

dejà

review

Obstetrics & Gynecology

- Great for last-minute clerkship and board review
- Written by medical students who just aced the USMLE Step 2 CK
- Rapid-fire, quick-hit format for maximum retention
- Clinical vignettes chapter preps you for cases you'll see on the exam

REMEMBER
WHAT YOU ALREADY
KNOW

Catherine J. Lee
Emily S. Miller

DEJA REVIEW™

Obstetrics & Gynecology

NOTICE

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The authors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the authors nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they disclaim all responsibility for any errors or omissions or for the results obtained from use of the information contained in this work. Readers are encouraged to confirm the information contained herein with other sources. For example and in particular, readers are advised to check the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this work is accurate and that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in connection with new or infrequently used drugs.

DEJA REVIEW™

Obstetrics & Gynecology

Catherine J. Lee, MD

New York University School of Medicine
New York, New York

Emily S. Miller, MD

New York University School of Medicine
New York, New York



Medical

New York Chicago San Francisco Lisbon London Madrid Mexico City
Milan New Delhi San Juan Seoul Singapore Sydney Toronto

Copyright © 2008 by The McGraw-Hill Companies, Inc. All rights reserved. Manufactured in the United States of America. Except as permitted under the United States Copyright Act of 1976, no part of this publication may be reproduced or distributed in any form or by any means, or stored in a database or retrieval system, without the prior written permission of the publisher.

0-07-159401-9

The material in this eBook also appears in the print version of this title: 0-07-148122-2.

All trademarks are trademarks of their respective owners. Rather than put a trademark symbol after every occurrence of a trademarked name, we use names in an editorial fashion only, and to the benefit of the trademark owner, with no intention of infringement of the trademark. Where such designations appear in this book, they have been printed with initial caps.

McGraw-Hill eBooks are available at special quantity discounts to use as premiums and sales promotions, or for use in corporate training programs. For more information, please contact George Hoare, Special Sales, at george_hoare@mcgraw-hill.com or (212) 904-4069.

TERMS OF USE

This is a copyrighted work and The McGraw-Hill Companies, Inc. ("McGraw-Hill") and its licensors reserve all rights in and to the work. Use of this work is subject to these terms. Except as permitted under the Copyright Act of 1976 and the right to store and retrieve one copy of the work, you may not decompile, disassemble, reverse engineer, reproduce, modify, create derivative works based upon, transmit, distribute, disseminate, sell, publish or sublicense the work or any part of it without McGraw-Hill's prior consent. You may use the work for your own noncommercial and personal use; any other use of the work is strictly prohibited. Your right to use the work may be terminated if you fail to comply with these terms.

THE WORK IS PROVIDED "AS IS." McGRAW-HILL AND ITS LICENSORS MAKE NO GUARANTEES OR WARRANTIES AS TO THE ACCURACY, ADEQUACY OR COMPLETENESS OF OR RESULTS TO BE OBTAINED FROM USING THE WORK, INCLUDING ANY INFORMATION THAT CAN BE ACCESSED THROUGH THE WORK VIA HYPERLINK OR OTHERWISE, AND EXPRESSLY DISCLAIM ANY WARRANTY, EXPRESS OR IMPLIED, INCLUDING BUT NOT LIMITED TO IMPLIED WARRANTIES OF MERCHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE. McGraw-Hill and its licensors do not warrant or guarantee that the functions contained in the work will meet your requirements or that its operation will be uninterrupted or error free. Neither McGraw-Hill nor its licensors shall be liable to you or anyone else for any inaccuracy, error or omission, regardless of cause, in the work or for any damages resulting therefrom. McGraw-Hill has no responsibility for the content of any information accessed through the work. Under no circumstances shall McGraw-Hill and/or its licensors be liable for any indirect, incidental, special, punitive, consequential or similar damages that result from the use of or inability to use the work, even if any of them has been advised of the possibility of such damages. This limitation of liability shall apply to any claim or cause whatsoever whether such claim or cause arises in contract, tort or otherwise.

DOI: 10.1036/0071481222

*To my family, friends, and teachers—thank you for being a
constant source of light in my journey to becoming
an excellent physician.*

Catherine J. Lee

*To all those who have taught me the art of medicine, inspired
me to make change, and supported me throughout the process
of becoming a physician.*

Emily S. Miller

Contents

Contributing Authors	x
Illustrators	x
Faculty Reviewers	x
Acknowledgments	xii
Introduction	xiii
SECTION I: USEFUL FACTS FOR THE WARDS	1
Chapter 1 LAB VALUES	3
Chapter 2 COMMON PROCEDURES	7
Chapter 3 FEMALE ANATOMY	23
Bony Pelvis / 23	
Pelvic Organs / 25	
Perineum / 31	
Abdominal Wall / 33	
SECTION II: TOPICS IN GYNECOLOGY	35
Chapter 4 GENERAL GYNECOLOGY	37
Contraception Overview / 41	
Sterilization Methods / 43	
Barrier Methods of Contraception / 52	
Intrauterine Devices / 54	
Elective Abortion / 56	
Sexually Transmitted Infections of the Lower Genital Tract / 61	
Sexually Transmitted Infections Causing Genital Ulcerations / 63	
Cervicitis / 71	
Infections of the Upper Genital Tract / 73	
Vaginitis / 81	
Other Gynecologic Infections / 86	
Chronic Pelvic Pain / 89	
Acute Pelvic Pain / 94	
Endometriosis / 95	
Benign Cervical Masses / 103	
Benign Uterine Masses / 105	
Sexual Dysfunction / 111	

	Dystrophies / 116	
	Benign Cysts / 118	
	Pyodermas and other Nonsexually Transmitted Infections of the Vulva and Vagina / 120	
	Other Vulvar Disorders / 121	
Chapter 5	GYNECOLOGIC ONCOLOGY	123
	Introduction to Cervical Cancer / 123	
	Cervical Cancer Screening and Prevention / 128	
	Squamous Cell Carcinoma of the Cervix / 132	
	Adenocarcinoma of the Cervix / 135	
	Vulvar Cancer Overview / 136	
	Squamous Cell Vulvar Cancer / 138	
	Vaginal Cancer Overview / 144	
	Endometrial Cancer / 148	
	Uterine Sarcoma / 153	
	Introduction to Ovarian Cancer / 154	
	Epithelial Ovarian Cancer / 154	
	Nonepithelial Ovarian Cancer / 158	
	Fallopian Tube Cancer / 163	
Chapter 6	UROGYNECOLOGY	165
	Acute Cystitis / 165	
	UTI in Pregnancy / 168	
	Urethritis / 169	
	Acute Pyelonephritis / 169	
	Pelvic Organ Prolapse / 171	
	Introduction to Urinary Incontinence / 178	
	Patient Evaluation / 180	
	Stress Incontinence / 182	
	Urge Incontinence / 183	
	Mixed Incontinence / 184	
	Overflow Incontinence / 185	
	Treatment / 185	
Chapter 7	REPRODUCTIVE ENDOCRINOLOGY AND INFERTILITY	189
	Introduction to Adolescent Development / 189	
	Congenital Anomalies / 191	
	Accelerated Sexual Maturation / 192	
	Delayed Sexual Maturation / 194	
	PMS / 195	
	Dysfunctional Uterine Bleeding / 198	
	Amenorrhea / 201	
	Evaluation of Infertility / 204	
	Treatment of Infertility / 207	

Assisted Reproductive Technologies /	209
Osteoporosis /	209
Perimenopause and Menopause /	213
Hirsutism and Virilization /	217

SECTION III: TOPICS IN OBSTETRICS 223

Chapter 8 GENERAL OBSTETRICS 225

Embryology /	225
Fetal Circulation /	226
Cardiovascular /	227
Respiratory /	229
Renal/Urinary /	230
Gastrointestinal /	231
Hematologic /	232
Endocrine /	232
Dermatology /	233
Reproductive /	234
Diagnosis and Terminology of Prenatal Care /	234
Management /	238
Screening /	243
Prenatal Testing and Monitoring of the Fetus /	247
Diagnosis and Definitions of Labor and Delivery /	251
Fetal Position /	254
Pelvimetry and Vaginal Examination /	259
Stages of Labor /	260
Cardinal Movements of Labor /	264
Induction of Labor /	266
Intrapartum Assessment and Monitoring /	267
Management /	269
Operative Vaginal Delivery /	271
Cesarean Delivery /	273
Puerperium Management /	274
Breastfeeding and Infant Care /	277
Physiologic Changes and Resolution /	279
Postpartum Clinical Care /	281
Fertility and Contraception /	282

Chapter 9 COMPLICATIONS OF PREGNANCY 283

Congenital Anomalies /	283
Spontaneous Abortions /	286
Ectopic Pregnancy /	290
Gestational Trophoblastic Disease /	295
Hypertensive Disorders of Pregnancy /	302

Abnormal Placentation /	307
Third Trimester Bleeding /	309
PROM and PPROM /	311
Postpartum Hemorrhage /	321
Placenta Accreta /	323
Uterine Inversion /	324
Postpartum and Puerperal Infections /	325
Endometritis /	326
Endocrine Disorders /	327
Renal and Urinary Tract Disorders /	343
Neurologic Disorders /	344
Hematologic Disorders /	346
Pulmonary Disorders /	351
Cardiovascular Disorders /	352
Acute Abdomen and Abdominal Trauma /	355
TORCH Infections /	357
Other Viral Infections /	364
Bacterial Infections /	374
UTI in Pregnancy /	377
Rhesus (Rh) Alloimmunization /	378
Fetal Growth Restriction /	383
Fetal Macrosomia /	386
Dystocia /	392
Abnormal Fetal Testing /	396
Meconium /	399
Postterm Pregnancy /	400
SECTION IV: WOMEN'S HEALTH ISSUES	407
Chapter 10 ISSUES IN WOMEN'S HEALTH	409
Domestic Violence /	409
Sexual Assault /	411
Ethics and Law /	413
SECTION V: REVIEW QUESTIONS AND ANSWERS	417
Chapter 11 REVIEW QUESTIONS AND ANSWERS	419
Suggested Readings and Sources of Information	467
Index	469

Contributing Authors

Jessica Maria Atrio, MD

Department of Obstetrics and Gynecology
New York University School of Medicine
New York, New York
General Obstetrics

Ricky Darnell Grisson II, MBA, MD

Harvard Medical School
Boston, Massachusetts
Women's Health

Illustrators

Alex M. Kotylar

New York University
College of Arts and Sciences
New York, New York

Pavel Kotylar

Touro College
Brooklyn, New York

Faculty Reviewers

Kristin L. Atkins, MD

Assistant Professor
Division of Maternal Fetal Medicine
Department of Obstetrics and Gynecology
New York University School of Medicine
New York, New York
*Adolescent Development, Maternal-Fetal
Physiology, Prenatal Care*

Stephanie V. Blank, MD

Assistant Professor
Division of Gynecologic Oncology
Department of Obstetrics and Gynecology
New York University School of Medicine
New York, New York
*Cervical Cancer, Vulvar and Vaginal Cancer,
Uterine Cancer, Fallopian Tube Cancer*

Yoni Barnhard, MD, FACOG, MFM

Vice Chairman
Department of Obstetrics and Gynecology
Lenox Hill Hospital
New York, New York
Medical Conditions of Pregnancy

Asaf Ferber, MD

General Obstetrician-Gynecologist
Department of Obstetrics and Gynecology
Lenox Hill Hospital
New York, New York
Benign Conditions of the Vulva and Vagina

Jonathan S. Berek, MD

Professor and Chair
Department of Obstetrics and Gynecology
Stanford University School of Medicine
Division of Gynecologic Oncology
Stanford Cancer Center
Stanford, California
Ovarian Cancer

Debra A. Keegan, MD

Associate Physician
IVF Florida Reproductive Associates
Margate, Florida
*Disorders of the Menstrual Cycle, Uterus, and
Endometrium; Infertility; Osteoporosis;
Perimenopause and Menopause; Hirsutism,
Virilization and PCOS*

Chad Klauser, MD

Assistant Professor
 Division of Maternal-Fetal Medicine
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
*STDs and Pelvic Infections, Postpartum Care,
 Multifetal Gestation, Teratogens*

Christina Kwon, MD

Assistant Professor
 Division of Urogynecology
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
*Urinary Infections, Pelvic Organ Prolapse,
 Urinary Incontinence*

Ruth Lahti, MD

Assistant Professor
 Reproductive Endocrinology and Infertility
 Program
 Department of Obstetrics and Gynecology
 Stanford University School of Medicine
 Stanford, California
Endometriosis

Rachel Masch, MD

Assistant Professor
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
Menstrual Cycle, Family Planning

Victoria Ort, PhD

Assistant Professor
 Department of Cell Biology
 New York University School of Medicine
 New York, New York
Female Anatomy

John C. Petrozza, MD

Chief, Reproductive Medicine & IVF
 Obstetrics and Gynecology Service
 Massachusetts General Hospital
 Harvard Medical School
 Boston, Massachusetts
Pelvic Pain

Michael Plakogiannis, MD

Faculty Staff
 Department of Obstetrics and Gynecology
 Lenox Hill Hospital
 New York, New York
Late Complications, Postpartum Complications

Florencia Greer Polite, MD

Assistant Professor
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
Benign Masses

Ashley S. Roman, MD, MPH

Assistant Professor
 Division of Maternal-Fetal Medicine
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
*Labor and Delivery, Early Complications,
 Obstetric Infections, Abnormal Labor and
 Delivery*

David Seubert, MD

Associate Professor of Obstetrics and
 Gynecology
 Director, Division of Maternal-Fetal
 Medicine and Medical Student Clerkship in
 Obstetrics and Gynecology
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
Isoimmunization, Fetal Growth Abnormalities

Tomer Singer, MD

Resident Physician
 Department of Obstetrics and Gynecology
 Lenox Hill Hospital
 New York, New York
*Common Procedures, Medical Conditions of
 Pregnancy*

Erin E. Tracy, MD, MPH

Assistant Professor
 Obstetrics, Gynecology, and Reproductive
 Biology
 Harvard Medical School
 Massachusetts General Hospital
 Boston, Massachusetts
Domestic Violence

Ming C. Tsai, MD

Assistant Professor
 Department of Obstetrics and Gynecology
 New York University School of Medicine
 New York, New York
Sexual Dysfunction

Acknowledgments

The authors would like to thank all the contributors, artists, and faculty reviewers for their invaluable time and effort in contributing to this book and making it a useful resource for all medical students.

The authors would like to recognize all the faculty and staff at New York University School of Medicine, Harvard Medical School, Stanford University School of Medicine, and Lenox Hill Hospital for their endless commitment and dedication to educating medical students. We would also like to thank the students who used this text in preparation for the boards and provided essential feedback necessary to write an improved high-yield, comprehensive book. Finally, a special thanks to the editors and publishers at McGraw-Hill for their extraordinary patience and guidance at each step of the process to see this project through.

Introduction

Thank you for using *Deja Review: Obstetrics & Gynecology* to assist you in your preparation for the core clerkship in Obstetrics and Gynecology, the in-service exam, and for the Ob and Gyn-related questions in Step 2 CK of the United States Medical Licensing Exam (USMLE). We have worked diligently to bring you a concise and rigorous review of obstetrics and gynecology. This book is a compilation of essential facts, organized into an easy-to-read question and answer format, with difficult concepts illustrated by figures and mnemonics. Furthermore, this review book encourages step-by-step logical problem-solving skills, necessary for good performance on the boards and success on the wards, by combining clinical scenarios with fundamental principles of these topics. Considering the huge volume of information that you must synthesize in order to perform successfully as a clinician, we recommend the use of all the books in the *Deja Review* series to help you form that required foundation.

Organization

Section I includes Chapters 1–3, “Useful Facts for the Wards.” These chapters include common laboratory values and essential knowledge of female anatomy and commonly performed procedures in obstetrics and gynecology.

Sections II and III are divided into several chapters that contain thorough and high-yield material on obstetrics and gynecology. It has been our goal to fulfill the third-year learning objectives recommended by the American Professors of Gynecology and Obstetrics (APGO) and the American College of Obstetrics and Gynecology (ACOG).

Section IV is dedicated to Women’s Health Issues, which contains information on domestic violence, sexual assault, and ethics and law.

Section V of this book provides the reader with many clinical vignettes that students may face on the USMLE Step 2 CK and on the wards. This section encourages students to apply their knowledge of obstetrics and gynecology to relevant clinical scenarios.

Topics are covered in a question and answer format with buzz words highlighted in bold for each response. This format is designed for rapid review during the clerkship and before exams. Furthermore, we have included questions that require students to think about a problem in a logical and clinically appropriate manner.

It is our hope that this book will supplement topics learned in the clinical setting and will facilitate success on the clerkship shelf exam and the USMLE Step 2 CK. We also hope to stimulate interest in the fascinating fields of obstetrics and gynecology through exposure to some of the exciting clinical cases that are typical of this field.

How to use this book

We recommend the use of this book alongside a standard textbook to test your comprehension of the material and to assist in the organization of these pertinent medical facts into a cohesive whole. When preparing for exams, this book can be used as a quick, last-minute review of high-yield facts. Please remember that while this book will be very useful for the USMLE Step 2 CK and for reviewing the fundamentals of medical science, this review book should neither replace standard medical texts or lecture notes, nor substitute for sound clinical judgment. Rather, it is intended to clarify difficult concepts, review high-yield topics, and to provide you with a small portable book that is easy to use to quiz yourself and classmates on these concepts.

We hope you will find this review book helpful during your preparations for the USMLE Step 2 CK and throughout your medical education. Thank you for letting us help with your medical education!

SECTION I

Useful Facts for the Wards

This page intentionally left blank

Lab Values

Common Lab Values in the Nonpregnant Woman and Their Change During Pregnancy

Chemistries	Nonpregnant	Compared to Nonpregnant Woman
Sodium	135–145 mEq/L	Decreased
Potassium	3.5–5.1 mEq/L	Decreased
Chloride	98–106 mEq/L	Decreased
Bicarbonate	22–29 mEq/L	Decreased
Blood urea nitrogen (BUN)	7–18 mg/dL	Decreased
Creatinine	0.6–1.2 mg/dL	Decreased
Glucose	70–115 mg/dL	Decreased
Calcium	8.4–10.2 mg/dL	Decreased (due to decrease in albumin)
Phosphate	2.7–4.5 mg/dL	Unchanged
Magnesium	1.3–2.1 mg/dL	Decreased
Anion gap	7–16 mEq/L	
Osmolality	275–295 mOsm/kg	Decreased
Lipase	10–140 U/dL	
Amylase	25–125 U/dL	Unchanged
SGOT/AST	7–40 U/L	Unchanged
SGPT/ALT	7–40 U/L	Unchanged
GGT	9–50 U/L	Unchanged
Alkaline phosphatase	38–126 U/L	Increased
LDH	120–240 U/L	Unchanged
Uric acid	2.0–6.9 mg/dL	
Free T ₄	0.71–1.85 ng/dL	Unchanged
TSH	0.32–5.00 mIU/mL	Unchanged
Free T ₃		Unchanged
Total thyroxine, T ₄		Increased
Free T ₄		Increased

(Continued)

Common Lab Values in the Nonpregnant Woman and Their Change During Pregnancy (*Continued*)

Chemistries	Nonpregnant	Compared to Nonpregnant Woman
Urinalysis		
Color	Yellow	
Turbidity	Clear	
Specific gravity	1.003–1.035	
pH	4.5–8.0	
Ketones	Negative	Unchanged
Protein	Negative	Minimal increase
Blood	Negative	Unchanged
Glucose	Negative	Minimal increase
Nitrite	Negative	Unchanged
Leukocyte esterase	Negative	Unchanged
Osmolality	50–1400 mOsm/kg	
Sodium	40–220 mEq/day	
Potassium	25–125 mEq/day	
Hematology		
WBC	4,700–11,000/mm ³	Increased
RBC	3.8–5.7 × 10 ⁶ /mL	Increased
Hemoglobin	13.5–17.0 g/dL	Decreased
Hematocrit	39–50%	Decreased
Mean corpuscular volume (MCV)	80–96 fL	Increased
Mean corpuscular hemoglobin (MCH)	27–33 pg/cell	
Mean corpuscular hemoglobin concentration (MCHC)	32–36% hgb/cell	
Platelets	150–400 × 10 ³ /mL	Decreased due to dilution increases
Erythrocyte sedimentation rate (ESR)		
Red blood cell distribution width (RDW)	11.0–16.0%	
Segs (neuts)	35–73%	
Lymphocytes	15–52%	
Monocytes	4–13%	
Eosinophils	1–3%	
Basophils	0–1%	

Common Lab Values in the Nonpregnant Woman and Their Change During Pregnancy (*Continued*)

Chemistries	Nonpregnant	Compared to Nonpregnant Woman
Coagulation		
PT	12.3–14.2 seconds	Reduced
PTT	25–34 seconds	Reduced
Fibrinogen	200–400 mg/dL	Increased
D-dimer		Increased
Bleeding time	2–7 minutes	
Thrombin time	6.3–11.1 seconds	
Arterial Blood Gas		
pH 7.35–7.45		Increased (chronic, corrected respiratory alkalosis)
Paco ₂	35–45 mm Hg	Decreased
PaO ₂	80–100 mm Hg	Increased
HCO ₃ ⁻	21–27 mEq/L	Decreased
O ₂ saturation	95–98%	Unchanged

This page intentionally left blank

Common Procedures

These tables list several of the most common procedures performed in obstetrics and gynecology. Procedures not listed here are reviewed in topic-specific chapters.

Common Obstetric Procedures

Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Amniocentesis	Withdrawal of fluid from the amniotic sac to obtain fluid and cells for a variety of tests	Determine the presence of genetic diseases (e.g., Down syndrome, Tay-Sachs), fetal structural abnormalities (neural tube defects), fetal lung maturity, or intrauterine infection (i.e., chorioamnionitis)	0.5% risk of fetal loss because of bleeding, infection, preterm labor, or fetal injury	Usually performed using ultrasonographic guidance to reduce the risk of fetal loss
Cerclage	It is the placement of a suture into and around the cervix to hold it closed	Used to prevent cervical opening in an incompetent cervix and prevent preterm delivery or miscarriage	It is controversial whether a cerclage reduces the likelihood of a preterm delivery	It is usually performed between 12 and 14 weeks and removed before labor begins
8 Cesarean delivery	It is the delivery of the fetus by making an incision through the abdomen and uterus	It is used when a vaginal delivery would be harmful to the mother or fetus or it can be requested by the patient (elective)	The mortality rate is less than vaginal delivery; however, complications include postoperative adhesion, infection, and problems with the next birth (i.e., placenta previa, uterine rupture)	The incision can be made in two ways: (1) Classical-midline longitudinal incision (2) Lower uterine segment section—transverse cut above the bladder; more commonly used and less bleeding

Chorionic villus sampling (CVS)	A small cannula is passed through the cervix or transabdominally, and villus cells are aspirated for genetic analysis	Cells are taken for genetic studies	0.5% risk of fetal loss. Usually reserved for patients with a greater than 0.5% chance of having an abnormality in the fetus	CVS is usually performed in early pregnancy to allow earlier decision making regarding possible pregnancy termination
Circumcision	It is the removal of the foreskin from the penis of a newborn male	It has been performed as part of religious and social customs, and health reasons	The same as for any surgical operation (bleeding, infection, surgical damage)	There is controversy over the health value of the procedure
Episiotomy	It is a surgical incision made through the perineum to widen the vagina and facilitate delivery	Used when there are signs of fetal distress while in the vaginal canal, when the baby's head or shoulders are too large to pass, to lessen perineal trauma and reduce postpartum pelvic floor dysfunction	It is controversial whether episiotomy may cause more morbidity (postpartum pain, trouble defecating, dyspareunia)	Perineal massage with oils beginning around the 34th week may help relax the perineum and avoid an episiotomy
External cephalic version	It is the application of constant gentle pressure (between 36 and 39 weeks) to the abdomen of the mother with a breech fetus to place it in cephalic presentation	To position a breech fetus into cephalic presentation		Success rates are from 50–75%. Fetal monitoring is advised after the procedure as well as administration of Rh-immune globulin to Rh-negative women

(Continued)

Common Obstetric Procedures (*Continued*)

Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Fern test (amniotic fluid crystallization test)	Vaginal secretion from the posterior vaginal pool is collected with a sterile swab and placed on a clean slide to dry	It detects ruptured membranes or the leakage of amniotic fluid from the membranes surrounding the fetus during pregnancy. It is helpful to diagnose premature rupture of membranes. If positive, the amniotic fluid will form a fernlike pattern of crystallization		To be used in conjunction with the Nitrazine test (pH test to determine presence of amniotic fluid. pH paper will turn blue, demonstrating an alkaline pH)
Forceps delivery	It is an instrument applied to the fetal head used for assistance in vaginal delivery	Used to provide traction to augment and/or direct the expulsive forces during the second stage of labor (i.e., prolonged second stage of labor)	Maternal complications: lacerations of the cervix and birth canal, blood loss, and hematomas. Fetal complications: bruising, cephalohematomas, facial and head lacerations	Four types of forceps are classified as outlet, low, mid, and high depending on the station of the fetal head. High forceps is not recommended by American College of Obstetricians and Gynecologists (ACOG)
Pelvimetry	It is the assessment of the size of the female pelvis in relation to the space needed for the birth of a baby	It has been used to determine whether a natural or vaginal delivery is possible, or whether a caesarean delivery is indicated		Can be performed by physical exam, radiography, or MRI

Percutaneous umbilical blood sampling (PUBS)	A needle is placed transabdominally and fetal blood is obtained from the umbilical cord under real-time ultrasonographic guidance	Fetal blood gas and metabolic status, fetal hemogram, fetal blood chemistries, and fetal genetic studies can be performed	The risk is similar to that of an amniocentesis (0.5%). There may also be bleeding at the umbilical puncture site	Also referred to as <i>cordocentesis</i>
Tubal ligation	It is a permanent form of female sterilization where the fallopian tubes are severed, sealed, or “pinched shut” to prevent fertilization	It is used when fertility is no longer desired	No major complications	Tubal reversal is possible. Tubal ligation does not affect hormone production, libido, or menstrual cycle
Ultrasound imaging	Specific parts of the body are exposed to low-energy sound waves which produce images reflective of the structure and movement of the body’s internal organs	Establish the presence of a living embryo/fetus, estimate the age of the pregnancy, diagnose congenital abnormalities, evaluate the position of the baby, evaluate the position of the placenta, determine if there are multiple pregnancies, determine the amount of amniotic fluid around the baby, check for opening or shortening of the cervix or mouth of the womb	It is a painless procedure and does not involve any ionizing radiation	Doppler ultrasound can be used in conjunction to evaluate blood flow through a blood vessel
Vacuum extraction	It is a suction cup device that is applied to the fetal head to help in delivery	Indications are similar to that of forceps delivery	Less complications as compared to forceps delivery, but hematomas and abrasions to the fetal scalp can occur	

Common Gynecologic Procedures

Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Angiography	It is a medical imaging technique in which a radiographic contrast medium is injected into the blood vessel and an x-ray picture is taken to visualize the blood vascular system	It can be used to locate the source of continued bleeding from postoperative procedures, visualize bleeding from sites infiltrated by cancers, help assist in the embolization of the uterine arteries in hematomas, and to reduce the size of uterine myomas		
Bimanual pelvic examination	Two fingers are placed in the vagina and the flat of the opposite hand is placed on the lower abdominal wall. Gentle palpation and manipulation should delineate the position, shape, mobility, tenderness, and size of the uterus and adnexal structures. See Figure 2-1, page 22	Part of routine pelvic exam and part of investigation for gynecologic pathology. Tenderness may be elicited on direct palpation or on movement/stretching of pelvic structures (i.e. acute salpingitis or pelvic inflammatory disease [PID])	Difficult to elicit any information on obese patients or uncooperative patients	
Cervical conization	It is a surgical procedure that involves excising a cone-shaped sample of tissue that includes the entire cervical	It is used for either diagnostic or therapeutic reasons. The test is done when results of a cervical biopsy indicate precancerous cells in the area or cervical	An early complication is excessive bleeding. Infrequent complications include cervical stenosis or incompetence	Conization can be performed using a knife (cold knife cone), laser excision, and

	transformation zone and a portion of the endocervical canal. The sample is then examined for any signs of malignancy	cancer. It may also be done if the cervical biopsy has not revealed the cause of an abnormal Pap smear		electrocautery (large loop excision of the transformation loop electrosurgical excision procedure [LEEP])
Colposcopy	It is the direct magnified inspection of the surface of the cervix, vagina, and vulva, using a light source and a binocular microscope	Used to facilitate detailed evaluation of a suspect malignancy and to assist in directed biopsies of suspicious areas. It can also be used to detect inflammatory or infectious changes, and traumatic injuries to the cervix, vagina, and vulva	Minimal risk	Can be performed in the office and rarely requires any anesthesia
Computed axial tomography (CT)	It is a form of imaging that uses x-ray information to generate detailed cross-sectional images of internal structures	Can help evaluate for pelvic masses, signs of adenopathy, and plan for radiation therapy CT heavily relies on the use of IV contrast and this contrast can cause kidney damage. Should not be used in patients with advanced renal failure	Superior contrast resolution which provides a significant amount of information. However, it is considered as a moderate to high radiation diagnostic technique	CT is best suited to study bone and calcifications in the body, or vessels and bowel which have been enhanced with contrast

(Continued)

Common Gynecologic Procedures (*Continued*)

Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Cryotherapy	It is a technique to destroy tissue by freezing with liquid nitrogen or liquid carbon dioxide	Most frequently used to destroy dysplastic sections of the cervix and other benign lesions (i.e., condyloma)	Minimal risk. Inexpensive and generally effective, although not as precise as laser ablation	Not as useful for treating changes in the upper cervix; a cone biopsy is recommended instead
Culdocentesis	It is the passage of a needle into the cul-de-sac to obtain fluid from the pouch of Douglas	It is a diagnostic procedure to check for abnormal fluid. Blood fluid may indicate a ruptured ectopic pregnancy; pus-filled fluid indicates acute infection; ascetic fluid may indicate cancer	There is a slight risk of puncturing any mass or uterine wall	Not as commonly performed today with advances in ultrasound technology
Dilation and curettage (D&C)	It is a process of opening and dilating the cervix using a series of graduated dilators followed by curettage, or scraping of the uterine lining	It may be used to take an endometrial biopsy, remove polyps, other tumors, or excess endometrial lining, or to treat cases of incomplete spontaneous abortion	If done roughly, uterine perforation may occur or scarring could develop leading to infertility	It is generally performed under local or general anesthetic in the operating room
Dilation and evacuation (D&E)	It is a procedure used in the second semester to remove the products of conception by	It is used in pregnancy termination	Mild bleeding, cramping. There is also slight risk of uterine perforation and scarring	D&E is the most common and safest

Fern test for ovulation	<p>first dilating the cervix and then using destructive grasping forceps. Vacuum aspiration may also be used to facilitate the removal</p> <p>Cervical mucus is spread on a clean, dry slide and allowed to dry in the air. A fern-frond pattern is seen under the microscope when ovulation has failed to occur. A non-frond-like pattern is seen when ovulation has occurred</p>	To determine the presence or absence of ovulation at the ovulatory time of cycle	Minimal risk	<p>procedure used (95% of the time) for an abortion in the second trimester</p> <p>A fern-frond pattern is induced by unopposed estrogen. Progesterone inhibits the effects of estrogen on ferning and makes the mucus thick and cellular (seen in ovulation). See Figure 2-2, page 22</p>
Genital tract biopsy	It is the removal of tissue from lesions of the vulva, vagina, cervix, and endometrial cavity	Allows for histopathologic assessment of the specimen to evaluate any malignancy and may also serve to fully excise small lesions	Minimal risk	Usually performed in the office and requires only local anesthetic

(Continued)

Common Gynecologic Procedures (*Continued*)

Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Hysterectomy	It is the removal of the uterus. It can be performed by entering the abdomen (abdominal hysterectomy) or extracting the uterus through the vagina (vaginal hysterectomy)	It may be indicated for patients with benign or malignant changes in the uterine wall or cavity, for menstrual disturbances or abnormal bleeding, endometriosis, uterine prolapse, or chronic pelvic pain It is not recommended for the sole purpose of sterilization	Advantages include the elimination of future pregnancies, cessation of menses, and possibility of uterine and cervical cancer	There are different types of hysterectomy: <i>Total:</i> removal of all of the uterus and not the fallopian tubes or ovaries <i>Subtotal or supracervical:</i> body of the uterus is removed near the level of the internal cervical os, leaving the cervix in place <i>Radical:</i> is a cancer surgery procedure where the uterus is removed with wide margins of surrounding tissues

Hysterosalpingography	It is an x-ray of the uterus, fallopian tubes, and abdominopelvic cavity that involves the injection of dye through the cervix	It is useful to assess the size, shape, and anatomy of the uterine cavity for evaluation of infertility or genital anomalies	There is a risk of infection of the uterus or pelvis, bleeding, pain, and allergic reaction to the dye	Can be performed with hysteroscopy and hysterosonography
Hysteroscopy	It is a small endoscope which has a built-in viewing camera that allows direct visualization of the endocervix and endometrial cavity	It is used for evaluation of bleeding or structural abnormalities which may cause infertility, or location of missing intrauterine devices; it is also used for therapeutic reasons (i.e., polypectomy, endometrial ablation, removal of the uterine septum)	Mild bleeding, cramping	Fluid may be used to distend the uterine cavity. Usually performed as an outpatient procedure under local or general anesthesia
Hysterosonography	It is a new minimally invasive technique used to visualize the uterine cavity by use of ultrasound and slow infusion of sterile saline	It is used to evaluate abnormal growths inside the uterus; abnormalities of the tissue lining the uterus (the endometrium); or disorders affecting deeper tissue layers	Does not use ionizing radiation, contrast media, or invasive surgical techniques. Mild bleeding and cramping may occur postprocedure	It is useful as a screening test to minimize the use of more invasive diagnostic procedures, such as tissue biopsies and D&C

(Continued)

Common Gynecologic Procedures (*Continued*)

Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Laparoscopically assisted vaginal hysterectomy (LAVH)	It is a procedure using laparoscopic surgical techniques and instruments to remove the uterus and/or tubes and ovaries through the vagina	Indications are similar to that of a hysterectomy	Advantages include avoiding a large abdominal incision, less postoperative pain and recovery time. Disadvantages include technical difficulty leading to increased operative time and increased anesthesia exposure	Recently, laparoscopic supercervical hysterectomy (LSH) has been advocated by some. This preserves the cervix and its supporting structures, and allows for lubrication and maintenance of sexual response
Laparoscopy	It is the inspection and manipulation of tissue within the abdominal cavity using endoscopic instruments (camera)	It is used for diagnostic and therapeutic purposes. It can help in diagnosis of the source of pelvic pain, pelvic masses, infertility, and congenital abnormalities. It can treat endometriosis, lyse adhesions, and be used to perform minimally invasive surgeries (eg, bilateral salpingo-oophorectomy [BSO])	Less morbidity than laparotomy, but injury to the bowel and vessels can occur. Postsurgical infection and bleeding is possible	The abdominal cavity is usually distended with carbon dioxide or nitrous oxide gas to facilitate viewing and prevent bowel injury

Laparotomy	It is a surgical maneuver involving an incision through the abdominal wall to gain access into the abdominal cavity	It is for both diagnostic and therapeutic purposes. Exploratory laparotomy is used to identify the cause of disease; whereas, in therapeutic laparotomy, the nature of disease is known	The operative procedure time may be less time consuming compared to laparoscopic technique, but there is more postoperative pain and recovery time	The Pfannenstiel's incision is a transverse incision below the umbilicus and just above the pubic symphysis. It is the incision of choice for cesarean delivery and abdominal hysterectomy for benign disease
Laser vaporization	High-energy light waves are used to destroy abnormal cells and dysplastic tissue	Can be used to treat cervical dysplasia. Also used to make incisions	Painless can replace cryotherapy. However, due to its sophistication it is expensive	Infrared and CO ₂ lasers are commonly used in the office, and can be coupled to colposcopes
Loop electro-surgical excision procedure (LEEP)	It is an instrument that consists of a thin wire loop electrode and an electro-surgical generator, and uses electrical current to cut away cervical tissue in the immediate area of the loop wire	It has both diagnostic and therapeutic uses. It is most commonly used to excise vulvar condylomas and cervical dysplasias. It is also used for cone biopsies of the cervix	Minimal pain, minimal damage to the surrounding tissue, and low morbidity	It is an office procedure and requires only local anesthetic

(Continued)

Common Gynecologic Procedures (*Continued*)

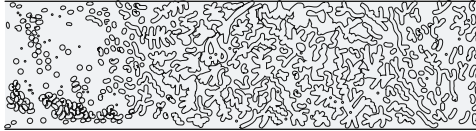
Procedure	Description	Indications/Contraindications	Benefits/Risks	Comments
Magnetic resonance imaging (MRI)	It is a form of imaging that is based on the magnetic characteristics of various atoms and molecules in the body. It uses nonionizing radiofrequency signals	Used to evaluate any soft tissue mass but emerging clinical applications include assessment of breast lesions and staging of cervical cancer	There are no harmful effects to the fetus	MRI is best suited to evaluate soft tissue or non calcified tissue
Mammography	It is an x-ray examination of the breasts. The breasts are placed between two plates and pressed flat. An x-ray is taken of each	It is used as a diagnostic and screening modality for breast masses	Lower dose radiation exposure makes this exam safe	Women should receive screening mammograms every 1–2 years when they reach age 40

Pap smear	It is a microscopic examination of cells scraped from the ectocervix and endocervix	Mainly used as a screening modality, it can detect cancerous or precancerous conditions of the cervix	ACOG recommends annual Pap smear screenings from 3 years after the start of sexual intercourse but not later than age 21 years. Women at risk should have annual Pap smears; women who have had three consecutive negative tests can be screened every 2–3 years
Schiller’s test for neoplasia	Iodine solution is placed on areas of the cervix and vagina that are suspect for dysplasia. Any portion of the tissue that does not absorb the dye is biopsied for signs of cancer	It is performed on areas of the cervical or vaginal mucosa where malignant changes are suspected	Colposcopy is a more accurate diagnostic tool.
Suction and curettage (S&C)	It is a procedure used in the first trimester to remove the products of conception by first dilating the cervix, followed by suction and scraping inside the uterus	It is used in pregnancy termination	Suction curettes are preferred because they are less likely to cause damage to the uterus. Other risks may include bleeding and cramping
			This is an outpatient procedure and may be done in the office using local anesthetics

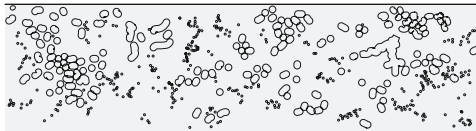


Figure 2-1 Bimanual examination of the uterus and adnexa.

Normal cycle, 14th day



Midluteal phase, normal cycle



Anovulatory cycle with estrogen present

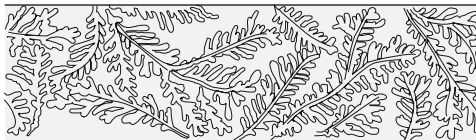


Figure 2-2 Fern test for ovulation.

Female Anatomy

BONY PELVIS

What forms the bony pelvis?

Sacrum
Coccyx
Paired hip bones (ilium, ischium, and pubis)

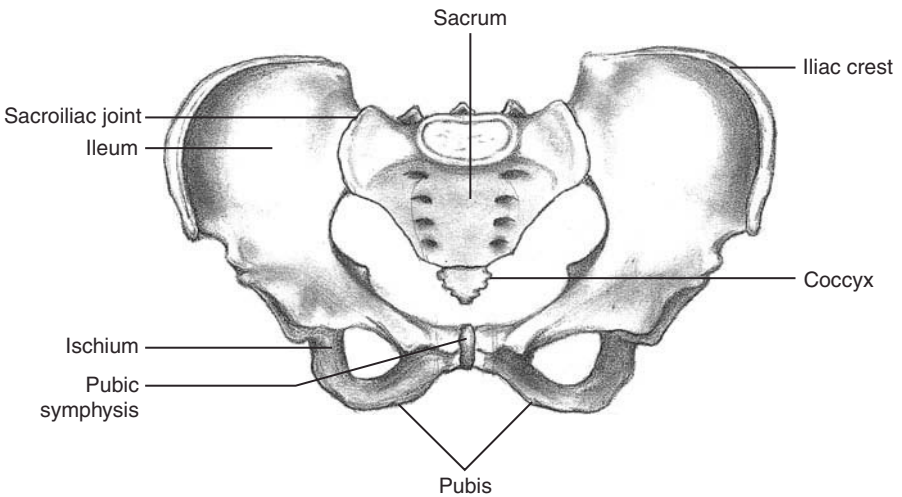


Figure 3-1 The bony pelvis.

What is the sacral promontory and what is its significance?

The most anterior projection of the sacrum, it is a landmark for the insertion of a laparoscope as it demarcates the point of bifurcation of the common iliac arteries

What is the arcuate line and what is its significance?

Also called the linea semicircularis, it is the line that marks the pelvic brim. It lies between the first two segments of the pelvis and demarcates the site

What is the ischial spine and what is its significance?

of entry of the inferior epigastric artery into the rectus sheath

It is the medial protrusion of the ischium and an important landmark for giving a pudendal nerve block. It also provides a good landmark by which to assess progression of fetal descent during labor

What are the four pelvic configurations found in females and how common are each?

Gynecoid (50%); anthropoid (25–50%); android (16–33%); platypelloid (3%)

Describe the shape of a gynecoid pelvis.

It is **wider** and **lower** than the male pelvis. The pubic arch is wide and round, the iliac bone is flatter, and the ischial spines are not prominent. The anterior-posterior and transverse diameters are roughly equal. All of this makes the pelvic basin more spacious

Describe the shape of an anthropoid pelvis.

It is **heart-shaped** with a wider anterior-posterior diameter than a transverse diameter

Describe the shape of an android pelvis.

It is **narrower** and **taller** than the gynecoid pelvis

Describe the shape of a platypelloid pelvis.

It has a wider transverse diameter than an anterior-posterior diameter

What is the pelvic inlet?

The superior circumference of the lesser pelvis. Its boundaries include the sacral promontory, the pubic ramus and symphysis pubis, and the linea terminalis

Describe the following:

Obstetric conjugate

The shortest pelvic diameter through which the fetal head passes; it can only be measured radiographically; the distance from the sacral promontory to the symphysis pubis; the normal diameter is >10 cm

True conjugate

The AP diameter that lies between the sacral promontory and the superior symphysis pubis

Diagonal conjugate

Only one measured clinically; the distance between the sacral promontory and the inferior margin of the symphysis pubis

PELVIC ORGANS

What is the major blood supply to the pelvic organs?

Internal iliac artery (aka hypogastric artery)

Describe the branches of the internal iliac artery.

Coming off the common iliac artery, the internal iliac typically but not always divides into anterior and posterior trunks

- Anterior trunk:
- Superior vesical
 - Middle vesical
 - Inferior vesical
 - Middle hemorrhoidal
 - Obturator
 - Internal pudendal
 - Inferior gluteal
 - Uterine
 - Vaginal

- Posterior trunk:
- Iliolumbar
 - Lateral sacral
 - Superior gluteal

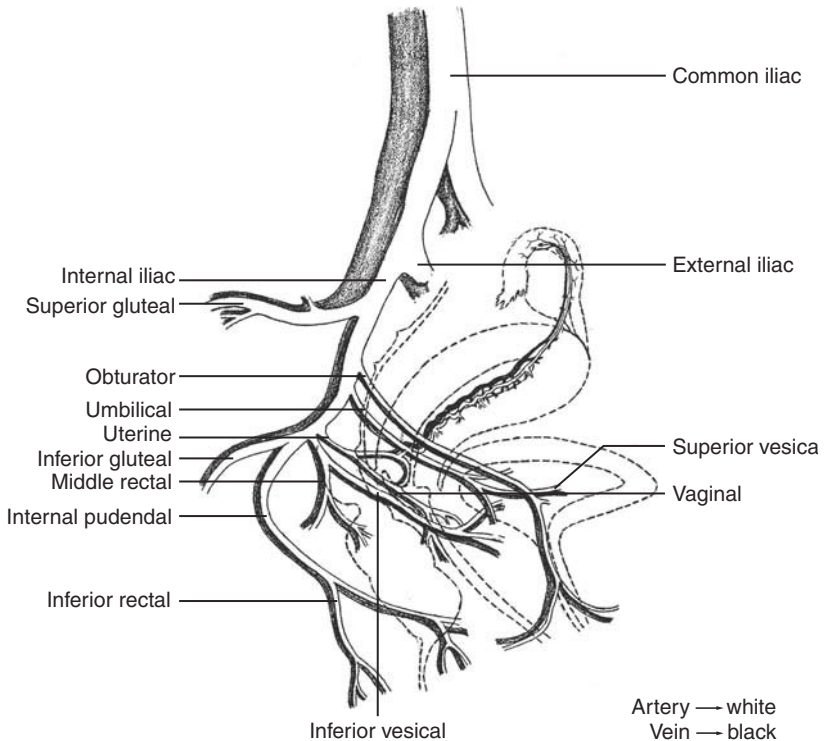


Figure 3-2 Arteries and veins of the female reproductive system.

Describe the position of the vagina in the pelvis.

It extends from the vulva to the cervix. The bladder lies anterior, separated from the vagina by the vesicovaginal septum. The rectum lies posterior, separated from the vagina by the rectovaginal septum

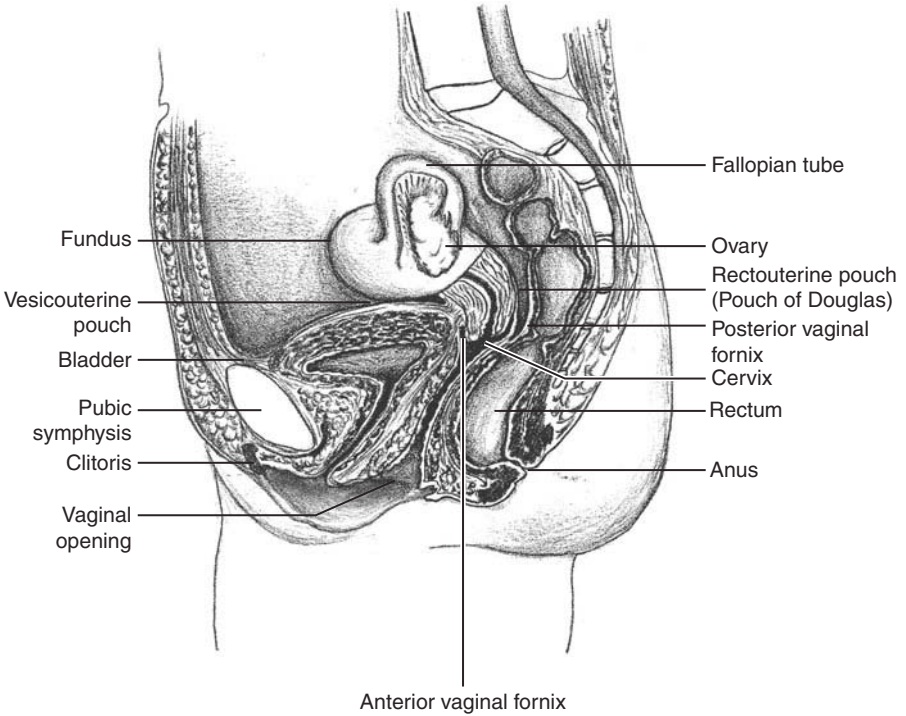


Figure 3-3 Midsagittal view of pelvic viscera.

What is the major blood supply to the vagina?

The vaginal artery (can also arise from hypogastric artery or the uterine artery)

What is the innervation to the vagina?

Parasympathetics via S2-S4 for the upper two-thirds and general somatic efferent to the lower one-third via the pudendal nerve; there are no other specific nerve endings

What is the lymphatic drainage of the vagina?

The upper two-thirds drain into the internal and external iliac nodes. The lower one-third drains into the inguinal nodes

Describe the layers of the vaginal wall (from interior to exterior).

It is composed of a smooth muscle layer (arranged in an outer longitudinal layer, circumferential layer, and a poorly differentiated inner longitudinal layer) followed by a mucosal layer. The mucosa of the vagina is lined by stratified squamous epithelium

Describe the vaginal fornices.

The area of the vagina surrounding the cervix; divided into anterior, posterior, and two lateral fornices

Describe the position of the uterus in the pelvis.

It is covered by the peritoneum (vesicouterine peritoneum anteriorly and the peritoneal reflection called the pouch of Douglas posteriorly) and is between the bladder and rectum, directly touching the bladder

What ligaments support the uterine position in the pelvis?

Uterosacral, cardinal, round, and broad

What are the anatomic portions of the uterus?

Fundus (most superior portion of the uterus [above the entrance of the fallopian tubes]); Corpus (the uterine body); Isthmus (area where the uterus begins to constrict); Cervix (inferior portion of the uterus that extends into the vagina)

What is meant by flexion of the uterus?

The angle between the long axis of the uterine body and the cervix; can be anteflexed or retroflexed

What is meant by the terms anteversion and retroversion?

The angle between the cervix and the vagina

What are the histological layers of the uterine wall (from interior to exterior)?

Endometrium (ciliated columnar epithelium, glands, and spiral arteries); Myometrium (smooth muscle and connective tissue); Serosa

What are the major blood supplies to the uterus?

Uterine and ovarian arteries (there are extensive anastomoses which allow for ligation of the uterine or internal iliac during hemorrhage to control bleeding without compromising blood supply to the uterus)

Describe the course of the uterine artery in the pelvis.

Arises from the internal iliac → divides into cervicovaginal and uterine branches → uterine branch

What is the relationship of the uterine artery with the ureter?	divides into the fundal, ovarian, and tubal arteries and provides radial arteries that perforate into the uterus
What is the lymphatic drainage of the uterus?	The ureter crosses under the artery ("water under the bridge") 2 cm lateral to the cervix
Describe the following portions of the cervix:	Internal and external iliac nodes (although the fundus can drain into the para-aortic lymph nodes)
Portio	The portion of the cervix that is visible from the vagina
Cervical canal	Area in between the internal and external os
External os	Inferior opening of the cervix into the vagina
Internal os	Superior opening of the cervix into the uterine cavity
What is the major blood supply to the cervix?	Cervical branch of the uterine artery
What is the innervation to the cervix?	Autonomics, sympathetics (T12–L3), and parasympathetics from S2, S3, S4
What is the lymphatic drainage of the cervix?	Internal and external iliacs to the common iliacs
What are the fallopian tubes?	These are 8–10 cm tubes that extend laterally from the body of the uterus
What are the histological layers of the fallopian tubes (from interior to exterior)?	Mucosal layer (ciliated columnar epithelium covered in cilia); Muscular layer (external longitudinal layer and internal circular layer); Serosa
What are the segments of the fallopian tubes (from medial to lateral)?	Interstitial, isthmic, ampullary, infundibular, and fimbrial
What is the major blood supply to the fallopian tubes?	Uterine and ovarian arteries
What is the lymphatic drainage of the fallopian tubes?	Aortic nodes
Describe the position of the ovaries in the pelvis.	They lie in the ovarian fossae, which are lateral to the uterus in the pelvic sidewall where the common iliac artery bifurcates

What are the histological layers of the ovary (from interior to exterior)?

Medulla (connective tissue and blood supply); cortex (ova and tunica albuginea); germinal epithelium

What is the major blood supply to the ovaries?

Branches of the uterine artery and the ovarian arteries

Where do the right and left ovarian veins drain?

The right ovarian vein drains through the inferior vena cava (IVC) and the left drains through the left renal vein

Describe the position of the bladder in the pelvis.

It sits between the abdominal wall/pelvic bones (anteriorly) and the vagina/cervix (posteriorly)

What are the major blood supplies to the bladder?

Superior vesical artery, inferior vesical artery, and middle hemorrhoidal artery

What is the innervation to the bladder?

Parasympathetics, sympathetics, and the pudendal nerve (innervates the external urethral sphincter)

What is the lymphatic drainage of the bladder?

Internal iliac nodes

Describe the course of the ureter through the abdomen and pelvis.

In the abdomen, it is retroperitoneal on the anterior surface of the psoas muscle. It then crosses the common iliac artery (the right at the bifurcation and the left 1–2 cm above the bifurcation) and then follows the pelvic wall laterally to the medial broad ligament. It then crosses under the uterine artery and courses medially to enter the cardinal ligaments. It enters the bladder at the trigone

How is the ureter distinguished during surgery?

(1) Ureteral peristalsis
(2) Auerbach's plexus (only on the ureteral anterior surface)

What are the most common sites of ureteral injury?

(1) Pelvic brim
(2) Where the ureter crosses the uterine artery
(3) Bladder trigone

How can the rectum be differentiated from the rest of the bowel?

It begins after the sigmoid colon where the mesentery ends and it does not have teniae coli or appendices epiploicae

Why is the rectum sometimes injured during vaginal surgery?

Because of its proximity to the posterior vaginal wall

What is the blood supply to the rectum?

Hemorrhoidal arteries (superior, middle, and inferior)

What is the lymphatic drainage of the rectum?

Iliac nodes

Name the ligamentous structures in the pelvis. Describe each.

Broad ligaments

Reflections of peritoneum from the lateral margin of the uterus to the pelvic sidewall; contain the uterine vessels and ureters in their base

Round ligaments

Homologue of the male gubernaculum; also contain an artery of the round ligament, connects lateral uterine fundus to the upper labia majora

Uterosacral ligaments

Extend from posterior-inferior uterus to the presacral fascia

Cardinal ligaments

Support the uterus; at base of broad ligament, contain uterine artery

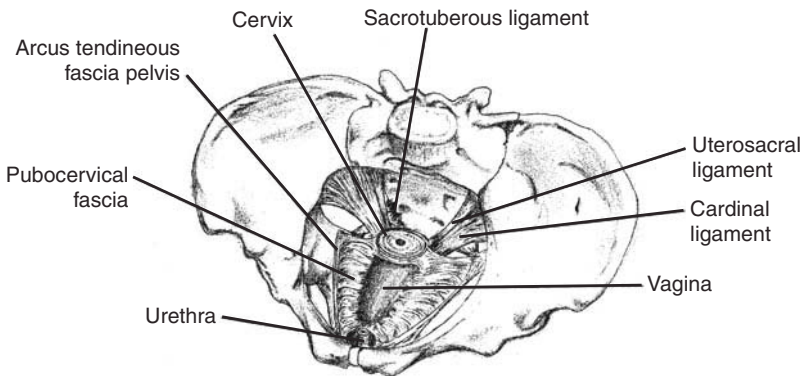


Figure 3-4 Ligaments of the female pelvis.

Describe the clinical manifestations of damage to each of the following nerves:

Femoral nerve

Problems with hip flexion

Obturator nerve

Problems with adduction of thigh/hip; loss of sensation in medial thigh

Genitofemoral nerve

Loss of sensation in perineum

Peroneal nerve

Foot drop; loss of sensation in lateral lower leg and dorsum of the foot

During which types of surgeries can each of the following nerves be injured?

Femoral nerve

Abdominal surgery or inguinal node dissection

Obturator nerve

Radical hysterectomy or node dissection

Genitofemoral nerve

Radical hysterectomy or node dissection

Peroneal nerve

Improper placement of legs in stirrups

PERINEUM

What is the perineum?

Area between the mons pubis and the buttocks

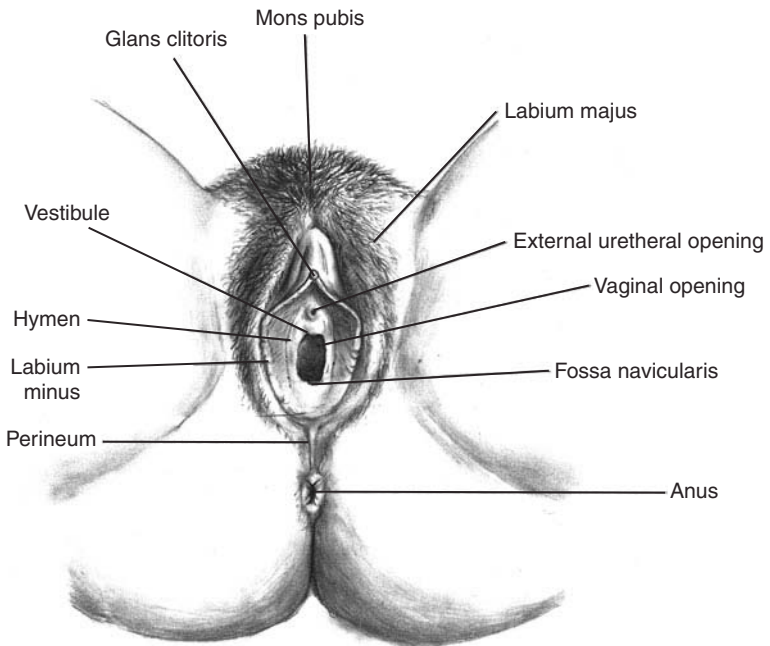


Figure 3-5 External genitalia of the adult female.

What is the major blood supply to the perineum?

Internal pudendal artery

What is the major innervation to the perineum?

Pudendal nerve

What is the lymphatic drainage of the perineum?

Inguinal nodes

Name the muscles of the perineum

Superficial and deep transverse perineal

Bulbocavernosus

Ischiocavernosus

External anal sphincter

Urethral sphincter

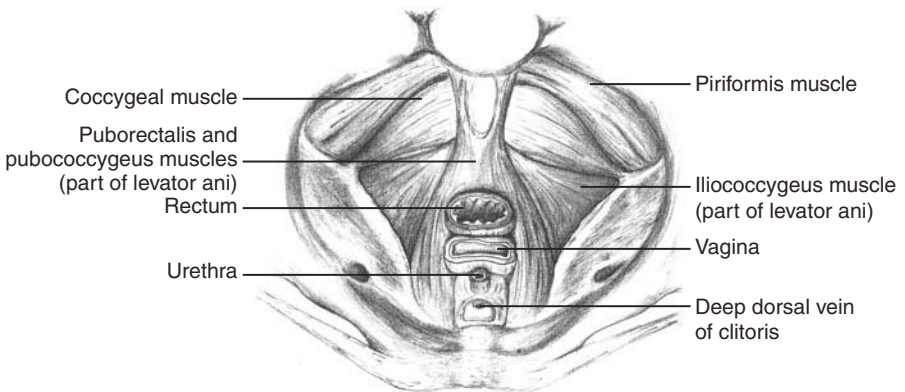


Figure 3-6 Pelvic diaphragm—superior view.

What is the urogenital diaphragm?

Muscles between the pubis symphysis and the ischial tuberosities that support the perineum

Name the muscles of the urogenital diaphragm.

Urethral sphincter and deep transverse perineal

What is the pelvic diaphragm?

Muscles from the upper pubis and ischium to the rectum that support the perineum

What muscles comprise the pelvic diaphragm?

Levator ani; coccygeal

What structures comprise the external genitalia of the female?

Mons pubis, labia majora, labia minora, clitoris, vestibule, urethral opening, vagina, perineum, and perineal body

What are the muscles of the vulva?	Superior transverse perineal Bulbocavernosus Ischiocavernosus (All lie superficial to the urogenital fascia [perineal membrane])
What is the difference between the labia minora and the labia majora?	Labia majora are skin folds with adipose tissue that connect with the mons anteriorly and the perineum posteriorly. They contain hair follicles, sweat glands, and sebaceous glands. Labia minora are narrower skinfolds medial to the labia majora that contain sweat and sebaceous glands but no hair follicles or adipose tissue. They connect with the prepuce and frenulum of the clitoris anteriorly and the labia majora and perineum posteriorly
What is the frenulum?	The anterior junction of the labia minora; ventral to the clitoris
What is the vestibule?	The area between the labia minora, clitoris, and perineum where the vaginal and urethral openings are located
What are Bartholin's glands?	Glands found deep to the vestibule, lateral to the vagina that open into the vestibule
Where is the urethral opening in relation to other perineal structures?	It is located in the anterior aspect of the vestibule
What are Skene's glands?	Also called paraurethral glands; they are homologues of the prostate that surround the urethral opening and empty into the vestibule
What are the embryologically homogenous male structures for the labia majora and the clitoris?	Scrotum for the labia majora and glans penis for the clitoris

ABDOMINAL WALL

What are the layers of the abdominal wall?	Skin → subcutaneous fat → outer fatty layer (Camper) of superficial fascia → inner membranous layer (Scarpa) of superficial fascia →
---	--

	<p>anterior rectus sheath → rectus abdominus muscle → posterior rectus sheath → preperitoneal fat subserous or extraperitoneal fascia → parietal peritoneum</p>
Describe the blood supply to the lower abdomen.	<p>Deep circumflex iliac (off of the external iliac) and external pudendal artery</p>
What is the arcuate line?	<p>Also called the linea semicircularis; above it the posterior aponeurosis of the internal oblique and the aponeurosis of the transversus abdominus comprise the posterior portion of the rectus sheath. Below the arcuate line they contribute to the anterior leaflet of the rectus sheath</p>
What is the linea alba?	<p>The medial aspect of the rectus abdominus; formed by the fusion of the aponeuroses of the anterior abdominal muscles</p>
What are the linea semilunara?	<p>The lateral borders of the rectum abdominus muscles</p>
What structures pass through the inguinal canal in the female?	<p>Round ligament An artery and vein passing to the uterus Extraperitoneal fat</p>

SECTION II

Topics in Gynecology

This page intentionally left blank

General Gynecology

Menstrual Cycle Physiology

What is the average duration of the menstrual cycle, duration of menses, and amount of blood loss during menses?

The average duration of the menstrual cycle is **28 days**. The average duration of menstrual flow is **4 days**. On an average, women lose less than **60 mL of blood** during each menses

What are the two phases of the menstrual cycle and how long does each last?

Follicular (or **proliferative**) phase and the **luteal** (or **secretory**) phase, separated by **ovulation**. (Follicular/luteal describe the ovarian changes, proliferative/secretory describe the endometrial changes)

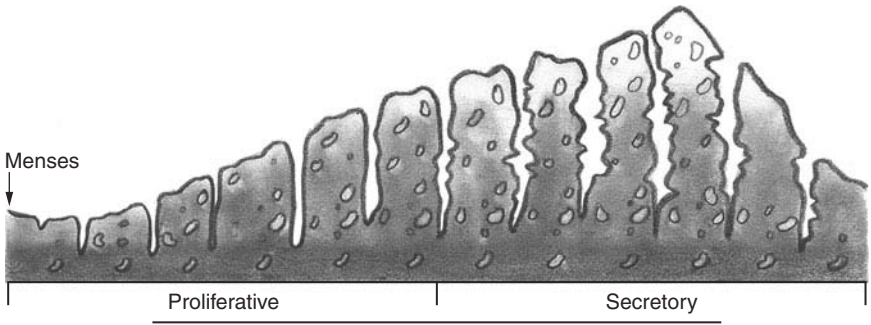
What causes the variability in the length of the menstrual cycle?

By convention, day 1 marks the onset of menses. The follicular phase begins on day 1 and lasts approximately **14 days** (days 1–14) in a 28-day cycle, until ovulation occurs. The luteal phase then commences and lasts until approximately day **28** (days 14–28)

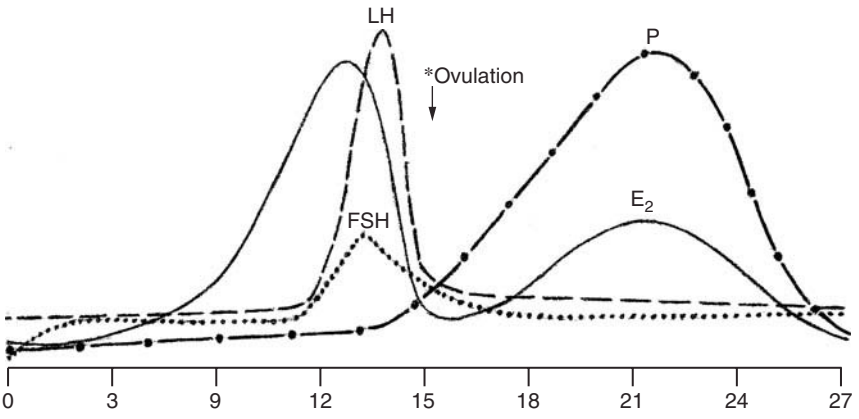
Describe the hormone pathway involved in the menstrual cycle (see Fig. 4-1) and name which structures produce each hormone

The duration of the follicular phase (the luteal phase is constant)

The cycle begins in the arcuate nucleus of the **hypothalamus** where **gonadotrophin-releasing hormone (GnRH)** is released in a pulsatile fashion. GnRH stimulates the **anterior pituitary** to release **follicle-stimulating hormone (FSH)** and **luteinizing hormone (LH)**. These gonadotropins then cause the ovaries to release the sex steroid hormones **estradiol** and **progesterone**. Estrogen



Endometrial histology



Endocrine cycle



Average basal body temperature

Figure 4-1 The menstrual cycle.

and progesterone feedback negatively on both the hypothalamus and the pituitary gland

What is happening in the ovary during the menstrual cycle?

The ovary begins with approximately one million **primordial follicles at birth** (20 million at week

20 in utero). Each follicle contains an oocyte arrested in **prophase of meiosis**. The oocyte is surrounded by pre-granulosa cells and these are surrounded by pre-theca cells. In the follicular phase, FSH stimulates the pre-granulosa cells to become granulosa cells. The **granulosa cells** secrete **estradiol**. The pre-theca cells in turn become **theca cells** and secrete **androgens**, which are aromatized by the granulosa cells into estradiol.

One follicle with the highest number of granulosa cells, FSH receptors, and estradiol production becomes the **dominant follicle** and all other follicles become atretic. This follicle is released during ovulation and becomes the **corpus luteum**. The corpus luteum secretes progesterone and a smaller amount of estrogen during the follicular phase of the cycle. If fertilization does not occur, it degenerates into the **corpus albicans**.

What is the function of the corpus luteum?

Secretion of **progesterone** and **estradiol**. It is the only structure that produces progesterone in significant quantities which sustains the pregnancy until the placenta is developed.

What is happening to hormone levels in the follicular phase?

At menstruation, concentrations of **estradiol**, **progesterone**, and **LH** are at their **lowest** point. **FSH** and **LH** levels begin to rise in response to the low estrogen and progesterone.

Estradiol levels, secreted from the dominant ovarian follicle, begin to rise by day 4. Just before ovulation, estradiol levels peak. This peak causes a positive feedback on LH secretion, leading to the **LH surge** and a **smaller FSH surge**, which results in **ovulation** 30–38 hours later. **Progesterone** levels remain **low** throughout the follicular phase.

What is happening to hormone levels in the luteal phase?

The LH surge causes granulosa and theca cells to secrete **progesterone**.

and smaller amounts of **estrogen**. **Progesterone peaks** 3–4 days after ovulation.

Estrogen levels decrease immediately after ovulation but slowly rise with the growth of the corpus luteum. Progesterone and estrogen (at low to moderate levels) both act via negative feedback to **suppress LH and FSH**. If fertilization and implantation do not occur, progesterone and estradiol levels diminish after 11 days. FSH increases as the corpus luteum regresses

What is happening to the endometrium in the proliferative phase?

At menses, the **endometrium sloughs off** until it becomes a thin line. During the proliferative phase estradiol levels rise, resulting in the proliferation of the uterine endometrium. The endometrium becomes **thicker** and **more glandular** and the **spiral arteries elongate**. On ultrasound, it appears as a “**triple stripe**” pattern

What is happening to the endometrium in the secretory phase?

The progesterone released from the corpus luteum leads to **slowing of endometrial proliferation**, reorganization of the glands (resulting in a more **edematous stroma**), and further **coiling of the spiral arteries**. This results in the loss of the “triple stripe” pattern and its replacement with a **uniformly bright** endometrium. If pregnancy does not occur, the endometrium degenerates

What are the primary clinical manifestations of estradiol and progesterone during the menstrual cycle?

Estradiol

Endometrium: thickens stroma and elongates glands (creates **proliferative endometrium**); Endocervix: stimulates secretion of thin, **watery mucus**. Produces “**ferning**” pattern when spread on a glass slide; Vagina: promotes **vaginal thickening**

Progesterone

Endometrium: causes tissue to become edematous and blood vessels to thicken and twist (creates **secretory endometrium**); Endocervix: **thickens** endocervical

<p>What layer of the endometrium sloughs off during menses?</p> <p>What hormone mediates menstrual cramps and how is it synthesized?</p>	<p>mucus, causing it to become stringy; Breast: stimulates acinar glands, causing breasts to round; Other: raises basal body temperature by 0.6–1°F. Causes some women to have the emotional, physical, and behavioral changes of premenstrual syndrome (PMS)</p> <p>The functionalis layer (inner layer) sloughs off after glandular and stromal degeneration</p> <p>Prostaglandins, especially PGF_{2α}. It is released by the secretory endometrium in response to progesterone and causes uterine contractions</p>
--	--

Family Planning

CONTRACEPTION OVERVIEW

<p>How many American women use contraception and what are the rates of unintended or mistimed pregnancy in the United States?</p> <p>What are some of the reasons for contraception failure?</p> <p>What are the two measures for effectiveness of a contraceptive method?</p> <p>What factors about each method must be considered when counseling about contraception?</p> <p>What are the five main classes of contraception?</p>	<p>Between 89% and 93%; yet 53% of births are either unintended or mistimed</p> <p>Failure of the method; Incorrect use; Nonadherence</p> <ol style="list-style-type: none"> Theoretical effectiveness (perfect use)—the pregnancy rate among women who use the method correctly every time Actual effectiveness (typical use)—includes the chances of inconsistent or incorrect use <p>Coital dependence, convenience, cost, duration of action, protection against sexually transmitted infections (STIs), effect on menses, religion, reversibility, acceptability, and side effects</p> <ol style="list-style-type: none"> Rhythm method Barrier methods
---	--

3. Hormonal contraceptives
4. Intrauterine devices (IUDs)
5. Sterilization

Rank the following contraceptives from the highest to the lowest with respect to actual effectiveness. What is the failure rate associated with each?

Condom, diaphragm/cervical cap, injectable/implantable hormonal contraception, oral contraception, periodic abstinence (fertility awareness method), spermicide, sterilization, withdrawal

IUDs (0.1–2%)
 Sterilization (0.4–4%)
 Injectable hormonal contraception (<1%)
 Oral contraceptives (5%)
 Male condom (14%)
 Diaphragm or cervical cap (20%)
 Female condom (21%)
 Periodic abstinence (25%)
 Withdrawal (19%)
 Spermicide (26%)

What is the fertility awareness method?

Predicting the time of month when a woman is **most fertile** and **abstaining** during that time. Also known as the rhythm method

What three methods can be used to predict fertility and which days are women presumed to be fertile?

1. **Date of the last menstrual period:** days 8–19 of the cycle (peri-ovulation)
2. **Changes in body temperature:** after menstruation until 3 days after an increase in basal body temperature by 0.5–1°F
3. **Changes in cervical mucus:** when the mucus becomes clear and stretchy (peri-ovulation) known as spinbarkeit

Why are fertility awareness methods unreliable?

Because people **do not always abstain** during this time and because there is always **some chance of fertility** on the “non-fertile” days. Body temperature and cervical mucus (especially if used together) are more effective than the calendar method because of irregular cycle lengths

What is the withdrawal method?

The withdrawal method is when the male **withdraws** from the vagina **before ejaculation**. It is ineffective when not timed correctly or when the pre-ejaculatory fluid contains sperm

Is lactation an effective means of birth control?

Lactation is somewhat effective as a means of birth control because of the **prolactin-induced inhibition of GnRH**, which leads to a delay in return to ovulation. Additional contraception should be used by breast-feeding women to prevent pregnancy

What factors affect the effectiveness of lactation as a means of birth control?

The number of times a woman breast feeds each day; How effective breast feeding is (i.e., how much milk she is producing); If the child is getting any supplemental feeding

STERILIZATION METHODS

What methods are available for surgical sterilization?

Male: vasectomy—ligation of vas deferens preventing passage of sperm into seminal fluid

Female:

1. **Ligation/removal** of a section of the fallopian tube—involves laparotomy or laparoscopy
2. **Mechanical blockage**—using rings, coils, clips, or plugs
3. **Coagulation-induced blockage**—usually through cauterization methods

What are the overall risks and benefits of female sterilization?

Risks:

1. Anesthesia/surgical complications
2. Ectopic pregnancy—failed procedures can result in an increased risk of ectopic pregnancies
3. Regret of the procedure (especially in younger people)

4. Does not stop the spread of HIV or other STIs

- Benefits:
1. Not coitally dependent
 2. Decreased risk of ovarian cancer
 3. No evidence of menstrual irregularity or dysmenorrhea

What are the risks and benefits of each of the female sterilization procedures?

Ligation is one of the **oldest methods** of sterilization with the **lowest failure rate (0.8%)** but it is not easily reversed

Mechanical blockage with a clip is the **most readily reversed method** but it also has the **highest failure rate (3.7%)**

Coagulation-induced blockage with electrocautery is the **fastest procedure** with a **low failure rate (2.5%)**, but there is increased risk of electrical damage to surrounding structures

What are some advantages and disadvantages of a vasectomy?

Advantages: effectiveness is very high—typical first-year failure rate 0.15%; simpler, surgically safer, more cost-effective than female sterilization; Males share contraception responsibility with females

Disadvantages: does not protect against STIs

Is reversibility after a female sterilization procedure and vasectomy possible?

Reversibility after a female sterilization procedure is generally very difficult and has been reported as only **60%** effective.

As for vasectomy, men are generally counseled that it is permanent. About **50–70%** of men who have a reversal become fertile. The chance of becoming fertile decreases with increasing time after the procedure

If a woman in her early twenties with two children requests tubal sterilization, what is the next appropriate recommendation?

Considering the woman's age, you must inform her of the risk for regret of the procedure and of the permanence of tubal sterilization/difficulty of reversal

Hormonal Contraceptives

What types of hormonal contraceptives are available?

Oral contraceptives; Injectable contraceptives; Contraceptive patch; Implants; Vaginal ring

What is the general mechanism of action of hormonal methods of contraception?

Hormonal contraceptive methods act primarily by **inhibiting** the midcycle surge of **gonadotropin secretion** and thereby **inhibiting ovulation**. They also **alter the endometrium and cervical mucus to decrease sperm transport and implantation**, and **decrease tubal motility**

Do hormonal contraceptives protect against STIs?

No. Condoms must be used to prevent transmission of STIs

Oral Contraceptive Pills

What are the types of oral contraceptive pills (OCPs) available and which is the most effective?

Combined estrogen-progesterone pills are the most effective (2–3% actual failure rate). **Progestin-only pills** are indicated for women who have contraindications to estrogen-containing pills

How are OCPs dosed?

Cyclic—OCPs are taken for 21 consecutive days, followed by 7 days of placebo

Extended cycle—OCPs are taken for 84 consecutive days, followed by 7 days of placebo

When should OCPs be started?

On the **first day of menstruation**

Sunday starter schedule: take the pill on the Sunday after the beginning of menses. This method requires **another nonhormonal form of contraception** for the first **7 days of OCP use**

Quick-start regimen: begin taking the pill ASAP, even in the doctor's office/clinic; increases adherence, decreases failures, side effects the same

What type of OCP should be started first?

Low-estrogen OCPs (20–35 µg of ethinyl estradiol [EE]) or very low estrogen OCPs (20 µg of EE) should be started in the average patient

In addition to contraception, what other advantages are conferred by taking OCPs?

Decreases menorrhagia, dysmenorrhea, and ovulation pain

(mittelschmerz); regulates menses; Lessens anemia; Improves acne (with estrogen-containing pills); protects against benign breast disease, formation of new corpus luteum cysts, osteoporosis, hirsutism; may reduce fibroids (controversial); decreases risk for ovarian cancer, endometrial cancer, pelvic inflammatory disease (PID), colorectal cancer, ectopic pregnancy

What are the risks of taking OCPs?

Increased risk of **thromboembolic disease/stroke; hepatocellular adenoma**; adenocarcinoma of the cervix; myocardial infarction (MI) (especially in high estrogen formulations); subarachnoid hemorrhage; hypertension; insulin dependence in diabetics

Is there a risk of developing breast cancer when using OCPs?

Experts agree there is most likely **no** risk between birth control pills and breast cancer

What are the absolute contraindications to the use of estrogen-progesterone OCPs?

History of thromboembolic disease/thrombophlebitis; smokers >age 35; cerebrovascular disease/coronary artery disease; congenital hyperlipidemia; history of an estrogen-dependent tumor; liver disease; pregnancy

What are the relative contraindications to the use of estrogen-progesterone OCPs?

Diabetes mellitus (may need to change insulin dose)

Epilepsy (can use the higher dose E2 pills if their medicines stimulate the P₄₅₀ system; if their medicines do not, there is no contraindication)

Gallbladder disease (although benign disease is not a contraindication)

Hypertension (requires close monitoring)

Use of some antibiotics

Postpartum state (women may be susceptible to clots following delivery)

Migraines/vascular headaches with aura

Obesity

What are the possible side effects of OCPs?	Breakthrough bleeding (especially first 3 months) Amenorrhea Depression/mood changes Nausea Bloating Breast tenderness Headaches Acne (with some progestin-only pills)
Do OCPs cause weight gain?	OCPs have not been proven to cause weight gain
How do OCPs affect acne?	Progestins worsen acne by increasing sebum production whereas estrogen relieves acne . Combined OCPs are used to treat acne. Use of a less androgenic progesterone-only pill will lower the risk of acne
What mediates the increased risk of thromboembolic disease with OCPs?	Estrogen . It increases the levels of clotting factors VII and X and decreases levels of antithrombin III
What is a hepatocellular adenoma?	A rare benign liver tumor associated with long-term OCP use, diagnosed by a right upper quadrant pain or a palpable mass on physical examination. Rupture of the adenoma can lead to hemodynamic instability
How is hepatocellular adenoma treated?	Discontinuation of OCPs is the treatment for hepatocellular adenoma . Pregnancy should be avoided in patients with this diagnosis unless the tumor is surgically removed
What is breakthrough bleeding?	Intermenstrual bleeding that occurs because of an imbalance of estrogen and progesterone . Occurs in 10–30% of women on low-dose OCPs. Early occurrences usually resolve spontaneously , but if bleeding does not resolve within 4 months, the patient can be switched to a pill with increased estrogen or a lower dose

- What is post-pill amenorrhea?**
- progesterone.** Pathologic causes of abnormal bleeding should also be considered.
- How is post-pill amenorrhea managed?**
- The **failure to menstruate by 6 months after cessation of OCPs.** Most cases **resolve spontaneously**
- An endocrine workup is necessary after 6 months to rule out pregnancy and functional causes of amenorrhea (anorexia, excessive exercise). It should also include a **progesterin challenge test** to rule out hypothalamic-pituitary dysfunction
- What is a progesterin challenge test?**
- 100 mg of progesterone is administered. Withdrawal bleeding should occur within a few days if ovulation is occurring
- What drugs interfere with OCP metabolism?**
- OCPs are metabolized by the **cytochrome P₄₅₀.** Drugs including **anticonvulsants** (phenytoin), **antibiotics** (isoniazid, rifampin, penicillin, tetracycline), and some **anti-retrovirals** decrease their effectiveness by increasing their metabolism
- What should be done if the patient must take these drugs: for a short duration, for a long duration?**
- Back-up contraception** should be used if the drugs are used for a short duration; however, one must seek an **alternative contraceptive modality if the drugs are used for long duration**
- What should a patient do if she forgot to take pills in following cases?**
- One pill:** take the missed dose as soon as possible and take the following dose at her regular time
- Two pills:** take two pills immediately and then continue the following day
- Three or more pills:** stop taking the pills in order to have menses and then restart the cycle
- Note:** In all of the above cases, an **additional contraceptive measure for the rest of that cycle is required.** A **pregnancy test is recommended if menstruation does not occur**

Injectable Contraceptives

<p>What injectable contraceptive is available in the United States?</p>	<p>Depot medroxyprogesterone acetate (DMPA or Depo-Provera)—a progestin-only injectable</p>
<p>What is its typical use failure rate in the first year?</p>	<p>Less than 1%—similar (if not lower) to that of a tubal ligation</p>
<p>How is DMPA administered?</p>	<p>An intramuscular (IM) injection of 150 mg every 3 months</p>
<p>When should DMPA be started?</p>	<p>DMPA should be begun within 5 days of the onset of menses. If received after this time window, another nonhormonal method must be used for the first 7 days</p>
<p>What are the contraindications to the use of DMPA?</p>	<p>Undiagnosed abnormal uterine bleeding; cerebrovascular disease/ coronary artery disease; liver disease; pregnancy; thromboembolic disease/ thrombophlebitis (relative contraindication)</p>
<p>In addition to contraception, what other advantages are conferred by DMPA?</p>	<p>Decreased risk of endometrial cancer and pelvic inflammatory disease (PID); decreased volume of menstrual bleeding; decreased pain associated with endometriosis; can be used while breast-feeding and in cases where estrogen is contraindicated</p>
<p>What are the potential side effects of taking DMPA?</p>	<p>Amenorrhea (eventually occurs in most women within 1 year of consistent use); irregular bleeding (spotting); decreased bone density (fully reversed once DMPA is stopped); acne; headache; depression</p> <p>There is no evidence to date that DMPA leads to weight gain</p>
<p>How long does it take to restore fertility after DMPA is stopped?</p>	<p>Most women return to fertility in 9–12 months after their last injection; however fertility can be delayed for up to 18 months after cessation</p>

Hormonal Implants, Patches, and Rings

<p>What are contraceptive implants?</p>	<p>Small rods placed surgically in the inner part of the upper arm subdermally. They release progestin in a controlled manner and can be left in place for 3–5 years, depending on the implant</p>
<p>What is the typical use failure rate in the first year?</p>	<p>0.05%</p>
<p>What are the absolute contraindications to hormonal implants?</p>	<p>Pregnancy, undiagnosed vaginal bleeding, liver disease, breast cancer, thrombophlebitis/embolism</p>
<p>What are the main side effects of contraceptive implants?</p>	<p>Irregular bleeding (common), amenorrhea, headache, acne, weight gain</p>
<p>What is the contraceptive patch ?</p>	<p>A transdermal patch that delivers estrogen and progestin in a controlled manner daily</p>
<p>How is the contraceptive patch administered?</p>	<p>It is placed on the buttock, abdomen, upper arm, or upper torso (but not breast). It is changed once per week for 3 weeks, followed by a patch-free week</p> <p>Note: If detached for more than 24 hours, it must be replaced and an additional method of contraception should be used for 7 days</p>
<p>What is the overall failure rate for the contraceptive patch?</p>	<p>0.3–1%</p>
<p>What is the main advantage of the patch over OCPs?</p>	<p>Better compliance by avoiding daily pill taking</p>
<p>What are the side effects of the contraceptive patch?</p>	<p>Skin reactions at site of placement; breakthrough bleeding; breast discomfort; dysmenorrhea; headache; abdominal pain; nausea</p>
<p>Is there an increased risk of blood clots with the contraceptive patch?</p>	<p>Theoretically, yes. The patch delivers a higher dose of estrogen (area under the curve) than typical OCPs and so it may increase the risk for blood clot development. However, no studies to date have documented this effect.</p>
<p>What is the contraceptive vaginal ring?</p>	<p>An intravaginal ring that releases estrogen and progestin daily</p>
<p>How is the vaginal ring administered?</p>	<p>It is inserted within 5 days of the onset of menses and requires an</p>

additional form of contraception for the first 7 days. The ring is taken out after **3 weeks** to allow 1 week of withdrawal bleeding

Note: If the ring is displaced for more than 3 hours, an additional contraceptive method must be used for 7 days

What are the potential side effects of the vaginal ring?

Device-related discomfort; irregular bleeding; headache; vaginal discharge/irritation; nausea; breast tenderness; mood changes

Emergency Contraception (“Morning-After Pill”)

What is emergency contraception (EC)?

A postcoital method of contraception

How does EC work?

EC works by preventing ovulation and fertilization. **EC will not terminate a pregnancy** if implantation has already occurred

Which methods are recommended in the United States?

High-dose progestin-only contraceptive pills (**Plan B**)
High-dose estrogen and progesterone pills (**Yuzpe method**)

Plan B is routinely recommended over the Yuzpe method

How is EC administered?

It can be taken up to 3 days after unprotected intercourse, but is more effective if taken sooner. It is now recommended that both doses of Plan B be taken together ASAP!

How effective are the Yuzpe method and Plan B at preventing pregnancy?

The Yuzpe method **reduces the risk of pregnancy by approximately 75%** whereas Plan B **reduces the risk of pregnancy by up to 89%**. Both methods are more effective the closer they are taken to the unprotected act of coitus

How effective is Plan B at preventing pregnancy if started:

Within 24 hours of unprotected intercourse?

95% effective

Within 72 hours of unprotected intercourse?

58% effective

Who should be prescribed EC?	Every sexually active woman at risk of pregnancy should be prescribed EC as a backup in the event of primary contraceptive failure
What are the major side effects of EC?	Nausea and vomiting. Both are seen more commonly with the Yuzpe method. Antiemetics may be used prophylactically prior to EC use
Can EC prevent against future pregnancies?	No. Barrier or hormonal contraceptives should be used to prevent future pregnancy
Is EC teratogenic to the fetus if pregnancy occurs?	No. There is no evidence that emergency contraceptive pills (ECPs) increase the risk of fetal anomalies or miscarriages
Are there any nonhormonal methods of emergency contraception?	Yes—an IUD can also be used. It is 92–98% effective in preventing pregnancy if placed within 120 hours of unprotected intercourse

BARRIER METHODS OF CONTRACEPTION

What are the types of barrier methods available?	Male condom; female condom; diaphragm; cervical cap; Lea's shield; contraceptive sponge; spermicide
What are the advantages of barrier contraception over other methods?	Many prevent STI transmission and they have no hormonal side effects
What are the disadvantages of barrier contraception over other methods?	Coital dependence (less reliable); Risk of breakage
Which barrier method is the most effective at preventing STI transmission?	Male condoms are the most effective . The diaphragm, cervical cap, and sponge have more limited protection against STIs because they do not cover the entire genital tract
What are the two types of male condoms and what are the disadvantages of each?	<ol style="list-style-type: none"> 1. Latex—the most common type of condom; Disadvantages: risk of latex sensitivity, breaks with oil-based lubricants 2. Synthetic—such as polyurethane; Disadvantages: less effective, more likely to break
	Male condoms have a typical use failure rate of 15% in the first year

Do spermicidal condoms offer more protection compared to non-spermicidal condoms?

No. They have no added protection against STIs or pregnancy. Many manufacturers have stopped producing spermicidal condoms

What is the female condom and how it is used?

The female condom is a **lubricated polyurethane sheath** with a flexible ring on each end. One ring covers the cervix and the other remains external, partly covering the labia. It should be **left in place for 6–8 hours after intercourse**

*Female condoms have a typical use failure rate of 21% in the first year

What is a diaphragm, what is a cervical cap, and how are they used?

Cup-shaped rubber barriers that, when **used with spermicide**, are effective contraceptive modalities

How are the diaphragm and cervical cap used?

Both require initial fitting by a clinician and are inserted to cover the cervix. They should be inserted no more than 6 hours before coitus. Both must be **left in the vagina 6–8 hours after intercourse**. The diaphragm can be left in place for up to 24 hours and the cervical cap can be left in place for up to 48 hours

What are the side effects of the diaphragm and cervical cap?

Increased risk of UTI (with diaphragm)

Possible increased risk of **toxic shock syndrome**

Increased risk of **vaginal irritation/discharge**

For which women are diaphragms and caps not recommended and why?

Women who are at risk for HIV or who are infected with HIV. The nanoxynol-9 placed on the device may induce vaginal irritation and destroy normal vaginal flora thereby increasing the risk for HIV infection

What is Lea's shield?

A dome-shaped silicone bowl placed over the cervix. It has a one-way valve that allows egress of secretions but keeps sperm out. It does not protect against STDs

How is Lea's shield used?

It does not need to be fitted by a physician. It is inserted before sexual intercourse and must be taken out 8–48 hours after intercourse

What is the contraceptive sponge?	A circular polyurethane foam sponge containing nonoxynol-9 attached to a loop for removal
How is the contraceptive sponge used?	It is moistened and placed intravaginally up to 24 hours before coitus. The sponge can be left in place for up to 24 hours after intercourse
What are the side effects of the cervical sponge?	Increased risk of toxic shock syndrome
What type of spermicide is available over the counter in the United States?	Nonoxynol-9. It is available as a vaginal cream, film, foam, and gel
How is spermicide used?	It is placed intravaginally at least 15 minutes prior to intercourse and remains effective for up to 1 hour
What are the disadvantages of spermicide?	Relative high failure rate (29% typical use failure rate in first year) It is not an effective vaginal microbicide and does not protect against HIV, gonococcus, or chlamydia transmission

INTRAUTERINE DEVICES

What are IUDs and how are they administered?	T-shaped devices placed by a clinician through the cervix and into the uterus . They have a small string that hangs down from the external cervical os into the vagina for removal (see Fig. 4-2). They can be left in place for 1–10 years (depending on specific device)
What are the two types of IUDs available and how do they work?	Copper IUDs (<i>Paragard</i>)—prevent sperm from reaching the fallopian tubes by inducing a sterile inflammatory reaction in the endometrium . They can be left in place for up to 10 years Levonorgestrel-releasing IUDs (<i>Mirena</i>)— prevent sperm from reaching the ovum by causing the cervical mucus to become thicker. Also can cause anovulation after 1 year as well as decreased menstrual flow and amenorrhea. They can be left in place for up to 5 years

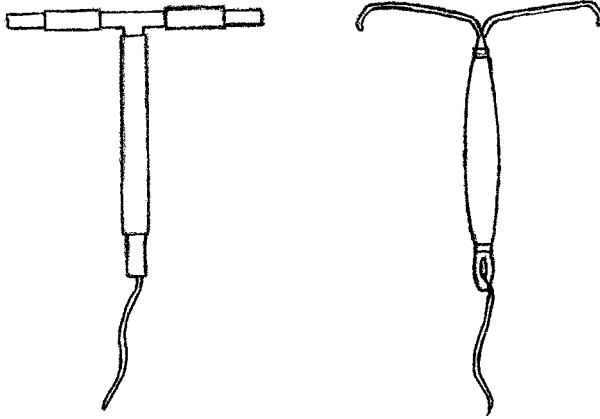


Figure 4-2 Examples of commonly used intrauterine devices.

What are the benefits of IUDs?

Very effective (typical use failure rate in first year <0.1%); Decreased risk for ectopic pregnancy (compared to non-contraception users); possible protective effect against endometrial and cervical cancer; highly cost-effective over a 5-year period; immediate return to baseline fertility after removal; menorrhagia and dysmenorrhea improvement (with Mirena*). Mirena may help reduce the risk of pelvic inflammatory disease (PID) because of its cervical mucus effects

***It has the highest level of user satisfaction of any contraception being used by women**

What are the risks of IUD placement?

Uterine perforation; infection (risk of PID highest in first 20 days after placement); expulsion (patients should be encouraged to look/feel for string monthly); potential complications if pregnancy does occur

What are the side effects of IUD use?

Menorrhagia and dysmenorrhea (with Paraguard); can be relieved with NSAIDs; amenorrhea (with Mirena); no protection from STIs or HIV

What are the contraindications to IUD placement?

Absolute contraindications: pregnancy, endometrial cavity distortion, undiagnosed uterine bleeding, active, recent, or recurrent pelvic infection, copper allergy/Wilson disease (for Paraguard only), pelvic malignancy

Relative contraindications: multiple sexual partners, dysmenorrhea/menorrhagia (for Paraguard only)

What is the relationship between IUD use and PID?

PID development risk is greatest in the **first 20 days** after IUD insertion and rare thereafter. If a woman develops a gonococcal or chlamydial infection, she is **much more likely to develop PID** if she has an IUD in place. IUD use is related to the development of PID with **actinomycosis** infection

Should the IUD be removed if a woman is found to have a positive gonorrheal or chlamydial culture?

Yes, and the patient must be treated promptly

What are the four signs of IUD expulsion and what should be done?

1. Cramping
2. Vaginal discharge
3. Irregular spotting
4. Dyspareunia

However, expulsion may be asymptomatic and so it is important to check monthly for **elongation or absence of the vaginal string** to ensure continued placement

What should be done if expulsion occurs?

The patient should immediately begin to **use another form of contraception** until she sees her physician

ELECTIVE ABORTION

How common is elective abortion?

Very common; in the United States 50% of all pregnancies are unintended and half of these are terminated

Is there an impact of elective abortion on future pregnancies?

No—there is no evidence to prove that a single termination has any

	<p>impact on future fertility or the risk of spontaneous abortion. However there is an increased risk of Asherman syndrome (intrauterine synechiae) if the dilation and curettage (D&C) is done in the presence of infection or if multiple procedures are done</p>
<p>What examinations/tests need to be done before an elective termination of pregnancy?</p>	<p>Pelvic examination (to assess uterine size and position); β-hCG or ultrasound (to confirm pregnancy); ultrasound (to assess dates and confirm intrauterine pregnancy); hematocrit and Rh(D) status.</p> <p>Some providers screen and treat for STIs prior to the procedure</p>
<p>What are the surgical procedure options for evacuation of products of conception (POC)?</p>	<p>Manual vacuum aspiration (MVA); suction dilation and curettage (suction D&C); dilation and evacuation (D&E); dilation and extraction (D&X)</p>
<p>What are the risks of these procedures?</p>	<ul style="list-style-type: none"> • Risks of anesthesia • Infection • Hemorrhage • Embolus (pulmonary or other) • Cervical laceration • Uterine perforation with/without pelvic • Organ damage • Potential for adhesion development
<p>Does an abortion increase the risk of breast cancer?</p>	<p>No</p>
<p>What are the mortality rates associated with elective terminations?</p>	<p>In the United States, mortality rates for first trimester abortions are 0.1–0.4 per 100,000 and 1.7–8.9 per 100,000 in the second trimester. However, internationally, where many abortions are done illegally, one in eight maternal deaths is because of abortion-related complications</p>

What should be done if uterine perforation is suspected?	Immediate laparoscopy or laparotomy to examine the abdominal contents to assess bowel injury
How and when is an MVA performed?	Anytime before 12 weeks. The cervix is dilated using cervical dilators and the uterine contents are evacuated using a cannula that is attached to a handheld syringe which has the vacuum source
How is a suction D&C performed?	The cervix is dilated using cervical dilators and the uterine contents are evacuated using an electrically powered vacuum device
When can a D&C be performed?	Anytime before 14 weeks
How is a D&E performed?	The cervix is dilated using osmotic dilators placed intracervically 1–2 days prior to the procedure to slowly soften and dilate the cervix and thus minimize mechanical damage to the cervix. At the time of the procedure, these are removed and mechanical dilators are used for further dilation as needed. The uterine contents are evacuated using specialized forceps (sophers and bierers). Many people confirm completion by performing a curettage and feeling a good “cri” (gritty texture) throughout the cavity. Vacuum can also be used to remove any remaining blood or tissue
When can a D&E be performed?	Anytime before 24 weeks, depending on the legal limit in the state
How is a D&X performed?	It is similar to a D&E except that the fetus is delivered in breech presentation through the dilated cervix and the cranial contents are suctioned before delivery of the fetal head
When can a D&X be performed?	From approximately 18–24 weeks
What is the advantage of a D&X procedure over a D&E?	It potentially minimizes uterine and cervical injury from the fetal bones and from instrumentation
Are prophylactic antibiotics indicated to prevent infection?	Yes. Women given antibiotics peri-abortion have a lower risk of postoperative infection

What types of antibiotics can be used prophylactically?	Doxycycline, ofloxacin, or ceftriaxone
What is a medical abortion?	Termination of pregnancy or evacuation of POCs using medications only
How effective is medical abortion?	Very effective—it is successful in 90–98% of women
What are the various modalities for medical abortion?	Intravaginal misoprostol alone Oral or IM methotrexate followed by intravaginal, oral, or buccal misoprostol 3–7 days later Oral mifepristone followed by intravaginal, oral, or buccal misoprostol 6–72 hours later (up to 49–63 days)
How do each of the following agents work?	Misoprostol: a prostaglandin analogue; increases contractility by directly stimulating the myometrium Methotrexate: blocks dihydrofolate reductase, an enzyme necessary for the production of thymidine during DNA synthesis, thus affecting the rapidly growing cytotrophoblast Mifepristone: binds to the progesterone receptor with a greater affinity than progesterone itself, and therefore blocks the “pro-gestation” action of progesterone
What are the side effects of a medical abortion?	Cramping, bleeding (often heavier than a menstrual period) Gastrointestinal (GI) distress (nausea, vomiting, diarrhea)
What are the contraindications to a medical abortion?	Allergies to any of the medications An in situ IUD Severe anemia Coagulopathy/use of an anticoagulant Active liver disease Cardiovascular disease Uncontrolled seizure disorder Adrenal disease
Is there any effect of medical termination on subsequent pregnancies?	No

What is a septic abortion?	Evidence of localized infection and systemic infection after a spontaneous or elective abortion
How is a septic abortion managed?	<p>Stabilize the patient</p> <p>Take blood and endometrial cultures</p> <p>Administer parenteral broad-spectrum antibiotics</p> <p>Surgically evacuate the uterine contents</p> <p>Administer anti-D immunoglobulin if it is warranted</p>
What antibiotic combinations can be used to treat septic abortion?	<p>Clindamycin and gentamicin with or without ampicillin</p> <p>Ampicillin and gentamicin and metronidazole</p> <p>Ticarcillin-clavulanate or piperacillin-tazobactam or imipenem alone</p>
What are the indications for laparotomy in the management of a septic abortion?	<p>Failure to respond to evacuation/antibiotics</p> <p>Pelvic abscess</p> <p>Gas gangrene or other fulminant disease (i.e., <i>Clostridium sordelii</i>)</p>
Who should be given Rh(D)-immune globulin postabortion?	All Rh(D)-negative women who are unsensitized
When does ovulation resume postabortion?	As early as 2 weeks after an abortion. Therefore if pregnancy is undesired, contraception must be started immediately
When do menses resume postabortion?	Usually within 6 weeks (average 4 weeks)
What conditions need to be considered if menses do not resume within 6 weeks?	Pregnancy and gestational trophoblastic disease; less likely are Asherman syndrome or other systemic diseases (e.g., thyroid disorder)
What instructions should be given postabortion?	Women should be advised to have nothing per vagina for 2 weeks after the procedure/passage of POC. Some recommend deferment of pregnancy (if desired) for 2–3 months, although there is no data to support this recommendation
When should a woman return to clinic postabortion?	If she experiences heavy bleeding, fever, or abdominal pain

Sexually Transmitted Diseases and Pelvic Infections

SEXUALLY TRANSMITTED INFECTIONS OF THE LOWER GENITAL TRACT

Condyloma Accuminata

What is condyloma accuminata?

Genital warts. It is the most common sexually transmitted infection

How is condyloma accuminata transmitted?

Through skin-to-skin contact; it is primarily transmitted through sexual activity

What is the pathogen and which subtypes are associated more commonly with genital warts?

A DNA virus called human papillomavirus (HPV); subtypes **6 and 11**

What are the clinical manifestations of condyloma accuminata?

Warts located on the external genitalia, perineum, anus, cervix, mouth, inside the vagina, and urethra; These are generally raised, pedunculated/**cauliflower-shaped** lesions but they can vary in number, size, and color (flesh-colored, pinkish-white, grayish-white)

What other disease should be excluded?

Condyloma lata of secondary syphilis

How is condyloma accuminata diagnosed?

Visualization by colposcopic examination and cytologic smear. Biopsy can be performed if the diagnosis is uncertain

What would a biopsy of the specimen reveal?

Koilocytosis (vacuolated keratinocyte with peri-nuclear halo). It is often also associated with atypia and dysplasia

What is the treatment?

