, also termed wolffian ducts, which connect to the cloaca.

The early urinary tract develops from the mesonephros and its mesonephric ducts (Fig. 3-2A). Recall that evolution of the renal system passes sequentially through the pronephric and mesonephric stages to reach the permanent metanephric system. Between the 4th and 5th weeks, each mesonephric duct gives rise to a ureteric bud, which grows cephalad toward its respective mesonephros (Fig. 3-2B). As each bud lengthens, it induces differentiation of the metanephros, which will become the final kidney (Fig. 3-2C). Each mesonephros degenerates near the end of the first trimester, and without testosterone, the mesonephric ducts regress as well.

The cloaca begins as a common opening for the embryonic urinary, genital, and alimentary tracts. By the 7th week it becomes divided by the urorectal septum to create the rectum and the urogenital sinus (Fig. 3-2D). The urogenital sinus is considered in three parts: (1) the cephalad or vesicle portion, which forms the urinary bladder; (2) the middle or pelvic portion, which creates the female urethra; and (3) the caudal or phallic part, which gives rise to the distal vagina and to the greater vestibular (Bartholin) and paraurethral glands.

Embryology of the Genital Tract

The fallopian tubes, uterus, and upper vagina derive from the müllerian ducts, also termed paramesonephric ducts, which form adjacent to each mesonephros (see Fig. 3-2B). These ducts extend downward and then turn medially to meet and fuse together in the midline. The uterus is formed by this union of the two müllerian ducts at approximately the 10th week (Fig. 3-2E). Fusion to create the uterus begins in the middle and then extends both caudally and cephalad. With cellular

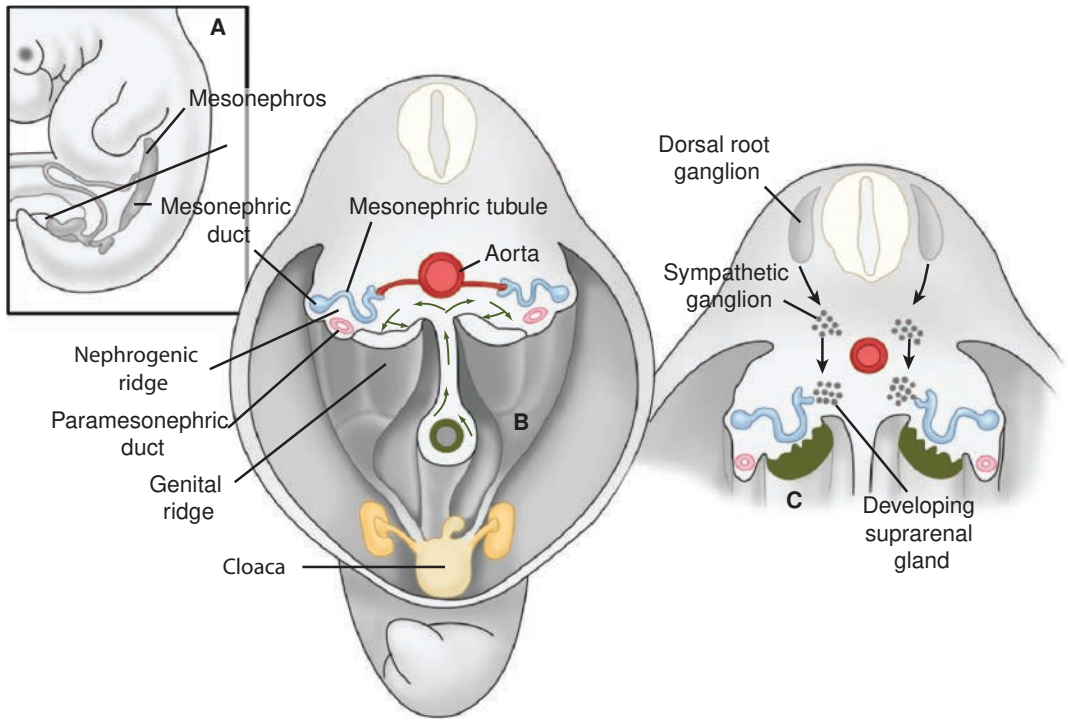

FIGURE 3-1 **A.** Cross-section of an embryo at 4 to 6 weeks. **B.** Large ameboid primordial germ cells migrate (*arrows*) from the yolk sac to the area of germinal epithelium, within the genital ridge. **C.** Migration of sympathetic cells from the spinal ganglia to a region above the developing kidney.

