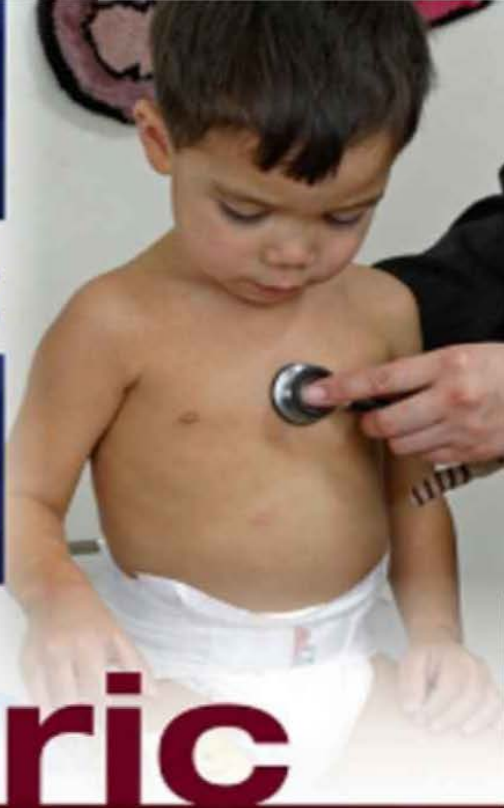
Karen G. Duderstadt



Pediatric Physical Examination

An Illustrated Handbook

EDITION 3

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An Illustrated Handbook

EDITION THREE

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Dedication

To all the students who have shaped my work as a teacher and a mentor; To the children and families whom I have had the privilege of caring for and who have enriched my life; and To Chris, my constantly patient husband, who has supported my life's work.

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Preface

Pediatric Physical Examination: An Illustrated Handbook, third edition, is written for students, educators, and pediatric health care providers dedicated to mastering the art and technique of the comprehensive physical examination of infants, children, and adolescents. Facing increasing pressures, primary health care providers need astute assessment skills combined with quick references to assist them in caring for children and families. This third edition of *Pediatric Physical Examination: An Illustrated Handbook* will provide the novice or experienced provider with pediatric content from experts in the field, useful examination techniques from birth to adolescence, and pediatric clinical pearls not covered in other texts on health assessment across the life span. In response to suggestions by students and experienced clinicians who have used the text as a resource across pediatric clinical settings, the third edition includes a *red flag* feature in many of the chapters to alert readers to key findings across systems. Also included in some chapters of the third edition is a note on *interprofessional collaboration* to highlight important collaborations with pediatric specialists, physicians, behavioral health, nutritionist, and therapists to provide comprehensive care to children and families. There are also many new photos and illustrations of important assessment findings.

The initial chapters of this handbook begin with an overview of the developmental approach to information gathering and assessment of children from birth to adolescence. Chapter 3 focuses on development surveillance and presents evidence on reliable and valid developmental and behavioral screening tools. Chapter 4 presents comprehensive information on history taking in infants,

children, and adolescents, including expanded coverage of pediatric mental health assessment. Chapter 5 reviews the unique vulnerability of children to environmental hazards and focuses on the importance of the environmental health history in identifying and reducing environmental risk factors that have an impact on child health.

The most exciting addition to the text is Chapter 6, *Newborn Assessment*. This chapter features a head-to-toe assessment of the term newborn and reviews pertinent health history and common newborn conditions. Chapter 7 presents assessment of the skin and common pediatric findings. The chapters that follow are organized in the pediatric-oriented "quiet-to-active" approach to physical examination. Pediatric experts consider this to be the most effective approach to assessing young children. This format begins with the quieter parts of the exam—cardiac and respiratory—which require astute listening skills and less active participation of the young child. Then, Chapters 10 through 14, which cover assessment of the head and neck, eyes, ears, nose, and throat, require more active participation from the child and are better performed after the quiet parts of the physical examination. Chapter 15 reviews abdominal assessment from birth to adolescence.

A unique feature of the text remains the developmental approach to examination of the male and female genitalia and the developing breast in males and females, presented in Chapters 16 through 18. Chapter 19 includes a comprehensive assessment of the child and adolescent athlete for sports participation, as well as the recent guidelines on assessment for concussion. Finally, Chapter 20 presents neurological assessment from a pediatric developmental perspective.

Working with children and families is a hopeful endeavor, and health in childhood builds the foundation for health promotion and health protection throughout life. Pediatric primary care providers have a key role in protecting and improving the health of the next generation. Through astute physical assessment, providers build trust with children and families and preserve the provider/patient relationship, ultimately improving health outcomes and decreasing health care costs. This handbook assists the pediatric health care provider with this most important undertaking: promoting the

health of the next generation.

Acknowledgments

The publication of the third edition would not have occurred without encouragement from my students, pediatric colleagues, family members, and the “very patient” editors at Elsevier. I would like to first acknowledge Melissa Rawe, Content Developmental Specialist, who has patiently and pleasantly mentored me through the publication of the third edition. Her attention to detail and willingness to accept my numerous edits has made this edition a wonderful resource for students and educators. Lee Henderson, Executive Content Strategist, has loyally carried my vision through to publication for the text and eBook! Marquita Parker was the Senior Project Manager for the text during the final stages of publication.

I am pleased to have some new contributors in the third edition—Annette Carley and Julianne Kristyne. I am very thankful to them and my UCSF faculty colleagues for bringing new pediatric and adolescent specialty expertise to this edition of the text. Their contributions were invaluable at a very busy time during the academic year. Pat Jackson Allen, my longtime mentor and friend, was willing to again support the text as a distinguished contributor. Naomi Schapiro was willing to return as a key contributor during a very busy time for her research work and grant duties. All of these pediatric colleagues were instrumental to the third edition with their excellent contributions. Tina Chang PNP and Julia Chang PNP were my devoted research assistants on the third edition and contributed to the searches yielding many of the excellent references and resources used in updating the text.

Finally, I would like to acknowledge my dear husband and life companion, Chris. Without his constant patience, love, and support,

this third edition would not have happened. My hope is that the knowledge in this text will help to shape the next generation of pediatric health care providers who advocate for and promote optimum health for children and families

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CHAPTER 1

Approach to care and assessment of children and adolescents

Karen G. Duderstadt

Unique role of the pediatric provider

Pediatric health care providers have a unique role in the development of a child's health over a lifetime. Health is an interactive, dynamic process shaped by genetics, exposures, human experiences, and individual choice.[1] From a life course perspective, early experiences can "program" a child's health and development. Protective factors, such as a nurturing family and safe neighborhood, improve health and contribute to healthy development, whereas risk factors, such as limited access to quality health care and social services, have a negative effect on a child's development and ability to reach his or her full developmental potential.[1]

A significant body of literature indicates that many chronic conditions in adulthood and disparities in adult health have their origins during childhood and increase over time.[2] Physical,

cognitive, and social-emotional health are established in the early years of life. Pediatric health care providers are now charged with identifying developmental concerns early and focusing on interventions that promote the health and well-being of children and families and limit the adverse effects of toxic stress on brain development.[2] Toxic stress, in contrast to tolerable stressors during childhood, is defined as prolonged activation of the physiologic stress response system that is not buffered by environmental protections or by stable, responsive parent and caregiver relationships.[2] Early investments in health promotion can empower healthy choices for both child and family, greatly improve child health outcomes, alter the life course, and decrease the cost of health care services.

Establishing a caring relationship

Children have unique needs because of their long period of dependency and development, which presents a unique challenge to the pediatric health care provider. Children's health and well-being depend greatly on the care received from their family units and the environment in which they live. Addressing the needs of the parent or caregiver while caring for the child and fostering a healthy relationship in the family unit are among the most important and challenging tasks in pediatrics.

Care is best provided in a *patient-centered medical or health care home,* which promotes holistic care of the child and family by partnering with a primary pediatric health care provider. The patient-centered health care home allows the provider to create a trusting partnership, to limit the effects of psychosocial adversity in childhood, and to improve the quality of care through empowering and supporting the family unit.[3] Children, adolescents, and families benefit from motivational and anticipatory guidance in health promotion, and care provided over time by the same provider is associated with higher odds of receiving multiple preventive services, particularly for adolescents.[4] The caring relationship established by the primary care provider reinforces positive parenting and provides behavioral consultation to the child, adolescent, and family at critical periods of development.

A caring relationship includes a patient-provider relationship without bias in regard to racial, ethnic, or socioeconomic difference. The attitudes and behaviors of health care providers has been identified among many factors contributing to health disparities.[5] Implicit attitudes are those thoughts and feelings that exist outside of conscious awareness and are difficult to acknowledge and control.[5] Implicit biases can influence behavior and clinical decision making without the provider's awareness.[5, 6] The perception of bias in clinical encounters undermines the caring relationship. Health care providers must make conscious efforts to overcome bias in their actions. Educating providers on racial, cultural, and socioeconomic biases, self-regulating behavior, and helping them to develop new mental habits can ensure the highest quality of care for all children and families.[7]

Parent and child interaction

One of the most important aspects of the health interview is observing and eliciting interactions between the parent and child or adolescent. Analyzing verbal responses and interactions during the encounter gives the health care provider an idea of how the parental relationship fosters child development and child self-esteem. It also gives the health care provider a window into the child's world. Interactions between the parent or caregiver, the family members, and the health care provider reveal family dynamics, family connectedness, family authority, and the approach to problem solving.

Nonverbal cues provide the most revealing picture of the child's demeanor and of the parent-child relationship and give the health care provider additional understanding of the context in which the child lives. Stop and observe these cues, and verbalize your concerns to the parent and child: "You look sad today. Can you tell me what that is about?" Ask yourself the following: Is the parent or caregiver disengaged with the child or infant during the encounter? Does the parent appear depressed or angry? Does the data from the health interview fit with the demeanor of the child during the encounter?

A child who is withdrawn, refuses to make eye contact, or is

consistently stressed when communicating with a parent is exhibiting signs of strain in her or his environment. This should alert the health care provider to communicate her or his concern to the family and provide support, counseling, and referral when indicated. Children often mirror the emotions of the adults around them. Families involved in conflict, who have experienced violence, or who are under stress often cannot see their own interactions clearly or the effect that their interactions have on the child. Pediatric health care providers should assess parenting behaviors by asking open-ended questions, such as "How do you handle negative child behaviors in the home?" or "How do you negotiate limiting screen time?"[8] By fostering and supporting effective parenting in early childhood and providing problem-based counseling, the pediatric health care provider offers a service that is critical to the healthy psychosocial development of the child.[8]

Children and the media

A large part of the social and emotional development of children and adolescents is now occurring while they are using mobile devices. Children's virtual lives constitute a larger part of their social interactions daily. It is important for parents and caregivers to monitor children's social media, gaming, web, and cell phone activities for their potential impact on the child's emotional health. Population-based studies have shown associations between excessive screen time and cognitive, language, and social-emotional developmental delay.[9] Infants with more handheld screen time are at increased risk for expressive speech delay.[10] Excessive screen time in middle childhood and adolescence may involve bullying or peer-to-peer communications that pose potential risks to their privacy and safety.

Heavy parental use of mobile devices is associated with fewer verbal and nonverbal interactions between parents and children; therefore, reducing parental media use and enhancing parent-child interactions may be an important area of family behavioral change.[9] The quality of parenting can modify the associations between media use and the child's speech and language development. Introducing high-quality programming and viewing programs

together with children is the recommended approach, along with limiting daily screen time.[9] Temperament may also influence how much media children consume. Excessive media use is often more likely in young children with self-regulation problems, and these children will present greater challenges with parental limit setting.[9]

The American Academy of Pediatrics (AAP) has a Media and Children Communication Toolkit that provides parent with resources to help them manage screen time. Further information about the tool kit and a Family Media Use Plan in English and Spanish is available at https://www.aap.org/en-us/advocacy-and-policy/aap-health-initiatives/Pages/Media-and-Children.aspx

A child's perspective

The view of the parent or caregiver and the view of the child may differ quite remarkably, and this difference can often affect health care decisions and treatment outcomes. Promoting opportunities for children and adolescents to contribute to health care decisions creates a child-centered approach to care that empowers and enables the child or adolescent within the family unit.

A model to facilitate children's participation in the health history provides a framework for communication that is attentive, sensitive, and values the child's perspective while capturing the shared responsibility of families, health care professionals, and health care organizations.[11] The model proposes the following:

- Irrespective of age the child is listened to.
- The child is supported in expressing his or her views.
- The child's views are taken into account.
- The child is involved in the decision-making process.
- The child can share power and responsibility in decision making.

This framework provides a child-centered approach to health care while considering the needs and views of the family unit.[11]

Family, cultural, racial, and ethnic considerations

It is essential that health care professionals attend to the increasing

diversity within a globally connected society.[12] The increasing global immigration of populations will continue to pose challenges to health care professionals working with diverse populations. Providers will be required to assess the extent to which culture plays a role in health care decisions in the family and how it impacts treatment plans and adherence to these plans. An understanding of diversity must include not only knowledge of the culture but also respect and the tailoring of communications to embrace and value diversity.[12]

Cultural beliefs also affect care-seeking behavior and the delivery of clinical care by health care providers. Effective models of care incorporate sensitivity to cultural differences and enhance the protective factors of cultural practices within families. Studies demonstrate that differences within ethnic groups are as great as the cultural differences between groups; therefore no valid assumptions can be made in health care on the basis of physical appearance or surname. It is important for providers to acknowledge cultural differences, understand their own background and culture, and understand how that influences the care they deliver. They also must engage in self-assessment, acquire cultural knowledge and skills consistent with the practice setting, and value diversity within the health care team. *Cultural humility* involves a change in overall perspective around diversity and increasing awareness of power imbalances in the provider-patient relationship.[12]

Ideally, delivery of health care services should occur in the first language of the client. When this is not possible, a model framework for the encounter between the health care provider, child, and family should include the following:

- Recognizing language barriers and effectively using interpreters
- Exploring parental beliefs and their impact on the child
- Building on family strengths
- Recognizing and exploring the use of cultural beliefs and alternative therapies, how they influence child health, and the effect on clinical care
- Understanding, as the health care provider, how your personal values and beliefs influence care delivery and affect health outcomes

- Altering care practices to eliminate disparities in health care delivery related to race/ethnicity and economic status

Cultural humility is an essential component of responsible health care services for multicultural populations. Embracing and valuing cultural diversity in the health care encounter will build honesty and trust in the caring relationship. Throughout this text, attention is given to how culture and ethnicity influence the assessment process in child health.

Effective communication

Children are accepting of many different styles of interaction and will adapt to the health care provider who is at ease and competently engages the child. The skilled pediatric health care provider learns to make eye contact when a child is interested and to avoid eye contact when a child is fearful. Initially directing attention to the parent or caretaker allows the infant or young child time to adjust to the environment.

Children are comfortable when they know what to expect in an environment. Explaining clearly to the young child or adolescent exactly what you are going to assess during the physical exam will decrease anxiety and build trust. With each part of the exam, explain or "talk through" the assessment and the findings. Reassure the child or adolescent of your findings when normal and explain abnormal findings as appropriate for the child's age. Talking through every step of the encounter decreases anxiety in the child and adolescent. Even busy practitioners in time-pressured environments will find the child or adolescent to be a more willing participant when this *talk-through* format is added to the health care encounter.

Interviewing children

Engaging children in the interview process can reveal their understanding of health, allow their participation in the health care encounter, and provide insight into their social-emotional world. Eye-level encounters are the most effective with young children and make the health care provider appear more approachable.

Many young children are effective communicators and can

respond to questions about their dietary habits, daily activities, school, or child care and relationships with school friends. It is important to engage children early in gathering health information. Health education can easily follow when this interview technique is used, and the child's responses create a dialogue that establishes a supportive provider-patient relationship.

Children 6 to 11 years old can be directly interviewed and can be participants in their health care. Health care providers can be role models for parents as they engage the child in the health interview and teach aspects of health and safety education. This approach teaches children from a young age to understand and care about their health and establishes the importance of building healthy habits for life.

Adolescents should always be interviewed separately from the parent. It is important to prepare the parent for this transition to a more independent role for the adolescent during the health care encounter. Allowing time to engage the adolescent independently will provide the best opening for discussion of personal or sensitive concerns that need to be voiced and for discussion of any conflicts in the home or school environment that are affecting the adolescent's well-being.

Use of the following clear communication techniques when interviewing will build a caring, trusting relationship with children/adolescents and their families.

- **Question indirectly** to encourage children and adolescents who are reluctant to discuss feelings. Engage the young child with, "I am going to tell you a story about a 5-year-old who lost his favorite pet. How do you think he feels?" or the adolescent with, "Some 15-year-olds have tried vaping or smoke marijuana. Do you have any friends who smoke?"
- **Pose scenarios** to the child or adolescent. "What would you do if . . . ?" is appropriate for the young child, in contrast to, "How would you feel if . . . ?" which is appropriate for the older school-age child and adolescent.
- **Begin with less threatening topics** and move slowly to more sensitive topics for the child or adolescent. "Tell me how things are going at school this year," in contrast to, "Has anyone ever hurt you?" or directed to the adolescent, "Has anyone forced

you to have sex against your will?"

- **State your expectations clearly.** Say to the child, "I need you to be very quiet now so I can listen to . . . " Or to the adolescent, "To take care of you, I need you to tell me . . . "
- **Do not offer a choice** to the child or adolescent when in reality there is no choice.
- **Use "I"** when speaking to the child or adolescent. "I need to ask you this question because I want to help you . . . " in contrast to, "You must tell me what is going on." Avoid using the word *you*, which creates a defensive atmosphere when interviewing children or adolescents. This will provide positive role modeling for parents and also build the caring relationship between the health care provider, the child, and the parent.
- **Ask the young child to draw a picture or draw his or her family.** This captures the child's attention and establishes your interest in the child's abilities. Children often reveal feelings or communicate important family issues through their art.[13]

The importance of physical examination

As health care providers increasingly rely on imaging and technology, the practice of diagnosing a health condition or disease from a physical examination has taken a back seat. The physical exam may be a ritual, but it is an important diagnostic tool when applied by skilled hands and can yield important diagnostic information and decrease the cost of health care.[14] The physical exam helps providers to ask better questions of the diagnostic tests they order.[15] Further, performing the physical examination provides important psychosocial benefits to the patient and can relieve stress and provide reassurance. Diagnostic evidence is important, but it is most valuable when decisions about diagnostic testing are made after completing a thorough health history and performing a thorough physical examination.

Besides the importance of focusing the diagnostic testing, what is also lost in eliminating the physical examination is the meaningful encounter between the patient and provider.[15] Recent studies have indicated that the trusting relationship between the provider and the family is the most influential factor in assisting families in

making sound decisions about the care of their child. This provider-patient relationship is an important part of the therapeutic effect when working with families. It is crucial for pediatric health care providers to master the skills of history taking and physical examination in order to decrease or prevent medical errors and to increase the efficiency in ordering diagnostic testing and treating patients and families to decrease health care costs.

"Quiet to active" approach to the physical examination

"Quiet to active" is an important mantra that should be adopted by the health care provider who will be caring for infants and young children. It refers to the approach of beginning with the parts of the physical examination that require the child to be quiet or silent in order for the health care provider to differentiate physical findings. The "quiet" parts of the physical examination in infants and young children include pulse and respiratory rate, auscultation of cardiac sounds and respiratory sounds, and auscultation and assessment of the abdomen. Respiratory and cardiac sounds are subtle, and accurate assessment requires a relatively cooperative child. Therefore, approaching these areas first during the physical examination produces the best results. Assessment of the genitalia and cranial nerves in the infant and young child can also be completed before the more invasive examination of the ears and mouth and before measuring height, weight, temperature, and blood pressure when indicated. Varying the sequence of the physical examination to fit the temperament and activity level of the child is an essential part of pediatrics, but omitting an aspect of the physical examination does not serve the health care needs of the child and risks a diagnosis made on the basis of an incomplete assessment.

Developmental approach to assessment

Preterm infants and newborns

In the clinical setting, it is important to begin the physical

assessment with the infant initially swaddled on the examining table or in the parent's arms to maintain body temperature. In this manner, auscultation of the cardiac and respiratory sounds can be accomplished before disturbing a sleeping infant or cooling the infant significantly. After the "quiet" parts of the examination are completed, transfer the infant to the examining table if in the parent's arms and begin a complete assessment with the infant wearing only a diaper. Observing the movements of the newborn for symmetry, strength, and coordination must be accomplished with the infant undressed. Assessment of overall appearance, skin color, breathing pattern, and degree of alertness or responsiveness should be noted. The health care provider should remain flexible in regard to the order of the exam throughout the encounter, because often the physical examination is performed between the eating and sleep cycles of the newborn.

Infants up to 6 months of age

Until 6 months of age, infants are most effectively assessed on the examination table. It provides a firm surface to support the infant's head during the physical exam and also provides a stable surface for the examination techniques required during a complete physical assessment. A calm, gentle approach works well and avoids possibly frightening the infant. The "quiet" parts of the exam may be accomplished with the infant in the parent's arms, but other parts are difficult to accomplish effectively in that position. Remember never to leave the examining table while you are evaluating a young infant.

Children 6 months to 2 years of age

The inspection of infants and young children necessitates using a completely different social approach than with any other age groups. In establishing a therapeutic relationship with an adult, etiquette requires immediate eye contact. With infants 7 to 9 months of age, a progressive approach to eye contact is required because of the developmental phenomenon of stranger anxiety. First, observe the infant or young child covertly while speaking with the parent or caretaker, so as to allow the child to adjust to your presence in the environment. If the young child is looking at

you and listening, then make glancing eye contact. If you are not rejected, then speak to the young child and, finally, reach out to touch the child. This approach will produce the best results in establishing the caring relationship. By offering puppets or small, washable toys to the child or using dolls to demonstrate parts of the physical examination, you will also often provide a calming effect at the beginning of the encounter.

Once the infant is able to sit stably, normally around 6 to 8 months of age, the examination can proceed with the child in the parent's lap, so as to decrease fear and stranger anxiety. Clothing should be removed gradually as the physical examination progresses from "quiet" to "active." Optimal examination of the abdomen and genitalia occurs with the child on the examining table, but in the fearful child, the exam may proceed with the child still in the parent's lap and with the examiner seated at the same level as the parent in a knee-to-knee position to create a surface for the child to lie on. With the young child's head and shoulder on the parent or caretaker's lap, the examiner proceeds with assessment of the abdomen, genitalia, and hips, thereby avoiding the need to place the child on the examination table and thus eliciting anxiety. When the infant is old enough to begin walking, it is important to observe the infant toddling in only a diaper to evaluate gait and musculoskeletal coordination.

Young children

By 3 years of age, most children, though still apprehensive, are able to make eye contact and separate briefly from the parent. Observe their ability to be comforted, evaluate their response to the environment, their level of social interaction, and their relationships with parents or caretakers and siblings if present. How appropriate is their behavior in the setting? What is the quality and variety of their verbal responses? What is their level of activity and attention span? Young children generally respond best to a slow, even, steady voice. Give the young child time to warm to the situation before undressing him or her. The confident young child should be able to be examined sitting on the examining table. The "quiet" to "active" approach is advisable with this age group also; that is, beginning with the cardiac and respiratory examination and then

proceeding head to toe. Give children 3 to 5 years of age clear directions, allow them to respond, and recognize success. Young children particularly enjoy games, drawing, and role playing the physical examination with dolls or stuffed animals (Fig. 1.1). Modesty sets in during the preschool years, and health care providers need to be respectful and mindful of this developmental stage.

FIGURE 1-1 Use of play to decrease anxiety in a young child. Source: (© StockLite, 2013. Used under license from Shutterstock.com.)

Children 6 to 11 years of age

Children 6 to 11 years old benefit most from the talk-through approach to the physical exam. They are interested in learning

about their bodies and are forming a body image of themselves. They are becoming more independent from their parents. School-age children gain the most from education about good health habits. Learning more about their bodies helps them connect their health with their health habits. Allow the child to participate in all aspects of the physical exam and respect his or her modesty. A "head-to-toe" assessment with the child on the examining table is most effective during middle childhood.

Adolescents

The approach used with the adolescent during the health care encounter should be based on the child's developmental stage rather than his or her age. This is true for all children but particularly those in adolescence. Development during early, middle, and late adolescence proceeds unevenly and can vary widely among 11- to 18-year-olds. Respect and confidentiality are essential components of developing a trusting relationship with the adolescent. Parental input is important during the health encounter, but adolescents should be interviewed and examined separately from the parent or peers. Avoid power struggles and give the adolescent control whenever possible. Involve adolescents in planning their health care and in establishing realistic goals and health habits. Chapter 4 further discusses evidence-based practice for the adolescent health visit.

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CHAPTER 2

Assessment parameters

Karen G. Duderstadt

Normal growth

Growth is the most dynamic aspect of childhood. In the early years, height and weight measurements are key indicators of health. Abnormal progression of weight gain and/or height parameters is often the first indicator to the health care provider of abnormalities in growth and maturation patterns. Growth normally proceeds in a predictable pattern during childhood, from *cephalocaudal* (head to tail) and *proximodistal* (near to far). Plotting serial measurements regularly on a standardized growth curve for age and gender is a reliable method of monitoring growth and is an essential component of comprehensive well-child care. Gender- and age-specific standardized growth charts are available at https://www.cdc.gov/growthcharts/clinical_charts.htm

Accurate assessment of growth begins in the newborn period by evaluating progress on the growth curve and continues throughout childhood and adolescence by evaluating growth in relation to age. An accurate assessment of gestational age in the newborn begins with the prenatal and birth history; it can help the provider anticipate conditions associated with preterm birth. Infants can be classified by gestational age as *term infant*, *preterm infant*, or *postterm infant*. See Chapter 6 for a review of newborn assessment

parameters. It is important to use the correct technique when gathering measurements of infants, children, and adolescents; doing this is one of the significant challenges in pediatrics. Table 2.1 presents average weight, height, and head circumference gains from infancy through middle childhood and adolescence growth patterns.

TABLE 2.1

Average Weight, Height, and Head Circumference Gains in Infancy Through Adolescence

	WEIGHT	HEIGHT	HEAD CIRCUMFERENCE	
Age	Average Weekly Gain	Average Monthly Gain	Average Monthly Gain	Comments
0–3 months	210 g (8 oz)	3.5 cm	2.0 cm	Regain or exceed birth weight by 2 weeksBirth weight doubles in 4–6 months
3–6 months	140 g (5 oz)	2.0 cm	1.0 cm	—
6–12 months	85–105 g (3–4 oz)	1.2–1.5 cm	0.5 cm	—
	Average Yearly Gain	Average Yearly Gain	Average Yearly Gain	
1–3 years	2–13 kg (4.4–6.6 lb)	12 cm	3.0 cm	Height at 2 years, approximately half of adult height
3–6 years	2 kg (4.5 lb)	3–7 cm	1.0 cm	—
6–12 years	3–3.5 kg (7 lb)	6–7 cm	2–3 cm during middle childhood	Growth occurs in spurts lasting about 8 weeks, occurring 3–6 times per year
	Average Total Gain	Average Yearly Gain		
Girls 9.7–13.5 years	17.7 kg (39 lb)	8–14 cm/year	—	95% of growth achieved by onset of menarche
Boys 11.7–15.3 years	22.2 kg (50 lb)	8–14 cm/year	—	95% of growth achieved by 15 years; weight gain follows linear growth and is delayed by several months

Adapted from data in Kleigman RM, Stanton BF, St. Geme JW, Shor FM: *Nelson Textbook of Pediatrics*, ed 20, Philadelphia, 2016, Elsevier.

Measurement

Head circumference

Measurement of *head circumference* is a routine part of growth assessment in the first 2 years of life. An accurate measurement of the head is taken with the measuring tape placed around the head at the point of greatest circumference from the occipital protuberance above the base of the skull to the midforehead or point of greatest bossing of the frontal bone (Fig. 2.1). The head

circumference measurement is then plotted on the growth curve specific for sex and age at each well-child visit to determine if the growth pattern is normal. If the initial head measurement is plotted on the growth chart and indicates a concerning pattern of growth, it is important to remeasure the head to ensure accuracy of head size. A *head circumference* that plots 1 to 2 SD above height and weight on the growth curve or >95th percentile or <5th percentile for age should be evaluated. *Microcephaly* may be indicative of a small-for-gestational (SGA) infant, intrauterine growth retardation (IUGR), or premature closure of the cranial sutures, termed *craniosynostosis*, which requires immediate referral. *Macrocephaly,* a large head in proportion to the body size, may indicate increased intracranial pressure or may be a familial variant. Consistent and accurate assessment of the head circumference is a critical part of the evaluation of normal growth and development in the first year of life.

FIGURE 2.1 Accurate measurement of head circumference.

Chest circumference

Chest circumference is smaller at birth than *head circumference. Chest circumference* is measured at the nipple line. The head circumference is normally 2 cm greater than the chest circumference in the first 6

months of life. Molding of the head in the term newborn may make it appear as though the measurements are equal. By 1 year of age, the *chest circumference* should closely equal the head circumference. With the progression of growth, the *chest circumference* becomes larger than the head circumference at about 2 years of age and continues to grow more rapidly during childhood. Chest measurements are not routinely taken unless an infant has abnormal physical findings at birth or demonstrates abnormal growth.

Height

Height is the most stable measurement of growth and maturation in childhood. Linear growth is genetically predetermined, and therefore adult height generally occurs within a predictable range if accurate family history is available. Linear growth often occurs in spurts followed by long quiescent periods in which no growth occurs. Infants and young children may demonstrate an increase in appetite before a growth spurt, followed by an increased need for sleep.

For the infant or toddler, *recumbent* height is required for accurate measurement of linear growth. Place the infant supine on a flat surface or examination table equipped with a *measuring device* or *length board* (Box 2.1). Accurate measurement requires the infant's legs to be flat against the measuring device with the foot in the level position and head held erect against the measuring device (Fig. 2.2). Term newborns vary in height between 45 to 55 cm (18 and 22 inches) at birth, and height increases by approximately 2.54 cm (1 inch) per month over the first few months of life. Although infant measurement is often taken on the examination table by marking the position of the top of the head and the bottom of the foot on table paper and then determining length with a measuring tape, this method can result in inaccurate measurements. Always compare linear measurements with previous growth trends to assure accuracy.[1]

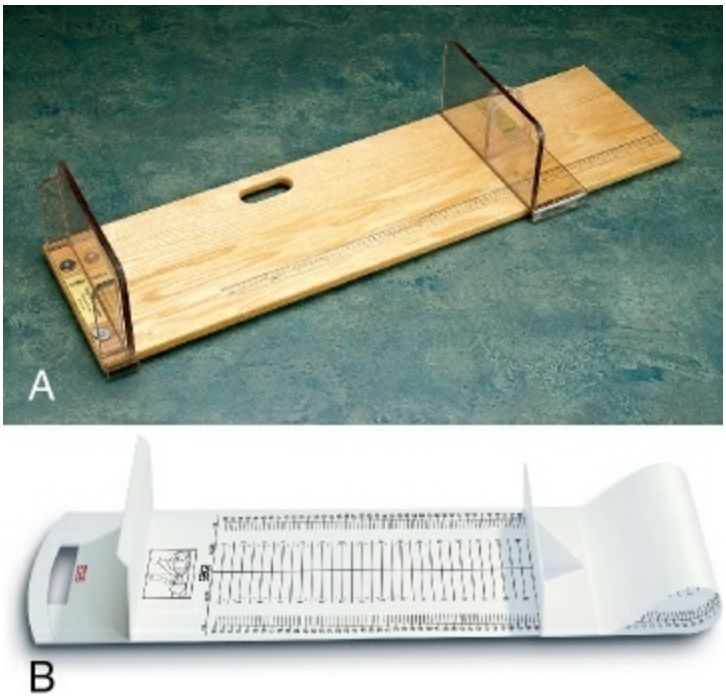

FIGURE 2.2 Length board (A) and measure mat (B). Source: (A, Courtesy Perspective Enterprises, Inc., Portage, MI. B, Courtesy Seca North America, Medical Scales and Measuring Systems, Seca Corp.)

BOX 2.1

Accurate Measurement of Height and Weight

Height

Birth–24 months

- Infant's head must be held firm on flat surface against top of measuring device.
- Push knees, with legs extended, gently toward table. Bottom of foot is placed directly against footboard of measuring device.

24– 36 months

- Transitioning to a stadiometer at 24 months of age is most accurate to assess linear growth; use of a measuring device for fearful children from 24 to 30 months can be used with child

lying on a flat surface or examination table.

36 months through school age

- Child should be standing erect with buttocks and back against stadiometer or wall.
- For accurate height measurement in the young child, maintain the head erect by placing slight upward pressure under the chin.

Weight

Birth–12 months

- Infant should be undressed and weighed consistently with clean, dry diaper or without diaper when placed on infant balance scale.
- *Safety* is of primary concern. Examiner cannot leave infant unattended at any time.

12–24 months

- Before 2 years of age, weight is measured most accurately on infant balance scale with dry diaper. *Exceptions:* when child is very large or more cooperative/stable, a standing scale may be used.

2– 6 years

- At 2 years, when child is cooperative, weight can be measured accurately on a standing balance scale.
- Weight, until 3 years of age, should be measured with child in underwear consistently when using a standing balance scale.
- From 3 to 6 years, children can be weighed in clothing without shoes.

Term infants generally increase in length by 50% in the first year. The increase is primarily in truncal growth. Doubling the height at 2 years of age can give an estimate of adult height (see Table 2.1). After 2 years of age transition to standing height is most accurate and appropriate, however, recumbent height with a measuring device is often easier to obtain in the first part of the second year if the child is fearful. The increase in height averages 3 inches (7.5 cm) over the second and third years. Measurement is recorded without

shoes to the nearest 0.1 cm or 0.25 inches. Standing height should be taken using a wall-mounted or portable *stadiometer* for accuracy. Accurate measurement requires a cooperative child to stand erect with head level, feet flat, and heels against the measuring surface (Fig. 2.3). From the end of the third year until the onset of puberty, the increase in height averages 3 to 7 cm (2 to 3 inches) per year. In adolescence, girls' height potential is generally realized by 16 years of age, and boys' growth potential continues until 18 years of age. Linear growth ceases when the maturation of the skeleton is complete (see Chapters 16 and 18 for discussions of growth and sexual maturity rating).

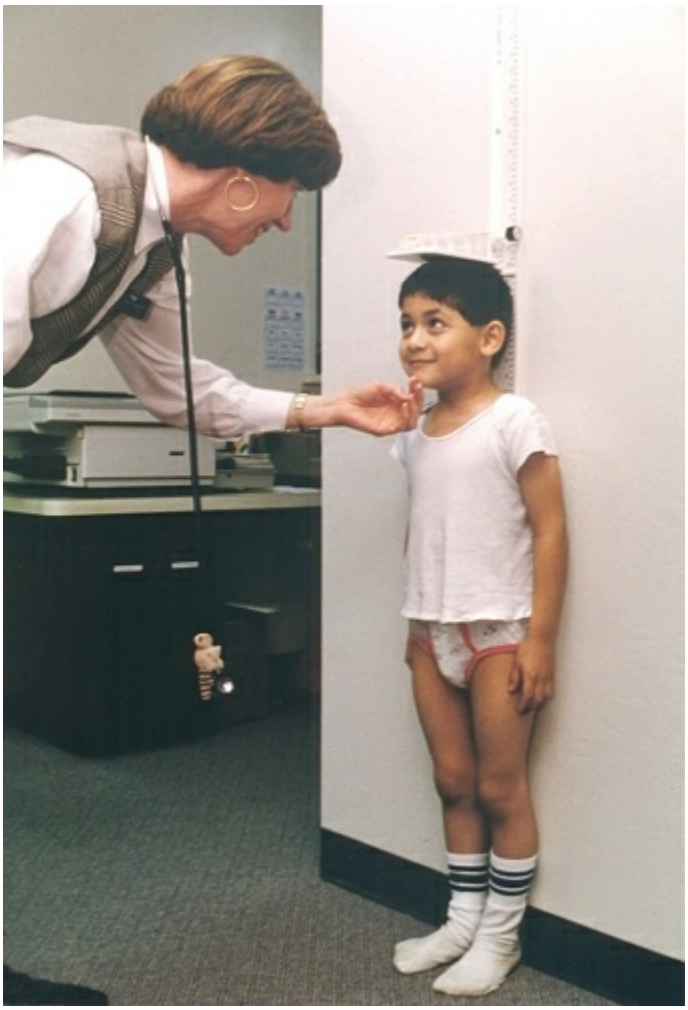

FIGURE 2.3 Measuring standing height.

In *familial short stature*, growth follows a curve below the normal growth curves, and both parents and infant are small. Familial short

stature is often not recognized as the most likely assessment for slow linear growth when parents are anxious about their child's height. Poor nutritional status is less often the cause of short stature. *Linear growth deficiency* (stunting) or *short stature* may be a key indicator in children with chronic conditions including cardiac and renal disease, fetal alcohol syndrome, methadone exposure, metabolic abnormalities, growth hormone deficiency, or chromosomal abnormalities.[1, 2]

PEDIATRIC PEARLS

Height at 2 years of age is approximately 50% of adult height. To estimate adult height, double the height at 2 years of age.[1]

Arm span

An adolescent male or female of tall stature with a disproportionate arm length should be evaluated for *arm span* measurement. This should be taken with the arms outstretched. Measure the distance from the tip of the middle finger across the crest of the shoulders to the other middle fingertip. The arm span should equal the height. In *Marfan syndrome,* the arm span exceeds the height and is associated with a disproportionate appearance.

Weight

The average birth weight in term infants is 3175 to 3400 g (7 to 7.5 lb). The average range of weight in a healthy term newborn is from 2500 to 4000 g (5 lb 8 oz to 8 lb 13 oz). Infants may lose up to 10% of their birth weight in the first week of life and normally regain it by the end of the second week.[1] Poor weight gain in early infancy is indicative of *failure to thrive* and may be caused by poor feeding patterns, malnutrition, neglect, cardiac or renal disease, chronic infection, or chromosomal and congenital anomalies.

Infants can be accurately weighed lying on an infant balance scale while wearing a dry diaper. Infants should be weighed in the sitting position only with extreme caution and only after they are able to sit without support. Weight is recorded to the nearest 0.5 oz or 0.01 kg (10 g). After 2 years of age, standing weight should be recorded

to the nearest 0.25 lb or 0.1 kg (100 g; see Box 2.1). If a young child is very fearful or irritable, parent and child can be weighed together on standing scale; then parental weight is subtracted from total weight to obtain an *estimate* of child's weight. For children with special health care needs or disabilities, accommodations for wheelchair scales or special purpose scales should be made available in the clinical setting.

Adiposity

Adipose tissue is a unique form of connective tissue with cells that maintain a large intracellular space. These *adipocytes,* or fat cells, store large quantities of triglycerides and are a repository of energy in the body. More than 90% of the body's energy is stored in the adipose tissue, and lipids are the main source for stored fuel in the body.[2] Although *adipocytes* do not reproduce, they do have a long life span, and infants born with a large numbers of fat cells are at risk for obesity in childhood. In a typical pattern of growth and development in children, adipose tissue typically declines in later infancy, with the lowest point in early childhood; it then rebounds in middle childhood through the adolescent growth spurt, as shown by the upturn in the age- and sex-specific child growth charts and the *body mass index* (BMI) curve. This upturn in the growth curve is known as *adiposity rebound* and is normally expected at about 5 to 7 years of age.[3] Large weight gains in infancy and early childhood alter the normal pattern of growth and adiposity rebound. Thus, early adiposity rebound is associated with increased depositions of fat in middle childhood, and risks associated with early adiposity rebound persist at least until early adulthood.[3] Gestation, early infancy, middle childhood, and adolescence are critical periods for the development of adiposity; the focus for pediatric health care providers is to closely monitor the pattern of the growth curve and BMI to help parents and families maintain healthy weights throughout childhood.[3]

Body mass index for age in children

BMI provides a guideline for health care providers to determine the healthy weight of a child beginning at 2 years of age based on height. The formula for determining BMI is weight in kilograms

divided by height in meters squared (BMI = weight [kg]/height squared [m^2]). Calculating and plotting BMI during the routine health visit and discussing this information with parents and families is an important part of providing comprehensive well-child care. BMI is used to determine whether a child or adolescent is underweight, overweight, or obese, and interpretation of the BMI growth chart in children depends on age and sex. The BMI growth curve is easier to use than the standard weight-for-height on the age-and-sex growth charts to point out weight trends to parents. Children with a BMI greater than or equal to the 95th percentile for age and sex or a BMI greater than or equal to 30 are considered obese, and children with a BMI between the 85th and 94th percentiles for age and sex are considered overweight (Table 2.2). Electronic health records (EHRs) include growth charts and calculate and plot BMIs, making it easily available to review with parents and families. There are no BMI parameters for children under 2 years of age. However, children younger than 2 years of age who have a weight-to-height ratio on the standard growth curve of ≥95th percentile for age and sex are considered overweight.

TABLE 2.2

Interpretation of Body Mass Index Standards

Weight	BMI for Age and Sex (Percentile)
Underweight	<5th percentile
Overweight	85th–94th percentile
Obese	≥95th percentile or BMI ≥30

BMI, Body mass index.

Data from Barlow SE: Expert Committee: Expert Committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity, *Pediatrics* 120(Supp14):S164–S192, 2007.

The overall prevalence of obesity in children and adolescents between 2 and 19 years old in the United States is 17%. For children 6 to 11 years of age, the overall prevalence of obesity is 17.5%; in adolescence, it is 20.5%. There are significant differences by race and ethnicity for the prevalence of overweight and obesity, particularly among non-Hispanic black children (19.5%) and Hispanic (21.9%) children 2 to 19 years of age.[4] Overweight is

defined as a BMI ≥85% and obesity is defined as a BMI ≥95% for age-sex-specific growth charts.[4] [4]

The prevalence of childhood overweight has remained stable over the past decade, and presents a serious pediatric health concern. Increased BMI in childhood is associated with metabolic and cardiovascular disease in adulthood and the early onset of type 2 diabetes.[5]-[7] Obesity has its origins in early life, and routine serial measurements of height and weight in the growing infant and child are most important in evaluating trends in weight gain early and communicating this information to parents and families. Rapid weight gain in early infancy, early adiposity rebound in childhood, and early pubertal development all influence the risk of becoming overweight and have been implicated in the development of childhood obesity.

The categories of BMI percentiles may not adequately define risk of obesity-related comorbid conditions, such as diabetes and hyperlipidemia, in children.[8] Overweight children have different levels of body fatness and degrees of risk factors. Obese children are taller than children of average weight, often 1 standard deviation above their counterparts for age. Abdominal obesity is identified as an important factor in the adverse health effects of childhood obesity. A high ratio of waist circumference to height has been associated with *metabolic syndrome* and *type II diabetes* in adolescents. Among overweight children, a high ratio of *waist circumference to height* is more strongly associated with adverse risk factors than a high BMI for age or skinfold thickness.[8]

Family, cultural, racial, and ethnic considerations in height and weight

Asian children may be at risk for comorbidities due to obesity at lower BMI thresholds than other ethnic groups. The risk of comorbidity is modified by actual body composition, family history of comorbidity, birth weight, and other variables, including race/ethnicity.[9] A recent study of South Asian children has shown BMI cutoffs at ≥95% for obesity and ≥85% for overweight on the age-and-sex growth charts may not approximate risks, indicating a need for additional anthropometric measures and/or ethnic-specific

criteria. Waist circumference-to-height ratio has been identified as a more accurate predictor for ethnic-specific BMI identifiers for overweight and obesity, but further research is needed.[9] South Asian and East Asian children have a higher percentage of body fat than other racial and ethnic groups of the same age, sex, and BMI and may experience greater obesity-related comorbidities at lower BMI cutoffs on the age-and-sex growth charts.[10]

Developmental considerations

Temperament

Assessment of *temperament* is a key part of comprehensive health assessment in infants, children, and adolescents. Temperament is the inborn tendency to react to one's environment in certain ways and is thought to be generally constant and at least partially genetically determined. The personality of an individual child reflects the interaction between the child's temperament and environment. Temperament can be assessed by report, clinical observation, or by a formal assessment tool. The term *goodness of fit* describes the concept of how well the child's temperament meets the expectations of his or her parents and caregivers. *Goodness of fit* promotes healthy development in the family unit through adaptation to the infant's personality, and it has a critical influence on a child's emotional well-being and behavior. Table 2.3 identifies the nine characteristics of temperament. Certain temperamental characteristics may be associated with resiliency in children, whereas other characteristics may signal emotional difficulties. Characteristics of frustration tolerance and intensity may be specific indicators of emotional dysregulation in some children and may indicate an underlying dysfunction in affective processes that significantly increases risk for mood disorders in later childhood or in adulthood.[11] An easy temperament with strong characteristics of regularity and adaptability acts as a protective factor for social-emotional development and could be related to resilience in children living in an adverse social environment.[12]

TABLE 2.3

Temperament Characteristics

Characteristics	Description
Activity	Amount of motor activity and proportion of active to inactive periods
Intensity	Amount of emotional energy released with responses
Sensitivity	Amount of sensory stimuli required to produce response
Approach/withdrawal	Nature of initial response to new stimuli
Adaptability	Ease of accepting new situation after initial response
Frustration tolerance	Length of time activity is pursued
Mood	Amount of pleasant versus unpleasant behavior child exhibits
Distractibility	Effectiveness of extraneous stimuli in altering direction of ongoing behavior
Regularity	Predictability of physiological functions such as hunger, sleep, elimination

An understanding of temperamental characteristics removes judgment and blame and helps parents to recognize that all rules do not work equally well with all children. Setting limits and time out is more difficult with some children, and some children are more difficult to discipline and require more parental ingenuity. Temperament theory objectifies these differences. A comprehensive approach to assessing development includes assessing and understanding temperament and sharing this information with parents and caregivers. A temperament assessment tool for parents may be accessed online at http://www.preventiveoz.org.

Vital signs

Temperature

Measurement of temperature continues to be a dynamic process in the infant and young child. Much discussion has occurred on the accuracy of temperature measurement and the ideal instrument to use. Currently there is a range of methods for measuring temperature in the infant and young child.

Axillary temperature measurement is the recommended method for the healthy newborn. Place the electronic digital thermometer under the arm at the base of the axilla for 3 to 5 minutes and hold the arm firmly against the side of the body with swaddling.

Research has shown parental report of axillary temperature measurement can be considered reliable and has shown a high correlation with measurement by trained, experienced staff.[13]

Rectal temperature remains the standard and most reliable method for detecting core body temperature in infants and young children. Rectal temperature aligns more closely with the core body temperature than axillary, temporal, or tympanic measurements. In early infancy to 8 to 9 months of age, rectal temperature is most accurately measured with the infant placed in the supine position on the examining table with knees flexed toward the abdomen. The infant can see the practitioner and be secured more easily in this position. Proper positioning prevents injury. In males, stimulation often elicits urination, so the penis should be covered. A child from 9 months to 2 years of age can be placed in the parent's arms or laid supine on the parent's lap. Insert the lubricated tip of thermometer into the anal opening of the rectum a distance of 0.5 to 1 inch. Rectal measurement of temperature remains the recommended standard in clinical practice for infants under 1 month of age to determine fever and when considering further diagnostic evaluation. Oral measurement is the preferred method for cooperative children over 4 years of age. Oral temperature is typically 0.6°C (1.0°F) lower than rectal temperature. Oral temperature readings can vary by thermometer position and in children with mouth breathing, tachypnea, or respiratory distress.

Tympanic membrane thermometer readings can vary depending on proper positioning in the ear, impacted cerumen, and/or otitis media. Recent studies comparing tympanic membrane and rectal temperatures in children have had contradictory results; it has been concluded that tympanic membrane thermometry shows insufficient agreement with established methods of core body temperature measurement to be used in infants and children where detection of fever has clinical implications and requires diagnostic workup.[14, 15]

Noncontact infrared thermometers are often used by parents and in some clinical settings as a noninvasive method for measuring temperature for infants and young children. Temperature is measured by pointing the infrared beams of light approximately 3 cm from the forehead or naval. Research comparing the accuracy of

the noncontact infrared thermometers with rectal temperatures has been inconclusive. Infrared thermometry has tended to overestimate the temperature of afebrile children and underestimate the temperature of febrile patients (P <.01).[16] Temperatures measured with noncontact infrared thermometers in comparison with rectal temperatures varied significantly and did not reliably predict fever in infants and young children.[17] The digital thermometer provides the best agreement with the traditional mercury-in-glass thermometer in detecting fever in infants and young children.[18]

For smart phones, apps are now available for measuring temperature in the pediatric population. An external sensor is required and the accuracy of the temperature depends on the quality of the sensor. Currently there is no standardization and no studies are available on the specificity and sensitivity of the external sensors.

Pulse

Although pulse is most often monitored electronically, pulses should be assessed for the quality of rate, rhythm, and volume or strength, and it is important to detect any differences in pulse between the upper and lower extremities. Children under 2 years of age require apical pulse (AP) measurements. Readings should be taken when the child is quiet. AP measurements are taken with the stethoscope placed over the heart below the nipple at the apex. For children over 2 years of age, the radial pulse is a satisfactory measurement. In infants and young children, the pulse should be counted for a full minute to account for irregularities in rhythm. Fig. 2.4 illustrates the location of the pulses. The radial and femoral pulses should be evaluated and compared for strength and quality. Detecting the femoral pulse in newborn and young infants requires focus and concentration by the examiner. The femoral pulse is located in the midinguinal area over the head of the femur. In children who are overweight or obese, locating the femoral pulse is challenging and requires palpating through adipose tissue to determine the presence and strength of the pulse. An absent or weak pulse in the lower extremities compared with the upper extremities is diagnostic of *coarctation of the aorta.* Table 2.4 presents

the grading of pulses used to evaluate strength and quality.

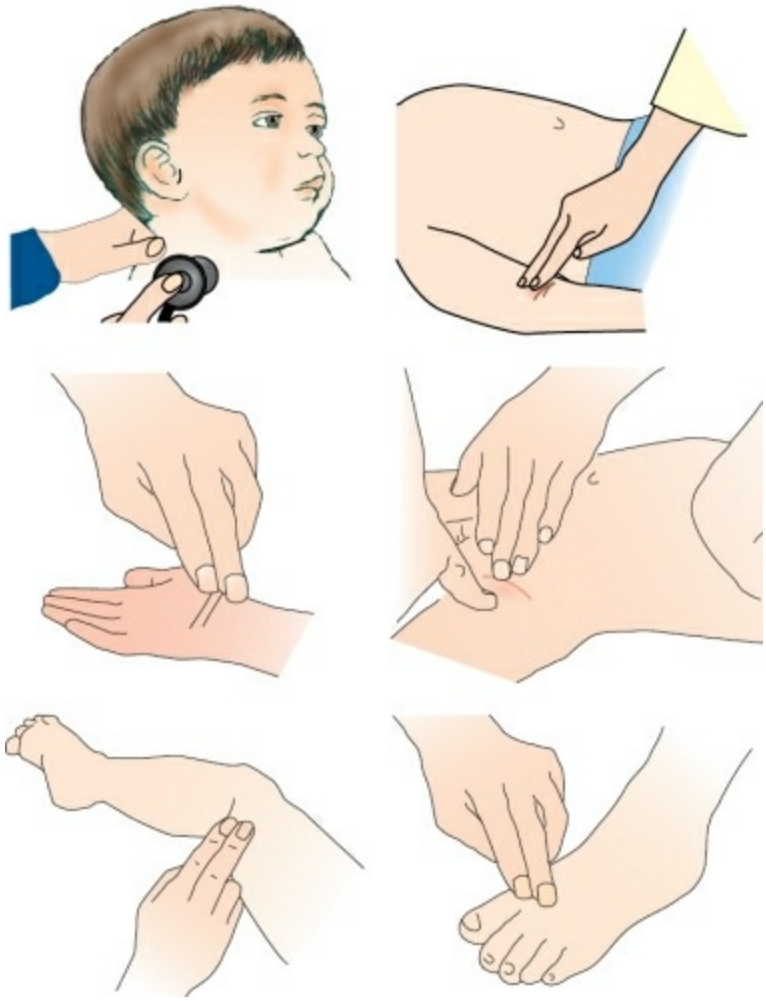

FIGURE 2-4 Assessment of pulses.

TABLE 2.4
Strength and Quality of Pulses

Strength	Quality
0	Not palpable
1+	Difficult to palpate, thready, obliterated by pressure
2+	Weak, difficult to palpate, may be obliterated by pressure
3+	Palpable, normal strength
4+	Strong, bounding, not affected by pressure

Respirations

Respirations should be assessed for rate and pattern. They should be taken when the child is quiet to accurately assess the rate and quality of breathing. In infants and young children, observe the abdominal movements when you are evaluating respiratory rate. An infant's breathing is primarily diaphragmatic. Also evaluate the use of any accessory muscles in the upper chest or any difficulty breathing. Respirations should be assessed for a full minute, because the typical respiratory rate is irregular in the newborn and very young infant. Table 2.5 shows the normal range of vital signs in children.

TABLE 2.5
Expected Range of Vital Signs

	Temperature	Respirations (breaths/min)	Heart Rate (breaths/min)	Blood Pressure
Newborn	36.5°C–37.5 °C, clothed and swaddled	30–60	120–160 AP[a] (range 80 when sleeping)	65/44 mean 75/49 90th percentile 80/52 95th percentile
1 month to 1 year	37.4°C–37.7°C	24–40	80–150 AP	86/41 mean 101/54 90th percentile 105/57 95th percentile
2–6 years	37°C–37.2°C	20–30	75–120	95/56 mean 108/67 90th percentile 112/71 95th percentile
6–12 years	36.8°C–37°C	16–25	70–110	112/73 mean 114/72 90th percentile 120/80 95th percentile
12 years to adult	36.6°C–37°C	14–20	60–105	119/78 mean 130/80 90th percentile 140/90 95th percentile

[a]Apical pulse rate taken in children younger than 2 years of age.

Data from Engorn B, Flerlage J: The Harriett Lane Handbook: A Manual for Pediatric House Officers, ed 20th, Philadelphia, 2015; Saunders; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW, editors: Seidel's Guide to Physical Examination, ed 8, St. Louis, 2015, Mosby. Flynn JT, Kaelber DC, Baker-Smith CM, et al; Subcommittee on screening and management of high blood pressure in children. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. Pediatrics. 2017; 140(3):e20171904.

AP, Apical pulse.

Oxygen saturation

Measurement of *oxygen saturation* has become a standard in most clinical settings for respiratory assessment in the pediatric patient. *Pulse oximetry* is a noninvasive method of determining oxygen saturation (Spo_2) and should be part of the criteria for evaluating any infant for respiratory distress or cardiovascular defects or any child presenting with wheezing, respiratory distress, respiratory compromise, or for preoperative evaluation. A normal Spo_2 of 95% to 96% is adequate, and 97% is generally considered to be normal. A Spo_2 ≤92% is considered *hypoxemic*. Spo_2 assists the practitioner in clinical decision making when the need for prolonged observation or hospital admission is being determined. *Pulse oximetry* alone may detect cyanotic heart disease in the asymptomatic newborn, preventing morbidity and delayed diagnosis of cardiac disease.[2]

Blood pressure

Blood pressure should be assessed at all routine well-child visits beginning at 3 years of age. In infants and children younger than 3 years of age, blood pressure should be evaluated in children who are at risk for cardiac defects or who experience trauma or have chronic conditions. The size of the blood pressure cuff is critical to obtaining an accurate measurement. The blood pressure cuff should cover about two thirds of the upper arm and should encircle the arm once. A cuff that is too large will result in a low blood pressure reading. If the cuff is too small, the blood pressure reading may be too high. For obese children and adolescents, an extra-large adult cuff may be needed to obtain an accurate measurement. Blood pressure readings can increase in children who are crying and in children and adolescents who are feeling anxious. Table 2.5 reviews the normal range of blood pressure at different ages. For healthy children and adolescents with a blood pressure reading at or above the 90th percentile for age, three independent readings should be taken to confirm the diagnosis of *hypertension.*

Pain

An accurate assessment of pain response in infants and young children requires strategies specific to the developmental level of the child. The use of the pain scale in children 3 years of age and

older has greatly improved the ability of the health care practitioner to accurately assess and treat pain in the pediatric patient. Fig. 2.5 presents the FACES pain rating scale used across most pediatric clinical settings.[19] For nonverbal children, cognitively impaired children, or children with special health care needs, the revised Face, Legs, Activity, Cry, Consolability (FLACC) Behavioral Scale has been validated for evaluating postoperative pain in the acute care setting and for pain secondary to trauma or other chronic conditions.[20] Zero to two points is assigned to each of the five categories on the scale, and total score from 0 to 10 reflects no pain to severe pain. Table 2.6 presents the FLACC Behavioral Scale.

FIGURE 2.5 FACES pain rating scale. *Brief word instructions:* Point to each face using the words to describe the pain intensity. Ask the child to choose the face that best describes its own pain, and record the appropriate number. *Note*: Use of these instructions is recommended. A rating scale can be used with children 3 years of age and older.
Original instructions: Explain to the child that each face is for a person who feels happy because he has no pain (hurt) or sad because he has some or a lot of pain. FACE 0 is very happy because he doesn't hurt at all. FACE 1 hurts just a little bit. FACE 2 hurts a little more. FACE 3 hurts even more. FACE 4 hurts a whole lot. FACE 5 hurts as much as you can imagine, although you don't have to be crying to feel this bad. Ask the child to choose face that best describes how much hurt he has. Record the number under the chosen face on the pain assessment record. Source: (From Hockenberry MJ, Wilson D: *Wong's Nursing of Infants and Children*, ed 9, St. Louis, 2011, Copyrighted by Mosby. Reprinted by permission.)

TABLE 2.6

Face, Legs, Activity, Cry, Consolability Behavioral Scale

	SCORING		
Categories	0	1	2
Face	No particular expression or smile	Occasional grimace or frown, withdrawn or disinterested	Frequent to constant frown, clenched jaw, quivering chin
Legs	Normal position or relaxed	Uneasy, restless, or tense	Kicking, or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, or tense	Arched, rigid or jerking
Cry	No cry	Moans, whimpers, or occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging, or being talked to; distractible	Difficult to console or comfort

From Merkel SI, Voepel-Lewis T, Shayevitz JR, Malviya S: The FLACC: a behavioral scale for scoring postoperative pain in young children, *Pediatr Nurs* 23:293–297, 1997.

Physical assessment techniques

Inspection

Inspection is about *looking*. *Inspection* is a skill acquired by developing detailed and meticulous observation of children and learning to see the whole as well as the parts. It involves not only the sense of sight but also the senses of hearing and smell. Inspection requires good room lighting and complete visibility of the body part to be examined to accurately assess symmetry, shape, color, and odor. It is an essential skill for the pediatric health care provider, particularly when interacting with the nonverbal child, the young pediatric patient, or a child who is ill.

Palpation

Palpation is about *touching* and *feeling*, a skill used to detect temperature, vibration, position, and mobility of body organs and glands. *Palpation* appreciates shape, pulsation, texture, and hydration of the skin and tenderness. *It* detects masses and differences in size and shape of glands, organs, muscles, and bones in all parts of the body. The fingertips are most sensitive to tactile differences, the backs of the fingers are most sensitive to temperature, and flattened fingers and palm on the chest detect

vibrations. The examiner's hands should move smoothly over the body without hesitation, first using light palpation, then followed by deep, firm pressure with palpation. It is important to know the distinction between palpation and massage. Massage incorporates *rubbing*, in contrast to the technique of *palpation,* which is the movement of the fingers over an area using light to firm pressure for the purpose of identifying size, location, mobility, sensitivity, and temperature of lymph nodes, muscles, tissues, and body organs.

Percussion

Percussion is a helpful skill for mapping out the borders of the organs or sternum and for determining the presence of solid tumors. *Percussion* requires using the examiner's fingers and hands to produce sounds on the area of the body being examined. The density of the body parts is determined by the sounds emitted when the examiner's finger strikes the middle finger of the opposite hand. The fingers produce sounds ranging from the least dense sound, *tympany* or *resonance,* as heard over the stomach or intestines, to the most dense sound, *dullness* or *flatness,* produced by striking over bone. This technique can be useful when examining the abdomen to detect the size of an organ prior to diagnostic imaging.

Auscultation

Auscultation is listening to body sounds transmitted through the stethoscope. With infants and small children, low-pitched cardiac sounds are heard best with the bell-shaped side of the stethoscope, and high-pitched lung and bowel sounds are best heard with the diaphragm, or flat portion, of the stethoscope. The bell shape is effective in isolating cardiac sounds from stomach sounds in the young infant. For best results during the physical examination of a child, it is essential to match the size of the stethoscope head to the size of the child proportionately, or the pressure on the head of a dynamic stethoscope. In listening to the lungs and heart, it is important to develop the skill to screen out adventitious sounds that occur in infants and children. The close proximity of the organs requires the examiner to screen out stomach and abdominal sounds

in listening to the heart and the respirations in the lung.

The techniques of inspection, palpation, percussion, and auscultation are among the most important tools available to the examiner for accurate diagnosis. When applied correctly, they can limit unnecessary and expensive diagnostic testing for the child or adolescent and family.

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CHAPTER 3

Developmental surveillance and screening

Abbey Alkon

Children achieve developmental milestones at their own pace, yet the sequence of developmental milestones is expected to be incremental and stepwise for all children. Screening children for developmental delay and emotional and behavioral difficulties is one of the most important components of preventive health care. Screening is an important step toward referral for evaluation and diagnosis of developmental delay and emotional and behavioral problems, and health care professionals are mandated by the Individuals with Disabilities Education Act (IDEA) and Title V of the Social Security Act to provide screening, early identification, and intervention for children with developmental delays and disabilities.[1] Early treatment of developmental delays leads to improved outcomes, enhanced function, and reduced prevalence of secondary behavior problems later in life.[2]

In the United States, 12% to 16% of children have at least one developmental delay.[3] The most common childhood developmental and behavioral problems are speech and language delay, hearing loss, emotional and behavioral concerns, learning disabilities, and delay in other developmental milestones.[4] Pediatric primary care

providers can identify young children with these developmental and mental health problems early in life by providing surveillance at every well-child visit and implementing standardized developmental screening tests at the 9-month, 18-month, and 24- or 30-month well-child visit.[1] Timely identification of developmental and behavioral problems and prompt referral for early intervention can resolve or lessen the impact of a delay or disability on the functioning of the child and family, and promote their growth and readiness to progress toward the next developmental milestone. Effective interventions in early childhood increase a child's readiness for school entry and for optimum learning in the classroom.

PEDIATRIC PEARLS

The stability and quality of the child's home and child care environment vastly impact the development of a child's full potential.

Developmental surveillance in early childhood

The first component of a developmental screening program is developmental surveillance. Developmental surveillance includes eliciting parental concerns, collecting and documenting a developmental history, and identifying risks and protective factors for developmental delay or emotional and behavioral problems in the child's environment. Observations of the child's development include speech and language skills, social skills, activity level, communication, and parent/child relationship. It is important to identify strengths in the parent and caregiver that are protective factors in the child's developmental progress. The primary care provider must include input from parents, other caregivers, and teachers in child care and school settings to make informed observations of the child's developmental progress and accurately document the ongoing developmental surveillance in the health record.

Surveillance for vision and hearing development includes asking parents about any concerns they may have about their infant or young child's developing vision or response to voices or sounds in the environment. Observable normal visual behaviors include fixing on and following near faces at 6 to 8 weeks of age; visually tracking at 4 months of age, including seeing the parent or caregiver at a distance of 5 feet; and visually fixating at 5 months on a 1-inch cube or small object at a distance of 12 inches.[5] Pediatric primary care providers should ask parents about their child's ability to hear quiet sounds and turn and locate voices. All children referred for behavioral concerns should have formal hearing and vision screening tests conducted to rule out deficits or delays. Reviews of vision and hearing screening practices are in Chapters 12 and 13, respectively.

Speech refers to the mechanics of oral communication, and *language* encompasses the understanding, processing, and production of communication during the early developmental years and throughout childhood. Surveillance of speech and language skills in childhood includes eliciting any parental concerns about communication problems, dysfluency, stuttering, articulation disorders, or an unusual voice quality.[6] Approximately 10% to 15% of 2 year olds have language delays, and 4% to 5% remain language delayed after 3 years of age.[7] Early referral and intervention is the key to optimizing speech and language development in early childhood. Pediatric primary care providers need to conduct ongoing surveillance at each health encounter and screen children whose parents have concerns about their speech and language development. Table 3.1 presents normal developmental speech and language patterns in infancy and early childhood.

TABLE 3.1

Normal Range of Developmental Speech and Language Patterns in Infancy and Early Childhood

Age	Preverbal Communication to Developing Language and Attention to Speech Sounds
8 weeks	Grunting, crying, cooing

4 months	Squeals, yells, repeating vowel sounds or sounds in repeated patterns Turns head to look at the speaker by 4 months
6 months	More complex consonant-vowel combinations ("da," "ba") Vocal play with parent or caregiver repeating sounds Responding to own and family names by 6 months
8–10 months	Multisyllable babble, including vowel-consonant combinations Appears to listen to conversation of others by 8 months Looks at or gives common objects used at home by 8 months Word-like sounds with intonation 9 months Waves bye-bye and/or plays patty-cake by 10 months
10–12 months	Infant learns favored sounds of parent and caregivers language and responds in a predictable manner
12–13 months	Emergence of first word ranges from 8 to 18 months (mean 13 months) Consonant-vowel word 12 months Single words, other than mama/dada, with consistent meaning 12–13 months Infant communicates actively by pointing
13–15 months	Follows familiar requests by 12–15 months
18–20 months	Understands about 50 words Speaks about 20 words
24 months	Median speaking vocabulary 300 words 10th percentile speaking 60 words 90th percentile speaking 500 words

From Sharma A: Developmental examination: birth to 5 years, *Arch Dis Child Educ Pract Ed* 96:162-175, 2011; and Davies D: *Child Development: A Practitioner's Guide*, ed 3. New York, 2011, The Guildford Press.

The acquisition of language is a critical developmental skill, and pediatric primary care providers can promote literacy as a component of developmental surveillance for language deficits. The *Reach Out and Read* (ROR) program, available at www.reachoutandread.org, was designed to target children in early childhood at risk for poor early school performance and provide families with books and anticipatory guidance about the importance of reading to infants and young children. Several studies have shown that ROR can significantly enhance a young child's early literacy environment by increasing the frequency of parent-child book-sharing activities and facilitating language development.[8, 9] Pediatric health care settings interested in establishing a pediatric literacy promotion program may contact the national ROR program.

The clinical assessment or surveillance of development without using standardized screening testing identifies less than 50% of children with developmental delays.[10] The ultimate goal of

developmental surveillance and screening is to improve outcomes for children with developmental delays and disorders. Table 3.2 presents normal developmental milestones of communication, and fine and gross motor, problem-solving, and social/emotional skills from 6 months to 5 years of age in accordance with *Ages and Stages Questionnaire (ASQ-3)*.[11] Guidelines for surveillance and screening during primary care visits are available at the American Academy of Pediatrics' (AAP) *Bright Futures: Guidelines for Health Supervision for Infants, Children and Adolescents*, available at http://brightfutures.aap.org.[12]

TABLE 3.2
ASQ-3 Developmental Milestones By Age

Age	Communication-Linguistic Skills	Gross Motor	Fine Motor	Problem Solving	Social-Emotional
6 months	Makes sounds like "da", "ba"; makes high-pitched squeals; turns in direction of loud noise	Rolls from back to tummy, sits with support of own hands, bears weight with feet flat on surface	Grabs toy, picks up small toy with one hand, reaches for crumb with thumb or raking motion	Reaches or grasps toy using both hands, looks for fallen object, picks up toy and mouths it	Smiles or coos in front of mirror; grabs own foot while lying on back; tries to get toy out-of-reach
12 months	Makes 2 similar Sounds, such as "ba-ba," "da-da"; plays "peek-a-boo"; says 3 words; points to objects	Walks holding on to furniture or holding both your hands; stands alone	Picks up string; uses pincer grasp; throws ball with forward arm motion; turns pages of book	Claps 2 toys together; looks for hidden object (object constancy); copies scribbles on paper	Helps with dressing; rolls or throws a ball; hugs stuffed animal or doll
18 months	Points to objects and pictures in book; says 8 or more words; imitates 2-word sentence	Picks up object from the floor; walks; climbs; walks down stairs; kicks large ball	Throws small ball; stacks 2–3 blocks; marks paper; turns pages of book; uses spoon	Dumps things out of container; copies single line drawing	Gets attention by pulling on your hand or clothes; comes to you for help; drinks from a cup; copies activities
2 years	Points to correct picture in book; imitates 2-word sentence; follows directions; names objects	Walks down stairs holding rail; kicks large ball; runs; jumps with both feet	Uses spoon without spilling; turns pages of book; flips switches on and off; stack 7 blocks; string beads	Copies line drawing; pretends play; puts things away; climbs on chair to reach object	Drinks from cup without spilling; copies activities such as sweep, comb hair; pushes toys on wheels
3 years	Points to 7 body parts; speaks with 3–4 word sentences; follows directions; identifies action in picture book; knows own first and last name	Kicks ball by swinging leg forward; jumps forward at least 6 inches; walks up stairs; stands on one foot for 3 to 5 sec; throws ball overhand	Draws line in vertical direction and circle; strings beads; cuts paper; hold crayon between fingers and thumb	Lines up 4 objects in a row; repeats 2 numbers; makes a bridge with blocks	Pushes and steers wagon or toy on wheels; dresses self; knows sex; takes turns
4 years	Follows simple directions; follows 3 directions; speaks in complete sentences	Jumps forward at least 6 inches; catch a large ball with both hands; climbs a ladder	Traces a circle; cuts paper with scissors; puts together 5–7 piece puzzle	Repeats 3 numbers; identifies small versus large circle; pretends play	Dresses self with coat and shirt; takes turns; serves self food; washes hands using soap and water
5 years	Follows 3 unrelated directions; uses 4–5 word sentences; used past tense and comparison words; answers questions	Throws ball at least 6 feet away; catches large ball with both hands; stands on one foot for 5 s; walks on tiptoes for 15 feet; hops on one foot 4–6 feet; skips	Draws picture of person with 3 or more body parts; cuts with scissors; copies shapes and letters; prints first name	Identifies colors; counts up to 15; knows opposites; names 3 numbers and 4 letters	Serves self food; washes and dries hands and face without help; knows phone number and city of residence; dresses and undresses self; uses toilet by self; takes turns and shares with others

From Squires J, Bricker D. *Ages and Stages Questionnaires*, ed 3 (ASQ-3), Baltimore, MD, 2009, Brooks Publishing Company.

Surveillance also includes "close and considerable observation," where the primary care provider compares a child's growth or development from one point to another. While a 3-year-old who has been in speech therapy may not meet all of the speech milestones expected of a 3-year-old, it would be critical to note the progress they have made since the last screen or assessment.

INTERPROFESSIONAL COLLABORATION

Close collaboration with speech therapists, occupational therapists, behavioral health and other developmental specialists provides optimum team based care for children with developmental delay.

EVIDENCE-BASED PRACTICE TIP

Children who participate in *Reach Out and Read* demonstrate increased receptive and expressive language in preschool and are less likely to suffer school failure. [13]

PEDIATRIC PEARLS

Parental concern regarding delays in fine and gross motor skills, language skills, and social/emotional development are often highly accurate and should always warrant further evaluation.

Developmental screening

Over 25% of children under 5 years of age are at risk for developmental, emotional, and behavioral problems, but fewer than one in five children receive the recommended developmental

screening.[14] Many parents with children under 6 years of age are concerned about their child's development or behavior, yet only 20% of parents report that developmental screening was conducted at their child's primary care visits prior to entering kindergarten.[15] The AAP's recommendations for developmental testing are based on evidence that indicates standardized screening tools capture up to 80% of children with early developmental delays.[1]

Developmental screening focuses on positive development and also identifies children with delays or problems early in life. It assists the health care provider with anticipatory guidance for parents and caregivers by identifying the developmental milestones expected and met at each age as the infant grows and develops (Fig. 3.1). Identification of a deficit or delay on a developmental screening test does not provide a diagnosis, but it identifies children who need further evaluation, referral, and intervention services. Table 3.3 presents standardized developmental screening tools used in infancy and early childhood, and Table 3.4 presents standardized tools used to screen for emotional, behavioral, and mental health problems during early and middle childhood and adolescence.

FIGURE 3.1 Developmental tasks for toddler.