Cryotherapy, laser excision, trichloroacetic acid, podophyllum, or imiquimod cream. If left untreated, visible lesions may resolve on their own, remain unchanged, or increase in size or number

	The virus cannot be eradicated once present in the genital tract; therefore, warts can recur
What are Buschke-Lowenstein tumors?	Giant condylomas caused by HPV that are found in immunocompromised patients
What else can HPV cause?	Preinvasive and invasive cervical cancer. Dysplasia is more commonly caused by subtypes 16 and 18
How can HPV infection be prevented?	A quadrivalent recombinant vaccine against HPV serotypes 6, 11, 16, 18 (Gardasil) can be given to females between the ages of 9 and 26

Molluscum Contagiosum

What is genital molluscum contagiosum?	A benign, usually asymptomatic, infection of the vulva caused by poxvirus
How is genital molluscum transmitted?	Through sexual contact, casual skin-skin contact, fomites (substances that absorb and transport infectious disease particles, i.e., underwear), or auto-inoculation
What is the typical clinical presentation?	Single or multiple (<30) small, white, pink, or flesh-colored dome-shaped papules with central umbilication in the genital/inguinal area. It is usually asymptomatic but eczema may surround the lesion and pruritus may occur
How does genital molluscum contagiosum present in an immunocompromised person?	Large lesions (>1 cm) or a clustering of numerous small lesions; intra-oral or peri-oral lesions can arise
What are the differential diagnoses?	Multiple small lesions: condyloma acuminata Large, solitary lesions: basal or squamous cell carcinoma
How is the diagnosis made?	Usually based on clinical manifestations. Biopsy can be performed for definitive diagnosis and will reveal intracytoplasmic inclusions
What are the treatment options?	It is usually self-limiting; cosmetic options include curettage or

cryotherapy of lesions with liquid nitrogen; topical imiquimod is also used

Sexual partners do not need to be treated

Pediculosis Pubis (Crabs) and Scabies

What are pediculosis pubis and scabies?	Parasitic infections of the pubic area
What causes pediculosis pubis?	The crab louse <i>Phthirus pubis</i> —a blood-sucking parasite that lives on hair shafts and lays eggs (nits)
What causes scabies?	The itch mite <i>Sarcoptes scabiei</i> —a parasite that burrows underneath the skin and lays eggs
How are pediculosis pubis and scabies transmitted?	Primarily through sexual contact. Can also be through fomites (clothing, sheets)
What is the typical presentation of these infestations?	Intense pruritus over the genital area, especially at night. Polymorphic papules may be seen in scabies.
How are pediculosis pubis and scabies diagnosed?	Visualization of the parasites or eggs (nits) under a microscope (skin scrapings) or magnifying glass
What is the treatment?	Permethrin 1% cream (first line) Lindane 1% shampoo (second line) (contraindicated in pregnant/lactating women) All clothing and sheets should be washed in hot water and isolated for at least 3 days after treatment. All contacts must be treated
What other tests should be ordered?	Screening tests for other sexually transmitted diseases (HIV, syphilis, gonorrhea, <i>chlamydia</i>)

SEXUALLY TRANSMITTED INFECTIONS CAUSING GENITAL ULCERATIONS

What are the most common infectious causes of genital ulcers in the United States?	Herpes simplex virus (HSV) > syphilis > chancroid >>> lymphogranuloma venereum and granuloma inguinale
--	--

Is coinfection with multiple organisms common?

Yes. Making a diagnosis based on the history and physical can be very difficult and requires a thorough workup and knowledge of the most likely causal organism in the specific patient population

What are some of the most common noninfectious causes of genital ulcers?

Behcet disease, drug reactions, trauma, neoplasma

What are the tests used to diagnose a genital ulcer?

Darkfield microscopy (syphilis)
Serologic tests (syphilis and lymphogranuloma venereum [LGV])
Gram stain and **viral culture** on selective media (*Haemophilus ducreyi*)
Tzanck preparation, direct fluorescence antibody (DFA), viral culture, or polymerase chain reaction (PCR) for HSV
Tissue biopsy (syphilis, granuloma inguinale)

With what other disease are these genital ulcer diseases associated?

HIV

Which of these diseases must be reported?

Syphilis (and HIV)

Genital Herpes

What is genital HSV?

A DNA virus **transmitted through infectious secretions** that causes recurrent, **lifelong disease**

What is the difference between HSV-1 and HSV-2?

Both are implicated in genital herpes and have the same clinical presentation. However, **HSV-2** more commonly infects the **genitalia** whereas **HSV-1** more commonly affects the **oral mucosa**

What is the natural history of the HSV infection?

Primary episode: mucocutaneous infection occurs via direct sexual contact with an infected person. After a 4-day incubation period, ulcerating pustular lesions erupt, often associated with local pain, lymphadenopathy, and systemic symptoms

Viral latency: after primary infection, there is a period of **viral latency** as the virus ascends along the sensory

	nerve roots and becomes latent in the dorsal root ganglion
	Reactivation: can occur at any interval when the virus travels back down the sensory nerve. This can either cause a mucocutaneous outbreak (recurrence) or sometimes no symptoms may be detected
What is the strongest risk factor for genital HSV infection?	A large number of lifetime sex partners
What clinical presentation is suggestive of primary genital herpes?	A prodrome of 2–24 hours characterized by localized/regional burning and pain Systemic symptoms including fever, malaise, and bilateral inguinal lymphadenopathy Grouped vesicles, uniform in size, mixed with multiple, shallow, severely tender ulcers around the vulva, perineum, and perianal area are pathognomonic Cervical lesions are also common and cause intermittent bleeding and vaginal discharge Dysuria and urinary retention syndromes may occur because of contact with urethral and vulvar ulcers
Are all primary outbreaks clinically symptomatic?	No. 70–80% of infected persons have unrecognized symptoms or completely asymptomatic infections; therefore, transmission of this virus can be high and unrecognized
What laboratory tests help make a diagnosis of HSV?	Type-specific viral cultures (highest sensitivity and specificity; highest yield if done early when you can attain more vesicle fluid) Direct fluorescence antibody test Type-specific serologic testing (best for those with a questionable hx, subclinical infection, or suspicion of a false-negative viral culture) Tzanck smears Polymerase chain reaction (PCR)
What is the presentation of a secondary outbreak?	They vary widely in their frequency, are milder and shorter in duration ,

What are some precipitating factors of a recurrent outbreak?

and may not have prodromal symptoms of pain, burning, and itching. Viral shedding can occur for weeks after the appearance of lesions.

What are the treatment options for genital herpes?

Immunodeficiency, trauma, fever, nerve damage, concurrent infection, and sexual intercourse

Oral acyclovir for primary and recurrent episodes. It reduces viral shedding and shortens the clinical course. Can also be used prophylactically for patients with frequently recurring episodes, decreasing the recurrence rates by 70–80%

Intravenous (IV) acyclovir should be considered for severe/disseminated disease or in immunocompromised patients

Other therapies include keeping the affected area clean and dry, wearing loose clothing and undergarments, washing hands after contact with affected areas, and using an ice pack or sitz bath for soothing sores

All sexual partners should be evaluated for infection

Screening tests should be considered for what other diseases?

Syphilis and HIV

What is the most common cause of urogenital complaints in the emergency room setting?

Genital herpes

If a pregnant woman carries HSV, what is the risk of transmission to the neonate?

The risk of transmission by an infected mother is 30–50% among women with a primary outbreak during the third trimester, and <1% in those with recurrent infection or a primary infection during the first half of the pregnancy

What precautions should be considered to decrease neonatal transmission from an affected mother?

In the presence of a genital lesion or prodrome, delivery should be via cesarean delivery

Prophylactic antivirals may be administered in the third trimester to reduce the incidence of HSV recurrence

Syphilis

What organism causes syphilis?

Treponema pallidum

How is it transmitted?

Usually via sexual transmission. It enters the body by penetrating intact mucous membranes or by invading epithelial abrasions

Is it *always* sexually transmitted?

No. Skin contact between any skin abrasions and an ulcer infected with *T. pallidum* can result in infection

Which population is highest at risk?

Black heterosexual women and homosexual men in urban areas

What are the clinical manifestations for the three stages of syphilis?

Primary, secondary, and tertiary syphilis

Describe primary syphilis

Symptoms occur approximately 3 weeks after infection and include a **painless chancre** (a single, clean-based ulcer usually on labia/vaginal wall/cervix) and **painless lymphadenopathy**

Describe secondary syphilis

Systemic disease results from hematogenous dissemination that occurs 6–8 weeks after infection. Symptoms include a **maculopapular rash on the palms and soles**, **condyloma latum** (moist, grayish papules-like warts), malaise, fever, arthralgias, pharyngitis, and generalized lymphadenopathy

Describe tertiary syphilis

Occurs 3–10 years after initial infection. Symptoms include **gummas** (noninfectious granulomatous lesions found in skin and bones), cardiovascular syphilis (**aortitis** or an **aortic aneurysm**), and neurosyphilis (**general paresis**, **tabes dorsalis**, or an **Argyll-Robertson pupil**)

What is the latent period?

A period of anywhere from 2–20 years that occurs **between the second and third stages** of syphilis. Most patients are asymptomatic (some have recurrences) and are considered noninfectious, although their serologic tests remain positive

What is the gold standard for diagnosis of syphilis and when can it be used?

Dark field microscopy of a specimen from the primary chancre,

	condyloma latum, or the maculopapular rash reveals spirochetes . It is only useful during the active stages of primary and secondary syphilis
What other tests are available to help with diagnosis?	Nonspecific and specific serological screening tests: Nonspecific: venereal Disease Research Laboratory (VDRL) and rapid plasma reagin (RPR). A positive result must be confirmed with a specific treponemal antibody test given high rate of false positives! Specific: Fluorescent treponemal antibody absorption (FTA-ABS), microhemagglutination assay (MHA-TP), and treponemal hemagglutination tests for syphilis (HATTS)
How would you interpret the following laboratory results:	
Positive nonspecific test and positive specific test	Active treponemal infection
Positive nonspecific test and negative specific test	False positive
Negative nonspecific test and positive specific test	Successfully treated syphilis
Negative nonspecific test and negative specific test	Syphilis unlikely
What other common conditions may cause a false positive nonspecific test result?	Systemic lupus erythematosus (SLE), rheumatic heart disease, pregnancy, infectious mononucleosis, intravenous drug use, viral hepatitis, recent immunization
What is the treatment for syphilis?	Penicillin or tetracycline (for nonpregnant penicillin allergic patients) for primary, secondary, and early latent syphilis are first-line drugs. Erythromycin is second-line but is contraindicated in pregnant patients
What is Jarisch-Herxheimer phenomenon?	A self-limiting, acute worsening of symptoms after antibiotics are started . Symptoms include headache, fever, chills, muscle aches, and other flu-like symptoms

What diagnosis should be considered for unexplained rashes, arthralgias, neurologic or systemic complaints? Syphilis

Chancroid

What is chancroid?

An **acute, curable, sexually transmitted disease** caused by *H. ducreyi*. It is uncommon in the United States but is a predominant cause of genital ulcer disease in sub-Saharan Africa

What clinical presentation highly suggests chancroid?

One to three extremely painful ulcers around the perilabial area that are deep, purulent, and have ragged edges. These are associated with **unilateral, suppurative, painful swollen inguinal lymph nodes** that, in 25% of cases, will rupture, releasing a heavy, foul discharge that is contagious (**suppurative adenopathy = bubo**). Systemic symptoms (fevers, myalgias) are typically **not** present

What other infections must be ruled out before a diagnosis of chancroid can be made?

Syphilis, HSV, lymphogranuloma venereum (LGV), and granuloma inguinale

What laboratory tests help make a diagnosis of chancroid?

Culture on selective media isolates *H. ducreyi*

Gram stain of a specimen from the ulcer base or bubo aspirate: reveals gram-negative rods in a chain; referred to as a “**school of fish**” pattern

PCR

What is the treatment for chancroid?

Azithromycin (oral) or **ceftriaxone** (IM)

Treat sexual partners

Lymphogranuloma Venereum

What is LGV?

A **sexually transmitted ulcerative disease** that occurs in **three stages** and involves **infection of the lymphatic tissue** in the genital region

What is the causal agent?

Chlamydia trachomatis; serotypes L1, L2, and L3 are most common

What are key risk factors one should be aware of in the history and physical (H&P)?

Travel and unprotected sex in tropical regions or regions where LGV is endemic (Africa, Southeast Asia, India); anal sex

What are the key physical findings at each stage of this disease?

Stage 1: **small, painless papules/shallow ulcerations** typically on the vaginal wall that often go unnoticed

Stage 2: **painful unilateral inguinal lymphadenopathy** (typically in men); women may complain of lower back/abdominal pain because of deep pelvic node involvement; **bubo** (matted nodes that adhere to the overlying skin); systemic symptoms; **groove sign** (enlargement of the nodes above and below the inguinal ligament)

Stage 3: **rupture of the bubo** leads to **genitoanorectal syndrome** (strictures and fistulas in the anogenital tract); constitutional symptoms; **proctocolitis**; abscesses

At what stage do most women present?

Stage 3

Which finding is pathognomic for LGV?

Groove sign (inguinal buboes with nonsignificant ulcers = LGV)

What other diseases may present with similar cutaneous lesions?

Granuloma inguinale, tuberculosis (TB), early syphilis, and chancroid

What are the most common laboratory tests used to diagnose LGV?

Complement fixation tests (used most often)

Serologic tests for IgG antibodies

Immunofluorescence on aspirates from bubo for the presence of inclusion bodies

PCR for *C. trachomatis* or DNA swab from lesion

Genital or lymph node specimen tested by culture

How is LGV treated?

Oral **doxycycline** or erythromycin for 3 weeks

Lymph node aspiration if needed

What are some complications of LGV?

Fistulas, strictures, tissue ischemia and necrosis; elephantiasis of the female genitalia (esthiomene)

Granuloma Inguinale (Donovanosis)

What is granuloma inguinale?	A slow, progressive genital ulcerative disease that is primarily sexually transmitted. It is most common in the developing world; it is rare in the United States
What is the cause?	<i>Klebsiella granulomatis</i> (previously known as <i>Calymmatobacterium granulomatis</i>), a gram-negative pleomorphic bacillus
What are the typical manifestations of granuloma inguinale?	Large, painless , and spreading ulcers typically in the vulva area; the lesions are clean but have friable bases with raised, rolled margins that bleed easily . They are typically beefy red in appearance and exude a malodorous discharge. Inguinal lymphadenopathy is rare
What is the classic finding for establishing a diagnosis of granuloma inguinale?	Donovan bodies (intracytoplasmic safety pin shaped organisms seen after Giemsa or Wright staining of tissue specimens)
What is the treatment?	Doxycycline or trimethoprim-sulfamethoxazole . (Tetracycline is no longer recommended because of bacterial resistance)

CERVICITIS

Chlamydia, Gonorrhea, and Other Causes of Cervicitis

What cell types make up the cervix and where are they located?	Columnar epithelium—endocervix Non-keratinizing squamous epithelium—ectocervix
What is mucopurulent cervicitis (MPC)?	Inflammation of the endocervix most commonly caused by sexually transmitted organisms. It is characterized by a yellow-greenish mucopurulent discharge on visual inspection or on an endocervical swab specimen.
What are the two most common infectious etiologies of MPC and what kind of organisms are they?	1. <i>C. trachomatis</i> : gram-negative, obligate intracellular bacterium

What is ectocervicitis?	2. <i>Neisseria gonorrhoeae</i> : gram-negative diplococci
What are some infectious causes of ectocervicitis and what are some key clues, if any, that lead you to that etiology?	Inflammation of the ectocervical epithelium. This squamous epithelium is an extension of the vaginal epithelium and can be infected by the same organisms that cause vaginal infections Trichomonas: strawberry cervix (small petechiae to large punctuate hemorrhages on the ectocervix) HSV: ulcerative and hemorrhagic lesions/vesicles during the primary infection HPV: genital warts, cervical dysplasia on Pap smear
What age group is most frequently affected with cervicitis?	15–25 year olds
What are the typical symptoms of MPC?	Vaginal discharge, dysuria, urinary frequency, dyspareunia, postcoital bleeding
What findings are found on clinical examination?	A tender, friable cervix that may also be erythematous and/or edematous
What laboratory test supports a diagnosis of MPC?	Patients infected with <i>C. trachomatis</i> or <i>N. gonorrhoeae</i> are frequently asymptomatic! >30 polymorphonuclear (PMN) leukocytes on a Gram-stained specimen from the endocervix
How is the diagnosis of each of the following organisms made?	<i>C. trachomatis</i> : cell culture (gold standard, but difficult), nucleic acid amplification tests (NAAT) from urine, DNA probe, enzyme immunoassay <i>N. gonorrhoea</i> : Thayer-Martin agar culture (gold-standard), DNA probe, enzyme immunoassay <i>T. vaginalis</i> : visualization of motile trichomonads on wet mount HSV: Tzanck smear test, serologic testing, viral culture HPV: clinical appearance, cytology

What is the treatment for each of these causes of cervicitis?

C. trachomatis: doxycycline × 7 days or azithromycin (single dose)

N. gonorrhoeae: ceftriaxone (IM), ciprofloxacin plus doxycycline, azithromycin (single dose)

T. vaginalis: metronidazole (PO)

HSV: acyclovir (PO)

For all of the above infections, sexual partners also must be treated

What is the treatment for *C. trachomatis* in a pregnant patient or a noncompliant patient?

Azithromycin (single dose PO)

What other organism should be treated in a patient diagnosed with *N. gonorrhoeae*?

C. trachomatis

What are some serious complications of cervicitis?

Pelvic inflammatory disease (PID), pregnancy and neonatal complications, cervical cancer, increased risk of HIV transmission

What are the guidelines for STI screening?

Annual screening of *C. trachomatis* and *N. gonorrhoeae* for all sexually active women <25 years of age, or those at high risk >25 (new sex partner or multiple sex partners)

What other necessary steps should be taken with a patient diagnosed with an STD-induced cervicitis?

Treat all partners and test for HIV, syphilis, hepatitis B and C

INFECTIONS OF THE UPPER GENITAL TRACT

Pelvic Inflammatory Disease

What is PID?

An **acute infection** that may involve parts or all of the female genital tract, including the **cervix, endometrial cavity (endometritis), fallopian tubes (salpingitis), ovaries (oophoritis), parametrial tissues/ligaments (parametritis), and/or peritoneal cavity (peritonitis)**. It is *typically* initiated by **sexually transmitted agents**

What are the usual presentation/symptoms/signs of this disease?

The patient is typically a **13–35-year-old sexually active female**

who presents with **lower abdominal pain** (not more than of 2 weeks' duration), **adnexal tenderness** (usually bilateral), or **cervical motion tenderness** on physical examination (PE) (only one must be present). While not required to establish the diagnosis of PID, **the presence of one or more of the following enhances the specificity of the minimum criteria: fever (>101°F); purulent cervical discharge; elevated erythrocyte sedimentation rate (ESR)/C-reactive protein level; leukocytosis**

What are other immediate differential diagnoses of a patient who presents with lower abdominal pain?

Appendicitis, endometriosis, (ruptured) ectopic pregnancy, irritable bowel syndrome, inflammatory bowel disease, ruptured/hemorrhagic ovarian cyst, abortion, gastroenteritis, ovarian torsion, renal colic, tubo-ovarian abscess (TOA), UTI, somatization, mesenteric adenitis

For each of the following differential diagnosis related to Ob-Gyn, list the main symptoms/signs that would differentiate between that diagnosis and PID

Endometriosis: medical history of chronic pelvic pain, dysmenorrhea, deep dyspareunia, low sacral backache, dischezia, perimenstrual bleeding, cystic ovarian enlargement, tender adnexae, uterosacral tenderness and nodularity, retroflexed uterus

(Ruptured) ectopic pregnancy: history of amenorrhea, crampy abdominal pain, abnormal uterine bleeding, nausea/vomiting (N/V), dizziness/light-headedness, palpable tender adnexal mass; other signs depend on extent of rupture and hemorrhage (peritoneal signs, tachycardia, tachypnea, and orthostatic changes)

Ruptured/hemorrhagic ovarian cyst: sudden-onset bi/unilateral lower abdominal pain, rebound tenderness, guarding, N/V

Ovarian torsion: intense, progressive unilateral pain combined with tense,

<p>What are the risk factors for PID and what pathogens (if any) are associated with these risk factors?</p>	<p>tender, and enlarged ovarian mass. History of repetitive, transitory pain. “Wave-like” episodes of N/V may also be experienced</p> <p>TOA: usually associated with PID. Symptoms can be consistent with sepsis: high fever, N/V, tachycardia, abdominal rigidity and guarding, rebound tenderness</p> <p>Abortion: amenorrhea, vaginal spotting, crampy abdominal pain</p> <p><35 years old (recent reports state <25 years old); multiple sexual partners; sexual partners with <i>Chlamydia</i>, gonorrhea, or other urethritis, nonbarrier protection</p> <p>-common pathogens: <i>C. trachomatis</i> and <i>N. gonorrhoeae</i></p> <p>Instrumentation of the cervix</p> <p>-common pathogen: <i>Actinomyces israelii</i></p> <p>Bacterial vaginosis (BV)</p> <p>-common pathogens: <i>Bacteroides</i>, <i>Peptostreptococcus</i>, <i>Escherichia coli</i></p> <p>Alterations in vaginal flora (i.e., douching, changes in vaginal pH, necrotic tissue, foreign body reaction)</p> <p>-common pathogens: <i>Bacteroides</i>, <i>Clostridia</i></p>
<p>What is Fitz-Hugh–Curtis (FHC) syndrome?</p>	<p>Focal perihepatitis, causing right upper quadrant tenderness in 15–30% of patients with PID. Right upper quadrant pain does not rule out PID!</p>
<p>Name the diagnostic tests and the expected results that help you make a diagnosis of PID</p>	<p>β-hCG pregnancy test (rule out ectopic pregnancy or complications of an intrauterine pregnancy)</p> <p>Microscopic examination of vaginal discharge in saline (78% for ≥3 WBC/hpf)</p> <p>Gram stain tests for <i>Chlamydia</i> and gonococcus</p> <p>Urinary analysis (UA) (rule out UTI)</p>

	Complete blood count (CBC) (leukocytosis)
	Fecal occult blood tests (r/o acute abdomen)
	C-reactive protein (CRP) (elevated in PID)
	ESR (elevated in PID)
What other methods of evaluation could you consider?	Culdocentesis, ultrasound, endometrial biopsy, laparoscopy
How is the diagnosis of PID for empiric treatment made?	The minimum criteria include cervical motion tenderness or adnexal tenderness . No other causes of these symptoms/signs should be present
Additional criteria that increase the suspicion for PID include:	Oral temperature of $>101^{\circ}\text{F}$ ($>38.3^{\circ}\text{C}$) Abnormal cervical or vaginal mucopurulent discharge Presence of white blood cells on saline
What is the gold standard for diagnosis of PID?	Laparoscopy (usually used in severe cases, patients who have tubo-ovarian drainage, or when the diagnosis is in question)
What are important points to remember regarding treatment?	Always rule out pregnancy! Always use multiple antimicrobial agents to provide coverage for <i>N. gonorrhoeae</i> , <i>C. trachomatis</i> , gram-negative facultative bacteria, streptococci, and anaerobes Better to “over diagnose” to prevent sequelae such as scarring, infertility Reassess in 48–72 hours after initiating treatment! If no improvement, change treatment or diagnosis Always treat sexual partners (asymptomatic or symptomatic)
When should you consider inpatient management?	Pregnancy Inability to exclude surgical emergency (i.e., appendicitis) or uncertain diagnosis Failure to respond to outpatient oral therapy within 72 hours, requiring IV medicines

	Inability to tolerate oral therapy (e.g., severe N/V)
	Severe illness (e.g., high fever, peritonitis)
	Presence of a TOA
	Noncompliance, +IUD, +peritoneal signs, +pelvic mass
	Nulliparity with initial infection will also be a case for hospitalization to avoid sequelae (i.e., scarring and infertility)
What are the first-line regimens for inpatient therapy of PID?	Cefotetan and doxycycline
What are the first-line regimens for outpatient therapy of PID?	Clindamycin and gentamicin
	Ofloxacin ; or levofloxacin ± metronidazole
	Ceftriaxone (or cefoxitin with probenecid) plus doxycycline ± metronidazole
What are complications of PID?	Tubal factor infertility (tubes are scarred), ectopic pregnancy, chronic pelvic pain, TOA, perihepatitis (FHC syndrome), adhesions
What is the most likely cause of infertility in a normally menstruating woman <30 years old?	Pelvic inflammatory disease
How can PID be prevented?	Education of young women and teenagers-at-risk (primary prevention)
	Annual screening for <i>Chlamydia</i> in all sexually active women <25 and women >25 if they have new or multiple partners (secondary prevention)
	Consistent use of barrier contraception
	Oral contraception
	Treatment of sexual partners

Fitz-Hugh–Curtis Syndrome

What is FHC syndrome?

Inflammation of the liver capsule and diaphragm most often associated as an extrapelvic manifestation of PID

What organisms are typically cultured from the infection?	<i>C. trachomatis</i> > <i>N. gonorrhoeae</i>
What are the typical presenting symptoms?	Acute phase: right upper quadrant pain that may radiate to the right shoulder Chronic phase: chronic abdominal pain (commonly over the right upper quadrant)
How is the diagnosis of FHC syndrome made?	Clinical presentation Elevated WBC count and ESR Positive cervical and/or abdominal cultures of <i>C. trachomatis</i> and/or <i>N. gonorrhoeae</i>
What is the gold standard for diagnosis and what are the expected findings?	Diagnostic laparoscopy Acute phase: inflammation (gray and flaky exudates) of the peritoneum and liver capsule Chronic phase: “ violin-string ” adhesions of the anterior liver capsule to the anterior abdominal wall or diaphragm
What is the treatment for FHC syndrome?	Medical (same as for PID: doxycycline plus ceftriaxone or ofloxacin plus metronidazole) or surgical (lysis of adhesions)

Tubo-Ovarian Abscess

What is a TOA?	An abscess of the ovary and fallopian tube that almost always arises as a complication of PID in premenopausal women
In what settings does a TOA develop?	Most commonly, it occurs in the setting of a history of chronically damaged adnexal tissue with a superimposed recurrent infection . Secondary TOA results from intraperitoneal spread of infection by bowel perforation (appendicitis or diverticulitis) or in association with a pelvic malignancy
What are the most common pathogens associated with TOA?	Mixed polymicrobial infection with a high prevalence of anaerobes (<i>Bacteroides</i> and <i>Peptostreptococcus</i>)

<p>What is the typical presentation of a patient with TOA?</p>	<p>and gram-negative organisms (<i>E. coli</i> and streptococcal species)</p>
<p>How does a ruptured TOA present?</p>	<p>The patient is usually young and of low parity with a history of PID. Typical symptoms and signs include severe abdominal and/or pelvic pain, fever, leukocytosis, N/V, rebound tenderness in lower quadrants, diminished bowel sounds, distension, and tympany</p>
<p>What is on the list of differential diagnoses for an unruptured TOA?</p>	<p>As septic shock: fever, chills, tachycardia, hypotension, disorientation, tachypnea, and oliguria</p>
<p>What tests are used to diagnose TOA?</p>	<p>Unruptured ectopic pregnancy, ovarian torsion, pelvic neoplasm, cul-de-sac (pelvic) abscess, acute appendicitis, septic incomplete abortion, perforation of a diverticular abscess or a diverticulum, perforation of a peptic ulcer</p>
<p>What are the findings on culdocentesis?</p>	<p>Pelvic ultrasound (first choice) CT (used if ultrasound is uninformative) Exploratory laparoscopy (gold standard) Culdocentesis</p>
<p>What is the difference between a TOA and a tubo-ovarian complex (TOC)?</p>	<p>Unruptured TOA: “cloudy reaction” fluid Ruptured TOA: grossly purulent material</p>
<p>What are the appropriate steps in management of TOA?</p>	<p>TOC is an inflammatory pelvic mass consisting of living tissue from adherent, infected pelvic structures in PID. Unlike TOA, there is no abscess wall or pus contained within a cavity. It can be distinguished from TOA by ultrasound and it is responsive to medical treatment</p>
	<p>Admit the patient Begin IV fluids followed by IV antibiotics (ampicillin plus gentamicin plus metronidazole <i>or</i> imipenem-cilastin)</p>

Monitor for signs of sepsis (vital signs, CBC, chest x-ray/EKG, urine output)

If there is no response to antibiotics, drain transvaginally or perform exploratory laparotomy

Pelvic Actinomycosis

What is pelvic actinomycosis?

It is a very rare infection of the upper genital tract caused by *Actinomyces israelii*, a **gram-positive anaerobic organism**. It is usually part of a polymicrobial infection

Is it normally part of the female genital tract?

Yes—the presence of *Actinomyces* in the vagina or cervix is neither diagnostic nor predictive of disease

With which gynecologic diseases has *Actinomyces* been associated?

PID, TOA, chronic endometritis, retroperitoneal fibrosis

With what gynecologic procedure has *Actinomyces* been associated?

IUD placement—*Actinomyces* has been identified in 8–20% of women with an IUD

How is the diagnosis of actinomycosis infection made in a symptomatic patient?

Microscopically. A hematoxylin and eosin (H&E) stain reveals sulfur granules and a Gram stain reveals gram-positive filaments

If a Pap smear returns positive for *Actinomyces* on an asymptomatic patient with an IUD, what are the next steps in management?

IUD removal or antibiotic treatment. Repeat the Pap in 1 year

When are both removal of the IUD and treatment with antibiotics necessary?

In a patient showing symptoms/signs of a pelvic infection

Pelvic Tuberculosis

Which bacteria commonly cause pelvic TB?

Mycobacterium tuberculosis or *Mycobacterium bovis*

How does TB reach the pelvic organs?

Via hematogenous dissemination from either the lung or the GI tract

What parts of the upper genital tract does TB usually affect?

Oviducts and endometrium

Which population is most affected?

Pre-menopausal women; immigrants from Asia, the Middle East, and Latin America

<p>What are the most common presenting complaints of patients with a chronic infection of pelvic TB?</p>	<p>Infertility, abnormal uterine bleeding, pelvic pain, and abdominal distension (ascites)</p>
<p>What are some findings on the PE?</p>	<p>Pelvic examination is normal 50% of the time; however, patients may have mild adnexal tenderness and/or bilateral adnexal masses</p>
<p>How is the diagnosis of pelvic TB made?</p>	<p>Positive chest x-ray and lung scan, positive purified protein derivative (PPD), and positive sputum smears/cultures are suggestive. A positive acid-fast stain and culture from menstrual discharge or biopsy of the endometrium is diagnostic</p> <p>*Suspect TB if a patient is not responding to conventional antibiotics for bacterial PID</p>
<p>What are the histologic findings of the endometrial biopsy?</p>	<p>Classic giant cells, granulomas, and caseous necrosis</p>
<p>What are the next steps in management?</p>	<p>Chest x-ray, intravenous pyelogram, serial gastric washings, and urine cultures (for urinary tract TB)</p>
<p>What is the treatment?</p>	<p>A multidrug regimen consisting of isonicotinyl hydrazine (INH), rifampicin, pyrazinamide, and ethambutol</p>
<p>What are some complications of pelvic TB?</p>	<p>Infertility and chronic endometritis</p>

VAGINITIS

Introduction

<p>What are the characteristics of normal vaginal discharge?</p>	<p>White or transparent in color, thick, and odorless</p>
<p>What is the normal vaginal pH?</p>	<p><4.5</p>
<p>What is the microbiology of normal vaginal flora?</p>	<p>There is an average of six different species of bacteria, which are predominantly aerobic. The most common is the hydrogen peroxide-producing lactobacilli</p>
<p>What does microscopy of normal vaginal secretions reveal?</p>	<p>Predominantly squamous epithelial cells, few white blood cells (<1 per epithelial cells), and possibly a few clue cells</p>

What are the typical symptoms of vaginitis?	Increased vaginal discharge, pruritus, irritation, soreness, odor, dyspareunia, bleeding, dysuria, and mucosal erythema
What are some of the most important etiologies to consider in your differential diagnoses?	<p>Most common infectious causes (bacterial vaginosis > vulvovaginal candidiasis > trichomoniasis)</p> <p>Less common “infectious” causes (desquamative inflammatory vaginitis, foreign body with secondary infection)</p> <p>Noninfectious causes (atrophic vaginitis, contact dermatitis, allergens, irritants, hypersensitivity)</p>
What laboratory tests are typically ordered to diagnose the etiology of vaginitis?	pH, amine test (whiff test), saline microscopy

Bacterial Vaginosis (Nonspecific Vaginitis)

What is BV?	The most common cause of infectious vaginitis and results from an alteration of the normal vaginal bacteria flora . Loss of the normal hydrogen peroxide-producing lactobacilli results in an overgrowth of anaerobes such as <i>Gardnerella vaginalis</i> and <i>Mycoplasma hominis</i>
What are the key distinguishing symptoms?	“Musty” or “fishy” vaginal odor; thin, homogenous, gray-white discharge ; less inflammatory symptoms; dyspareunia absent
What are risk factors for BV?	<p>Multiple sex partners</p> <p>A new sex partner</p> <p>Douching</p> <p>Lack of vaginal lactobacilli</p>
What are the three major diagnostic findings in BV?	<p>Vaginal pH >4.5</p> <p>Positive amine/whiff test (release of fishy, amine-like odor when vaginal fluid is alkalized with KOH)</p> <p>Saline microscopy reveals >20% of clue cells (vaginal epithelial cells with adherent bacterial clusters)</p>

What are some important complications of BV?

Cervicitis, increased risk of PID, increased risk of HIV infection, preterm delivery, intrapartum and postpartum infections, first trimester miscarriages

What is the treatment for BV?

Oral or intravaginal metronidazole or clindamycin

*Pregnant patients should not receive topical clindamycin

Do sexual partners need to be treated?

No. BV is not a sexually transmitted disease

Vulvovaginal Candidiasis

What is vulvovaginal candidiasis (VVC)?

It is a **yeast infection** of the vagina primarily caused by *Candida albicans* because of a change in the vaginal flora

What are the major risk factors for VVC?

Immunosuppression (**corticosteroids**, AIDS)

Changes in normal vaginal flora (**antibiotics**)

Hormonal changes (**pregnancy**, menstruation, higher dose estrogen OCP)

Intrauterine devices and vaginal sponges

Diabetes mellitus

What key symptoms and signs distinguish candidiasis from other causes of infectious vaginitis?

Intense vulvovaginal pruritis, soreness, vulvar mucosal erythema and edema, and vaginal discharge that resembles **white “cottage cheese”**

What are other conditions that must be considered in the differential diagnosis?

Hypersensitivity, allergic or chemical reactions, and contact dermatitis

What is the pH of the vagina in patients with VVC?

Normal pH (4–4.5)

How would you definitively diagnose vaginal candidiasis?

Microscopic evaluation of a wet saline or KOH prep of vaginal fluid reveals **hyphae, pseudohyphae, or budding yeast (“spaghetti and meatballs”)**

What is the treatment?

Either **oral fluconazole** (single dose in nonpregnant women) or **topical or intravaginal antifungal drugs**

(3–7 days). About 1% hydrocortisone may be used to relieve external irritative symptoms

When is oral fluconazole contraindicated?

During pregnancy. A 7-day course of topical antifungal therapy is recommended for pregnant patients

Does the patient's sexual partner need to be treated as well?

No. Candidiasis is not typically a sexually transmitted disease

Trichomonas Vaginalis

What is trichomoniasis?

A sexually transmitted vaginal infection caused by the flagellated protozoan, *T. vaginalis*. There is a strong association with BV

What are the key presenting characteristics of trichomoniasis?

Malodorous, purulent, greenish, frothy, profuse watery discharge; vulvovaginal erythema and irritation, dyspareunia, dysuria; punctate hemorrhages on the cervix ("**strawberry cervix**")

How is the diagnosis usually made?

pH of vaginal secretions >5.0
Microscopic examination of a wet saline prep of vaginal fluid reveals **motile trichomonads**

What are some complications of trichomoniasis?

Increased HIV transmission; increased risk of PID; preterm delivery; premature rupture of membranes; low birth weight infants

What is the treatment?

Oral metronidazole

Pregnant patients should receive oral metronidazole (category B drug)

All sexual partners should be treated

What other tests should be considered?

Tests for *N. gonorrhoeae*, *C. trachomatis*, syphilis, HIV

Desquamative Inflammatory Vaginitis

What are the three characteristics of desquamative inflammatory vaginitis?

Diffuse, exudative vaginitis
Vaginal-epithelial cell exfoliation
Profuse and purulent vaginal discharge

What causes inflammatory vaginitis?	Replacement of normal lactobacilli with gram-positive cocci (usually streptococci)
What type of patients present with desquamative inflammatory vaginitis?	Premenopausal women with normal estrogen levels
How do these patients typically present?	With a purulent vaginal discharge, vulvovaginal burning, dyspareunia, vaginal erythema, and a vulvovaginal-cervical spotted rash
What are some laboratory findings?	Vaginal secretion pH >4.5 Increased number of parabasal cells Gram-positive cocci (usually streptococci) on Gram-staining
What is the treatment?	2% clindamycin cream intravaginally × 7 days

Atrophic Vaginitis

Which women are more likely to be affected by atrophic vaginitis?	Postmenopausal women
What causes atrophic vaginitis?	Thinning of the vaginal epithelium because of a reduction of endogenous estrogen. Reduction of lactobacilli and lactic acid increases the vaginal pH and leads to an overgrowth of non-acidophilic organisms
What are some common symptoms?	Mild vaginal atrophy is usually asymptomatic. Advanced vaginal atrophy can present with vaginal soreness, purulent vaginal discharge, dyspareunia, and postcoital irritation and bleeding
What does a physical examination reveal?	Atrophy of external genitalia, loss of vaginal folds, thin and diffusely erythematous vulvovaginal mucosa with some ecchymoses, and watery or serosanguineous discharge
What do laboratory tests of the vaginal secretions reveal?	pH >5.0–7.0 Increased number of leukocytes and parabasal epithelial cells Increased gram-negative rods
What is the treatment?	Topical estrogen vaginal cream

Noninfectious Causes of Vaginitis

What are some causes of noninfectious vaginitis?

Topical antimycotic drugs
Spermicides
Mini-pads/pantyliners and other feminine products
Soaps
Povidone-iodine
Latex condoms
Seminal fluid

What are the symptoms of noninfectious vaginitis?

Pruritus, irritation, burning, soreness, and variable discharge (similar to infectious vaginitis)

With what disease can noninfectious vaginitis be confused?

Acute *Candida* vaginitis

How is the diagnosis made?

Based upon symptoms and exclusion of all infectious etiologies

What is the treatment?

Removal of causative agent
Local relief by sodium bicarbonate, sitz baths, and topical vegetable oils

OTHER GYNECOLOGIC INFECTIONS

Postoperative Pelvic Infection

What are some gynecologic postoperative infections?

Cuff and pelvic cellulitis
Salpingitis
Suppurative pelvic thrombophlebitis
TOA with and without rupture

What are the five major causes of fever in the postoperative gynecology patient?

The five "W"s:
Wind = pulmonary atelectasis or pneumonia
Water = urinary tract infection
Walk = deep vein thrombosis (DVT) or superficial phlebitis
Wound = infection from the abdominal incision or from a pelvic source
Weird drugs = drug-causing fevers

On what postoperative day does fever because of pelvic infection commonly occur?

Between postoperative day (POD) 2–4

What should one think of if fevers continue and there is no clinical response to antibiotics?

Pelvic abscess

Toxic Shock Syndrome

What is toxic shock syndrome (TSS)?

An **acute** illness characterized by **high fevers** which may quickly lead to **hypotensive shock** and **multisystem failure**

What is the cause of toxic shock syndrome?

Preformed exotoxins produced by *Staphylococcus aureus*

In which patients has this syndrome been associated?

Menstruating women between ages 12–24 who use **superabsorbent tampons**

Postpartum women

Women who use a diaphragm

In both men and women following surgical procedures

What are the major clinical findings in TSS?

Abrupt onset of **high fevers**, **vomiting**, and **diarrhea**. Within 48 hours, **signs of shock** occur (temperature $\geq 102.2^\circ\text{F}$, dehydration, tachycardia, hypotension) and a **diffuse “sunburn-like” rash** appears over the face, trunk, and proximal extremities. **Desquamation (particularly affecting the palms and soles)** may occur 1–2 weeks after the onset of illness

Involvement of three or more organ systems (GI, CNS, renal, mucous membrane, skin, cardiac, hepatic) is essential for diagnosis

What information should be sought during the H&P that is pertinent to this diagnosis?

Ask the patient if she is **menstruating or using tampons!!!** You must perform a vaginal examination and remove the tampon immediately if one is present

What laboratory should be ordered?

Complete panel of blood tests

	Cultures from blood, sputum, CSF, and the vagina
What will the vaginal culture yield in TSS?	Penicillinase-producing <i>S. aureus</i>
What is the management and treatment for a patient with TSS?	Assess hemodynamics Replace fluid volume and electrolytes (may require dopamine infusion) Monitor urine output IV antibiotics: β -lactamase-resistant antibiotic (nafcillin or oxacillin) Vancomycin (for penicillin-allergic patients)
What are the three most common causes of death from TSS?	Acute respiratory distress syndrome Intractable hypotension Hemorrhage secondary to DIC

HIV/AIDS

What is HIV?	A single-stranded RNA retrovirus that infects CD4 receptor lymphocytes and other target cells and causes a progressive decrease in cellular immunity leading to AIDS
What are the three means of HIV transmission?	Sexual contact Parenteral exposure to blood or bodily fluids (i.e., IV drugs, occupational exposure) Vertical transmission (from an infected mother to her fetus)
How do HIV-infected patients initially present?	With mononucleosis-like symptoms such as fever, weight loss, night sweats , pharyngitis, lymphadenopathy, erythematous maculopapular rash. This is followed by a long asymptomatic period lasting from months to years
What percent of those diagnosed with AIDS in the United States are women?	25%
What are some symptoms of HIV infection in females?	Difficult-to-treat vaginal infections (candidiasis , BV, and common STDs) Increase in frequency, severity, and recurrence of HSV ulcers, HPV infections, and cervical dysplasia

<p>How is it diagnosed?</p>	<p>Presence of idiopathic genital ulcers</p> <p>Screening test: ELISA (detects antibodies to HIV)</p> <p>Confirmation test: Western blot</p>
<p>When can the ELISA test give a false-negative result?</p>	<p>In early infection (<12 weeks). All patients with recent exposure need to be tested again after this window period</p>
<p>What vaccinations should be offered to HIV-infected patients?</p>	<p>Hepatitis B, influenza, and pneumococcus</p>
<p>What organism commonly causes pneumonia in HIV-infected patients?</p>	<p><i>Streptococcus pneumoniae</i></p>
<p>What is the most common opportunistic pneumonia in HIV-infected patients? How is it detected and treated?</p>	<p><i>Pneumocystis carinii</i> (PCP)</p> <p>Diagnosis: bronchoalveolar lavage with silver stain (Giemsa)</p> <p>Treatment: trimethoprim-sulfamethoxazole or pentamidine</p>
<p>What is the current treatment regimen for HIV infection?</p>	<p>Two anti-retrovirals and one protease inhibitor</p>

Pelvic Pain

CHRONIC PELVIC PAIN

Etiologies

<p>How is chronic pelvic pain (CPP) defined?</p>	<p>It is noncyclic pain of nonmenstrual origin that lasts ≥ 6 months duration and is located below the umbilicus. The pain is severe enough to cause functional disability or require medical treatment</p>
<p>What is the prevalence of chronic pelvic pain?</p>	<p>Approximately 15–20% of women aged 18–50 years have chronic pelvic pain of greater than 1 year duration</p>

What are the most common gynecologic conditions that cause chronic pelvic pain?

Pelvic inflammatory disease (18–35%); endometriosis; adenomyosis; gynecologic malignancies (late stages); tuberculous salpingitis

How often is endometriosis diagnosed by laparoscopy in women with chronic pelvic pain?

Diagnosis of endometriosis by laparoscopy is made in 33% of women with chronic pelvic pain

What are the most significant non-gynecological causes of chronic pelvic pain?

Consistent scientific evidence has shown that interstitial cystitis, irritable bowel syndrome, chronic coccygeal or back pain, and depression may cause or exacerbate chronic pelvic pain

What is the relationship between chronic pelvic pain and involvement of more than one organ system?

Pain is more severe when more than one organ system is involved. Women with chronic pain are more often found to have dysmenorrhea and dyspareunia compared to the general population. The severity of pain is greater in women who have gastrointestinal or urologic symptoms in addition to the chronic pelvic pain

What is the association between physical or sexual abuse, and chronic pelvic pain?

About 40–50% of women with chronic pelvic pain have a history of some form of abuse. A direct causal relationship and mechanism to chronic pelvic pain has not been established

What other mental disorders should be considered in a patient with chronic pelvic pain?

Somatization, opiate abuse, depression

What type of obstetrical history may lead to chronic pelvic pain?

Known as “peripartum pelvic pain syndrome,” this is a musculoskeletal source of pain that manifests in women with lumbar lordosis, delivery of a large infant, muscle weakness and poor physical conditioning, a difficult delivery, vacuum or forceps delivery, and use of gynecologic stirrups for delivery

Workup

What information should be gathered when taking the history of the patient's pain?

A thorough review of systems should be performed with emphasis on urinary tract disease, bowel disease, reproductive tract disease, musculoskeletal disorders, and psychoneurologic disorders

What characteristics of the pain should be investigated as part of the detailed history?

Location, intensity, quality, duration, temporal pattern, precipitating patterns (exertion, sexual activity, menses, pregnancy), radiation, and relationship to urination and defecation

How do the nature and the quality of pain give a clue to the source of the pain?

Somatic pain is usually localized and sharp, indicating a musculoskeletal origin

Visceral pain is usually vague, aching, and difficult to localize; this may indicate an intraperitoneal or upper reproductive tract etiology

What should be looked for on physical examination?

The physical examination should concentrate on looking for points of localized or generalized tenderness, surgical scars, masses, and hernias. The pelvic examination should focus on identifying physical findings consistent with pelvic floor dysfunction, painful bladder syndrome, endometriosis, adenomyosis, or leiomyoma

What are the physical findings that suggest the following etiologies of chronic pelvic pain?

Endometriosis: uterosacral ligament nodularity or thickness, cervical stenosis, lateral displacement of the cervix because of shortening of one of the ligaments

Adenomyosis: slightly enlarged, globular, tender uterus on examination

Leiomyoma: enlarged, mobile uterus with an irregular contour on bimanual or abdominal examination

Pelvic inflammatory disease (PID): uterine tenderness or cervical motion tenderness on examination

What is Carnett's sign?

Neuropathy: burning, shock-like, paresthesia, and dysesthesia

Neoplasm: adnexal mass, ascites

It refers to **increased local tenderness during muscle tensing by raising both legs straight up while lying supine**. This maneuver tightens the rectus abdominis muscles, increasing pain if there is **myofascial pain** (e.g., trigger points, entrapped nerve, hernia, myositis), while true visceral sources of pain are associated with less tenderness when abdominal muscles are tensed

Which laboratory and imaging tests should be ordered in a patient with chronic pelvic pain?

Complete blood count (CBC) with differential and ESR

Pregnancy test

Urinalysis

Occult blood test

Pelvic ultrasound

Chlamydia and gonorrhea infection

CA-125

When should MRI or CT scan be used?

Only when abnormalities are found on ultrasound examination

What is the role of laparoscopy in evaluation of women with chronic pelvic pain?

It is indicated in women who have symptoms and signs of endometriosis and/or adhesions. It is also indicated in women with **chronic pelvic pain** who have not had relief of symptoms with nonsteroidal anti-inflammatory drugs (NSAIDs) or estrogen-progestin treatment and have no strong contraindications to laparoscopic surgery

What is conscious laparoscopic pain mapping?

It refers to laparoscopy performed under local anesthesia in which the tissues are probed and pulled with surgical instruments while the patient is asked about the severity and nature of any pain she perceives. It can lead to the treatment of subtle or atypical areas of disease that might have been previously overlooked. It may also help prevent surgical treatment when no painful lesions are identified

Treatment

How is endometriosis-associated chronic pelvic pain treated?

Empirical sequential treatment with nonsteroidal anti-inflammatory drugs, oral contraceptives administered in monthly or longer cycles, GnRH agonist analogues (nafarelin, goserelin, and leuprolide), progestins (medroxy-progesterone acetate)

In what other sources of chronic pelvic pain are GnRH agonists effective?

Irritable bowel syndrome, pelvic congestion syndrome, and interstitial cystitis also respond to GnRH agonists

When are oral contraceptives indicated for treatment of chronic pelvic pain?

To decrease pain from primary dysmenorrhea

What is the role of antidepressants in treatment of chronic pelvic pain?

Currently, evidence is insufficient to substantiate efficacy of antidepressants for the treatment of chronic pelvic pain. However, adding psychotherapy to medical treatment of chronic pelvic pain appears to improve response over that of medical treatment alone and should be considered

What is the role of surgery in treatment of chronic pelvic pain?

Excision or destruction of endometriotic tissue, lysis of adhesions, and hysterectomy (in women who have finished childbearing) have been proposed to relieve chronic pelvic pain

What is presacral neurectomy and is it effective for treatment of chronic pelvic pain?

It is the surgical resection of the superior hypogastric plexus which innervates the cervix, uterus, and proximal fallopian tubes with afferent nociception. It may be used to treat centrally located dysmenorrhea but has limited efficacy for chronic pelvic pain or pain that is not central in its location

What is the main take-home point of treatment for chronic pelvic pain?

Multimodal therapy (medical therapy, surgical therapy, and behavioral/mental health treatment, along with pain consultation) is superior to treatments that emphasize a single approach

ACUTE PELVIC PAIN

Etiologies

What are the obstetric and gynecologic differential diagnoses for acute pelvic pain?

Pelvic inflammatory disease
 Adnexal cysts/masses/abscesses with bleeding, torsion, or rupture
 Ectopic pregnancy
 Spontaneous abortion
 Endometritis
 Degeneration, infarction, or torsion of leiomyomas (fibroids)

What are non-gynecologic differential diagnoses for acute pelvic pain?

Appendicitis
 Diverticulitis
 Urinary tract infection or obstruction
 Renal colic

Which of these diagnoses are life threatening and must be ruled out quickly?

Ectopic pregnancy
 Tubo-ovarian abscess
 Ruptured ovarian cyst
 Appendicitis

On what additional points should the history taking focus to determine a pelvic etiology?

The regularity and timing of menstrual periods, possibility of pregnancy, presence of vaginal discharge or bleeding, and a recent history of dyspareunia or dysmenorrhea

What findings on physical examination may lead you to the appropriate gynecologic/obstetric etiology for acute pelvic pain?

Ectopic pregnancy: unruptured pregnancy leads to localized unilateral pain because of fallopian tube dilatation. If it ruptures, the pain becomes generalized because of peritoneal irritation. A pelvic examination may reveal cervical motion tenderness that is exaggerated on the side of the tubal pregnancy

Tubo-ovarian abscess: patients present with generalized abdominal pain and rebound tenderness caused by peritoneal inflammation. Bilateral, palpable, fixed, and tender masses are commonly found on examination

What labs or tests should be considered in the workup of acute pelvic pain?

How do differences in the clinical and laboratory findings help point out the etiology of acute gynecologic pelvic pain?

What is mittelschmerz?

Acute PID: lower abdominal tenderness, cervical motion tenderness, adnexal tenderness

Ovarian torsion/rupture: severe unilateral, lower abdominal pain, adnexal mass, nausea and vomiting

Uterine infection or pathology (torsion, degeneration, infarction): irregular enlargement and painful uterus on palpation

Endometriosis: localized pain in the cul-de-sac or uterosacral ligaments, palpable tender nodules in the cul-de-sac, uterosacral ligaments, or rectovaginal septum, possible fixation of adnexa or uterus in a retroverted position

CBC; urinary analysis (UA); pregnancy test; tests for *Chlamydia* and gonorrhea in women with suspected PID; microscopic examination of (wet mount) of any abnormal vaginal discharge; culdocentesis; vaginal ultrasound; laparoscopy

See Table 4.1

It is recurrent acute midcycle abdominal pain because of leakage of prostaglandin-containing follicular fluid at the time of ovulation. The pain associated is typically mild, unilateral, midway between menstrual periods, and lasts for a few hours to a couple of days

Endometriosis

What is endometriosis?

A benign yet very debilitating gynecologic disease where **endometrial glands and stroma** are present in an **extrauterine location**

Table 4-1 Differential Diagnosis of Acute Gynecologic Pelvic Pain

Disease	Clinical and Laboratory Findings					
	CBC	UA	Pregnancy Test	Culdocentesis	Fever	Nausea and Vomiting
Ruptured ectopic pregnancy	Hematocrit low after treatment of hypovolemia	Red blood cells rare	Positive, beta-hCG low for gestational age	High hematocrit Defibrinated, nonclotting sample with no platelets Crenated red blood cells	No	Unusual
Salpingitis/PID	Rising white blood cell count	White blood cells occasionally present	Generally negative	Yellow, turbid fluid with many white blood cells and some bacteria	Progressively worsening; spiking	Gradual onset with ileus
Hemorrhagic ovarian cyst	Hematocrit may be low after treatment of hypovolemia	Normal	Usually negative	Hematocrit generally <10%	No	Rare
Torsion of adnexa	Normal	Normal	Generally negative	Minimal clear fluid if obtained early	No	Rare
Degenerating leiomyoma	Normal or elevated white blood cell count	Normal	Generally negative	Normal clear fluid	Possibly	Rare

Reprinted, with permission, from Pearlman MD, Tintinalli JE, eds. *Emergency Care of the Woman*. New York, NY: McGraw-Hill; 1998: 508.

To which hormone does ectopic endometrial tissue respond? What is the significance of this?

Estrogen. It changes in a cyclic manner according to menstrual changes in estrogen levels. The ectopic tissue can release a small amount of blood into the surrounding tissues leading to repeated **tissue inflammation, pelvic pain, scarring, and eventually adhesions in the reproductive organs, pelvis, and other intestines**

Who develops endometriosis?

Reproductive-age women and postmenopausal women on estrogen-replacement therapy

What is the prevalence of endometriosis in the United States?

7% of reproductive-age women are diagnosed with endometriosis

What are the two most common symptoms associated with endometriosis and what percentage of women with these complaints are found to have endometriosis?

Chronic pelvic pain (71–87%); Infertility (38%)

How does endometriosis lead to infertility?

1. It causes the **formation of adhesions** that distort the normal uterine/tubal/ovary anatomy, inhibit tubo-ovarian motility, and block ovum release
2. The release of other substances (such as **cytokines**) may be “toxic” to normal ovarian function/fertilization/implantation

What other common complication can endometrial adhesions cause?

Small bowel obstruction

For what other disease is endometriosis a risk factor?

Epithelial ovarian cancer (EOC)

Since both are estrogen-dependent diseases, the presence of endometriosis may indicate a risk for developing EOC

Do genetic factors increase the risk for endometriosis?

Yes. There is a seven- to tenfold increased risk of developing endometriosis if a first-degree relative has been affected by endometriosis. The mode of inheritance is polygenic and multifactorial

What three leading theories explain the etiology of endometriosis?

1. **Retrograde menstruation:** a reverse flow of endometrial tissue through the fallopian tubes

during menses leads to the seeding of endometrial cells in the peritoneal cavity

2. **Lymphatic and vascular spread:** spread of endometrial cells through lymphatic and vascular channels
3. **Coelomic metaplasia:** transformation of coelomic epithelium or undifferentiated peritoneal cells into endometrial tissue by an endogenous undefined biochemical factor

In what sites are endometrial implants found?

Most common: **ovaries**, anterior and posterior cul-de-sac, uterosacral ligaments, posterior uterus, posterior broad ligaments, sigmoid colon, appendix, round ligaments

Less common: vagina, cervix, rectovaginal septum, cecum, ileum, inguinal canals, abdominal or perineal scars, ureters, urinary bladder, and umbilicus

Rare: breast, pancreas, liver, gallbladder, kidney, urethra, extremities, vertebrae, bone, peripheral nerves, lung, diaphragm, and central nervous system

What are the common presenting symptoms of endometriosis and how do ectopic lesions explain these symptoms?

Chronic pelvic pain: local peritoneal inflammation secondary to formation of adhesions and fibrosis

Acute exacerbations of pelvic pain: peritoneal inflammation secondary to the release of hemolyzed blood and other contents of ruptured endometriomas

Secondary dysmenorrhea: cyclical changes in ectopic lesions secondary to changes in estrogen levels

Dyspareunia: deep infiltration and scarring of the uterosacral ligaments and rectovaginal septum by endometriotic lesions. Can also be caused by a retroverted uterus secondary to adhesions (leads to deep dyspareunia)

	<p>Infertility: invasive endometriosis and the formation of adhesions distorts the normal uterine/tubal/ovary anatomy, inhibits tubo-ovarian motility, and blocks ovum release</p> <p>Many women are asymptomatic</p> <p>Cyclical rectal bleeding</p>
What symptom is pathognomonic for endometriosis?	
What are endometriomas?	Invasive endometriotic lesions found inside the ovary. Often described as a “chocolate cyst” because it contains old blood that has undergone hemolysis
What are the complications of endometriomas?	Rupture of endometriomas can cause peritoneal inflammation, scarring, and pelvic adhesions
What findings are typically found on physical examination?	<p>Tenderness upon pelvic examination during menses (most common finding)</p> <p>Uterosacral or cul-de-sac nodularity</p> <p>Retroverted uterus and limited motion of ovaries and fallopian tubes</p> <p>Tender, enlarged adnexal mass (unilateral)</p> <p>There are often no abnormal findings on physical examination</p>
What is the differential diagnosis for the presentation of endometriosis?	<p>Pelvic inflammatory disease (PID)</p> <p>Acute or chronic inflammation</p> <p>Ectopic pregnancy</p> <p>Kidney stones</p> <p>Cystitis</p> <p>Pelvic tumors</p>
What laboratory tests would help you rule out the diseases on your differential diagnoses list?	Complete blood count (CBC) and erythrocyte sedimentation rate (ESR); cervical Cx; urine β -hCG; urinalysis and urine culture
How do the following tests help you in your diagnostic decision making?	
CBC and ESR	Positive findings are nonspecific; leukocytosis may be seen in infection

Cervical Cx	Positive cultures for guanylyl cyclase c (GC/C) may indicate PID
Urine β -hCG	Positive test indicates pregnancy, ruling out ectopic pregnancy is necessary
Urinalysis and urine culture	Ruling out UTI
What value does CA125 have in the clinical management of endometriosis?	CA125 is not a sensitive diagnostic test for endometriosis (especially in minimal to mild disease). However, it may be used as a marker to follow medical treatment response for endometriosis. CA125 levels may decrease during treatment compared to pretreatment values; however, normal posttreatment values do not confirm the absence of endometriosis nor are they useful markers for predicting disease recurrence
Are imaging tests a sensitive modality for diagnosing endometriosis?	No. However, ultrasound, CT, and MRI are useful for detecting pelvic or adnexal masses (i.e., endometriomas) or ruling out other causes of pelvic pain. Deeply infiltrating endometriosis that involve the uterosacral ligaments and the cul-de-sac may be detected by MRI
How is the diagnosis of endometriosis made?	Direct visualization by laparoscopy/ laparotomy with histologic confirmation
What are the characteristic findings of endometriosis found during laparoscopy/ laparotomy?	<p>Classic lesions: brown/black/ blue “powder burn,” “gun shot” lesions, nodules or small cysts containing old hemorrhage surrounded by fibrosis on the serosal surfaces of the peritoneum</p> <p>Atypical/subtle lesions: clear vesicles, white opacifications, red, white, yellow/ brown plaques, excrescences, scars or lesions of varying sizes</p> <p>Note: Normal appearing peritoneum may have microscopic evidence of endometriosis</p>

What histologic findings confirm the diagnosis of endometriosis?

The **presence of two or more** of the following histologic features: endometrial epithelium, endometrial glands, endometrial stroma, hemosiderin-laden macrophages

What is the current classification system for endometriosis staging?

One created by the American Society for Reproductive Medicine. It uses point scores for disease staging and is used primarily for the uniform recording of operative findings

What are the limitations of this system?

It is not good for correlating disease stage with pain and/or dyspareunia. It also does not predict the chance of pregnancy following treatment

What is the typical clinical outcome of endometriosis and what is the goal of treatment?

Endometriosis is a progressively deteriorating disease that rarely improves. Elimination of endometriotic implants by medical or surgical intervention provides only temporary relief. However, **the goal of treatment should be to eradicate the lesions, treat endometriosis-related pelvic pain and infertility, and prevent bowel and extra-pelvic complications**

What factors must be considered when choosing the appropriate treatment regimen?

Patient's desire for future fertility, side effects of medication, cost, and patient's tolerance

What type of medical therapy is most effective for endometriosis?

Hormonal therapy that **suppresses estrogen synthesis** (induces atrophy of ectopic lesions) and/or **disrupts the menstrual cycle** (abrogates or diminishes retrograde bleeding)

What are the most commonly used hormonal medications for the treatment of endometriosis?

Oral contraceptive pills (OCPs): best for minimal to mild pain, reduces menstrual flow

Progestins: good for moderate to severe disease, excellent pain relief, induces atrophy of endometrial implants, few side effects, inexpensive

Danazol: effective for mild to moderate disease, relieves dysmenorrhea, inhibits midcycle

	LH/FSH surge, induces endometrial atrophy within uterus and ectopically ultimately leads to amenorrhea
	GnRH agonists: inhibit pituitary gonadotropin secretion which suppresses ovarian estrogen production, induces amenorrhea, relieves pain
What other nonhormonal medication treats endometriosis-related pain?	Nonsteroidal anti-inflammatory drugs
When is surgery indicated for the treatment of endometriosis?	<ol style="list-style-type: none"> 1. When symptoms have not improved or worsened with medical management 2. When symptoms of endometriosis are severe and incapacitating 3. For advanced disease 4. To treat endometriomas, bowel or urogenital tract obstruction, or anatomically distorted pelvic structures because of adhesions/invasive endometriotic lesions
What are the indications for conservative surgery (preserves uterus and ovarian tissue)?	Less severe disease or for those who want to preserve fertility
What are the indications for definitive surgery (hysterectomy with or without removal of the fallopian tubes and ovaries)?	For patients with advanced disease and for those who no longer want/need fertility conservation
What is the impact of medical management versus surgical management on recurrence and infertility?	Similar or higher rates of recurrence have been reported with medical management versus surgical management. Surgery has been reported to improve rates of fertility compared to expectant management
What is the most likely cause of infertility in a menstruating woman without a history of PID?	Endometriosis
What is the management of endometriosis-related infertility?	Assisted reproduction technologies (ART) such as IVF
What is adenomyosis?	The presence of ectopic endometrial glands and stroma within the uterine musculature
What is the prevalence of adenomyosis?	It is thought to affect 20% of all women

What is the classic presentation of a patient with adenomyosis?	A parous, middle-aged woman with menorrhagia and dysmenorrhea with a symmetrically enlarged, tender, and “boggy” uterus
How is the diagnosis of adenomyosis made?	A presumptive diagnosis is based on the clinical presentation in the absence of endometriosis or leiomyomas. A definitive diagnosis requires histologic assessment of the uterine tissue
What is the differential diagnosis for adenomyosis?	Leiomyoma Intra-abdominal neoplasia Endometriosis PID
Is adenomyosis related to endometriosis?	Although they are both disorders of the ectopic endometrium, they are unrelated
What is the definitive treatment for adenomyosis?	Hysterectomy
What is the prognosis for adenomyosis?	Good—it is a self-limited process that often becomes asymptomatic after menopause

Benign Pelvic Masses

BENIGN CERVICAL MASSES

Cervical Cysts

What four types of cervical cysts are there?	1. Nabothian 2. Mesonephric 3. Endometrial 4. Adenosis
What are the symptoms of a cervical cyst?	Usually asymptomatic but can cause dyspareunia
What is a nabothian cyst and how does it develop?	A discrete mucus-filled cyst that appears grossly as a small translucent or yellow elevation on the cervix. Nabothian cysts are often discovered on pelvic examination. They occur

<p>How are nabothian cysts treated?</p>	<p>via metaplasia, a process by which cervical cells convert from glandular cells that secrete mucus to squamous cells. Sometimes a cleft of columnar endocervical epithelium becomes covered by squamous epithelium, trapping the mucus secretions and forming cysts.</p> <ul style="list-style-type: none"> • Most are asymptomatic and therapy is not needed • If symptomatic, treatment can be via excision, electrocautery or cryotherapy
<p>Which other cervical cyst may be confused with a nabothian cyst? How can it be distinguished from a nabothian cyst?</p>	<p>The mesonephric (Wolffian) cysts are found deep in the stroma of the ectocervix and are lined by Wolffian-type cells</p>
<p>What conditions are most associated with cervical cysts?</p>	<p>Pregnancy, menopause, and cervicitis</p>

Cervical Polyps

<p>What are cervical polyps?</p>	<p>Small, pedunculated, benign neoplasms of the cervix composed of a vascular connective tissue stroma covered by epithelium. They commonly arise via focal hyperplasia of the endocervix and protrude from the cervical canal out of the external os.</p>
<p>How common are they and in whom do they develop?</p>	<p>Relatively common—especially in multigravidas over 20 years of age. They are rare before menarche but may develop after menopause</p>
<p>How are cervical polyps diagnosed and managed?</p>	<ul style="list-style-type: none"> • Asymptomatic polyps often are discovered on routine pelvic examination. Otherwise they usually present with intermenstrual or postcoital bleeding or discharge • Although carcinoma developing in a polyp is rare, polyps should be removed close to their attachment and pathologically reviewed

BENIGN UTERINE MASSES

Leiomyomas

What are uterine leiomyomas?

Benign tumors of the smooth muscle cells (myometrium) of the uterus; Also known as **fibroids**, **fibromyomas**, or **myomas**

What are the three types of leiomyomas? (see Fig. 4-3)?

1. **Intramural**—located **within the wall of the myometrium** and may distort the shape of the uterine cavity and surface
2. **Submucosal**—originate in the myometrium and **grow toward the endometrial cavity**, protruding into the uterine lumen
3. **Subserosal**—originate in the myometrium and grow out toward **the serosal surface** of the uterus; extend from the uterine surface into the peritoneum and abdominal cavity

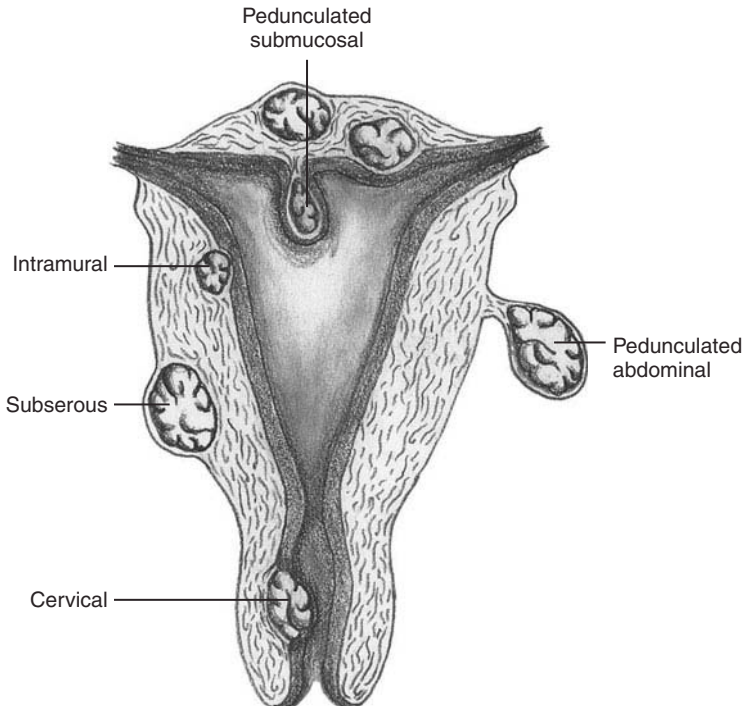


Figure 4-3 Types of leiomyomas.

What is the prevalence of leiomyomas and in whom do they occur?

Leiomyomas are **hormonally responsive** and are found in **20–30% of reproductive-age women**. Most women diagnosed with leiomyomas are **between 30 and 50 years old**. **Black women** are 2–3 times more likely to develop fibroids than white women.

What are the risk factors for leiomyomas?

Black race

Nulliparity

Oral contraceptives (OCPs) are mostly **protective**

Diet—a diet high in red meat and low in green vegetables has been suggested to increase the risk for fibroids

Genetics

Alcohol

What are the usual presentation/symptoms/signs of fibroids?

Most are **asymptomatic** and so are diagnosed based on the finding of an **irregularly enlarged, mobile uterus** on gynecologic examination

If symptoms do occur, the most common are:

Abnormal uterine bleeding (menorrhagia or metrorrhagia)

Pelvic pressure/pain (dysmenorrhea, urinary frequency, constipation, or dyspareunia)

Infertility

Aside from leiomyomas, what are other differential diagnoses of a patient who presents with menorrhagia and pelvic pressure/pain?

Adenomyosis; primary dysmenorrhea; endometriosis; tubo-ovarian abscess; malignancy (cervical, endometrial, or ovarian)

How is the diagnosis of fibroids usually made?

Via ultrasonography

Describe the theoretical mechanisms for increased bleeding associated with leiomyomas.

1. An **alteration in myometrial cells contractile ability** leading to lack of control of bleeding from the endometrial arterioles
2. The endometrium is **unable to respond to hormonal changes** in the menstrual cycle, leading to sloughing off of excess tissue

What other methods of evaluation could you consider for the diagnosis of fibroids?

3. Increased pressure causes **necrosis of the endometrium**, which leads to exposure of the vasculature surfaces and increased bleeding

Hysterosalpingography allows for visualization of submucosal fibroids
MRI or CT

What causes the development and growth of leiomyomas?

Hysteroscopy and/or dilation and curettage (D&C)

It is unknown what causes their development. Their growth is responsive to both **estrogen** and **progesterone**. Estrogen may increase the production of extracellular matrix. Progesterone increases the mitotic activity of myomas and inhibits apoptosis

What is the molecular pathogenesis of leiomyomas?

First, **normal myocytes transform into abnormal myocytes** either via somatic mutation or in response to injury

Second, the abnormal myocytes **grow via clonal expansion** into clinically significant tumors

Molecular alterations that lead to **increased or abnormal vasculature** are also involved in fibroid formation

What is degeneration of a leiomyoma and what types can occur?

Various histologic and gross changes occur in leiomyomas when they outgrow their blood supply. The types are as follows:

Red degeneration—hemorrhagic changes that occur as a result of rapid growth. Can cause acute pain, low-grade fever, and an elevation in white blood cell (WBC) count; most common during pregnancy

Hyaline degeneration—mildest form of degeneration: hyalization of the smooth muscle of the leiomyoma represented by loss of the whorled pattern and an overall homogenous appearance

When do fibroids need to be treated?

Calcification—occurs especially in inactive smooth muscle elements after menopause

Most women **do not need surgical or medical treatment**. Expectant management with annual pelvic examinations to monitor growth is warranted. Intervention is indicated with worsening symptom or with a rapidly enlarging uterus

What are the treatment options for symptomatic leiomyomas?

Progesterin supplementation
Prostaglandin synthetase inhibitors—reduce bleeding

Other NSAIDs

GnRH analogs → inhibit estrogen which reduces the size of the leiomyoma

Myomectomy → indicated in symptomatic women desiring future fertility

Hysterectomy → indicated in symptomatic women not desiring future fertility

Embolization of the uterine arteries → indicated in symptomatic premenopausal women not desiring fertility and who want to preserve their uterus and avoid surgical intervention

What are the risks of myomectomy?

Intraoperative blood loss;
postoperative hemorrhage;
postsurgical adhesions; risk of recurrence. These risks are higher with myomectomy than with hysterectomy

What types of complications can occur which are secondary to leiomyomas?

Iron-deficiency anemia

Acute blood loss

Hydroureter or hydronephrosis

Can leiomyomas ever metastasize?

Rarely. In those cases, leiomyomas can grow beyond the uterus or even invade intravascularly to spread to the peritoneum or lung

What types of metastatic disease can develop from a leiomyoma?

Intravenous leiomyomatosis
Benign metastasizing leiomyoma
Leiomyomatosis peritonealis disseminate

Who is most likely to develop a malignancy from a leiomyoma?

Postmenopausal women, especially those presenting with rapid tumor growth, vaginal bleeding, and/or pain

What is a leiomyosarcoma?

A **malignant neoplasm of the uterine smooth muscle**. It occurs most commonly in postmenopausal women, is rapidly expanding, and carries a poor prognosis

Benign Ovarian Masses

What is the differential diagnosis of an ovarian mass?

Functional ovarian cysts; dermoid cyst; PCOS; endometriomas; ectopic pregnancy; tubo-ovarian complex; malignancy

Functional Ovarian Cysts

What are functional ovarian cysts?

Benign anatomic variations resulting from irregularities in normal ovarian function

What are the three types of functional ovarian cysts and how do they form?

1. **Follicular cysts:** ovulation fails to occur and the remaining fluid does not become reabsorbed but gets accumulated into a cystic structure
2. **Corpus luteum cysts:** the corpus luteum formed after ovulation persists and grows larger than 3 cm
3. **Theca lutein cysts:** most frequently occur iatrogenically following ovulation induction or in young girls with hypothyroidism. Also may be seen with high levels of hCG (e.g., in patients with hydatidiform mole or choriocarcinoma)

What are the typical symptoms (if any) of each of the following types of ovarian cysts?

Follicular

Typically asymptomatic; can cause midcycle pelvic pain, dyspareunia, and abnormal uterine bleeding

Corpus luteum

Localized tenderness, amenorrhea, and delayed menstruation (often confused with ectopic pregnancy)

Theca lutein

Pelvic heaviness/aching, hyperemesis, and breast paresthesias

What is the appearance of each of the following types of cyst?

Follicular

Smooth, thin walled, unilocular

Corpus luteum

More complex, usually yellowish-orange lining consisting of luteinized granulosa and theca cells

Theca lutein

Usually bilateral, multicystic, filled with clear, straw-colored fluid. All are usually <10 cm

What is the diagnostic workup for a patient with a suspected functional ovarian cyst?

Pelvic exam; ultrasound; repeat examination and sonography at 6–8 weeks; laparoscopy if symptomatic, concern for torsion or persistence after 3–6 months

What findings raise your suspicion that a cyst may be neoplastic?

- Patient is prepubescent or postmenopausal
- Patient has history of another malignancy (especially breast or gastric)
- Patient has ascites
- Ultrasound findings are significant for: large size, loculations, septa, papillae, or increased blood flow
- Continued presence of cyst at 3–6 months follow-up

How are asymptomatic functional ovarian cysts treated?

Expectant management with analgesics as needed (usually resolve within weeks); OCPs are often used—they do not promote faster resolution of the cyst, but reduce the risk of future cyst development

When do functional ovarian cysts need to be treated?

When there is severe pain or when there is suspicion of malignancy, rupture, or torsion

Benign Ovarian Neoplasms

How are ovarian neoplasms categorized?

Epithelial; germ cell; stromal cell

How are benign ovarian neoplasms treated?

Laparoscopy with unilateral cystectomy or oophorectomy (if the patient wishes to preserve fertility). Conversion to laparotomy and staging if malignancy is found

Benign Fallopian Tube Masses

What are the types of benign fallopian tube neoplasms?

Paraovarian cysts; paratubal cysts

What is another name for paratubal cysts? Describe them.

Hydatid cysts of Morgagni. They are located near the fimbriated end of the tube, filled with clear fluid, and ~1 cm in diameter

How do these masses usually present?

Usually asymptomatic and the diagnosis is usually made as an incidental finding in the OR

Sexual Dysfunction

Describe what happens during the four phases of sexual response described by Masters and Johnson

1. **Excitement: internal or external stimuli** → activation of the central nervous system (CNS) → deep breathing, increase in heart rate, blood pressure, and sexual tension; generalized vasocongestion → skin flush, breast engorgement, nipple erection, engorgement of labia and clitoris, vaginal transudation, and uterine tenting
2. **Plateau: marked degree of vasocongestion throughout the body** → further engorgement of the labia, lower third of vagina, breast, and areolae. Secretion from

What is the biopsychosocial model of female sexual response?

- the Bartholin glands, retraction of the clitoris, vagina lengthens with dilation of the upper two-thirds, muscle tension begins to build up
- 3. Orgasm:** release of sexual tension, generalized myotonic contractions, perivaginal muscles and anal sphincter contract at precise intervals, vaginal and uterine contractions
 - 4. Resolution:** a gradual diminution of sexual tension and response

The biopsychosocial nature of female sexual response is influenced by the dynamic interaction of four components: **biologic, psychologic, sociocultural influences,** and **interpersonal relationships.** All of these components must be addressed in order to achieve sexual satisfaction

What are the possible etiologies of sexual dysfunction?

1. Change in vascularity (atherosclerosis, pudendal artery insufficiency affecting vaginal vasocongestion)
2. Neurogenic causes (spinal cord dysfunction or injuries)
3. Depression or anxiety disorders
4. Medications (selective serotonin reuptake inhibitor [SSRI], tricyclic antidepressants, H₂ blocker, and some antihypertensive medication)
5. Psychosocial factors (prior history of sexual abuse, religious or cultural expectation, fear of rejection or intimacy, and distorted body image)
6. Hormonal changes (premature ovarian failure and menopause)

What is the prevalence of sexual dysfunction?

Studies show a range of 10–60%; the average is 43%

What are the types of female sexual dysfunction and what is the main symptom of each?

- 1. Sexual desire disorders:** decreased sexual fantasy and/or desire, sexual aversion
- 2. Sexual arousal disorders:** decreased genital vasocongestion and lubrication

How should the question of sexual dysfunction be addressed?

3. **Orgasmic disorders:** anorgasmia
4. **Sexual pain disorders:** vaginismus, dyspareunia, noncoital sexual pain

The evaluation should involve an interview of the couple and each partner separately. A complete assessment should include past medical, psychological, sexual history and physical examination including gynecologic examination. Each patient should be asked if she has any questions or concerns about her sexual activity. The most important aspect of taking a sexual history is to make the patient feel comfortable

What hormones influence vaginal blood flow?

Estrogen and **testosterone** increase vaginal blood flow; **Progesterone** diminishes vaginal blood flow

What types of therapies are available for the treatment of sexual dysfunction?

1. **Nonpharmacologic therapy:**
Patient education, lifestyle and behavioral changes—should be tried first

2. **Pharmacologic therapy:**
 Hormones: **estrogen**—increases genital blood flow and enhanced lubrication

Testosterone—may improve libido, data nonconclusive

Herbal therapy: (e.g., St. John's wort, ginseng, yohimbine) generally ineffective

L-Arginine: increases nitric oxide (NO) leading to genital vasocongestion; needs further study

Tibolone: used for osteoporosis; has androgenic activity that may improve sexual function

Sildenafil: a vasodilator; data inconclusive on its benefit for women, not FDA approved

Describe what changes occur with aging that affect sexual function

1. **Decreased libido**
2. **Hormonal changes**—estrogen levels gradually drop leading to vaginal atrophy and dryness. Testosterone levels decrease,

	<p>leading to a decrease in arousal and intensity and frequency of orgasm</p> <p>3. Medical issues—increase in medical problems and use of medications that may affect sexual function</p> <p>4. Past experiences—for example, recurrent dyspareunia can lead to introital spasm, which can further impede sexual function</p> <p>5. Relationship issues</p> <p>6. Self-esteem changes</p>
<p>What types of medication or substances can lead to sexual dysfunction?</p>	<p>Alcohol; antihypertensives; illicit drugs; SSRIs</p> <p>Psychotropic</p> <p>Antihistaminic</p>
<p>What are the adverse effects of SSRI use on sexual function?</p>	<p>SSRIs have been reported to reduce libido in women and men, to cause anorgasmia in women, and to increase ejaculation latency in men</p>
<p>What types of changes occur under the following circumstances that may affect female sexual function?</p>	<p>During pregnancy: breast tenderness, mild cervical bleeding during intercourse, and uterine contractions with orgasm</p> <p>Postpartum: fatigue, vaginal dryness, bleeding, vaginal discomfort</p>
<p>What is hypoactive sexual desire disorder (HSDD)?</p>	<p>Recurrent and persistent lack of sexual fantasies or desires or receptivity to sexual activity that causes personal distress</p>
<p>How should HSDD be evaluated?</p>	<p>Take a careful history including medications, medical illness, depression, substance abuse, and stress. Thyroid test and prolactin levels may be indicated if there is any suggestion of hyperprolactinemia. Androgen levels are not useful in the majority of cases</p>
<p>How should HSDD be treated?</p>	<p>Physiologic causes should be assessed and managed. Further treatment may require individual therapy or relationship therapy</p>
<p>What is sexual aversion disorder?</p>	<p>It is characterized by a phobia with avoidance of sexual contact and severe anxiety associated with contemplation of sexual activity</p>

What are sexual arousal disorders and how are they treated?

When women experience desire and orgasm, but **lack signs of sexual stimulation**, such as lubrication and genital vasocongestion. Treatment includes masturbation, vaginal lubricants, vibrator to increase stimulation, foreplay, distraction technique to alleviate anxiety, and/or estrogen replacement therapy for postmenopausal women

What is orgasmic dysfunction and how is it treated?

A **persistent delay in or absence of orgasm** after sufficient stimulation and arousal resulting in distress or interpersonal difficulty. Treatment involves orgasm goal directed sexual counseling

What types of orgasmic dysfunction exists?

Primary anorgasmia is found in 5–10% of women and is lifelong

Secondary anorgasmia is often related to relationship problems, medications, medical illness, depression, substance abuse, and self-monitoring/anxiety during arousal

What types of sexual pain disorders exist and what are they?

Vaginismus (recurrent involuntary contraction of the vaginal musculature during vaginal penetration)

Dyspareunia (general pain that occurs before, during, or after intercourse)

What organic disorders must be ruled out when vaginismus is diagnosed?

Endometriosis; PID; partial imperforate hymen; vaginal stenosis

How is vaginismus treated?

Education; relaxation techniques; kegel exercises; progressive vaginal dilatation

What organic disorders must be ruled out when dyspareunia is diagnosed?

Bartholin cysts; vulvitis; vestibulitis; vaginitis; clitoral irritation/hypersensitivity; rigid hymenal ring/introital scar tissue; vaginal atrophy and dryness; pelvic adhesion; fibroid; endometriosis

How is dyspareunia treated?

The treatment depends on the etiology

Benign Conditions of the Vulvavagina

DYSTROPHIES

What are vulvar dystrophies?

A group of disorders characterized by lesions that are **white, intensely pruritic** with or without pain, and may occur with **vulvar epithelial changes**. Lesions should be biopsied to rule out malignancy

What is vulvar lichen sclerosis?

A **benign, progressive, and chronic dermatologic condition** more common in older women and characterized by **intense pruritus and pain**, which may be so severe that it leads to **sleep disturbances**

What are other sequelae of lichen sclerosis?

Painful defecation, anal pruritus, dyspareunia, and dysuria. **They may also develop into invasive squamous cell cancer of the vulva**

How does lichen sclerosis appear on clinical examination?

Thin, white, wrinkled skin often resembling **“parchment paper” or “cigarette paper”** that is localized to the labia minora and/or labia majora. It may extend toward the anus

What is the treatment for lichen sclerosis?

Super potent topical corticosteroids (Clobetasol)

What other additional steps should be considered in management of these patients?

Strong encouragement of vulvar hygiene. Given the risk of malignant vulvar cancer, the skin should be examined yearly and suspicious lesions should be biopsied

What is vulvar lichen planus?

An inflammatory dermatologic condition with unknown etiology that mainly affects the skin and mucous membranes of the oral cavity and genital area

How does lichen planus clinically present on the vulva?	With either violaceous papules , hyperkeratosis, or bright erythematous erosions with a white border or white striae along the margins
How does lichen planus clinically present on the skin and oral mucous membranes?	Skin —eruption of multiple, shiny, polygonal, flat-topped, purple papules with white striae Oral mucous membranes —white plaques
How is lichen planus different from lichen sclerosis?	The vagina is involved 70% of the time in lichen planus
What is vulvovaginal-gingival syndrome?	A variant of lichen planus that involves lesions on the vulva, vestibule, vagina, gingival epithelium, and/or skin. These affected areas may or may not occur concurrently
What is the treatment of lichen planus?	Ultra-potent topical corticosteroids
What is vulvar dermatitis?	Also known as vulvar eczema, it is the most common inflammatory skin disease characterized by intense pruritus and irritation, leading to chronic scratching and eventual changes in the dermis. It can have a familial predisposition (atopic dermatitis) or occur with allergens (contact dermatitis)
What is the end result of constant irritation and scratching in vulvar dermatitis?	Lichen simplex chronicus. It appears as a raised, hyperkeratotic white lesion. Biopsy reveals hyperkeratosis and acanthosis
What is the difference between lichen simplex chronicus, and lichen sclerosis and lichen planus?	Lichen simplex chronicus is a reactive change whereas lichen sclerosis and lichen planus are primary dystrophies
What is the treatment?	Medium-to-high-potency topical steroids
What other vulvar dystrophy has the same histologic appearance as lichen simplex chronicus?	Squamous cell hyperplasia
In what setting does lichen simplex chronicus arise?	In patients who have chronic vulvovaginal infections or other causes of chronic irritation

What is the treatment for squamous cell hyperplasia?	The goal is symptomatic relief. Sitz baths and lubricants are recommended to restore moisture to cells. Medium potency topical steroids are used to decrease inflammation and pruritus
What is the gold standard for diagnosis of any of these dystrophies?	Biopsy of the lesion
How does psoriasis appear on physical examination of the vulva?	Red moist plaques covered by silver scales. Topical corticosteroids are the treatment of choice
What are the most common differential diagnoses among all of the vulvar dystrophies?	Lichen sclerosis, squamous cell hyperplasia, lichen planus, lichen chronicus, vulvar eczema, psoriasis, vulvovaginal candidiasis, vitiligo, desquamative inflammatory vaginitis, pemphigus, Behcet disease

BENIGN CYSTS

What are the most common benign vulvovaginal cysts?	Bartholin ducts cysts, epidermal, sebaceous, and apocrine sweat gland cysts
Where are Bartholin glands and ducts located?	Deep in the labia majora at about four and eight o'clock positions. They are not palpable in healthy women
What is the role of Bartholin glands?	As a homologue to the male bulbourethral glands, they secrete mucus to provide moisture for the vagina
How do Bartholin gland cysts develop?	Infection can cause inflammation and obstruction of the main duct of Bartholin glands leading to cystic dilation. They may enlarge up to 1–3 cm and are usually asymptomatic
What may Bartholin gland cysts develop into?	Bartholin gland abscesses
When symptomatic, what are the acute symptoms and signs of Bartholin gland cysts and abscesses?	Pain, dyspareunia, and difficulty ambulating or sitting. Physical examination may reveal swelling, erythema, edema, and a large fluctuant mass in the medial labia majora. This is usually because of infection or swelling

How are Bartholin gland cysts treated?

Asymptomatic cysts need no intervention or antibiotic treatment

Symptomatic cysts are incised and drained, and **a catheter is placed to form a tract for the drainage of glandular secretions (Word catheter)**.

If this procedure fails, then a marsupialization can be done (the creation of a new ductal orifice). The most definitive procedure for a Bartholin cyst after failure of all previous methods is complete excision of the gland

What microbes are commonly implicated in Bartholin gland abscesses?

Escherichia coli, *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and several anaerobes

How are Bartholin gland abscesses managed and treated?

An aspirate and culture of the abscess should be done. Treatment includes antibiotics such as ceftriaxone and clindamycin, and surgical drainage of the pus (with possible supplementation of a Word catheter)

For the following descriptions, list the most appropriate type of cyst or cyst-related condition

1. Occurring mostly beneath the labia majora, this cyst occurs when the **sebaceous gland duct becomes obstructed**. They are multiple, smooth, and palpable masses that are generally asymptomatic. Acute infection can be treated with incision and drainage
2. These cysts are lined by **squamous epithelial cells and contain oily material**. They occur in the setting of vulva surgery or arise from obstruction of pilosebaceous ducts. They are usually small, solitary, and asymptomatic
3. Apocrine sweat gland cysts are mainly found in the labium majus and become functional after puberty. They are usually small, multiple, and extremely pruritic. This disease occurs when **keratin obstructs the duct**

1. Sebaceous cyst**2. Epidermal cyst****3. Fox-Fordyce disease**

4. This condition occurs with chronic infection of the apocrine glands and is manifested by multiple painful, pruritic, and subcutaneous abscesses. It may be treated with antibiotics or incision
 5. This cystic vulvar tumor is a rare **congenital anomaly** and located near the urethral meatus. It can be treated with partial excision
 6. Occlusion of a **persistent processus vaginalis** may cause this hydrocele or cystic tumor
 7. These lateral vaginal wall cysts result from dilation of the **mesonephric duct remnants**
 8. This is a benign outgrowth of normal skin. More of a solid tumor than a cyst, it must be removed when it causes discomfort
4. **Hidradenomas**
 5. **Skene duct cyst**
 6. **Cyst of the canal of Nuck**
 7. **Gartner duct cyst**
 8. **Acrochordon (skin tag)**

PYODERMAS AND OTHER NONSEXUALLY TRANSMITTED INFECTIONS OF THE VULVA AND VAGINA

- What are the specific types of pyodermas?** Cellulitis, impetigo, folliculitis, furuncles, and carbuncles
- What is the most common bacterial etiology for all of these pyodermas?** *Staphylococcus aureus*
- How does impetigo manifest?** Vesicles and pustules form in an area that was recently traumatized. They often rupture and form a characteristic golden crusting
- How is it treated?** Erythromycin or dicloxacillin (oral)
- What is folliculitis? How does it manifest?** An infection of the hair follicles. It can occur with exposure to whirlpools/hot tubs, antibiotic therapy, and shaving of pubic hair. The lesions are multiple, "5 mm, cluster in groups, erythematous, and pruritic
- What is the recommended treatment for folliculitis?** Warm saline compresses and topical antibiotics
- What other pyodermas can occur after an episode of folliculitis? Describe them.** Furuncles: a painful nodular lesion that involves the hair follicle and drains pus

What is the preferred treatment for furuncles and carbuncles?	Carbuncles: clusters of furuncles or subcutaneous abscesses that drain pus through multiple hair follicle openings
What is erysipelas?	Warm compresses to promote spontaneous drainage in furunculosis. Patients with systemic symptoms of furuncles and/or carbuncles warrant empiric oral antibiotic therapy followed by guided therapy based on cultures and sensitivity
How is erysipelas treated?	Rapidly spreading erythematous lesions of the skin caused by invasion of the superficial lymphatics by β-hemolytic streptococci . It usually occurs after trauma or a surgical procedure to the vulva. Pustules, vesicles, and bullae may appear
What is hidradenitis suppurativa? How is it treated?	Oral penicillin or tetracycline
What is the agent that commonly causes nocturnal perineal itching in children? How is it diagnosed and what is the treatment?	A more severe condition of hidradenomas that occurs when the cysts develop into abscesses and rupture. Draining sinus tracts develop deep within the skin, and scars, fibrosis, hyperpigmentation, and pitting can be seen over the vulva. It is treated by drainage and antibiotic therapy
	<i>Enterobius vermicularis</i> (pinworm). Apply adhesive tape to the perineum and look for ova under the microscope for diagnosis. Mebendazole is the treatment of choice

OTHER VULVAR DISORDERS

Trauma to the vulva can occur in both children and women. Straddle-type injuries are the most common cause of vulvar hematomas in children whereas trauma incurred during vaginal delivery is the main etiology in women of reproductive age.

What vessels are most commonly involved in vulvar hematomas?

Branches from the pudendal artery

What are the common symptoms?

Severe perineal pain within the first 24 hours is usually the first symptom of a rapidly expanding vulvar hematoma. Rapid appearance of a tense, palpable, fluctuant, and sensitive tumor of varying size covered by discolored skin readily gives a diagnosis of vulvar hematoma

How are vulvar hematomas managed?

The two primary modalities are

1. Conservative management with analgesia and ice packs
2. Surgical drainage is warranted for large hematomas (>3 cm)

Gynecologic Oncology

Cervical Cancer

INTRODUCTION

Describe the three histologic regions of the cervix

1. **Ectocervix:** the inferior portion of the cervix that is continuous with the vagina; covered with **squamous epithelium**
2. **Squamocolumnar junction (SCJ):** separates two regions of the cervix (often with overlap)
3. **Endocervix:** the superior portion of the cervix that begins at the external os and continues to the endocervical canal; covered with mucin-secreting **columnar epithelium** and continues into the cuboidal epithelium of the endometrium

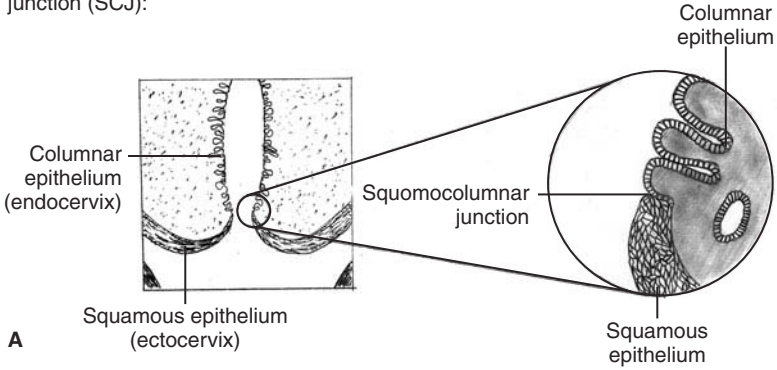
Describe the location of the SCJ

It varies between the ectocervix, the cervical canal, and the vaginal fornices. Throughout a woman's life, the SCJ **migrates internally** toward the endocervix via the metaplasia

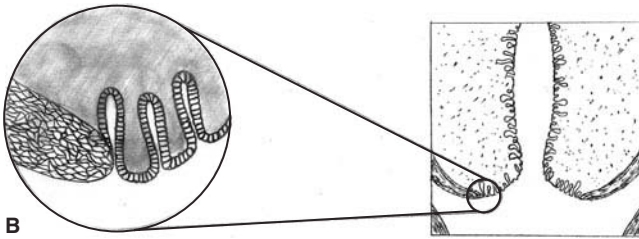
What is the transformation zone?

The 1–3 cm area of **squamous metaplasia** that separates the endocervix from the ectocervix created from the internal migration of the SCJ. It is the area **most susceptible to the development of cervical neoplasia** because of the metaplastic changes.

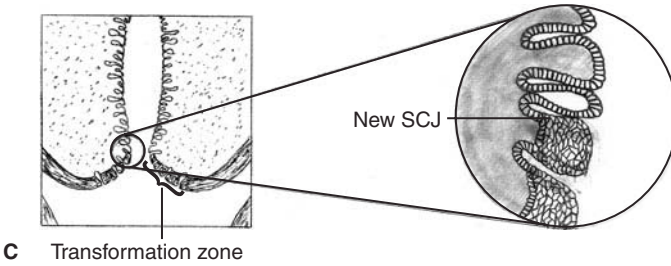
Original squamocolumnar junction (SCJ):



Outward growth of the SCJ:



Squamous metaplasia:



Inward growth of the SCJ:

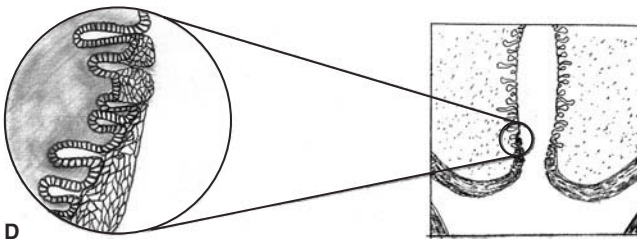


Figure 5-1 The cervical transformation zone.

What is the incidence of cervical cancer?

It is the second leading cause of malignancy in women worldwide but comprises only 1% of all cancer deaths of women in the United States

Who does cervical cancer affect?

Sexually active women, usually in a bimodal age distribution with peaks in the late 30s and in the early 60s

What are the risk factors for cervical cancer?

HPV infection

Sexual history: Early onset of sexual activity; multiple sexual partners; history of sexually transmitted infections (STIs); sex with high-risk partners

Smoking

High parity

Immunosuppression

Low socioeconomic status

Pelvic radiation

Prior history of vulvar or vaginal squamous dysplasia

What is the epidemiologic relationship between HPV infection and cervical cancer?

Human papillomavirus (HPV) is found in nearly all cases of squamous cell cervical cancer and is thought to **contribute to the pathogenesis of dysplasia**. While most HPV infections self-resolve, some progress to genital warts or cervical dysplasia. HPV infection is therefore deemed **necessary but not sufficient** for the development of cervical cancer

How does HPV infection lead to cervical cancer?

HPV initially infects the basal layer cervical cells, forming **koilocytes** within the cells. HPV can then **integrate into the cells genome**, altering the expression of the cell's regulatory genes. This leads to intraepithelial neoplasia and/or cancer

What serotypes of HPV most commonly cause cervical cancer?

HPV 16, 18, 31, 33, and 45

In what other types of neoplasia is HPV thought to be a causative agent?

1. **Vaginal intraepithelial neoplasia (VAIN) and vaginal malignancies**
2. **Vulvar intraepithelial neoplasia (VIN) and vulvar malignancies**
3. **Penile neoplasia**

What are the types of cervical cancer, how common are each worldwide, and where in relation to the SCJ do they occur?

1. **Squamous cell carcinomas:** 80–90% of all cervical cancers, occur below the SCJ
2. **Adenocarcinomas:** 15% of all cervical cancers, occur above the SCJ
3. **Adenosquamous carcinomas:** 3–5% of all cervical cancers
4. Neuroendocrine, small cell carcinomas, clear cell carcinomas, melanomas, lymphomas, and sarcomas can all originate in the cervix but are rare

What are the signs/symptoms of cervical cancer?

Early stages of cervical dysplasia and cancer are asymptomatic. There is **no classic presentation for cervical cancer** but the most common symptom is **abnormal vaginal bleeding** (either postcoital, postmenopausal, or intermenstrual)

Symptoms of late stage disease include: **vaginal discharge** pain (usually pelvic or lower back pain), weight loss, hematuria (vaginal passage of blood in urine) or hematochezia (passage of blood in stool)

Signs on cervical examination can range from a normal gross appearance with aberrant cytology to a cervix entirely replaced with tumor

What is the differential diagnosis of these symptoms?

Cervicitis, vaginitis, STI

How is the definitive diagnosis of cervical cancer made?

Cervical biopsy if the lesion is grossly visible. If the lesion is only diagnosable microscopically, a **colposcopy-directed biopsy** or diagnostic **conization** (for microinvasive disease) are modalities for diagnosis

Upon diagnosis, what other tests need to be done?

1. A comprehensive physical examination to evaluate metastases, including cervical and vaginal inspection, a rectovaginal examination, and palpation of the liver and lymph nodes (inguinal and supraclavicular)
2. Lab and imaging tests to evaluate for metastases

What do each of the following contribute to the evaluation of cervical cancer?

Chest x-ray:

Identifies lung metastasis

Intravenous pyelogram:

Identifies bladder involvement, but has been largely replaced by CT, MRI, or cystoscopy

MRI, CT, or lymphangiography:

Identifies nodal involvement, tumor size, and abdominal/retroperitoneal spread; CT and MRI cannot be used for staging purposes

Renal function tests:

Identifies urinary tract metastasis

Liver function tests:

Identifies liver metastasis

Barium enema:

Identifies colonic metastasis

How and to where does cervical cancer spread?

1. **Direct extension** to contiguous structures
2. **Lymphatic dissemination** to any of the pelvic lymph node groups
3. **Hematogenous dissemination** most commonly to the **lungs, liver, and bone** although it can spread to the large intestine, adrenals, spleen, or brain as well
4. **Intraperitoneal implantation**

What are the stages of cervical cancer?

The **FIGO system** is based on the histological assessment, the physical examination, and the laboratory results. See Table 5-1.