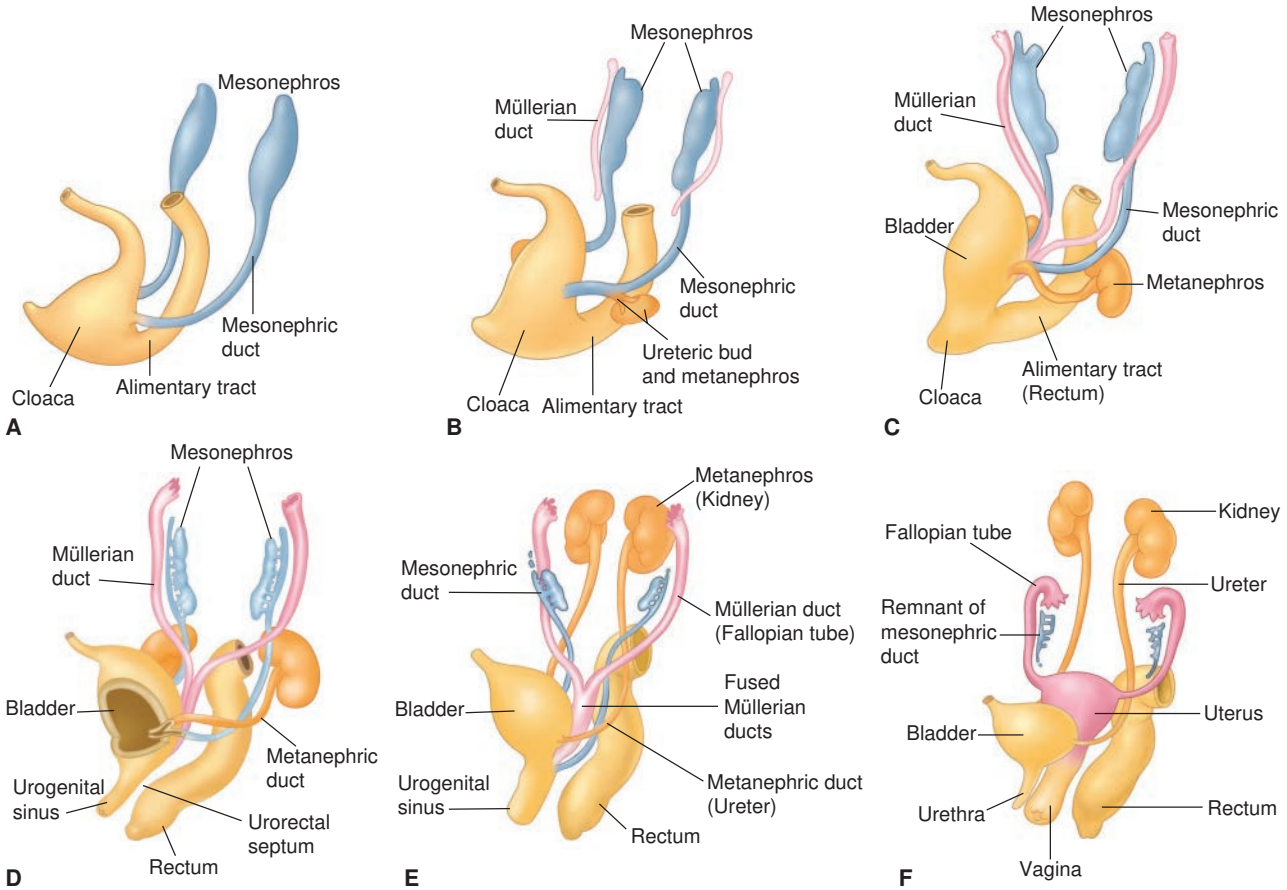

FIGURE 3-2 Embryonic development of the female genitourinary tract **(A-F)**. (Reproduced with permission from Shatzkes DR, Haller JO, Velcek FT: Imaging of uterovaginal anomalies in the pediatric patient, Urol Radiol 1991;13(1):58–66.)

proliferation at the upper portion, a thick wedge of tissue creates the characteristic piriform uterine shape. At the same time, dissolution of cells at the lower pole forms the first uterine cavity (Fig. 3-2F). As the upper wedge-shaped septum is slowly reabsorbed, the final uterine cavity is usually formed by the 20th week. If the two müllerian ducts fail to fuse, then two separate uterine horns remain. In contrast, resorption failure of the common tissue between them results in various degrees of persistent uterine septum.

As the distal end of the fused müllerian ducts contacts the urogenital sinus, this induces endodermal outgrowths from the sinus termed the sinovaginal bulbs. These bulbs proliferate and fuse to form the vaginal plate, which later resorbs to form the vaginal lumen. This vaginal canalization is generally completed by the 20th week. However, the lumen remains separated from the urogenital sinus by the hymeneal membrane. This membrane further degenerates to leave only the hymeneal ring.

The close association of the mesonephric (wolffian) and paramesonephric (müllerian) ducts explains the simultaneous abnormalities in their end organs. Kenney and colleagues (1984) showed that up to half of females with uterovaginal malformations have associated urinary tract defects. Anomalies most frequently associated with renal defects are unicornuate uterus, uterine didelphys, and agenesis syndromes, whereas arcuate and bicornuate are less commonly linked (Reichman, 2010). When müllerian anomalies are identified, the urinary system can be evaluated with magnetic resonance (MR) imaging, sonography, or intravenous pyelography (Hall-Craggs, 2013). With müllerian anomalies, ovaries are functionally normal but have a higher incidence of anatomical maldescent into the pelvis (Allen, 2012; Dabirashrafi, 1994).