TABLE 3.3

General Developmental Screening Tools

Tool	Age Range	Informant; Time to Administer	Description	Access
Ages and Stages Questionnaires-3 (ASQ-3)	0–5 years	Parent; 5 min	10–15 items for each age range available in multiple languages and using simple directions Screens communication, gross motor, fine motor, problem solving, personal-social skills Available in multiple languages Sensitivity = 70%–90%; Specificity = 76%–91%	Paul H. Brooks Publishers P.O. Box 10624 Baltimore, MD 21285 (800) 638-3775; http://agesandstages.com/products-services/asq3/
Parent's Evaluation of Developmental Status (PEDS)	0–8 years	Parent; 2 min	10 items elicit parent's concerns for children 0–8 years Screens for developmental and behavioral problems Available in multiple languages Sensitivity = 75%; Specificity = 74%	Ellsworth and Vandemeer Press, Ltd. P.O. Box 68164 Nashville, TN 37206 615-226-4460; http://www.pedstest.com/default.aspx
Bayley Infant Neurodevelopmental Screen (BINS)	3–24 months	Provider; 10–15 min	Trained examiner uses 10–13 directly elicited items to assess neurological processes, neurodevelopmental skills, developmental achievements Sensitivity = 75%–86% and Specificity = 75% and 86%	Pearson Assessments http://www.pearsonclinical. com
Survey of Wellbeing in Young Children (SWYC)	2–60 months	The Milestones 10 items per age; BPSC 18 items; PPSC 25 items; 10–15 adm.	Comprehensive tool made up of several scales: The Developmental Milestones, Baby Pediatric Symptom Checklist (BPSC), Preschool Pediatric Symptom Checklist (PPSC), Parent's Observations of Social Interactions (POSI), and Family Risk Factors; Covers cognitive, motor, language, social-emotional-behavioral functioning, autism, family factors; Available in English. Sensitivity (55%–100%) Specificity (63%–96%)	http://www.theswyc.org

TABLE 3.4
Emotional, Behavioral and Mental Health Screening Tools

Tool	Age Range	Informant; Time to Administer	Description	Access
Ages and Stages Questionnaires-SE-2 (ASQ-SE-2)	1–72 months	Parent;10–15 min	30 items; Screens self-regulation, compliance, communication, adaptive behaviors, autonomy, affect, interaction with people Available in English and Spanish Test-retest reliability = 89% Sensitivity = 81% Specificity = 83%	Paul H. Brooks Publishers P.O. Box 10624 Baltimore, MD 21285 (800) 638-3775; http://agesandstages. com/products-services/asqse-2/
Brief Infant/Toddler Social Emotional Assessment (BITSEA)	12–36 months	Parent; 5–7 min	42-item report measure for identifying social emotional/behavioral problems with domains for externalizing, internalizing, dysregulation, and competence Available in different languages Sensitivity = 80%–85%; Specificity= 75%–80%	Pearson Assessments; http://www. pearsonclinical.com
Modified Checklist for Autism in Toddlers (M-CHAT-R/F)	18–24 months	Parent; <5 min stage 1; Professional and parent 5–10 min stage 2	2-stage screen: (1) 20 Pass/Fail items; (2) Structured follow-up questions for children with positive screen in stage 1 Identifies children at risk of autism spectrum disorders (ASD) Sensitivity = 91%; Specificity= 96%	http://www.mchatscreen.com
Pediatric Symptom Checklist (PSC35; Y-PSC)	4–16 years	Parent (PSC) or child over 11 years (Y-PSC); 5 min	35 items elicit parent or teen's response on short statements about problem behaviors including conduct, depression, anxiety, adjustment Available in English, Spanish, Japanese Sensitivity = 80%–95%; Specificity = 68%–100%	https://www.brightfutures.org/mentalhealth/pdf/professionals/ped_symption_chklst.pdf
Children's Depression Inventory 2 (CDI 2)	7–17 years of age	Parental report, teacher report, self-report; 5–10 min for short form	12–28 items Measures cognitive, affective and behavioral signs of depression	http://www.mhs.com

Standardized developmental screening tools

Psychometrics

The psychometric properties of developmental screening tools are important considerations when deciding which screening tool to use in the pediatric health care setting.[16] Psychometric properties include test-retest reliability, validity, sensitivity, and specificity.[17] Test-retest reliability is the ability of a measure to produce consistent results. The validity of a screening test is its ability to discriminate between a child with a problem and a child without such a problem. Sensitivity is the accuracy of the test in identifying a problem, and specificity is the accuracy of the test in identifying individuals who do not have a problem. Tables 3.3 and 3.4 list the

sensitivity and specificity, developmental domains, number of items, available languages, and time to complete and score the developmental, behavioral, and emotional screening tools. Other important considerations are the costs to administer the screening tools.

The AAP endorses the use of standardized, reliable (≥80%), well-validated, and accurate screening tools with a sensitivity and specificity of ≥70% for the early identification of developmental and behavioral problems.[16] Screening tools should be directly administered by providers or trained staff, or be parent-report questionnaires. Use of developmental screening tests with lower sensitivity and specificity may identify children without significant delays and may result in unnecessary referrals. Emotional and behavioral screening tests with low specificity may identify mental health symptoms that are below the level of a *Diagnostic and Statistical Manual of Mental Disorders* diagnosis (DSM-V).[18] However, these children may benefit from early interventions in the home or improved developmental learning opportunities to address developing behavioral problems. Continual tracking of a child's developmental status and follow-up on referrals is critical to optimize child health outcomes.

Developmental screening tools for early and middle childhood

The Parents' Evaluation of Developmental Status (PEDS)[19] and the ASQ-3[11] use parental report for identifying children with possible developmental delays. Parent-completed developmental screening tools are found to be efficient, feasible, and cost-effective compared with provider-completed screening tools.

The Bayley Infant Neurodevelopmental Screener (BINS) is a tool designed specifically for high-risk infants and assesses cognitive, social, language, and gross and fine motor skills (Fig. 3.2). It has a high sensitivity and specificity in identifying delays in premature and low birth weight infants and is reliable as an indicator for referral for developmental delays in preterm infants at 6 months that remain delayed at 12 months of age; thus it is reliable as an indicator for referral.[20]

FIGURE 3.2 Scribbling to assess handedness.

The Denver-II Developmental Screening Test (DDST-II) was a widely used developmental screening tool by primary care providers, but it was found to have lower sensitivity (56% to 83%) and specificity (43% to 80%) than other developmental screening tools, which may result in overreferral of those infants and children who do not have a developmental delay and underidentification of those children who have developmental deficits. It is no longer recommended for routine use for developmental surveillance.

A new comprehensive parent-completed screening tool is the Survey of Wellbeing of Young Children (SWYC) that is composed of several scales: the Developmental Milestones, Baby Pediatric Symptom Checklist (BPSC), Preschool Pediatric Symptom Checklist (PPSC), Parent's Observations of Social Interactions (POSI), and Family Risk Factors. Together these scales cover cognitive, motor, language, social-emotional-behavioral functioning, autism, and family risk factors. The tools are available at https://www.floatinghospital.org/The-Survey-of-Wellbeing-of-Young-Children/Overview.aspx.

Mental health, emotional and behavioral screening tools for early and middle childhood and adolescence

The Ages and Stages Questionnaire-Social/Emotional (ASQ-SE) is a parent-completed questionnaire designed to monitor the social and emotional development of infants and young children and to screen for adaptive behaviors, communication, autonomy, affect, and

interaction with adults. The Brief Infant/Toddler Social Emotional Assessment (BITSEA) is a parent-report screening tool of children's social emotional and behavioral development.[21] The ASQ-SE and BITSEA both identify children with developing social and emotional disorders who may benefit from a more in-depth evaluation.

The Modified Checklist for Autism in Toddlers—Revised With Follow-Up (M-CHAT-R/F) is a two-stage screening tool to identify children at risk for autism spectrum disorder (ASD).[22] The initial stage is a parent-completed 20-item pass/fail survey followed by a second stage for positive screens with a 5- to 10-minute interview by a professional. It has a high sensitivity and specificity for identifying children with possible ASD, and assists primary care providers in assessing the need for referral for early intervention services.

The M-CHAT parent-completed survey includes the following items: Does your child play pretend or make-believe? Does your child point with one finger to ask for something or to get help? Does your child like climbing on things? Does your child respond when you call his or her name? Does your child try to copy what you do? Does your child get upset by everyday noises? If a child fails three items, the follow-up questions are administered. If a child has more than seven fails, referral for a diagnostic evaluation is indicated. Children at risk for other developmental disorders or delays may be identified with the M-CHAT screening tool.

The Pediatric Symptom Checklist (PSC) is the most commonly used mental health screening tool for children in middle childhood and adolescents in pediatric primary care practices.[23] The PSC has 35 items and screens children and adolescents from 4 years to 16 years of age. Beginning at 11 years of age, the PSC can be completed by the child or adolescent.

Approximately one in every four to five adolescents have one or more mental, emotional, and behavioral problem, including depression, conduct disorder, and substance abuse.[24] Routine screening for mental health and emotional and behavioral disorders is a primary focus for primary care providers caring for adolescents. The Children's Depression Inventory 2 (CDI 2) is used to screen children and adolescents from 7 years to 17 years of age for

cognitive, affective, and behavioral signs of depression. It is administered as a parental report, teacher report, and/or self-report screening tool.

Comprehensive health history screening for anxiety and depression in adolescence is presented in Chapter 4.

Developmental and behavioral health conditions

Developmental risk factors

Certain biological, family, and social risk factors increase the likelihood that a child will exhibit developmental delays. Biological risk factors that influence a child's development include prenatal conditions, such as maternal substance abuse, infection, chronic health conditions, medications, and severe toxemia. Neonatal risk factors include a history of prematurity, gestational age less than 33 weeks, birth weight less than 1500 g, Apgar score less than 3 at 5 minutes, neonatal infections (sepsis or meningitis), and severe hyperbilirubinemia. Social risk factors include a family history of maternal depression, low maternal education, poverty, lack of maternal bonding, child abuse and neglect, and lack of developmentally appropriate opportunities for learning. Environmental risk factors, such as disadvantaged neighborhoods, high lead levels, exposure to environmental toxins, and limited access to health care, may also contribute to developmental delays.[5]

Children at risk require especially diligent developmental surveillance, including implementing the recommended use of developmental screening tools in the first 3 years of life, genetic testing, and prompt referral for early intervention services, including a pediatric neurologist or pediatric developmental specialist when indicated. Table 3.5 presents developmental red flags in infancy and early childhood that indicate developmental deficit and the need for referral and early intervention. Box 3-1 provides further resources for the pediatric primary care provider.

TABLE 3.5

Developmental Red Flags in Early Childhood Indicating Need for

Referral

Age	Developmental Red Flag	Developmental Deficit and Need for Referral
3 months	Rolling over prior to 3 months Persistent fisting Failure to alert to environmental stimuli Failure to alert to visual/auditory stimuli	Evaluate for neuromotor dysfunction Evaluate for sensory impairment
4–6 months	Poor head control while sitting Failure to reach for objects by 5 months No social smile Lack of fixation Lack of visual tracking by 4 months Failure to turn to sound or voice at 6 months	Evaluate for hypotonia Evaluate for motor, visual or cognitive deficits Evaluate for visual loss, attachment problems, maternal depression, consider child neglect or abuse Evaluate for visual loss Requires evaluation for hearing loss
6–12 months	Persistence of primitive reflexes after 6 months Inability to sit by 9 months No babbling by 6 months No reciprocal vocalizations by 9 months Inability to localize sound by 10 months Absent stranger anxiety by 7 months	Evaluate for neuromuscular disorder Evaluate for unilateral or bilateral hearing loss Evaluate for maternal attachment or may be related to multiple care providers
12–18 months	Not verbalizing consonant by 15 months Hand dominance before 18 months Lack of imitation by 16 months Simple commands not understood Lack of pointing by 18 months Advanced noncommunicative speech Inability to walk independently by 18 months	Evaluate for mild hearing loss May indicate contralateral weakness with hemiparesis Evaluate for cognitive or socialization deficit Problem in social relatedness Evaluate for autism spectrum disorder (ASD) Evaluate for pervasive developmental disorder (PPD) Evaluate for neuromotor dysfunction
18–24 months	No two-word sentences by 24 months No word other than mama/dada by 18 months Unable to follow simple command by 24 months Cannot name one picture in book by 24 months; Echolalia beyond 24 months Does not associate toys with function-push car to make it go	Evaluate for hearing deficit Evaluate for cognitive or socialization deficit Evaluate for cognitive or socialization deficit May lack opportunity rather than motor deficit

	Inability to walk up/down stairs by 24 months	

Adapted from Gerber R, Wilks T, Erdie-Lalena C: Developmental milestones: motor development, *Pediatr Rev* 31(7):267-277, 2010; Wilks T, Gerber R, Erdie-Lalena C: Developmental milestones: cognitive development, *Pediatr Rev* 31(9):364-367, 2010; Moses S: Developmental red flags, *Family Practice Notebook,* 2012, available at http://www.fpnotebook.com/Peds/Neuro/DvlpmntlRdFlgs.htm.

BOX 3.1

Web Resources

American Academy of Pediatrics (AAP) Children's Mental Health in Primary Care; https://www.aap.org/en-us/advocacy-and-policy/aap-health-initiatives/Mental-Health/Pages/default.aspx Addressing Mental Health Concerns in Primary Care: A Clinician's Toolkit; http://www.aap.org/bookstore

AAP National Center for Medical Home Implementation; Autism Information; http://www.medicalhomeinfo.org/about/cocwd/autism.aspx

CDC's Learn the Signs Act Early; Includes fact sheets on developmental screening, resource kits, growth charts; http://www.cdc.gov/ncbddd/actearly

First Signs; Information on autism and developmental problems; http://www.firstsigns.org

U.S. Department of Health and Human Resources and U.S. Department of Education (2014). Birth to 5: Watch Me Thrive! A primary care provider's guide for developmental and behavioral screening. Washington, D.C.; https://www.acf.hhs.gov/sites/default/files/ecd/pcp_screening_guide_march2014.pdf

Autism spectrum disorders

ASD is a complex neurodevelopmental disorder that is characterized by persistent deficits in social communication and social interaction, repetitive patterns of speech or motor behavior,

and delays in communication and language skills and social and behavioral development that limit and impair everyday functioning.[25] Some of the concerning behaviors identified by 18 months of age are not smiling in response to your face, not responding to his/her name, not using the index finger to point to objects, not taking an interest in other children, not playing peek-a-boo or hide-and-seek, making unusual finger movements near his/her face, and wandering or staring without a purpose.

ASDs include autism; pervasive developmental disorder, not otherwise specified (PDD-NOS); and Asperger syndrome. The onset of ASD is before 3 years of age. ASD is at least partially genetically linked, though no single gene abnormality or mode of inheritance has yet been identified. The Centers for Disease Control and Prevention reports the prevalence of ASD is currently 1 in 68 children, and the ratio of ASD in children is 5:1 male to female.[26] According to the AAP, autism screening is recommended for all children before 24 months of age, and the M-CHAT parent-completed survey should be completed at the 18 and/or 24-month well-child visit.[27, 28] Early identification and intervention has been shown to be highly effective in helping children with ASD gain social and emotional skills.[29, 30]

Learning & neurodevelopmental disorders

Learning disorders may result from neurodevelopmental or genetic causes, or occur as comorbidities with other physical or mental disabilities (Fig. 3.3). *Learning disability* refers to difficulty in acquiring and using basic reading skills, reading comprehension, oral expression, listening comprehension, mathematical reasoning, and mathematical comprehension that occurs without an environmental precipitant in otherwise normally intelligent children. Learning disorders are neurologically based and persist into adulthood. The definition requires at least a 2-year discrepancy between the child's expected level of achievement and his or her performance.

FIGURE 3.3 A girl, 3 years and 3 months old, with macrocephaly, macrosomia, and Autism Spectrum Disorder. A missense mutation was found in the *PTEN* gene. Source: (From Zitelli BJ, McIntire SC, Nowalk AJ: *Zitelli and Davis' Atlas of Pediatric Physical Diagnosis*, ed 6, St. Louis, 2012, Saunders.)

The incidence of learning disorders is 10% to 15% of the population, with twice as many boys affected compared with girls.[31] Pediatric primary care providers assist with the diagnosis of learning disabilities by performing an initial thorough history and physical examination, including a complete neurological examination and an evaluation of school performance. The confirmation of learning disabilities includes referral for neuropsychometric testing and evaluation by a licensed psychologist.

Attention deficit hyperactivity disorder

Attention deficit hyperactivity disorder (ADHD) is a cluster of behaviors that appear early in a child's life and persist throughout childhood and adolescence. ADHD occurs in 4% to 12% of the pediatric population in the community, with males outnumbering females 3:1 to 6:1.[32] The diagnosis of ADHD is based on a characteristic clinical presentation and observable behaviors. The

problematic behaviors include inattention, hyperactivity, and impulsivity. The practitioner must rule out the possibility that such behaviors merely represent variations in normal development or temperament, and that they are not attributable to environmental factors such as a poor fit with the teacher and/or classroom. Furthermore, ADHD may coexist with other disorders or conditions that must be addressed before an appropriate diagnosis can be given. Between 10% and 40% of children with ADHD have learning disabilities. Schools are federally mandated to perform learning evaluations if a child is suspected of having a disability, such as ADHD. Pediatric primary care providers should initiate an evaluation for ADHD by gathering a comprehensive history, with careful attention given to developmental level; performing a complete physical examination, including neurological exam; vision and hearing screening; lead and hematocrit levels in preschool children; and obtaining input from teachers regarding behavior and classroom performance. Parents should be assisted in initiating an individual education plan (IEP) coordinated with the child's school. Follow-up and referral to a developmental-behavioral specialist, mental health professional, and pediatric neurologist, when indicated, initiate the further evaluation and treatment of children with ADHD.

Summary of developmental evaluation

- The most common childhood developmental and behavioral problems are speech and language delay, hearing loss, emotional and behavioral concerns, learning disabilities, and delays in other developmental milestones.
- Developmental surveillance is an important component of all pediatric primary care visits, and developmental psychometric testing should be performed routinely at the 9-month, 18-month, and 24- or 30-month well-child visit.
- Timely identification of developmental delay and behavioral problems and prompt referral for early intervention can resolve or lessen the impact of a deficit or disability on the long-term

functioning of the child and family.

DOCUMENTATION

A healthy 9-month-old infant

Crawling, pulls to stand, bangs two cubes, demonstrates thumb-finger grasp, exhibits some stranger anxiety, consolable, verbalizing consonants. Passed ASQ for communication, fine motor, gross motor, and social/emotional behaviors.

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CHAPTER 4

Comprehensive information gathering

Karen G. Duderstadt, Naomi A. Schapiro

Despite the many technologic advances in health care, the most important diagnostic tool is obtaining a comprehensive, holistic health history. Accurate diagnosis is most influenced by observing, listening, and thinking critically in a clinical setting. Recognizing patterns of health and illness in infants, children, and adolescents requires obtaining relevant data from the health history, thinking about their meaning, and explaining them logically. Taking a comprehensive history with families not only develops a profile to guide physical assessment, diagnosis, and treatment, but also contributes to the development of a trusting, continuity relationship between the family and the pediatric health care provider.

Information gathered from the family history is also key to identifying genetic patterns of inheritance in health conditions and is a guide to anticipatory guidance and promoting responsible health behaviors in the child and adolescent. Identifying and counseling children and adolescents at risk for chronic health conditions such as obesity, diabetes, hypertension, fatty liver, and cardiovascular disease begins with gathering a comprehensive family history.

Identifying and counseling for childhood overweight and obesity remain a top priority in pediatrics. Identifying early childhood factors in the comprehensive history that are significant predictors of obesity in adulthood is an important role for providers in prevention and anticipatory guidance. Probable early markers of obesity include maternal body mass index (BMI); childhood growth patterns, particularly early rapid growth and early adiposity rebound; childhood obesity; and socioeconomic status of the family.[1] In taking the health history, it is important to gather information from children and adolescents about any regular physical activity and their dietary routines in order to counsel them and foster development of positive health habits.

Another important component of comprehensive information gathering is assessing oral health. Tooth decay or *caries* is the single most common chronic health condition in childhood.[2] Tooth decay affects almost one fourth of US children from 2 to 5 years of age and half of adolescents 12 to 19 years of age.[2, 3] Children living in low-income families have a higher incidence of dental caries and unmet dental needs, particularly Hispanic and African American children.[4] Assessing children's oral health and access to dental health services is key to establishing positive dental health behaviors early in life and improving oral health outcomes in children and adolescents.

The comprehensive health history in children and adolescents incorporates screening for psychosocial conditions. The top five chronic health conditions in pediatrics currently are speech and language delays, learning disabilities, attention-deficit/hyperactivity disorders, developmental disabilities, and emotional, behavioral, and mental health problems.[5] These findings indicate the importance of screening during every well-child and adolescent encounter for learning and attention problems, mental health, and emotional/behavioral problems to assess need for further evaluation and referral.

Health equity, attaining optimum health for all, is linked to social, economic, and environmental factors, which affect the life course of the child and family.[6, 7] Trauma exposure or adverse childhood experiences (ACEs) including physical or emotional abuse or neglect, sexual abuse, domestic violence, parental substance abuse, and parental separation and divorce result in toxic

stress, which negatively affect the life course.[8, 9] Further, inclusion of an assessment of the *social determinants of health* for families at risk (e.g., food insecurity, unstable housing, family or neighborhood violence, family substance abuse) has become a priority in the comprehensive health history and an important part of the family-centered encounter, along with assessment of family strengths and protective factors.[7, 10]

The genetic family history

The *genetic family history* remains the most important tool in the comprehensive health history to identify a child's risk for developing diabetes, hypertension, cardiac disease, and a wide range of chronic health conditions.[11] Family patterns of behavioral and environmental factors that influence the occurrence of health conditions also often emerge in the course of developing a *family pedigree*.[11] The genetic family history establishes a family pedigree or *genogram* as a visual way to enhance recognition of patterns of inheritance. This approach leads to insights in patterns of inheritance across generations; it gives pediatric health care providers an opportunity to counsel families on prevention and offer referrals for further genetic testing. Knowledge of the genetic family history can aid in the diagnosis of rare single-gene disorders such as cystic fibrosis, fragile X syndrome, Huntington disease, and familial hypercholesterolemia.

It is important to be aware of the emotional and ethical issues that may arise when taking a genetic family history, such as the hereditary link between breast cancer in female relatives and familial gene testing. Pediatric health care providers can provide support to individuals and families when they are making decisions about seeking genetic testing. Also, pediatric providers are well positioned to counsel families as they provide primary care and anticipatory guidance for children from birth to young adulthood, the period during which many genetically linked disorders emerge.

Table 4.1 presents the SCREEN mnemonic for an initial genetic family history.[12] This mnemonic represents an initial series of questions used to quickly identify potential genetically influenced health conditions in the family that require further intervention,

counseling, referral, or screening by a geneticist. Fig. 4.1 is an example of a family pedigree with a multigenerational inheritance of cardiovascular disease.

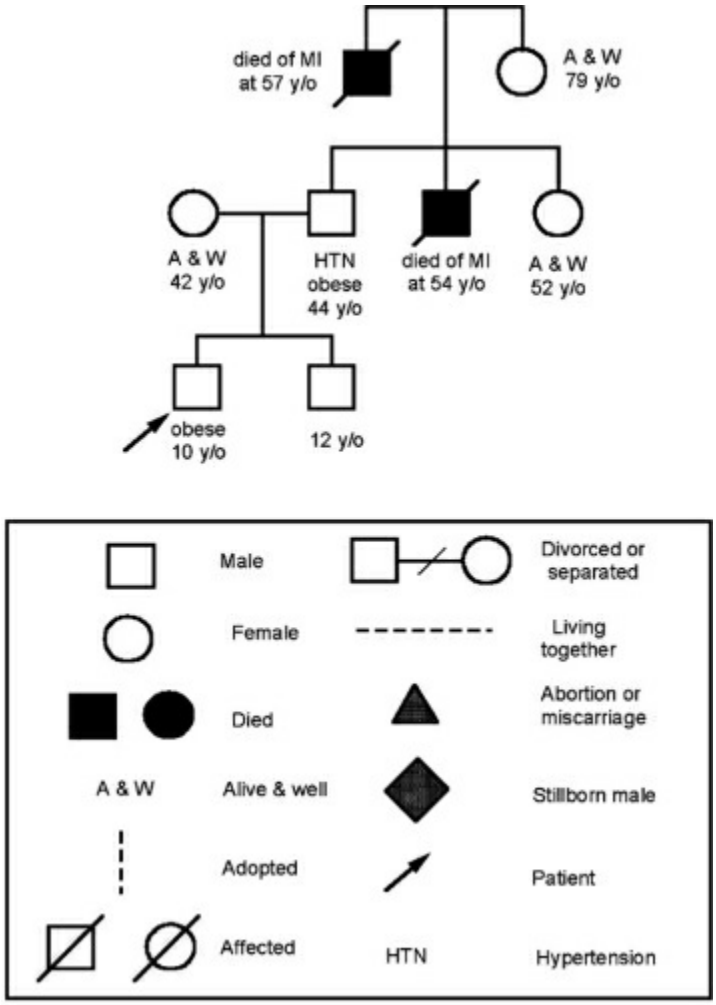

FIGURE 4.1 Family pedigree with a multigenerational inheritance of cardiovascular disease. Source: (From Bennett RL: *The Practical Guide to the Genetic Family History,* New York, 1999, Wiley-Liss, Inc.)

TABLE 4.1
The Screen Mnemonic for Family History Collection

SC	Some concerns	"Do you have any (some) concerns about diseases or conditions that run in the family?"
R	Reproduction	"Have there been any problems with pregnancy, infertility, or birth defects in your family?"

E	Early disease, death, or disability	"Have any members of your family died or become sick at an early age?"
E	Ethnicity	"How would you describe your ethnicity?" or "Where were your parents born?"
N	Nongenetic	"Are there any other risk factors or nonmedical conditions that run in your family?"

From Trotter TL, Martin HM: Family history in pediatric primary care, *Pediatrics* 120(Suppl 2):S60–S65, 2007.

EVIDENCE-BASED PRACTICE TIP

The *genetic family history* remains the most important tool in the comprehensive health history to identify a child's risk for developing diabetes, hypertension, cardiac disease, and a wide range of chronic health conditions. [11]

Family-centered history

The following key strategies are involved in developing a successful family-centered relationship with children and families in the pediatric setting:

- Listen actively to the concerns of the family. A caring relationship is established by developing an understanding of the feelings and values within the family context.
- Understand the family's expectations for the encounter. To successfully establish a caring relationship, the parent's agenda must be identified and addressed during the encounter.
- Ask open-ended questions, thereby allowing relevant data to unfold.
- Personalize your care. Ask about the health and well-being of other family members and extended family members to personalize your care and demonstrate your connectedness with the family unit and to assist in building the family profile and family-centered relationship.
- Learn and understand the role and importance of cultural influences in the family and the primary language spoken in the home. The health interview should be conducted in the family's primary language, if possible, to promote family engagement.

- Identify protective factors in the family that create a positive environment and focus on the child's strengths. Identify social or family supports for the parent and shared family interest in activities such as sports, cultural events, or religious services. These often assist in forming a supportive and protective community for children and adolescents.
- Build a sense of confidence in parents by confirming and complimenting their strengths in caring for their child. This approach also builds a trusting relationship between the family and health care provider.

With the increased workload in health care settings and the implementation of electronic health records, the process of speaking with children and families becomes more challenging. Remember to sit rather than stand while gathering the health history, maintain eye contact with the child and family in between screen time, and share screen health information such as growth charts and laboratory results as you engage with families. Families trust the pediatric provider; their relationship is based on the knowledge, understanding, respect, and care that the pediatric provider demonstrates during the encounter with their child.

Box 4.1 approaches the family-centered interview from three levels to assess family strengths, stressors, and threats to the family unit. By integrating the social context in the family-centered interview, you may identify unmet needs such as food insecurity or parental unemployment, which are important child health determinants and may be addressed by referral to community services.[10]

BOX 4.1

The Three Levels of Family-Centered Assessment

Level 1: Micro level

The child, parent or parents, partner, caregiver, other adults or children in household

- *Consider* intersecting characteristics and temperament of the individuals in the family unit, style of parenting, relationship between parents, and impact of family dynamics on physical, developmental, behavioral, and emotional health of child.

Level 2: Mezzo level

The extended family, family supports and resources, community-based systems

- *Consider* housing, school, childcare, parental work and income, health care access, church and religious community, safety of community, immigration status, and impact on child well-being.

Level 3: Macro level

The social context of child and family

- *Consider* impact of the community, cultural influences, economic status, environmental health, political climate, and impact on physical and mental health of child.

Family, cultural, racial, and ethnic considerations

The *family cultural assessment* should include the family's beliefs about the origins of wellness or illness and their perceptions of the child's health. Cultural values and beliefs strongly influence a family's perception of cause and effect of common and chronic health conditions. Establishing positive health behaviors within the family may be influenced by a family's understanding of the cause of an illness and their beliefs about the impact of medications or therapies on the illness. Integrating respect for culture is a continual process in the provider/family relationship. Understanding cultural diversity includes not only knowledge of the culture but also respect, supportive interaction, and tailoring communication, which embraces and values diversity.[13]

The following questions should be included in a *family cultural assessment* with the goal of developing an understanding of the importance of culture within the context of the family:

- Where was the child born? *If an immigrant:* How long has the

child lived in this country? What is the family's cultural identity?
- Has the child always lived with the parent/current caregiver?
- *If multiracial family:* What culture(s)/race(s) does the family identify with most closely? *If interviewing an adolescent:* What culture(s)/race(s) do you identify with most closely?
- What are the child's primary and secondary languages? What is the family's speaking and reading ability of the primary language (languages) in the home?
- What is the family's religion, and do they practice their religion daily or weekly?
- Are the family's food preferences linked to cultural or religious preferences?
- Are there beliefs about health or illness related to the family's culture?
- *If interviewing an adolescent:* Are there conflicts with parents or peers concerning expected cultural norms or customs? Have you experienced racial discrimination?

Components of information gathering

The information gathered during a health history reflects the parent's or caretaker's opinions and experience and, therefore, needs to be viewed as *subjective* information. The subjective information guides the *objective* findings of the physical examination and assists the health care provider in evaluating the family functioning, the family's approach to health and illness, and the reliability of the historian—parent, partner of the biologic parent, guardian, or grandparent. Children beginning at 3 years of age can effectively participate in the health interview. Involving children at this age establishes the health care provider as an advocate for the child and gives voice to the concerns of the child. It also allows the provider to observe the parent/child relationship during the interview process.

The type of health history gathered during an encounter depends on whether the child and family are presenting for a *comprehensive well-child visit, acute care visit, symptom-focused visit*, or for a *preparticipation sports physical examination* (see Chapter 19).

Information gathering of subjective data

Child profile

The demographic and biographical information for a child and family should be gathered at the initial health visit and verified at each health encounter. In an initial visit, the child's profile should include information on the previous health care provider, date of the child's or adolescent's last health care visit, and contact information on parents or stepparent, partner of biologic parent, caregiver, guardian, or grandparent. Family units change and this information should be updated at each health encounter.

The open-ended question

Begin the encounter with an open-ended question such as, "What brings you here today?" This allows the family to tell their story in their own words and centers the health visit around the family concerns and establishes the basis of the *family-centered interview.* Although the child or adolescent may have presented for a well visit or with a primary complaint, it is important to be open to other health issues that may arise during the encounter. Clarifying the expectations for the child or adolescent and family for the health visit and negotiating a time frame and plan for care are important to establishing trust in a provider/parent relationship.

Present concern

After summarizing the family concerns gathered in the initial interview, transition to the *provider-centered interview* to complete gathering information on the present health history and past medical history. For a *symptom-focused* or *acute care visit*, begin gathering information in the following areas to clarify information the family has not already addressed:

- When did you first notice the symptoms? Or date/time child was last well?
- Character of symptoms (time of day, location, intensity, duration, quality)?
- Progression of symptoms (How is child doing now? Symptoms getting better or worse?)

- Associated symptoms (vomiting, fever, rash, cough etc.)? Anything else bothering child?
- Exposure to household member, classmates, or others who have been ill? Pets in home?
- Any recent travel?
- Changes in appetite or activity level (eating regularly, school/day care attendance, sleeping pattern)?
- Medications taken (dosage, time, date)? Did the medication help or relieve the symptoms?
- Home management (What has the family tried? What has helped?) Use of alternative therapies or healing practices? Family belief about the illness?
- Pertinent family medical history? (Is anyone in the family immunosuppressed or does anyone have a chronic illness?)
- What changes have occurred in the family as a result of this illness (effects or secondary gain)?
- Has the family seen other health care providers for the concern?

For a *symptom-focused* or *acute care visit*, the health care provider should include only pertinent parts of the comprehensive history presented in the following sections.

Components of comprehensive health history

Prenatal and birth history

The prenatal and birth history is important in relation to the first 2 years of life and particularly relevant for preterm infants, and term infants and children with developmental delays, abnormal neurologic findings, or congenital syndromes.

Prenatal history

- GTPAL (Gravidity, number of pregnancies; Term deliveries; Premature deliveries; Abortions, spontaneous or induced; Living children)
- Maternal and paternal age, month prenatal care initiated, wanted pregnancy?
- Length of pregnancy, weight gain, history of fetal

movement/activity
- Maternal health before and during pregnancy—overweight or obese; hypertension; gestational diabetes; history of infectious diseases including tuberculosis (TB), human immunodeficiency virus (HIV), hepatitis, sexually transmitted infections (STIs), maternal group B streptococcal (GBS) status; asthma or allergies, or other chronic health conditions; hospitalizations
- Maternal substance use or abuse; tobacco use; prescription drug use; over-the-counter (OTC) drug use or abuse; intimate partner violence or exposure to abuse or family violence
- History of maternal depression or anxiety disorders

Birth and neonatal history

- Length of labor, location of birth, cesarean or vaginal delivery, epidural/anesthesia? Vacuum-assisted vaginal birth? Breech or shoulder presentation?
- Infant born on or near expected due date? Born preterm or postterm? *If preterm*: Was infant in neonatal intensive care unit (NICU)? Intubated? Length of stay in NICU?
- Birth weight and length? Gestational age?
- Apgar score, if known? Breathing problems after birth? Cardiac or other problems noted?
- Who was present at delivery, how soon after birth did mother or parent(s) touch or hold baby?
- Difficulties in feeding or stooling? Irritability or jitteriness? Jaundice?
- Length of hospitalization if term infant? Was infant discharged with mother?

Past medical history

A comprehensive health history includes a review of the *past medical history* from birth to adolescence. For symptom-focused or acute care visits, past medical history information is often focused on system-specific symptoms, which are presented in the system chapters that follow. The following are components of the past medical history:

- **Childhood conditions:** Frequent upper respiratory infections (URIs) or viral infections, history of ear infections? Frequent sore

throats; streptococcal or bacterial infections? Recurrent urinary tract infections (UTIs)? Eczema or frequent skin rashes? Dental caries?

- **Chronic conditions:** Seasonal or household allergies, wheezing or asthma; recurrent cough; frequent ear infections or fluid in ears; hearing or vision problems; overweight or obese; diabetes; bed-wetting; dental decay or poor oral health? Immunosuppressed? Chronic constipation? Chronic abdominal pain? Onset of chronic condition?
- **Hospitalizations:** Date and reason for hospitalization, length of stay, complications after hospitalization? History of surgery, trauma, or head injury?
- **Unintentional injuries:** Falls, nature of injury, age of child when injury occurred, problems after injury? Motor vehicle, bicycle, scooter/skateboard, or pedestrian-related injuries?
- **Intentional injuries:** History of family violence, physical abuse, or intimate partner violence? Child interview should include the following questions: Has anyone hurt you? Have you felt afraid someone would harm you? Is there any bullying or verbal abuse from family members or during school, after-school programs, or at childcare?
- **Allergies:** Allergic to prescription medications or antibiotics; reaction to OTC medications? Any food allergies noted? What type of reaction occurred? Severity of the reaction? Was an epinephrine pen (EpiPen) recommended? Reaction to insect bites or bee stings? Pets in home? Environmental triggers (dampness, mold, household dust, dust mites)?
- **Medications:** Is child taking vitamins, fluoride, or medications regularly? Type of medication? Use of OTC medications? Use of herbs or natural or homeopathic medicines? Cultural healing practices?
- **Immunization:** Review immunization dates and current status including status of annual flu vaccine; ask parent/caregiver about reactions to vaccines; travel history and date of last Tuberculin purified protein derivative (PPD) or TB symptom screening? If underimmunized, reasons for withholding vaccines or parental concerns about vaccine safety?
- **Laboratory tests:** Result of newborn hearing screening? Review

of newborn screening results? Hemoglobin or hematocrit screening for anemia? Lead screening? Blood test for TB (QUANTIferon)?

Family history

- **Family health history:** Hypertension, heart disease, diabetes, stroke, cancer, asthma, nasal allergy, eczema, mental health problems, STIs or HIV-positive, family history of headaches or migraine, congenital anomalies or mental retardation, sickle cell trait or disease, kidney disease, learning problems, neonatal loss or death in infancy, early death from heart attack or any cause before 50 years of age, seizures or epilepsy, or treatment for TB. (Information gathered can include age of family member and be incorporated into family pedigree and genetic family history.)
- **Socioeconomic history:** Employment status, parental occupations, parental or caregiver work schedules, parental education and parental literacy level in native language, access to health insurance, medical or health care home, uninsured or underinsured. Does family experience periods of food insecurity?
- **Home and housing situation:** Who lives in the household? Currently living with spouse or partner? Parental separation or divorce? Number of adults and children living in home? Type of home, apartment, or housing situation? Living with extended family members or relatives? Is housing secure or temporary? Unstable housing problems? Is the neighborhood and community safe? Sleeping arrangements?
- **Support systems:** With whom do you talk when you have a problem? Family support or extended family living near or in home? Help with childcare? Supportive or close friends?
- **Family violence or intimate partner violence:** Do you feel safe in the home? Is there a history of family violence or intimate partner violence in home? Has the child witnessed or been involved in family violence? Is a family member currently incarcerated?
- **Family substance use:** Is smoking, alcohol, e-cigarette, or drug use a concern in your family? Is your child or adolescent exposed to substance abuse?[7]

- **Maternal history:** Any feelings of depressed mood, feeling down, or loss of interest or pleasure in activities? Feeling anxious or unable to control worrying?

Family history should include a *family cultural assessment* (see earlier section titled "Family, Cultural, Racial, and Ethnic Considerations").

Review of systems

The *review of systems* is often included in the comprehensive health history for children and adolescents, particularly those with chronic health conditions affecting multiple systems in the body. Table 4.2 presents a guide for the information gathered in a review of systems. Experienced providers will incorporate the *review of systems* questions into the physical examination to prompt families to remember areas of past medical history that may have been overlooked in the initial interview. Interview questions for the review of systems are also found in the system-specific chapters that follow.

TABLE 4.2

Review of Systems for Middle Childhood and Adolescence

General	General health/well-being, weight gain/loss, fevers, appetite, sleep, malaise, fatigue, mood
Skin	Rashes, eruptions, skin infections, nodules or lumps, easy bruising, variations is skin pigment, congenital birthmarks
Eyes	Itching, redness, dryness, mucous discharge, tearing, rubbing eyes frequently, vision screening results, vision problems, squinting, trouble reading and/or holding screen close, sitting close to TV, has glasses or wears glasses, has contact lenses
Ears	History of frequent infections, ear pain, pulling at ears, ear drainage, ear wax impaction, trouble hearing, newborn hearing screening results, history of failed audiogram
Nose	Frequent nasal congestion, runny nose or nasal allergy, frequent upper respiratory infection, nose bleeds, difficulty breathing at night, snoring
Mouth/throat	Frequent sore throats, large tonsils, mouth breathing, speech difficulties, number of teeth, signs of teething, most recent dental visit, history of dental caries, thumb sucking
Neck/lymphatic	Pain or stiffness in neck, swollen or tender lymph nodes or glands, any lumps or masses noted
Chest	Any breast development or breast swelling –noted at what age? For pubertal males: Any breast swelling or gynecomastia?

Respiratory	Frequent cough, nighttime cough, shortness of breath when exercising, respiratory distress, nasal flaring, retractions, rate, wheezing, cyanosis, pain on respiration, tuberculosis exposure and testing, previous chest x-rays, history of asthma, sleep apnea, snoring, secondhand smoke exposure
Cardiovascular	Shortness of breath, tire easily with exercise, history of heart murmur, any chest pain, hypertension, history of anemia
Gastrointestinal	Appetite, weight gain/loss, food intolerance, frequent abdominal pain or stomachaches, vomiting (description), constipation, history of colic, lactose intolerance, frequent loose stool or diarrhea, anal itching, blood or mucus in stool
Genitourinary	Frequency of urination, blood in urine, burning/dysuria, urgency, hesitancy, wetting during day or at night, family history of bedwetting, history of urinary tract infections
Gynecologic	Females: age of puberty, age of first menses, frequency of periods and onset of last menstrual period, duration of periods, pain with menses, vaginal discharge, vaginal itching Females/Males: sexual debut, sexual history, questions about sex, attracted to males, females or both Males: wet dreams, testicular pain
Musculoskeletal	Pain, redness, or swelling around joints; sprains or strains; history of injuries; recent change in gait; hip or feet deformities; family history of flat feet; weakness; awkwardness; or clumsiness when walking or running
Neurologic	Headaches, dizziness, fainting, tremors, tics, breath-holding spells, night terrors, sleepwalking, history of head trauma, convulsions or seizures, concussions, unconsciousness, falls
Endocrine	Polyuria, polydipsia, polyphagia, any hair/skin changes (including acne, skin pigmentations, extra body hair), parental or child concerns about rate of growth, early or late puberty, elevated blood glucose
Hematologic	History of anemia, blood transfusions, any problems with bleeding, frequent bruising, sickle cell trait or disease, hepatitis exposure
Psychosocial and behavioral	Behavioral problems, frequent mood changes, concerns about unusual behavior, difficulty focusing, learning problems, anxiety, nervousness, extreme shyness, fearful, or depressed

Activities of daily living

Age-specific nutritional information gathering

Infancy

Adequate nutrition in the first 2 years of life is critical for the period of rapid growth and brain development. A thorough dietary history in the infant recognizes problems early and allows the health care

provider to counsel families for developing issues of overweight, underweight, or failure to thrive. Review gender and age-specific growth charts and height-to-weight ratios at every health encounter during infancy. Dietary history for the first year of life includes the following:

- **Breastfeeding:** Frequency and duration of infant feeds; use of supplemental formula feedings or water; difficulties with latching or feeding patterns? Concern about infant weight gain? Any vomiting after feeds? Mother's diet or dietary restrictions? Father's participation in feeding routines? Mother receiving adequate rest? Experiencing nipple soreness? Plans for return to work? Plan for expression of breast milk or weaning?
- **Formula feeding:** Type of infant formula, how is formula stored? How is powdered formula prepared? Amount and frequency of feeds? Concern about infant weight gain? Bottle-feeding at night? How often? Difficulty feeding or slow feeder? Plans for bottle weaning and transition to cup?
- **Formula and breastfeeding:** Feeding both breast milk and formula? How often and quantity of formula daily? Frequency and length of breastfeeding? Number of nighttime feedings with breast or bottle?
- **Fluids:** Drinking juice? Amount and type of juice? Drinking juice in cup or bottle? Amount of water daily?
- **Solid foods:** Age at introduction of solid foods? Portion size/amount of baby foods? Introduction of table foods and finger foods? Regular meal pattern? Infant interested in table foods? Family members frequently feeding infant table foods? Introduction of cup?

Early childhood (1 to 4 years of age)

During early childhood, it is particularly important to establish weaning from the bottle. Persistent bottle-feeding may be associated with childhood overweight or obesity, iron-deficiency anemia, and persistent bottle-feeding and breastfeeding are associated with early childhood caries. Therefore, reducing milk intake during this period is key to supporting overall healthy nutrition patterns and protecting oral health. Eating habits are established during the early years, and a balanced diet is important

to maintain a healthy weight and for optimal growth and development. Review gender and age-specific growth charts at every health encounter during early childhood and BMI after 2 years of age. Dietary history for early childhood includes the following:

- Obtain a 24-hour dietary history.
- Document servings of fruits and vegetables, sources of protein, iron, vitamin C, vitamin D and calcium (Ca^{2+}).
- Food likes and dislikes?
- Snacking and meal pattern? Frequency of juice and soda?
- Any parental concerns about the child's appetite or overeating? Any food struggles developing around mealtime or portion size?

Identify any parental concerns and challenges around the child's eating and review the importance of introducing a variety of foods often. New foods must be introduced repeatedly to gain acceptance. Portion size should be reviewed. If food is used for discipline or reward, caution the parent that this may establish an early unhealthy relationship with food. It is also important to discuss avoidance of foods commonly associated with choking or inhalation—round slices of hot dogs/sausage, popcorn, peanuts, chips, grapes, chewing gum, hardy candy, gummy candies, and carrot sticks. It is best to avoid feeding a child while driving so as to prevent choking incidents. In early childhood, only 100% juice is recommended, and this should be limited to 4 to 6 ounces daily. Soda, sugar-sweetened beverages, and carbonated beverages should be avoided. Caffeinated drinks should not be consumed in early childhood.

Middle childhood (5 to 10 years of age)

In middle childhood, the prevalence of obesity is 17.5% in children 6 to 11 years of age, with the same pattern of obesity in males and females.[14] There are significant differences by race and ethnicity for youth 6 to 19 years of age, with the prevalence of obesity for non-Hispanic black males at 18.4% and 22.4% for Hispanic males.[14] Overweight children and adolescents have a 70% chance of becoming overweight or obese adults. Therefore, during the health history interview with the school-age child and parent, it is

important to obtain a comprehensive history of eating choices and snacking habits. Determine the use of food as a reward or punishment and what the parental and family attitude is toward the weight of the child. Review gender and age-specific growth charts and BMI at every health encounter during middle childhood. Dietary history for middle childhood should include the following:

- Obtain a 24-hour dietary history. Begin with the child's recall of dietary intake and then elicit dietary information from parent or caregiver.
- Document servings of fruits and vegetables, sources of protein, iron, vitamin C, vitamin D, and calcium (Ca^{2+}).
- Review daily meal pattern and snacking habits, amount of fast foods and carry-out foods weekly, daily quantity of juice and water, amount of sugar-sweetened beverages, soda and carbonated beverages daily or weekly, consumption of sports drinks or caffeinated drinks daily or weekly.
- Review amount of high-fat, salty, and sugary foods in the diet.
- How many meals does child eat away from home daily or weekly? Does child contribute to preparation or planning of family meals at home? Does the child have any interest in cooking?
- Any parental concerns about appetite overeating, or portion size at meals? Any parental struggles around food and weight gain or weight loss?

Adolescence (11 years of age to young adulthood)

"Disordered eating" is unfortunately the norm for many adolescents who do not necessarily have an eating disorder. According to the 2015 National Youth Risk Behavior Surveillance System, 6.7% of adolescents reported no vegetable intake, 13.8% reported no breakfast intake in the 7 days before the survey, only 10.2% reported drinking the recommended amount of milk daily, and 20.4% drank one or more cans or bottles of soda daily.[15] Teens may skip breakfast, either because of lack of time, lack of available food, or the misconception that skipping breakfast will aid in weight loss. Although a vegetarian diet is often a healthy choice for adults or for children who have been raised as vegetarians, in teens a sudden switch to a vegetarian diet may actually be a red flag for a

developing eating disorder.[16] Even though eating disorders are more common among adolescent girls, boys are also at risk. A 24- or 48-hour diet recall can be helpful in evaluating nutrition status. While an assessment of the context of nutrition and activity can take longer than a traditional diet recall (e.g., "Tell me how eating and activity fit into your day yesterday"), this contextual information can be very useful in client-centered counseling techniques, such as motivational interviewing, for improved nutritional and dietary habits.

In asking adolescents about body image, the provider should avoid the assumptions that an adolescent with a low BMI for age is satisfied with his or her weight or that an adolescent with a high BMI wants to lose weight (Box 4.2). See Table 4.3 for a screening tool for evaluating adolescents for eating disorders.

TABLE 4.3

Scoff Screening Tool for Eating Disorders

S	Do you make yourself **S**ick (vomit) because you feel uncomfortably full?
C	Do you worry you have lost **C**ontrol over how much you eat?
O	Have you recently lost more than **O**ne stone (14 pounds) in a 3-month period?
F	Do you believe yourself to be **F**at when others say you are thin?
F	Would you say that **F**ood dominates your life?

From Morgan JF, Reid F, Lacey JH: The SCOFF questionnaire: a new screening tool for eating disorders, *West J Med* 172(3):164, 2000.

BOX 4.2

Body Image and Dieting Behavior in the Adolescent

Ask the following questions to gather nutritional information when assessing an adolescent.

Satisfaction with weight

- Is adolescent happy with his or her weight?

- What has he or she done to gain/lose weight?
- Exercise history?

Diet and dieting history

- Number of diets in past year? Does adolescent feel he or she should be dieting? Dissatisfaction with body size?
- Is adolescent eating in secret? Using supplements, laxatives, or diuretics?

Self-image

- How much does weight affect how adolescent feels about herself or himself?
- Has a specific binge/purge cycle been established?

Data from Anstine D, Grinenko D: Rapid screening for disordered eating in college-aged females in the primary care setting, *J Adolesc Health* 26(5):338–342, 2000.

Family meal patterns

- Is there a time daily when the family has a common meal? Are there usual family meal patterns? How often does the family eat a meal together? Who does the food shopping? How often are meals prepared at home? Daily or number of times per week? Number of fast food or carry out meals per week?
- Does family have a vegetarian or vegan diet? Are there any special cultural or religious food rituals or preferences?
- Does the family participate in any supplemental food programs? WIC (Special Supplemental Nutrition Program for Women, Infants, and Children) or SNAP (Supplemental Nutrition Assistance Program)? Does the family run out of food during the month or are they worried about running out of food?

Stooling and elimination patterns

- **Infancy:** Stooling pattern, frequency, and consistency for breastfed or formula-fed infants? Irregular or hard stool pattern?
- **Early childhood (1 to 4 years of age):** Stooling pattern and frequency? Signs of readiness for toilet training? Plan for initiating toilet training? Any resistance to toilet training or

difficulty with bowel or bladder control? Age when bowel and bladder control was attained for daytime? Age of bladder control for nighttime?

- **Middle childhood (5 to 10 years of age):** History of constipation, frequent stooling, or diarrhea? Daytime or nighttime wetting? Parental concern or attitude toward wetting incidents? Soiling or difficulty with bowel control? History of encopresis? History of diurnal or nocturnal enuresis?

Sleep

Sleep is essential for the optimal health of children and adolescents, and sleeping the recommended number of hours regularly is associated with improved attention, memory, and learning; behavior and emotional regulation; improved immune function and improved health outcomes.[17] Insufficient sleep has been associated with an increased risk for childhood overweight and obesity and also an increase in risk in adolescents of depression and self-harm.[17] Table 4.4 shows the recommended guidelines for the amount of sleep needed each night to promote optimal health across the pediatric age span.[17]

- **Infancy:** What are the infant's sleeping patterns and amount of sleep during the day and at night in the first year? Where does the infant sleep? Back to sleep on firm surface? Avoiding soft bedding?
- **Early childhood (1 to 4 years of age):** Sleep pattern and amount of sleep? Bedtime routines? Regular bedtime? Hours of sleep nightly? Where does the child sleep? Co-sleeping until what age? Always sleeps in the same household? Concerns about nightmares, night terrors, night waking, somnambulism (sleepwalking)?
- **Middle childhood (5 to 10 years of age) & adolescents:** Regular bedtime? Hours of sleep nightly? Always sleeps in the same household? Share bed, bedroom or sleep in common areas of household? Concerns about difficulty falling asleep, insomnia, nightmares, night terrors, night waking, somnambulism (sleepwalking)? Screen or cell phone time after bedtime?

TABLE 4.4

Recommended Guidelines for Amount of Sleep Per Age

4–12 months	12–16 h daily including naps
1–2 years of age	11–14 h daily including naps
3–5 years of age	10–13 h daily including naps
6–12 years of age	9–12 h daily
13–18 years of age	8–10 h daily

Data from Paruthi S, Brooks LJ, D'Ambrosio C, et al: Recommended amount of sleep for pediatric populations: a consensus statement of the American Academy of Sleep Medicine, *J Clin Sleep Med* 12(6):785–786, 2016.

Oral health and dentition

- **Infancy:** Number of teeth? Use of night bottle or frequent breastfeeding? Weaning occurred at what age? Does infant have juice in a bottle or cup? Cleaning or brushing teeth daily?
- **Early childhood to adolescents:** Number of teeth? Tooth eruption and tooth loss pattern? Brushing teeth daily? Flossing daily? Use of sugar sweetened beverages/sodas/juices? Sugary snacks and snacking frequency? Has child or adolescent seen a dentist? Does the family have a dental provider? Last dental appointment? History of dental caries? Recent application of fluoride varnish or dental sealants?

School/after-school programs/child care

- **Infancy (birth to 11 months):** Who is the infant's primary caregiver? For infants in day care or childcare, is the infant in home day care or in a childcare center? Number of children in home day care and ratio of caregivers to children? Hours spent daily in childcare?
- **Early childhood (1 to 4 years of age):** Where does the child spend the day? Who is primary caregiver? What is your child's school experience? Day care or childcare, preschool, or prekindergarten experience? How well did your child adapt to child care or school entry? Assessment of child's performance in childcare or preschool?
- **Middle childhood (5 to 10 years of age):** School performance? Likes school? What are child's strengths? Number of missed

school days? Reason for school absence? Teacher has concerns about learning? Attends special education classes? Any history of bullying in the classroom or during school hours? Attends after-school program?
- **Adolescence (11 years to young adulthood):** See adolescent-specific content later in this chapter.

Interests/hobbies/sports

- Screen time? Number of hours daily of TV, video gaming, cell phone use, iPad or tablet, laptop or computer use, and computer gaming?
- After-school activities? At school or in the community? Interests or focus (music, art, etc.)?
- Interested in sports? Involved in competitive or recreational sports? Participates in after-school sports programs?
- Daily amount of physical activity?

Safety[7]

- **Infancy:** *Car seat:* Rear-facing car seat in back seat of car? Any concern about installation of car seat? Safely installed? If preterm infant or infant with special health care needs, is infant using supports in car seat or car bed for safe transport?[18] Using sunscreen for outdoor activities? *Household:* Is household water temperature set at 120°F or less? Avoiding choking hazards? Installation of safety locks and gates? Avoiding screen time? Is infant in smoke-free environment? Discuss avoidance of parental tobacco or drug use and counsel on smoking cessation. Adults should avoid drinking hot liquids while holding an infant.
- **Early childhood (1 to 4 years of age):** *Car seat:* Rear-facing car seat in back seat of car until age 2 years? Child meets manufacturer's weight/height for front-facing infant car seat? Any concern about installation of car seat? Safely installed? Transition to booster seat and belt-positioning? Bicycle or scooter safety? Helmet use? *Household:* Childproofing of household? Preventing burns and falls? Number for poison control? Choking hazards? Tobacco use in home? Presence of

gun in home, loaded/unloaded, how stored? Stranger safety taught? Teaching pet safety? Drowning risks? Home or community pool? Empty cleaning buckets? Sunscreen use? Monitoring screen time and content (cell phone, iPad, tablet, and TV)?

- **Middle childhood (5 to 10 years of age):** *Car safety*: Transition to booster seat at what age? Belt-positioning booster seat required until child's height reaches 4 feet 9 inches or until child is at least 8 years of age. Seat belt used regularly? Riding in back seat of motor vehicle? Pedestrian safety? Bicycle, skateboard, or scooter safety? Helmet use? Sports safety equipment used? Swimming safety? Sunscreen use? Stranger safety taught? Supervision with friends/peers? Presence of gun in home? *Household:* Loaded/unloaded, how stored? Fire safety? Smoke detectors? Monitoring screen time and content (cell phone, iPad, tablet, computer, and TV)? Discussion about tobacco, alcohol, and drug use risks with child?
- Further information for parents on safe car seats and booster seats is available at https://www.safercar.gov/parents/CarSeats/Car-Seat-Safety.htm?view=full/
- **Adolescence (11 years to young adulthood):** See later section on adolescent health history.

Developmental history

Normal developmental progress and achieving developmental milestones on time is a critical marker of health during infancy and early childhood. Parental concerns about delayed physical growth or delayed psychomotor or cognitive development are of primary importance in the health history interview. Parental expectations of developmental markers vary widely in families, so it is important to elicit in the history not only what the infant or child is doing but also what the parent or family expects and allows.[7] Always note in the electronic health record the ages at which key developmental milestones were attained.

Infancy

- **Gross motor skills:** Holds head up while prone; supervised tummy time? Rolls over; sits without support? Crawling or cruising? Pulls to standing? Starting to stand and walk?
- **Fine motor skills:** Hand-eye coordination; reaching and grasping? Beginning to feed self? Pincer grasp for finger foods? Scribbling by 1 year of age?
- **Communication:** Is infant responding to sounds/hearing? First word? How many words by 1 year of age? Pointing?
- **Social/emotional:** Smiles? Eye contact and socially engaged with family? Points? Stranger anxiety?
- **Sexual development:** Normal genital exploration in infant? Terms used by parent for genitalia?

Early childhood (1 to 4 years of age)

Explore parental concerns about the child's temperament and behavior and about temper tantrums and parental reactions during the health care encounter. Discussions about establishing age-appropriate disciplinary methods and parental response to child's increasing demands are important during this developmental period. Breath-holding spells can occur during early childhood, and habits such as thumb sucking are common and should be reviewed.

- **Gross and fine motor skills:** Manual dexterity, handedness, uses utensils/self-feeding, drawing and writing? Coordination when walking and running? Learning to dress independently and tie shoes?
- **Communication:** Concerns about the child's speech or language skills? Primary language in home and number of languages spoken? Do others understand the child's speech? Do others? Does child follow directions? Does child combine words? Does child get confused, stutter, or often repeat words?
- **Social/emotional:** Dresses without help, separates from mother or parent? Names friend, peer interaction?
- **Temperament/personality:** Activity level, attention span, consolability?
- **Discipline:** What type of discipline is used when the child misbehaves? When is it used? Is the discipline effective?
- **Sexual development:** Normal self-pleasure exploration, masturbation, and sexual play in preschooler, parental attitudes,

toward normal self-pleasure exploration? Parental comfort or concerns with child's gender expression and fantasy play?[7]

Middle childhood (5 to 10 years of age)

Middle childhood provides an opportunity to foster positive health habits, particularly in the areas of nutrition and physical activity.[7] The health care provider should explore developing interests with the school-age child and parent and encourage participation in recreational and a variety of competitive sports. Exploring the quality of the child's friendships and the school performance and experience (current grades, relationship with teacher, responsibility for homework) gives a view into the child's world. Children who are bullying, being bullied, or both should be counseled and considered for possible referral. Habits such as nail biting and tics should be explored.

- **Gross and fine motors skills:** Physical balance and coordination appropriate for age? Participating in sports? Handwriting ability?
- **Communication:** Readiness to learn? Speech or language delays? Communication patterns with peers and adults? Problem-solving skills?
- **Social/emotional:** Increasing independence? Self-esteem? Relationships with parents, siblings? Peer relationships and best friend? Experienced bullying in school or from family members? Mood or symptoms of depression? History of suicidal ideation?
- **Temperament/personality:** Congenial with peers? Positive self-image? Able to name best friend? Ability to adapt to change?
- **Discipline:** What type of discipline is used when your child misbehaves? When is it used? Is it effective? Are parenting styles consistent? Does child live at more than one home? Are there behavior issues at home or at school?
- **Sexual development:** Gender and sexual identity, comfort with biologic sex, child or family concerns about gender expression; sex education provided in the home and/or at school? Signs of breast development or pubic hair noted?

Adolescence (11 years to young adulthood)

See adolescent-specific content later in this chapter.

Children with special health care needs

Children with special health care needs (CSHCNs) are those who have a chronic physical, developmental, behavioral, or emotional condition and who also require health and related services of a type or amount beyond that required by children generally.[19] In gathering information on health history, CSHCNs require special consideration. Assessing the level of impact on family functioning, family cohesion, sibling relationships, and care management required for a child with special health care needs is an important part of the health history. The role of the pediatric health care provider is to assist families in optimizing the daily functioning of the child in the home and school environment; in focusing on the strengths of the child and family; in coordinating pediatric specialty referral as indicated; and in providing support and a framework for continuity of care in a medical home or health care home setting.[7] The CSHCN screener is a five-item survey-based tool completed by the parent; it is an efficient and flexible standardized method of identifying children across the range and diversity of chronic health conditions and special needs.[20] Implementing the CSHCN screener can be an effective part of the comprehensive health history and child and family assessment. The CSHCN screener is available at http://www.ndcpd.org/ndis/pdf/tools/CAHMIScreener.pdf/

Adolescent psychosocial history and confidentiality

The most important caveat of the adolescent psychosocial history is that it should be conducted without the parent or guardian in the room. A review of studies on adolescent access to health care services shows that a perceived lack of confidentiality is a barrier to care and discussing sensitive topics improves the adolescent's satisfaction with care, yet only a minority of adolescents have ever discussed the importance of the confidentiality of the health visit with a health care provider.[21]

All 50 states have laws allowing adolescents to consent to testing for STIs and to treatment; however, confidential access to contraception, prenatal care, abortion, adoption, medical care of the

minor's child, and mental health services vary from state to state, as do laws restricting or requiring disclosure of confidential care to a parent.[22] In all 50 states, there are some limitations to confidentiality. Providers are generally required to notify parents and/or police or child protective services if an adolescent under 18 years of age expresses a desire to harm himself or herself or others or if an adolescent has been abused or neglected. In addition, some states have mandatory reporting laws about consensual sexual activity, depending on the teen's age and the age discrepancy between the teen and the sexual partner; other states encourage reporting but give the provider some discretion. It is crucial for the provider to become familiar with the specific state laws pertaining to adolescent consent, confidentiality, privacy, child abuse reporting, and the amount of control the adolescent has over the release of medical records related to confidential services. Federal regulations under the Health Insurance Portability and Accountability Act (HIPAA), as well as the adoption of electronic health records, affect the control and privacy of medical records for services the adolescent may have accessed confidentially.[23]

Adolescent psychosocial screen: From headsss to sshadess

The SSHADESS psychosocial history (Box 4.3) is a key part of a comprehensive adolescent health history and assessment.[24] The SSHADESS psychosocial history is preferred by many adolescent providers to the more familiar HEADSSS mnemonic, which covers a similar health history.[25] The SSHADESS offers the advantages of a strength-based approach and a more holistic exploration of the adolescent's emotional states.[24] The order of questioning, in general, proceeds from less private and sensitive questions to more sensitive ones, giving the provider and adolescent an opportunity to establish rapport. The SSHADESS can be tailored to early, middle, or late adolescents by modifying the questions. Remember that early adolescents and occasionally middle adolescents can have concrete thinking[26] and may wonder why the provider is asking such unusual questions. It is important to explain to early adolescents that you ask all teens the same questions; it is also important that you start the health history interview by asking

questions about the activities of peers before asking about the teen directly. Avoid medical jargon and try to use the teen's own terminology without sounding as though you are trying to talk like a teen. Remember that adolescents tend to be oriented in the here and now. A "long time ago" may refer to years or months ago or as little as a few weeks ago.

BOX 4.3

Sshadess—Review of Adolescent History

Strengths

- What are some of your strengths that help you cope with stress?
- How would your friends describe you?
- If you were applying for a job, how would you describe yourself to encourage someone to hire you?

School

- Are you in school? (Regular or continuation? English learner/bilingual? Special education/504 plan?) Attend regularly? Suspensions?
- Favorite/most difficult subjects?
- What are your grades/GPA?
    - Low: Recent if changes in GPA? Is work too difficult? Not doing homework?
    - High: Any stress about college goals or grades?
- Plans after high school graduation? (Realistic? Is teen taking right courses/activities? Taking SATs, sending college applications on time? Taking vocational training?)
    - If no specific plans, end of high school can be a difficult, vulnerable time.

Home

- Who lives with you? (One or both parents, grandparents, aunts/uncles, adult siblings, group home, foster care?) Do you

live with boyfriend/girlfriend and family?
    - Immigrant teens may live with adult siblings/extended family while parents live in home country.
- Have you lived with ...your whole life? Changes because of divorce or death of parent? Separation/reunification with parents resulting from immigration? Conflict with parents/guardians? Illness, incarceration, homelessness of family members?
- How do you get along with... ? How are conflicts handled at home?

*A*ctivities

- How do you spend free time? What do you do for fun? Sports/other extracurricular activities? Exercise? Hobbies? Church or community activities?
    - Responses reflect a measure of connection to school, extra motivation for attendance/grades
- Jobs? (Number of hours/week, schedule, location, hazards)
- Names of friends, best friend?

*D*rugs/alcohol/tobacco

Introduce the subject gently, especially with young teens; can be more direct with older teens

- Does anyone at your school... ? Do any of your friends... ? Then, have you...?
- If yes, use CRAFFT questions (see Box 4.4).
- Attempts to quit?
- Family members using drugs/alcohol/tobacco?

*E*motions

- How would you describe your moods? (Elicits rich information if teen is given time to elaborate)
- Depression/anger: Changes in energy, appetite, weight? Sleep disturbances, difficulty concentrating? Irritability is hallmark of depression in teens. Difficulty with homework, school? How teen copes with anger?
- Present/past suicidal ideation, attempts? Suicide gesture versus

self-cutting without suicidal intent?
    - Suicidal gestures/attempts may be impulsive acts after disagreement with parents, peers; teen may not self-identify as depressed.
- Warn parent/guardian if teen contemplating suicide, even if not at immediate risk.

Sexuality and sexual abuse

Warn the teen of limits of confidentiality in your setting or state.

- "Have you ever had sex or ever come close to having sex?"
    - "Come close to" covers a broad range, includes oral/anal sex, which teens often do not define as sex. For teens with the intention to initiate sexual activity, it is important to explore choice and decisions about sex in relationship. It is important to elicit history before discussing safer sex, contraception, and need for pelvic exam.
- "Are your partners girls, boys, or both?" or "Are you attracted to girls, boys, or both?"
    - Ask of everyone. Be sensitive to teens engaging in same-sex activities.
- Condom/barrier (if appropriate): "At what point in the sexual encounter do you use condoms?" ("late use" problem). Condom education in school? Knowledge of other barriers (gloves, dental dams)? Difficulties in negotiating condom use with partner?
    - Teens with less formal education may lack awareness of anatomy/physiology of genitals and reproductive organs.
- Are you doing anything to prevent pregnancy (if indicated)? Are you experiencing any pressure from partners or family to not use birth control or interference with condom or birth control use?
- "Has anyone ever touched you sexually without permission or tried to force you to have sex?" *If yes*, history of childhood sexual abuse? Acquaintance or date rape? Stranger assault?
- "Has anyone you were seeing ever put you down or made you feel ashamed? Pressured you to go the next step when you are not ready? Grabbed your arm, yelled at you, or pushed you

when they were angry or frustrated? Treated you badly when you were alone but acted differently in front of friends and family? Pushed you to have sex or do sexual things when you didn't want to?"

Safety issues

- Do you have to do anything special to be safe in your home, at school, or in your neighborhood (e.g., positive climates, presence of gangs, etc.)?
- Guns or other weapons in home or school?
- Physical fighting/abuse in home (between siblings, parents, parent-child)?
- Teen involved in physical fights at home, neighborhood, school?
- Adolescent relationship abuse? (See previous questions under Sexuality.)
- Friends in gangs? Have you ever been a gang member or do you wear a color/insignia? Teen, peers, siblings/cousins involved in gangs?
    - Be sensitive to potential reluctance to disclose.

Data from Ginsburg KR, Carlson EC: Resilience in action: an evidence-informed, theoretically driven approach to building strengths in an office-based setting, *Adolesc Med State Art Rev* 22(3):458–481, 2011; Griswold KS, Aronoff H, Kernan JB, Kahn LS: Adolescent substance use and abuse: recognition and management, *Am Fam Physician* 77(3):331–336, 2008; Miller E, Levenson R: *Hanging out or Hooking Up: Clinical Guidelines on Responding to Adolescent Relationship Abuse, an Integrated Approach to Prevention and Intervention*, San Francisco, 2012, Futures Without Violence.

Begin the interview with an opening such as "I'm going to ask you questions that I ask all teens about sex, drugs, and feelings. What you tell me is private, unless you tell me that you have been hurt by someone else, you are thinking of hurting someone else, or you are thinking of hurting yourself." Questions are asked in as neutral and nonjudgmental manner as possible to avoid making assumptions about the family structure, kinds of sexual activity, or sexual orientation of the adolescent. Asking open-ended questions such as, "How are things at home?" sets a neutral tone.

Sshadess assessment for adolescence[24]

(S) Strengths: Personal characteristics that help youth cope and succeed
(S) School: Connection to or disconnection from school
(H) Home: Family structure and living arrangement, supports, and any problems at home
(A) Activities: Sports, school activities (school connection), hobbies, church involvement, youth groups, jobs, and hours per week for each
(D) Drugs: Drug, alcohol, tobacco experimentation and abuse (Box 4.4)
(E) Emotions/eating/depression: Positive and negative emotional states, including potential depression and suicidal ideation, healthy and unhealthy eating habits
(S) Sexuality: Sexual attractions, sexual activity or intentions, and any history of coercion or sexual abuse
(S) Safety issues: Protective factors (seat belts, helmets, problem-solving skills) and risk factors (guns in home, engaging in fights, gang activity), home and neighborhood safety

BOX 4.4

Crafft Substance Abuse Screening Test

Begin: *"I'm going to ask you a few questions that I ask all my patients. Please be honest. I will keep your answers confidential."*

Part A

During the PAST 12 MONTHS, on how many days did you:	No. of Days
1. Drink more than a few sips of beer, wine, or any drink containing **alcohol**? Say "0" if none.	▭
2. Use any **marijuana** (pot, weed, hash, or in foods) or **"synthetic marijuana"** (like "K2" or "Spice")? Say "0" if none.	▭
3. Use **anything else to get high** (like other illegal drugs, prescription or over-the-counter medications, and things that you sniff or "huff")? Say "0" if none.	▭

Did the patient answer "0" for all questions in Part A?	
Yes ☐	No ☐
	
Ask CAR question only, then stop	Ask all six CRAFFT* questions below

Part B	No	Yes
C Have you ever ridden in a **Car** driven by someone (including yourself) who was "high" or had been using alcohol or drugs?		
R Do you ever use alcohol or drugs to **Relax**, feel better about yourself, or fit in?		
A Do you ever use alcohol or drugs while you are by yourself, or **ALONE**?		
F Do you ever **Forget** things you did while using alcohol or drugs?		
F Do your **Family** or **Friends** ever tell you that you should cut down on your drinking or drug use?		
T Have you ever gotten into **Trouble** while you were using alcohol or drugs?		

NOTICE TO CLINIC STAFF AND MEDICAL RECORDS: The information on this page is protected by special federal confidentiality rules (42 CFR Part 2), which prohibit disclosure of this information unless authorized by specific written consent. A general authorization for release of medical information is NOT sufficient
The **CRAFFT** test is intended specifically for adolescents. It draws upon adult screening instruments, covers alcohol and other drugs, and calls upon situations that are suited to adolescents.

Health history for female teens

- Age at menarche, last menstrual period, regularity of menses, days of menstrual flow, pain during menstruation?

Health history for sexually active male and female teens

- Sexually active? Gender of partner(s)—male, female, both, other?

- Type of activity (oral, vaginal, anal), number of sexual partners, change of recent partners, any coercion
- History of gonorrhea/*Chlamydia* screen (urine or genital probe), other STI screens (syphilis, HIV, hepatitis, HPV, herpes simplex)?

Sexually active female teens

- In addition to other STI screening (described previously), testing for trichomoniasis, bacterial vaginosis, *Candida*?
- *If history of pregnancy:* GTPAL (*G*ravidity [number of pregnancies], *T*erm deliveries, *P*remature deliveries, *A*bortions [spontaneous or induced], *L*iving children)

See Chapter 18 for detailed information on female health history.

Adolescent depression and anxiety

Anxiety and depression place affected adolescents at greater risk for poor developmental and health outcomes, such as lifelong anxiety and depression, attention deficit hyperactivity disorder (ADHD), conduct disorders, and substance abuse.[27] Adolescents who suffer from anxiety and depression also have difficulty forming important peer relationships, tend to suffer from low self-esteem, and lack self-advocacy skills due to their developmental stage.[28] Anxiety and depression can lead to school absenteeism, poor performance, and underachievement.[27] Screening for these conditions in primary care has become an important part of routine adolescent health care. Table 4.5 provides information on the Patient Health Questionnaire Screening for Depression-9 (PHQ-9), Screen for Child Anxiety Related Disorders (SCARED), and Generalized Anxiety Disorder 7-Item Scale (GAD-7). These are frequently used screening tools for adolescent psychosocial screening for anxiety and depression across clinical settings.[16, 29]

TABLE 4.5

Screening Tools for Depression and Anxiety in Adolescents

Ages	Completed by	Screening Tools
13 years and	Adolescent	Generalized Anxiety Disorder 7-Item Scale (available at http://www.phqscreeners.com/overview.aspx?

up		Screener=03_GAD-7)
13 years and up	Parent, adolescent	Patient Health Questionnaire Screening for Depression-9 (available at http://www.phqscreeners.com/overview.aspx?Screener=02_PHQ-9)
8 years and up	Parent, child, adolescent	Screen for Child Anxiety Related Disorders (available at http://www.psychiatry.pitt.edu/sites/default/files/Documents/assessments/SCARED) Child.pdf)

It is important to assess adolescents for exposures to ACEs which put them at risk for poor long-term physical health outcomes as well as increased risk for emotional and mental health conditions. Additional screening questions for identification of adolescents exposed to ACEs are available at http://www.centerforyouthwellness.org/healthcare-professionals/how-we-screen-for-aces/

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CHAPTER 5

Environmental health history

Karen G. Duderstadt

Environment is a key determinant of health, and children are uniquely vulnerable to environmental hazards. Children's genetic predisposition, social milieu, and nutrition play an important role in their susceptibility to environmental hazards.[1] The burden of disease and cost of environmental hazards stems primarily from exposure to toxic chemicals and air pollutants, and the related health conditions affecting children include lead poisoning, exposure to mercury pollution, childhood cancers, asthma, autism, intellectual and learning disabilities, and attention-deficit/hyperactivity disorder.[2]

Toxic substances are those chemicals in the environment capable of causing harm. *Toxicants* are environmental hazards from chemical pollutants, and *toxins* are environmental hazards from biologic sources. Children have a larger ratio of surface area to body mass than adults which increases their susceptibility to pesticides and other environmental toxicants. Therefore, children absorb larger amounts of environmental toxins, kilogram for kilogram, than adults. Infants have three times as large a surface area and children have twice the surface area to body mass compared with an adult.[1] Young children breathe more air and drink more water per pound of body weight than adults. They have greater exposure to toxic chemicals and air pollutants for their body

weight than adults, and they *absorb* toxic substances at a higher rate than adults.[3] The skin, respiratory tract, and gastrointestinal tract in the child are particularly vulnerable to toxic substances and absorb them more readily and efficiently than those in the adult. The high gastric pH in children facilitates absorption of environmental toxins. The developing fetus and the young child are particularly vulnerable to the neurodevelopmental effects of environmental toxins because of their rapid periods of brain growth and development in the first 2 years of life.