What are the overall survival rates for cervical cancer?

The survival rates **depend most significantly on the stage of disease** at diagnosis. The 5-year survival rates for each stage are:

Stage I: over 90%

Stage II: 75%

Stage III: 40%

Stage IV: under 15%

What is the differential diagnosis of a cervical mass?

Nabothian cysts

Glandular hyperplasia

Mesonephric remnants

Reactive glandular changes

Endometriosis

Table 5-1 The Stages of Cervical Cancer

I	Carcinoma confined to the cervix
IA	Invasive carcinoma diagnosable only by microscopy
IA1	Minimal microscopic invasion <3 mm deep and <7 mm horizontally
IA2	Microscopic lesions <5 mm deep and <7 mm horizontally
IB	Clinically visible lesions that are confined to the cervix
II	Carcinoma extends beyond the cervix, but not to the pelvic wall or lower third of the vagina
IIA	No parametrial involvement
IIB	Parametrial involvement
III	Carcinoma extends to the pelvic wall and/or lower third of the vagina. Also includes all cases with hydronephrosis
IIIA	No extension to pelvic wall, but involvement of lower third of vagina
IIIB	Extension to pelvic wall or hydronephrosis
IV	Carcinoma extends beyond the true pelvis or clinically involves the mucosa of the bladder or rectum
IVA	Spread to adjacent pelvic organs
IVB	Spread to distant organs

CERVICAL CANCER SCREENING AND PREVENTION

What is a Pap smear and how is it done?

A cytological examination of the cervix. A small brush scrapes cells from the endocervix and ectocervix. These cells are either spread on a microscopic slide and fixed or placed in a liquid medium for evaluation

What is the difference between a Pap smear and the thin-layer liquid-based cytology (ThinPrep, SurePath)?

In a **traditional Pap smear**, a spatula or brush is used to collect cells on the ectocervix and then from the endocervix. The specimen is rolled or smeared onto a slide and rapidly fixed. A single slide can be used to examine both ectocervical and endocervical cells

Liquid-based cytology involves taking cells from the ectocervix and endocervix and placing the specimens in vials containing preservative solutions. The vials are placed in a ThinPrep processor machine and ultimately, the cells are transferred to a slide. This technique results in a monolayer of cells on the slide,

How often should a Pap smear be done?

which can be read more quickly than conventional cytology slides

The general consensus to date is an **annual examination beginning at coitus or by the age of 21**. If a female is low-risk and has three negative Pap smears, she can then get repeat evaluations every 3 years

What are some of the limitations of Pap smear screening?

Not all patients have access (the most important limiting factor); it is less successful in diagnosing adenocarcinoma; it has a low sensitivity and therefore a high false-negative rate

Describe the histologic definition of cervical intraepithelial neoplasia (CIN) and of each of the stages

CIN refers to precancerous pathology that can slowly progress to cervical cancer.

CIN I: formerly known as mild dysplasia, is a **cellular dysplasia confined to the basal one-third of the epithelium**

CIN II: refers to **lesions confined to the basal two-thirds of the epithelium** that used to be referred to as moderate dysplasia

CIN III: formerly known as severe dysplasia and carcinoma in situ (CIS), is a **cellular dysplasia that affects more than two-thirds of the epithelium**

Describe the cytological definitions of the Bethesda system?

Low-grade squamous intraepithelial lesion (**LSIL** or **LGSIL**): includes CIN I and condylomatous atypia

High-grade squamous intraepithelial lesion (**HSIL** or **HGSIL**): includes CIS, CIN II, and CIN III

Atypical squamous cells of undetermined significance (**ASCUS**)

Atypical squamous cells that cannot exclude HSIL (**ASCH**). See Table 5-2.

How rapidly does cervical dysplasia progress to cervical cancer?

Cervical cancer in general progresses slowly and mild dysplasia (CIN I or LSIL) often spontaneously regresses

It is estimated that it takes 3–10 years for women with CIN III to progress to cervical cancer

Table 5-2 The Bethesda System

Bethesda System	Dysplasia/CIN System
Atypical squamous cells of undetermined significance	Squamous atypia
Low-grade squamous intraepithelial lesion	HPV atypia
High-grade squamous intraepithelial lesion	CIN I
	CIN II
	CIN III

What is colposcopy?

A binocular stereomicroscope that **magnifies the cervix** that is used to visualize changes consistent with dysplasia, allowing for directed biopsy

What must be seen in order to consider colposcopy satisfactory?

The entirety of the SCJ and the entire lesion in question

What findings on colposcopy are suggestive of cervical cancer?

Abnormal blood vessels; abnormal appearing surface of the cervix; color change

What is endocervical curettage?

A sampling procedure of the endocervix used to retrieve cells further inside the cervical canal that can be visualized with colposcopy

What should be done if the ECC is positive?

Because there is no tissue orientation, **a positive ECC warrants conization**

What is cervical conization?

A cone-shaped biopsy of the entire SCJ that allows diagnosis via histologic criteria

How is cervical conization done?

Cold knife conization (via scalpel); **Loop electrosurgical excision procedure (LEEP)** (via heated wire)

What are the four common indications for conization in order of their frequency?

1. CIN III
2. Unsatisfactory colposcopy
3. Positive endocervical curettage
4. A discrepancy between Pap smear and biopsy results

What are the risks of cervical conization?

Infection, blood loss, risks from anesthesia, cervical stenosis, and cervical incompetence

What is the appropriate workup of each of the following abnormal Paps?

LSIL:

Colposcopy with local ablation or repeat Pap smears to rule out a high-grade lesion. If \leq CIN I, the patient can be just followed-up over time. Conization can also be used, both diagnostically and therapeutically, but it is not necessary

HSIL:

Warrants a **colposcopy followed by direct ablation or excision**

ASCUS:

HPV DNA testing. If high-risk HPV+, colposcopy should be done. If high-risk HPV-, then a repeat Pap smear can be done in 12 months. Alternatively, a repeat Pap smear can be done every 3–4 months until there are three consecutive negative smears

Menopausal women may have an atrophic component leading to this cytology, and they can be given **intravaginal estrogen** and followed up every 3–4 months

Women with an infection should be reexamined once their infection is treated

Immunosuppressed women with ASCUS need colposcopy

Glandular cell abnormalities:

All glandular cell abnormalities warrant **colposcopy and endocervical sampling**

How often should patients be followed up after treatment of noninvasive abnormalities?

Because of the greater risk of recurrence, patients need to be followed **every 3–6 months for 2 years after ablation or excision**

What types of vaccination is now available to prevent cervical cancer?

Gardasil, a quadravalent vaccine protecting against HPV 6, 11, 16, and 18

Who should get this vaccination?

Females ages 9–26. Whether males should also be vaccinated is controversial and it is not FDA approved for males

Do vaccinated individuals still need to get Pap smears?

Yes! The vaccine only protects against four of the many HPV serotypes that cause cervical cancer

SQUAMOUS CELL CARCINOMA OF THE CERVIX

Who gets squamous cell carcinoma (SCC) of the cervix?

There is a bimodal age distribution with peaks between 35 and 40 and between 60 and 65

What are the five prognostic indicators for SCC?

1. **Stage of disease** (most important)
2. Lymph node involvement
3. Tumor size
4. Depth of stromal invasion
5. Invasion of lymphovascular space

What are the treatment options for invasive SCC of the cervix and what are the indications of each?

1. **Cervical conization:** used for women with stage Ia1 with no involvement of the lymphovascular space who wish to preserve fertility
2. **Radical trachelectomy:** involves surgical removal of the cervix and parametria and placement of a cerclage, and can be combined with a laparoscopic or open diagnostic/therapeutic lymphadenectomy with para-aortic lymph node sampling; used for women with stage Ia1 with involvement of the lymphovascular space, stage Ia2 or Ib1 disease who wish to preserve fertility
3. **Radical hysterectomy:** used for premenopausal women with early stage (up to stage IIa) cervical cancer who wish to preserve ovarian function
4. **Chemoradiotherapy alone:** used for women with stage Ia, Ib, or IIa cancer who are poor surgical candidates or who decline a surgical approach
Chemoradiotherapy versus surgery for Ib2 is controversial
5. **Chemoradiotherapy and hysterectomy:** women with advanced disease. Can be done via:
 - a. Chemoradiotherapy followed by hysterectomy
 - b. Neoadjuvant chemotherapy followed by hysterectomy and, if indicated, radiation

c. Hysterectomy followed by chemoradiotherapy

*This is rarely an ideal choice as the morbidity is increased when this combination is used.

Patients with locally advanced disease should be treated with primary chemoradiotherapy. Women with PID, another pelvic mass, or anatomic alterations should be treated with primary hysterectomy.

What is the recommended treatment for each of the following stages of cervical cancer?

Ia1: Conization or simple hysterectomy

Ia2–early IIb: Radical hysterectomy with a pelvic lymphadenectomy or chemoradiotherapy

Late IIb–IV: Chemoradiotherapy or combination chemotherapy

What are the indications for adjuvant radiotherapy without chemotherapy?

Patients with any two of the following risk factors should be considered candidates for adjuvant radiotherapy without chemotherapy: large tumor size; deep stromal invasion; lymphovascular space invasion

What are the indications for adjuvant chemoradiotherapy?

If a patient has any of the following:

Positive resection margins

Positive lymph nodes

Parametrial involvement

What types of chemotherapy are used for cervical cancer treatment and how do they work?

Cisplatin with or without **5-FU**.

Cisplatin is a cycle-nonspecific alkylating agent that cross-links DNA. 5-FU is a cycle-specific DNA synthesis inhibitor

What are the main side effects of the following?

Cisplatin:

Leukopenia, ototoxicity, nephrotoxicity, and peripheral neuropathy

5-FU:

Fatigue, diarrhea, nausea, vomiting, and myelosuppression

The two main methods of radiation delivery for cervical cancer are external photon beam radiation therapy (RT) and intracavitary brachytherapy. When is each indicated?

Intracavitary brachytherapy alone is adequate treatment for stage Ia1 disease, external beam RT is generally added to brachytherapy to improve pelvic control with more advanced

	disease, such as stage Ib and IIa disease
What are the side effects of radiation?	Acute side effects: nausea, diarrhea, and skin damage Long-term complications: cystitis, proctitis , vaginal foreshortening, stenosis, and dryness (which leads to sexual dysfunction), development of fistulae, small bowel obstruction
What are the complications of surgery?	Postoperative complications: hemorrhage, fever, sepsis, pulmonary embolus, and infection Long-term complications: bladder dysfunction, strictures of the ureter, lymphocyst formation, and fistula formation
What type of surveillance is indicated in posttreatment for SCC?	Patients need to be evaluated every 3 months for the first 2 years after treatment. After these 2 years, they must be seen every 4 months in the third year, every 6 months for 5 years, and annually thereafter. These evaluations must include careful attention to the supraclavicular and inguinal lymph nodes, a rectovaginal examination, and an abdominal examination. A Pap smear must be done at each visit
What is the treatment for recurrence?	Depends on patient and presentation, however in general, patients who were initially surgically treated may receive radiotherapy. Patients initially treated with radiotherapy may be surgically treated if their recurrence is localized or they may receive chemotherapy
What are the 5-year survival rates for the following stages of SCC?	
Stage I:	85–90%
Stage II:	60–80%
Stage III:	30%
Stage IV:	10%

ADENOCARCINOMA OF THE CERVIX

<p>What are the types of adenocarcinoma?</p>	<p>Mucinous; endometrioid; clear cell; serous</p>
<p>What is the incidence of cervical adenocarcinoma?</p>	<p>Accounts for 15% of cervical cancers, however this rate has been rising</p>
<p>What are the risk factors for the development of cervical adenocarcinoma?</p>	<p>The same risk factors as for SCC</p>
<p>How is cervical adenocarcinoma classified?</p>	<p>Using the Bethesda 2001 system which divides it into four subgroups:</p> <ol style="list-style-type: none"> 1. Atypical glandular cells (AGC)—either endocervical, endometrial, or unspecified 2. AGC favoring neoplasia—either endocervical, endometrial, or unspecified 3. Endocervical adenocarcinoma in situ (AIS) 4. Adenocarcinoma <ol style="list-style-type: none"> 1. Improved detection 2. An increase in the prevalence of the pathogenic serotype of HPV (HPV 18) 3. An increase in oral contraceptive use (more associated with adenocarcinoma than SCC)
<p>What are some of the theories for the rise in the diagnosis of AIS?</p>	<p>Improved detection</p> <p>An increase in the prevalence of the pathogenic serotype of HPV (HPV 18)</p> <p>An increase in oral contraceptive use (more associated with adenocarcinoma than SCC)</p>
<p>What is the epidemiologic relationship between AIS and SCC of the cervix?</p>	<p>About 50% of women found to have adenocarcinoma have concomitant SCC or CIN</p>
<p>How is AIS diagnosed?</p>	<p>It is usually asymptomatic and is found through the following:</p> <ol style="list-style-type: none"> 1. Cervical cytology with colposcopy-directed biopsy (not sensitive) <ol style="list-style-type: none"> a. cold knife conization—best method b. electro-excision—may obscure margins 2. Endocervical curettage (improves the detection rate if used with the above methods)
<p>What is the microscopic appearance of cervical adenocarcinoma?</p>	<p>Endocervical glands crowded together in a cribriform pattern, lined by atypical columnar epithelial cells. Multifocal disease is often found</p>

What is the gross appearance of cervical adenocarcinomas?

Half are exophytic, some are ulcerative, and some have the lesion within the endocervical canal (and so it is not visible)

What are the treatment options for microinvasive disease/carcinoma in situ?

There is not a consensus; however the following options are acceptable:

1. **Simple hysterectomy** (generally recommended if fertility need not be preserved)
2. Radical hysterectomy
3. Conization (however, margins have poor predictive value for residual disease)
4. Radical trachelectomy

How should a patient be followed after the diagnosis of AIS if she does not have a hysterectomy?

With cervical cytology and endocervical sampling, every 6 months indefinitely

What are the treatment options for adenocarcinoma?

Similar to that of SCC. However, adenocarcinomas tend to be more bulky and therefore more often warrant radiotherapy, even with earlier stage disease

What are the prognostic indicators for adenocarcinoma?

Prognosis primarily depends on **stage**. However, in general, adenocarcinoma is more aggressive than SCC and so survival rates are slightly lower

What are the 5-year survival rates for the following stages of adenocarcinoma?

Stage I:	70–75%
Stage II:	30–40%
Stage III:	20–30%
Stage IV:	<15%

Vulvar and Vaginal Cancer

VULVAR CANCER OVERVIEW

What are the designations of preinvasive vulvar malignancy?

Vulvar intraepithelial neoplasia, classified into three levels:

VIN-I: mild dysplasia

VIN-II: moderate dysplasia

How do each of the preinvasive lesions typically present?	<p>VIN-III: severe dysplasia or carcinoma in situ</p> <p>VIN-I and VIN-II present most commonly with itching, chronic irritation, and development of a palpable lesion. The lesion typically appears as localized, isolated, raised, whitish areas found most commonly along the posterior vulva and perineal body. Perineal pain and dysuria can also be presenting symptoms. VIN-III typically presents with intractable itching and irritation along with the gross lesion(s) described above. Occasionally, the vulvar involvement is extensive</p>
How is VIN treated?	<p>Complete excision. Early cases with limited involvement can be treated with local excision, cryocautery, electrodesiccation, or laser ablation. VIN-III is treated with wide local excision with/without laser ablation</p>
What is the incidence of vulvar cancer?	<p>Vulvar cancer is the fourth most common gynecologic cancer that affects almost 4000 women in the United States annually</p>
Whom does vulvar cancer affect?	<p>Typically, postmenopausal women, usually around 65 years of age</p>
What are the risk factors for vulvar cancer?	<p>Infection with certain types of HPV history of cervical cancer Immunocompromise; northern European ancestry Cigarette smoking; diabetes Vulvar dystrophy; obesity Vulvar intraepithelial neoplasia; hypertension</p>
How is the diagnosis of vulvar cancer made?	<p>Careful history-taking and inspection are essential. Inspection must include all of the vulvar skin, peri-anal skin, cervix, and vagina. A biopsy of each lesion leads to a definitive diagnosis as gross appearance is often inconsistent with the underlying cellular morphology. If there is no obvious lesion, colposcopy may be</p>

What other test should be ordered after biopsy?

used. Also, the skin can be washed with a dilute **acetic acid solution** to accentuate any lesions or aberrant vascular patterns

What is the differential diagnosis of a suspected vulvar cancer?

A synchronous secondary malignancy is found in over 20% of patients with vulvar cancer and so screenings for a secondary malignancy as well as for metastasis should be done

Screenings may include: **CXR; IVP; LFTs; renal function tests; CT scan.**

If the lesion is located near the anus then a **barium enema** is warranted and if the lesion is located near the urethra then **cystoscopy** is warranted

What are the types of vulvar cancer?

Epidermal inclusion cysts; seborrheic keratoses

Lentigo condyloma acuminata

Bartholin gland disorders; lichen sclerosus

Acrochordons; hidradenomas

Over 90% of vulvar cancers are primary cancers.

Types of primary vulvar cancers include: **squamous cell carcinomas (90%); melanomas (5%); Bartholin's gland carcinomas; basal cell carcinomas; sarcomas; lymphomas; endodermal sinus tumors**

Metastasizing cancers to the vulva include **cervical, endometrial, renal, and urethral cancers**

SQUAMOUS CELL VULVAR CANCER

What are the signs/symptoms of squamous cell vulvar cancer?

Itching, burning, irritation, and/or the development of a **palpable lesion** (plaque, ulcer, or mass) usually on the labia majora. Bleeding, ulceration, abnormal discharge, dysuria, or an enlarged groin lymph node are usually associated with more advanced disease

What are the two subtypes of squamous cell carcinoma of the vulva?

1. **Keratinizing type**—occurs in older women; associated with other vulvar dystrophies such as lichen sclerosis but not with HPV; usually unifocal
2. **Warty or basaloid type**—found in younger women; associated with HPV, VIN, and cigarette smoking; usually multifocal

How does squamous cell vulvar cancer spread?

1. **Direct extension:** to the vagina, urethra, or anus
2. **Lymphatic dissemination:** occurs via the superficial inguinal nodes into the deep inguinal and femoral nodes and finally into the pelvic lymphatics
3. **Hematogenous dissemination:** rare; occurs mostly in patients who also have lymphatic dissemination; spreads to lung, liver, and bone

How common is lymphatic spread and what factors predict whether it occurs?

Lymphatic spread **occurs in 30%** of vulvar cancers and its likelihood increases with the size of the lesion and the depth of invasion

What are the stages of vulvar cancer?

The FIGO system is used to stage vulvar cancer and is based on both the microscopic evaluation of the removed tumor as well as of the regional lymph nodes. See Table 5-3.

How is squamous cell vulvar cancer treated?

In general, treatment needs to be **individualized** using the most conservative treatment that will lead to cure of the disease. The possible modalities are listed below:

1. **Groin node dissection** with either **wide local excision** or **radical vulvectomy** is the mainstay of treatment
2. **Adjuvant radiation therapy** can be added postoperative in patients with either positive inguinal nodes or close/positive surgical margins or preoperatively in patients with advanced disease

Table 5-3 The Stages of Vulvar Cancer

Stage 0	Tis	Carcinoma in situ; intraepithelial carcinoma
Stage I	T1 N0 M0	Tumor confined to the vulva and/or perineum; <2 cm in diameter; no palpable nodes
Stage II	T2 N0 M0	Tumor confined to the vulva and/or perineum; >2 cm in diameter; no palpable nodes
Stage III	T3 N0 M0 T3 N1 M0 T1 N1 M0 T2 N1 M0	Tumor of any size with: (1) adjacent spread to the lower urethra and/or the vagina, or the anus; and/or (2) unilateral regional lymph node metastasis
Stage IVA	T1 N2 M0 T2 N2 M0 T3 N2 M0 T4 N-any M0	Tumor invades any of the following: upper urethra, bladder mucosa, rectal mucosa, pelvic and/or bilateral regional lymph node metastasis
Stage IVB	T-any N-any M1	Any distant metastasis including pelvic lymph nodes

- Chemoradiotherapy** can be used for advanced stage disease where surgery is associated with high morbidity and mortality
- Adjunctive chemotherapy alone is only of limited value

When is node dissection warranted and how is it done?

All patients with more than 1 mm of stromal invasion require an inguinal femoral lymphadenectomy. Unilateral lesions only need node dissection on the ipsilateral side if these nodes are negative. Midline or bilateral lesions warrant bilateral node dissection. Sentinel node biopsy is currently under investigation as a method to evaluate degree of treatment

What is the difference between the following:

Wide local excision:

Surgical excision of the lesion down to the layer of underlying fascia that must include at least 1 cm negative surgical margins

Radical vulvectomy:

Excision of both the labia majora and the mons pubis down to the layer of underlying fascia

What are the complications of radical vulvectomy and groin dissection?

1. **Wound infection, necrosis, and breakdown** (occurs in 40–50% of cases)
2. **Chronic leg edema** (occurs in 30% of cases)
3. **Depression**, poor body image, sexual dysfunction
4. Less commonly: UTIs, deep vein thrombosis (DVT), PE, MI, seromas, hemorrhage, femoral nerve injury, chronic cellulitis, femoral hernias, and fistula development

What are the most important predictors of recurrence and survival?

Lymph node involvement (most important predictor); **lesion size**

What is the treatment for recurrent disease?

Further surgical resection or radiotherapy. Metastatic recurrences are treated with chemotherapy (usually cisplatin, cyclophosphamide, methotrexate, bleomycin, or mitomycin C)

What type of surveillance is indicated posttreatment for vulvar cancer?

Biannual gynecologic evaluations. They must include a thorough inspection as well as palpation of the vulva and inguinal lymph nodes

What are the survival rates for squamous cell vulvar cancer?

The average 5-year survival rate for all stages is around 70%. If there is no lymphatic spread, survival rates reach up to 90%, but if spread has extended to the deep pelvic nodes, 5-year survival rate drops below 20%

What is a verrucous carcinoma and how does it present?

A type of squamous cell cancer that grossly has a cauliflower-like appearance

Who develops verrucous carcinoma?

Usually postmenopausal women; it is associated with HPV infection

How is verrucous carcinoma treated?

Radical local excision

What is the prognosis for verrucous carcinoma?

Good—it grows slowly, does not metastasize often. However it can be locally destructive

Vulvar Melanoma

How does vulvar melanoma present?

Typically in postmenopausal Caucasian women as a **raised pigmented lesion**, usually on the labia minora or clitoris. It is often asymptomatic, however it can present with **itchiness, irritation, or bleeding**

What is the etiology of a vulvar melanoma?

Melanomas can arise either de novo or from a prior nevus. They are rare (2–10% of vulvar cancers)

How is vulvar melanoma staged?

Using the **Breslow** criteria. Because of the direct association of survival with depth, this system measures the thickness of the lesion from the surface to the deepest point of invasion. The Clark and Chung staging system are also used

How is vulvar melanoma treated?

Local excision with wide margins
Groin lymph node dissection is needed for lesions with more than 1 mm invasion

Sentinal lymph node sampling may also be warranted in women with central primary lesions

Chemotherapy and radiotherapy do not have good results

What are survival rates for vulvar melanoma?

Survival is near 100% if the melanoma has not invaded more than 1 mm, but rates decline with increasing dermal depth

Bartholin Gland Carcinoma

What is Bartholin gland carcinoma?

An uncommon type of **adenocarcinoma** that arises mainly from either the squamous epithelium of the Bartholin's ducts/glands

How does a Bartholin gland carcinoma present?

It typically presents either as an **asymptomatic vulvar mass** or as **perineal pain**, usually in women in their 50s. Because of this, any Bartholin gland enlargement in postmenopausal women must be biopsied

What are the criteria required to diagnose a vulvar tumor as a Bartholin gland carcinoma?

1. Tumor in the posterior vulva
2. Tumor is deep in the labia majora
3. The overlying skin is inflected
4. There is some recognizable normal gland present

How is Bartholin gland carcinoma treated?

Because metastasis is common, treatment usually involves a **radical vulvectomy, bilateral lymphadenectomy,** and postoperative **radiation therapy**

What is the survival rate for a Bartholin gland carcinoma?

The 5-year survival is only 50–60% as recurrences are common

Basal Cell Carcinoma

What is basal cell carcinoma of the vulva and how does it present?

A rare vulvar cancer that typically presents in postmenopausal Caucasian women as a **vulvar lesion** that has **rolled edges with central ulceration** (a “**rodent ulcer**”) usually on the labia majora. While usually asymptomatic, symptoms can include pruritus, bleeding, soreness, or irritation

How is basal cell carcinoma treated?

Wide local excision without lymph node dissection. They are associated with the presence of other antecedent or concomitant malignancies, so treatment also involves a search for concomitant disease

What are the survival rates for basal cell carcinoma?

Survival rates are overall good; while they are locally aggressive, lymphatic metastasis is rare

How is basal cell carcinoma diagnosed?

Via biopsy

Vulvar Sarcoma

What are the most common types of vulvar sarcoma?

Leiomyosarcoma and **rhabdomyosarcoma**

How does leiomyosarcoma usually present?

As an **enlarging, painful lesion** of the labia majora

How is a leiomyosarcoma treated?

Wide local excision (lymph node involvement is rare)

In whom is vulvar rhabdomyosarcoma most common?	Children
How is vulvar rhabdomyosarcoma treated?	Primary chemotherapy followed by surgery

Paget Disease of the Vulva

What is Paget disease of the vulva and how does it present?	An extensive intraepithelial adenocarcinoma. It is most commonly found in white women aged 60–80 and it grossly appears as multifocal, well-demarcated areas of a bright red background with white, hyperkeratotic areas
How does Paget disease usually present?	With pruritis and vulvar soreness
What is the histological appearance of Paget lesions?	Similar to Paget disease of the breast—large pale apocrine cells directly below the epithelium
What is the diagnostic workup for Paget disease?	About 20–30% of Paget disease patients will have a noncontiguous carcinoma (especially of the breast, colon, cervix, bladder, or gallbladder) and so evaluation should include an extensive evaluation
What is the treatment for Paget disease?	Either wide local excision or a simple vulvectomy. Long-term follow-up and lifelong surveillance for tumors at other sites are warranted
What is the prognosis of Paget disease?	Local recurrence is common. Increased depth of invasion and involvement of the lymph nodes lead to a poorer prognosis

VAGINAL CANCER OVERVIEW

Describe preinvasive vaginal malignancy	Called vaginal intraepithelial neoplasia (VAIN) . It is squamous atypia without invasion and is classified by depth of involvement
What are the designations of preinvasive vaginal malignancy?	VAIN I: involves lower one-third of the epithelium

<p>How does VAIN typically present?</p>	<p>VAIN II: involves lower two-thirds of the epithelium</p> <p>VAIN III: involves more than two-thirds of the epithelium; includes carcinoma in situ</p> <p>Usually asymptomatic, but can present as postcoital spotting or vaginal discharge</p>
<p>What are the options for VAIN treatment and what are the benefits of each?</p>	<ol style="list-style-type: none"> 1. Excision: the mainstay of therapy; allows histologic diagnosis 2. Ablation: well tolerated but may require repeat administration 3. Topical chemotherapy: good for multifocal disease or difficult to access lesions 4. Radiation therapy: a last resort therapy if all else has failed or is contraindicated 5. Observation: for mild cases
<p>What factors should be considered when deciding on therapeutic modalities for VAIN?</p>	<p>Previous treatment failures; presence of multifocal disease; risks of surgery; desire to preserve sexual function; whether invasive disease has been definitively excluded</p>
<p>What is the incidence of vaginal cancer and VAIN?</p>	<p>One case per 100,000 women in the United States; 3% of all female genital malignancies. However, the diagnosis of VAIN has been steadily increasing (likely secondary to increased awareness)</p>
<p>What is the most common type of vaginal cancer?</p>	<p>Metastatic cancer from other primary sites, especially cervix</p>
<p>What are the types of primary vaginal cancer?</p>	<p>Squamous cell carcinomas (most common); melanoma; sarcoma; adenocarcinoma</p>
<p>How is the diagnosis of vaginal cancer made?</p>	<p>Digital palpation and colposcopy (with acetic acid and Lugol's iodine to enhance visualization). Biopsy of suspicious areas is the definitive diagnostic tool</p>
<p>In whom should you aggressively search for VAIN/vaginal cancer?</p>	<p>Postmenopausal women with an abnormal Pap smear; any woman with an abnormal Pap smear without any cervical lesions</p>

Squamous Cell Vaginal Carcinoma

What are the risk factors for squamous cell vaginal cancer?

HPV infection; HIV infection; increased number of sexual partners; early age at coitarche; smoking; prior lower genital neoplasia

What are the explanations for the association between squamous cell vaginal cancer and other genital neoplasia?

1. **Extension of disease** → CIN of VIN may spread contiguously to the vagina
2. **Common etiologic factors** → vulvar, cervical, and vaginal cancer share many common etiologies (such as HPV exposure)
3. **Prior radiation** → treatment of prior neoplasia increases susceptibility to later cancers

What are the symptoms of squamous cell vaginal cancer?

Up to one-fifth of all women are **asymptomatic** but are found to have a vaginal mass or atypical Pap smear. Symptoms can include **abnormal vaginal bleeding, vaginal discharge, vaginal mass, urinary/GI complaints, pelvic pain**

Where are squamous cell vaginal lesions typically located?

In the **upper one-third of the vagina**, especially the **posterior wall**

How is vaginal cancer staged?

Using the FIGO system which is clinically done using information obtained from the physical examination, cystoscopy, proctoscopy, and x- rays. See Table 5-4.

What are the common primary sites for metastatic vaginal cancer?

Endometrium, breast, Cervix, rectum, Vulva, kidney, ovary

Table 5-4 The Stages of Vaginal Cancer

I	Carcinoma confined to the vaginal mucosa
II	Submucosal infiltration into parametrium, but not extending out to pelvic wall
IIA	Subvaginal infiltration; no parametrial involvement
IIB	Parametrial infiltration; not extending to pelvic wall
III	Carcinoma extends to the pelvic wall
IV	Carcinoma extends beyond the true pelvis
IVA	Infiltration of the mucosa of bladder or rectum or extends outside true pelvis
IVB	Distant metastatic disease

How do these cancers spread to the vagina?

Either through direct extension or via lymphatic or hematogenous spread

To where and by what routes does primary vaginal cancer spread?

1. **Direct extension** to bladder, rectum, urethra, parametria, and bony pelvis
2. **Lymphatic spread** to pelvic and paraaortic lymph nodes
3. **Hematogenous dissemination** to lungs, liver, and bone (usually occurs late)

What are the treatment options for vaginal cancer and when is each used?

1. **Radiation:** the main treatment modality; used with or without surgery for large stage I or all stage II–IV lesions
2. **Surgery:** used for small, localized stage I cancer in the upper vagina
3. **Chemotherapy:** sometimes used in combination with radiation with advanced disease, however not shown to be more effective

What are the side effects of treatment?

Fistulas (rectovaginal or vesicovaginal); vaginal or rectal strictures; radiation cystitis or proctitis; vaginal stenosis; radiation-induced menopause

What considerations need to be taken into account when deciding on a treatment modality?

Stage of tumor; proximity of tumor to structures that preclude radiotherapy; anatomical constraints of surgery; future sexual function

What predicts survival rates?

Stage of cancer at presentation

What are the average 5-year survival rates for each stage?

1. Stage 0: 95%
2. Stage 1: 67%
3. Stage 2: 39%
4. Stage 3: 33%
5. Stage 4: 19%

What is a verrucous carcinoma?

An uncommon variant of squamous cell carcinoma that presents as a **large, verrucous mass**. It has a low **malignant potential**, although it is locally aggressive

Other Vaginal Cancers

What is vaginal melanoma and how does it typically present?

A rare but aggressive malignancy of the vaginal mucosa. Occurs in middle-aged Caucasian women and presents

<p>What is the 5-year survival rate of vaginal melanoma?</p>	<p>as a dark-colored mass or ulceration, usually in the distal one-third of the vaginal wall</p> <p>A dismal 10%</p>
<p>When does adenocarcinoma of the vagina typically present?</p>	<p>Usually in women under 20</p>
<p>In utero exposure to what leads to vaginal adenocarcinoma?</p>	<p>DES (diethylstilbestrol)—it leads to the clear cell variant of adenocarcinoma</p>
<p>What is the risk of developing clear cell adenocarcinoma if exposed?</p>	<p>1 in 1000; higher risk is associated with those exposed before 12 weeks of gestation</p>
<p>How does clear cell carcinoma of the vagina present?</p>	<p>As a polypoid vaginal mass in a young woman; DES was discontinued in 1971 and so most cases have already been discovered</p>
<p>What are the major types of vaginal sarcomas?</p>	<p>Rhabdomyosarcomas; leiomyosarcomas; endometrial stromal sarcomas; malignant mixed Müllerian tumors</p>
<p>What is the most common form of vaginal sarcoma?</p>	<p>Embryonal rhabdomyosarcoma or sarcoma botryoides</p>
<p>What is sarcoma botryoides and how does it present?</p>	<p>A very aggressive malignant tumor that presents in early childhood as a vaginal mass that resembles a bunch of grapes</p>
<p>How is sarcoma botryoides treated?</p>	<p>Preoperative chemotherapy followed by surgery or radiation</p>

Uterine Cancer

ENDOMETRIAL CANCER

<p>What is endometrial cancer?</p>	<p>An estrogen-dependent neoplasm of the endometrium that begins with proliferation of normal endometrial tissue. Over time, hyperplasia of the glandular elements becomes anaplastic and then neoplastic, with invasion of</p>
------------------------------------	--

There are two types of endometrial cancer based on light microscopic appearance, clinical behavior, and epidemiology, Type 1 and Type II. What are these types?

How common is endometrial cancer and in whom does it occur?

What are the major symptoms of endometrial cancer?

What are the roles of estrogen and progesterone in endometrial cancer?

What are the main etiologies for chronically elevated estrogen levels?

What are the risk factors for endometrial cancer?

What are protective factors against endometrial cancer?

What is the relationship between endometrial hyperplasia and endometrial cancer?

the underlying stroma, myometrium, and vascular spaces

Type 1: These have an endometrioid histology and comprise 70–80% of newly diagnosed cases of endometrial cancer in the United States. They are associated with unopposed estrogen exposure and are often preceded by premalignant disease

Type II: These have nonendometrioid histology (usually papillary serous or clear cell) with an aggressive clinical course. Hormonal risk factors have not been identified, and there is no readily observed premalignant phase

It is the **most common** gynecologic cancer; 75% of these cancers occur in **postmenopausal women**

Abnormal uterine bleeding (primarily postmenopausal bleeding)

Estrogen stimulates endometrial growth while **progesterone has anti-proliferative** effects. Long-term unopposed estrogen stimulation can eventually lead to atypical endometrial hyperplasia and endometrial cancer

Exogenous unopposed estrogen (i.e., estrogen replacement therapy); chronic anovulation (i.e., polycystic ovarian syndrome [PCOS]); estrogen-producing tumors (i.e., granulosa cell tumors); obesity

Obesity; diet; diabetes; hypertension; early menarche/late menopause; nulliparity; PCOS (infertility); tamoxifen treatment for breast cancer; family predisposition (hereditary nonpolyposis colorectal cancer [HNPCC]/Lynch syndrome II)

Combined oral contraceptives; exercise; multiparity; smoking (through increased hepatic metabolism of estrogen)

Endometrial hyperplasia is a benign condition associated with hyperestrogenic states. Atypical

What characteristics are used to classify endometrial hyperplasia?

hyperplasia is a precancerous condition

1. Glandular/stromal architecture (simplex or complex)
2. Nuclear atypia (present or absent)

Which histologic feature of endometrial hyperplasia confers the greatest risk for cancer?

Nuclear atypia

What are the four types of endometrial hyperplasia and what is the risk of cancer for each?

1. Simple hyperplasia without atypia 1% (penny)
2. Complex hyperplasia without atypia 3% (nickel)
3. Simple hyperplasia with atypia 8% (dime)
4. Complex hyperplasia with atypia 40% (~quarter)

What are the general guidelines for the treatment of endometrial hyperplasia?

If nuclear atypia is absent, treat with progesterone. If nuclear atypia is present, hysterectomy is recommended

Describe your classic patient diagnosed with endometrial cancer

A postmenopausal woman who is obese, nulliparous/infertile, diabetic, and hypertensive

How should atypical endometrial cells on a Pap smear be worked up?

They are not diagnostic for endometrial cancer, but they do warrant an endometrial biopsy for further evaluation

What is the differential diagnosis for abnormal uterine bleeding in the premenopausal woman?

Complications of early pregnancy; other gynecologic neoplasms; leiomyomata; endometrial hyperplasia/polyps; cervical polyps; Intrauterine device; hemophilias

What are your differential diagnoses for abnormal uterine bleeding in the postmenopausal woman?

Atrophic vaginitis; exogenous estrogens; other gynecologic neoplasms; endometrial hyperplasia/polyps

What is the gold standard for diagnosing endometrial cancer?

Hysteroscopy with dilation and curettage (D&C)

Is CA-125 a useful laboratory test to help with diagnosis?

In advanced disease, CA-125 may be elevated; however, it is not useful in the diagnosis or management

What are the different subtypes of endometrial cancer?

1. **Endometrioid adenocarcinoma (80%)**

How does endometrial cancer spread?	<ol style="list-style-type: none"> 2. Papillary serous carcinoma (5–10%) 3. Clear cell carcinoma (1–5%)
What lymph nodes are involved in endometrial cancer metastases?	<p>Most commonly through direct extension, but it can also spread transtubally, lymphatically, or hematogenously</p>
How is tumor grading for endometrial cancer determined?	<p>Pelvic and paraaortic lymph nodes</p>
What are the three different tumor grades for endometrial cancer?	<p>By tumor histology (architecture and nuclear atypia)</p>
How is endometrial cancer staged?	<ol style="list-style-type: none"> 1. Grade 1: Well differentiated (~95% glandular tissue, 5% solid pattern) 2. Grade 2: Moderately differentiated (~50–95% glandular tissue, 5–50% solid pattern) 3. Grade 3: Poorly differentiated (<50% glandular tissue, >50% solid pattern)
What is the staging system for endometrial cancer?	<p>Surgically (by the spread of the cancer) based on abdominal exploration, pelvic washings, total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH/BSO), and pelvic and periaortic lymph node biopsies</p>
How is endometrial cancer treated?	<p>See Table 5-5.</p>
What is the role of adjuvant therapy in endometrial cancer?	<p>TAH/BSO with lymph node dissection</p> <p>It is dependent on whether the patient is at low, intermediate, or high risk for disease recurrence. Below are general guidelines for adjuvant postoperative treatment:</p> <ol style="list-style-type: none"> 1. Low risk (Stage Ia, Ib): no adjuvant treatment warranted (Stage Ia, grade 3) Vaginal brachytherapy 2. Intermediate-risk (Stage Ic, Stage II): vaginal brachytherapy 3. High risk (Stage III, IV): pelvic/abdominal radiation therapy ± chemotherapy

Table 5-5 Stages of Endometrial Cancer

Stage	Description	Endometrial tumors	5-Year survival
Stage I: Involvement limited to the uterus	Ia: tumor limited to the endometrium Ib: invasion < half of myometrium Ic: invasion > half of myometrium	75%	80–95%
Stage II: Extension to and involvement of the cervix	IIa: extension to endocervical glands only IIb: cervical stromal invasion	11%	65–75%
Stage III: Local spread	IIIa: invasion of serosa and/or adnexa, and/or positive peritoneal cytology IIIb: vaginal metastases IIIc: metastasis to pelvic and/or para-aortic lymph nodes	11%	30–60%
Stage IV: Distant spread	IVa: invasion of the bladder and bowel IVb: distant metastasis including intra-abdominal and/or inguinal lymph nodes	3%	5–20%

Name the chemotherapeutic agents used in endometrial cancer and their major toxicities

1. Doxorubicin (cardiotoxicity)
2. Cisplatin (ototoxicity, neuropathy, nephrotoxicity, nausea)
3. Paclitaxel (bone marrow suppression, allergic reaction)

What is the recommended treatment for women who have early-stage disease and wish to preserve their fertility?

Trial of progestins after careful counseling

What factors worsen the prognosis for endometrial cancer?

Advanced stage; higher pathologic grade; histologic subtype; increase in myometrial invasion; lymph/vascular space invasion

What is the appropriate follow-up after treatment of endometrial cancer?

Monitor patients every 3–4 months for the next 2–3 years, then twice yearly with a comprehensive pelvic examination

What is the overall prognosis for endometrial cancer?

Without major adverse risk factors, treatment with a TAH/BSO may result in a 5-year survival greater than 95%

What is the screening recommendation for the general population?

Screening is not warranted for women who are asymptomatic

What is the screening recommendation for women with/at risk for HNPCC?

Annual endometrial biopsies by age 35

UTERINE SARCOMA

What is a uterine sarcoma?

A very aggressive tumor that arises from the myometrium

How common is it and who does it affect?

It is a very rare cancer (3–4% of all uterine malignancies) and commonly affects women over age 40

What are risk factors for uterine sarcoma?

History of pelvic radiation; Long-term use of tamoxifen

What are the main types of sarcomas?

Mixed müllerian tumors (carcinosarcoma); leiomyosarcoma; endometrial stromal sarcoma (ESS); undifferentiated sarcoma

How do uterine sarcomas present clinically?

Abnormal uterine bleeding; rapidly enlarging uterus; malodorous vaginal discharge; pelvic pressure and pain; part of the tumor may protrude from the cervical os

What is the major differential diagnosis of uterine sarcoma?

Uterine leiomyomas

What preoperative examinations may help with the diagnosis of a uterine sarcoma?

Endometrial biopsy; dilation and fractional curettage

How is the definitive diagnosis of uterine sarcoma made?

Exploratory laparotomy with a pathologic diagnosis

What is the role of imaging studies in uterine sarcomas?

Imaging studies cannot distinguish between a sarcoma or other uterine tumors. A CT scan of the thorax is recommended because uterine sarcoma commonly metastasizes to the lung

What is the staging system for uterine sarcomas?

Staging is done surgically and is a modification of the system used for endometrial cancer

What is the treatment for uterine sarcomas?

TAH/BSO, pelvic/paraortic lymph node dissection, and pelvic washings

What is the role of adjuvant therapy?

Stage I and II pelvic radiation therapy

What is the overall prognosis for sarcomas?

Generally they have worse prognosis than endometrial cancers of a similar stage

Ovarian Cancer

INTRODUCTION

How common is ovarian cancer?

It is the second most common gynecologic malignancy, but the **leading cause of mortality** among women who develop a gynecologic malignancy, and the **fifth most common cause of cancer-related female deaths in the United States**. The lifetime risk of developing ovarian cancer is 1.4% (1 in 70)

Why is ovarian cancer the leading cause of mortality among women with gynecologic cancers?

Symptoms of early-stage ovarian cancer are ill-defined and many women will not seek medical attention. Often, the cancer is detected at its advanced stages when it has spread beyond the ovaries

How are ovarian tumors categorized?

Epithelial origin; germ cell tumors; sex cord-stromal tumors; cancer metastatic to the ovary (usually from primary tumors of the breast, GI tract, or genital tract)

Which ovarian tumor is the most common?

Epithelial (~90% of all ovarian tumors)

EPITHELIAL OVARIAN CANCER

What age group does EOC affect?

Predominantly **postmenopausal** women. Median age is 62 years old

What is thought to be the underlying hypothesis in the development of EOC?

It is not well understood, but one hypothesis is that repetitive ovulation where disruption and repair of the ovarian epithelium may allow opportunity for genetic mutation and malignant transformation

What are the risk factors for development of EOC?

Family history of breast cancer or EOC; history of breast cancer; advanced age; obesity/high fat diet; infertility; nulliparity; talc; endometriosis

What factors are protective against development of EOC?

OCPs; tubal ligation; breastfeeding (history of); pregnancy; multiparity

What percent of EOC develops sporadically? What percent are because of genetic predisposition?

90% are sporadic; 10% are genetic predisposition

What is one feature that distinguishes hereditary ovarian cancer from a somatic cause?

The onset of ovarian cancer occurs **10 years earlier** in hereditary syndromes

With what hereditary syndromes has ovarian cancer been associated? Describe them.

- Breast and ovarian cancer (BOC):** associated with BRCA-1 mutation (autosomal dominant), more susceptibility to BOC, higher frequencies in Ashkenazi Jews
- Lynch II Syndrome (hereditary nonpolyposis colon cancer—HNPCC):** colorectal cancer is hallmark, also commonly found are endometrial, urogenital, and other GI primaries
- Site-specific ovarian cancer:** strong genetic link, autosomal dominant, usually two or more first-degree relatives have the disease

What are current screening recommendations for women at high risk?

- Prophylactic genetic screening
- CA-125 and transvaginal ultrasound every 6–12 months beginning between ages 25 and 35 years

What other preventative surgical procedure is recommended for women with a presumed hereditary ovarian cancer syndrome?

Prophylactic oophorectomy who have completed childbearing or by age 35

With what other gynecologic disease is ovarian cancer associated?

Endometriosis (25%)

What are the subtypes of EOC and how common is each type?

Serous 75–80%
Mucinous 10%
Endometrioid 10%
Clear cell <1%
Transitional cell <1%
Undifferentiated <10%

What are some key features that describe each tumor type?

Serous: often bilateral if malignant, resembles the endosalpinx, extra-ovarian spread common, histological finding of **psammoma bodies**

Mucinous: unilateral, **large size**, resembles the endocervix

Endometrioid: resembles the endometrium

Clear cell: “mesonephroid,” “**clear cells**” similar to renal carcinoma, **hypercalcemia**

Transitional cell: “Brenner tumors,” **transitional epithelium**, unilateral, benign, or malignant

Undifferentiated: No distinguishable histologic features

At what cancer stage is EOC usually diagnosed?

75% of ovarian cancers are detected at **stage III or IV**. Five-year survival rate is 5–35%

What is the typical presentation of a patient with ovarian cancer?

A **postmenopausal woman** who presents with any or a combination of these:

1. **Abdominal distension** from ascites or pelvic mass
2. **Pelvic pressure**
3. **Nausea, early satiety**, weight loss, bowel obstruction
4. Dyspnea because of a pleural effusion (metastasis to the lungs)

How does ovarian cancer spread?

Ovarian cancer cells are found in peritoneal fluid (ascites) which carries them to distant abdominal sites

Should the ovaries on a postmenopausal woman be palpable on examination?

Women who are more than 1 year postmenopause should have atrophic, nonpalpable ovaries. An ovarian enlargement in a postmenopausal woman is cancer until proven otherwise

What findings on the physical examination would lead you to believe this is an ovarian malignancy?

A **solid, fixed, irregular pelvic mass** associated with an upper abdominal mass (omental caking) and/or ascites

What is the following?

Omental caking: ovarian cancer metastasis to the omentum causing a fixed upper abdominal mass with ascites, very common in advanced disease

What is the initial step to evaluate an ovarian lesion?	Ultrasound
What other radiographic tests should be ordered in a patient suspected of having EOC?	Abdominal-pelvic CT: to determine the extent of metastases Chest x-ray: to determine the presence of a pleural effusion
What tumor marker is often elevated in advanced stage ovarian cancer? What is its value?	CA-125 is elevated in 80% of cases with advanced ovarian cancer. It is not as useful in premenopausal women or for early stage disease. It is most useful to evaluate the progression/regression of disease following treatment
What is the “gold standard” to diagnose EOC?	Exploratory laparotomy/laparoscopy and histopathologic diagnosis
How is ovarian cancer staged?	Surgically
Describe the stages of ovarian cancer and their prognoses	See Table 5-6.

Table 5-6 The Stages of Ovarian Cancer

Stage	Description	5-Year survival
Stage I: Tumor limited to the ovaries	1a: one ovary involved and capsule intact 1b: both ovaries involved and capsule intact 1c: 1a or 1b and tumor on the ovary; ruptured capsule; malignant ascites and positive peritoneal washings for malignant cells	>90%
Stage II: Spread of the tumor to the pelvis	IIa: uterus/oviducts are involved IIb: other pelvic structures are involved IIc: IIa or IIb and tumor on the ovary; ruptured capsule; malignant ascites and positive peritoneal washings for malignant cells	60–75%
Stage III: Spread to the abdominal cavity	IIIa: microscopic seeding of the abdominal peritoneal surfaces; negative nodes IIIb: <2 cm implants on the abdominal peritoneal surface IIIc: >2 cm implants on the abdominal peritoneal surface and/or positive retroperitoneal or inguinal lymph nodes	25–40%
Stage IV: Distant metastasis	Liver, splenic, or pulmonary parenchymal metastasis Malignant pleural effusion Metastases to the supraclavicular lymph nodes or skin	5%

Which lymph nodes are typically involved in advanced ovarian cancer?

Pelvic and para-aortic lymph nodes

What is the optimal treatment for advanced ovarian cancer disease?

Surgical debulking and chemotherapy. Debulking includes TAH/BSO, omentectomy. In epithelial tumors, cytoreductive surgery does not include uterus removal

What is the most effective chemotherapeutic regimen?

A combination of **paclitaxel plus cisplatin**

What are the most common side effects of chemotherapy?

Nausea, vomiting, alopecia, diarrhea, nephrotoxicity, myelosuppression

What are poor prognostic factors?

Stage III or IV disease; advanced age; short disease-free interval; residual tumor after primary surgery

NONEPITHELIAL OVARIAN CANCER

Ovarian Germ Cell Tumors

What percent of all ovarian neoplasms are germ cell tumors (GCTs)?

20–25%

From what embryologic origin do ovarian GCTs (OGCTs) arise?

They arise from the totipotential germ cells that normally differentiate into the three germ layers

What are the OGCT subtypes?

Dysgerminoma; endodermal sinus tumor (yolk sac tumor); immature teratoma; mature teratoma; embryonal carcinoma; choriocarcinoma; mixed GCTs

At what age do these malignancies occur?

It is a disease of children and young women (usually between 10 and 30 years)

What are the typical clinical symptoms and signs?

Acute abdominal pain (from rupture or torsion); rapid abdominal enlargement (because of either the mass or ascites); fever; vaginal bleeding

How does the time of diagnosis differ from that of EOC?

Patients usually present at Stage Ia
For each of the following scenarios, identify the correct GCT:

A **20-year-old** woman presents with acute onset of lower abdominal pain and pelvic mass. Serum tests reveal an **elevated level of alpha-fetoprotein**

(AFP). Intraoperative examination reveals **bilateral ovarian masses and intraperitoneal dissemination**. Tumor biopsy reveals **Schiller-Duval bodies**

Endodermal sinus tumor (yolk sac tumor)

These tumors are **rare**, found in **young females**. They may **secrete estrogen**, causing **precocious puberty** and **abnormal vaginal bleeding**. **β -hCG is commonly elevated** and serves as a tumor marker

Embryonal carcinoma and choriocarcinoma

A **young female** presents with abdominal enlargement. Radiographic films reveal a **unilateral mass with calcification**. Tumor markers are not present. Histology of the removed specimen reveals the presence of a poorly differentiated tumor consisting of **hair, teeth, cartilage, bone, and muscle**

Immature (malignant) teratoma

This dysgerminoma is the **female counterpart of the male seminoma** and commonly occurs in **adolescents** and young females. It is the most common of the malignant OGCTs and typically presents as a rapidly growing unilateral mass. Lactic dehydrogenase (**LDH**) **may be elevated**

Dysgerminoma

A young female presents with **tachycardia, palpitations, anxiety, and tremors**. The pituitary and thyroid appear normal on examination and radiographic films. The patient is later found to have a **mature teratoma consisting of thyroid tissue**

Mixed GCTs

These neoplasms contain **two or more germ cell elements**. A dysgerminoma and endodermal sinus tumor occur together most frequently. **LDH, AFP, and β -hCG may be elevated**

Struma ovarii

Which GCTs are benign?	Gonadoblastomas and mature cystic teratomas
What laboratory test should be ordered prior to definitive diagnosis and treatment?	Serum ovarian tumor marker panel for β -hCG, AFP, LDH
What is the treatment plan for malignant OGCTs?	Unilateral adnexectomy (fertility preserving) and complete surgical staging; adjuvant chemotherapy
How are OGCTs staged?	Similar to EOC staging
How effective is chemotherapy in malignant GCTs?	GCTs are very chemosensitive. 90% of patients with early-stage OGCTs and 80% of patients with advanced disease are long-term survivors
For which tumors is chemotherapy recommended?	For all resected Stage I GCTs except Stage Ia dysgerminoma and Stage Ia, grade 1 unruptured immature teratoma
What chemotherapeutic agents are used?	Bleomycin, etoposide, cisplatin is the preferred regimen
List the chemotherapeutic agent that causes the toxicities listed below:	
Nephrotoxicity	Cisplatin
Pulmonary fibrosis	Bleomycin
Blood dyscrasias	Etoposide
How do you monitor the response to chemotherapy?	Based on the physical examination and decrease in the serum tumor marker levels (if initially elevated)
What is the prognosis of GCTs?	Much better than EOC. Depending on the GCT, 5-year survival ranges from 60% to 95%

Ovarian Sex Cord-Stromal Tumors

What percent of all ovarian tumors are sex cord-stromal tumors?	5–8% of all primary ovarian neoplasms
From what embryologic origin do ovarian sex cord-stromal tumors arise?	They develop from the cells that surround the oocytes, including those that produce ovarian hormones
What are the ovarian sex cord-stromal tumor subtypes?	Granulosa cell tumors; ovarian thecoma; ovarian fibroma; Sertoli-Leydig cell tumors

What are the typical clinical symptoms and signs?

Estrogen-producing tumors may cause **precocious puberty** in young girls, and **endometrial hyperplasia** and **abnormal vaginal bleeding** in postmenopausal women. Androgen-producing tumors cause **virilization**, **acne**, and **gynecomastia**

In the following scenarios, identify the correct sex cord-stromal tumor

Most women present with a **unilateral adnexal mass** between the second and third decade. **Virilization**, **hirsutism**, **acne**, and **menstrual abnormalities** commonly occur. **Testosterone** levels may be elevated
Sertoli-Leydig cell tumors

A postmenopausal woman presents with a **unilateral benign solid mass** that is not hormone secreting
Ovarian fibroma

These tumors are **estrogen producing** and cause **precocious puberty** in young females and **abnormal vaginal bleeding** in postmenopausal women. **Inhibin** may be elevated
Granulosa/thecoma

What is Meigs' syndrome?

It is the association of an **ovarian fibroma** with **ascites** and/or **pleural effusion**

With what other syndrome are ovarian fibromas associated?

Gorlin syndrome (nevroid basal cell carcinoma syndrome): an autosomal dominant disease characterized by its association with **basal cell cancers**, **brain tumors**, **odontogenic keratocysts**, and **mesenteric cysts**

What are Call-Exner bodies and for what tumor are they nearly pathognomonic?

They are small, fluid-filled cavities in between granulosa cells of ovarian follicles that contain eosinophilic fluid. They are associated with **granulosa cell tumors**

What other cancer is associated with granulosa cell tumors/ovarian thecomas?

With estrogen-producing tumors, **endometrial adenocarcinoma** must be considered. **These women must have an endometrial biopsy**

How are ovarian sex cord-stromal tumors staged?

Similarly to EOC

What is the surgical treatment?	TAH/BSO or unilateral oophorectomy for women who wish to preserve their fertility and who have low-stage/grade neoplasms. A dilation & curettage or an endometrial biopsy must be performed for estrogen-secreting tumors
Is adjuvant therapy recommended?	Chemotherapy is not recommended for low-stage/grade neoplasms. Data is inconclusive for more advanced stages
What is the prognosis for these tumors?	In general, very good. For early-stage cancers, 5-year survival rates range between 70–90%
What follow-up procedures are recommended for the hormone-producing cancers?	Serial pelvic examinations and serum tumor marker levels

Tumors Metastatic to the Ovary

What percent of ovarian tumors are metastatic?	5%
What are the primary tumors that metastasize to the ovary?	Breast; gastrointestinal tract; acute leukemia and lymphoma (rare)
How do metastatic tumors to the ovary clinically present?	Gastrointestinal cancers present like primary ovarian: pelvic mass, ascites, early satiety, and change in bowel habits. Recurrent metastatic breast cancer may present as an asymptomatic pelvic mass
What are gastric tumors metastatic to the ovaries called?	Krukenberg tumors
What primary ovarian tumor do Krukenberg tumors resemble in histology?	Mucin-secreting adenocarcinoma of the ovary
What histologic feature is pathognomonic for Krukenberg tumors?	Signet ring cells
What can be used to differentiate between a primary mucinous ovarian tumor and metastatic colon cancer?	Immunohistochemistry (cytokeratin expression)
Describe the cytokeratin expression pattern of each of the following:	Primary mucinous ovarian: CK7 positive; CK20 negative

	Metastases from a primary mucinous adenocarcinoma of the colon: CK7 negative; CK20 positive
What is the overall prognosis in patients who have cancers metastatic to the ovary?	Survival following surgery and chemotherapy is very poor, ranging from 4 to 12 months

Fallopian Tube Cancer

How common is fallopian tube cancer?	Primary carcinoma is very rare , accounting for 0.3% of primary gynecologic malignancies. Secondary carcinoma from metastases of ovarian, endometrium, gastrointestinal (GI), or breast cancers are more common
What are risk factors for fallopian tube cancers?	A mutation in the BRCA1 or BRCA2 gene. Women with these mutations who are planning prophylactic oophorectomies should also have their oviducts removed
What is the most common type of fallopian tube cancer?	Papillary serous adenocarcinoma
What other types of fallopian tube cancers are there?	Adenosquamous carcinoma and sarcoma
How does the cancer spread?	It spreads through the tubal ostia into the peritoneal cavity and also through lymphatics to the para-aortic and pelvic lymph nodes
Where does fallopian tube cancer commonly metastasize?	Ovaries, uterus, and pelvic and para-aortic nodes
What is the classic clinical presentation?	A woman between the ages of 40 and 60 who presents with Latzko triad: serosanguineous vaginal discharge, pelvic pain, and pelvic mass . However, this triad is seen in less than 15% of patients
What is hydrops tubae profluens?	The spontaneous or pressure-induced release of clear or blood-tinged vaginal discharge followed by shrinkage of an adnexal mass and relief of cramping pain. It is pathognomonic for fallopian tube cancer

What preoperative evidence suggests fallopian tube cancer?

Positive vaginal cytology for abnormal cells with a negative workup for endometrial or cervical cancer suggests fallopian tube carcinoma

What is the role of CA-125 in fallopian tube cancer?

Preoperative CA-125 levels may determine prognosis. CA-125 levels are also sensitive markers for response to chemotherapy and recurrence

How is staging for fallopian cell cancers determined?

Surgically; it is staged similarly to epithelial ovarian cancer

What is the treatment?

Total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH/BSO) and tumor debulking. Retroperitoneal lymph node sampling, infracolic omentectomy, and peritoneal washings should also be performed for staging purposes. Chemotherapy consisting of carboplatin and paxlitaxel is recommended after surgery

What is the prognosis?

Similar to ovarian cancer. The overall 5-year survival rate is 56%

Urogynecology

Urinary Infections

ACUTE CYSTITIS

What is the prevalence and incidence of acute cystitis?

50–60% of women report having had cystitis at some point in their life. Young, sexually active women have on an average 0.5 episodes per year; postmenopausal women have on an average of 0.1 episodes per year

Describe the symptoms of acute cystitis

Abrupt onset of: **dysuria, urinary frequency and urgency, suprapubic or low back pain**, possible hematuria (in hemorrhagic cystitis)

What is the differential diagnosis for these symptoms?

Urethritis; vaginitis; pyelonephritis

For each differential diagnosis, list the main symptoms/signs that would differentiate it from acute cystitis

Urethritis: urethral discharge, more gradual onset, history of new sexual partner

Vaginitis: vaginal discharge/odor, pruritis, dyspareunia, external dysuria, absence of frequency or urgency

Pyelonephritis: elevated temperature, costovertebral angle (CVA) tenderness, nausea/vomiting

How is the diagnosis of acute cystitis definitively made?

1. Most cases can be diagnosed on **history and physical examination alone**. The examiner should specifically look for signs of vaginitis, cervicitis, urethral discharge, or herpetic ulcerations
2. **Gonococcus (GC) and Chlamydia cultures** should be done if urethritis is suspected

<p>Describe the pathophysiology of acute cystitis</p>	<p>3. Urinalysis should also be done, looking for pyuria or hematuria. Urine culture is typically not always needed, unless the infection is complicated or it is a recurrent infection</p> <p>Coliform bacteria from the rectum colonize the vaginal introitus, enter the urethra, and ascend toward the bladder. This migration can be facilitated by sexual intercourse</p>
<p>What are the most common pathogens that cause acute cystitis?</p>	<p><i>Escherichia coli</i> (80% of cases); <i>Staphylococcus saprophyticus</i> (5–15% of cases); <i>Proteus mirabilis</i>, <i>Klebsiella</i>, enterococci, <i>Pseudomonas</i>, <i>Serratia</i>, <i>Providencia</i>, staphylococci, and fungi (rare, more common in complicated cases)</p>
<p>What are the risk factors for the development of acute cystitis?</p>	<p>Increased sexual intercourse; newly sexually active; postmenopausal; diabetes, sickle cell anemia, immunosuppressed conditions; abnormalities of the genitourinary tract; use of spermicide, especially with a diaphragm; history of a recent UTI; recent hospitalization</p>
<p>What factors suggest the presence of a complicated urinary infection?</p>	<p>Patient characteristics: elderly patient; pregnancy</p> <p>Medical history: hospital-acquired infection; indwelling catheter/recurrent instrumentation; functional or anatomic abnormality of the urinary tract; recent antibiotic use; diabetes; immunosuppression</p>
<p>What is the prognostic significance of a complicated urinary infection?</p>	<p>Complicated infections are associated with increased rates of failing therapy</p>
<p>What are the treatment options for uncomplicated acute cystitis?</p>	<ol style="list-style-type: none"> 1. Trimethoprim-sulfamethoxazole for 3 days is first line therapy in geographic regions with resistance rates <20% 2. Nitrofurantoin for 3-5 days 3. Fluoroquinolone for 3 days <p>No follow up is needed unless symptoms recur</p>

What are the treatment options for complicated acute cystitis?

Treat initially with **broad spectrum antibiotics** (such as fluoroquinolones) and then tailor therapy based on the culture results. These patients should be treated for **7 to 14 days** and they need to be followed up to ensure resolution of symptoms

How often does cystitis recur?

In 20% of premenopausal women; the vast majority of these are because of exogenous reinfection. Postmenopausal women also have frequent reinfections

What is considered recurrent cystitis?

More than three UTIs per year or more than two UTIs within a 6-month period

What are the risk factors of recurrent cystitis?

Sexual intercourse; use of spermicide, especially with a diaphragm; recent antimicrobial use; genetic predisposition; in postmenopausal women: urinary incontinence, cystocele, elevated post-void residual volume

How is recurrent cystitis treated?

Cultures need to be taken to rule out resistance to prior treatment

For patients with multiple reinfections, treatment consists of one of three options:

1. **Behavioral changes** (change contraceptive methods, early postcoital voiding)
2. **Continuous low-dose antibiotic prophylaxis**
3. **Postcoital low-dose antibiotic prophylaxis**
4. **Self-start therapy** when symptoms begin

In postmenopausal women with frequent infections, **topical estrogen** is often used, with or without concomitant use of prophylactic antibiotics

Is cranberry juice an effective home remedy for preventing UTIs?

There is some evidence which suggests that cranberry juice inhibits pathogens from adhering to uroepithelial cells, which can reduce the incidence of UTI. Cranberry has not been found to be effective in the treatment of UTI

What percentage of reproductive-age women have asymptomatic bacteriuria?	3–6%
What are the risk factors for the development of asymptomatic bacteriuria?	Diabetes; advanced age; presence of an indwelling bladder catheter
Should asymptomatic bacteriuria be treated?	No, unless the woman is high risk
Who are the high-risk women that need treatment for their asymptomatic bacteriuria?	Pregnant women; women with indwelling catheters; renal transplant recipients; women with spinal cord injury; prior to invasive procedures

UTI IN PREGNANCY

UTI is the most common medical complication of pregnancy.

Why is pregnancy considered a high-risk condition with asymptomatic bacteriuria?	Both hormonal and mechanical changes predispose the pregnant woman with asymptomatic bacteriuria to develop acute pyelonephritis, which is associated with preterm birth and perinatal death. Pyelonephritis in pregnancy will lead to septicemia in 10–20% and acute respiratory distress syndrome (ARDS) in 2% of cases
How prevalent is asymptomatic bacteriuria in pregnancy?	It is estimated to occur in 4–7% of pregnant patients. If left untreated, up to 40% of cases will progress to pyelonephritis
How is this condition detected?	Screening for asymptomatic bacteriuria using urine culture is recommended at the first prenatal visit
How is asymptomatic bacteriuria treated?	Nitrofurantoin for 70 days is first line. If ineffective, amoxicillin, trimethoprim-sulfamethoxazole or a cephalosporin can be used for 3 days. Quinalones are generally not used during pregnancy. A urine culture should be done 1–2 weeks after completion of therapy. Monthly urine cultures should be done and suppressive antibiotics should be considered.

How is symptomatic cystitis treated in pregnancy?

Treatment and follow up are similar to asymptomatic bacteriuria. Acute pyelonephritis should be treated with intravenous (IV) antibiotics and hospitalization. Suppressive antibiotics should be given following treatment to any pregnant patient treated for acute pyelonephritis

URETHRITIS

Describe the signs and symptoms of urethritis

Dysuria, but with a more **gradual** onset and **milder** symptoms than with acute cystitis. **Hematuria is rare** and points to cystitis more than urethritis. Urethritis is often associated with abnormal vaginal discharge or bleeding (because of related cervicitis). A **mucopurulent discharge from the urethral os** is usually found on examination

What are the risk factors for urethritis?

History of an STD; new sexual partner in past weeks; partner with urethral symptoms

How is the diagnosis definitively made?

Pyuria on urinalysis and mucopurulent cervicitis or herpetic lesions are usually found. A positive GC, *Chlamydia*, or herpes test result can confirm the diagnosis

What are the most common pathogens that cause urethritis?

Most common: *Chlamydia trachomatis* (5–20%)
Neisseria gonorrhoea (<10%)
Genital herpes

Others: *Candida*, *Trichomonas*

What is irritant urethritis?

Dysuria that occurs as a reaction to an irritant such as a condom, tampon, or any other product inserted into the vagina

ACUTE PYELONEPHRITIS

Describe the symptoms of acute pyelonephritis

Typical presentation: flank pain, fever (>38°C), CVA tenderness, nausea/vomiting, ± cystitis symptoms

What is the differential diagnosis for acute pyelonephritis?

Gastrointestinal: **cholecystitis, appendicitis, pancreatitis, diverticulitis**

Gynecologic, pelvic inflammatory disease (**PID**), **ectopic pregnancy**

Pulmonary: **lower lobe pneumonia**

Genitourinary: **urinary calculi**

How is the diagnosis definitively made?

Most patients can be diagnosed based on history and physical examination alone

Urinalysis: **pyuria** is almost always found; **white cell casts**, if found, indicate renal origin; **gram-negative bacteria** on Gram stain

Urine cultures and possible blood cultures should be taken at time of diagnosis for antimicrobial susceptibility

What is the most common pathogen that causes acute pyelonephritis?

E. coli (70–95%); *S. saprophyticus* (5–20%)

Describe the pathophysiology of acute pyelonephritis

Uropathogens from the fecal flora colonize the vaginal introitus and infect the urethra. They then ascend via the urethra to the bladder and then into the kidneys. It is also possible that the kidneys could be seeded from bacteria in the lymphatics

What are the risk factors associated with developing uncomplicated acute pyelonephritis?

Increased sexual intercourse; history of UTI within past year; diabetes; stress incontinence within the past 30 days; a new sexual partner within the past year; spermicide use; family history of UTIs

How is acute pyelonephritis treated?

Outpatient treatment: **trimethoprim-sulfamethoxazole** or a **quinolone** for 10–14 days; if enterococcus is suspected, add amoxicillin until cultures return

Inpatient treatment: indicated if patient is noncompliant, cannot tolerate PO, is severely ill, or is pregnant; initial treatment is **ceftriaxone, ampicillin** and **gentamycin**, or **aztreonam**

What should be done if flank pain does not resolve after 2–3 days?

An ultrasound or CT to rule out perinephric or intrarenal abscess or ureteral obstruction

When should a patient be followed up after treatment for acute pyelonephritis?

Two weeks after completion of therapy or as soon as symptoms recur

Pelvic Organ Prolapse

How significant is pelvic organ prolapse (POP) in women in the United States?

Approximately 200,000 procedures for POP are performed annually. The lifetime risk for undergoing surgery for urinary incontinence or prolapse is 11%. The risk of requiring a repeat procedure may be as high as 29%

What is meant by POP?

Refers to the relaxation of the normal connective tissue supports of any of the pelvic organs (uterus, vaginal apex, bladder, rectum) and its associated vaginal segment from its normal anatomic location

What are the most common POP abnormalities? Describe each defect

Cystocele:

An anterior vaginal wall defect/prolapse that includes the bladder. See Fig. 6-1.

Enterocoele:

An apical vaginal wall defect/prolapse in which bowel is contained within the prolapsed segment. See Fig. 6-2.

Rectocele:

A posterior vaginal wall defect/prolapse that includes the rectum. See Fig. 6-3.

Uterine prolapse:

An apical vaginal wall defect where the cervix/uterus descend with strain from its normal anatomic site. Sometimes associated with cervical elongation

Vault prolapse:

A defect of the apex of the vagina, most commonly found posthysterectomy

Defects in which pelvic floor muscles/supporting structures allow POP?

The uterosacral and cardinal ligaments, the levator ani muscles, and the endopelvic connective tissue. See Figs. 6-4 and 6-5.

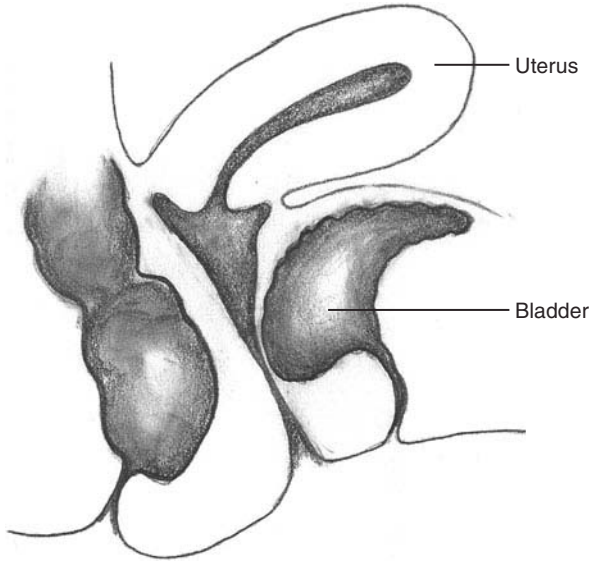


Figure 6-1 Anterior vaginal prolapse—cystocele.

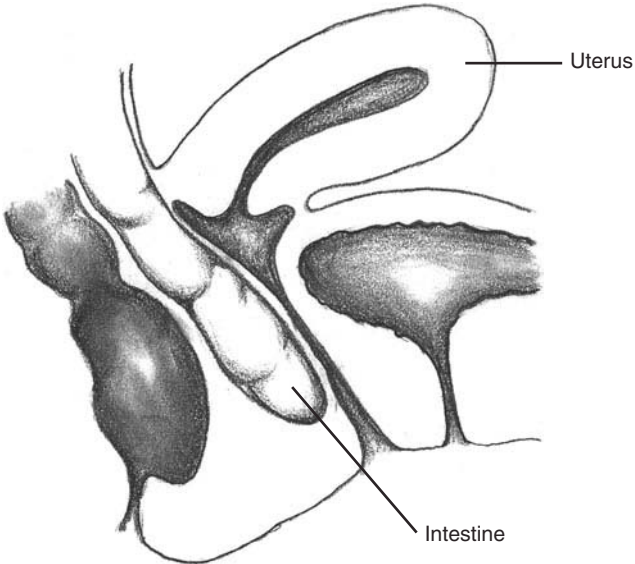


Figure 6-2 Apical prolapse—enterocele.

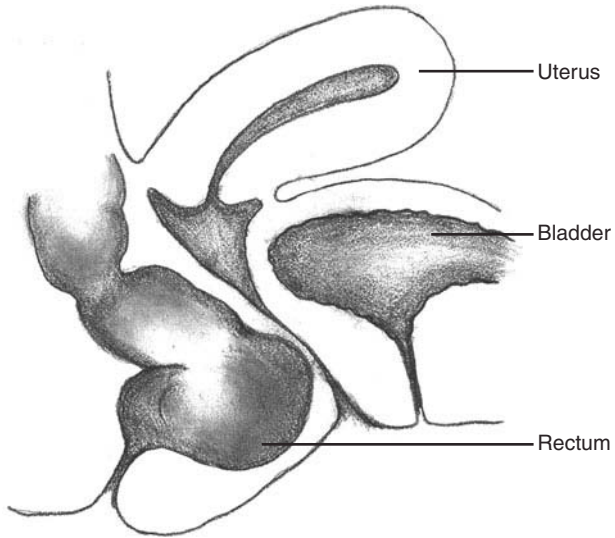


Figure 6-3 Posterior vaginal wall prolapse—rectocele.

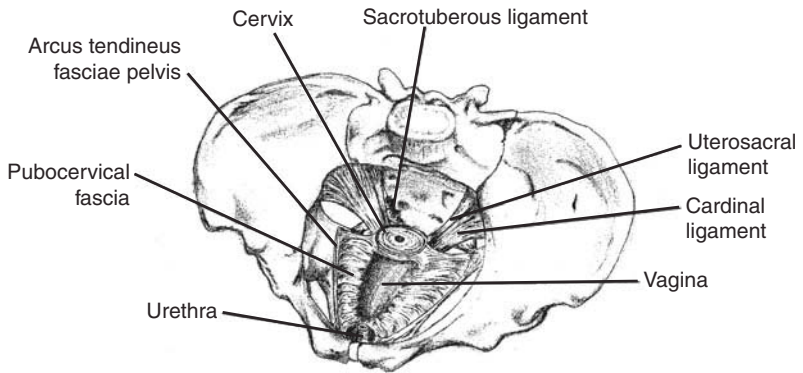


Figure 6-4 Ligaments of the female pelvis.

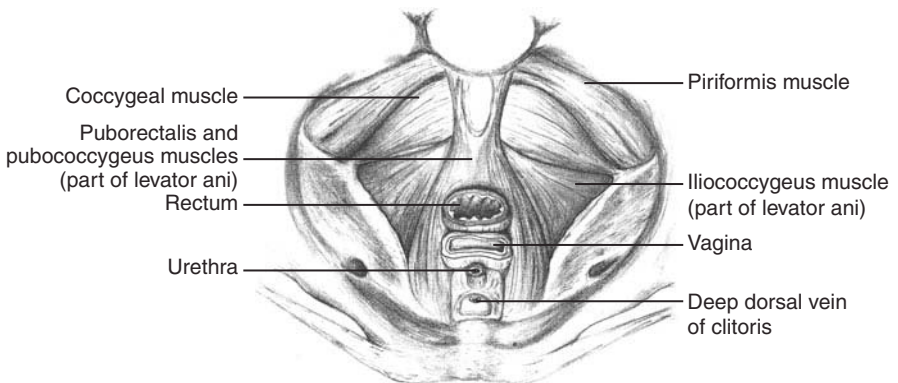


Figure 6-5 Pelvic diaphragm—superior view.

What are the risk factors for POP?

Advancing age; multiparity (vaginal delivery route); obesity; history of pelvic surgery (particularly hysterectomy); increased intra-abdominal pressure (i.e., chronic straining during constipation, chronic coughing (COPD), heavy lifting); postpartum muscle weakness (i.e., levator ani); loss of innervation; connective tissue disorders; tumor/masses; genetic predisposition

What is the most common symptom of POP?

Many women are asymptomatic and are found to have prolapse on exam.

The most specific complaint for POP is when the woman can see or feel a bulge of tissue that protrudes to or past the vaginal opening.

Patients sometimes describe this as a pressure or heaviness. This may be relieved by lying down, may be less noticeable in the morning and worsen later in the day because of extended exertion or standing

Other symptoms include: stress incontinence, urinary frequency or hesitancy, feeling of incomplete emptying of the bladder, defecatory symptoms, coital laxity

How is POP diagnosed?

It is based upon the findings of physical examination. The patient should be examined in both upright and recumbent (dorsal lithotomy) positions and each area of vaginal anatomy should be described separately

What are the grading systems used to evaluate and diagnose POP?

While there are several different classifications, currently the two most commonly used are:

- (1) The Pelvic Organ Prolapse Quantification (POPQ) system
- (2) The Baden–Walker system

What does the POPQ system involve?

The POPQ system was created to use objective measurements in centimeters using the hymenal ring as a reference point. It involves quantitative measurements of various points representing anterior, apical, posterior, and basal vaginal prolapse. These points are then used to determine the stage of the prolapse

Pro: it is helpful in standardizing the reporting of prolapse outcomes by relying on objective measurements

Con: it can be confusing to those not familiar with the system

Explain the Baden–Walker System

It is a less detailed grading system for evaluation of POP displacement compared to the POPQ system, but it is still widely used in clinical practice. See Table 6-1

Pro: it is straightforward and familiar to most practitioners

Con: it uses a subjective description of the prolapse that is not quantifiable

What is the management for women with asymptomatic POP?

Reassurance and observation at regular intervals. Pelvic floor muscle exercises (i.e., Kegel exercises) can in theory be of some benefit by improving the tone of the muscular floor upon which the pelvic organs rest

Table 6-1 Baden–Walker System for the Evaluation of POP on Physical Examination

Grade	Displacement of Prolapse
0	No prolapse
1	Halfway to the hymen
2	At the hymen
3	Halfway out of the hymen
4	Total prolapse

What is the management for women with symptomatic POP?

- (1) Nonsurgical treatment such as pessaries and symptom-directed therapies (i.e., weight loss, pelvic floor muscles rehabilitation, behavior modifications)
- (2) Surgical intervention

What is a pessary?

It is a prosthesis usually made out of rubber or silicone-based material with a spring frame that is placed in the vaginal vault to support the prolapsing vaginal walls or uterus. There are several types available which come in varying shapes and sizes. See Fig. 6-6.

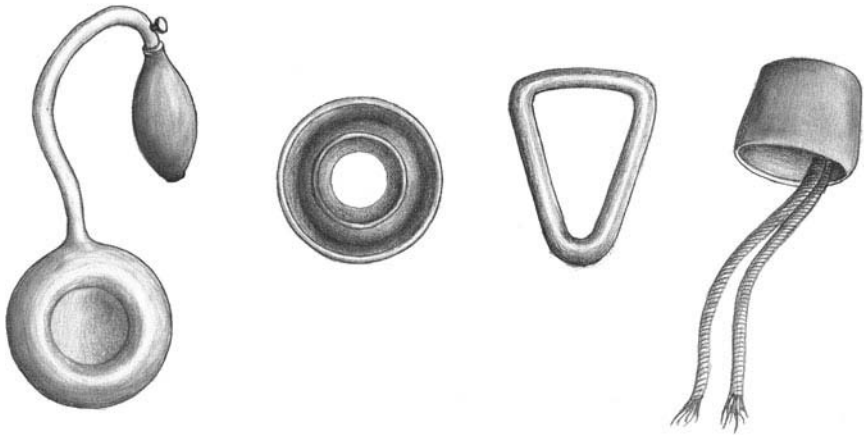


Figure 6-6 Types of pessaries.

When is a pessary contraindicated?

There are few contraindications to using a pessary: allergy to the product material untreated vaginitis or pelvic inflammatory disease and in a patient who is unable/unlikely to follow up

When is surgery indicated?

When a patient is symptomatic from her prolapse and has been counseled regarding her treatment options

**What is the traditional procedure for an anterior vaginal prolapse (cystocele)?
What does it involve?**

Anterior vaginal colporrhaphy. It involves dissecting the vaginal epithelium from the underlying fibromuscular connective tissue and bladder, and plicating the vaginal muscularis across the midline

**What is the traditional procedure for a posterior vaginal prolapse (rectocele)?
What does it involve?**

Posterior vaginal colporrhaphy. It involves dissecting the vaginal epithelium from the underlying fibromuscular connective tissue and rectum, and plicating the vaginal muscularis across the midline.

If a site-specific defect is noted, this is repaired

What are the different surgical options for treatment of an apical prolapse (enterocele)?

Colpocleisis: usually reserved for severe uterovaginal prolapse. It is an obliterative procedure that closes off the genital hiatus, and therefore should only be considered in patients who do not desire future sexual intercourse. It is minimally invasive, so it is a good consideration for patients in whom major surgery is contraindicated

Sacrospinous ligament fixation: attachment of the vaginal apex to the sacrospinous ligament, usually unilaterally

Iliococcygeus suspension: attachment of the vaginal apex to the iliococcygeus muscle, either uni- or bilaterally

Uterosacral ligament suspension: attachment of the vaginal apex to the uterosacral ligament bilaterally

Abdominal sacral colpopexy: an abdominal procedure attaching the vaginal apex to the sacral promontory

When is a hysterectomy warranted?

For treatment of uterine prolapse or if concomitant uterine pathology is present at the time of prolapse surgery

Urinary Incontinence

INTRODUCTION

Name the factors involved in normal urethral closure

Extrinsic factors: the levator ani muscles, endopelvic fascia, ligamentous attachments
 Intrinsic factors: striated and smooth muscle of the urethral wall, vascular congestion of the submucosal venous plexus, urethral epithelial folds, urethral elasticity, α -adrenergic receptors in the urethra (urethral tone)

Describe the factors involved in normal bladder function

Bladder filling: increased outlet resistance via muscle fiber recruitment; the detrusor is inactive

Full bladder: a micturition reflex is sent to the brain by tension-stretch receptors, cortical control mechanisms then permit or do not permit this reflex

Bladder emptying: voluntary relaxation of the pelvic floor and urethra, sustained contraction of the detrusor muscle

Describe the action of the detrusor muscle?

It is the **involuntary smooth muscle** wall of the bladder that **reflexively contracts** after voluntary relaxation of the pelvic floor and urethral musculature

Describe the innervation to the lower urinary tract

1. **Sympathetic** system—controls **bladder storage**
 - a. α -receptors: located mainly in the urethra and bladder neck, stimulation increases urethral tone (promotes closure)
 - b. β -receptors: located mainly in the bladder body, stimulation decreases bladder tone
2. **Parasympathetic** system—controls **bladder emptying**
 - a. stimulation of muscarinic receptors increases bladder contraction

- What is the prevalence and incidence of urinary incontinence?**
- Over 13 million Americans carry the diagnosis, with approximately 1 million new cases diagnosed each year
- What are some of the causes of urinary incontinence?**
- Anatomic abnormalities; infection; drugs; atrophy; neurologic issues
- What is the differential diagnosis of urinary incontinence?**
- Infection: cystitis
 Anatomic: fistula (vesicovaginal or ureterovaginal), ectopic ureter
 Neuro/Psych: dementia, normal pressure hydrocephalus
 Medications: diuretics, caffeine, sedatives
 Endocrine: diabetes mellitus, diabetes insipidus
- What is meant by the following symptoms?**
- Urgency:** sudden pressing desire to urinate
- Nocturia:** waking at night more than one time to urinate
- Overactive bladder:** a syndrome of urgency, frequency, and nocturia \pm urge incontinence
- Continuous urinary incontinence:** constant dribbling of small amounts of urine
- Daytime frequency:** subjective voiding too often by day
- Stress incontinence:** involuntary leakage with increased intra-abdominal pressure (e.g., with exertion, sneezing, or coughing)
- Urge incontinence:** involuntary leakage immediately preceded by a feeling of urgency; often precipitated by running water, hand washing, or cold temperatures
- Mixed incontinence:** involuntary leakage associated with urgency and increased intra-abdominal pressure
- 3. Somatic system—innervation of the pelvic floor and the external urethral sphincter**
- Name and describe the symptoms of the following major types of incontinence**

What type of incontinence do the following symptoms suggest?

Overflow incontinence: leakage associated with significant urinary retention which exceeds the storage capacity of the bladder (usually associated with neurogenic conditions)

Leakage when coughing/sneezing/laughing/exercising: stress incontinence

Leakage associated with a strong desire to void: urge incontinence

Many voids per day: urge or overflow incontinence or possible infection

Continual dripping with shifting positions: overflow incontinence or sphincter impairment

Nocturia or incontinence associated with intercourse: urge incontinence

Painful urination: obstruction, infection, or urge incontinence

What are the known risk factors for urinary incontinence?

Childbearing; advanced age; obesity; family history; chronic cough; ascites; pelvic masses

What age-related changes contribute to the development of urinary incontinence?

1. The prevalence of detrusor overactivity increases with age
2. Total bladder capacity diminishes
3. Urinary flow rate decreases
4. Postvoiding residual increases
5. The urethral mucosal epithelium becomes atrophic secondary to low estrogen levels

Is incontinence a part of normal aging?

No, however the prevalence and severity of incontinence increases with age. Thus, patients should be queried regarding the presence of these symptoms. Infection should be ruled out when there is any sudden change or onset of incontinence symptoms

PATIENT EVALUATION

What are the pertinent factors to ask about when taking a history?

Review of symptoms: how often leakage occurs, what provokes urine loss, how much urine is leaked, what

	<p>makes the problem better/worse, any prior treatment that has been tried</p> <p>General medical history: systemic illnesses (such as diabetes, vascular insufficiency, chronic pulmonary disease, or any neurologic condition)</p> <p>Past surgical history</p> <p>Current medications</p>
<p>What types of medications can sometimes cause urinary incontinence?</p>	<p>Benzodiazepines; α-agonists (OTC cold medications); α-antagonists (antihypertensives); calcium channel blockers; ACE inhibitors (by increasing cough); alcohol</p>
<p>What should be done on physical examination and what tests should be done?</p>	<p>General examination: look for signs of other medical problems as well as for alertness/functional status</p> <p>Pelvic examination: look for signs of atrophy, infection, fistulae, diverticulum, pelvic organ prolapse</p> <p>Urinalysis: routine urinalysis and culture, urine cytology (in women over 50 with urinary tract irritation or hematuria)</p> <p>Labs: metabolic panel (renal function, glucose, calcium), vitamin B₁₂ (in the elderly), glucose</p> <p>Measurement of postvoid residual urine (especially in high-risk patients)</p> <p>Give out a frequency/volume bladder diary for the patient to record her symptoms</p>
<p>What are normal values for the following?</p>	
<p>Daily urine output:</p>	1500–2500 mL
<p>Average void volume:</p>	250 mL
<p>Functional bladder capacity:</p>	400–600 mL
<p>Voids per day:</p>	7–8 times
<p>Is routine urodynamic testing indicated in the evaluation of urinary incontinence?</p>	<p>No. While it is the gold standard, it is not always necessary to make the diagnosis. It should be considered if empiric therapy has failed or prior to any surgical intervention</p>

Describe the following urodynamic tests:

Multichannel urodynamic testing: produces a biophysical profile of the patient's bladder and urethra. Used for patients with mixed incontinence, prior bladder surgeries, or suspected intrinsic sphincter deficiency

Cystometry: measures the pressure-volume relationship of the bladder.

Filling cystometry measures bladder pressure during filling; fluid is infused while pressure is continually monitored. The point of urgency, point of leakage, and total capacity are recorded. **Voiding cystometry** (also known as pressure-flow study) measures the urine flow rate and correlates it with the detrusor pressure

Urethral pressure profile: measures urethral closure and sphincteric integrity

During a bladder filling test, what type of incontinence does a woman have if:

She feels urinary urgency and involuntary voiding upon filling?

Urge incontinence

She leaks small amounts upon coughing?

Stress incontinence

STRESS INCONTINENCE

What are the typical symptoms of stress incontinence?

Leakage of a small amount of urine with any activity that increases intra-abdominal pressure (coughing, sneezing, laughing, etc)

What is the prevalence of stress incontinence?

20–30% of women complain of stress incontinence. It is the **most common type** of urinary incontinence in younger women and the second most common cause of incontinence in older women

Describe the pathophysiology of stress incontinence

With weakening of the pelvic muscles and endofascia, **urethral support is impaired**. With rising intra-abdominal pressure (because of sneezing, coughing, or exercise), the intravesicular pressure rises higher

What are the risk factors for the development of stress incontinence?

than the pressure of the urethral closure mechanism, leading to urine leakage

Advanced age; Denervation and/or connective tissue injuries from childbirth or surgery; **previous surgery for prolapse and/or incontinence; low estrogen levels; chronically increased intra-abdominal pressure; collagen vascular disorders**

What is intrinsic urethral sphincter deficiency?

A less common cause of stress incontinence wherein there is **complete failure of urethral closure**; usually because of **scarring** from a past operation or occasionally from severe atrophy in postmenopausal women

Does demonstration of urine leakage during coughing always indicate stress incontinence?

No. It could also be detrusor overactivity, detrusor hyperactivity with impaired contractility (DHIC), or overflow incontinence

What is the Q-tip test?

Insertion of a cotton swab into the urethra at the bladder neck. It is positive (indicates stress incontinence) if the angle of the cotton swab changes by more than 30° with strain

How does the timing between the increased pressure and incontinence affect the diagnosis?

Immediate leakage with pressure suggests **stress incontinence**. A **delay** between the maneuver and the leakage suggests **detrusor overactivity**

What is the treatment for stress incontinence?

Nonsurgical: muscle strengthening, pessaries, medications (usually ineffective)

Surgical: **pubovesical sling or collagen injections**

URGE INCONTINENCE

What are the typical symptoms of urge incontinence?

Leakage of urine preceded by a feeling of urgency

How common is urge incontinence?

It is the second most common form of incontinence and the most common form of incontinence in older women

Describe the pathophysiology of urge incontinence

Involuntary **detrusor overactivity** or bladder contractions

What are some of the causes of detrusor overactivity?

Advanced age; disruption of inhibitory pathways (stroke, cervical stenosis, diabetes, Alzheimer disease, Parkinson disease); **bladder irritation** (infection, stones, cancer); **idiopathic**

What is interstitial cystitis?

A spectrum of symptoms, characterized by **urgency** and **frequent voiding of small amounts** of urine and often associated with dysuria or pain. Most commonly diagnosed in younger women

What is DHIC?

Detrusor hyperactivity with impaired contractility. It is characterized by both urgency and an elevated post-void residual

What is the treatment for urge incontinence?

Behavioral: bladder retraining

Medication: anticholinergics and/or β -adrenergics; botox injections into the detrusor

Surgical: neuromodulation implants; surgical denervation (usually ineffective); correction pelvic organ prolapse if significant

MIXED INCONTINENCE

What are the symptoms of mixed incontinence?

Leakage associated with urgency and with increased intra-abdominal pressure

Describe the pathophysiology of mixed incontinence

Combination of both **detrusor overactivity and stress incontinence**, sometimes compounded by impaired **urethral sphincter function**

What is the treatment for mixed incontinence?

Individualized care based on the primary complaint and the individual's characteristics

OVERFLOW INCONTINENCE

What are the symptoms of overflow incontinence?

Continual **dribbling** associated with incomplete bladder emptying

Describe the pathophysiology of overflow incontinence

There is **underactivity of the detrusor muscle** resulting in failure of full bladder emptying. This leaves a **large postvoid residual** which leads to leakage with increased intra-abdominal pressure. It can also be caused by bladder outlet **obstruction**, usually in women with significant pelvic organ prolapse

What are some of the causes of underactivity of the detrusor muscle?

Neurogenic bladder (e.g., from diabetes, vitamin B₁₂ deficiency, Parkinson's disease); prior bladder surgery; chronic, long-standing overdistention

TREATMENT

What are the three categories of treatment options for urinary incontinence?

Lifestyle/behavioral changes; medications; surgery (should be tried in that order)

What types of lifestyle changes are associated with an improvement in urinary incontinence?

1. Limiting fluid intake (no more than 2 L/day)
2. Avoidance of caffeine and alcohol
3. Smoking cessation
4. Treatment of cough (for stress incontinence)
5. Treatment of constipation
6. Weight loss and increased physical activity

What types of behavioral changes are associated with an improvement in urinary incontinence?

Bladder training; pelvic muscle exercises

Describe bladder training

Timed voiding while awake, with gradually increasing intervals between voids; **relaxation and distractive techniques** to suppress urgency in between voids

What is the theory behind bladder training?

The central nervous system and pelvic musculature can be **trained to inhibit detrusor contractions by becoming gradually accustomed to accommodating larger bladder volumes**

What is supplemental biofeedback?

Vaginal or anorectal biofeedback mechanisms used to help patients identify and use their pelvic musculature in response to a sense of urgency. It can be used as a primary therapy or as a supplement to either medication or bladder training therapy

For which type of incontinence is bladder training most useful?

Urge incontinence

What are pelvic muscle exercises?

Also known as **Kegel** exercises, they involve isometric repetitions of exercises in order to strengthen the levator ani muscles, which support the pelvic organs and significantly contribute to urethral closure mechanisms

For which types of incontinence are pelvic muscle exercises effective?

Urge, stress, or mixed incontinence

In which types of incontinence is pharmacotherapy warranted?

Urge and mixed incontinence. They are often ineffective in treating stress incontinence

What types of medications are used to treat urinary incontinence?

Antimuscarinics: oxybutynin; tolterodine; trospium; solifenacin; darifenacin

α -adrenergic agonists: duloxetine; tricyclic antidepressants (TCAs) (imipramine); topical estrogens; botulinum toxin

What is a continence pessary?

A device that can be used to treat incontinence because of pelvic floor prolapse

What types of surgical options are effective for urge incontinence?

Sacral nerve modulation; augmentation cystoplasty

What types of surgical options are effective for stress incontinence?

1. Minimally invasive midurethral anti-incontinence procedures

Should an indwelling urinary catheter be used to alleviate symptoms of incontinence?	<ol style="list-style-type: none"> 2. Retropubic bladder neck suspension (Burch or Marshall–Marchetti–Krantz [MMK] colposuspension) 3. Sling procedures 4. Periurethral bulking injections <p>As these are associated with high morbidity, they should only be considered in select cases as a last resort</p>
Describe the midurethral anti-incontinence procedures	<p>These are minimally invasive, vaginal surgical procedures using synthetic mesh material placed at the level of the midurethra in a tension-free manner</p>
What complications are associated with the midurethral anti-incontinence procedures?	<p>Postoperative voiding dysfunction Urinary retention Erosion of mesh material</p>
Describe the retropubic bladder neck suspension procedures	<p>An abdominal procedure that uses the periurethral vaginal tissue to support the urethrovesical junction via sutures to Cooper’s ligament (Burch) or to the pubic symphysis (MMK)</p>
What complications are associated with the retropubic bladder neck suspension procedure?	<p>Enterocoele formation; postoperative voiding dysfunction; urinary retention</p>
Describe suburethral sling procedures	<p>Elevation of the urethrovesical junction and introduction of mechanical compression of the urethra using autologous or synthetic graft material</p>
What are the complications of a sling procedure?	<p>Postoperative voiding dysfunction; urinary retention; erosion of graft material</p>
Describe periurethral bulking agents procedures	<p>Elevation of the urethrovesical junction and introduction of mechanical compression of the urethra using autologous or synthetic graft material</p>

This page intentionally left blank

Reproductive Endocrinology and Infertility

Adolescent Development

INTRODUCTION

What types of changes occur during puberty?

1. Development of secondary sexual characteristics
2. Growth
3. Changes in body composition
4. Achievement of fertility

What is meant by the following terms?

1. **Thelarche:** breast development
2. **Pubarche:** development of pubic hair
3. **Menarche:** beginning of menses

What is the typical sequence of events in puberty for girls?

Thelarche (breast development) → **pubarche** (pubic hair growth) → peak growth spurt → **menarche**

What is the average time duration of puberty and when does it start?

4.5 years is the average duration of puberty. On an average, thelarche begins at 10 years and menarche at 12 years of age

What are the Tanner stages?

A **staging system** for the sequence of **pubertal development**. Divides the development of secondary sex characteristics into five stages

Describe the Tanner stages for breast development in females

1. Stage 1: Prepubertal
2. Stage 2: Elevation of breast and papilla; enlargement of areola; breast bud stage
3. Stage 3: Further enlargement of breast and areola; increased glandular tissue
4. Stage 4: Areola and papilla project above the breast
5. Stage 5: Recession of areola, projection of papilla only, mature stage

Describe the Tanner stages for pubic hair development

1. Stage 1: Velus hairs only, prepubertal
2. Stage 2: Sparse growth of slightly pigmented hair
3. Stage 3: Hair growth spreads and becomes darker, coarser, and more curly
4. Stage 4: Adult hair type, but no spread to medial thigh
5. Stage 5: Medial spread with inverse triangle distribution

What happens to the external genitalia during puberty?

Estrogen stimulation leads to thickening of the mons pubis, growth of the labia majora, rounding of the labia minora, thickening of the hymen, elongation of the vagina

What happens to the uterus during puberty?

The myometrium thickens, altering the uterine shape. The endometrium thickens gradually and then more rapidly before menarche

Describe the pattern of growth that occurs in puberty

Growth affects both the trunk and the limbs, with the limbs (especially the distal portions thereof) growing before the rest of the body. Girls have their growth spurt before boys and the peak growth occurs approximately 6 months prior to menarche

What happens to the long bones during puberty?

Long bones lengthen and their epiphyses close

What are some of the unwelcome physiological changes that occur as a result of puberty?

Acne; psychological changes—boys develop a more positive and girls experience a diminished self-image; scoliosis; myopia

CONGENITAL ANOMALIES

What is suggested by clitoral enlargement?

Exposure to high levels of **androgens**. Proper evaluation to exclude any intersex conditions is warranted

What is imperforate hymen?

A persistent urogenital membrane

What are the symptoms of an imperforate hymen?

It is rare to make this diagnosis before puberty. Symptoms include **primary amenorrhea, pelvic pain,** and difficulty with urination/defecation

What are some of the potential complications associated with an untreated imperforate hymen?

Endometriosis and vaginal adenosis

What is a vaginal septum?

An absence of fusion of the müllerian ducts and/or urogenital sinuses leading to an obstructive lesion in the vagina. This leads to obstructive symptoms similar to those of an imperforate hymen

What is vaginal agenesis?

Usually associated with Mayer-Rokitansky-Kuster-Hauser syndrome. It involves normal-appearing external genitalia with agenesis of the vagina superior to the hymen. It is usually associated with uterine and cervical agenesis. Also associated with testicular feminization, yet these patients have testes instead of ovaries and need to have them removed

What are the symptoms of vaginal agenesis?

Primary amenorrhea and pelvic pain

How is vaginal agenesis treated?