As discussed, the mesonephric ducts usually degenerate, however, persistent remnants may become clinically apparent. Mesonephric or wolffian vestiges can persist as Gartner duct cysts. These are typically located in the proximal anterolateral vaginal wall but may be found at other sites along the vaginal length. They can be further characterized by MR imaging, which provides excellent image resolution at soft tissue interfaces. Most cysts are asymptomatic and benign and usually do not require surgical excision.

Intraabdominal wolffian remnants in the female include a few blind tubules in the mesovarium—the epoöphoron—and similar ones adjacent to the uterus—paroöphoron (see Fig. 3-2F) (Moore, 2013). The epoöphoron or paroöphoron may develop into clinically identifiable cysts in the adult.

Embryology of the Gonads

At approximately 4 weeks, gonads derive from coelomic epithelium covering the medial and ventral surface of the nephrogenic cord at a site between the eighth thoracic and fourth lumbar segments. Because of this separate gonadal and müllerian derivation, women with müllerian defects typically have functionally normal ovaries and are phenotypic females. The coelomic epithelium thickens to form the genital ridge, also known as the gonadal ridge. Strands of these epithelial cells extend into the underlying mesenchyme as the primary sex cords. By the sixth week, primordial germ cells have migrated from the yolk sac to enter the genital ridge mesenchyme (Fig. 3-3). The primordial germ cells are then incorporated into the primary sex cords.