Children also live and play closer to environmental hazards on the ground, which increases their concentration of inhaled toxic substances. The breathing zone of a child is lower than adults, and chemical pollutants such as lead or mercury and chemicals vaporizing from carpets, flooring, or nap mats affect children at a greater rate than adults.[1] Children's metabolic pathways are immature, and they *metabolize* toxic chemicals differently than adults because they lack the enzymes to break down and remove toxic chemicals from the body.[3] Their higher metabolic rate increases their oxygen consumption and production of carbon dioxide (CO_2). The increased CO_2 requires higher minute ventilation in infants and children and increases their exposure to particulate matter in the air.[1] The *dose-response* rate for exposure in children is far more rapid than in adults.

Environmental health is defined broadly; the long-term goal is to safeguard the children's health and improve the environments where children live, learn, and play.[3] It is critically important for pediatric health care providers to understand the impact of environmental hazards and exposures on the healthy growth and development of infants, children, and adolescents. Health care providers have a professional responsibility to identify and understand the environmental health risks present in the communities in which they work, to access available health risk data from community surveillance programs, and to report exposures to appropriate local and state authorities. Furthermore, providers are mandated to conduct appropriate screening tests and to educate children and families about toxic environmental health risks.

Environmental risk factors

Children can encounter environmental hazards and be exposed to many different toxic substances in the home, car, school, child care setting, play environments, and community. Young children spend 80% to 90% of their time indoors, so environmental hazards in the home and child care environments are the primary sources of exposures.[1] Environmental health hazards include physical agents; chemical agents; outdoor and indoor air contaminants; water, soil, or dust contaminants; biologic irritants; allergens; toxins; and infectious agents. Box 5.1 presents common indoor and outdoor air pollutants, contaminants in water and soil, food contaminants, and hazardous substances that children may be exposed to through parental or family employment or hobbies.

BOX 5.1

Risk Categories of Environmental Hazards

Household exposures and indoor air pollutants

- Mold spores
- Animal dander
- Carbon monoxide
- Tobacco smoke
- Mercury vapors
- Radon
- Smoke from wood-burning stoves
- Lead
- Phthalates and plasticizers
- Personal care products or cosmetics

Outdoor air pollutants

- Pesticides
- Air particulates
- Ozone

- Insecticides
- Herbicides

School or day care exposures

- Polychlorinated biphenyls
- Arsenic from pesticide-treated wood
- Pesticides
- Friable asbestos

Community and outdoor exposures

- Insecticides
- Herbicides

Water pollutants

- Bacteria
- Parasites

Food contaminants

- Mercury
- Pesticides

Unintentional ingestions or poisonings

Family members' occupations/hobbies

- Paint contractors
- Car mechanics
- Smelters
- Agricultural workers or farm workers
- Miners
- Jewelry artists
- Stained-glass artists

Data from Health AAoPCoE: *Pediatric Environmental Health*, ed 3, Elk Grove Village, IL, 2012, American Academy of Pediatrics.

Developmental vulnerabilities

Different developmental stages put children at risk for different types of exposures to environmental hazards. Prenatal exposure of the fetus to maternal smoking, substance use, and chemical or biologic agents increases the risk of absorption of toxicants and toxins. Toxicants such as illicit drugs, alcohol, cotinine from environmental tobacco smoke, mercury, and lead, all of which cross the placental barrier, contribute to low birth weight, intrauterine growth retardation, cognitive and developmental delays, and congenital birth defects.[4] In the newborn, particular attention should be given to toxicants in breast milk or infant formula, dermal contacts, and parental occupations.

Infants and toddlers have expanded mobility, giving them increased exposure to their environment. They are particularly vulnerable to oral exposures because of their hand-to-mouth activity and inhaled substances within the physical zone they occupy near the ground. Children in early and middle childhood become susceptible to toxicants in the school, child care settings, and playground environments. Occupational hazards are of particular concern in adolescents and young adults, as are harmful exposures that occur through experimentation with illicit drugs, alcohol, and intentional inhalation of other substances.

In addition to traditional cigarettes, e-cigarettes and hookahs are increasing in popularity among youth. *E-cigarettes* are battery-powered devices that deliver a nicotine-containing aerosol or vapor by heating a solution of nicotine, propylene glycol or glycerol used as a preservative, and a flavoring agent.[5] Exogenous nicotine exposure and cigarette use in adolescence has been associated with long-term structural and functional changes in the brain as cognitive maturation occurs throughout adolescence. Lasting cognitive and behavioral impairments, disrupted memory, selective attention, and reduced executive function and activity of the prefrontal cortex have been associated with early uptake of traditional cigarettes and e-cigarettes.[6] Smoke from water pipes or *hookahs* appears to contribute to significantly more particulate matter than traditional cigarettes. There is as much as a 73-fold increase in nicotine in the urine following water pipe smoking and a significant increase in exposure to lung carcinogens.[7, 8]

Sources of environmental toxins

Children are at risk for environmental toxins from dust in homes containing lead-contaminated soil, paint chips or peeling paint; industrial toxicants in or near neighborhoods, landfill sites, or waste treatment sites; pre-1989 plumbing with presence of lead pipes or lead solder; well water or contaminated tap water; and drinking water contaminated with lead. Children that play near high-traffic areas risk exposure to soil contaminated with old deposits from leaded gasoline. Children are also at increased risk for pesticide and organophosphate exposure, as they consume more fruits and vegetables per pound of body weight than adults. Children of farm workers are particularly vulnerable to pesticide and organophosphate exposure.

Children are at risk for indoor air pollutants such as environmental tobacco smoke, mold, pesticides in the home or school, products containing lead including pottery with lead glaze and imported pottery, and food products containing lead. Children's exposure to toxins may also occur through contact with a parent's workplace or work clothes and hobbies of family members and household contacts such as soldering or refinishing old cars and painted furniture can also put children at risk.

Children with asthma are at higher risk from exposure to outdoor air pollutants. *Particulate matter* in the air consists of extremely small particles and liquid droplets that can have adverse effects on developing lungs and respiratory function. Increased levels of particulate matter created by air pollution from gasoline and diesel engines and wood-burning stoves, release of known environmental toxins from industrial sources, and increased allergens due to extreme weather events can trigger and exacerbate childhood asthma. Children living in low-income communities are at highest risk for exposure to outdoor air pollutants and environmental toxins. Children living in low-income families with food insecurity are at risk for poor nutrition, resulting in iron or calcium deficiency, which may enhance the uptake of lead and lead toxicity in the body.

Environmental health screening history

Environmental exposures in early childhood may influence health and disease across the adult life span.[3] All children and adolescents should have an environmental health screening history taken during routine primary care visits to establish the risk of exposures.[9] An environmental health screening history establishes known home, school, and/or community environmental health risks, and a family history of parental or sibling exposure in the workplace. Table 5.1 presents an environmental health screening history for use in establishing a risk profile for exposure to pesticides, poor indoor or outdoor air quality, contaminated drinking water, or chemical toxicants. A Pediatric Environmental History form for children from birth to 18 years of age with categories of environmental exposures and questions for parents and children with a positive screen for environmental risks and a Pediatric Asthma Environmental Health History form are available at https://www.neefusa.org/resource/pediatric-environmental-history

TABLE 5.1

Quick Environmental Screening Questionnaire

Source	Exposure
Where does your child spend time during the day?	
Home	
Do you have a basement where children sleep or play?	Asbestos, radon
Do you have water damage or visible mold in home?	Indoor air pollutants
Do you use pesticides in lawn/garden area or in home?	Pesticide
Do you have a gas stove or wall heater?	Carbon monoxide
Do you live near a freeway, industrial area, or polluted site?	Outdoor air/water pollutants
Smoking	
Does anyone smoke in the home environment?	Environmental tobacco smoke
Food and Water	
Do you use tap water or well water? Do you wash fresh fruits/vegetables?	Pesticides, nitrates, lead, biologic agents
Workplace	
What do family members and household contacts do for a living?	Chemical, physical, and biologic agents
Is anyone in the household involved in a hobby at home?	

Sun Exposure	
Do you use sun protection for your child?	Ultra Violet UV index

Data from American Academy of Pediatrics, Etzel RA, Balk SJ: *Pediatric Environmental Health*, ed 3, Elk Grove Village, IL, 2012, American Academy of Pediatrics.

The concept of *health risk communication* is particularly important to assessing environmental health in children and is part of a holistic approach to working with families in the clinical setting. *Health risk communication* requires active listening to identify parental concern and the public health risks of exposure to indoor and outdoor hazardous substances in the surrounding community. It requires determining the presence of an environmental hazard, assessment of the health risk, the severity of the exposure or dose, acceptability of the health risk, the impact on the health of the child or adolescent, and communicating the health risk effectively to the family.[1]

Lead exposure

The Centers for Disease Control and Prevention (CDC) recently supported ending the use of the term *level of concern* in discussing blood lead levels (BLLs) in children because evidence indicates that no level of lead is safe; therefore all elevated levels of lead are of concern.[10] Health care providers should take the primary role in educating individual families and screening for risk of environmental exposure to lead. Lead screening in children is routinely performed in most pediatric health care settings at 1 year and 2 years of age. Health care providers should monitor the health status of children and infants with BLLs above the 97.5th percentile or greater than 2.0 μg/dL until environmental investigation and mitigation have been implemented.[10] The risk of lead exposure must be reviewed with families at routine well-child health visits and in culturally and linguistically appropriate health education materials.

Immigrant and refugee children in the United States are at higher risk for elevate BLLs. Recent evidence indicates that the median age of immigrant and refugee children with elevated BLLs was 4.9 years, with an age range from 14 months to 13 years.[11] Since this age

range is considerably older than the ages of recommended screening for most children in the United States, it is particularly important for health care providers to screen the pediatric immigrant population for lead toxicity. Malnutrition and food insecurity are a known risk factors for increased BLL, and immigrant children have an increased risk of iron deficiency anemia which predisposes them to the increased intestinal adsorption of lead.[11]

Infants and children can be exposed to lead contaminants from a variety of substances including household dust, household products, candies, and medications. Children living in immigrant families are particularly vulnerable to household and medicinal exposures to lead. Box 5.2 provides questions for assessing health risks for lead exposure in children and families. Table 5.2 presents common substances associated with elevated BLLs in immigrant and refugee children.

BOX 5.2

Quick Lead Screening Questionnaire For Children

1. Within the last 6 months, has your child lived in or regularly visited a house, apartment, or school built before 1978 or before 1950? Are there paint surfaces that are peeling or chipped in the home or school?
2. Does your child live in or regularly visit a house or school built before 1978 or before 1950 that is undergoing renovation or has recently been renovated?
3. Have you ever seen your child eating paint chips or other nonfood substances such as paper?
4. Has your child ever taken home remedies such as *azarcon, pay-loo-ah, carol, ghasard, kohl, greta, bala goli, shurma,* or *rueda* (see Table 5.2)?
5. Do you use ceramic pottery from Mexico, Central America, South America, or Asia for cooking, serving, or storing food or beverages?

6. Have you ever been told that your child has an elevated blood lead level?

Data from National Environmental Education Foundation: Pediatric environmental history. https://www.neefusa.org/resource/pediatric-environmental-history; 2017 Accessed 01.02.17.

TABLE 5.2

Examples of Culture-Specific Exposures Associated With Elevated Lead Levels In Children

Exposure	Area of Origin	Reported Uses	Description
Pay-loo-ah	Southeast Asia	Treatment of fever and rash	Orange-red powder Administered by itself or mixed in tea
Greta	Mexico	Treatment of digestive problems	Yellow-orange powder Administered with oil, milk, sugar, or tea Sometimes added to baby bottles or tortilla dough
Azarcon	Mexico	Treatment of digestive problems	Bright orange powder Administered similarly to greta
Liturgirio	Dominican Republic	Deodorant/antiperspirant; treatment of burns and fungal infections of the feet	Yellow or peach-colored powder
Surma	India	For improvement of eyesight	Black powder administered to inner lower eyelid
Unidentified ayurvedic	Tibet	Treatment for slow development	Small gray-brown balls administered several times a day
Lozeena	Iraq	Added to rice and meat dishes for flavor	Bright orange spice
Tamarind candies (multiple brand names)	Mexico	Lollipops, fruit rolls, candied jams	"Bolirindo" lollipops are soft and dark brown Candied jams are typically packaged in ceramic jars
Lead-glazed ceramics	Often made in Latin America	Bean pots, water jugs	
Makeup and beauty products	Multiple cultures	Decoration	Many types

Modified from Walker P, Barrett E: *Immigrant Medicine*, Philadelphia, 2008, Saunders. In Centers for Disease Control and Prevention: Refugee health guidelines: lead screening. Available at http://www.cdc.gov/immigrantrefugeehealth/guidelines/lead-guidelines.html; 2010 Accessed 03.01.13.

Children with excess lead levels usually show no unique features on physical examination. Environmental exposures are often insidious and affect the internal organs and brain. Untreated elevated BLLs in early and middle childhood are associated with reduced intelligence quotient (IQ).[4] Children with lead toxicity may present with one or more of the following symptoms: fatigue, malaise, abdominal pain, loss of appetite, constipation, irritability, headache, weakness, or clumsiness. Any signs of developmental

delay, neurobehavioral disorders such as tics, persistent hand-to-mouth activity such as pica, unexplained seizures, anemia, chronic abdominal pain, learning difficulties, or attention-deficit/hyperactivity disorder warrant an in-depth environmental health history to relate positive exposure to environmental toxins and hazards. Fig. 5.1 illustrates the primary organs and body systems affected by exposure to environmental hazards.

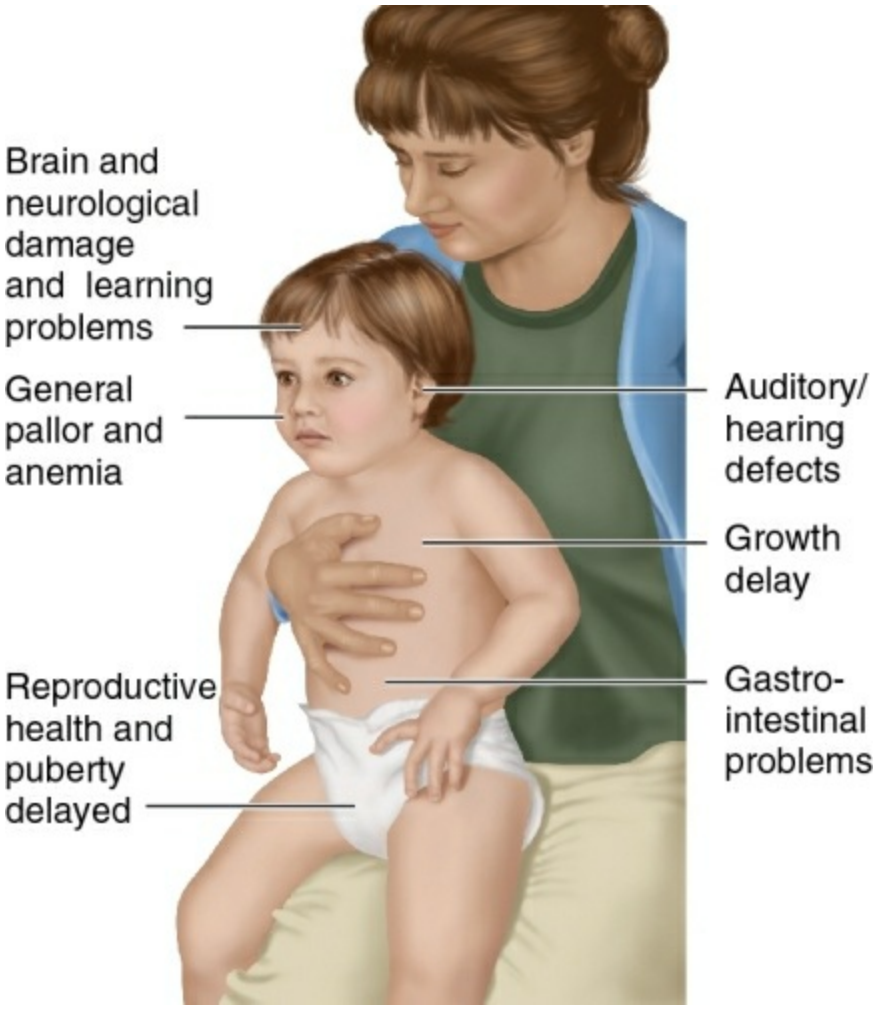

FIGURE 5.1 Effects of lead exposure on a child's body.

Endocrine-disrupting chemicals

Endocrine-disrupting chemicals are chemically manufactured or naturally occurring substances that alter the hormonal system through environmental or developmental exposures resulting in

adverse health effects.[2] They can mimic naturally occurring hormones such as estrogens (female sex hormones), androgens (male sex hormones), and thyroid hormones, producing overstimulation of the endocrine system; they affect the normal function of tissues and organs, which can result in abnormal gonadal development.[2] Low-dose exposure over time and extremely high-dose exposures to endocrine-disrupting chemicals have significant effects on the fetus and growing and developing child, including a decline in the age of the onset of puberty, decreased sperm counts, infertility, testicular cancer, cryptorchidism, hypospadias, thyroid disorders, diabetes, and some childhood cancers.[2, 4, 12]

Endocrine-disrupting chemicals are present in the environment in food, water, soil, plastics, personal care products, and drugs. Exposure can be through ingestion, topically, and transplacentally to the fetus. Phthalates, bisphenol A (BPA), and polychlorinated biphenyls (PCBs) are three of the high-volume chemicals present in the environment and are known endocrine disruptors. Phthalates and BPA are chemicals used in the production of soft plastics, polycarbonated plastics, and epoxy resins. Epoxy resins are used as coatings for liners in food and beverage cans. Phthalates are also found in personal care products, plastic toys, food packaging, car seats, floor coverings, medical tubing, and many other products. BPA is an endocrine-disrupting chemical that can cause hormonal abnormalities and adverse effects on neurodevelopment. It is found in plastic water bottles, baby bottles, water main filters, dental sealants, and paint products.[4] PCBs are synthetic organic chemicals associated with harmful effects in humans. Although PCBs are no longer manufactured in the United States, they are resistant to degradation and remain in the environment, where they are hazardous to human and animal health. Phytoestrogens, which are naturally occurring substances found in some food and soy products, also can have hormone-like activity.

EVIDENCE-BASED PRACTICE TIP

Breast milk and polycarbonate feeding bottles are the primary sources of BPA exposure among infants, and canned foods are

the primary source of BPA exposure in children. [13]

Diagnostics

For most environmental toxins, valid and reliable laboratory tests have not been developed.[4] Testing for toxins is not performed at all laboratories. Further, there may be a lapse between exposure and testing, and the levels may not reflect the total burden of the environmental contaminant on the developing child. See Table 5.3 for available laboratory tests for environmental hazards. Exposure to significant levels of toxicants should be reported to local and state authorities and the regional Pediatric Environmental Health Specialty Units (PESHUs) at http://www.aoec.org/pehsu.htm.

TABLE 5.3

Laboratory Testing For Environmental Toxins

Environmental Hazard Exposure	Diagnostic Study
Lead	Blood lead level, free erythrocyte protoporphyrin, zinc protoporphyrin
Carbon monoxide	Carboxyhemoglobin
Mercury	Blood mercury level, 24-h urine sample, hair analysis with atomic absorption spectrometry
Pesticide metabolites and organophosphates	Plasma cholinesterase levels
Tobacco metabolites	Urine cotinine assays
Polychlorinated biphenyls	Gas-liquid chromatography
Heavy metals, arsenic	24-h urine sample

Data from Dunn AM, Bevacqua J, Burns CE: Environmental health issues. In Burns CE, Dunn AM, Brady MA, B. SN, Blosser CG, Garzon DL, eds: *Pediatric Primary Care*, Philadelphia, 2017, Elsevier, pp 1170–1198.

Resources

Resources on environmental health and the impact on children and families are available to pediatric health care providers through governmental, public health, and environmental health agencies and are important in assisting with comprehensive screening for

environmental hazards (Box 5.3). Having access to evidence-based research and resources on environmental health is key to responsible health-risk screening and health-risk communication to parents, caregivers, and families.

BOX 5.3

Environmental Health Resources

Children's Environmental Health Network
http://www.cehn.org

National Center for Environmental Health
http://www.cdc.gov/nceh

Center for Health, Environment and Justice
http://www.chej.org

Columbia University's Center for Children's Environmental Health
http://www.ccceh.org

The National Environmental Education Foundation
http://www.neefusa.org/

Healthy Schools Network, Inc.
http://www.healthyschools.org

U.S. Environmental Protection Agency: Ground Water and Drinking Water Topics
https://www.cdc.gov/nceh/information/safe_water_programs.htm

World Health Organization: Health Impact Assessment
http://www.who.int/hia/en/

National Institute of Environmental Health Sciences: Endocrine Disruptors
http://www.niehs.nih.gov/health/topics/agents/endocrine/index.cfm

U.S. Department of Health & Human Services: Bisphenol A (BPA) Information for Parents
http://www.hhs.gov/safety/bpa/

Association of Occupational and Environmental Clinics
http://www.aoec.org/pehsu.htm

Summary of environmental health screening

- Children are uniquely vulnerable to environmental hazards. Children have a larger surface area/body mass ratio than adults.
- The child's skin, respiratory tract, and gastrointestinal tract are particularly vulnerable to toxic substances and absorb substances more readily and efficiently than those of an adult.
- Children live and play closer to environmental hazards on the ground, which increases their concentrations of inhaled toxic substances.
- Children's metabolic pathways are immature, and they metabolize toxic chemicals differently than adults do because they lack the enzymes to break down and remove toxic chemicals from the body.[3]
- Environmental health hazards include physical agents; chemical agents; outdoor and indoor air contaminants; water, soil, or dust contaminants; biologic irritants; allergens; toxins; and infectious agents.[1]
- Children are at risk for indoor air pollutants such as environmental tobacco smoke, mold, contaminated dust particles, pesticides in the home or school, and products containing lead, such as pottery with lead glazes and imported pottery and food products containing lead.
- Children living in low-income communities are at highest risk for exposure to environmental toxins. Ongoing lead toxicity among immigrant and refugee children in the United States has been well documented.[11]
- An environmental health history establishes known home, school, and/or community environmental health risks and a family history of parental exposure in the workplace.

DOCUMENTATION

Environmental exposure history of a 2½-year-old

A 2½-year-old healthy-appearing female lives in subsidized

housing built before 1978. Her mother gives a history of obvious mold on the bedroom and bathroom walls. For the past 2 years the building has had water damage on the walls, which has not been repaired. The building overlooks a large gas station and a high-traffic area adjacent to a freeway.

DOCUMENTATION

Environmental exposure history of a 5-year-old

A 5-year-old healthy-appearing male lives on a farm where pesticides are used seasonally on crops. His father works part-time as a crop duster. Their house was built before 1950 with some restoration under way in the family living area. The house is partially heated with a wood stove. The parents refinish old furniture as a hobby in the garage area adjacent to house. Well water is the primary source of drinking water for family.

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CHAPTER 6

Newborn assessment

Annette Carley, Karen Duderstadt

Fetal transition

The transition to extrauterine life marks a critical event for the newborn and requires readily adapting from a dependent state to one capable of newly expected independence. Important physiologic adjustments occur in pulmonary, cardiovascular, thermoregulatory, and immunologic adaptation to the external environment.[1] During fetal life with development of the respiratory system, pulmonary fluid occupies the evolving alveoli due to secretions from pulmonary epithelial cells necessary to ensure alveolar growth. Although production of this fluid normally decreases close to the time of delivery, residual fluid must be cleared to allow the lungs to expand with gas postnatally. Chemical stimuli such as decreased oxygen concentration and pH prompt respiratory center receptors to facilitate initiation of respirations. Paralleling these pulmonary changes are adaptations in circulatory patterns to redirect blood flow postnatally. In utero pulmonary blood vessels are constricted to facilitate flow away from unventilated lungs not yet meant to participate in oxygenation. A series of circulatory shunts, the *ductus arteriosus*, *ductus venosus,* and *foramen ovale*, facilitate optimized delivery of highly oxygenated blood from the placenta to the systemic circulation while bypassing the fetal lung (Fig. 6.1).[1]

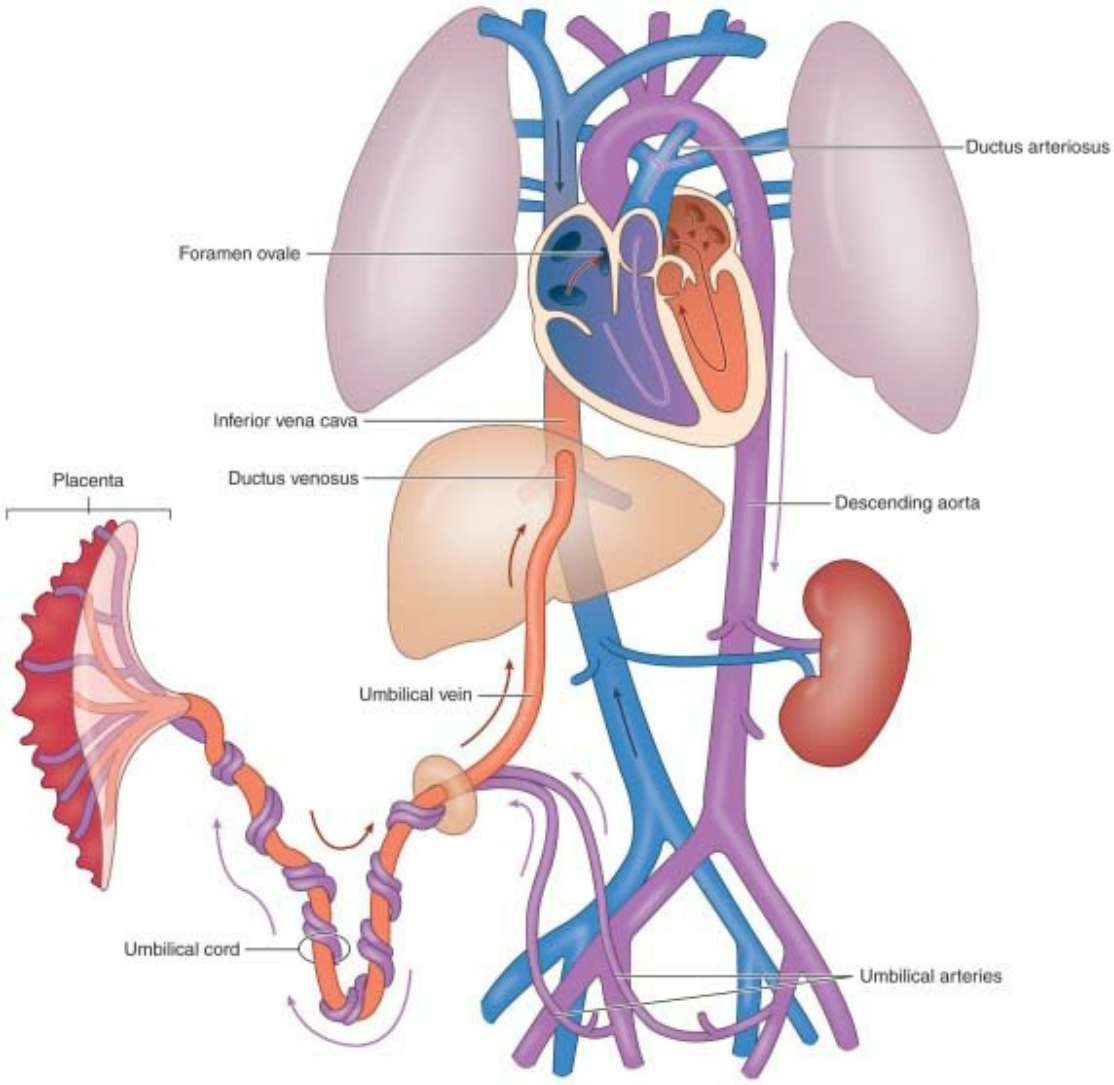


FIGURE 6.1 Fetal circulation.

Postnatally, the newborn must transition to sustained pulmonary ventilation by establishing adequate respirations for the exchange of gases. Postnatal changes in pulmonary and systemic pressure dynamics include closure of the fetal circulatory shunts to allow establishment of an adult circulatory pattern and may result in transient murmurs in some newborns.[2] The *Apgar score* reflects the transition of the newborn postnatally and is performed at 1 minute and 5 minutes of life. The score provides a summative assessment of reliable indicators of successful transition, including heart rate, respiratory effort, muscle tone, reflex irritability, and color (Tables 6.1 and 6.2). Although not predictive of long-term perinatal or neurologic outcome, it remains the standard for assessing the newborn immediately after birth.[3]

TABLE 6.1
Apgar Scoring System

Sign	0	1	2
Heart rate	Absent	Slow <100	>100
Respiratory rate	Absent	Irregular, slow, weak cry	Good, strong cry
Muscle tone	Limp	Some flexion of extremities	Well flexed
Reflex irritability	No response	Grimace	Cry, sneeze
Color	Blue, pale	Body pink, extremities blue	Completely pink

Data from Apgar V: Evaluation of the newborn infant, second report, *JAMA* 168(15):1985–1988, 1958.

TABLE 6.2
Interpretation of Apgar Scores

Total Score	Assessment
0–2	Severe asphyxia
3–4	Moderate asphyxia
5–7	Mild asphyxia
8–10	No asphyxia

Most often postnatally, the intricate transition to extrauterine life occurs rapidly and effectively. However, infants with system immaturity or other prenatal/birth complications or anomalies may not achieve adequate or complete transition. Approximately 10% of newborns will need some assistance to achieve successful pulmonary transition, although only 1% will require extensive respiratory support measures.[1, 3] Although tachypnea may be present during the first postnatal hours, from efforts to reabsorb alveolar fluid, persistence of this symptom may indicate poor transition. Clinical findings such as nasal flaring, expiratory grunting, or chest wall retractions may indicate respiratory distress needing intervention.[1]

Although most systems are anatomically complete by approximately 24 weeks gestation, many systems including the pulmonary system are functionally immature until closer to term. The pediatric health care provider must acknowledge the influences

of gestational age, as well as circumstances of ongoing fetal environment, labor and/or delivery, and early postnatal life in assessing the newborn to determine the need for intervention to support adequate transition.

Information gathering of prenatal and birth history

The Information Gathering table presents pertinent questions to ask about the maternal and prenatal history, neonatal history, and birth history. In addition, see prenatal and birth history in Information Gathering in Chapter 4.

Information Gathering for Prenatal and Birth History

	Questions to Ask
Maternal and Prenatal History	• History of prenatal care access? Maternal weight gain during pregnancy? General health during pregnancy? Any maternal infections or chronic health conditions? History of gestational diabetes or thyroid disease? Any use of drugs or alcohol during pregnancy? Maternal smoking during pregnancy? Any partner violence? History of maternal depression or anxiety disorder? Immunization status? Term birth? GTPAL (Gravidity, number of pregnancies; Term deliveries; Premature deliveries; Abortions, spontaneous or induced; Living children)
Birth History	Vaginal or cesarean birth? Prolonged labor with prolonged third stage? Precipitous delivery? Vacuum-assisted delivery? Breech or shoulder presentation? History of premature membrane rupture or maternal intrapartum fever? Maternal group B streptococcal (GBS) screen? Respiratory distress at birth? Apgar score, if known?
Neonatal History	Risk factors for sepsis? Newborn screening results? Difficulties in feeding or stooling? Irritability or jitteriness? Jaundice? Length of hospitalization? Discharged with mother?

Assessment of gestational age

An important component of newborn assessment is establishing gestational age. Obtaining an accurate assessment of gestational age begins with the prenatal and birth history, and can assist the provider in anticipating conditions associated with preterm birth. Infants can be classified by gestational age as *term infant*, *preterm infant*, or *postterm infant* depending on the number of completed

postmenstrual weeks. Gestational growth can be affected by maternal, placental, and fetal factors.[4] *Term infants* are born between the start of the 38th and completion of the 41st gestational week, whereas *preterm infants* are born prior to completing the 37th week and postterm infants are born following completion of the 41st week (Fig. 6.2). Infants born preterm and postterm are at increased risk for postnatal complications. Preterm infants are at risk for system immaturity, notably the pulmonary, gastrointestinal, and neurologic systems. Especially at lower gestational ages, preterm infants may develop complications such as respiratory distress syndrome, necrotizing enterocolitis, and intraventricular hemorrhage that can substantially impact survival. Postterm infants are at risk for birth complications, such as shoulder dystocia, if they are also macrosomic, in addition to being at added risk for poor tolerance of labor, pulmonary complications such as meconium aspiration, and hypoglycemia. Accuracy in assessment of gestational age is thus essential for developing effective management plans.[2, 5-7]

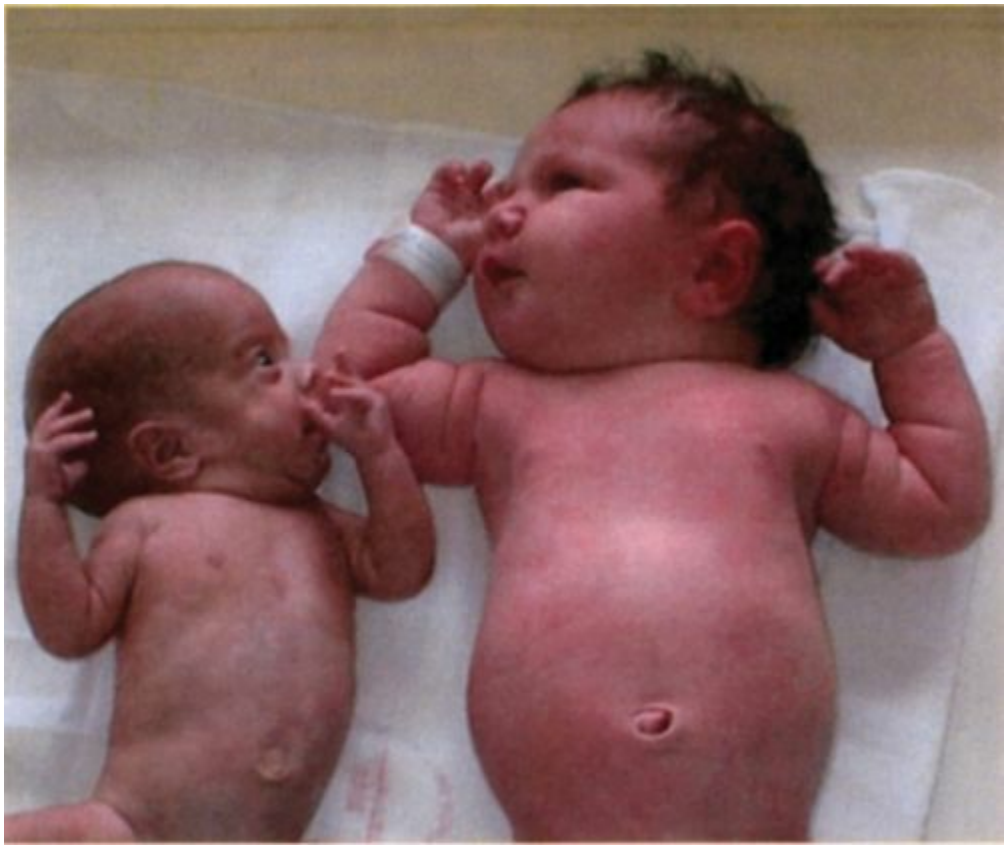


FIGURE 6.2 Preterm and postterm infant.

Gestational age can be estimated based on prenatal assessments with calculated dates based on last menstrual period, or postnatally using a standardized assessment tool that considers neuromuscular and physical criteria.[6] The New Ballard Score (NBS) is the tool most commonly used to evaluate gestational age postnatally (Fig. 6.3 and Box 6.1). The NBS, consisting of six neuromuscular and six physical criteria, is validated to accurately estimate gestational age within 2 weeks when performed within the first 48 hours following birth.[2,6] An increasing population to be aware of is the late preterm infant, defined as the infant whose postmenstrual age is 34 weeks 0/7 days to 36 weeks 6/7 days. These infants are not expected to be as physiologically competent as term infants and may have issues that prolong hospitalization, including jaundice, poor feeding, hypoglycemia, apnea, and thermal instability.[1, 6]

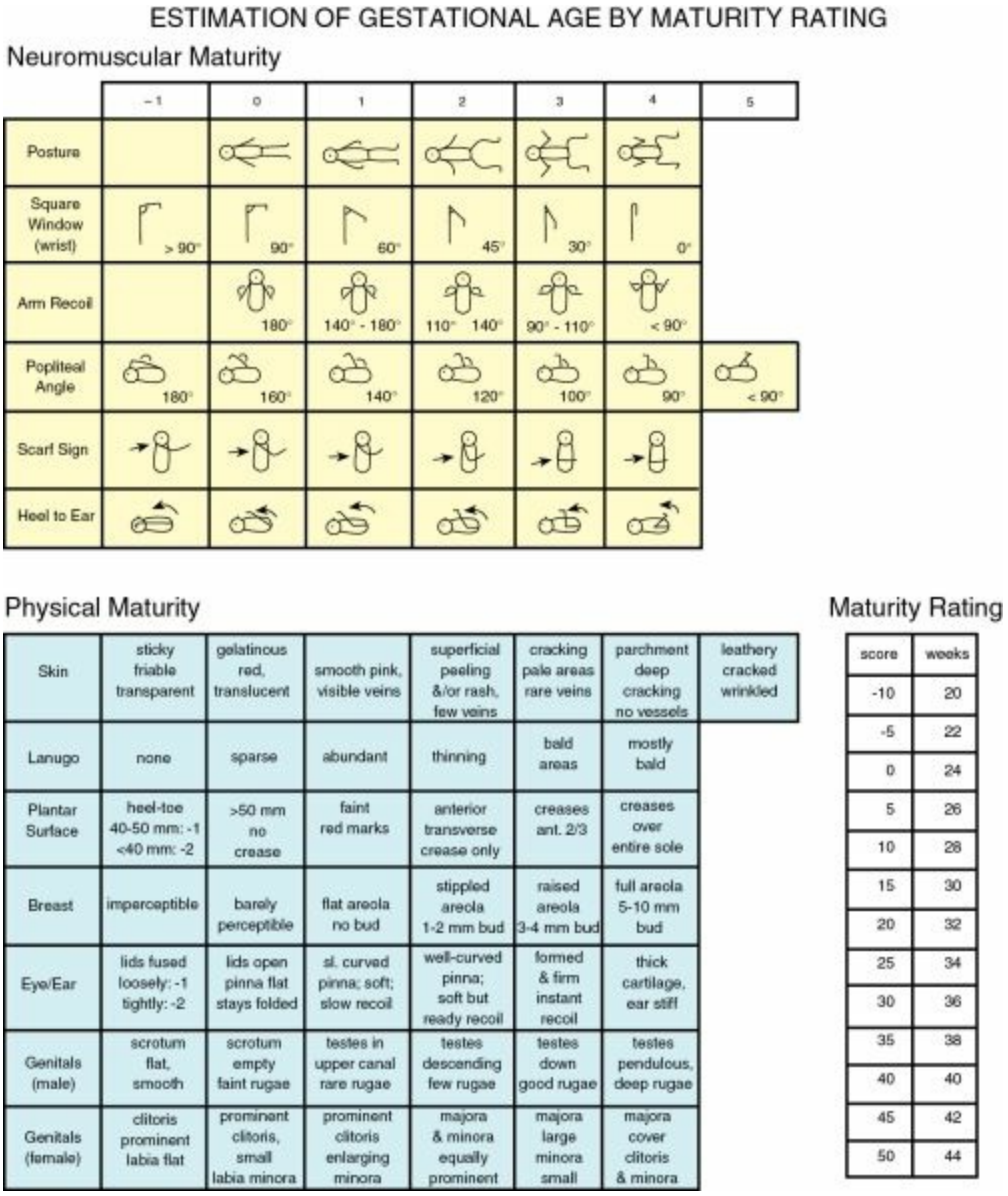


FIGURE 6.3 New Ballard Score. Source: (From Ballard JL, Khoury JC, Wedig K et al.: New Ballard Score, expanded to include extremely premature infants, *J Pediatr* 119(3):418, 1991.)

BOX 6.1

Tests for Assessing Neuromuscular Maturity in the Newborn

Posture. With infant quiet and in supine position, observe degree of flexion in arms, legs. Muscle tone and degree of flexion

increase with maturity. Full flexion of the arms, legs = 4.

Square window. With thumb supporting back of arm below wrist, apply gentle pressure with index and third fingers on dorsum of hand without rotating infant's wrist. Measure angle between base of thumb and forearm. Full flexion (hand lies flat on ventral surface of forearm) = 4.

Arm recoil. With infant supine, fully flex forearms on upper arms, hold for 5 s; pull down on hands to fully extend and rapidly release arms. Observe rapidity and intensity of recoil to state of flexion. A brisk return to full flexion = 4.

Popliteal angle. With infant supine and pelvis flat on firm surface, flex lower leg on thigh, then flex thigh on abdomen. While holding knee with thumb and index finger, extend lower leg with index finger of other hand. Measure degree of angle behind knee (popliteal angle). Angle of <90 degrees = 5.

Scarf sign. With infant supine, support head in midline with one hand; use other hand to pull infant's arm across shoulder so that infant's hand touches shoulder. Determine location of elbow in relation to midline. Elbow does not reach midline = 4.

Heel to ear. With infant supine and pelvis flat on firm surface, pull foot as far as possible toward ear on same side. Measure distance of foot from ear and degree of knee flexion (same as popliteal angle). Knees flexed with popliteal angle of <90 degrees = 4.

Data from Hockenberry MJ, Wilson D: *Wong's essentials of pediatric nursing*, ed 10, St. Louis, 2016, Mosby; Figures from Brozanski BS, et al: Neonatology. In Zitelli BJ, McIntire SC, Nowalk AJ, editors: *Zitelli and Davis' Atlas of Pediatric Physical Diagnosis*, ed 6, St. Louis, 2012, Mosby.

Congruence between growth measures and maturity is an important consideration in newborn assessment. When a newborn demonstrates expected growth at a given gestational age (if within 2 standard deviations [SDs] from the mean),[5] the infant is classified as *appropriate for gestational age* (AGA). Infants whose growth exceeds standards for a given gestational age are considered *large for gestational age* (LGA), and conversely those whose growth is less than the standard for a given gestational age are classified as *small for gestational age* (SGA).[5, 6]

Newborns are also classified by birth weight, and the terms *normal birth weight*, *macrosomic*, *low birth weight* (LBW), *very low birth weight* (VLBW), and *extremely low birth weight* (ELBW) are applied. Tables 6.3 and 6.4 present newborn weight and gestational age classifications. The etiologies of gestational age at birth and birth weight vary and may include maternal, placental, or fetal influences that can impact successful transition. Each of the gestational age and birth weight subgroups, especially at extremes, carries potential for neonatal complications.

TABLE 6.3
Classifications of Newborn By Weight

Gestational Age	Expected Growth Parameters
Appropriate for gestational age (AGA)	Growth parameters for gestational age between 10th and 90th percentile
Small for gestational age (SGA)	Weight below 10th percentile for gestational age
Large for gestational age (LGA)	Weight above 90th percentile for gestational age
Normal term birth weight	Weight between 10th and 90th percentile
Low birth weight (LBW)	Weight below 2.5 kg
Very low birth weight (VLBW)	Weight below 1.5 kg
Extremely low birth weight (ELBW)	Weight below 1 kg

Data from Hummel P: Newborn assessment. In Chiocca EM, editor: *Advanced Pediatric Assessment*, ed 2. Philadelphia, PA, 2013, Lippincott Williams & Wilkins, pp 199–229; Grossman S, Porth CM: *Porth's Pathophysiology: Concepts of Altered Health States,* ed 9, Philadelphia, 2014, Wolters Klumer/Lippincott Williams & Wilkins; and Gomella TE: *Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs,* ed 7. Philadelphia, PA, 2013, Lippincott Williams & Wilkins.

TABLE 6.4
Classification of Newborns By Gestational Age

Gestational Age	Completed Post-Menstrual Weeks
Postterm	≥ 42 weeks
Term	Completed > 37 weeks
Preterm	Born prior to 37 completed weeks
Late preterm	Born between 34 weeks 0/7 days and 36 weeks 6/7 days

Data from Smith JR, Carley A: Common neonatal complications. In Simpson KR, Creehan PA, editors: *Perinatal Nursing,* ed 4. Philadelphia, PA, 2014, Wolters Kluwer-Lippincott Williams & Wilkins, pp 662–698; Gomella TE: *Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs,* ed 7. Philadelphia, PA, 2013, Lippincott Williams & Wilkins; and Trotter CW: Gestational age assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn*, ed 5. Petaluma, CA, 2015, NICU INC, pp 23–43.

Low birth weight infants

LBW infants are those infants born weighing less than 2500 g. VLBW infants are those infants born weighing less than 1500 g. at birth, and infants weighing ≤1000 g are considered ELBW.[6] Factors that contribute to the risk for LBW include race and ethnicity, maternal age, maternal health and nutrition, substance abuse during pregnancy, environmental toxins, and access to prenatal care.[8] Box 6.2 presents maternal risk factors for LBW and VLBW infants.

BOX 6.2

Maternal Risk Factors for Low Birth Weight and Very Low Birth Weight Infants

Maternal risks

- Maternal age less than 16 or greater than 35 years of age
- Race and ethnicity
- Maternal chronic health conditions
- Maternal medications
- Nutritional status
- Environmental toxins or occupational chemical toxins
- Access to prenatal care

Maternal substance abuse

- Alcoholism
- Tobacco use
- Illicit drug use

- Over-the-counter drug use

Data from LaBronte KH: Recording and evaluating the neonatal history. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination*. Petaluma, 2015, NICU INC.

Small for gestational age

Infants with birth weight below the 10th percentile for age are considered SGA.[6] The head may be *microcephalic*, or small in proportion to the body, and *head circumference* may be below the fifth percentile for age. Fetal growth restriction may be symmetrical with all growth parameters smaller than expected, or asymmetrical with head sparing. The etiology of fetal growth abnormalities varies. Symmetric growth restriction may be due to intrinsic fetal conditions such as chromosomal abnormalities, whereas asymmetric growth restriction is often due to impaired placental perfusion or nutrient deprivation during fetal development.[6] With adequate nutrition, SGA infants experience overall catch-up growth. *Head circumference* is normally the first growth parameter to show catch-up growth, followed by weight and then length.

Newborns with intrauterine growth restriction (IUGR) have restricted fetal weight, length, or head circumference (*occipitofrontal head circumference* [OFC]) due to a pathophysiologic process in utero (Box 6.3).[6] IUGR infants are not necessarily SGA because an IUGR infant may be below expected weight but not below the 10th percentile for age.

BOX 6.3

Maternal Risk Factors for Intrauterine Growth Retardation

Maternal factors

- Maternal hypertension
- Maternal diabetes—poorly controlled

- Autoimmune disease
- Cardiac or respiratory disease
- Maternal infection
- Maternal chronic health conditions

Placental factors

- Abnormal cord insertion or cord abnormalities
- Abnormal placentation
- Placental abruption

Fetal factors

- Multiple gestations
- Chromosomal abnormalities
- Fetal infection

Data from Gabbe, S: Intrauterine growth restriction. In Gabbe S, editor: *Obstetrics: Normal and Problem Pregnancies*, ed 6, Philadelphia, 2012, Saunders.

Large for gestational age

LGA is defined as birth weight greater than the 90th percentile for age.[6] The increased weight may result from fetal factors, which include genetic and chromosomal disorders, or from maternal factors, such as obesity or diabetes. *Maternal hyperglycemia* exposes the fetus to increased levels of glucose, which increases fetal insulin secretion. Increased insulin levels increase fat deposits in the fetus and often result in *macrosomia*, birth weight greater than 4000 g.[4] Diabetic mothers who are insulin dependent and in poor control during the early trimesters of pregnancy have characteristically large infants (Fig. 6.4). LGA infants are at risk for birth trauma, neonatal asphyxia, hypoglycemia, polycythemia, and hyperbilirubinemia.[4]

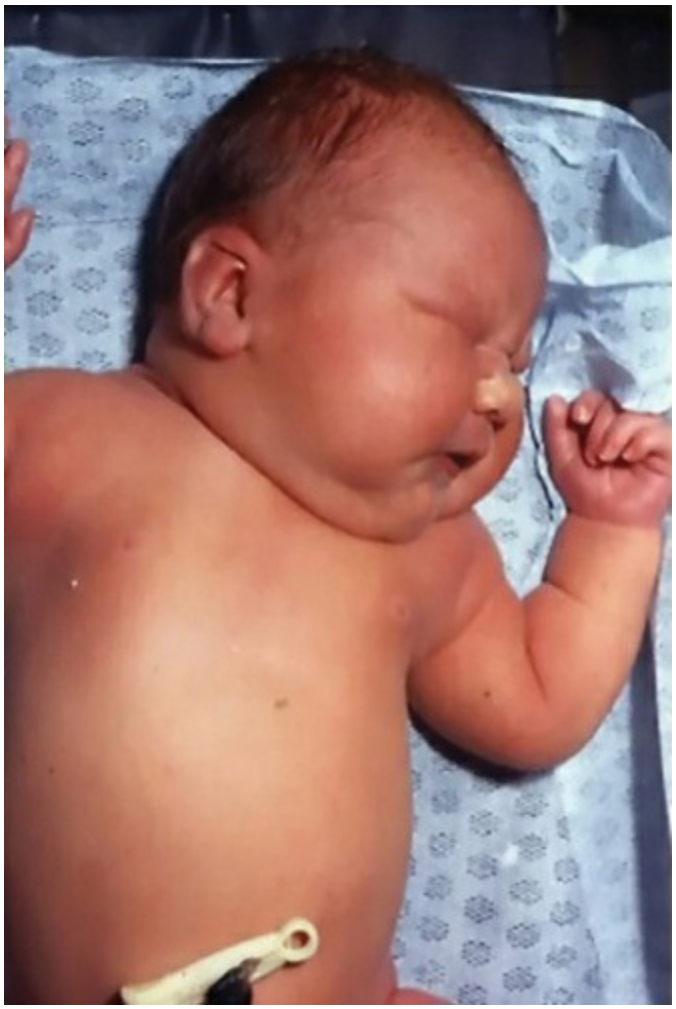


FIGURE 6.4 Macrosomic infant of diabetic mother. Source: (From Meur S: Infant outcomes following diabetic pregnancies. *Paediatr Child Health (Oxford)* 17(6): 217–222, 2007.)

Assessment of growth parameters

The assessment of the newborn begins with an evaluation of the stage of maturity and growth parameters. Measures normally included in the newborn assessment are weight, length, and the OFC. Table 6.5 shows the reference values for common growth measures in term infants. The abdominal and chest circumference are not typically included in the regular newborn examination in the absence of suspected clinical concerns.

TABLE 6.5

Term Newborn Growth Parameters

Measurement	Normal Range
OFC	32–38 cm[2,7] Head approaches half the total body length
Length	44–55 cm[7]
Weight	2500–3900 g[2]
Chest circumference	30–36 cm (~ 1–3 cm < OFC)[3,7] Average 2 cm less than OFC
Abdominal circumference	32–36 weeks gestation equal to OFC After 36 weeks gestation, greater than OFC

Data from Hummel P: Newborn assessment. In Chiocca EM, editor: *Advanced Pediatric Assessment*, ed 2. Philadelphia, PA, 2013, Lippincott Williams & Wilkins, pp 199–229; Fraser D: Chest and lungs assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination,* ed 5. Petaluma, CA, 2015, NICU INK, pp 79–91; and Gooding JR, McClead RE: Initial assessment and management of the newborn. *Pediatr Clin North Am* 62(2):345–365, 2015.

OFC, *Occipitofrontal head circumference*.

For most accurate assessment of these measurements and subsequent plotting on a reference growth chart, the following is recommended:

- Length: measured as heel to crown with infant lying on flat surface[2]
- Head circumference: measured with tape just above the brow line to the occipital prominence. Note that transient head molding or overlapping of the cranial bones may affect this measurement.[9]
- Weight: obtained using a standardized scale; with infant unclothed or standardized measurement of diaper. Note that the newborn can lose up to 10% of birth weight during the first 72 hours following birth, due to contraction of the extracellular space.[9]

The current recommendation in the United States is to use the WHO growth record for infants up to 2 years of age.[7]

Physical assessment

Preparation for the examination

Preparation and examination of the infant should ideally be in a well-lit room, while the infant is calm, and ensure that you are

applying warm hands and warmed equipment.[10, 11] The infant should be weighed on an appropriately calibrated weight scale, height measurement should be obtained on a firm surface, and flexible tape measure used for head circumference. See Chapter 2 for further discussion on accurate measurement. It is best to use a pediatric or neonatal stethoscope for auscultation, and an otoscope and ophthalmoscope is needed for the inspection of the ears and eyes.

The newborn assessment uses multiple examination techniques, most commonly inspection, auscultation, and palpation.[9] Percussion and transillumination may be helpful in assessing certain organ systems (e.g., percussion with liver assessment) or to better evaluate some findings (e.g., transillumination for suspected hydrocele).[10]

Approach to physical examination

The best approach to physical examination of the newborn is to approach the infant in the least disruptive manner and consider the infant's behavioral state to obtain the best results. Newborn behavioral states include: two sleep states, deep/quiet sleep, light/active sleep, as well as four awake states: drowsy, quiet alert, active alert, and crying. The examination should be conducted in a systematic manner because a consistent approach ensures that all aspects are evaluated. The provider also acknowledges that components of the exam itself (e.g., eye or kidney exam) may disturb the infant, and the unclothed infant can easily become cold stressed. Prior to touching the infant, much can be learned by observing the appearance of the undressed infant in the resting, nonstimulated state. General inspection should include determination of gender, identification of any deformities, determination of fetal nutrition, assessment of color, and position and movement.

The sequence of the physical assessment begins with the quiet parts of the examination—the cardiac and respiratory systems. The overall sequence of the exam may vary with the infant's behavioral state but may then proceed systematically from head to toe and may integrate inspection of multiple systems to take best advantage of infant's behavior and tolerance of the examination. Each body

system has relevant embryology, development, anatomic, and physiologic variations, and the pediatric health care provider is directed to the relevant system chapters in the text.

Skin

The term newborn's skin is structurally similar to the adult. However, the immature infant's skin is thinner, friable and permeable, and more easily disrupted. The skin is assessed with the techniques of inspection and palpation.[9] Although most skin findings are evident with inspection, palpation will allow additional characteristics, such as skin thickness and turgor, to be determined.[12] Be sure to assess the entire skin surface including skin folds and scalp for subtle findings.[9] The newborn's skin is typically covered with vernix, a cheesy-white odorless protective sebaceous secretion that may collect in the skin folds. Skin pigmentation varies with ethnic origin and is highly variable.

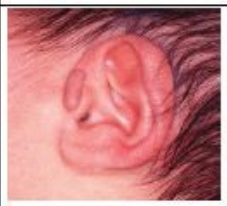
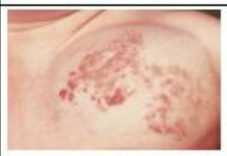
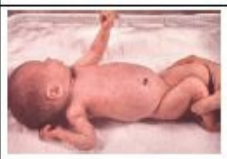
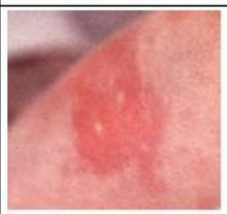
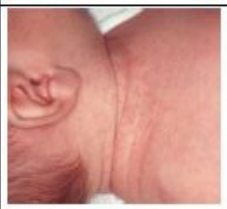
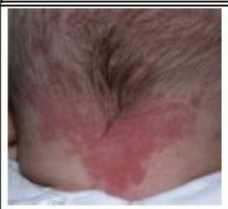
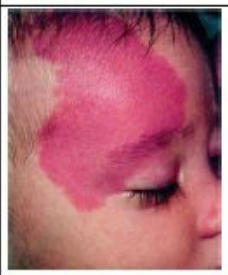
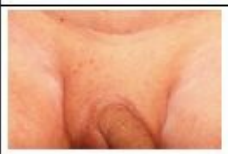
One essential skin assessment to make in the newborn is color, and the most reliable color indicator in the newborn is the mucous membranes and tongue. Newborn infants commonly exhibit *acrocyanosis*, cool and cyanotic hands and feet due to vasomotor instability of peripheral circulation; this is a normal finding in the otherwise healthy infant.[9] Persistence of this finding beyond the first 24 to 48 hours may indicate poor perfusion due to cold stress, hypovolemia, or polycythemia. *Central cyanosis* is always considered abnormal and is evident when less than 5 g percent of desaturated hemoglobin is present. There may be associated etiologies such as cardiac or pulmonary disease, infection, metabolic and neurologic conditions, and hematologic issues such as anemia or polycythemia. *Pallor*, a pale or ashen appearance, can be due to poor perfusion, acidosis, or anemia. *Plethora,* a ruddy appearance, may indicate polycythemia. Both anemia and polycythemia may be associated with hypoxemia, although the appearance of cyanosis may be misleading. Because cyanosis becomes evident when there is 3 to 5 g of desaturated hemoglobin, cyanosis may present readily in polycythemic infants with otherwise normal oxygenation, whereas its presence with anemia requires substantial hypoxemia and low oxygen content of the blood.

Jaundice, a yellow discoloration of the skin, may occur from deposition of bilirubin. Clinical jaundice is a common newborn finding, occurring in up to 60% of healthy term infants during the first week of life. Jaundice typically follows a cephalocaudal distribution and is initially seen in the face and upper trunk. However, exaggerated clinical jaundice with associated hyperbilirubinemia in the newborn may also result from hemolysis, and jaundice occurring in the first 24 hours of life is considered pathologic and warrants investigation.[9, 11-13]

The newborn skin assessment may reveal lesions or rashes, most benign in nature and requiring no intervention (Table 6.6). *Ecchymoses* may be evident over presenting parts, such as the head or buttocks, or occur related to use of assistive devices such as forceps for delivery. *Milia* are pearly white papules typically found on the nose, brows, and cheeks. These epidermal cystic lesions are the result of accumulated sebaceous gland secretions and resolve spontaneously. *Erythema toxicum* occurs in nearly three-quarters of newborns and presents at approximately 24 to 48 hours as small yellowish-white papular lesions with erythematous bases on the face and trunk. The papules may persist intermittently until 2 weeks of age. Neither of these transient skin lesions requires intervention.

TABLE 6.6

Common Conditions in Newborn and Infant Skin

Condition	Photo	Description	Significance/Treatment
Acrocyanosis		Bluish coloration of hands and feet present at birth; may persist up to 24 h; circumoral cyanosis also may be present	Benign color variation in newborn; no treatment needed if gone after 24 h
Accessory tragi		Pedunculated, flesh-colored, soft, round papules usually arising on or near the tragus	May occur anywhere from corner of ear to mouth and require removal by careful surgical dissection; do not confuse with skin tags, do not tie off with suture
Cavernous hemangiomas		A raised, soft, lobulated bluish red tumor with irregular borders and involves the dermis and subcutaneous dermis layers of skin	Increases in size from 6–12 months and then involutes spontaneously
Cutis marmorata		Reddish-blue mottling or marbling of skin in response to changes in temperature; caused by dilation of capillaries and venules	Benign color variation; no treatment needed unless it does not disappear with skin warming
Erythema toxicum		Small white to yellow papules, vesicles with erythematous base; occurs in response to rubbing; starts as early as 24 h of life, may continue until 2 weeks old	Common benign skin lesion in newborn; eosinophils in smear from papule confirms diagnosis
Miliaria rubra, m. crystallina, m. pustulosa		Clear, thin vesicles or discrete erythematous papules seen primarily over forehead, neck, or groin or in creases; occurs as a result of obstructed sweat glands in humid environment	Benign skin lesion in newborn; can be treated by eliminating precipitating factors such as heat, humidity, too many clothes
Nevus simplex, Nevus flammeus		Red to purple macule with irregular borders often over the upper eyelids, bridge of nose and upper lip, increases with crying; when occurs over nape of neck called stork bite	Increases until 6 months of age and then involutes spontaneously
Sturge-Weber, Port Wine nevi		Purplish red lesion with clearly defined borders under the epidermis generally on face; usually unilateral; may be located over trigeminal nerve	Does not resolve spontaneously and size remains stable; may be associated with Sturge-Weber syndrome
Transient neonatal pustular melanosis		Vesicles that rupture leaving collaret of scale and pigmented macule; macules may remain for up to 3 months after birth	Benign skin lesion requiring no treatment

Unn Fig 6-3 Cavernous hemangiomas. From *Dermatopathology: High-Yeild*

Pathology, Saunders, 2011. Fig 1. **Unn Fig 6-7 Nevus Simplex**. Dermatology Essentials. Bolognia, Jean L., MD; Schaffer, Julie V., MD . . . Show all.; Duncan, Karynne O., MD; Ko, Christine J., MD . . . Published January 1, 2014. Pages 812-833. © 2014. Fig. 85.17 B, Courtesy, Julie V. Schaffer, MD; C, Courtesy, Odile Enjolras, MD. **Unn Fig 6-8 Sturge-Weber, Port Wine nevi**. From Cohen B: Pediatric dermatology, ed 3, Philadelphia, 2005, Mosby, p 49.

Nevi and other hyperpigmented macular lesions may be common findings on the newborn skin. *Blue-black spots*, formerly termed *mongolian spots*, are hyperpigmented macules seen in 90% of Hispanic, Asian, and African American infants, and approximately 10% of white infants. These large lesions are found most commonly over the buttocks but may extend to the back and shoulders. These lesions represent infiltration of the dermis with melanocytes, generally fade over time, but may persist to adulthood.[12] *Nevus simplex* is a common vascular lesion seen in up to 40% of newborns. It is commonly referred to as stork bite or salmon patch, is an irregularly edged macular lesion caused by dilated capillaries, and is most often found at the nape of the neck (stork bite), eyelid, bridge of nose, or lip. Due to the distended underlying capillaries, these lesions blanch with pressure and may increase in size over the first 1 to 2 years of life. *Nevus flammeus*, also known as port-wine nevus, is a flat pink to purple lesion of dilated capillaries with a regular edge. This lesion does not typically increase in size but also does not regress in size spontaneously and may require treatment with laser for elimination or reduction. Most of these lesions are isolated; however, an important variant is the lesion that follows the branches of the trigeminal nerve in the forehead and upper eyelid. This lesion may be associated with Sturge-Weber syndrome, a condition that may result in seizures and other system complications.[9, 11–13]

In addition to hyperpigmented macules, the skin assessment may reveal strawberry or *cavernous hemangiomas* that consist of dilated capillaries and associated endothelial proliferation of dermis and subdermal skin layers. These lesions grow rapidly for the first 6 months and then typically begin to regress spontaneously. Important complications due to their dense capillary network are bleeding, ulceration, and infection. Depending on the location, hemangiomas may also grow in size to the point of obstructing the airway, necessitating treatment. As with *port-wine nevi*, there is an

association of cavernous hemangiomas with syndromes.[12]

Head

Using the techniques of inspection and palpation, the infant's head is initially assessed for size, shape, presence of abnormal hair patterns, scalp defects, unusual lesions or protuberances, lacerations, and abrasions or contusions. Measurement of head circumference is performed and contrasted with anticipated norms (see Chapter 2, Fig. 2.2). The head size is considered normal, *microcephalic* (<2 SD or less than the 10th percentile), or *macrocephalic* (>2 SD or greater than the 90th percentile) compared with established norms. Note that macrocephaly or microcephaly can be associated with other pediatric chromosomal and developmental conditions.[14] The scalp is examined for bruising or other disruptions, such as lacerations, that might have occurred during delivery. Due to the influences of the birth process, there may be transient deformation in head shape and size. Table 6.7 and Fig. 6.5 presents some transient deformations of the head in newborns. (See Chapter 10 for further assessment of the head.)

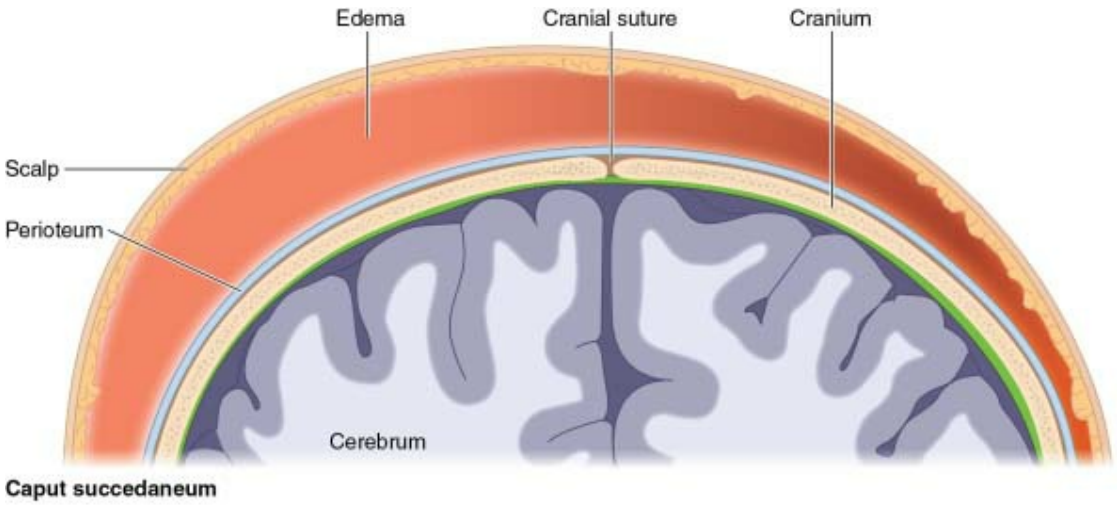


FIGURE 6.5 Caput succedaneum.

TABLE 6.7

Head Assessment—Common Findings

Condition	Description
Caput Succedaneum	Diffuse, soft swelling superior to cranial bones; common finding due to head compression during delivery that will resolve without intervention; not confined by suture lines and is maximally evident at birth. Does not require intervention[4, 12, 13]
Cephalhematoma	Subperiosteal collection of fluid or blood primarily associated with assisted deliveries; occurs in approximately 2% of deliveries; distinguished on exam as a soft swelling that can increase in size over the first 24–72 h but remains confined by the suture lines. Does not typically require intervention (but may be associated with skull fracture in up to 5% of cases)[4, 12, 13]
Cranial Molding	Temporary skull asymmetry due to compression during delivery; common with prolonged labor; rarely associated with other anomalies; resolved without intervention[4]
Subgaleal Hemorrhage	Subaponeurotic blood collection; occurs in 1.5/30,000 births; associated with mechanically assisted deliveries or coagulopathy; distinguished on exam as boggy, shifting mass that extends over cranial surface to neck and behind ears; may increase substantially in size; need for intervention varies with degree of blood loss into potential space[4, 12]

Data from Grossman S, Porth CM: *Porth's Pathophysiology: Concepts of Altered Health States*, ed 9, Philadelphia, 2014, Wolters Klumer/Lippincott Williams & Wilkins; Witt C: Skin assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination,* ed 5. Petaluma, CA, 2015, NICU INK, pp 45–59; Vargo L: Cardiovascular assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination,* ed 5, Petaluma, CA, 2015, NICU INK, pp 93–110; and Johnson PJ: Head, eyes, ears, nose, mouth, and neck assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination,* ed 5. Petaluma, CA, 2015, NICU INK, pp 1–8.

One important cause of external cranial swelling is a *subgaleal hemorrhage*.[5, 14-16] This uncommon complication, estimated at 1.5 to 30 per 10,000 births,[16] represents venous bleeding between the aponeurosis and the periosteum and can increase considerably in size over the first hours to days of life (Fig. 6.6). Subgaleal hemorrhage has an association with mechanically assisted deliveries, due to external shearing forces.[15, 16] The clinical presentation of subgaleal hemorrhage is a large boggy, shifting collection of fluid over the cranial surface unrestricted by the suture lines. The swelling may extend to the neck and behind the ears, lifting the ears forward. Because this subaponeurotic space is large, infants may quickly develop hypovolemia as a complication and must be vigilantly watched for this, as well as development of hyperbilirubinemia after red blood cell breakdown occurs.[5, 16]

However, infants may remain asymptomatic, and the stable infant with a subgaleal hemorrhage may be observed clinically for 24 to 48 hours. Outcomes for infants who remain hemodynamically stable are good.[16]

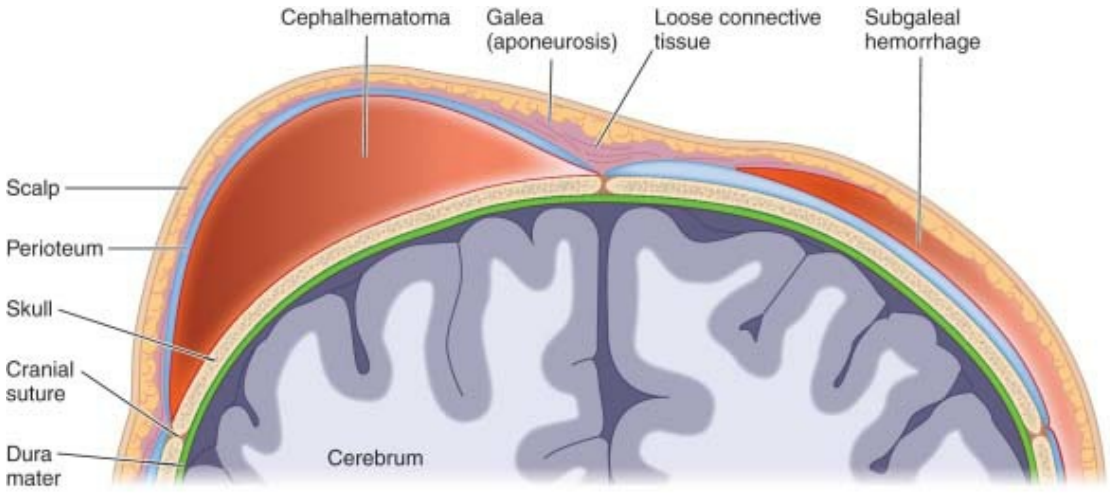


FIGURE 6.6 Subgaleal hemorrhage and cephalhematoma.

The cranial bones are typically approximated but not fully fused until adolescence. This allows for some overlapping to occur during vaginal delivery or expansion to occur in the case of elevated intracranial pressure. There should be some slight movement appreciated at the level of the suture lines. *Overlapping sutures* can occur during vaginal birth in the term infant (Fig. 6.7). *Craniotabes*, a softening of the cranial bones, may be evident as a ping-pong ball sensation when pressure is exerted along the parietal or occipital margins. This is often a finding in preterm infants related to incomplete ossification and also in breech deliveries in term infants due to pressure during delivery. In term infants, it generally disappears within a few weeks.[9]

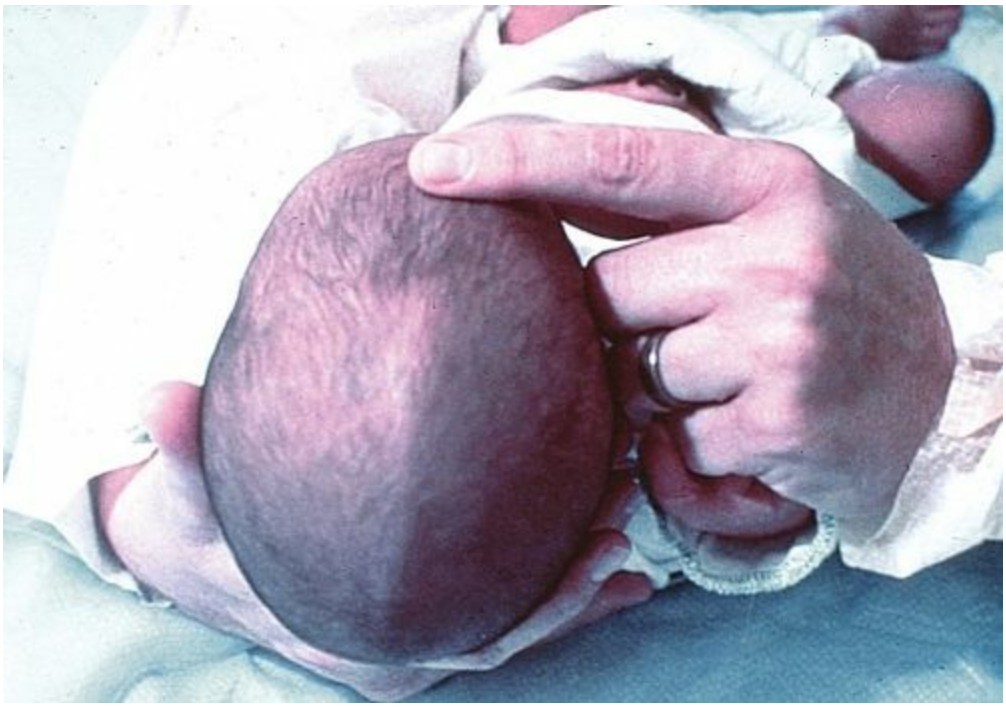


FIGURE 6.7 Newborn with overlapping sutures.