Creation of a vagina by use of either **serial vaginal dilation** or surgical reconstruction with a **vaginoplasty procedure**

What are the various congenital anomalies of the uterus and how common are each? Describe each

Arcuate uterus (15%): small septum with minimal cavity indentation
 Incomplete septum (13%): partial fusion resulting in a septum that does not completely divide the horns

<p>What are the symptoms of these uterine anomalies?</p>	<p>Complete uterine septum (9%): partial fusion that completely divides the uterine horns</p> <p>Bicornate uterus (37%): partial fusion leading to a midline septum (can be partial or complete)</p> <p>Uterine didelphys (11%): failure of fusion resulting in two separate uterine bodies</p> <p>Unicornuate uterus (4%): agenesis of one Müllerian duct, with an absence of the corresponding fallopian tube and round ligament</p>
<p>What does the palpation of an inguinal mass in an adolescent patient suggest?</p>	<p>Usually asymptomatic; can occasionally cause retention of menstrual flow or infertility</p>
<p>How should an aberrant gonad be managed?</p>	<p>Possible aberrant gonad (often with testicular elements)</p> <p>A karyotype should be done and a biopsy of the gonad should be done. If it is an ovary, it should be returned to the peritoneal cavity. If it is a testis, it should be removed</p>

ACCELERATED SEXUAL MATURATION

<p>What is premature thelarche and how is it managed?</p>	<p>Isolated development of breasts before 8 years of age. No intervention is required if other signs of precocious puberty do not develop</p>
<p>What is premature pubarche and how is it managed?</p>	<p>Isolated appearance of pubic or axillary hair before 6–7 years of age. It generally represents premature secretion of androgens from the adrenal gland. Evaluation of the adrenal and gonadal function should be done to exclude precocious puberty</p>
<p>What is premature menarche and how is it managed?</p>	<p>Isolated cyclic vaginal bleeding without any other signs of sexual development. It is usually related to increased end-organ sensitivity to estrogen and it does not require any intervention</p>

What is sexual precocity?	Onset of sexual maturation before 2.5 times the standard deviation of the normal age for that population
How is precocious puberty classified?	GnRH dependent and GnRH independent
What is GnRH-dependent precocious puberty?	Also called central precocious puberty (CPP); premature activation of the hypothalamic-pituitary axis with premature gonadotropin secretion
What is GnRH-independent precocious puberty?	Sex steroids are present independent of the release of pituitary gonadotropins. This is a pseudoprecocious puberty
What are some of the causes of GnRH-independent precocious puberty?	Congenital adrenal hyperplasia; tumors of the adrenal gland; tumors of the gonads; hCG-secreting tumors; McCune-Albright syndrome; Exogenous steroids
What is the workup for precocious puberty?	<ol style="list-style-type: none"> 1. History: age of onset, progression, family history 2. Physical examination (PE): growth velocity changes, acne, breast development, genital changes 3. Laboratory: luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, GnRH-stimulation test 4. Imaging: skeletal survey and bone scan to determine bone age, pelvic ultrasound, abdominal CT (of adrenals), brain MRI
What are the results of the laboratory tests in GnRH-dependent precocious puberty?	Normal pubertal range
What is the treatment for GnRH-dependent precocious puberty?	GnRH analogues, which induce down-regulation of the receptor function, creating an inhibition of the hypothalamic-pituitary-ovarian axis
What is McCune-Albright syndrome?	A genetic disorder with a classic triad of precocious puberty , polyostotic fibrous dysplasia , and café-au-lait skin lesions

DELAYED SEXUAL MATURATION

<p>What is delayed sexual maturation?</p>	<p>Absence of pubertal changes after 2.5 times the standard deviation of the mean age for a population (e.g., absence of thelarche by age 13 and menarche by age 15)</p>
<p>What is the proper evaluation for a patient with delayed sexual maturation?</p>	<ol style="list-style-type: none"> 1. History: previous growth patterns and pubertal development, other medical disorders 2. PE: height and weight, Tanner staging, pelvic examination (for congenital anomalies and obstruction) 3. Labs/imaging: vaginal smear (for cytohormonal evaluation), karyotype, pelvic ultrasound, FSH
<p>What does breast development signify?</p>	<p>Prior gonadal function</p>
<p>What does the absence of pubic hair signify?</p>	<p>Androgen insensitivity</p>
<p>What is the differential diagnosis of a patient with delayed menarche and adequate secondary sexual characteristics?</p>	<p>Pregnancy; anatomic genital abnormalities; inappropriate positive feedback; complete androgen insensitivity syndrome</p>
<p>How are patients with delayed menarche and inadequate secondary sexual characteristics classified?</p>	<p>By their FSH level</p>
<p>What is the differential diagnosis of a patient with delayed menarche and inadequate secondary sexual characteristics?</p>	<p>Low FSH: constitutional delay; weight loss (extreme dieting, drug abuse, extreme exercise); Kallman syndrome; pituitary destruction High FSH: Turner syndrome; Ovarian destruction (chemotherapy, radiation, infection, autoimmune); Resistant ovary syndrome</p>
<p>What is Kallman syndrome?</p>	<p>A rare genetic syndrome causing hypogonadotropic hypogonadism and anosmia</p>
<p>What is the differential diagnosis of a patient with delayed menarche and virilization?</p>	<ol style="list-style-type: none"> 1. Enzyme deficiency (such as 21α-hydroxylase deficiency) 2. Neoplasia 3. Male pseudohermaphroditism

Disorders of the Menstrual Cycle, Uterus, and Endometrium

PMS

What is premenstrual syndrome?

Premenstrual syndrome (PMS) is a cyclical pattern of **emotional, physical, and/or behavioral** changes that occur in the **luteal phase** of the menstrual cycle and remit during menses. Symptoms cause **significant disability**, and it is *not* an exacerbation of an underlying psychiatric disorder. It is best characterized as an abnormal response to normally fluctuating hormones. A more severe variant is described in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)* as **premenstrual dysphoric disorder**.

What is the incidence of PMS and in whom does it occur?

The reported incidence of PMS ranges from 5% to 90% of women. Approximately 70% have some symptoms of PMS and 5% have the more severe PMDD. It occurs mostly in women in their mid-20s to 40s

What are the symptoms of PMS?

PMS is characterized by various constellations of symptoms including at least one of the following which is temporally related to the menstrual cycle:

1. **Affective lability**
2. **Anxiety or tension**
3. **Depressed mood, hopelessness, or self-deprecating thoughts**
4. **Persistent anger or irritability**

Other symptoms include decreased interest/avoidance of usual activities, decreased productivity, lethargy, changes in appetite/cravings, reproducible patterns of physical complaints (such as headaches, weight gain, breast

How is PMS diagnosed?

tenderness), difficulty concentrating, sleep disturbances (hypersomnia or insomnia), and a sense of being overwhelmed

The patient is asked to keep a daily **menstrual diary** to document her symptoms and their severity. The patient must demonstrate at least 5 of the above symptoms. She also must demonstrate a **symptom-free follicular phase** and problems with notable changes in the **luteal phase**. A comprehensive history and physical examination must be done to rule out any other illness

What are the treatment options for PMS?

Education of the patient and her family, **dietary changes** (emphasizing fresh foods), **exercise**, medications to **prevent ovulation** (such as oral contraceptive pills), **progesterone suppositories**, **NSAIDs**, **diuretics**, **anxiolytic/antidepressant medications**, and **vitamin B₆**. **GnRH agonists** and surgical intervention are a last resort for severe refractory PMS

Dysmenorrhea

What is dysmenorrhea and what are the two types?

Dysmenorrhea is **pain with menses** either due to **pelvic pathology** (**secondary dysmenorrhea**) or **without pelvic pathology** (**primary dysmenorrhea**)

What is the incidence of dysmenorrhea and in whom does it occur?

Dysmenorrhea occurs in approximately **15% to 75% of women**. **Primary dysmenorrhea** is most common in **younger women**, whereas the incidence of **secondary dysmenorrhea** increases with age

What is the etiology of primary dysmenorrhea?

Primary dysmenorrhea is caused by an excess of **prostaglandin F_{2α}** produced in the endometrium. It stimulates smooth muscle, leading to uterine contractions and uterine ischemia

What are the clinical signs and symptoms of primary dysmenorrhea?

Pain typically begins **at menstruation** and subsides after

	<p>1–3 days. It is characterized as cramping, labor-like pain, typically in the lower abdomen and suprapubic area, radiating to the back. The pain can be associated with nausea, vomiting, and diarrhea. Fatigue and headache are often also associated. In general, the physical examination is normal</p>
<p>What is the treatment of primary dysmenorrhea?</p>	<p>NSAID therapy is first line. Other options include heat, exercise, and oral contraceptives. In severe, refractory cases, presacral neurectomy is a last resort. However, secondary dysmenorrhea should be suspected if symptoms are refractory to NSAIDs or OCPs</p>
<p>How do NSAIDs and OCPs work to treat dysmenorrhea?</p>	<p>NSAIDs work by inhibiting prostaglandin production. OCPs work by decreasing prostaglandin production through suppression of ovulation</p>
<p>What are the gynecologic causes of secondary dysmenorrhea?</p>	<p>Extrauterine causes: Endometriosis Neoplasm Inflammation (e.g., PID) Adhesions Intramural causes: Adenomyosis Leiomyomata Intrauterine causes: Leiomyomata Polyps IUDs Infection Cervical stenosis/lesions</p>
<p>For a patient with dysmenorrhea, what primary diagnosis do each of the following findings suggest:</p> <ul style="list-style-type: none"> Uterine asymmetry Symmetrical enlargement of uterus Painful nodules in posterior cul-de-sac Restricted motion of the uterus 	<ul style="list-style-type: none"> Myomas or other tumors Adenomyosis Endometriosis Endometriosis or pelvic scarring/adhesions from prior infection
<p>What is the treatment for secondary dysmenorrhea?</p>	<p>Treatment of the underlying condition is the primary modality.</p>

What other underlying diseases are exacerbated by menstruation?

However, when this is not possible, **NSAIDs** and low-dose **oral contraceptives** offer symptomatic therapy

Women with **migraines**, tension, or vascular can have increased frequency of headaches at the time of menstruation. Asthma patients occasionally report worsening of symptoms with menses. These effects are thought to be due to increased prostaglandin production

DYSFUNCTIONAL UTERINE BLEEDING

Describe what is meant by the following patterns of abnormal bleeding:

Hypomenorrhea

A **decreased amount** of bleeding at **regular intervals**

Intermenstrual bleeding

Bleeding of variable amounts occurring **between regular menstrual periods**

Menorrhagia

Prolonged or excessive uterine bleeding occurring at **regular intervals**. Synonymous with **hypermenorrhea**

Metrorrhagia

Uterine bleeding occurring at **irregular intervals**

Polymenorrhea

Uterine bleeding occurring at **intervals of less than 21 days**

Oligomenorrhea

Menstrual bleeding at intervals of **longer than 35 days**

What is dysfunctional uterine bleeding?

Dysfunctional uterine bleeding (DUB) is a diagnosis of exclusion that consists of **irregular menstruation unrelated to anatomic lesions***

What is the physiologic cause of DUB?

Women with DUB may be in a **hyperestrogenic state**. Because of

*Patients may also bleed from lack of estrogen (atrophy), or too much or too little progesterone

	<p>anovulation, these women have noncyclic estrogen levels that stimulate endometrial growth. Once the endometrium outgrows its blood supply, it sloughs off, leading to irregular, often heavy bleeding</p>
How can you clinically differentiate between ovulatory cycles and anovulatory cycles?	<p>Ovulatory cycles are regular in their length, duration of menses, and amount of bleeding. Anovulatory cycle bleeding is much more irregular</p>
What must be ruled out before the diagnosis of DUB can be made?	<p>Organic causes of abnormal bleeding such as leiomyomas, inflammation (cervicitis, endometritis), carcinomas, cervical/endometrial polyps, vaginal lesions, blood dyscrasia, and iatrogenic causes (IUDs, fertility drugs)</p> <p>Note: Abnormal uterine bleeding in a postmenopausal woman is cancer until proven otherwise</p>
What conditions are often associated with DUB?	<p>Obesity Polycystic ovarian disease Adrenal hyperplasia Perimenopause</p>
In whom does DUB occur?	<p>Half of all patients with DUB are peri- or postmenopausal. Another 20% are adolescents</p>
What type of bleeding pattern is seen in women with high estrogen levels and with low estrogen levels?	<p>High levels of estrogen cause the endometrium to build up beyond its blood supply. This leads to prolonged amenorrhea followed by profuse bleeding</p> <p>Low levels of estrogen cause intermittent spotting of a prolonged duration, but can cause heavy bleeding from denuded, dysynchronous endometrium</p>
What is the initial workup for abnormal bleeding?	<p>All women with abnormal uterine bleeding need a thorough menstrual</p>

In each scenario, what additional tests should be done and why?	history, bimanual examination, and Pap smear. A TSH may also be ordered as thyroid disease can affect bleeding patterns.
An ovulatory woman	Transvaginal ultrasound, sonohysterogram, or D&C with hysteroscopy (gold standard) should be done to rule out anatomic causes of abnormal bleeding
An anovulatory woman	<p>β-hCG: pregnancy</p> <p>CBC: infection</p> <p>Coagulation profile: blood dyscrasia</p> <p>Endocrine tests (FSH, LH, TSH, prolactin): premature ovarian failure, polycystic ovarian syndrome (PCOS), prolactinoma, hypothyroidism</p>
All postmenopausal women	Endometrial biopsy: endometrial carcinoma
What are the risks of unrecognized DUB?	Hemorrhage, endometrial hyperplasia, and/or cancer
What is the goal of treatment for DUB?	To convert the proliferative endometrium into secretory endometrium
How is anovulatory DUB treated?	<p>Anovulatory DUB is treated with OCPs or cyclic progesterone therapy (such as medroxyprogesterone daily for 10 days of each month)</p> <p>DUB associated with heavy bleeding is treated with high-dose estrogen or progestins. Endometrial ablation can also be used for treatment if patient is refractory to oral and IV estrogen therapy. A D&C can be done if patient is hemodynamically unstable</p>
How is ovulatory DUB treated?	Ovulatory DUB is treated primarily with OCPs

AMENORRHEA

What is amenorrhea and what are the two subtypes?

Amenorrhea is the **absence of menstruation**. It can be subdivided into two subtypes. If a woman has **never menstruated**, she has **primary amenorrhea**, whereas if she has previously menstruated but has **failed to menstruate within 6 months or for 3 cycle intervals** she has **secondary amenorrhea**

What are the four main etiologies of amenorrhea?

Pregnancy: Since it is the most common cause of amenorrhea, this is an essential diagnosis to exclude via a pregnancy test

Hypothalamic-pituitary dysfunction: When the pulsatile secretion of GnRH is altered, the pituitary gland ceases secreting FSH and LH. This results in the absence of regular ovulation and menstruation. Most hypothalamic-pituitary dysfunction is of functional origin

Ovarian dysfunction: The ovarian follicles are resistant to stimulation by FSH and LH

Anatomic dysfunction: Genital outflow tract obstruction can be congenital or from the development of scar tissue. The most common cause of obstruction is scarring of the uterine cavity (Asherman syndrome)

What are the common causes of hypothalamic-pituitary dysfunction?

Functional: Weight loss

Excessive exercise

Obesity

Drug induced: Marijuana

Sedatives

Malignancy: Prolactinoma

Craniopharyngioma

Hypothalamic hamartoma

Psychogenic: Anxiety

Anorexia

Other: Head injury

<p>How is amenorrhea due to hypothalamic-pituitary dysfunction diagnosed?</p>	<p>Chronic disease Empty sella syndrome Sheehan syndrome</p> <p>A history of any of the above conditions suggests this etiology. However, low serum levels of FSH and LH are diagnostic. Prolactin is usually normal, except in a prolactinoma</p>
<p>What are the common causes of ovarian dysfunction that lead to primary amenorrhea?</p>	<p>Gonadal dysgenesis: Turner syndrome Mosaicism 17-hydroxylase deficiency</p>
<p>What are the common causes of ovarian dysfunction that lead to secondary amenorrhea?</p>	<p>Gonadal agenesis</p> <p>Pregnancy Menopause Autoimmune ovarian failure (Blizzard syndrome) Premature ovarian failure Alkylating chemotherapy</p>
<p>How is amenorrhea due to ovarian dysfunction diagnosed?</p>	<p>As the ovaries fail, serum FSH level rises</p>
<p>How can estrogen deficiency due to hypothalamic-pituitary failure be differentiated from that due to ovarian failure?</p>	<p>Estrogen deficiency secondary to hypothalamic-pituitary failure usually does not cause hot flashes. Ovarian failure may cause hot flashes</p>
<p>What genital outflow obstructions lead to primary amenorrhea?</p>	<p>Müllerian anomalies (e.g., imperforate hymen) Müllerian agenesis</p>
<p>What genital outflow obstructions lead to secondary amenorrhea?</p>	<p>Asherman syndrome</p>
<p>What laboratory tests are indicated in the evaluation of a patient with secondary amenorrhea?</p>	<p>β-hCG TSH Prolactin FSH Progesterone challenge test</p>
<p>What diagnoses are suggested by the following lab results:</p>	
<p>↑ β-hCG?</p>	<p>Pregnancy</p>
<p>↑ TSH?</p>	<p>Hypothyroidism</p>

↑ Prolactin?

Hyperprolactinemia from a pituitary tumor, idiopathic, or due to medications (e.g., dopamine antagonists)

↑ FSH ?

Postmenopausal state or ovarian failure

↓ FSH ?

Hypothalamic disorder or pituitary dysfunction

Key: In general, if the gonadotropins are high, the problem is at the level of the ovary. If the gonadotropins are low, the problem is in the hypothalamus or pituitary

What further test must be done if the patient is found to be hypogonadotropic?

Imaging of the sella turcica, either via **CT scan** or **MRI**. Abnormal imaging suggests a pituitary tumor, whereas normal imaging is assumed to signify a hypothalamic problem

What further test must be done if the patient is found to be hypergonadotropic?

A **karyotype** to diagnose mosaicism with a Y chromosome

What is the progesterone challenge test and what is the procedure?

A test done if all the preceding lab test are found to be normal. It assesses if an amenorrheic patient has **adequate estrogen** levels and an **intact outflow tract**. The patient is given oral **progesterone**. **Withdrawal bleeding** is observed after a few days

What does withdrawal bleeding in the progesterone challenge test indicate?

Withdrawal bleeding indicates that the patient is **chronically anovulatory** or **oligo-ovulatory**. Ovulation had not occurred and so no endogenous progesterone was made. The exogenous progesterone allowed menses to occur after withdrawal. Causes of anovulation include hypothalamic dysfunction, polycystic ovarian syndrome, Cushing syndrome, or an ovarian tumor

What does the absence of withdrawal bleeding in the progesterone challenge test indicate?

The absence of withdrawal bleeding indicates that the patient is either **hypoestrogenic** or has an **anatomic obstruction**

What is the next step if withdrawal bleeding does not occur?

An **estrogen-progesterone test, imaging**

What is an estrogen-progesterone test and what do the results indicate?

The estradiol and progesterone test entails giving a small dose of **estradiol daily for 28 days** and adding **progesterone for the last 7 days**. This differentiates a hypoestrogenic state from an anatomic obstruction. **Withdrawal bleeding** indicates **inadequate estrogen**. If withdrawal bleeding does **not** occur within a few days, **anatomic obstruction** is the diagnosis

What is the treatment for amenorrhea due to the following?

Hypothalamic anovulation?

Induction of ovulation with **gonadotropins if pregnancy desired, otherwise OCPs**

A prolactinoma?

Dopamine agonists. Rarely, surgical excision

Premature ovarian failure?

Exogenous **estrogen** replacement

Genital tract obstruction?

Surgery to restore genital tract integrity

Infertility

EVALUATION OF INFERTILITY

How is infertility defined?

It is defined as the **inability to conceive after 12 months** of frequent intercourse without use of contraception

What is the definition of fecundability?

It is the probability of achieving pregnancy in one menstrual cycle

What is the prevalence of infertility?

About 13%, with a range from 7% to 28% depending on the age of the woman

What is the difference between primary and secondary infertility?

Primary infertility pertains to those who have never conceived, whereas secondary infertility is applied to those who have conceived in the past

What percent of infertility is because of male factors, female factors, both and unknown etiology?

1. Male factor (23%)
2. Female factors (40–50%)
3. Both (27%)
4. Unknown etiology (15–20%)

What are the most common factors that comprise female factor infertility?

- Ovulatory disorders (25%)
- Endometriosis (15%)
- Pelvic adhesions (12%)
- Tubal blockage (11%)
- Other tubal abnormalities (11%)
- Hyperprolactinemia (7%)

What is the first test to perform for infertility if the history and physical from the infertile couple offer no clues?

Semen analysis

What are the relevant characteristics of normal semen?

Volume: 2–5 mL

Concentration: ≥ 20 million per mL

Motility: 50% should be motile, 25% should have rapid progressive motility

Morphology: lower limit of normal is 10–15%

What hormone tests should be ordered if the sperm concentration is less than 5 million per ml?

Follicle-stimulating hormone (FSH), luteinizing hormone (LH), and testosterone

What is the next step in evaluation of infertility if the semen analysis is normal?

Ovulatory function

What percent of female factor fertility can be attributed to ovulatory dysfunction?

25%

What are risk factors for premature ovarian failure?

Exposure to cytotoxic drugs, pelvic radiation therapy, autoimmune diseases, previous ovarian surgery, family history of early menopause

What are the methods to evaluate ovulatory function?

Menstrual history, basal-body-temperature (BBT) chart, serum progesterone levels in the mid-luteal phase, sonogram, and/or endometrial biopsy

How is anovulation classified and what is the mechanism of that type of anovulation?

1. **Hypogonadotropic hypogonadal anovulation (hypothalamic amenorrhea)**—low secretion of GnRH or pituitary unresponsiveness to GnRH, resulting in low serum FSH and estradiol levels. Can be seen in stress- or exercise-related amenorrhea, anorexia nervosa, Kallman syndrome, or CNS tumors

2. **Normogonadotropic normo-estrogenic anovulation**—most prevalent subtype. Normal GnRH secretion, estradiol and FSH levels with either normal or elevated LH concentrations. Includes women with polycystic ovary syndrome (PCOS)
3. **Hypergonadotropic hypoenestrogenic anovulation**—ovarian failure is the primary cause
4. **Hyperprolactinemic anovulation**—hyperprolactinemia inhibits gonadotropin and, therefore, estrogen secretion

What other endocrine abnormalities may result in infertility?

Hypothyroidism, androgen excess, diabetes/obesity, starvation

How do each of these methods help evaluate a woman's ovulatory function?

A **menstrual cycle history** that reveals amenorrhea indicates anovulation and a cause of infertility, whereas a normal monthly menstrual cycle is a strong indicator of normal ovulation

A **basal-body-temperature chart** that indicates normal ovulation is reflected by a biphasic curve during ovulation, with a rise in temperature by 0.5–1.0°F during the luteal phase (because of an increased level of progesterone). The highest temperature in one menstrual cycle occurs during ovulation

Urinary LH kits. Over-the-counter monitors that detect the LH surge in the urine. This method allows for appropriate timing of intercourse

Measurement of a **serum progesterone level in the mid-luteal phase** (18–24 days after the onset of menses) is the definitive confirmation for ovulation. Normal levels are between >5 ng/mL

Sonogram and/or endometrial biopsy may be used to evaluate whether the

If a female's ovulatory function is intact, what other factors may cause female factor infertility and what is their relative frequency?

endometrium is optimal for normal implantation of the egg

1. Tubal damage (14%)
2. Endometriosis (9%)
3. Coital problems (5%)
4. Cervical factor (3%)

What is the major cause of tubal infertility and by what method may tubal abnormalities and/or other uterine cavity abnormalities be evaluated?

Pelvic inflammatory disease. Hysterosalpingogram should be obtained for evaluation

What are other causes of tubal infertility?

Prior abdominal or pelvic surgery, ectopic pregnancy, severe endometriosis

When should laparoscopy be used?

It should be considered for couples with otherwise unexplained infertility or in women with known or suspected endometriosis and/or pelvic adhesions

How should a woman who presents with infertility and early stage endometriosis be treated for fertility?

Surgical resection of endometriosis lesions and other pelvic adhesions, followed by a trial of clomiphene or gonadotropin plus IUI (intrauterine insemination)

What is meant by assessing a female's ovarian reserve and what are the most commonly used tests to evaluate this?

This assessment is to determine quantity and possibly quality of remaining oocyte pool. The most commonly used tests are the day 3 FSH test and the clomiphene citrate challenge test (CCCT)

TREATMENT OF INFERTILITY

What is the general approach to treatment of infertility?

A stepwise approach, from least to most invasive (and expensive). It may start with use of clomiphene citrate to induction with gonadotropins, and then to in vitro fertilization (IVF). For patients with anovulation, the goal is monofollicular development with ovulation. For patients over 35 and those with unexplained infertility, the goal may be 2–4 follicles, with ovarian stimulation

What modifications in lifestyle habits may improve fertility?

Smoking and alcohol cessation, decrease in caffeine consumption,

What is clomiphene citrate and in which patients is it used?

increased frequency of coitus. Maintaining a healthy weight and diet not only helps with conception but predicts a healthier pregnancy

When is induction with human menopausal gonadotropins (hMG) indicated?

It is a selective estrogen receptor modulator (SERM) that ultimately increases gonadotropin release. It is the agent of choice for women less than 36 years of age and in normogonadotrophic normo-estrogenic anovulation. It has been effective in inducing ovulation for 60–85% of anovulatory women

How is follicle development and ovulation induced in women in hypogonadotropic hypogonadal anovulation with normal pituitary function?

Subcutaneous injection of hMG, specifically FSH, is used in women who have failed with clomiphene and in women with hypothalamic amenorrhea with hypopituitarism

What agent should be used in women who have anovulation because of hyperprolactinemia?

Pulsatile administration of GnRH stimulates endogenous production of FSH and LH

Do these induction agents have an associated risk for ovarian cancer and breast cancer?

Dopamine agonists such as bromocriptine or cabergoline

What is intrauterine insemination (IUI)?

Although historical evidence may indicate an association between clomiphene use and ovarian cancer, more recent well-controlled studies do not support the concept of increased risk. However, it is recommended that induction with clomiphene be limited to 12 cycles. There has been no reported risk between fertility drugs and breast cancer

It consists of collecting a specimen of ejaculated sperm, followed by washing and concentration of the sperm. The concentrated sperm is then injected directly into the upper uterine cavity using a small catheter through the cervix. The pregnancy rate is 4–6% per cycle when used alone. It is frequently used in combination with ovulation induction to achieve pregnancy rates of 5–15%

ASSISTED REPRODUCTIVE TECHNOLOGIES

What are the different modalities of assisted reproductive technologies (ARTs)?

1. In vitro fertilization-embryo transfer (IVF-ET)
2. Intracytoplasmic sperm injection (ICSI)
3. Gamete intra-fallopian tube transfer (GIFT)
4. Zygote intra-fallopian tube transfer (ZIFT)
5. Oocyte donation

What is the mechanism of each of the following technologies and their success rate of pregnancy?

IVF-ET: The female's ovary is hyperstimulated by daily FSH injections. Multiple mature eggs are collected by a transvaginal procedure and combined with sperm in the laboratory to allow fertilization to occur. After fertilization selected embryos are placed in the female's uterus to allow development. Pregnancy is achieved 28% of the time and 82% result in one or more births

ICSI: This procedure combines the technique of IVF and involves taking a single sperm from the male partner and injecting it directly into the cytoplasm of the egg in the laboratory. It is commonly used as treatment for male infertility factor. Overall fertilization rate is 60%

Osteoporosis

What is osteoporosis?

A disorder of the skeleton that involves **low bone mass** and **micro-architectural disruption**, both of which lead to skeletal fragility and increased fracture risk

What is the prevalence of osteoporosis?

Over 30% of women over 50 years have radiographic evidence of osteoporosis; over 44 million men and women are currently diagnosed with osteoporosis in the United States

Who does osteoporosis affect?	Predominately thin, postmenopausal women and more often Caucasian and Asian women
Why does osteoporosis affect women more than men?	Women have a lower peak bone mass and have accelerated bone loss after menopause
When does bone mass peak?	At age 30–35
What is the pathogenesis of osteoporosis?	Bone loss, which is a result of a mismatch between bone resorption and bone formation. Most is a result of either age-related or menopause-related bone loss
How does osteoporosis typically present?	Often with hip fracture , a vertebral compression fracture (leading to kyphosis), or a wrist fracture after minimal trauma
What is the differential diagnosis of osteoporosis?	Osteomalacia Hyperparathyroidism Multiple myeloma Pathological fracture secondary to metastatic cancer
What is “high turnover” osteoporosis and what are the causes?	Osteoporosis primarily secondary to increased bone resorption Caused by estrogen deficiency , hyperparathyroidism, hyperthyroidism, hypogonadism, cyclosporine, heparin
What is “low turnover” osteoporosis and what are the causes?	Osteoporosis primarily secondary to decreased bone formation Caused by advanced age , liver disease, heparin
What drug causes both decreased formation and increased resorption of bone, leading to osteoporosis?	Glucocorticoids
What drugs are known to cause osteoporosis?	Glucocorticoids; heparin; medroxyprogesterone acetate; vitamin A; certain retinoids
Describe age-related bone loss	Loss of both the cortical and trabecular bone that begins around age 30–40 in both men and women; partially because of decreased calcium absorption

Describe menopause-related bone loss	Loss of all but especially trabecular bone that occurs for 10 years beginning after the onset of menopause because of the decline in estrogen levels
Can estrogen be used to prevent menopause-related bone loss?	Yes, however it is not first-line therapy because of its cardiovascular and breast cancer risks
What is the relationship between lactation and bone loss?	Lactation is associated with a 1–4% bone loss; however, it is regained after lactation is completed. There is no association between osteoporosis and lactation earlier in life
What is the relationship between calcium and osteoporosis?	Calcium has been shown to reduce bone loss when a, 1000–1500 mg supplement is taken by postmenopausal women daily
How is osteoporosis diagnosed?	Either by the presence of a fragility fracture or by testing for bone mineral density (BMD) with a dual-energy x-ray absorptiometry (DEXA) scan of the spine and/or hip. Osteoporosis is defined as a BMD of less than 2.5 standard deviations below the mean
Are there any abnormal lab tests in osteoporosis?	No
What is osteopenia?	Decreased BMD; diagnosed on a DEXA scan as a BMD between 1 and 2.5 standard deviations below the mean
Who should be screened for osteoporosis using a DEXA scan?	Women under 65 years who have one or more risk factors for osteoporosis (in addition to menopause); All women over 65 years of age
In women with osteoporosis, what are risk factors for fracture?	Medical history: previous fracture or fracture in first-degree relative , inflammatory bowel disease (IBD), celiac disease, cystic fibrosis, history of hyperthyroidism, use of anxiolytic, anticonvulsant, or neuroleptic drugs, type II diabetes mellitus (DM), dementia Social history: cigarette smoking , consumption of lots of caffeine,

	sedentary lifestyle, inadequate calcium intake
	Physical examination: low body weight , tall stature
What are the categories of treatment for osteoporosis?	Reduce risk of falls; nonpharmacologic therapies; drug therapies
What types of nonpharmacologic therapies are useful for the treatment of osteoporosis?	<ol style="list-style-type: none"> 1. Diet: adequate calcium/vitamin D intake 2. Exercise: any weight-bearing exercise (including walking) at least 30 minutes three times per week 3. Cessation of smoking 4. Avoidance of drugs that increase bone loss
Who should be considered for pharmacologic therapy?	Postmenopausal women with diagnosed osteoporosis or with high risk for its development
What types of drugs are available?	Bisphosphonates; selective estrogen receptor modulators (SERMs); estrogen; calcitonin; vitamin D
How do bisphosphonates work?	They increase bone mass and reduce the incidence of fracture by inhibiting resorption of bone . They are first-line therapy for osteoporosis treatment
What are the side effects of bisphosphonates?	Pill-induced esophagitis, osteonecrosis of the jaw (rare)
How do SERMs work?	They increase BMD and reduce the risk of vertebral fractures
What other benefits do SERMs confer?	Lower risk of breast cancer; decrease total cholesterol and LDL
What are the side effects of SERMs?	Increased risk of venous thromboembolism
What are the indications for estrogen/progesterone therapy?	Persistent menopausal symptoms Inability to tolerate other antiresorptive medications
How does parathyroid hormone (PTH) work and how is it administered?	If given intermittently, PTH stimulates bone formation more than it causes resorption
When is calcitonin used?	Not a first-line treatment; used in women with pain secondary to a

What are the potential side effects of calcitriol that limit its use?	fracture because it offers analgesia in addition to its antiosteoporotic effects
What are isoflavones?	Hypercalcemia, hypercalciuria, and renal insufficiency
How are thiazide diuretics used in the prevention of osteoporosis?	A type of phytoestrogen; a micronutrient substance that has many similar properties to estrogen. Their effectiveness has not been proven
What is tibolone and how is it used in osteoporosis?	Used in postmenopausal women with hypertension because they decrease bone loss slightly
	A synthetic steroid that metabolizes to have the effects of estrogens, androgens, and progestins. It improves BMD ; however, it may increase the risk of stroke

Perimenopause and Menopause

How is menopause defined?	12 months of amenorrhea after the final menstrual period because of a loss of ovarian activity
What is the average age of menopause in the United States?	51 years
What are some factors that cause an earlier age of onset for menopause?	Smoking Genetic factors Nulliparity Hysterectomy Living at higher altitudes
What is premature menopause?	Also known as premature ovarian failure; the spontaneous cessation of menses before the age of 40
What are the other phases of menopause?	Perimenopause: the phase preceding menopause; reflected by menstrual cycle irregularities Postmenopause: phase of life that follows menopause

When does perimenopause begin?	Between 5 and 10 years before menopause
How does the menstrual cycle length change in the perimenopause?	<p>Early phase: the menstrual cycle remains regular but is shortened by 7 days or more</p> <p>Late phase: more menstrual cycle variability; may skip two or more cycles</p>
How do the following hormones change during the perimenopausal state?	
FSH/LH	Increases
Estrogen	Remains normal (until follicular growth stops)
Progesterone	Decreases
Why does FSH change during perimenopause?	The decline in the number of follicles and the irregular maturation of follicles leads to a decreased concentration of inhibin (which normally inhibits FSH secretion). This causes the rise in FSH levels
What are the symptoms of perimenopause?	Hot flashes (episodic sensation of warmth on chest/face that becomes generalized; associated with sweating) Sleep disturbances
How do the following hormones change at menopause?	
Androstenedione	Decreases
FSH/LH	Markedly increases
Testosterone	Increases
Estrogen	Decreases (to <20 pg/mL)
Why do estrogen levels change during menopause?	Because of the loss of ovarian follicles
Compare the changes in the levels of FSH and LH during menopause	Both increase; however FSH >> LH
Why does FSH increase more than LH?	Renal clearance of FSH is less than that of LH
What disorder presents with the same hormonal abnormalities as menopause (increased serum FSH > LH)?	Primary ovarian failure , which can be seen in Turner syndrome

What are the chief complaints from women going through menopause?

Hot flashes
 Hair/skin changes
 Atrophic urogenital system (symptoms of urinary tract infection, dyspareunia)
 Vaginitis (atrophic)
 Variable moods
 Osteoporosis
 Coronary artery disease

***Can be remembered by the mnemonic "menopause causes HHAVVOC"**

Describe the pathophysiology and the treatment for each of the symptoms of menopause

See Table 7-1

What complaint is often associated with hot flashes?

Insomnia (because symptoms often occur at night)

What are the clinical manifestations of an atrophic urogenital system?

Symptoms similar to those seen in UTIs (including urinary frequency, urgency, dysuria, pyuria, and urge incontinence)

Where does menopause-associated osteoporosis typically begin?

In the spine

What is the rate of bone loss associated with osteoporosis?

Before menopause, bone loss occurs at a rate of 0.5% per year; after menopause it increases to over 1% per year

What type of bone is most sensitive to the changes in estrogen levels associated with menopause?

Trabecular bone

What diagnosis is most likely when a postmenopausal woman presents with severe itching around the vagina? How is it diagnosed and how is it treated?

Lichen sclerosus. A biopsy is required for diagnosis. Treatment is high-potency corticosteroids

What are the two most commonly used hormone replacement therapies?

Unopposed estrogen therapy
 Continuous combined therapy with conjugated estrogen and medroxyprogesterone acetate

Is there cardiovascular benefit to using unopposed estrogen therapy (ET) and continuous combined oral estrogen-progestin therapy (CCE-MPA)?

According to data from the Women's Health Initiative (WHI) study, there is no benefit from estrogen therapy and a slight increased risk with combined therapy

Table 7-1 The Pathophysiology and Treatment for Symptoms of Menopause

Symptom/Condition	Pathophysiology	Treatment
Hot flashes	Estrogen withdrawal → thermoregulatory dysfunction	Short-term estrogen replacement therapy
Hair and skin changes	Decreased collagen → wrinkles Decreased estrogen, no change in testosterone → male growth patterns	Estrogen replacement therapy
Atrophic urogenital system	Decreased estrogen → atrophy of urethral epithelium → loss of urethral tone, shrinkage of the uterus, cervix, vagina, ovaries, and bladder	Estrogen replacement therapy
Vaginitis (atrophic)	Decreased estrogen → epithelial atrophy → soreness, burning, dyspareunia, sexual dysfunction	Lubricants (mild) vaginal estrogen (severe)
Variable moods	Hormonal/life changes → nervousness, anxiety, depression	Estrogen replacement therapy
Osteoporosis and joint pain	↑ Bone resorption phase and ↑ osteoclasts; ↓ bone formation phase and ↓ osteoblasts	Raloxifene or bisphosphonates
Coronary heart disease	↑ LDL, ↓ HDL → increased atherosclerosis	Hormone replacement therapy does not have any benefit
Cognitive decline	Change in areas important for memory → memory loss, possible link to Alzheimer's disease	Estrogen or combined hormonal therapy has no benefit

What is the risk of breast cancer when using unopposed estrogen therapy or combined estrogen-progestin HRT?

In the WHI **unopposed estrogen trial**, there is **no increase in risk of breast cancer** in 10,000 women who had a hysterectomy. In the **combined estrogen-progestin group**, there was a **significant increase in the risk of breast cancer**. The presence of breast cancer was seen in year 3 in women

What are the relative risks of endometrial cancer when using unopposed estrogen therapy versus combined estrogen-progestin hormone therapy?

who had previously used menopausal hormones and in year 4 who had no history of previous use

Treatment with estrogen alone greatly increases the risk of endometrial hyperplasia and cancer. Adding a progestin diminishes this excess risk of endometrial hyperplasia and carcinoma

When is estrogen replacement therapy (ERT)/hormone replacement therapy (HRT) contraindicated?

Women who have abnormal vaginal bleeding

History of breast cancer

History of coronary heart disease (CHD)

History of estrogen-dependent neoplasia

History of DVTs or thromboembolic event

History of liver dysfunction/disease

What are complications of estrogen replacement therapy?

Endometrial cancer

Breast cancer

Thromboembolic disease

Stroke

Uterine bleeding

Gallbladder disease

Hirsutism, Virilization, and Polycystic Ovarian Syndrome

HIRSUTISM AND VIRILIZATION

What is hirsutism?

Excessive growth of **androgen-dependent** hair (e.g., on the upper lip, chest, chin)

What is virilization?

Excessive androgen-induced changes in addition to hirsutism. These include clitoromegaly, voice

What is hypertrichosis?

deepening, increasing muscle mass, and other masculinizing signs

It is a rare disease that refers to **diffusely increased androgen-independent** fine body hair, usually caused by drugs or systemic illnesses. It does not represent hirsutism

How does the clinical presentation of hirsutism differ from that of virilization?

Hirsutism manifests with **increased “midline” hair** on the upper lip, chin, ear, cheeks, lower abdomen, chest, back, and upper arms. Amenorrhea is seen in severe cases. *Virilization* is excess hair and additional characteristics such as **deepening of the voice, acne, breast atrophy, clitoromegaly, balding, and increased strength**

Which androgens cause and are elevated in hirsutism?

Testosterone and dehydroepiandrosterone sulfate (DHEAS)

What two organs may be involved in hirsutism and what steroid does each mainly secrete?

Ovary: testosterone
Adrenal gland: DHEAS

What is the role of 17OH progesterone in the development of hirsutism?

17OH progesterone is a precursor to the biosynthesis of cortisol and can be converted peripherally into androgens if found in excess

What is the most common disorder that causes hirsutism?

Polycystic ovarian syndrome (PCOS)

What other underlying diseases may cause hirsutism?

Congenital adrenal hyperplasia (CAH; 21-hydroxylase deficiency)
Androgen-secreting ovarian tumors (Sertoli-Leydig or granulosa-theca tumors)
Adrenal tumors
Cushing syndrome
Exogenous androgens (danazol)
Hyperprolactinemia
Other rare disorders (hyperthecosis)

What is idiopathic hirsutism?

It is a diagnosis given to women with hirsutism without adrenal or ovarian dysfunction, normal serum androgen concentrations, normal menstrual cycles, and no other identifiable cause of their hirsutism. There is often a positive family history

How may the presenting signs and symptoms of a patient help specify the disorder causing hirsutism?

Ovarian tumor: pelvic mass, sudden onset of amenorrhea, virilization

PCOS: obesity, acne, long history of irregular menses, slow onset of hirsutism beginning at puberty, acanthosis nigricans

Theca-lutein cysts: hirsutism develops during pregnancy

CAH: gradual onset of anovulation, positive family history

Adrenal tumor: rapid onset, virilization, abdominal-flank mass

Cushing syndrome: moon facies, buffalo hump, centripetal obesity, striae, extremity wasting

Hyperprolactinemia: galactorrhea or visual changes with menstrual irregularities

What laboratory studies assist in the diagnosis of the etiology of hirsutism?

Testosterone >200 ng/mL → androgen-secreting ovarian tumor

DHEAS >700 ug/dL → androgen-secreting adrenal tumor

17 α -hydroxyprogesterone > 200 ng/dL → 21-hydroxylase deficiency

LH:FSH \geq 3 → PCOS

Prolactin > 200 μ g/dL → prolactinoma

24-hour urinary free cortisol > 100 ng/24 h → Cushing syndrome

What imaging studies are warranted?

Pelvic ultrasound may reveal polycystic ovaries or ovarian tumors/cysts

CT/MRI of the abdomen to look for an adrenal mass when DHEAS levels are elevated

How is hirsutism generally treated?

Treat the underlying disorder. The most common medications are **oral contraceptives (OCPs)**, GnRH analogs, and antiandrogens (first-line spironolactone, second-line flutamide, finasteride)

What are the specific treatments for each of the following causes of hirsutism?

Ovarian tumor: surgical removal

PCOS: combination OCPs

CAH: continuous corticosteroid replacement

Adrenal tumor: surgical removal

Idiopathic hirsutism: spironolactone

How do combination OCPs work as antiandrogens?

They suppress LH stimulation of the theca cells and they increase sex hormone-binding globulin (SHBG) (thus decreasing free testosterone)

How does spironolactone act as an antiandrogen?

It is an androgen receptor blocker and it suppresses 5 α -reductase in hair follicles

Polycystic Ovarian Syndrome (Stein-Leventhal Syndrome)

Describe polycystic ovarian syndrome (PCOS)

It is the most common chronic endocrine condition characterized by **persistent anovulation** which leads to **secondary amenorrhea** and other menstrual irregularities, and **androgen excess** which may cause hirsutism and virilization

What is the pathophysiology behind PCOS?

It is a **dysfunction of the hypothalamic-pituitary axis**. **Increased pulsatile secretions of GnRH** → **excess production of LH** → excess production and secretion of androgens → virilization

How does PCOS lead to anovulation?

Some of the excess androgens are converted to estrogen. High estrogen levels increase LH (by blocking the inhibitory feedback mechanism of progesterone on the pituitary). High LH levels stimulate the immature follicles to produce more androgens, which then become converted to estrogen. The cycle then repeats

What are the two most common presenting complaints of patients with PCOS?	FSH is inhibited by high estrogen levels. This allows the early growth of multiple follicles, but failure of the development of the mature follicle and its ovulation
What other clinical manifestations are associated with PCOS?	Hirsutism and infertility Chronic anovulation Obesity Insulin resistance Irregular bleeding (from a chronically estrogen-stimulated endometrium)
What dermatologic condition may be associated with PCOS?	Acanthosis nigricans
How is the diagnosis of PCOS made?	It is suspected in the presence of menstrual irregularity (anovulation), evidence of androgen excess, and exclusion of other causes of menstrual irregularities and hyperandrogenism
What test should be ruled out first in a woman who presents with secondary amenorrhea?	Pregnancy test (β -hCG)
What biochemical test confirms the diagnosis of PCOS?	An LH/FSH ratio > 3:1
How do polycystic ovaries appear on pelvic ultrasound?	They appear with eight or more, small (2–8 mm), subcapsular fluid-filled follicle cysts that look like a “ black pearl necklace ”
How is infertility in PCOS patients treated?	Weight loss is recommended first. Ovulation induction with clomiphene citrate can be used. Metformin and/or gonadotropins can be added if clomiphene alone fails
How is hirsutism treated in PCOS patients?	Combination OCPs are first line (for women who do not desire pregnancy) Antiandrogen therapy (spironolactone, flutamide, finasteride)

What are complications associated with PCOS?

Increased risk of early onset of Type II diabetes mellitus

Increased risk of endometrial hyperplasia and endometrial cancer because of unopposed estrogen stimulation

What is HAIR-AN syndrome?

It is a variant of PCOS that leads to hyperandrogenism, insulin resistance, and acanthosis nigricans

SECTION III

Topics in Obstetrics

This page intentionally left blank

General Obstetrics

Maternal-Fetal Physiologic Adaptation to Pregnancy

EMBRYOLOGY

What are the developmental stages of early pregnancy?

Fertilization and cleavage result in a **zygote** (an 8-cell mass) which then divides to form the **morula** (a 16-cell mass) 3 days after fertilization. A **blastocyst** develops and implantation occurs approximately 10 days after fertilization. An **embryonic disk** develops after the first week and a **yolk sac** after 5 weeks

What are the placental membranes and uterine layers (fetal to maternal)?

Amnion

Chorion (with villi composed of syncytial trophoblasts and cytotrophoblasts)

Decidua parietalis (endometrium)

Myometrium (undergoes hyperplasia early in pregnancy with subsequent hypertrophy and distension)

Serosa (See Fig. 8-1)

How does the timing of division affect the development of monozygotic twins?

If division occurs <72 hours: two embryos, two amnions, two chorions (“di-di”)

If division occurs between 4-7 days: two embryos, two amnions, one chorion (“mono-di”)

If division occurs after 7 days: one shared amnion and chorion (“mono-mono”)

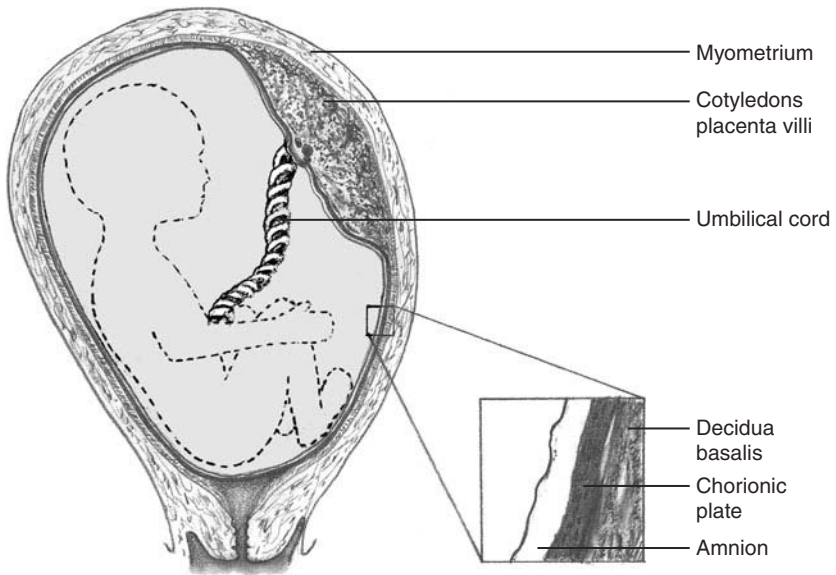


Figure 8-1 Embryologic layers of the human fetus at 3 months.

If division occurs after the embryonic disk has developed, the twins will be conjoined

FETAL CIRCULATION

What are the major shunts involved with fetal circulation?

Ductus venosus
Foramen ovale
Ductus arteriosus

What are the patterns of fetal blood flow (starting from the umbilical vein)?

1. Umbilical vein → ductus venosus → inferior vena cava → right atrium → foramen ovale → left atrium → left ventricle → cephalic/systemic circulation
2. superior vena cava → right atrium → right ventricle → fetal lungs → pulmonary artery → ductus arteriosus → aorta → hypogastric arteries → umbilical arteries (See Fig. 8-2)

Do each of the following vessels carry oxygenated or deoxygenated blood?

Umbilical artery

Deoxygenated

Umbilical vein

Oxygenated

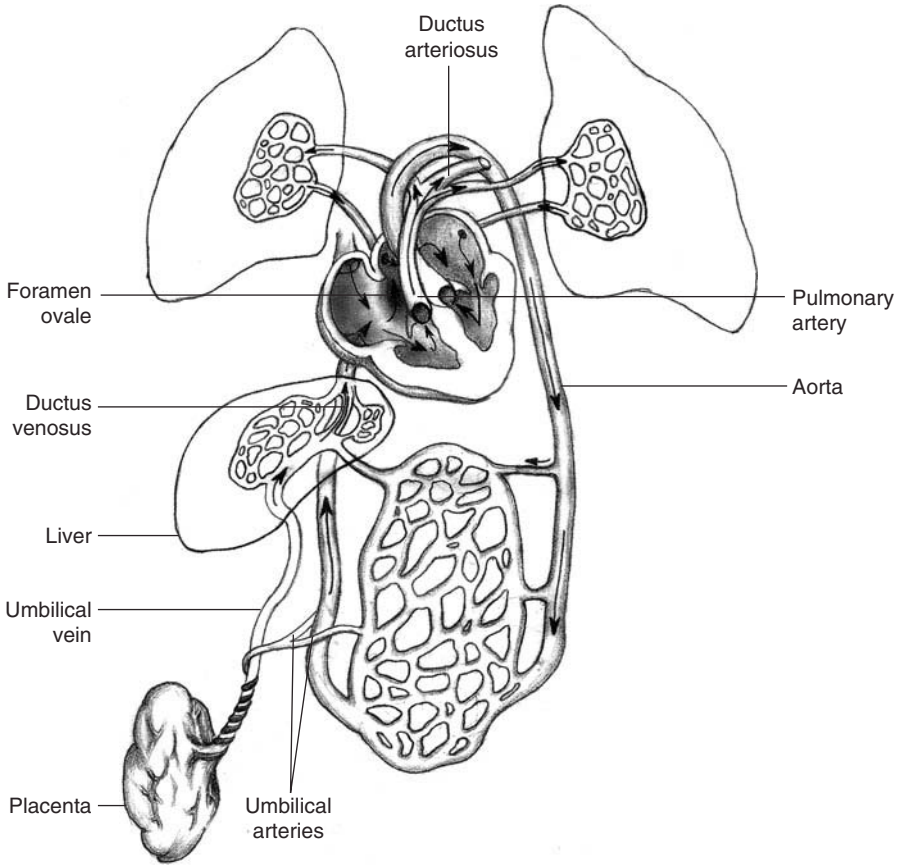


Figure 8-2 Fetal circulation.

What are the embryologic remnants associated with each of the following?

Umbilical vein

Ligamentum hepatis

Ductus venosus

Ligamentum venosum

Ductus arteriosus

Ligamentum arteriosum

Umbilical arteries

Medial umbilical ligaments

CARDIOVASCULAR

How does pregnancy affect cardiac output (CO), blood volume, and blood pressure (BP)?

CO increases up to 50% (1.8 L/min)

Blood volume increases by 50%

BP decreases

	<p>These changes peak during the second trimester and remain relatively constant until delivery; they are mediated by hormones (aldosterone, progesterone, human chorion gonadotrophin (HCG), relaxin, nitric oxide [NO])</p>
Pregnancy affects which components of CO?	<p>Remember, $CO = SV \times HR$. Both stroke volume (SV) and heart rate (HR) increase; however, SV increases more than HR</p>
How does pregnancy affect SV?	<p>Preload is increased with an increase in blood volume; afterload is reduced by the low-resistance uteroplacental circulation and by peripheral vasodilatation</p>
When do HR and SV peak during pregnancy?	<p>HR peaks around term; SV peaks during the second trimester</p>
What percentage of CO goes to the uterus during pregnancy?	<p>Up to 20% at term (compared to <1% in the nonpregnant state). Most of this goes to the placenta</p>
How does the cardiac examination change during pregnancy?	<p>Heart sounds are louder and split, there is often a systolic murmur and an S3. This manifests as a slight left axis deviation on EKG and as mild cardiomegaly on chest x-ray (CXR)</p> <p>(A diastolic murmur is never normal and should be evaluated)</p>
Why does the cardiac examination change?	<p>All four cardiac chambers and valve diameters are increased and hyperdynamic. This may lead to mild pulmonic, tricuspid, and/or mitral regurgitation or a small pericardial effusion</p>
How does the gravid uterus affect CO?	<p>In the supine position the inferior vena cava is compressed, which may decrease CO by up to 30%. Maximum venous return and CO is maintained in the left lateral decubitus (LLD) position</p>
Describe the hematologic changes of pregnancy	<p>Red blood cell mass increases by 15–30% (because of increased erythropoietin)</p> <p>Plasma volume increases by 30–50% (because of increased aldosterone)</p>

	Hct and Hb decrease , causing the physiologic “anemia of pregnancy” (because of increased plasma volume)
	*An Hb <11 is never normal and must be evaluated for iron/folate deficiency or a hemoglobinopathy
When does the anemia of pregnancy reach its nadir?	Around 24 weeks
What is the effect of pregnancy on BP?	BP decreases in the first trimester , nadirs at approximately 24 weeks, and then almost normalizes by term. Diastolic is affected more than systolic

RESPIRATORY

How does pregnancy affect pulmonary function tests (PFTs)?	<p>Tidal volume increases by 40%</p> <p>Minute ventilation increases by 50%</p> <p>Functional residual capacity decreases by 20%</p> <p>Total lung capacity slightly decreases</p> <p>Expiratory reserve volume and total lung capacity decrease</p> <p>Forced expiratory volume (FEV1) does not change</p> <p>Respiratory rate does not change</p>
What hormone mediates these effects and how does it act?	Progesterone. It acts centrally (through direct stimulation of the medulla and respiratory centers) as well as peripherally (through pulmonary vasodilation)
How do these respiratory changes affect the blood gas levels?	<p>Alveolar and arterial oxygen levels increase</p> <p>Arterial carbon dioxide levels decrease</p>
How is the acid base equilibrium affected during pregnancy?	Respiratory alkalosis with metabolic compensation (pH between 7.40 and 7.45)
What is the effect of pregnancy on oxygen consumption?	Increases by over 20% owing to the placenta, fetus, and maternal organs
How does the gravid uterus affect respiratory function?	The thorax becomes more barrel-chested and dyspnea is a common symptom in advanced pregnancy because of the pressure of the gravid uterus and the central effects of progesterone

RENAL/URINARY

<p>What is the effect of pregnancy on renal function?</p>	<p>Glomerular filtration rate (GFR) increases by 50% (because of the increase in glomerular plasma flow)</p> <p>Renal blood flow increases</p> <p>Plasma creatinine, BUN, and uric acid levels decrease (because of the increased GFR)</p> <p>[*Normalization of Cr, BUN, or UA may indicate underlying pathology (preeclampsia, renal insufficiency)]</p>
<p>Why is the pregnant woman at increased risk for urinary tract infections?</p>	<p>Progesterone reduces ureteral tone and peristalsis, and relaxes the bladder wall allowing reflux through the incompetent vesico-ureteral valves. This results in stasis of urine, dilated ureters, increased pressure in the renal pelvis, and bacterial proliferation</p>
<p>What are the common causes of bacteriuria in pregnancy?</p>	<p><i>Escherichia coli</i> >> <i>Klebsiella</i> > <i>Proteus</i> > <i>Group B streptococcus</i> (GBS) > Enterococci > Staphylococci</p>
<p>What is a complication of pyelonephritis during pregnancy?</p>	<p>Preterm labor</p>
<p>Where does hydronephrosis in pregnancy most commonly occur?</p>	<p>On the right side (because of right uterine dextrorotation and right ureter compression)</p>
<p>What happens to plasma osmolality during pregnancy?</p>	<p>It falls because of increased levels of relaxin (stimulated by HCG) and antidiuretic hormone (ADH). These lead to an increase in intravascular fluid which then decreases plasma osmolality</p>
<p>What is the clinical affect of this change in plasma osmolality?</p>	<p>Pitting edema in gravity-dependent areas</p>
<p>What is the significance of trace protein or glucose in the second or third trimester?</p>	<p>It is normal, because of increased GFR. In 24 hours protein may increase to 200 mg (normal is less than 100 mg/24 hours). However, higher values suggest renal pathology or preeclampsia</p>

GASTROINTESTINAL

When does morning sickness occur?	During the first trimester , as levels of hCG double every 2 days. After hCG levels peak (~10 weeks), morning sickness decreases
What is hyperemesis gravidarum?	An idiopathic, noninfectious, severe nausea and vomiting that causes dehydration, ketone formation, weight loss, and metabolic acidosis
How does progesterone affect the GI tract?	Lower esophageal sphincter tone decreases (can lead to GERD) Small bowel and colon transit decreases (can lead to bloating and constipation) Venous congestion (can lead to hemorrhoids) Biliary tract peristalsis decreases (increases risk of cholelithiasis and cholecystitis)
What impact does estrogen have on hepatic metabolism?	It increases the production of hormone-binding globulins (sex hormone-binding globulin [SHBG], thyroxine-binding globulin [TBG], ceruloplasmin [copper], transferrin total iron-binding capacity [TIBC], increases up to 100% and coagulation factors)
How do liver function tests (LFTs) change in pregnancy?	Alkaline phosphatase is increased (because of placental production). Other enzymes are within normal limits [*Abnormalities may suggest pathology: hepatitis, HELLP, acute fatty liver of pregnancy, cholestasis of pregnancy, or other pathologies]
How does pregnancy affect the risk of pancreatitis?	Increases the risk (because of increased risks of cholelithiasis and hyperlipidemia)
Why is the mortality associated with appendicitis higher in pregnancy?	Because of the gravid uterus, the appendix is pushed higher leading to a delay in the diagnosis

HEMATOLOGIC

How is the coagulation system affected by pregnancy?

Decreased protein S
Resistance to protein C

Increased factor I (fibrinogen), II, V, VII, VIII, X, and XII

Because of these, **pregnancy is a hypercoagulable state**

What are the potential complications of this hypercoagulable state?

Venous thrombosis (occurs 0.7 per 1000 women in pregnancy) and subsequent **pulmonary embolism**. Both occur predominantly in the **third trimester**

What is the effect of pregnancy on immunity?

Cell-mediated immunity is weakened but humoral immunity is strengthened

ENDOCRINE

What is hCG and where is it formed?

Human chorionic gonadotropin; in the **syncytiotrophoblast** of the placenta

hCG is structurally related to what other glycoprotein hormones?

Luteinizing hormone (**LH**), follicle-stimulating hormone (**FSH**), and thyroid-stimulating hormone (**TSH**) (they all have the same α subunit but different β subunits)

How does hCG promote pregnancy?

1. **Maintains the corpus luteum** for first trimester
2. Causes sexual differentiation in the male fetus
3. Ensures adequate T_3 and T_4 production
4. Increases relaxin secretion by the corpus luteum (leading to decreased vascular resistance)

What are the three major types of estrogen are they predominately produced?

Estrone: adrenals (dehydroepiandrosterone [DHEA] is the precursor)

Estriol (least bioactive): **placenta**

Estradiol (most bioactive): **ovaries**

What happens to the adrenal hormones during pregnancy?

Adrenocorticotrophic hormone (ACTH), corticotropin-releasing hormone (CRH), and cortisol all increase

What is oxytocin?

A hormone produced by the supraoptic and paraventricular nuclei of the hypothalamus and released by the posterior pituitary. Concentrations rise throughout gestation, during labor, and with lactation or nipple stimulation

What are the major actions of oxytocin?

Contraction of uterine myometrium, ductal myoepithelial contraction (milk expression), orgasm, and bonding

What peptide is similar to oxytocin and what are the clinical consequences?

Antidiuretic hormone (ADH). Induction/augmentation of labor with oxytocin may cause severe fluid retention and pulmonary edema

What happens to thyroid hormone levels during pregnancy?

TBG increases
Total T_3 and T_4 levels increase but **free T_3 and T_4 levels** (and thus thyroid function) **remain unchanged**
TSH does not change

What mediates these thyroid hormone changes?

hCG stimulates TSH receptors to increase total T_4 and T_3 levels. Estrogen increases the hepatic production of TBG

What is hPL and where is it formed?

Human placental lactogen; in the **syncytiotrophoblast** of the placenta

What is the function of hPL?

It **antagonizes insulin** and **increases lypolysis** to ensure adequate glucose delivery to the fetus

How does maternal glucose metabolism change during pregnancy?

hPL causes maternal insulin resistance

Why does gestational diabetes mellitus (GDM) occur?

There is insufficient maternal insulin to counter the hyperglycemic effects of placental hPL. It occurs most commonly in the third trimester, when hPL levels are highest

DERMATOLOGY

What are striae distensae or striae gravidarum?

Stretch marks; caused from a diminution of elastin fibers and fibrillin microfibrils

What other skin changes occur during pregnancy?

Chloasma or **melasma** (“mask of pregnancy;” hyperpigmentation of the face)

Linea nigra (darkening of the linea alba from the pubic symphysis to the xiphoid process)

Hyperpigmentation of the axilla, genitalia, perineum, anus, inner thighs, neck, scars, nevi, and lentigo

Palmar erythema and **telangiectasias**

Hirsutism and **acne**

REPRODUCTIVE

What is Chadwick sign?

Bluish color of vulvar and vaginal membranes because of venous congestion; a normal finding in pregnancy

What other vaginal changes occur in pregnancy?

Increased vascularity and distensibility; increased vaginal discharge (because of increased capillary permeability and desquamation)

What changes occur to the cervix and uterus during pregnancy?

Hegar sign (softening of the lower uterine segment) that occurs in early pregnancy)

Increased eversion of the cervical columnar epithelium

What breast changes occur in pregnancy?

Enlargement, increase in cystic components, darkening of areolae, hypertrophy of sebaceous glands, colostrum production (in late pregnancy)

Prenatal Care

DIAGNOSIS AND TERMINOLOGY

When should prenatal care begin?

Preconception or as soon as pregnancy is suspected

What are the major goals of preconceptive counseling?	<p>Minimize unplanned pregnancies</p> <p>Optimize chronic medical disorders (diabetes mellitus [DM], epilepsy, hypothyroid, cardiovascular disorders)</p> <p>Promote healthy behaviors</p> <p>Counsel regarding adequate diet, exercise, and nutritional supplements (folic acid and iron)</p> <p>Offer appropriate vaccinations (rubella, diphtheria, hepatitis B virus)</p> <p>Screen for genetic or chromosomal abnormalities</p> <p>Improve patient's readiness for pregnancy and parenting</p>
What are the major goals of prenatal care?	<p>Prevention, ensure a healthy mother, ensure a healthy baby</p>
What are some important medical conditions that adversely impact pregnancy?	<p>DM, hypertension (HTN), cardiovascular disease, autoimmune disorders, kidney disease/UTIs, pulmonary disease (asthma or TB), seizures or other neurologic disorders, psychiatric disorders, hepatitis, phlebitis, thyroid abnormalities, blood transfusions, hemophilia or blood disorders (thalassemia or sickle cell)</p>
What does folic acid supplementation help prevent?	<p>Neural tube (NT) defects</p>
Who should take folic acid supplementation?	<p>All women of reproductive age; the NT closes 26 days after fertilization (often before a woman is aware of pregnancy)</p>
How much folic acid should women of reproductive age consume?	<p>A balanced diet plus 0.4 mg daily</p>
What are the early signs and symptoms of pregnancy?	<p>Amenorrhea or irregular bleeding</p> <p>Fatigue</p> <p>Nausea/vomiting</p> <p>Breast tenderness</p> <p>Urinary frequency</p> <p>Chadwick sign (bluish discoloration of the vagina)</p> <p>Hegar sign (softening of the cervix)</p>
How is pregnancy diagnosed?	<p>β-hCG</p>

<p>When does β-hCG become positive?</p>	<p>Serum tests become positive following implantation (about 10 days after fertilization); urine tests become positive 2–3 weeks after fertilization or around on the first day of a missed period</p>
<p>Where is hCG formed?</p>	<p>In the placental trophoblasts</p>
<p>What is the primary function of hCG?</p>	<p>To maintain the corpus luteum and progesterone production (it is “pro-pregnancy”)</p>
<p>Why is the β subunit of hCG measured?</p>	<p>β subunit of hCG differentiates it from LH, FSH, and TSH, which all share the same α subunit</p>
<p>What are some reasons that β-hCG may be abnormally elevated?</p>	<p>Multiple gestations Trophoblastic disease Molar pregnancy Choriocarcinoma</p>
<p>At what rate does serum β-hCG increase?</p>	<p>Serum concentrations double every 48 hours during the first trimester and peak at 100,000 IU/L; levels then regress to 30,000 IU/L from the 20th week until term</p>
<p>A woman with a positive pregnancy test presents to the ER with vaginal bleeding. She has been trying to become pregnant and cannot remember her last menstrual period (LMP). Her serum β-hCG is 200 IU/L and no gestational sac is visualized on transvaginal ultrasound. What is your next step in management?</p>	<p>Ask the patient to return in 2 days to recheck the β-hCG. If it is not doubling appropriately she may have an ectopic pregnancy or a spontaneous abortion (SAB). A gestational sac should be visualized with a β-hCG > 1000 IU/L</p>
<p>What is the estimated gestational age (EGA)?</p>	<p>Duration of the pregnancy dated from first day of the LMP. The EGA of a normal pregnancy at term is 40 weeks</p>
<p>What is the developmental age (DA) of a pregnancy?</p>	<p>Duration of the pregnancy dated from fertilization; typically 14 days less than the EGA (such that the DA of a normal pregnancy at term is 38 weeks)</p>
<p>What is the estimated date of confinement (EDC)?</p>	<p>The “due date” when the pregnancy is full term</p>
<p>What is Nügele rule?</p>	<p>For women with a regular 28-day menstrual cycle:</p>

What gestational ages are defined by the following terms:

EDC = LMP – 3 months + 7 days + 1 year

First trimester: <14 weeks

Second trimester: 14–28 weeks

Third trimester: >28 weeks

Viability: 24 weeks

Prematurity: 24–36 weeks

Term: 37–42 weeks

Post dates: >40 weeks

Post term: >42 weeks increased risk of perinatal morbidity and mortality

What ages are defined by the following terms:

Neonate: birth to 28 days of life

Infant: birth until 1 year of life

What are the patient's Gs and Ps?

Gravidity: total number of pregnancies

Parity: all viable and nonviable pregnancies, including spontaneous, therapeutic, and voluntary abortions

What are the four numbers that follow parity?

First: deliveries >37 weeks EGA

Second: viable deliveries before 37 weeks

Third: abortions and ectopic pregnancies

Fourth: living children (including twins or adoptions)

What are the Gs and Ps for a mother of three with a history of an normal spontaneous vaginal delivery (NSVD) at term, preterm twins, a fetal demise at 23 weeks, and an ectopic pregnancy at 6 weeks?

G4 P1213

How is pregnancy dating confirmed?

Ultrasound (most accurate in the first trimester) and **fundal height** on physical examination

What size-date discrepancy* would require further evaluation?

1. Ultrasound should be within 1 week of the EGA during the first trimester, within 2 weeks during the second trimester, and within 3 weeks in the third trimester
2. FH should be within 3 weeks of the EGA (determined by LMP)

	*A size-date discrepancy suggests either underlying pathology or an error in dating (requiring the EDC to be changed)
To what EGA does each fundal height correspond?	Symphysis pubis: 12 weeks EGA Umbilicus: 20 weeks EGA Xiphoid process: 36 weeks EGA
	Fundal height in centimeters above the symphysis pubis corresponds to EGA after 20 weeks
What fetal parameter is most important in assessing viability?	Fetal cardiac activity
With each of the following tools, when can fetal cardiac activity be detected?	
Transvaginal ultrasound	6 weeks EGA
Abdominal ultrasound	10 weeks EGA
Doppler fetal heart monitor	12 weeks EGA
Ascultation	20 weeks EGA
What fetal parameters are measured on ultrasound to assess EGA?	First trimester: crown-rump length Second/third trimesters: femur length; abdominal circumference; biparietal diameter; head circumference
Why is dating important?	Monitoring growth, appropriate timing of screening markers (quad screen, gestational diabetes mellitus [GDM]), delivery planning (fetal lung maturity, postdates)
A woman presents to the emergency room with abdominal pain. Her period is 1 week late and her serum β-hCG measures 1000 IU/L. Transvaginal ultrasound (TVUS) visualizes thickened endometrial tissue and fluid in the cul-de-sac. What is the greatest concern?	Ruptured ectopic pregnancy (with possible hemorrhage and shock) TVUS can visualize a gestational sac at 5 weeks EGA, which corresponds to a serum β -hCG level of 1000–1500 IU/L

MANAGEMENT

What information should be incorporated in a patient's prenatal care (PNC) records and the labor admission note?	Age, Gs & Ps, LMP, EDC (determined by LMP and ultrasound)
---	---

Chief complaint or presenting issue
(routine prenatal care, SOB, CP)

Four cardinal questions (ctx, LOF,
vaginal bleed, fetal movements)

Prenatal care: doctor's name, number
of visits

Complications during this
pregnancy: GDM, AMA, IUGR,
teenage pregnancy, and so on

Prenatal labs (PNL)

Ultrasound: date/placental
location/amniotic fluid volume
(AFV)/estimated fetal weight
(EFW)/abnormalities/presentation

Past obstetric history (ObHx), past
gynecologic history (GynHx), past
medical history (PMHx), psychiatric
history, past surgical history (PSHx),
meds, allergies, family history (FHx),
social history (SHx)

Physical examination: vital signs:
BP, P, T, R, FS (diabetic), urine dip
(for everyone if possible, *must* for
preeclampsia)

General, cardiac, lungs, abdomen:
typically soft, NT, gravid, **Leopold:**
vertex, left lateral lie, EFW

Extremities: Homans +/-, no calf
tenderness

Electronic fetal monitoring (EFM):
baseline, +/- accelerations, +/-
decelerations, minimal/moderate/
marked long-term variability

Tocolysis history (TOCO):
contractions q minutes

SSE: normal EFG (external female
genitalia), discharge, bleeding, fluid,
lesions

Vaginal examination (VE): cervical
(cx) dilation/ effacement/station of
the presenting fetus, cx consistency
(firm, soft), cx position (posterior,
anterior), clinical pelvimetry

Assessment and plan: age (y/o)
GxPxxxx @(EGA) weeks with
(diagnosis . . . early labor, active
labor, SROM, PROM, induction of

labor (IOL) secondary to _____)
admit to labor and delivery

- Antibiotic prophylaxis (Pphx) if GBS+
- CBC/Type and Screen (T&S)
- NPO except ice chips
- Intravenous fluid (IVF): lactate ringers (LR) @ 120 cc/h
- Plan for pain management
- Fetal heart rate (FHR) tracing reactive/reassuring/ concerning. Continue EFM/TOCO
- Any additional issues that will need to be addressed postpartum

What are relevant aspects of a patient's past obstetrical history (ObHx)?

For each prior pregnancy:
date of delivery, mode of delivery or outcome, gestational age and weight at delivery (weeks), anesthesia complications, (maternal and fetal)

History of infertility

Type of uterine incision with prior C-sections

Year and EGA of all abortions and procedures (spontaneous abortion [SAB], elective termination of pregnancy [ETOP] or voluntary termination of pregnancy [VTOP], intrauterine demise [IUD] c dilation and curettage [D&C], dilation and evacuation [D&E])

What are relevant aspects of a patient's past gynecologic history (GynHx)?

Gyn triad: age of menarche/cycle length/duration of menstruation

History of cysts, fibroids, abnormal Pap smears, gyn surgeries

STIs

Prior use of contraception

What are relevant aspects of a patient's social history (SH)?

Domestic violence

Social support

Occupation

Highest level of education

Nutrition

Tobacco, alcohol, drug use

What are relevant aspects of a patient's genetic & family history (FHx)?

Relatives with pregnancy-related disorders (e.g., pregnancy losses)

	<p>Family history of chronic medical conditions (diabetes, thyroid disorders, hemoglobinopathies)</p> <p>Consanguinity</p> <p>Twins</p> <p>Congenital, chromosomal, or metabolic abnormalities (blood disorders, mental retardation)</p> <p>Ethnicity of the mother and father (for possible screening tests)</p>
<p>How much weight should a woman be advised to gain in pregnancy?</p>	<p>Average women should gain between 25–35 lbs (underweight women should gain more and overweight women should gain less)</p>
<p>When does a fetus experience the most rapid weight gain?</p>	<p>In the third trimester; the fetus gains approximately $\frac{1}{2}$ lbs/week</p>
<p>What is the recommended daily nutritional intake during pregnancy?</p>	<p>Calories: an additional 300 kcal/day for each fetus</p> <p>Protein: increase daily intake by 5–6 g</p> <p>Iron: requirements double to 30 mg/day</p> <p>Calcium: increases by 1000 mg in the third trimester during fetal bone calcification</p> <p>Daily intakes of copper (2 mg), folate (0.4 mg), vitamin C (50 mg), vitamin D (10 mcg or 400 IU), and vitamin B₁₂ (2 mcg) should continue</p>
<p>Which substances and foods should be limited or avoided?</p>	<p>Tobacco, alcohol, street drugs</p> <p>Herbal medications</p> <p>Caffeine > 500 mg or > 4 cups a day (can cause SAB or small for gestational age [SGA])</p> <p>Iodine (can cause fetal goiter)</p> <p>Large amounts of vitamins A, D, E, and K</p> <p>Unpasteurized dairy</p> <p>Methylmercury (in raw fish, shark, swordfish, king mackerel, and tilefish)</p>
<p>Should exercise and sexual behaviors change during pregnancy?</p>	<p>Activity can be maintained at the same intensity prior to pregnancy, in</p>

Should pregnant women continue to wear seat belts?	the absence of obstetric or medical complications. If a patient does not, she should be encouraged to begin light exercise
How often are prenatal care visits scheduled?	Yes! All patients should be encouraged to continue to wear seat belts during pregnancy. The lap belt can be placed below the uterus
What are the four cardinal questions asked during each prenatal care visit?	Every 4 weeks during the first and second trimesters Every 2 weeks in the third trimester (28–36 weeks) Once a week near term (36–40 weeks) Postdates will require more involved monitoring (Adequate prenatal care requires more than nine visits, with the first visit during the first trimester)
What are important parameters in evaluating the pelvic shape?	Presence of: Contractions; leakage of fluid; vaginal bleeding; fetal movement (after 20 weeks) Pelvic inlet (diagonal conjugate = distance from the pubic symphysis to the sacral promontory) Prominence of ischial spines Pelvic sidewalls (convergent vs parallel) Shape of sacrum
What measurements are taken during each standard prenatal care visit?	Weight Blood pressure Urine dip (protein, glucose, leukocytes) Fundal height Abdominal doppler fetal heart rate (after 12 weeks)
Which vaccines should be offered to pregnant women?	Any required inactivated vaccines and the influenza vaccine (in the second or third trimester) Note: Do not give vaccines that contain active viral components (measles, mumps, oral polio vaccine [OPV], or rubella)

<p>What is advanced maternal age (AMA)?</p>	<p>>35 years of age at the time of delivery (with a singleton pregnancy)</p>
<p>What symptoms must a patient be educated about? What do each signify?</p>	<p>Vaginal bleeding (SAB in first trimester, placental abruption/previa in second or third trimester)</p> <p>Edema, headache, black spots, blurry vision, right upper quadrant pain, epigastric pain (preeclampsia)</p> <p>Dysuria, fever, chills (pyelonephritis)</p>
<p>What are the signs and symptoms of preterm labor?</p>	<p>Any of the following symptoms between 20 and 37 weeks EGA:</p> <p>Abdominal, vaginal, or lower back pain or pressure that does not improve after hydration/rest</p> <p>Uterine contractions every 10 minutes for more than 1 hour</p> <p>A sudden thinning or increase in vaginal discharge</p> <p>Bleeding from the vagina</p>
<p>What should a patient be advised to do if she experiences these symptoms?</p>	<p>Contact her health care provider and present to the clinic or hospital for further evaluation</p>
<p>What is the prevalence of domestic violence (DV) among pregnant women?</p>	<p>DV is more prevalent among pregnant than nonpregnant women; it affects approximately 1 in 6 women</p>

SCREENING

<p>What are routine intake prenatal labs?</p>	<p>Hematology: blood type and screen, Hemoglobin and hematocrit</p> <p>Immunity and infectious: rubella status (immune or nonimmune)</p> <p>HIV</p> <p>HBV (HBsAg & HBsAb)</p> <p>Syphilis (RPR or VDRL)</p> <p><i>Chlamydia</i> and gonorrhea cervical cultures</p> <p>tuberculosis (PPD)</p> <p>urine culture</p> <p>Cytology: Pap smear</p>
--	---

What are some appropriate screening tests for the following groups?

Mediterranean descent MCV or Hgb electrophoresis for thalassemia (defect in the α or β chain)

African descent Hgb electrophoresis for sickle cell

Ashkenazi Jews descent

Hexosaminidase A leukocyte assay for Tay-Sachs

DNA analysis for: Canavan disease, Bloom syndrome, cystic fibrosis, familial dysautonomia, Fanconi anemia, Gaucher disease, mucopolysaccharidosis Type IV, Niemann-Pick disease, or Tay-Sachs disease

Caucasian descent Delta F 508 mutations for cystic fibrosis (autosomal recessive)

Serum phenylalanine level for phenylketonuria

In an Rh(−) pregnant woman with an Rh(+) father of the baby, would her first or second child be at greatest risk?

Her **second child**, because of isoimmunization and the development of anti-Rh antibodies. The first Rh(+) fetus will only be mildly affected, if at all

What is the major complication associated with isoimmunization?

Hemolytic disease of the newborn (with possible hydrops, anasarca, or death)

How is Rh isoimmunization prevented?

With Rh immune globulin (RhoGAM)—an antibody to the D antigen

Who is given RhoGAM?

All Rh(−) mothers with a possible Rh(+) fetus

When is RhoGAM given?

At 28 weeks EGA

At delivery

Within 72 hours of an abortion or vaginal bleeding at any gestational age

Following all invasive procedures (CVS or amniocentesis)

Maternal isoimmunization to which antibodies affect fetal outcome?

Blood cell antigens: C, D (Duffy), E, K (Kell), and Rh

“Duffy dies, Kelly kills, Lewis lives” (L antigen does not have a significant deleterious effect on the fetus)

Name some additional screening and diagnostic tests. At what EGA are these routinely completed?

Nuchal translucency screen (with serum free β -hCG and PAPP-A)
11–13 weeks

Quad or triple screen 15–18 weeks

Anatomic survey with ultrasound (to assess for fetal anomalies)
18–20 weeks

Glucose challenge test (GCT)
24–28 weeks

Streptococcus group B (perineal and rectal culture) 36 weeks

Hgb, Hct, and syphilis >28 weeks (third trimester)

In appropriate patients what tests should be repeated? At what EGA?

Gonorrhea, *Chlamydia*, and HIV >28 weeks (third trimester)

Antibody testing in unsensitized Rh(D[–])women 28–30 weeks

What is the nuchal translucency?

A cystic space dorsal to the cervical spine measured by ultrasound at 11–13 weeks. It is a **sensitive indicator of chromosomal abnormalities**; a larger diameter signifies a greater risk of aneuploidy and poor fetal outcome

What other tests enhance the sensitivity of a nuchal translucency?

Maternal serum free β -hCG and plasma protein A (PAPP-A) measurements

What is measured in the triple screen? In the quadruple screen?

Triple screen: α -fetoprotein (AFP), β -hCG, and estriol (E3)

Quad screen: all of above plus **inhibin A**

When are these tests employed and what do they assess?

Between 16–20 weeks
Abnormal values correlate with various chromosomal, genetic, and developmental disorders

How should an abnormal triple/quad screen be followed-up?

With a detailed ultrasound and possibly an amniocentesis

What do the following triple screen results suggest?

↓ AFP, ↑ hCG, ↓ E3: Trisomy 21

↓ AFP, ↓ hCG, ↓ E3: Trisomy 18

An elevated AFP may indicate what fetal conditions?

Neural tube defects

Gastroschisis (abdominal wall defect, often lateral to the rectus on the right)

	Omphalocele (midline umbilical hernia covered by peritoneum)
A decreased AFP may indicate what fetal condition?	Down syndrome
What are the most common neural tube defects (NTDs)?	Anencephaly and spina bifida
Acetylcholinesterase is a specific marker for NTD when it is increased or decreased in the amniotic fluid?	Increased
What is a Glucose tolerance test (GCT)?	A screening test for gestational diabetes. The patient drinks 50 g of glucose and her blood sugar is tested 1 hour later
What is the cutoff for the GCT?	Serum glucose >130–135mg/dL
What should be done if a patient has an abnormal GCT?	A diagnostic test called the glucose tolerance test (GTT)
What is a Glucose tolerance test (GTT)?	After an overnight fast, blood sugar is tested. 100 g glucose is then given and blood sugar is checked every hour for 3 hours
What are the normal values for a GTT?	Fasting <95 mg/dL 1 hour <180 mg/dL 2 hours <155 mg/dL 3 hours <140 mg/dL
When is gestational diabetes diagnosed?	If there are two or more abnormal values on the GTT
What is chorionic villus sampling (CVS)?	A diagnostic procedure for chromosomal and genetic anomalies that involves transvaginal or trans-abdominal aspiration of placental cells at 9–12 weeks EGA
What is the risk of CVS?	There is a 1/200 risk of adverse fetal outcome or demise
What is an amniocentesis?	A diagnostic procedure done at >15 weeks that can detect chromosomal/genetic abnormalities, amniotic infection, inflammation, and fetal lung maturity. It involves trans-abdominal aspiration of the amniotic fluid from the uterine cavity
What is the risk of amniocentesis?	There is a 1/300 risk of adverse fetal outcome or demise

PRENATAL TESTING AND MONITORING OF THE FETUS

What is quickening and when does it occur?

The mother's first **perception of fetal movement (FM)**; it usually occurs **between 16–20 weeks** (may be earlier in a multipara)

What is a "kick count"?

After 26–32 weeks, fetal well-being can be assessed by asking the mother to count FM, "kick counts," which should occur eight times every 2 hours

What are the commonly used tests of fetal well-being and when are they used?

Non-stress test (NST) and biophysical profile (BPP)

They are used most commonly for: **decreased fetal movements, diabetic mother, post-dates, chronic HTN, intrauterine growth restriction (IUGR)**

What is an NST?

Placement of an EFM to trace the fetal heart variability (which rises with fetal movement). A tocodynamometer monitor is also placed to assess for uterine contractions (See Fig. 8-3)

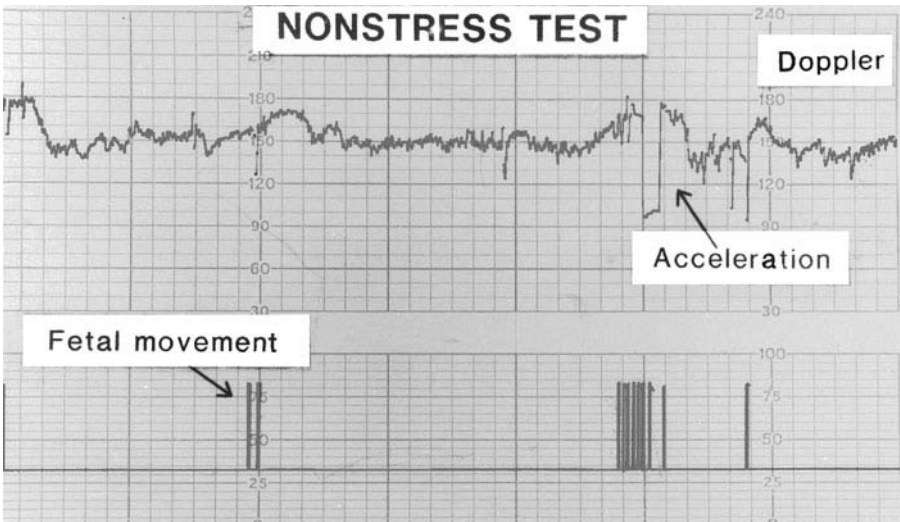


Figure 8-3 Reactive nonstress test. (Reproduced with permission, from Cunningham FG et al: *Williams Obstetrics*, 22nd ed. New York. McGraw-Hill, 2005:279.)

What is a reactive NST?	Two accelerations above the baseline of 15 beats per minute (bpm) for 15 seconds within 20 minutes
What does a reactive NST indicate?	Good vagal tone and reassuring fetal status
What is fetal tachycardia?	A baseline FHR > 160 bpm
What is the most common cause of fetal tachycardia?	Maternal tachycardia
What are some other causes of fetal tachycardia?	Maternal fever Anemia Asphyxia Infection Autoimmune disorders Adrenergic medications Cardiac anomalies (e.g., SVT)
What is fetal bradycardia?	A baseline FHR < 110 bpm
What are some causes of fetal bradycardia?	Physiologic (short episodes because of transient compression of the fetal head/umbilical cord) Maternal hypotension Local anesthesia (e.g., paracervical block) Uteroplacental insufficiency (e.g., placental abruption, uterine rupture, cord prolapse) Cardiac anomalies
How is fetal heart rate variability assessed?	10 minutes of fetal heart tracing is reviewed to assess peak-to-trough long-term variability. Variability may be: absent minimal (<5 bpm) moderate (normal = 6–25 bpm) marked (>25 bpm)
What is suggested by a sinusoidal pattern on EFM?	Severe fetal anemia, hypoxia, or exposure to sedative hypnotics
What are the five components of the bio-physical profile BPP?	1. Breathing: 30 seconds after 30 minutes of observation 2. Movement: three gross body movements in 30 minutes

What do each of the following BPP scores signify?

3. **Tone:** extension and flexion of an extremity

4. **Amniotic fluid:** vertical pocket > 2 cm

5. **Reactive NST**

Each is worth 2 points for 10 possible points. The fetus is given 30 minutes to demonstrate each variable

8–10 Reassuring fetal status with an intact CNS

6 Equivocal test; repeat in a few hours

4 High risk of fetal hypoxia; consider delivery

0–2 Fetal hypoxia; delivery immediately regardless of EGA

Other than fetal hypoxia, what are some other causes of a low BPP?

Fetal sleep cycle

Transplacental sedatives

Corticosteroids

Which of the BPP elements are lost first as the fetus becomes progressively more hypoxic?

Breathing is lost early, then FHR accelerations, movement, and finally tone

How is chronic fetal stress manifested?

Oligohydramnios (AFI <5 cm or vertical pocket <2 cm), because of several days of decreased renal perfusion

During hypoxic stress, where is fetal blood preferentially shunted?

Brain, heart, adrenals, and placenta

How is fetal perfusion assessed with doppler ultrasound?

Systolic versus end diastolic flow velocity (**S/D flow**) in the umbilical vessels, middle cerebral artery, and ductus venosus

What is a normal S/D ratio?

Less than three (because of low placental resistance) in the third trimester; however, this ratio is dependent on gestational age

What is a high S/D ratio associated with?

PEC and IUGR. Reversed diastolic flow suggests a poor fetal outcome

Before the development of NST/BPP, what test was performed to assess uteroplacental insufficiency?

A **contraction stress test (CST)**.

Nipple stimulation or oxytocin was administered to induce three contractions (ctx) every 10 minutes with concurrent FHM. A normal test

	has no late decelerations. This is rarely performed today
What are the three types of decelerations?	Early, late, and variable
Describe early decelerations.	A gradual decrease from baseline that mirrors a contraction
What do early decelerations signify?	A vagal response from compression of the fetal head during uterine ctx; they are normal
Describe variable decelerations.	A rapid decline of more than 15 beats from the baseline that is unrelated to uterine ctx
What do variable decelerations signify?	Usually cord compression (can be relieved by changing the mother's position)
Describe late decelerations.	A gradual decrease from baseline that starts at the peak of the contraction and persists until the ctx is finished
What do late decelerations signify?	Uteroplacental insufficiency
Fetal metabolic acidosis and hypoxia are suggested by what findings on a fetal heart tracing?	Recurrent (>three), prolonged (>3 minute), late decelerations Minimal (>5 bpm), decreased or absent long-term variability Tachycardia (>160 bpm), which may also be associated with infection or maternal fever
What is the significance of irregular contractions in the third trimester?	If the fetal status is reassuring, there is no cervical change (patient remains closed/long/high), and ctxs are less than 4 per hour (they are likely insignificant Braxton Hicks contractions .) If the contractions are painful, the patient may be in latent labor
What is fetal fibronectin?	A glycoprotein that is normally present in maternal circulation and amniotic fluid. If it is present in the cervicovaginal secretions (>50 ng/mL), it suggests that the cervix is undergoing structural change and the patient may go into labor in the next 2 weeks
How is fetal fibronectin used clinically?	It has a high negative predictive value, so it is used to rule out preterm labor

Labor and Delivery

DIAGNOSIS AND DEFINITIONS

What percentage of deliveries at U.S. hospitals are normal spontaneous vaginal deliveries (NSVDs) with no intervention?	Approximately 50%
How is labor defined?	Regular uterine contractions that result in cervical effacement and dilation and eventual expulsion of the fetus and placenta
How is labor diagnosed?	With tocodynamometry and serial cervical examinations
What are some of the signs of labor?	Painful contractions Bloody show Spontaneous rupture of membranes (SRM)
What is bloody show?	Vaginal passage of blood-tinged mucus
What are the four characteristics of cervical change?	Change in consistency (from firm to soft) Change in position (from posterior to anterior) Progressive effacement (cervix becomes shorter and thinner) Dilation of the internal and external os (from 0 to 10 cm)
The following terms refer to rupture of fetal membranes (ROM) under what conditions?	PROM ; premature rupture of membranes; at least 1 hour before the onset of labor PPROM ; preterm, premature rupture of membranes; EGA <37 weeks with rupture at least 1 hour before the onset of labor Prolonged PROM ; rupture of membranes >18 hours without the onset of labor
What are the four signs and symptoms of spontaneous rupture of membranes?	Initial gush with continued loss of fluid

	<p>Pooling of vaginal fluid on sterile speculum examination</p> <p>Positive nitrazine blue test (indicating that the vaginal fluid is alkaline, with a pH greater than 6)</p> <p>Ferning of dried fluid under low power magnification (because of fetal urine salt crystals in the amniotic fluid [AF])</p>
<p>What can cause a false-positive nitrazine blue test?</p>	<p>Anything that causes the vagina to become more alkaline, such as:</p> <ul style="list-style-type: none"> Sperm Blood Infection Douching
<p>What is the pH of amniotic fluid?</p>	<p>7.0</p>
<p>Describe the following terms:</p>	<p>Nulligravida; a woman who never conceived a fetus</p> <p>Nullipara; a woman who never carried a fetus to viability</p> <p>Primipara; a woman who has delivered a viable fetus in the past, regardless of the outcome of the fetus</p> <p>Multigravida; a woman who has carried more than one fetus to viability, regardless of the outcome of the fetus</p> <p>Grand multiparity; given birth five or more times</p>
<p>What is the term for a difficult delivery, protracted labor, or arrest of labor?</p>	<p>Labor dystocia</p>
<p>How do labor forces and the passage of the fetal head through the birth canal affect the fetal head (in cephalic presentations)?</p>	<p>The fetal calvarium undergoes molding, where the bones of the skull shift to minimize the diameter that must pass through the bony pelvis</p>
<p>On vaginal examination, the presenting fetal vertex is noted to be soft without any identifiable sutures or fontanelles. What is the term for this fetal finding (which often occurs in prolonged labors with slow cervical dilation)?</p>	<p>Caput succedaneum; the tissues overlying the fetal calvarium become edematous and swollen</p>

How is cardiac output (CO) affected during labor?	There is a 50% increase in CO during the second stage (preload increases as blood from the uterine sinusoids enters systemic circulation)
How much does maternal blood pressure increase during labor?	Systolic blood pressure increases up to 25% and diastolic blood increases up to 15%. It is increased during uterine contractions, with pain or anxiety, and with maternal positions that affect venous return. The pressure in the amniotic fluid, cerebrospinal fluid, and dural compartments also increase with labor
Why are women in labor predisposed to gastric aspiration?	Increased intra-abdominal pressure Relaxation of the lower esophageal sphincter Recumbent laboring position
What are some of the consequences of gastric aspiration?	Pneumonia Bronchospasm Adult respiratory distress syndrome (ARDS)
What precaution is taken in labor to minimize this risk?	Oral intake is restricted to occasional ice chips
What is a pudendal block?	A method of administering local anesthesia to the pudendal nerve (sacral nerve roots 2, 3, and 4), with subsequent decreased vulvar sensation
How is a pudendal block administered?	Transvaginally; the anesthetic is injected medial and inferior to the ischial spines through the sacrospinous ligaments bilaterally
Where is an epidural catheter placed?	A guide needle is used at the interspinous space between the fourth and fifth lumbar vertebra. The ligamentum flavum is penetrated and the catheter is threaded into the potential epidural space, which is comprised of lymphatics and venous plexuses. An epidural injection enters the extradural or peridural space; it does not penetrate the dura mater
Where is a spinal block placed?	It enters the dura and then the subarachnoid space, bathed by

	cerebral spinal fluid. Only the pia mater separates the cord from the injected substance
What are the risks associated with epidurals and spinal blocks?	Spinal headache Hypotension Infection Hematoma High spinal blockade Cord compression (a surgical emergency)
What advantages does a spinal block offer?	A faster onset and requires a lower dose of anesthetics
What risks are increased with a spinal block?	Hypotension (sympathectomy causing peripheral vasodilatation) Nausea Compromised placental perfusion Ascending respiratory paralysis (because of anesthesia reaching the cervical nerve roots 3, 4, & 5)
Does regional, local, or general anesthesia increase the rate of cesarean delivery?	No

FETAL POSITION

What is the fetal lie?	The crown-rump axis of the fetus in relation to the longitudinal axis of the uterus
What types of fetal lie are there?	Longitudinal Transverse Oblique
What is the fetal presentation?	The fetal part closest to the cervix and pelvic inlet
What is the most common fetal presentation?	Cephalic, followed by breech
What are the other types of fetal presentation? Describe them.	Types of cephalic presentation: Vertex Brow Mentum

Face (depending on which part is leading through the cervix)

Types of breech presentation:

Frank breech (flexed hips and extended knees)

Complete breech (flexed hips and knees)

Footling breech (one knee flexed, one knee extended) (See Fig. 8-4)

Other:

Hand

Shoulder

Funic

Compound (involves more than one fetal part leading) e.g., cord and head or hand and head)

Less than 4%

What is the incidence of mal-presentation (any presentation not cephalic) at the onset of labor?

Fetal attitude or posture describes what characteristic?

The degree of flexion of the fetal neck, back, and joints of the limbs

Describe the fetal postures from most flexed to most extended

Vertex → Military → Brow (forehead) → Face

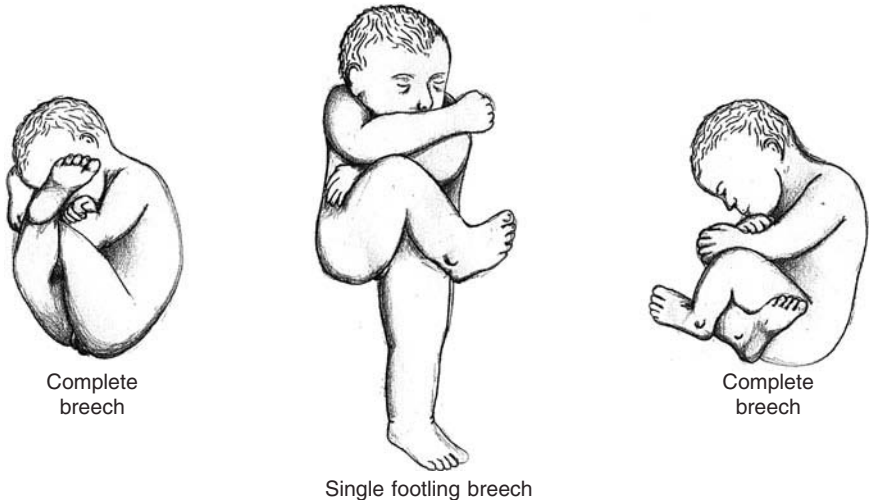


Figure 8-4 Breech presentation.

What are the risks associated with an extended neck?

The fetus requires a larger leading diameter and thus has less ability to negotiate the birth canal. This can lead to **labor dystocia**

What are the shapes of the two fetal fontanels and when do they close?

The **anterior** or frontal fontanel is **diamond shaped** and closes late in infancy (near 13 months). The **posterior** or occipital fontanel is **triangle shaped** and closes early in infancy (near 2 months)

During labor how are the fetal fontanels examined? What is the significance of this examination?

The fetal scalp is examined through the dilating cervix on sterile vaginal examination (SVE). The **location of the posterior fontanel** is noted with regard to **maternal left/right** and **anterior/posterior/transverse orientation**. This describes the specific **position** of the presenting fetal head. (See Fig. 8-5)

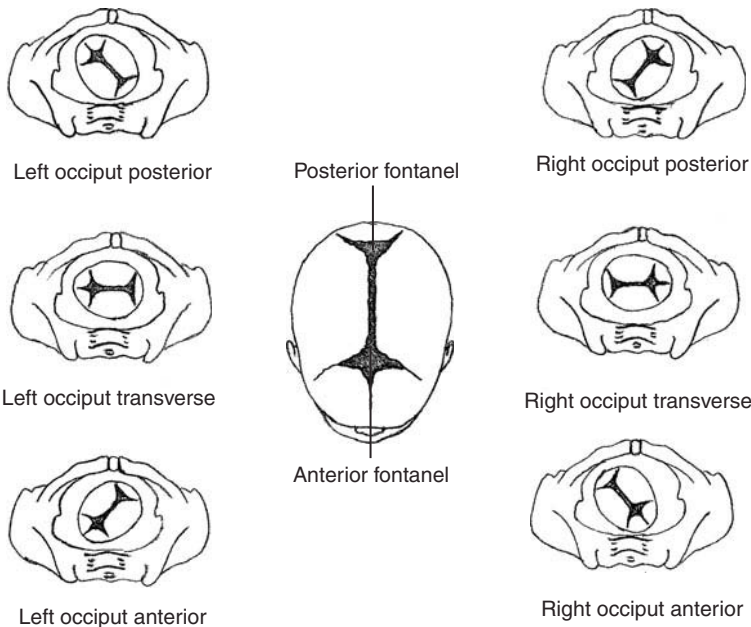
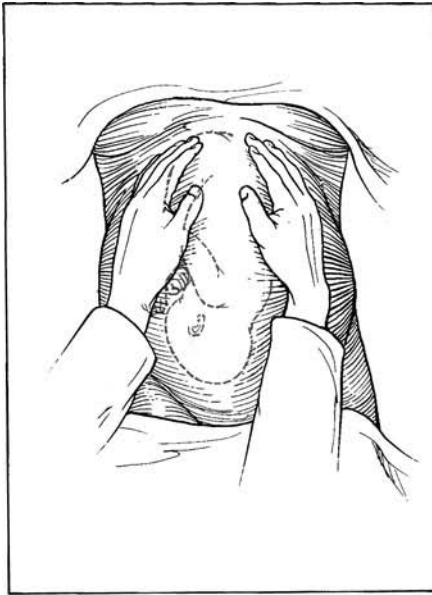


Figure 8-5 Vertex presentations.

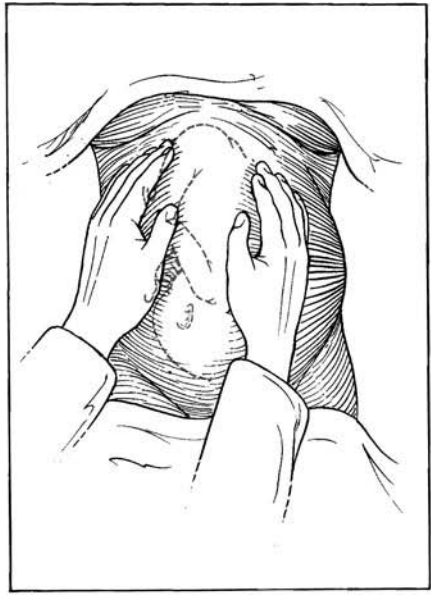
Why does the occiput posterior (OP) position frequently cause labor dystocia?