In the seventh week, the sexes can be distinguished, and testes are recognized during microscopic sectioning by their well-defined radiating testis cords. These cords are separated from the coelomic epithelium by mesenchyme that is to become the tunica albuginea. The testis cords develop into the seminiferous tubules and rete testis. The rete testis establishes connection with small tubes arising off the mesonephric duct. These small tubes become the efferent ducts that drain into the epididymis and then into the vas deferens, which are main mesonephric duct derivatives.

In the female embryo, the primary sex cords give rise to the medullary cords, which persist only for a short time. The coelomic epithelium again proliferates into the underlying mesenchyme, and these strands are the cortical cords. By the fourth month, the cortical cords begin to form isolated cell clusters called primordial follicles. These follicles contain the oogonia, which derive from primordial germ cells and are surrounded by a single layer of flattened follicular cells derived from the cortical cords. Follicular cells serve as supporting nutrient cells. By 8 months, the ovary has become a long, narrow, lobulated structure that is attached to the body wall by the mesovarium. The coelomic epithelium has been separated by a band of connective tissue—tunica albuginea—from the cortex. At this stage, the cortex contains follicles and is well defined from the inner medulla, which is composed of abundant blood vessels, lymphatic vessels, and nerve fibers.

Embryology of the External Genitalia

Early development of the external genitalia is similar in both sexes. By 6 weeks' gestation, three external protuberances have developed surrounding the cloacal membrane. These are the left and right cloacal folds, which meet ventrally to form the genital tubercle (Fig. 3-4). With division of the cloacal membrane into anal and urogenital membranes, the cloacal folds become the anal and urethral folds, respectively. Lateral to the urethral folds, genital swellings arise, and these become the labioscrotal folds. Between the urethral folds, the urogenital sinus extends onto the surface of the enlarging genital tubercle to form the urethral groove. By week 7, the urogenital membrane ruptures, exposing the cavity of the urogenital sinus to amnionic fluid.

The genital tubercle elongates to form the phallus in males and the clitoris in females. Still, it is not possible to visually differentiate between male and female external genitalia until week 12. In the male fetus, dihydrotestosterone (DHT) forms locally by the 5-α reduction of testosterone. DHT prompts the anogenital distance to lengthen, the phallus to enlarge, and the labioscrotal folds to fuse and form the scrotum.

In the female fetus, without DHT, the anogenital distance does not lengthen, and the labioscrotal and urethral folds do not fuse (Fig. 3-4C). The genital tubercle bends caudally to become the clitoris, and the urogenital sinus forms the vestibule of the vagina. The labioscrotal folds create the labia majora, whereas the urethral folds persist as the labia minora. Female external genital differentiation is complete by 11 weeks, whereas male external genital differentiation is complete by 14 weeks.