The fontanels are evaluated for size, shape, and fullness. The diamond-shaped *anterior fontanelle*, at the juncture of the frontal and parietal bone, varies in size from 1 to 4 cm and typically closes by 18 months of age. The triangular-shaped *posterior fontanelle*, at the juncture of the single occipital and parietal bones, may be dimple sized at birth, typically closes by 4 months, and is no longer appreciable by 1 year of age (see Chapter 10, Fig. 10.2).[9] The hair is assessed for distribution, texture, amount and presence of whorls, or hypopigmentation.[9]

Face

The facies are inspected for symmetry, gross presence of anomalies or deformation, and movement during the transition between infant behavioral states. Transient distortions can occur due to compression during delivery, prolonged delivery, and the use of forceps; these will typically resolve in a few days without intervention. Asymmetry of facies during crying, or inability to close the eye may be an indication of nerve injury occurring during delivery or from absence of musculature controlling facial movements. Typically the infant will have decreased movement on the ipsilateral side.[5, 7]

Eyes

Assessment of the eyes in the newborn is achieved by inspection, palpation, and selected use of the ophthalmoscope. Determine eye spacing and the intracanthal distance between the eyes. A narrower than normal distance between the eyes is known as *hypotelorism*, and wide-set eyes, *hypertelorism*, may be associated with other facial and systemic anomalies. The eyes are also assessed for slanting, determined by the relationship of the outer canthus with the helix of the ear. Upward or downward slanting of the eyes may be associated with congenital anomalies or syndromes.[9, 14]

The color of the newborn's iris varies, from gray to brown to dark blue; actual eye color becomes permanent at approximately 6 months of age.[9] The sclera is blue-white but may commonly have associated *subconjunctival hemorrhages* resulting from ruptured capillaries during delivery. Yellow sclera suggests hyperbilirubinemia and should be evaluated further. Tears are typically absent in the newborn due to incomplete lacrimal duct patency for the first few months of life.

The initial examination of the newborn's eyes may be difficult to perform because the eyelids often are edematous after delivery. Most infants will open their eyes spontaneously when held vertically in an environment with low ambient light. Pupillary reactivity can be evaluated in a dark room using a penlight; pupils are expectedly equal in size and reactive to light. The *red light reflex* or *retinal light reflex* should be present, indicating an intact cornea and lens. Note that in the infant, the retinal light reflex may vary from pale to slightly yellow depending on the racial/ethnic variability. Unequal or white reflexes are abnormal and may indicate cataracts or other opacity.[9] Chapter 12 provides additional detail about the eye examination in the infant.

Ears

The newborn ear exam applies the techniques of inspection and palpation.[9] The term newborn demonstrates ready recoil when the pinna is folded inward. The ears are assessed for positioning, presence of deformities, swelling, bruising, pits, dimples, and tags. Malformed ears may be associated with other abnormalities, including chromosomal anomalies and congenital syndromes.

Uncommonly, preauricular or postauricular pits and tags may be associated with deafness or renal defects, although more often are inconsequential unless another anomaly is detected.[9] The external auditory canals are grossly inspected for patency. The pinna is typically situated at or above an imaginary line from the inner to outer canthus of the eye; those ears situated below this line are considered low set. The tympanic membranes are not typically examined in the newborn due to difficulty visualizing as a consequence of short, collapsible canals and canals obscured with vernix.[9, 10] However, patency of the external canal should be determined during the newborn assessment. Hearing acuity can be determined by evaluation of cochlear response to or transmission of sound (Table 6.8).[5]

TABLE 6.8

Common Newborn Diagnostic Screening Tests

Screening	Diagnostic Test
Hearing screening	Universal screening recommended (but not mandated) prior to hospital discharge; testing done by either automated auditory brainstem response (ABR) or otoacoustic emission (OAE); ABR tests cochlear response to sound and auditory pathways; OAE tests cochlear transmission of sound[4, 7]
Screening for critical congenital heart disease	Universal screening recommended prior to discharge and after 24 h of age; testing done by pulse oximetry determination of oxygen saturation simultaneously in upper and lower extremity. Positive result warranting additional evaluations: saturation <90%; saturation 90%–95% on repeat measurements, saturation >3% difference between upper and lower sites on repeat measurements[4]
Newborn screening	Mandated state-administered blood screening for identification an early treatment of genetic and metabolic conditions (including amino acid metabolism disorders, organic acid metabolism disorders, fatty acid oxidation disorders, hemoglobinopathies, and select other disorders). Screening for 29 conditions required in all states; others optional state by state. Test performed 48 h after birth[2, 4]

Data from Hummel P: Newborn assessment. In Chiocca EM, editor: *Advanced Pediatric Assessment*, ed 2. Philadelphia, PA, 2013, Lippincott Williams & Wilkins, pp 199–229; Grossman S, Porth CM: *Porth's Pathophysiology: Concepts of Altered Health States,* ed 9. Philadelphia, 2014, Wolters Klumer/Lippincott Williams & Wilkins; and Gooding JR, McClead RE: Initial assessment and management of the newborn, *Pediatr Clin North Am* 62(2):345–365, 2015.

Nose

The nares are inspected grossly for patency and symmetry. Patency can be assessed noninvasively by listening with a stethoscope at the level of the nares or suspending a thin cotton wisp under the nare and looking for rhythmic movement of the wisp. Attention is paid to not allowing the stethoscope or wisp to touch the nare directly, and the other nare should be occluded at the time of assessment. If the results of either of these tests is inconclusive, but discontinuity of the passage suspected, a small catheter can be introduced into the nares to the nasopharynx.[9, 14]

Mouth

The mouth, lips, gums, and palate are inspected and palpated. Assessment of the interior of the mouth includes examination of the gingiva, tongue, palate, and uvula. *Cleft lip* may be observed unilaterally or bilaterally; unilateral cleft lip primarily occurs left sided. A bilateral cleft lip is most commonly associated with cleft palate as well.[5, 14] Natal teeth that erupt prior to the first month of life may be present, typically as central incisors. These are similar to normal teeth but typically lack a developed root system and may fall out. Unless there is concern for aspiration or interference with feeding, there is no indication for removal.[5] The sublingual frenulum should be inspected when the infant cries to determine position of attachment. If the sublingual frenulum is attached to the anterior portion or tip of the tongue, *ankyloglossia* or tongue-tied, this may interfere with infant feeding and require treatment.[14]

Neck

The techniques of inspection and palpation are used to determine suppleness, symmetry, and range of motion and to detect any extraneous tissue or masses. The examiner assesses for the presence of cysts, clefts, dimples, masses, and redundant skin that may suggest a syndrome. The neck is manipulated side to side; limitations of full range of motion or head tilt may indicate *congenital torticollis*.[7]

Chest

The chest is inspected and palpated for size, shape, symmetry, and

contour. If measured, the chest circumference at the nipple line is approximately 2 to 3 cm less than the head circumference.[9, 13] There should be symmetric movement with respiratory effort. Due to the compliant newborn chest (an advantage during delivery), the sternum is drawn inward during inspiration. The clavicles are inspected and palpated to detect any swelling, tenderness, or crepitus. Any decreased movement or asymmetry of the Moro reflex on the affected side may suggest clavicular fracture. Clavicular fractures, for example, are a common birth injury, occurring in 0.2%–3.5% of births. These bony disruptions are evident as crepitus palpable in the clavicular region and may be confirmed by radiographic imaging. No treatment is indicated.[5]

The xyphoid process may be evident protruding at the distal end of the sternum. The measured spacing between the nipples is typically one-half of the anteroposterior diameter of the chest. The chest is inspected for presence of *supernumerary nipples*. Use of accessory muscles, evident as retractions, may indicate inability to sustain adequate inflation and may be a finding of pulmonary disease that needs further evaluation. The soft nature of the newborn's bony structures increases risks for disruptions.[3] Chest deviations may include *pectus excavatum*, an inverted sternum that is usually not medically significant, and *pectus carinatum*, a protruding sternum that occurs predominantly in males 4:1 and can be associated with other abnormalities, including scoliosis, Marfan syndrome, and congenital heart disease. [2, 3]

Lungs

The lungs are auscultated for presence of bilateral, equal breath sounds. During the first postnatal hours, due to residual presence of alveolar fluid, the breath sounds may be moist. Respirations are typically "regularly irregular," with short periods of rapid rate followed by short pauses. However, pauses longer than 20 seconds are abnormal and warrant additional evaluation to rule out pulmonary disease or other disorders of transition.[2, 3] Normal respiratory rate is presented in Table 6.9.

TABLE 6.9

Normal Range of Newborn Respiratory and Cardiac Rates

Heart Rate	Respiratory Rate	Blood Pressure
80–180 beats/min (70–80 during sleep; 120–160 awake)[4]	30–60 breaths/min[3]	57–69 systolic 44–52 diastolic[4]

Data from Fraser D: Chest and lungs assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn: A Comprehensive Approach to the Art of Physical Examination,* ed 5. Petaluma, CA, 2015, NICU INK, pp 79–91; and Grossman S, Porth CM: *Porth's Pathophysiology: Concepts of Altered Health States,* ed 9, Philadelphia, 2014, Wolters Klumer/Lippincott Williams & Wilkins.

Heart

The cardiac examination commences with an inspection of the chest and palpation to determine the point of maximum impulse (PMI) that will shift following the first hours after birth as elevated fetal right-sided heart forces diminish once pulmonary ventilation is established. Heart sounds are auscultated, and determination of S1 and S2 and any adventitious sounds. Murmurs are common in the early newborn period due to changing cardiopulmonary dynamics postnatally. For example, a *patent ductus arteriosus* may be evident as a murmur best heard along the left lower sternal border. This sound typically decreases by 48 hours of age and is not associated with any other evidence of cardiac instability.[9, 13] Normal cardiac rates are presented in Table 6.10. Chapter 8 reviews innocent or physiologic murmurs that may be present during infancy.

TABLE 6.10
Common Newborn Symptoms and Conditions

Presentation	Signs and Symptoms
Transient tachypnea of newborn	Excessively rapid respirations usually due to retained fetal lung fluid; tachypnea subsides with absorption of fluid
Acrocyanosis and circumoral cyanosis	Bluish discoloration of the surrounding the lips and of the hands and feet due to changes in newborn temperature
Transient jitteriness	Rapid movements of newborn's arms and legs often with vigorous crying generally in response to stimuli; does not persist after early neonatal period and does not involve eye movements

The cardiac exam also includes an assessment of pulses, including brachial and radial in the upper extremities and femoral

and pedal in the lower extremities. Perfusion is determined by blanching the skin over the abdomen or chest and allowing for return of color; a capillary filling time less than 3 seconds is considered within normal limits.[9, 13] Although norms exist for newborn blood pressure, it is not a typical component of the routine newborn exam.[9]

Abdomen

The abdomen is inspected for contour and palpated for normal presence of internal organs and abnormal presence of masses. The abdominal circumference is measured just superior to the umbilicus at the largest diameter; it is approximately equal to head circumference by term.[17] The newborn abdomen is rounded and soft. Use light and deep palpation in a circular area examining the four regions in sequence. Bowel sounds are evident within several hours postnatally, as swallowed air progresses distally. *Diastasis recti*, a separation of the abdominal musculature, may be evident as a midline bulge extending from the xiphoid process to the umbilicus. This is due to failed fusion of the muscle and will resolve without intervention. The umbilicus is inspected for color and state of healing, as well as the presence of vessels. Typically, the cord remnant is opaque, blue-white, and odorless with evidence of two thick-walled arteries and one thin-walled vein. The presence of swelling or masses is noted because this may represent a herniation of abdominal contents.[9, 13]

The liver is palpated as an upper right to midline region organ; the newborn liver may extend down 1 to 3 cm below the right costal margin.[9] An alternative technique to direct palpation is to use the "scratch" technique and listen indirectly with a stethoscope to detect a change in percussion note. The spleen is also palpated, although typically not appreciated on the normal exam. Kidneys in the newborn are palpated; they are expectedly smooth, round, and approximately 4 to 5 cm in size. A bimanual technique is the easiest method for assessing the kidneys, approached with deep palpation at a 45-degree angle just below and lateral to the umbilicus.[9, 17]

Genital and rectal

The perianal area is assessed for the presence of a patent anus. Most

newborns will spontaneously pass stool within the first 48 hours of life and void within the first 24 hours of life.[9] Failure to stool beyond this time may indicate obstruction and is an indication for further assessment. The femoral area is inspected and palpated. Bruising and swelling of the genitalia may be a common finding following delivery. However, an *inguinal hernia* that represents incomplete obliteration of the *processus vaginalis* may be evident as a compressible mass or swelling and may contain abdominal viscera. When examining the female genitalia, note the clitoris and labia minora are normally covered by the labia majora at term, and the urinary meatus is situated just below the clitoris. *Hymenal tags* are common, and pseudomenses may occur transiently due to the residual effects of maternal hormones. For the male, the average penile length measures 2.5 to 3.5 cm at term, and the urinary meatus is situated just ventral to the tip. Deviations of placement of the meatus include *hypospadias*, a ventral urethral opening, and *epispadias*, a dorsal urethral opening that occurs less commonly are indications for referral. The scrotum is inspected, and rugae as well as color and symmetry of the scrotal sac are assessed. The testes are palpated bilaterally for size, firmness, and tenderness. A *hydrocele* may be evident as a fluid-filled scrotal mass that transilluminates; this will resolve spontaneously (see Chapter 16 for review of male genitourinary conditions).[2, 9]

Musculoskeletal, extremities, and spine

Normal resting position of the term newborn is inward flexion of upper and lower extremities, although postnatal positioning is affected by the lie in utero. The extremities should be assessed for size, shape, range of motion, alignment and symmetry of movement, and presence/number of digits.[9] The skin overlying the spine is inspected for lesions, pits, tags, and hair tufts; the dermis should be continuous without breaks. Abnormal curvature of the spine should be noted. The hips are assessed for stability and an evaluation for *developmental dysplasia* of the hips. The *Barlow maneuver* provokes an unstable hip to dislocate by adducting the hip and applying pressure posteriorly to force the femoral head over the posterior rim of the acetabulum. Conversely, the *Ortolani maneuver* reduces the dislocated hip by abducting the hip and

allowing the femoral head to slip back toward the acetabulum, creating an audible click and palpable sensation to the examiner (see Chapter 19, Fig. 19.15).[2, 5, 18]

Neurologic

The neurologic assessment can be integrated throughout the physical assessment and takes notes of changes in posture, activity, state, or muscle tone during handling. Infants should exhibit changes, especially in response to stimuli. The infant who does not increase alertness when handled or exhibits tremors, clonus, or poorly coordinated activity, such as suck/swallow, deserves careful watching.[2] Observing an infant successfully feeding, at breast or bottle, provides information about intactness of the cranial nerves. See Chapter 20, Table 20.5 for cranial nerve testing in newborn. Assessment of primitive reflexes is included in the newborn exam. Most primitive reflexes generate symmetric responses, and this provides important information about brainstem function. Common reflexes include the Moro (startle) reflex, stepping and placing, rooting and sucking, and plantar and palmar grasping (see Chapter 20, Table 20.3). These reflexes generally disappear by 3 to 4 months postnatally.[15]

Summary of examination

A comprehensive neonatal assessment helps the pediatric provider to anticipate potential problems, detect subtle findings in the newborn that may be meaningful, and intervene readily to prevent or minimize further problems. The assessment should be generally performed within first 24 hours of life to identify any abnormality that would alter the normal newborn course or identify a medical condition that should be addressed.

Consistency in technique and organizational skill, as well as integration of infant behavioral state into the physical exam can help to ensure completeness in the comprehensive assessment. The comprehensive assessment of the newborn integrates components of the prenatal, perinatal, birth and early neonatal history with a thorough physical examination.

Summary of Newborn Physical Examination

Vital signs	Temperature, apical pulse, respirations
Measurements:	Birth weight and discharge weight, length, head circumference, chest circumference if indicated
General	Gestational Age New Ballard Score & Apgar score Inspection & Observation: posture, state of arousal/alertness, activity level, tone, symmetry, self-soothing measures
Skin	Color: presence of jaundice, cyanosis, acrocyanosis, lanugo, vernix caseosa, Skin turgor, amount of subcutaneous tissue Presence of birthmarks or lesions, rashes, bruises, pustules, petechiae
Head	Shape, symmetry, scalp hair Anterior & posterior fontanelles, sutures Evidence of trauma: molding, caput succedaneum, cephalhematoma Face: symmetry, unusual or dysmorphic features
Eyes	Shape, edema, drainage Sclera, conjunctiva: color, scleral hemorrhages Retinal or red light reflex, corneal light reflex, eye movement
Ears	Shape and position of pinna, skin tags, patency of external canal, visualization of tympanic membrane
Nose	Patency, discharge, nasal flaring
Mouth and Throat	Symmetry of movement, cyanosis, intact hard and soft palate Presence of teeth, cysts, Epstein pearls, thrush Size of chin and tongue, position of tongue in mouth Moistness of mucus membranes
Neck	Presence of webbing, masses, range of motion Evidence of fractured clavicle Presence of lymph nodes
Chest	Respiratory rate, rhythm, retractions Shape, size, symmetry of movement Breath sounds Breast: amount of tissue, engorgement, nipples; size, symmetry, discharge
Cardiovascular	Apical pulse location and quality Heart rate, rhythm, identify S1 S2 Presence of murmurs, extra heart sounds Radial, femoral, pedal pulses symmetry, quality
Abdomen	Shape, size, symmetry, presence of bowel sounds Palpate for liver, spleen, kidneys, evidence of diastasis recti Umbilical cord: number of vessels, color, drainage, odor, presence of hernia
Extremities	Symmetry, shape, strength, range of motion, number of digits Nails: texture, color Creases: palms, soles Abduct hips for dislocation, perform Barlow and Ortolani
Back	Spinal contour Presence of cysts, sinuses, dimples, tufts of hair

	Uneven skin folds (check without diaper) Presence of nevi
Rectum	Patency, presence of fissures and innervation with anal wink Passage of meconium
Genitalia	*Genitalia—male* Position of urethral meatus, passage of urine Prepuce covering glans, scrotum Testes–descended or undescended, *Genitalia–female* Size of labia majora, labia minora, vaginal discharge, skin tags Labial masses, passage of urine *Note: Consult for any concern with gender ambiguity*
Neurologic	Posture, muscle tone, movement, seizure activity Head control, quality of cry Response to light and sound Ankle clonus *Primitive Reflexes*
Screening Tests and Labs	Passage of meconium and urine Newborn screening test, newborn hearing screening results, point-of-care glucose testing, complete blood count or hematocrit, blood type, Coombs, bilirubin

NEWBORN HEALTH HISTORY DOCUMENTATION

Newborn 8 hours of age

38 weeks 2 days male infant, BW 3500 gm, AGA, mother 32 y/o $G_3P_{2\text{-}3}A_0L_{3,}$ O pos. Rh neg, Rubella, HepBsAg RPR, GBS, GC/CT—neg., breastfeeding initiated, good suck, intermittent transient tachypnea noted.

BW, birth weight; *AGA,* appropriate gestational age; *GPAL,* gravidity, parity, abortion, live births; *HepBsAg,* hepatitis B surface antigen; *RPR,* rapid plasma regain; *GBS,* Group B Streptococcus; *GC/CT,* Neisseria gonorrhea and Chlamydia trachomatis

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UNIT II

System-specific assessment

OUTLINE

17. Male and female breast
18. Female genitalia
19. Musculoskeletal assessment
20. Neurologic assessment

CHAPTER 7

Skin assessment

Renee P. McLeod

It is easy to underestimate the importance of a thorough examination of the skin. Careful inspection of the skin gives the examiner clear insight into the overall health of the child. Along with inspection and a thorough health history, examination of the skin, hair, and nails provides clues to oxygenation, tissue perfusion, nutritional and hydration status of the child, and any underlying disease pathology or injury.[1] The skin of an infant, child, and adult share similarities in structure and function, but a child's skin reacts differently to environmental demands because of the unique skin properties of each age group. All skin, regardless of age, is affected by seasonal factors such as the heat and humidity of summer or the dryness and low humidity of winter, but the differences in an infant's skin and ability to sweat compared with an adult's skin can create many more problems associated with these seasonal changes. Therefore, the manifestations of a skin disorder observed in an infant or child may vary widely from what may be seen in an adult.[2]

The skin is the largest organ in the body and has five distinct functions. The skin controls fluids, regulates temperature, protects against invasion from microbial and foreign bodies, and protects against damage from the ultraviolet (UV) rays of the sun. Finally, the skin is an organ of communication. Touch and skin-to-skin contact is one of the ways we bond with our mothers and families at birth and later bond with our sexual partners. Research conducted over the past 50 years has proven that touch is more

important to humans than food in regard to optimal development.[3-5] Having a disease of the skin, hair, or nails that prevents or decreases human touch can be devastating to a child's self-esteem.

Anatomy and physiology

The skin consists of three layers: the *epidermis*, the *dermis*, and the *subcutaneous layer* (Fig. 7.1). The *epidermis* is the outermost layer of the skin and consists of two main layers: the *stratum corneum* and the *cellular stratum.* The *stratum corneum* is the very top layer of the skin and is composed of stacked, overlapping nonnucleated keratinized cells called *corneocytes.* The thickness of this layer depends on the region of the body, being thinnest on the face and thickest over the soles of the feet.[2] This layer forms the protective barrier of the skin and contains the waterproofing protein *keratin,* which restricts water loss and penetration of a variety of substances through the skin. The innermost layer of the epidermis consists of a single row of columnar cells called *basal cells,* which reside in the *stratum basale.* These cells divide to form the *keratinocytes* that move to the surface through the *stratum spinosum*, *stratum granulosum*, and *stratum lucidum* to replace the cells that are sloughed off every day in the stratum corneum.[2, 6] The stratum basale also contains *melanocytes*, which synthesize melanin to provide color and protect the skin from damage by the UV rays of the sun. The dermal-epidermal junction lies beneath the stratum basale and is an important site of attachment in the skin. This junction allows nutrients to pass through the dermis to the avascular epidermis. *Langerhans cells* lie in the suprabasal layer of the dermis and are the immunologic cells responsible for recognizing harmful antigens to the body. They process the antigen and enter into the blood stream residing in the regional lymph nodes and stimulate the production of T lymphocytes.[2, 6]

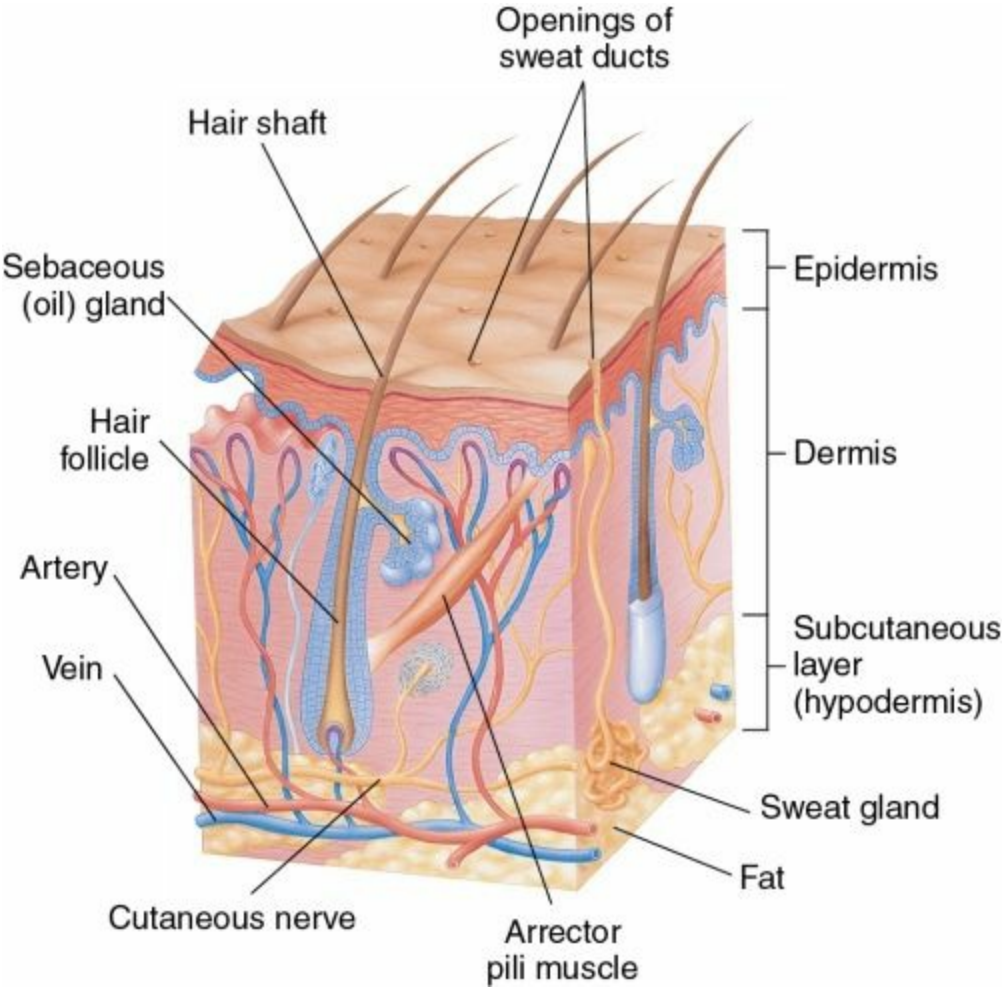


FIGURE 7.1 Anatomy of the skin. Source: (From Thibodeau GA, Patton KT: *Anatomy and Physiology*, ed 9, St. Louis, 2016, Mosby.)

The *dermis* is a richly vascular layer consisting largely of fibroblasts and collagen. The collagen matrix of the dermis supports and separates the epidermis from the subcutaneous fat layer. Papillae project up into the epidermis to provide nourishment to the living epidermal cells. In addition, the dermis contains a large network of sensory nerve fibers. These fibers provide sensations of pain, itch, and temperature. *Meissner corpuscles* are encapsulated end organs of touch found in the dermal papillae close to the epidermis. They are most numerous in hairless portions of the skin such as the volar surfaces of the hands, fingers, feet, toes, lips, eyelids, nipples, and tip of the tongue. The dermis also contains autonomic nerve fibers that innervate blood vessels, the *arrectores pilorum* muscles, the sweat glands, sebaceous glands, hair, and nails.

The *sweat glands* in the dermis control thermoregulation by

releasing water through the skin. The *eccrine sweat glands* are distributed throughout the body except for the lip margins, eardrums, nail beds, inner surface of the prepuce, and the glans penis.[7] They are most abundant on the palms and soles of the feet. The *apocrine sweat glands* are larger and deeper than the eccrine glands and secrete an odorless white fluid (sweat) in response to emotional or physical stimuli. They are located in the axillae, around the nipples or areolae, anogenital area, eyelids, and external ears. Body odor in adolescence comes from bacterial decomposition of the sweat produced by these glands; activation of these glands earlier than adolescence should be investigated. Neonates have the ability to respond to thermal stress by sweating, though it requires a greater thermal stimulus. This response is less developed in premature infants and increases with postnatal age.[8] Full-term infants are also able to respond to emotional stress by sweating, though this is not developed in the premature infant. This has clinical implications related to increased insensible water loss and thermoregulation in infants at risk.[9]

The *sebaceous glands* arise from the hair follicles deep within the dermis. The oil produced by these glands is called *sebum*, a lipid-rich substance that helps lubricate the skin and hair. Sebaceous glands depend on hormonal stimulation and are activated by androgens at puberty. The level of oil produced varies throughout the life span. In the newborn, the production of sebum is accelerated while still under the influence of maternal hormones, and the glands themselves become hyperplastic until maternal hormones wane in the infant's body. This stimulated activity results in skin conditions in the newborn such as *neonatal acne.* Overactive sebaceous glands appear again in adolescence and contribute to the common skin conditions *acne vulgaris* and *tinea versicolor*.[2]

The nail bed starts to keratinize to form a hard, protective plate around 8 to 10 weeks of gestation. It sits on a highly vascular bed that gives each nail its color. The *cuticle*, or *eponychium*, the white crescent-shaped area at the end of the nail matrix, is the root and site of nail growth (Fig. 7.2A and B). It is covered by a layer of stratum corneum that pushes up and over the lower part of the nail body. The *perionychium* is the soft tissue that surrounds the nail border on each digit. Nail health can be affected by several factors,

including nutrition, hydration, local infection/irritation, and systemic disease.

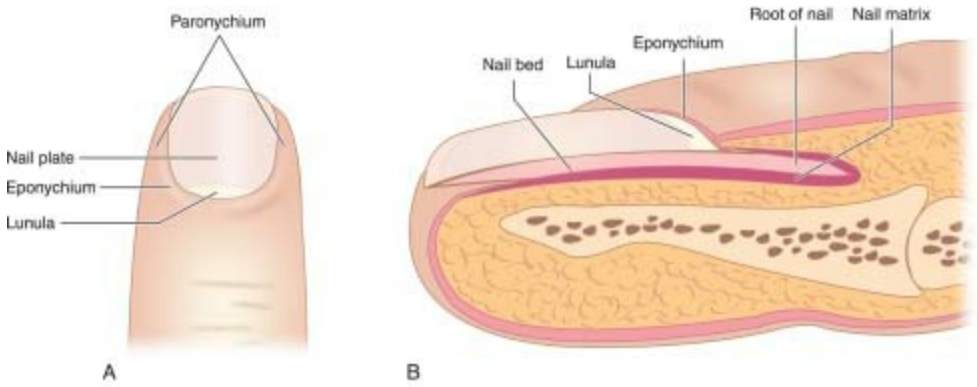


FIGURE 7.2 (A and B) Anatomy of nail.

The *subcutaneous* layer of the skin is composed of adipose tissue. This layer connects the dermis to underlying organs, provides insulation and shock absorption, and generates heat for the body. It also provides a reserve of calories for use by the body.[9] Premature and small for gestational age infants often lack this critical layer of insulation, which causes difficulty with thermoregulation.

Hair, eyebrows, and nails are part of the anatomical structure of the skin. Hair is formed by epidermal cells that go deep into the dermal layer of the skin and consists of a *root*, a *shaft*, and a *follicle* (Fig. 7.3). The *papilla* is the structure at the base of the hair follicle and is made of connective tissue and loop of capillaries, which supply and nourish the hair to promote growth. *Melanocytes*, which lie in the hair shaft, supply color to the hair.

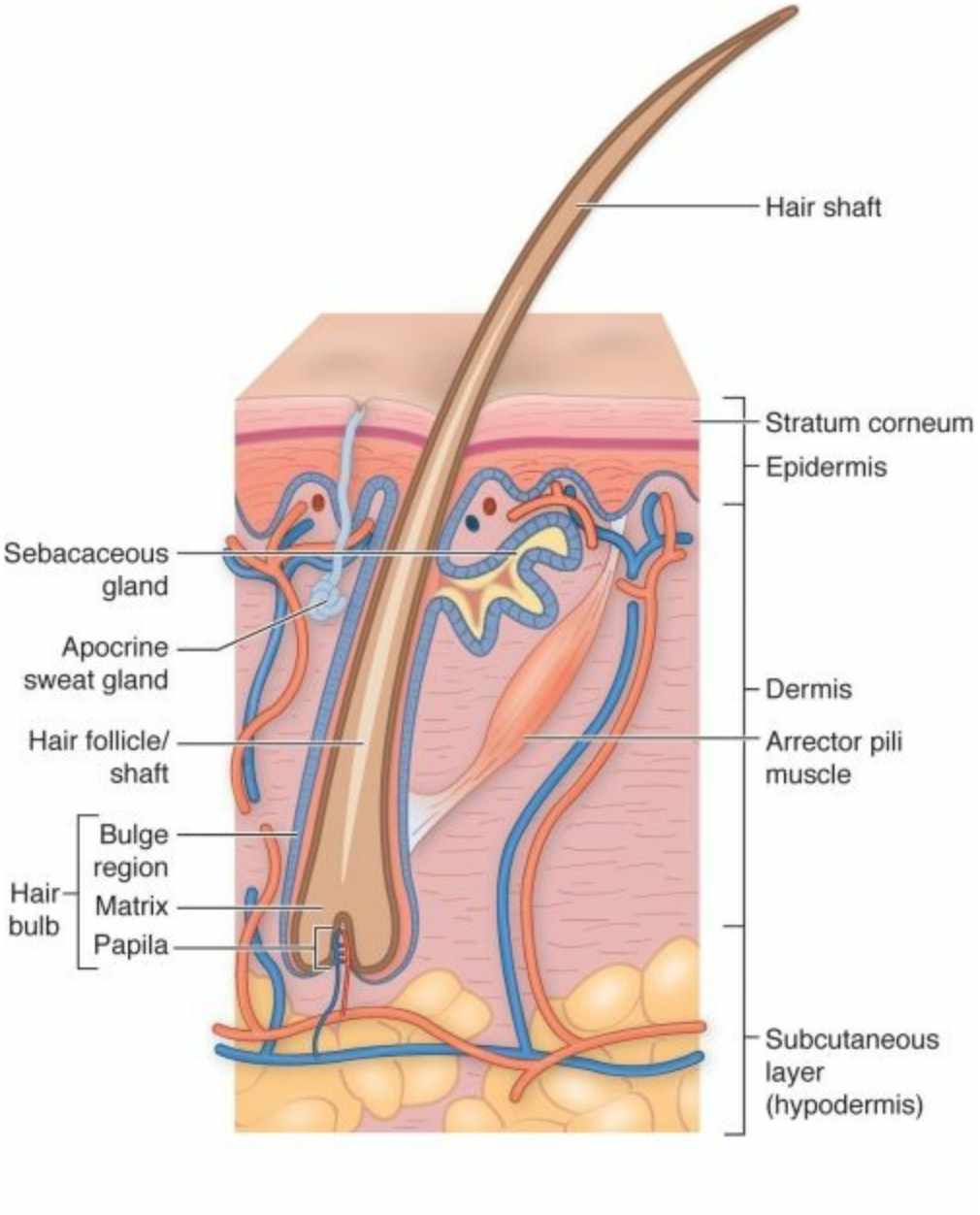


FIGURE 7.3 Anatomy of hair follicle.

Developmental variations

In the newborn, all the hair on the body consists of fine *lanugo* hair —all of the hair is in the same phase of growth. Within the first 4 months after birth, infants undergo a period of physiologic hair shedding to convert actively growing hairs to the resting phase and start the process of nonsynchronized growth phases. This normal process may already be complete at birth for infants with light hair who often appear completely bald at birth. This process may be

delayed for up to 4 months in dark-haired individuals.[9] As the lanugo is shed, it is replaced by hair that is increased in diameter and coarseness; the first to form are *vellus* hairs, which are short, fine, soft, and nonpigmented hairs on the body. Then adult-type *terminal* hairs, which are coarse, thick, longer, pigmented, and grow on the scalp and eyebrows. During adolescence, vellus hairs located in androgen-sensitive areas, pubic area, axillae, and the face in males undergo a similar transition to terminal hairs.

Hair follicles grow in a nonsynchronized fashion, with 85% to 95% in the growth phase lasting 2 to 6 years, 10% to 15% in the resting phase lasting 2 to 3 months, and less than 1% in the involution phase which lasts 2 to 3 weeks. There are approximately 100,000 to 150,000 hairs on the scalp. Daily hair loss varies between shampoo days and nonshampoo days, with adults losing 40 to 100 hairs on a nonshampoo day and 200 to 300 hairs on a shampoo day. Hair density has to decrease by half before there is noticeable hair thinning.[10, 11]

Physiological variations

The stratum corneum does not develop until between 23 and 25 weeks of gestation. Extremely premature infants are born without this critical top layer of skin and therefore have no protective barrier and are not able to control water loss. They need protection from the environment and can tolerate only the least amount of touching. The term newborn has a fully functional stratum corneum, but it is only about 60% of the thickness of adult skin depending on the location. A thin stratum corneum with the larger body surface area/weight ratio of the newborn may allow substances placed on the skin to pass more easily through to the bloodstream.[9, 12] The blood vessels continue to mature into a more adult pattern until 3 months after birth. The nerves in the skin are small and poorly myelinated at birth. The growth and myelination of the nerve fibers continue on into puberty (Table 7.1).

TABLE 7.1

Structural and Functional Differences of Skin

Structure	Term Newborn Infant	Child	Adolescent	Significance/Implications
Epidermis	Stratum corneum appears as adherent cell layer Greater absorption because of higher skin surface/body weight ratio	Stratum is getting thicker, starts to appear as separate sheet of cells	Stratum corneum appears as separate sheet of cells Adult-like pattern	Thin skin of infant and child allows for easy absorption of products placed on skin Apply thin layer of topical medications
Dermis	Fewer immature elastin fibers Thinner than adult	Elastin fibers are maturing	Full complement of elastin fibers	Decreased elasticity Increased tendency to blister
Melanosomes	Melanin production low Final overall skin tone is shown in genitalia where the scrotum, labia have darker pigment	Melanin production after 6 months of age like adult	Adult pattern of melanin production	Infants, young children need sunscreen/complete sun block because can sunburn easily
Eccrine sweat glands	Equivalent in structure to adult Dense distribution due to body surface area	Distribution starts becoming less dense as child grows, but has decreased neurological control until 2–3 years old	Distribution is less dense than in infant, child	Reduced sweating capability, especially first 13–24 days of life Decreased response to thermal stress
Apocrine sweat glands	Small, nonfunctional Devoid of secretory granules	Start to appear but generally nonfunctional in childhood	Apocrine sweating in response to mechanical, pharmacological stimuli	Secrete oily substance in adolescent
Sebaceous glands	Large and active but diminish rapidly in size and activity several weeks after birth	Decreased activity throughout childhood	Large, active, produce sebum in large amounts	Infants get acne and tinea versicolor as do teens because of hormone activity and large active sebaceous glands
Nervous and vascular systems	Vascular system fully organized after 3 months Most nerves are small in diameter Sensory autonomic nerves are unmyelinated Cutaneous nerve network not fully developed Meissner touch receptors [a] not fully formed	Cutaneous network continuing to develop	Cutaneous network of nerves may continue to develop into adolescence Rest of nervous, vascular systems are in adult pattern	
Hair	Lanugo covering body often shed within 10–14 days Vellus and terminal hairs appear quickly after birth Hair growth is synchronous	Vellus and terminal hairs present Hair growth is asynchronous	Vellus and terminal hairs present Hair growth is asynchronous	Dry, dull, and brittle hair may indicate protein-calorie malnutrition

[a]Meissner touch receptors are encapsulated end organs of touch found in dermal papillae close to epidermis.

Family, cultural, racial, and ethnic considerations

Recent research has shown skin *pigmentation* or *melanin* should be considered as a factor for understanding some underlying skin properties and characteristics, particularly in children of multiracial ancestry. Understanding how underlying skin color and pigmentation may affect the examination and diagnosis of skin conditions allows the clinician to adjust assessment techniques and interventions using the child or adolescent's physiological characteristics rather than racial or ethnic categorization to guide care.[1]

Assessing skin pigmentation allows the health care provider to make clinical decisions based on tissue perfusion, jaundice, pallor, cyanosis, and blanch response. The *blanch test* differs widely based on underlying skin color. The blanch test differentiates healthy skin

from erythematous skin that is nonblanchable when gentle pressure from fingertips is exerted on the skin, blood is temporarily forced out of the region, causing the skin to appear lighter than the normal skin color. In individuals with lighter skin pigmentation, the skin color returns swiftly as the blood refills the dermal capillaries. It may be difficult to determine this response in a darker pigmented or darker skinned individual. Individuals with darker skin often have a purplish tinge rather than a reddish tinge when erythema is present and often have a more follicular pattern (Fig. 7.4).

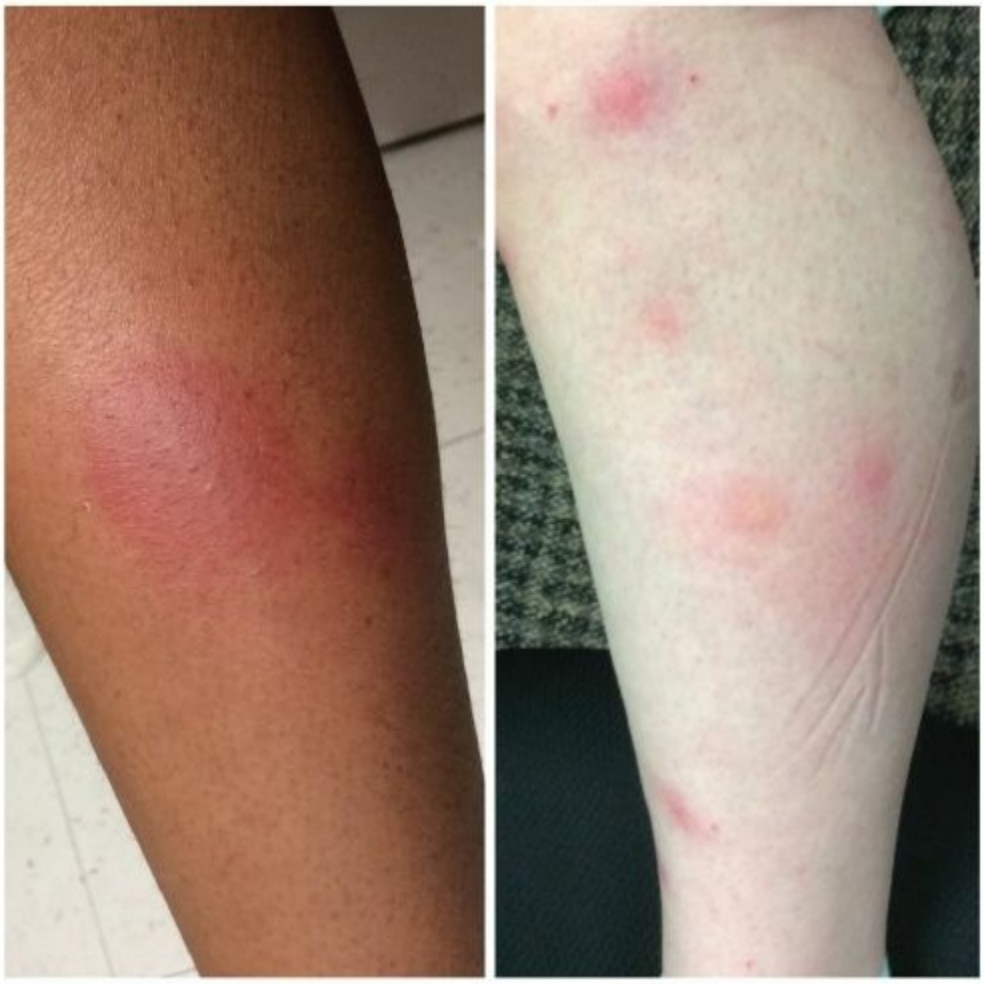


FIGURE 7.4 Insect bites on lower leg of pigmented and nonpigmented skin.

Postinflammatory hypopigmentation, absence of normal melanin, and hyperpigmentation, increased melanin, occur more frequently in darker skinned individuals, and conditions such as

acne or eczema can produce significant skin color changes from postinflammatory hyperpigmentation. African American, Asian, and Hispanic children are also at higher risk of *keloid* scarring. A keloid is a type of scar at the site of a healed skin injury resulting from an overgrowth of granular tissue composed of collagen (see Table 7.3). Lighter skinned individuals may have more recognizable signs of skin breakdown, sun exposure, and tissue perfusion than darker skinned individuals. Looking at the sclerae, conjunctivae, buccal mucosa, lips, tongue, and nail beds will assist the health care provider in assessing children and adolescents with significant clinical variations in pigmentation and skin color. Variations of skin pigmentation with darker skinned individuals normally occur on the palms, soles of feet, nail beds, and the genital area. Freckling of the buccal cavity, gums, and tongue is also common. Areas that get regular exposure to the sun may have much darker pigmentation.

Other variations related to skin pigmentation or melanin may exist in the barrier properties of the skin and in the distribution of hair follicles. Variations in barrier properties and hair follicle distribution are significant because skin in darker pigmented individuals may provide more of a barrier against absorption of topically applied drugs and cosmetics. The barrier function may also prevent the penetration of some toxins.[12-14] The variation in barrier properties of the skin contributes to darker skinned individuals having an increased incidence of *xerosis,* abnormal dryness of the skin with a loss of natural skin shine, which presents as a whitish visual appearance in darkly pigmented skin.[13, 14]

Hair follicle distribution and hair quality differ greatly with racial and ethnic background. African Americans have hair that is often coated in natural oils, and the frequent use of hair oils and braiding may make it difficult to accurately assess lesions on the scalp. African American children and adolescents have a higher incidence of *tinea capitis*, which may be due to the increased coiling of the hair shaft, and require more prolonged medical management. Some of these hair qualities are present in multiracial children, and it may be confusing to a mother or parent who does not have similar hair qualities. Hair, skin, and nail care practices vary widely among cultures and within cultures. Timing of a child's first haircut is one such cultural variation. In many Asian and Latino cultures, it is

common to shave the infant's head between 3 and 9 months of age in the belief the hair will grow in thick and long. In some cultures, shaving of the head is part of a religious ceremony. Some African American communities believe an infant's hair should not be cut until he or she begins walking.

Health care providers should be sensitive and attentive to the differences in skin quality and skin integrity when caring for children, adolescents, and families from diverse racial and ethnic backgrounds, and should encourage and support cultural practices that preserve the family culture except when a skin care practice or treatment is harmful and impacts the healthy development of the skin.

System-specific history

A careful age-appropriate history is critical to making an accurate assessment of the skin (see the Information Gathering table). The health care provider needs to gather information related to current skin conditions, any significant past medical history, and family history of chronic skin conditions. Skin care routines and any recent changes in skin, hair, or nail care habits should be assessed. Sun exposure habits and application of sunscreen are also important considerations in the assessment of the skin. Box 7.1 presents symptom-focused information gathering for skin conditions in children.

BOX 7.1

Symptom-Focused Information Gathering of Skin Conditions

- Any recent changes in skin, hair, or nails? Any dryness, pruritus, sores, rashes, lumps, color changes, or changes in texture or odor noted?
- What signs or symptoms are present (rash, single or multiple skin lesions, itching, pain, exudates, bleeding, color changes)? Other symptoms of fever, malaise, loss of appetite? Upper

respiratory symptoms?

- Where is the skin problem located?
- When did it start, sequence of occurrence, rapidity of onset? Is this a recurrence? Any known allergies? Any history of recent illness like strep throat?
- Any recent exposure to drugs, new skin products including insect repellent, detergent products, new foods, other environmental or occupational toxins, or family member or contact with similar condition? Family history of diseases like psoriasis? History of recent travel?
- What has been done to treat the problem, including medications (over-the-counter [OTC] or prescription) and/or lotions or other emollients applied? Did the problem get better or worse?

Information Gathering at Key Developmental Stages

Age Group	Questions to Ask
Newborn	*At birth:* History of skin trauma at birth or significant bruises to face/body? Presence of skin tags, dimples, cysts? Any extra digits? Moles or nevus? Hair or nail variations present at birth? Received any phototherapy?
Infant to 6 months of age	*Diaper history:* Type of disposable wipes used? Type of diapers used? *Skin care history:* Types of soap, moisturizing/cleansing lotion, other lotions, emollients, creams, oils? *Dressing habits:* Amounts/types of clothing in relation to environmental temperature, how clothing is washed, use of detergents, fabric softeners, dryer sheets? *Home environment:* Temperature, humidity, type of home heating? Air conditioning? *Feeding history:* Breast or bottle, type of formula, what foods introduced and when?
6 months to 2 years of age	History of eating large amounts of yellow fruits, vegetables? History of prolonged crawling on hands, knees without protective clothing? History of rubbing head against furniture/walls?
Early childhood	Eating habits/types of food? History of exposure to communicable diseases? Pets/animal exposure? History of dry skin, eczema, urticaria, pruritus, nasal allergy, asthma? History of nail biting, hair twisting?
Middle childhood	History of skin injuries: cuts, falls, fractures, need for sutures? Any unexplained scarring or bruises? Outdoor exposure to plants during hiking, camping, picnics? Bee stings, contact with plants resulting in allergic reactions, dog bite? Undiagnosed rashes?
Adolescence	History of skin/hair changes, acne? Acne treatments used? Sports-related

	injuries? Body tattooing, ritual scarring, piercing? Were they done professionally using sterile techniques/supplies? Problems/infections related to these practices?
Environmental Risks	Exposure To Tobacco Smoke? Contact With Chemical Cleaning Agents/Other Chemicals At Home, School, Work? Exposure To Chemicals, Toxins From Parent's Work?

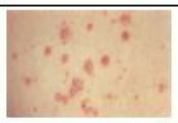
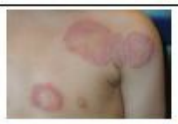
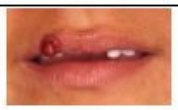
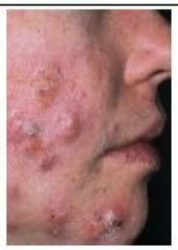
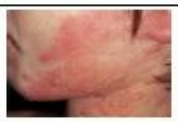
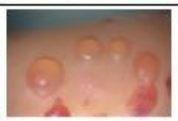
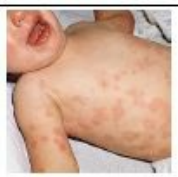
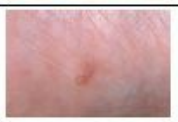
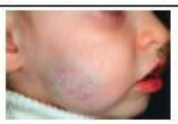
Physical assessment

The skin is one of the most accessible and easily examined organs of the body and is often the organ of most concern to children, adolescents, and parents. A complete examination of the skin using a consistent, systematic approach will increase the likelihood that important findings critical to making a diagnosis will not be missed. Always avoid making a quick diagnosis after only a brief inspection of an area of exposed skin or examining only the lesion of concern. A deliberate and methodical assessment of the skin will lead to a correct diagnosis and prevent missing important clues. During inspection, any changes in the skin should be palpated, inspected, and classified by morphology, size, color, texture, firmness, configuration, location, and distribution. The color, turgor, texture, temperature, and moisture of the skin and the growth, and texture of the hair and nails should also be noted.

Dermatology has its own language. Using the correct terminology facilitates accurate description of skin lesions. A *skin lesion* refers to any variations or skin changes. If the inspection and palpation of the skin reveal a lesion, more examination is necessary. Skin lesions may be *primary* or *secondary* (Tables 7.2 and 7.3). A *primary lesion* is the initial lesion of a skin condition, and identifying the *primary lesion* is the most important step in assessing skin conditions in children and assists clinicians in diagnosis. A *secondary lesion* often develops as a skin condition progresses such as infection, trauma or with skin therapy.

TABLE 7.2

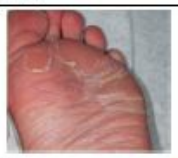
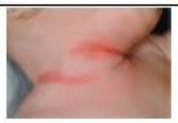
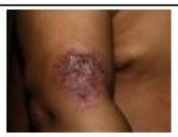
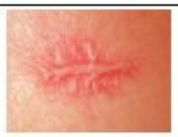
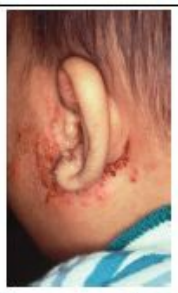
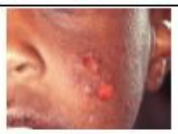
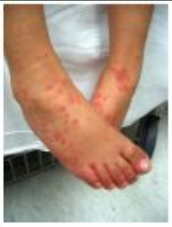
Primary Lesions

Name	Photo	Description	Examples of Conditions
Macule/patch		Flat, circumscribed lesion of any size, <1 cm is macule; >1 cm is patch; lesions usually rounded but may be oval, can be vascular, hyperpigmented, or hypopigmented	Freckle, café au lait spots, vitiligo, flat mole (nevus), blue-gray macules of the neonate (Mongolian spots), port-wine stain
Papule		Palpable, circumscribed elevated lesions <1 cm	Molluscum contagiosum, papular urticaria, elevated moles, wart
Plaque		Circumscribed, elevated, disc-shaped lesion >1 cm; commonly formed by confluence of papules	Atopic dermatitis, lichen simplex chronicus (neurodermatitis), tinea corporis
Nodule		Circumscribed, elevated, usually solid lesion that measures 0.5–2 cm; may be in epidermis or extend deeper	Fibromas, neurofibromas, intradermal nevi, erythema nodosum, hemangioma, pyogenic granuloma
Cyst		Elevated, circumscribed, encapsulated lesion in dermis or subcutaneous layer filled with liquid/semisolid material	Sebaceous cyst, cystic acne
Vesicle		Sharply circumscribed, elevated, fluid-containing lesion that measures ≤0.5 cm	Herpes simplex, varicella, insect bite, herpes zoster
Bulla		Sharply circumscribed, elevated, fluid-containing lesion that measures ≤1 cm	Contact dermatitis, epidermolysis bullosa, pemphigus vulgaris, burn, bullous impetigo
Wheal		Distinctive type of solid elevation formed by local, superficial, transient edema; white to pink–pale red in color; blanches with pressure, varies in size, shape	Urticaria, insect bite, dermographia, erythema multiforme
Comedones		Plugged secretions of horny material retained within pilosebaceous follicle; may be flesh-colored, closed (whiteheads); brown/black, open (blackheads)	Acne
Burrows		Linear lesion produced by tunneling of animal parasite in stratum corneum	Scabies, cutaneous larva migrans (creeping eruption)
Telangiectasia		Fine, irregular, red lines produced by capillary dilation	Rosacea

Papule, vesicle, and bulla images are from Weston W, Lane A, Morelli J: *Color Textbook of Pediatric Dermatology*, ed 4, St. Louis, 2007, Mosby; Cyst image is from Habif T: *Clinical Dermatology*, ed 6, St. Louis, 2010, Mosby; Wheal image is from Eichenfield L, Frieden I, Esterly NB: *Neonatal and Infant Dermatology*, ed 3, Philadelphia, 2015, Saunders; Comedone image is from Brinster NK, Liu V, Diwan AH, et al.: *Dermatopathology: High-Yield Pathology*, Philadelphia, 2011, Saunders; Burrow image is from White G, Cox N: *Diseases of the Skin*, ed 2, St. Louis, 2006,

Mosby; and Telangiectasia image is from Paller A, Manicini A: *Hurwitz Clinical Pediatric Dermatology: A Textbook of Skin Disorders of Childhood and Adolescence*, ed 4, Philadelphia, 2011, Saunders.

TABLE 7.3
Secondary Lesions

Name	Photo	Description	Example of Diseases
Scale		Formed by accumulation of compact desquamation of stratum corneum layers; may be greasy, yellowish; silvery, fine, barely visible or large, adherent, and lamellar	Seborrheic dermatitis Psoriasis Pityriasis alba Tinea versicolor Ichthyosis
Fissure		Dry, moist, linear, often painful, cleavage from epidermis to dermis that results from marked drying; long-standing inflammation, thickening, loss of elasticity of integument	Chronic dermatoses Intertrigo Atopic dermatitis Ichthyosis
Lichenification		Rough, thickened epidermis secondary to persistent rubbing, itching, or skin irritation; often involves flexor surface of extremity	Atopic dermatitis Chronic dermatitis
Scar		Permanent fibrotic skin changes that develop following damage to dermis; initially pink/violet, fading to white, shiny, sclerotic area *Keloid:* pink, smooth, rubbery; often traversed by telangiectatic vessels; increases in size long after healing of lesion; differentiated from hypertrophic scars because surface of keloid scar tends to be beyond original wound area	Surgery Healed wound Stretch marks Keloid Herpes zoster Burn
Crust		Dried exudate on epidermis composed of serum, blood, or pus overlying a ruptured bulla or vesicle; caused by staphylococcal or streptococcal bacteria	Impetigo Epidermolysis bullosa
Erosions		Moist, slightly depressed vesicular lesion in which all or part of epidermis has been lost; may have surrounding erythema or edema; heals without scarring	Impetigo Eczematous diseases Intertrigo Candidiasis Methicillin-resistant *Staphylococcus aureus* (MRSA)
Purpura		Flat lesion but may be palpable; petechiae if pinpoint; does not blanch to pressure; larger areas of bruising may be present	Henoch-Schönlein Purpura fulminans

Data from Eichenfield L, Frieden I, Zaenglein A, et al.: *Neonatal and Infant Dermatology*, ed 3, Philadelphia, 2015, Saunders; Ball JW, Dains JE, Benedict GW: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby; Infoderm.com,

Galderma Laboratories, LP, 2012; Scale and fissure images are from Paller A, Manicini A: *Hurwitz Clinical Pediatric Dermatology: A Textbook of Skin Disorders of Childhood and Adolescence*, ed 4, Philadelphia, 2011, Saunders; Lichenification image is from Rudikoff D: Differential diagnosis of round or discoid lesions, *Clin Dermatol* 29(5): 489-497, 2011; Scar image is from Arndt K: *Procedures in Cosmetic Dermatology Series: Scar Revision*, Philadelphia, 2006, Saunders; and Purpura image is from Marx J, Hockberger R, Walls R, et al.: *Rosen's Emergency Medicine: Concepts and Clinical Practice*, ed 8, Philadelphia, 2014, Elsevier. (Courtesy of Marianne Gausch-Hill, MD.)

A rash cannot be diagnosed by listening to a description over the phone, and pediatric health care providers should avoid this type of assessment. It is important to conduct a complete skin examination before making a diagnosis. It is usually very easy to do a complete skin assessment on a newborn, but there may be a great deal of resistance from many adolescents to the idea of a complete skin exam. When the child or adolescent is uncomfortable being completely undressed because of developmental stage or cultural belief, then the assessment of the skin must be conducted using a systematic approach that divides the skin into areas that are sequentially uncovered, examined, and then re-covered before going on to the next area. In infants and young children, this may also prevent unnecessary cooling of the skin.

Telemedicine, as a method to deliver healthcare, may allow a complete assessment of the skin using a computer and peripheral devices. Patients that are not able to physically come to the clinical or office may now have an option that allows both initial assessment and diagnosis and follow-up of skin diseases. Patients are often more comfortable showing their skin, hair, and nails in the home setting using a computer. The high-resolution cameras that are available today on personal computers and tablets and the magnifier peripheral devices (dermascopes) available provide excellent visualization of the skin with proper instructions to the patient for lighting and technique. The "store and forward" feature of most telemedicine software allows the practitioner to easily forward pictures for consultation with a pediatric dermatologist when a diagnosis is uncertain.[15, 16]

Inspection

Inspection of the skin is best conducted using natural light. If that is

not available, a well-lit room with fluorescent or incandescent lighting may be satisfactory. A magnifying glass and a measuring tool such as paper tape measure will be helpful for examining small skin lesions and moles. It may be necessary to follow the progress of lesions over time to determine an accurate diagnosis. A light for transillumination of lesions or for closer inspection may also be helpful. A photo inserted in the medical chart or electronic medical record may be useful when following a skin condition or using Telederm to consult with dermatology. In addition, a photo showing a ruler next to the skin lesion can assist in evaluating changes in lesions over time.

Hair should be examined carefully by sitting the child in a chair rather than on the examination table so the hair can be viewed from above. Use a magnified light source whenever possible as overhead lighting or natural light is often not sufficient for examination of the scalp and hair. Be sure to assess terminal and vellus hairs for changes. Note distribution, color, and quantity. Check for any lesions, dryness, oiliness, scaling, or infestation on the scalp.

A tool that is very important in examining the hair is a *hair card*, which is a 3 × 5 inch^2 card, white on one side and black on the other, with a centimeter ruler down one side to demonstrate new hair growth. The hair card allows the provider to assess new hair growth, temporal recession, and the dimensions of hair loss in both dark hair and light hair (Fig. 7.5). To assess hair density, part the hair starting at the frontal hairline and repeat the parts at 1-inch intervals, noting the spacing between the hairs. While noting color, scale, papules, pustules, and crusting on the scalp, note hair density from front to back of the scalp.

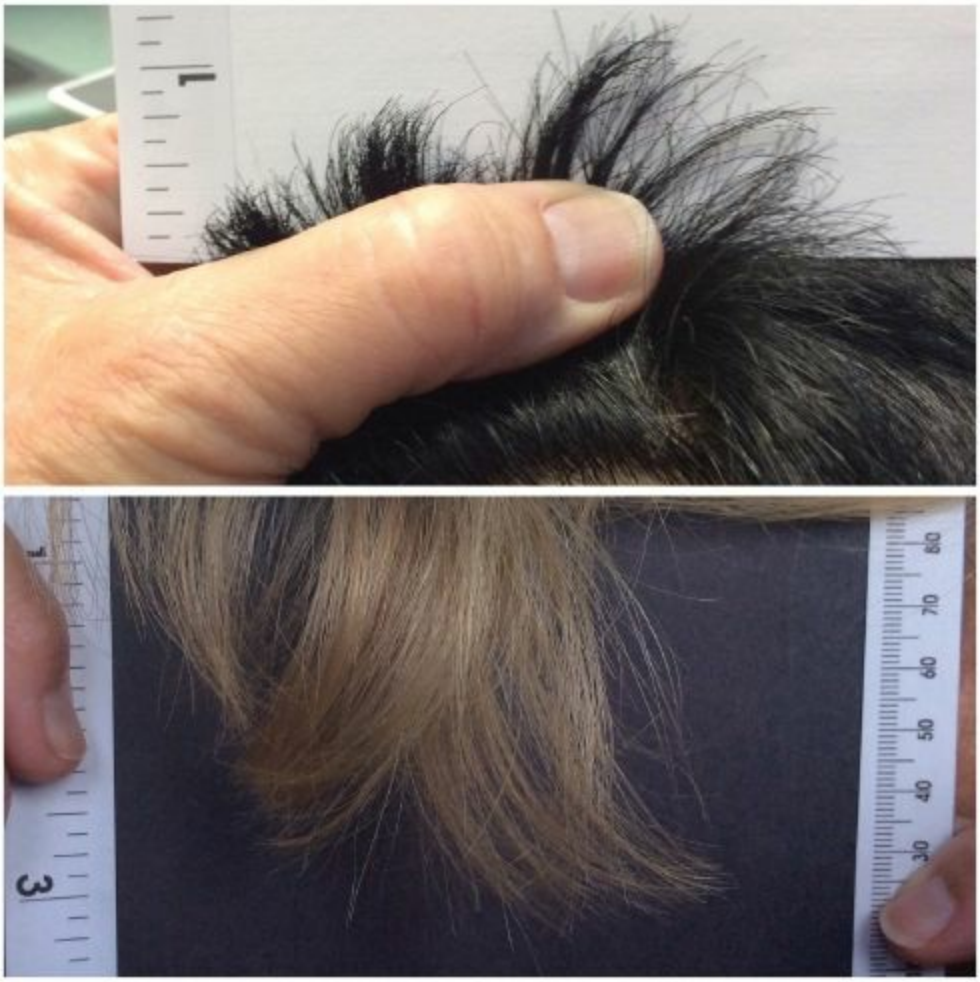


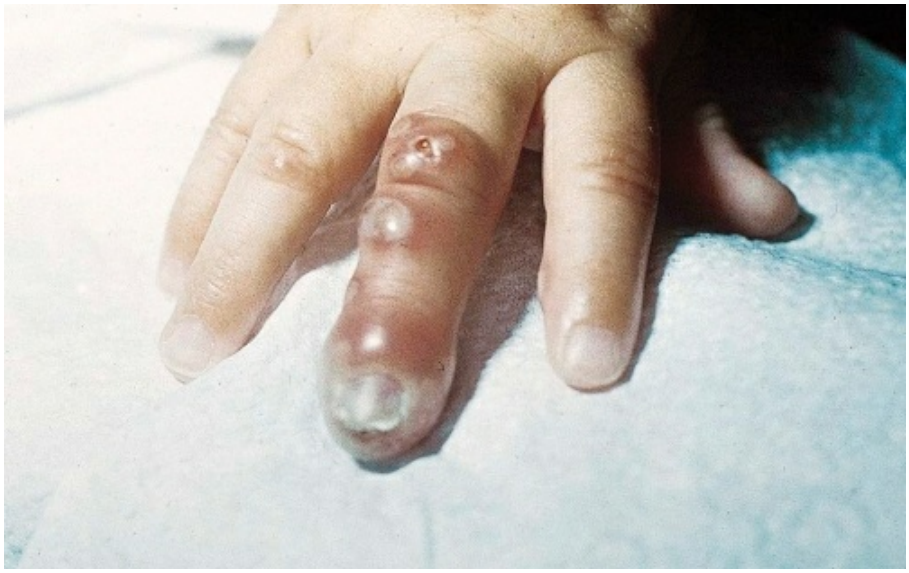
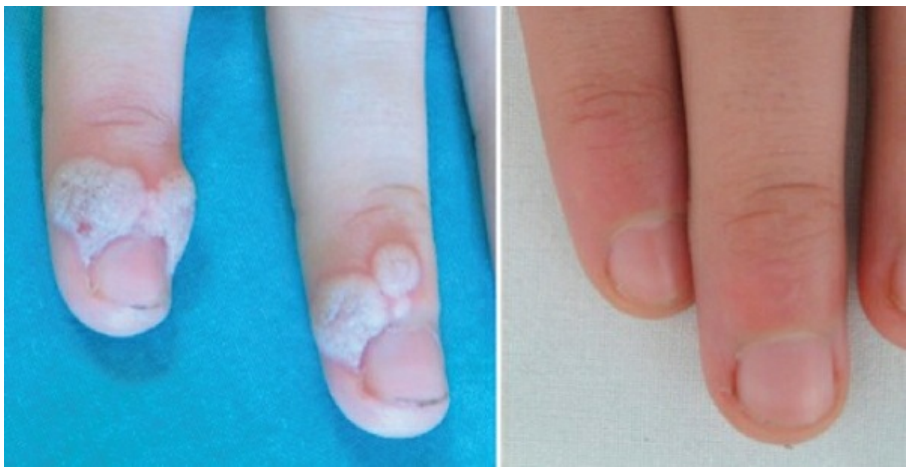
FIGURE 7.5 Hair card measuring hair growth.

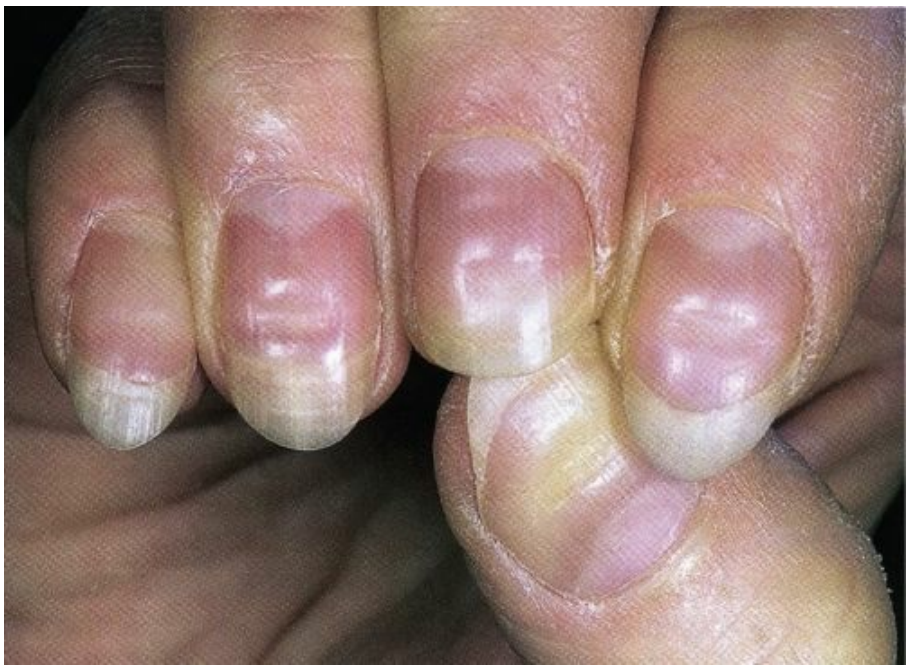
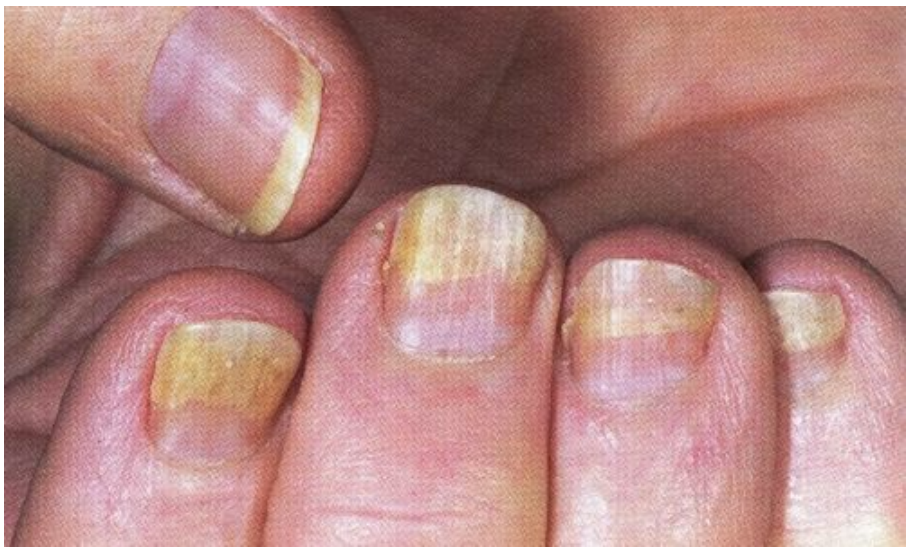
Some hair loss is common in older infants and children. In infants, hair loss after the neonatal period may be due to birthmarks, birth trauma, inflammation such as seborrheic dermatitis, or infections such as *tinea capitis*. The majority of hair loss in infants and children is nonscarring, and the hair grows back once the underlying condition is corrected.[11, 15] Excessive hair shedding can be measured by the *hair pull* test. Grasp 30 to 40 closely grouped hairs at the scalp using the thumb and index finger and gently but firmly pulling the hair away from the scalp. For the *hair tug* test, gather a cluster of hair at the scalp and use the other hand to tug the distal ends of the hair. This assesses hair fragility, and fragile hair will break off in small clusters.[10, 11] Excessive hair loss or excessively fragile hair requires further diagnostics; refer as indicated.

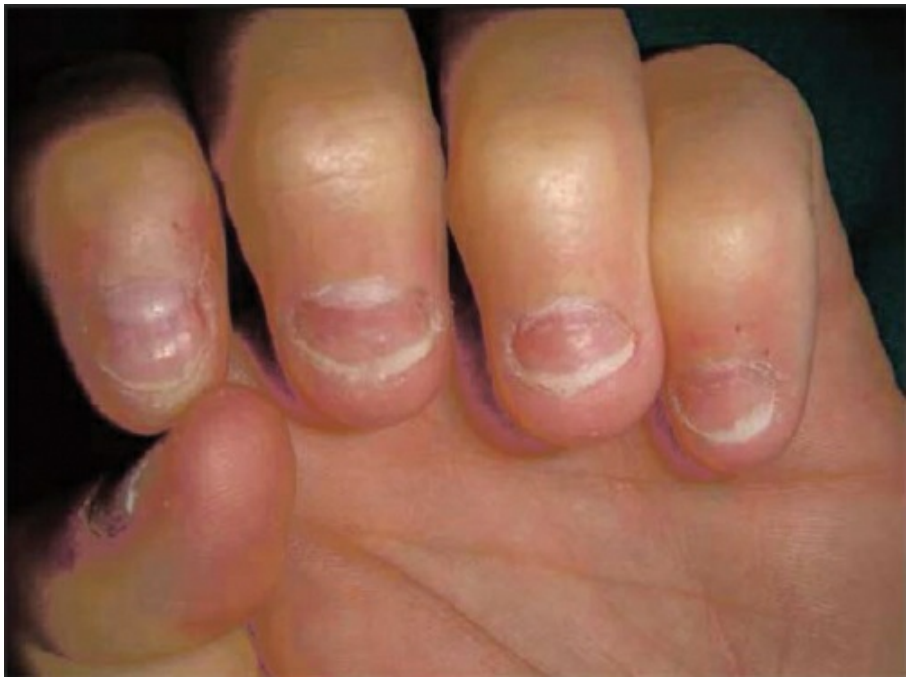
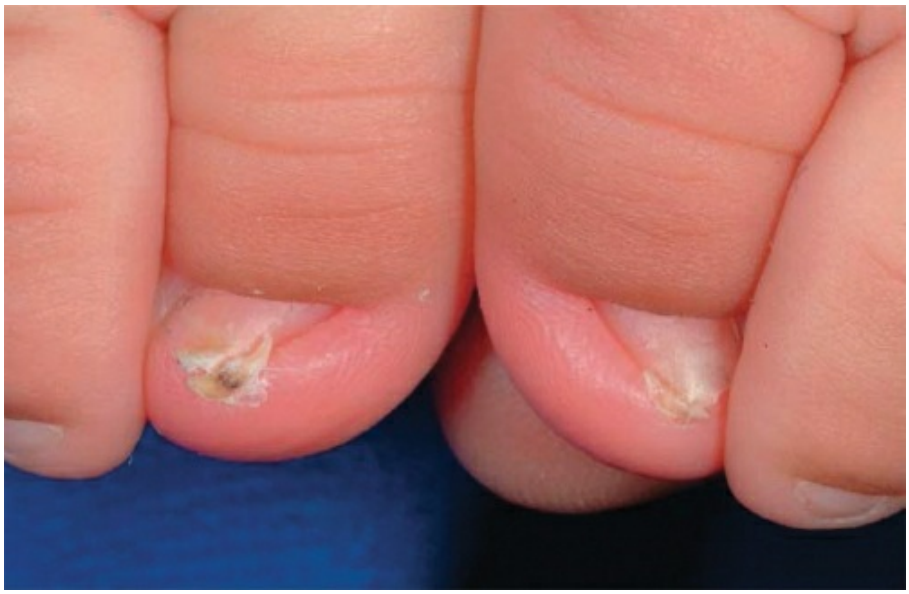
Nails should be examined for shape, color, and texture. Nail

changes may be an early sign of systemic disease (Table 7.4). Artificial nails or nail polish can interfere with the assessment. Inspect the curvature of the nail for *clubbing* (see Chapter 8, Fig. 8.5) or spooning and feel the surface for ridges. Changes in coloration or splinter hemorrhages in the nail should be noted. Finally, check the periungual tissue of the nail and note any redness, edema, induration, or tenderness. Absence or atrophy of the nails in the newborn period may indicate a congenital syndrome and requires consultation and referral for further diagnostics.[17, 18]

TABLE 7.4
Nail Lesions

Nail Symptoms	Photo
Periungal blister that has coalescent tapioca-like vesicles Vesicles become turbid and opaque colored with time. Usually at distal phalanx of finger Often painful, edema and erythema may be present	
Dry, cauliflower like thickened skin The absence of skin lines crossing the surface is a diagnostic sign of all warts as is pinpoint black marks, which represent subkeratotic capillary thrombosis. Usually located around the	

cuticle but can originate in the longitudinal grooves of the proximal or lateral nail folds Can cause fissures in the nail, pain, and embarrassment Can cause nail elevation and thickening of the nail	
Thick yellow appearance to nail most often affects toenails, but check finger nails that have had acrylic or gel nails Nails are often brittle and break off or separate from the nail plate	
Transverse grooves or furrows that originate under the proximal nail fold. Lines first appear at the cuticle and grow out with the nail Onychomadesis is proximal separation of the nail resulting from full but temporary arrest of the growth matrix.	