Because the fetal head must be more flexed and rotate more extensively (135 degrees instead of 90 degrees)

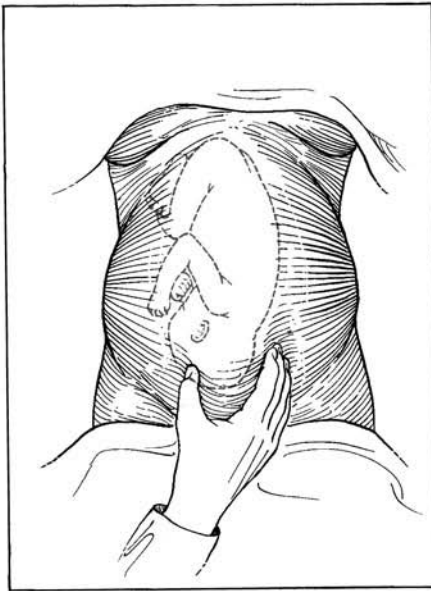
What is a normal synclitism?	to pass under the symphysis pubis. Additionally, this position is often associated with brow or face presentations
What is anterior asynclitism?	When the leading sagittal suture is parallel to the pelvic outlet
What is posterior asynclitism?	When the sagittal suture is deflected toward the sacrum , allowing more of the parietal bone to be palpated anteriorly
How is the position of a fetus described in a face or breech presentation?	Deflection of the fetal sagittal suture toward the maternal symphysis pubis. It is normal unless the tilt is severe
Leopold maneuvers convey what information about the fetus?	The relationship of the fetal mentum (chin) or sacrum are described in relation to maternal right, left, anterior, and posterior
What are the four Leopold maneuvers?	Estimated fetal weight (EFW) Fetal presenting part Fetal lie Engagement
	First position: hands are placed at the cephalic margins of the fundus, to determine the nonpresenting part that occupies the fundus, and the fetal lie
	Second position: hands are placed at the right and left margins of the fundus, to feel for small fetal parts, to confirm fetal position
	Third position: thumb and finger are placed just above the symphysis pubis to assess engagement of the presenting part
	Fourth position: facing the patient's feet, the examiner's fingers trace the fundus toward the pelvic inlet to identify the anterior shoulder with cephalic presentation and to assess the degree of descent of the presenting part (See Fig. 8-6)



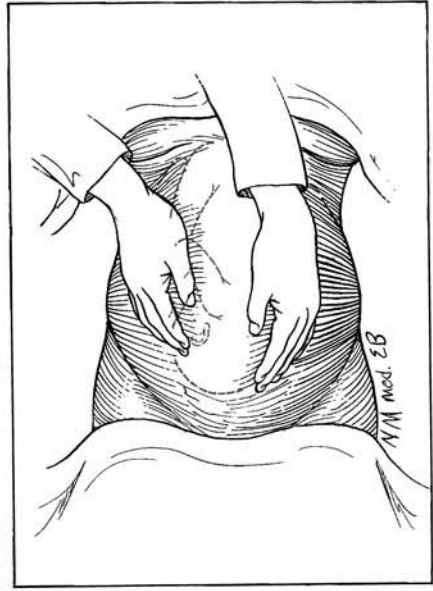
First maneuver



Second maneuver



Third maneuver



Fourth maneuver

Figure 8-6 Leopold maneuvers. (Reproduced with permission, from Cunningham FG et al.: *William Obstetrics*, 22nd ed. New York: McGraw-Hill, 2005:416.)

PELVIMETRY AND VAGINAL EXAMINATION

The pelvis is composed of what eight bones?	Ilium ($\times 2$), ischium ($\times 2$), pubis ($\times 2$), sacrum, coccyx
When is an SVE indicated?	During PNC visits at term Upon presentation to labor and delivery with symptoms of labor Periodically throughout the course of labor (approximately every 2 hours) to assess progress
When should a manual pelvic examination <i>not</i> be performed?	When there is bright red blood per vagina (because of risk of increased trauma with vasa previa or placenta previa), ultrasound should be performed first to confirm no previa
What information is gathered during a manual pelvic examination?	Fetal position Cervical dilation/cervical effacement/station of the presenting fetus Cervical consistency (firm, soft) Cervical position (posterior, anterior) Clinical pelvimetry (diagonal conjugate, pelvic sidewalls, interspinous diameter, and a wide pubic arch)
What is the interspinous diameter and what is its significance?	It is the distance between the ischial spines and is used to estimate the station of the presenting fetal part
What station is associated with each of the following positions?	A leading edge that is -3 cm above the ischial spines (-3 station) A leading edge that is at the level of the spines (0 station) A leading edge that is 3 cm past the spines (+3 station)
What is zero station (determined on SVE)?	When the leading fetal edge is at the level of the maternal ischial spines
What is its obstetrical significance of zero station?	It represents the most narrow sagittal obstetric diameter and so signifies that the largest fetal diameter has engaged the bony pelvis
What modalities can be used for pelvimetry?	X-ray, computed tomography, magnetic resonance imaging, and physical examination

What is the significance of pelvic classification and clinical pelvimetry?

Clinical pelvimetry seeks to describe the pelvic inlets, angles, and diameters. However, pelvic type and pelvimetry are not reliable predictors of vaginal delivery, labor dystocia, or cesarean section and so they are rarely employed in contemporary obstetrics

What is the best indicator of pelvic adequacy?

Prior vaginal delivery, prior progress of labor, or family history of cephalopelvic disproportion (CPD)

From what reference point are all three anterior posterior (AP) pelvic diameters measured?

From the sacral promontory

List the following from narrowest to widest: true, obstetric, and diagonal conjugates

Obstetric < true < diagonal

Which conjugate can be directly measured on physical examination?

The **diagonal conjugate** (or transverse diameter)

How is the diagonal conjugate measured?

From the inferior margin of the pubic bone to the sacral promontory (average diameter 12.5 cm)

How is the diagonal conjugate used?

To indirectly assess the obstetric conjugate (which is 2 cm shorter and cannot be measured by clinical examination)

What is the obstetric conjugate? What is its significance?

Measured from the middle of the pubic bone to the sacral promontory, it is the **shortest AP diameter** of the bony pelvis (average diameter 10.5 cm)

What is the true conjugate?

Also known as the anteroposterior diameter or conjugate vera, it is the **diameter at the inlet of the true bony pelvis** (average diameter 11 cm). It is measured from the superior margin of the symphysis pubis to the sacral promontory

STAGES OF LABOR

What is the biggest difference between the labor of nulliparous and multiparous women?

Labor tends to be longer in nulliparous women, who are also more likely to experience a failure to progress

What defines the first stage of labor?

The onset of **consistent painful contractions** until the cervix is

The first stage of labor is considered to be abnormal when it exceeds what length? **completely dilated (10 cm) and 100% effaced**
Over 24.7 hours in nulliparas; over 18.8 hours in multiparas (based on Friedman data)

How is the first stage of labor further subdivided? **Into latent and active phase**
(See Fig. 8-7)

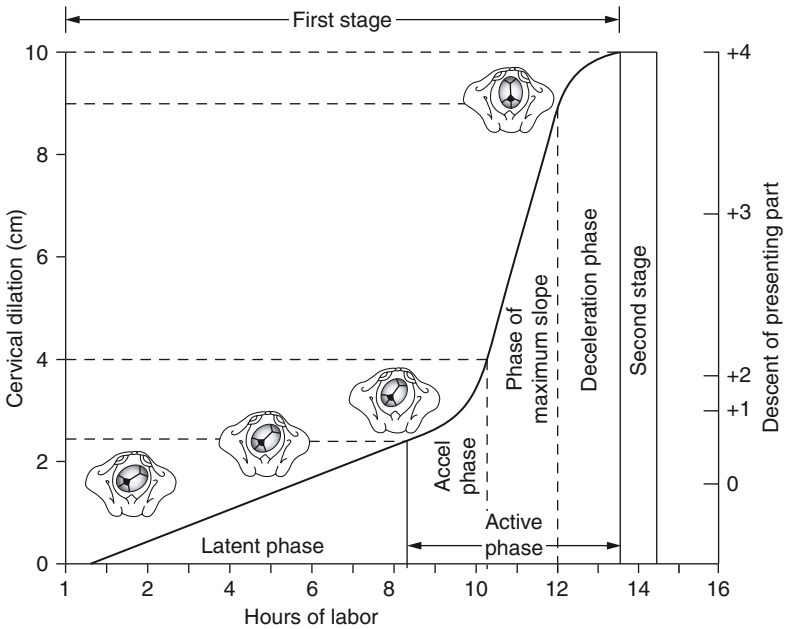


Figure 8-7 Schematic illustration of progress of cervical dilation against time in the successive stages of labor.

Describe the latent phase

Regular painful contractions every 5–10 minutes that result in **cervical dilation up to 3–5 cm**

Describe the active phase

A faster rate of cervical dilation which typically begins after 3–5 cm dilation in the presence of regular uterine contractions that **continues through full cervical dilation of 10 cm**

What are minimum adequate rates of cervical dilation during the active phase?

For **multiparas**: at least **1.5 cm/h**
 For **nulliparas**: at least **1.2 cm/h**

What are some conditions that slow the active phase?	Uterine dysfunction Fetal malposition CPD
How is the active phase of the first stage of labor further subdivided?	Acceleration phase: an increasing rate of dilation from 3-5 cm cervical dilatation to 8 cm Deceleration phase: a slower rate of cervical dilation after 8 cm until full dilation
How is arrest of dilation defined?	>2 hours with no cervical change in the presence of adequate contractions
What are adequate contractions?	More than 200 Montevideo units (MU) as calculated by internal fetal monitoring
What is the average duration of the active phase in nulliparous patients?	3.3 hours
What is the second stage of labor?	From a fully dilated and fully effaced cervix until delivery of the fetus
What is the average duration of the second stage of labor?	In nulliparas: 54 minutes (but can be up to 146 minutes) In multiparas: 18 minutes (but can be up to 64 minutes)
What is a protracted labor?	When there continues to be some cervical change or descent of the fetus; however, it is progressing slower than would be expected for the parity
Define protraction of the second stage in terms of fetal descent	In nulliparas: fetal head descent <1 cm/h In multiparas: fetal head descent <2 cm/h
Define protraction of the second stage in terms of length of labor (without anesthesia)	In nulliparas: >118 minutes (or 2 hours) In multiparas: >47 minutes (or 1 hour)
What is arrest of labor?	Complete cessation of dilation (no change for 2 hours) or descent (no change for 1 hour) despite adequate contractions
What are the causes of secondary arrest of labor?	The three "P"s: Power (inadequate uterine contractions)

Define arrest of descent	<p>Passage (pelvic disproportion)</p> <p>Passenger (malpresentation or hydrocephalus)</p> <p>More than 2 hours in a nullipara or >1 hour in a multipara with no descent of the fetus despite adequate contractions</p> <p>If the patient has had an epidural or regional anesthesia she is given 1 additional hour</p>
When should a woman begin pushing to aid in the descent and delivery of the fetus?	During the second stage (after full cervical dilatation)
Why is pushing avoided in the first stage of labor?	To prevent cervical lacerations and maternal exhaustion
A nulliparous patient at term has dilated from 6 to 7 cm over 2 hours. Is this adequate change, protracted dilation, or arrest of dilation during the active phase of labor?	Protracted dilation (a nullipara should dilate at least 1.2 cm/h during the active phase of the first stage of labor)
In the above patient, what should be done?	Her contraction pattern should be evaluated and, in the setting of reassuring fetal status, her labor can be augmented if her contractions do not appear to be adequate
A rate of cervical dilation >5 cm/h in a nulliparous or 10 cm/h in a multiparous patient is considered what type of labor?	Precipitous labor
What risks are associated with precipitous labor?	An increased risk of fetal hypoxia, brain injury, and maternal morbidity such as hemorrhage and vaginal/cervical lacerations
A multiparous patient progressed from 3 cm on admission to 10 cm in 30 minutes, and pushed for 2 hours to deliver a viable baby girl. How would you describe the first and second stage of her labor?	A precipitous first stage with a significantly protracted second stage of labor. Greater than 20 minutes for multiparas is considered a prolonged second stage
How is the third stage of labor defined?	It begins after delivery of the fetus and continues until the placenta is delivered
What is the acceptable duration of the third stage of labor?	Less than 30 minutes (although some clinicians will wait up to 60 minutes)
What are the three Gs that indicate the placenta has separated and is ready to be delivered?	<p>Globular uterus</p> <p>Growing cord</p> <p>Gush of blood</p>

What is the fourth stage of labor?

The acute maternal **hemodynamic adjustment to the fluid shifts** associated with labor that lasts 1–2 hours after delivery of the placenta

Why is the woman at increased risk for in the fourth stage of labor?

Postpartum hemorrhage

CARDINAL MOVEMENTS OF LABOR

What are the seven cardinal movements of labor?

The positions that describe the behavior of the fetal head during the second stage of labor. They include **engagement, descent, flexion, internal rotation, extension, external rotation** (or restitution), and **expulsion** (See Fig. 8-8)

Fetal engagement occurs at what station?

0 station (when the leading fetal edge has reached the ischial spines)

What is considered to be inadequate downward passage of the fetus during the second stage of labor?

Less than 1 cm/h in a nulliparous patient and less than 2 cm/h in a multiparous woman

Why does a fetus flex and rotate during labor?

To negotiate the 90 degree concave curvature of the pelvic passage (the curve of Carus)

The fetus presents the smallest diameter of its head (suboccipito-bregmatic diameter) by engaging what position?

Tight anterior **flexion** of the head (the inability to flex the head may lead to a dystocia)

How does the fetal head undergo internal rotation?

The fetus rotates so as to turn the saggital suture from a transverse to an anteroposterior position, it is a passive movement

When does the fetal head undergo extension?

As the fetus is crowning at the introitus and the head has passed under the symphysis pubis

What cardinal movements occur while the fetal head is at the introitus?

Extension and external rotation

After the fetal head is delivered what subsequent steps should the clinician take?

1. Check for nuchal cord (umbilical cord around the neck)
2. Apply gentle downward pressure to assist the delivery of the anterior shoulder

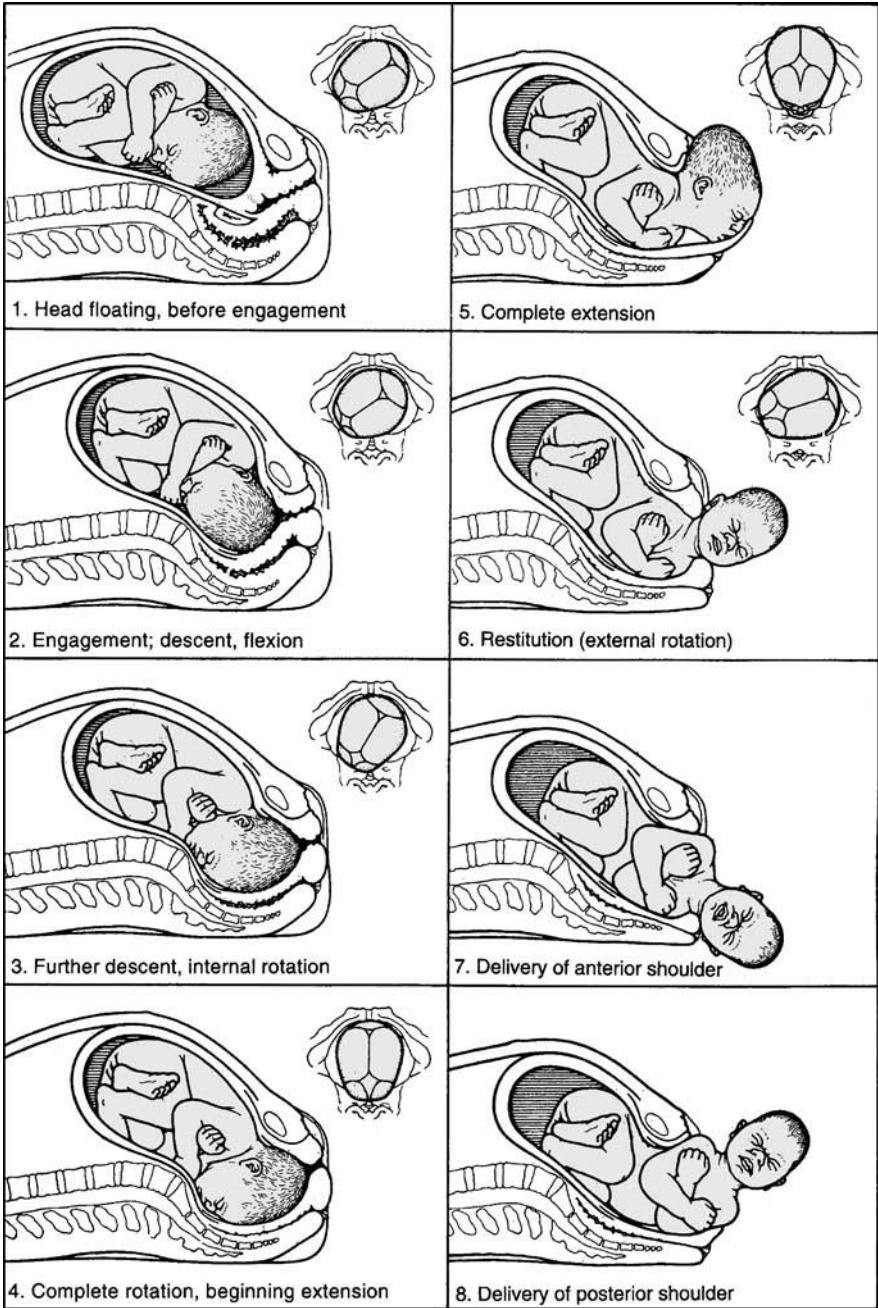


Figure 8-8 Cardinal movements of labor. (Reproduced with permission, from Cunningham FG et al: *Williams Obstetrics*, 22nd ed. McGraw-Hill, 2005:418.)

3. Gentle upward pressure for the posterior shoulder
4. Support the subsequent delivery of the fetus's body

INDUCTION OF LABOR

What are some indications for induction of labor (IOL)?

Prolonged pregnancy (postterm)
 Diabetes mellitus
 Rh alloimmunization
 Preeclampsia
 ROM at term
 Placental insufficiency
 Non-reassuring fetal status
 Oligohydramnios
 Intrauterine growth restriction

What are some contraindications to IOL?

Previous cesarean with a classical uterine incision
 Prior uterine surgery involving the myometrium
 EFW >4500 g
 Severe fetal hydrocephalus
 Malpresentation (breech)
 Cervical cancer
 Active genital herpes
 Placenta or vasa previa

What is the Bishop score?

A rating system that evaluates a woman's cervix on SVE to **predict the likelihood that she will be able to complete a vaginal delivery following IOL**; a score of 5 or greater is favorable

What are the five cervical components of the Bishop score?

Consistency: firm 0, medium 1, soft 2
Position: posterior 0, middle 1, anterior 2
Effacement: 0–100% (0–3)
Dilation: 0–10 cm (0–3)
Station: –3 to +2 (0–3)
 Mnemonic: “see peds” CPEDS

What Bishop scores correspond to the following probabilities of failed IOL?

Less than 50%: 0–4

Less than 10%: 5–9

Less than 1%: 10–13

Preinduction cervical ripening can be achieved with what medications?

Prostaglandin PGE1: **misoprostol** (tablets)

PGE2 or dinoprostone: **prepidil** (cervical gel) and **cervadil** (vaginal insert)

What mechanical techniques are used to ripen the cervix for IOL?

Hygrosopic cervical dilators (laminaria)—seaweed sticks that absorb water and expand

Balloon; tipped transcervical catheter or Foley bulb

Membrane stripping or rupture to increase endogenous prostaglandins

INTRAPARTUM ASSESSMENT AND MONITORING

Labor depends on the coordination of what three variables?

Passage, powers, and passenger

What are the components of the maternal pelvis (passage)?

Soft tissues (cervix, vagina, pelvic floor muscles)

Bony pelvis (ilium, ischium, pubis, sacrum, coccyx)

What is defined as adequate uterine contractions (power)?

3–5 contractions every 10 minutes (or 5–7 q 15 minutes)

What techniques can be used to count the number of contractions?

Palpation

External tocodynamometry

Intrauterine pressure catheter (IUPC)

Which of these devices is able to assess the force of the contractions?

Only IUPC

What are Montevideo units?

Quantifications of intrauterine pressure (IUP) as determined by IUPC. They are calculated by **multiplying the average peak strength** of the contraction **by the number of contractions** over 10 minutes

At what target MU are contractions considered adequate?

>200

What is uterine tachysystole?

More than five contractions in 10 minutes with a non-reassuring fetal heart tracing

What is uterine hyperstimulation?	More than five contractions in 10 minutes with a non-reassuring fetal heart tracing
What is a tonic contraction?	A contraction that lasts more than 3 minutes (in the context of a non-reassuring fetal tracing or fetal decelerations)
Why does uterine hyperstimulation/tonic contraction compromise placental blood flow?	The pressure in the placental sinuses exceeds maternal systolic BP, resulting in insufficient perfusion and fetal hypoxia
What aspects of the fetus can impact labor (passenger)?	Size (macrosomic >4000 g) Lie, presentation, attitude, position, asynclitism Number of fetuses Anatomic anomalies (sacroccygeal teratoma)
What are the ideal fetal characteristics for negotiating the maternal pelvis?	A small fetus, longitudinal lie, flexed OA at 0 station
What is the average EFW at term?	3000–4000 g
Fetal scalp electrode (FSE) monitoring offers what benefits to electronic fetal monitoring (EFM)?	Continuous monitoring of the fetal heart rate in an obese patient or difficult to monitor fetus Precise beat-to-beat assessment of variability and decelerations
How is the FSE applied?	Placental membranes must be ruptured and the electrode is adhered to the fetal calvarium
In what clinical setting is fetal blood sampling (fetal scalp pH) performed?	To acutely assess hypoxemia in the setting of non-reassuring FHR tracing, meconium, or other signs of fetal distress
What is suggested by the following range of fetal scalp pHs?	pH >7.25 reassuring; a low probability of hypoxic-ischemic encephalopathy pH 7.24-7.20 indeterminate; should be repeated in 30 minutes pH <7.20 a non-reassuring pH; delivery should be expedited with operative intervention
What other clinical information can be utilized to improve the predictive value of a low scalp pH?	FHR variability

MANAGEMENT

<p>What is the active management of labor?</p>	<p>The proactive utilization of various techniques and augmenting interventions, with the goal of shortening the duration of labor and reducing cesarean deliveries</p>
<p>What can be used to augment latent labor or prolonged early labor?</p>	<p>Cervical ripening agents (prostaglandins)</p> <p>Cervical dilators (laminaria or a Foley)</p> <p>Intravenous oxytocin</p> <p>An amniotomy</p>
<p>Within what time frame following ROM is it recommended to administer antibiotic prophylaxis to GBS(+) patients?</p>	<p>Within 6 hours of ROM or at the onset of labor</p>
<p>What is the effect of oxytocin (Pitocin) on labor?</p>	<p>It increases the frequency and force of uterine contractions</p>
<p>What is the half-life of oxytocin?</p>	<p>3–5 minutes</p>
<p>A nulliparous patient is receiving 15 mU/min of intravenous oxytocin to augment her labor. Mother and fetus are tolerating labor well. There is no evidence of CPD, and an IUPC indicates 180 MU. What is the next step in management?</p>	<p>Increase the oxytocin (up to 30–50 mU/min); the goal is to achieve >200 MU</p>
<p>What are the risks involved with prolonged high-dose oxytocin administration?</p>	<p>Maternal SIADH and excessive fluid retention (pulmonary edema)</p> <p>Hyperstimulation of the uterus</p> <p>Postpartum atony and hemorrhage</p> <p>Hyperbilirubinemia of the infant</p>
<p>Why is an amniotomy or assisted rupture of membranes (AROM) performed?</p>	<p>To induce or augment labor</p> <p>To assess for meconium in the amniotic fluid</p> <p>To place an IUPC or FSE</p>
<p>If the fetal head is not engaged in the pelvis or well applied to the cervix why is an amniotomy contraindicated?</p>	<p>Because of the risk of umbilical cord prolapse, compression by the presenting fetal part, and subsequent fetal hypoxia</p>
<p>A woman at 37 weeks EGA presents with painful uterine contractions, reassuring fetal status, no signs or symptoms of ROM,</p>	<p>Braxton Hicks contractions (prodromal or false labor)</p>

and an unchanged SVE after 3 hours. The cervix remains long, closed, and high. Irregular contractions occur at 15–20 minute intervals, what is your diagnosis?

How should the above woman be managed?

She should be **hydrated**, scheduled for **close clinical follow-up**, and advised to **go home** with labor precautions

In the presence of a non-reassuring fetal heart rate what initial interventions should be tried?

Position the mother in left lateral decubitus position with flexed knees
Supplemental oxygen with a face mask
Refrain from active pushing
Turn off the oxytocin and consider tocolytics

What is tocolysis?

Relaxation of the uterine smooth muscle in the context of persistent fetal distress

What substances are commonly used for tocolysis?

Terbutaline, magnesium sulfate, nifedipine, indomethacin

What are the risks of applying traction on the cord during the third stage of labor?

Avulsion of the cord or placenta and uterine inversion

How can this risk be reduced?

By **applying suprapubic pressure** when applying gentle traction to the cord (fundal pressure should not be applied). The placenta and uterus should be examined, to ensure that no membranes or accessory lobes are retained

After delivery of the placenta what is given to prevent postpartum hemorrhage?

20 units (2 mL) of **oxytocin** in 1 L of lactate ringers

What are the four classifications of vaginal and perineal lacerations?

First degree limited to vaginal mucosa and skin of the introitus

Second degree extends to the fascia and muscles of the perineal body

Third degree trauma involves the anal sphincter

Fourth degree extends into the rectal lumen, through the rectal mucosa

What are the indications for an episiotomy?

To expedite delivery in the setting of non-reassuring fetal heart tracing or maternal exhaustion

	Shoulder dystocia (to facilitate operator maneuvers)
	Breech delivery
	Operative vaginal delivery (forceps or vacuum)
	OP positions
Why is an episiotomy only performed with clear indication?	Third and fourth degree lacerations and anal incontinence of stool or flatus are more common with an episiotomy than with a spontaneous laceration
What are advantages of midline episiotomies when compared with mediolateral episiotomies?	They are associated with fewer infections, faster healing, less pain, less blood loss, less dyspareunia, and better anatomical results
What is the one advantage of the mediolateral episiotomy?	Decreased risk of extension to a third or fourth degree laceration
What muscles are affected by second degree lacerations?	Bulbocavernosus and ischiocavernosus laterally Superficial transverse perineal muscle

OPERATIVE VAGINAL DELIVERY

What are the two major types of operative vaginal delivery?	Forceps and vacuum
What is required for operative vaginal delivery?	Fully dilated cervix Ruptured membranes Engaged fetal head (at or below zero station) Known absence of CPD Known position of the fetal head Experienced operator The capability to perform an emergency cesarean delivery if necessary
What are the various types of forceps deliveries?	Outlet: fetal scalp is visible at the introitus and rotation does not exceed 45 degrees Low: +2 station and may require more than 45 degrees of rotation to AP orientation

<p>Operative vaginal delivery is appropriate or indicated in what context?</p>	<p>Mid-pelvic: head is engaged at or below 0 station (but above +2 station), rotation is often required to mimic the cardinal movements of labor</p>
<p>What are the most common maternal morbidities involved with forceps delivery?</p>	<p>High: above 0 station</p> <p>Maternal or fetal distress (e.g., infection, heart, or lung disease, exhaustion, prolonged second stage)</p>
<p>Vacuum delivery confers what advantages over forceps?</p>	<p>Increased perineal trauma (third and fourth degree extensions)</p> <p>Increased need for blood transfusion (these risks are increased with more rotation or at a higher station)</p>
<p>What are some contraindications to vacuum-assisted delivery?</p>	<p>Can be achieved with minimal analgesia</p> <p>Low birth weight fetus (estimated fetal weight <2500 g)</p> <p>Prematurity <34 weeks</p> <p>Suspected fetal coagulopathy</p> <p>Recent scalp blood sampling</p> <p>Face or breech presentation</p> <p>An inability for the mother to engage in expulsive efforts</p> <p>Cessation of contractions (vacuum traction must be coordinated with maternal effort)</p>
<p>What are the most common fetal morbidities involved with forceps and vacuum delivery?</p>	<p>Scalp lacerations, bruising, subgaleal hematomas, cephalohematomas, intracranial hemorrhage, neonatal jaundice, subconjunctival hemorrhage, clavicular fracture, shoulder dystocia, facial nerve injury, Erb palsy, retinal hemorrhage, and fetal death</p>
<p>Which operative vaginal delivery is associated with more trauma to the fetus?</p>	<p>Vacuum delivery—there is an increased risk of cephalohematoma and retinal hemorrhage when compared with forceps delivery</p>
<p>Which operative vaginal delivery method is associated with more maternal trauma to the perineum?</p>	<p>Forceps (except when electively applied to facilitate outlet delivery of the fetus)</p>

CESAREAN DELIVERY

What are some indications for scheduled cesarean delivery?

Previous classical uterine incision
 Prior uterine surgery that involved the myometrium
 EFW >4500 g
 Severe fetal hydrocephalus
 Malpresentation (breech)
 Cervical cancer
 Active genital herpes
 Placenta previa

What percentage of deliveries in the United States are emergent or scheduled cesarean deliveries?

Over 20%

What is the risk of a trial of labor in a woman with a prior classical uterine incision?

A 12% risk of **uterine rupture**, which is associated with fetal death, maternal shock, and a 10% maternal mortality rate

What is the risk of uterine rupture in a laboring patient with a prior lower uterine segment incision?

<1%

At what EGA is a cesarean section scheduled?

After 39 weeks in a patient with accurate dating of her pregnancy. Fetal lung maturity tests should be performed if delivery is planned earlier or if the dating method is not adequate

What is the lower uterine segment? What are the advantages of using this in a C-section?

The region of the uterus just superior to the cervix. The myometrium is **significantly thinner** than the uterine fundus (especially during labor) and is associated with a **lower risk of uterine rupture**

What are the layers (from exterior to interior) that are incised with a Pfannenstiel or low transverse abdominal incision?

Skin → subcutaneous fat → superficial fascia (Camper) → deep fascia (Scarpa) → anterior rectus sheath → rectus abdominus muscle → preperitoneal fat → parietal peritoneum (with bladder flap) → visceral peritoneum → uterus

What percentage of VBACs (vaginal births after cesarean) are successful?

60–80% are successful (depending on the indication for prior cesarean)

What term describes a pathologically thin lower uterine segment through which fetal membranes or fetal parts can be visualized prior to uterine incision?

What risk may be associated with a uterine window?

What are some indications for a vertical lower uterine incision?

Can the type of previous uterine incision be determined from the skin incision?

A **uterine window**

Imminent risk of uterine rupture and expulsion of the fetus into the peritoneum

Premature breech fetus

Poorly developed lower uterine segment

Extensive fibrosis of lower uterine segment (following multiple cesareans)

No

Postpartum Care

PUERPERIUM MANAGEMENT

What is the duration of the postpartum period, also known as the puerperium?

What is uterine involution?

What are common uterotonics and their contraindications?

When is a pregnant woman at highest risk of developing venous thrombosis?

6–8 weeks following delivery, when maternal physiology returns to the pre-pregnancy state

The contraction of interlacing myometrium bundles, with subsequent atrophy, so as to constrict vessels and prevent hemorrhage, as well as gradually returning the uterus to its prepregnancy size

Oxytocin (Pitocin): pulmonary edema

Carboprost (Hemabate): asthma

Methylergonovine (Methergine): preeclampsia, pregnancy-induced HTN or HTN

Misoprostol (Cytotec)

During the immediate puerperium period because of vessel trauma,

<p>Stool softeners, intermittent strait catheterization (to relieve urinary retention), and ice packs are commonly employed to treat which complications of vaginal delivery?</p>	<p>immobility, increased fibrinogen, factor VII, VIII, IX, X, and platelets. Approximately 0.7 per 1000 women experience a venous thrombosis during pregnancy or postpartum</p>
<p>Which nerve can be injured during delivery, with subsequent urinary retention?</p>	<p>Peri-urethral, third or fourth degree lacerations, which are also associated with dyspareunia and bowel incontinence. Additionally, ice packs and nonsteroidal anti-inflammatory drugs (NSAIDs) decrease swelling and induration from the inflammatory reaction to suture used to repair lacerations</p>
<p>Following a 300-minute second stage of labor, a G1P1 is unable to flex her hips against tension and has difficulty walking. What type of neuropathy is likely responsible?</p>	<p>The pudendal nerve; the neuropathy normally resolves within 2 months</p> <p>Femoral nerve compression, secondary to hyperflexion of the hips during a prolonged second stage</p>
<p>Which episiotomy is associated with perineal or pelvic hematoma?</p>	<p>Mediolateral episiotomies</p>
<p>How soon should an Rh(D–) mother be given Rhogam (anti-D immune globulin), following delivery of an Rh+ fetus?</p>	<p>Within 72 hours of delivery</p>
<p>Where is the fundus during the postpartum period?</p>	<p>Immediately after delivery: near the umbilicus</p> <p>After 24 hours below the umbilicus</p> <p>1 week near the symphysis pubis</p> <p>2 weeks in the pelvis</p> <p>6–8 weeks it has assumed its nonpregnant size</p>
<p>What is lochia?</p>	<p>A combination of blood, serous exudate, erythrocytes, leukocytes, necrotic decidua, epithelial cells, and bacteria</p>
<p>What is the progression of the various types of lochia?</p>	<p>Lochia rubra (red), is followed by lochia serosa (brown), and ultimately lochia alba (yellow white). Lochia rubra consists of primarily blood from the placental bed, mixed with tissue, lasting 3–5 days. Lochia serosa consists of smaller amounts of older blood mixed with serous</p>

	drainage, leukocytes, and cervical mucus, ending by the 10th day after delivery. Lochia alba lasts from the second to sixth week after delivery and has a small amount of blood, combined with leukocytes, epithelial cells, cholesterol, fat, and mucus
How long does a woman shed lochia?	4–6 weeks however, it should not be red for more than 2 weeks
What is suggested if a woman continues to have lochia rubra for over 2 weeks?	Retained products of conception (POC) must be considered and her uterus, cervix, and vagina should be examined with possible dilation and curettage
How much lochia is shed during the puerperium?	Initially it may be as heavy as a period and should subside; the total amount lost is approximately 500 mL
What is the management of wound dehiscence?	Dehiscence of a vaginal laceration repair should be evaluated for infection, irrigated, and debrided of necrotic tissue. Sitz baths should be used liberally. If discovered in the first 2–3 days after delivery, the wound can be resutured; however, if the tissue is friable or has evidence of infection, a secondary repair should be delayed for 6–8 weeks. Antibiotics should be utilized if infection is noted
What are some signs and symptoms of endometritis?	Elevated temperature ($>100.4^{\circ}\text{F}$ or $>38^{\circ}\text{C}$) in the presence of uterine tenderness. Additionally, purulent vaginal discharge may be noted, as well as leukocytosis. It is usually noted within the first 3–5 days after delivery. It occurs in 4% of all vaginal deliveries and up to 10% of cesarean deliveries
Who is at increased risk for endometritis?	Risk factors include: prolonged ROM, prolonged labor, multiple internal examinations, internal monitoring (FSE or IUPC), retained POC, lower socioeconomic status, poor nutrition, maternal anemia, and concurrent genital tract infection
What is the treatment for endometritis?	It is a polymicrobial infection, with a mixture of aerobic and anaerobic bacteria found. Treatment usually

What are some common causes of puerperal fever (postpartum or post-cesarean-section fever)?

consists of gentamicin and clindamycin until the patient is afebrile for 24–36 hours

Wind (atelectasis or aspiration PNA)

Water (cystitis or UTI)

Wound (surgical site infection or laceration)

Walking (PE or a DVT)

Wonder drugs (medication SE or adverse reaction)

Womb (endometritis)

Wet nurse (engorgement or mastitis: infection often with *S. aureus* or *Streptococcus*)

Phlebitis or septic pelvic thrombophlebitis

BREASTFEEDING AND INFANT CARE

What are the five components of the Apgar score at 1, 5, and 10 minutes?

Appearance (blue, acrocyanosis, pink)

Pulse (absent, <100, >100)

Grimace (none, present, vigorous irritability)

Activity (flaccid, flexed, moving)

Respirations (absent, slow, crying)

Each component is given 0, 1, or 2 points

Silver nitrate, erythromycin, or tetracycline ointments are applied to the newborn's eyes to prevent what ocular infection?

Chlamydia and gonorrhea

Why is it required to emergently assess an infant with ambiguous genitalia?

Congenital adrenal hyperplasia is a life-threatening condition requiring mineralocorticoid supplementation to prevent salt wasting (hypovolemic-hyponatremia). The most common form is 21-alpha-hydroxylase deficiency. Electrolytes, 17-alpha hydroxyprogesterone, and dehydroepiandrosterone sulfate should be checked

How does an infant benefit from skin-to-skin contact at birth?	Better temperature and glucose control, as well as an increased likelihood of maternal breastfeeding
Which vitamin is absent from human breast milk?	Vitamin K. Infants are given a vitamin K shot at birth to prevent hemorrhagic disease of the newborn
What is the effect of decreased dopamine production in the hypothalamus following nipple stimulation?	Decreased prolactin-inhibiting factor, thereby increasing prolactin from the anterior pituitary
How does prolactin impact breastfeeding?	Stimulates milk production by the terminal exocrine glands
Nipple stimulation also increases oxytocin release from the posterior pituitary, which impacts breastfeeding. How?	Causing contraction of the myo-epithelial cells of the lactiferous ducts, allowing milk letdown
How does breastfeeding prevent ovulation?	Prolactin inhibits the pulsatile gonadotropin-releasing hormone from the hypothalamus
What is colostrum?	Thick yellow breast secretions that contain plasma exudates, immunoglobulins (IgA), lactoferrin, albumin, and electrolytes. It is secreted during the first 2 days postpartum
Why is an ELISA or Western blot repeated at 6 months and 1 year following delivery of an infant to an HIV+ mother?	Maternal transplacental IgG persists for several months in the infant's serum. The above tests check for antibodies to HIV, not the actual virus
When does a breastfeeding woman's milk "come in"?	3–5 days postpartum. It contains protein, lactose, water, fats, and immunoglobins (IgA)
Are mothers who are seropositive for the following conditions advised to breastfeed?	<p>Hepatitis A: Yes</p> <p>Hepatitis B: Yes, with vaccination and hepatitis B IgG administration to the infant at birth. The virus is present in breast milk, although the benefits outweigh the risk of transmission, which is minimized with treatment</p> <p>Hepatitis C: Yes</p> <p>HIV: No</p> <p>Cytomegalovirus (CMV): No</p>

Are mothers who are exposed to the following substances advised to breastfeed?

Alcohol	No
Heroin and other analgesics	No
Tobacco	Yes
Hepatitis B vaccination	Yes
Rubella vaccination	Yes
Rubeola vaccination	Yes
Ibuprofen	Yes
Tetracyclines	No
Sulfa drugs	No, because they displace bilirubin and increase risk of kernicterus
Quinolones	No
Chemotherapy (antimitotic medications)	No
Radiation	No
Lithium or heavy metals	No

Is previous mammoplasty (reduction or implantation) a contraindication to breastfeeding? No. If the integrity of the nipple ducts are preserved, a woman can breastfeed; however, the surgical technique and subsequent scarring may make this difficult

What is the caloric demand of lactation? 640 kcal/day

How much should breastfeeding women increase their daily caloric intake? 300–500 kcal, so as to ensure that loss of gestational weight

How much calcium should a breastfeeding woman consume? 1200 mg/day of calcium

A mother does not want to breastfeed although her breasts are extremely tender 5 days postpartum. What is appropriate management? Tight brassiere, avoidance of nipple stimulation, cool compresses, acetaminophen or ibuprofen will offer comfort and suppress lactation. She is experiencing ductal, venous, and lymphatic engorgement

PHYSIOLOGIC CHANGES AND RESOLUTION

How is cardiac output (CO) affected during the first day postpartum?

A 60–80% increase in CO occurs with the autotransfusion of uteroplacental blood to the intravascular space and decompression of the vena cava, during the fourth stage of labor

A leukocytosis <25,000 and low-grade fevers <101 are considered normal or abnormal 24 hours postpartum?

Normal

Over what time period does the CO and systemic vascular resistance gradually return to nonpregnant levels?

3–4 months, with concurrent reduction in left ventricular size and contractility

How does a pre-partum cervix (cx) differ from a parous cx?

The pre-partum cervix is composed of fibrous connective tissue without muscle, and the cervical os can be described as a “pinpoint.” Over several weeks following delivery, the cx slowly contracts, with the cervical os appearing as a transverse, stellate slit, which can be dilated with greater ease

Why is a woman who has delivered vaginally at risk for pelvic relaxation, cytocele, rectocele, or incontinence?

The musculature and female genitalia slowly contract; however, connective tissue and fascial stretching may not return to the pre-gravid state, resulting in persistent trauma or changes

What is a persistent defect of the abdominal wall musculature caused by the gravid uterus known as?

Diastasis recti

How much weight is lost following delivery?

Almost half of the gestational weight gain is lost following delivery (13 lbs), with the additional weight loss occurring over the next 6 months (15 lbs)

Do women typically gain weight following pregnancy?

Most women maintain 10% of their gestational weight following the postpartum period

Why do some women note increased alopecia postpartum?

Scalp hair shifts from the predominant anagen phase (growing) during pregnancy to a predominant telogen phase (resting). This **telogen effluvium** typically resolves within 5 months

What is the condition in which a woman displays hypopituitarism following a delivery with postpartum hemorrhage?

Sheehan’s syndrome, because of infarction and necrosis of the pituitary

What are some symptoms of central diabetes insipidus?

Polydipsia, polyuria, hypernatremia (>140 mEq/L), normal serum osmolality (<280 mOsm/kg), and dilute urine osmolality (<380 mOsm/kg)

When does serum human chorionic gonadotropin (HCG) return to normal (nondetectable levels)?	Within 4–6 weeks of delivery or abortion
What is suggested by a rising HCG postpartum?	Gestational trophoblastic disease
Why do some women note vaginal atrophy in the puerperium?	Prolactin inhibition of systemic estrogens
How is the thyroid affected postpartum?	Hormone levels are within normal limits by 4 weeks and the thyroid gland decreases to pre-pregnancy size over 3 months

POSTPARTUM CLINICAL CARE

What is postpartum thyroiditis?	Acute hyperthyroidism and subsequent hypothyroidism, each lasting approximately 1 month in duration. Women typically recover spontaneously
How many women with gestational diabetes mellitus (GDM) develop Type II diabetes later in life?	Up to 50%. A woman with GDM should be screened for diabetes postpartum
How common are postpartum blues?	50–70% of women
When do they occur?	Within 2 weeks of delivery and typically resolve within 2 weeks; they are attributed to dramatic shift in hormones and decreased progesterone
How common is postpartum depression?	4–10%
When does a woman present with postpartum depression?	A majority of cases occur between 2 weeks and 4 months postpartum. Depression may last 3–14 months
How is postpartum depression diagnosed?	The criteria is similar to major depressive disorder; five of the following symptoms must be present for 2 weeks: SIGECAPS Sleep changes Loss of Interest (anhedonia) Guilt Decreased Energy Decreased ability to Concentrate

	Appetite increase or decrease
	Psychomotor increase or decrease
	Suicidal ideation
Who is at increased risk for postpartum depression?	An older, primiparous woman, without social support, with an unplanned pregnancy, a history of psychiatric hospitalizations, suicide attempts, depression or other mood disorders, and a family history of suicide
How common is postpartum psychosis?	0.1–0.2%, it can last up to 2 months

FERTILITY AND CONTRACEPTION

When does ovulation and menstruation begin in a postpartum nonlactating woman?	Ovulation typically resumes within 45 days, but it may occur within 4 weeks. Average duration to menstruation is 8 weeks postpartum
When does ovulation and menstruation begin in a postpartum breastfeeding woman?	A woman who is breastfeeding more than 5 times a day can remain anovulatory for the duration of breastfeeding. Amenorrhea, feeding schedules > 8 times daily, maximum of 6 hours between feeds, and minimal supplementation are key predictors of anovulation
How long should the woman maintain “pelvic rest,” by refraining from coitus or inserting anything in the vagina?	6 weeks postpartum, so as to decrease the risk of an infection ascending through the patent cervical os, and to reduce the risk of infection or trauma to healing lacerations
What type of birth control will not affect breastfeeding?	IUD, barrier methods, progestin only pills (mini pill), Depo-Provera
Why do oral contraceptive pills (OCPs) and Norplant affect breastfeeding?	Estrogen decreases breast milk production. It can be safely started 6 weeks after delivery if milk production is adequate
Why do some clinicians recommend that women obtain an IUD 6 weeks postpartum instead of insertion at the time of delivery?	Decreased risk of expulsion because of patent os and uterine cramping

Complications of Pregnancy

Early Complications

CONGENITAL ANOMALIES

What is the prevalence of major congenital anomalies in the United States?

Between 2 and 4%

What are the causes of congenital anomalies and what are their relative frequencies?

Single gene disorders (15–20%)

Chromosomal abnormalities (5%)

Teratogens (either maternal illness, infection, drugs, or chemicals; 10%)

Unknown (60–70%)

What is the overall incidence of chromosomal anomalies in the United States?

0.7% of all live births; however, it is more common among abortuses and stillbirths (up to 50%)

What happens to most fetuses with chromosomal abnormalities?

Most do not survive to term. If they do, they are often with congenital abnormalities (with multiple organ system involvement), growth deficiency, and mental retardation

Do the same cytogenetic abnormalities produce the same phenotype in each fetus?

No. While it will produce the same pattern of malformations, there is significant variability

What are the genetic etiologies of chromosomal abnormalities?

Nondisjunction

Unequal recombination

Inversion

<p>What is the most common etiology of chromosomal abnormality?</p>	<p>Deletions/duplications Translocations</p>
<p>What is the major risk factor for nondisjunction?</p>	<p>Nondisjunction—the loss or gain of a chromosome resulting in trisomy or monosomy Advanced maternal age</p>
<p>Describe the syndrome associated with trisomy 21</p>	<p>Known as Down syndrome, it is the most common chromosomal abnormality found in live births. It is associated with mental retardation, hypotonia, a single palmar crease, early-onset Alzheimer, and other serious congenital features such as duodenal atresia, congenital heart disease, and leukemia</p>
<p>What is the cytogenetic etiology of trisomy 21 and what are the relative frequencies?</p>	<p>True trisomy (94%) Robertsonian translocation (3–4%) Trisomy mosaicism (2–3%)</p>
<p>What are the risk factors for trisomy 21?</p>	<p>Advanced maternal age</p>
<p>Describe the syndrome associated with trisomy 18</p>	<p>Major features include: Severe mental retardation Hypertonia Prominent occiput Micrognathia Short sternum Flexed fingers Congenital heart disease Ectopic pancreatic tissue Small pelvis Horseshoe kidney Meckel diverticulum or malrotation Half of trisomy 18 infants die within 1 week of life and the vast majority die within 1 year</p>
<p>Describe the syndrome associated with trisomy 13</p>	<p>It primarily includes malformations of the midface, eyes, and brain. However, omphalocele, genitourinary (GU) anomalies, hemangiomas,</p>

polydactyly, rocker-bottom feet, and congenital heart defects are also often found

80% die within the first month and the vast majority die within the first 6 months

What is genomic imprinting?

Differential expression of genetic information, depending on whether it is from the mother or father. It is the basis of diseases such as Prader-Willi syndrome and Angelman syndrome, which involve the same microdeletion on chromosome 15 but result in a different phenotype depending on which parent the deletion came from

Describe the following sex chromosome abnormalities

Turner syndrome (45, X) characterized by short stature, streak gonads, mild MR, and other abnormalities such as webbed neck, lymphedema, pigmented nevi, and congenital heart defects

Klinefelter syndrome (47, XXY) characterized by tall stature, microorchidism, and azospermia

What is a teratogen?

Any agent that can lead to abnormalities in a developing fetus. They can be from maternal illness, microbial infections, drugs, or other chemicals in the environment

What types of birth defects are associated with the following maternal illnesses?

Insulin-dependent diabetes mellitus (IDDM): congenital heart disease, spina bifida, caudal regression, focal femoral hypoplasia

Phenylketonuria: microcephaly, mental retardation, congenital heart disease

Adrenal/ovarian tumors: virilization of female fetuses if they secrete androgens

Autoimmune disease: a similar disease in fetus as in the mother if

What are some of the sonographic signs that suggest fetal infection?

the antibodies cross the placenta, congenital heart block

Obesity: neural tube defects

Microcephaly: cardiac malformations

Cerebral calcifications:

hepatosplenomegaly

Hepatic calcifications: limb hypoplasia

Intrauterine growth restriction: hydrocephalus

SPONTANEOUS ABORTIONS

What is a spontaneous abortion?

Also called a **miscarriage**, it is a pregnancy that spontaneously ends **prior to 20 weeks of gestation or before the fetus has reached 500 g** resulting in expulsion of all or any part of the products of conception

How common is spontaneous abortion?

It occurs in 8–20% of known pregnancies under 20 weeks and in an even higher percentage of subclinical pregnancies

When do most spontaneous abortions occur?

80% occur prior to 12 weeks

Chromosomal abnormalities (aneuploidy) are the most common cause of spontaneous abortion in the first trimester. What are the two most common genetic abnormalities that cause this?

Autosomal trisomies

Monosomy X

Are there any therapeutic interventions that prevent first trimester pregnancy loss?

No, nothing has been proven in randomized controlled trials to prevent this

What are some causes of second trimester spontaneous abortion?

Maternal factors such as cervical insufficiency, infection, chromosomal or structural malformation, or maternal thrombophilia

What increases the risk of spontaneous abortion?

Advanced maternal age
Prior spontaneous abortion

What are the three most common symptoms of a spontaneous abortion?

Describe each of the following types of spontaneous abortions:

What percentage of women exhibit signs of threatened abortion during their pregnancy? What percentage of these women will spontaneously abort?

What must be considered in the differential diagnosis of a threatened abortion?

What is the first diagnostic test you should perform in a woman with a known intrauterine pregnancy who presents with vaginal bleeding?

Heavy cigarette use

Short interpregnancy interval

Maternal medical disease (e.g., celiac dz, IDDM)

Heavy alcohol or caffeine intake

Trauma

Increased parity

Advanced paternal age

History of amenorrhea, vaginal bleeding, pelvic pain

Complete abortion: uterine bleeding with complete expulsion of the product of conception (POC) and closed cervical os

Inevitable abortion: uterine bleeding with cervical dilation but without expulsion of any POC

Incomplete abortion: uterine bleeding with cervical dilation but with incomplete expulsion of the POC

Missed abortion: embryonic demise without expulsion of POC and with closed cervical os

Septic abortion: embryonic demise with evidence of infected products of conception (e.g., fever, uterine tenderness)

Threatened abortion: uterine bleeding with closed cervical os and normal cardiac activity

Approximately 25% and 50%, respectively

Ectopic pregnancy

Cervical, vaginal, or uterine pathology

Bleeding related to implantation

Ultrasound

How is vaginal bleeding in the first trimester evaluated?

Via history, physical examination, hCG levels, and ultrasound. The following algorithm (Fig. 9-1) can be used:

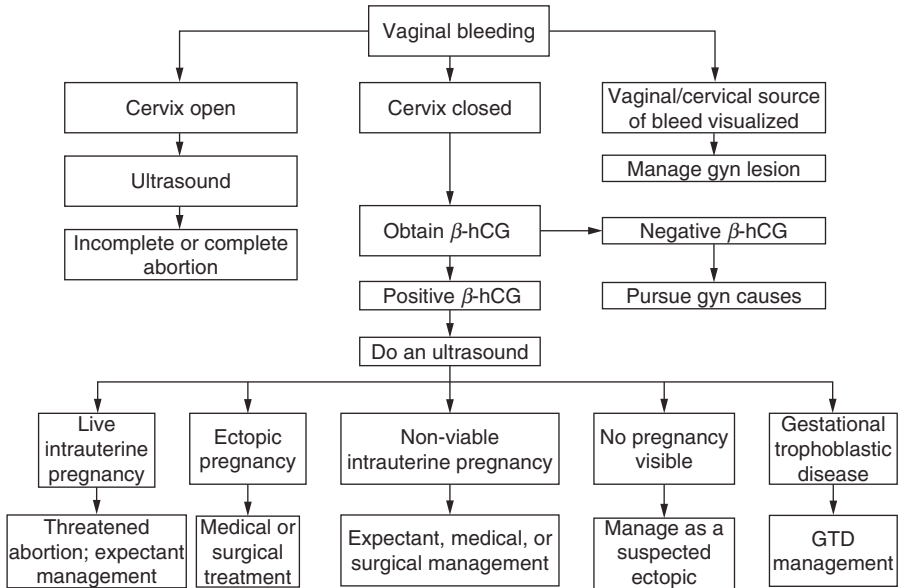


Figure 9-1 Evaluation of vaginal bleeding in the first trimester. (Adapted, with permission, from uptodateonline.com *Clinical manifestations, diagnosis, and management of ectopic pregnancy* by Togas Tulandi).

What are the ultrasonographic criteria for diagnosis of a nonviable intrauterine pregnancy?

Absence of fetal cardiac activity in an embryo with a crown-rump length >5 mm

-or-

Absence of a fetal pole when the mean sac diameter >25 mm (transabdominally) or >18 mm (transvaginally)

What are the treatment options for an incomplete, inevitable, or a missed abortion? What are the benefits of each?

Surgical, medical, or expectant management

What are the advantages and disadvantages surgical, medical, or expectant management?

See Table 9-1

Table 9-1 Treatment Options for Abortion

	Advantages	Disadvantages
Surgical Management	Greatest efficacy; quickest evacuation	Invasive; procedural/anesthesia risks
Medical Management	Less invasive; avoids anesthesia; patient can do it in the privacy of her own home	Requires more than two visits; less quick
Expectant Management	Least invasive	Timing is unpredictable; risk of needing an unplanned procedure

Why is it imperative that spontaneous abortions be evacuated?

Because after several weeks-months, the trophoblastic tissue might enter the maternal bloodstream, triggering the coagulation cascade and causing a **coagulopathy**

What lab tests must be done after a spontaneous abortion?

Type and screen (because Rh immunoglobulin must be given to Rh[-] women)

Can a woman do anything to prevent a spontaneous abortion?

Typically no, and so all women need to be reassured that it is not their fault

Should a woman who has had a spontaneous abortion undergo an evaluation for the cause?

No. These are fairly common and are often sporadic

What is recurrent pregnancy loss (RPL)?

Three or more consecutive losses of pregnancy prior to 20 weeks

What is the incidence of RPL?

Approximately 0.5–1%

What is the risk of spontaneous abortion?

After one prior loss: 10–20%

After two consecutive losses: 25–45%

What does the diagnostic workup of RPL consist of?

A complete medical, surgical, genetic, and family history

A physical examination with attention to signs of **endocrinopathies** or **pelvic organ anomalies**

Laboratory evaluation including: **uterine assessment** (sonohysterography)

Anticardiolipin antibodies and **lupus anticoagulant**

Evaluation of ovarian reserve

Possibly other tests (e.g., an evaluation for inherited thrombophilias, thyroid function tests [TFTs], and a karyotype)

What is the treatment for RPL?

It is dependent on the etiology; however, it can include:

Surgery (for pelvic organ abnormalities)

Coumadin and aspirin (for antiphospholipid syndrome)

Synthroid (for hypothyroidism)

ECTOPIC PREGNANCY

What is an ectopic pregnancy (see Fig. 9-2)?

Implantation of the pregnancy into a site outside of the endometrial lining of the uterine cavity

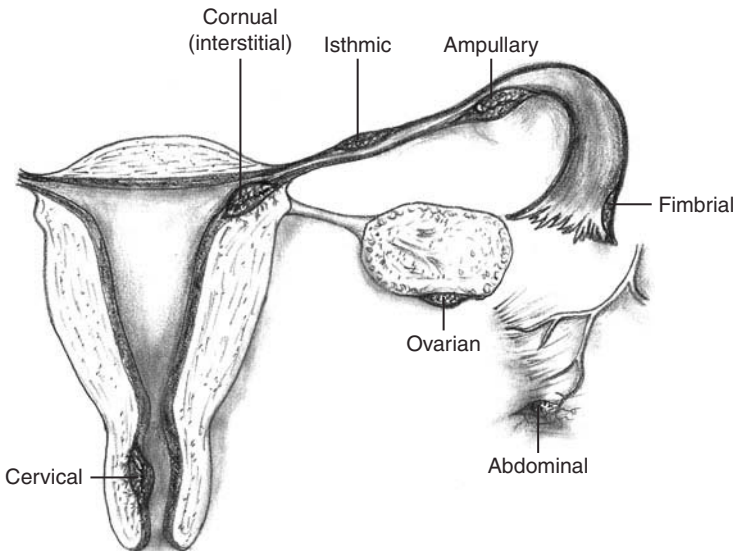


Figure 9-2 Locations of ectopic pregnancies.

What is the most common cause of pregnancy-related death in the first trimester?

Rupture of an ectopic pregnancy

What is the incidence of ectopic pregnancy?

Approximately 2% of pregnancies in the United States are ectopic

What is the mortality rate associated with an ectopic pregnancy?

Approximately 3–4 deaths per 10,000 cases result in death

What are the major risk factors for ectopic pregnancy?

History of pelvic inflammatory disease (PID), (most common)

History of ectopic pregnancy

History of tubal surgery or pathology

Diethylstilbestrol (DES) exposure in utero

Current intrauterine device (IUD) use

Current oral contraceptive pill (OCP) use

Infertility

History of cervicitis

Multiple sexual partners

Cigarette smoking

Describe the relationship between IUD use and ectopic pregnancy

Women using an IUD are much less likely to conceive compared to women not using any form of birth control. However, in the unlikely event that they do conceive, there is a greater probability that it will be ectopic

What are the major symptoms of ectopic pregnancy?

Classic symptoms: **amenorrhea, abdominal/pelvic pain, vaginal bleeding**

Other symptoms: dizziness, nausea, vomiting, diarrhea

40% of cases present acutely and 60% of cases present as chronic symptoms. Many women are asymptomatic until rupture

What are the signs on physical examination of ectopic pregnancy?

Many women have a normal physical examination; however, the common signs are:

Adenexal mass and/or tenderness

Mild uterine enlargement

Cervical motion tenderness

Abdominal tenderness

Orthostatic hypotension, tachycardia, and rebound tenderness are all signs of rupture

What is the differential diagnosis of these symptoms?

Threatened abortion

Torsion

Ruptured corpus luteum cyst

Abnormal uterine bleeding

	<p>Tubo-ovarian abscess (TOA)</p> <p>Molar pregnancy</p> <p>PID</p> <p>UTI or stones</p> <p>Pyelonephritis</p> <p>Diverticulitis</p> <p>Appendicitis</p> <p>Pancreatitis</p>
<p>What diagnostic tests can be used to distinguish between these conditions?</p>	<p>β-hCG</p> <p>Transvaginal ultrasound</p>
<p>What is the difference in β-hCG levels between an intrauterine and ectopic pregnancy?</p>	<p>The rate of β-hCG rise is lower in most cases of ectopic pregnancy</p>
<p>Where are ectopic pregnancies located and what are the relative frequencies of each (see Fig. 9-2)?</p>	<p>Fallopian tube (95%; ampulla > isthmus > fimbria)</p> <p>Ovarian (3.2%)</p> <p>Interstitial or cornual (2.4%)</p> <p>Abdominal (1.3%)</p> <p>Cervix (rare)</p> <p>Hysterotomy scar</p>
<p>What are the causes of tubal implantation?</p>	<p>Conditions that delay transport of the egg through the tube</p> <p>Conditions in the embryo that lead to premature implantation</p>
<p>What types of conditions cause a delay in transport of the egg through the tube?</p>	<p>Chronic salpingitis</p> <p>Salpingitis isthmica nodosa (SIN)</p>
<p>What happens to the endometrium during an ectopic pregnancy?</p>	<p>It still responds to pregnancy hormones and so often exhibits signs of decidual reaction or endometrial thickening</p>
<p>What is a heterotopic ectopic pregnancy?</p>	<p>A concurrent intrauterine and extrauterine pregnancy. While rare, it is more common in women pregnant through in vitro fertilization (IVF)</p>
<p>What is the natural course of an ectopic pregnancy?</p>	<p>Rupture, spontaneous regression, or tubal abortion</p>
<p>What is a tubal abortion?</p>	<p>The expulsion of the POC through the fimbria into the abdominal cavity. The POC can then either regress or reimplant in the abdominal cavity or in the ovary</p>

At what gestational age do the clinical manifestations of an ectopic pregnancy begin?

At least 6–8 weeks after the LMP

Describe the algorithm for management of a suspected ectopic pregnancy

See Fig. 9-3

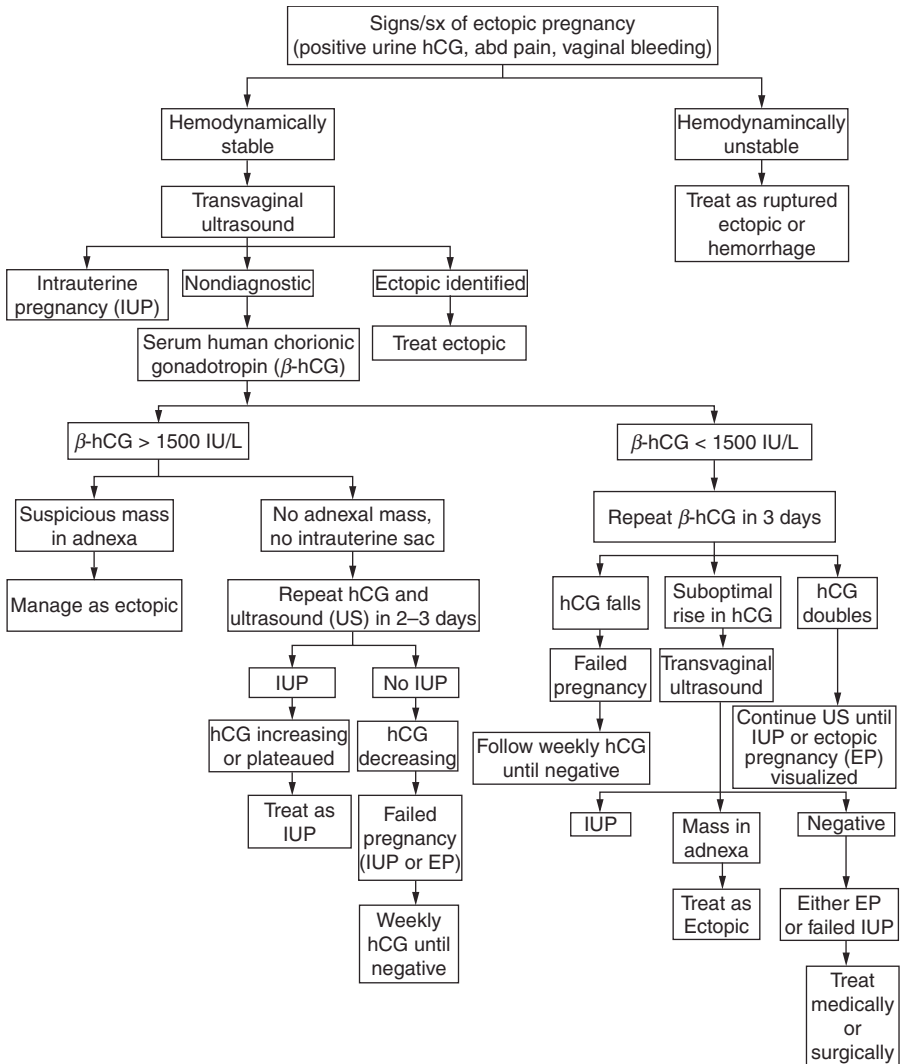


Figure 9-3 Management of suspected ectopic pregnancy. (Adapted, with permission, from uptodateonline.com *Spontaneous abortion: Risk factors, etiology, clinical manifestations, and diagnostic evaluation* by Togas Tulandi and Haya M Al-Fozan)

What are the treatment options for an ectopic pregnancy?

Methotrexate (for early, unruptured, ectopic pregnancies)

What are the contraindications of methotrexate used to treat an ectopic pregnancy?

Surgery (laparoscopy with salpingostomy or salpingectomy)

Active hemorrhage

Pregnancy larger than 4 cm

Breastfeeding

Alcoholism

Peptic ulcer disease

Liver or renal disease

Blood dyscrasias

Immunodeficiency

Active pulmonary disease

What is methotrexate's failure rate?

5–10%, but higher in more advanced pregnancies

What are the two methods of methotrexate administration for the treatment of an ectopic pregnancy?

Give a single intramuscular (IM) dose of methotrexate and then follow β -hCG levels at days 4 and 7. hCG levels should decline by 15% between days 4 and 7.

Alternate day IM administration of methotrexate until β -hCG level decreases by 15% in 48 hours

What are the differences between a salpingostomy and a salpingectomy?

Salpingostomy: an incision is made on the antimesenteric part of the fallopian tube and the POCs are evacuated. The incision is closed by secondary intention

Salpingectomy: a tubal resection that involves partial removal of the oviduct, salvaging as much as possible

What is the discriminatory zone?

A range of β -hCG levels (1000–2000 IU/L) in which a **gestational sac should be seen** if there is an intrauterine pregnancy. If no sac is seen at β -hCG levels above the discriminatory zone, an ectopic pregnancy can be diagnosed in most cases

How should a patient with a low β -hCG without a visible intrauterine pregnancy be managed?

Both the ultrasound and the β -hCG should be repeated in 3 days. If the pregnancy is intrauterine and viable,

Does a negative pelvic ultrasound rule out the diagnosis of an ectopic pregnancy?	the β -hCG should double in 1.5–2 days
What is the difference in progesterone levels between an intrauterine and an ectopic pregnancy?	No, an extrauterine pregnancy is visualized in only 50% of ectopic pregnancies
What percentage of patients with an ectopic pregnancy experience tubal rupture?	Serum progesterone levels are lower in ectopic pregnancies compared with viable intrauterine pregnancies. However, the sensitivity and specificity of progesterone levels are too low to make it a screening or diagnostic test for ectopic pregnancy
In a patient with an ectopic pregnancy, what are the risk factors for tubal rupture?	Approximately 18%
How does the presence of an ectopic pregnancy affect subsequent pregnancies?	History of tubal damage/infertility Induction of ovulation Never having used contraception High β -hCG

GESTATIONAL TROPHOBLASTIC DISEASE

Introduction

What is gestational trophoblastic disease (GTD)?	A group of tumors that arise from placental tissue and secrete β-hCG
What are the types of GTD?	Hydatidiform mole Persistent/invasive gestational trophoblastic neoplasia (GTN) Choriocarcinoma Placental site trophoblastic tumor (PSTT)
Which of the types of GTD are benign and which are malignant?	Hydatidiform moles are usually benign, whereas invasive GTN, choriocarcinoma, and PSTT are all malignant

What are the major risk factors for GTD?	Extremes of maternal age History of prior GTD
When should GTD be suspected clinically?	If there is unusual bleeding after a pregnancy or abortion
What are the signs and symptoms of GTD?	First trimester bleeding Uterine size/date discrepancy Pelvic pressure or pain First trimester preeclampsia Higher β -hCG than expected Hyperemesis gravidarum Hyperthyroidism Passage of hydropic (grape-like) vesicles
What is the differential diagnosis of GTD?	Normal pregnancy Spontaneous abortion Preeclampsia Placenta previa Placental abruption Endometriosis Ectopic pregnancy Ovarian tumor Prolapsed fibroid Cervical neoplasia
How should post-pregnancy bleeding be investigated?	Using curettage, serum hCG measurements, and a chest x-ray (CXR) (to assess if there are nodules associated with metastasis)
What is the major sign that GTD disease is metastatic?	hCG levels that do not decrease or that increase
Describe the following stages of GTD:	
Stage I	Disease that is limited to uterus
Stage II	Disease that extends outside uterus but stays within pelvis or vagina
Stage III	Disease with pulmonary metastases
Stage IV	Disease spreads to other sites
Where does metastatic GTD spread?	In order from most likely to least likely: Lung (80%)

	Vagina (30%)
	Brain (10%)
	Liver (10%)
	Other (bowel, kidney, spleen)
What are the good prognostic indicators for GTD?	Short duration between last pregnancy and disease (<4 months)
	Low hCG level (<40,000 IU/L)
	No metastatic disease to brain or liver
	No prior chemotherapy
What are the poor prognostic indicators for GTD?	Long duration between last pregnancy and disease (>4 months)
	High hCG level (>40,000 IU/L)
	Brain or liver metastases
	Prior chemotherapy
	Development of disease after a term pregnancy
What is the overall cure rate for GTD?	Over 90%
How does the diagnosis of GTD affect the prognosis for future pregnancies?	It increases the risk of subsequent molar pregnancies; however, this risk is still low (1%)

Hydatidiform Mole

What is a hydatidiform mole?	A localized, usually noninvasive tumor of the placenta that results from aberrant fertilization leading to proliferation of the trophoblastic tissue
What is the incidence of a molar pregnancy?	In the United States the incidence is 1 in 1500 pregnancies; however, it is much more common in the developing world
What are the signs and symptoms of a hydatidiform mole?	Amenorrhea Positive β -hCG Signs and symptoms consistent with early pregnancy Pregnancy-induced hypertension early in pregnancy Hyperthyroidism Vaginal bleeding

What is the difference between a complete and a partial hydatidiform mole? See Table 9-2

Table 9-2 Difference Between Complete and Partial Mole

	Complete Mole	Partial Mole
Synonyms	Classic or true mole	Incomplete mole
Karyotype	Diploid (46XX or 46XY)	Triploid (XXY or XYY)
Fetal/embryonic tissue	Absent	Present
Uterine size	Often large for dates	Often small for dates
Appearance of trophoblast	Diffuse hyperplasia, often atypia	Focal hyperplasia
Appearance of villi	Diffusely swollen	Focal swelling
Theca lutein cysts	Sometimes present	Never present
hCG levels	>50,000 IU/L	<50,000 IU/L
Placental alkaline phosphate levels	Normal	High
Malignant potential	15–25%	5–10%

Describe the pathogenesis of a complete mole

An “empty” egg (caused by inactivated or absent maternal chromosomes) is **fertilized by a haploid sperm that then duplicates**, or occasionally by two sperm. This leads to a “normal” diploid karyotype with all chromosomes of paternal origin

Describe the pathogenesis of a partial mole

A haploid ovum is fertilized by two haploid sperm, leading to a triploid karyotype

What is the typical sonographic appearance of a complete mole and a partial mole?

A central heterogeneous mass without an embryo/fetus or any amniotic fluid but with theca lutein cysts; classically described as a **snowstorm pattern**

A growth-restricted fetus, reduced amniotic fluid, and a Swiss cheese pattern of chorionic villi; no theca lutein cysts

What are the complications associated with a high hCG?

Ovarian enlargement (with theca lutein cysts)

Hyperemesis gravidarum

Early preeclampsia

Hyperthyroidism (because hCG may stimulate TSH receptors)

How is a molar pregnancy definitively diagnosed?

It is suspected based on ultrasound and hCG levels; however, it must be confirmed with histologic studies of the tissue with analysis of the DNA content

What is the treatment for a molar pregnancy?

Dilation and curettage (**D&C**; suction curettage is preferred)

Serum hCG levels must be followed weekly until there are three consecutive normal values and then monthly for a total of 6 months of negative levels. During this time, effective contraception *must* be used to avoid misinterpretation of a rising hCG level

What should be done before surgical evacuation of a complete molar pregnancy?

A **CXR** to assess for metastatic disease

When can another pregnancy be attempted after the diagnosis of GTN?

The patient should wait for at least a year

In what percentage of molar pregnancies will trophoblastic tissue persist after evacuation?

19–28% of complete moles
2–4% of partial moles

When is malignant disease suspected?

After a molar pregnancy when hCG levels rise. This usually represents an invasive mole (75%), but can also represent choriocarcinoma (25%) or a placental site trophoblast tumor (rare)

After a nonmolar pregnancy when hCG levels rise. This usually represents choriocarcinoma, but can (rarely) represent a PSTT

What are the risk factors for development of malignant disease?

Theca lutein cysts >6 cm
Larger uterine size for dates
Advanced maternal age (>40 years)
History of GTD
Initial hCG >100,000 IU/L
Histologic findings of atypia or hyperplasia

When does malignant disease develop?

50% of cases follow a hydatidiform mole, 25% follow a normal pregnancy, and 25% follow an abortion

What is the major complication associated with malignant GTD? Hemorrhage—these lesions are highly vascular and often have AVMs which readily bleed

Invasive Gestational Trophoblastic Neoplasia

What is an invasive mole? A molar pregnancy whose chorionic villi invade into the myometrium

Is an invasive mole more common after a complete mole or a partial mole? A complete mole

What is the treatment of an invasive mole? **Methotrexate** and hCG follow-up as with a molar pregnancy (actinomycin-D or etoposide are alternative therapies)

Can an invasive mole regress spontaneously? Yes

Choriocarcinoma

What is choriocarcinoma? A malignant carcinoma of the chorionic epithelium, usually after a molar pregnancy

What is the incidence of choriocarcinoma after a normal gestation, an abortion, and a complete mole? 1 in 16,000; 1 in 15,000; 1 in 40

What are the common signs/symptoms of choriocarcinoma? **Irregular vaginal bleeding** (typically late postpartum bleeding, but it can present later)
Enlarged uterus with bilateral ovarian cysts

What is the ultrasonographic appearance of choriocarcinoma? An enlarging, heterogeneous, hypervascular uterine mass with areas of hemorrhage and necrosis

What is the histologic appearance of choriocarcinoma? Proliferation of cytotrophoblasts and syncytiotrophoblasts that penetrate the musculature and vasculature; no villi present

What percentage of patients with choriocarcinoma develop metastases? Approximately 50%

What is the treatment for choriocarcinoma? Chemotherapy (typically **methotrexate** or **actinomycin-D** for low-risk disease and a combination of **methotrexate**, **actinomycin-D**,

What is the recommended follow-up after treatment of an invasive mole?	<p>cyclophosphamide, etoposide, and vincristine for high-risk disease)</p> <p>Adjuvant hysterectomy decreases the dose of chemotherapy required for remission</p> <p>Serial hCG follow-up (weekly for 3 months, then monthly) is recommended for at least a year. If there are metastases outside of the lung, follow-up is recommended for 2 years</p> <p>In both cases, the patient <i>must</i> use effective contraception in order to ensure appropriate interpretation of a rise in hCG</p>
---	--

Placental Site Trophoblastic Tumor

What is a PSTT?	A rare malignant tumor that arises from placental intermediate cytotrophoblastic cells
What are the signs/symptoms of PSTT?	<p>Irregular vaginal bleeding (can be massive hemorrhage)</p> <p>An enlarging uterus</p> <p>Amenorrhea</p> <p>Virilization</p> <p>Nephrotic syndrome</p> <p>Metastatic lesions</p>
Is PSTT always associated with high hCG levels?	No, because there is no syncytiotrophoblast proliferation
What is the ultrasonographic appearance of PSTT?	A hyperechoic intrauterine mass that is invading the myometrial wall and has both cystic and solid areas
What is the histological appearance of PSTT?	Many mononuclear cells that invade the myometrium; proliferation of intermediate trophoblast cells (no cytotrophoblasts or syncytiotrophoblasts)
What is the treatment for PSTT?	Hysterectomy is the first-line therapy because chemotherapy is fairly ineffective and the tumor is usually confined to the uterus

Late Complications

HYPERTENSIVE DISORDERS OF PREGNANCY

Hypertensive disorders in pregnancy are defined by blood pressures that are persistently elevated above what values?

Systolic >140 mm Hg or diastolic >90 mm Hg

What percentage of United States pregnancies are complicated by hypertensive disorders?

10–20%

What are the various hypertensive disorders observed during pregnancy?

Chronic hypertension (cHTN), which affects 3% of all U.S. pregnancies
 Pregnancy-induced hypertension (PIH) or gestational hypertension (GHTN), which affects 6% of all United States pregnancies
 Preeclampsia (PEC), which affects 5–8% of all U.S. pregnancies
 Eclampsia

If a woman at 20 weeks estimated gestational age (EGA) presents with a systolic pressure \geq 140 mm Hg or diastolic pressure 90 mm Hg, what is her probable diagnosis?

Chronic hypertension (cHTN), because it was likely present and undiagnosed prior to pregnancy. She should be evaluated for a molar pregnancy

A woman was noted to have multiple elevated blood pressures (BP) during the third trimester. At her IUD insertion 12 weeks postpartum, her BP is 140/90. Was her hypertension pregnancy induced?

No, she meets criteria for cHTN because her elevated BPs persisted >12 weeks postpartum. cHTN was likely present prior to her pregnancy

What antihypertensives are used to lower the BP of a pregnant woman?

Methyldopa for management of cHTN. Hydralazine for the acute management of hypertension associated with PEC. Additional agents include nifedipine and labetalol

How is GHTN diagnosed (dx)?

The onset of two BPs >140/90, separated by 6 hours, after 20 weeks EGA

What is the difference between GHTN and PEC?

Proteinuria is present in PEC, and it is absent in GHTN

Who are most at risk for PIH or toxemia?

Adolescent primiparous women, multiparous women >35 years of

	age, women with a history of PIH (there is a 33% chance of recurrence), and women with a different partner between pregnancies
What percentage of women with GHTN develop PEC?	50%
Does GHTN and PEC resolve postpartum?	Yes, they typically resolve by 12 weeks postpartum
How is PEC diagnosed?	The new onset of hypertension (>140/90) and proteinuria (≥ 0.3 g protein in a 24-hour urine specimen or persistent 1+ on dipstick) after 20 weeks of gestation. The elevated blood pressure should be documented on two occasions at least 6 hours. Edema is no longer one of the criteria evaluated for diagnosis
What is the cure for PEC?	Delivery, though a woman remains at risk for the development of eclampsia postpartum
What are other signs and symptoms of PEC?	<p>Endothelial damage: Elevated uric acid, plasma urate level >5.5 mg/dL (327 mmol/L) Sudden and rapid weight gain (>5 lbs/week) Facial edema</p> <p>Uteroplacental insufficiency: Intrauterine growth restriction (IUGR) Oligohydramnios (AFI <5) Alkaline phosphates in maternal serum because of placental vascular stress</p>
Once PEC is suspected why is 24-hour urine collected to assess protein excretion?	PEC is a dynamic process and single dipstick values do not correlate well with the degree of end-organ pathology present. 24-hour urine collection offers a more accurate reflection of the volume of protein lost by the kidneys. Proteinuria is defined as >300 mg/24 hours or >100 mg/dL
What are risk factors for the development of PEC?	Multiple gestations Obesity

	<ul style="list-style-type: none"> Personal or family history of PEC Primigravid state Pregestational diabetes Existing hypertension (GHTN, cHTN) Prolonged interval between pregnancies Renal disease (focal glomerulosclerosis) Advanced maternal age (greatest risk >40 years of age) Collagen vascular disease Antiphospholipid syndrome Other coagulation abnormalities (protein C or S deficiency, factor V Leiden mutation, and hyperhomocysteinemia)
	Note: smoking is not a risk factor
In the setting of cHTN, how is PEC diagnosed?	<p>With the onset of proteinuria or other signs and symptoms of end-organ disease.</p> <p>In hypertensive nephrosclerosis, protein excretion is typically <1 g/day.</p> <p>In the setting of new onset PEC proteinuria is typically >1+ on urine dip</p>
What are the two types of PECs?	Mild and severe
What percent of cases are mild in the United States?	75%
How is severe PEC defined?	PEC with any of the signs or symptoms of severe eclampsia, that are mentioned below
What are the systemic and clinical features of severe PEC?	<p>Endothelial damage</p> <ul style="list-style-type: none"> Severe hypertension >160 mm Hg systolic or >100 mm Hg diastolic Pulmonary edema because of capillary leak <p>Liver abnormalities</p> <ul style="list-style-type: none"> Elevated transaminases Epigastric or right upper abdominal pain, because of hepatic congestion and pressure on the capsule

Hematologic abnormalities

Hemoconcentration

Thrombocytopenia because of formation of microthrombi, platelets <100,000

Microangiopathic hemolysis, with schistocytes and helmet cells

Increased serum lactate dehydrogenase and bilirubin

CNS manifestations

Brisk deep tendon reflexes

Persistent headache, which does not resolve with medication, hydration, or rest

Visual changes (scotoma, blurring, cortical blindness)

Renal dysfunction

Nephritic range proteinuria (>5 g/day) because of the impaired integrity of the glomerular barrier

Glomerular capillary endotheliosis because of deposition of proteinaceous material

Oliguria <400 mL in 24 hours

What percentage of women with a history of PEC have a recurrence in subsequent pregnancies?

25–65% in severe PEC

5–7% in mild PEC

What are considered to be maternal complications of PEC?

Cerebral hemorrhage

Disseminated intravascular coagulation (DIC)

Eclamptic seizure

Pulmonary edema

Oliguria and renal failure

Rupture of the hepatic capsule

What is considered to be the most severe manifestation of PEC, without the onset of eclampsia (without seizures)?

HELLP syndrome (hemolysis, elevated liver function tests, low platelets), it is speculated that GHTN and eclampsia occupy a spectrum of hypertensive disorders that involve endothelial damage

How many women with PEC develop HELLP?

2% of patients

Can a woman develop HELLP without a prior diagnosis of PEC?	Yes, a small percentage of women do not exhibit hypertension or proteinuria
What laboratory abnormalities are associated with HELLP?	Lactate dehydrogenase >600 IU/mL Bilirubin >1.2 mg/dL Platelets <150 Elevated alanine and aspartate aminotransferase (AST and ALT)
What is the appropriate management of severe PEC, HELLP, or eclampsia?	Immediate delivery, through induction of labor or cesarean delivery (cxs), and anticonvulsant prophylaxis
What is the difference between PEC and eclampsia?	Grand mal or tonic-clonic seizures, which are not attributed to any other pathology. Eclampsia is associated with significant maternal and neonatal morbidity and mortality
What is the most common symptom prior to the onset of seizures?	Intense headache, which does not resolve with medication, hydration, or rest
Do all women who develop eclampsia have proteinuria?	No, 10% lack proteinuria; however, they typically have other clinical and histologic manifestations
How often do women with mild or severe PEC progress to eclampsia?	1 in 200 mild PEC patients and 2% of severe PEC patients seize if they are not treated with anticonvulsant prophylaxis. A woman is at increased risk for eclamptic seizures as she approaches delivery
What percentage of seizures occur prior to the onset of labor, during delivery, and within 48 hours postpartum?	25%, 50%, and 25% respectively
What medication is used to treat eclamptic seizures, in addition to its use as an anticonvulsant prophylaxis?	Magnesium sulfate (MgSO ₄) is the primary treatment. Morphine, barbiturates, benzodiazepines (diazepam), and antiepileptics have also been used
What are the signs and symptoms of magnesium toxicity?	Decreased deep tendon reflexes at 8 mg/dL, they are lost at 10 mg/L Lethargic and blunted mental status Respiratory depression at 15 mg/dL Pulmonary edema and shortness of breath Chest pain and cardiac arrest at levels of 30 mg/L

Why is urine output monitored during magnesium treatment?

Magnesium is renally excreted and blood levels may quickly become toxic if the patient become oliguric, <0.5 cc/kg/h

How is magnesium toxicity managed?

Calcium gluconate IV, hydration, respiratory and cardiac support

ABNORMAL PLACENTATION

What is a velamentous insertion of the cord or a velamentous umbilical cord?

Umbilical vessels that are surrounded only by fetal membranes, with no Wharton jelly. For this reason they extend to the maternal membranes, beyond the normal placental margin

Is a velamentous cord more common in twins?

Yes, it occurs in 10% of twins and 1% of singleton gestations

What is placenta previa?

The edge of the placenta is in close proximity to or overlies the internal os of the cervix

What is the incidence of placenta previa?

4 per 1000 pregnancies and 1 in 200 births

What are the various types of placenta previa?

Complete previa placenta covers the internal os of the cervix; accounts for approximately 30% of all previas

Marginal previa <2 cm from the cervical os

Low-lying placenta >2 cm, has no significance

Vasa previa velamentous fetal vessels cross the internal os (see Fig. 9-4)

Can placenta previa diagnosed early in pregnancy resolve during the course of the pregnancy?

Yes, as the lower uterine segment enlarges and the placenta grows; 90% will resolve by term

Rupture of membranes (ROM) or vaginal manipulation in vasa previa is associated with what risk?

Fetal exsanguinations, with a concomitant finding of non-reassuring fetal heart rate (FHR) and a sinusoidal pattern

What are risk factors for placenta previa?

Endometrial scarring caused by increasing parity, increasing maternal age, prior cxs, prior curettages, increased maternal age

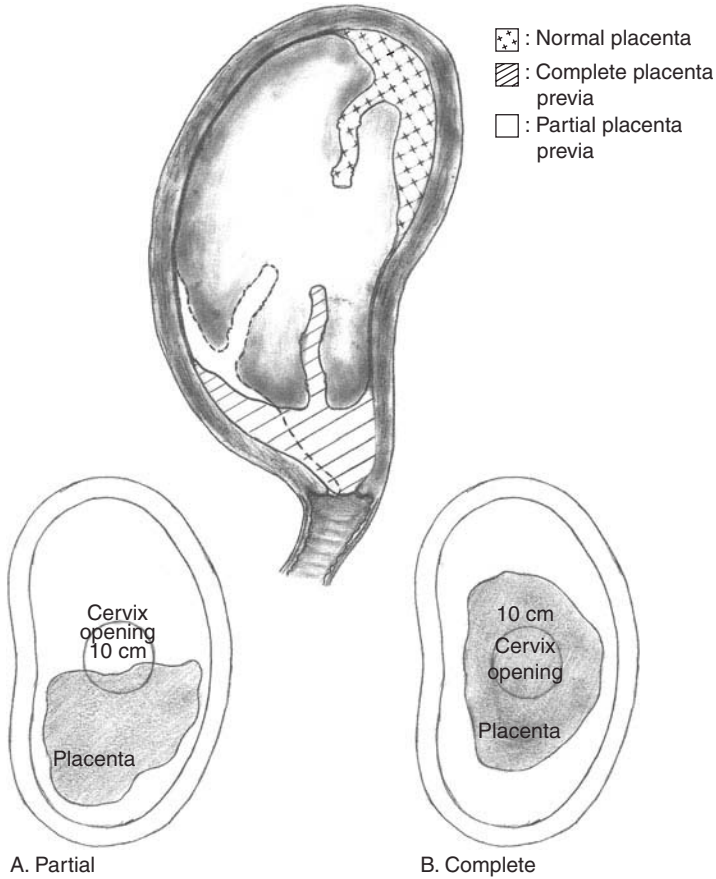


Figure 9-4 Types of placenta previa.

Increased uteroplacental transport demand, which may be caused by maternal smoking, cocaine abuse, higher altitudes, or multiple gestations

It is more common with male infants

Hemorrhage, placenta accrete (10%), malpresentation, and preterm premature rupture of the membranes (PPROM)

Yes, the recurrence rate is 4–8%

Cesarean delivery, amniocentesis is performed at 36 weeks to assess

What are some of the complications of placenta previa?

Are women with a history of placenta previa at risk for previa in subsequent pregnancies?

What type of delivery is indicated in previa?

What are the other forms of abnormal placentation?

pulmonary maturity. If immature, it is repeated weekly until maturity is confirmed with subsequent delivery

Bipartite placenta: composed of two equal portions separated by membranes and exposed large vessels

Battledore placenta: involves a marginal insertion of the cord

Fenestrated placenta: involves a central defect

Placenta diffusa (membranous): all fetal surfaces are covered by villi

Succenturiate placenta: has a marginal accessory lobe(s) or cotyledon

Circumvallata placenta: is small and limited by an amniotic ring

What abnormal placentation may cause retained products of conception and subsequent postpartum hemorrhage?

Succenturiate placenta, examination of the delivered placenta will exhibit vessels that do not taper; instead they extend past the edge

What abnormal placentation may cause increased risk of preterm delivery?

Circumvallata placenta, which is more common in older, multiparous patients

THIRD TRIMESTER BLEEDING

What is the most common cause of bleeding in the second and third trimester?

Bloody show associated with cervical insufficiency or labor (term and preterm)

Antepartum hemorrhage refers to what type of bleeding?

Vaginal bleeding after 20 weeks. EGA that is caused by some process other than labor and delivery. By definition this excludes preterm labor (PTL) and preterm delivery (PTD)

What are the major causes of antepartum bleeding?

Abruptio placentae 30%

Placenta previa 20%

Uterine rupture

Vasa previa

Other causes, such as, infections, trauma, polyps or neoplasia, and bloody show are associated with cervical changes because of insufficiency

How often is antepartum hemorrhage observed in the third trimester?

What is the initial management of painless blood per vagina in the third trimester?

Laboratory tests that identify the etiology of the vaginal bleeding (fetal vs. maternal) include:

What is the difference between a small marginal placental separation and placental abruption?

In 4% of pregnancies

Transabdominal ultrasonography is used for initial placental localization and evaluation for abnormalities. A manual examination should *not* be performed until previa is ruled out

Ogita, Londersloot, Apt, or Kleihauer—Bentke tests identify fetal cells in maternal circulation

EGA, or when the event occurs during pregnancy. The former is used to describe noncatastrophic placental hemorrhage before 20 weeks EGA. The latter refers to any placental separation after 20 weeks (see Fig. 9-5)

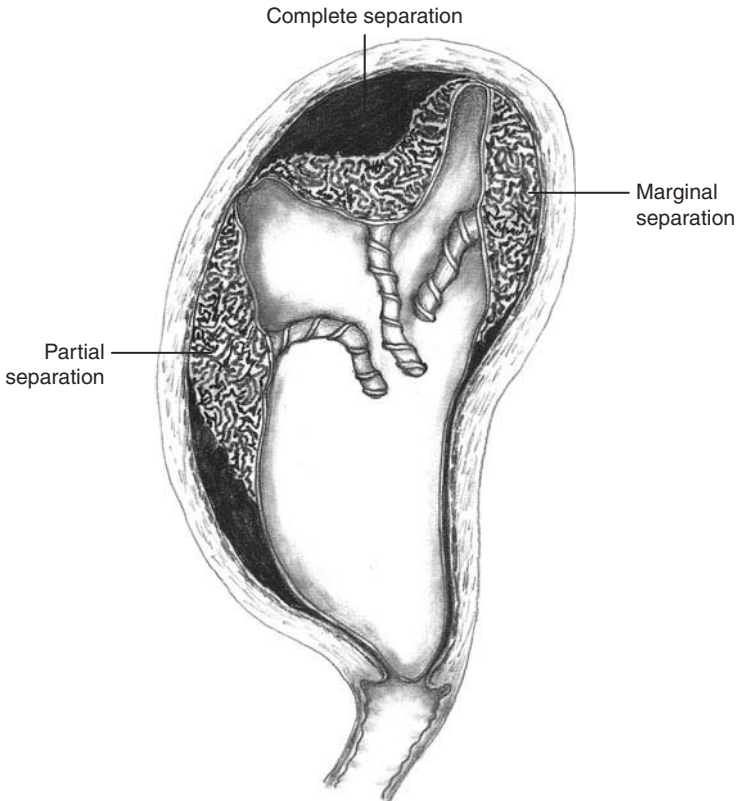


Figure 9-5 Types of placental previa separation.

What is the classic presentation of placenta previa?

Painless blood per vagina after 20 weeks EGA, which may range from spotting to hemorrhagic. This presentation occurs in 70–80% of patients

How often are painful uterine contractions reported in the setting of vaginal bleeding because of placenta previa?

10% of presentations. However, vaginal bleeding and painful uterine contractions are more suggestive of abruption, especially in the setting of recent trauma

Delivery should be expedited in which situations?

Persistent non-reassuring FHR, hemodynamic instability because of refractory maternal hemorrhage, significant bleeding with known fetal pulmonary maturity (>34 weeks EGA), disseminated intravascular coagulopathy or other consumptive coagulation disorder

In the context of intermittent contractions, third trimester bleeding, immature fetal lung maturity (FLM) results, and <32 weeks EGA, what conditions must be met to allow for 24 hours of corticosteroids with possible tocolysis?

The mother must be hemodynamically stable and uninfected. The fetus should have a reassuring FHR. There are conflicting data regarding the benefit of steroids after 32 weeks EGA

PROM AND PPROM

A term patient reports a sudden gush of fluid with continued loss of fluid and no uterine contraction, what is the initial management?

Ask her to come to the hospital for further evaluation, confirm membrane rupture, confirm the gestational age, and assess the fetal status

What is the definition of premature rupture of membranes (PROM)?

Rupture of placental membranes (ROM), or amniorrhexis, in a full-term pregnancy, with the absence of labor

How prevalent is PROM?

It occurs in 8% of U.S. pregnancies

Pregnant women occasionally report leakage of fluid without ROM; this is because of what side effects of pregnancy?

Sporadic urinary incontinence (stress incontinence), because of progesterone and increased pressure on the bladder by the gravid uterus. The patient should still be evaluated for possible ROM

What physical findings support the diagnosis of ROM?

Pooling of vaginal fluid or leakage with coughing on sterile speculum examination, positive nitrazine test of

What laboratory studies support the diagnosis of ROM?

the vaginal fluid, ferning of dried fluid under low power magnification, ultrasound with decreased amniotic fluid (AF), amnio dye test (pigment is injected into the AF and a tampon is examined for leakage)

Do women who present with PROM go into spontaneous labor?

Placental alpha microglobulin-1, insulin-like growth factor binding protein-1 (IGFBP-1), alpha-fetoprotein (AFP), fetal fibronectin, creatinine, human chorionic gonadotropin, di amino-oxydase, prolactin, ceruloplasmin, and lactate are all substances found at higher concentrations in the AF

Yes, 70% will go into labor within 24 hours and 85% within 48 hours

What is considered prolonged PROM?

PROM for more than 18–24 hours, without the onset of labor

In a patient with PROM, what is suggested by expectant management?

The fetus and mother are monitored or closely followed, with the expectation that the patient is likely to go into spontaneous labor. No efforts are made to immediately induce delivery

What are the benefits of immediate induction upon presentation to the hospital with PROM, instead of expectant management?

Reduced fetal and maternal infections, such as chorioamnionitis, endometritis, neonatal sepsis, and intensive care unit admission

What are the risks of induction with a poor Bishop score?

Failed induction, cxs, protracted labor, cost of prolonged hospitalization, maternal or neonatal infection, uterine rupture because of hyperstimulation with tocolytics

What is the definition of preterm premature rupture of membranes (PPROM)?

ROM in the absence of labor at <37 weeks EGA

How prevalent is PPRM?

It affects 1–3% of U.S. pregnancies or 25% of PROM

What is the most common risk factor for the development of PPRM?

Genital tract infections and chorioamnionitis

What are other risk factors for the development of PPRM?

Placenta previa, smoking, cervical incompetence, multiple gestations, polyhydramnios, antepartum hemorrhage, personal history of PPRM

What is the rate of PPROM recurrence in subsequent pregnancies?	13.5%
Is PPROM an indication for hospitalization?	Yes, from the time of diagnosis until delivery
Prior to delivery, what type of adverse outcomes are seen following PPROM?	Intrauterine infection 13–60% Placental abruption 4–12% Umbilical cord prolapse 1–2% Precipitous delivery
What are the most severe adverse neonatal outcomes associated with PPROM?	Hyaline membrane disease Acute respiratory distress syndrome (ARDS) Intraventricular hemorrhage (IVH) Infection (sepsis, pneumonia, meningitis) Necrotizing enterocolitis (NEC) Other adverse events include: thermal instability, fluid and electrolyte disturbances (hypocalcemia, hypokalemia), hypoglycemia, hyperbilirubinemia, patent ductus arteriosus, irregularities in pulmonary and cardiac function (apnea and bradycardia), poor feeding
In the setting of EGA ≥ 32 weeks, documented fetal lung maturity, and PPROM is it advisable to engage in immediate delivery or expectant management?	In this context, immediate delivery has been shown to result in better outcomes, as long as expert neonatal care is available
What are fetal lung maturity tests?	Lecithin/sphingomyelin ratio (L/S) $> 2-3.5$ Acidic phospholipids phosphatidylinositol (PI) or foam stability index (TDX) > 48 Phosphatidylglycerol (PG) present Percent perceptible lecithin or fluorescence polarization > 55 mg/g, optical density Surfactant to albumin ratio (S/A) Saturated phosphatidyl choline (SPC)
What is surfactin?	A phospholipid produced by type II pneumocytes at 29 weeks EGA, which decreases surface tension in

When and why is betamethasone or dexamethasone used?

In the context of immature fetal lung maturity results, <32 weeks EGA, and PPROM, delivery may be delayed 24 hours so as to offer what treatments?

What effect does prophylactic antibiotic administration have in the setting of PPROM?

What conditions would require immediate delivery of PPROM, regardless of lung maturity?

Corticosteroid administration increases the risk of:

the alveoli. Insufficient surfactin production will result in ARDS

They are corticosteroids that cross the placenta and stimulate production of surfactin and lung maturation in the 27–32-week EGA fetus. It is given in two doses 12 hours apart and is believed to attain its maximal impact after 12 hours

Two doses of corticosteroids, to hasten the production of surfactant in the type II pneumocytes. Antibiotics are often concurrently administered. There are conflicting data regarding the benefit of steroids after 32 weeks EGA, though several physicians will offer it to their patients through 34 weeks EGA

It reduces the rate of maternal and neonatal infection, and prolongs the latency period between the time of membrane rupture and the onset of labor. Ampicillin is often used with erythromycin

The development of advanced labor, abruption placentae, maternal hemodynamic instability (likely because of hemorrhage), intrauterine infection, non-reassuring fetal testing, fetal compromise (severe oligohydramnios or cord prolapse)

Chorioamnionitis

Preterm Labor and Preterm Delivery

What is the definition of preterm labor (PTL)?

Labor (regular uterine contractions with cervical change) that starts between the 20th and 37th week of pregnancy

What is the most significant risk factor for PTL?

Previous history of preterm birth (PTB) or preterm delivery (PTD)

What are other risk factors for PTL?

Multiple gestations (the risk increases with increasing number of fetuses)

	<p>Adolescent pregnancy (greatest risk <15 years of age)</p> <p>Advanced maternal age (>35 years of age)</p> <p>Cervical insufficiency</p> <p>Maternal genital tract infections</p> <p>Uterine malformations</p> <p>Cigarette smoking</p> <p>Maternal substance abuse (cocaine is the most common illicit substance associated with PTL)</p> <p>A change in partners between pregnancies</p> <p>Low socioeconomic status (education, occupation, and family income)</p> <p>Race/ethnicity, with black women having the highest rates</p>
What is the definition of preterm birth (PTB) or preterm delivery (PTD)?	<p>Birth that occurs before 37 completed weeks (less than 259 days) of estimated gestation age (EGA). Less than 28 weeks of gestation is considered an extreme PTB</p>
What are the causes of PTD?	<p>20% Iatrogenic, for maternal or fetal indications</p> <p>Intrauterine growth restriction</p> <p>Severe PEC</p> <p>Placenta previa with unstable hemorrhage</p> <p>Non-reassuring fetal testing</p> <p>30% Preterm premature rupture of the membranes (PPROM)</p> <p>20–25% Intra-amniotic infection</p> <p>25–30% Other pathology or idiopathic preterm labor</p>
What is the prevalence of PTD in the United States?	<p>12% of all deliveries, 2% occur before 32 weeks</p>
What is the impact of PTD on neonatal morbidity and mortality?	<p>In the United States, PTB accounts for 85% of all perinatal morbidity and mortality. Disability occurs in 60% of survivors of birth at 26 weeks EGA</p>

Hydration may decrease preterm uterine contractions based on dilution of what hormone?