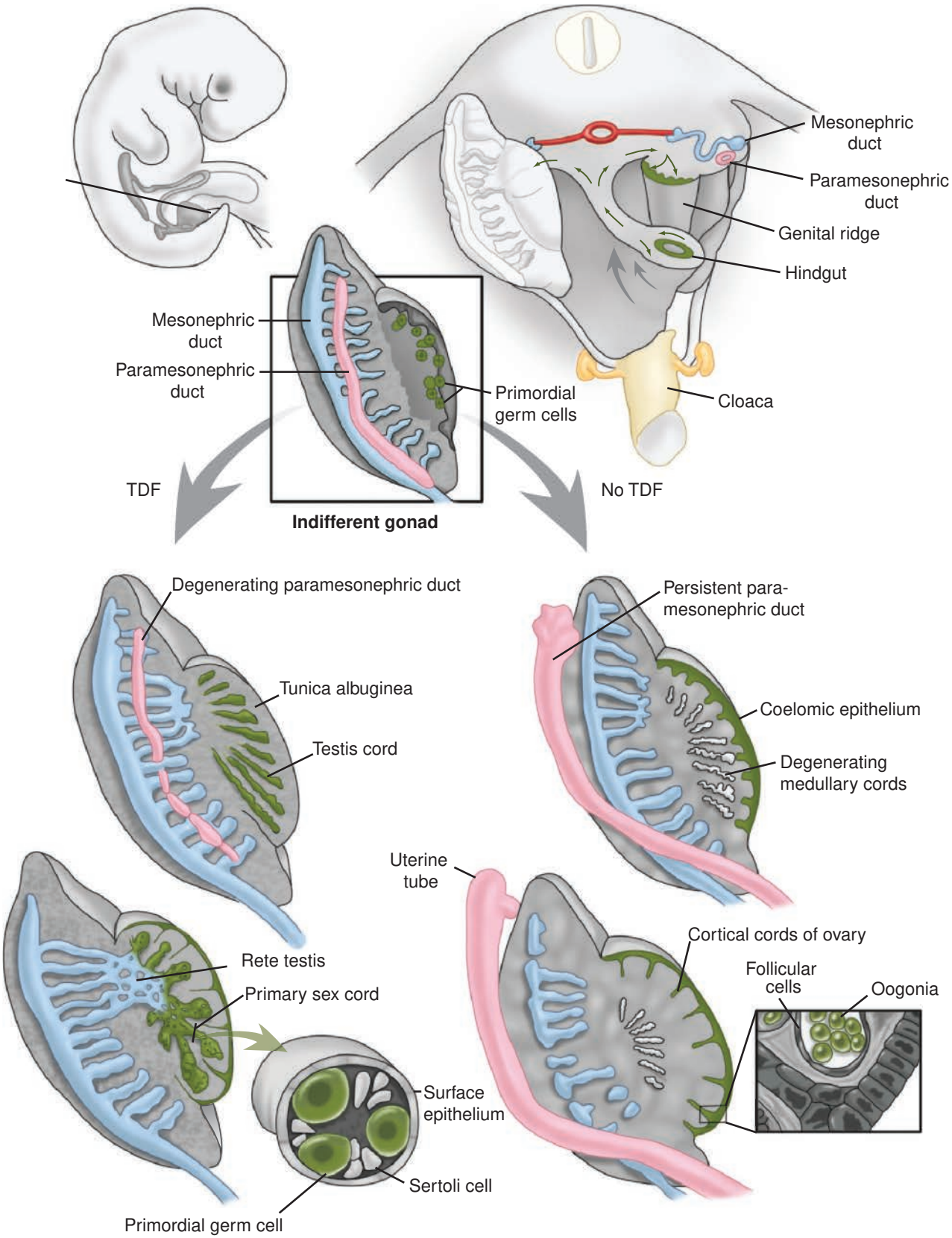

FIGURE 3-3 Embryonic gonad differentiation. TDF = testis-determining factor.