Punctate depressions resulting from alterations in the proximal matrix. May vary from small to large and dip pits to clusters of pits. One or more nails may be affected.	
Lateral edge of the nail is curved inward and penetrates the underlying tissue Erythema, edema, pain and in chronic conditions granulation tissue are present Most commonly seen on the great toes but may occur on other nails Pseudo-ingrown nails present in newborns is self-correcting by 12 months of age	

Modified from Luckenberg EL, Silverman RA: Nail disorders in children. *Dermatol Nurs* 22(1), 2011; available at http://www.medscape.com/viewarticle/718695_1; Richert B, Andre J: Nail disorders in children diagnosis and management. *Am J Clin Dermato* 12(2):101-112, 2011; Figures: Herpetic whitlow from Mendoza N: Human herpesviruses. *Dermatology* 80:1321-1343.e1, 2012. Courtesy of Louis A. Fragola, Jr., MD; Periungal warts from Martin JM: Spontaneous remission of recalcitrant warts

in girls after human papillomavirus vaccination. *Dermatology* (Actas Dermo-Sifiliograficas, English Edition) 107(6);533-535, 2016; Onchomyocosis and Beau's lines from Habif TP: *Clinical Dermatology*, ed 6, Philadelphia, 2015, Mosby; and nail pitting from Richert B, Andre J: Nail disorders in children diagnosis and management. *Am J Clin Dermatol* 12(2):101-112, 2011.

Palpation

Palpation of the skin should be done with warm hands using gloves if you think the child or adolescent may have an infectious lesion. Palpate skin temperature using the back of your hand, and compare the temperature of one area of skin to another area of skin using both hands. Temperature cannot be assessed accurately through gloves, and the presence of a fever should always be checked using a thermometer. Check for *skin turgor*, resiliency, or elasticity of the skin, by gently pinching a fold of the child's skin over the abdomen between your thumb and forefinger and then releasing it. *Skin turgor* can give important clues to the hydration and nutritional status of a child. How long the skin remains tented after it is released will provide clues to the degree of dehydration (Table 7.5).

TABLE 7.5

Estimating Dehydration in an Infant or Young Child

Return to Normal After the Pinch	Degree of Dehydration
<2 s	<5% loss of body weight
2–3 s	5%–8% loss of body weight
3–4 s	9%–10% loss of body weight
>4 s	>10% loss of body weight

Data from Ball JW, Dains JE, Solomon BS, et al: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby.

Skin conditions

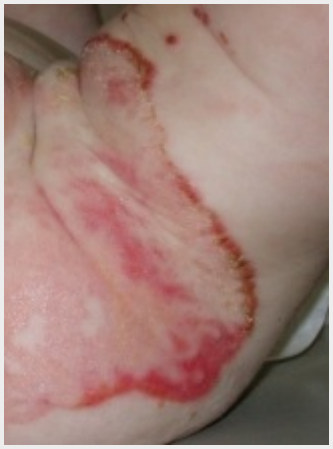
Skin lesions

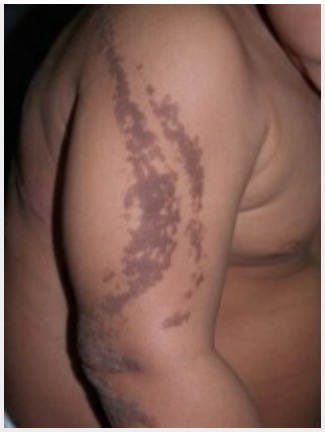
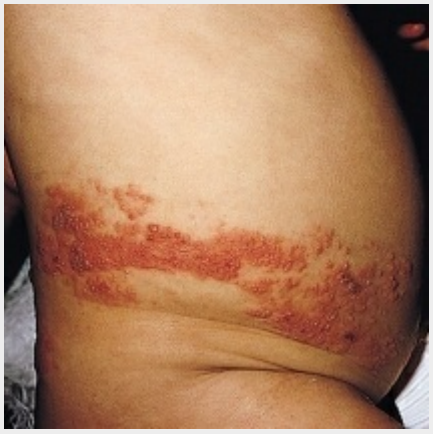
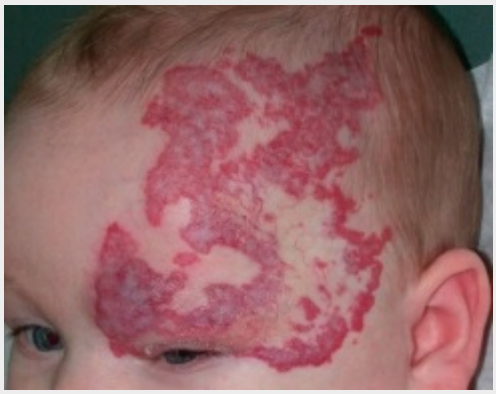
The *morphology* or characteristic form and structure of skin lesions should be identified when any condition is noted during the assessment of the skin. Attention should be made to the distribution

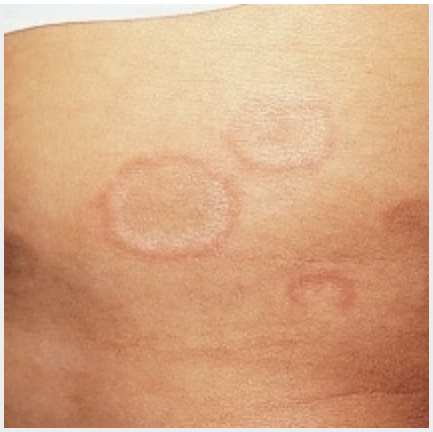
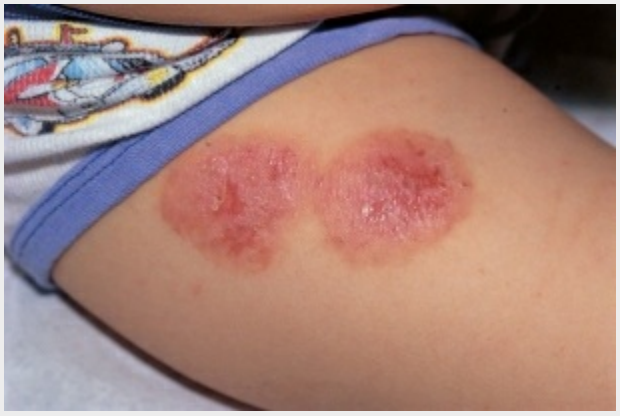
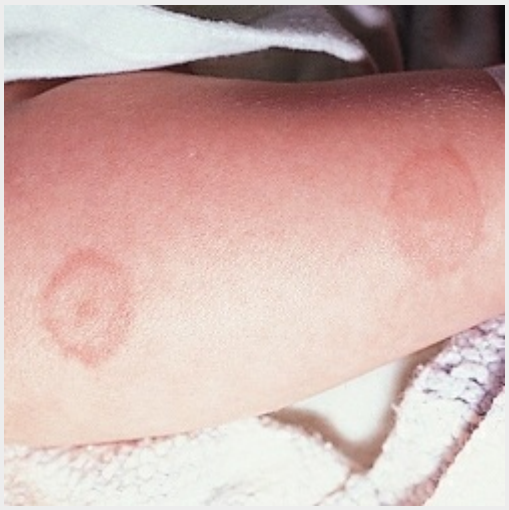
and pattern of lesions. Becoming familiar with four or five distinct morphologic patterns such as acneiform, annular, or linear eruptions and the associated findings will assist in making a diagnosis. The *distribution* refers to the location of skin findings, whereas *pattern* refers to the specific anatomical or physiological arrangement of the lesions. Note the shape of skin lesions and whether they are clustered together or scattered. The border or margin, any associated findings such as central clearing, and the pigmentation of the lesion also should be identified. Find and study the primary lesion and examine the distribution of any other skin lesions or skin variations. Skin lesions should be classified as primary or secondary (see Tables 7.2 and 7.3). Box 7.2 illustrates the color, borders, configuration, and distribution of lesions. Common newborn and infant skin conditions are presented in Chapter 6, Table 6.6.

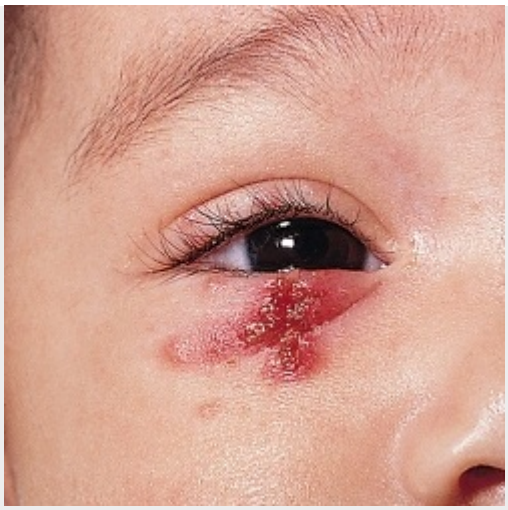
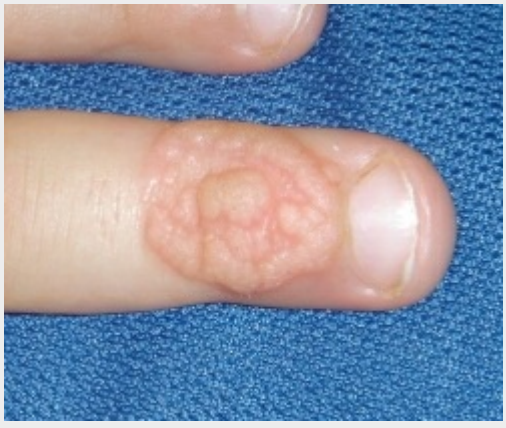
BOX 7.2

Borders, Configuration, and Distribution of Lesions

Border	
	Borders of lesion may be raised or indurated, as in granuloma annulare and neonatal lupus, or indistinct, as in cellulitis or atopic dermatitis
Acrodermatitis enteropathica	
Configuration ***Blaschko (Linear)***	
	Linear lesions do not follow any known vascular, nervous, or lymphatic pattern. V- and S-shaped lines may

	represent patterns of neuroectodermal migration, and distribution indicates a cutaneous mosaicism
Linear epidermal nevus	
Dermatomal/Zosteriform (Linear)	
	Lines demarcating a dermatome supplied by one dorsal root ganglia
Herpes zoster	
Segmental Patterns	
	The configuration of segmental lesions is thought to be determined by the location of embryonic placodes or other embryonic territories, as can be seen in PHACE syndrome
Infantile hemangioma	
Annular	
	A round, ring-shaped lesion, where the periphery is distinct from the center, as in tinea corporis or neonatal lupus

	
Annular lesions of neonatal lupus	
Nummular	
	A coin-shaped lesion, with homogeneous character throughout, as in nummular eczema
Nummular eczema	
Targetoid	
	Concentric ringed lesions, often with a dusky or bullous center characteristic of erythema multiforme
Early lesions of erythema multiforme	
Herpetiform	

	Clusters of erythematous occasionally scabbed lesions, as in herpes simplex
Herpes simplex infection	
Corymbiform	
	Defined as a central cluster of lesions surrounded by scattered individual lesions, as in verrucae
Verrucae	

Modified from Eichenfield L, Frieden I, Zaenglein A, et al.: *Neonatal and Infant Dermatology*, ed 3, Philadelphia, 2015, Saunders; Acrodermatitis enteropathica, linear epidermal nevus, herpes zoster, infantile hemangioma, neonatal lupus, erythema multiforme, herpes simplex, and verrucae images are from Eichenfield L, Frieden I, Zaeglein A, et al.: *Neonatal and Infant Dermatology*, ed 3, Philadelphia, 2015, Saunders; Nummular eczema image is from Weston W, Lane A, Morelli J: *Color Textbook of Pediatric Dermatology*, ed 4, St. Louis, 2007, Mosby.

Documentation

Avoid the use of a specific diagnosis when describing a lesion in the objective physical findings, such as diaper rash or *candidiasis*, a

common fungal infection in infants. Accurate charting using the correct terminology and photos allows other health care providers to visualize the skin lesions and provide the necessary follow-up to evaluate a change or improvement in skin lesions. The wide use of smartphones with high-resolution digital cameras by individuals and families from all economic and social backgrounds makes the tracking of skin lesions and skin conditions at home a possibility for health care providers. The ability to digitally visualize lesions over time may prevent the child and family from returning to the clinical setting for frequent follow-up visits. In electronic medical record (EMR) systems, customizing a charting template for assessment of the skin with dermatological terms and adding a glossary of dermatological definitions provides accurate, consistent descriptions of skin lesions by different providers, and saves time when charting. Many EMR systems allow photos to be inserted into the medical record. These photos may be taken by the provider or by the family and submitted by text message or e-mail to the provider to enhance accurate diagnosis, treatment, and follow up.

Summary of examination

- Skin disorders may vary widely when observed in an infant or child, as compared with the same skin disorder when seen in an adult.
- Extremely premature infants are born without the *stratum corneum*, have limited ability to control water loss, and can tolerate a limited amount of touch.
- Term newborns have a fully functional stratum corneum, which is about 60% the thickness of adult skin.
- Skin pigmentation or melanin should be considered as a factor for understanding some underlying skin properties and characteristics.
- A careful age-appropriate history is critical to making an accurate assessment of the skin (see the Information Gathering table and Box 7.1).
- A skin lesion refers to any variations or skin changes. On inspection, note distribution or location of skin findings and pattern of skin lesions.

- Identify primary lesions, the initial lesion of a skin condition, and secondary lesions, which develop over time as a skin condition progresses.
- Palpation of the skin should be done with warm hands or with gloves when infection is suspected.
- Assessment of the skin must be conducted using a systematic approach that divides the skin into areas that are sequentially uncovered, examined, and then re-covered.
- Avoid making a quick diagnosis after only a brief inspection of exposed skin.
- A rash in children cannot be diagnosed by listening to a description over the phone.
- Accurate charting using the correct terminology and photos allows other health care providers to visualize the skin lesions and provide the necessary follow-up to evaluate change or improvement in skin lesions.
- When charting, avoid use of a specific diagnosis when describing a lesion in the objective physical findings.

DOCUMENTATION

14 month old with candidiasis

Skin: Discrete, red papules and pustules over the perineum with satellite lesions over the legs and abdomen; otherwise skin lightly pigmented and clear.

DOCUMENTATION

15 year old with moderate acne vulgaris

Skin: Moderate amount of open and closed comedones over nose and cheeks, discrete pustular lesions on forehead, no nodules or cyst noted. Skin oily with moderate papular, erythematous lesions over upper back.

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CHAPTER 8

Heart and vascular assessment

Patricia O'Brien, Julianne Evangelista

Embryological development

The heart begins to form in the fetus by the end of the third week after conception. A crescent-shaped structure is formed that fuses at the midline to create a single linear heart tube (Fig. 8.1). As the primitive heart tube elongates, it differentiates into the *atria*, *ventricles*, *bulbus cordis*, and *truncus arteriosus.* The conduction system also begins to form at this time. Valve formation begins around the fourth to fifth week after conception, and the formation of the heart is complete by the eighth week after conception. Any early changes in this process caused by genetic, maternal, or external environmental factors can lead to structural malformations of the heart.

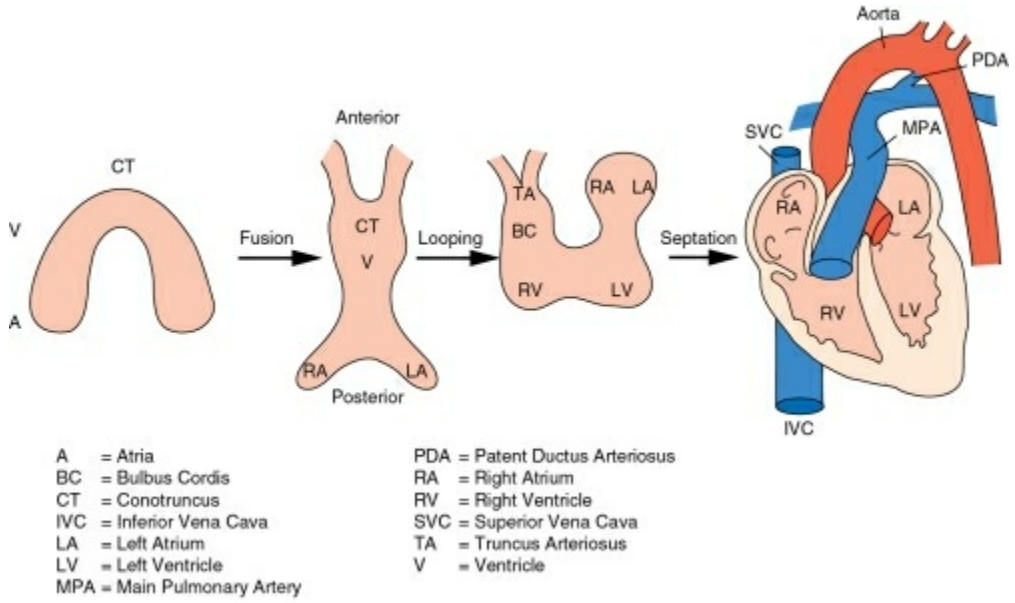


FIGURE 8.1 Fetal development of the heart.

During fetal life, the lung sacs are collapsed and blood is oxygenated through the placenta. Oxygenated blood travels from the placenta to the heart via the umbilical veins and *ductus venosus* to the *inferior vena cava* (IVC) and into the *right atrium* (RA). Blood then streams to the *left atrium* (LA) through a *patent foramen ovale* (PFO) and into the *left ventricle* (LV), which pumps it out the *aorta* (Fig. 8.2). The less saturated venous blood traveling from the *superior vena cava* (SVC) and *coronary sinus* also flows to the RA, but is directed toward the *right ventricle* (RV) and *pulmonary artery* (PA). High pulmonary vascular resistance limits blood flow into the lungs, which are not yet involved in ventilation, and redirects it through the *patent ductus arteriosus* (PDA) to the descending aorta and lower body.

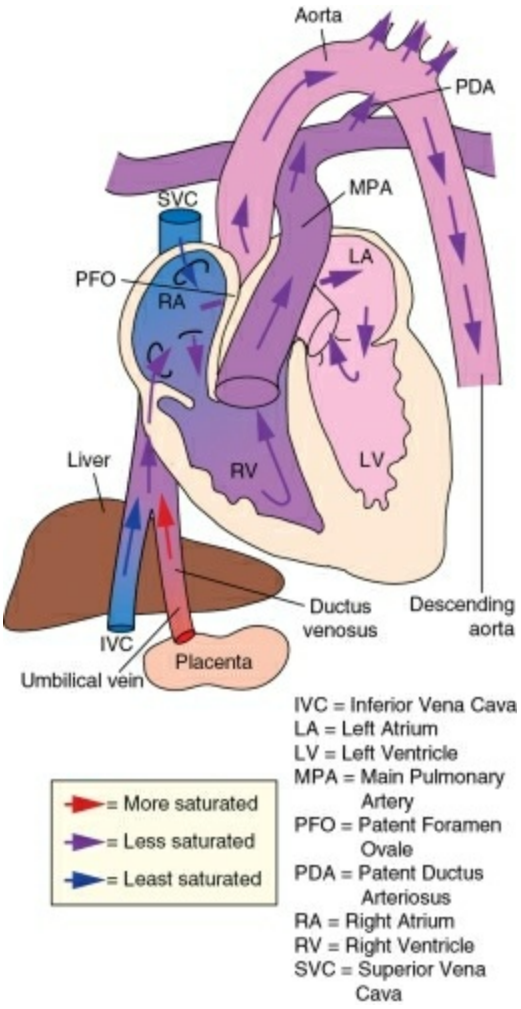


FIGURE 8.2 Fetal cardiac circulation.

With a baby's first breaths, pulmonary vascular resistance falls, causing a dramatic increase in pulmonary blood flow. The ensuing increase in pulmonary venous return to the heart raises LA pressure, causing closure of the PFO. Arterial oxygen saturation increases as a result of improved oxygenation by the lungs. This higher saturation promotes functional closure of the PDA by 48 to 72 hours after birth, with complete anatomical closure occurring by 2 to 3 weeks of age.[1]

Anatomy and physiology

Anatomy of the postnatal heart

The heart is composed of four chambers. The upper chambers (atria) are low-pressure receiving chambers, and the lower

chambers (ventricles) are high-pressure pumping chambers. The heart is further divided into right and left sides. The *RA* receives deoxygenated blood from the body, and the *RV* pumps it out the pulmonary artery to the lungs to become oxygenated. The *LA* receives oxygenated blood from the lungs, and the *LV* pumps it out the aorta to the body (Fig. 8.3). The LV operates at a higher pressure than the RV. This normal circulation occurs in series, and there is no mixing of deoxygenated and oxygenated blood.

There are four valves in the heart that regulate blood flow between the atria and ventricles. The atrioventricular (AV) valves regulate blood flow between the atria and ventricles, and the semilunar valves regulate blood flow between the ventricles and great vessels. The *tricuspid valve* on the right and the *mitral valve* on the left are the AV valves (see Fig. 8.3). The *pulmonic valve*, located at the base of the pulmonary artery between the right ventricle and the pulmonary artery, and the *aortic valve*, located at the base of the aorta between the aorta and LV, are the semilunar valves. Closure of these valves produces the heart sounds commonly referred to as "lub-dub" (S_1, S_2).

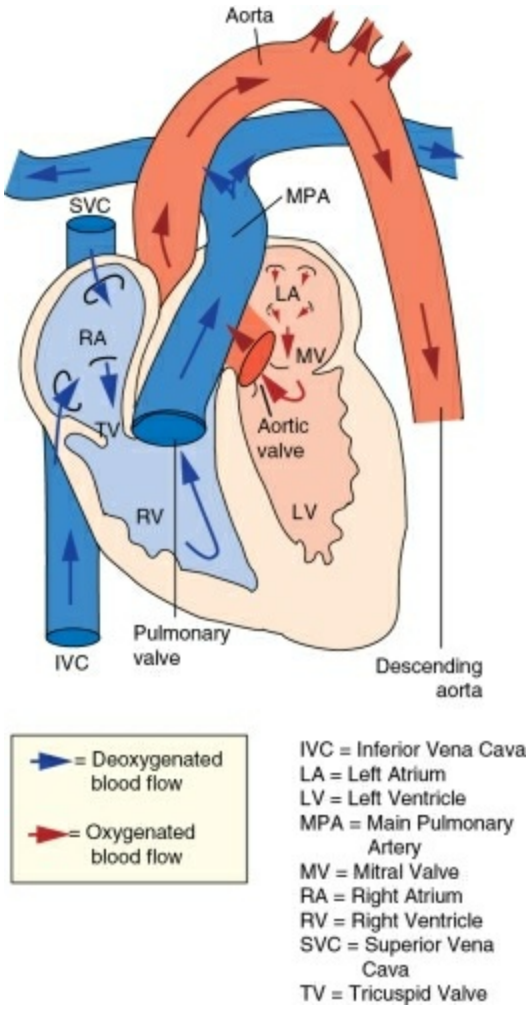


FIGURE 8.3 Postnatal cardiac circulation.

Physiological variations

In preterm infants, the ductus arteriosus may remain open for several weeks after birth, causing hemodynamic instability. Medical intervention with a prostaglandin inhibitor, such as the drug indomethacin, or surgical ligation of the ductus is required to stabilize the infant.

System-specific history

A careful and systematic history needs to be performed at each developmental stage to monitor the overall health status of infants and children, to identify those with cardiac symptoms, and to recognize signs of cardiac disease. The Information Gathering table presents the relevant age-related questions to ask about the

cardiovascular system and questions related to maternal infections and maternal medical conditions that place infants at high risk for *congenital heart disease* (CHD).

PEDIATRIC PEARLS

Infants with CHD may have tachypnea (rapid, shallow breathing) and tachycardia, but typically do not present in respiratory distress (i.e., retractions, grunting, nasal flaring) unless there is a significant increase in pulmonary blood flow or poor systemic output with acidosis.

Information Gathering for Assessing the Cardiovascular System at Key Developmental Stages

Age Group	Questions to Ask	Rationale
Prenatal, infancy, early childhood	Any family history of CHD/chromosomal abnormalities, sudden/premature death; maternal illness/infections (both chronic and during pregnancy); maternal medications/drug use	CHD caused by interaction between genetic and environmental factors (systemic lupus, rubella, diabetes mellitus, anticonvulsants, alcohol, etc.)
	Apgar scores if known	Usually normal with CHD except for color if cyanotic
	Any problems with poor feeding, sweating (especially on the forehead) during feeding, poor weight gain, FTT, decreased activity level	Symptoms of CHD often occur with feeding because of increased oxygen consumption and the need for greater cardiac output.
	Any pallor or blueness in color; any changes in color, especially when crying	Babies with cyanotic heart disease turn dark blue or ruddy in color when crying because of prolonged expiratory phase and resulting increase in right-to-left shunting. Hypercyanotic spells are often associated with extreme irritability and rapid, deep, and sometimes labored respirations.
	Any pattern of rapid breathing; frequent	Left-to-right shunting lesions (VSD, AVSD, PDA) cause increased blood flow to the

	respiratory infections	lungs, resulting in frequent respiratory infections, FTT, and decreased exercise tolerance.
Middle childhood and adolescence	Any inability to keep up with the activity level of peers, need for frequent periods of rest, anorexia, cough, wheezing, rales, chest pain, leg cramps, syncope, light-headedness, palpitations; any history of drug use; any family history of sudden death, syncope, or arrhythmias	CHD can decrease exercise intolerance. Left ventricular outflow obstructive lesions (AS, coarctation of the aorta) can cause CHF. Undetected coarctation of the aorta can cause leg cramps. Coronary artery abnormalities (including Kawasaki disease) and cocaine use can cause chest pain. Structural and dysrhythmic heart disease can first present as syncope. Some dysrhythmias are genetic in origin and run in families.
	Any history of recent infections, prolonged fever or malaise, recent dental work	Untreated streptococcal infections, Kawasaki disease, and subacute endocarditis can result in CHF or acquired heart disease.

AS, Aortic stenosis; *AVSD*, atrioventricular septal defect; *CHD*, congenital heart disease; *CHF*, congestive heart failure; *FTT*, failure to thrive; *PDA*, patent ductus arteriosus; *VSD*, ventricular septal defect.

Physical assessment

The cardiac examination must be systematic and tailored to the child's developmental level (Fig. 8.4). A complete assessment of the cardiac system must be done in order to make conclusions about the significance of any single abnormality. Cardiac findings should not be taken in isolation.

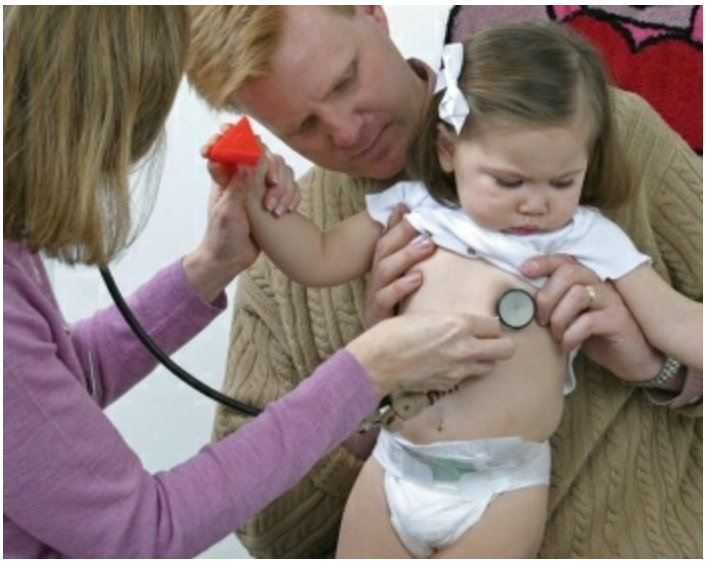


FIGURE 8.4 Cardiac examination of the toddler.

It is important to consistently plot height, weight, and head circumference in infants and children up to 2 years of age to evaluate whether the growth rate is proportional and to monitor for failure to thrive (FTT). For infants, any drop-off in weight percentiles as compared with length and head circumference values should raise the suspicion of CHD.[1] Temperature, heart rate, and respiratory rate should be measured and assessed. Fever and respiratory distress both can elevate heart rate.

Blood pressure should be measured whenever possible during physical examinations, but definitely once in infancy and then routinely after 3 years of age.[2] *Coarctation of the aorta* and systemic *hypertension* can go undetected if blood pressure measurements are omitted during well-child visits. If measured, blood pressure in infants should be taken in all four extremities or at a minimum in the right arm and in one leg to detect a coarctation of the aorta. In children, simultaneous palpation of the radial and femoral pulses is also important in assessing whether a coarctation may be present.

Inspection

Note general appearance and activity level and whether the child is alert, lethargic, or appears ill. Note nutritional status and the proportion of weight to height and head circumference. Also, note whether any unusual facial or other external features are present that may indicate the presence of a chromosomal syndrome or

congenital anomaly. Examine for any surgical scars on the sternum and chest area that would indicate a previous surgical procedure. Note shape of chest and any presence of *pectus excavatum* or *pectus carinatum* (see Chapter 9).

Color

Note whether the child is pale or cyanotic, and assess color under natural light if possible. Pallor can occur in infants who are anemic, who have vasoconstriction due to *congestive heart failure* (CHF), or who are in shock. Children with cyanotic heart disease will appear blue or ruddy, especially around the perioral area—tongue and mucous membranes—because of right-to-left shunting of blood at the arterial level.

Clubbing

Clubbing occurs when arterial desaturation has been present for at least 6 months or longer. The fingers and toes become red and shiny, and progress to wide, thick digits with eventual loss of the normal angle between the nails and the nail beds (Fig. 8.5).[2] With early surgical treatment of cyanotic heart defects in infancy, severe clubbing is an unusual finding.

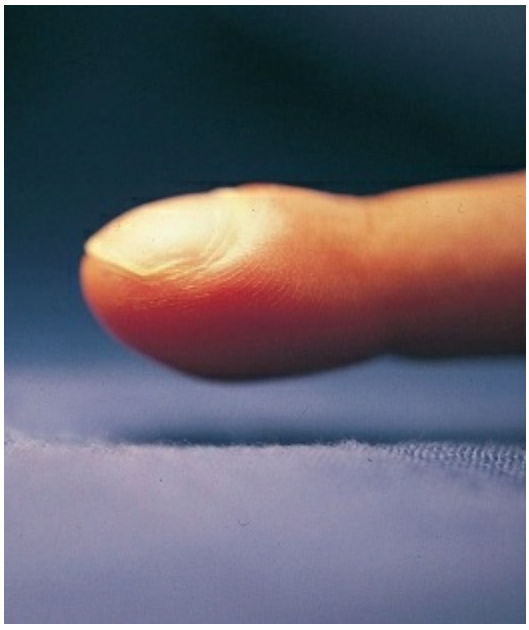


FIGURE 8.5 Clubbing of nails resulting from arterial desaturation. Source: (Modified from Hochberg MC, Silman AJ,

Smolen JS, et al.: *Rheumatology,* ed 3, St. Louis, 2003, Mosby.)

Palpation

Pulses need to be evaluated for their presence or absence, intensity, timing, symmetry, and whether the pulse is regular or irregular, weak or bounding. A comparison also should be made as to right and left symmetry and quality of pulses in the upper and lower extremities. A pulse that is absent or weaker in the lower extremities compared with the upper extremities is diagnostic of *coarctation of the aorta*. A strong pedal pulse is a good indication that there is no coarctation. Irregular pulses may be due to an *arrhythmia.* Weak and thready pulses may indicate poor perfusion or shock, whereas bounding pulses are usually noted with aortic runoff lesions such as a PDA, AV malformation, or aortic insufficiency.[2] *Peripheral perfusion* is also important to assess, especially in infants. Normally, extremities should be warm to the touch and have a brisk capillary refill time (CRT) of less than 3 seconds—a quick measure of cardiac output. Older children with cardiac conditions may have weak distal pulses on one side or the other because of previous cardiac catheterizations or cardiac surgeries.

Normal liver size is usually 1 to 2 cm below the right costal margin. In conditions of abnormal cardiac position, or situs (positional abnormalities), the liver edge is midline or on the left side of the abdomen. *Hepatomegaly*, or liver engorgement, is a consistent indicator of right heart failure when noted in conjunction with other cardiac findings.

The precordium should be palpated to determine the location of the *point of maximal impulse*, or PMI. The PMI is important in determining ventricular overload, cardiomegaly, and the presence or absence of thrills. Normally, the PMI is felt at the apex in the left midclavicular line, indicating LV dominance. However, it is normal for newborns and infants to have a greater RV impulse, with the PMI felt at the left lower sternal border (LLSB). A PMI that is diffuse and rises slowly is called a *heave*, and a PMI that is sharp and well localized is known as a *tap*.[2]

A *thrill* indicates turbulent blood flow and is never normal. It is

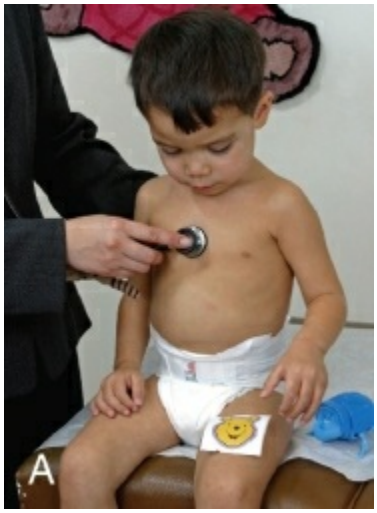
felt as a vibratory sensation on palpation and should be examined not only on the precordium but also in the suprasternal notch and over the carotid arteries. Precordial thrills are best felt with the palm of the hand, whereas thrills in the suprasternal notch and over the carotid arteries are best felt with the fingertips.[2]

PEDIATRIC PEARLS

Palpation of the liver for enlargement is a critical indicator of overall fluid status in infants and children with CHF.

Auscultation

Auscultation of heart sounds in children should be done in a stepwise fashion, and with both the diaphragm and the bell of a stethoscope to elicit both high (diaphragm) and low (bell) frequency sounds (Fig. 8.6). Children with a thin chest wall have heart sounds that are louder than in adults. However, the faster heart rate can make it difficult to accurately distinguish the heart sounds from other adventitious sounds, particularly in early infancy. In children who are overweight or obese, heart sounds are softer due to the increase in adipose tissue. Thus it is recommended that the individual heart sounds be identified first and then analyzed before identifying murmurs.



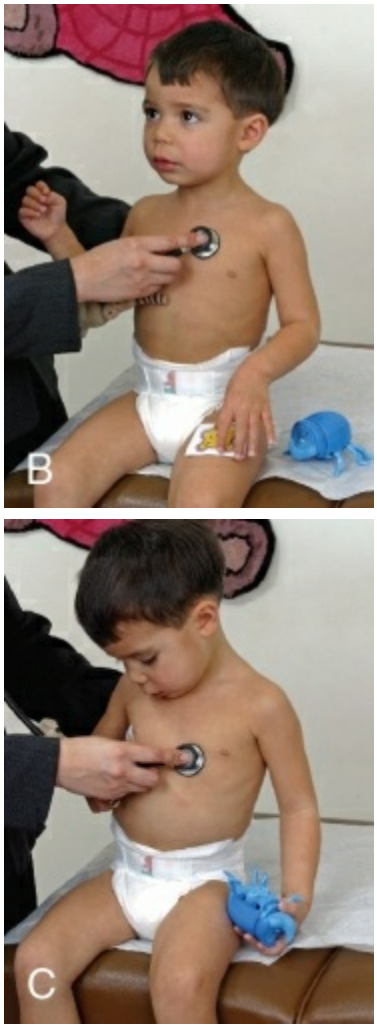


FIGURE 8.6 Auscultation of the heart sounds.

Heart sounds

The *first heart sound* is called S_1 and is created by the closure of the tricuspid and mitral valves. It is usually heard best at the LLSB or at the apex. A split S_1 can be a normal but uncommon finding in children.[2]

The *second heart* sound is called S_2 and is created by the closure of the aortic and pulmonic valves. It is usually heard best at the left upper sternal border (LUSB). Evaluation of S_2 is critical in children because it provides important clues as to the presence of structural defects and to the pressures in the heart. S_2 normally varies with respiration—split with inspiration and single or narrowly split in expiration. A fixed split, single S_2, or loud S_2 warrants referral and further evaluation by a cardiologist. Abnormal splitting of the S_2

may indicate increased pulmonary blood flow, pulmonary valve abnormality, or a cyanotic heart condition. A loud single S_2 may indicate pulmonary hypertension or malposition of the great arteries.[2]

A *third heart sound* (S_3) can be a common finding in children and young adults. S_3 can be heard at the apex and is caused by vibrations in the ventricle as it fills rapidly during diastole.

A *fourth heart sound* (S_4 or *gallop rhythm*) is rare in infants and children. An S_4 is an abnormal finding and suggests decreased ventricular compliance in conditions such as CHF.[2]

Ejection clicks are extra heart sounds that occur between S_1 and S_2. They are heard best at the upper sternal border and are usually associated with stenotic semilunar valves or dilated great arteries.[2]

Murmurs

Murmurs are produced when blood flows across an area that has a pressure difference and causes turbulence or disturbed flow. Murmurs should be assessed and evaluated according to their timing in the cardiac cycle, location, transmission, intensity, frequency, and quality.[2] It is always important to note whether a murmur radiates to the lung fields, axillae, clavicles, or neck. A normal grading scale is used to describe a murmur's intensity (Table 8.1).

TABLE 8.1

Grading Scale for Cardiac Murmurs

Grade	Sound
1	Barely audible and softer than usual heart sounds
2	Still soft, but about as loud as usual heart sounds
3	Louder than usual heart sounds, but without a thrill
4	Louder than usual heart sounds, and with a thrill
5	Can be heard with stethoscope barely on chest (rare)
6	Can be heard with stethoscope off chest, or with naked ear (extremely rare)

Data from Park M: *Pediatric Cardiology for Practitioners*, ed 6, St. Louis, 2014, Mosby/Elsevier.

Murmurs are described in relation to their timing during the cardiac cycle—systolic, diastolic, or continuous. Systolic murmurs occur between S_1 and S_2, and diastolic murmurs are heard after S_2. Systolic murmurs are further described as *ejection* crescendo-decrescendo, or *regurgitant* long systolic-decrescendo. Fig. 8.7 illustrates the difference between ejection and regurgitant murmurs in relation to when they occur in the cardiac cycle.

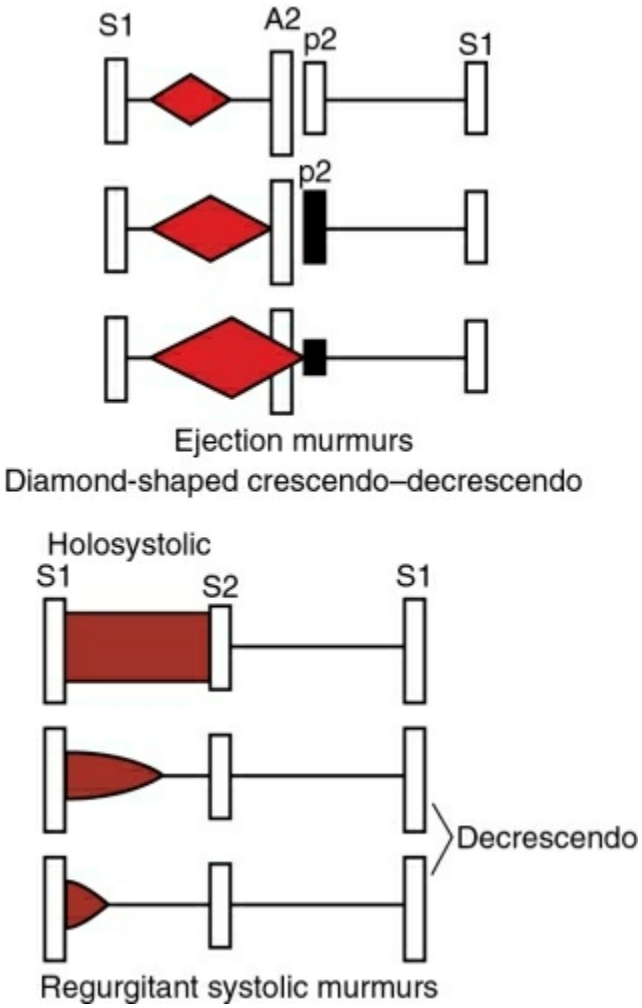


FIGURE 8.7 Systolic ejection and regurgitant murmurs. Source: (Modified from Park M: *Pediatric Cardiology for Practitioners*, ed 6, St. Louis, 2014, Mosby/Elsevier, p. 28.)

Systolic ejection murmurs begin shortly after the first heart sound, are due to semilunar valve or great vessel stenosis, usually vary in intensity, and are diamond-shaped. They can be short or long in duration but usually end before S_2. Regurgitant murmurs

typically begin with S_1, although they usually do not obscure it; are the result of mitral or tricuspid valve insufficiency; and can be long or short in duration. Holosystolic murmurs obscure S_1 at their maximal or loudest point and are usually caused by a *ventricular septal defect* (VSD).[2]

Diastolic murmurs occur between S_2 and S_1 and are described as early, mid, or late. Diastolic murmurs are usually caused by aortic or pulmonic regurgitation or mitral stenosis and are never normal.[2]

Continuous murmurs begin in systole and continue without interruption through S_2 and into diastole. They are usually caused by conditions in which vascular shunting occurs throughout the cardiac cycle, such as in PDA or a surgical aorta-to-pulmonary shunt. A continuous murmur from a PDA has a machinery-like quality, is best heard in the left clavicular area or back, and has a crescendo-decrescendo shape.[2]

The origin of a murmur is usually found at the point where the murmur is heard the loudest. The location of the murmur provides valuable information regarding the cardiac malformation. If a murmur is heard throughout the chest, the area of highest frequency will define its origin. For example, a systolic ejection murmur that radiates to the axillae and back is usually pulmonary in origin, and one that radiates to the neck and carotid arteries is typically aortic in origin. The frequency or pitch of a murmur is a good indicator of the pressure gradient across a valve or septal defect. The higher the pressure gradient, the higher the frequency of the murmur.

PEDIATRIC PEARLS

Innocent murmurs occur in up to 50% of normal children. [1] They are a common finding in infants and children, and do not always signify heart disease.

Physiologic versus pathologic murmurs

It is important to distinguish physiologic, or innocent, murmurs from pathologic murmurs (Table 8.2). Innocent murmurs occur in

up to 50% of normal children.[1] They are systolic ejection murmurs that are usually heard best at the LLSB and have a vibratory or musical quality (Fig. 8.8). They tend to be short and well located. They are usually no louder than grade 2 to 3 in intensity and are often accentuated during high output states such as exercise, stress, anemia, or febrile illness. Innocent murmurs are never purely diastolic except in the case of a venous hum, which is a continuous diastolic physiologic murmur. Innocent murmurs are usually not associated with a diastolic murmur, a thrill, abnormal electrocardiogram (ECG) or chest x-ray, cyanosis, or other symptoms of heart disease. Although innocent murmurs usually occur between 2 to 6 years of age, they also may be heard from infancy to early adolescence.

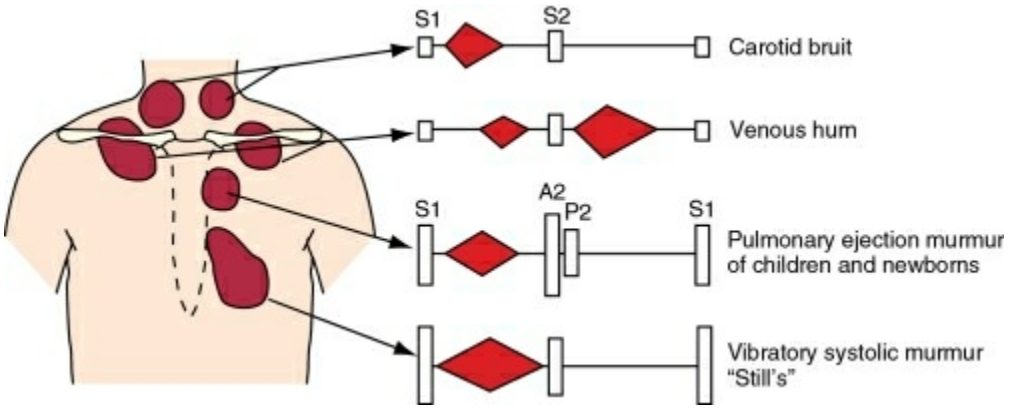


FIGURE 8.8 Anatomical locations of physiologic murmurs. Source: (Modified from Park M: *Pediatric Cardiology for Practitioners*, ed 6, St. Louis, 2014, Mosby/Elsevier, p. 37.)

TABLE 8.2
Physiologic or Innocent Murmurs

Murmur	Characteristics/Evaluation	Age of Occurrence
Still's murmur	Localized between LLSB and apex Grade 1-3/6 systolic ejection (outflow murmur), decreasing with inspiration, when upright, or disappearing with Valsalva maneuver Low frequency, vibratory, musical in quality Often confused with VSD murmur	Most commonly heard at 2–7 years old
Peripheral	Also known as *newborn pulmonary flow murmur*	Often heard in

pulmonic stenosis (PPS)	Heard at LUSB with radiation to back, axillae Grade 1-2/6 systolic ejection, crescendo-decrescendo	premature infants, infants with low birth weight, and infants up to 4 months of age Need to document resolution by 4–5 months of age to rule out organic cause or valve involvement
Pulmonary ejection	Well localized to LUSB Grade 1-3/6 systolic ejection crescendo-decrescendo Heard loudest when supine and decreases or disappears with Valsalva maneuver Does not radiate Similar to ASD murmur, but S_2 is normal	Common in 8- to 14-year-olds with greatest frequency in adolescents
Venous hum	Heard best just below clavicles at either RUSB or LUSB Grade 1-3/6 low-frequency continuous murmur Loudest when sitting, diminishes or disappears when supine; can be increased by turning patient's head away from the side of the murmur, and can be obliterated by light jugular vein compression; can be mistaken for PDA	Common in 3- to 6-year-olds
Supraclavicular carotid bruit	Heard above the right or left clavicle with radiation to the neck Grade 1-3/6 holosystolic, crescendo-decrescendo Decreases or diminishes with shoulder hyperextension Can be confused with murmur of aortic stenosis	Common at any age

Data from Park M: *Pediatric Cardiology for Practitioners*, 6th ed., St. Louis, 2014, Mosby/Elsevier.

ASD, Atrial septal defect; *LLSB*, left lower sternal border; *LUSB*, left upper sternal border; *PDA*, patent ductus arteriosus; *RUSB*, right upper sternal border; *VSD*, ventricular septal defect.

Common diagnostic tests

Pulse oximetry

Pulse oximetry is used to verify and document the degree of central cyanosis and is an accurate way to assess arterial oxygen saturation, especially in infants. Using pulse oximetry to screen newborns in the well infant and intermediate care nurseries for critical CHD was

added to the US Recommended Uniform Screening Panel in 2011 and has been adopted by 46 states.[3] In addition to screening for 12 or more congenital heart defects, the identification of infants with other causes of hypoxia, such as infections, lung disease, or hemoglobinopathies, has been documented as an added benefit of pulse oximetry screening.[4]

Pulse oximetry readings from the right hand and one foot (together or in sequence), taken on the second day of life before discharge, and using a motion tolerant pulse oximeter approved by the US Food and Drug Administration are recommended.[5] Box 8.1 presents the criteria for a positive result for pulse oximetry screening. A positive screening result is defined as any identified low oxygen level, and requires follow-up with a comprehensive history and physical examination to determine the cause of hypoxia. Critical CHD is excluded using a diagnostic echocardiogram, and it should be read and interpreted by a pediatric cardiologist.[6]

BOX 8.1

Criteria for a Positive Pulse Oximetry Screening Result

- Any oxygen saturation measure <90%
- Any oxygen saturation <95% in both extremities on three measures taken 1 hour apart
- Greater than 3% absolute difference between readings in the upper extremity (right hand) and lower extremity (foot) on three measures taken 1 hour apart (e.g., 96% right hand and 93% foot, or 88% right hand and 92% foot)

Data from Kemper AR, Mahle WT, Martin GR, et al: Strategies for implementing screening for critical congenital heart disease, *Pediatrics* 128(5):e1259–e1267, 2011.

Pulse oximetry is also commonly used to monitor oxygen saturations in children with known cardiac disease. Infants and

children who have complex cyanotic heart defects in which blood mixes in a common ventricle, or who are dependent on a surgically placed aorta-to-pulmonary artery shunt to supply pulmonary blood flow, normally have oxygen saturation levels between 75% and 85%. It is important to know an infant's baseline oxygen saturation to accurately assess their oxygen status. Increasingly, pulse oximeters are used at home to monitor changes in oxygen saturation in infants with hypoplastic left heart syndrome (HLHS) and other single-ventricle (SV) heart defects as part of home surveillance programs.[7]

Chest x-ray

Chest x-rays have been the standard to assess heart size, pulmonary congestion, position of the great vessels, and abdominal situs. Although this diagnostic tool is frequently used in the inpatient or ICU setting, it is becoming less practical and valuable in the outpatient setting with the advanced echocardiography, which gives more precise information of the cardiac structure and function without radiation exposure.

Heart size is determined by comparing the width of the cardiac silhouette at its widest diameter to the width of the chest at its maximal internal dimension (Fig. 8.9). This is referred to as the cardiothoracic (CT) ratio. A CT ratio greater than 0.65 is considered cardiomegaly.[2] It is important to have a good inspiratory x-ray for diagnosis, as an expiratory film may make the heart appear larger than it is. Also, prominent thymic tissue in newborns is often falsely interpreted as cardiomegaly.

The position of the cardiac apex provides information about ventricular enlargement. Normally the apex points down and to the left. An upward turned apex is indicative of right ventricular enlargement, whereas an apex that is pushed more downward and leftward than normal is caused by left ventricular enlargement. The *main pulmonary artery* (MPA) is normally seen as a small knob at the LUSB (see Fig. 8.9). The prominence or absence of this shadow provides clues about the size, position, and presence of the MPA.[2] Pulmonary vascular markings provide important information about the degree of pulmonary blood flow and should be noted as normal, increased, or decreased.

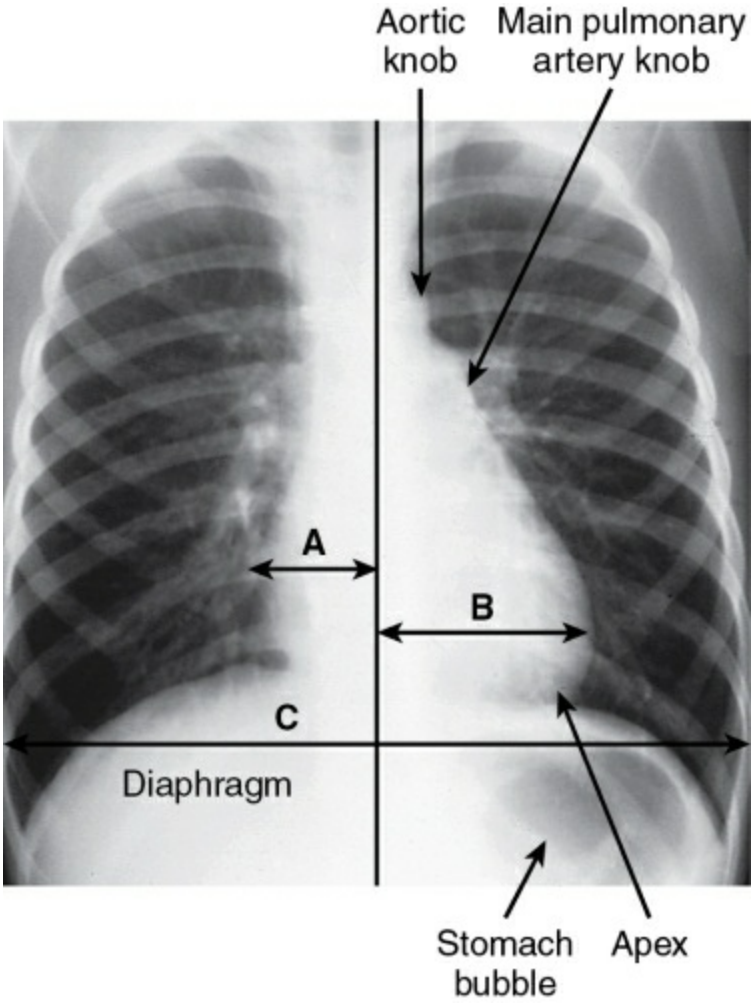


FIGURE 8.9 Anatomical landmarks for chest radiographs. Source: (Modified from Zitelli BJ, McIntire SC, Nowalk AJ: *Zitelli and Davis' Atlas of Pediatric Physical Diagnosis*, ed 6, Philadelphia, 2012, Elsevier; Park M: *Pediatric Cardiology for Practitioners*, ed 5, St. Louis, 2008, Mosby, p. 66.)

Chest x-rays can also determine abdominal situs. The location of the cardiac apex is normally on the left and should be on the same side as the stomach bubble and opposite the liver shadow. When these structures are misaligned (with the stomach bubble on the right and the apex on the left), or the liver is midline, then heterotaxy or situs abnormality is present, which is often associated with serious heart defects.

Electrocardiogram

An ECG is a screening tool that provides information about heart rate, heart rhythm, conduction, and forces of contraction in the

heart. It is particularly useful in the diagnosis of arrhythmias and ventricular hypertrophy. However, ECG patterns can vary within diagnostic groups. ECGs can be a sensitive test they do not always identify or rule out cardiac disease. **Therefore, except for arrhythmias, ECGs are used most often to confirm a diagnosis of structural CHD as opposed to establishing one.**

Echocardiogram

Cardiac ultrasound, or *echocardiogram*, has become the primary diagnostic tool for patients suspected of having CHD. An echocardiogram can be done safely and noninvasively in the outpatient setting or at the bedside and provides accurate information for all age groups including premature infants. The addition of Doppler ultrasound and three-dimensional (3D) images makes it possible to evaluate valve anatomy, function, and flow patterns throughout the heart and proximal vessels. This is particularly useful in quantifying degrees of shunting and obstruction. Advanced fetal cardiac echocardiography has become a standard prenatal evaluation tool for parents with a family history of CHD or other risk factors. Echocardiograms are expensive, however, and should only be ordered by a cardiologist after a thorough evaluation has been done to determine whether one is warranted. Referral to a cardiologist should be as complete as possible and include data about the history, physical examination, chest x-ray, ECG, and oxygen saturation that have raised suspicion of CHD. Table 8.3 presents the common tests used to diagnose CHD.

TABLE 8.3
Overview of Common Diagnostic Tests in CHD

Test	Applications/Specific Modalities
Chest x-ray	Information on heart size and shape, enlargement of cardiac chambers, size and position of the great vessels, degree of pulmonary blood flow, position of abdominal organs
Electrocardiogram (ECG)	Graphic measure of electrical activity with information on rhythm, conduction, and force of contraction, chamber hypertrophy **Holter:** 24-hour continuous ECG recording used to assess arrhythmias

Echocardiogram	Noninvasive imaging using high frequency ultrasound to assess cardiac structures and ventricular function **Transthoracic echocardiogram (TTE):** Transducer is on the chest, most common. Infants and toddlers may need sedation for full study. • *M-mode:* One-dimensional graphic display to estimate chamber size and ventricular function, assess valve motion and pericardial fluid • *2-D:* Real-time cross-sectional images to assess cardiac anatomy • *3-D:* Imaging primarily to assess valve anatomy • *Doppler:* Demonstrates blood flow patterns and pressure gradients **Transesophageal echocardiogram (TEE):** Transducer placed in esophagus to obtain images of posterior cardiac structures, used in patients with poor thoracic imaging. Widely used in the operating room after cardiac surgical repairs. Patient must be sedated or under general anesthesia. **Fetal:** Images fetal cardiac structures in utero
Cardiac MRI	Provides real time 3D imaging and measurement of intracardiac structures and extracardiac vascular anatomy, assesses function • Often used in adolescents or young adults with limited imaging by echo. Takes an hour or more in a confined magnet. • Children younger than 8 years of age or those with claustrophobia or anxiety may need anesthesia or sedation.
Cardiac computerized tomography (chest CT, cardiac CT)	Computer-processed x-rays to obtain detailed images of the heart and chest structures. • Recent improvements in technology (decreased image acquisition time and decreased radiation exposure) has increased the use of chest CT. • Used to evaluate aortic arch abnormalities, vascular rings, coronary arteries, and pulmonary veins
Exercise (stress) test	Monitors heart rate, blood pressure, ECG, oxygen saturation, oxygen consumption, and presence of symptoms (i.e., chest pain, dizziness) at rest and with progressive exercise on bicycle or treadmill • Done with physician supervision and emergency equipment available • 6-minute walk test: Assess maximum distance walked in 6 minutes while monitoring heart rate and oxygen saturation; used in patients with severe exercise limitations or those with pulmonary HTN.
Cardiac catheterization	Invasive imaging modality using radiopaque catheters placed in peripheral vessels (femoral access most common) and advanced into the heart to visualize cardiac structures, measure chamber pressures and oxygen levels, and assess blood flow patterns **Hemodynamics:** Assessment of pressures and oxygen levels in cardiac chambers **Angiography:** Injection of contrast material to image heart structures and flow patterns under fluoroscopy **Biopsy:** Bioptome catheters used to obtain tiny samples of heart muscle for microscopic examination; used to assess for infection, inflammation or muscle dysfunction disorders, and posttransplant rejection **Electrophysiology:** Special catheters with electrodes used to record electrical activity from inside the heart and assess rhythm abnormalities

CHD, Congenital heart disease; *HTN,* hypertension; *MRI,* magnetic resonance imaging.

Cardiac symptoms

Arrhythmias and palpitations

It is not unusual for children to complain of skipped heartbeats, a fast heart rate, or extra heartbeats. Most complaints are benign in origin, but a history of chest pain, light-headedness, or syncope can be indicative of a serious arrhythmia.[2] Family history should be directed toward any structural heart disease or history of sudden death, and the patient history should include any association of symptoms with exercise, food intake (especially caffeine), medications (especially cough preparations, and stimulants), or specific positioning. Physical findings, except the arrhythmia, are usually not present. Evaluation begins with an ECG. Documentation of the cardiac rhythm on an ECG is essential for determining diagnosis and treatment; however, obtaining this documentation can be challenging, especially if the symptoms are infrequent. For a complete evaluation, referral to a pediatric cardiologist or electrophysiologist is essential, and if the symptoms persist or worsen, additional testing with continuous Holter monitoring or event recording is recommended.

Smartphones are another way to monitor heart rhythms. There are several smartphone applications that can be used to document subjective complaints of palpitations. Depending on the application, the information can be programmed to forward the data to a pediatric health care provider or become a personal ECG monitor. In 2013 the AliveCor application was Food and Drug Administration approved for detection of atrial arrhythmias. The device accurately detects an irregular pulse by analyzing data signals recorded with an iPhone.[8] Additionally, even personal fitness trackers, like Fit Bit, Garmin, and Fuelband, are helpful in monitoring objective heart rates in older children and adolescents.

Syncope

Syncope is a common complaint in older children and adolescents (especially adolescent females).[2] *Vasovagal syncope*, also called *simple syncope* or *simple faint or neurocardiogenic syncope* or *vasodepressor syncope*, is the most common cause of syncope in children and

adolescents. It is usually characterized by a loss of consciousness, falling, and then a quick recovery once the child or adolescent is lying down. The episodes are usually preceded by dizziness, light-headedness, pallor, weakness, blurred vision, or cold sweats. The majority of syncopal events are benign, but a careful evaluation is always warranted, because it may be the first symptom of serious cardiac, neurologic, or metabolic disease. Syncope is unusual in children less than 6 years of age unless it is related to seizures, breath-holding, or cardiac arrhythmias.[2] The most common neurological cause is a seizure disorder, and possible metabolic causes include hypoglycemia, electrolyte imbalance, or profound anemia. In toddlers, the causes include breath-holding, and in adolescents, hyperventilation techniques.

Arrhythmias, long QT syndrome, and *hypertrophic cardiomyopathy* (HCM) are other potential cardiac causes of syncope. Information gathering should include careful details of the event and a family history of similar events or sudden cardiac death. Medication history and past medical history are important to elicit. Often the only evidence of HCM on physical examination may be the increased intensity of a cardiac murmur from supine to standing. If the neurological examination is negative, an ECG should be obtained. Referral to a cardiologist is warranted if a murmur is heard on physical exam, if there is an abnormal ECG, a family history of sudden death, or cardiomyopathy.[2]

Chest pain

Chest pain in children is a common complaint, causing anxiety in both patients and their parents. However, chest pain due to a cardiac cause is rare, occurring in less than 4% of cases.[9] The most common cause of chest pain is musculoskeletal in origin, including muscle strain, trauma, and costochondritis, which accounts for close to 70% of cases of chest pain in children.[10] *Costochondritis* is an inflammation of the chest wall, causing sharp, short, and well-localized pain that can be reproduced with pressure on palpation. Other causes of chest pain in children are pulmonary (especially asthma or illnesses associated with coughing), gastrointestinal (esophagitis or gastroesophageal reflux), or psychogenic (less often in children less than 12 years of age but more frequent in adolescent

females).[2]

A careful history is important in assessing the cause of chest pain in children (Box 8.2). It is important to elicit the onset and duration of the pain, and how long the child has been experiencing pain, if the chest pain occurs at rest or only with exercise and sports participation. Document the severity, location, and radiation of the chest pain. A family history of sudden death, CHD, cardiomyopathy, or a hypercoagulable state is concerning for cardiac disease. Chest pain associated with fever may have an infectious cause. Illicit drug use, particularly cocaine, can cause myocardial ischemia with chest pain as a presenting symptom.

BOX 8.2

Information Gathering for Presenting Chest Pain in Children

Questions to ask:

- Has the child had a fever or recent illness?
- Is the pain related to breathing or activity, does it occur with exercise or at rest?
- Any sports activities or injuries that may have preceded the onset of chest pain?
- Is the chest pain accompanied by palpitations, dizziness, or fainting?
- Anything improve the pain? Anything make the pain worse?
- Any illicit drug use or use of caffeine supplements or drinks?

The physical exam should note the child or adolescent's color, perfusion, pulses, respiratory effort, and degree of acute pain. Auscultation includes the evaluation of breath sounds and their symmetry, as well as the identification of abnormal heart sounds, murmurs, rubs, gallops, or muffled heart sounds.

Cardiac origins of chest pain include ischemic causes (coronary artery abnormalities or cardiomyopathies), inflammatory

conditions such as pericarditis or myocarditis, arrhythmias, aortic dissection, or pulmonary embolus.[10] *Pericarditis*, an inflammation of the pericardium, is characterized by chest pain that is worse lying down and improves with sitting and leaning forward.[2] Careful cardiac history, family history, physical examination, and selected use of the ECG can identify potentially life-threatening or other nonemergent causes of cardiac chest pain, which require further evaluation.[10] If acute chest pain of cardiac origin is suspected, an ECG should be obtained, with immediate referral to a pediatric cardiologist.

PEDIATRIC PEARLS

Chest pain with exertion, associated with dizziness, fainting, or palpitations, and chest pain that radiates to the back, jaw, or left arm could be indicative of cardiac disease and requires prompt evaluation by a cardiologist. [10]

Cardiovascular disease

Cardiovascular disease in children is normally divided into two categories: congenital or acquired. CHD comprises the majority of cardiovascular disorders in the pediatric population. CHD is not a singular entity, but rather a myriad of structural anomalies in the heart that develop during fetal life and thus present at birth. *Acquired heart disease* (Kawasaki disease, HCM, myocardial infections, rheumatic fever, hypertension) occurs after birth and develops during an individual's lifetime.

Congenital heart disease

CHD is prevalent in at least 10 per 1000 live births, close to 1% of all births in the United States.[11, 12] Forty percent of CHD malformations are diagnosed during a child's first year of life. Adults now account for two-thirds of patients with severe and other chronic forms of CHD in the general population.[12] Maternal infections such as rubella, coxsackievirus, and other viruses contracted during pregnancy can be associated with CHD or myocarditis.

Medications, alcohol, and other drugs may act as teratogens on the developing fetus. Maternal medical conditions associated with an increased risk of the fetus developing CHD include diabetes mellitus (cardiomyopathy, transposition of the great vessels) and systemic lupus erythematosus (congenital heart block).[2] See Table 8.4 for common presentations of CHD in children.

TABLE 8.4
Common Presentations of Congenital Heart Disease

Presentation	Physiology	Signs and Symptoms	Potential Diagnoses
Cyanosis (infants)	• Central, arterial (vs. peripheral) desaturation • Desaturated blood mixes with saturated blood in the heart due to right-to-left shunting	• Bluish or deeply ruddy color around mouth/lips • Usually not visible unless O_2 saturations are <85% • O_2 saturations for infants with cyanotic heart disease are normally 75%–85% • Supplemental O_2 will not raise saturations to normal levels of 95%–100% • Hct usually higher than normal • Cyanosis not evident if patient is anemic and will be greater with polycythemia • Can be tachypneic without respiratory distress	• TOF • PA
Congestive heart failure (CHF)	• Heart is unable to meet metabolic demands (output) of the body • Etiologies can be both cardiac and non-cardiac-related	• Tachycardia • Tachypnea with or without respiratory distress (grunting, wheezing, rales) • Cough, wheezing, rales (older children) • Gallop rhythm • Hepatomegaly (liver palpable >2 cm below right costal heart margin) • Peripheral edema plus hepatomegaly (older children) • Pallor due to vasoconstriction • Poor feeding, failure to thrive (infants) • Sweating, especially on forehead, during feeding (infants) • Anorexia, somnolence (older children) • Frequent respiratory infections • Fatigue, exercise intolerance, inability to keep up with peers	• VSD • HCM • Decreased ventricular function post-CHD surgical repair or intervention • Arrhythmia • Myocardial infection • Anemia
Shock	• Severe obstruction of blood flow out of the heart • Can occur suddenly in newborn infants with undetected coarctation of the aorta or interrupted aortic arch when the PDA closes at 2–3 weeks of age	• Hypotension • Extreme pallor • Poor ventricular function, circulation • Hypovolemia • Weak pulses • Poor urine output	• Coarctation of the aorta • Interrupted aortic arch

CHD, Congenital heart disease; *HCM*, hypertrophic cardiomyopathy; *PA*, pulmonary atresia; *PDA*, patent ductus arteriosus; *TOF*, tetralogy of Fallot; *VSD*, ventricular septal defect.

CHD is caused by the interaction between genetic and environmental factors, with single gene mutations accounting for 3% of CHD, gross chromosomal anomalies accounting for 5%, environmental factors (rubella, fetal alcohol syndrome, other maternal illness/infections) accounting for 3%, and multifactorial genetic random event mediation for the remainder.[1] Genetic etiologies include genetic syndromes, deletion/duplication syndromes, and both syndromic and nonsyndromic single gene

disorders (i.e., *NKX2.5*). At least one or more additional congenital malformations can be found in 20% to 30% of infants with CHD.[1] Although knowledge and understanding of the role of genetics in CHD has advanced significantly, it is still an evolving science, and current data can change or become quickly outdated. Table 8.5 presents some examples of the more common syndromes associated with CHD diagnoses.

TABLE 8.5

Common Pediatric Syndromes Associated with Congenital Heart Disease

Type	Clinical Entity	% CHD	Associated CHD Diagnosis
Chromosomal	Trisomy 21 (Down syndrome)	40–50	AVSD, VSD, ASD, PDA, TOF
	Deletion 22q11 (DiGeorge syndrome; velocardiofacial syndrome [VCFS])	75	IAA-B, TA, aortic arch abnormalities, TOF, VSD
	Deletion 7q11.23 (Williams-Beuren syndrome)	50–85	Supravalvar or Supravalvular AS (SVAS) and PS, PPS
	Deletion 20p12 (Alagille syndrome)	85–94	PPS, pulmonary artery hypoplasia, PS, TOF
	Turner syndrome (45 XO)	25–35	CoA, BAV, valvar or valvular AS, aortic dissection, mitral atresia, HLHS
Single gene disorders	CHARGE (Hall-Hittner syndrome)	60-90	Conotruncal malformations, ASD, VSD, PDA
	Holt-Oram syndrome	75	Secundum ASD, VSD, progressive AV conduction delay
	LEOPARD syndrome	85	PS, HCM, rhythm abnormalities
	Marfan syndrome and other connective tissue disorders	80	Aortic dilation/dissection, MVP, MPA dilation, TVP

Data from Ruppel K: Disorders of the cardiovascular system. In Rudolph C, et al., editors: *Rudolph's Pediatrics*, ed 22, New York, 2011, McGraw-Hill.

AS, Aortic stenosis; *ASD,* atrial septal defect; *AV,* atrioventricular; *AVSD,* atrioventricular septal defect; *BAV,* bicuspid aortic valve; *CoA,* coarctation of the aorta; *HCM,* hypertrophic cardiomyopathy; *HLHS,* hypoplastic left heart syndrome; *IAA-B,* interrupted aortic arch type B; *MPA,* main pulmonary artery; *MVP,* mitral valve prolapse; *PDA,* patent ductus arteriosus; *PPS,* peripheral pulmonic stenosis; *PS,* pulmonic stenosis; *TOF,* tetralogy of Fallot; *VSD,* ventricular septal defect.

Common presentations of congenital heart disease

Cyanosis in infants

Cyanosis indicative of CHD is due to arterial desaturation and may not be visible unless the oxygen saturation is 85% or less.[1, 2] Arterial desaturation or *central cyanosis* is best detected in the perioral area, the mucous membranes of the mouth, lips, and gums. Central cyanosis should be distinguished from *peripheral cyanosis*, which can

occur in a cold environment, and *acrocyanosis*, which in newborns is due to sluggish circulation in the fingers and toes.[1]

PEDIATRIC PEARLS

The intensity of cyanosis is dependent on the concentration of desaturated hemoglobin and not on the actual arterial oxygen saturation.

An infant who has *polycythemia*, an abnormal increase in circulating erythrocytes, will appear more cyanotic than an infant who is anemic in the presence of the same degree of arterial desaturation. Therefore, it is important to follow a cyanotic infant's hemoglobin (Hgb) and hematocrit (Hct) levels, particularly at 2 to 3 months of age, the time of normal physiological anemia.

Congestive heart failure

CHF is the inability of the heart to adequately meet the metabolic demands of the body. It occurs in 15% to 25% of children with CHD, and about 40% of patients with cardiomyopathy can have CHF so severe that it results in the need for heart transplantation or results in death.[13] The causes of CHF can be related to both cardiac disease (VSD, AV septal defect, aortic stenosis, coarctation of the aorta, cardiomyopathy, myocarditis, arrhythmia, and Kawasaki disease) and noncardiac disease (anemia, sepsis, hypoglycemia, and renal failure). The multiple causations of CHF manifest in a variety of clinical presentations that are usually age specific. Infants most commonly present with pallor, tachycardia, tachypnea, poor feeding, FTT, and hepatomegaly. Older children are often unable to keep up with their peers, and may exhibit peripheral edema, hepatomegaly, anorexia, and respiratory symptoms such as cough, wheezing, and rales.[13]

Acquired heart disease

Hypertension

The incidence of *hypertension* (HTN) is increasing in children and

adolescents in the United States—in large part related to the increase in childhood obesity.[14] HTN is more prevalent in non-Hispanic blacks and Mexican-American youth, and it occurs more often in males than females.[14] There is now evidence that blood pressure in childhood tracks into adulthood, so children who are hypertensive in their younger years are frequently hypertensive as adults.[15] Screening for HTN in childhood and effective management is aimed at decreasing the risk of cardiac disease in adulthood. The definition of HTN is derived from blood pressure percentiles based on gender, age, and height (Table 8.6).[16] Chapter 2 reviews the proper method for obtaining blood pressure measurements, and both the systolic and diastolic values are of equal importance.

TABLE 8.6
Classifications of BP and Hypertension

Classification	Criteria
Normal	Both systolic and diastolic BP are <90th percentile
Elevated hypertension	Systolic and/or diastolic BP ≥90th percentile but <95th percentile OR BP exceeds 120/80 mm Hg
Stage 1 HTN	Systolic and/or diastolic BP measures ≥95th percentile measured on three separate occasions OR BP exceeds 130/80 mm Hg
Stage 2 HTN	Systolic and/or diastolic BP ≥ 95th percentile OR BP exceeds 140/90 mm Hg

Data from Flynn JT, Kaelber DC, Baker-Smith CM, et al, Subcommittee on Screening and Management of High Blood Pressure in Children. Clinical practice guidelines for screening and management of high blood pressure in children and adolescents. *Pediatrics* 140:1-72, 2017.

BP, Blood pressure. *HTN,* Hypertension.