Antidiuretic hormone (ADH) is increased in the setting of dehydration and it may have some crossreactivity with uterine oxytocin receptors. Diluting ADH and decreasing the systemic production may decrease preterm uterine irritability

What class of medications is used to prevent uterine contractions?

Tocolytics, such as, magnesium sulfate (increases extracellular calcium), calcium channel blockers (nifedipine), beta adrenergic agonists (terbutaline, ritodrine)

In what scenario is there a clear indication for tocolytics?

To delay delivery 48 hours in PTL with EGA <32 weeks so as to administer 24 hours of corticosteroids and hasten the production of surfactant in the type II pneumocytes

Why is it not advisable to use more than one tocolytic concurrently?

Increased risk of pulmonary edema. The use of two tocolytics has not been show to benefit maternal or fetal outcomes

What do contraindications to tocolysis include?

Fetal distress, severe oligohydramnios, fetal demise, intrauterine infection

Tocolysis with an antiprostaglandin, such as indomethacin, can prolong the interval to labor in a PTL patient; however, it carries what significant risk to the fetus?

Premature closure of the ductus arteriosus, with subsequent pulmonary hypertension

What type of patient has been shown to benefit from progesterone supplementation in randomized controlled trials?

Patients with a history of spontaneous or idiopathic PTL. Progesterone injections are administered to asymptomatic patients as weekly injections 17 alpha-hydroxyprogesterone caproate (250 mg) or daily progesterone vaginal suppositories (100 mg) beginning in the second trimester and continuing through term

Prophylactic progesterone injections reduce the rate of PTB by what percent?

15–70%, but there is no significant impact on perinatal mortality, morbidity, or miscarriage

A woman who presents with painless cervical dilation and bulging fetal membranes during the second trimester is consistent with what diagnosis?

Cervical insufficiency, formerly known as cervical incompetence

What sonographic findings are consistent with cervical insufficiency?

Short cervical length, dilated internal cervical os, and funneling of the fetal membranes

How prevalent is cervical insufficiency?

2% of all U.S. pregnancies

What treatment may be offered to patients with cervical insufficiency?

Prophylactic or emergent cerclage, the efficacy of this intervention may be specific to certain subgroups; however, it has not been consistently demonstrated in trials

What are risk factors for cervical insufficiency?

History of cervical colonization, multiparity, history of cervical trauma

What is considered a highly specific test for PTL?

Fetal fibronectin (fFN) assay because of a false-positive rate of 3–4% or a low prevalence of positive results in asymptomatic low-risk women. Unfortunately, the sensitivity and positive predictive value (41%) are not as impressive

What is fetal fibronectin?

Fetal fibronectin is a glycoprotein present in maternal circulation and AF, if present in the cervicovaginal secretions >50 ng/mL; it suggests that the cervix is undergoing structural change and the patient may go into labor in the next 2 weeks. Positive predictive value is 41%

Would fetal fibronectin be indicated in a 32-week EGA patient with painful uterine contractions?

Yes. The test is more valuable for its negative predictive value of 96%. If the protein is low or absent there is a low probability that she will go into PTL in the next 2 weeks. The swab must be collected prior to any other vaginal cultures, examination, or procedure. It can be repeated in 2 weeks if she presents with signs or symptoms consistent with PTL

What common interventions have *not* proven to prevent PTL?

Bed rest, home uterine monitoring, prophylactic broad-spectrum antibiotic therapy, abstinence from sex, hydration, long-term or prophylactic tocolytic therapy

Maternal and Fetal Pathology in Pregnancy

What is round ligament pain?

As the gravid uterus increases rapidly during the second and third trimester, tension on the round ligament may cause sharp shooting inguinal and groin pain. Rest, warm compresses, and acetaminophen may relieve the pain

In what circumstances is fundal height greater than dates?

Twins, polyhydramnios, macrosomia, fibroids, hydatidiform mole (molar pregnancy)

What conditions or anomalies are associated with polyhydramnios?

Gestational diabetes mellitus (GDM), fetal anomalies of the nervous (anencephaly), or gastrointestinal system (tracheosophageal fistula or atresia)

What conditions or anomalies are associated with oligohydramnios?

Bilateral renal anomalies (Potter), posterior urethral valves in males, hypertensive disorders of pregnancy (gHTN, PEC, HELLP)

What are amniotic bands?

Placental membrane defects that result in fetal deformities or amputations; they are believed to be because of sclerosis and are associated with oligohydramnios

How prevalent are birth defects of medical, surgical, or cosmetic significance?

2–4% of all viable infants, irrespective of ethnic background. Most birth defects are multifactorial

What is the definition of a low birth weight infant?

Less than 2500 g

What are causes for infants born less than 2500 g?

PTD, small for gestational age (SGA), intrauterine growth restriction (IUGR), infection (rubella or CMV), teratogen exposure, chromosomal or congenital anomalies. The smaller the infant the higher the mortality

When is a fetus considered small for gestational age?

When the estimated fetal weight is less than the 10th percentile at a given EGA

Is symmetric or asymmetric IUGR more prevalent?

Asymmetric is observed in 80% of cases

What type of insult or anomaly is believed to contribute to symmetric IUGR?

A long-standing pathologic process, which began early in the pregnancy. Possible causes of IUGR include: first-trimester infections, fetal anomalies (severe cardiac defect), systemic maternal disease (DM type I, HTN), or chromosomal abnormalities (trisomy 13, 18, 21)

What type of insult or anomaly is believed to contribute to asymmetric IUGR?

Asymmetric IUGR is often attributed to a pathologic process occurring later in pregnancy such as: uteroplacental insufficiency because of PEC, twin transfusion syndromes, or teratogen exposure

Following intrauterine fetal demise, what is the greatest maternal risk?

Disseminated intravascular coagulation (DIC) because of increased thromboplastin release from the products of conception

Why is a mother with multiple sclerosis likely to experience a flare postpartum?

Pregnancy induces a state of weakened cell-mediated immunity. Th1, Il-2, and TNF are all decreased. For this reason certain autoimmune diseases seem to subside during pregnancy. However, humoral Th2 immunity is strengthened, with increased Il-4 and Il-10. This may result in lupus flares during pregnancy

The following sonographic findings are characteristic of what fetal anomalies?

Double-bubble duodenal atresia, the appearance is because of fluid collections in the stomach and truncated intestine

Banana sign communicating spina bifida, the appearance is because of the flattened cerebral hemispheres and cisterna magna

Lemon sign neural tube defects, the appearance is because of an

	indentation of the calvarium or scalloping of the frontal bones
What is the incidence of stillbirth in the United States?	6.4 per 1000 total deliveries
What are the most common causes of stillbirth 24–27 weeks EGA?	Infection Abruptio placentae Anomalies
What are the most common causes of stillbirth <28 weeks EGA?	Unexplained Fetal malnutrition Abruptio placentae
What are the risk factors for stillbirth?	Prepregnancy obesity Advanced maternal age Socioeconomic factors (low education attainment) Hypertensive disorders Diabetes Systemic lupus erythematosus Renal disease Thyroid disorders Thrombophilia Cholestasis of pregnancy Smoking >10 cigarettes per day History of an SGA fetus History of stillbirth Multiple gestations Race (black women have twice the risk)
In North Americans what percentage of stillbirths are because of intrapartum asphyxia?	Less than 1% of stillbirths. The rate was dramatically decreased by the implementation of intrapartum monitoring during the 1950s. Unfortunately, intrapartum monitoring has not reduced the rate of cerebral palsy
What are the leading causes of traumatic maternal morbidity and mortality during pregnancy?	Motor vehicle accidents (MVAs) account for 55–70% of injuries according to national hospital registries

What percentage of U.S. pregnancies experience significant nonobstetrical trauma?

What is the risk of fetal death following abruption?

Seat belt use was reported in less than half of these cases

Falls account for 10–20%

Interpersonal and domestic violence accounts for 12–31%

However, the CDC considers interpersonal violence to be the leading cause of traumatic maternal injury

6–7%, with the incidence of trauma increasing throughout the course of the pregnancy

42%

Postpartum Complications

POSTPARTUM HEMORRHAGE

What is the significance of postpartum hemorrhage?

It is the most significant cause of maternal death worldwide. There are approximately 140,000 deaths per year because of severe bleeding—otherwise estimated as 1 death every 4 minutes

How is postpartum hemorrhage defined?

It is excessive bleeding following delivery. Although uncomplicated vaginal deliveries may have an average blood loss of 700 mL, blood loss > 500 mL following a vaginal birth or a blood loss of > 1000 mL following cesarean birth qualifies as hemorrhage

Postpartum hemorrhage is classified as primary or secondary. What is meant by each type?

Primary: occurs within the first 24 hours of delivery and occurs in 4–6% of pregnancies

Secondary: occurs between 6–12 weeks postpartum in about 1% of pregnancies

What are the etiologies for primary postpartum hemorrhage and secondary postpartum hemorrhage?

Primary: uterine atony (80%), obstetric lacerations (episiotomy involving large arteries), retained placenta (i.e., placenta accreta), coagulation defects, uterine inversion

What are risk factors for postpartum hemorrhage?

Secondary: subinvolution of placental site, retained products of conception, infection, inherited coagulation defects

Prolonged labor, augmented labor, rapid labor, history of postpartum hemorrhage, overdistended uterus (macrosomia, twins, hydramnios), episiotomy, operative delivery, Asian or Hispanic ethnicity, chorioamnionitis

In addition to mortality, what major morbidities follow postpartum hemorrhage?

Note: postpartum hemorrhage can often occur without warning

Adult respiratory distress syndrome (ARDS), coagulopathy, shock, loss of fertility, and pituitary necrosis (Sheehan syndrome)

How does Sheehan syndrome clinically manifest?

Failure to lactate, amenorrhea, decreased breast size, loss of pubic and axillary hair, hypothyroidism, and adrenal insufficiency

How should each of the following be evaluated as a cause of excessive bleeding immediately after placental separation? How is each etiology managed?

Uterine atony: pelvic exam reveals a soft, poorly contracted (“boggy”) uterus. Compression or massage of the uterus can diminish bleeding

Obstetric-related lacerations: careful visual assessment of the lower genital tract is necessary. Proper patient positioning, operative assistance, anesthesia, and proper repair of lacerations are indicated

Genital tract hematomas: patient complains of pelvic pressure and pain; mass enlargement may be visualized. The hematoma should be surgically drained. The bleeding vessels may also be embolized

What are proper supportive measures that should be instituted early in women suspected to have postpartum hemorrhage?

How can uterine atony and subsequent bleeding be prevented?

If uterotonic agents with or without vaginal tamponade measures fail to control bleeding, what procedure is indicated next?

What is the definitive measure to control postpartum hemorrhage?

Retained products of conception: ultrasound can help diagnose a retained placenta. This may be removed by forceps, guided by ultrasonography

Coagulopathy: patient and family history of clotting disorders, CBC, PT/PTT, fibrinogen levels. Type and cross should be ordered. Surgery and blood transfusion may be necessary

IV access, type & cross, blood products requests from the blood bank, crystalloid infusions, communication with anesthesiologists, preparation of operating room

Administer uterotonic agents, such as oxytocin, immediately after delivery. If uterotonic agents fail to control the bleeding, packing or tamponade of the uterus may help

Exploratory laparotomy

Hysterectomy

PLACENTA ACCRETA

Placenta accreta is one of the two most common reasons (the other being uterine atony) for postpartum hemorrhage and hysterectomy. Describe placenta accreta

What is placenta increta and placenta percreta?

What are risk factors for placenta accreta?

It is the abnormal attachment of the placenta to the inner uterine wall without an intervening decidual layer

Placenta increta: the placenta invades the myometrium

Placenta percreta: the placenta penetrates the full thickness of the myometrium

Placenta previa in current pregnancy
Previous cesarean delivery
 Previous myomectomy

<p>Are there any tools that can diagnose placenta accreta?</p>	<p>Asherman syndrome Submucous leiomyoma Maternal age > 35 years</p> <p>Though ultrasonography and color doppler studies may help in the diagnosis, they are not definitive for placenta accreta</p>
<p>What is another consequence of placenta accreta that is life-threatening?</p>	<p>Uterine rupture</p>
<p>In what other situations can uterine rupture occur?</p>	<p>History of a previous cesarean delivery</p> <p>Excessive intrauterine manipulation</p> <p>Congenital malformations of the uterus (small uterine horn)</p> <p>Spontaneously</p>

UTERINE INVERSION

<p>What is uterine inversion?</p>	<p>It is when the uterine corpus/fundus prolapses to (incomplete inversion) and sometimes through (complete inversion) the uterine cervix, so that it is in effect turned inside out. It is also associated with severe hemorrhage</p>
<p>In addition to postpartum hemorrhage, what is the immediate morbidity associated with uterine inversion?</p>	<p>Endomyometritis</p>
<p>What are several conditions that predispose to uterine inversion?</p>	<p>Fundal implantation of the placenta</p> <p>Partial placenta accreta</p> <p>Uterine anomalies</p> <p>Weakness of the myometrium</p> <p>Strong traction exerted on the umbilical cord</p> <p>Fundal pressure</p>
<p>What findings on physical examination suggest an inverted uterus?</p>	<p>A bimanual examination may reveal a firm mass at or below the cervix, and an abdominal exam will reveal a depression in the location or the absence of the uterine fundus</p>
<p>If uterine inversion occurs before placental separation, what should <i>not</i> be done?</p>	<p>Removal or detachment of the placenta should <i>not</i> be performed. This will cause profound hemorrhage</p>

How should uterine inversion be managed?

Either manual or surgical repositioning of the uterus

POSTPARTUM AND PUERPERAL INFECTIONS

How is puerperal infection and puerperal morbidity because of infection defined?

Puerperal infection is used to describe any bacterial infection of the genital tract after delivery

Puerperal morbidity because of infection occurs when the patient develops a **temperature >38°C (100.4°F)** on **two separate occasions at least 24 hours apart following the first 24 hours of delivery**

What are several risk factors for puerperal morbidity because of infection?

Lower socioeconomic status
 Cesarean delivery
 Premature rupture of the membranes
 Long labors
 Multiple pelvic examinations

What are several types of postpartum infections?

Uterine infection (endometritis)
 Respiratory Infections (ARDS, aspiration pneumonia, bacterial pneumonia)
 Urinary tract infections (acute pyelonephritis)
 Wound infections (cesarean delivery incision)
 Mastitis
 Thrombophlebitis

Most postpartum infections are caused by organisms that are present in the female genital tract and which also normally cause female genital tract infections.

What are these common bacterial agents?

Gram-positive cocci: group A, B, and D streptococci, *Staphylococcus aureus*

Gram-positive bacilli: *Clostridium* species

Aerobic gram-negative bacilli: *Escherichia coli*, *Klebsiella*, *Proteus* species

Anaerobic gram-negative bacilli: *Bacteroides fragilis* group

Other: *Mycoplasma* species, *Chlamydia trachomatis*, *Neisseria gonorrhoeae*

ENDOMETRITIS

What does endometritis in the postpartum period refer to?

It refers to infection of the decidua (i.e., pregnancy endometrium), and also the myometrium (endomyometritis) and parametrial tissues (parametritis)

What is the most significant risk factor for endometritis?

The route of delivery. The risk of infection is 5–10 times higher in cesarean delivery compared to vaginal delivery

Which two bacterial agents have been found to be specific for endometritis?

Bacterial vaginosis and group B streptococcus

How is the diagnosis of endometritis determined?

Fever (> 100.4°F)

Uterine tenderness

Foul lochia

Leukocytosis (increased neutrophil count with left shift)

Typically presents on postpartum day 2 or 3

What is the “gold-standard” treatment?

IV clindamycin and gentamycin q8h. Improvement is usually seen 48–72 hours after treatment has begun

If the fever has not subsided after 72 hours of antibiotic administration or after a change in antibiotic therapy, what other sources of fever must be considered?

Wound infection

Septic pelvic thrombophlebitis

Pelvic abscess

Drug-induced fever

Are prophylactic antibiotics indicated in women undergoing cesarean delivery?

Yes. Prophylaxis reduces the rate of endometritis by two-thirds to three-quarters. Single agents such as **ampicillin and first-generation cephalosporins** are ideal prophylactic antibiotics

In what setting does chronic endometritis occur?

It occurs when there are retained products of conception after spontaneous abortion, pregnancy termination, or delivery

What are the clinical manifestations of chronic endometritis?

Fever

Irregular vaginal bleeding

Pelvic pain

Malaise

<p>What is the treatment of chronic endometritis?</p>	<p>A tender, boggy, and enlarged uterus with bloody and/or purulent discharge on physical examination</p> <p>Curettage to remove the necrotic material</p>
---	--

Medical Conditions of Pregnancy

ENDOCRINE DISORDERS

Gestational Diabetes Mellitus

<p>What is pregestational diabetes mellitus (GDM) and how is it characterized?</p>	<p>It is a diagnosis of diabetes mellitus prior to pregnancy. It occurs in 1% of all pregnancies and includes both Type I diabetes and Type II diabetes. Type II pregestational diabetes is more common and is characterized by onset later in life, obesity, peripheral insulin resistance, some insulin deficiency, and end-organ complications (renal, vascular, nervous). Type I pregestational diabetes is less common and occurs most often early in life. It is characterized by an autoimmune process that destroys the pancreatic β cells</p>
<p>What is GDM and how is it characterized?</p>	<p>It is a diagnosis of diabetes mellitus defined as glucose intolerance with onset or first recognition during pregnancy. 90% of diabetes cases encountered during pregnancy are GDM and more than one half of those patients at risk will end up developing pregestational diabetes later in life</p>
<p>How may pregnancy predispose some women to GDM?</p>	<p>Placental secretion of anti-insulin and diabetogenic hormones that contribute to the diabetic state include:</p> <ul style="list-style-type: none"> Growth hormone Corticotropin-releasing hormone Human placental lactogen Prolactin Progesterone

What are several risk factors for the development of GDM?

Tumor necrosis factor- α and leptin have also been implicated in creating the insulin-resistant state of pregnancy

Age >25

Obesity (BMI >30 in the nonpregnant state)

Prior history of GDM

Family history of diabetes (especially in a first-degree relative)

Previous stillbirth or child with a congenital malformation

Birth of a prior infant with weight >9 lbs (or history of macrosomia)

Polycystic ovary syndrome (contributes to the insulin resistance state)

2⁺ glycosuria (debatable in the literature)

In addition to diabetic retinopathy, nephropathy, and neuropathy, what are several obstetric-related maternal complications associated with GDM?

Preeclampsia

Preterm birth

Macrosomia and birth trauma (especially shoulder dystocia)

Intrauterine growth restriction

Polyhydramnios

First trimester abortions and stillbirths

Asymptomatic bacteriuria

Higher incidence of cesarean section, vacuum, and forceps deliveries

Higher incidence of neonatal respiratory distress syndrome (delay in the fetal lung maturity)

With what other endocrine disorders is Type I diabetes mellitus associated?

There is a 5–8% incidence of hypothyroid disease as well as ~25% risk of developing postpartum thyroid dysfunction

What are several adverse neonatal outcomes associated with hyperglycemia?

Congenital malformations

Macrosomia

Intrauterine fetal demise

Hypoglycemia

Hypocalcemia

Respiratory distress syndrome

What congenital malformations are associated with maternal pregestational diabetes mellitus?

Polycythemia
 Organomegaly (cardiac)
 Hyperbilirubinemia
 Heart defects (**transposition of the great vessels**, ventricular septal defect [VSD], atrial septal defect [ASD])
 Neural tube defects
Caudal regression (pathognomonic, but very rare)
 Situs inversus
 Anal/rectal atresia
 Renal anomalies (duplex ureter)

How does hyperglycemia cause congenital malformations?

Hyperglycemia is teratogenic during the period of organogenesis (first 8 weeks of pregnancy); therefore, preconceptual glucose control and monitoring is crucial for normal development

Glycosylated hemoglobin (HbA_{1c}) levels correlate directly with the frequency of congenital anomalies. What is this relationship?

HbA _{1c} Levels (%)	Frequency of Anomalies (%)
5–6	2–3
8.9–9.9	8.1
10	20–25

How does GDM cause fetal macrosomia?

Maternal glucose crosses the placenta and creates a hyperglycemic environment for the fetus. In response, the fetus produces more insulin. Insulin is a potent growth hormone and leads to increased somatic growth, macrosomia, central fat deposition, and enlargement of internal organs (i.e., heart)

Which women should be screened for gestational diabetes?

Though controversial, **universal screening of all pregnant women is recommended by ACOG**. However, low-risk women may be exempt from screening. These women should have all of the following characteristics:

Age <25 years
 Normal weight or BMI before pregnancy
 No first-degree relative with diabetes mellitus
 No history of abnormal glucose tolerance test
 No history of poor obstetric outcome, macrosoma, vacuum, forceps, shoulder dystocia (even with good outcome)
 Member of an ethnic group with a low prevalence of GDM (i.e., patient is *not* Hispanic, African, Native American, South or East Asian, Pacific Islander)

When should screening be performed?

If there is a high suspicion of GDM, screening should be done at the first antenatal visit; otherwise, screening can be performed at 24–28 weeks of gestation. (If a high-risk patient has a negative screening test at the first antenatal visit, she should be rescreened at 24–28 weeks.)

What screening test is recommended for GDM?

A **50 g oral glucose challenge test (GCT)** is given. Plasma or serum glucose level is measured 1 hour later without regard to the time of the prior meal. A value 140 mg/dL (7.8 mmol/L) is the most commonly used parameter and is considered abnormal (some authors recommend using a cut-off as low as ≥ 130)

If the screening test is positive, what are the next recommended tests for diagnosing gestational diabetes?

A **100-g, 3-hour oral glucose tolerance test (GTT)** performed after an overnight fast. GDM is present if a diagnosis of two or more of the following are met or exceeded:

Status	Plasma or Serum Glucose Level (mg/dL)	Plasma or Serum Glucose Level (mmol/L)
Fasting	95–105	5.3
1 hour	180	10.0
2 hours	155–165	8.6
3 hours	140	7.8

What is the White Classification of Diabetes?

The White Classification System initially attempted to predict perinatal risk according to the age of onset of diabetes, duration of diabetes, and type of end-organ damage. The American College of Obstetricians and Gynecologists has recommended a single diabetes classification system as follows:

Class	Onset	Fasting Plasma Glucose	2-Hour Postprandial Glucose	Therapy
A ₁	Gestational	<105 mg/dL	<120 mg/dL	Diet
A ₂	Gestational	>105 mg/dL	>120 mg/dL	Insulin

Class	Age of Onset (yr)	Duration (yr)	Vascular Disease	Therapy
B	>20	<10	None	Insulin
C	10–19	10–19	None	Insulin
D	"10	≥20	Benign retinopathy	Insulin
EF	Any	Any	Neph (NEF) ropathy	Insulin
R	Any	Any	Proliferative retinopathy	Insulin
H	Any	Any	Heart disease	Insulin
RT	Any	Any	Renal transplant	Insulin

What are the main considerations in the management of GDM?

1. Preconceptional counseling, achievement of normal hemoglobin A_{1c} levels before pregnancy, glucose monitoring and control, diet and exercise adjustments, and insulin if necessary
2. In addition to a routine prenatal checkup, the patient should be assessed for glycosylated hemoglobin concentration; baseline renal function with serum creatine level and a 24-hour urine collection analysis; asymptomatic bacteriuria by urine culture; thyrotropin and free thyroxine; electrocardiogram; dilated and comprehensive eye examination by an

What antepartum fetal assessment is appropriate in women with

- ophthalmologist; and first trimester ultrasound examination to confirm gestational age
3. Monitoring of and intervention for fetal or obstetrical complications

1. An ultrasound assessment for fetal growth and anatomy **pregestational diabetes?** (i.e., heart) and a fetal echocardiogram should be completed around 18–20 weeks
2. Testing for fetal malformations should also be performed in the first trimester (nuchal translucency and serum screening for neural tube defects and/or second trimester triple or quadruple screening)
3. At 32 weeks, a weekly nonstress test and/or biophysical profile, and amniotic fluid volume should be performed increasing to two times a week beginning at 36 weeks
4. Another ultrasound at 38 weeks of gestation to estimate fetal weight, reevaluate cardiac morphology, and assist with delivery plans

How common is diabetic ketoacidosis (DKA) in women with pregnancy-related diabetes and what is the typical presentation?

It is found in 5–10% of all pregnancies complicated with pregestational diabetes mellitus. It is more common in Type I pregestational diabetes mellitus. The typical presentation includes abdominal pain, nausea and vomiting, altered sensorium, low arterial pH, low serum bicarbonate, serum and urine ketones, and increased anion gap. Recurrent late decelerations may be seen on fetal heart monitoring (improves when maternal ketoacidosis is corrected) and are signs of fetal distress (acidemia)

How is DKA managed in the pregnant woman?

The same as in nonpregnant women. Aggressive hydration and IV insulin is mandatory. Glucose, potassium, and bicarbonate levels should be monitored closely and replenished

appropriately. Admission to the hospital is mandatory and establishment of fetal well-being is crucial

When is cesarean delivery indicated for women with gestational diabetes? If the expected fetal weight is greater than 4500 g, to prevent birth trauma from shoulder dystocia

Thyroid in Pregnancy

What are the two major changes in thyroid function during pregnancy? An increase in serum thyroxine-binding globulin (TBG) concentrations and stimulation of the thyrotropin (TSH) receptor

What two hormones have thyroid-stimulating activity? Estrogen (increases serum TBG concentration); hCG

How do thyroid function test results change in normal pregnancy, and in hyperthyroid and hypothyroid states? See Table below

Maternal Status	TSH	FT ₄	TT ₄	TT ₃	RT ₃ U
Normal	NC	NC	↑	↑	↓
Hyperthyroid	↓	↑	↑	↑ or NC	↑
Hypothyroid	↑	↓	↓	↓ or NC	↓

TSH: thyroid stimulating hormone; FT₄: free thyroxine; FTI: free thyroxine index; TT₄: total thyroxine; TT₃: total triiodothyronine; RT₃U: resin T3 uptake; NC: no change

Which of the thyroid-related hormones does *not* cross the placenta? TSH

Which thyroid hormones or thyroid-related molecules can cross the placenta? Thyroid hormone, T₃, T₄, TRH, iodine, TSH receptor immunoglobulins

Hyperthyroidism

What are the clinical manifestations of hyperthyroidism? Nervousness, tachycardia, palpitations, hypertension, weight loss, tremors, flushing, frequent bowel movements, excessive sweating, and insomnia

What are several pregnancy-related complications associated with poorly controlled hyperthyroidism?

Spontaneous abortion or stillbirth
 Preeclampsia
 Preterm delivery
 Placental abruption
 Cardiac arrhythmias, congestive heart failure
 Low birth weight
 Thyroid storm
 Hyperemesis gravidarum

What are several etiologies of hyperthyroidism?

Graves' disease (90%)
 Toxic nodular goiters
 Iatrogenic
 Iodine induced
 Subacute thyroiditis
 hCG-mediated

What three syndromes are associated with hCG-mediated hyperthyroidism?

See Table below

Syndrome	Description	Treatment
Transient subclinical hyperthyroidism	It occurs in 10–20% of normal pregnant women during the period of highest serum hCG concentrations.	None needed
Hyperemesis gravidarum	It is a syndrome that is characterized by nausea and vomiting with weight loss of more than 5% during early pregnancy. Women may have either subclinical or mild overt hyperthyroidism.	Includes IV fluids, IV vitamins, NPO to Clears, IV anti-nauseam, thiamine, and tx. of hyperthyroidism
Trophoblastic hyperthyroidism	It occurs in about 60% of women with a hydatidiform mole or choriocarcinoma	Removal of the mole or therapy directed against the choriocarcinoma (IV/IM methotrexate + repeat β -hCG until it trends to 0

What additional clinical manifestations suggest Graves' disease?

Exophthalmos; goiter; pretibial myxedema

How is hyperthyroidism diagnosed?

Elevated levels of serum FT₄ or elevated FTI, and low serum TSH (<0.01 mU/L)

What is the management and treatment for pregnant women with hyperthyroidism?

The goal of treatment is to maintain the mother's serum free T₄ concentration in the high-normal range for nonpregnant women using the lowest drug dose

1. **Radioiodine is absolutely contraindicated**
2. **Iodine is contraindicated as it can cause fetal goiter**
3. Propylthiouracil (PTU) 50 mg bid or less is recommended for treatment of moderate to severe hyperthyroidism-complicating pregnancy. If treatment fails, consider methimazole
4. Beta blockers may be given to ameliorate the symptoms of moderate to severe hyperthyroidism in pregnant women (low-dose atenolol may be appropriate to begin)
5. Thyroidectomy during pregnancy may be necessary in women who cannot tolerate thionamides because of allergy or agranulocytosis (preferably during second trimester)

What percent of neonates born to women with Graves' disease have hyperthyroidism because of transplacental transfer of TSH receptor-stimulating antibodies?

1–5%

What are the clinical manifestations of fetal hyperthyroidism?

Fetal tachycardia (>160 beats/min)

Fetal goiter

Advanced bone age

Poor growth

Craniosynostosis

Cardiac failure

Fetal hydrops

Though rare, what are several manifestations of fetal and neonatal thyrotoxicosis?

Fetal tachycardia and intrauterine growth restriction are the most common signs

What is a thyroid storm?

It is an acute, life-threatening medical emergency characterized by a high metabolic state in patients with thyrotoxicosis

What is a major consequence of thyroid storm?

Maternal heart failure

What are the clinical signs and symptoms of a thyroid storm?

Fever, tachycardia out of proportion to the fever, changed mental status, confusion, nervousness, nausea and vomiting, seizures, diarrhea, and cardiac arrhythmias

(It can be initiated by infections, stress, surgery, labor, and/or delivery)

How is a thyroid storm treated?

Transfer to the ICU

PTU, 600–800 mg PO STAT, followed by 150–200 mg PO for 4–6 hours

IV sodium iodine or 2–5 drops of supersaturated solution of potassium iodide is given PO 1 hour after PTU

IV or IM dexamethasone

Propranolol IV or PO

Phenobarbital for restlessness (if needed)

Supportive measures (O₂, antipyretics, cooling blankets, IV hydration)

Hypothyroidism

How often do hypothyroidism-complicating pregnancies occur?

Hypothyroidism-complicating pregnancy is rare (1:1000–1:1600 deliveries) because many women with hypothyroidism are anovulatory and infertile, and, in addition, the rate of first-trimester miscarriages is high

What are the clinical manifestations of maternal hypothyroidism?

Fatigue, constipation, intolerance to cold, dry skin, muscle cramps, hair loss, weight gain, myxedema, carpal

What are the most common etiologies of hypothyroidism in pregnant or postpartum women?

tunnel syndrome, and prolonged relaxation of deep tendon reflexes

Hashimoto disease (most common in developed countries)

Iron deficiency (worldwide)

Subacute thyroiditis

Thyroidectomy

Radioactive iodine treatment

Type I diabetes mellitus

What other endocrine disease is hypothyroidism associated with?

What are the several pregnancy-related complications associated with hypothyroidism?

Preeclampsia

Preterm delivery

Low-birth weight

Placental abruption

Postpartum hemorrhage

What is the management and treatment for pregnant women with hypothyroidism?

Levothyroxine. TSH levels should be checked 4 weeks later. Levothyroxine doses should be adjusted at 4-week intervals until the TSH level is stable

What is a significant impact of maternal hypothyroidism on the fetus and/or neonate?

Congenital cretinism

Mental retardation

Intrauterine growth restriction

Small for gestational age

Growth failure, mental retardation, floppy baby, macroglossia, other neuropsychologic deficits

What are the signs of congenital cretinism?

1:4000 births and only 5% of neonates are identified by clinical symptoms at birth

How common is congenital hypothyroidism in neonates?

75% of hypothyroid infants have some form of thyroid agenesis

What is the common cause of congenital hypothyroidism in neonates?

Lethargy and slow movement

Hoarse cry

Feeding problems

Constipation

Macroglossia

Umbilical hernia

Large fontanelles

Hypotonia

What are the clinical manifestations of hypothyroidism in neonates?

Dry skin
Hypothermia
Prolonged jaundice

Which neonates should be screened for hypothyroidism?

Screening of all newborns is now routine in all 50 states of the United States

Other Thyroid Disorders

How common is postpartum thyroiditis?

It occurs in 5–10% of women during the first year of childbirth or pregnancy loss. It is directly related to increasing serum levels of thyroid autoantibodies

What are the clinical phases and characteristics of postpartum thyroiditis?

Phase 1 is characterized by thyrotoxicosis-like symptoms (small, painless goiter; fatigue; palpitations) between 1 and 4 months after delivery. Some women return back to a euthyroid state, while others go into **Phase 2—the development of transient hypothyroidism or permanent hypothyroidism** (4–8 months postpartum). See Table below.

Phases and Characteristics of Postpartum Thyroiditis

Clinical Phase	Onset	Incidence	Mechanism	Symptoms	Treatment	Sequelae
Thyrotoxicosis	1–4 months postpartum	4%	Destruction-induced hormone release	Painless goiter, fatigue, palpitations	β -blocker for symptoms	Return to euthyroid state or develop hypothyroidism
Hypothyroidism	4–8 months postpartum	2–5%	Thyroid insufficiency	Goiter, fatigue, depression	Thyroxine for 6–12 months	Transient or permanent hypothyroidism

What laboratory results help make the diagnosis of postpartum thyroiditis?

New-onset abnormal values of TSH and T4 may be present. The presence of antimicrosomal and/or

<p>Postpartum thyroid dysfunction is seen 25% of the time with what other endocrine disorder?</p> <p>How is the presence of a thyroid nodule or thyroid cancer assessed and managed during pregnancy?</p>	<p>thyroperoxidase—antithyroid peroxidase antibodies confirm the diagnosis</p> <p>Type I diabetes mellitus</p> <p>The same as in a nonpregnant woman. Thyroid radionuclide scanning is contraindicated, and fine-needle-aspiration biopsy of the nodule should be done. Benign nodules are followed; if required, surgery is best performed in the second trimester</p>
---	---

Other Endocrine Disorders of Pregnancy

<p>What is Sheehan syndrome?</p> <p>What are the characteristic signs and symptoms of Sheehan syndrome?</p>	<p>It is partial or complete pituitary insufficiency because of postpartum necrosis of the anterior pituitary gland following severe intrapartum or early postpartum hemorrhage</p> <p>Failure of lactation, amenorrhea, breast atrophy, loss of pubic and axillary hair, hypothyroidism, and adrenal cortical insufficiency</p>
---	--

GASTROINTESTINAL DISORDERS

Acute Fatty Liver of Pregnancy

<p>What is acute fatty liver of pregnancy (AFLP)?</p> <p>In which trimester does this disease typically appear?</p> <p>Deficiency of which enzyme has been associated in the pathogenesis of acute fatty liver of pregnancy?</p> <p>What are the common clinical manifestations of this disease?</p>	<p>It is a rare complication of pregnancy (1:5000 to 16,000) characterized by microvesicular fatty infiltration of hepatocytes</p> <p>Third trimester</p> <p>Long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD)</p> <p>Nausea and vomiting (most frequent symptoms; indicative of acute fatty liver of pregnancy if onset in third trimester)</p>
--	--

	Abdominal pain (commonly epigastric)
	Malaise
	Anorexia
	Jaundice
What laboratory findings may be seen in patients with acute fatty liver of pregnancy?	Elevated AST and ALT
	Elevated bilirubin
	Prolonged prothrombin time
	Elevated liver enzyme levels
	Low platelets and fibrinogen
	Elevated serum creatinine
	Low glucose levels (30% of patients)
What other major disorder with similar laboratory results must be ruled out and how is this condition characterized?	HELLP syndrome. It is characterized by hemolysis, elevated liver enzymes, and low platelets
What is the gold standard for diagnosis of acute fatty liver of pregnancy?	Liver biopsy revealing microvesicular fatty infiltration of hepatocytes. However, this is rarely performed since it is an invasive procedure
What is the management and treatment?	Maternal stabilization (fluids, blood products, antibiotics) Supportive care (mechanical ventilation, dialysis) Prompt delivery of the fetus
What test should be considered for newborns of mothers with acute fatty liver of pregnancy?	Deficiency of LCHAD
What is the rate of recurrence?	It can recur in future pregnancies but the rate is unclear

Intrahepatic Cholestasis of Pregnancy

What is intrahepatic cholestasis of pregnancy (ICP)?	It is a condition characterized by jaundice and pruritis secondary to the accumulation of bile acids in the liver and plasma
In what trimesters does it usually occur?	Onset is more common in the third trimester, but sometimes occurs in the second
What genetic factors may be involved in the pathogenesis of ICP?	Defects in either the ABCB4 (adenosine triphosphate-binding cassette, subfamily 4, member 4)

<p>What hormonal factors may be involved in the pathogenesis of ICP?</p>	<p>gene or multidrug resistance 3 (MDR3) gene (encoding for a canalicular phospholipid translocator) may be involved in ICP</p> <p>High concentrations of estrogen and excess progesterone may be risk factors for ICP</p>
<p>What is the cardinal clinical manifestation of ICP that helps distinguish this disease from other liver conditions?</p>	<p>Severe pruritis (especially on the palms and soles of the feet) (Jaundice is present 10% of the time; jaundice without pruritis warrants other causes of liver disease.)</p>
<p>What do laboratory values reveal in patients with ICP?</p>	<p>Increased serum total bile acids (chenodeoxycholic acid, deoxycholic acid, cholic acid)</p> <p>Marked elevation of the cholic/chenodeoxycholic acid ratio</p> <p>Elevated alkaline phosphatase</p> <p>Elevated total and direct bilirubin</p> <p>Elevated AST and ALT</p> <p>Normal prothrombin time (usually)</p>
<p>What are the main liver conditions that must be ruled out?</p>	<p>Hepatitis (autoimmune and viral)</p> <p>Biliary tract disease</p> <p>Acute fatty liver of pregnancy</p> <p>HELLP syndrome</p>
<p>What is the treatment of ICP?</p>	<p>Treatment is focused on relieving symptoms and preventing maternal and fetal complications.</p> <p>Ursodeoxycholic acid (500 mg bid until delivery) has been shown to alleviate pruritis and normalize bile acids and improve liver function test results. Early delivery (36–38 weeks) improves symptoms in majority of patients suffering from ICP</p>
<p>Can oral contraceptives that contain estrogen be given postpartum?</p>	<p>Yes. Oral contraceptives containing low-dose estrogen can be given after normalization of liver function tests. Women should be advised of a potential recurrence of pruritis</p>
<p>What are several fetal complications of ICP?</p>	<p>Fetal death</p> <p>Spontaneous preterm birth</p> <p>Postpartum hemorrhage</p>

	Neonatal respiratory syndrome
	Meconium-stained amniotic fluid
What is the rate of recurrence?	It recurs 60–70% in future pregnancies, but may be milder in severity

Hyperemesis Gravidarum

What is hyperemesis gravidarum?	It is persistent vomiting typically in the first trimester that is severe enough to cause weight loss, dehydration, acidosis from starvation, alkalosis from vomiting, and hypokalemia
What hormones may be associated with hyperemesis?	It may be related to high or rapidly rising levels of serum estrogen or hCG, or both
What abnormalities are seen on laboratory tests?	Elevated levels of hCG or serum thyroxine Elevated levels of serum transaminases (typically <200 IU/L, and ALT > AST) Elevated levels of bilirubin, amylase, lipase, and electrolytes
What is the association between hyperthyroidism and hyperemesis gravidarum?	Although there are biochemical signs of hyperthyroidism (elevated thyroxine levels), this is most likely the effect of hCG on the TSH receptor (seen in 60–70% of patients). Clinical symptoms of hyperthyroidism during hyperemesis gravidarum are not seen
How is hyperemesis gravidarum treated?	First-line pharmacotherapy consists of vitamin B₆ or vitamin B₆ plus doxylamine. Vitamin B ₁ should also be administered to prevent Wernicke encephalopathy. Antiemetics, IV crystalloids, and IV fluids should be given until the vomiting is controlled
What are several life-threatening complications of hyperemesis gravidarum?	Mallory-Weiss tears Esophageal rupture Pneumothoraces Pneumomediastinum

What two vitamin deficiencies are a result of prolonged vomiting and can have severe consequences?

Vitamin B₁ (thiamine) deficiency can cause Wernicke encephalopathy
Vitamin K deficiency can cause coagulopathy with epistaxis

What is the effect of hyperemesis gravidarum on the fetus?

IUGR and fetal death may occur in the setting of persistent vomiting and maternal weight loss

What should mothers be advised regarding recurrence of hyperemesis gravidarum?

It often recurs in subsequent pregnancies

RENAL AND URINARY TRACT DISORDERS

Urinary Tract Infections

UTI is the most common medical complication of pregnancy.

Why is pregnancy considered a high-risk condition with asymptomatic bacteriuria?

Both hormonal and mechanical changes predispose the pregnant woman with asymptomatic bacteriuria to develop acute pyelonephritis, which is associated with preterm birth and perinatal death. Pyelonephritis in pregnancy will lead to septicemia in 10–20% and ARDS in 2% of cases

How prevalent is asymptomatic bacteriuria in pregnancy?

It is estimated to occur in 4–7% of pregnant patients. If left untreated, up to 40% of cases will progress to pyelonephritis

How is this condition detected?

Screening for asymptomatic bacteriuria using urine culture is recommended at the first prenatal visit

How is asymptomatic bacteriuria treated?

Any FDA category B drug such as **cephalosporins, nitrofurantoin, or trimethoprim-sulfamethoxazole** can be used. Quinolones (FDA category C) are generally not used during pregnancy. Seven-day courses are recommended, along with a follow-up culture to document sterile urine. Persistent bacteriuria should be treated based on sensitivities. Suppressives antibiotics (most commonly nitrofurantoin) should then be considered in these patients. See the following table.

Antimicrobial Used for Treatment of Pregnant Women with Asymptomatic Bacteriuria

7-Day Course (Recommended)	3-Day Course
Amoxicillin, 3 g	Amoxicillin, 500 mg tid
Ampicillin, 2 g	Ampicillin, 250 mg qid
Cephalosporin, 2 g	Cephalosporin, 250 mg qid
Nitrofurantoin, 200 mg	Ciprofloxacin, 250 mg bid
Trimethoprim-sulfamethoxazole, 320/1600 mg	Levofloxacin, 250 mg daily
	Nitrofurantoin, 100 mg bid

How is symptomatic cystitis treated in pregnancy?

Treatment and follow-up are similar to asymptomatic bacteriuria. Acute pyelonephritis should be treated with IV antibiotics and hospitalization. Suppressive antibiotics should be given following treatment to any pregnant patient treated for acute pyelonephritis

NEUROLOGIC DISORDERS

Epilepsy and Seizure Disorders

Seizure disorders are the most frequent major neurologic complications encountered in pregnancy and more than 95% of patients who have seizures during pregnancy have a history of epilepsy or have been receiving anticonvulsant therapy.

What are the two major pregnancy-related threats to women with epilepsy?

Increase in seizure frequency
Increased risk of congenital malformations in the fetus

Increased seizure frequency is associated with subtherapeutic anticonvulsant levels and/or a lower seizure threshold. What are the several factors that cause these characteristics?

Pregnancy-related changes such as nausea and vomiting, decreased gastrointestinal motility, increased intravascular volume, increased drug metabolism from induction of hepatic and placental enzymes, antacid use (reduces drug absorption because of abnormal protein binding), and increased glomerular filtration rate decrease anticonvulsant concentrations

Decreased seizure threshold can be affected by sleep deprivation, and hyperventilation and pain during labor

What are the maternal and fetal side effects for the following traditional anticonvulsant medications?

See the table below.

Anticonvulsant	Maternal Side Effects	Fetal Side Effects
Phenytoin	Nystagmus, ataxia, hirsutism, gingival hyperplasia, megaloblastic anemia	Fetal hydantoin syndrome: craniofacial anomalies (upturned nose, mild midfacial hypoplasia, thin philtrum, facial clefts), fingernail hypoplasia, growth deficiency, developmental delay, cardiac defects
Carbamazepine	Drowsiness, leukopenia, ataxia, mild hepatotoxicity	Fetal hydantoin syndrome, spina bifida, fingernail hypoplasia, IUGR
Valproate	Ataxia, drowsiness, alopecia, hepatotoxicity, thrombocytopenia	Neural-tube defects, heart and kidney malformations, hypospadias, polydactyly
Phenobarbital	Drowsiness, ataxia	Clefts, cardiac anomalies, urinary tract malformations
Primidone	Drowsiness, ataxia, nausea	Possible teratogenesis, coagulopathy, neonatal depression

What is the association between anticonvulsants and folic acid deficiency?

All anticonvulsants interfere with folic acid metabolism and patients on anticonvulsants may become folic acid deficient and develop macrocytic anemia. Folic acid deficiency has been associated with neural tube defects and other congenital malformations. Folic acid supplementation (4 mg/day) should be begun before pregnancy if possible

What are the recommendations regarding management and therapy with anticonvulsants?

1. Women with epilepsy should have preconception counseling regarding the optimal anticonvulsant during pregnancy, a switch to the least teratogenic drug and the least

number of medications prescribed, and the lowest dose needed

2. Blood levels of anticonvulsants should be measured every trimester and prior to delivery to maintain a therapeutic range
3. Patients should be screened for neural tube defects and other fetal anomalies associated with anticonvulsants

What is the effect of anticonvulsants on vitamin K levels?

Many of the anticonvulsants, particularly phenytoin, induce a deficiency of vitamin K-dependent clotting factors (II, VI, IX, X). This places the patient and her fetus at risk for hemorrhage. The patient should receive vitamin K supplementation (10 mg/day) from week 36 until delivery, and the newborn should also receive an IM injection of vitamin K after delivery

HEMATOLOGIC DISORDERS

Thromboembolism

All the elements of Virchow triad, venous stasis, vascular damage, and hypercoagulability are present during venous thrombosis. How does pregnancy increase the risk for venous thromboembolism?

Increased venous capacity, relaxation of vascular smooth muscle, and compression of the pelvic veins by the gravid uterus cause venous stasis. Increased risk for endothelial damage occurs during delivery, especially during cesarean delivery. Lastly, estrogen stimulation of coagulation factors and a decrease in fibrinolytic activity favors coagulation during pregnancy

Thrombosis of the superficial veins of the saphenous system can occur during pregnancy. What are the clinical manifestations of patients with superficial thrombophlebitis?

Tenderness, pain, or erythema along the vein

What diagnostic test should be ordered?

In order to rule out DVT, a compression **ultrasound** of the affected extremity should be performed

What is the treatment for superficial thrombophlebitis?

Treatment consists of compression stockings, ambulation, leg elevation, and analgesics

Does deep venous thrombosis (DVT) commonly occur in the left or right lower extremity?

Greater than 80% of DVTs occurs in the **left lower extremity**. This is because of compression of the left iliac vein by the right iliac vein as it branches off the aorta

What are the clinical features of DVTs?

Although the presentation is variable, signs include **lower extremity tenderness, erythema, swelling, and a palpable cord**. Homan sign may be present (pain on passive dorsiflexion of the foot).

How is DVT diagnosed?

Doppler ultrasound is the gold standard

What is the treatment for DVT that occurs during pregnancy?

Initial anticoagulation with IV unfractionated or low-molecular weight heparin for 5–7 days followed by subcutaneous adjusted-dose low-molecular weight heparin therapy for the remainder of the pregnancy. Warfarin should be started postpartum for 6–18 weeks. Bed rest, analgesia, and ambulation with compression stockings should be initiated

What effects does Coumadin have on the fetus, thus making it a contraindication for DVTs during pregnancy?

Fetal warfarin syndrome (FWS) is possible. This is characterized by hemorrhage and embryopathy, which includes ventral midline dysplasia, nasal hypoplasia, CNS abnormalities, cardiac and renal anomalies

What is a major complication of DVTs?

If untreated, 24% of DVTs will result in **pulmonary embolus**. This can occur in 0.5–3.0 per 1000 pregnancies

What are the clinical manifestations of pulmonary embolism (PE)?

Dyspnea, pleuritic chest pain, cough, syncope, hemoptysis, tachypnea, and tachycardia

How is a pulmonary embolism evaluated and diagnosed in the pregnant woman?

It usually consists of an ABG, chest x-ray, EKG, spiral CT pulmonary angiography, V/Q scan

What is the treatment of pulmonary embolism?

Anticoagulation with low molecular weight heparin for at least 4–6 months, including up to

	6 weeks postpartum. Vena caval filter should be considered in those with recurrent embolization
What is septic pelvic thrombophlebitis?	It is thrombosis in the veins of the pelvis because of infection. About 90% of cases occur after a cesarean delivery; however, the incidence is low, affecting 1:2000 pregnancies. It is a diagnosis of exclusion for postpartum fever
What is the most common vein at risk for septic thrombosis?	The pelvic veins (most commonly the ovarian veins)
When should septic pelvic thrombophlebitis be suspected?	Continuous and wide-swinging fevers (from normal to 105.8°F) in the puerperium and which do not respond to antibiotics
How is it diagnosed and treated?	A pelvic examination is often not helpful for diagnosis. A chest x-ray may reveal multiple, small septic emboli. CT and MRI should be considered for diagnosis. Treatment includes a combination of antibiotics and heparin
What are the most significant complications of septic pelvic thrombophlebitis?	Septic pulmonary emboli Extension of the venous clot in the pelvis Renal vein thrombosis Ureteral obstruction Death

Thrombophilias

What are the most common inherited thrombophilias?	Factor V Leiden mutation Antithrombin deficiency Prothrombin G20210 mutation Protein C deficiency Protein S deficiency Hyperhomocysteinemia Methylene-tetra-hydro-folate-reductase (MTHFR)
What is antiphospholipid syndrome?	It is an autoimmune disorder characterized by the presence of certain clinical features, including venous and arterial

What are the two most well-characterized antiphospholipid antibodies?

thromboembolism, and the presence of specified levels of circulating antiphospholipid antibodies

What are the most common medical problems associated with antiphospholipid syndrome?

Lupus anticoagulant and anticardiolipin antibodies

What are several obstetrical complications associated with antiphospholipid syndrome?

The most common and most serious medical problems are venous and arterial thromboses, most commonly in the lower extremities.

Autoimmune thrombocytopenia occurs 40–50% of the time

Recurrent spontaneous abortions (most common)

Preeclampsia and eclampsia

Intrauterine growth restriction

Uteroplacental insufficiency

Preterm delivery

Placental abruption

Infertility

Intrauterine fetal demise

Who should be tested for antiphospholipid syndrome?

Those who have a complicated obstetrical history characterized by recurrent spontaneous abortions or stillbirths, or preterm delivery resulting from eclampsia, preeclampsia, or uteroplacental insufficiency. Those who have a history of unexplained venous or arterial thromboses should also be evaluated for antiphospholipid syndrome

Anemias and Hemoglobinopathies

What is physiologic anemia of pregnancy?

During the course of pregnancy, there is an expansion in plasma volume greater than that of the RBC mass. This reflects in a decrease in hematocrit during pregnancy; however, it is not a true anemia

How is true anemia in pregnancy defined?

It is generally defined as an Hct <30% or a hemoglobin <10 g/dL

What are the effects of maternal anemia on pregnancy?

Several associations with maternal anemia have been found, including preterm birth and fetal growth restriction

What are the two most common causes of anemia during pregnancy?

What red cell indices, laboratory results, and characteristics on a peripheral blood smear indicate an iron deficiency anemia? What is the treatment for this anemia?

What is the most common cause of macrocytic anemia in pregnancy? What red cell indices, laboratory results, and characteristics on a peripheral blood smear indicate this anemia?

What is the recommended folate level in pregnant women and what is the treatment for folate deficiency anemia?

What is the significance of folic acid deficiency on the fetus?

What are examples of hereditary hemolytic anemias?

What are the most common hemoglobinopathies?

What other infections are patients with sickle-cell hemoglobinopathies at an increased risk for?

What is a significant pulmonary complication in patients with sickle-cell disease?

Iron deficiency anemia and anemia from acute blood loss

Red cell indices include a mean cell volume (MCV) <80 f/L, and mean corpuscular hemoglobin concentration (MCHC) $<30\%$. Serum iron is decreased (<50 mg/dL), total iron-binding capacity (TIBC) is increased, and serum ferritin is decreased (a level <15 ug/L is confirmatory of iron deficiency anemia). The classic findings on a blood smear include small, pale erythrocytes (microcytic and hypochromic). Treatment is iron therapy consisting of ferrous sulfate 325 mg bid PO

Folate acid deficiency. Red cell indices reveal an MCV >80 f/L. Serum folate levels <4 ng/mL and erythrocyte folate activity <20 ng/mL are diagnostic. A smear of peripheral blood demonstrates macrocytes, hypersegmentation of neutrophils, and peripheral nucleated erythrocytes

A recommended level of folate during pregnancy is 400 ug/day and treatment is 1 mg of folic acid PO once daily

The development of neural tube defects

Hereditary spherocytosis, glucose 6-phosphate dehydrogenase deficiency (G6PD), pyruvate-kinase deficiency

Sickle-cell anemia (SS disease), sickle-cell hemoglobin C disease (SC disease), sickle-cell α -thalassemia disease, β -thalassemia, and $\alpha\beta$ -thalassemia

Urinary tract infections. Diagnosis should be established in first trimester

Acute chest syndrome. It is characterized by pleuritic chest pain, fever, cough, lung infiltrates, and hypoxia which all lead to hypoxemia and acidosis. Pathology of this complication includes infection,

Which thalassemia is associated with fetal hydrops, intrauterine death, and preeclampsia?

vaso-occlusion and infarction, pulmonary sequestration, and fat embolization from bone marrow

Hb Bart (α -thalassemia major, absence of both α -globin chains)

PULMONARY DISORDERS

Asthma

What percentage of women experience worsening of their asthma during pregnancy?

40%, while another 40% of women experience no change and 20% of them report improvement of their asthma

What are the potential maternal complications of an acute exacerbation of asthma?

Pneumonia, hyperemesis gravidarum, preeclampsia, vaginal bleeding, complicated labor, and cesarean delivery

What are fetal complications of an acute exacerbation of asthma?

Intrauterine growth restriction, low birth weight, preterm birth, neonatal hypoxia, and increased overall perinatal mortality

How is acute asthma managed in a pregnant woman?

It is similar to that of a nonpregnant woman

IV fluids and supplemental O₂ (keep O₂ saturation >95%)

α -agonists and high-dose steroids (IV prednisone 40–60 mg q6h)

Continuous pulse oximetry and electronic fetal monitoring

Which vaccine is recommended for all pregnant women, especially those with asthma?

Influenza vaccination

What medications can be used to treat pregnant asthmatics?

Beta-2 agonists, terbutaline SQ, theophylline, corticosteroids

Which medications should be avoided in pregnant asthmatics during labor and delivery?

Analgesics, which cause histamine release, respiratory depression, and bronchospasm, should be avoided. Prostaglandin F₂ can cause bronchospasm

Aspiration Pneumonitis

Why are pregnant women at risk for aspiration of gastric contents?

Elevated intra-abdominal pressure, decreased gastroesophageal

How common are maternal deaths related to aspiration?	sphincter tone, delayed gastric emptying, and diminished laryngeal reflexes during pregnancy increases the risk of aspiration
What are symptoms and signs of aspiration?	It accounts for 30–50% of maternal deaths related to anesthetic complications
	Dyspnea, bronchospasm, tachycardia, cyanosis, hypoxia, hypercapnea, and acidemia. Respiratory arrest is possible

CARDIOVASCULAR DISORDERS

Heart disease complicates approximately 1% of all pregnancies. Pregnant patients with functional Class III and IV heart disease (see the table below for the New York Heart Association classification of heart disease) have high event rates and succumb to heart failure, arrhythmias, and stroke.

Which heart diseases are associated with the highest maternal morbidity during pregnancy?	Patients with septal defects, PDA, and mild mitral and aortic valvular disorders are often in classes I and II and have minimal complications during pregnancy. Primary pulmonary hypertension, uncorrected tetralogy of Fallot, Marfan syndrome, and Eisenmenger syndrome are associated with a worse prognosis, and patients are advised against becoming pregnant
--	--

New York Heart Association Functional Classification of Heart Disease

Class	Description
Class I	No signs or symptoms of cardiac decompensation
Class II	No symptoms at rest; minor limitation with mild to moderate activity
Class III	No symptoms at rest; marked limitation with less than ordinary activity
Class IV	Symptoms at rest; increased discomfort with any physical activity

What are the signs and symptoms indicative of significant cardiovascular disease?

See the table below.

Signs and Symptoms Indicative of Significant Cardiovascular Disease

Symptoms	Progressively worsening shortness of breath Cough with frothy pink sputum Paroxysmal nocturnal dyspnea Chest pain with exertion Syncope preceded by palpitations or exertion
Physical examination	Hemoptysis Abnormal venous pulsations Rarely audible S ₁ Single S ₂ or paradoxically split S ₂ Loud systolic murmurs, any diastolic murmur Ejection clicks, late systolic clicks, opening snaps Friction rub Sustained right or left ventricular heave Cyanosis or clubbing
Electrocardiogram	Significant arrhythmias Heart blocks
Chest radiograph	Cardiomegaly Pulmonary edema

Reprinted, with permission, from DeCherney AH et al: *Current Obstetrics & Gynecologic Diagnosis Treatment*, 10th ed. New York: McGraw-Hill, 2007:362.

What are the potential risks to the fetus of patients with functionally significant cardiac disease?

They are at risk for low-birth weight and prematurity. Congenital heart disease in the fetus is more likely to occur in patients with congenital heart disease (1–5%).

For the following heart diseases, what are their pregnancy-related complications, associated signs and symptoms, and treatment modalities?

See the table on the following page.

Table 9-3 Common Heart Diseases Occuring During Pregnancy

Heart Disease	Signs/Symptoms during		Treatment	Comment
	Decompensation	Complications		
Mitral regurgitation	Chest pain, palpitations, tachycardia, anxiety, late systolic murmur with a late systolic click	Generally, pregnancy is well-tolerated and unaffected	β -Blockers (propranolol) may aid in control of associated symptoms	May occur in as many as 5% of pregnancies
Mitral stenosis	Dyspnea on exertion and at rest, hemoptysis, loud S ₁ , an opening snap, low diastolic rumble at the apex, right ventricular lift	Pulmonary hypertension, right heart failure, hemoptysis, low cardiac output, atrial fibrillation, CHF during labor	Medical management is first line: digitalis, quinidine, β -adrenergic blocking agents, anticoagulation with heparin. Severe mitral stenosis may warrant mitral valvotomy, mitral valve replacement, or balloon valvuloplasty	Most common valvular lesion found in pregnancy. Mitral stenosis with a-fibrillation has a high likelihood of congestive failure
Rheumatic heart disease	Dependent on the affected valve	Patients are at high risk for thromboembolic disease, SBE, cardiac failure, and pulmonary edema; fetal loss is also more common	Corrective surgical procedures, such as balloon valvuloplasty, surgical commissurotomy, and valve replacement	90% of rheumatic heart disease patients have mitral stenosis
Peripartum cardiomyopathy	Dyspnea; other frequent symptoms include cough, orthopnea, paroxysmal nocturnal	Heart failure and death are potential complications	Combination of digoxin, diuretics and sodium, β -blockers, and after-load reducers.	Usually affects women in late pregnancy (>36 weeks) or early puerperium

(Continued)

Common Heart Diseases Occuring During Pregnancy (*Continued*)

Heart Disease	Signs/Symptoms during Decompensation	Complications	Treatment	Comment
	dyspnea, and hemoptysis		Anticoagulants, such as heparin should be considered	(in first 5 months). Etiology is unknown, though myocarditis is responsible sometimes
Marfan syndrome	Dyspnea, chest pain, aortic diastolic murmur, and midsystolic click	Aortic aneurysm and possible rupture	Surgical intervention	25–50% of maternal mortality and 50% chance that offspring will inherit the disease

Describe the classic patient at increased risk for peripartum cardiomyopathy

A black, multiparous woman who is over the age of 30, and who has had a multifetal gestation in the past and/or preeclampsia

Which cardiac medications are considered safe during pregnancy? Which are contraindicated during pregnancy?

Acceptable cardiac medications: digoxin, loop diuretics, thiazides, hydralazine, β -adrenergic blockers, calcium-channel blockers

Contraindicated: nitrates, angiotensin-converting enzyme inhibitors, and angiotensin II receptor blockers

ACUTE ABDOMEN AND ABDOMINAL TRAUMA

List the differential diagnoses for the pregnant woman who presents with acute lower abdominal pain

Gynecologic etiologies: ruptured corpus luteum cyst; adnexal torsion; ectopic pregnancy; abruptio placentae; early labor; salpingitis

Nongynecologic etiologies: pyelonephritis; cholecystitis; diverticulitis

What is the most consistent clinical symptom encountered in pregnant women with appendicitis?

Signs and symptoms of acute appendicitis are not dramatic in the pregnant woman and also not as

	<p>specific compared to the nonpregnant woman. In pregnancy, the appendix is commonly upwardly displaced. Vague pain on the right side of the abdomen is the most consistent clinical symptom of acute appendicitis in the pregnant woman, and there should be a high index of suspicion for appendicitis for any abdominal pain</p>
<p>What is the first-line imaging modality in pregnant women with suspected appendicitis?</p>	<p>MRI. an appendiceal CT may be used if an MRI is not feasible; it is also more accurate than a graded compression ultrasonography</p>
<p>What are potential consequences of a ruptured appendix?</p>	<p>Preterm labor, maternal and fetal sepsis which may lead to fetal neurologic injury, and spontaneous abortion/fetal demise</p>
<p>What is the treatment for nonperforated acute appendicitis in the pregnant woman?</p>	<p>Appendectomy is the gold standard. Laparoscopic appendicitis is being used more frequently, especially in the first half of the pregnancy</p>
<p>How does pregnancy lead to more gallstones in pregnant women?</p>	<p>Increased estrogens cause an increase in cholesterol saturation, which, in addition to biliary stasis and decreased gallbladder contraction, lead to more gallstones</p>
<p>What percentage of pregnancies is complicated by trauma?</p>	<p>7%. Motor vehicle accidents account for 40%, falls account for 30%, direct assaults to the maternal abdomen (20%), and others (10%)</p>
<p>What is the most common nonobstetric cause of maternal death during pregnancy?</p>	<p>Automobile accidents</p>
<p>What is the most common cause of fetal death?</p>	<p>Death to the mother</p>
<p>What is the second most common cause of fetal death?</p>	<p>Abruptio placentae</p>
<p>What is the management of pregnant women who experienced abdominal trauma?</p>	<p>Admit for observation for at least 24 hours Monitor mother; an fetal well-being should be obtained (nonstress test, BPP, FHR monitor) If bleeding is present, obtain Rh status by using the Kleihauer-Betke test. For the woman who is</p>

D-negative, administration of anti-D immunoglobulin should be considered

Monitor for contractions

Obstetric Infections

TORCH INFECTIONS

Introduction

What are the TORCH infections?

A group of nonbacterial perinatal infections that lead to significant neonatal morbidity and mortality

T: *Toxoplasma gondii*

O: **Other** (such as syphilis, VZV, HIV, hepatitis, parvovirus B19)

R: **Rubella**

C: **Cytomegalovirus**

H: **Herpes simplex virus**

How does the fetus become infected with the TORCH diseases?

Usually through **transplacental migration of the infection**; however, infection can also occur via ascending chorioamnionitis, maternal exposure intrapartum, external contamination intrapartum, or nosocomial neonatal exposure

What modalities can be used to diagnose these infections?

Direct isolation of the pathogen via amniocentesis with culture or PCR of amniotic fluid

Indirect tests—ultrasound for fetal manifestations, maternal antibody titers (IgM, IgG), or testing of fetal blood for IgM via cordocentesis

What is the difference between IgG and IgM?

IgG: rises slowly and often persists for long periods of time

A better marker for **overall exposure** to a disease

Crosses the placenta (starting at 16 weeks) to provide passive immunity

IgM: rises acutely and disappears from the circulation relatively soon after acute infection

A better marker for **recent infection**

Cannot cross the placenta

Synthesized by the fetus to provide primary immunity starting at 9–15 weeks

Toxoplasmosis

What is toxoplasmosis?

Infection with the **protozoa** *T. gondii* that causes an asymptomatic infection in adults but congenital disease if the primary infection occurs during pregnancy

What is the incidence of toxoplasmosis?

0.01–0.1% in the United States

What is the life cycle of *T. gondii*?

The domestic cat is the host and it passes the eggs in its feces. Humans can become infected by exposure to the **cat feces, which can occur with gardening, handling a cat's litter box.** Alternatively, many animals consume the eggs which are found in soil and humans also become infected by handling or eating the raw or **undercooked meat** of infected animals

What is the chance of fetal infection if primary infection occurs during pregnancy?

Approximately 33%

What is the difference between first and third trimester infection?

First trimester: low (15%) congenital infection rate

Most of those infected have **severe symptoms** and 5% result in fetal demise

Third trimester: high (65%) congenital infection rate

Most infections are **mild**

What are the symptoms of maternal toxoplasmosis?

Usually none; however, many patients report a syndrome of fevers, fatigue, headache, muscle pains, and lymphadenopathy after exposure

What is the classic triad of congenital toxoplasmosis?

Chorioretinitis, hydrocephalus, and intracranial calcifications

What are the possible clinical manifestations of toxoplasmosis?

Most infants are asymptomatic at birth. However, if signs are present at birth, they can include:

- Low birth weight
- Fever
- Maculopapular rash
- Hepatosplenomegaly
- Microcephaly
- Seizures
- Jaundice
- Thrombocytopenia
- Generalized lymphadenopathy

What abnormalities can develop after birth in an infected and untreated infant?

- Chorioretinitis
- Mental retardation
- Deafness
- Seizures
- Spasticity

Is toxoplasmosis screening routinely recommended for pregnant women?

No

What are the options for prenatal diagnosis of toxoplasmosis?

- Serology of maternal blood (high IgG titers suggest recent infection)
- Detection of the parasite
- Toxoplasma* PCR of amniotic fluid
- Serology of fetal blood for *Toxoplasma* IgM—not as sensitive

How can toxoplasmosis be diagnosed postnatally?

- Ophthalmologic, auditory, and neurologic examinations, an LP, and a head CT
- Neonatal serum test for **IgM and/or IgA antibodies**
- Isolation of organism** from placental tissue, umbilical cord tissue, or infant blood

How is congenital toxoplasmosis prevented?

If suspicion is high for congenital infection, spiramycin should be given until amniocentesis can be performed for toxoplasmosis PCR. Spiramycin prevents transplacental transmission of the parasite. Spiramycin, however, does not help treat or lessen the effects of congenital toxoplasmosis. If PCR of amniotic fluid is positive for *Toxoplasma*,

pyrimethamine and sulfadiazine should be added. Leucovorin is given to avoid the bone marrow suppression associated with the last two of these drugs. **If PCR of amniotic fluid is negative for *Toxoplasma*, spiramycin should be continued, and additional agents are not necessary**

How is toxoplasmosis in the newborn treated?

With a combination of **pyrimethamine and sulfadiazine. Leucovorin** is given to avoid the bone marrow suppression associated with these drugs

What is the prognosis of congenital toxoplasmosis?

Untreated, there is a poor prognosis. Chorioretinitis can eventually lead to blindness. These infants also suffer from seizures and severe psychomotor retardation

Rubella

What is rubella?

A single-stranded RNA virus of the togavirus family that is also known as **German measles**. It causes a mild, self-limited infection in adults but leads to severe disease if congenitally acquired

What is the incidence of congenital rubella?

Because of vaccinations, congenital rubella is extremely rare in the United States. However, it is slightly more common in the developing world

When is congenital rubella infection most likely to cause serious sequelae?

When maternal infection occurs in the first 8 weeks of pregnancy. Infection after the first trimester has almost no risk of developing anomalies in utero

How is rubella diagnosed in the pregnant state?

With history and physical examination, as well as by assessing the development of positive rubella antibody titers (which peak 1–3 weeks after the development of symptoms, if there are any)

What tools are used to diagnose congenital rubella?

Maternal history, physical examination of the neonate, CBC, x-rays of long bones, ophthalmologic examination, auditory examination, head CT, and LP

Viral isolation from nasal secretions

What is the difference between rubella IgM and IgG?	<p>Rubella-specific IgM or monthly rising IgG levels</p> <p>Both increase after exposure; however, IgM disappears after 1–2 months and so can distinguish acute infection from prior immunization. IgG is present for life</p>
What are the signs and symptoms of rubella in adults?	<p>Most are asymptomatic, but many women present with a rash</p>
What are the major clinical manifestations of congenital rubella and how common are each?	<p>Sensorineural deafness (50–75%)</p> <p>Cataracts (20–50%)</p> <p>Cardiac malformations (PDA [20–50%] or pulmonary arterial hypoplasia [20–50%])</p> <p>Neurologic sequelae (meningoencephalitis, behavior disorders, or MR [10–20%])</p> <p>Growth retardation</p> <p>Hepatosplenomegaly</p> <p>Thrombocytopenia</p> <p>Dermatologic manifestations (purpura, known as “blueberry muffin” lesions)</p> <p>Hyperbilirubinemia</p> <p>How is congenital rubella prevented? Through vaccination strategies and by testing all pregnant women for immunity</p> <p>Should the vaccination be given during pregnancy? No—it is a live, attenuated virus</p> <p>How is congenital rubella treated? Supportively—there is no known treatment for rubella</p>

Cytomegalovirus

What is cytomegalovirus (CMV)?	<p>A DNA herpes virus that is the most common cause of perinatal infection in the developed world</p>
What is the incidence of CMV?	<p>30–60% of Americans are seropositive; 1–4% of women seroconvert during pregnancy</p>
What increases the risk of seroconversion?	<p>Immune compromise</p>
What is the incidence of congenital CMV?	<p>40,000 infected infants are born in the United States annually</p>

How is CMV transmitted?	Via respiratory droplets, saliva, urine, blood, or sexual contact. It is commonly acquired in daycare centers
How is congenital CMV transmitted?	Transplacentally or through breast milk
What is the risk of transplacental transmission for a woman who was previously seropositive or who is newly infected during pregnancy?	A woman who was previously seropositive is at approximately 1% risk while one infected during pregnancy is approximately 40% at risk
How is maternal CMV diagnosed?	With antibody titers—a rise in IgG or the development of IgM antibodies predicts primary infection
How is congenital CMV diagnosed prenatally?	Amniocentesis for PCR or culture (preferred method) Ultrasound (microcephaly, hepatosplenomegaly, intracranial calcifications, etc.) IgM studies of fetal blood (less sensitive)
How is congenital CMV diagnosed in the neonate?	Viral isolation from urine or saliva in the first 3 weeks of life
When is congenital CMV infection most likely to cause serious sequelae?	When infection is acquired early in pregnancy
What percentage of infected neonates are symptomatic?	5–20%
What is the mortality rate associated with congenital CMV infection?	Almost 30%
What are the clinical manifestations of maternal CMV infection?	Most are asymptomatic ; however, some women have a mononucleosis-like syndrome
What are the possible clinical manifestations of congenital CMV infection?	Most newborns are asymptomatic at birth, but eventually develop the following anomalies: Hearing loss Impaired speech Chorioretinitis/visual impairment Mental retardation Microcephaly Seizures Paralysis or paresis Death

What can be used for treatment of congenital CMV infection?

Supportive—there is no treatment. Ganciclovir, CMV immune globulin, and alpha-interferon have been studied, but results are inconclusive to date and so they are not routinely recommended.

How can congenital CMV infection be prevented?

Through good hygiene and proper precautions for health care workers; routine prenatal screening is not recommended.

Herpes Simplex Virus

What is the incidence of herpes simplex virus (HSV)?

Over 20% of women are seropositive for HSV.

What are the two types of HSV?

HSV-1: causes most nongenital herpes infections.

HSV-2: causes most genital herpes infections (and most neonatal infections).

What is the incidence of congenital HSV?

0.01–0.04%

How is congenital HSV transmitted?

Mostly transplacentally; however, it can also be transmitted through ascending infection.

What is the rate of transmission if the mother has primary infection or a recurrent infection?

In the case of primary infection the rate of transmission is 50% while it is 4% in the case of recurrent infection.

What can be done to reduce the risk of transmission of HSV if there is an active lesion when the patient is in labor?

Cesarean delivery (reduces transmission from 8% to 1%)

What can be done to reduce the risk of needing a cesarean section in a patient with known recurrent HSV?

Prophylaxis with acyclovir from 36 weeks until delivery *or* initiated at an episode of preterm labor or PPRM.

How is congenital HSV diagnosed?

Viral cultures (from skin, nose, mouth, eyes, urine, blood, stool, and/or CSF).

What complications of pregnancy can occur as a result of HSV infection?

Preterm labor or spontaneous abortion

What are the three major groups of clinical manifestations of congenital HSV?

Disease localized to the skin, eyes, and mouth (SEM)

Disease localized to the CNS

Disseminated disease

What are other sequelae of congenital HSV?

Temperature instability
Respiratory distress
Poor feeding
Lethargy
Hypotension
Jaundice
DIC
Apnea

When do symptoms of congenital HSV develop?

Most newborns are asymptomatic and symptoms begin within the first 4 weeks of life

What is the mortality rate associated with neonatal HSV infection?

Close to 60%

What is the treatment for congenital HSV infections?

Supportive care and high-dose IV acyclovir

What is the prognosis for congenital HSV?

Disseminated infection carries a mortality rate of 57% but localized infection is generally not fatal

Encephalitis affects many of the survivors

OTHER VIRAL INFECTIONS

Syphilis

What is syphilis?

Infection with the **spirochete** *Treponema pallidum*

What is the incidence of congenital syphilis?

Less than 40 per 100,000 live births in the United States

How is the fetus infected with syphilis?

Transplacental transmission, typically during the second half of pregnancy. Women with primary or secondary syphilis are the most likely to transmit the disease

What are the possible sequelae of congenital syphilis?

Fetal manifestations: stillbirth
Spontaneous abortion
Hydrops fetalis
Prematurity
Manifestations before 2 years: lesions on palms and soles
Hepatosplenomegaly

	Jaundice
	Anemia
	Snuffles
	Manifestations after 2 years: congenital anomalies
	Active congenital syphilis
	Hutchinson triad
What is the Hutchinson triad?	Hutchinson teeth (blunted upper incisors), interstitial keratitis, and eighth nerve deafness
How is congenital syphilis diagnosed?	Veneral Disease Research Laboratory (VDRL) or rapid plasmin reagin (RPR) are used as screening tests. If positive, microhemagglutination T. pallidum (MHA-TP) or fluorescent treponemal antibody absorbed (FTA-ABS) are done as confirmatory tests
How is congenital syphilis prevented?	With adequate screening and then treatment (with penicillin) of infected mothers
How is syphilis treated?	With IV or IM penicillin G
How should a PCN-allergic pregnant woman be treated for syphilis?	With desensitization, because alternative drugs are teratogenic
How should a woman be followed after treatment?	Non-treponemal antibody serologic titers should be checked at 1, 3, and 6 months, 1 year, and 2 years after treatment to ensure proper falls of levels. If they do not fall, she should be treated for reinfection
What is the Jarisch-Herxheimer reaction?	An acute febrile reaction precipitated by treatment of syphilis thought to result from the release of large amounts of treponemal lipopolysaccharide (LPS). It may precipitate preterm labor, contractions, or non-reassuring fetal heart tracings in pregnant women

Varicella-Zoster Virus

What is varicella-zoster virus?

A double-stranded, linear, DNA herpes virus that causes both chickenpox (varicella) and shingles (zoster)

<p>What is the incidence of VZV in pregnancy?</p>	<p>1–5 cases per 10,000 pregnancies (most women are immune secondary to prior infection)</p>
<p>What are the clinical features of varicella in adults?</p>	<p>It includes a prodrome of fever, malaise, and myalgia followed, 1–4 days later, by a vesicular rash</p>
<p>What are the complications of varicella infection?</p>	<p>Bacterial superinfection of vesicles Pneumonia Arthritis Glomerulonephritis Myocarditis Ocular disease Adrenal insufficiency Death CNS abnormalities</p>
<p>Why is varicella in pregnancy a medical emergency?</p>	<p>Because varicella pneumonia can develop and it is very severe in pregnancy</p>
<p>What is varicella pneumonia?</p>	<p>An infection that develops within a week of the varicella rash that presents as cough, dyspnea, fever, pleuritic chest pain, and/or hemoptysis. It is very severe in pregnant women as it can rapidly progress to respiratory failure</p>
<p>What is the incidence of varicella pneumonia during pregnancy?</p>	<p>It occurs in 10–30% of all VZV cases in pregnancy</p>
<p>What are the chest x-ray (CXR) findings of varicella pneumonia?</p>	<p>A diffuse or miliary/nodular infiltrative pattern usually in a peribronchial distribution in both lungs</p>
<p>What is the treatment of varicella pneumonia during pregnancy?</p>	<p>Supportive care and acyclovir. All women with VZV require a CXR to rule out varicella pneumonia</p>
<p>What is the mortality rate associated with varicella in pregnancy?</p>	<p>Untreated, it carries a 40% mortality rate</p>
<p>What are the features of congenital varicella syndrome?</p>	<p>Chorioretinitis Cortical atrophy Dermatologic conditions Hypoplastic lower limbs Hydronephrosis Clubbed feet</p>

	Optic atrophy
	Failure to thrive
	Cataracts
	Horner syndrome
	Microphthalmos
	Nystagmus
	Low birth weight
	Mental retardation
	Early death
What is the congenital infection rate if the mother has VZV?	25–50% if the mother develops a rash 5 days before or 2 days after birth
When is the fetus most likely to be affected by congenital varicella syndrome?	When the mother is infected between 8 and 20 weeks of gestation
How can varicella infection during pregnancy be prevented in a patient with a recent exposure?	By giving VariZIG , a purified human varicella zoster immune globulin if she is IgG seronegative (not previously immune)
When should VariZIG be given?	To the mother within 96 hours after viral exposure if she is IgG seronegative To the neonate (if there is exposure) within 4 days prior or 2 days after delivery
How should a patient with varicella be managed if she is more than 5 days from delivery?	With close observation (for any signs/sx of varicella pneumonia) and possibly with acyclovir
How should a pregnant woman exposed to zoster be treated?	Reassurance—it is only contagious from direct contact with open lesions

Parvovirus B19

What is parvovirus B19?	A single-stranded DNA virus
What is the incidence of parvovirus B19 infection in pregnant women?	3–4%
What is the transmission rate of parvovirus B19 from an infected woman to her fetus?	Approximately 20%—it is the highest if infection occurs between 10 and 20 weeks of gestation
How is parvovirus diagnosed?	Serologic testing of IgM and IgG antibodies
Describe the major manifestations of infection with parvovirus B19	A self-limited infection known as erythema infectiosum or fifth disease that consists of:

<p>What are the manifestations of parvovirus B19 during pregnancy?</p>	<p>Dermatologic manifestations—a “slapped cheek” appearance on the face and a “lace-like” erythematous rash on the trunk and extremities</p> <p>Symmetric arthropathy</p> <p>Flu-like symptoms</p>
<p>What is the risk of fetal loss?</p>	<p>Fetal loss or hydrops fetalis may ensue; however, there are no long-term developmental sequelae if a normal pregnancy ensues</p> <p>If infection occurs before 20 weeks, it is 11%. If infection occurs after 20 weeks, it is <1%</p>
<p>What is hydrops fetalis?</p> <p>What is the etiology of hydrops in the setting of congenital parvovirus infection?</p>	<p>Generalized fetal edema</p> <p>Destruction of RBC precursors leading to fetal anemia</p> <p>Myocarditis leading to fetal myocardial dysfunction</p>
<p>What is the risk of hydrops fetalis?</p> <p>What do the following serologic results signify?</p>	<p>Less than 4%</p> <p>Positive IgG and negative IgM: Prior maternal immunity</p> <p>Positive IgM and negative IgG: Acute infection</p>
<p>How should an infected pregnant woman be managed?</p>	<p>Prior to 20 weeks, no action is necessary. After 20 weeks, women should receive weekly ultrasounds to look for signs of hydrops for 10 weeks after infection</p>
<p>What sign on ultrasound indicates severe fetal anemia?</p>	<p>Elevated peak systolic velocity on the fetal middle cerebral artery dopplers</p>
<p>How is severe fetal anemia secondary to parvovirus treated?</p>	<p>With intrauterine fetal blood transfusion</p>

HIV

<p>What is HIV?</p>	<p>An RNA retrovirus that incorporates into the host genome and affects primary T cells leading to immunocompromise</p>
<p>How is HIV infection diagnosed?</p>	<p>Enzyme-linked immunosorbent assay (ELISA) testing followed by a Western blot, if the ELISA is positive</p>

How do most pediatric HIV infections develop?	Through mother-to-child transmission via transplacental infection (50%), peripartum infection (30%), or via breast-feeding (20%)
What is the rate of vertical transmission in an HIV positive woman who is untreated?	25%
What is the rate of vertical transmission in an HIV positive woman on zidovudine (azidothymidine) ZDV (AZT) alone?	8%
What is the rate of vertical transmission in an HIV positive woman on HAART (highly active antiretroviral therapy)?	1–2%
What are the rates of perinatal HIV transmission in the United States?	1–2%—they have been dramatically reduced via multiple public health efforts
What increases the risk of vertical HIV transmission?	Preterm birth Prolonged ROM Concurrent syphilis infection Chorioamnionitis
What are the two mainstays of prevention of vertical transmission?	1. Antiretroviral therapy 2. Cesarean delivery
Are adverse pregnancy outcomes more common in HIV infected women?	Yes. Preterm birth and fetal growth restriction are more common, and these rates increase with a decreased CD4 count
Which pregnant patients should be given antiretroviral therapy?	All pregnant patients , regardless of CD4 count or viral load, should be offered antiretroviral therapy in order to reduce the risk of transmission
When should CD4 counts be measured during pregnancy?	Once each trimester
When should the viral load be measured during pregnancy?	4 weeks after any change in therapy, monthly until viral levels are undetectable, every 3 months while the viral load remains undetectable, and then near term
What are some of the intrapartum precautions that should be taken?	Avoid artificial rupture of membranes (HIV transmission increased with increased time after rupture)

	Use labor augmentation sooner (HIV transmission increased with longer interval to delivery)
	Give IV zidovudine intrapartum
How should the neonate of an HIV infected mother be treated?	With oral zidovudine
How much does cesarean delivery reduce the risk of vertical HIV transmission?	By half

Hepatitis

What are the hepatitis viruses?	A group of both DNA and RNA viruses that invade hepatocytes , leading to hepatocellular inflammation, scarring, and sometimes death
What are the symptoms of acute hepatitis infection?	Most are asymptomatic or mild. Symptoms, when present, include: Nausea Vomiting Headache Malaise Fatigue Right upper quadrant (RUQ) pain
What are the signs of hepatitis infection?	Jaundice RUQ tenderness Hepatomegaly
What are the laboratory values which can be associated with hepatitis infection?	Elevated transaminases Elevated bilirubin Coagulation abnormalities—increased PT and PTT (in severe cases) Elevated ammonia (in severe cases)
What are the major differential diagnoses of hepatitis infection in pregnancy?	Acute fatty liver of pregnancy, severe preeclampsia, HELLP syndrome
What is chronic hepatitis?	Hepatic necrosis, inflammation, and scarring that eventually can lead to cirrhosis and liver failure
How is chronic hepatitis diagnosed?	It is usually asymptomatic and so it is presumptively diagnosed by

How is chronic hepatitis because of viral infection treated?

elevated transaminase levels. The diagnosis can be confirmed via liver biopsy

With **interferon** (a cytokine with immunoregulatory effects) and **ribavirin**. Recently, some nucleoside analogues (lamivudine and adefovir dipivoxil) have been found effective in many patients. None of these medications can be used in pregnancy

Hepatitis A

What is hepatitis A and how is it transmitted?

An RNA picornavirus that is transmitted by the **fecal–oral spread**. It causes about one-third of hepatitis cases in the United States and leads to infection in 1 in 1000 pregnancies in the United States

How is hepatitis A diagnosed?

IgM antibody suggests **recent infection**; **IgG** antibody suggests **prior exposure**

How is hepatitis A prevented?

Vaccination for high-risk women (it is an inactivated vaccine and so is safe to administer in pregnancy)

How is hepatitis A exposure treated?

With passive immunity via hepatitis A immune globulin

What is the effect of hepatitis A on perinatal outcomes?

Hepatitis A is not teratogenic; however, infection can lead to preterm birth and neonatal cholestasis

Hepatitis B

What is hepatitis B and how is it transmitted?

A DNA hepadenavirus transmitted through **infected blood or bodily fluids**. Its viral genome is covered by a middle portion and then an outer shell

What is the relationship between hepatitis B and HIV infection?

There is often coinfection, as they have similar modes of transmission. Coinfection with HIV leads to increased liver-related disease

What is the prevalence of hepatitis B?

There are 1.2 million carriers in United States and 350 million carriers globally

What percentage of those exposed to hepatitis B develop chronic infection?

In adults: 5–10%

In infants: 70–90%

How is hepatitis B diagnosed?

Through antibody testing

Describe each of the following hepatitis B antigens

HBsAg Hepatitis B surface antigen; it is on the outer viral shell and circulates in the serum

HBcAg Hepatitis B core antigen; it is in the middle portion of the virus and does not circulate in serum although it is expressed in infected hepatocytes

HBeAg Hepatitis B 'e' antigen; it correlates with infectivity

Describe the series of hepatitis B antibody responses

The first marker of infection is **HBsAg**. Then **HBeAg** is detectable, which also denotes early infection. Both of these resolve within 3–6 months after infection. One month after infection, **IgM anti-HBc** begins to develop. It peaks at around 4 months and, within 6 months after exposure, **IgG anti-HBc is detectable**. Around the same time, **anti-HBs** develops. Persistence of HBsAg for more than 6 months is considered to be chronic infection

How is an acute hepatitis B infection diagnosed serologically?

Anti-HBc IgM and HBSAg

How is chronic hepatitis B infection diagnosed serologically?

HBsAg without anti-HBc IgM

What are the major sequelae of hepatitis B infection?

Chronic hepatitis, cirrhosis, and hepatocellular carcinoma

What is the percentage of risk of vertical transmission in a patient that is HBeAg positive?

90%

What is the percentage of risk of vertical transmission in a patient that is HBeAg negative?

<10%

How does infection with hepatitis B affect pregnancy?

It does not affect the pregnancy course; however, it can increase the likelihood of preterm delivery

How does neonatal infection with hepatitis B occur?

Through **peripartum exposure to infected maternal fluids** and through **breast-feeding**. Transplacental infection is rare

What is the likelihood of developing disease after neonatal hepatitis B exposure?

Most infected neonates are asymptomatic; however, **85% become chronic carriers**

How is neonatal infection prevented?

Through **prenatal screening** and administration of **hepatitis B immune globulin** as well as the **hepatitis vaccination** of newborns of infected mothers (vaccination is safe in pregnancy)

Is breast-feeding safe in women with Hepatitis B?

There is a theoretic risk of transmission; however, hepatitis B immune globulin and neonatal vaccination should eliminate this risk

Hepatitis C

What is hepatitis C and how is it transmitted?

A single-stranded Flaviviridae virus that is transmitted through exposure to blood and bodily fluids

What is the incidence of hepatitis C in pregnancy?

Depending on the population studied, it ranges from approximately 1–5%

How is hepatitis C diagnosed?

Serum antibody testing (those seropositive for anti-HCV have chronic hepatitis)

How does hepatitis C infection affect pregnancy?

It has no effect on pregnancy, although it may increase the risk of cholestatic jaundice

What is the incidence of vertical transmission of hepatitis C?

Between 3% and 6%

How can vertical transmission be prevented?

There are currently no known methods to prevent transmission at birth

Hepatitis D

What is hepatitis D?

A defective RNA virus that is only infectious if it coinfects with hepatitis B and is transmitted in a similar manner

What percentage of patients infected with hepatitis B are coinfecting with hepatitis D?

Approximately 25%

What are the sequelae of infection with hepatitis B and D?

The same as hepatitis B, however the infection is **more severe** than infection with hepatitis B alone

Hepatitis E

What is hepatitis E?

An RNA virus transmitted via contaminated water

What are the effects of hepatitis E infection in pregnancy?

There may be a high rate of vertical transmission and there is some evidence that infection is more severe in pregnancy

BACTERIAL INFECTIONS

Group B Streptococcus

What is group B *Streptococcus* (GBS)?

Streptococcus agalactiae, a gram-positive infection of the GI, upper respiratory and genital tracts. It leads to severe disease in neonates if they are infected right before or during birth

What percentage of women are asymptomatic carriers of GBS?

Approximately 15–30%

Where does colonization typically occur?

Primarily in the **rectum**, with secondary infection in the bladder, vagina, and cervix

What are the symptoms of GBS colonization in women?

Usually none

How is GBS colonization diagnosed?

Via routine screening with urine culture and rectovaginal swab. A sample is swabbed from the vagina and rectum for culture and latex agglutination or ELISA

What is the incidence of invasive GBS infection in pregnancy?

Because of intrapartum chemoprophylaxis, the incidence of invasive disease is low. GBS affects only approximately 1 in 4000 births in the United States

What is the rate of maternal-neonatal transmission of GBS if left untreated?

35–75% depending on the degree of infection

How does symptomatic GBS infection manifest in pregnant women?

UTI, chorioamnionitis, or postpartum wound infection, bacteremia, or endomyometritis

What are the clinical manifestations of neonatal GBS infection?

Bacteremia, sepsis, pneumonia, respiratory distress, meningitis, shock, or death can ensue. If the neonate survives, 33% have long-term neurodevelopmental problems

What is the difference between early onset and late onset GBS disease?

Early onset refers to infection before 7 days of age and it carries the highest mortality rate. Late onset disease occurs from 1 week to 3 months after birth

What is the mortality rate associated with neonatal GBS infection?

5%–15%

What are the major risk factors for neonatal GBS infection that necessitate treatment in a woman who has not been screened for GBS?

Preterm labor

GBS bacteriuria during the pregnancy

Prolonged ROM (>18 hours)

Having a neonate affected by early-onset GBS infection in a previous pregnancy

Maternal fever >38°C during labor

What is the recommended treatment for GBS colonization?

IV penicillin or ampicillin

What are the alternatives for patients allergic to penicillin?

Cefazolin and vancomycin. Clindamycin or erythromycin are acceptable alternatives only if culture and sensitivities have demonstrated the bacteria to be sensitive

Listeria Monocytogenes

What is *Listeria*?

A gram-positive, aerobic bacillus that is transmitted via fecal-oral transmission

What is commonly implicated in *Listeria* infection?

Infected foods such as raw vegetables, milk, smoked fish, soft cheeses, and some processed meats

What are the clinical manifestations of listeriosis in adults?

Can be **asymptomatic** or it can cause a **flu-like illness**

What are the clinical manifestations of fetal infection with *Listeria*?

Disseminated granulomatous lesions with microabscesses

Chorioamnionitis

Sepsis

Sudden abortion or stillbirth

What is the difference between early onset and late onset *Listeria* infection?

Early onset disease occurs within the first week of life and it typically presents after a **preterm** delivery with **respiratory distress**, fever, and neurologic abnormalities. **Late onset disease** occurs after the first week, and it typically presents as **meningitis**

What is the treatment for *Listeria* infection in pregnancy?

Ampicillin or trimethoprim-sulfamethoxazole or vancomycin (in PCN-allergic patients)

Gonorrhea

What is gonorrhea?

Caused by *Neisseria gonorrhoeae*, it is an STI that can have serious consequences in pregnancy

What are the possible pregnancy complications that can occur as a consequence of gonorrhea infection?

Spontaneous abortion
Stillbirth
Preterm labor
PROM
Chorioamnionitis
Postpartum infection

What are the possible neonatal effects of gonorrhea infection in pregnancy?

Gonococcal ophthalmia, arthritis, and sepsis

How is gonococcal ophthalmia prevented?

By giving all infants prophylactic **erythromycin** or **silver nitrate** eye drops

Chlamydia

What is *Chlamydia*?

Infection with *Chlamydia trachomatis*, an obligate intracellular bacterium

What are the possible pregnancy complications that can occur as a consequence of chlamydial infection?

Preterm labor, PROM, and/or postpartum endometritis

What are the possible neonatal effects of chlamydial infection in pregnancy?

Conjunctivitis, **pneumonia**, and/or **otitis media**

How should pregnant women infected with *Chlamydia* be treated?

With **erythromycin** or **azithromycin** (tetracycline should be avoided because of teratogenicity)

How is chlamydial ophthalmia prevented? By giving all infants prophylactic **erythromycin** or **silver nitrate** eye drops

UTI IN PREGNANCY

UTI is the most common medical complication of pregnancy.

Why is pregnancy considered a high-risk condition with asymptomatic bacteriuria? Both hormonal and mechanical changes predispose the pregnant woman with asymptomatic bacteriuria to develop acute pyelonephritis, which is associated with preterm birth and perinatal death. Pyelonephritis in pregnancy will lead to septicemia in 10–20% and ARDS in 2% of cases

How prevalent is asymptomatic bacteriuria in pregnancy? It is estimated to occur in 4–7% of pregnant patients. If left untreated, up to 40% of cases will progress to pyelonephritis

How is this condition detected? Screening for asymptomatic bacteriuria using urine culture is recommended at the first prenatal visit

How is asymptomatic bacteriuria treated? Any FDA category B drug such as cephalosporins, nitrofurantoin, or trimethoprim-sulfamethoxazole can be used. Quinolones (FDA category C) are generally not used during pregnancy. 7-day courses are recommended, along with a follow-up culture to document sterile urine. Persistent bacteriuria should be treated based on sensitivities. Suppressives antibiotics (most commonly nitrofurantoin) should then be considered in these patients

How is symptomatic cystitis treated in pregnancy? Treatment and follow-up is similar to asymptomatic bacteriuria. Acute pyelonephritis should be treated with IV antibiotics and hospitalization. Suppressives antibiotics should be given following

treatment of any pregnant patient treated for acute pyelonephritis

Rhesus (Rh) Alloimmunization

What is alloimmunization of pregnancy?

It is when the fetus inherits a blood group factor from the father which the mother does not possess. Exchange of blood during fetal-maternal bleeding causes the mother to become sensitized to the foreign antigen and stimulates the **formation of maternal antibodies (alloimmunization) during that pregnancy**. These antibodies may enter the fetal circulation of her **next pregnancy** and lead to **hemolytic disease in the fetus** and neonate by sensitizing the fetal RBCs for destruction by macrophages of the fetal spleen (see Fig. 9-6)

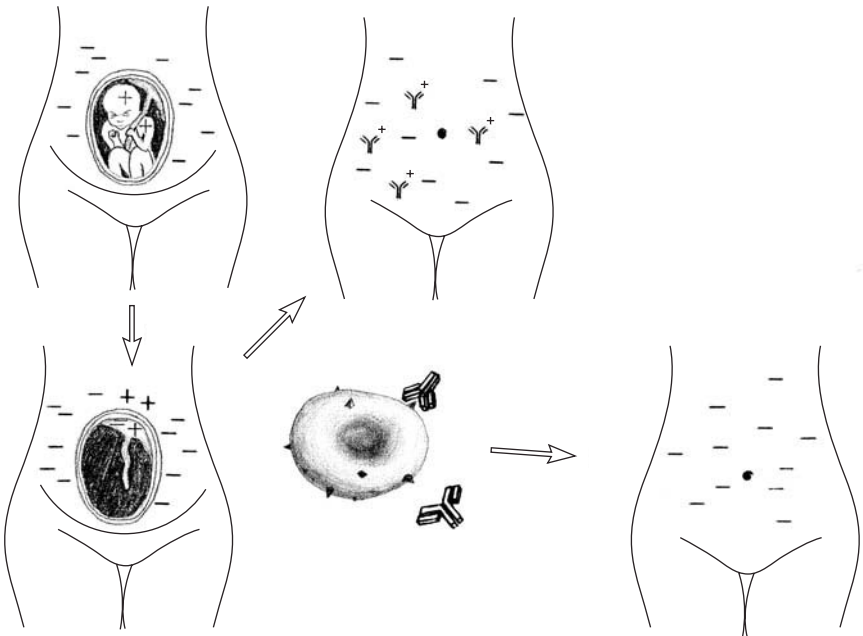


Figure 9-6 Isoimmunization and RHOGAM treatment.

How is erythroblastosis fetalis described?	It is when fetal red blood cell destruction far exceeds production and severe anemia occurs. This disease is characterized by extramedullary hematopoiesis, heart failure, edema, ascites, and pericardial effusion. Hemolysis also produces heme and bilirubin, both of which are neurotoxic and may lead to kernicterus
What is the correct nomenclature for designating a gravida's blood type?	ABO blood type and either Rh(D+) or Rh(D-)
What is the nomenclature of the Rh blood group system?	The Rh blood system consists of five antigens (C, c, D, E, e). The D antigen is most commonly implicated in Rh alloimmunization and, therefore, Rh positive usually indicates the presence of the D antigen and Rh negative indicates the absence of D antigen on erythrocytes
By what mechanisms can Rh alloimmunization occur?	<ol style="list-style-type: none"> 1. Transplacental fetomaternal hemorrhage during any pregnancy (most common) 2. Injection with needles contaminated by Rh(D+) blood 3. Inadvertent transfusion of Rh(D+) blood
What is the most common scenario for fetomaternal hemorrhage?	Uncomplicated vaginal delivery in 15–50% of births. A minimum volume of 0.1 mL of fetal blood entering the maternal circulation can result in alloimmunization
What are several less common situations where fetomaternal hemorrhage can occur?	Cesarean delivery, multifetal gestation, bleeding placenta previa or abruption, manual removal of the placenta, and intrauterine manipulation, first-trimester spontaneous and induced abortion, threatened abortion and ectopic pregnancy, obstetrical procedures such as chorionic villus sampling, pregnancy termination, amniocentesis, and external cephalic version
When should a blood and Rh(D) typing and antibody screen be performed?	Always at the first visit! These tests should also be repeated with every subsequent pregnancy. The American

What test is the most sensitive test for determining antibody titers and diagnosing Rh(D) alloimmunization? How does it work?

What titer levels are considered critical for significant risk of severe erythroblastosis fetalis and hydrops?

What is the direct Coomb's test?

Who should be given anti-D immune globulin (RhoGAM)?

How does RhoGAM prevent alloimmunization?

How are sensitized Rh(-) patients evaluated?

Association of Blood Banks also recommends repeated **antibody screening before administration of anti-D immune globulin at 28 weeks of gestation, postpartum**, and at the time of any event in pregnancy

Indirect Coomb's test. Incubation of a known specimen of Rh(D+) RBCs with maternal serum is the first step in the indirect Coomb's test. Maternal anti-Rh(D) antibodies, if present, will adhere to the RBCs. The RBCs are then washed and suspended in serum containing antihuman globulin (Coomb's serum). Red cells coated with maternal anti-Rh(D) will be agglutinated by the antihuman globulin, which is referred to as a positive indirect Coomb's test

Critical titers may vary from laboratory to laboratory. In general, the critical titer that poses a significant risk for erythroblastosis fetalis and hydrops is 1:16 to 1:32

It is similar to the indirect Coomb's test and is done after birth to detect the presence of maternal antibody on the neonate's RBCs. It is performed by placing the infant's RBCs in Coomb's serum; maternal antibody is present if the cells are agglutinated

It is not effective once alloimmunization to the Rh(D) antigen has occurred. Therefore, it is essential that it be given to an **Rh(D-) woman whose fetus is or may be Rh(D+) whenever there is a risk of fetomaternal hemorrhage**

It is an IgG that will attach to the Rh antigen and prevent an immune response by the mother (see Fig. 9-6)

There are three options once the gravida reaches the critical titer:

1. MCA doppler can be performed weekly. MCA doppler is sensitive in diagnosing moderate to severe

hemolytic disease. Once the fetus reaches a value indicating moderate to severe anemia, cordocentesis with transfusion is indicated.