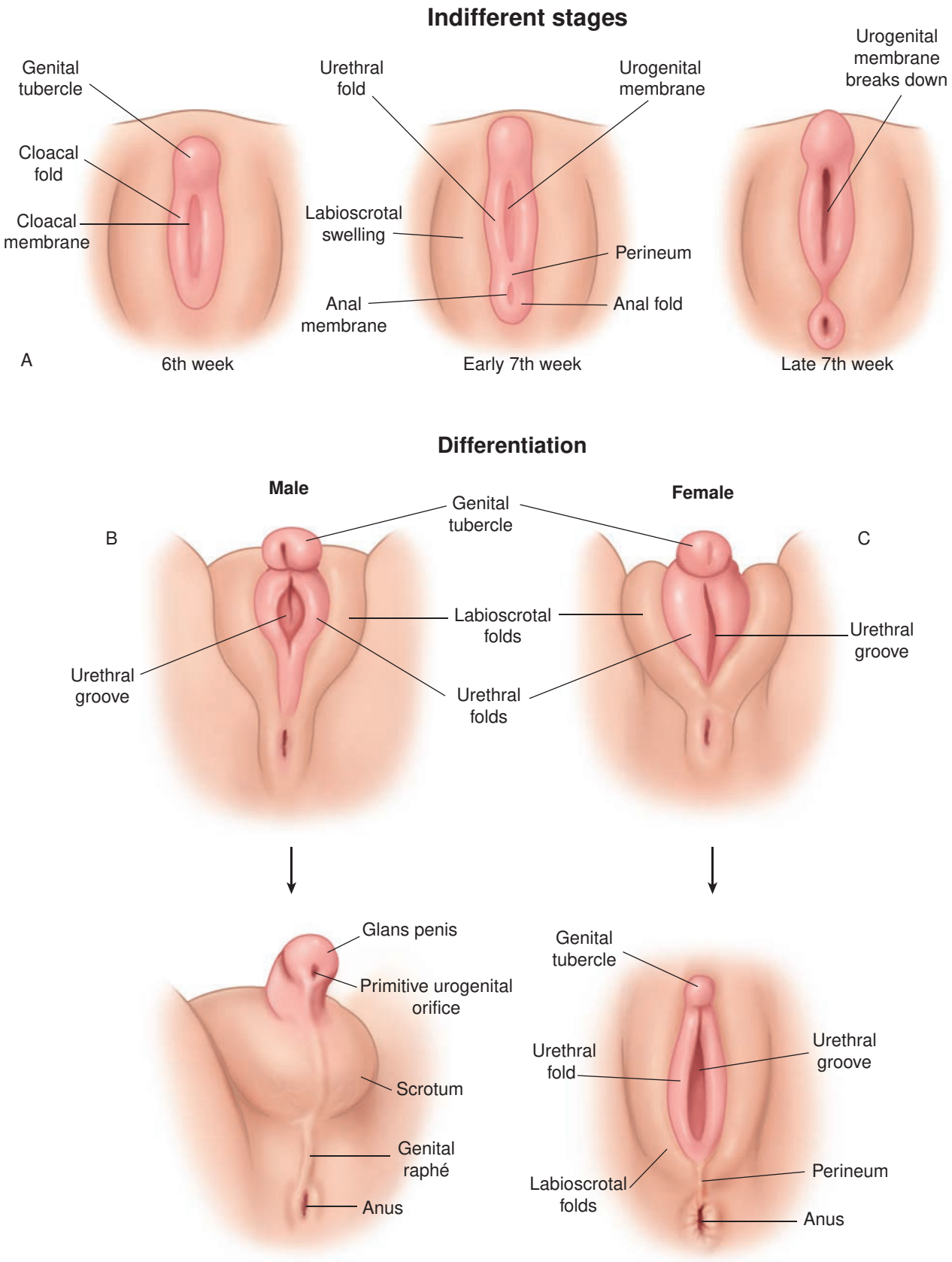

FIGURE 3-4 Development of the external genitalia. **A.** Indifferent stage. **B.** Virilization of external genitalia. **C.** Feminization. (Reproduced with permission from Bradshaw KD: Anatomical disorders. In Hoffman BL, Schorge JO, Bradshaw KD, et al (eds): Williams Gynecology, 3rd ed. New York, McGraw Hill Education, 2016.)

SEXUAL DIFFERENTIATION

Defining gender incorporates genetic gender, gonadal gender, and phenotypic gender. *Genetic gender*—XX or XY—is established at fertilization. However, for the first 6 weeks, development of male and female embryos is morphologically indistinguishable.

Gonadal gender is heralded by the differentiation of the primordial gonad into a testis or an ovary. If a Y chromosome is present, the gonad begins developing into a testis. Testis development is directed by a protein called the *testis-determining factor (TDF)*, which modulates the transcription of several genes involved in gonadal differentiation. TDF is encoded by the *sex-determining region (SRY) gene,* located on the short arm of the Y chromosome. But testis development is much more complex and requires other autosomal genes (Nistal, 2015a).