HTN can be classified as primary or essential (without a clear cause) or secondary to an underlying disorder (often related to renal or vascular disease). In secondary HTN, treatment of the underlying cause can cure the HTN. Secondary HTN has a higher incidence in prepubertal children and likely underlies the more severe or stage 2 HTN. Primary or essential HTN is more often seen in older school-age children and adolescents and is often associated

with excess weight gain, obesity, and/or a positive family history of HTN.[14]

The goal of HTN screening is to identify children and adolescents with HTN and distinguish between primary and secondary causes. Evaluation of HTN includes a comprehensive history and physical examination, as well as baseline laboratory studies including complete blood count, serum electrolytes, blood urea nitrogen, creatinine, lipids, glucose, and urinalysis. Renal and cardiac ultrasounds are performed to assess for secondary causes or to document end-organ involvement such as increased left ventricular mass. An additional diagnostic tool is a 24-hour ambulatory blood pressure monitor. The device automatically measures blood pressures every 20 to 30 minutes over a 24-hour period while patients undergo normal daily activities. The portable device uses an oscillometric technique to document mean systolic and diastolic blood pressure measurements. The device is very helpful in identifying "white coat hypertension" or masked hypertension versus essential hypertension.

It is also important to identify those with other risk factors for premature atherosclerosis such as HTN, smoking, overweight/obesity, dyslipidemia, family history of premature cardiovascular disease, diabetes, and chronic renal disease. The target blood pressure for children with other cardiovascular risk factors is a systolic and diastolic blood pressure less than 90% (see Table 8.6).[16]

EVIDENCE-BASED PRACTICE TIP

Both blood pressure and lipid levels track from childhood to adulthood. Elevated blood pressure and elevated lipid levels in childhood frequently remain elevated in adulthood.[14, 15]

Dyslipidemia

Recent evidence supports the correlation between lipid disorders in childhood and the onset and severity of atherosclerosis in children and young adults.[14] The National Health and Nutrition Examination Survey (NHANES) indicate that 7.8% of children aged

8 to 17 years have elevated levels of total cholesterol (≥200 mg/dL), and 7.4% of adolescents aged 12 to 19 years have elevated low-density lipoprotein (LDL) cholesterol (≥130 mg/dL).[17] Like HTN, elevated lipid levels track from childhood to adulthood. In the past, children with a strong family history of lipid disorders or premature cardiovascular disease were screened for lipid disorders, but the increase in childhood obesity has contributed to a larger population of children at risk for dyslipidemia. Dyslipidemia, genetic factors, and childhood obesity are contributing factors to coronary artery atherosclerosis along with diabetes, nephrotic syndrome, chronic renal disease, postcardiac transplant, history of Kawasaki disease with aneurysms, and chronic inflammatory disease.

The US Preventive Services Task Force (USPSTF) recent recommendation concluded there was insufficient evidence for routine lipid screening in children and adolescents; however, the American Academy of Pediatrics (AAP) continues to recommend routine screening between 9 to 11 years of age and at 17 to 20 years of age, with the initial screen either a fasting or nonfasting lipid profile measuring total cholesterol and high-density lipoprotein (HDL) cholesterol.[14, 17, 18] Non-HDL cholesterol level is then calculated from these two values (non-HDL cholesterol = total cholesterol – HDL cholesterol).[14] Normal values are a non-HDL cholesterol value less than 145 mg/dL, with an HDL cholesterol above 40 mg/dL. If the non-HDL cholesterol is abnormal, children should have a fasting lipid profile and further evaluation and follow-up.

Metabolic syndrome

Metabolic syndrome, a combination of dyslipidemia, abnormal glucose regulation, obesity, and hypertension, has been described in obese adults and is known to significantly increase the risk for coronary artery disease and type 2 diabetes.[18] Research suggests a strong interaction between obesity, insulin resistance, and inflammatory markers which contribute to metabolic syndrome and later heart disease and diabetes. Metabolic syndrome in adults is often defined as the presence of three or more of the following risk factors: elevations in waist circumference, triglyceride levels, blood

pressure, reduced levels of HDL cholesterol, and/or fasting glucose levels.[14]

With the increased incidence of obesity in children and adolescents, a similar constellation of conditions is being described as metabolic syndrome in children. There is not universal agreement on the diagnostic criteria for *pediatric metabolic syndrome*, but the International Diabetes Federation has proposed a pediatric definition (Table 8.7).[19] Some of the risk factors for the development of metabolic syndrome in childhood include a family history of a parent with metabolic syndrome, Hispanic background, inactivity and sedentary lifestyle, and smoking.[18] The presence of obesity should prompt a comprehensive evaluation for other cardiovascular risk factors, including family history of premature cardiovascular disease, hypertension, dyslipidemia, diabetes, and tobacco exposure.[15] Longitudinal studies have demonstrated an increased incidence of both type 2 diabetes and cardiovascular disease when elements of metabolic syndrome are present in childhood.[20] The International Diabetic Foundation (IDF) consensus group recognizes that there are ethnic, gender, and age differences, and more research is needed to establish outcomes in high risk populations.[18] Preventive treatment for pediatric metabolic syndrome includes weight loss, diet, and exercise.

TABLE 8.7

Definition of Metabolic Syndrome in at-Risk Children and Adolescents [a]

Age Group (years)	Obesity (WC)	Triglycerides	HDL-C	Blood Pressure	Glucose (mmol/L) or Known T2DM
6–10 years	≥90th percentile	Metabolic syndrome cannot be diagnosed, but further measurements should be made if there is a family history of metabolic syndrome, T2DM, dyslipidemia, cardiovascular disease, hypertension, and/or obesity.			
10–16 years—Metabolic syndrome	≥90th percentile or adult cutoff if lower	≥1.7 mmol/L (≥150 mg/dL)	<1.03 mmol/L (<40 mg/dL)	Systolic ≥130/diastolic ≥85 mm Hg	≥5.6 mmol/L (100 mg/dL) (If ≥5.6 mmol/L [or known T2DM], recommend an OGTT.)
16 years—Young adult metabolic syndrome *	≥90th percentile or adult cutoff if lower	≥.7 mmol/L (≥150 mg/dL)	<1.03 mmol/L (<40 mg/dL) in males and <1.29 mmol/L (<50 mg/dL) in females	Systolic ≥130/diastolic ≥85 mm Hg	Glucose (FPG) = 5.6 mmol/L (≥100 mg/dL), or known T2DM
	Use existing IDF criteria for adults including waist circumference central obesity (defined as waist circumference ≥94 cm for Caucasian men and ≥80 cm for Caucasian women, with ethnicity specific values for other groups *) and any two of the four factors listed previously				

[a]*HDL-C*, High-density lipoprotein cholesterol; *IDF*, International Diabetes Federation; *OGTT*, oral glucose tolerance test; *T2DM*, type 2 diabetes mellitus; *WC*, waist circumference.

Data modified from Zimmet P, Alberti KGMM, Kaufman F, et al.: IDF Consensus Group. The metabolic syndrome in children and adolescents—an IDF consensus report. *Pediatr Diabetes* 8:299-306, 2007.

There are several clinical features of metabolic syndrome in the pediatric population. *Obesity* is a crucial component and defined as a body mass index (BMI) above the 95% for age and gender. *Body fat distribution*, most commonly assessed by waist circumference, is often associated with increased insulin resistance.[18] *Dyslipidemias*, defined as an increase in triglycerides or a decrease in HDL cholesterol, can be detected on cholesterol screening. Blood pressure measurements should be done at least yearly to screen for *hypertension*. *Insulin resistance* is often found in obese children, and some may progress to glucose intolerance and diabetes. *Inflammatory markers* such as CRP may be elevated, but the relationship between increased CRP and metabolic syndrome has not been well defined in children.[16] *Nonalcoholic fatty liver disease* (characterized by intrahepatic fat accumulation) and *polycystic ovary syndrome* (characterized by hyperandrogenism) are both associated with insulin resistance and are more prevalent in obese children and adolescents.[16] Physical examination includes pulse, blood pressure, calculation of BMI, assessment for obesity, increased waist size, hepatomegaly, and *acanthosis nigricans* (see Chapter 7).

Summary of examination

- Obtain a thorough history, including gestational history, birth history, family history, and any presenting symptoms.
- Routine cardiac assessment begins with evaluation of weight, height, BMI, and head circumference infants.
- Assess temperature, heart rate, respiratory rate, color, activity level, and oxygen saturation *when indicated*.
- Accurately assess blood pressure in all children whenever possible, and definitely once in infancy, and routinely beginning at 3 years of age (see Table 8.6).
- Approach the inspection systematically, starting from the periphery and moving inward and upward toward examination of the chest assessing perfusion, and pulses in upper and lower extremities.

- Palpate cardiac area for any heaves and presence of a thrill.
- Palpate liver edge for enlargement and position. Note: 1 to 2 cm below the right costal margin is normal in infants.
- Inspect for head and neck for signs of any syndromes; inspect the chest for scars, signs of respiratory distress, and pectus excavatum or pectus carinatum.
- Auscultate lungs for quality of breath sounds, any wheezing, grunting, or rales.
- Auscultate systematically through the four precordial areas moving from LLSB to apex listening for *first heard sound* to the LUSB listening for *second heart sound*, noting variation with respiration and any *third* or *fourth heart sounds* or *ejection clicks.*
- Findings of tachypnea, respiratory distress, tachycardia, bradycardia, low oxygen saturation with pallor or hypoperfusion, hepatomegaly, and syncope require immediate further evaluation and consultation.
- Diagnostic chest x-ray and ECG are performed *as indicated.*
- Refer to cardiology for further evaluation *as indicated* by examination findings, diagnostic results, or parental anxiety/concern and before echocardiogram.

DOCUMENTATION

1-month-old infant with murmur

Cardiac: Increased RV (right ventricular) impulse, normal S1, split S2, Grade 2-3/6 low frequency SEM (systolic ejection murmur) heard best at lower left sternal border. No diastolic murmur, extra heart sounds, thrill, or clicks, nonradiating.

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CHAPTER 9

Chest and respiratory assessment

Concettina (Tina) Tolomeo

Embryological development

Knowledge of lung development is critical when performing a respiratory assessment in early childhood, especially in a newborn infant who is born preterm. This information, along with an understanding of respiratory anatomy and physiology, will provide you with a foundation for interpreting pulmonary symptoms.

Lung development begins in utero at approximately 4 weeks' gestation. It occurs in five stages: embryonic, pseudoglandular, canalicular, saccular, and alveolar. During the embryonic phase, the lungs form from a sac on the ventral wall of the alimentary canal. Right and left branches form through budding and dividing. This is also the time when the vascular system begins to develop. During the pseudoglandular stage, branching of the lung bud occurs, as the trachea, bronchi, and bronchioles are formed by 16 to 17 weeks' gestation. In addition, the intrapulmonary arterial system begins to branch, and cilia and cartilage begin to form. During the canalicular stage, branching of the bronchioles continues and alveoli begin to form. By approximately 25 weeks, the number of bronchial generations with cartilage is the same as the adult lung. This is also the period of capillary bed expansion. Growth of the pulmonary parenchyma and *surfactant* system occurs during the saccular

phase. By 24 weeks the alveolocapillary membrane is ready to begin gas exchange. Maturation and expansion of the alveoli occur during the alveolar period and persist through early childhood. This is when the alveolocapillary membrane matures and the gas exchange surface area increases. Alveolization is complete by about 8 years of age.[1-3]

Breathing movements occur in utero. The movements are irregular and do not open the alveoli. Fetal gas exchange occurs via the placenta. At birth, the lungs fill with air for the first time and take on the role of ventilation and oxygenation. The fluid in the lungs moves into the tissues surrounding the alveoli and is absorbed into the lymphatic system. At this point, gas exchange occurs via diffusion across the alveolar-pulmonary capillary membranes.[1, 3]

Anatomy and physiology

Thorax

The thorax is the bony cage that surrounds the heart, great vessels, lungs, major airways, and esophagus.[3] It is composed of the sternum and ribs (Fig. 9.1). The *sternum* is a flat, narrow bone made up of three parts: the *manubrium,* the body, and the *xiphoid process*.[4] The *manubrium* is somewhat triangular and attaches to the body of the sternum; the angle at which the manubrium and body meet is termed the *manubriosternal angle* or the *angle of Louis*. This angle is in line with the second rib and therefore serves as an important landmark. The *xiphoid process* is the small, thin, cartilaginous end of the sternum, which varies greatly in shape and prominence in infants and children because of the influence of heredity, intrauterine environment, and nutrition. It sits at the level of T9.[3-5] *Pectus carinatum*, pigeon breast, is the abnormal protrusion of the xiphoid process and sternum, and *pectus excavatum*, funnel chest, is the abnormal depression of the sternum (Fig. 9.2; Fig. 9.1).[3, 4, 6, 7] The chest cavity is divided, with the middle portion known as the *mediastinum*.

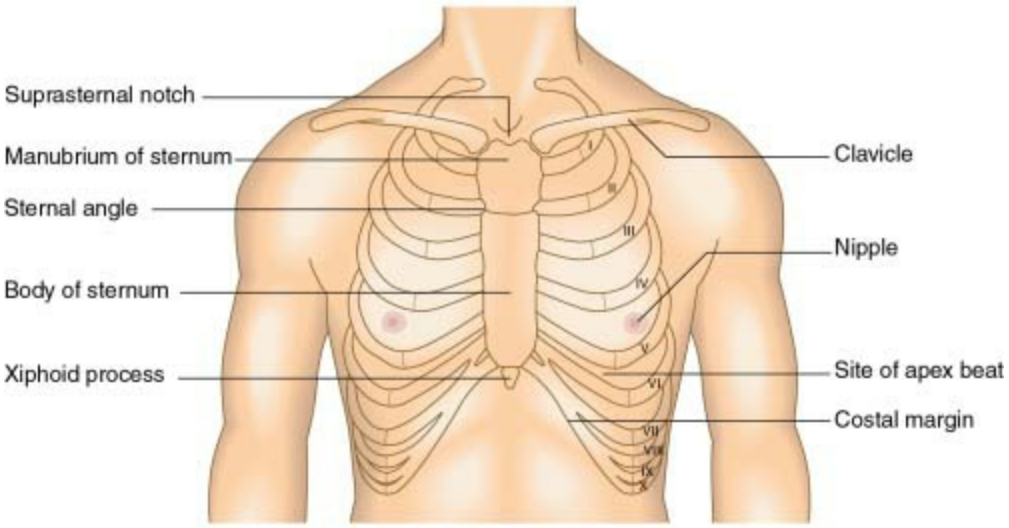

FIGURE 9.1 Anatomy of the rib cage and thorax. Source: (From Revest P: *Medical Sciences*, Edinburgh, 2009, Saunders Ltd.)

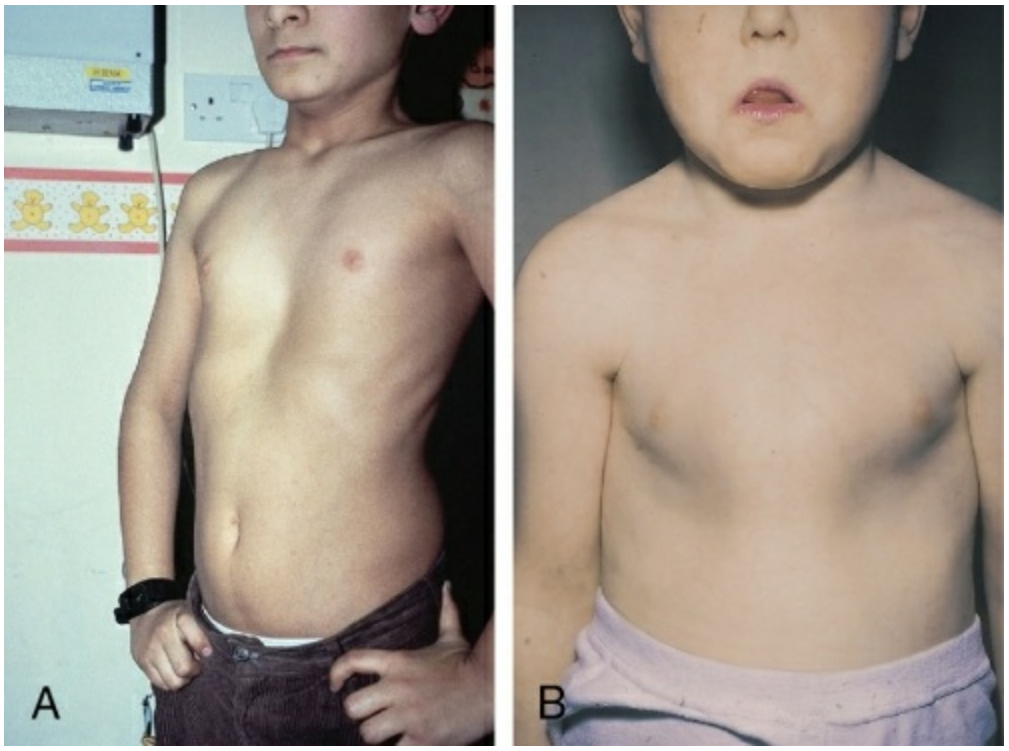

FIGURE 9.2 **(A)** Pectus excavatum. **(B)** Pectus carinatum. Source: (**[A]** From Chaudry B, Harvey D: *Mosby's Color Atlas and Text of Pediatrics and Child Health,* St. Louis, 2001, Mosby. **[B]** From Lissauer T, Clayden G: *Illustrated Textbook of Paediatrics,* ed 2, St. Louis, 2001, Mosby.)

There are 12 pairs of ribs; the first 7 pairs attach anteriorly via their corresponding costal cartilages to the sternum. Ribs 8, 9, and 10 are attached to the costal cartilage on the rib above them; ribs 11 and 12 do not attach anteriorly and are known as floating ribs. All 12 pairs of ribs attach posteriorly to the thoracic vertebrae. The diaphragm sits at the bottom of the rib cage and is the major muscle of respiration. There are 11 intercostal muscles anteriorly and posteriorly and 8 thoracic muscles, all of which help to increase the volume of the rib cage with inspiration and decrease the thoracic volume with expiration (Fig. 9.3).[3, 5]

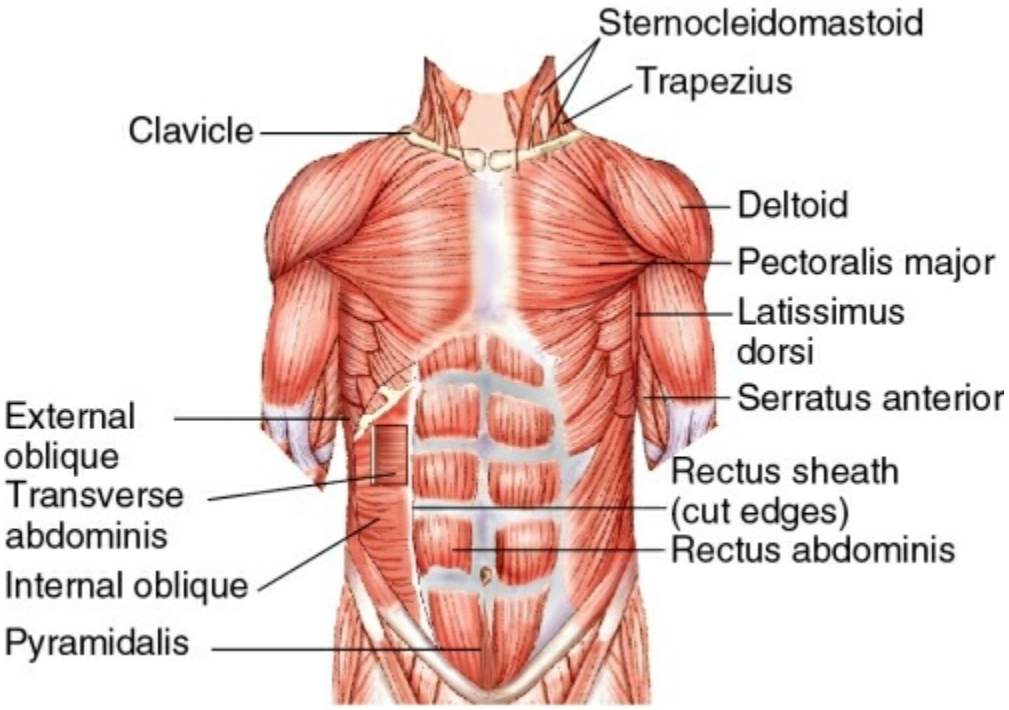

FIGURE 9.3 Anterior thoracic muscles.

The following landmarks are often used in describing the location of physical findings of the chest: the midsternal line (MSL), which runs down the middle of the sternum; the midclavicular line (MCL), located on the right and left sides of the chest, runs parallel to the MSL and through the middle of the clavicles (midway between the jugular notch and acromion) bilaterally. Laterally there are three lines on each side, the anterior axillary line (AAL), the midaxillary line (MAL), and the posterior axillary line (PAL). The AAL begins at the anterior axillary folds, the MAL begins at the

middle of the axilla, and the PAL begins at the posterior axillary folds. Posteriorly is the vertebral line that runs down the middle of the spine and the scapular line, which runs down the inferior angle of each scapula (Fig. 9.4).[3-5, 7]

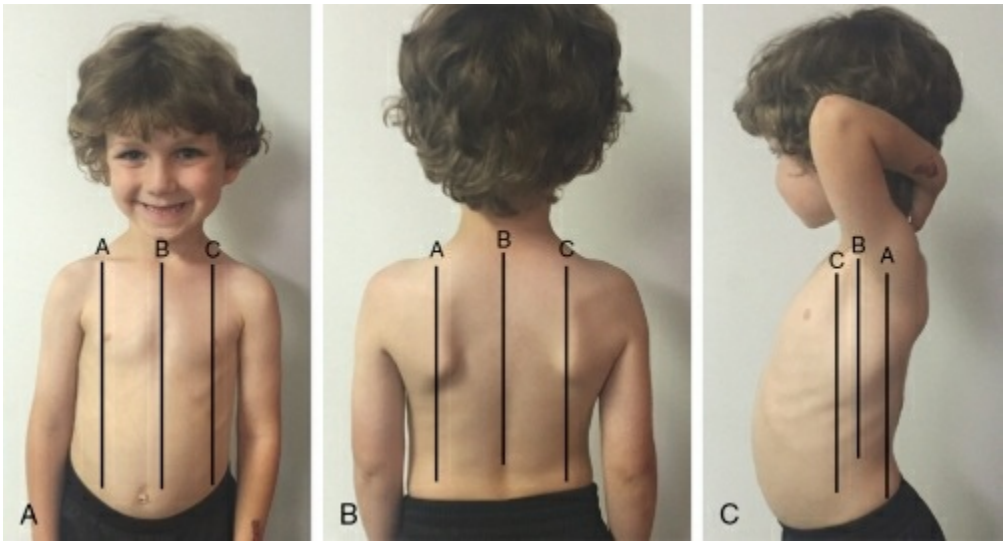

FIGURE 9.4 Anatomical landmarks of the chest. **(A)** Anterior chest. **(B)** Posterior chest. **(C)** Lateral chest.

Lower respiratory tract

The respiratory system is divided into two parts: the *upper respiratory tract* and the *lower respiratory tract*. The upper respiratory tract consists of the nasal cavity, pharynx, and larynx and is reviewed in Chapter 14. The *lower respiratory tract* consists of the trachea, bronchi, bronchioles, alveolar ducts and sacs, and alveoli (Fig. 9.5). The *trachea* is a tube that lies anterior to the esophagus. The distal end of the trachea splits into the right and left mainstem/primary *bronchi*. This bifurcation occurs at the level of T3 during infancy and childhood. By the time the child is an adult, this bifurcation occurs at T4 or T5. The right mainstem bronchus is shorter and more vertical than the left and, therefore, more susceptible to aspiration of foreign bodies in the young child. Beyond the bifurcation, the bronchi continue to branch into lobar/secondary bronchi. There are three branches on the right and two on the left; each branch supplies one of the lung lobes. These

branches further divide into segmental/tertiary smaller bronchi to supply each segment of the lungs and finally the terminal *bronchioles*. Ultimately, the respiratory tract terminates with the alveolar ducts, alveolar sacs, and alveoli, where gas exchange takes place. The bronchial arteries branch from the aorta and supply blood to the lung parenchyma. The blood supply is returned primarily by the pulmonary veins.[1, 3, 5]

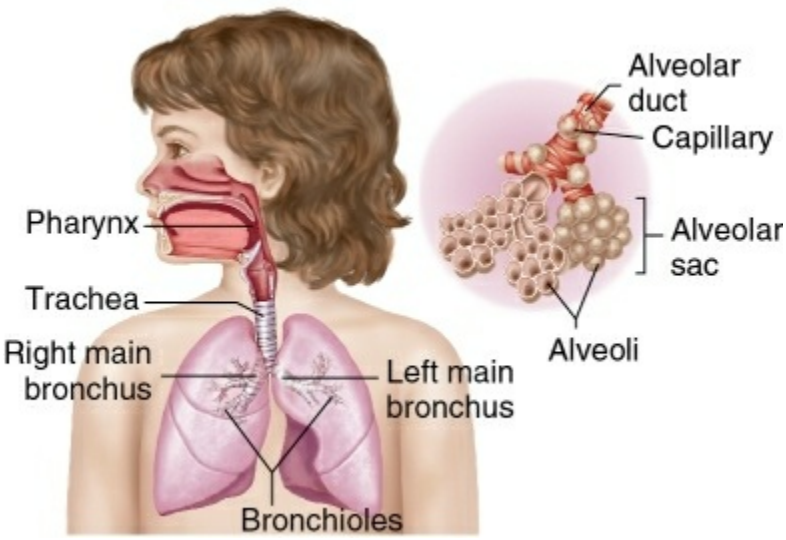

FIGURE 9.5 Lower respiratory tract.

Lungs

The lungs are positioned in the lateral aspects of the thorax, separated by the heart and the mediastinal structures. The right lung has three lobes (upper, middle, and lower), and the left lung has two lobes (upper and lower). The *apex* is the top portion of the upper lobes, which extends above the clavicles. On the right side, the minor or horizontal fissure, located at the fourth rib, divides the right upper lobe (RUL) from the right middle lobe (RML). On the left side, there is a tongue-shaped projection that extends from the left upper lobe (LUL), called the *lingula.* Laterally, the right lower lobe (RLL) and the left lower lobe (LLL) occupy most of the lower lateral chest area. Only a small portion of the RML extends to the MAL, and it does not go beyond that point. Posteriorly, the vertebral column helps in identifying the underlying lung lobes. T4

marks the inferior portion of the upper lobes and the superior portion of the lower lobes. The *base* is the bottom portion of the lower lobes and is marked by T10 or T12, depending on the phase of respiration.[3] The principal function of the lungs is gas exchange between the atmosphere and the blood. Oxygen is carried into the lungs, and carbon dioxide is eliminated. The lungs play a significant role in acid-base balance.

Physiological variations

Table 9.1 presents variations in growth and development that impact the function of the respiratory system in the infant and young child.

TABLE 9.1

Physiological Variations of the Chest and Lungs

Age Group	Physiological Variation
Preterm infant	Respiratory muscles are weak, poorly adapted for extrauterine life; periodic breathing occurs that is similar to fetal breathing; preterm infants become easily hypoxic and apnea occurs
Newborn	Diaphragm is flatter, more compliant; paradoxical breathing occurs in the neonate with inward movement of chest during inspiration due to the compliance of the chest wall; predominantly nose breathers until 4 weeks of age; chest circumference very close in size to head circumference at birth
Infancy	Smaller airways with increased resistance to airflow; rapid respiratory rate; minimal nasal mucus causes mild to moderate upper airway obstruction
Early childhood (1–4 years of age)	Rapid growth and maturation of alveoli improve ventilation; respiratory rate decreases dramatically from newborn period
Middle childhood (5–10 years of age)	Alveoli continue to increase in number
Adolescence	Alveolar size matures to adult capacity

Data from Nakra N: Pediatric pulmonary anatomy and physiology. In Tolomeo C, editor: *Nursing Care in Pediatric Respiratory Disease*, Ames, 2012, Wiley-Blackwell; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW: Chest and lungs. In Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW, editors: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby; and Sarnaik AP, Heidemann SM, Clark JA: Respiratory pathophysiology and regulation. In Kliegman RM, Stanton BF, St

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System-specific history

Complete and accurate information gathering is essential when assessing an infant, child, or adolescent with respiratory symptoms. The Information Gathering table presents respiratory focused questions based on developmental stages. Questions should be open-ended and age-specific to allow the parent or caregiver the opportunity to give a full explanation of past and present concerns. In addition, always obtain information directly from the older child or adolescent when possible. Table 9.2 presents a symptom-focused assessment of respiratory conditions for children and adolescents.

TABLE 9.2

Symptom-Focused Assessment of Respiratory Conditions

Symptom	Questions to Ask
Cough	History: Onset of cough symptoms, was it sudden or gradual? How long has the cough been present? Is cough worsening or changing character? Is cough wet, dry, hacking, barking, whooping? Worse during day or at night? Worse with feeding, sleeping, running? Any other symptoms: Shortness of breath, chest pain/tightness, or wheeze? Choking episodes? History of aspiration (small toy, food, etc.)? Rhinorrhea or nasal congestion? In the older child/adolescent: Is cough productive with sputum or nonproductive? If productive, what color is the sputum?
	Pattern: Occasional, persistent, or coughing spasms?
Wheeze	History: Onset of wheezing, was it sudden or gradually worsening? Any other symptoms: Cough? Shortness of breath? Chest pain/tightness? Is it associated with upper respiratory infection symptoms? Where is wheezing heard—nose, neck, chest? History of aspiration (small toy, food, etc.)?
	Pattern: Occasional, increase with exercise?
Shortness of breath	History: Onset of shortness of breath—was it sudden or gradual? Does it occur with activity or rest? Is it difficult to get air in, out, both? History of aspiration (small toy, food, etc.)? Accompanying symptoms of cough, wheezing? Any diaphoresis?
Chest pain	History: Onset of chest pain—was it sudden or gradual? Does it occur on inspiration, expiration, or both? Chest pain occurs with movement or rest? Type of pain (sharp, dull)? Ask verbal child to point to area of pain. History of trauma or recent sports injury or weight lifting? Any other symptoms—cough, wheezing, shortness of breath, syncope?
	Pattern: Occasional with respirations or persistent?

Information Gathering for Chest and Lung Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant	How many weeks' gestation? Admitted to newborn intensive care unit? Length of stay? Any episodes of apnea or tachypnea? Need for oxygen? Need for ventilation? For how long? Infant discharged to home on a ventilator/on oxygen? Infant discharged home on any medications? Any maternal substance abuse?
Newborn	How many weeks' gestation? Birth weight? Any birth complications? Meconium aspiration? Breathing problems at birth? Any episodes of apnea or tachypnea?
Infancy	History of respiratory infections as infant (respiratory syncytial virus [RSV], rhinovirus, etc.)? Frequent upper respiratory infections (URIs)? History of wheezing? History of noisy breathing? Hospitalizations? History of intubations? History of eczema/skin allergy? In daycare? Immunization status? Frequent vomiting after feeds/"choking" episodes? Arches back after feeding?
Early childhood	History of apnea/breath-holding spells? Does child's speech have a nasal or congested sound? History of nasal congestion, chronic URIs, allergy symptoms, tonsillitis, frequent ear infections? In daycare or preschool? Does child frequently put objects in mouth/nose? Exposure to group A streptococcal infection? Does child snore at night? Exposure to ill contacts? Foreign travel or recent immigrant?
Middle childhood	History of nasal congestion, chronic rhinorrhea, asthma, sinusitis, tonsillitis, chronic URIs, recurrent pneumonia? Does child snore at night? Exposure to group A streptococcal infection? History of asthma? History of gastroesophageal reflux? Exposure to ill contacts? Foreign travel or recent immigrant?
Adolescence	History of chronic URIs, allergic rhinitis, asthma, recurrent tonsillitis? History of oral sex? Tobacco use? How many cigarettes per day? Marijuana use? Any oral piercings? Does the child snore at night?
Environmental risks	Year home was built? Location (inner city, suburb, rural, near highway/high traffic areas)? Is there a basement? Number of people in home? Anyone smoke in home? Pets in home? Type of heating system? Wood burning stove? Humidifier? Mold? Carpets? Drapes? Mice or roaches? Presence of chemicals/fumes in home or near home?

EVIDENCE-BASED PRACTICE TIP

The Test for Respiratory and Asthma Control in Kids (TRACK) for children less than 5 years of age is a five-item questionnaire that has been validated to assess respiratory and asthma control in children. It is intended for children in this age group who have a history of two or more episodes of wheezing, coughing, or

shortness of breath lasting more than 24 hours, and have been treated with a quick relief medication for respiratory symptoms or have a diagnosis of asthma. The first three questions pertain to asthma symptoms over the last 4 weeks. The fourth question asks about the use of quick relief medications over the past 3 months, and the last question asks about the use of oral steroids over the past year. Each question has five response levels. A score of less than 80 indicates that respiratory problems are not well controlled, and a score of 80 or more indicates good control.[8, 9] The TRACK is available at http://imaging.ubmmedica.com/consultantlive/pdfs/TRACKbrocCompliance.pdf.

EVIDENCE-BASED PRACTICE TIP

The Childhood Asthma Control Test (C-ACT) for children 4 to 11 years of age is a seven-item questionnaire that assesses asthma control over the previous 4 weeks. The first four questions are completed by the child and the last three by the parent/guardian. Each question has four response levels. The Asthma Control Test (ACT) for children 12 years of age and older is a five-item questionnaire that assesses asthma control over the previous 4 weeks. Each question has five response levels. Questions for both pertain to daytime and nighttime symptoms, activity limitations, use of short-acting beta agonists, and perception of asthma control. Both questionnaires have been validated to measure asthma control. [10] A score of 19 or less indicates the asthma is not well controlled. The questionnaires are available in print and web format (http://www.asthma.com/additional-resources/childhood-asthma-control-test.html) in a number of languages.

Physical assessment

Equipment

When auscultating breath sounds, use the diaphragm of the

stethoscope. The size of the stethoscope being used is extremely important when evaluating respiratory sounds. Stethoscopes with a smaller diaphragm should be used on infants and toddlers. Isolating cardiac and respiratory sounds is difficult in small children with too large a diaphragm, and using a diaphragm that is too small on adolescents or on children who are overweight or obese causes practitioners to miss findings of cardiac and respiratory sounds on auscultation due to increased adipose tissue, which obscures respiratory sounds.

Positioning

If possible, the child should sit upright for the respiratory assessment. Young children, especially toddlers, may be more relaxed sitting on their parent's lap during the examination of the chest. Children should be allowed to help as much as possible during the exam. Have them hold the stethoscope in place once you position it in the appropriate location. Other techniques include letting children role-play by allowing them to listen to their parent, a doll, or a stuffed animal. School-age children are very curious. Therefore, explain what you will be doing during the exam and why, using developmentally appropriate vocabulary. Taking a few extra minutes to incorporate the child's developmental level into your exam will result in more thorough and reliable findings. In addition, pictures of lungs in the exam room can be helpful with older children when you are explaining what you are looking for and listening to during the examination.

Physical examination

Astute physical examination skills are important when making a diagnosis. Medical oversights have been associated with failure to perform a physical examination, misinterpretation of physical examination findings, and missing or not eliciting a physical examination sign.[11]

Pediatric health care providers should begin the physical examination by assessing the chest. The "quieter" parts of the exam, the cardiac and respiratory exam, require astute listening skills and less active participation of the child. Therefore, they are best

performed first—an effective approach to assessing children among pediatric experts. The pediatric provider must take a systematic approach to examining the chest and use all the components of physical assessment. This includes inspection, palpation, percussion, and auscultation. Examination of the chest should always include both the anterior and the posterior chest.

A thorough assessment of the chest and lungs is not complete without examination of the upper airway and the extremities. Examine the nasal passages to determine the size of the airway passage and the appearance of the nasal turbinates, as well as the presence of rhinitis, nasal secretions, nasal polyps, or a foreign body. When looking at the oropharynx, note the presence of postnasal drip and tonsillar size. Tonsillar size is often graded on a scale of 0 to 4+, where 0 indicates tonsils that fit in the tonsillar fossa and 4+ indicates tonsils that occupy greater than 75% of the oropharyngeal width.[12] An abnormal finding in any of these areas can be a cause of respiratory symptoms. Finally, you need to examine the extremities for signs of digital clubbing. *Clubbing* is the bulbous-shaped enlargement of the soft tissue of the distal phalanges (Fig. 9.6). To assess for *clubbing* in a simple way, ask the child to place the first phalange of each thumb together (nails facing each other). Normally this position results in a diamond shape between the two thumbs. With clubbing, the diamond shape disappears and the space between the two thumbs is decreased or absent, depending on the degree of clubbing. This is termed the *Schamroth sign*. Other methods for measuring clubbing include the phalangeal depth ratio and they hyponychial angle. A distal phalangeal diameter (DPD) to interphalangeal diameter (IPD) ratio of less than 1 is normal. A ratio greater than 1 is seen with clubbing. A hyponychial angle of less than 180 degrees is normal, while an angle of greater than 195 degrees is indicative of clubbing.[13] Clubbing can be hereditary or can be the result of cardiac disease, respiratory disease, or severe malnutrition.

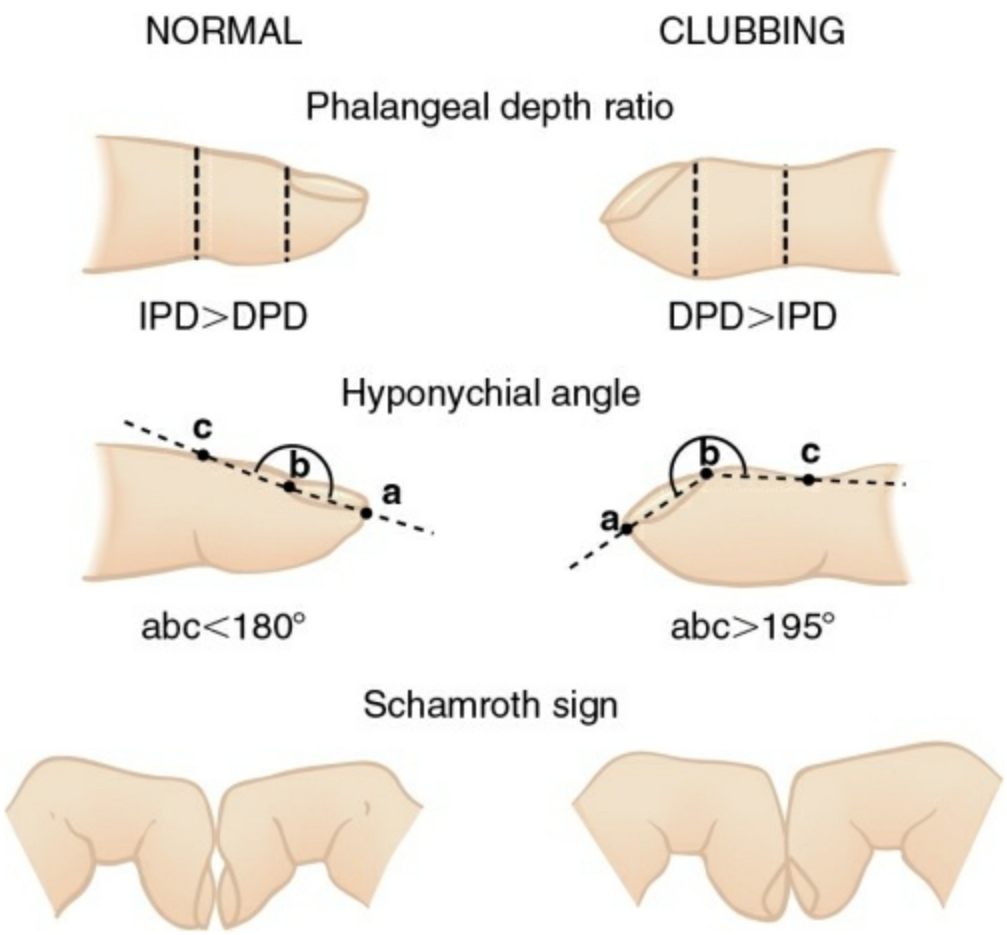

FIGURE 9.6 Digital clubbing. *DPD*, Distal phalangeal diameter; *IPD*, interphalangeal diameter. Source: (From Pasterkamp H: The history and physical examination. In Wilmott RW, Boat TF, Bush A, et al., editors: *Kendig and Chernick's Disorders of the Respiratory Tract in Children*, ed 8, Philadelphia, 2012, Elsevier.)

EVIDENCE-BASED PRACTICE TIP

Diagnosing asthma during the infant and preschool years is difficult because many children wheeze during their first year of life. The Asthma Predictive Index (API) is a useful tool to help rule out the likelihood of asthma during the school age years in young children with wheezing. Recurrent episodes of wheezing during the first 3 years of life, plus one of two major criteria (provider diagnosed eczema or parental asthma) or two of three minor criteria (provider diagnosed allergic rhinitis, wheezing without colds, or peripheral eosinophilia ≥4%) constitute a positive API and increase the likelihood of asthma. [14]

Inspection

Ideally, start the exam by visually inspecting the infant or child undressed from the waist up. This allows you to make general observations about the child's respiratory rate, breathing pattern, respiratory effort, accessory muscle use, inspiratory to expiratory ratio (I:E ratio), skin color, presence of noisy breathing, chest symmetry, and chest shape. In an irritable, ill, or fearful child, observation of respiratory pattern and rate may be the most helpful part of the examination if the child exhibits resistance to auscultation.

Assess the shape of the chest and note any abnormalities. The normal anterior-posterior (AP) to transverse ratio is 1:2. In the infant, the chest is round with a diameter roughly equal to the head circumference until 2 years of age and has a 1:1 AP/transverse ratio, giving a barrel chest appearance. As the child grows, the chest takes on the shape of the adult chest.[3, 6] A barrel chest shape also can be seen when chronic air trapping is present, such as in advanced stages of *cystic fibrosis*. Other deformities of the chest that can have an impact on the child's respiratory status and decrease expansion of the lungs include *pectus carinatum*, *pectus excavatum*, or *scoliosis*.

Assessment of respirations

Resting respiratory rates vary with the age of the child: the younger the child, the higher the respiratory rate (Table 9.3). The child's rhythm of breathing should be regular. Many factors can increase or decrease the respiratory rate, such as fever, pain, exercise, and medications.

TABLE 9.3

Expected Range of Respiratory Rates for Age

Age	Rate
Preterm neonate	40–70
0–12 months	24–55
1–5 years	20–30
5–9 years	18–25

9–12 years	16–22
12 years and older	12–20

Data from Hughes DM: Evaluating the respiratory system. In Goldbloom RB, editor: *Pediatric Clinical Skills*, ed 4, Philadelphia, 2011, Elsevier/Saunders; Engorn B, Flerlage J: *The Harriett Lane Handbook: A Manual for Pediatric House Officers*, Philadelphia, 2015, Saunders; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW: Chest and lungs. In Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW, editors: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby; and Cox LC: Examination of the respiratory system. In Cox LC, editor: *Physical Assessment for Nurses,* West Sussex, 2010, Wiley-Blackwell.

PEDIATRIC PEARLS

Assess respiratory rate before the physical exam portion of the visit when the child is calm; this is best accomplished when the child is sitting in the parent's/guardian's lap.

Periodic breathing is characterized by rapid breathing followed by periods of *apnea*, cessation of breathing. This is normal in the first few hours of life in healthy full-term newborns. Periodic breathing is more likely to persist in preterm infants, but the episodes of apnea should improve as infants approach term age.[3, 15] *Apnea* is considered clinically significant if it lasts greater than 20 seconds or is characterized by shorter pauses associated with bradycardia (heart rate <100 beats per minute), cyanosis, or pallor.[16] *Paradoxical breathing*, or seesaw breathing, is often seen in newborns and infants because they use abdominal muscles more than intercostal muscles.[3, 15] *Cheyne-Stokes breathing* is characterized by cycles of increasing and decreasing tidal volume separated by apnea. It occurs in children with congestive heart failure and increased intracranial pressure.[3, 7, 15]

Noisy breathing includes stridor, grunting, and snoring. *Stridor* is a high-pitched, loud, inspiratory sound produced by upper airway obstruction. Causes of upper airway obstruction include edema status post intubation, subglottic stenosis, laryngotracheobronchitis, and foreign body aspiration.[17] *Grunting* is a low-pitched expiratory sound present with respiratory distress and is the result of a partial closure of the glottis.[13] Snoring is a rough, snorting sound during

sleep, usually on inspiration, but can also be present on expiration. It may be present during sleep in healthy children who have an upper respiratory infection. Snoring is often heard in the presence of adenoidal and tonsillar hypertrophy or congenital anomalies that involve the upper airway or facies.[6]

Inspect for nasal flaring and use of accessory muscles in the infant and toddler. Mild nasal flaring can be seen in newborns because they are obligate nose breathers in the first month of life. However, increased nasal flaring should be investigated because it is a sign of labored breathing. Other signs of increased effort and respiratory distress include retractions, bulging of the intercostal muscles, and head bobbing.[18] Although mild retractions may be seen in some healthy young children, increased retractions can be a sign of airway obstruction. The chest wall of newborns and infants is more compliant than that of older children, making them more prone to retractions. Bulging of the intercostal spaces also may be seen with airway obstruction as a consequence of increased expiratory effort.[3, 6] *Head bobbing*, the forward movement of the infant's head, is a sign of respiratory distress due to the contraction of the *scalene* and *sternocleidomastoid* muscles.

An abnormal inspiratory to expiratory (I:E) ratio is an additional sign of respiratory distress. A normal I:E ratio in the infant is 1:2 seconds, except in the newborn, when it is variable. The inspiratory phase is greater than the expiratory phase when there is an extrathoracic (from the nose to the mid-trachea) obstruction. When there is an intrathoracic obstruction, the expiratory phase is more prolonged and accessory muscles are often used.[13] This is seen in obstructive diseases that cause air trapping, such as cystic fibrosis, or an acute asthma exacerbation can increase the expiratory time.

Assess for *cyanosis*, a bluish color to the skin or mucous membranes. *Acrocyanosis*, cyanosis of the hands and feet, is normal in the newborn and can persist for days if the infant is in a cool environment. *Central cyanosis,* which occurs in the conjunctiva, lips, mucous membranes, and nail beds, is an abnormal finding at any age and warrants immediate further evaluation. In the anemic child, it may be difficult to detect cyanosis early on because the arterial oxygen saturation at which cyanosis becomes apparent varies with the total hemoglobin level.[6] In addition, in a dark-

skinned child, cyanosis is best assessed by looking at the mucous membranes and/or nail beds.

Auscultation

Auscultation is best performed at the beginning of the examination when the infant/child is more cooperative and attentive. Auscultation is performed with the diaphragm of the stethoscope placed firmly and directly on the chest. The child's chest should be bare because clothing can change the quality of the breath sounds. Remember to start at the apex which sits slightly above the clavicles (Fig. 9.7) then work down to the base. You want to auscultate moving from side to side across the chest so that you can compare one side to the other. Be sure to listen at each location for one full breath. Breath sounds are identified by their intensity, pitch, and duration. In children, breath sounds tend to be louder because of the thinness of the chest wall.[6] There has been much confusion about the terminology used to describe breath sounds. Table 9.4 presents the most common description of normal breath sounds in the respiratory cycle.

FIGURE 9.7 Auscultation of left apex.

TABLE 9.4
Normal Breath Sounds

Sound	Description	Duration of Inspiration and Expiration	Sound Diagram
Vesicular	Soft, low-pitched sound heard over entire surface of lungs; inspiration louder	Inspiration > expiration	
Bronchovesicular	Moderately loud and pitched sounds heard over intrascapular area; heard on inspiration and expiration	Inspiration = expiration	
Bronchial (tubular)	Loud and high-pitched sounds heard over trachea near suprasternal notch; louder on expiration	Inspiration < expiration	
Tracheal	Loudest and highest pitched sounds heard over the trachea; heard on inspiration and expiration	Inspiration = expiration	

Data from Hughes DM: Evaluating the respiratory system. In Goldbloom RB, editor: *Pediatric Clinical Skills,* ed 4, Philadelphia, 2011, Elsevier/Saunders; Berger N, Conroy ML, Hanlon Rafter R, et al.: Respiratory system. In Berger N, Conroy ML,

Hanlon Rafter R, et al., editors: *Health Assessment Made Incredibly Visual*, ed 2, Philadelphia, 2011, Lippincott Williams & Wilkins; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW: Chest and lungs. In Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW, editors: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby; and Cox LC: Examination of the respiratory system. In Cox LC, editor: *Physical Assessment for Nurses,* West Sussex, 2010, Wiley-Blackwell.

Transmitted voice sounds or an infant's cry *vocal fremitus* also can be assessed with a stethoscope. Voice sounds are typically muffled on auscultation. If you hear the voice sound or cry loud and clear, it is termed *bronchophony.* This technique can be used to examine an infant or uncooperative child even while he or she is crying. If the verbal child speaks the sound "ee" and it sounds like "ay," it is called *egophony.* If the child whispers a word and it is heard loud and clear, it is called *whispered pectoriloquy.* If any of these signs are positive, it is evidence of a consolidation indicated by fluid or exudate in the alveolar spaces.[4,7]

Abnormal lung sounds

In addition to normal lung sounds, you may hear adventitious or abnormal breath sounds (Table 9.5). Adventitious lung sounds are sounds that are superimposed on normal breath sounds.

TABLE 9.5
Abnormal Breath Sounds

Sound	Description
Crackles	Discontinuous sounds, bubbly, heard primarily on inspiration, do not clear with cough; associated with pneumonia, pulmonary edema, cystic fibrosis
	• Fine crackles—soft and higher in pitch, generally indicative of fluid in smaller airways in infants andchildren
	• Coarse crackles—loud and lower in pitch, usually signify fluid in larger airways
Wheezes	Continuous, high-pitched musical sounds heard primarily on expiration; associated with foreign body aspiration, bronchiolitis, asthma
Rhonchi	Continuous low-pitched sounds; clears with coughing; caused by secretions/mucus in larger airways as in bronchitis and lower respiratory tract infections
Stridor	High-pitched, harsh sounds; heard primarily with inspiration but can be biphasic; associated with laryngotracheobronchitis, laryngomalacia, subglottic stenosis, and vocal cord dysfunction
Stertor	Noisy breathing heard on inspiration and expiration. Caused by the echoing of soft structures at the level of the nasopharynx and pharynx. Can occur with stridor, especially when there is external compression of the larynx and supraglottis.

Data from Hughes DM: Evaluating the respiratory system. In Goldbloom RB, editor: *Pediatric Clinical Skills*, ed 4, Philadelphia, 2011, Elsevier/Saunders; Berger N, Conroy ML, Hanlon Rafter R, et al.: Respiratory system. In Berger N, Conroy ML, Hanlon Rafter R, et al., editors: *Health Assessment Made Incredibly Visual*, ed 2, Philadelphia, 2011, Lippincott Williams & Wilkins; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW: Chest and lungs. In Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW, editors: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby; Cox LC: Examination of the respiratory system. In Cox LC, editor: *Physical Assessment for Nurses,* West Sussex, 2010, Wiley-Blackwell; Engorn B, Flerlage J: *The Harriett Lane Handbook: A Manual for Pediatric House Officers*, Philadelphia, 2015, Saunders; and Ida JB, Thompson DM: Pediatric stridor, *Otolaryngol Clin N Am* 47:795-819, 2014.

Palpation

Palpation is performed to identify anatomical landmarks, respiratory symmetry, and areas of tenderness or abnormalities. Begin by counting the ribs, locate the *angle of Louis,* and move your fingers laterally to feel the second rib and corresponding costal cartilage. Directly below this rib is the second intercostal space. From there, count downward to the other ribs and their respective intercostal spaces.[3, 4, 7]

To assess chest excursion, place your hands along the lateral rib cage and squeeze the thumbs toward each other so that you gather a small amount of skin in between your thumbs. As the child inhales, note the symmetry of the chest excursion. Again, this should be done both anteriorly and posteriorly. Asymmetry is an abnormal finding. In the newborn period, asymmetrical chest excursion may be a sign of a diaphragmatic hernia. Other possible abnormalities associated with asymmetrical chest excursion during the newborn period or later include diaphragmatic dysfunction, pneumothorax, mass, foreign body, or abnormal chest wall shape. An important part of the exam that should not be ignored is palpation of the trachea to assess for a mediastinal shift. To perform this exam, place fingers in the suprasternal notch on both sides of the trachea (Fig. 9.8). The distance between the trachea and the sternocleidomastoid tendons should be equal on both sides. A shift in the trachea occurs when there is a difference in volume or pressure between the two sides of the chest, as is seen in a pneumothorax or pleural effusion.[3, 5, 7]

FIGURE 9.8 Palpation of trachea.

To complete the palpation portion of the exam, assess for tactile fremitus. To do this, place your palms, the ulnar surface of your hands, or your fingers, depending on the size of the chest wall, on the child's back (right and left side), and ask the verbal child to say "1-2-3." With an infant or uncooperative child, this technique can be performed while the child is crying. Perform this exam both anteriorly and posteriorly. Vibrations or *fremitus* should be of equal intensity bilaterally. A pneumothorax or hyperinflation can decrease fremitus; a mass or pneumonia can increase fremitus.[4, 7]

Percussion

Percussion is used to determine the sounds of the underlying organs and tissues. It helps distinguish whether the tissue is air filled, fluid filled, or solid. There are five sounds that are produced with percussion: *resonance*, *hyperresonance*, *dull*, *flat*, and *tympany*.

The sounds are distinguished by their intensity, pitch, and duration. In infants and toddlers, the sound produced is more resonant because the chest wall is thinner than in older children and adolescents (Table 9.6). To perform percussion, hyperextend the middle finger of your nondominant hand and press the distal interphalangeal joint firmly on the chest. With the middle finger of your dominant hand, strike down on the hyperextended interphalangeal joint. The movement must be sharp and quick, and the only portion of the nondominant finger that should be touching the chest should be the hyperextended joint. Strike each area two or three times and then move to the opposite side for comparison (Fig. 9.9). Lastly, it may be necessary to repeat auscultation to confirm findings that were revealed during palpation and percussion. Percussion is often deferred in infants and young children because the examiner often cannot discriminate between sounds that originate from organs that are proximate to each other.[4, 7, 16]

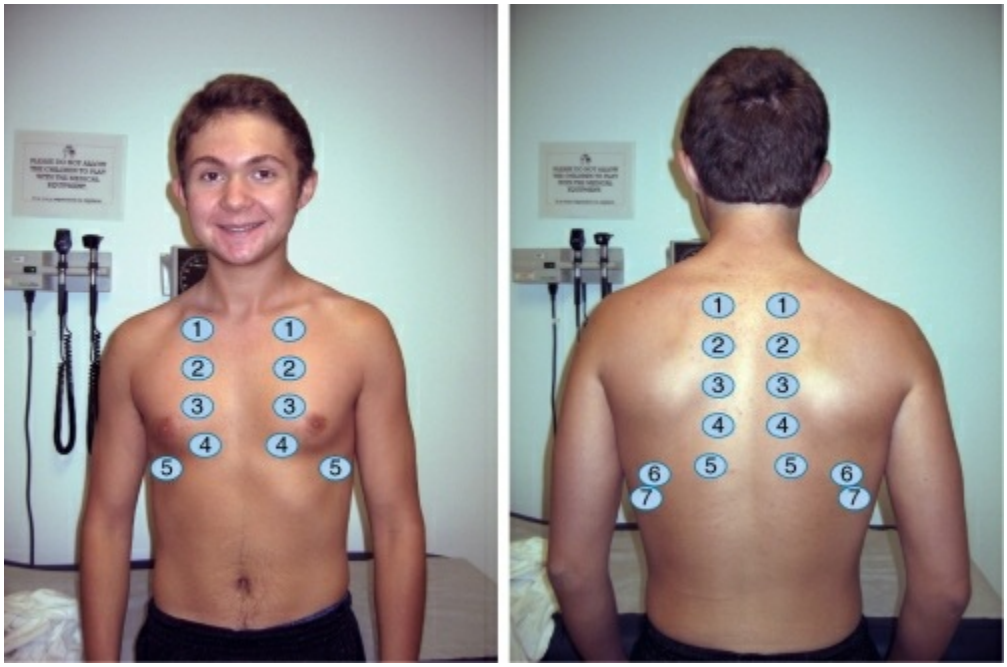

FIGURE 9.9 Sequence for percussion of the thorax.

TABLE 9.6
Percussion Sounds

Tone	Intensity	Pitch	Quality	Clinical Implication
Tympanic	Loud	High	Drumlike	Air collection (i.e., large pneumothorax)
Resonant	Loud	Low	Hollow	Normal lung
Dull	Moderate	Moderate	Dull thud	Solid area (i.e., mass)
Flat	Soft	High	Very dull	Consolidation (i.e., pneumonia)

Data from Berger N, Conroy ML, Hanlon Rafter R, et al.: Respiratory system. In Berger N, Conroy ML, Hanlon Rafter R, et al., editors: *Health Assessment Made Incredibly Visual*, ed 2, Philadelphia, 2011, Lippincott Williams & Wilkins; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW: Chest and lungs. In Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW, editors: *Seidel's Guide to Physical Examination*, ed 8, St. Louis, 2015, Mosby; and Cox LC: Examination of the respiratory system. In Cox LC, editor: *Physical Assessment for Nurses,* West Sussex, 2010, Wiley-Blackwell.

Diagnostics

Pulse oximetry

For a discussion on pulse oximetry, see Chapter 8.

Chest radiograph

Chest radiography is often used to identify pulmonary pathology in the presence of pulmonary symptoms such as coughing, wheezing, shortness of breath, and chest pain. A comparison of inspiratory to expiratory radiographs may be useful when evaluating a child with a suspected foreign body. If a localized area of the lung does not empty on the expiratory view, it is indicative of a bronchial obstruction.[13] A finding of hyperinflation is indicative of air trapping and is frequently seen in children with bronchiolitis or an acute asthma exacerbation. Atelectasis or collapse may indicate mucus retention and can be present in children with bronchiolitis, asthma, or bronchomalacia. Consolidation can be caused by pneumonia, and an area of hyperlucency without normal lung markings is indicative of a pneumothorax or other air-containing pathology such as a cyst.

Pulmonary function testing

Pulmonary function testing is performed to evaluate pulmonary symptoms and assess for obstructive and/or restrictive lung disease.

It is also used to monitor disease progression and response to therapy. Although infant pulmonary function testing is available at specialized pediatric respiratory medicine centers, pulmonary function tests are generally reserved for children ≥5 years of age. Predicted values are based on a reference population and are dependent upon age, height, sex, and ethnicity. There are specific guidelines for the performance of lung function testing; an experienced technician is of paramount importance in the testing process. An assessment of patient technique and effort is necessary when interpreting results; in addition, acceptability criteria must be met before a test can be considered interpretable.[19] Severity of lung dysfunction is categorized as normal, mild, moderate, moderately severe, severe, and very severe.[20] While there are many types of pulmonary function tests available, this chapter will focus on the most common tests performed in the pediatric population.

Spirometry

The most common pulmonary function test is spirometry. Portable spirometers are available for office use. *Spirometry* is defined as "a physiological test that measures how an individual inhales or exhales volumes of air as a function of time."[21] Measures include (1) forced vital capacity (FVC), which is the maximum volume of air forcefully exhaled after a maximal inhalation; (2) forced exhaled volume (FEV_1) in the first second, which is the maximum volume of air exhaled in the first second of FVC; (3) FEV_1/FVC, which is the ratio of air exhaled in the first second to the total volume of air exhaled; and (4) forced expiratory flow 25-75 (FEF 25-75), which is the forced expiratory flow between 25% and 75% of FVC.[21] The FEV_1 is a measure of large airways and is effort dependent, while the FEF 25-75 is a measure of small airways and is effort independent. A reduced FEV_1 and FEV_1/FVC is indicative of an obstructive pattern, as seen in asthma. A reduced FVC and FEV_1 and a normal FEV_1/FVC is indicative of a restrictive pattern as seen in chest wall abnormalities. A mixed pattern may be seen in someone with advanced cystic fibrosis. The *Expert Panel Report 3: Guidelines on the Diagnosis and Management of Asthma* recommends performing spirometry at the initial assessment after treatments

have been initiated and symptoms have stabilized, during periods of loss of asthma control, and at least every 1 to 2 years.[22]

Evaluation of the spirogram is as important as the evaluation of the spirometry values. The spirogram is a graphic depiction of the spirometry values. The flow-volume curve and the time volume curve provide the expiratory phase of the maneuver. The flow volume loop provides both the inspiratory and the expiratory phases of the maneuver (Fig. 9.10). An evaluation of the curve shapes can provide you with a sense of the quality of the test, as well as whether an obstructive or restrictive pattern is present.[23] If the expiratory curve has a concave shape, it is indicative of an obstructive pattern. If the expiratory curve has a steep slope, it is indicative of a restrictive pattern (Fig. 9.11). If the inspiratory loop is blunted, it is indicative of vocal cord dysfunction.

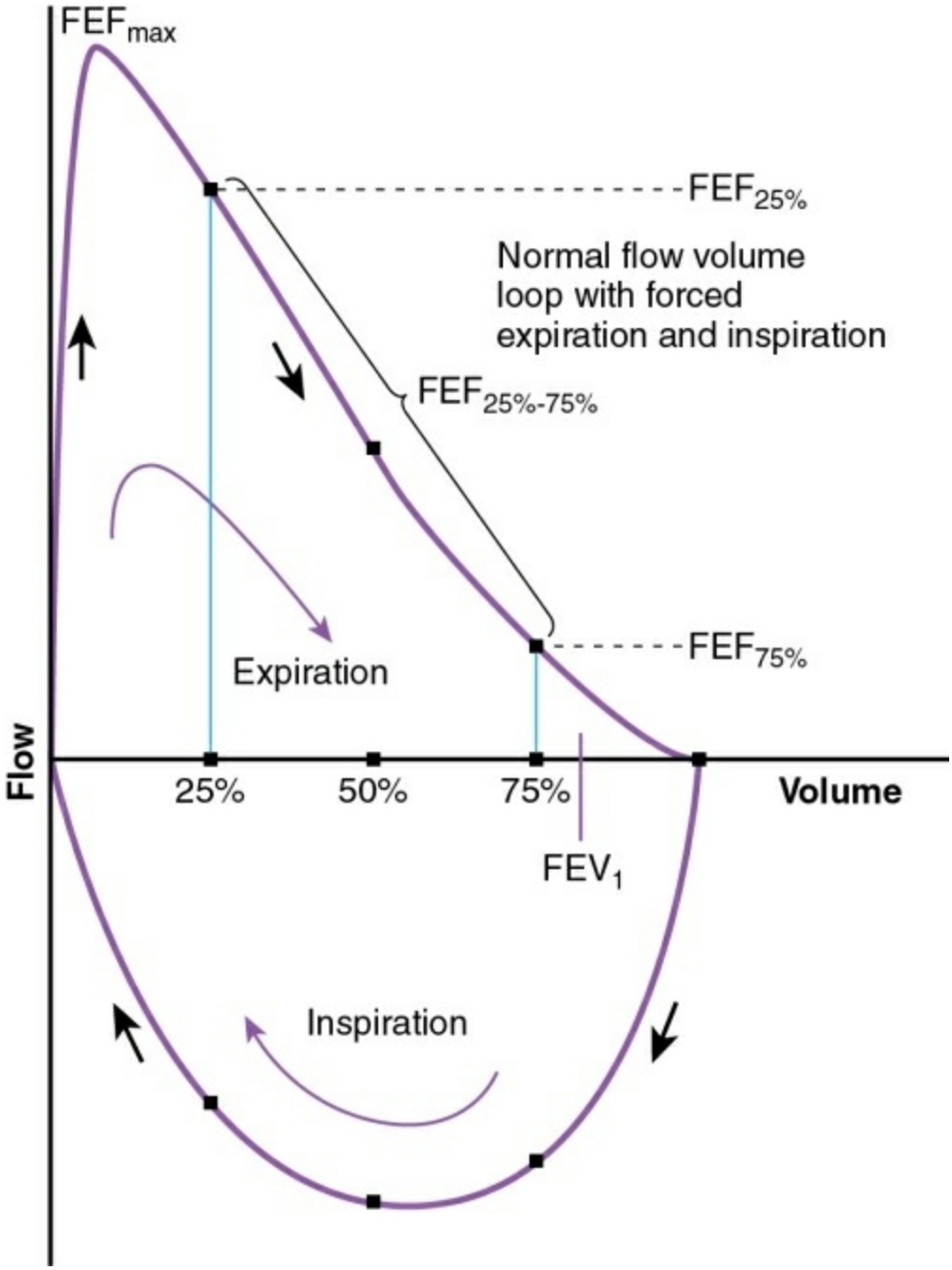

FIGURE 9.10 Normal flow volume loop. *FEF*, Forced expiratory flow; *FEV*, forced exhaled volume. Source: (From Sarnaik AP, Heidemann SM, Clark JA: Respiratory pathophysiology and regulation. In Kliegman RM, Stanton BF, St Geme JW, Schor NF, Behrman RE, editors: *Nelson Textbook of Pediatrics*, ed 20, Philadelphia, 2016, Elsevier, Inc.)

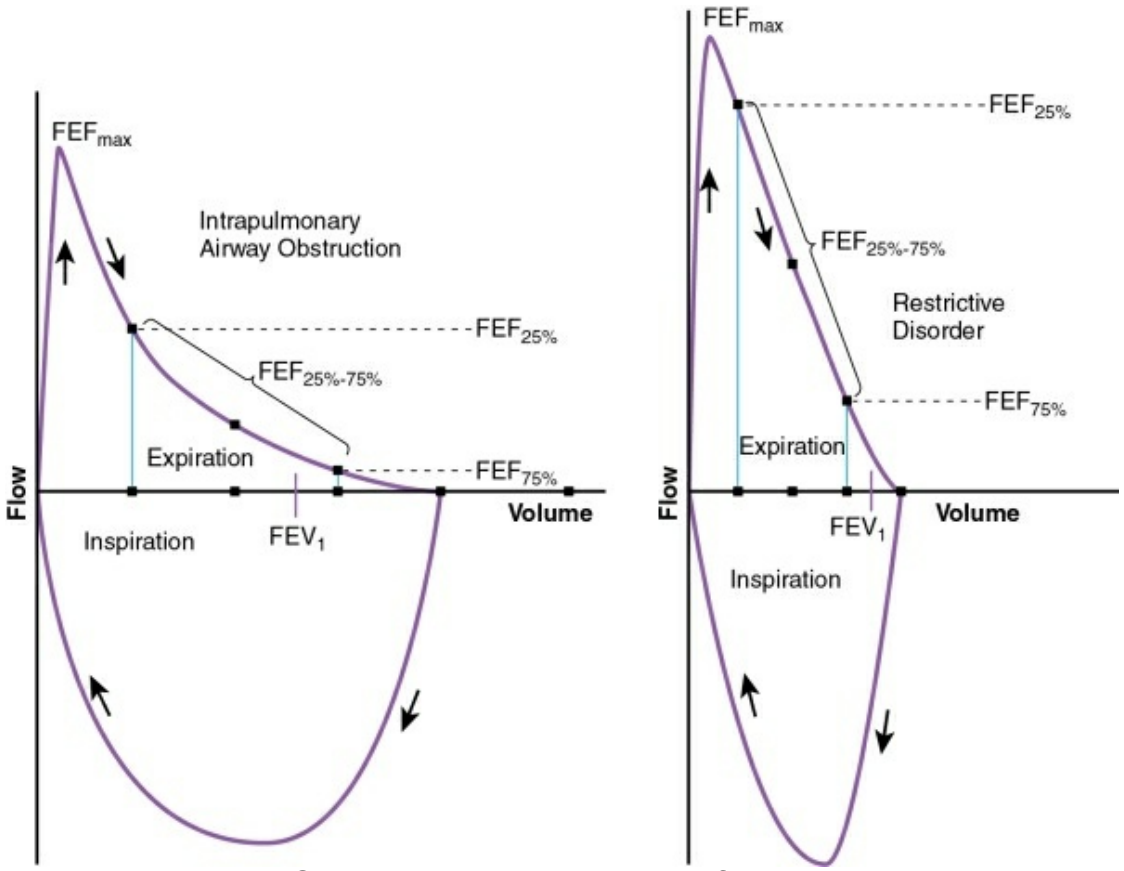

FIGURE 9.11 Obstructive and restrictive flow volume loops. *FEF*, forced expiratory flow; *FEV*, forced exhaled volume. Source: (From Sarnaik AP, Heidemann SM, Clark JA: Respiratory pathophysiology and regulation. In Kliegman RM, Stanton BF, St Geme JW, Schor NF, Behrman RE, editors: *Nelson Textbook of Pediatrics*, ed 20, Philadelphia, 2016, Elsevier, Inc.)

Lung volumes

A restrictive pattern on spirometry must be confirmed with *lung volume testing*. Common lung volume measures include (1) total lung capacity (TLC), which is the volume of air in the lungs after a maximal inspiration; (2) functional residual capacity (FRC), which is the volume of air in the lungs at the end of expiration during tidal breathing; and (3) residual volume (RV), which is the volume of air in the lungs at the end of a maximal expiration. A decreased TLC is indicative of a restrictive pattern. An increased RV is indicative of an obstructive pattern.[24]

Bronchodilator response testing

Bronchodilator response testing is used to assess for the presence of

reversible airflow limitation. To perform the test, the patient should first undergo baseline spirometry. This is followed by the administration of a short-acting beta agonist and repeat spirometry 15 minutes after medication administration. The American Thoracic Society considers a change in the FEV_1 and/or FVC of greater than 12% and 200 mL from baseline as a significant response.[21] The *Expert Panel Report 3: Guidelines on the Diagnosis and Management of Asthma* considers an increase in FEV_1 of ≥12% from the baseline or an increase of ≥10% of predicted after the bronchodilator as significant reversibility.[22]

Quantitative pilocarpine iontophoresis sweat chloride testing

The *sweat test* is the gold standard for diagnosing cystic fibrosis. The test measures the concentration of chloride in sweat after stimulation with pilocarpine. Specific guidelines for collection and analysis have been established by the Cystic Fibrosis Foundation and must be adhered to in order to ensure accuracy of results. A test is considered negative if the concentration of chloride is less than 40 mmol/L, borderline if it is 40 to 60 mmol/L, and consistent with cystic fibrosis if it is greater than 60 mmol/L. In infants, values greater than 30 mmol/L require further evaluation. In addition, values less than 40 mmol/L have been reported in some patients with genetic evidence of cystic fibrosis.[25]

Chest and respiratory conditions

Table 9.7 presents respiratory conditions seen in infants, children, and adolescents by the pediatric health care provider.

TABLE 9.7

Chest and Respiratory Conditions

Condition	Description
Bronchiolitis	Inflammatory obstruction of small airways caused by edema, mucus production; occurs during first 2 years of life with peak incidence at 6 months of age
	Etiology: Viral etiology common with a large percentage

	caused by respiratory syncytial virus (RSV)
	Symptoms: Wheezing, crackles, cough, and rhinorrhea; when severe, nasal flaring, retractions and tachypnea can be present
Epiglottitis	Obstructive inflammatory process of airway that is supraglottic; abrupt onset of high fever, sore throat, drooling, dysphagia, dyspnea, increasing airway obstruction; occurs between 2 and 6 years of age
	Etiology: Bacterial with marked decrease in incidence because of widespread use of *Haemophilus influenzae* vaccine; most cases post–*H. influenzae* due to streptococci and staphylococci
	Symptoms: High fever, difficulty swallowing, drooling, sore throat, rapidly progressing respiratory distress
Asthma	Inflammatory process characterized by airway obstruction and hyperresponsiveness; inflammation plays key role in factors leading to symptoms
	Symptoms: Cough, wheezing, tachypnea, chest tightness, and dyspnea with prolonged expiration
Croup/laryngotracheobronchitis	Acute upper airway obstruction; inflammation and edema of the airway leads to symptoms
	Etiology: 75% due to Para influenza virus; others include influenza A and B, adenovirus, respiratory syncytial virus, and measles
	Symptoms: Hoarse, barky cough that is worse at night, stridor; respiratory distress can occur
Cystic fibrosis	A multisystem disease related to thick secretions that lead to airway obstruction
	Etiology: Inherited, autosomal recessive
	Symptoms: Chronic or recurrent cough, wheeze, recurrent pneumonia or bronchitis
Foreign body aspiration	Lodging of object in larynx, trachea, bronchi (most common site), with degree of obstruction dependent on size/location of object in respiratory tract
	Etiology: Food and small objects are the most common causes; possibility of foreign body must be considered in infants and young children with acute respiratory distress regardless of history; common in children <3 years of age
	Symptoms: Choking, prolonged cough, dyspnea, pneumonia that does not resolve
Laryngomalacia	Immature cartilage of the supraglottic larynx leads to symptoms; it slowly resolves by 12–18 months of age
	Symptoms: Inspiratory stridor with activity/feeding that improves when the child is calm
Tracheomalacia	Weakened/"floppy" trachea that leads to symptoms

	Symptoms: Harsh noise/stridor on expiration caused by airway collapse; onset in early neonatal period; diagnosed by bronchoscopy.
Pneumonia	Infection of lung parenchyma or interstitium; may be primary condition or manifestation of another illness
	Etiology: Bacterial (streptococcus, staphylococcus, etc.) or viral (RSV, parainfluenza, adenovirus, etc.); most commonly caused by viral microorganisms—bacterial pneumonia is less common, but *Mycoplasma pneumoniae* accounts for ~70% of all pneumonias in 9- to 15-year-olds; noninfectious causes such as aspiration should be considered
	Symptoms: Crackles, decreased breath sounds; more severe cases may also see tachypnea, nasal flaring, and retractions
Neonatal respiratory distress syndrome	A condition related to decreased number of branching airways and alveoli, surfactant deficiency (this is the main cause of the disease), atelectasis, impaired gas exchange, and hypoxemia
	Symptoms: Tachypnea, retractions, cyanosis, apnea
Vocal cord dysfunction	Transient obstruction of the upper airway associated with inappropriate adduction of the vocal cords during inhalation. Often mimics asthma.
	Etiology: Can be psychological, physiological (hyperresponsiveness to a trigger), or neurological
	Symptoms: Can be asymptomatic; however, symptoms can range from mild dyspnea to acute respiratory distress.
Primary ciliary dyskinesia	Impaired ciliary function that results in sinus disease, ear infections, and infertility
	Etiology: Typically autosomal recessive
	Symptoms: In infants can present with respiratory distress. A daily productive cough is present in young children.