2. Amniocentesis can be performed with two indicated tests: PCR on the fetal amniocytes will determine the fetal blood type. If the fetus is Rh(-), no additional testing is indicated. Indirect measurement of hemolytic disease can be determined by measuring bilirubin concentration in the amniotic fluid using spectrophotometry at a wavelength of delta-OD 450. The absorbance measurements indicate the degree of fetal anemia. Measurements in zone III and high range of zone II indicate a need for cordocentesis with transfusion.
3. Cordocentesis can be performed initially to determine fetal blood type and hemoglobin concentration. However, this procedure can have up to a 1% risk of fetal loss per procedure
1. All pregnant women should undergo type and antibody screening for the ABO and Rh group at the first prenatal visit
2. Another antibody screen is obtained at 28 weeks of gestation to detect women who have become alloimmunized in the interval since the first screen
3. Anti-D immune globulin should be administered early in the third trimester, 300 µg at 28 weeks of gestation
4. Anti-D immune globulin is effective for 12 weeks, and if the gravida received an injection at 28 weeks' gestation, she should have a repeat injection at 40 weeks' gestation

What is the management of the unsensitized Rh(-) pregnancy (the Rh(-) patient who has a negative antibody screen)?

What are additional indications to give 300 µg anti-D immune globulin to any Rh(D-) woman whose fetus is or may be Rh(D+)?

Whenever there is increased risk of fetomaternal hemorrhage such as spontaneous abortion, ectopic pregnancy, invasive procedures such as amniocentesis, chorionic villus sampling and cord blood sampling, antepartum hemorrhage, external cephalic version

What is the Kleihauer-Betke test and when should it be used?

It is a test to quantify the amount of fetal RBCs in the maternal circulation in circumstance where excessive fetomaternal hemorrhage has occurred and Rh sensitization is positive for persistent antibody after initial administration of RhIgG. Additional doses of RhIgG is given according to the amount of excess hemorrhage

What is the role of postpartum administration of anti-G immune globulin?

It is recommended to administer 300 µg of anti-D immune globulin within 72 hours of delivery of an Rh (D+) infant

What should one do if anti-D immune globulin is inadvertently omitted after delivery?

It is still recommended to give it as soon as possible. Partial protection is afforded with administration within 13 days of the birth, and there may be an effect as late as 28 days after delivery

How are sensitized Rh(-) patients (positive Rh antibody screen on initial visit) evaluated?

It depends on whether the patient has a history of an affected fetus in a previous pregnancy.

No history of a previous pregnancy affected by Rh isoimmunization:

1. Antibody screen and titers at 0, 20 weeks' EGA, and then every 4 weeks
2. Determine the paternal Rh(D) type and if Rh(D+), determine zygosity
3. Amniocentesis should be performed if titers reach critical levels (1:32)
4. Doppler ultrasound of the MCA should be performed every 2 weeks beginning at 24 weeks. High peak velocity blood flows correlates with severe fetal anemia

History of a previous pregnancy affected by Rh isoimmunization:

1. Maternal titers are not helpful in following the degree of fetal anemia after the first affected gestation
2. Determine the paternal Rh(D) type and if Rh(D+), determine zygosity
3. In cases of a heterozygous paternal phenotype, perform amniocentesis at 15 weeks of gestation to determine the fetal Rh(D) status
4. If the father is a homozygote or the fetus is Rh(D+), begin MCA doppler velocity assessment at 18 weeks of gestation. Repeat at 1–2 week intervals

How is the severity of disease predicted in fetuses in which isoimmunization has occurred?

Bilirubin present in amniotic fluid derives from fetal pulmonary and tracheal effluents and correlates with the degree of fetal hemolysis. Amniotic fluid is analyzed by spectrophotometer, which measure the light absorbance by bilirubin. Absorbance measurements are plotted on a **Liley curve**, which predicts the severity of disease

Fetal Growth Abnormalities

FETAL GROWTH RESTRICTION

What are other common terms to describe fetuses with disproportionately small growth?

Small for gestational age (SGA); intrauterine growth restriction (IUGR); low birth weight (LBW); and fetal growth restriction (FGR)

How is FGR commonly defined?

It is defined as **estimated fetal weight (ESW) at or below the 10th percentile for gestational age**. This definition is controversial because it does not make a distinction among fetuses who are constitutionally

FGR or LBW is associated with increased perinatal mortality. At what percentile is this risk greatest?

Abnormal fetal growth may be classified as symmetrical or asymmetrical. What is meant by these terms?

What are the several causative factors for both symmetric and asymmetric FGR?

FGR may be caused by fetal, maternal, or placental factors. What are several fetal etiologies that cause FGR?

What are some genetic diseases or syndromes that typically manifest with FGR?

small, growth restricted and small, and growth restricted but not small. In addition, birth weight is also related to maternal height, parity, paternal height, and the fetus's sex

Weights below the third percentile for gestational age

In symmetric FGR, all fetal organs including the brain are proportionally small because of abnormalities in early fetal cellular hyperplasia. There is cellular hypoplasia or a reduction in the total number of cells. This comprises 20–30% of all growth-restricted fetuses

In asymmetrical FGR, there is a relatively greater decrease in abdominal size than head circumference. This is thought to occur from redistribution of blood from non-vital organs (liver, abdominal viscera) to vital organs (heart, brain). There is redistribution away from the kidneys in asymmetrical growth restriction which may give lower AFI and oligohydramnios. This comprises 70–80% of growth-restricted fetuses

Symmetric: early insults such as chromosomal abnormalities, early teratogenic exposure, and early exposure to TORCH infections

Asymmetric: maternal conditions such as hypertension, vasculopathies, diabetes with vascular disease, and placental abnormalities

Genetics, congenital anomalies, multi-fetal pregnancy

Trisomy 21 (Down syndrome), Trisomy 18 (Edwards syndrome), Trisomy 13 (Patau syndrome), cri-du-chat syndrome, Turner syndrome,

Abnormal placental conditions cause FGR because of mismatch between fetal nutritional or respiratory demands and placental supply. What are some placental etiologies?

Several maternal conditions affect the microcirculation causing fetal hypoxemia, vasoconstriction, or a reduction in fetal perfusion, thus causing FGR. What are these common maternal etiologies?

What other maternal etiologies cause FGR?

What are some maternal complications associated with an FGR pregnancy?

What are some fetal complications associated with an FGR pregnancy?

What are some neonatal complications associated with an FGR pregnancy or complications found in small-for-gestational age infants?

Fanconi syndrome, skeletal dysplasias

Chorioangioma or hemangioma of the placenta, placenta previa, placental mosaicism, single umbilical artery, velamentous umbilical cord insertion, placenta biloba

Hypertension, both chronic and acute (as in preeclampsia), cyanotic heart disease, and severe anemia (as with sickle cell anemia), renal insufficiency, systemic lupus erythematosus, thrombophilias (acquired such as antiphospholipid antibody syndrome and inherited), chronic anemia, and pregestational diabetes

Infections (rubella, toxoplasmosis, cytomegalovirus, varicella-zoster, malaria)

Teratogens (trimethadione, phenytoin, methotrexate, and warfarin)

Poor nutrition

Substance abuse (cocaine, alcohol)

Socioeconomic factors (race, pregnancy at the extremes of reproductive life, and previous delivery of an FGR neonate)

Complications because of underlying disease, preeclampsia, premature labor, cesarean delivery

Stillbirth, hypoxia and acidosis, malformations

Difficult cardiopulmonary transition: perinatal asphyxia, meconium aspiration, or persistent pulmonary hypertension

Complications with prematurity: neonatal death, necrotizing enterocolitis, and respiratory distress syndrome

Impaired thermoregulation

Hypoglycemia

<p>What are the long-term complications for the child of an FGR pregnancy?</p>	<p>Polycythemia and hyperviscosity Impaired immune function Lower IQ, learning and behavior problems, major neurologic handicaps</p>
<p>How is the diagnosis of FGR determined?</p>	<p>Patient history, clinical assessment of fundal height, and sonographic evaluation of the fetus, placenta, and amniotic fluid is necessary</p>
<p>What sonographic measurements are diagnostic and predictive of FGR?</p>	<p>Estimation of fetal weight is the single best morphometric test for identifying fetuses whose birth weight is likely to be below the 10th percentile for gestational age (FGR)</p>
<p>What is oligohydramnios and what role does it have in the diagnosis of FGR?</p>	<p>Oligohydramnios may be a consequence of FGR and refers to amniotic fluid volume that is less than expected for gestational age. It occurs when there is diminished fetal urine production, and in FGR this may be because of hypoxia-induced redistribution of blood flow to vital organs at the expense of less vital organs, such as the kidney. Severe oligohydramnios is associated with high perinatal mortality (15–80% of fetuses with FGR do not have decreased amniotic fluid volume)</p>
<p>What is the role of doppler assessment of the umbilical arteries in evaluating FGR?</p>	<p>They are not useful for screening and diagnosis of the small fetus. They are useful for identifying the small fetus that is at risk for adverse perinatal outcome (non-reassuring fetal heart rate patterns, cesarean delivery, preterm birth, neonatal intensive care admission, asphyxia)</p>

FETAL MACROSOMIA

What is the definition of fetal macrosomia and large-for-gestational age (LGA)?

Macrosomia implies growth beyond a specific threshold, usually 4000 g or 4500 g, regardless of gestational age. However, maternal and fetal morbidity rises sharply with birth weights >4500 g

What is the incidence of fetal macrosomia?

Large-for-gestational age implies a birth weight equal to or greater than the 90th percentile for a given gestational age

The worldwide prevalence of the birth of infants ≥ 4000 g is approximately 9%, with wide variations between countries. 1.5% of all infants weigh >4500 g

What are maternal and fetal complications associated with macrosomia?

Maternal: protracted labor, cesarean delivery, genital tract lacerations, postpartum hemorrhage, uterine rupture

Fetal: shoulder dystocia, brachial plexus injuries, fractures of the clavicle, asphyxia

Neonatal: increased risk of depressed 5-minute APGAR scores, increased risk of admission to the NICU, jaundice, hypoglycemia

Long-term: obesity

What are risk factors for macrosomia?

In decreasing order of importance:

Prior history of macrosomia

Maternal prepregnancy weight

Maternal weight gain during pregnancy

Multiparity

Male fetus

Gestational age >40 weeks

Ethnicity

Maternal birth weight, height, age

Positive 50 g glucose screen with a negative result on the 3-hour glucose tolerance test

What pregnancy-related disease is an independent factor for and highly associated with macrosomia?

Pregestational diabetes and gestational diabetes

Which genetic and congenital syndromes are associated with macrosomia?

Beckwith-Wiedemann syndrome (pancreatic islet cell hyperplasia)

Fragile X syndrome

How is macrosomia diagnosed?

An accurate diagnosis of macrosomia can only be made after weighing the newborn. However, prenatal

Are there any available interventions for treating fetal macrosomia?	diagnosis of fetal macrosomia is best diagnosed by evaluation of maternal risk factors, clinical examination (Leopold maneuvers), and ultrasound measurements
Is prophylactic cesarean delivery indicated for pregnant women suspected to have macrosomic fetuses?	For mothers without diabetes, there are no reported interventions For diabetic mothers, addition of insulin to diet therapy may treat early macrosomia
When should elective cesarean delivery be considered?	Prophylactic cesarean delivery to prevent shoulder dystocia may be considered for an ESW >5000 g in nondiabetic women and >4500 g in women with diabetes (induction of labor for macrosomia is also not recommended) For women whose previous delivery was complicated by shoulder dystocia, particularly when a brachial plexus injury occurred

Multi-fetal Gestation

What is the incidence of <i>spontaneous</i> twins and multiple births in the United States?	Twins: 1:80 Triplets: 1:8000
How has the incidence of multifetal gestation changed over time?	Since 1980 there has been a 65% increase in the frequency of twins and a 500% increase in triplet and high-order births
What is causing this increasing trend in multi-fetal gestation?	Increased use of ART and ovulation-induction agents
What is the role of maternal age in increased multi-fetal pregnancy?	The rate of multiples increases with increasing age. This is most likely the result of older women undergoing antiretroviral therapy (ART) and higher levels of FSH in advancing age
What is the significance of multi-fetal gestation on fetal morbidity and mortality?	It causes a significant effect on preterm delivery and low birth weight of newborns

How significant is the impact of multi-fetal gestation on maternal morbidity and mortality?

They account for 17% of all preterm births (before 37 weeks of gestation)

It increases maternal morbidity and mortality significantly, as well as hospital costs. Women with multiple gestations are six times more likely to be hospitalized with complications. Hospital costs are on an average 40% greater compared to women with single gestations. Neonatal intensive care unit (NICU) admissions are significantly more frequent and are of longer duration. Multi-fetal gestations contribute to maternal conditions such as preeclampsia and gestational diabetes

What is meant by monozygosity and dizygosity?

Monozygotic (identical or maternal) twins form from a fertilization of a single ovum that subsequently divides into two separate individuals. **Dizygotic (nonidentical or fraternal) twins** result from fertilization of two separate ova by two separate sperm during a single ovulatory cycle

Monozygotes can either be monoamnionic/monochorionic or diamnionic/monochorionic depending upon the timing of division of embryos

When does division of the embryo occur in these two types of twins?

Conjoined twins: division after 13 days post-fertilization results in conjoined twins where the twins share a single cavity and have one placenta, one chorion, one amnion, and one shared umbilical cord

Monoamnionic/monochorionic placentation occurs with division after amnion formation (between days 8 and 12 postfertilization). These gestations have a single placenta and are at increased risk of cord entanglement during the pregnancy

Diamnionic/monochorionic placentation occurs with division (after trophoblast differentiation and before amnion formation between days 4 and 8 postfertilization). Twins are in separate cavities and have one

placenta, one chorion, two amnions, and two umbilical cords. These gestations are at increased risk for twin-to-twin transfusion syndrome (TTTS) (see Fig. 9-7).



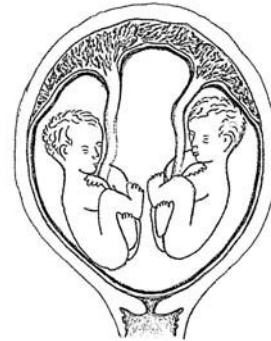
Dichorionic diamniotic



Monochorionic diamniotic



Dichorionic diamniotic



Monochorionic monoamniotic

Figure 9-7 Twin pregnancies.

What type of twinning may occur in dizygotic twins? Explain these types of twins

Dichorionic/diamniotic separate or fused development. In both types, division of embryos occurs before differentiation of the trophoblast (within 3 days of fertilization). In fused development, the chorions may fuse. Sexes may be different in dizygotic twins

What percentage of same sex twins with monochorionic placentas are identical?

100%

What is twin-to-twin transfusion syndrome?

TTTS is a syndrome in which there is unequal flow of blood across the shared placenta, typically resulting

What are several complications of pregnancy that are increased in multi-fetal gestation?

in one twin who is smaller (donor twin) and has decreased amniotic fluid and another twin (recipient twin) who is larger with excess amniotic fluid. Perinatal mortality associated with TTTS is high, and twins who survive are at risk of severe cardiac, brain, or developmental disorders

What is the association between multi-fetal gestation and cerebral palsy?

Spontaneous preterm delivery, intrauterine growth restriction, small for gestational age (SGA), gestational diabetes, hypertension, preeclampsia, pulmonary embolism, pulmonary edema, and acute fatty liver (which is marked by severe coagulopathy, hypoglycemia, and hyperammonemia) are increased among multi-fetal gestations

There is a three-fold greater risk of cerebral palsy. Causes that may contribute to this risk include low birth weight, congenital anomalies, cord entanglement, preterm delivery, and abnormal vascular connections

What technique is used to reduce the number of fetuses in a multi-fetal gestation?

Fetal reduction or selective fetal termination

Preterm delivery is a common and serious complication of multiple births. What are some methods to predict pre-term delivery?

Cervical length measurement by ultrasound and the presence of fetal fibronectin in cervical-vaginal fluids can predict those at highest risk for preterm delivery

How may a multiple gestation pregnancy be prolonged?

While there are no proven methods to delay delivery in multi-fetal pregnancies, a prophylactic cerclage may benefit those with proven cervical insufficiency, decreasing strenuous activity levels, and closer monitoring may have small benefits in these gestations

How is preterm labor managed in multiple gestation?

Judicious use of tocolytics is recommended with the administration of antenatal corticosteroids to accelerate fetal lung maturity

Abnormal Labor and Delivery

DYSTOCIA

What is dystocia and what is its incidence?	Abnormal labor; occurs in approximately 25% of nulliparous women
What are the risk factors for abnormal labor?	Advanced maternal age Non-reassuring fetal heart tracing Epidural anesthesia Macrosomia Occiput posterior position Nulliparity Short stature High station with full dilatation Chorioamnionitis Post-term pregnancy Obesity
What are the three major categories of causes of dystocia?	Problems with the three “P”s: power , passage , and passenger
What is meant by abnormal power?	Inadequate or uncoordinated contractions
What is meant by abnormal passage?	Abnormal size or shape of maternal pelvis leading to cephalopelvic disproportion (a disproportion between the fetal head and maternal pelvis)
What is meant by abnormal passenger?	Malposition, malpresentation, macrosomia, or multiple gestations
What are the two major categories of failure to progress?	Protraction disorders —slower than normal progress Arrest disorders —complete cessation of progress
Name and describe two of the major causes of failure to progress	Uterine hypocontractility: the most common cause of failure to progress; refers to uterine activity that is not strong enough or is not coordinated enough to dilate the cervix; quantified as uterine contractions <200 Montevideo units

	Epidural anesthesia: leads to an increased duration of the first and second stages of labor and an increased incidence of fetal malposition	
What are the criteria for the diagnosis of abnormal labor in each of these categories?		
Duration of labor:	Nullipara >24.7 hours	Multipara >18.8 hours
Protracted dilation:	<1.2 cm/hr	<1.5 cm/hr
Arrest of descent (with epidural):	>3 hours	>2 hours
Arrest of descent (without epidural):	>2 hours	>1 hour
When is an arrest of dilation diagnosed?	Cessation of dilation after 4 cm or more despite adequate uterine contractions (>200 Montevideo units for 2 hours or more)	
How is poor progression in the first stage of labor managed?	With an amniotomy (if membranes are intact) and/or oxytocin (for hypocontractile uterine activity)	
If protraction persists despite these interventions, how should the patient be managed?	Cesarean delivery—she is in active phase arrest	
What is a prolonged latent phase?	A latent phase (in the first stage of labor) of over 20 hours for a nullipara or 14 hours for a multipara	
What are the risks associated with a prolonged latent phase?	Increased risks of cesarean delivery Newborn requiring NICU admission Thick meconium Depressed apgars	
What are the risk factors for poor progression in the second stage of labor?	Nulliparity Diabetes Macrosomia Epidural anesthesia Use of oxytocin Chorioamnionitis	
How is poor progression in the second stage of labor managed?	Fetal surveillance and expectant management or active pushing by the patient	
How is a hypocontractile uterus treated?	With oxytocin	
What is assisted vaginal delivery?	Also known as operative vaginal delivery, it involves the use of forceps or a vacuum device	

<p>What fetal position is most associated with abnormal labor?</p>	<p>Occiput posterior</p>
<p>How is occiput posterior position managed?</p>	<p>Most spontaneously rotate. If it doesn't, it can be managed with manual/instrumental rotation to occiput anterior, operative vaginal delivery, or spontaneous delivery in the occiput posterior position</p>
<p>What types of abnormal presentations are possible and what are the relative incidences of each?</p>	<p>Face (~1/700) Brow (~1/1400) Breech (~1/30) Compound (~1/1500)</p>
<p>Describe face presentation</p>	<p>The fetal neck is sharply extended, causing the face to lead into the birth canal</p>
<p>What are the risk factors for face presentation?</p>	<p>Cephalopelvic disproportion Macrosomia Contracted maternal pelvis Platypelloid pelvis Multiparity Abnormal fetal head (e.g., anencephaly)</p>
<p>What are the three types of breech presentation?</p>	<p>Frank breech: fetus has hips flexed and knees extended (feet near head) Complete breech: fetus has hips and knees flexed Footling/incomplete breech: fetus has one or both feet present below the buttocks (see Fig. 9-8)</p>
<p>How common is breech presentation?</p>	<p>Approximately 3–4% of fetuses at term are breech (increased rates at earlier gestational ages)</p>
<p>What are some of the factors that affect presentation?</p>	<p>Uterine shape (fibroids, placenta previa, poly/oligohydramnios, müllerian anomaly, etc.) Fetal shape (anomalies such as anencephaly) Fetal mobility (asphyxia, impaired growth, fetal structural malformation, fetal chromosomal anomaly, etc.)</p>
<p>How is breech presentation diagnosed?</p>	<p>With abdominal palpation and ultrasound</p>

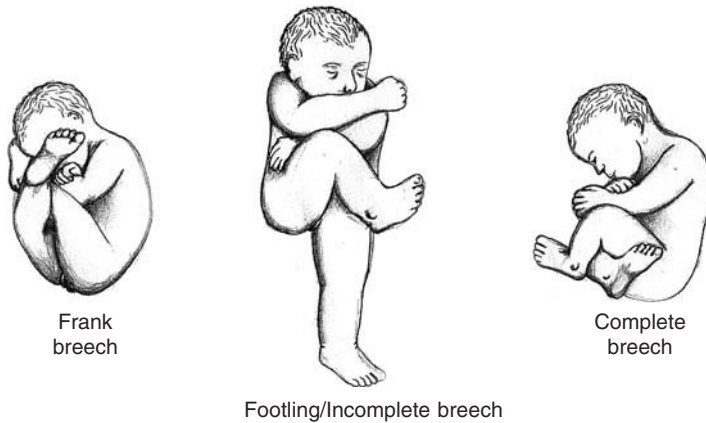


Figure 9-8 Breech presentations.

What are the options for management of a breech presentation near term?

External cephalic version
 Cesarean delivery
 Vaginal delivery (rarely done)

What is external cephalic version?

A procedure that externally rotates the fetus from the breech presentation to the cephalic presentation

When is external cephalic version done?

After 36 weeks

What is the success rate of external cephalic version?

Approximately 65%

What is a transverse lie?

When the fetus's longitudinal axis is perpendicular to the long axis of the uterus

What are the two types of transverse lie?

Back down: fetal back facing toward the cervix
 Back up: fetal back facing away from the cervix

What is the incidence of transverse lie?

1 in 300 deliveries. Many more are transverse early in gestation, but convert spontaneously before term

What are the options for the intrapartum management of transverse lie?

Cesarean delivery
 External cephalic version

What potential problems can be associated with transverse lie?

Placenta previa
 Prolapsed umbilical cord
 Fetal trauma
 Prematurity

ABNORMAL FETAL TESTING

<p>What causes non-reassuring fetal testing?</p>	<p>Fetal hypoxia or acidosis, which in turn can be caused by:</p> <ul style="list-style-type: none"> Maternal diseases (e.g., hypotension, hypoventilation) Placental problems (e.g., insufficiency, abruption) Uterine factors (e.g., hyperstimulation, uterine rupture) Fetal factors (e.g., umbilical cord prolapse, arrhythmia, infection)
<p>What is the fetal response to transient hypoxia?</p>	<p>Initially, slowing of the fetal heart rate resulting in decelerations</p>
<p>What are the fetal responses to prolonged hypoxia?</p>	<ul style="list-style-type: none"> Persistent bradycardia Repetitive late decelerations Loss of heart rate variability Loss of fetal biophysical activities (low BPP) (see Fig. 9-9)
<p>What is a reassuring fetal heart rate pattern?</p>	<ul style="list-style-type: none"> Rate between 110 and 160 beats per minute (bpm) with accelerations No decelerations Variability between 6 and 25 bpm
<p>Fetal acidosis is suggested by what findings on a fetal heart tracing?</p>	<ul style="list-style-type: none"> Decelerations: prolonged (>2 minutes and <10 minutes) decelerations, late decelerations, or periodic severe variable decelerations Minimal (<5 bpm) or absent long-term variability Tachycardia (>160 bpm for more than 10 minutes) or bradycardia (<110 bpm for more than 10 minutes)
<p>How is fetal acidosis directly assessed?</p>	<p>Blood is sampled from fetal presenting part</p>
<p>What is the most common cause of fetal tachycardia?</p>	<p>Maternal tachycardia</p>
<p>What are some other causes of fetal tachycardia?</p>	<ul style="list-style-type: none"> Maternal fever Fetal anemia Asphyxia Infection Autoimmune disorders

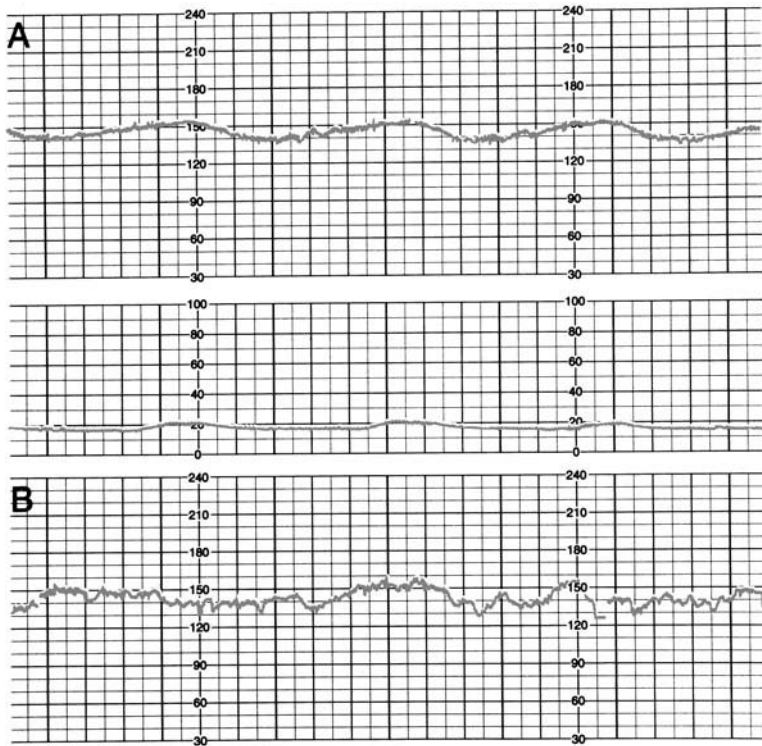


Figure 9-9 Antepartum fetal heart rate tracings at 28 weeks' gestation in woman with diabetic ketoacidosis. A. During maternal and fetal acidemia B. Return of normal accelerations. (Reproduced, with permission, from Cunningham FG et al: *Williams Obstetrics*, 22nd ed. New York: McGraw-Hill, 2005:378.)

What is fetal bradycardia?

What are some of the causes of fetal bradycardia?

Adrenergic medications

Fetal cardiac arrhythmia (e.g., sinoventricular tachycardia [SVT])

A baseline FHR <110 bpm

Physiologic (short episodes—because of transient compression of the fetal head/umbilical cord)

Maternal hypotension

Local anesthesia (e.g., paracervical block)

Uteroplacental insufficiency (e.g., placental abruption, uterine rupture, cord prolapse)

Fetal cardiac arrhythmia

What are the different classifications of fetal heart rate variability?	Absent Minimal (<5 bpm) Moderate (6–25 bpm) Marked (>25 bpm)
What is suggested by a sinusoidal pattern on electronic fetal monitoring (EFM)?	Fetal anemia, hypoxia, or exposure to sedative hypnotics
How can chronic fetal stress be manifested?	Oligohydramnios (AFI <5 cm or maximum vertical pocket <2 cm), decrease in BPP score, intrauterine growth restriction, or abnormal umbilical artery doppler assessment
During hypoxic stress, where is fetal blood preferentially shunted?	Brain, heart, adrenals, and placenta
How is feto-placental perfusion assessed with doppler ultrasound?	Systolic versus end diastolic flow velocity (S/D flow) in the umbilical vessels, middle cerebral artery, and ductus venosus
What is a normal umbilical artery S/D ratio?	It is gestational age-dependent. At term, <3
What is a high umbilical artery S/D ratio associated with?	Placental insufficiency
What are the three types of decelerations?	Early, late, and variable
Describe early decelerations	A gradual decrease from baseline that mirrors a contraction with the nadir of the heart rate at the same time as the peak of the contraction
What do early decelerations signify?	A vagal response from compression of the fetal head during uterine ctx; they are normal
Describe variable decelerations	A rapid decline of more than 15 beats from the baseline. The shape and timing of the deceleration is variable. They may or may not occur with contractions
What do variable decelerations signify?	Usually cord compression (can be relieved by changing the mother's position)
How are variable decelerations managed?	If mild or moderate: expectant management If severe and periodic: move mother to left or right lateral decubitus position; consider stopping oxytocin

What is an amnioinfusion?	and starting amnioinfusion to alleviate cord pressure Catheter administration of sterile saline into the uterine cavity
Describe late decelerations	A gradual decrease from baseline with an onset, nadir, and recovery after the beginning, peak, and end of a contraction
What do late decelerations signify?	Fetal hypoxia
How are late decelerations managed?	Move mother to left or right lateral position Supplemental O ₂ Stop oxytocin (and potentially start tocolytics) Increase IV hydration Monitor maternal BP to ensure maternal hypotension is not the cause Fetal blood sampling to assess for acidosis (if available)
What are the indications for operative delivery because of fetal distress?	Fetal acidosis as determined by fetal scalp sampling Persistent late decelerations or repetitive severe variable decelerations in a tracing without reassuring features (i.e., accelerations)

MECONIUM

What is meconium?	Fetal feces released into the amniotic fluid
How often is meconium noted before or during delivery?	10–15% of births
What are some of the causes of meconium passage in utero?	Placental insufficiency Cord compression Infection
What is meconium aspiration syndrome (MAS)?	A chemical pneumonitis caused by inhalation of meconium into the fetal tracheobronchial tree during the antepartum or intrapartum period
What are some of the potential complications of MAS?	Respiratory distress, persistent pulmonary hypertension, death

POST-TERM PREGNANCY

What is the definition of post-term pregnancy?	Also called prolonged pregnancy; a pregnancy beyond 42 weeks gestation
What is the incidence of post-term pregnancy?	Approximately 7–10% in the United States
What are factors associated with post-term pregnancy?	Incorrect dating Primiparity Prior post-term pregnancy Fetal congenital anomaly Placental sulfatase deficiency
What are the risks to the fetus in post-term pregnancy?	Perinatal death Intrauterine infection Macrosomia and associated risks Asphyxia Fetoplacental insufficiency Anencephaly Fetal dysmaturity syndrome
What is fetal dysmaturity syndrome?	Also known as postmaturity syndrome , it is placental insufficiency resulting in wrinkly, peeling skin (especially on palms and soles); long, thin body—wasting of subcutaneous tissue; alert appearance with open eyes; long nails
What are the fetal risks of post-term pregnancy?	Oligohydramnios (and subsequent umbilical cord compression) Non-reassuring fetal heart tracing Meconium aspiration Complications at birth (hypoglycemia, seizures, respiratory insufficiency) Long-term neurologic sequelae
What are the maternal risks of post-term pregnancy?	Labor dystocia Macrosomia-related perineal injury Increased rate of cesarean delivery
How should post-term pregnancies be monitored?	With antenatal fetal monitoring (either via NST and AFI, BPP, or oxytocin challenge test) twice

When should delivery be entertained?	weekly beginning at 41 weeks gestation At 41 weeks of gestation if the cervix is favorable After 42 completed weeks or earlier if there is evidence of fetal compromise or oligohydramnios
What factors must be considered when deciding whether to induce?	Results of antepartum fetal assessment Favorability of the cervix Gestational age Maternal preference
Are there any long-term consequences of post-term pregnancy?	No

Teratogens

What is the definition of a teratogen?	An agent that acts during the embryonic or fetal development to produce structural abnormalities in the fetus
What percentage of congenital abnormalities are caused by teratogens?	Approximately 10%
What properties of drugs allow their toxic effects on fetuses to occur?	Lipid-soluble molecules readily cross the placenta compared to water-soluble substances Molecules bound to carrier proteins are less likely to cross the placenta
What is meant by embryopathy versus fetopathy?	Embryopathy refers to exposure to a teratogen within the first 8 weeks, whereas fetopathy refers to exposure after 8 weeks
What is the general effect of teratogens on each of the three developmental periods of gestation?	Gestation is divided into three periods known as: 1. Preimplantation period —the period from fertilization to implantation or bilaminar disk formation. An insult causing damage to a large

number of cells at this period usually causes death of the embryo (all or nothing phenomenon)

2. **Embryonic period**—the period from the second through the eighth week following conception. This period is the critical stage of organ development (see Fig. 9-10) and is most crucial in regards to structural malformations
3. **Fetal period**—the period where maturation and functional development continue after 9 weeks. Certain organs, such as the brain, remain vulnerable to teratogens during this period

What are the FDA Pregnancy Drug Categories?

Category A: safety has been established using human studies

Category B: presumed safety based on animal studies

Category C: uncertain safety; though animals studies show an adverse effect, there are no human studies

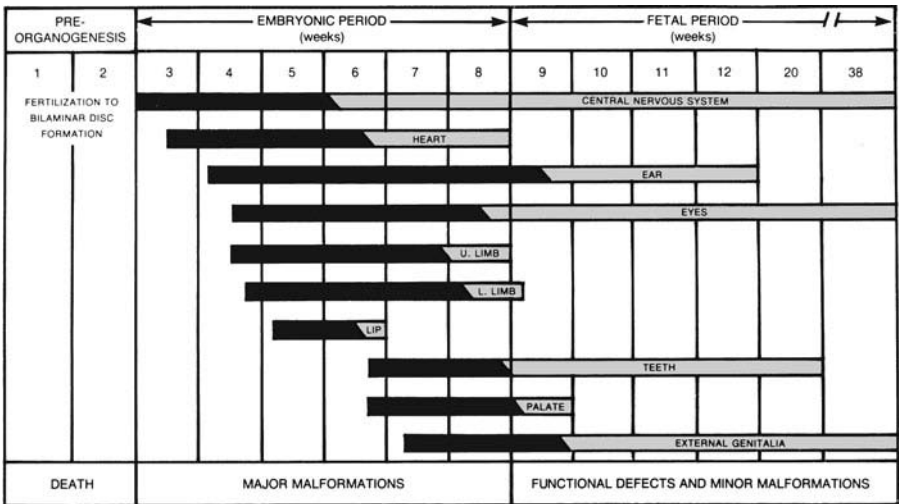


Figure 9-10 Illustrates the critical development period of each organ system.

Category D: unsafe—evidence of risk that may in certain clinical circumstances be justifiable

Category X: highly unsafe—risk outweighs any possible benefit

What are the major abnormalities associated with each known teratogen?

See the following table.

Teratogen	Syndrome and/or Well-Known Effects of the Teratogen	Other Comments
Alcohol	Fetal alcohol syndrome: congenital heart and brain defects, failure to thrive, developmental delay, mental retardation, ADHD, craniofacial anomalies (absent/hypoplastic philtrum, broad upper lip, micrognathia, microphthalmia, short palpebral tissues, short nose). Growth restriction before and after birth	A safe threshold dose for alcohol has not been established. Fetuses of women who consume six drinks/day are at 40% risk of having characteristics of fetal alcohol syndrome
Androgens and testosterone derivatives (i.e., danazol)	Virilization of females; advanced genital development in males	Effects are dose-dependent and related to the time of exposure during the developmental period
Phenytoin	Fetal hydantoin syndrome: craniofacial anomalies (upturned nose, mild midfacial hypoplasia, thin philtrum, facial clefts), fingernail hypoplasia, growth deficiency, developmental delay, cardiac defects	Syndrome results from accumulation of free oxide radicals in fetal tissues
Carbamazepine	Fetal hydantoin syndrome, spina bifida, fingernail hypoplasia, IUGR	
Valproate	Neural-tube defects	Lowers fetal folate levels
Phenobarbital	Clefts, cardiac anomalies, urinary tract malformations	Lowers fetal folate levels
Trimethadione, paramethadione	Cleft palate, V-shaped eyebrows, microcephaly, growth deficiency, mental retardation, speech disturbance, cardiac defects	70% of newborns affected by this drug
Warfarin	<i>Exposure between 6–9 weeks:</i> nasal and midface hypoplasia, stippled vertebral and femoral epiphyses, phenotypically identical to chondrodysplasia punctata	Fetal malformations are significant when doses exceeding 5 mg are taken during the first semester. The

(Continued)

Teratogen	Syndrome and/or Well-Known Effects of the Teratogen	Other Comments
ACE inhibitors	<p data-bbox="314 283 663 455"><i>Exposure between second and third trimester:</i> hemorrhage, organ scarring, agenesis of the corpus callosum, Dandy-Walker malformation, optic atrophy, blindness, mental retardation</p> <p data-bbox="314 462 663 634">Renal ischemia, renal tubular dysgenesis, anuria, oligohydramnios, lung hypoplasia, limb contractures, hypoperfusion, growth restriction, limb shortening</p>	<p data-bbox="713 283 986 342">incidence of spontaneous abortion is 72%</p> <p data-bbox="713 462 986 634">These changes mostly occur during the fetal period, ACE inhibitor fetopathy. Enalapril is the most teratogenic of the ACE inhibitors</p> <p data-bbox="713 641 986 718">Vitamin A doses higher than 5000 IU should be avoided</p> <p data-bbox="713 725 986 784">No safe first trimester exposure period or dose</p>
Retinol-vitamin A	No conclusive data available for dose toxicity and birth defects	Vitamin A doses higher than 5000 IU should be avoided
Isotretinoin	<p data-bbox="314 725 680 784">First trimester use: high rate of fetal loss</p> <p data-bbox="314 792 680 964">Strongly associated malformations include bilateral microtia, agenesis or stenosis of the external ear canal, cleft palate, abnormal facial bones/calvarium, cardiac defects, hydrocephalus.</p>	No safe first trimester exposure period or dose
Diethylstilbestrol (DES)	Clear-cell adenocarcinoma of the cervix and vagina and abnormalities of the female genital tract (T-shaped uterus)	
Cyclophosphamide	Absent or hypoplastic digits on the hands and feet, cleft palate, single coronary artery, imperforate anus, fetal growth restriction with microcephaly	Should be avoided in the first trimester but can be given in the second and third trimester
Methotrexate	Growth restriction, craniosynostosis, failure of calvarial ossification, hypoplastic supraorbital ridges, micrognathia, and severe limb deformities	Drug alters folate metabolism. Used as an abortifacient. Should not exceed a dose >10 mg/week Contraindicated for treatment of psoriasis
Tetracyclines	Yellow-brown discoloration of teeth, hypoplasia of tooth enamel	Effects occur if drug is used in second or third trimester. Should only be used for tx of maternal syphilis in penicillin-allergic women

(Continued)

Teratogen	Syndrome and/or Well-Known Effects of the Teratogen	Other Comments
Aminoglycosides Streptomycin, kanamycin	Nephrotoxicity and ototoxicity in preterm newborns treated with gentamicin or streptomycin Hearing loss, eighth nerve damage	No confirmation regarding congenital defects from prenatal exposure No ototoxicity has been reported with use of gentamicin or vancomycin
Lead Tobacco	Increased abortion rates, stillbirths Dose-dependent intrauterine fetal growth reduction, spontaneous abortion, preterm delivery, placenta previa, cleft lip/palate, hydrocephaly, microcephaly, omphalocele, gastroschisis, and hand abnormalities, sudden infant death syndrome (SIDS)	Cessation of smoking throughout and after pregnancy should be strongly advised
Cocaine	Placental abruption, porencephaly, subependymal and periventricular cysts, ileal atresia, cardiac anomalies, urinary tract defects, and visceral infarcts	
Thalidomide	Upper limb reduction, lower limb reduction, gall bladder aplasia, duodenal atresia	Excellent example shows timing of drug exposure and type of birth defect
Methyl mercury	Ranges from developmental delay to microcephaly and severe brain damage	Not a drug but a major pollutant found in fish. Pregnant women should abstain from seafood thought to be exposed to mercury
Marijuana	Possible low birth weight; otherwise, no evidence of association with human anomalies	
Amphetamines	Symmetrical fetal growth restriction, SIDS	
Heroin	Fetal-growth restriction, perinatal death, small head circumference, developmental delays, SIDS	40–80% of newborns have typical heroin withdrawal symptoms
Lithium	Ebstein anomaly, diabetes insipidus, hypothyroidism, and hypoglycemia	

Can heparin be used for anticoagulation in pregnant women?

Yes. Heparin does not cross the placenta because of its large negative charge and thus does not have any adverse fetal effects. Low-molecular weight heparins also do not cross the placenta

Has paternal exposure to these drugs been found to be teratogenic?

There is no evidence for this

SECTION IV

Women's Health Issues

This page intentionally left blank

Issues in Women's Health

DOMESTIC VIOLENCE

Describe the epidemiology of domestic violence?

When violence occurs within the household, 90–95% of the victims are women. Annually, nearly 5 million women are victims of domestic or intimate partner violence and **one-fifth of American women will be abused by an intimate partner within their lifetime**

Domestic or intimate partner violence perpetuated against women in family or intimate relationships typically follows a predictable cycle. Describe the three stages of the cycle of violence

The three phases of the cycle include the following: **Tension-building phase**—usually contains intense arguing and blaming
Battering phase—characterized by verbal threats, physical battering, sexual abuse, or assault with weapons

Honeymoon phase—is characterized by the abuser's attempt to apologize, deny, or offer gift compensation for previous violence. With time, the tension-building phase gets longer and more frequent, and the honeymoon phase gets shorter and less frequent

What are four major social risk factors for domestic violence?

Although domestic violence spans all socioeconomic groups, **poverty, unemployment, alcohol, and substance abuse** tend to be highly associated with a history of domestic violence

How do battered women often present for medical care?

Women who are being battered present for medical care with a wide variety of complaints, ranging from **sexual dysfunction** (decreased interest or arousal, dyspareunia, etc) and **persistent somatic complaints** (headaches, abdominal pain, sleep or eating disorders) to **psychiatric illnesses** (depression, post-traumatic stress disorder [PTSD], or multiple personality disorder). Because there is no pathognomonic presentation of domestic violence, many cases go undiagnosed

* Other factors that should trigger practitioners to inquire regarding possible violence in the home include apparent **noncompliance, frequent emergency room visits, or frequently cancelled appointments**

What screening questions can be asked of all women to increase the likelihood of diagnosing domestic violence despite its various presentations?

Women presenting for routine preventative care or urgent care visits should be routinely screened for domestic violence regardless of their socioeconomic background

Stage 1 screening should be directed at specific behaviors. Sample questions include:

Has anyone close to you ever threatened to hurt you?

Has anyone ever hit, kicked, choked, or hurt you physically?

Has anyone, even your partner, ever forced you to have sex against your will?

Are you afraid of your partner?

What questions should be asked after violence has been determined to be present in a household?

Stage 2 screening should **assess safety and lethality of violence and develop a safety plan**. Patients who are unsafe at home should be offered shelter.

Has your partner ever threatened to kill you or your children?

Are there weapons in the house?

Does your partner abuse drugs or alcohol?

Is it safe for you to go home?

Are your children safe at home?

What agencies should be utilized in the referral of a patient who is a victim of domestic violence?

Contact information for local police and emergency departments, women's shelters, rape crisis centers, counseling services, self-help, and advocacy agencies should be given to battered women

SEXUAL ASSAULT

What is sexual assault?

Sexual assault is the performance of genital, anal, or oral penetration by one person on another without the person's consent

Describe the epidemiology of sexual assault?

Although some authors estimate that less than 50% of sexual assaults are reported, **nearly 1 million women are sexually assaulted annually.**

Furthermore, 20% of adult women, 15% of college-age women, and 12% of adolescents have been sexually assaulted in their lifetime

Sexual assault can happen under a variety of conditions and relationships. Describe four special variants of sexual assault

Marital rape is forced sexual acts within a marital relationship without the consent of a partner. **Acquaintance rape** is sexual assault committed by someone known to the victim. **Incest** involves sexual assault perpetuated by a family member. **Date rape** is sexual assault occurring in the context of a dating relationship

What is statutory rape?

Statutory rape occurs when an adult has intercourse with a minor, whose age makes him or her legally incapable of consenting to sexual intercourse. Many states mandate physician reporting of statutory rape. Legal definitions for a minor, or someone at the age of consent, vary depending on jurisdiction

What is child sexual abuse?

Child sexual abuse is any contact between a child and an adult where the child is being used for sexual stimulation of the adult. This type of behavior must be immediately reported to child protection services

Date rape drugs diminish a woman's ability either to consent to sexual activity or remember an assault. What drugs are considered date rape drugs?

Many of the **benzodiazepines**, because of their sedative/hypnotic properties and their propensity to cause amnesia can be considered to be date rape drugs. Currently, **flunitrazepam (Rohypnol)** and **gamma-hydroxybutyrate (GHB)** are the two most frequently used date rape drugs; however, ketamine, chloral hydrate, and MDMA (Ecstasy) have the potential to be abused in this manner

What is the Rape Trauma Syndrome (RTS) and how does it differ from PTSD?

Rape Trauma Syndrome is a biphasic PTSD-like condition that occurs within hours to days after a sexual assault and can persist for months to years. In the acute or disorganization stage, which occurs over 2 weeks following a sexual assault and can have a cyclical relapsing-remitting presentation, the victim's coping mechanisms are impaired leading to either an emotionally labile, expressive catharsis or a controlled, emotional detachment. In the late or reorganization phase, minimal symptoms of PTSD emerge, but do not disrupt the victim's life as in PTSD. During this phase, some victims experience nightmares, flashbacks, feelings of alienation and isolation, depression, and anxiety

After obtaining informed consent to do a careful history and full, chaperoned physical examination, what specimen will be collected from the patient to look for DNA evidence to identify the perpetrator?

Victim's clothing
 Air-dried swabs and smears from the oropharynx, vagina, and rectum
 Cervical mucus for a Pap smear
 Washings from the skin and vagina
 Combed specimen from scalp and pubic hair with control samples of the victim's hair from each site
 Fingernail scrapings and clippings
 Whole blood samples
 Saliva samples
 The patient should be counseled regarding the ability to photograph

any physical findings. The consent process for taking pictures should include a discussion of the disposition and confidentiality surrounding these photographs

What substances contained within the collected specimen will be used as evidence of sexual assault and to identify the perpetrator?

Motile and nonmotile sperm and hair to provide evidence of sexual assault and for DNA

Acid phosphatase to provide evidence of sexual assault

The following are conditions and their prophylactic treatments the victim should receive immediately after verifying the patient's allergy history:

Condition	Treatment
<i>Chlamydia</i>	Azithromycin (1 g PO) or Doxycycline (100 mg, PO, bid × 7 days)
Gonorrhea	Ceftriaxone (125 mg, IM)
Trichomoniasis	Metronidazole (2 g, PO × 1 or 500 mg bid, PO × 7 days)
Hepatitis B	Hepatitis B immune globulin and Hepatitis B vaccine (0, 1, and 6 months)
HIV	Low risk (basic regimen): ZDV (TDF, d4T or ddI) and 3TC or FTC High risk: basic regimen and either LPV/RTV, SQV/RTV, NFV, ATV ± RTV, IDV ± RTV, or FPV ± RTV
Pregnancy	Yuzpe (100 mcg ethinyl estradiol, 0.5 mg levonorgestrel): Q12H × 2 Plan B (0.75 mg levonorgestrel): Q12H × 2 doses Mifepristone (RU 486): 600 mg, PO

ZDV, zidovudine; TDF, tenofovir; d4T, stavudine; ddI, didanosine; 3TC, lamivudine; FTC, emtricitabine; LPV, lopinavir; RTV, ritonavir; SQV, saquinavir; NFV, nefinavir; ATV, atazanavir; IDV, indinavir; FPV, fosamprenavir

ETHICS AND LAW

Two forms of advanced directives include the living will and a durable power of attorney for health care. These structures allow patients to state their preferences for future medical treatment in the event of loss of capacity because of medical illness. How does the living will differ from the power of attorney?

The living will offers a competent adult patient a means to express her wishes and offer informed consent governing the use of life-sustaining treatments in writing in advance of a medical condition that leads to incapacity or incompetence. However, in making a power of

<p>The doctrine of informed consent requires physicians to obtain the patient's permission for treatment, operations, or some diagnostic procedures. What conditions need to be met in order to validate the patient's offering of consent?</p>	<p>attorney, the patient appoints a surrogate decision maker to stand in her place and express her wishes or give informed consent in the event of incapacity or incompetence</p>
<p>Under what circumstances is the requirement of informed consent waived?</p>	<p>A valid informed consent must be voluntarily granted by a competent patient who has full comprehension of the risk, benefits, alternatives, and consequences of the relevant, available diagnostic or treatment options</p> <p>Informed consent is often not required before administering treatment or performing lifesaving procedures in a medical emergency, preventing suicide, or attending to minors in the absence of a parent</p>
<p>In general, the information disclosed by a patient during a physician consultation is strictly confidential, and thus should not be revealed without the patient's consent, unless disclosure is required by law. Under what circumstances is the maintenance of strict patient confidentiality not required?</p>	<p>Patient confidentiality can be broken under the following circumstances:</p> <p>When a patient discloses an intention to inflict serious bodily harm on herself or another person</p> <p>In the event of a life-threatening emergency</p> <p>When a reportable, communicable disease has been diagnosed</p> <p>However, in these and other cases, disclosure is permitted only to those for whom it is medically or legally necessary</p>
<p>Laws governing malpractice are formed by two mechanisms, from legislative action or from judicial opinion rendered during precedent cases. Under which type of law do most malpractice claims proceed?</p>	<p>Most malpractice claims proceed under the body of law defined by judicial opinion derived from precedent cases, or common law. A dynamic body of law, common law is constantly changing and thus continually redefining the grounds for potential litigation</p>
<p>What are the four principles that must be proven in order to establish medical liability in a negligence suit?</p>	<p>Medical liability requires the plaintiff to demonstrate the presence of duty, breach of duty, causation, and consequent damages. Causation is defined as the link between the alleged breach of duty and an injury</p>

Identify the elements needed to prove malpractice in a wrongful birth and wrongful conception claim

and must be supported by proof of causation. Damages constitute demonstrable injuries and can either be purely economic (e.g., lost wages) or noneconomic (e.g., pain and suffering)

In a wrongful birth claim, a clinician interviewing a pregnant couple (duty) omits the family history and fails to recognize a serious disability that has a hereditary or genetic basis (breach of duty). Consequently, a baby with appreciable disabilities is born (damages) to parents who would have sought termination, but for the clinician's failure which prevented proper counseling. In a wrongful conception claim, a clinician treating a nonpregnant couple (duty) fails to provide histologic evidence of sterilization during a tubal ligation or provides improper contraceptive counseling or techniques (breach of duty). Consequently, a normal but unwanted child is born (damages) to the couple seeking sterilization or effective contraception

This page intentionally left blank

SECTION V

Review Questions and Answers

This page intentionally left blank

Review Questions and Answers

- (1) At which of the following time periods of zygotic division does the formation of dichorionic, diamniotic twins occur following fertilization? Answer: d
- (a) >264 hours
 - (b) >120 and \leq 240 hours
 - (c) >72 and \leq 120 hours
 - (d) \leq 72 hours
- (2) Which surgical procedure is most commonly performed in the second trimester of pregnancy? Answer: d
- (a) Ovarian cystectomy
 - (b) Cholecystectomy
 - (c) Laparoscopy
 - (d) Appendectomy
- (3) A 21-year-old female presents to the emergency room (ER) stating that she “was raped.” Which medications should she be given for prevention of sexually transmitted diseases (STDs) in this situation? Answer: a
- (a) Ceftriaxone, azithromycin, and metronidazole
 - (b) Ceftriaxone plus cefixime
 - (c) Ceftriaxone plus azithromycin
 - (d) Ceftriaxone plus penicillin

- (4) Maternal obesity is a risk factor for all of the fetal complications *except*: Answer: c
- (a) fetal macrosomia
 - (b) neural-tube defects
 - (c) dizygotic twinning
 - (d) stillbirth
- (5) Your pregnant patient has mitral stenosis and is New York Heart Association functional class II. Which of the following sets of vaccinations, cultures, and antibiotics is indicated during her pregnancy? Answer: b
- (a) Influenza, pneumococcal, intrapartum bacterial endocarditis prophylaxis
 - (b) Influenza, pneumococcal, group B streptococcal vaginal and rectal culture at 36 weeks
 - (c) Influenza, pneumococcal, intrapartum bacterial endocarditis prophylaxis, group B streptococcal vaginal and rectal culture at 36 weeks
 - (d) Influenza and pneumococcal vaccine
- (6) Your patient states that she is comfortable at rest but begins to experience shortness of breath and chest pain after walking three blocks. What New York Heart Association classification would you assign her? Answer: b
- (a) I
 - (b) II
 - (c) III
 - (d) IV
- (7) Which of the following analgesics is a non-histamine-releasing narcotic and therefore should be used for asthmatics? Answer: c
- (a) Fentanyl
 - (b) Codeine

(c) Morphine

(d) Meperidine

(8) Your current patient is pregnant and has a history of deep vein thrombosis (DVT) in her previous pregnancy. How should she be managed during this pregnancy? Answer: c

(a) Low-dose aspirin

(b) Careful observation

(c) Mini-dose subcutaneous heparin or LMWH

(d) Full prophylactic dose subcutaneous heparin or LMWH

(9) Your patient is 18 weeks pregnant and presents with idiopathic hematuria. Which of the following outcomes is she at increased risk of developing? Answer: a

(a) Preeclampsia

(b) Pyelonephritis

(c) Chronic renal disease

(d) Preterm labor

(10) Which of the following pregnancy complications is most common in women with chronic renal insufficiency? Answer: b

(a) Fetal growth restriction

(b) Anemia

(c) Preeclampsia

(d) Preterm delivery

(11) During a first trimester surgical abortion, signs of complete evacuation include all of the following except: Answer: d

(a) Gritty sensation

(b) Contraction around the uterus

(c) Bubbles in the cannula and hose

(d) Bleeding from the os

- (12) Typically, postabortal infections present: Answer: b
- (a) within the first 24–36 hours
 - (b) within the first 48–96 hours
 - (c) within the first 6 days
 - (d) after at least 1 week post-procedure
- (13) During a suction dilation and curettage (D & C), the surgeons note a midline perforation. If the uterus is not completely evacuated, all of the following options may be indicated except: Answer: a
- (a) continue with the same procedure
 - (b) exploratory laparotomy
 - (c) perform ultrasound
 - (d) exploratory laparoscopy
- (14) The method of abortion at 15 weeks' gestation with the least psychologic and emotional impact on the patient is: Answer: c
- (a) intra-amniotic hypertonic saline
 - (b) intra-amniotic prostaglandin
 - (c) dilation and evacuation
 - (d) prostaglandin vaginal suppositories
 - (e) abdominal hysterectomy
- (15) The most common complication arising from a first-trimester surgical pregnancy termination is: Answer: d
- (a) uterine perforation
 - (b) cervical trauma
 - (c) retained products of conception
 - (d) pelvic infection
 - (e) vaginal laceration

- (16) A 21-year-old G0, with a history of irregular menses presents for her first gynecologic examination. Pelvic examination reveals fullness in the right adnexa. The examination is otherwise unremarkable. You obtain a transvaginal ultrasound which reveals a thin-walled 4-cm unilocular, clear fluid-appearing cystic structure in the right ovary. How do you manage this patient?
- Answer: b
- (a) Send tumor markers
 - (b) Follow-up sonogram in 3–6 months
 - (c) Drainage of the cyst via transvaginal approach
 - (d) Laparoscopy and cystectomy
- (17) An 18-year-old female presents to the ER with acute right-sided pain. She reports nausea and vomiting and states that her pain is unrelenting when it is present, though it seems to “come and go” over the last few hours. Her examination is significant for involuntary rebound and guarding. You obtain a transvaginal ultrasound, which reveals an adnexal mass consistent with a 7-cm unilocular structure with both hyperechoic and hypoechoic components. How do you manage this patient?
- Answer: d
- (a) Call general surgery consult for suspected appendicitis
 - (b) Paracentesis of adnexal structure
 - (c) Laparoscopy and oophorectomy
 - (d) Laparoscopy and cystectomy

(18) A 41-year-old G3P3 presents to the ER with complaints of unrelenting lower abdominal pain. She denies nausea and vomiting but reports subjective fevers. In the ER, she is febrile to 100.9. Pelvic examination reveals a mobile 12-week size uterus with point tenderness midline in the lower abdomen, no cervical motion tenderness, no discharge. Urinalysis (UA) is significant for few white cells with many squamous cells. You obtain an ultrasound which reveals a multityomatous uterus with a subserosal anterior myoma of 6-cm size with internal components suggestive of calcification and necrosis; adnexal structures are within normal limits. What is this patient's diagnosis?

- (a) PID
- (b) Cystitis
- (c) Degenerating fibroid
- (d) Appendicitis

Answer: c

(19) How would you manage the patient above?

- (a) Admission and IV antibiotics
- (b) Discharge home and PO antibiotics
- (c) Discharge home with NSAIDs
- (d) Admission and myomectomy
- (e) Admission and hysterectomy

Answer: c

(20) How does progesterone affect various organ systems during pregnancy?

- (a) Lowers diastolic blood pressure less than systolic
- (b) Increases lower esophageal sphincter tone causing painful spasms

Answer: d

- (c) Decreases the central respiratory drive resulting in dyspnea of pregnancy
- (d) Reduces ureteral tone, decreases peristalsis, and relaxes the bladder wall

(21) Why is there an increase in total T_4 and T_3 concentrations in pregnancy? Answer: b

- (a) Pregnancy is a state of physiologic stress with upregulation of all hormones
- (b) β -hCG stimulates TSH receptors
- (c) Liver production of binding globulins is decreased
- (d) Fetal production results in increased maternal concentrations

(22) Which of the following hemodynamic values remains unchanged in pregnancy? Answer: d

- (a) Pulmonary vascular resistance
- (b) Colloid osmotic pressure
- (c) Pulmonary capillary pressure
- (d) Systemic vascular resistance

(23) Which of the following is not a cause of DVT in pregnancy? Answer: d

- (a) Decreased protein S
- (b) Resistance to protein C
- (c) Increased factor I (fibrinogen), II, V, VII, VIII, X, and XII
- (d) Compression of the left iliac vein by the right iliac artery

(24) Which of the following complications in pregnancy is increased in women with a female fetus? Answer: d

- (a) Hepatitis
- (b) Cholelithiasis
- (c) Reflux esophagitis
- (d) Hyperemesis gravidarum

- (25) Your patient is pregnant and is found to be positive for hepatitis C. Which of the following outcomes is associated with hepatitis C? Answer: c
- (a) Abruptio placentae
 - (b) Fetal growth restriction
 - (c) Vertical transmission of hepatitis C
 - (d) Preterm birth
- (26) Which class of gestational diabetes is *not* at increased risk for unexplained stillbirth? Answer: a
- (a) A1
 - (b) A2
 - (c) B
 - (d) C
- (27) What fetal malformation is most strongly associated with diabetes? Answer: c
- (a) Neural-tube defects
 - (b) Congenital heart defects
 - (c) Caudal regression
 - (d) Renal agenesis
- (28) What is the most common etiology of pregnancy-associated osteoporosis? Answer: d
- (a) Bed rest
 - (b) Corticosteroids
 - (c) Heparin therapy
 - (d) Idiopathic
- (29) A pregnant patient presents with symptoms and signs of thyrotoxic storm. All of the following medications are indicated *except*: Answer: b
- (a) potassium iodide
 - (b) magnesium sulfate
 - (c) dexamethasone
 - (d) propylthiouracil

- (30) Your patient has a history of epilepsy and is also found to be pregnant. She refuses to take antiepileptic medications. Of which of the following complications are fetuses at an increased risk? Answer: c
- (a) Fetal growth restriction
 - (b) Congenital malformation
 - (c) Seizure disorder
 - (d) Perinatal death
- (31) What is the most common cause of female sexual dysfunction? Answer: b
- (a) Vaginismus
 - (b) Inhibited sexual desire
 - (c) Arousal disorder
 - (d) Anorgasmia
- (32) Which antidepressant listed below is the least likely to cause sexual dysfunction? Answer: c
- (a) Prozac
 - (b) Zoloft
 - (c) Wellbutrin
 - (d) Effexor
- (33) An 18-year-old female with no history of STIs and who has had two sexual partners presents to her gynecologists' office for her last dose of HPV vaccine. She asks how often she needs to have a Pap smear. The correct response is: Answer: c
- (a) every 3 years
 - (b) every 5 years
 - (c) annually
 - (d) never again—she is now immune to HPV

- (34) A 62-year-old G2P2002 presented for her gynecologic visit and was found to have high-grade squamous intraepithelial lesions (HSIL). A colposcopy was performed and found to be negative; however, the squamocolumnar junction was unable to be visualized. What is the next appropriate step in management?
- Answer: a
- (a) Conization
 - (b) Repeat Pap smear within 3 months
 - (c) Endocervical curettage
 - (d) Biopsy of cervical tissue
- (35) The above patient underwent conization via loop electrosurgical excision procedure (LEEP), which was found to be negative. Which of the following follow-up management is the most appropriate?
- Answer: a
- (a) Repeat Pap smear in 4–6 months
 - (b) Repeat Pap smear in 1 year
 - (c) Repeat colposcopy in 6 months
 - (d) Repeat conization in 6 months
- (36) A 37-year-old female is diagnosed with invasive squamous cell carcinoma of the cervix. She is found to have minimal microscopic stromal invasion that is confined to the cervix. What are the most appropriate treatment options? (Choose all that apply.)
- Answer: a or b
- (a) Conization
 - (b) Simple hysterectomy
 - (c) Radical hysterectomy
 - (d) Radiotherapy
 - (e) Combination chemotherapy
- (37) All of the following increase during pregnancy *except*:
- Answer: c
- (a) tidal volume
 - (b) minute ventilation

- (c) total lung capacity
- (d) alveolar partial pressure of oxygen

(38) What screening test is listed with the appropriate condition? Answer: a

- (a) Maternal serum alpha fetal protein and neural tube defects
- (b) Urine dip and gestational diabetes
- (c) Magnetic resonance imaging and cleft lip
- (d) Percutaneous umbilical blood sampling and Rh isoimmunization

(39) What diagnostic test is listed with the appropriate condition? Answer: b

- (a) Maternal serum alpha fetal protein: aneuploidy
- (b) Amniocentesis: Down syndrome
- (c) Glucose challenge test: gestational diabetes
- (d) Biophysical profile: fetal lung maturity

(40) All of the following cause persistent or increasing levels of β -hCG *except*: Answer: d

- (a) retained products of conception
- (b) trophoblastic disease
- (c) choriocarcinoma
- (d) complete spontaneous abortion

(41) All of the following drugs can be used for the treatment of migraine headaches during pregnancy *except*: Answer: d

- (a) propranolol
- (b) meperidine
- (c) amitriptyline
- (d) ergonovine

- (42) What is the best treatment for syphilis in a penicillin-allergic patient during pregnancy? Answer: a
- (a) Penicillin desensitization
 - (b) Tetracycline
 - (c) Ceftriaxone
 - (d) Erythromycin
- (43) Your pregnant patient presents with chlamydial cervicitis. Which of the following treatments is appropriate? Answer: d
- (a) Tetracycline, 500 mg PO qid × 7 days
 - (b) Ciprofloxacin, 500 mg PO bid × 14 days
 - (c) Erythromycin estolate, 250 mg PO qid × 4 days
 - (d) Erythromycin base, 500 mg PO qid × 7 days
- (44) How does breast-feeding affect the risk of HIV transmission? Answer: a
- (a) Increases
 - (b) Decreases
 - (c) Unaffected
 - (d) Unknown
- (45) Which of the following is increased in pregnancies complicated by sickle-cell trait? Answer: a
- (a) UTI
 - (b) Low birth weight
 - (c) Perinatal mortality
 - (d) Spontaneous abortion

- (46) A 65-year-old female presents to her gynecologist with complaints of progressive vulvar itching and perineal pain. On examination, there is diffuse atrophy with one raised, whitish area on the posterior aspect of the vulva. What is the most appropriate first step in her management?
- Answer: d
- (a) Trial of low-dose corticosteroid cream
 - (b) Trial of high-dose corticosteroid cream
 - (c) Trial of topical antifungal cream
 - (d) Vulvar biopsy of the affected area
- (47) The biopsy results for the above patient demonstrate vulvar intraepithelial neoplasia II (VIN II). What is the most appropriate treatment for her?
- Answer: b
- (a) Chemotherapy
 - (b) Complete local excision
 - (c) Wide excision with laser ablation
 - (d) Chemotherapy followed by localized radiation
- (48) A 72-year-old female presents to her gynecologist with pruritus and soreness of the vulva. On inspection, there are multiple well-demarcated white hyperkeratotic areas on a bright red background. What is the most likely diagnosis?
- Answer: c
- (a) Squamous cell carcinoma of the vulva
 - (b) Basal cell carcinoma of the vulva
 - (c) Paget disease of the vulva
 - (d) Vulvar melanoma

- (49) A 17-year-old female presents to her gynecologist with complaints of cramping lower abdominal pain that begins with menstruation. She also admits to mild nausea and diarrhea around the same time. Her physical examination is unremarkable. What should be used as first-line therapy?
- Answer: a
- (a) NSAIDs
 - (b) Oral contraceptive pills
 - (c) Presacral neurectomy
 - (d) Antispasmodic agents
- (50) A 28-year-old female presents with increasing pelvic pain with menstruation that is not relieved with NSAIDs. Physical examination reveals some uterine immobility as well as tender nodularities in the posterior cul-de-sac. What is the most likely diagnosis?
- Answer: b
- (a) Primary dysmenorrhea
 - (b) Endometriosis
 - (c) Leiomyomas
 - (d) Adenomyosis
- (51) Match each of the following terms to their correct description
- Answers 1-c, 2-a, 3-b
- (1) Prolonged, irregular menstrual bleeding
 - (2) Prolonged, regular menstrual bleeding
 - (3) Irregular menstrual bleeding
- (a) Menorrhagia
 - (b) Metrorrhagia
 - (c) Menometrorrhagia

- (52) What is the fetal lie, presentation, and position if the head is down, flexed, and the fetal back is near the left maternal pelvis? Additionally, on sterile vaginal examination a diamond fontanel is noted near the right ischial spine, a triangular fontanel is the leading edge, and the sagittal suture is not parallel with the floor
- Answer: a
- (a) Longitudinal lie and left occiput anterior (LOA)
 - (b) Longitudinal lie and right occiput anterior (ROA)
 - (c) Transverse lie and left occiput anterior (LOA)
 - (d) Transverse lie and right occiput anterior (ROA)
- (53) On examination, the sagittal suture is deflected toward the sacrum, which allows more of the parietal bone to be palpated anteriorly. What is the term that describes this physical finding?
- Answer: d
- (a) Transverse diagonal
 - (b) Posterior diagonal
 - (c) Posterior asynclitism
 - (d) Anterior asynclitism
- (54) How is zero station determined on sterile vaginal examination?
- Answer: b
- (a) The leading fetal edge is flush with the introitus
 - (b) The leading fetal edge is parallel with the maternal ischial spines
 - (c) The leading fetal edge is engaged in the maternal pelvis
 - (d) The leading fetal edge is engaged in a fully dilated cervix

- (55) A multigravid mother has a history of previous group B streptococcus (GBS)-negative pregnancies, a GBS-positive urinary tract infection during the current pregnancy, and a negative GBS culture at 36 weeks. She presents to labor and delivery with spontaneous rupture of membranes and contractions every 5 minutes. Which of the following is indicated?
- Answer: d
- (a) Immediate urinalysis for signs of current infection
 - (b) Immediate urine culture and rectal swab for identification of GBS status
 - (c) Empiric treatment for unknown GBS status
 - (d) Antibiotics immediately due to history of GBS colonization
- (56) A group B *Streptococcus*-positive G2P1 with a penicillin allergy presents at 38 weeks with loss of fluid for 3 hours and contractions every 5 minutes. What is the next step in management?
- Answer: b
- (a) Place the mother and infant on cardiac monitors
 - (b) Assess for rupture of membranes
 - (c) Begin ampicillin prophylaxis
 - (d) Desensitize patient to penicillin
- (57) How often is meconium noted during labor and what does it signify?
- Answer: d
- (a) Rarely, and only in the presence of anoxic brain injury
 - (b) Often, without any significance
 - (c) Half of the time with variable outcomes
 - (d) Occasionally, and it is suggestive of some degree of fetal stress

- (58) Your patient, a 21-year-old G2P1 with a prior cesarean delivery and currently with a singleton gestation, is found to have placenta previa. Which of the following is not associated with an increased incidence of placenta previa?
- (a) Advanced maternal age
 - (b) Grand multiparity
 - (c) Prior cesarean delivery
 - (d) Singleton gestation
- Answer: d
- (59) Your patient presents at 30 weeks with complaints of vaginal bleeding. There is currently no active vaginal bleeding. She is admitted to a labor room for evaluation. A fetal heart strip is obtained and reveals an FHR of 130 bpm with no accelerations or decelerations. An ultrasound is obtained and reveals a partial previa. What is the next step in management?
- (a) Observation in labor and delivery (L&D)
 - (b) Assessment of fetal lung maturity
 - (c) Cesarean delivery
 - (d) Gentle cervical examination to assess dilation and amnionic membrane status
- Answer: a
- (60) During the third stage of labor, your patient's uterus inverts and the placenta becomes detached from the uterus. Which of the following is the next best step in management?
- (a) Prompt oxytocin administration
 - (b) Attempt to manually replace the uterus
 - (c) Prompt hysterectomy
 - (d) Administration of inhalation anesthetics prior to manual replacement
- Answer: b

- (61) A 62-year-old postmenopausal woman presents to your office with episodic vaginal bleeding over the past 3 months. You are most suspicious of:
- (a) perimenopausal spotting
 - (b) adrenal hyperplasia
 - (c) cancer
 - (d) fibroids
- Answer: c
- (62) A 26-year-old female presents with 8 months of irregular bleeding. Bleeding occurs at markedly irregular intervals and varies in its quantity. A progesterone challenge test demonstrates withdrawal bleeding. Which of the following laboratory tests are indicated for the evaluation of dysfunctional uterine bleeding in this woman? (Mark all that apply.)
- (a) CBC
 - (b) Coagulation profile
 - (c) Endocrine profile (TSH, LH, FSH, prolactin)
 - (d) Endometrial biopsy
- Answer: a, b, and c
- (63) A 45-year-old woman presents to her gynecologist with complaints of amenorrhea for 7 months. She denies any symptoms of vasomotor instability. Her lab work reveals a negative β -hCG and a decreased LH and FSH. What is the most likely cause of her amenorrhea?
- (a) Menopause
 - (b) Hypothalamic-pituitary dysfunction
 - (c) Outflow obstruction
 - (d) Pregnancy
- Answer: b

- (64) A patient with amenorrhea does not have withdrawal bleeding after a progesterone challenge test. What is the next test that is indicated? Answer: a
- (a) An estrogen-progesterone test
 - (b) Head CT
 - (c) Hysteroscopy
 - (d) Laparoscopy
- (65) Which of the following lower genital tract organisms in *not* associated with increased puerperal infection? Answer: a
- (a) *Trichomonas vaginalis*
 - (b) Group B *Streptococcus*
 - (c) *Gardnerella vaginalis*
 - (d) *Mycoplasma hominis*
- (66) What uterotonic is not appropriate to administer a woman with preeclampsia? Answer: c
- (a) Oxytocin (Pitocin)
 - (b) Carboprost (Hemabate)
 - (c) Methylergonovine (Methergine)
 - (d) Misoprostol (Cytotec)
- (67) All of the following mothers are advised to breast-feed *except* those with: Answer: a
- (a) HIV with a low viral count
 - (b) Fluctuant and indurated mastitis
 - (c) Current hepatitis A infection
 - (d) Hepatitis B following infant vaccination and IgG administration
- (68) When does serum hCG return to normal (non-detectable levels) after delivery? Answer: c
- (a) Within hours
 - (b) Within days
 - (c) Within weeks
 - (d) Within months

- (69) When does menstruation begin in a postpartum woman? Answer: d
- (a) Average duration is about 20 weeks postpartum in a breast-feeding woman
 - (b) Average duration is about 8 weeks postpartum in a breast-feeding woman
 - (c) Average duration is about 20 weeks in a postpartum nonlactating woman
 - (d) Average duration is about 8 weeks postpartum in a non-lactating woman
- (70) What is the most likely diagnosis in a woman with frequency, urgency, pyuria, dysuria, and a sterile urine culture? Answer: a
- (a) *Neisseria gonorrhoeae* urethritis
 - (b) *Chlamydia trachomatis* urethritis
 - (c) *Escherichia coli* cystitis
 - (d) Group B *Streptococcus* cystitis
- (71) What is the treatment for condylomata accuminata during pregnancy? Answer: d
- (a) Interferon
 - (b) 5-Fluorouracil
 - (c) Podophyllin resin
 - (d) Trichloroacetic acid
- (72) What is the “gold standard” for the diagnosis of genital herpesvirus in adults? Answer: d
- (a) ELISA or serology
 - (b) DNA probes
 - (c) Tissue culture
 - (d) Cervical smear cytologic examination

- (73) What is the major cause of menopause-related bone loss? Answer: c
- (a) Decline in calcium production
 - (b) Decline in calcium absorption
 - (c) Decline in estrogen levels
 - (d) Rise in LH and FSH levels
- (74) When following up on the results of a DEXA scan for one of your postmenopausal patients, you notice her bone mineral density (BMD) is 1.5 standard deviations below the mean. Her diagnosis is: Answer: b
- (a) normal age-related bone loss
 - (b) osteopenia
 - (c) osteoporosis
 - (d) osteomalacia
- (75) For a patient with osteoporosis, which of the following lab abnormalities would you expect? Answer: d
- (a) High calcium, low phosphorus, low PTH
 - (b) Low calcium, high phosphorus, high PTH
 - (c) Low calcium, low phosphorus, low PTH
 - (d) No lab abnormalities
- (76) A 22-year-old G1P0 presents with uterine bleeding at 8 weeks' gestation. On physical examination, her cervix is found to be dilated to 2 cm. Vaginal ultrasound reveals the products of conception in the uterine cavity. What is her diagnosis? Answer: b
- (a) Complete abortion
 - (b) Inevitable abortion
 - (c) Incomplete abortion
 - (d) Missed abortion

- (77) A 28-year-old G3P1011 at 10 weeks' gestation presents to the ER with uterine bleeding. Her cervix is found to be closed and her β -hCG is at an appropriate level for the stated gestational age. Ultrasound reveals a nonviable fetus in the uterine cavity. What is her diagnosis? Answer: d
- (a) Complete abortion
 - (b) Inevitable abortion
 - (c) Incomplete abortion
 - (d) Missed abortion
- (78) For the above patient, which of the following management options are appropriate? Answer: d
- (a) Surgical management
 - (b) Medical management
 - (c) Expectant management
 - (d) Any of the above
- (79) A 21-year-old female with a history of PID presents to the ER with right-sided pelvic pain and vaginal bleeding. She is hemodynamically stable. Her LMP was 8 weeks ago and her urine β -hCG is positive. What is the next appropriate step in management? Answer: b
- (a) Serum β -hCG
 - (b) Transvaginal ultrasound
 - (c) Immediate laparoscopy
 - (d) Immediate laparotomy
- (80) The above patient has a nondiagnostic ultrasound. Her serum β -hCG is found to be 1000. What is the next step in management? Answer: b
- (a) Repeat β -hCG in 24 hours
 - (b) Repeat β -hCG in 72 hours
 - (c) Repeat transvaginal ultrasound in 24 hours
 - (d) Immediate laparoscopy

- (81) A 16-year-old G1P1 presents to her obstetrician 2 weeks postpartum with increased vaginal bleeding. Which of the following steps should be used in management? (Choose all that apply.)
- Answers: a, b, and c
- (a) D and C
 - (b) Serum β -hCG level
 - (c) CXR
 - (d) Expectant management
- (82) A 42-year-old G3P3 presents with 12 weeks of amenorrhea, nausea, vomiting, and mild tremors. Ultrasound reveals a heterogeneous intrauterine mass with theca lutein cysts that appear like a snowstorm. What is the most likely diagnosis?
- Answer: d
- (a) Early intrauterine pregnancy
 - (b) Perimenopause
 - (c) Partial mole
 - (d) Complete mole
- (83) A G3P3 female presents 2 months postpartum with irregular vaginal bleeding. Physical examination reveals an enlarged uterus with bilateral ovarian cysts. Ultrasonographic evaluation reveals an enlarging, heterogeneous, hypervascular mass in the uterus with areas of hemorrhage and necrosis. What is the most likely diagnosis?
- Answer: d
- (a) Pregnancy
 - (b) Partial mole
 - (c) Complete mole
 - (d) Choriocarcinoma

- (84) What is the difference between gestational hypertension and preeclampsia? Answer: a
- (a) Proteinuria is present in preeclampsia, and it is absent in gestational hypertension
 - (b) Patient has a history of hypertension in gestational hypertension
 - (c) Patient exhibits sustained elevated blood pressures in preeclampsia
 - (d) Patient reports lower extremity pitting edema
- (85) What is the cure for preeclampsia? Answer: b
- (a) Magnesium sulfate
 - (b) Delivery
 - (c) Nifedipine
 - (d) Diazepam
- (86) What is not a complication of placenta previa? Answer: c
- (a) Maternal hemorrhage
 - (b) Placenta accret
 - (c) Gestational diabetes
 - (d) Preterm premature rupture of the membranes (PPROM)
- (87) What intrapartum obstetrical maneuvers are used to treat a shoulder dystocia? Answer: a
- (a) McRoberts maneuver
 - (b) Fundal pressure
 - (c) Decreasing anesthesia to facilitate maternal effort
 - (d) Placing the mother in the left lateral decubitus position
- (88) An 18-year-old woman with no prenatal care presents to labor and delivery in labor. After delivery, her neonatal infant is found to have sensorineural deafness, Answer: b

cataracts, PDA, hepatosplenomegaly, hyperbilirubinemia, and blue purpura that appear like a blueberry muffin. What congenital infection is the most likely culprit?

- (a) *Toxoplasma*
- (b) Rubella
- (c) Cytomegalovirus
- (d) Syphilis

(89) Which of the following are modalities of mother-to-child HIV transmission? Answer: d

- (a) Transplacental infection
- (b) Peripartum infection
- (c) Breast-feeding
- (d) All of the above

(90) A 29-year-old G1P0 female presents to L & D at 33 weeks' gestation in preterm labor. Her GBS status is unknown and she has no known drug allergies. What is the best management for this patient? Answer: a

- (a) Penicillin
- (b) Clindamycin
- (c) Erythromycin
- (d) No antibiotics are required

(91) A 23-year-old G1P0 at 39 weeks gestation presents to L & D with painful contractions every 3 minutes. She has a history of HSV-2, and on sterile speculum examination she is found to have an active lesion on the right labia. Which of the following is an appropriate next step in management? Answer: a

- (a) Cesarean section
- (b) Treatment with acyclovir
- (c) Careful delivery with pediatrics present at birth
- (d) All of the above

- (92) A 29-year-old G1P0 presents to L & D after 3 hours of painful contractions occurring every 3 minutes. Her initial cervical examination on the floor was 5/80%/-2. Two hours later, no change is noted. An IUPC is placed and her contractions are found to have 250 Montevideo units over the next 2 hours. What is the most appropriate diagnosis?
- (a) Normal latent labor
(b) Arrest of labor
(c) Protraction of labor
(d) Inadequate contractions
- Answer: b
- (93) An ultrasound is preformed at a 28-week prenatal visit, the fetus is found to be in breech presentation with its hips and knees flexed. What type of breech presentation is this?
- (a) Frank breech
(b) Complete breech
(c) Footling breech
- Answer: b
- (94) For the above patient, which of the following are appropriate management options?
- (a) Cesarean delivery
(b) External cephalic version
(c) Vaginal delivery
(d) All of the above
- Answer: a and b
- (95) A G2P1001 is in active labor with her last cervical examination 2 hours prior of 3/90%/-1. You begin to notice decelerations on the monitor. The decelerations rapidly drop approximately 20 bpm below baseline, quickly return to baseline, and appear unrelated to uterine contractions. What causes this type of deceleration?
- (a) Fetal scalp compression
(b) Uteroplacental insufficiency
- Answer: c

- (c) Umbilical cord compression
- (d) Fetal acidosis

(96) The amniotic membranes in the above patient are artificially ruptured in order to accelerate labor. The fluid is noted to be clear with a slightly greenish tint. What is the fetus at risk of? Answer: d

- (a) Renal failure
- (b) Conjunctivitis
- (c) Toxoplasmosis
- (d) Chemical pneumonitis

(97) Which method is used to reliably diagnose fetal alcohol syndrome prenatally? Answer: a

- (a) Cannot be diagnosed prenatally
- (b) Ultrasound
- (c) Amniocentesis
- (d) History of heavy alcohol consumption

(98) A neonate who was born preterm is found to have hyperbilirubinemia. Which antibiotic was most likely given to the mother near delivery? Answer: d

- (a) Cephalosporins
- (b) Macrolides
- (c) Penicillins
- (d) Sulfonamides

(99) Which of the following antiviral drugs used to treat HIV infection is teratogenic? Answer: d

- (a) Zidovudine
- (b) Amprenavir
- (c) Didanosine (DDI)
- (d) None of the above

- (100) The “double-bubble” sign is an ultrasonographic finding of which of the following anomalies? Answer: c
- (a) Aqueductal stenosis
 - (b) Cystic hygroma
 - (c) Duodenal atresia
 - (d) Two-vessel umbilical cord
- (101) A 23 year old GO presents to your office for an annual exam. Her Pap comes back as ASCUS. She has never had any other abnormal Pap smears. Which of the following management options could you do? Answer: d
- (a) Repeat Pap in 6–12 months
 - (b) Send a reflex HPV and triage based on those results
 - (c) Send patient directly to colposcopy
 - (d) All of the above
- (102) A 32 year old female presents to the ED with vaginal bleeding. Her LMP was 4 weeks ago and her β hCG is 1200. An ultrasound is done and no intrauterine pregnancy is seen. Her cervix is closed and the bleeding resolves. Which of the following management options is most appropriate? Answer: b
- (a) Immediate laparoscopy for a presumed ectopic pregnancy
 - (b) Repeat serum β hCG in 48 hours
 - (c) Discharge home with prenatal follow up in 4 weeks
 - (d) Dilation & curettage for missed abortion
- (103) Which of the following characteristics on US is most suspicious for malignancy when evaluating an ovarian mass? Answer: c
- (a) Size < 6 cm
 - (b) Unilocular mass

- (c) Complex mass
- (d) Minimal flow on Doppler

(104) A 33 year old G1P0 presents to L & D in labor. She progresses to the second stage of labor and pushes for 3 hours with an epidural. The decision is made to apply a vacuum as the fetal station is +3. The vacuum pops off 3 times. Which of the following management options is most appropriate?

Answer: c

- (a) Reapply to vacuum and reattempt delivery
- (b) Apply forceps
- (c) Immediate cesarean delivery
- (d) All of the above are appropriate

(105) A 28-year-old patient presents to you with symptoms of a leiomyoma. Which set of symptoms is most consistent with a leiomyoma?

Answer: b

- (a) Hirsutism, acne, amenorrhea, virulization
- (b) Pelvic pain, dyspareunia, urinary incontinence, menorrhagia
- (c) Dysmenorrhea, dyspareunia, infertility, painful defecation

(106) What disease is most associated with the symptoms listed under option (c) in the above question?

Answer: b

- (a) Pelvic inflammatory disease
- (b) Endometriosis
- (c) Uterine sarcoma

- (107) A distraught couple visits your office, upset at not being able to conceive after 1 year of regular, unprotected intercourse. The female is a nulligravid, takes no medication, and denies any medical illnesses. The husband reports that he is healthy as well and has never fathered a child before. What is the most appropriate initial step in the evaluation of this couple?
- Answer: d
- (a) Basal body temperature charting
 - (b) Postcoital test
 - (c) Semen analysis, including sperm antibodies
 - (d) History and physical examination of both partners
 - (e) Laparoscopy
- (108) A 25-year-old woman presents to the ER immediately after being rescued from a motor vehicle accident. She states that she is pregnant and ultrasound reveals an 18-week-old fetus. A fetal heart rate is appreciated. The patient looks stable on physical examination and has sustained cuts and scrapes on her arms, face, and legs. Her clothes and exposed skin are soiled. The cervix remains long and closed. While reading her medical record, you notice that her MMR, varicella, and tetanus are not up to date. Which shot is the most appropriate to give at this time?
- Answer: d
- (a) Measles
 - (b) Mumps
 - (c) Rubella
 - (d) Tetanus
 - (e) Varicella

- (109) Your patient presents at 30 weeks of pregnancy and requests an elective cesarean delivery. While discussing why she wants this procedure, you describe to her the complications that can arise from a cesarean delivery. Of all the following complications, which one has the highest association with cesarean delivery?
- (a) Bladder injuries
 - (b) DVT
 - (c) Aspiration pneumonia
 - (d) Wound infections
 - (e) Postpartum endometritis
- Answer: e
- (110) Which of the following can be visualized on a sonogram during the first trimester?
- (a) Diencephalon
 - (b) Mesencephalon
 - (c) Prosencephalon
 - (d) Rhombencephalon
 - (e) Telencephalon
- Answer: d
- (111) A 44-year-old woman is diagnosed with epithelial ovarian cancer. She is found to have cancer that is limited to one ovary with extension to the uterus and fallopian tubes. What ovarian cancer stage should be assigned to this patient?
- (a) Stage I
 - (b) Stage IIA
 - (c) Stage IIB
 - (d) Stage IIC
 - (e) Stage III
- Answer: c

- (112) A 34-year-old woman had a colposcopic examination with a negative biopsy result. Her endocervical canal curettage result returned positive. What is the next appropriate step in management?
- Answer: e
- (a) Observe for the next 4–6 months
 - (b) No follow-up is needed
 - (c) Repeat colposcopic examination in the next 2–3 months
 - (d) Vaginal hysterectomy
 - (e) Perform conization of the cervix
- (113) Which of the following sets contain the most important risk factors for cervical cancer?
- Answer: d
- (a) The use of oral contraceptives
 - (b) Family history of cervical cancer
 - (c) Marriage at an early age and having HSV-1
 - (d) History of smoking, multiple sex partners, and first intercourse in the adolescent years
 - (e) Multiparous woman with marriage at a late age
- (114) Which of the following sets describe the clinical appearance of lichen sclerosis?
- Answer: c
- (a) Moist, thick, white, scaly plaques
 - (b) Nontender ulcerative lesions
 - (c) White, thin, atrophic-appearing plaques
 - (d) Excoriated, thickened, and erythematous epithelium

- (115) A 31-year-old woman presents to your office with complaints of intense vaginal itching and frothy discharge with malodorous odor. Pelvic examination reveals greenish-gray discharge with numerous “strawberry-like” punctate marks on the cervix. What is the proper treatment for this patient?
- Answer: c
- (a) Fluconazole
 - (b) Estrogen cream
 - (c) Metronidazole
 - (d) Penicillin
 - (e) Doxycycline
- (116) A 62-year-old woman presents to your office with complaints of a 6-month history of intense and painful vulvar itching. She also states that she can feel a “small lump.” She denies any vaginal bleeding. Her past medical, surgical, and family histories are unremarkable. Examination of the vulva reveals a small fleshy outgrowth on her left labia majora. A biopsy is performed and pathology reveals squamous cell carcinoma in situ. Which of the following statements is most appropriate in the management of this patient?
- Answer: c
- (a) Postoperative radiation is recommended
 - (b) Careful observation for the next 6 months to see if the growth enlarges or remains the same is recommended
 - (c) Colposcopy of the vagina and cervix should be done postoperatively
 - (d) Total vulvectomy is recommended
 - (e) Groin dissection is necessary

- (117) A 30-year-old woman presents to the office with complaints of intense itching in the vulva and axillae. She states that she can feel small bumps in these areas and that they appeared suddenly when the weather became hot and humid. She asks for some kind of treatment as the itching prevents her from sleeping well. On examination there are multiple, smooth, flesh-colored papules with excoriations around the vulva and bilateral axillae. There is small whitish vaginal discharge and wet mount reveals epithelial cells, with no hyphae or clue cells. What is the most likely diagnosis?
- Answer: a
- (a) Fox-Fordyce disease
 - (b) Behcet disease
 - (c) Contact dermatitis
 - (d) Folliculitis
- (118) A 21-year-old female visits you complaining of a 1-month history of pyuria and dyspareunia. She is newly married and returned from her honeymoon 2 weeks ago. She is concerned that this will affect her marriage. What is the most likely cause of her discomfort?
- Answer: c
- (a) *Trichomonas*
 - (b) *Candida*
 - (c) *E. coli*
 - (d) *Lactobacillus*
 - (e) Inadequate vaginal lubrication
- (119) A 23-year-old female comes to your office with complaints of intense vaginal itching over the past 4 days that has been getting progressively worse. She states that she had been in good health until 1 week ago when she was treated with ampicillin for a urinary tract infection. Her past
- Answer: b

medical history is unremarkable, and she believes she is in a monogamous sexual relationship with her partner. What is the most appropriate step in management?

- (a) Repeat urinalysis to rule out urinary tract infection
- (b) Perform a pelvic examination and evaluate the discharge
- (c) Reassure the patient that it is a natural response to the antibiotics and will diminish over time
- (d) Prescribe antifungals for presumed candidiasis
- (e) Perform a Pap smear

(120) Which of the following is the most common cause of precocious puberty in females? Answer: c

- (a) Adrenal tumor
- (b) Ovarian tumor
- (c) Idiopathic
- (d) Functional ovarian cyst

(121) Your 23-year-old female patient has mild endometriosis. Her symptoms include irregular menses with no pelvic pain. Which of the following is the best treatment for her? Answer: d

- (a) Oral estrogen
- (b) GnRH agonists
- (c) Danazol
- (d) Oral contraceptive pills
- (e) Medroxyprogesterone acetate

(122) A 35-year-old African-American woman presents to your office with complaints of increasingly heavy and prolonged menstrual periods over the past year. She also describes feeling a “fullness” in her pelvic area. In addition, she reports having mild dyspareunia. She is happily married to her husband and they have two children. They are considering having another child in the future. Presently, she uses oral contraceptive pills for birth control.

In addition to her chief complaint, she reports feeling “more tired” and having difficulty sleeping. She has associated this with her job, as she is a vice-president for a consulting company and work has been more stressful recently. Her past medical history is unremarkable and she takes no prescription medications.

What is the next most appropriate step in management?

Answer: c

- (a) Reassurance that her symptoms will resolve when her job-related stress subsides
- (b) Obtain LH and FSH levels
- (c) Obtain an ultrasound
- (d) Obtain cultures for *Chlamydia* and gonorrhea
- (e) Perform an exploratory laparoscopy

(123) For the above scenario, what is the most likely diagnosis?

Answer: d

- (a) Endometriosis
- (b) Polycystic ovarian syndrome
- (c) Pelvic inflammatory disease
- (d) Uterine leiomyoma
- (e) Endometrial cancer

(124) Considering that the patient in the above question is considering having another child in the future, which of the following statements is true for this patient?

Answer: e

- (a) A myomectomy should be performed as soon as possible to allow as much time as possible for uterine healing prior to the stress of pregnancy
- (b) An open-surgery myomectomy is a common cause of infertility because of the formation of postoperative adhesions
- (c) Following a myomectomy, cesarean delivery is required to avoid uterine rupture
- (d) Myomectomy is expected to increase this patient's fertility rate by 50% in 1-2 years
- (e) Uterine artery embolization (UAE) has been shown to diminish bleeding symptoms and recovery time, but it is not recommended for women who desire children in the future

(125) A 36-year-old woman has heavy, painless bleeding every 4-5 months. She comes to your office asking for contraceptives. An examination of her cervix is normal and her Pap smear is class I. What is the most appropriate procedure?

Answer: d

- (a) Oral estrogen only
- (b) Bilateral-salpingo-oophorectomy
- (c) Cyclic oral contraceptive agents
- (d) Fractional dilation and curettage (D & C)
- (e) Conization of the cervix

- (126) A 26-year-old female visits your office for a routine gynecologic examination. She has no complaints and is in good health. Her menstrual periods began when she was 12 years old and are regular with a 28-day interval and duration of 5 days. She is single but sexually active and uses oral contraceptive pills and barrier contraception. During the pelvic examination, you palpate a mass on the right side. An ultrasound is performed and reveals a complex, cystic tumor approximately 5 cm in diameter in the right ovary. There is no free fluid in the peritoneum and there is no family history of cancer. What is the most appropriate step in her treatment?
- Answer: e
- (a) Laparoscopy and cystectomy
 - (b) Hormonal treatment with progesterone
 - (c) Pelvic examination in 6 months
 - (d) Ovariectomy of the right ovary
 - (e) Sonogram surveillance over the next two menstrual cycles
- (127) Which of the following ovarian tumors is most sensitive to radiation therapy?
- Answer: b
- (a) Gonadoblastoma
 - (b) Dysgerminoma
 - (c) Choriocarcinoma
 - (d) Serous cystadenocarcinoma
- (128) Which neoplasm is most sensitive to chemotherapy?
- Answer: e
- (a) Ovarian dysgerminoma
 - (b) Uterine sarcoma
 - (c) Ovarian serous carcinoma
 - (d) Fallopian tube cancer
 - (e) Gestational trophoblastic disease

- (129) The release of clear or blood-tinged vaginal discharge, whether spontaneously or pressure induced, followed by shrinkage of an adnexal mass and relief of cramping pain is pathognomonic for which neoplasm? Answer: a
- (a) Fallopian tube cancer
 - (b) Ovarian cystadenocarcinoma
 - (c) Epithelial ovarian cancer
 - (d) Dermoid cyst
 - (e) Krukenberg tumor
- (130) Herniation of the peritoneum between the uterosacral ligaments through the pouch of Douglas into the rectovaginal septum represents which of the following defects? Answer: b
- (a) Uterine prolapse
 - (b) Rectocele
 - (c) Enterocele
 - (d) Urethrocele
 - (e) Cystocele
 - (f) Retrodisplacement of the uterus
- (131) A 65-year-old menopausal woman visits your office with complaints of urinary difficulty and discomfort in the vagina. She complains that the feeling in her vagina is like she is "sitting on an egg." What is the appropriate therapy? Answer: b or c (Depending on patient's preference. However, b is preferred if there is other uterine pathology, whereas c is preferred for those who want to avoid surgery.)
- (a) Remove the foreign body
 - (b) Perform a hysterectomy
 - (c) Recommend a pessary
 - (d) Reassurance

- (132) A 26-year-old multiparous patient at 33 weeks' estimated gestational age reports significant abdominal pain of 40-minutes duration. The pain is improved when sitting up and was not associated with exertion. The patient has a history of alcohol dependence and had just finished eating a spiced curry tomato dish. On presentation, her vital signs and physical examination are within normal limits. Which of the following lab values is abnormal?
- Answer: a
- (a) Amylase 300 U/dL
 - (b) White blood cells 13,000 per mm³
 - (c) Fibrinogen 500 mg/dL
 - (d) Alkaline phosphatase 17 U/L
- (133) An obese 22-year-old woman has a history of irregular periods. She was placed on oral contraceptive pills and after two normal cycles she has begun to miss her period again. Her BhCG is zero. What is the next course of action?
- Answer: d
- (a) Begin metformin so as to better control the metabolic derangements associated with polycystic ovarian syndrome
 - (b) Check FSH and LH levels for possible premature ovarian failure
 - (c) Do a transvaginal ultrasound to look for ovarian cysts
 - (d) Change the oral contraceptive to a monophasic instead of a triphasic

(134) A 19-year-old, obese, Type A1 diabetic at 30 weeks of pregnancy complains of nausea, dizziness, and shortness of breath when lying down. Her vital signs and physical examination are within normal limits, urine dip reveals trace protein. What is the next step in her treatment?

Answer: d

- (a) Urgent biophysical ultrasound with evaluation of the fetus for macrosomic presentation and likely labor dystocia
- (b) Lower-extremity doppler and electrocardiogram for possible DVT and pulmonary embolism
- (c) Neurologic evaluation with attention paid to signs of end-organ damage related to preeclampsia
- (d) Request that the patient sleep on her left side so as to assume a left lateral displacement of the gravid uterus and schedule the patient for clinic visit the following day

(135) After completing an abdominal x-ray, a woman discovers that she is several weeks pregnant. She is concerned regarding the status of the fetus. Which of the following is accurate?

Answer: c

- (a) Organogenesis is completed by the 15th week
- (b) The bulk of neural development occurs during the third trimester
- (c) Preimplantation exposure to radiation typically results in a spontaneous abortion
- (d) Radiation exposure of less than 5 rads has been associated with neurologic defects

- (136) A 26-year-old Type I diabetic class B reports decreased fetal movement. She is at 37 weeks' estimated gestational age and has had poor glycemic control throughout pregnancy. The manual estimated fetal weight is 2000 grams and the fundal height is somewhat less than the estimated gestational age. What type of screening should be performed to assess fetal well-being and placental sufficiency?
- Answer: a and c
- (a) Non-stress test followed by abdominal ultrasound to assess for presentation, amniotic fluid, and placental location
 - (b) Biophysical profile
 - (c) Amniocentesis with analysis for fetal lung maturity
 - (d) Cordocentesis with analysis of pH, lactic acid, and immunoglobulins
- (137) An obese 38-year-old with chronic hypertension and Type II diabetes is found to be 6 weeks pregnant. Her medications include: an ACE inhibitor, beta-blocker, thiazide diuretic, metformin, folic acid, and a multivitamin. Which of the following medications (choose all that apply) are safe in pregnancy?
- Answer: a and c
- (a) Insulin
 - (b) ACE inhibitors
 - (c) Glyburide
 - (d) Thiazide
- (138) An Rh(-) mother has just undergone a normal spontaneous vaginal delivery with significant postpartum hemorrhage attributed to atony. What test would assist in determining how much RhoGam should be administered postpartum?
- Answer: c
- (a) Quantitative titer analysis of maternal circulating antibodies
 - (b) Flow cytometry to determine which isoforms the mother has developed

(c) Kleihauer-Betke serum stain

(d) Indirect Coombs test

(139) A woman with gestational diabetes class A1 and poor glycemic control requests a repeat cesarean delivery. Fetal lung maturity (FLM) at 37 weeks shows a lecithin/sphingomyelin ratio of 2.2/ and phosphatidylglycerol negative. Following delivery, the infant develops respiratory distress syndrome (RDS). What about the patient's history would suggest that the infant was at risk for RDS? Answer: d

requests a repeat cesarean delivery.

Fetal lung maturity (FLM) at 37 weeks shows a lecithin/

sphingomyelin ratio of 2.2/ and phosphatidylglycerol negative.

Following delivery, the infant develops respiratory distress syndrome (RDS). What about the patient's history would suggest that the infant was at risk for RDS?

(a) Two or more types of fetal lung maturity tests should be used when determining the risk of RDS

(b) Elective cesarean delivery should only take place after 39 weeks

(c) The amniocentesis was likely contaminated with blood, resulting in a false L/S ratio

(d) Surfactant function and production is adversely affected by increased fetal insulin and maternal diabetes

(140) A 36-year-old female presents with a single, firm, well-delineated, round, nontender nodule in her left upper breast. When her gynecologist palpates, it is very mobile with respect to its surrounding tissue. What is the most likely diagnosis of this mass? Answer: d

a single, firm, well-delineated, round, nontender nodule in

her left upper breast. When her gynecologist palpates, it is very

mobile with respect to its surrounding tissue. What is the

most likely diagnosis of this mass?