The importance of the *SRY* gene is demonstrated in several paradoxical conditions. First, 46,XX phenotypic males can result from translocation of the Y chromosome fragment containing *SRY* to the X chromosome during meiosis of male germ cells (Wu, 2014). Similarly, 46,XY individuals can appear phenotypically female if they carry a mutation in the *SRY* gene (Helszer, 2013).

Last, *phenotypic gender* begins at 8 weeks' gestation. Before this, urogenital tract development in both sexes is indistinguishable. Thereafter, differentiation of the internal and external genitalia to the male phenotype is dependent on testicular function. In the absence of a testis, female differentiation ensues irrespective of genetic gender (Table 3-1).

In males, the fetal testis secretes a protein called müllerian-inhibiting substance (MIS), also called antimüllerian hormone (AMH). It acts locally as a paracrine factor to cause müllerian duct regression. Thus, it prevents the development of uterus, fallopian tube, and upper vagina. AMH is produced by the Sertoli cells of the seminiferous tubules. Importantly, these tubules appear in fetal gonads and secrete AMH before differentiation of Leydig cells, which are the cellular site of testosterone synthesis. AMH is secreted as early as 7 weeks, and müllerian duct regression is completed by 9 to 10 weeks. Because AMH acts locally near its site of formation, if a testis were absent on one side, the müllerian duct on that side would persist, and the uterus and fallopian tube would develop on that side.

Apparently through stimulation initially by human chorionic gonadotropin (hCG), and later by fetal pituitary luteinizing hormone (LH), the fetal testes secrete testosterone. This hormone acts directly on the wolffian duct to effect the development of the vas deferens, epididymis, and seminal vesicles. Testosterone also enters fetal blood and acts on the external genitalia anlage. In these tissues, testosterone is converted to 5α-DHT to cause virilization of the external genitalia.

DISORDERS OF SEX DEVELOPMENT

Definitions

As evident from the prior discussion, abnormal sex development may involve the gonads, internal duct system, or external genitalia. Rates vary and approximate 1 in every 1000 to 4500 births (Murphy, 2011; Ocal, 2011). The nomenclature used to describe disorders of sex development (DSDs) has evolved. Current classification of these disorders include: (1) sex chromosome DSDs, (2) 46,XY DSDs, and (3) 46,XX DSDs (Table 3-2) (Hughes, 2006).

Other important terms describe the abnormal phenotypic findings that can be found. First, some disorders of sexual development are associated with abnormal, underdeveloped gonads, that is, *gonadal dysgenesis*. With this, if a testis is poorly formed, it is called a *dysgenetic testis*, and if an ovary is poorly formed, it is called a *streak gonad*. In affected patients, the underdeveloped gonad ultimately fails, which is indicated by elevated gonadotropin levels. Another important clinical sequela is that patients bearing a Y chromosome are at high risk of developing a germ cell tumor in the dysgenetic gonad.

A second term, *ambiguous genitalia*, describes genitalia that do not appear clearly male or female. Abnormalities may include

TABLE 3-1. Embryonic Urogenital Structures and Their Adult Homologues

Indifferent Structure	Female	Male
Genital ridge	Ovary	Testis
Primordial germ cells	Ova	Spermatozoa
Sex cords	Granulosa cells	Seminiferous tubules, Sertoli cells
Gubernaculum	Uteroovarian and round ligaments	Gubernaculum testis
Mesonephric tubules	Epoöphoron, paroöphoron	Efferent ductules, paradidymis
Mesonephric ducts	Gartner duct	Epididymis, ductus deferens, ejaculatory duct
Paramesonephric ducts	Uterus, fallopian tubes, upper vagina	Prostatic utricle, appendix of testis
Urogenital sinus	Bladder, urethra	Bladder, urethra
	Vagina	Prostatic utricle
	Paraurethral glands	Prostate glands
	Greater (Bartholin) and lesser vestibular glands	Bulbourethral glands
Genital tubercle	Clitoris	Glans penis
Urogenital folds	Labia minora	Floor of penile urethra
Labioscrotal swellings	Labia majora	Scrotum