Data from Tolomeo C: *Nursing Care in Pediatric Respiratory Disease*, Ames, 2012, Wiley-Blackwell; Hoyte FCL: Vocal cord dysfunction, *Immunol Allergy Clin N Am* 33:1-22, 2013; MacConnell LS, Danielsen RD, Symington S: Vocal cord dysfunction, unmasking the asthma pretender, *Clin Rev* 18-24, 2014; Ferkol TW: Primary ciliary dyskinesia (immotile cilia syndrome, Kartagener syndrome). In Kliegman RM, Stanton BF, St Geme JW, Schor NF, Behrman RE, editors: *Nelson Textbook of Pediatrics*, ed 20, Philadelphia, 2016, Elsevier, Inc.; and Roosevelt GE: Acute inflammatory upper airway obstruction (croup, epiglottitis, laryngitis, and bacterial tracheitis). In Kliegman RM, Stanton BF, St Geme JW, Schor NF, Behrman RE, editors: *Nelson Textbook of Pediatrics*, ed 20, Philadelphia, 2016, Elsevier, Inc.

Summary of examination

- Obtain a detailed history (including onset, aggravating factors, associated symptoms, etc.) of the symptom(s) from the parent/guardian and, when possible, the child; a thorough family and past medical history should also be obtained.
- Start the physical examination with inspection of the chest wall for shape and symmetry as well as respiratory rate and effort. Also assess skin for cyanosis and atopy. Relate findings to child's age and symptoms.
- Systematically auscultate breath sounds bilaterally. Note any adventitious sounds and correlate findings with clinical history.
- Systematically palpate chest bilaterally. Note excursion and vocal fremitus; correlate findings with clinical history.
- Systematically percuss chest bilaterally. Note sounds; correlate findings with history.
- All respiratory findings should be related to the child's age and clinical symptoms.

Documentation

Documentation is an important component of the physical examination, and should be organized and complete. Electronic health records have many benefits and allow for charting structured information that is readily available to the healthcare team, and is legible.[26]

DOCUMENTATION

7-month-old infant

Chest: AP diameter 1:1. Respiratory rate 32, rate regular, respirations quiet. No nasal flaring, retractions or intercostal bulging. I:E 1:2, Chest excursion symmetrical. Trachea midline. Vesicular lung sounds across all lung fields.

DOCUMENTATION

8-year-old with acute asthma exacerbation

Chest: Normal AP diameter. Respiratory rate 30. Audible wheeze present. Mild nasal flaring. Coughing spasms present. I:E 1:3. Mild intercostal retractions. Symmetrical chest excursion. Breath sounds equal. Inspiratory and expiratory wheezes scattered throughout all lung fields. No crackles or stridor noted.

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CHAPTER 10

Head and neck assessment

Karen G. Duderstadt

Embryological development

The rapid growth of the head begins during the third to fifth week of embryonic life, as the brain simultaneously undergoes a similar period of rapid growth. By the eighth week, the embryo is humanlike in form, but the head size is dispropo rtionate to the body. During this early fetal development, the head is the fastest growing part of the body and constitutes 50% of the body length at 8 weeks' gestation. The head growth then slows during the period from the 9th to the 12th week in the developing fetus while spine growth accelerates. Disturbances or deficiencies during this early fetal period can result in brain and spinal defects. During the 13th week, ossification of the cranium begins in the skull, which is one of the primary ossification centers of the skeletal system. As the bones of the cranium grow medially from the ossification centers, sutures form at the junctions of the *calvaria* or skullcap, and fontanels form at the intersection of the sutures. The hair patterns on the scalp develop during the 13th through 16th week of fetal development, and the scalp hair present in the term infant is established by the twentieth week of gestation.[1] The fetus continues to grow, and the head size becomes proportional to the body during the second and third trimesters of pregnancy.

Developmental variations

The normal growth of the skull depends on placental function, familial, and hereditary factors, growth potential within the uterus, and optimum nutrition during pregnancy and early childhood. Insufficient vitamin D supplementation during pregnancy and insufficient intake in early infancy is associated with an increased risk of skull deformity in first year of life.[2] The contour of the cranium of the newborn reflects fetal positioning and the effects of the delivery presentation. The cranial bones are pliable and are loosely connected by the sutures and the fontanels, which allow the head to be molded during delivery. Depressed skull fractures are rare, but they can occur with forceps delivery or vacuum-assisted vaginal births. If intrauterine growth retardation does occur as a result of either intrinsic or extrinsic factors in fetal growth, skull growth and brain development are impacted and result in long-term consequences of delayed growth and development in the infant.

Anatomy and physiology

At birth, there are seven movable and separate skull plates, or bones, joined together by sutures that accommodate brain growth (Fig. 10.1). The *fontanels* are the membranous spaces between the frontal and parietal bones and the parietal and occipital bones. The *anterior fontanel* lies along the *coronal*, *sagittal*, and *frontal* or *metopic* sutures (Fig. 10.2). The *posterior fontanel* lies at the juncture of the *sagittal* and *lambdoidal* sutures. There are smaller fontanels located bilaterally in the lower skull. The *sphenoid fontanel* is located at the lower juncture of the frontal and parietal bones superior to the ear and along the coronal suture. The *mastoid fontanel* is posterior to the ear at the juncture of the occipital and posterior parietal bones. The cranium is supported by the first cervical vertebra, the *atlas*, a solid vertebra, and rests on the second vertebra, the *axis.* These bones form the rotational bones of the skull. Ossification of the skull continues throughout infancy and childhood and into adulthood.

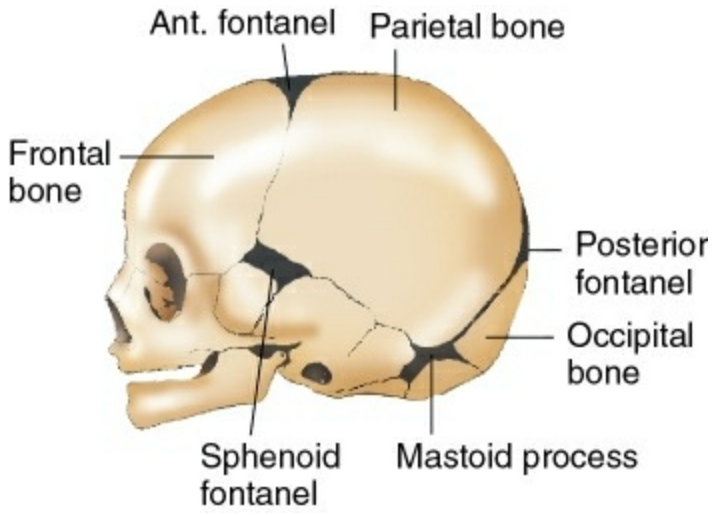


FIGURE 10.1 Anatomy of the skull in the newborn.

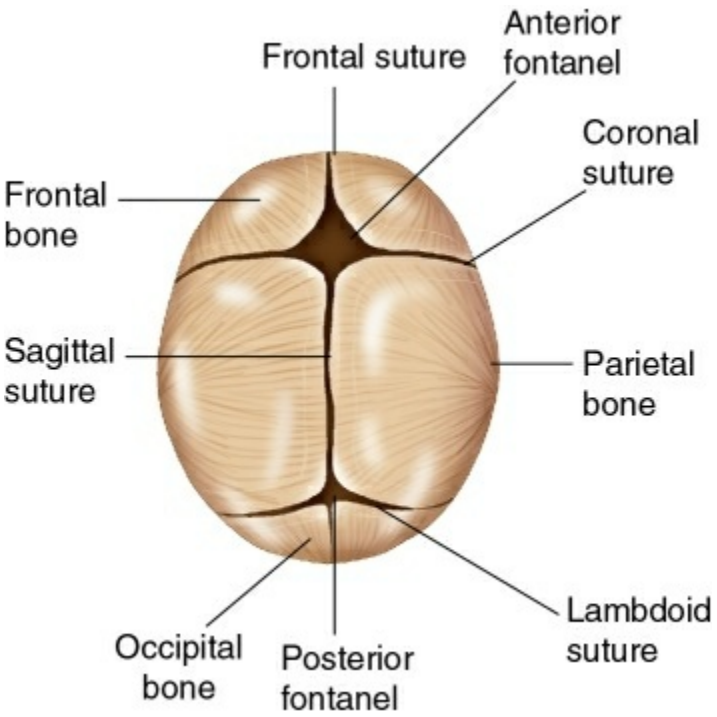


FIGURE 10.2 Fontanels and sutures.

The facial bones are also pliable at birth, except for the maxilla and the mandible, which are very small and underdeveloped in the newborn. The facial skeleton consists of the larger bones of the frontal area, zygomatic processes, *maxilla*, and *mandible* (Fig. 10.3). The two *nasal plates* and the *lacrimal*, *ethmoid*, and *sphenoid* bones comprise the smaller bones in the head. The maxillary and ethmoid

sinuses are present at birth but are small, and the sphenoid and frontal sinuses develop during infancy and childhood.

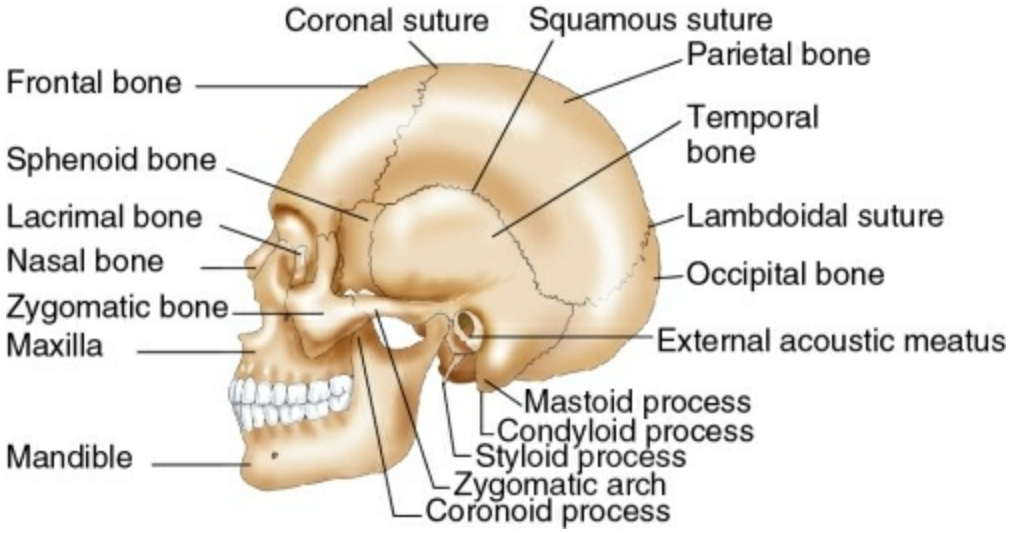


FIGURE 10.3 Facial skeleton.

The muscular structure of the head and neck is an intricate part of the underlying fascia of the cranium and neck structures. The connections between the muscular fascia and the facial orifices control facial expressions such as smiling, raising the eyebrows, and wrinkling the forehead. The superficial and deep muscles of the neck support the pivotal rotation of the head. The *sternocleidomastoid* or *sternomastoid* muscle is the largest muscle in the neck, running from the mastoid area at the base of the ear to the clavicle and sternum, and is primarily responsible for turning the head from side to side. The *trapezius* muscle lies at the back of the neck and is triangular. The origin of the *trapezius* muscle is in the back at the 12th thoracic vertebra extending to the lateral border of the clavicle and attaches at the posterior edge of the occipital bone. It supports the head movement from side to side and the shoulder movement.

The structures in the neck are protected by the deep vertebral muscles, which support the side movements of the head. The *trachea* is the cartilaginous tube that extends from the larynx to the bronchi in the upper chest beneath the sternum. In infants and children, it is more mobile and more deeply recessed in the vertebral muscles than in adults.

The *thyroid gland* lies in the anterior middle region of the neck just below the larynx surrounding the fifth or sixth tracheal ring. There are two butterfly-shaped lobes, joined or connected by an anterior *isthmus* that lies across the trachea below the *cricoid cartilage*. The thyroid gland is composed of tiny follicles filled with *thyroglobulin*, which binds with iodine in thyroid synthesis. A dietary intake of 150 to 200 μg of iodide is enough to produce sufficient quantities of thyroid hormone, and large reserves of iodide are concentrated in the thyroid gland. Thyroid-stimulating hormone (TSH) is necessary for normal growth and development in children and normal sexual maturation. The thyroid gland is extremely vascular and secretes *thyroxine* directly into the bloodstream, which promotes normal growth.

The four *parathyroid glands* are small endocrine glands generally located on the posterior side or dorsal surface of the thyroid gland. Like the thyroid gland, they do not contain ducts, and they secrete *parathyroid* hormone (PTH), which regulates calcium metabolism. Infants born with abnormal development of the parathyroid gland have an embryological defect known as DiGeorge syndrome caused by a chromosomal microdeletion (22q11). It is often associated with congenital heart defects, facial abnormalities, and other systemic defects.

The head and neck region are perfused by the *carotid arteries.* The *external carotid* supplies the head, face, and neck, and the *internal carotid* supplies the cranium. Blood from the cranium is drained through the *subclavian* and *jugular* veins. The thyroid and parathyroid are perfused by the superior and inferior *thyroid arteries.*

Physiological variations

Rapid growth in the infant brings rapid changes in the head and neck. At birth, the *anterior fontanel* ranges in size from an average of 2 cm to 4 to 5 cm in the term infant. The anterior fontanel may be small at birth, about the size of a fingertip, because the skull is compressed during vaginal delivery, and then the fontanel enlarges in the early neonatal period. The *posterior fontanel* may or may not be palpable at birth. It enlarges by a range of 0.5 to 1 cm and closes

by 6 weeks to 2 months of age. Suture lines can be overlapping or protuberant after birth and often are palpable until 6 months of age from molding at birth. Gestational age at birth and sex are not associated with significant differences in fontanel size.[3]

Slow progression of closure of the anterior fontanel occurs during the first year of life. However, closure in some infants may begin by 9 months of age with only a fingertip concavity present at the crown of the scalp, and 25% to 50% of fontanels are closed by 12 months of age. Normally, closure of the *anterior fontanel* occurs by 18 months of age, and 98% to 100% are closed by 24 months of age.[3] Head growth in the first year is determined not by the size of the fontanel, but by the normal increase of the occipital-frontal circumference (OFC) of the head. *Microcephaly*, a small head for gestational age, is considered 1 to 2 standard deviations (SD) *below* the norm for age and size, and *macrocephaly*, a large head for gestational age, is 1 to 2 SD *above* the norm for age and size.

Cephalhematoma is a soft, fluctuating effusion of blood trapped beneath the pericranium caused by rupture of the blood vessels over the parietal area (Fig. 10.4). It is usually unilateral and does not cross the sagittal suture (Fig. 10.5). The bleeding into the periosteum of the cranium may occur slowly, and therefore may not be apparent until the infant is 24 to 48 hours old. A cephalhematoma may be associated with hyperbilirubinemia. Resolution of a cephalhematoma may be slow, and it may persist until 6 weeks to 2 months of age. See Table 10.1 for developmental variations in the head and neck.

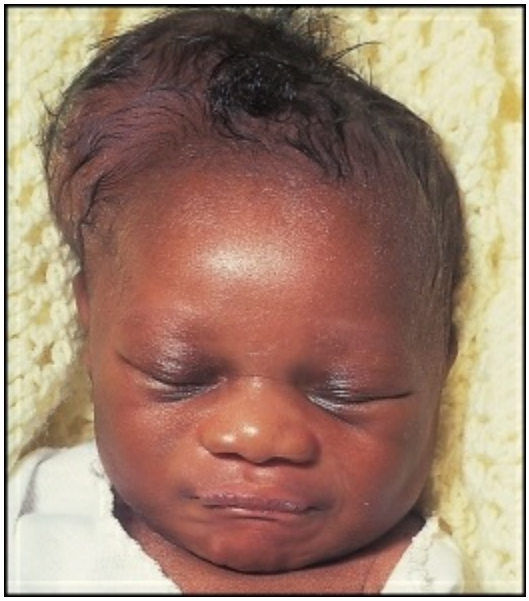


FIGURE 10.4 Cephalhematoma. Source: (From Lissauer TM, Clayden G: *Illustrated Textbook of Paediatrics*, 4 ed. London, 2012, Elsevier Ltd.)

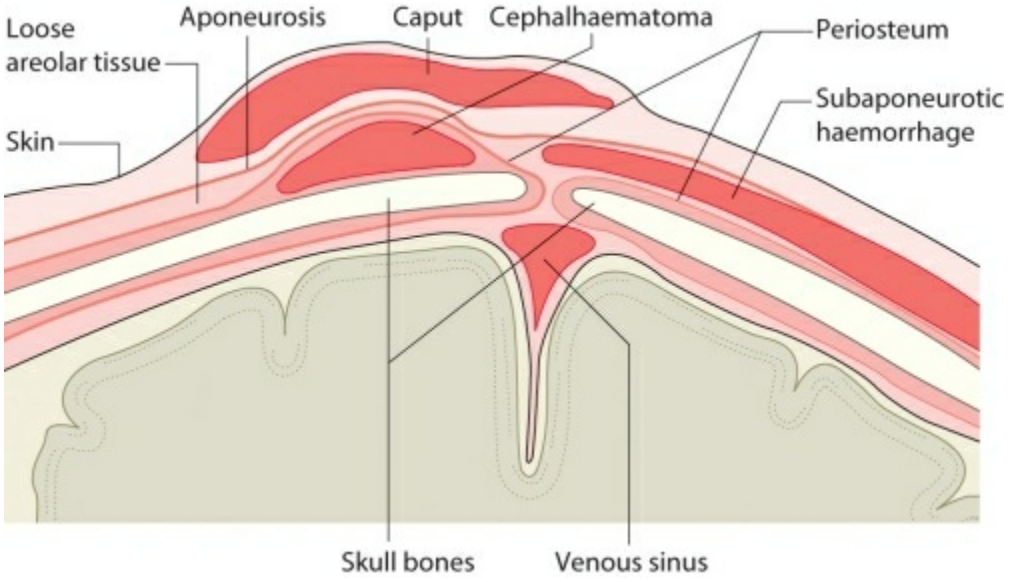


FIGURE 10.5 Location of extracranial hemorrhages. Source: (From Lissauer TM, Clayden G: *Illustrated Textbook of Paediatrics*, ed 4. London, 2012, Elsevier Ltd.)

TABLE 10.1
Developmental Variations of the Head and Neck

Age	Physiological Variations
Preterm infant	Symmetrical or asymmetrical head shape with a flattened temporal/parietal region giving head an elongated shape
Newborn	Symmetrical or asymmetrical head shape with head circumference > chest circumference
Infancy	Symmetrical brain growth is reflected in *occipital-frontal circumference* (OFC) of skull; abnormal growth patterns of skull are indicated by a misshapen cranium and/or a rapidly increasing OFC or slow growth of skull; closure of *anterior fontanel* is expected by 18 months of age in a healthy term infant.
Early childhood	After 18 months, chest circumference exceeds head circumference by 5–7 cm; brain reaches 80% of adult size by 2 years of age; cranium continues to ossify; sutures are proximate and immobile; neck lengthens by 3–4 years of age and neck-to-body proportion is closer to adult proportion.
Middle childhood	Nasal sinus cavities widen and deepen with skull growth, and size approximates those of an adult; thyroid gland is more readily palpable and approximates adult size.

Family, cultural, racial, and ethnic considerations

African American, Hispanic, and Chinese infants have a slightly larger anterior fontanel than do Caucasian infants secondary to familial and genetic factors.[3] Native American infants have an additional horizontal suture line over the occipital bone.

System-specific history

Obtaining a thorough history of growth and development of the head and neck area, including any injury or insult to the skull during the growth process, is an important part of accurate assessment and diagnosis. The Information Gathering table presents pertinent questions to ask about the growth and development of the head and neck area.

Information Gathering for Head and Neck Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant	Perinatal infections? History of intraventricular hemorrhage? History of maternal substance abuse?

Newborn	Vaginal or cesarean birth? Prolonged labor with prolonged third stage? Precipitous delivery? Vacuum-assisted delivery? Shoulder presentation? Respiratory distress at birth? Head tilt? Newborn screening results? History of maternal hyperthyroidism, thyroid disease, or gestational diabetes?
Infancy	Newborn screening results? History of maternal or neonatal infections? Any swelling in neck area? Quality of muscle tone and strength, head control? Achieving developmental milestones?
Early childhood	History of falls, head injury or concussions? Clumsiness or unstable gait? Head tilt, neck pain/stiffness? Persistent lymph gland swelling? History of head trauma or falls, neck pain/stiffness? Use of bike or scooter helmet?
Middle childhood	History of headache? Onset and duration? History of head injury or concussions? Neck pain/stiffness? Use of bike, scooter or skateboarding helmet? Other protective sports equipment?
Adolescence	History of head injury or concussions? Recurrent headaches? Blurred vision? Neck pain/stiffness? Swelling of lymph glands? Any recent weight loss? Use of bike, scooter or skateboarding helmet, other protective sports equipment?
Environmental risks	Maternal exposure to hazardous chemicals or hazardous waste materials? Childhood exposure to pesticides, insecticides, chemical cleaning agents, hazardous chemicals, tobacco smoke, or radiation? Limiting exposure to bisphenol A (BPA) and other plasticizers in the environment?

Physical assessment of the head and neck

The examination of the head and neck area involves inspection and palpation. In infants and young children, examination of the head and neck region may follow the quieter parts of the examination, the cardiac and respiratory exam, as palpation around the head and neck region often makes young children very fearful and uncomfortable, or ticklish, with a response of moving their shoulders upward. In the older child, the physical examination may proceed head to toe. Examination of the head and neck area requires good lighting particularly to view the areas of the neck region during examination. Gloves can be worn if skin lesions are noted.

Inspection of the head and neck

Inspection of the head includes observation by the health care provider for head movement and head control. Head lag when pulling the infant to the sitting position is normal until 3 to 4

months of age in the term infant. Head lag should be evaluated in all infants in the first 6 months of life as an indicator of muscle tone. Persistent head lag between 4 and 6 months of age in the term infant is concerning, and the infant should be monitored closely for the attainment of developmental milestones and referred when indicated.

Head alignment should be evaluated in the young infant with the head at midline on the examining table and in the older infant and young child while being supported in a sitting position by the parent or caregiver. Persistent head tilt from the normal position may indicate *hypotonia*, *congenital torticollis*, muscular abnormalities, gastrointestinal reflux, or visual and hearing deficits. Range of motion and movement of the head should be examined to determine tone and flexibility. In examining the infant younger than 3 months, the provider should move the head passively on the examining table to the left and right to determine mobility and range of motion. At 3 to 4 months of age, the infant can begin to follow a light or small toy to determine the full range of motion of the head and neck and the function of the musculature. Any limited range of motion, head bobbing or jerking, tremors, persistent downward gaze, or involuntary muscle contractions or spasms should be further evaluated and referred when indicated.

Observe the shape and size of the head in the infant and young child. Head shape varies widely in young infants and generally follows a normal growth pattern influenced by familial and genetic patterns and cultural influences. A flattened occipital region or a unilateral flattening of the parietal region can occur in young infants because of prolonged positional placement. Positional skull deformities are often benign and reversible and will normalize by 3 months of age.[4, 5] With the universal recommendation of supine or "back to sleep" positioning of infants to prevent sudden infant death syndrome, there has been an increase in reported cases of *positional or deformational plagiocephaly*. Plagiocephaly refers to an asymmetrical flattening or deformity of the skull and occurs when external forces are applied to the developing skull of an infant and it becomes misshapen, often resulting in asymmetry of the ears and face.[5, 6] Deformational *plagiocephaly* is most commonly seen on the lateral and central side of the occiput (Fig. 10.6).[6] It is more common

in infants with a lower level of motor activity, and with *congenital torticollis* or head tilt.[7] Deformational plagiocephaly may be prevented by varying the head position frequently when putting the very young infant down to sleep on the back and by giving supervised tummy time when awake.

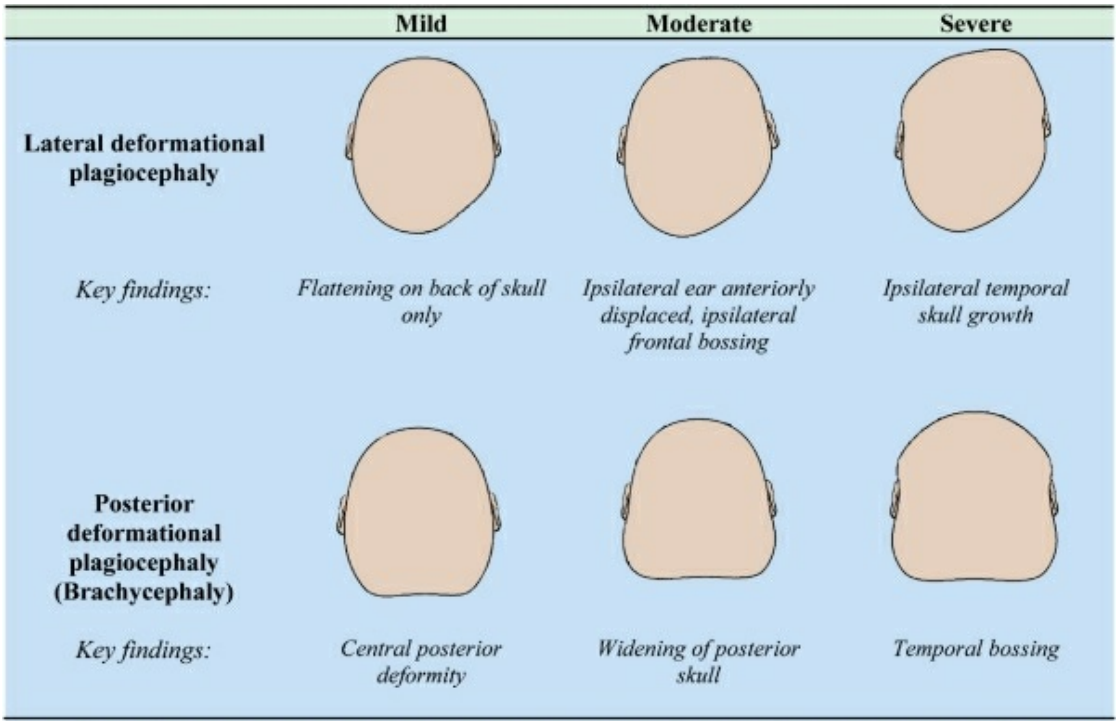


FIGURE 10.6 Deformational plagiocephaly. View of infant's head from crown. Source: (From Looman WS, Kack Flannery AB: Evidenced-based care of the child with deformational plagiocephaly, Part I: assessment and diagnosis, *J Pediatr Health Care* 26(4):242-250, 2012.)

EVIDENCE-BASED PRACTICE TIP

Most positional skull deformities can be prevented by alternating the infant's head position when supine from the left to right occipital area during sleep and providing short periods of supervised tummy time while the infant is awake. [4]

Craniosynostosis is the premature closure or fusion of sutures in the skull of the young infant, and causes a trapezoidal head shape

with flattening of both the occiput and frontal regions on the affected side, occurring in approximately 1 in 2000 births.[4, 7] Craniosynostosis often presents with palpable ridges along the cranial sutures.[6] Health care providers need to be able to properly differentiate infants with positional or deformational plagiocephaly from those with craniosynostosis (Table 10.2). *Lambodial craniosynostosis* occurs in 3 in 100,000 births, and it causes a parallelogram-shaped skull with the ear displaced posteriorly and inferiorly.[7] Always inspect the shape of the head in infants from above and with the infant supported in a sitting position with the head midline to verify the shape of the head and accuracy of the OFC of the head. If any significant abnormal shape of the head is noted, educate parents proactively to decrease the development of occipital and parietal flattening.[4] For moderate to severe deformational plagiocephaly and craniosynostosis, referral is indicated. Repositioning and molding or helmet therapy are recommended for infants over 4 months of age.[7]

TABLE 10.2

Assessment of Positional Plagiocephaly vs. Craniosynostosis

	Craniosynostosis	Deformational Plagiocephaly
Common type	Bilateral coronal Sagittal Metopic	Lateral Posterior
Cause	Premature closure of 1 or more cranial sutures	External force to the skull Prenatal: uterine compression, constrained intrauterine positioning Postnatal: congenital torticollis, sleeping position
Common features	Normal head shape at birth No palpable bony ridges Develops parallelogram shape to head and ipsilateral ear displaced anteriorly	Abnormal head shape at birth Palpable bony ridges Trapezoid shape to head and ipsilateral ear displaced posteriorly
Management/treatment	Referral Surgery Molding or helmet therapy in some cases	Repositioning head when sleeping Physical therapy for congenital torticollis

RED FLAG FINDING

Craniosynostosis often presents with palpable ridges along the cranial sutures. [6]

On inspection of the head, observe the level of the fontanel in the cranium. Normally, there is a slight pulsation in the *anterior fontanel*, and it is fuller or tenses slightly when the infant is crying but flattens when the infant calms. In the ill infant, a tense, bulging anterior fontanel can be a sign of increasing intracranial pressure due to *meningitis* or head trauma and is a medical emergency. Tumors in the brain and meninges can also cause increased intracranial pressure indicated by a bulging fontanel. Infants can have a mildly depressed fontanel, unaccompanied by other symptoms, which may indicate mild dehydration due to the metabolic demands of growth or fluid lost through heat and perspiration. A sunken fontanel, if accompanied by gastrointestinal symptoms, infection, or loss of normal turgor of skin and mucous membranes, may indicate severe dehydration and requires immediate further evaluation and treatment.

The neck should be inspected for symmetry, shape, and mobility. In the newborn and very young infant, the neck is shortened and the musculature is underdeveloped, so it is best to inspect the neck with the infant on a firm surface for examination. While supporting the neck and shoulders cradled with the thumb and forefingers, use the opposite hand to extend the infant's head back slightly to expose the shortened neck region and conduct a full inspection. This position allows the examiner to determine the symmetry and strength of the musculature of the neck, the alignment of the trachea, and the condition of the skin in the infant and young child, who are vulnerable to fungal and bacterial infection in the anterior neck region. *Torticollis*, or wryneck, causes the head to tilt to one side and limits range of motion of the neck muscles. Congenital torticollis is the most common associated finding in infants with deformational plagiocephaly, and early referral is indicated to support repositional therapy for the head.[6] Acquired torticollis in the infant or young child may be related to viral infection or gastroesophageal reflux disease (GERD) associated with

overfeeding, or it may be associated with hypotonia or neurological condition with rotation of the head and extension of the neck.

Infants and young children with unexplained fever, irritability, or a bulging fontanel associated with increased intracranial pressure may indicate pain or neck stiffness during examination of the neck region. *Meningismus* or *meningitis*, inflammation of the brain and spinal cord, can manifest in the neck. To assess for meningismus, flex the head forward or ventrally with the infant or young child lying on a flat surface or examining table. Pain, irritability, resistance to movement, and range of motion on flexion of the neck is a sign of *nuchal rigidity*. *Brudzinski sign* or flexion of the lower extremities will occur spontaneously, with flexion of the head forward when meningismus is present to guard or protect the body from pain. *Kernig sign*, pain or resistance to straightening legs or knees from the flexed position, also indicates a positive sign for meningeal irritation. *Opisthotonos*, hyperextension of the neck and spine, indicates severe meningeal irritation.

In the school-aged child, tilting the head back slightly during inspection of the neck will assist the health care provider in evaluating the structures of the neck. Inspect the *thyroid gland* for size and symmetry, and note any swelling or masses. Asking the child to swallow may assist the provider in distinguishing the structure of the neck and movement of the thyroid gland. The thyroid gland should rise as the child swallows. In children who are obese, adipose tissue in the neck region is common and should not be mistaken for an enlarged thyroid gland.

Inspection of the face

Examination of the facial area begins with initial observation of the infant, child, or adolescent for symmetry of facial features, ears, and facial movements. Observing the smile, laugh, facial creases, and nasolabial folds reveals normal innervation of the facial structures. Observing the symmetry of the facial features in the infant while crying will assist in assessing any facial or neurological injury that occurred during birth. During vaginal deliveries, excessive lateral traction of the head and neck away from the shoulder may injure the brachial plexus and the ventral root of the fifth cervical nerve,[1] and cause paralysis of the arm and shoulder. Injury to the facial

nerve before or during the birth process may cause asymmetrical nasolabial folds and/or asymmetrical facial expression. Any unusual facies with disproportional features, frontal bossing of the forehead, and small or low-set ears may be indicative of a genetic abnormality and require further evaluation and referral.

Palpation of the head

Begin palpation at the crown of the head and evaluate the scalp and the bony structures of the skull. In the infant, palpate fontanels for size, level of tenseness, and pulsations (Fig. 10.7). Note the length and width of the fontanel for infants with unusually large fontanels and monitor the head circumference. Infants and young children who present with a bulging fontanel and signs of increased intracranial pressure may have a resonant or cracked-pot sound, *Macewen sign*, when the scalp is tapped or percussed with the forefinger.

FIGURE 10.7 Palpation of the anterior **(A)** and posterior

fontanels **(B)**.

In the first few months of life, an infant may have a visible or palpable ridge at the sutures lines of the cranium. This is a *metopic ridge* and is normal variant when the head shape appears normal and the OFC is normal. Infants with a wide margin along the sagittal suture may have a communicating *anterior* and *posterior* fontanel, often referred to as a *metopic suture.* Suture lines are normally not palpable after 4 to 6 months of age in the term infant. Premature closure of the sutures may indicate *craniosynostosis*, or asymmetrical growth of the skull, which requires immediate evaluation and referral. Separation of the sagittal suture is one of the most common findings in infants with Down syndrome, along with a flattened occiput and a small rounded head.

When palpating the skull of an infant less than 6 months of age, an abnormal softness of the cranium known as *craniotabes* may be noted, and it is related to an incomplete ossification of bone in the cranium or widened sutures. It can be a normal variant in the term infant; however, if craniotabes is accompanied by abnormal facies or persists after 6 months of age, it may be associated with hydrocephalus or rickets, and further evaluation and referral is necessary.

Palpate for any tenderness, masses, skin lesions, or edema of the skull on examination, and note the size, location, and character of the mass or nodule, and evaluate for mobility and pain. Infants should be monitored closely for increasing head size, misshapen head, or positional deformities. Any abnormal skull findings require diagnostic evaluation and referral as indicated. Any depression on the scalp indicating a skull fracture requires urgent evaluation.

Palpation of the sinuses

Only the maxillary and frontal sinuses can be assessed in school-age children and adolescents by physical examination through inspection and palpation. Evaluate for swelling and tenderness by tapping with the forefingers or applying mild pressure with the thumb or forefinger over the maxillary and frontal sinus area (see Chapter 14).

Palpation and auscultation of the neck

If masses or nodules are noted during the inspection of the neck in the infant, then the provider should palpate the neck to evaluate the size, shape, and character of the mass or nodules. *Brachial cleft cysts* are smooth, nontender masses on the lateral neck area along the border of the sternocleidomastoid muscle. They may be fluctuant and require surgical removal. *Thyroglossal duct cysts* present higher in the neck region and require diagnostic evaluation and referral. Surgical removal may be indicated. Palpation of the lymph glands is reviewed in Chapter 11.

In the infant and young child, palpate the *sternocleidomastoid muscle* for masses, strength, and tone, including the clavicular area at the base of the sternocleidomastoid muscle. Any sign of pain or irritability, or resistance to range of motion of the neck or arm in the infant, child, or adolescent, indicates an abnormal finding and requires further evaluation. Resistance to lateral motion of the neck may indicate torticollis, lymph gland swelling, infection, or trauma to the sternocleidomastoid muscle. If webbing of the neck is noted, it may indicate *Turner syndrome* (Table 10.3).

TABLE 10.3
Head and Neck Conditions

Condition	Description
Bell palsy	Acute unilateral paralysis of cranial facial nerve VII related to postinfectious viral neuritis
Congenital hypothyroidism	Thyroid dysgenesis characterized by prolonged gestation, large for gestational age (LGA), delayed first stool and constipation, poor feeding; infant may have dysmorphic facial features, enlarged tongue, sparse hair/eyebrows with low-set hairline
Congenital syphilis	Bacterial infection transmitted placentally characterized by frontal bossing, depressed nasal bridge, chronic rhinitis, facial lesions circumorally
Congenital torticollis	Contracture of sternocleidomastoid muscle causing tilting of head to one side; occurs secondary to birth trauma, cervical spine, or spinal cord congenital deformities; may be associated with positional plagiocephaly
Craniosynostosis	Premature closure or fusion of sutures in skull of young infant causing a trapezoidal shape with flattening of occiput and frontal regions
Down syndrome	Microcephaly or small rounded head, thick epicanthal folds, almond-shaped eyes or oblique palpebral fissures; flattened nasal bridge,

	large protuberant tongue; ears are low-set, small, and protuberant
Facies of fetal alcohol syndrome	Fetal alcohol exposure characterized by dysmorphic features: microcephaly, short palpebral fissures, wide and flattened philtrum/thin lips; associated with developmental delay
Hydrocephalus	Ventricle enlargement in dura caused by increased production and blockage of or impaired absorption of cerebrospinal fluid; increased head circumference, bulging fontanel, widening fontanel
Hypothyroidism	Acquired thyroid condition usually caused by lymphocytic thyroiditis
Micrognathia	Underdeveloped mandible
Plagiocephaly	Asymmetrical flattening or deformity of the skull from external force or persistent positioning of infant causing misshapen head
Potter syndrome	Renal agenesis characterized by low-set ears, broad nose, underdeveloped chin line, blank appearance
Sandifer syndrome	Acquired torticollis in the infant related to gastroesophageal reflux disease (GERD) associated with overfeeding, or associated with neurological features including rotation of the head and extension of the neck
Torticollis	Contraction of sternocleidomastoid muscle causing tilting of head toward involved side; can be sequela of upper respiratory infection
Turner syndrome	Genetic disorder, a female phenotype; characterized by short stature, webbed neck, pectus excavatum, primary amenorrhea, no development of secondary sexual characteristics

The jugular vein is not normally distended in children who are sitting or standing upright. Pulsations in the jugular vein in the neck may be seen when the child is lying supine on the examining table. The jugular vein should appear full but not bulging, and it is normal to observe jugular venous pulsations (JVPs). The JVP is normally a gentle undulation visible in good lighting. The pulsations should be of normal rate and amplitude, without bruits, or blowing sounds, heard on auscultation over the vessel. Abnormal pulsations can indicate right-sided heart failure or pericarditis. In older children, *carotid pulsations* can be palpated with the finger pads of the second and third fingers in the area between the trachea and the sternocleidomastoid muscle. Evaluate for rate, rhythm, and intensity. Avoid significant pressure to the carotid artery, which may cause a vagal response or hypotension. Auscultation may be performed to detect bruits, but is not routinely performed on children.

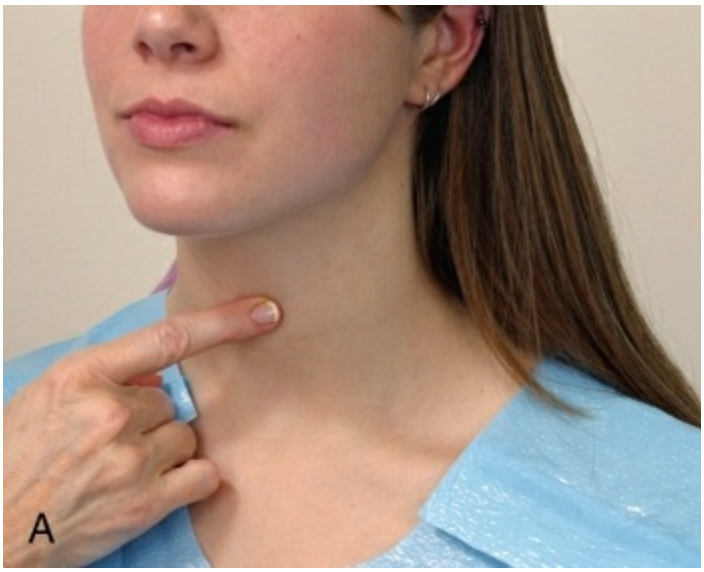
Palpation of the thyroid gland

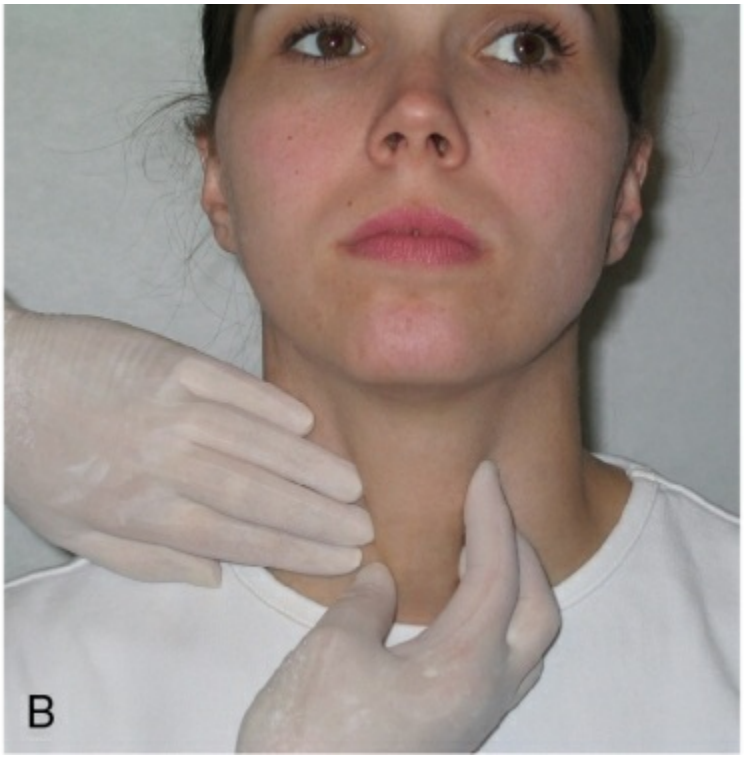
Palpation of the thyroid gland is not usually performed in infants

and young children, unless masses or nodular lesions are noted on inspection of the neck. The neck is short with strong musculature and difficult to palpate. Thyroid disease in infants and young children often presents with systemic symptoms including hypotonia, lethargy, distended abdomen, and enlarged tongue as in *congenital hypothyroidism* (see Table 10.3). Newborn screening for thyroid disease is required in all states, and results should be confirmed by the pediatric health care provider on the initial postpartum or well-child visit.

In school-aged children and adolescents, palpation of the *thyroid gland* is most easily approached from the front of the neck in the young child to minimize any fear that may occur during the examination. Tilt the head forward slightly while the child maintains a sitting position with the back straight. To guide the examination, the provider may gently support the child's head. Begin by using the forefinger to locate the prominent ring of the tracheal cartilage, the *cricoid cartilage*, which provides a landmark to determine the position of the thyroid gland (Fig. 10.8A). Just lateral to the carotid cartilage, palpate the thyroid gland by placing one hand on one side of the trachea and gently displacing the thyroid tissue to the contralateral side of the neck (see Fig. 10.8B). Repeat this movement to examine both sides of the thyroid gland. Once positioned, extend the forefingers of both hands and apply slight pressure for deep palpation along both sides of the trachea and medial to the sternocleidomastoid muscle, and ask the child to swallow. Fingernails must be groomed short for effective examination of the thyroid. Tilting the head to one side or rotating the neck very gently may help evaluate the size, quality, and firmness of the thyroid gland. The thyroid gland can also be examined from behind in adolescents and young adults with the head tilted slightly forward. Using the *cricoid cartilage* as a landmark, the provider uses the forefingers of both hands to palpate deeply, laterally to the trachea, and medial to the sternocleidomastoid muscle (see Fig. 10.8C). A soft, mushy gland, or any masses or nodules noted on examination of the thyroid gland, are abnormal and require prompt evaluation. A *goiter* is a firm, nontender, mobile, symmetrical mass in the neck. Enlargement of the thyroid gland may occur with *hyperthyroidism*,

Graves disease, or *Hashimoto thyroiditis.*





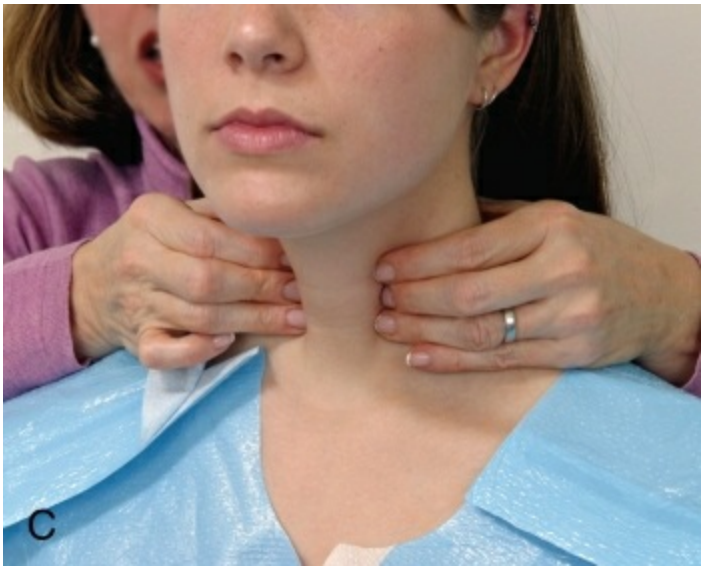


FIGURE 10.8 Examination of the thyroid gland. **A,** Locate prominent ring of cricoid cartilage. **B,** Palpate thyroid gland with forefingers. **C,** Alternative position for palpation of thyroid gland. Source: (**B,** From Fehrenbach M, Herring S: *Illustrated Anatomy of the Head and Neck*, ed 4, St. Louis, 2012, Elsevier.)

Diagnostic procedures

An ultrasound may be the most appropriate initial noninvasive test to perform in an infant with positional or deformational plagiocephaly, or for an infant or child with an abnormal mass in the neck region. Computed tomography (CT) imaging may be necessary in some cases for differentiation of cysts or solid neck lesions. On a child or adolescent with an enlarged thyroid or signs and symptoms of thyroid disease such as weight loss, fatigue, or growth failure, thyroid function test, thyroxine (T_4), and TSH should be performed. Chapter 11 reviews the diagnostics for the lymph glands in the neck region and lymphatic system.

For children or adolescents presenting with a history of moderate or severe head trauma, a CT scan may be indicated to aid in diagnosis. Children must remain immobilized during CT imaging. For diagnosis of possible tumors or malformations in the skull, magnetic resonance imaging (MRI) may be indicated. MRIs require sedation in children and may require injection of contrast for digital subtraction angiography. Skeletal radiography is used to evaluate

cranial abnormalities such as craniosynostosis.

Head and neck conditions

Table 10.3 presents the most common acute and chronic conditions of the head and neck in infants, children, and adolescents.

Summary of examination

- Observe the infant for head control. Head lag is normal until 3 to 4 months of age when pulling the infant to the sitting position.
- Observe shape and size of the head in the infant and young child. Monitor head circumference for abnormal growth. Note any misshapen skull or masses, nodules, or lesions on scalp.
- Fontanels should be palpated for size, pulsations, level of tenseness, or depression of fontanel.
- Inspect neck for symmetry, shape, and mobility.
- Palpate neck for any swelling, masses, or nodules.
- Palpation of the thyroid gland is often omitted in infants and young children unless masses or nodular lesions are noted on inspection.
- To examine the thyroid, begin by locating the cricoid cartilage as a landmark for examination of the thyroid gland. Palpate the thyroid gland by placing one hand on one side of the trachea and gently displacing the thyroid tissue to the contralateral side of the neck. Apply slight pressure with the forefingers. Ask the child or adolescent to swallow.
- Any masses or nodules on the scalp or masses in the neck area require diagnostic evaluation and referral when indicated.

DOCUMENTATION

Term newborn

Head: Normocephalic, anterior and posterior fontanel patent and soft, overriding sagittal suture

Neck: Supple, no masses palpable

DOCUMENTATION

9-year-old male

Head: Normocephalic, no masses or nodules noted, nontender

Neck: Full range of motion (ROM), no lymphadenopathy, palpable adipose tissue over anterior neck region, thyroid firm, without nodules or masses.

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CHAPTER 11

Lymphatic system assessment

Karen G. Duderstadt

Embryologic development

The lymphatic system is established in the mesoderm layer during the third week of embryonic development, and the development of the primary lymphoid organs, the thymus, and bone marrow begins during the fifth to sixth week of fetal development.[1] The secondary lymphoid organs—the spleen, lymph nodes, and lymphoid tissue—develop soon after the primary organs and are well developed at birth. The ectoderm gives rise to the epithelial linings of the glandular cells of the large organs that make up the lymphatic system.[1]

Developmental variations

The spleen, lymph nodes, and lymphoid tissue are small at birth and mature rapidly after exposure to antigens or microbes during the postnatal period. The thymus is the largest lymphoid tissue in the body at birth and continues to develop during the first year of life as the immune system develops. At puberty the thymus begins slowly regressing as the immune system is well established in the lymphoid tissue.[1]

Lymphatic malformation can occur during early embryonic

development as a congenital defect of the lymphatic vessels. The lymphatic channels may become blocked, and lymphatic fluid collects in the vessels during embryonic development, forming a mass or cyst which is present at birth but may not be detected until later in the first year of life or in early childhood. Fetal ultrasound (US) may detect large congenital malformations noted at birth. A lymphatic cyst or mass generally occurs in the neck, mouth, or tissues around the ear and may be associated with other chromosomal abnormalities (Table 11.1).

TABLE 11.1

Common Congenital Cysts and Masses

Condition	Findings
Lymphovascular malformation	Slow flow vascular lesion, formed, benign, usually soft and fluctuant Formed from dilated lymphatic channels of endothelial cells
Branchial cyst	Lateral congenital neck mass, usually anterior to sternocleidomastoid muscle, Formed from incomplete obliteration of pharyngeal pouches and clefts during embryologic development
Dermoid cyst	Midline congenital neck mass, nontender, mobile, submental neck mass Formed from epithelium entrapped in tissue during embryologic development
Thyroglossal duct cyst	Midline congenital anterior neck mass Typically presents as a painful, erythematous, and tender mass following an upper respiratory infection

Data adapted from Rajasekaran K, Krakovitz P: Enlarged neck lymph nodes in children. *Pediatr Clin North Am* 60(4):923–936, 2013.

Anatomy and physiology

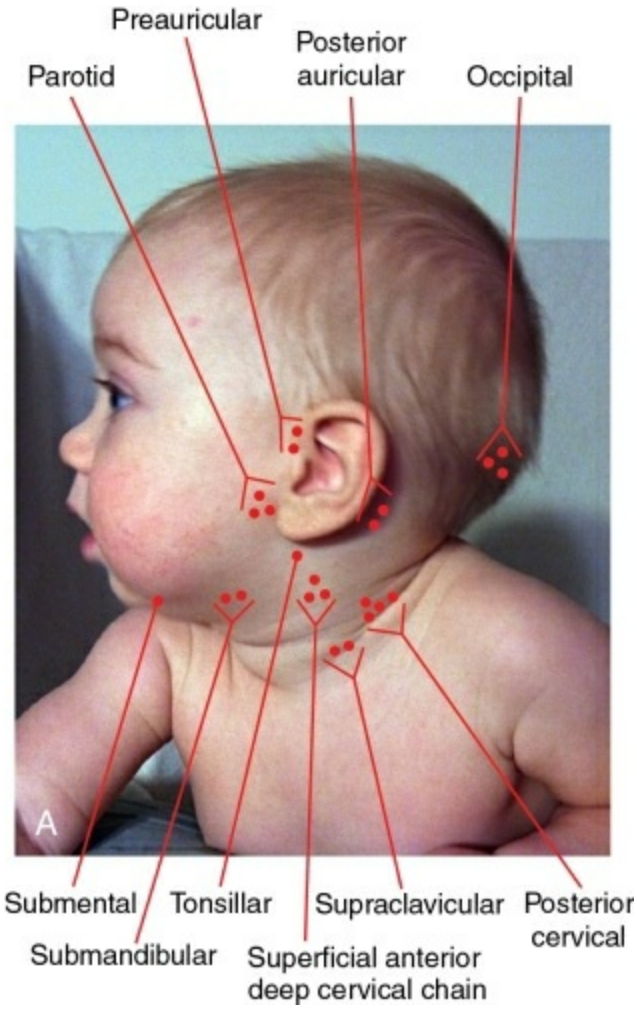
Lymphatic system

The *lymphatic system* is one of the most sensitive indicators of infection and toxins in children. The lymphatic tissue plays a role in the immune system as a first responder to fight infection through phagocytosis, the destruction of harmful cells, and the production of lymphocytes and antibodies. The *lymphatic system* forms an extensive network throughout the body and is composed of capillaries, collecting vessels, lymph nodes, and lymphoid organs.

The bone marrow and thymus, which are the central lymphoid organs, provide the center for the production and maturation of the immune cells.[1] The peripheral lymphoid organs—the spleen, tonsils, appendix, and lymphatic tissue in the respiratory, gastrointestinal, and reproductive systems—concentrate *antigens* or *immunogens* and promote the cellular interactions of the immune response throughout the body to seek out and destroy microbes.[1]

Lymph is a clear, colorless fluid filtered and collected through the *lymphatic capillaries* from the organs and tissues throughout the body. The fluid consists of white bloods cells and occasionally red blood cells. The *collecting vessels* or *lymphatic vessels* are small canals that lie near the blood vessels and carry lymph from the lymphatic capillaries to the lymph glands and back to the bloodstream. The *lymph* is deposited into the bloodstream through the jugular and subclavian veins in the neck. The lymphatic system also absorbs fat and fat-soluble substances from the intestinal wall. The lymph and fat are transported from the lymph glands to the larger ducts and through venous return to the heart. The lymphatic system plays a major role in the maintenance of fluid balance and filters fluid at the lymph nodes and removes bacteria. Obstruction of lymph flow or removal of lymph nodes causes lymphedema.

Lymph glands are small aggregates of lymphoid tissue lying along lymphatic vessels throughout the body and consist of outer cortical layers and an inner medullary layer. The terms *lymph gland* and *lymph node* are often used interchangeably, and both terms can be applied to the lymphatic system. A *gland* is an organ that produces a substance or secretion, and a *node* is a swelling or protuberance. The lymph nodes throughout the body are filters for the collection vessels. Each lymph node processes lymph from the surrounding anatomic area. Lymph nodes remove antigens and microbes from the lymph before it enters the bloodstream, serve as the site of the body's immune response, and aid in the maturation of lymphocytes and monocytes. The immune response within the lymph node ultimately leads to its increase in size.[2] Fig. 11.1 illustrates the lymph glands in the head and neck area and gives a view of the lymphatic chain in the body.


Preauricular
Parotid
Posterior auricular
Occipital
A
Submental
Tonsillar
Supraclavicular
Posterior cervical
Submandibular
Superficial anterior deep cervical chain

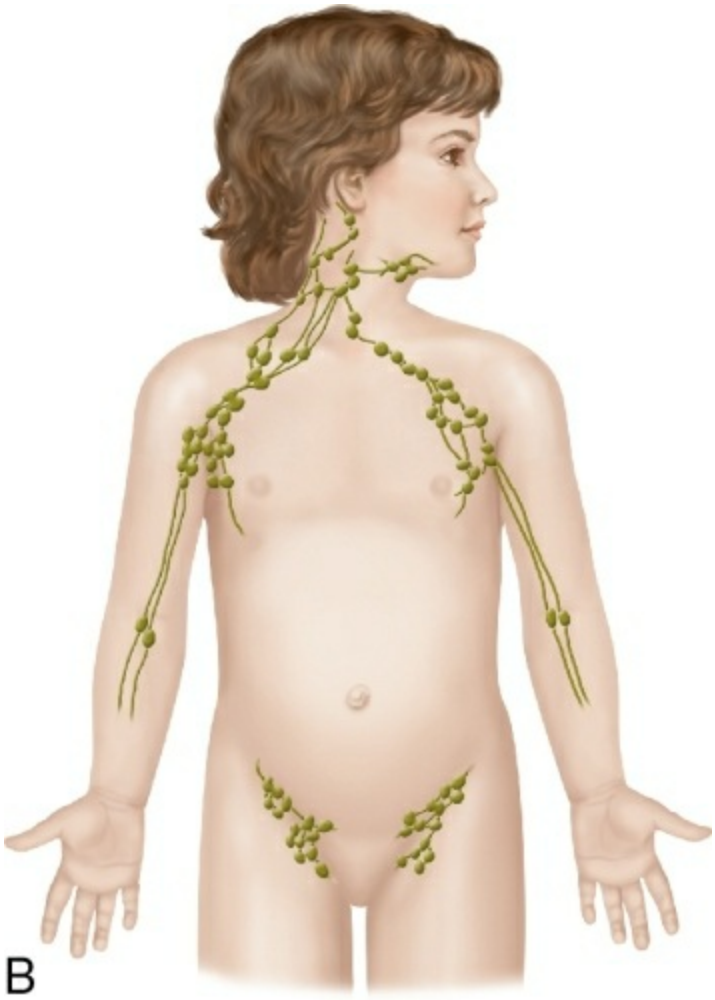


FIGURE 11.1 **(A)** Lymph glands in the head and neck area. **(B)** Lymph glands in the body.

The T lymphocytes are responsible for cell-mediated immunity and aid in antibody production. They are activated in the cortex of the lymph nodes and proliferate to fight antigens. The B lymphocytes are essential for humoral immunity. They interact with the T lymphocytes and migrate to the medulla to mature before releasing antibodies. Many interactions in the immune system depend upon the secretion of chemical mediators such as *cytokines* and *chemokines. Cytokines* are soluble proteins secreted by cells of the immune system and mediate many functions within the cell. *Chemokines* are cytokines that stimulate the immune system and activate inflammatory cells systemically in acute and chronic conditions activating the lymphatic system.[1]

PEDIATRIC PEARLS

Occipital nodes are located high above the hairline in the infant and are often missed by the examiner palpating too low at the nape of the neck. Occipital adenopathy may be an indicator in the newborn of maternal infection during pregnancy or in the infant of an acute viral infection. They may be visible on inspection and are often noted by the parent of a young infant.

The *tonsils* and *adenoids* are organs of the lymphatic system. The buds of *tonsillar tissue* are present in the oropharynx at birth but are underdeveloped. As the immune system develops and reacts to respiratory triggers, such as viral, bacterial, and fungal infections and environmental toxins, the *tonsils* are the first line of defense. The tonsils harbor the immune cells needed to respond to the constant exposure of microorganisms. The adenoidal tissue is a mass of lymphoid tissue situated posterior to the nasal cavity. The *adenoids* are also known as a *pharyngeal tonsil* or *nasopharyngeal tonsil.* Significant hypertrophy of the adenoidal tissue can partially or completely block airflow through the nasal passages and impact the quality of the voice. *Adenoiditis,* the inflammation of the adenoid tissue, often accompanies tonsillitis and occurs mainly in childhood. Incidence decreases with age, due to atrophy of adenoid tissue after adolescence.

The *thymus gland* is embedded beneath the upper sternum above the heart and is a fully developed organ in the term infant. The thymus gland is prominent in the mediastinum of the newborn and infant in the first year of life, often shadowing the cardiac silhouette on radiographs (Fig. 11.2). In the infant the thymus begins to produce mature T lymphocytes and plays an important role in cell-mediated immunity. In puberty, when the immune system is well established, the thymus decreases in size and is gradually mostly replaced by adipose tissue. Thymus tissue persists in the adult but has no demonstrated function.

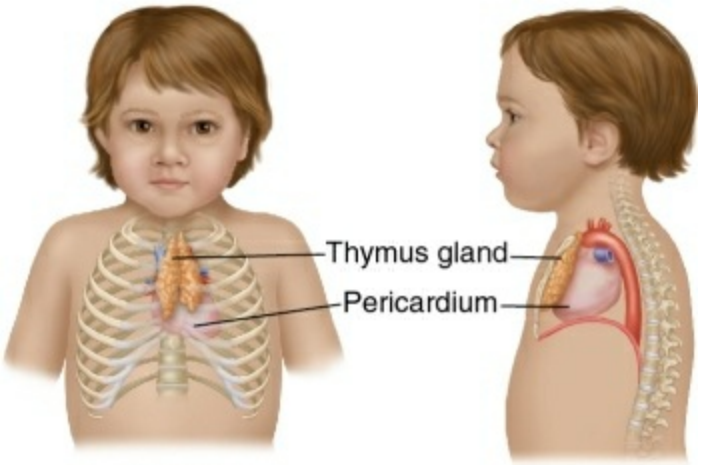


FIGURE 11.2 Thymus gland in young child.

The *spleen* lies in the upper left quadrant of the abdomen protected by the rib cage. It is composed of lymphoid tissue and reticuloendothelial cells and is a densely vascular organ. The spleen acts to filter antigens in the bloodstream and responds to systemic infections. The spleen acts as a part of the immune system and hosts a sequence of activation events similar to the lymph nodes to fight blood-borne infection. In the infant and young child the spleen stores erythrocytes and filters the blood through the large presence of phagocytes. The spleen plays an important role in the immune system and the storage of erythrocytes, but it is a nonvital organ in the body.

Physiologic variations

In the newborn the amount of lymphatic tissue is small, but *lymphadenopathy,* enlargement of the lymph glands, can often be detected particularly in the occipital region as a result of perinatal infections. Lymphoid tissue increases throughout the first year of life, and cervical lymph nodes become more pronounced with respiratory infections by 12 months of age. In the young child, splenomegaly may also occur with episodic viral illness. By school age, tonsillar and adenoidal tissues are approximately the same size as in an adult, and then they increase in volume during pubertal development when tonsillar tissue becomes twice the adult size. During late adolescence the volume of lymphatic tissue begins to decrease and resumes an adult level, which is 2% to 3% of total

body weight.

The variable size of the lymphoid tissue in early and middle childhood may be one of the contributing factors of *obstructive sleep apnea* or *sleep-disordered breathing*, but other common causes include congenital craniofacial abnormalities, chronic nasal allergy, recurrent respiratory infections, environmental irritants, and childhood obesity. All children and adolescents should now be screened for snoring.[2] It is important to differentiate primary snoring from snoring associated with disordered breathing. Pediatric sleep-disordered breathing is characterized by prolonged partial upper airway obstruction and intermittent obstructive apnea that disrupts normal sleep patterns.[3] The prevalence of obstructive sleep apnea is currently 1% to 3% in the pediatric population, and the prevalence of primary snoring is estimated to be 3% to 12%.[4]

System-specific history

When a child presents with *lymphadenopathy* or enlarged lymph glands, obtaining a complete history is key to an accurate assessment of infection in the pediatric population. The Information Gathering table reviews the areas of assessment that are pertinent for each age group and developmental stage and focuses on exposure to infection.

Information Gathering for Lymphatic System Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant and newborn	History of maternal substance/alcohol abuse? Maternal infections or autoimmune disease? HIV+ mother? Neonatal course in NICU? Neonatal infection-sepsis or meningitis? Immunization history in NICU?
Infancy	Neonatal screening results? History of maternal/neonatal infection? History of fever, respiratory infection, exposures? Poor growth or failure to thrive?
Early childhood	History of fever/respiratory infection, exposures? International travel? Persistent lymph gland swelling? Head tilt? Neck pain/stiffness? Anemia? Tonsillitis? Oral candidiasis or chronic diarrhea?
Middle childhood	Lymphadenopathy? History of fever/respiratory infection? Neck pain/stiffness? Exposures? Fatigue? Loss of appetite? History of anemia? International travel? Family history of infections,

	tuberculosis? Snoring? Disrupted sleep from snoring?
Adolescence	Lymphadenopathy? Swelling in extremity? History of fever/respiratory infection? Neck pain/stiffness? Fatigue? Weight loss or gain? Neck swelling? Disrupted sleep from snoring?
Environmental risks	Contact with chemical cleaning agents, hazardous chemicals, radiation, hazardous waste? Recent professional carpet cleaning? Animal dander? Contact with tobacco smoke, water pipes or hookahs?

HIV, Human immunodeficiency virus; *NICU*, neonatal intensive care unit.

Physical assessment

Lymph glands are distributed throughout the body and normally range in size from 3 mm in the head to 1 cm in the neck and inguinal area. In children, lymph glands are often palpable and the pediatric health care provider will often palpate small, firm, mobile lymph nodes along the *cervical* chain on physical examination. These are occasionally referred to as "shotty" nodes because of their pellet like distribution. The provider must be an astute observer when examining the lymphatic system, because each region of the body has clusters or chains of lymph glands that can signal infection in adjacent areas.

Inspection and palpation

It is important to include the *inspection* and *palpation* of the lymph glands regionally during the physical examination. The examination includes palpating lymph glands accessible in four areas: the head and neck, arms, axillae, and inguinal areas of the body. Most effective for palpation are the pads of the examiner's second, third, and fourth fingers. It is important to distinguish between massage and palpation. Superficially massaging an area may not detect nodes, but superficial then deep palpation with the forefingers moving over the neck regions can better determine the size and mobility of a lymph gland. Lymph nodes are generally mobile and nontender and do not feel warm to the touch. Lymph nodes >1 cm in diameter are considered enlarged and defined as *cervical lymphadenopathy*.[2] Inspect enlarged lymph nodes for erythema or edema. Lymph nodes that are immobile, tender, and warm to the touch indicate infection or an abscess and require further diagnostic evaluation and treatment. Table 11.2 reviews

sizing of the lymph nodes commonly used to describe findings on physical exam.

TABLE 11.2
Sizing of Lymph Glands on Examination

Size	Description
1+	Shotty, firm, nontender, <1 cm to >1–1.5 cm, requires deep palpation
2+	Mobile, detectable on superficial-to-deep palpation, >2–2.5 cm
3+	Palpable superficially, visible on inspection, >3–3.5 cm
4+	Lymph glands are walnut size or larger, nonmobile, tender; skin can be reddened and warm; >4–4.5 cm; visible on inspection

PEDIATRIC PEARLS

Visible swelling in the lymph glands on inspection is generally an ominous sign in the child or adolescent and indicates the need for further diagnostic work-up and treatment if indicated.

Head and neck

Assessment of the lymph glands begins with the inspection of the clusters of lymph glands in the head and neck area followed by palpation (Fig. 11.3). The *cervical nodes* constitute the largest collection of lymph glands in the body. In children, the cervical glands are often palpable because of the frequency of respiratory infections. The *posterior auricular* and *preauricular nodes* are often not palpable in the presence of common viral respiratory infections but are enlarged and palpable with infection related to the external and internal ear, the *pinna,* and surrounding skin. The *occipital nodes* in the infant and young child lie on either side of the occiput just above the base of the skull. In the young infant the occipital nodes are located well above the hairline, adjacent to the occipital bony prominence, and are commonly palpable with viral respiratory infections.

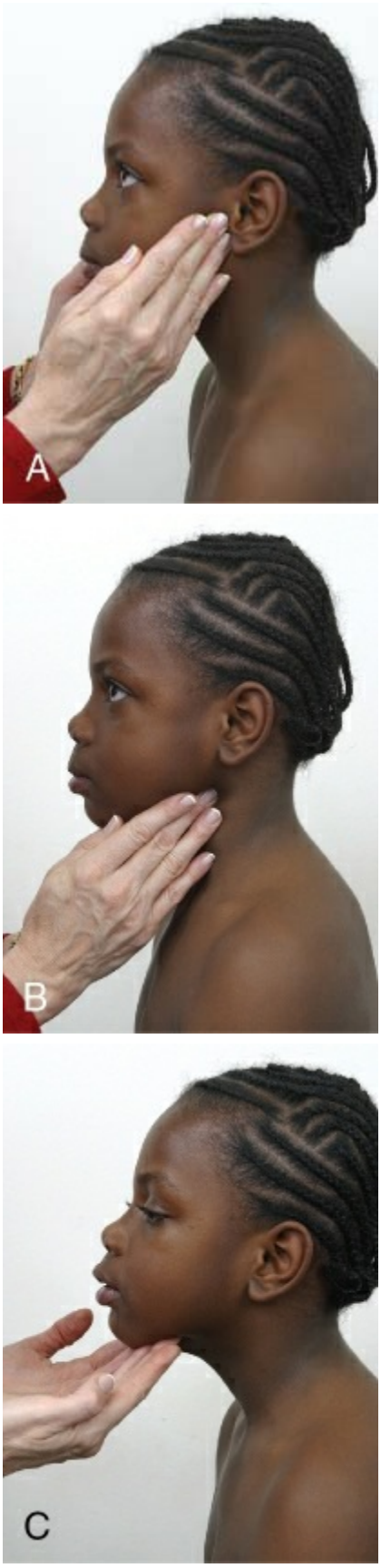


FIGURE 11.3 **(A–C)** Palpation of the cervical chain in a school-age child.

The *parotid* glands are located anterior to the ear and surround the oral cavity. The *submandibular, sublingual,* and *submental* glands are located along the anterior and posterior jaw line and under the anterior jaw. They are palpable when infection occurs in the tongue, mucous membranes of the mouth, sublingual area extending to the base of the tongue, or gums or when decay or abscess occurs in the teeth (Fig. 11.4).

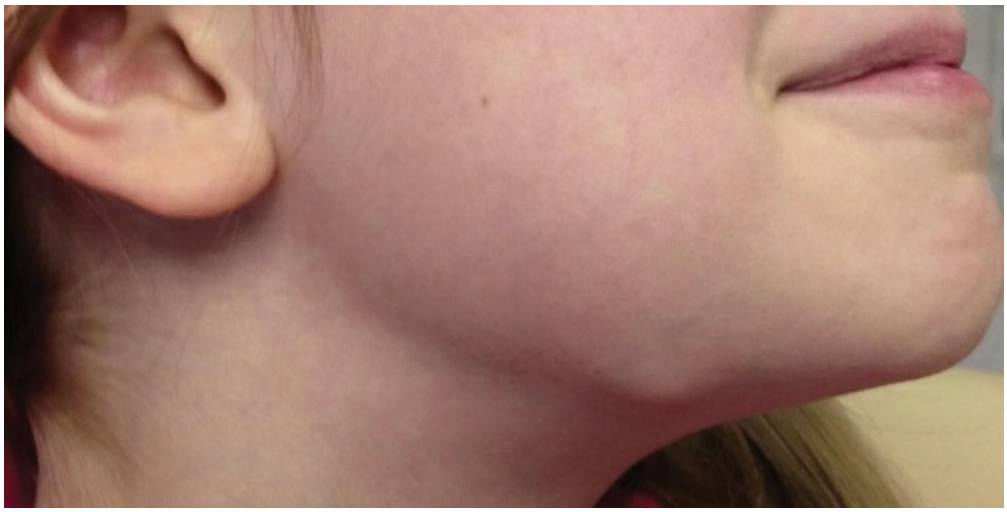


FIGURE 11.4 Enlarged sublingual lymph node. Source: (From Rajasekaran K, Krakovitz P. Enlarged neck lymph nodes in children. *Pediatr Clin North Am* 60(4):923-936, 2013.)

The *anterior superficial cervical nodes* are palpable at the juncture of the *mandible* and *sternocleidomastoid muscle* at the neck. This examination requires both superficial and deep palpation, depending on the age and developmental stage of the child or adolescent. These glands are almost always palpable during throat and respiratory infections, but may be missed due to incorrect positioning of the fingertips during the assessment of the neck. The *posterior superficial cervical nodes* along the posterior neck area are palpable with systemic infection (Fig. 11.5), and *posterior deep cervical nodes* are rarely palpable in children and are only identifiable with deep palpation in the older child. The *supraclavicular nodes,* along the clavicle, are rarely palpable and if enlarged may indicate a malignancy (Fig. 11.6). Visible swelling in the lymph glands on inspection is generally an ominous sign in the

child or adolescent and indicates the need for further diagnostics and prompt consultation and referral. Table 11.3 presents conditions associated with lymph node findings on physical exam.

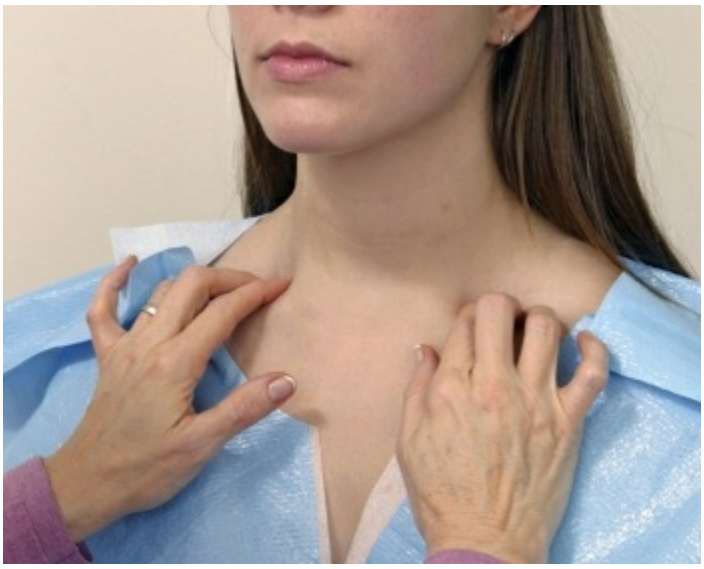


FIGURE 11.6 Palpation of the supraclavicular nodes.