(a) Ductal carcinoma in situ

(b) Papillary cystadenocarcinoma

(c) Breast cyst

(d) Adenocarcinoma

- (141) Tubal patency or “pelvic factor” in evaluation of infertility is best accomplished by:
- (a) Hysterosalpingogram (HSG)
 - (b) Hysteroscopy
 - (c) Pelvic magnetic resonance imaging (MRI)
 - (d) Transvaginal ultrasound
 - (e) Pelvic CT scan
- (142) You are seeing your 35-year-old patient, who has sought your help several times prior for vague but persistent gynecologic concerns. She currently complains of pelvic discomfort. You have evaluated her in the past for the same concern and found no gynecologic or gastrointestinal etiology. As you continue to ask her more questions, she begins to cry and reveals that she was sexually abused as a child.
- Which of the following statements about sexual abuse is most statistically accurate?
- (a) Her abuser was someone she did not know well
 - (b) Her abuser was a member of her family
 - (c) Approximately 5% of women have experienced childhood sexual abuse
 - (d) The abuse took place at a daycare center
 - (e) She was abused between the ages of 2 and 10
- (143) Through chromosome analysis, you find that your patient has a sex genotype of XO. What is the proper therapeutic regimen for this patient?
- (a) Androgens and cortisol
 - (b) Cortisol and human growth hormone
- Answer: a
- Answer: b
- Answer: e

- (c) Small doses of estrogen in early childhood
 - (d) Progesterone, estrogen, and cortisol
 - (e) Androgens, human growth hormones, small doses of estrogen, and later progesterone
- (144) In evaluating an infertile couple, the postcoital test is performed to assess which of the following? Answer: a
- (a) Interaction of sperm with cervical mucus prior to ovulation
 - (b) Interaction of sperm with cervical mucus anytime during the cycle
 - (c) Interaction of sperm with cervical mucus during ovulation.
 - (d) Interaction of sperm with cervical in midluteal phase
- (145) All of the following may be direct causes of female infertility except: Answer: a
- (a) previous uncomplicated abortion
 - (b) endometriosis
 - (c) pelvic inflammatory disease (PID)
 - (d) hyperprolactinemia
 - (e) polycystic ovarian syndrome (PCOS)
- (146) What is the most common presenting fracture in osteoporosis? Answer: a
- (a) Vertebral compression fracture
 - (b) Wrist fracture
 - (c) Tibial fracture
 - (d) Femoral head fracture
 - (e) Femoral neck fracture

- (147) What is the gold standard for the diagnosis of osteoporosis? Answer: d
- (a) Plain x-ray of the thoracic spine
 - (b) Qualitative ultrasound densitometry
 - (c) Peripheral dual x-ray absorptiometry (DXA)
 - (d) Central (DXA)
 - (e) A quantitated computed tomography (QCT)
- (148) A 52-year-old woman has been experiencing signs of menopause, including increasing hot flushes during the day and night, difficulty sleeping, emotional lability, and anxiety. She denies any other complaints or medical illnesses. Her last period was approximately 12 months ago. Her vital signs are all within normal range. Her pelvic examination reveals atrophic external genitalia, a small anteverted uterus, and no adnexal masses. The rest of her examination is normal. Answer: d
- What is the most effective treatment option for this patient?
- (a) Progestin alone
 - (b) Estrogen alone (ERT)
 - (c) Antidepressants
 - (d) Estrogen with progestin (hormone replacement therapy [HRT])
- (149) If this patient also complained of vaginal dryness and pain during intercourse, reasonable treatment options would include? Answer: d
- (a) Topical estrogen cream
 - (b) An estrogen ring (Estring)
 - (c) Vaginal moisturizers
 - (d) All of the above

(150) According to the World Health Organization (WHO), osteoporosis is defined as:

Answer: c

- (a) BMD between 1.5 and 2.0 standard deviations below the mean for young normal adults (T score)
- (b) BMD is between 1.5 and 2.0 standard deviations below the mean for age-matched adults (Z score)
- (c) BMD is less than 2.5 standard deviations below the mean for young normal adults (T score)
- (d) BMD is less than 2.5 standard deviations below the mean for age-matched adults (Z score)

This page intentionally left blank

Suggested Readings and Sources of Information

Beckmann C, Ling FW, Smith RP, Barzansky BM, Herbert W, and Laube DW. *Obstetrics and Gynecology*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.

Berek, Jonathan S. *Berek and Novak's Gynecology (Novak's Textbook Gynecology)*. 14th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.

Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Gilstrap LC III, and Wenstrom KD. *Williams Obstetrics*. 22nd ed. New York, NY: McGraw-Hill Companies; 2005.

Decherney AH, Nathan L, Goodwin TM, Laufer N. *Current Diagnosis & Treatment*. 10th ed. New York, NY: McGraw-Hill Companies; 2007

www.ACOG.org (Medical Students are eligible for Free Membership)

www.UpToDate.com

This page intentionally left blank

Index

Page numbers followed by *f* or *t* indicate figures or tables, respectively.

A

- Abdominal distension, in ovarian cancer, 156
- Abdominal hysterectomy, 16*t*. *See also* Hysterectomy
- Abdominal pain. *See also* Pelvic pain
 - in acute fatty liver of pregnancy, 340
 - in degenerating fibroid, 447
 - differential diagnosis, 74, 355, 424
 - in Fitz-Hugh-Curtis syndrome, 78
 - in ovarian germ cell tumor, 158
 - in pregnancy, 355–356
 - in tubo-ovarian abscess, 79
- Abdominal sacral colpopexy, 177
- Abdominal wall, 33–34
- Aberrant gonad, 192
- Abnormal uterine/vaginal bleeding.
 - See also* Dysfunctional uterine bleeding
 - in cervical cancer, 126
 - in choriocarcinoma, 300
 - differential diagnosis, 150, 199
 - in endometrial cancer, 149
 - in first trimester, 287–288
 - leiomyomas and, 106
 - in ovarian sex cord-stromal tumors, 161
 - in third trimester, 309–311
 - in uterine sarcoma, 153
 - in vaginal cancer, 146
- Abortion
 - differential diagnosis, 75
 - elective, 56–60, 421–422
 - infections after, 422
 - medical, 59
 - mortality rates, 57
 - septic, 59, 287
 - spontaneous, 286–289
 - tubal, 292
 - types, 287
- Abscess
 - Bartholin gland, 118–119
 - pelvic, 87
 - tubo-ovarian, 75, 78–80
- Abuse, sexual/physical, 90, 411, 462
- ACE inhibitors, teratogenic effects, 404*t*
- Acetylcholinesterase, neural tube defects and, 245
- Acid base equilibrium, in pregnancy, 229
- Acid-fast stain, in pelvic tuberculosis, 81
- Acidosis, fetal, 396
- Acne
 - in ovarian sex cord-stromal tumors, 161
 - in pregnancy, 234
- Acquaintance rape, 411
- Acquired immunodeficiency syndrome (AIDS), 88–89. *See also* Human immunodeficiency virus (HIV) infection
- Acrochordon, 120
- Actinomyces israelii*, 80
- Actinomycin-D, for choriocarcinoma, 300
- Actinomycosis, pelvic, 80
- Active phase, labor, 260*f*, 261
- Acute chest syndrome, 350–351
- Acute fatty liver of pregnancy (AFLP), 339–340
- Acyclovir
 - for congenital herpes infection, 364
 - for genital herpes, 66, 73
 - for herpes prophylaxis in pregnancy, 363
- Adenocarcinoma
 - breast, 462
 - cervical, 126, 135–136. *See also* Cervical cancer
 - endometrioid, 150
 - vaginal, 148
- Adenomyosis, 91, 102–103, 197
- Adenosquamous carcinoma, cervical, 126. *See also* Cervical cancer
- ADH. *See* Antidiuretic hormone
- Adnexectomy, for ovarian germ cell tumor, 160
- Adrenal hormones, in pregnancy, 232
- Adrenal tumor, 219
- Advanced maternal age, 242
- AGC (atypical glandular cells), 135
- Agensis, vaginal, 191
- Alcohol, teratogenic effects, 403*t*
- Alkaline phosphatase, in pregnancy, 231
- Alopecia, postpartum, 279
- Alpha-fetoprotein (AFP)
 - in ovarian germ cell tumors, 158–160
 - in prenatal screening, 246
- α -adrenergic agonists, for urinary incontinence, 186
- Ambiguous genitalia, 276
- Amenorrhea
 - etiology, 201, 436
 - evaluation, 202–204, 437–438
 - hypothalamic-pituitary dysfunction and, 201–202, 436
 - imperforate hymen and, 191
 - in menopause, 213. *See also* Menopause
 - ovarian dysfunction and, 202
 - post-DMPA, 49
 - post-pill, 48
 - subtypes, 201
 - treatment, 204
- Amine/whiff test, 82
- Aminoglycosides, teratogenic effects, 405*t*
- Amniocentesis, 8*t*
 - in cytomegalovirus infection, 362
 - definition, 246
 - in Rh alloimmunization, 381
- Amniotic bands, 318
- Amniotic fluid
 - analysis in Rh isoimmunization, 383
 - bilirubin in, 383
 - dye test, 311
 - fern (crystallization) test, 10*t*, 15*t*, 22*f*, 251, 311
 - pH, 251
- Amniotomy, 268–269, 393

- Amoxicillin, for asymptomatic bacteriuria
in pregnancy, 344f
- Amphetamines, teratogenic effects, 405f
- Ampicillin
for asymptomatic bacteriuria in
pregnancy, 344f
for group B streptococcus, 375
for *Listeria* infection, 376
prophylactic, in cesarean delivery, 326
for pyelonephritis, 170
for tubo-ovarian abscess, 79
- Anaerobic bacteria, in tubo-ovarian abscess, 78
- Androgen(s)
clitoral enlargement and, 191
in hirsutism, 218
insensitivity, 194
premature pubarche and, 192
secretion in menstrual cycle, 39
teratogenic effects, 403f
- Android pelvis, 24
- Androstenedione, in menopause, 214
- Anemia, in pregnancy, 349–350, 421
- Angelman syndrome, 285
- Angiography, 12f
- Anorgasmia, 115
- Anosmia, in Kallmann syndrome, 194
- Anovulation
causes, 203
classification, 205–206
dysfunctional uterine bleeding in, 198–200
infertility treatment in, 207–208
in polycystic ovarian syndrome, 220–221
progesterone challenge test and, 203
- Antepartum hemorrhage, 309
- Anterior asynclitism, 256
- Anterior vaginal colporrhaphy, 177
- Anteversio, 27
- Anthropoid pelvis, 24
- Anti-androgens
oral contraceptives as, 220
for polycystic ovarian syndrome, 221
spironolactone as, 220
- Antibiotics
oral contraceptives and, 48
in preterm premature rupture of
membranes, 314
prophylactic
for cesarean delivery, 326
for elective abortion, 58–59
in sexual assault victims, 413f
for pyelonephritis, 170
for toxic shock syndrome, 88
vulvovaginal candidiasis and, 83
- Anticholinergics, for urge incontinence, 184
- Anticonvulsants
oral contraceptives and, 48
in pregnancy, 345–346, 345f
- Antidepressants
for chronic pelvic pain, 93
sexual dysfunction and, 427
- Antidiuretic hormone (ADH)
oxytocin and, 233
preterm labor and, 315–316
- Antifungal agents, for vulvovaginal
candidiasis, 83–84
- Antihypertensives, in pregnancy, 302
- Antimuscarinics, for urinary
incontinence, 186
- Antiphospholipid syndrome, 348–349
- Antiretrovirals
for HIV prophylaxis after sexual assault, 413f
oral contraceptives and, 48
in pregnancy, 369, 445
- Apgar score, 276
- Appendicitis, in pregnancy, 231, 355–356, 419
- Apt test, 310
- Arcuate line, 23–24, 34
- Arcuate uterus, 191
- Arginine, for sexual dysfunction, 113
- Arrest of descent, 262
- Arrest of labor, 262
- Arterial blood gas, in pregnant vs.
nonpregnant woman, 5f, 229
- Ascites
in ovarian cancer, 156, 161
in ovarian germ cell tumor, 158
- Asherman syndrome, 202
- Ashkenazi Jews
breast and ovarian cancer syndrome in, 155
prenatal screening tests, 243
- Aspiration, gastric, 252–253, 351–352
- Assisted reproductive technologies (ARTs), 209
- Assisted rupture of membranes, 268–269
- Asthma, in pregnancy, 351, 420
- Asynclitism, 256
- Atenolol, for hyperthyroidism in pregnancy, 335
- Atrophic vaginitis, 85
- Atypical glandular cells (AGC), 135
- Atypical squamous cells of undetermined signifi-
cance (ASCUS), 129, 130f, 131
- Augmentation cystoplasty, for urge
incontinence, 186
- Autoimmune disease, birth defects and, 286
- Azithromycin
for *C. trachomatis*, 73, 413f
for chancroid, 69
for chlamydia in pregnancy, 376
for gonorrhoea, 73
- Aztreonam, for pyelonephritis, 170
- B**
- Back pain, in cystitis, 165
- Bacterial vaginosis, 82–83, 326
- Bacteriuria, asymptomatic, 168, 343, 344f, 377. *See*
also Urinary tract infections
- Bacteroides, 78
- Baden-Walker system, for pelvic organ prolapse
grading, 175, 175f
- Barium enema
in cervical cancer, 127
in vulvar cancer, 138
- Barrier methods, contraception, 52–54
- Bartholin glands
anatomy, 33, 118
carcinoma, 138, 142–143
cysts and abscesses, 118–119
- Basal body temperature, 41, 206
- Basal cell carcinoma, vulvar, 143
- Battledore placenta, 309
- Beckwith-Wiedemann syndrome, 387
- Benzodiazepines, as date rape drugs, 412
- β -adrenergic blockers, for hyperthyroidism, 335
- β -adrenergic(s), for urge incontinence, 184
- β -agonists, for asthma, 351
- β -human chorionic gonadotropin (β -HCG). *See*
Human chorionic gonadotropin

- Betamethasone, for fetal lung maturation, 313–314
- Bethesda system
 - cervical adenocarcinoma classification, 135
 - cervical intraepithelial neoplasia classification, 129, 130*t*
- Bicornuate uterus, 192
- Bimanual pelvic examination, 12*t*, 22*f*
- Biofeedback, for urinary incontinence, 186
- Biophysical profile (BPP), 248–249, 460
- Bipartite placenta, 309
- Bishop score, 265–266
- Bisphosphonates, 212
- Bladder, 29, 178. *See also* Urinary tract infections
- Bladder diary, 181
- Bladder neck suspension, retropubic, 187
- Bladder training, 185–186
- Bleeding, abnormal uterine/vaginal.
 - See* Abnormal uterine/vaginal bleeding
- Bleomycin, for ovarian germ cell tumors, 160
- Blizzard syndrome, 202
- Blood chemistry, pregnant vs. nonpregnant woman, 5*t*
- Blood pressure, in pregnancy, 227, 229.
 - See also* Hypertensive disorders, in pregnancy
- Blood volume, in pregnancy, 227
- Bloody show, 251
- Bone(s), in puberty, 190
- Bone mineral loss. *See also* Osteoporosis
 - calcium for, 211
 - lactation and, 211
- Bony pelvis, 23–24, 23*f*, 257
- Botulinum toxin, for urinary incontinence, 184, 186
- Brachytherapy, for cervical squamous cell carcinoma, 133
- Bradycardia, fetal, 247–248, 397
- Braxton Hicks contractions, 250, 269
- Breach of duty, 414–415
- Breakthrough bleeding, oral contraceptives and, 47–48
- Breast(s)
 - delayed development, 194
 - in pregnancy, 234
 - Tanner stages, 190
- Breast cancer
 - abortion and, 57
 - characteristics, 462
 - oral contraceptives and, 46
 - ovarian metastases, 162
- Breast milk, 277
- Breast and ovarian cancer (BOC) syndrome, 155
- Breastfeeding
 - as contraceptive method, 43
 - contraindications, 278
 - in cytomegalovirus infection, 277
 - in hepatitis infection, 277, 373
 - in HIV infection, 430, 437
 - menstruation and, 281
 - nutritional considerations, 278
- Breech presentation, 254, 255*f*, 394–395, 394*f*, 444
- Breslow criteria, melanoma staging, 142
- Broad ligaments, 30
- Bromocriptine, for infertility, 208
- Bulking agents, periurethral, 187
- Burch colposuspension, 187
- Buschke-Lowenstein tumors, 62
- C**
- CA-125
 - in endometrial cancer, 150
 - in endometriosis, 100
 - in fallopian tube cancer, 164
 - for ovarian cancer evaluation, 157
 - for ovarian cancer screening, 155
- Cabergoline, for infertility, 208
- Café-au-lait skin lesions, 193
- Calcification, leiomyoma, 108
- Calcitonin, for osteoporosis, 212–213
- Calcium, for bone loss prevention, 211
- Call-Exner bodies, 161
- Canal of Nuck cyst, 120
- Candida albicans*, 83
- Candidiasis
 - in HIV infection, 88
 - vulvovaginal, 83–84
- Caput succedaneum, 252
- Carbamazepine
 - side effects, 345*t*
 - teratogenic effects, 403*t*
- Carboprost, contraindications, 273
- Carbuncles, 121
- Cardiac medications, in pregnancy, 355
- Cardiac output
 - in labor, 252
 - postpartum, 278
 - in pregnancy, 227–228
- Cardinal ligaments, 30
- Cardiomyopathy, peripartum, 354–355*t*, 355
- Cardiovascular disease, in pregnancy, 352–355, 352*t*, 353*t*, 354–355*t*, 420
- Carnett sign, 92
- Cataracts
 - congenital rubella and, 361
 - congenital varicella and, 367
- Causation, in malpractice, 414–415
- Cefazolin, for group B streptococcus, 375
- Cefotetan, for pelvic inflammatory disease, 77
- Cefoxitin, for pelvic inflammatory disease, 77
- Ceftriaxone
 - for chancroid, 69
 - for Fitz-Hugh-Curtis syndrome, 78
 - for gonorrhea prophylaxis after sexual assault, 413*t*
 - for pelvic inflammatory disease, 77
 - for pyelonephritis, 170
- Cephalopelvic disproportion, 392
- Cephalosporins
 - prophylactic, for cesarean delivery, 326
 - for urinary tract infections in pregnancy, 168, 343, 344*t*
- Cerclage, 8*t*, 391
- Cerebral palsy, multi-fetal gestation and, 391
- Cervical canal, 28
- Cervical cancer
 - adenocarcinoma, 126, 135–136
 - diagnosis and evaluation, 126–127, 130
 - human papillomavirus and, 62, 125
 - incidence, 125
 - metastases, 127
 - prevention, 62, 131
 - risk factors, 125, 450
 - screening, 128–129
 - signs and symptoms, 126
 - squamous cell carcinoma. *See* Squamous cell carcinoma, cervical

- Cervical cancer (*Cont.*)
 stages, 127, 128*t*
 survival rates, 127
 types, 126
- Cervical cap, 53
- Cervical conization, 12–13*t*, 130, 132, 428, 450
- Cervical insufficiency, 316–317
- Cervical intraepithelial neoplasia, 10*t*, 88, 129–131
- Cervical masses
 cysts, 103–104
 differential diagnosis, 127
 malignant. *See* Cervical cancer
 polyps, 104
- Cervical ripening, 266
- Cervicitis, 71–73
- Cervidil, for cervical ripening, 266
- Cervix
 anatomy, 28
 changes after delivery, 279
 changes in labor, 251
 histologic regions, 123
 in pregnancy, 234
 transformation zone, 123, 124*f*
- Cesarean delivery, 8*t*
 complications, 449
 in fetal macrosomia, 388
 gestational diabetes and, 333
 in herpes simplex virus infection, 363
 in HIV infection, 368, 370, 443
 incidence, 272
 incisions for, 272, 273
 indications, 272, 326
 lower uterine segment in, 272
 scheduling, 272
 vaginal birth after, 273
- Chadwick sign, 234
- Chancre, 67
- Chancroid, 69
- Chemotherapy
 for cervical squamous cell carcinoma, 132–133, 133
 for endometrial cancer, 152
 for fallopian tube cancer, 164
 for ovarian cancer, 158
 for ovarian germ cell tumors, 160
 for vaginal squamous cell carcinoma, 147
- Chest radiograph
 in cervical cancer, 127
 in ovarian cancer, 157
 in varicella pneumonia, 366
- Child sexual abuse, 411
- Chlamydia trachomatis*
 lymphogranuloma venereum and, 69–70
 mucopurulent cervicitis and, 71–73
 in pregnancy, 376–377, 430
 prophylaxis after sexual assault, 413*t*
 screening test in pregnancy, 244
 urethritis and, 169
- Chloasma, 233
- Chocolate cyst, 99
- Choriocarcinoma, 158, 159, 300–301, 441
- Chorionic villus sampling (CVS), 9*t*, 246
- Chorioretinitis
 in congenital toxoplasmosis, 358, 360
 in congenital varicella, 366
- Chromosomal abnormalities, 283–284.
See also Congenital anomalies
- Chronic pelvic pain. *See* Pelvic pain, chronic
- Ciprofloxacin
 for asymptomatic bacteriuria in pregnancy, 344*t*
 for gonorrhoea, 73
- Circumcision, 9*t*
- Circumvallate placenta, 309
- Cisplatin
 for cervical squamous cell carcinoma, 133
 for endometrial cancer, 152
 for ovarian cancer, 158
 for ovarian germ cell tumors, 160
 side effects, 133, 152, 160
- Clear cell carcinoma
 endometrial, 151
 ovarian, 155–156
 vaginal, 148
- Clindamycin
 for bacterial vaginosis, 83
 for desquamative inflammatory vaginitis, 85
 for endometritis, 276, 326
 for pelvic inflammatory disease, 77
- Clitoris, enlargement of, 191
- Clomiphene citrate
 challenge test, 207
 for infertility, 208
 for polycystic ovarian syndrome, 221
- Clue cells, 82
- CMV (cytomegalovirus), 361–363
- Coagulation profile
 in dysfunctional uterine bleeding, 200
 in pregnant vs. nonpregnant woman, 4*t*, 232
- Cocaine, teratogenic effects, 405*t*
- Coelomic metaplasia, 98
- Cold knife conization, 130. *See also* Cervical conization
- Collagen injections, for stress incontinence, 183
- Colostrum, 277
- Colpocleisis, 177
- Colporrhaphy, vaginal, 177
- Colposcopy, 13*t*, 130
 in cervical cancer, 126, 130
 in cervical intraepithelial neoplasia, 131
 in vaginal cancer, 145
 in vulvar cancer, 137–138
- Complement fixation tests, in lymphogranuloma venereum, 70
- Complete abortion, 287. *See also* Abortion
- Complete breech, 254, 255*f*, 394, 444
- Computed axial tomography (CT), 13*t*
 in cervical cancer, 127
 in hirsutism, 219
 in hypogonadotrophism, 203
 in ovarian cancer, 157
- Condyloma acuminata, 61–62, 438
- Confidentiality, 414
- Congenital adrenal hyperplasia, 218, 219, 276
- Congenital anomalies
 causes, 283–284
 of female reproductive system, 191–192
 incidence and prevalence, 283
 maternal diabetes and, 329
 teratogens and, 401, 403–405*t*
- Congenital cretinism, 337
- Congenital infections
 cytomegalovirus, 361–363
 group B streptococcus, 375
 hepatitis B, 372–373
 herpes simplex virus, 363–364

- HIV, 369–370
 parvovirus B19, 368
 rubella, 360–361
 syphilis, 364–365
 toxoplasmosis, 358–359
 varicella, 366–367
- Conjoined twins, 289
- Conjugates, pelvic, 24, 259–260
- Conjunctivitis, neonatal, 376
- Conscious laparoscopic pain mapping, 92
- Continence pessary, 186
- Contraception
 barrier methods, 52–54
 breastfeeding and, 43, 277
 counseling, 41
 effectiveness measures, 41
 failure rates, 42
 hormonal
 emergency, 51–52
 implants, patches, and rings, 50–51
 injectable, 49
 oral contraceptives. *See* Oral contraceptives
 intrauterine devices. *See* Intrauterine devices
 overview, 41–43
 postpartum, 281
 sterilization, 43–44
- Contraceptive sponge, 54
- Contraction(s). *See also* Labor
 assessment, 266–267
 tonic, 267
- Contraction stress test, 249
- Copper intrauterine devices, 54. *See also*
 Intrauterine devices
- Corpus albicans, 39
- Corpus luteum, 39, 232
- Corpus luteum cysts, 109–110
- Corpus, uterine, 27
- Cortical atrophy, congenital varicella and, 366
- Corticosteroids
 bone loss and, 210
 for fetal lung maturation, 313–314
 for vulvar dystrophies, 117, 215
- “Cottage cheese” discharge, in vulvovaginal candidiasis, 83
- Cranberry juice, for prevention of urinary infections, 167
- Cryotherapy, 14f
- CT. *See* Computed axial tomography
- Culdocentesis, 14f, 79
- Cushing syndrome, 218, 219
- CVS (chorionic villus sampling), 9f, 246
- Cyclophosphamide
 for choriocarcinoma, 300
 teratogenic effects, 404f
- Cyst(s)
 canal of Nuck, 120
 cervical, 103–104
 epidermal, 119
 Gartner duct, 120
 ovarian, 74, 96f, 109–111, 423, 446, 456
 sebaceous, 119
 Skene duct, 120
 vulvovaginal, 118–120
- Cystic hygroma, 446
- Cystitis
 diagnosis, 165–166
 differential diagnosis, 165
 etiology, 166, 452
 incidence, 165
 interstitial, 184
 pathophysiology, 166
 in pregnancy, 169, 344, 377–378
 prevention, 167
 recurrent, 167
 risk factors, 166, 452
 treatment, 166–167
- Cystocele, 171, 172f, 177
- Cystometry, 182
- Cystoscopy, in vulvar cancer, 138
- Cytochrome P₄₅₀, oral contraceptives and, 48
- Cytokeratin expression, in ovarian tumors, 162–163
- Cytomegalovirus (CMV), 361–363
- D**
- Damages, in malpractice, 414–415
- Danazol
 for endometriosis, 101–102
 hirsutism and, 218
 teratogenic effects, 403f
- Darifenacin, for urinary incontinence, 186
- Darkfield microscopy, 64, 67–68
- Date rape, 411, 412
- Deafness, congenital rubella and, 361
- Decelerations, fetal heart rate, 249, 396, 398–399, 444–445
- Deep venous thrombosis, 346–347, 421, 425
- Delayed sexual maturation, 194
- Delivery. *See also* Labor
 cesarean. *See* Cesarean delivery
 operative vaginal, 270–272, 393
 physiologic changes following, 278–280
 postpartum care, 273–276
 postpartum complications
 endometritis, 326–327
 hemorrhage. *See* Postpartum hemorrhage
 infection, 325
 placenta accreta, 323–324
 uterine inversion, 324–325
 spontaneous vaginal, 250
- Depo-Provera. *See* Medroxyprogesterone acetate
- Depression, postpartum, 280–281
- Dermatitis, vulvar, 117
- Descent, arrest of, 262
- Descent, fetal, 263, 264f
- Desquamation, in toxic shock syndrome, 87
- Desquamative inflammatory vaginitis, 84–85
- Detrusor muscle
 function, 178
 overactivity, 183, 184
 underactivity, 185
- Developmental age, of pregnancy, 236
- Dexamethasone
 for fetal lung maturation, 313–314
 for thyroid storm, 336
- Diabetes insipidus, 279
- Diabetes mellitus
 characteristics, 327
 polycystic ovarian syndrome and, 222
 in pregnancy. *See also* Gestational diabetes
 birth defects and, 285, 329, 426
 management, 460
 vulvovaginal candidiasis and, 83
 White Classification System, 331, 331f
- Diabetic ketoacidosis, 332–333
- Diagonal conjugate, 24, 259

- Diaphragm
 contraceptive, 53
 pelvic, 32, 32f
 urogenital, 32, 32f
- Diarrhea, in toxic shock syndrome, 87
- Diastasis recti, 279
- Dicloxacillin, for impetigo, 120
- Didelphys, uterine, 192
- Diethylstilbestrol (DES), teratogenic effects, 148, 404f
- Dilation, cervical, 261, 262
- Dilation and curettage (D&C), 14t, 58
 in dysfunctional uterine bleeding, 200
 in endometrial cancer, 150
- Dilation and evacuation (D&E), 14–15t, 58, 422
- Dilation and extraction (D&X), 58
- Dinoprostone, for cervical ripening, 266
- Direct Coombs test, 380
- Direct fluorescence antibody test, for herpes simplex, 65
- Discriminatory zone, 294
- Dizygotic twins, 389, 390
- DMPA. *See* Medroxyprogesterone acetate
- Domestic violence, 243, 409–411
- Dominant follicle, 39
- Donovan bodies, 71
- Dopamine agonists, for infertility, 208
- Doppler fetal heart monitor, 238
- Down syndrome. *See* Trisomy 21
- Doxorubicin, for endometrial cancer, 152
- Doxycycline
 for *C. trachomatis*, 73, 413t
 for Fitz-Hugh-Curtis syndrome, 78
 for gonorrhea, 73
 for granuloma inguinale, 71
 for lymphogranuloma venereum, 70
 for pelvic inflammatory disease, 77
- Doxylamine, for hyperemesis gravidarum, 342
- Dual energy x-ray absorptiometry (DEXA), 211, 464
- Ductus arteriosus, 226–227
- Ductus venosus, 226–227
- Duloxetine, for urinary incontinence, 186
- Duodenal atresia, 319
- Durable power of attorney for health care, 413–414
- Duty, 414–415
- Dysfunctional uterine bleeding. *See also* Abnormal uterine/vaginal bleeding
 anovulatory, 200
 conditions associated with, 199
 differential diagnosis, 199
 ovulatory, 200
 pathophysiology, 198–199
 patterns, 198
 treatment, 200
 workup, 199–200, 436
- Dysgerminoma, 158, 159, 456
- Dysmenorrhea
 in adenomyosis, 103
 in endometriosis, 98
 etiology, 196, 197
 incidence, 196
 signs and symptoms, 196–197
 treatment, 197–198, 432
 types, 196
- Dyspareunia
 in Bartholin gland cysts and abscesses, 118
 differential diagnosis, 115
 in endometriosis, 98
 treatment, 115
- Dyspnea, in ovarian cancer, 156
- Dystocia
 categories, 392
 definition, 252
 diagnostic criteria, 393
 external cephalic version for, 95
 failure to progress, 392–393, 444
 fetal malposition and, 394
 fetal malpresentation and, 394–395, 394f
 hypocontractile uterus in, 393
 incidence, 392
 poor progression in first stage, 393
 poor progression in second stage, 393
 prolonged latent phase, 393
 risk factors, 392
 transverse lie and, 395
- Dysuria
 in cystitis, 165
 in urethritis, 169
- E**
- Early decelerations, 249
- Eclampsia, 306
- Ectocervicitis, 72
- Ectocervix, 123
- Ectopic pregnancy
 definition, 290
 diagnosis and evaluation, 238, 292, 440
 differential diagnosis, 74, 96t, 291–292
 heterotopic, 292
 incidence, 290
 intrauterine devices and, 291
 management, 293–295, 294f
 mortality rate, 290
 natural course, 292
 physical findings, 94, 291
 risk factors, 291
 signs and symptoms, 291
 subsequent pregnancy and, 295
 tubal rupture in, 295
- Eczema, vulvar, 117
- Edwards syndrome. *See* Trisomy 18
- ELISA (enzyme-linked immunosorbent assay), 89, 277
- Embryology, 225
- Embryonal carcinoma, ovarian, 159
- Embryonal rhabdomyosarcoma, 148
- Embryopathy, 401
- Emergency contraception, 51–52, 413t
- Endocervical curettage, 130, 135
- Endocervix, 123
- Endocrine system
 changes in pregnancy, 232–233
 disorders
 diabetes mellitus. *See* Diabetes mellitus;
 Gestational diabetes
 hyperthyroidism. *See* Hyperthyroidism
 hypothyroidism, 336–338
 postpartum thyroiditis, 338–339, 338t
 Sheehan syndrome, 279, 322, 339
- Endodermal sinus tumor, 158, 159
- Endometrial biopsy
 in dysfunctional uterine bleeding, 200
 in pelvic tuberculosis, 81

- Endometrial cancer
 characteristics, 148–149
 diagnosis, 150
 endometrial hyperplasia and, 149–150
 estrogen and, 148
 metastases, 151
 polycystic ovarian syndrome and, 222
 posttreatment surveillance, 152
 progesterone and, 148
 prognosis, 152
 protective factors, 149
 risk factors, 149
 screening, 153
 signs and symptoms, 148
 staging, 151, 152†
 subtypes, 150–151
 tumor grading, 151
- Endometriomas, 99
- Endometriosis
 chronic pelvic pain and, 90
 complications, 97
 definition, 95
 differential diagnosis, 74, 99
 dysmenorrhea and, 197
 epithelial ovarian cancer and, 97
 etiology, 97–98
 genetic factors, 97
 histologic findings, 101
 implant sites, 98
 infertility and, 97, 102
 laboratory tests, 99–100
 pathophysiology, 97
 physical findings, 91, 95, 97, 99
 staging, 101
 symptoms, 98–99, 448
 treatment, 101–102, 453
- Endometritis, 275–276, 326–327
- Endometrium
 hyperplasia, 149–150, 161
 in menstrual cycle, 40–41
 stromal sarcoma, 153
- Engagement, fetal, 263, 264f
- Enterobius vermicularis*, 121
- Enterocele, 171, 172f, 177
- Epidermal cyst, 119
- Epidural block, in labor, 253, 393
- Epilepsy, in pregnancy, 344–346, 345t, 427
- Episiotomy, 9t, 270, 274
- Epithelial ovarian cancer. *See* Ovarian cancer, epithelial
- Erysipelas, 121
- Erythema infectiosum, 367–368
- Erythroblastosis fetalis, 379
- Erythromycin
 for chlamydia in pregnancy, 376
 eye drops, prophylactic in neonates, 376, 377
 for impetigo, 120
 for lymphogranuloma venereum, 70
 for syphilis, 68
- Escherichia coli*
 cystitis and, 166, 452
 in pyelonephritis, 170
- Esophagitis, bisphosphonates and, 212
- Estimated date of confinement (EDC), 236
- Estradiol, in menstrual cycle, 39–41
- Estrogen
 dysfunctional uterine bleeding and, 199, 200
 endometrial cancer and, 149
 in endometriosis, 97
 hepatic metabolism and, 231
 hyperemesis gravidarum and, 342
 intravaginal
 for atrophic vaginitis, 85, 216†
 for atypical squamous cells of undetermined significance, 131
 for urinary incontinence, 186
 leiomyomas and, 107
 in menopause, 214
 in perimenopause, 214
 in pregnancy, 232
 in puberty, 190
 vaginal blood flow and, 113
- Estrogen-progestin replacement therapy, 215–217
- Estrogen replacement therapy
 complications, 217
 contraindications, 217
 for menopause-related bone loss, 211, 212
 for menopausal symptoms, 216†
 risks, 216–217
- Ethambutol, for pelvic tuberculosis, 81
- Ethics and law, 413–415
- Etoposide
 for choriocarcinoma, 301
 for ovarian germ cell tumors, 160
 side effects, 160
- Expiratory reserve volume, in pregnancy, 229
- Expulsion, fetal, 263, 264f
- Extension, fetal, 263, 264f
- External beam radiation therapy, for cervical squamous cell carcinoma, 133
- External cephalic version, 9t, 395
- External os, cervix, 28
- External rotation, fetal, 263, 264f
- F**
- Face presentation, 394
- Fallopian tubes
 anatomy, 28
 benign neoplasms, 111
 cancer, 163–164, 457
 ligation/blockage for sterilization, 11t, 43–44
- Family planning. *See* Contraception
- FDA pregnancy drug categories, 402–403
- Fecundability, 204
- Female condom, 42, 53
- Female reproductive system
 abdominal wall, 33–34
 arteries and veins, 25, 25f
 bony pelvis, 23–24, 23f, 257
 congenital anomalies, 191–192
 ligaments, 30, 30f
 pelvic organs, 25–31, 26f
 perineum, 31–33, 31f
- Femoral nerve, injury to, 30–31, 274
- Fenestrated placenta, 309
- Fern test, amniotic fluid, 10t, 15t, 22f, 251, 311
- Fertility awareness methods, 42
- Fetal acidosis, 396
- Fetal alcohol syndrome, 403t, 445
- Fetal attitude/posture, 255
- Fetal blood sampling, 267–268
- Fetal circulation, 226–227, 227f
- Fetal dysmaturity syndrome, 400
- Fetal fibronectin, 250, 317
- Fetal growth restriction. *See* Fetus, growth restriction

- Fetal hydantoin syndrome, 403f
- Fetal lie, 254, 433
- Fetal position, 254–256, 255–256f, 394, 433
- Fetal presentation, 254–255, 255f, 394–395, 394f, 433
- Fetal scalp
 - electrode monitoring, 267
 - pH, 267–268
- Fetal warfarin syndrome, 347
- Fetopathy, 401
- Fetus
 - assessment in maternal diabetes, 332
 - birth defects, 318. *See also* Congenital anomalies
 - cardiac activity detection, 238
 - cardinal movements of labor, 263–265, 264f
 - chronic stress, 249
 - congenital infections. *See* Neonate, congenital infections
 - fontanels, 255
 - forces on head during labor, 252
 - growth restriction
 - alcohol use in pregnancy and, 403f
 - classification, 318, 384
 - complications, 385–386
 - definition, 318, 383–384
 - diagnosis and evaluation, 386
 - fetal factors, 318, 384–385
 - maternal factors, 385
 - placental factors, 385
 - heart rate variability, 248
 - hyperthyroidism, 335
 - hypoxic stress, 249
 - intrauterine demise, 319–320
 - lung maturity tests, 313
 - macrosomia
 - complications, 387
 - definition, 386
 - diagnosis, 387–388
 - gestational diabetes and, 329
 - incidence, 387
 - management, 388
 - risk factors, 387
 - maternal thalassemia and, 351
 - meconium aspiration syndrome, 399
 - metabolic acidosis, 250
 - monitoring
 - abnormal results
 - bradycardia, 247–248, 397
 - causes, 396
 - decelerations, 249, 397–399, 444–445
 - tachycardia, 247, 396–397
 - biophysical profile, 248–249, 460
 - non-stress test, 246–247, 247f
 - in post-term pregnancy, 400–401
 - routine, in prenatal care, 246–247
 - perfusion assessment, 249
 - radiation exposure, 459
 - thyrotoxicosis, 335
 - weight gain, 240
- Fever
 - in postoperative gynecologic patient, 86–87
 - in pyelonephritis, 169
 - in toxic shock syndrome, 87
 - in tubo-ovarian abscess, 79
- Fibroids. *See* Leiomyomas
- Fibroma, ovarian, 160, 161
- Fibromyomas. *See* Leiomyomas
- Fibrous dysplasia, 193
- Fifth disease, 367–368
- FIGO system
 - cervical cancer stages, 127, 128f
 - vaginal cancer stages, 146, 146f
 - vulvar cancer stages, 139, 140f
- Filling cystometry, 182
- Finasteride, for polycystic ovarian syndrome, 221
- Fitz-Hugh-Curtis syndrome, 75, 77–78
- Flank pain, in pyelonephritis, 169
- Flexion, fetal, 263, 264f
- Flexion, uterus, 27
- Fluconazole, for vulvovaginal candidiasis, 83–84
- Flunitrazepam, 412
- Fluorescent treponemal antibody
 - absorption (FTA-Abs), 68, 365
- Fluoroquinolones. *See* Quinolones
- 5-Fluorouracil (5-FU), for cervical squamous cell carcinoma, 133
- Flutamide, for polycystic ovarian syndrome, 221
- Folic acid
 - deficiency
 - anticonvulsants and, 345
 - fetal effects, 350
 - supplementation, in pregnancy, 235, 345, 350
- Follicle-stimulating hormone (FSH)
 - in amenorrhea, 203
 - challenge test, 207
 - delayed menarche and, 194
 - in menopause, 214
 - in menstrual cycle, 37–40, 38f
 - in perimenopause, 214
- Follicular cysts, ovarian, 109–110
- Follicular phase, menstrual cycle, 37, 39
- Folliculitis, 120
- Fontanels, 255
- Footling breech, 254, 255f, 394
- Foramen ovale, 226–227
- Forceps delivery, 10f, 271–272
- Fox-Fordyce disease, 119, 452
- Fracture, in osteoporosis, 210, 464
- Fragile X syndrome, 387
- Frank breech, 254, 255f, 395
- Frenulum, 33
- Functional residual capacity, in pregnancy, 229
- Functional layer, endometrium, 41
- Fundus, uterine, 27
 - height, for pregnancy dating, 237–238, 318
 - in postpartum period, 274
- Furuncles, 120–121
- G**
- Gallstones, in pregnancy, 356
- Gamete intra-fallopian tube transfer (GIFT), 209
- Gamma-hydroxybutyrate (GHB), 412
- Gardasil, 62, 131
- Gardnerella vaginalis*, 82
- Gartner duct cyst, 120
- Gastrointestinal cancer, ovarian metastases, 162
- Gastrointestinal disorders, in pregnancy
 - acute fatty liver, 339–340
 - appendicitis, 231, 355–356, 419
 - hyperemesis gravidarum, 231, 334f, 342–343
 - intrahepatic cholestasis, 340–342
 - morning sickness, 231
 - pancreatitis, 231
- Gastroschisis, 245
- GBS. *See* Group B *Streptococcus*
- Genetic and family history, 240
- Genital tract biopsy, 15f

- Genital ulcers, 63–64
 chancroid, 69
 granuloma inguinale, 71
 herpes simplex virus, 64–66, 438
 in HIV infection, 89
 lymphogranuloma venereum, 69–70
 syphilis, 67–69
- Genital warts, 61–62
- Genitofemoral nerve, injury to, 31
- Genomic imprinting, 285
- Gentamicin
 for endometritis, 276, 326
 for pelvic inflammatory disease, 77
 for pyelonephritis, 170
 for tubo-ovarian abscess, 79
- Germ cell tumors, ovarian, 158–160
- German measles, 360–361
- Gestational age, categories, 236–237
- Gestational diabetes
 cesarean delivery in, 333
 characteristics, 327
 complications, 328–329, 426, 461
 fetal assessment in, 332, 461
 fetal macrosomia and, 329, 387
 management, 331–332
 pathophysiology, 233, 327–328
 risk factors, 328
 screening, 246, 329–330, 330†
 thyroid dysfunction and, 328
 type II diabetes risk following, 280
- Gestational hypertension, 302–303, 442
- Gestational trophoblastic disease
 benign vs. malignant, 295
 choriocarcinoma, 300–301, 441
 differential diagnosis, 296
 hydatidiform mole, 297–300, 441
 hyperthyroidism and, 334†
 invasive mole, 300
 placental site tumor, 301
 prognosis, 297
 risk factors, 296
 signs and symptoms, 296
 stages, 296–297
 subsequent pregnancy and, 297
 types, 295
- Giemsa stain, 89
- Glucocorticoids. *See* Corticosteroids
- Glucose challenge test, 244, 245, 330
- Glucose metabolism, in pregnancy, 233.
See also Gestational diabetes
- Glucose tolerance test, 245–246, 330
- Glycosylated hemoglobin (HbA_{1c}), 329†
- Gonadotropin-releasing hormone (GnRH)
 for infertility, 208
 for leiomyomas, 108
 in menstrual cycle, 37
 in precocious puberty, 193
- Gonadotropin-releasing hormone (GnRH) agonists
 for chronic pelvic pain, 93
 for endometriosis, 102
- Gonococcal ophthalmia, 376
- Gonorrhea
 diagnosis, 72
 mucopurulent cervicitis in, 71–72
 in pregnancy, 376
 prophylaxis after sexual assault, 413†
 screening, 73, 244
 treatment, 73
- Gorlin syndrome, 161
- Goserelin, for chronic pelvic pain, 93
- Gram-negative bacteria, in tubo-ovarian abscess, 79
- Grand multiparity, 252
- Granuloma inguinale, 71
- Granulosa cell(s), 39
- Granulosa cell tumors, 160, 161, 218
- Graves disease, 335
- Gravidity, 237
- Groove sign, 70
- Group B *Streptococcus* (GBS)
 diagnosis, 374
 in endometritis, 326
 incidence, 374
 in pregnancy, 374–375, 434
 screening test, 244
- Growth patterns, in puberty, 190
- Gynecoid pelvis, 24
- Gynecologic history, 240
- H**
- Haemophilus ducreyi*, 69
- HAIR-AN syndrome, 222
- hCG. *See* Human chorionic gonadotropin
- Heart rate
 fetal, 247–248, 396–399
 in pregnancy, 228
- Hegar sign, 234
- HELLP syndrome, 305–306, 340
- Hemagglutination assay tests (HATTS), 68
- Hematology tests/values
 in preeclampsia, 304–305
 in pregnant vs. nonpregnant woman, 4†, 228–229, 232
 in prenatal care, 243
- Hematoma, vulvar, 122
- Hematoxylin and eosin (H&E) stain,
 in actinomycosis, 80
- Hematuria, in cystitis, 165
- Hemoglobinopathies, 350–351
- Hemolytic disease of the newborn, 244
- Heparin, 406
- Hepatitis
 breastfeeding and, 277, 373
 chronic, 370–371
 differential diagnosis, 370
 false-positive syphilis test in, 68
 laboratory tests, 370
 prophylaxis after sexual assault, 413†
 signs and symptoms, 370
- Hepatitis A, 371
- Hepatitis B, 371–373
- Hepatitis C, 373, 426
- Hepatitis D, 373–374
- Hepatitis E, 374
- Hepatocellular adenoma, oral
 contraceptives and, 47
- Hereditary cancer syndromes, 155
- Hereditary nonpolyposis colon cancer, 155
- Heroin, teratogenic effects, 405†
- Herpes simplex virus (HSV)
 ectocervicitis and, 72
 genital infection, 64–66, 438
 in pregnancy, 66, 363–364
 urethritis and, 169
- Hidradenitis suppurativa, 121
- Hidradenomas, 120

- High-grade squamous intraepithelial lesion, 130*t*, 131, 428
- Hirsutism
 definition, 217
 disorders causing, 218, 219
 evaluation, 219
 idiopathic, 219
 in ovarian sex cord-stromal tumors, 161
 pathophysiology, 218
 in polycystic ovarian syndrome, 221
 in pregnancy, 234
 treatment, 220
 vs. virilization, 218
- HIV. *See* Human immunodeficiency virus (HIV) infection
- Hormone replacement therapy, 215–217, 216*t*, 464
- Horner syndrome, congenital varicella and, 367
- Hot flashes
 in ovarian failure, 202
 in perimenopause, 214
- Human chorionic gonadotropin (hCG)
 in amenorrhea, 202
 after delivery, 280, 437
 in dysfunctional uterine bleeding, 200
 in ectopic pregnancy, 236, 292, 294–295
 in hydatiform mole, 298
 in hyperemesis gravidarum, 342
 in ovarian germ cell tumors, 159, 160
 in pregnancy, 232, 235–236
- Human immunodeficiency virus (HIV) infection
 characteristics, 368
 diagnosis, 89, 368–369
 hepatitis B infection and, 371
 opportunistic infections in, 89
 in pregnancy
 antiretroviral therapy in pregnancy, 369
 cesarean delivery and, 369, 370, 443
 intrapartum precautions, 369–370
 mother-to-child transmission, 369, 443
 screening test, 244
 prophylaxis after sexual assault, 413*t*
 signs and symptoms, 88–89
 transmission, 88
- Human menopausal gonadotropin (hMG), for infertility, 208
- Human papillomavirus (HPV)
 cervical cancer and, 62, 125
 ectocervicitis and, 72
 in genital warts, 61
 HIV infection and, 88
 vaccine, 62, 131
- Human placental lactogen (hPL), 233
- Hutchinson triad, 365
- Hyaline degeneration, leiomyoma, 107
- Hydatid cysts of Morgagni, 111
- Hydatidiform mole, 297–300
- Hydralazine, for hypertension in pregnancy, 302
- Hydrocephalus, in congenital toxoplasmosis, 358
- Hydrocortisone, for vulvovaginal candidiasis, 83–84
- Hydronephrosis
 in congenital varicella, 366
 in pregnancy, 230
- Hydrops fetalis, 368
- Hydrops tubae profluens, 163
- 17-Hydroxylase deficiency, amenorrhea in, 202
- Hymen, imperforate, 191
- Hyperemesis gravidarum, 231, 334*t*, 342–343, 425
- Hyperglycemia. *See* Diabetes mellitus
- Hyperpigmentation, in pregnancy, 234
- Hyperprolactinemia
 infertility treatment in, 208
 sexual dysfunction and, 114
 signs and symptoms, 219
- Hyperstimulation, uterine, 267
- Hypertensive disorders, in pregnancy
 antihypertensives for, 302
 chronic hypertension, 302
 definition, 302
 gestational hypertension, 302–303, 442
 incidence, 302
 preeclampsia. *See* Preeclampsia types, 301
- Hyperthyroidism
 complications, 334, 334*t*
 diagnosis, 335
 etiology, 334
 fetal, 335
 management in pregnancy, 335
 signs and symptoms, 333, 335
- Hypertrichosis, 218
- Hypoactive sexual disorder, 114
- Hypogonadotropic hypogonadism
 infertility treatment in, 208
 in Kallmann syndrome, 194
- Hypomenorrhea, 198
- Hypopituitarism, after postpartum hemorrhage, 279
- Hypothalamic-pituitary dysfunction, 201–202, 436
- Hypothalamus, in menstrual cycle, 37
- Hypothyroidism, 336–338, 338*t*
- Hypoxia, fetal, 396
- Hysterectomy, 16*t*
 for adenomyosis, 103
 for cervical adenocarcinoma, 136
 for cervical squamous cell carcinoma, 132–133
 for chronic pelvic pain, 93
 for endometriosis, 102
 for leiomyomas, 108
- Hysterosalpingography, 17*t*, 107
- Hysteroscopy, 17*t*
 in endometrial cancer, 150
 in leiomyoma, 107
- Hysterosonography, 17*t*
- I**
- Iliococcygeus suspension, 177
- Imipenem/cilastin, for tubo-ovarian abscess, 79
- Immunity, in pregnancy, 232
- Immunoglobulin G (IgG)
 in hepatitis A, 371
 in parvovirus B19, 368
 in TORCH infections, 357
- Immunoglobulin M (IgM)
 in hepatitis A, 371
 in parvovirus B19, 368
 in TORCH infections, 357
- Immunosuppression, vulvovaginal candidiasis and, 83
- Imperforate hymen, 191
- Impetigo, 120
- Implants, contraceptive, 50
- Incest, 411
- Incomplete abortion, 287
- Indirect Coombs test, 380
- Indomethacin, for tocolysis, 269, 318

- Induction of labor, 265–266
 Inevitable abortion, 287, 439
 Infant, 237. *See also* Neonate
 Infections, in pregnancy. *See* Pregnancy, infections during
 Infectious mononucleosis, false-positive syphilis test in, 68
 Infertility
 assisted reproductive technologies for, 209
 definition, 204
 endometriosis and, 97, 99, 102, 207
 etiology, 204–205, 207, 463
 evaluation, 206–207, 448, 462, 463
 leiomyomas and, 106
 ovulatory dysfunction in, 205–207
 pelvic inflammatory disease and, 77
 pelvic tuberculosis and, 81
 in polycystic ovarian syndrome, 221
 prevalence, 204
 treatment, 207–209
 tubal, 207
 Influenza vaccine, in pregnancy, 242, 351
 Informed consent, 414
 Inguinal canal, 34
 Inguinal mass, 192
 INH (isonicotinyl hydrazine), for pelvic tuberculosis, 81
 Injectable contraceptives, 49
 Intermenstrual bleeding, 198
 Internal iliac artery, 25, 25f
 Internal os, cervix, 28
 Internal rotation, fetal, 263, 264f
 Interspinous diameter, 259
 Interstitial cystitis, 184
 Intimate partner violence, 243, 409–411
 Intracytoplasmic sperm injection (ICSI), 209
 Intrahepatic cholestasis of pregnancy, 340–342
 Intrauterine devices (IUDs)
 actinomycosis and, 80
 benefits, 55
 characteristics, 54, 55f
 contraindications, 56
 ectopic pregnancy and, 291
 as emergency contraception, 52
 expulsion, 56
 failure rate, 42, 55
 pelvic inflammatory disease and, 56
 postpartum insertion, 281
 risks, 55
 side effects, 55
 Intrauterine growth restriction. *See* Fetus, growth restriction
 Intrauterine insemination (IUI), 208
 Intrauterine pressure catheter (IUPC), 266
 Intravenous pyelogram, in cervical cancer, 127
 Invasive mole, 300
 In vitro fertilization-embryo transfer (IVF-ET), 209
 Iodine, contraindication in pregnancy, 335
 Iron deficiency anemia, in pregnancy, 350
 Ischial spine, 24
 Isoflavones, 213
 Isotretinoin, teratogenic effects, 404f
 Isthmus, uterine, 27
- J**
- Jarisch-Herxheimer phenomenon, 68, 365
- K**
- Kallmann syndrome, 194
 Kanamycin, teratogenic effects, 405f
 Kegel exercises, 175, 186
 Kick count, 246
Klebsiella granulomatis, 71
 Kleihauer-Betke test, 310, 382, 461
 Klinefelter syndrome, 285
 KOH prep, in vulvovaginal candidiasis, 83
 Koilocytes, 125
 Koilocytosis, 61
 Krukenberg tumors, 162
- L**
- Labetalol, for hypertension in pregnancy, 302
 Labia majora, 33
 Labia minora, 33
 Labor. *See also* Delivery
 abnormal. *See* Dystocia
 anesthesia in, 253–254
 assessment and monitoring, 266–268
 cardinal movements, 263–265, 264f
 cervical change in, 251
 definition, 250
 diagnosis, 250
 episiotomy in, 270
 fetal head during, 252
 induction of, 265–266
 management, 268–270
 preterm, 242–243, 314–317
 protracted, 261–262
 signs and symptoms, 250–251
 stages, 260–263
 Lacerations, vaginal/perineal, 269–270, 274
 Lactation. *See* Breastfeeding
 Lactic dehydrogenase (LDH), in ovarian germ cell tumors, 159, 160
 Lactobacilli, vaginal, 81
 Laminaria, 266
 Laparoscopically assisted vaginal hysterectomy, 18f. *See also* Hysterectomy
 Laparoscopy, 18f
 in chronic pelvic pain, 92
 in endometriosis, 100
 in infertility evaluation, 207
 Laparotomy, 19f
 in endometriosis, 100
 in ovarian cancer, 157
 in septic abortion, 60
 Large for gestational age, 387
 Laser vaporization, 19f
 Late decelerations, 249
 Latent phase, labor, 260f, 261
 Lead, teratogenic effects, 405f
 Lea's shield, 53
 Lecithin/sphingomyelin (L/S) ratio, 313
 Leiomyomas
 bleeding mechanism with, 106–107
 characteristics, 105
 complications, 108
 degeneration, 107–108
 differential diagnosis, 96f
 evaluation, 107, 424, 447, 454
 metastases, 108–109
 pathophysiology, 107
 physical findings, 91, 447
 prevalence, 106
 risk factors, 106

- Leiomyomas (*Cont.*)
 symptoms, 106, 197, 447
 treatment, 108, 424, 447, 455
 types, 105, 105f
- Leiomyosarcoma, 109, 143, 153
- Leopold maneuvers, 256–257, 258f
- Leucovorin, in toxoplasmosis, 360
- Leuprolide, for chronic pelvic pain, 93
- Levofloxacin
 for asymptomatic bacteriuria in pregnancy, 344f
 for pelvic inflammatory disease, 77
- Levonorgestrel-releasing intrauterine devices, 54.
See also Intrauterine devices
- Levothyroxine, for hypothyroidism, 337
- Lichen planus, 116–117
- Lichen sclerosis, 116, 215, 450
- Lichen simplex chronicus, 117
- Ligaments, of female pelvis, 30, 30f, 173f
- Liley curve, 383
- Lindane, 63
- Linea alba, 34
- Linea nigra, 234
- Linea semilunare, 34
- Liquid-based cytology, for cervical cancer screening, 128–129
- Listeria* infection, 375–376
- Lithium, teratogenic effects, 405f
- Liver biopsy, in acute fatty liver of pregnancy, 340
- Liver function tests
 in acute fatty liver of pregnancy, 340
 in cervical cancer, 127
 in hyperemesis gravidarum, 342
 in intrahepatic cholestasis of pregnancy, 341
 in pregnancy, 231
- Living will, 413
- Lochia, 274–275
- Londersloot test, 310
- Long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD), 339
- Loop electrosurgical excision procedure (LEEP), 19t, 130. *See also* Cervical conization
- Low birth weight infant. *See* Fetus, growth restriction
- Low-grade squamous intraepithelial lesion, 129, 130t, 131
- Lunelle, 49
- Luteal phase, menstrual cycle, 37, 39–40
- Luteinizing hormone (LH)
 in menopause, 214
 in menstrual cycle, 37–40, 38f
 urinary, 206
- Lymph node dissection
 in vulvar melanoma, 142
 in vulvar squamous cell carcinoma, 139
- Lymphadenopathy
 in chancroid, 69
 in lymphogranuloma venereum, 70
 in syphilis, 67
- Lymphogranuloma venereum, 69–70
- Lynch II syndrome, 155
- M**
- Macrocytic anemia, in pregnancy, 350
- Macrosomia, fetal
 complications, 387
 definition, 386
 diagnosis, 387–388
 gestational diabetes and, 329
 incidence, 387
 management, 388
 risk factors, 387
- Magnesium sulfate
 for eclampsia, 306
 for tocolysis, 269
 toxicity, 306–307
- Magnetic resonance imaging (MRI), 20t
 in appendicitis, 356
 in cervical cancer, 127
 in hirsutism, 219
 in hypogonadotropism, 203
- Male condom, 42, 52–53
- Malpractice, 414–415
- Mammography, 20t
- Mammoplasty, breastfeeding after, 278
- Manual vacuum aspiration, 58
- Marfan syndrome, 355t
- Marijuana, teratogenic effects, 405t
- Marital rape, 411
- Marshall-Marchetti-Krantz colposuspension, 187
- Mayer-Rokitansky-Kuster-Hauser syndrome, 191
- McCune-Albright syndrome, 193
- McRoberts maneuver, 442
- Mebendazole, for pinworm, 121
- Meconium, 399, 434
- Meconium aspiration syndrome, 399
- Medical abortion, 59
- Medroxyprogesterone acetate (DMPA)
 bone loss and, 210
 for chronic pelvic pain, 93
 for contraception, 49
- Meigs syndrome, 161
- Melanoma, vulvar, 138, 142
- Melasma, 233
- Menarche
 characteristics, 189
 delayed, 194
 premature, 192
- Meningitis, in *Listeria* infection, 376
- Menometrorrhagia, 432
- Menopause. *See also* Postmenopausal women
 age of onset, 213
 hormone replacement therapy in, 215–217, 216t, 464
 osteoporosis in, 215, 439
 phases, 213–214
 premature, 213
 symptoms, 215, 216t, 464–465
- Menorrhagia
 in adenomyosis, 103
 definition, 198, 432
 differential diagnosis, 106
- Menstrual cramps, 41
- Menstrual cycle
 conditions affected by, 198
 disorders
 amenorrhea. *See* Amenorrhea
 dysfunctional uterine bleeding. *See* Dysfunctional uterine bleeding
 dysmenorrhea. *See* Dysmenorrhea
 premenstrual syndrome, 195–196
 in perimenopause, 214
 physiology, 37–41, 38f
 in postpartum period, 281, 438
- Menstruation, retrograde, 97–98

- Mental disorders
 pelvic pain and, 90
 postpartum depression, 280–281
 postpartum psychosis, 281
- Metabolic acidosis, fetal, 250
- Metformin, for polycystic ovarian syndrome, 221
- Methotrexate
 for choriocarcinoma, 300
 for ectopic pregnancy, 293
 for medical abortion, 59
 teratogenic effects, 404f
- Methyl mercury, teratogenic effects, 405t
- Methyldopa, for hypertension in pregnancy, 302
- Methylergonovine, contraindications, 273
- Metronidazole
 for bacterial vaginosis, 83, 451
 for Fitz-Hugh-Curtis syndrome, 78
 for pelvic inflammatory disease, 77
 for *T. vaginalis*, 73, 84
 for tubo-ovarian abscess, 79
- Metrorrhagia, 198, 432
- Microhemagglutination assay (MHA-TP), 68, 365
- Midurethral anti-incontinence procedures, 187
- Mifepristone
 as emergency contraception after sexual assault, 413t
 for medical abortion, 59
- Migraine headache, in pregnancy, 429
- Minute ventilation, in pregnancy, 229
- Miscarriage, 286–289
- Misoprostol
 for cervical ripening, 266
 for medical abortion, 59
- Missed abortion, 287, 440
- Mitral regurgitation, 354t
- Mitral stenosis, 354t
- Mittelschmerz, 95
- Mixed incontinence, 179, 184. *See also* Urinary incontinence
- Mixed müllerian tumors, uterine, 153
- Molar pregnancy, 297–300, 441
- Molding, fetal head, 252
- Molluscum contagiosum, 62–63
- Monozygotic twins, 225, 389
- Montevideo units, 266–267
- “Morning-after pill,” 51–52, 413t
- Morning sickness, 231
- MRI. *See* Magnetic resonance imaging
- Müllerian agenesis, amenorrhea and, 202
- Müllerian anomalies, amenorrhea and, 202
- Multi-fetal gestation
 complications, 391
 diamniotic/monochorionic placentation, 389–390, 390f
 dichorionic/diamniotic placentation, 390, 390f, 419
 dizygotic twins, 387
 fetal morbidity and mortality and, 388–389
 fetal reduction in, 391
 incidence, 388
 maternal age and, 388
 maternal morbidity and mortality and, 389
 monoamniotic/monochorionic placentation, 389, 390f
 monozygotic twins, 225, 387
 preterm labor in, 391
 twin-twin transfusion syndrome in, 390–391
- Multigravida, 252
- Multipara, 260
- Multiple sclerosis, pregnancy and, 819
- Mycobacterium bovis*, 80
- Mycobacterium tuberculosis*, 80
- Mycoplasma hominis*, 82
- Myofascial pain, 92
- Myomas. *See* Leiomyomas
- Myomectomy, 108
- N**
- Nabothian cysts, 103–104
- Nafarelin, for chronic pelvic pain, 93
- Nafcillin, for toxic shock syndrome, 88
- Nagele’s rule, 236
- Nausea/vomiting
 in acute fatty liver of pregnancy, 339
 in ovarian cancer, 156
 in pregnancy, 231. *See also* Hyperemesis gravidarum
 in pyelonephritis, 169
 in toxic shock syndrome, 87
- Neisseria gonorrhoeae*, 72–73, 169, 438
- Neonate
 Apgar score, 276
 congenital cretinism, 337
 congenital infections
Chlamydia trachomatis, 376–377
 cytomegalovirus, 361–363
 group B streptococcus, 375
 hepatitis B, 372–373
 herpes simplex virus, 66, 363–364
 HIV, 369–370
Listeria, 375–376
 parvovirus B19, 368
 rubella, 360–361
 syphilis, 364–365
 toxoplasmosis, 358–359
 varicella, 366–367
 definition, 237
 eye ointments, 276
 hypothyroidism, 337–338
 large for gestational age, 387. *See also* Fetus, macrosomia
 skin-to-skin contact, 277
 small for gestational age, 385. *See also* Fetus, growth restriction
- Neural tube defects, 245, 319, 403t
- Neuropathy, 92
- New York Heart Association, heart disease classification, 352t, 420
- Nifedipine
 for hypertension in pregnancy, 302
 for tocolysis, 269
- Nitrazine blue test, 251
- Nitrofurantoin
 for cystitis, 166
 for urinary tract infections in pregnancy, 168, 343, 344t
- Nocturia, 179
- Non-stress test (NST), 246–247, 247f
- Nondisjunction, 284
- Nonoxynol-9, 53, 54
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
 for chronic pelvic pain, 93
 for dysmenorrhea, 197
 for endometriosis, 102

- Nuchal translucency screen, 244, 245
 Nulligravida, 251
 Nullipara, 252, 260
- O**
- Obstetric conjugate, 24, 259
 Obstetrical history, 240
 Obturator nerve, injury to, 30–31
 Occiput posterior (OP) position, 255, 394
 Ofloxacin
 for Fitz-Hugh-Curtis syndrome, 78
 for pelvic inflammatory disease, 77
 Ogita test, 310
 Oligohydramnios, 249, 318, 398
 Oligomenorrhea, 198
 Omental caking, in ovarian cancer, 156
 Omphalocele, 245
 Oocyte donation, 209
 Oophorectomy, prophylactic, 155
 Operative vaginal delivery, 270–272, 393
 Oral contraceptives
 advantages, 45–46
 amenorrhea following cessation, 48
 as anti-androgens, 220
 breastfeeding and, 281
 for chronic pelvic pain, 93
 contraindications, 46–47
 dosage, 45
 drug-drug interactions, 48
 for dysfunctional uterine bleeding, 200
 for dysmenorrhea, 197
 for endometriosis, 101
 failure rate, 42
 forgotten doses, 48
 in polycystic ovarian syndrome, 221
 risks, 46
 side effects, 47
 types, 45
 Orgasmic dysfunction, 115
 Osteopenia, 211, 439
 Osteoporosis
 causes, 210
 definition, 209, 465
 diagnosis, 210, 464
 DMPA and, 49, 210
 epidemiology, 210
 fractures in, 210, 464
 pathogenesis, 210, 439
 pregnancy-associated, 426
 prevalence, 209
 risk factors for fracture, 211–212
 signs and symptoms, 210, 464
 treatment, 212–213
 Ovarian cancer
 categories, 154
 clomiphene citrate and, 208
 epithelial
 endometriosis and, 97, 155
 epidemiology, 154
 pathogenesis, 154
 protective factors, 155
 risk factors, 155
 subtypes, 155–156
 germ cell tumors, 158–160
 hereditary syndromes, 155
 incidence, 154
 metastases, 156
 mortality rate, 154
 prognosis, 158
 prophylactic oophorectomy for, 155
 screening, 155
 sex cord-stromal tumors, 160–162
 signs and symptoms, 156
 staging, 157, 157*t*, 449
 treatment, 158
 Ovarian cyst
 functional, 109–111
 management, 423, 446, 456
 ruptured/hemorrhagic, 74, 96*t*
 Ovarian torsion, 74–75, 95, 96*t*
 Ovarian veins, 29
 Ovary(ies)
 anatomy, 28–29
 benign neoplasms, 111
 cancer. *See* Ovarian cancer
 cyst. *See* Ovarian cyst
 dysfunction
 amenorrhea and, 202
 evaluation, 205
 infertility and, 205–206
 in menstrual cycle, 38–39
 tumors metastatic to, 162–163
 Overactive bladder, 179
 Overflow incontinence, 180, 185. *See also* Urinary incontinence
 Oxacillin, for toxic shock syndrome, 88
 Oxybutynin, for urinary incontinence, 186
 Oxygen consumption, in pregnancy, 229
 Oxytocin
 actions, 233
 characteristics, 232–233
 contraindications, 273
 after delivery, 269, 323
 for hypocontractile uterus, 393
 in labor, 268
 for poor progression in first stage of labor, 393
- P**
- Paclitaxel
 for endometrial cancer, 152
 for ovarian cancer, 158
 side effects, 152
 Paget disease, of vulva, 144, 431
 Palmar erythema, in pregnancy, 234
 Pancreatitis, in pregnancy, 231
 Pap smear, 21*t*, 128–129, 427
 Papillary serous carcinoma, endometrial, 151
 Paramethadione, teratogenic effects, 403*t*
 Parasympathetic nervous system, bladder function and, 178
 Parathyroid hormone, for osteoporosis, 212
 Parity, 237
 Parvovirus B19, 367–368
 Patau syndrome. *See* Trisomy 13
 Patient confidentiality, 414
 PCOS. *See* Polycystic ovarian syndrome
 Pediculosis pubis, 63
 Pelvic abscess, 87
 Pelvic actinomycosis, 80
 Pelvic configurations, 24
 Pelvic diaphragm, 32, 32*f*, 173*f*
 Pelvic examination
 bimanual, 12*t*, 22*f*
 in labor, 257–258
 Pelvic infection, postoperative, 86–87
 Pelvic inflammatory disease (PID)

- cervicitis and, 73
- complications, 77
- definition, 73
- diagnostic tests, 75–76
- differential diagnosis, 74–75, 96f
- intrauterine devices and, 56
- prevention, 77
- risk factors, 75
- signs and symptoms, 73–74, 91
- treatment, 76–77
- Pelvic inlet, 24
- Pelvic masses
 - benign
 - cervical cysts, 103–104
 - cervical polyps, 104
 - fallopian tube, 111
 - leiomyomas. *See* Leiomyomas
 - ovarian cyst
 - ovarian neoplasms, 111
 - in ovarian cancer, 156
- Pelvic muscle exercises, 175, 186
- Pelvic organ prolapse
 - characteristics, 171
 - defects in, 171, 172–173
 - diagnosis, 174
 - grading systems, 174–175, 175t
 - incidence, 171
 - management, 175–177, 176f, 457
 - risk factors, 174
 - symptoms, 174
- Pelvic Organ Prolapse Quantification (POPQ)
 - system, 175
- Pelvic pain
 - acute
 - differential diagnosis, 96t
 - in endometriosis, 98
 - etiology, 94
 - physical findings, 94–95
 - workup, 95
 - in cervical cancer, 126
 - chronic
 - definition, 89
 - in endometriosis, 98
 - etiology, 89–90
 - prevalence, 89
 - treatment, 93
 - workup, 91–92
 - in imperforate hymen, 191
- Pelvic pressure, in ovarian cancer, 156
- Pelvic tuberculosis, 80–81
- Pelvimetry, 10t, 257–260
- Pelvis
 - bones, 23–24, 23f, 257
 - organs, 25–31, 26f
- Penicillin
 - for group B streptococcus, 375
 - for syphilis, 68, 365
- Penile neoplasia, human papillomavirus and, 125
- Pentamidine, for *P. carinii* infection, 89
- Peptostreptococcus, 78
- Perimenopause, 213–214
- Perineal lacerations, 269–270
- Perineum, 31–33, 31f
- Peripartum cardiomyopathy, 354–355t, 355
- Peripartum pelvic pain syndrome, 90
- Periumbilical artery blood sampling (PUBS), 11f
- Periurethral bulking agents, for urinary
 - incontinence, 187
- Permethrin, 63
- Peroneal nerve, injury to, 31
- Pessary(ies)
 - continence, 186
 - for pelvic organ prolapse, 176, 176f
- Pfannenstiel incision, 272
- Phenobarbital
 - side effects, 345t
 - teratogenic effects, 403t
- Phenylketonuria, birth defects and, 285
- Phenytoin
 - side effects, 345t
 - teratogenic effects, 403t
- Phthirus pubis*, 63
- Physical abuse, chronic pelvic pain and, 90
- Physiologic anemia of pregnancy, 349
- Pinworm, 121
- Pitting edema, in pregnancy, 230
- Pituitary, in hypogonadotrophism, 203
- Placenta
 - abnormal, 307–309, 308f, 385, 435
 - membranes, 225, 226f
- Placenta accreta, 323–324
- Placenta diffusa, 309
- Placenta increta, 323
- Placenta percreta, 323
- Placenta previa, 307–308, 308f, 310–311, 435, 442
- Placental abruption, 310, 320
- Placental site trophoblastic tumor, 301
- Plan B, 51–52, 413t
- Plasma osmolality, in pregnancy, 230
- Platypeloid pelvis, 24
- Pneumocystis carinii*, 89
- Pneumonia, varicella, 366
- Polycystic ovarian syndrome (PCOS)
 - anovulation in, 220–221
 - characteristics, 220
 - complications, 222
 - diagnosis and evaluation, 221
 - pathophysiology, 220
 - signs and symptoms, 219
- Polyhydramnios, 318
- Polymenorrhea, 198
- Polymerase chain reaction (PCR), for
 - lymphogranuloma venereum, 70
- Polyps, cervical, 104
- Portio, cervical, 28
- Postcoital test, 463
- Posterior asynclitism, 256
- Posterior vaginal colporrhaphy, 177
- Postmaturity syndrome, 400
- Postmenopausal women. *See also* Menopause
 - bone loss in, 211. *See also* Osteoporosis
 - osteoporosis in, 215, 439
 - uterine/vaginal bleeding in, 199, 200, 436
- Postpartum blues, 280
- Postpartum care
 - breastfeeding and infant care, 276–278
 - depression, 280–281
 - fertility and contraception, 281
 - physiologic changes and resolution, 278–280
 - puerperium management, 273–276
 - thyroiditis, 280
- Postpartum depression, 280–281

- Postpartum hemorrhage
 causes, 322–323
 definition, 321
 hypopituitarism following, 279
 management, 323, 441
 morbidities following, 322
 mortality rate, 321
 oxytocin for prevention of, 269
 placenta accreta and, 323–324
 prevention, 323
 primary vs. secondary, 322
 risk factors, 322
- Postpartum psychosis, 281
- Post-term pregnancy, 400–401
- Post-void residual urine, 181
- Potassium iodide, for thyroid storm, 336
- Power of attorney for health care, 413–414
- Poxvirus, in molluscum contagiosum, 62
- Prader-Willi syndrome, 285
- Precipitous labor, 262–263
- Precocious puberty
 causes, 193, 453
 classification, 193
 in ovarian sex cord-stromal tumors, 161
 treatment, 193
- Preconceptive counseling, 234–235
- Preeclampsia
 chronic hypertension and, 304
 diagnosis, 303
 vs. gestational hypertension, 302, 442
 management, 306, 437, 442
 maternal complications, 305
 risk factors, 303–304, 421
 severe, 304–305
 signs and symptoms, 303
 types, 304
- Pregnancy. *See also* Delivery; Labor; Postpartum care; Prenatal care
 arterial blood gases in, 5*t*
 blood chemistry in, 5*t*
 blood pressure in, 228, 229
 breast changes in, 234
C. trachomatis infection in, 73
 cardiovascular effects, 227–229, 425
 cervical changes in, 234
 complications
 abnormal placentation, 307–309, 308*f*
 gestational trophoblastic disease.
 See Gestational trophoblastic disease
 hypertensive disorders.
 See Hypertensive disorders, in pregnancy
 intrauterine fetal demise, 319–320
 oligohydramnios, 249, 318
 polyhydramnios, 318
 in post-term pregnancy, 400
 premature rupture of membranes,
 251, 311–312
 preterm labor and delivery, 242–243, 314–317
 preterm premature rupture of
 membranes, 251, 312–314
 third trimester bleeding, 309–311
 developmental age, 236
 diagnosis, 235–236
 domestic violence during, 243
 ectopic. *See* Ectopic pregnancy
 endocrine functions in, 232–233
 estimated date of confinement, 236
 exercise during, 241
 false-positive syphilis test in, 68
 fertilization and implantation, 225
 fetal monitoring in, 246–250
 folic acid in. *See* Folic acid
 gestational age classifications, 236–237
 hematologic changes in, 228–229, 232
 human chorionic gonadotropin in, 232
 infections during
 cytomegalovirus, 361–363
 gonorrhea, 376
 group B *Streptococcus*, 374–375
 hepatitis, 370–374
 herpes simplex virus, 66, 363–364
 Listeria, 375–376
 parvovirus B19, 367–368
 rubella, 360–361
 syphilis, 364–365
 toxoplasmosis, 358–360
 urinary tract infections, 168–169, 230, 343–344,
 344*t*, 377–378
 varicella-zoster virus, 365–367
 vulvovaginal candidiasis, 83, 84
 laboratory values, 3–5*t*
 loss
 first trimester, 286–289
 recurrent, 289–290
 second trimester, 286
 medical conditions in
 acute abdominal pain, 355–356
 acute fatty liver, 339–340
 anemia, 349–351
 appendicitis, 355–356
 aspiration pneumonitis, 351–352
 asthma, 351
 cardiovascular disease, 352–355, 352*t*, 353*t*,
 354–355*t*
 diabetes mellitus. *See* Diabetes mellitus;
 Gestational diabetes
 epilepsy, 344–346, 345*t*
 hyperemesis gravidarum, 231, 334*t*,
 342–343
 hyperthyroidism. *See* Hyperthyroidism
 hypothyroidism, 336–338
 intrahepatic cholestasis, 340–342
 multiple sclerosis, 319
 Sheehan syndrome, 279, 322, 339
 sickle-cell disease, 350–351, 430
 thromboembolism, 232, 346–348, 421
 thrombophilias, 348–349
 multi-fetal. *See* Multi-fetal gestation
 nutrition during, 241
 post-term, 400–401
 renal/urinary function in, 230
 Rh isoimmunization in, 244
 round ligament pain in, 317
 seat belt use in, 241
 signs and symptoms, 235
 size-date discrepancy in, 237
T. vaginalis infection in, 84
 trauma in, 320, 356–357
 urinalysis in, 3–4*t*
 uterine changes in, 234
 vaginal changes in, 234
 weight gain in, 240
- Premature ovarian failure, 205
- Premature rupture of membranes (PROM), 251,
 311–312
- Premenstrual syndrome, 195–196

- Prenatal care. *See also* Pregnancy
 cardinal questions, 242
 frequency of visits, 241
 goals, 235
 laboratory tests, 243
 measurements, 242
 patient education, 242
 patient history, 240
 pelvic shape evaluation, 242
 records, 238–240
 screening tests, 243–246, 429
- Prepidil, for cervical ripening, 266
- Presacral neurectomy, 93
- Preterm delivery, 315
- Preterm labor, 242–243, 314–317, 391
- Preterm premature rupture of membranes (PPROM), 251, 312–314
- Primary syphilis, 67
- Primidone, side effects, 345*t*
- Primipara, 252
- Primordial follicles, 38
- Probenecid, for pelvic inflammatory disease, 77
- Progesterone
 for dysfunctional uterine bleeding, 400
 in ectopic pregnancy, 295
 endometrial cancer and, 149
 gastrointestinal effects, 231
 leiomyomas and, 107
 in ovulatory function assessment, 206
 in perimenopause, 214
 in pregnancy, 229, 231, 424
 for preterm labor, 316
 secretion in menstrual cycle, 38*f*, 39–41
 vaginal blood flow and, 113
- Progesterone challenge test, 203
- Progestin(s)
 for chronic pelvic pain, 93
 for dysfunctional uterine bleeding, 200
 for endometrial cancer, 152
 for endometriosis, 101
 for leiomyomas, 108
- Progestin challenge test, 48
- Prolactin
 in amenorrhea, 203
 breastfeeding and, 277
- Prolactinoma, 204
- Proliferative phase, menstrual cycle, 40
- Prolonged premature rupture of membranes, 251
- Propranolol, for thyroid storm, 336
- Propylthiouracil (PTU)
 for hyperthyroidism, 335
 for thyroid storm, 336
- Prostaglandin(s)
 for cervical ripening, 266
 menstrual cramps and, 41
- Prostaglandin F₂, 351
- Prostaglandin F_{2α}, 196
- Prostaglandin synthetase inhibitors,
 for leiomyomas, 108
- Proteinuria
 in eclampsia, 306
 in preeclampsia, 303–305
- Proteus mirabilis*, cystitis and, 166
- Pruritus, in intrahepatic cholestasis of pregnancy,
 341
- Psammoma bodies, 156
- Psoriasis, vulvar, 118
- Psychosis, postpartum, 281
- Pubarche
 characteristics, 189
 premature, 192
- Puberty, 189–190
- Pubic hair
 absence, 194
 Tanner stages, 190
- Pubovesical sling, for stress incontinence, 183
- Pudendal block, 253
- Pudendal nerve injury, 274
- Puerperal fever, 276, 325
- Puerperium, 273
- Pulmonary embolism, in pregnancy, 232, 347–348
- Pulmonary function tests, in pregnancy, 229
- Pyelonephritis
 diagnosis, 170
 differential diagnosis, 165, 170
 pathophysiology, 170
 in pregnancy, 168, 230, 343, 377
 risk factors, 170
 symptoms, 169
 treatment, 170
- Pyodermas, 120–121
- Pyrazinamide, for pelvic tuberculosis, 81
- Pyrimethamine, for toxoplasmosis, 360
- Pyuria, in pyelonephritis, 169
- Q**
- Q-tip test, 183
- Quadruple screen, 244, 245
- Quickening, 246
- Quinolones
 for cystitis, 166–167
 for pyelonephritis, 170
- R**
- Radiation therapy
 for cervical squamous cell carcinoma,
 132–134
 side effects, 134
 for vaginal squamous cell carcinoma, 147
 for vulvar squamous cell carcinoma, 139
- Radical hysterectomy, 16*t*. *See also* Hysterectomy
- Radical trachelectomy, for cervical
 squamous cell carcinoma, 132
- Radioiodine, contraindication in pregnancy, 335
- Rape, 411–413, 413*t*, 419
- Rape Trauma Syndrome, 412
- Rapid plasma reagin (RPR), 68, 365
- Rash, in toxic shock syndrome, 87
- Rebound tenderness, in tubo-ovarian
 abscess, 79
- Rectocele, 171, 173*f*, 177, 457
- Rectum
 anatomy, 29–30
 injury to, 30
- Recurrent pregnancy loss, 289–290
- Red degeneration, leiomyoma, 107
- Renal dysfunction
 in preeclampsia, 305
 in pregnancy, 421
- Renal function tests
 in cervical cancer, 127
 in pregnancy, 230
- Respiratory distress syndrome, fetal, 461
- Respiratory system, in pregnancy, 229
- Retropubic bladder neck suspension, 187
- Retroversion, 27

- Rh alloimmunization
 laboratory tests, 379–380
 management of sensitized Rh(–) patients, 380–383
 management of unsensitized Rh(–) patients, 381
 mechanisms, 379
 pathophysiology, 378, 378f
- Rh blood group system, 379
- Rh(D)-immune globulin, 274
 after abdominal trauma in pregnancy, 356–357
 after abortion, 60
 guidelines for administration, 244, 380, 382, 461
 mechanism of action, 380
- Rh isoimmunization, fetal effects, 244, 383
- Rhabdomyosarcoma
 vaginal, 148
 vulvar, 143
- Rheumatic heart disease, 68, 354f
- RhoGAM. *See* Rh(D)-immune globulin
- Rhombencephalon, 449
- Rhythm method, 42
- Rifampicin, or pelvic tuberculosis, 81
- Round ligaments, 30, 317
- Rubella, 360–361, 442–443
- Rupture of membranes (ROM), 251
- S**
- S/D flow (systolic vs. end diastolic flow velocity), 249
- Sacral colpopexy, abdominal, 177
- Sacral nerve modulation, for urge incontinence, 186
- Sacral promontory, 23
- Sacrospinous ligament fixation, 177
- Salpingectomy, 293
- Salpingitis, 96f
- Salpingostomy, 293
- Sarcoma
 uterine, 153–154
 vaginal, 148
 vulvar, 143–144
- Sarcoma botryoides, 148
- Sarcoptes scabiei*, 63
- Scabies, 63
- Schiller test, 21f
- Seat belts, in pregnancy, 241
- Sebaceous cyst, 119
- Secondary syphilis, 67
- Secretory phase, menstrual cycle, 40
- Seizures, in pregnancy
 in eclampsia, 306–307
 frequency, 344. *See also* Epilepsy, in pregnancy
- Selective estrogen receptor modulators (SERMs), for osteoporosis, 212
- Selective serotonin reuptake inhibitors (SSRIs), sexual dysfunction and, 114
- Semen analysis, 205
- Sentinel lymph node mapping, in vulvar melanoma, 142
- Septic abortion, 60, 287
- Septic pelvic thrombophlebitis, 348
- Septic shock, tubo-ovarian abscess and, 79
- Septum
 uterine, 191–192
 vaginal, 191
- Serologic tests, 64, 70
- Sertoli-Leydig cell tumor, 160, 161, 218
- Sex chromosome abnormalities, 285
- Sex cord-stromal tumors, 160–162
- Sexual abuse, 90, 411, 462
- Sexual arousal disorders, 115
- Sexual assault, 411–413, 413f, 419
- Sexual aversion disorder, 114
- Sexual dysfunction
 aging and, 113–114
 in battered women, 410
 dyspareunia, 115
 etiology, 112
 evaluation, 113
 hypoactive sexual disorder, 114
 medications causing, 114, 427
 orgasmic dysfunction, 115
 prevalence, 112, 427
 sexual arousal disorders, 115
 sexual aversion disorder, 114
 treatment, 113
 types, 112–113, 427
 vaginismus, 115
- Sexual pain disorders, 115
- Sexual precocity, 193. *See also* Precocious puberty
- Sexual response, 111–112
- Sexually transmitted infections (STIs)
 barrier contraceptives and prevention of, 52
 chancroid, 69
 condyloma acuminata, 61–62
 genital herpes, 64–66
 granuloma inguinale, 71
 lymphogranuloma venereum, 69–70
 molluscum contagiosum, 62–63
 mucopurulent cervicitis, 71–73
 pediculosis pubis, 63
 prophylaxis after sexual assault, 413f, 419
 scabies, 63
 screening guidelines, 73
 syphilis, 67–69
- Sheehan syndrome, 279, 322, 339
- Shoulder dystocia, fetal macrosomia and, 387
- Sickle-cell disease, 350–351, 430
- SIGECAPS, 280–281
- Signet ring cells, 162
- Sildenafil, for sexual dysfunction, 113
- Silver nitrate eye drops, prophylactic use in neonates, 376, 377
- Silver stain, 89
- Skene duct cyst, 120
- Skene glands, 33
- Skin
 café-au-lait lesions, 193
 changes in pregnancy, 233–234
- Sleep disturbances, in perimenopause, 214
- Small bowel obstruction, in endometriosis, 97
- Small for gestational age. *See* Fetus, growth restriction
- Smoking, teratogenic effects, 405f
- Social history, 240
- Sodium iodine, for thyroid storm, 336
- Solifenacin, for urinary incontinence, 186
- Somatic pain, 91
- Sonohysterogram, in dysfunctional uterine bleeding, 200
- Spermicide, 42, 54
- Spina bifida, sonographic findings, 319
- Spinal block, in labor, 253–254
- Spiramycin, for toxoplasmosis, 359
- Spironolactone
 as anti-androgen, 220
 for polycystic ovarian syndrome, 221

- Spontaneous abortion, 286–289
- Squamocolumnar junction, cervix, 123
- Squamous cell carcinoma
 cervical. *See also* Cervical cancer
 epidemiology, 132
 incidence, 126
 posttreatment surveillance, 134
 prognostic indicators, 132
 recurrence, 134
 survival rates, 134
 treatment, 132–134, 428
- vaginal, 145, 146–147
- vulvar
 incidence, 138
 metastases, 139
 node dissection in, 140
 posttreatment surveillance, 141
 recurrence, 141
 signs and symptoms, 138
 stages, 139, 140*t*
 subtypes, 139
 survival rates, 141
 treatment, 139–141, 451
- Squamous cell hyperplasia, vulvar, 118
- Staphylococcus aureus*
 in pyodermas, 120–121
 in toxic shock syndrome, 87
- Staphylococcus saprophyticus*
 in cystitis, 166
 in pyelonephritis, 170
- Stations, 259
- Statutory rape, 411
- Sterilization
 failure rate, 42
 female, 43–44
 male, 44
 methods, 43
- Stillbirth, 319–320
- “Strawberry cervix,” 84, 451
- Streptococcus agalactiae*, 374. *See also* Group B
Streptococcus
- Streptococcus* group B. *See* Group B streptococcus
- Streptomycin, teratogenic effects, 405*t*
- Stress incontinence, 179–180, 182–183, 186–187.
See also Urinary incontinence
- Striae distensae, 233
- Striae gravidarum, 233
- Stroke, oral contraceptives and, 46
- Stroke volume, in pregnancy, 228
- Struma ovarii, 159
- Subtotal (supracervical) hysterectomy, 16*t*. *See also*
 Hysterectomy
- Suburethral sling procedures, 187
- Succenturiate placenta, 309
- Suction and curettage (S&C), 21*t*
- Sulfadiazine, for toxoplasmosis, 360
- Sulfonamides, 445
- SurePath, 128–129
- Surfactin, 313
- Sympathetic nervous system, bladder function
 and, 178
- Syphilis
 congenital, 364–365
 diagnosis, 67–68
 Jarisch-Herxheimer phenomenon,
 68, 365
 in pregnancy, 244, 430
 stages, 67
 transmission, 67
 treatment, 68
- Systemic lupus erythematosus,
 false-positive syphilis test in, 68
- T**
- Tachycardia
 fetal, 247, 396–397
 in thyroid storm, 336
- Tachysystole, uterine, 267
- Tampons, toxic shock syndrome and, 87
- Tanner stages, pubertal development, 189–190
- Telangiectasias, in pregnancy, 234
- Telogen effluvium, 279
- Teratogen(s), 285, 401–403, 402*f*, 403–405*t*
- Teratoma, ovarian, 158, 159
- Terbutaline
 for asthma in pregnancy, 351
 for tocolysis, 269
- Tertiary syphilis, 67
- Testosterone
 in menopause, 214
 teratogenic effects, 403*t*
 vaginal blood flow and, 113
- Tetanus vaccine, 448
- Tetracyclines
 for syphilis, 68
 teratogenic effects, 404*t*
- Thalassemia, 351
- Thalidomide, teratogenic effects, 405*t*
- Thayer-Martin agar culture, 72
- Theca cells, 39
- Theca lutein cysts, 109–110, 219
- Thecoma, ovarian, 160, 161
- Thelarche
 characteristics, 189
 premature, 192
- Theophylline, for asthma in pregnancy, 351
- Thiazide diuretics, for osteoporosis, 213
- ThinPrep, 128–129
- Thromboembolic disease
 oral contraceptives and, 46, 47
 in pregnancy, 232, 346–348, 421, 425
 in puerperium, 274
- Thrombophilias, 348–349
- Thrombophlebitis
 septic pelvic, 348
 superficial, 346–347
- Thyroid hormones. *See also* Hyperthyroidism;
 Hypothyroidism
 after delivery, 280
 postpartum dysfunction, 280, 338–339, 338*t*
 in pregnancy, 233, 333, 333*t*, 425
- Thyroid-stimulating hormone, in amenorrhea, 202
- Thyroid storm, 336, 426
- Thyroidectomy, for hyperthyroidism in pregnancy,
 335
- Thyroiditis, postpartum, 280, 338–339, 338*t*
- Thyrotoxicosis
 fetal and neonatal, 336
 postpartum, 338, 338*t*
- Tibolone
 for osteoporosis, 213
 for sexual dysfunction, 113
- Tidal volume, in pregnancy, 229
- Tobacco, teratogenic effects, 405*t*
- Tocolysis, 269, 316, 391
- Tolterodine, for urinary incontinence, 186

- Tonic contraction, 267
 TORCH infections, 357–358. *See also specific infections*
 Total abdominal hysterectomy/bilateral salpingo-oophorectomy (TAH/BSO)
 for endometrial cancer, 151
 for fallopian tube cancer, 164
 for ovarian cancer, 158
 for ovarian sex cord-stromal tumors, 162
 for uterine sarcoma, 153
 Total hysterectomy, 16t. *See also Hysterectomy*
 Total lung capacity, in pregnancy, 229
 Toxic shock syndrome, 54, 87–88
 Toxoplasmosis, 358–360
 Transdermal patch, contraceptive, 50
 Transformation zone, cervix, 123, 124f
 Transvaginal ultrasound
 in dysfunctional uterine bleeding, 200
 fetal cardiac activity detection on, 238
 for ovarian cancer screening, 155
 Transverse lie, 395
 Trauma, in pregnancy, 320, 356–357
Treponema pallidum, 67
Trichomonas vaginalis, 72–73, 84, 413t, 437
 Trimethadione, teratogenic effects, 403t
 Trimethoprim-sulfamethoxazole
 for cystitis, 166
 for granuloma inguinale, 71
 for *Listeria* infection, 376
 for *P. carinii* infection, 89
 for pyelonephritis, 170
 for urinary tract infections in pregnancy, 168, 343, 344t
 Triple screen, 244, 245
 Trisomy 13 (Patau syndrome), 284–285, 384
 Trisomy 18 (Edwards syndrome), 245, 284, 384
 Trisomy 21 (Down syndrome), 245, 284, 384
 Trophoblastic hyperthyroidism, 334
 Trospium, for urinary incontinence, 186
 True conjugate, 24, 259–260
 Tubal abortion, 292
 Tubal infertility, 207
 Tubal ligation/blockage, 11t, 43–44
 Tuberculosis, pelvic, 80–81
 Tubo-ovarian abscess, 75, 78–80, 94
 Tubo-ovarian complex, 79
 Turner syndrome
 amenorrhea in, 202
 characteristics, 285
 primary ovarian failure in, 214
 Twin-twin transfusion syndrome, 390–391
 Twins. *See Multi-fetal gestation*
 Tzanck preparation, 64, 65, 72
- U**
 Ultrasound imaging, 11t
 fetal anemia, 368
 fetal anomalies, 319
 fetal cardiac activity detection on, 238
 in hirsutism, 219
 hydatiform mole, 298
 for ovarian cancer evaluation, 157
 for ovarian cancer screening, 155
 of placental site trophoblastic tumor, 301
 for pregnancy dating, 237
 for prenatal screening, 244
 in tubo-ovarian abscess, 79
 Umbilical artery, 226, 398
 Umbilical cord
 compression, 398
 traction on, 269
 velamentous, 307
 Umbilical vein, 226
 Unicornuate uterus, 192
 Ureter(s), 29
 Urethra
 anatomy, 33
 intrinsic sphincter deficiency, 183
 normal function, 178
 Urethral pressure profile, 182
 Urethritis, 165, 169, 438
 Urge incontinence, 179–180, 183–184, 186. *See also Urinary incontinence*
 Urgency, urinary, 179
 Urinalysis
 in cystitis, 166
 in pregnant vs. nonpregnant woman, 3–4f
 in urinary incontinence evaluation, 181
 Urinary catheter, for urinary incontinence, 187
 Urinary incontinence
 aging and, 180
 differential diagnosis, 179
 etiology, 179
 evaluation, 180–182
 incidence, 179
 medications causing, 181
 mixed, 184
 risk factors, 180
 stress, 182–183
 symptoms, 179–180
 treatment, 185–188
 urge, 183–184
 Urinary tract
 innervation, 178–179
 in pregnancy, 230
 Urinary tract infections
 asymptomatic bacteriuria, 168
 complicated, 166, 167
 cystitis. *See Cystitis*
 in pregnancy, 168–169, 230, 343–344, 344t, 377–378
 pyelonephritis. *See Pyelonephritis*
 urethritis, 169
 Urodynamic testing, 181–182
 Urogenital diaphragm, 32, 32f
 Ursodeoxycholic acid, 341
 Uterine artery, 27–28
 Uterine artery embolization, 108
 Uterine atony, 322
 Uterine bleeding, abnormal. *See Abnormal uterine/vaginal bleeding*
 Uterine cancer
 endometrial. *See Endometrial cancer*
 sarcoma, 153–154
 Uterine hyperstimulation, 267
 Uterine hypocontractility, 392
 Uterine inversion, 324–325, 435
 Uterine involution, 273
 Uterine perforation, in elective abortion, 58
 Uterine prolapse, 171
 Uterine rupture, 272, 324
 Uterine tachysystole, 267
 Uterine window, 273
 Utero-tonics, 273

- Uterosacral ligament
 anatomy, 30
 suspension, for enterocele, 177
- Uterus
 anatomy, 26f, 27–28
 congenital anomalies, 191–192
 layers, 225
 in pregnancy, 234
 in puberty, 190
- V**
- Vaccine(s)
 hepatitis B, 373
 in HIV infection, 89
 human papillomavirus,
 62, 131
 in pregnancy, 242, 351, 420
 rubella, 361
 tetanus, 448
- Vacuum-assisted delivery, 271
- Vacuum extraction, 11f
- Vagina
 agenesis, 191
 anatomy, 26–27, 26f
 benign cysts, 118–120
 non-sexually transmitted infections, 120–121. *See also* Sexually transmitted infections
 normal flora, 81
 normal secretions, 81
 in pregnancy, 234
 septum, 191
- Vaginal birth after cesarean, 274
- Vaginal bleeding, abnormal. *See* Abnormal uterine/vaginal bleeding
- Vaginal cancer
 adenocarcinoma, 148
 human papillomavirus and, 125
 melanoma, 147–148
 preinvasive, 144–145
 sarcoma, 148
 squamous cell carcinoma, 146–147
 stages, 146f
 types, 145
 verrucous carcinoma, 147
- Vaginal colporrhaphy, 177
- Vaginal discharge. *See also* Vaginitis
 in cervical cancer, 126
 in uterine sarcoma, 153
 in vaginal cancer, 146
- Vaginal examination. *See* Pelvic examination
- Vaginal fornices, 27
- Vaginal hysterectomy, 16t, 18t. *See also* Hysterectomy
- Vaginal intraepithelial neoplasia, 125, 144–145
- Vaginal lacerations, 269–270
- Vaginal ring, contraceptive, 50–51
- Vaginismus, 115
- Vaginitis
 atrophic, 85
 candidiasis, 83–84
 desquamative inflammatory, 84–85
 differential diagnosis, 82, 165
 laboratory tests, 82
 noninfectious, 86
 nonspecific, 82–83
 symptoms, 82
T. vaginalis, 72–73, 84, 413t, 437
- Vaginoplasty, for vaginal agenesis, 191
- Valproate
 fetal side effects, 345t
 maternal side effects, 345t
 teratogenic effects, 403t
- Vancomycin
 for group B *Streptococcus*, 375
 for *Listeria* infection, 376
 for toxic shock syndrome, 88
- Variable decelerations, 249
- Varicella zoster immune globulin, 367
- Varicella zoster virus, 365–367
- Vasectomy, 44
- Vault prolapse, 171
- Venereal Disease Research Laboratory (VDRL), 68, 365
- Venous thrombosis. *See* Thromboembolic disease
- Verrucous carcinoma
 vaginal, 147
 vulvar, 141
- Vestibule, 33
- Vincristine, for choriocarcinoma, 300
- “Violin-string” adhesions, in Fitz-Hugh-Curtis syndrome, 78
- Viral latency, 64–65
- Virchow triad, 346
- Virilization
 definition, 217–218
 vs. hirsutism, 218
 in ovarian sex cord-stromal tumors, 161
- Visceral pain, 91
- Vitamin A, teratogenic effects, 404t
- Vitamin B₁, in hyperemesis gravidarum, 342–343
- Vitamin B₆, for hyperemesis gravidarum, 342
- Vitamin K deficiency
 anticonvulsants and, 346
 in hyperemesis gravidarum, 343
- Voiding cystometry, 182
- Vomiting. *See* Nausea/vomiting
- Vulva
 anatomy, 33
 benign cysts, 118–120
 dystrophies, 116–118
 hematoma, 122
 non-sexually transmitted infections, 120–121
 traumatic injury, 121–122
- Vulvar cancer
 Bartholin gland carcinoma, 138, 142–143
 basal cell carcinoma, 143
 diagnosis and evaluation, 137–138
 differential diagnosis, 138
 human papillomavirus and, 125
 incidence, 137
 melanoma, 142
 Paget disease, 144, 431
 preinvasive, 136–137
 risk factors, 137
 sarcoma, 143–144
 squamous cell carcinoma. *See* Squamous cell carcinoma, vulvar
 types, 138
 verrucous carcinoma, 141
- Vulvar intraepithelial neoplasia, 125, 136–137, 431
- Vulvovaginal candidiasis, 83–84
- Vulvovaginal-gingival syndrome, 117
- W**
- Warfarin, teratogenic effects, 347, 403–404t
- Weight loss, following delivery, 279

- Western blot, 89, 277
- Wet mount
 for *T. vaginalis*, 72
 in vulvovaginal candidiasis, 83
- Withdrawal method, for contraception, 42–43
- Wolffian cysts, 104
- Wound dehiscence, 275
- Wrongful birth, 415
- Wrongful conception, 415
- Y**
- Yolk sac tumor, 158
- Yuzpe method, emergency contraception,
 51–52, 413f
- Z**
- Zero station, 433
- Zygote intra-fallopian tube transfer
 (ZIFT), 209