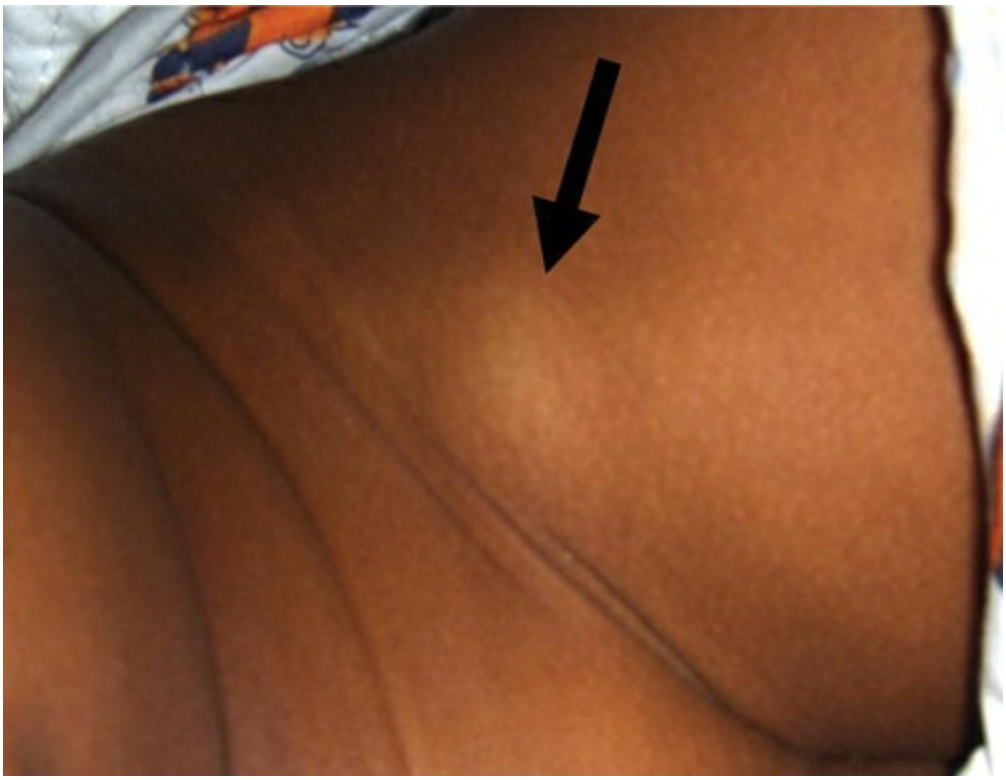


FIGURE 11.5 Enlarged posterior superficial cervical

nodes. Source: (From Kelly MN, Tuli SS, Usher S, Tuli SY. A 6-year-old with acute-onset generalized lymphadenopathy. *J Pediatr Health Care* 26(6):465-470, 2012.)

TABLE 11.3
Lymph Node Findings and Associated Conditions

Condition	Lymph Node Findings
Viral infection	Soft to palpation Not fixed to underlying tissue or structures
Bacterial infection	Tender Fluctuant Not fixed to underlying tissue or structures
Abscess formation	Tender, fluctuant lymph node Erythematous Warm to touch
Mycobacterium tuberculosis	Erythematous lymph node
Atypical mycobacterium	Matted Skin involvement
Malignancy	Hard to palpation Often fixed to underlying tissue No signs of acute inflammation

Data adapted from Rajasekaran K, Krakovitz P: Enlarged neck lymph nodes in children. *Pediatr Clin North Am* 60(4):923–936, 2013.

Abdomen

Palpation of the *spleen* is an important part of the examination for the lymphatic system, and tenderness and enlargement of the spleen on deep palpation indicates the need for further diagnostic evaluation. The examination of the spleen is reviewed in Chapter 15.

The *inguinal nodes* can be palpated along the juncture of the thigh and abdomen and along the inguinal ligament and the saphenous vein. The horizontal chain of inguinal nodes runs along the inferior groin, and the vertical chain can be palpated on deep palpation along the upper inner thigh. If lymph glands are enlarged in the inguinal area, they are often visible in normal weight children when lying supine on the examining table. Fig. 11.7 shows a young child in the supine position for examination of the inguinal nodes.

Inguinal lymphadenopathy often occurs with systemic viral infection in the pediatric patient.

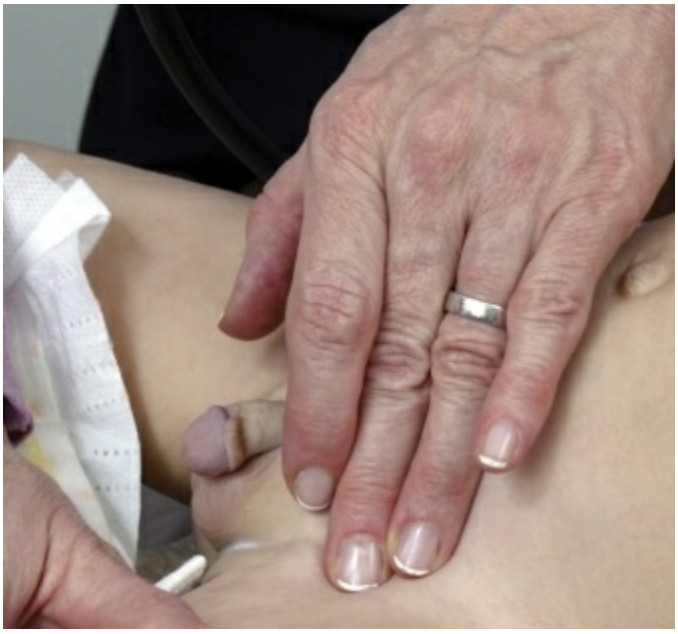


FIGURE 11.7 Palpation of inguinal nodes in a young child.

Extremities

The *axillary, brachial,* and *subscapular* nodes lie anteriorly along the brachial artery and in the axillae along the lateral edge of the pectoralis major muscle. They are generally noted in children only on deep palpation unless enlarged. Examination of the *subscapular* nodes of the axillary chain is illustrated in Fig. 11.8. The *epitrochlear* nodes lie along the medial aspect of the arm above the elbow and are palpable over the *humerus.* The *epitrochlear* and *popliteal* nodes are the only peripheral lymph nodes in the lymphatic system and act as collection ducts for the limbs. Table 11.4 reviews some of the causes of regional and systemic lymph node swelling in infants, children, and adolescents.

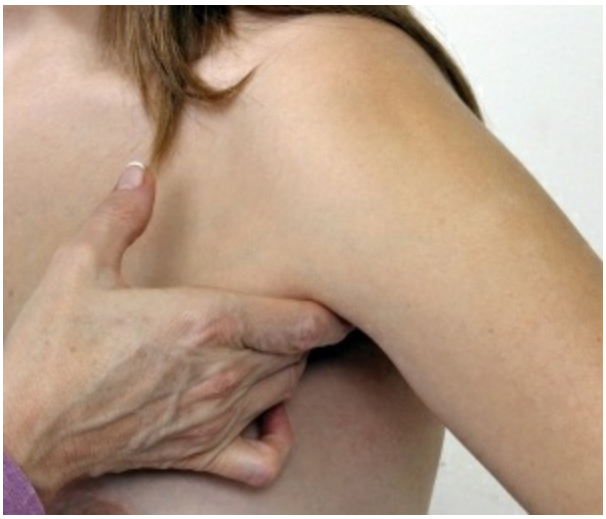


FIGURE 11.8 Palpation of the subscapular nodes of the axillary chain.

TABLE 11.4
Regional and Systemic Causes of Lymphadenopathy

Region	Related Causes
Occipital	Scalp infections such as seborrheic dermatitis, tinea capitis, pediculosis/head lice; viral syndromes such as varicella, measles, rubella, roseola; viral respiratory infections (i.e., rhinovirus, RSV, postimmunization)
Preauricular, parotid, postauricular, and superficial cervical	Infection of pinna (ear), otitis externa, middle ear infection; parotitis
Cervical glands—tonsillar, sublingual, submandibular, deep cervical	Tonsillitis, pharyngitis (Group A streptococcal (GAS), mycoplasma pneumonia), stomatitis; tooth decay, dental abscess; ear infection; oral mucosa/mucous membrane infections, tongue; cervical lymphadenitis from systemic infections (Epstein-Barr (EBV) virus, cytomegalovirus (CMV)); neoplasm or cancer; postimmunization response
Axillary	Breast infections, thoracic wall inflammation, infections of shoulder and arm, systemic infection, or neoplasm in lymphatic system
Supraclavicular and subclavian	Neoplasm or cancer—metastatic cancers from respiratory, gastrointestinal, or lymphatic system
Epitrochlear and popliteal	Forearm and finger infections, infection secondary to fractures, skin infections, neoplasm, or cyst in lower extremity, venous insufficiency, cardiac or renal disorder
Inguinal	Diaper rash, gluteal and perineal infections; skin infections in lower abdominal area; foot and leg infections, systemic viral infections
Generalized lymphadenopathy	Systemic disease occurring in lymphatic, circulatory, respiratory, gastrointestinal, or genitourinary system; infections such as

	tuberculosis, HIV

HIV, Human immunodeficiency virus; *RSV*, respiratory syncytial virus.

Diagnostic procedures

For evaluation of lymphadenopathy in infants and children, a complete blood count (CBC) with differential, peripheral blood smear, blood culture, erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP), rapid streptococcal antigen test and throat culture,[5] and purified protein derivative (PPD) intradermal testing or interferon-gamma release assay QuantiFeron testing are commonly used. Ultrasound (US) is used, if indicated, for initial imaging of palpable neck masses in infants and children to avoid radiation exposure with computed tomography (CT).[2] Magnetic resonance imaging (MRI) may be indicated for pediatric neck masses compared with CT because it limits radiation exposure but requires sedation and is less readily available. Biopsy of the lymph nodes may be indicated when the diagnosis is unclear after initial work-up, systemic symptoms persist, or an isolated, enlarged lymph node or nonmobile mass persists that does not respond to antibiotic therapy.

Children with primary snoring and symptoms of obstructive sleep apnea require a full-night sleep study or polysomnography (PSG) for confirmative diagnosis of pediatric sleep-disordered breathing.[6]

Lymphatic conditions

The extensive lymphatic system throughout the body provides the pediatric health care provider with a map in times of illness. The patterns of drainage leading to the regional lymph glands are indicative of infections that occur in different areas throughout the body. The head and neck region is the area of the body with the highest concentration of lymph glands, and even mild infections in children cause swelling in the regional lymph glands. However, generalized swelling of the lymph glands indicates a systemic source of infection, and general lymphadenopathy is more likely to occur in children than in adults. Accurate assessment and diagnosis

may depend on the pediatric health care provider's knowledge of the lymphatic drainage system.

Lymphadenopathy in children is generally episodic, benign, and self-limiting and is most often associated with viral infections. *Cervical lymphadenitis* refers to enlarged, inflamed, and tender lymph node(s) of the neck; although *lymphadenitis* refers to inflamed lymph nodes, the terms lymphadenitis and "lymphadenopathy" are often used interchangeably. Table 11.5 reviews common pediatric infectious conditions presenting with lymphadenopathy.[2, 7]

TABLE 11.5
Lymphatic Conditions

Condition	Description
Cat-scratch disease	Bacterial infection *(Bartenolla henselae)* caused by scratch or bite from contact with kitten or cat; initial lesion on face/arm area; fever may or may not be present; regional lymphadenopathy presents after incubation period of 7–60 days following the scratch, axillary is most common presentation
Cervical lymphadenitis	Marked swelling most commonly unilateral in tonsilar or retropharyngeal cervical node, although other lymph glands can be involved; characterized by tenderness, >4+ swelling *Etiology:* Group A β-hemolytic streptococci and *Staphylococcus aureus* are the most common causes of bacterial cervical lymphadenitis, in young infants Group B *Streptococcus* (GBS)
Hodgkin lymphoma	Malignant neoplasm of lymph system characterized by painless, enlarged lymph nodes often cervical, supraclavicular or axillary nodes, generally asymmetric, nontender, firm; swelling in supraclavicular nodes is ominous sign of disease; most common malignancy associated with cervical lymphadenopathy in 15 to 19 year old age group
Infectious mononucelosis	Acute clinical manifestation of Epstein-Barr virus characterized by splenomegaly with accompanying tenderness, cervical adenopathy 3+ to 4+; may be tender/firm; tonsillar hypertrophy, pharyngitis, malaise, fever
Acute Lymphoblastic Leukemia	Most common malignancy of childhood with peak incidence between 2 and 5 years of age characterized cervical lymphadenopathy, splenomegaly or hepatomegaly, pallor, fever, loss of appetite; may present with purpura or petechiae
Pediatric *sleep-disordered breathing*	Prolonged partial upper airway obstruction and intermittent obstructive apnea that disrupts normal sleep patterns characterized by frequent snoring (≥3 nights/week), labored breathing during sleep, gasps, snorting noises or observed episodes of apnea, and daytime sleepiness
Roseola infantum	Viral exanthem of infancy characterized by a prodromal of high fever for 3–4 days and swelling of occipital and postauricular nodes; with defervescence, mildly erythematous morbilliform rash appears over

	trunk
Group A streptococcal pharyngitis	Acute onset of bacterial pharyngitis with swollen and tender tonsillar or retropharyngeal cervical nodes, fever, malaise, positive history of abdominal pain, and scarlatiniform rash may appear over trunk.

Summary of examination

- Examination of the lymphatic system includes *inspection* and *palpation* of lymph glands regionally (head and neck, arms, axillae, and inguinal areas of the body) during the physical examination.
- In the young infant the *occipital nodes* are located well above the hairline, adjacent to the occipital bony prominence, and are commonly palpable with viral respiratory infections.
- The *inguinal nodes* can be palpated along the juncture of the thigh and abdomen over the inguinal area along the inguinal ligament.
- Lymph nodes are generally mobile and nontender and are not warm to the touch.
- Lymph nodes that are immobile, tender, and warm indicate infection or an abscess and require further diagnostic evaluation and treatment.
- Visible swelling in the lymph glands on inspection is generally an ominous sign in the child or adolescent and indicates the need for further diagnostics and prompt consultation and referral.

DOCUMENTATION

Adolescent with lymphadenopathy

Neck: 3+ tonsillar lymph nodes, mobile, warm, tender to touch, neck supple with full range of motion (ROM), no meningismus noted.

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3. Marcus, C Brooks, L Draper, K Gozal, D Halbower, AC Jones, J. *Diagnosis and management of childhood obstructive sleep apnea syndrome*. *Pediatrics* 2012;130(3):576-584.
4. Church, G. *The role of polysomnography in diagnosing and treating obstructive sleep apnea in pediatric patients*. *Curr Probl Pediatr Adolesc Health Care* 2012;42(1):2-25.
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7. Penn, Jr EB. *Goudy SL. Pediatric inflammatory adenopathy*. *Otolaryngol Clin North Am* 2015;48(1):137-151.

CHAPTER 12

Eyes

Karen G. Duderstadt

The eye is the most remarkable organ in the body as it is underdeveloped at birth and undergoes dramatic development in the first year of life. Although the eyes are anatomically complete, the sense of vision begins to develop over the first weeks and months of life. The development of normal vision depends on the reception of light rays that stimulate the internal eye during the early critical period after birth. Visual acuity is also determined by an infant's genetic predisposition, as well as physical and environmental health factors. Eye conditions that limit or occlude the process of visual development in the first year of life may affect the long-term visual health of the infant and child. A complete and thorough eye examination by the pediatric health care provider is critical to early detection and intervention to assure optimum visual development for infants and children.

Embryologic development

The development of the eye begins in the third week of embryonic development, with the optic grooves forming in the neural tube, and by the end of the fourth week the optic vesicle is formed and lies close to the ectoderm surface. The surface of the ectoderm thickens and forms the lens placode. By the fifth week of gestation the optic cup and the lens cavity are formed, and by the sixth week, invagination of the optic vesicles occurs, forming the choroid layer and vasculature of the eye. The formation of the cornea, lens, and

anterior chamber proceeds during the seventh and eighth week, and the eyelid folds develop and begin to cover the palpebral fissure. It is during this period of gestation that insults to the fetus, such as maternal rubella and varicella cause the development of congenital cataracts. Development of the retinal tissue continues with the differentiation of the nerve fiber layer, proliferative layer of the macula, and the pigmented layer of the retina that precedes the development of the iris and the ciliary body, which occurs between the 9th and 12th weeks of embryonic development. The ciliary body gives rise to the ciliary muscles, which control the accommodative reflex and pupillary aperture (Fig. 12.1). Development of the structures of the eye is complete by 15 weeks gestation. During the fourth month of gestation, the development of the retinal blood vessels is initiated and full vascularization of the retina occurs just before birth in a term infant and is incomplete in the preterm infant. The pupillary light reflex, which requires an intact optic nerve and oculomotor nerve, can be elicited by 30 weeks gestation.

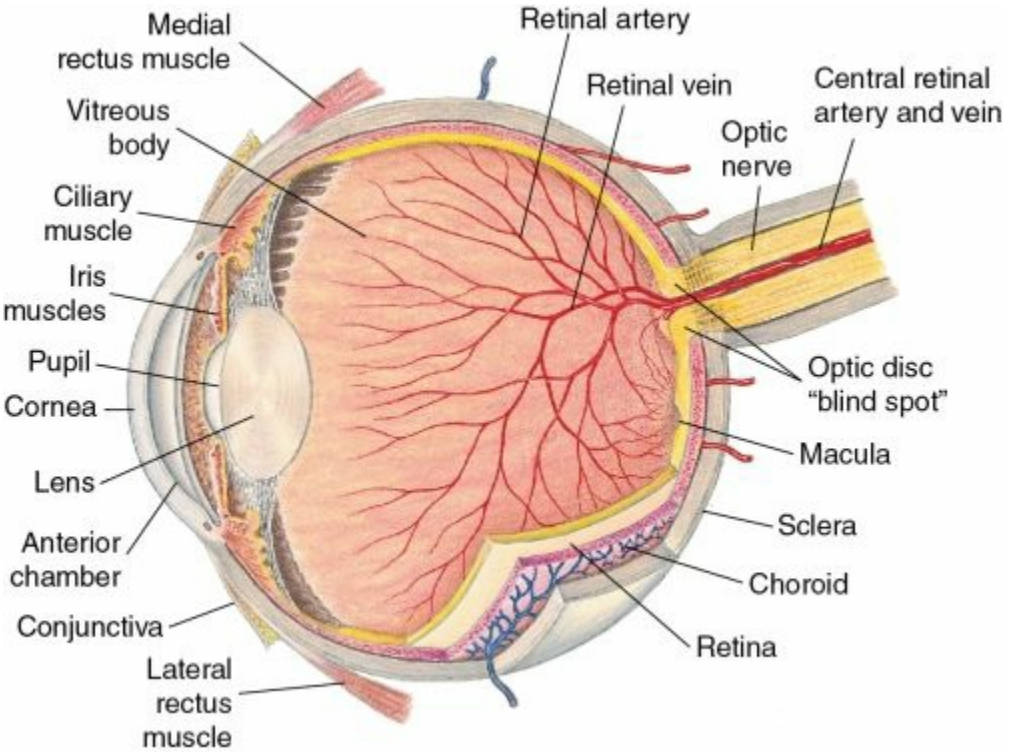

FIGURE 12.1 Anatomy of the human eye. Source: (From Seidel H, Ball J, Dains J, et al: *Mosby's Guide to Physical Examination*, ed

7, St. Louis, 2011, Mosby.)

Developmental and physiologic variations

Visual development begins after birth at the very center of the *retina* in the *macula.* The macula is a circular area surrounding the *fovea.* At birth, the macula is not fully developed but holds the genetic potential for 20/20 vision. The retina predominantly consists of rods at birth, and the cones are located near the outermost layer of the retina. As the retina is exposed to light, the cones migrate toward the center to become the anatomical macula on the *fundus* located on the posterior surface of the retina. The lens has a key role in focusing light on the neural layer of the retina, and the ciliary muscles assist in accommodation of the lens.

The *optic nerve* is also developing at the same time as the retina. There are as many as 8 million cells in the optic nerve and 5 million retinal cells in the optic neuron at birth that compete for synaptic sites on the nerve. Cells that are not oxygenated do not develop. In almost all term Infants the retina and retinal vasculature is fully developed.[1] *Hypoxia* or *hyperoxia* in preterm infants often impacts the normal development of the retina and causes a *retinopathy,* a disorder of the retinal vessels. Preterm infants have an avascular zone in the periphery of the retina that disrupts the normal proliferation of the retinal vessels and interrupts blood flow to the visual receptors. The disruption of blood flow may lead to retinal detachment from the choroid, causing visual impairment or blindness if left untreated. The extent of the immaturity of the retina and the avascular zone depends mainly on the degree of prematurity at birth.[1] Very low birth weight infants (≤1500 g) are at highest risk for *retinopathy of prematurity* (ROP), and infants born at 28 weeks gestation or weighing ≤1000 g are at risk for developing macular folds and retinal detachment.[2] Refractive anomalies also occur approximately eight times more frequently in preterm infants than in term infants.[3] Infants with a birth weight of ≤1500 g or a gestational age of 30 weeks or less and infants with a birth weight between 1500 and 2000 g or gestational age of greater than 30 weeks with an unstable neonatal course should be referred to ophthalmology for retinal screening.[1]

Term infants from 36 to 40 weeks of gestation can perceive shape, color, motion, and patterns at birth. Visual development in the newborn is dependent upon development of the visual pathways that link the eyes to the lateral geniculate nucleus in the thalamus and to the visual cortex located in the occipital lobe of the brain. Exposure to light begins the synaptic development of the neurons at birth.[3] Central fixation is present shortly after birth in the term infant, and the human face at a distance of 8 to 12 inches holds the most visual interest for a newborn. The term infant is *hyperopic,* or farsighted, at birth. Visual images are focused behind and not on the retina, so the visual image is blurred. For the infant to see near objects, the *ciliary muscles* of the eye must work hard to accommodate, or shape, the lens; in time these efforts of the *ciliary muscles* result in thickening of the lens, which makes accommodation of the image onto the retina possible.

The term infant's eyes often wander or deviate in the first 6 weeks when trying to achieve visual fixation in the *central field* of vision. After 6 weeks of early visual stimulus, an infant is able to focus and visually follow an object or the parent's movements. Any inability to visually focus after 6 weeks of age in a term infant is considered suspect and at 3 months of age is considered abnormal. As the eyeball or *globe* grows, the hyperopia decreases and the lens hardens, but detailed visual acuity is not present until 3 to 4 months of age in the healthy eye and not until 3 to 4 months corrected age for preterm infants (Table 12.1). Differentiation and maturation of the fovea and the retinal layers of the macula continues until 8 months of age.[3] The *fovea* is responsible for central vision, and the *macula* is responsible for colors and contrasts, and normal development of both are required for precise visual acuity. The critical window for normal development of full visual acuity is from birth to 5 to 6 years of age, and the synaptic development of the neuronal paths in the visual cortex continues until approximately 10 years of age. After this age, conditions that affect early visual development cannot be completely corrected (Table 12.2).

TABLE 12.1
Visual Development

Age	Developmental Stage of Vision
Birth	Awareness of light and dark
Neonatal	Rudiments of fixation on near object
2 weeks	Intermittent fixation
4 weeks	Follows moving objects
6 weeks	Fixates and follows moving objects
8 weeks	Convergence beginning to stabilize
4 months	Inspects hands and small held objects; vision 20/300
6 months	Retrieves small objects; hand-eye coordination appears
9 months	Binocular vision clearly established; beginning of depth perception
12 months	Vision 20/180; looks at pictures with interest; fusion is established
18 months	Convergence established; visual localization peripherally poor
2 years	Accommodation well developed; vision 20/40 to 20/50 in normal eyes

TABLE 12.2
Physiologic Variations of the Eye

Age Group	Variations
Preterm infant	• 24 weeks: Partially fused eyelids • 24–28 weeks: Eyelids open spontaneously • 28–30 weeks: Eyes have membranous embryonic vascular network over iris to protect lens, producing dull retinal or red light reflex • 36–40 weeks: Membrane over iris normally resolves; persistent membrane may result in anterior cataracts
Newborn	• Macula not fully developed, eyes tend to drift in initial newborn period • Benign scleral hemorrhage often present after birth • Definite ability to follow object not developed until 4–6 weeks • Lacrimation present at 6 weeks of age
Infancy	• 3–4 months: Fully fixates and follows object • Sucking often stimulates infant to open eyes and focus attention on surroundings • 3–5 months: Color discrimination present • 6 months: Eye color generally established • Infants may have visible sclera above and below cornea • Intermittent convergent strabismus common until 4 months of age
Early childhood	• 2 years of age: Binocular vision and depth perception are developed in healthy eye • Visual acuity should be 20/40 to 20/50 • >3 years of age: Visual acuity should be 20/30 • Accommodation and convergence are smooth and well established
Middle childhood	• Refractive error is common beginning at 9 years of age • Visual concerns about near vision in school-age child may be related to learning differences
Adolescence	• Hormonal changes during early and middle adolescence often cause change in visual acuity

Anatomy and physiology

External eye

The *bony orbit* is the structure surrounding the eye in the cranium. Only one-third of the eyeball, or optic *globe,* in the infant and child is exposed to the examiner, and the cranium protects the remainder of the globe in the orbital cavity. The *optic foramen* is the opening in the cranium that allows the passage of the optic nerve, ophthalmic artery, and ophthalmic vein to pass from the globe to the brain and visual cortex. The upper eyelid is shaped by connective tissue containing *tarsal plates.* The *meibomian glands* are located in the tarsal plates near the hair follicles of the eyelashes in the upper and lower lids. In infants and children with atopic or allergic reactions, the meibomian glands, one of the oil-producing sebaceous glands in the body, exude a whitish or yellowish sebaceous material onto the base of the eyelids.

The *lacrimal gland* is located in the lateral aspect of the frontal bone in the orbital cavity (Fig. 12.2). It is a peanut-sized gland similar to the salivary glands. In each eyelid, the *lacrimal duct* opens onto the eyelid margin, and the *nasolacrimal duct* opens into the *lacrimal sac,* which is buried in the frontal process of the maxillary bone. The *lacrimal puncta* are noted at the edge of the upper and lower eyelids at the *medial* or *inner canthus* and allow the drainage of tears into the *nasolacrimal duct* and the *lacrimal caruncle.* The *lacrimal caruncle* is the elevated area of tissue bordering the upper and lower medial canthus and assists in drainage of tears. Tear production is normally present by 6 weeks of age in the term infant. The eyelashes add further protection to the surface of the eye and assist in lubricating the exterior surface of the eye.

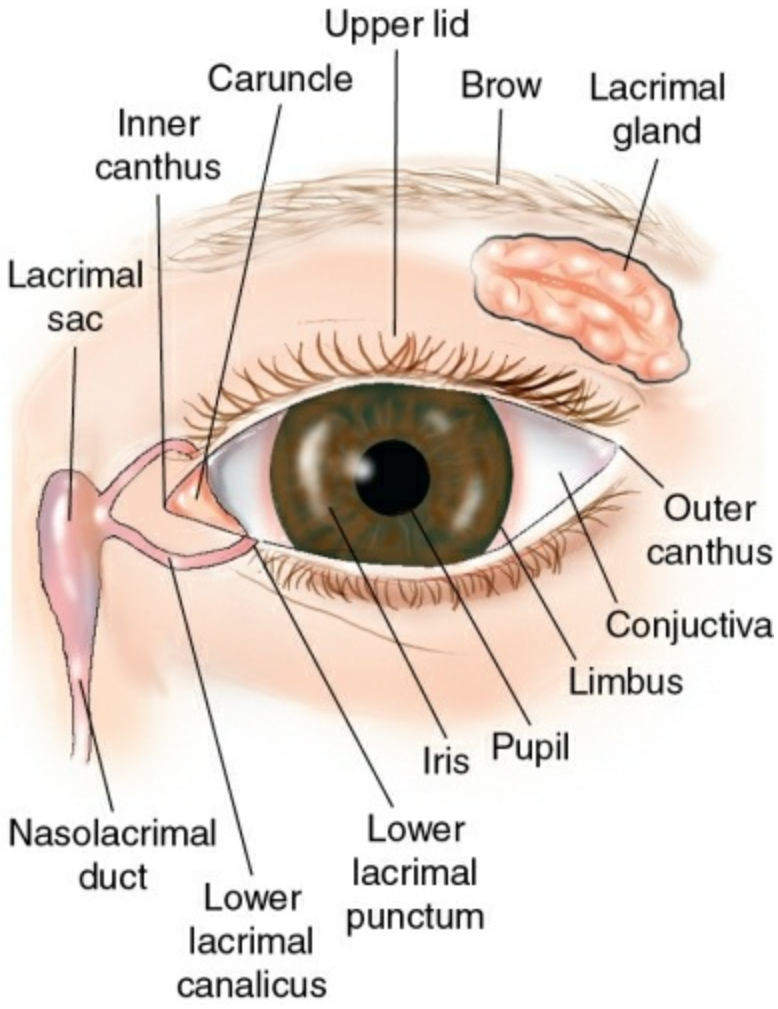

FIGURE 12.2 External eye and lacrimal apparatus.

The *sclera,* the outermost layer of the exterior structure of the globe or eyeball, is the firm collagenous layer that protects the intraocular structures (see Fig. 12.1). The *conjunctiva* is formed by thin mucous membrane lining the anterior surface of the sclera and inner eyelids and acts along with the tear film as a protective covering for the cornea (see Fig. 12.2). The conjunctiva has two surfaces: the palpebral and bulbar. The *palpebral conjunctiva* lines the inner eyelids, is vascular, and is covered by papillae. The *bulbar conjunctiva* is clear and contains no papillae and very few blood vessels. The *bulbar conjunctiva* covers the sclera up to the *limbus,* the juncture of the sclera and the cornea.

The *cornea,* the most anterior aspect of the external eye, is a lenslike structure that acts as a refractory surface for the eye and connects with the sclera at the limbus. The cornea is transparent and contains no blood vessels. The transparency allows light to be

focused on the retina. It derives oxygen from the aqueous humor and from tears. The *anterior chamber* is directly behind the cornea, and the *posterior chamber* is the thin area between the lens and the posterior iris. The anterior and posterior chambers contain *aqueous humor,* a clear nutrient fluid that circulates around the lens and the cornea and provides nutrition and oxygen. *Glaucoma* is increased intraocular pressure resulting from abnormalities in the ocular drainage of the aqueous humor, which damages the optic nerve. *Congenital glaucoma* may result from the abnormal development of the ocular drainage system and may cause clouding or enlargement of the cornea. It may be present at birth or develop during the first year of life and may result in blindness if untreated.

The *iris* is the pigmented structure containing the sphincter and dilator muscles, connective tissue, and pigmented epithelium. An absence of color in the iris may indicate *albinism.* The center of the iris is controlled by the *ciliary body* and *iris muscles* and forms the aperture, or pupil. The *pupil* constricts or dilates depending on the amount of light entering the eye. The ciliary body produces aqueous humor and controls accommodation. The iris lies behind the anterior chamber and in front of the *crystalline lens* and is protected by a thin clear capsule attached by small filaments to the ciliary body. A *cataract* is an opacity that occurs in the crystalline lens and obscures vision.

The upper eyelid is elevated by the *levator muscle,* which inserts into the tarsal plate in the upper eyelid and is innervated by cranial nerve III.

Extraocular muscles and internal eye

The six *ocular muscles,* inserted into the scleral surface, control the movement of the eye (Fig. 12.3). The four *rectus* muscles—the *superior, inferior, medial,* and *lateral recti*—originate at the top of the globe deep in the cranium and extend from the anterior to the posterior globe and insert at the back of the globe. The medial rectus muscle is responsible for the movement of the eye toward the midline, and the lateral rectus muscle innervated by cranial nerve VI moves the eye away from the midline. The inferior and superior rectus muscles move the eye up and down and also have overlapping functions with the oblique muscles. The two *oblique*

muscles—the *superior* and *inferior oblique*—insert at the anterior and posterior globe. The superior oblique tendon passes through the *trochlea,* the small, cartilaginous pulley on the frontal bone. The *superior oblique* muscles are responsible for movement of the eye downward and inward, and the *inferior oblique* moves the eye upward and inward. The superior oblique muscle is also innervated by cranial nerve IV, and paralysis of the cranial nerve IV causes a head tilt or torticollis in children to compensate for the weakened muscle.

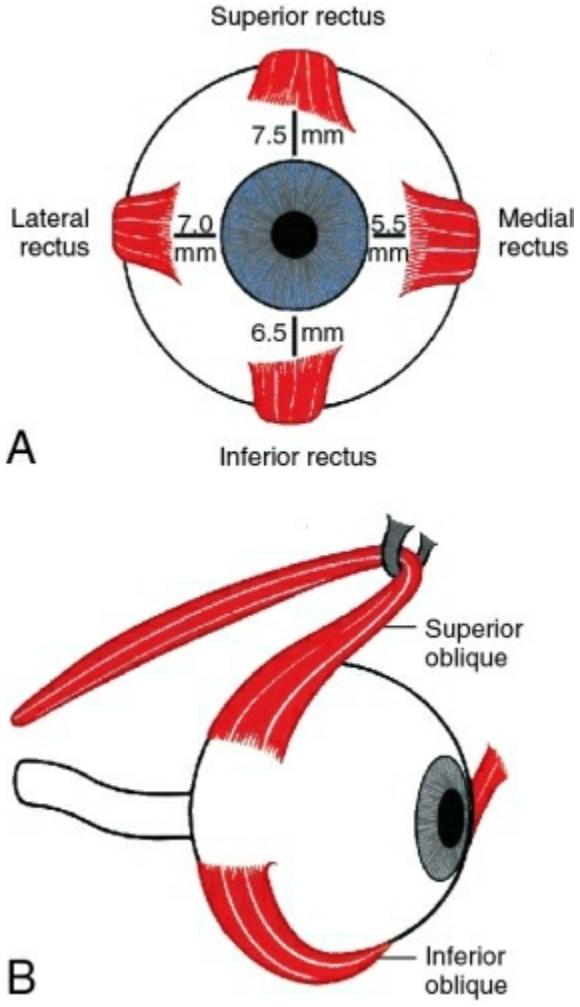

FIGURE 12.3 **(A)** Four extraocular recti muscles. **(B)** Oblique extraocular muscles. Source: (From Palay DA, Krachmer JH: *Primary Care Ophthalmology,* ed 2, St. Louis, 2005, Mosby.)

The *vitreous body* is the large interior cavity of the globe and

contains a clear gel or *vitreous humor.* The *choroid* is the interior layer of the eye between the sclera and the retina and is continuous with the iris and ciliary body. It is highly vascular and nourishes the receptor cells of the retinal epithelium. The outer layer of the *retina* contains photoreceptors, the rods and cones, which are stimulated by light focused by the lens and translate light energy or impulses into neuronal activity. The neurons activate the nerve fiber layer of the retinal epithelium and synaptic activity and transmit through the optic nerve to the brain, which perceives the visual image.

The retinal vessels and optic nerve fibers enter and exit through the *optic cup* and divide into two branches on the surface of the optic disc. The *optic disc,* the anterior aspect of the optic nerve, is pink to orange-red or pale to dark with a yellow cup at its center (Fig. 12.4). There are no photoreceptors in the optic disc, which creates a blind spot of 5 degrees in the visual field.[4] The *macula* lies medial to the optic nerve on the *fundus,* the posterior surface of the retina. The *fovea* or *fovea centralis* is a central depression in the macula without vessels and has darker pigmentation than the retina. This is the area where vision is most perfect, and the ciliary body works to accommodate the lens to focus an image on the fovea. The *arteries* on the fundus appear thinner and more orange-red than the *veins,* which are larger and darker (see Fig. 12.4). The normal arterial-to-venous ratio (A:V) is approximately 2:3 in the healthy individual. Vascular changes present in the retina that often reflect abnormal conditions in the systemic vasculature such as diabetes and hypertension. *Papilledema,* bilateral optic disc edema, is also associated with increased intracranial pressure. *Retinal hemorrhage* is associated with acute traumatic brain injury and inflicted traumatic brain injury. *Retinal hemorrhages* associated with *inflicted traumatic brain injury* in children involve deeper layers of the retina and are found more on the periphery of the retina (Fig. 12.5).[5]

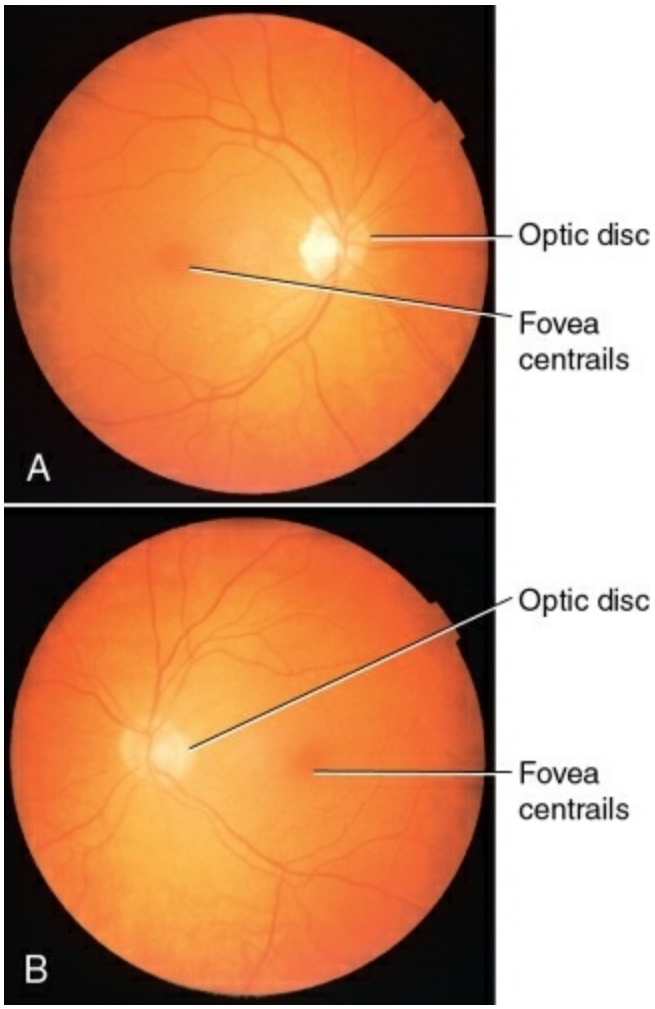

FIGURE 12.4 Normal fundus **(A)** Right. **(B)** Left. Source: (From Lemmi, FO, Lemmi, CAE: *Physical Assessment Findings CD-ROM*, Philadelphia, 2009, Saunders.)

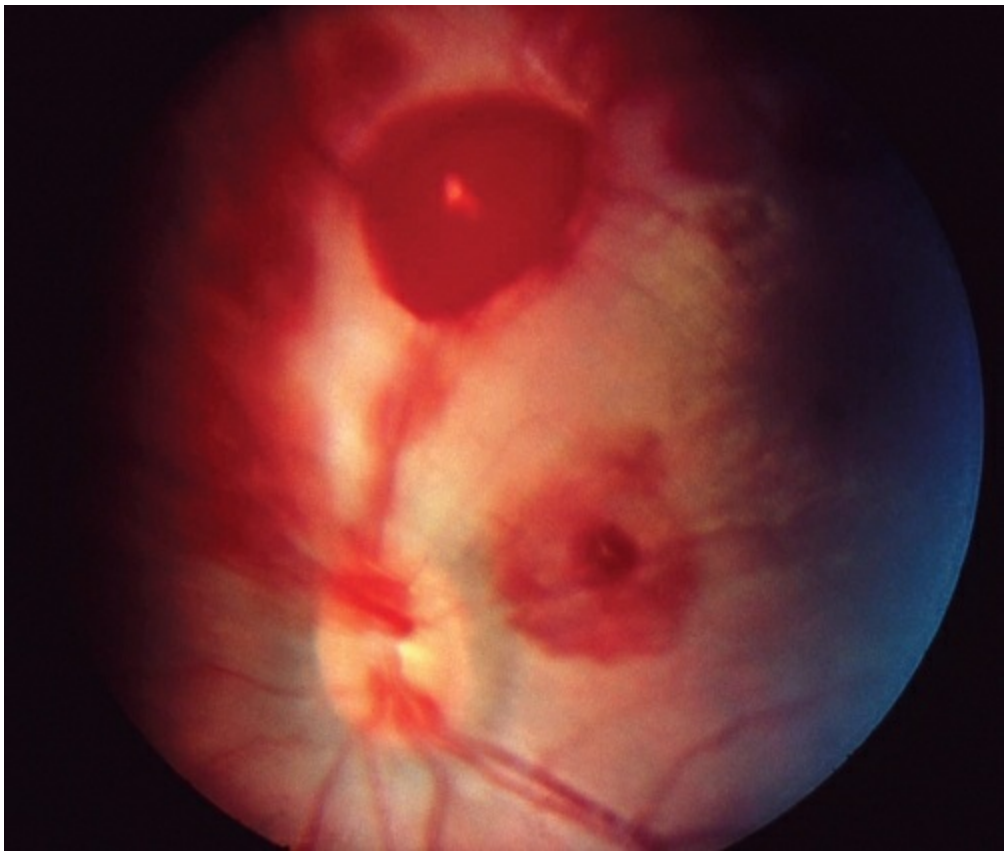

FIGURE 12.5 Extensive retinal hemorrhages in a shaken infant. Source: (From Ruttum MS: Managing the child with an ocular or orbital injury. In Albert DM, Miller JW, editors: *Albert & Jakobiec's Principles and Practice of Ophthalmology*, ed 3, London, 2008, Saunders.)

Family, cultural, racial, and ethnic considerations

Thick epicanthal folds are seen more commonly in infants and young children of Asian and Latino descent. They partially or completely cover the inner canthus at birth and diminish by middle childhood. Asian populations have a genetic predisposition to myopia. In one study, 68% of Asian children were found to be myopic.[3] The pediatric health care provider should focus particular attention to early vision screening in this population of children.

Ethnicity is a risk factor for severe ROP. Asian and African American infants have a higher risk of developing threshold ROP as compared with white infants.[5]

Retinal pigment or melanin is often found on the scleral surface of Latino and African-American children. The pigmented areas

usually become evident in early or middle childhood and persist into adulthood. This is within the range of normal variations of the eye and does not impact visual health. The *retinal light reflex* or "red" light reflex varies in color in darkly pigmented individuals. The *fundus* appears pale yellow or beige because of the increased melanin in the skin, and the optic disc is also often a pale yellow. *Scleral melanocytosis* is a common pediatric condition that characteristically features black or gray-blue pigmentation in the scleral tissue and becomes more prominent with increasing age. It is a benign condition common in Latino, African American, and Asian children and is usually bilateral (Fig. 12.6).

FIGURE 12.6 Scleral melanocytosis. Source: (From Kaiser PK, Friedman NJ, Pineda R: *The Massachusetts Eye and Ear Infirmary Illustrated Manual of Ophthalmology*, ed 4, London, 2014, Saunders.)

System-specific history

Detecting vision problems early is critical to the healthy development of the visual system in infants and young children and to timely referral and intervention. Obtaining a complete history is key to early identification of visual problems or visual changes in infants, children, and adolescents. The Information Gathering table reviews the pertinent areas of information gathering for each pediatric age group and developmental stage of childhood.

Information Gathering for Eye Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant	History of oxygen exposure in early neonatal period? History of retinopathy of prematurity? History of intraventricular insult? History of maternal substance or alcohol abuse?
Newborn	Significant neonatal or maternal infections? Does infant focus on face of parent when alert? Any eye discharge or swelling? Family history of congenital cataracts or glaucoma? History of maternal substance or alcohol abuse?
Infancy	When did infant begin visually following parent? Does infant blink/react to bright light? Any *rapid* involuntary movement of eyes? Persistent discharge or tearing on one or both sides? Any parental concern about visual development? Significant jaundice in neonatal period? History of maternal infection? Neonatal meningitis? History of abusive head trauma?
Early childhood	Does child sit close to TV? Amount of screen time? Able to see birds/plane in sky? Any clumsiness/bumping into objects? Holds books or cell phone close to face? Abnormal head positioning? Appropriate response to visual cues? Frequent eye rubbing? History of frequent stye or hordeolum? Repeated blinking? Difficulty with color recognition? Family history of color vision deficit? History of abusive head trauma?
Middle childhood	History of visual problems? Headache? Family history of myopia, strabismus? Is the child squinting? Does child have corrective lenses or refuse to wear them? Date of most recent eye exam? Where is child seated in classroom? Any difficulty reading? Does child learn at grade level? Protective eyewear for sports? Eye pain or strain? Amount of screen time?
Adolescence	History of eye trauma? Any difficulty with eyestrain when studying? Headache? Wears corrective lenses or refuses to wear them? Contact lens wearer? History of corneal abrasion? Date of most recent eye exam? Protective eyewear for sports? History of concussion or head trauma? Driver's license? Restricted license? Screen time?

Environmental risks	Exposure to eye irritants? Contact with chemical cleaning agents, hazardous chemicals, or smoke?

Physical assessment

Equipment

The traditional ophthalmoscope performs all necessary screening in the pediatric population. The PanOptic ophthalmoscope, if available, provides a larger view of the internal eye structures in children (Fig. 12.7). A penlight may be used for screening the corneal light reflex and testing the pupillary reflex. The Snellen E, Tumbling E, Lea symbols, Allen vision cards, color vision, and near vision testing cards may be used in the clinical setting for vision acuity testing for the young child through young adulthood. An eye cover will assist in proper vision testing. Children under 4 years of age can also benefit from instrument-based pediatric vision testing. Testing of cranial nerves is presented in Chapter 20.

FIGURE 12.7 PanOptic ophthalmoscope. Source: (Copyright Welch Allyn. Used with permission.)

Inspection of the external eye

Begin the inspection of the eyes with a general inspection of the periorbital region, globe position, and lid margins. Note any asymmetry in the eyelids or eyebrows and whether the eyelashes are normally distributed. Note the size and shape of the periorbital cavity. The *palpebral fissure* or opening should be on a horizontal plane between the medial and lateral canthus in infants, and slants upward laterally in infants and children of Asian descent or children with Down syndrome. The upper eyelids should appear symmetrical. Inspect the *conjunctiva* for erythema or irritation, and the lid margins for erythema, crusting, lash distribution, and cysts or lesions. The eyelashes should be carefully examined for nits or lice. Observe for *entropion* (eyelashes curl inward) or *ectropion*

(lower eyelid appears droopy and eyelashes curl under).

Conditions such as *ptosis,* an eyelid that droops or has an absent or faint lid crease, may be a normal familial variant or the result of a brachial plexus injury during a difficult delivery. *Ptosis* extending partially over the pupil and causing the infant to tilt the head in an effort to see constitutes an impact on the visual field and requires prompt referral to a pediatric ophthalmologist (Fig. 12.8). When closed, the upper eyelid should meet the lower eyelid and completely cover the cornea and sclera.

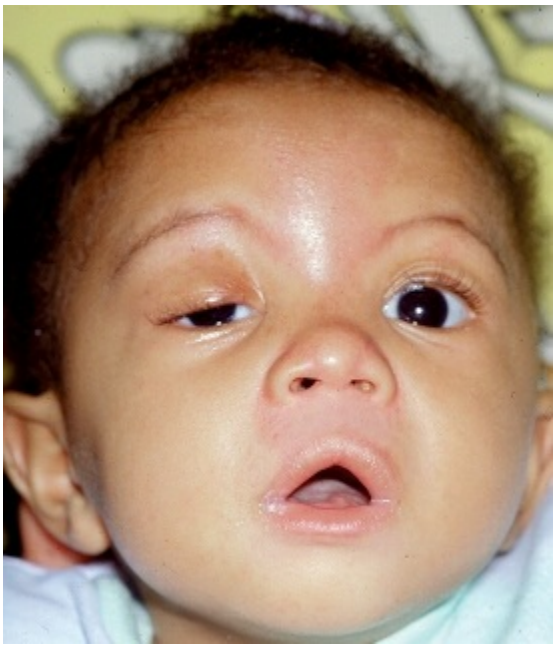

FIGURE 12.8 Congenital ptosis. Source: (From Palay DA, Krachmer JH: *Primary Care Ophthalmology,* ed 2, St. Louis, 2005, Mosby.)

Inspection of the upper eyelid

Inspection of the inside of the upper eyelid may be necessary when a child or adolescent presents with conjunctival irritation, infection, trauma, foreign body, or possible injury or abrasion of the cornea. To evert the upper eyelid for examination in the cooperative child, give the child a toy or bright object and ask the child to look down at the object. Grasp the upper eyelashes at the base and *gently* pull out and up while pushing in and down with a cotton applicator on

the upper *tarsal plate* (Fig. 12.9). Gently remove the cotton applicator and hold the eyelid while inspecting the *adnexa*. The palpebral conjunctiva should have a pink and glossy appearance and the adnexa should be clear. To return the lid to a normal position, have the child look up as the lid is released. This procedure is well tolerated by middle childhood and in cooperative school age children. In early childhood, administration of a topical anesthetic, such as proparacaine, may be necessary, or referral to an ophthalmologist may be made if indicated to ensure a thorough assessment of the eye. Newborn infants will occasionally have an inverted eyelid, which is within the normal range of variations.

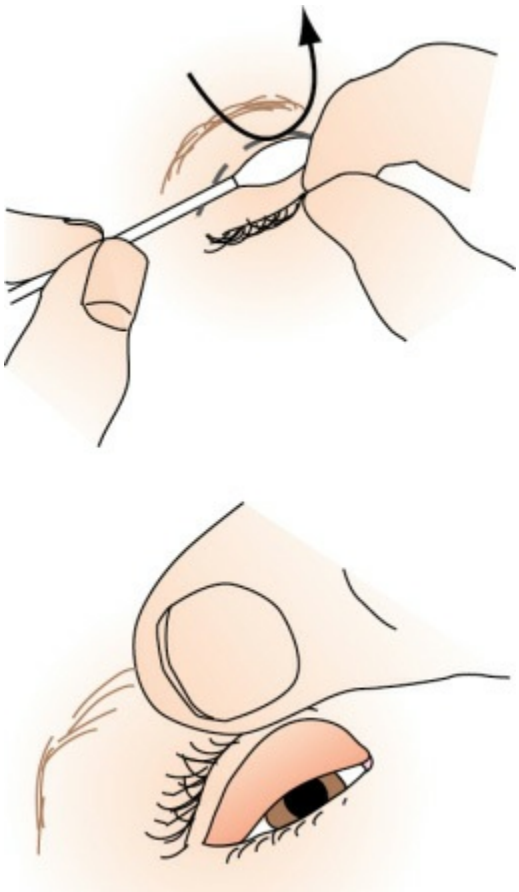

FIGURE 12.9 Examination of the upper eyelid.

Assessment of visual fields and visual alignment

Sight is the most complex of the senses, and any insult to the eye along the visual pathway during development of the sensory organ

can impact vision. The alignment and position of the *pupil* in the visual field and the clarity of the cornea is determined by the *corneal light reflex,* or *Hirschberg test.* It can be performed with a penlight or otoscope light without the speculum (Fig. 12.10). Focus the light source about 12 inches from the infant, and note the reflection of the light from the cornea at the center of the pupil. The light should be symmetrical in the center of the pupil. Any asymmetry of the light reflex could indicate ocular misalignment and could impact visual development.

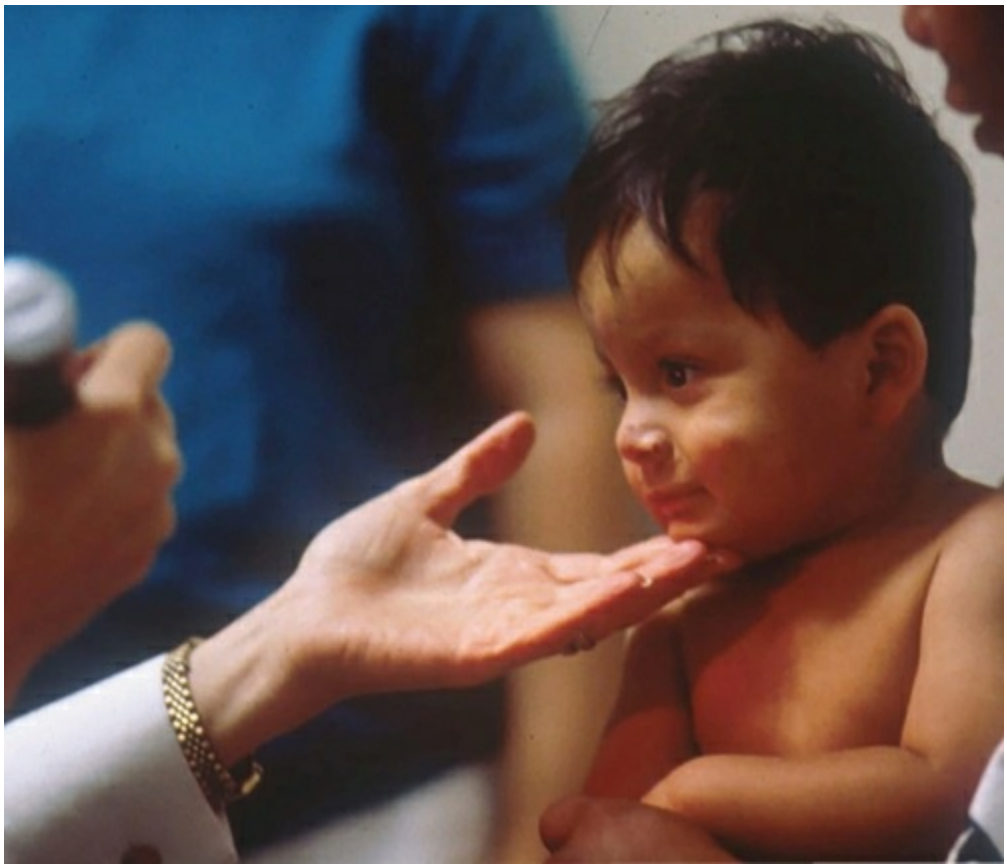

FIGURE 12.10 Testing corneal light reflex.

Next, evaluate the six extraocular muscles (EOMs) by having the child or adolescent follow a penlight or the examiner's finger through the six cardinal fields of gaze in the visual field (Fig. 12.11A). This testing evaluates the normal function of the oculomotor (cranial nerve III), trochlear (cranial nerve IV), and abducens (cranial nerve VI) nerves (see Fig. 12.11B–D). In infancy and early childhood, the examiner should focus the light

approximately 12 inches from the eyes. A cooperative infant should normally be able to follow the light on horizontal and vertical planes by 6 months of age.

FIGURE 12.11 **(A)** Six cardinal fields of gaze with associated cranial nerves. **(B)** Testing lateral rectus, CN VI. **(C)** Testing superior rectus, CN III. **(D)** Testing superior oblique, CN IV. *CN,* Cranial nerve.

The *cover-uncover test* further evaluates ocular alignment and can be performed as early as 6 months of age in an alert infant. The young infant should be assessed on the examination table. The

infant or toddler should be seated in the parent's lap. Begin by having the infant or child fixate on a penlight or light of the otoscope, the light of the large aperture on the ophthalmoscope, or a bright object. If the infant is alert but distracted, it is helpful to dim the lights and use a toy. Keep in mind infants will often reach for the toy but will often focus on a light. Use the nondominant hand or an occluder brought in laterally over the eye while the infant or child is fixating on the light or bright object. Observe the uncovered eye for fixation on the light or object. Remove the hand or occluder, and note any deviations or movement of the covered or uncovered eye from the central gaze. Any deviation or movement of the eye from the central gaze or focus on refixation on the light by either the covered or uncovered eye may indicate abnormal alignment and should be further evaluated (Fig. 12.12). An inward deviation of the eye is referred to as *esotropia* (Fig. 12.13); an outward deviation is referred to as *exotropia.* If a *phoria* is present, the covered eye will deviate and refixate when the hand or occluder is removed. This indicates a focusing abnormality and should be referred. As visual acuity develops, the cover-uncover test should be performed with the child fixating on a distant object or wall poster and should continue to be part of the physical examination until 10 years of age.

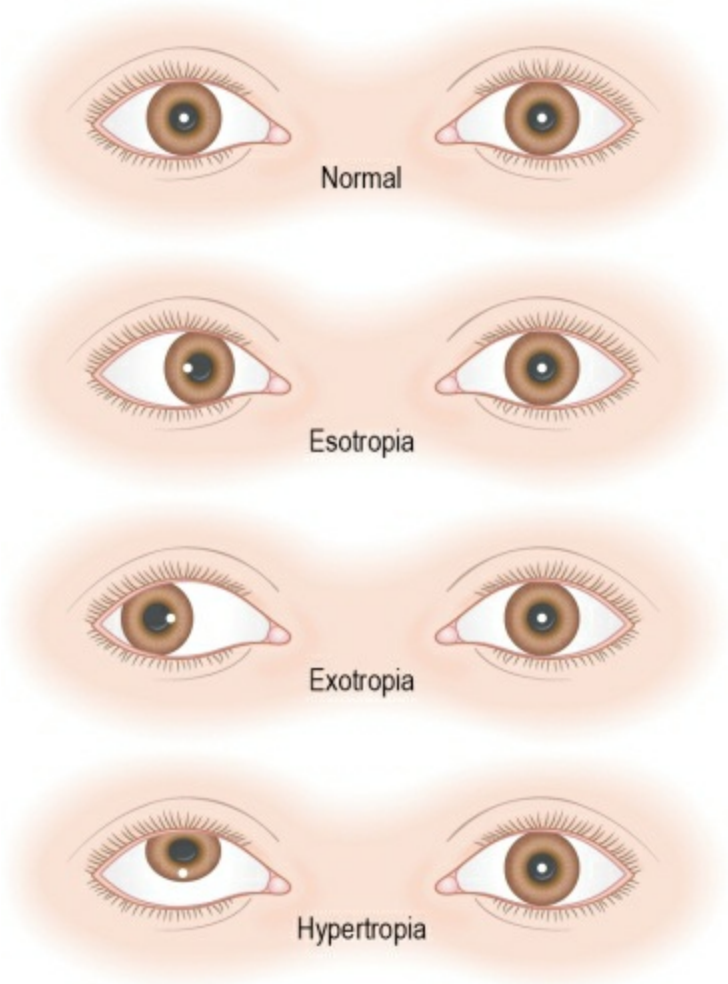

FIGURE 12.12 **(A)** Esotropia: outward movement of right eye. **(B)** Exotropia: inward movement of right eye. **(C)** Hypertrophia: upward deviation of eye. **(D)** Prism reflex test for nonalignment of eyes. Source: (From Levin LA, Albert DM: *Ocular Disease: Mechanisms and Management*, London, 2010, Saunders. Modified from Wright KW: *Pediatric Ophthalmology and Strabismus*, St. Louis, 1995, Mosby.)

FIGURE 12.13 **(A)** Esotropia. **(B)** Shortly after corrective muscle surgery. Source: (From Palay DA, Krachmer JH: *Primary Care Ophthalmology,* ed 2, St. Louis, 2005, Mosby.)

Conditions of visual alignment

Abnormalities in ocular alignment noted on examination may be congenital or acquired and may involve conditions affecting the muscle or nerve. *Congenital esotropia* occurs in the first 6 months and *accommodative esotropia* occurs between 12 months and 7 years of age.

Strabismus is nonalignment of the eyes causing the visual image to fall on the retina at a distance from the fovea. This disrupts binocular vision or visual fusion and keeps the eyes from working simultaneously. The resultant double vision and loss of depth perception from the blurred image impact normal binocular vision and visual development during the critical period of development in the first 5 to 6 years of life. Young children are often unaware of visual changes or deficits. If strabismus goes undiagnosed, amblyopia develops. A common finding in the young infant that prompts over referral to ophthalmology is *pseudoesotropia* or *pseudostrabismus,* a crossed appearance of the eyes caused by the large epicanthal folds covering the sclera (Fig. 12.14). The corneal light reflex or Hirschberg test differentiates pseudostrabismus from strabismus.

FIGURE 12.14 Pseudoesotropia. Source: (From Palay DA, Krachmer JH: *Primary Care Ophthalmology,* ed 2, St. Louis, 2005, Mosby.)

Amblyopia is a monocular loss of vision during the critical period of visual development in the first 6 to 8 years of life. The visual development in one eye is suppressed due to insufficient visual stimulation and the better functioning eye becomes the dominant eye. The common causes of loss of binocular vision during visual development are changes in the visual cortex due to unequal refraction in the eyes, cataracts, ptosis, congenital hemangioma of the eyelid, trauma, and untreated strabismus. The *cover-uncover test* and the *corneal light reflex* are both required in a complete ophthalmologic examination to detect strabismus and amblyopia.

Nystagmus is spontaneous, involuntary movement of one or both eyes and is an indication of poor visual acuity. In the preterm infant, *persistent* or *horizontal nystagmus* can indicate ROP, intracranial hemorrhage, or tumor. In the term infant, *congenital nystagmus* can be associated with Down syndrome, atrophy of the optic nerve, retinal dystrophy, congenital cataracts, abnormalities of the ocular muscles or nerves, vestibular disturbances, and decreased visual acuity. In the older child or adolescent, drug overdose or chemical toxicity is a possible cause. Slight horizontal nystagmus in the lateral fields of gaze is normal. Children with amblyopia exhibit nystagmus because of loss of vision in the affected eye. If nystagmus is noted, a thorough neurologic examination is warranted, as well as an evaluation by a pediatric ophthalmologist.

Assessment of the internal eye

The *retinal light reflex* or *red light reflex* determines the clarity of the posterior chamber of the eye, the receptivity to light, and the

sensitivity of the *retina* to visual stimulus. To elicit the retinal light reflex, the examiner brings the ophthalmoscope to touch the eyebrow and positions it obliquely at a 15- to 25-degree angle lateral to the eye approximately 12 inches from the infant or child. Use the ophthalmoscope on the "0" setting to view the fundus. Dim the lights, and bring the retinal light reflex into view in each eye. Inspect for symmetry and brightness, or brilliance, of the retinal light reflex. A lens that is congenitally dislocated or abruptly dislocated because of trauma appears as a darkened or asymmetrical retinal light reflex. Any asymmetry in size or color or darkness in the uniformity of the retinal light reflex indicates the need for immediate referral to an ophthalmologist.

The *Bruckner test* assesses the retinal light reflex through the ophthalmoscope simultaneously in both pupils from a distance of 2 feet to 3 feet from the infant or child. Compare the pupil and iris in both eyes for color, size, shape, movement, and clarity. To inspect for *opacities,* dim the light and illuminate the cornea by shining the light of the ophthalmoscope obliquely approximately 15 degrees from the lateral canthus. An alert infant with normal visual development should blink at a bright light directed at the eye.

PEDIATRIC PEARLS

To visualize the retinal or red light reflex in the newborn or in early infancy, hold the infant slightly upright from the exam table and, while cradling the head, gently rock the infant. As the head is lowered to the exam table, the eyes will usually open to examine the retinal light reflex.

To test for *visual accommodation* and *pupillary reaction,* shine a bright light momentarily into the eye. As the light approaches the iris, the pupil should begin to dilate. When bringing the light of the large aperture of the ophthalmoscope near the pupil from a distance, the pupils constrict as the light nears.

Conditions of the internal eye

Any serious defect of the cornea, aqueous chamber, lens, and

vitreous chamber can be detected in the infant and young child by assessing the quality of the retinal light reflex. *Leukokoria,* a whitish opacity of the pupil visible in dim light or in room light, is highly abnormal and appears as an absent retinal light reflex or a partially darkened reflex if the opacity does not cover the entire pupil (Fig. 12.15). This finding is usually *unilateral. Congenital cataracts* and *retinoblastoma* are associated with an absent or incomplete retinal light reflex and may have a presenting sign of leukocoria. A *coloboma* of the iris is an irregular or teardrop-shaped iris, which causes a deficit in the visual field and impacts visual acuity. Most colobomas are diagnosed at birth and are associated with chromosomal syndromes. If caused by trauma, colobomas requires immediate referral to a pediatric ophthalmologist (Fig. 12.16).

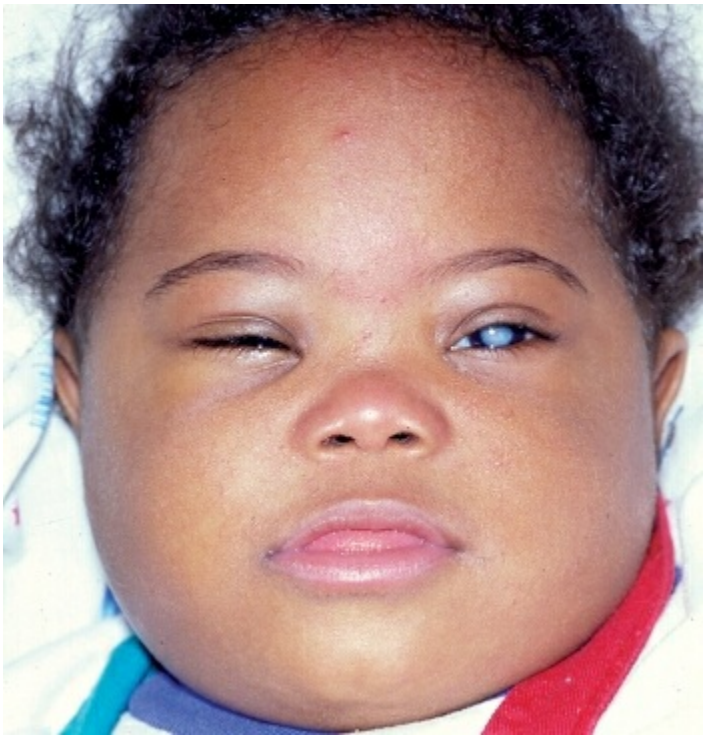

FIGURE 12.15 Leukokoria. Source: (From Palay DA, Krachmer JH: *Primary Care Ophthalmology,* ed 2, St. Louis, 2005, Mosby.)

FIGURE 12.16 Coloboma of left iris. Source: (From Femida K: Congenital and developmental abnormalities of the eye, orbit, and ocular adnexa. In Albert DM, Miller JW, editors: *Albert & Jakobiec's Principles and Practice of Ophthalmology,* ed 3, Philadelphia, 2008, Saunders.)

Visual acuity testing

Visual acuity testing should begin with verbal children at 2 to 3 years of age, with the Allen vision cards (Fig. 12.17 and Table 12.3). Children should be allowed to practice with a parent or caretaker to familiarize themselves with the figures. Then testing should proceed starting at a near distance, testing each eye separately using an occluder held by the parent. The examiner should then show the Allen vision cards while walking backward and continue testing one eye and then both eyes until the distance is 15 feet to 20 feet for the 3- to 5-year-old child. Allen vision cards test to 20/30 or 15/30 depending on the distance from the child and the figure size of the cards.

FIGURE 12.17 Allen vision cards. Source: (From Palay DA, Krachmer JH: *Primary Care Ophthalmology,* ed 2, St. Louis, 2005, Mosby.)

TABLE 12.3
Visual Acuity Testing

Age Group	Examination at All Well Visits	Referral Criteria
Preterm infant	Retinal or red light reflex Penlight exam of cornea Evaluate for nystagmus	Preterm infants ≤30 weeks with birth weight of <1500 g and infants with birth weight between 1500 g and 2000 g or gestational age of >30 weeks with an unstable neonatal course
Newborn	Retinal or red light reflex Penlight exam of cornea Evaluate for nystagmus	Asymmetrical, absent, or white reflexes Cloudiness of cornea Presence of *rapid* involuntary ocular movement
Infancy	Retinal or red light reflex Penlight exam of cornea Evaluate for nystagmus Corneal light reflex Cover/uncover test Fixation to light/follow 90 degrees	Asymmetrical, absent, or white reflexes Cloudiness of cornea Presence of *rapid* involuntary ocular movement Any ocular misalignment or deviation of eye from central axis after 3–4 months of age
Early childhood	Retinal or red light reflex Corneal light reflex Cover/uncover test Visual acuity: Allen vision cards,	Acuity of 20/50 to 20/40 in one or both eyes with accurate vision testing Difference of >20 between right and left eye Abnormal cover/uncover test

	Tumbling E, Lea symbols Funduscopic exam if indicated	
Middle childhood	Retinal or red light reflex Corneal light reflex Cover/uncover test Extraocular muscle testing Visual acuity: Tumbling E, Snellen E Funduscopic exam	Acuity of 20/40 in one or both eyes Difference of >20 between right and left eye Strabismus: Abnormal cover/uncover test Abnormal funduscopic exam
Adolescence	Visual acuity: Snellen E, Tumbling E for low literacy Extraocular muscle testing Funduscopic exam	Visual acuity of 20/40 in one or both eyes Abnormal funduscopic exam

The Snellen *Tumbling E test* begins in the prekindergarten age group and is used until the child knows the alphabet with accuracy. It is also used with children and adolescents with low literacy or learning problems. The examiner may ask the child which way the "legs of the table" are pointing. Using this directional approach may be difficult for children with learning disabilities or attention or behavior problems. The *Snellen E* distance acuity chart can be used when the child achieves literacy. Children of school age may become *myopic* (nearsighted) as the eye matures. Over the past decade, there has been an increased prevalence in *myopia* (nearsightedness) in children and young adults due to an interaction between environmental factors, including more near work or screen time, a decrease in exposure to outdoor light, and genetic predisposition.[6] The prevalence varies according to ethnicity and geographic region.[6]

Testing the eyes separately to detect a difference in refractive error is extremely important in young children. Occluding the eye properly is the key to accurate testing of visual acuity. *Anisometropia*, a difference in refraction between the eyes, can lead

to amblyopia. It is difficult to detect in the young child because the eye initially remains in alignment. Referral is indicated if accurate visual testing yields a 20/40 refraction bilaterally or a greater than 20 difference in refraction between the eyes (e.g., 20/30 in the left eye and 20/70 in the right eye).

Instrument-based pediatric vision screening

Instrument-based pediatric vision screening uses optical images of the retinal or red light reflex to estimate refractive error and ocular alignment, which put children at risk for developing amblyopia. Photoscreening instruments, which assess both eyes simultaneously, and autorefraction instruments, which screen one eye at a time, have been found to be accurate for vision screening in children 6 months to 3 years of age.[7] Portable photoscreening and handheld autorefraction instruments may result in earlier detection of amblyopia if performed and interpreted by trained individuals. They are also useful for screening older children who are developmentally delayed or unable to cooperate with routine acuity screening.[7] The use of vision charts and standard physical examination techniques to assess amblyopia in children 3 to 5 years of age remains standard practice currently.

EVIDENCE-BASED PRACTICE TIP

Humans normally blink approximately 15 times a minute, but using computers and other digital screen devices, they blink half to a third that often. The American Academy of Ophthalmology recommends following the 20-20-20 rule—for every 20 minutes of screen time, users should shift their gaze to an item approximately 20 feet away for at least 20 seconds to reduce eye strain and eye dryness. [8]

Evidence-Based Practice Tip

Vision problems can interfere with the process of reading, but

children with dyslexia or related learning problems have the same visual function and ocular health as children without such conditions. Currently, there is inadequate scientific evidence to support the view that subtle eye or visual problems cause or increase the severity of learning problems. [9]

Color vision testing

Visual testing for color sensitivity should occur at 4 years of age or before school entry. Children should be tested between 4 and 8 years of age for any history of difficulty with color recognition. Difficulty or confusion when identifying colors may be related to cognitive learning differences and should alert parents and teachers. The incidence of *color vision deficit*, previously referred to as *color blindness*, is 8% in white males and 4% in African American males. The incidence in females is from 0.4% to 1%. Testing should be completed with the *Hardy-Rand-Rittler (HRR) test.* The HRR test uses a series of symbols rather than numbers, which allows reliable testing to be done on young children. The *Ishihara test*, which uses a series of figures and letters composed of spots of certain colors, can be used on the older child. The child with a color vision deficit fails to see letters or figures of a certain color.

Funduscopic examination

The ophthalmoscopic exam permits the examiner to clearly visualize the internal structures of the eye in a child who is able to sit for examination and focus steadily on a distant point. This generally occurs by 5 to 6 years of age.

Using the lens selector disc, focus the ophthalmoscope on the palm at close distance before examining the child to determine the clarity of the image and accommodate for any visual deficit in the examiner. The lens indicator may read "0" or ± to produce the clearest image, depending on the visual acuity of the examiner. Resting the hand on the child's forehead just above the eyebrow, begin with the ophthalmoscope positioned laterally approximately 2 inches from the eye to decrease *miosis*, constriction of the pupils (Fig. 12.18). The rubber pad on the face of the ophthalmoscope should be resting on the eyebrow of the examiner. Use a distant

focal point to attract the child's attention and help him or her to fixate and look over the shoulder of the examiner. As the examiner moves medially toward the central field of vision, the vessels of the *fundus* should come into view. Once a vessel in the *retina* is in focus, follow along the vessel; where it "branches" will point toward the *optic nerve.* The *optic disc* should come into view in the medial aspect of the fundus. The macula is examined last to minimize miosis. Forcibly opening the eyes of a child results in a frustrated child and an incomplete examination. If examination is immediately necessary but cooperation is not achieved through verbal preparation or proper positioning of the child, then referral to a pediatric ophthalmologist is indicated.

FIGURE 12.18 Positioning ophthalmoscope for exam.

Eye conditions

Table 12.4 presents the most common and chronic eye conditions seen in infants, children, and adolescents by the pediatric health care practitioner.

TABLE 12.4
Eye Conditions

Condition	Description

Sundowning	Downward deviation of the eyes associated with hydrocephalus, intracranial hemorrhage, other pathologic brain conditions, or early sign of cerebral palsy; a sign of increased intracranial pressure when symptoms of lethargy, poor feeding, vomiting, bulging fontanel, or rapidly increasing head circumference are noted
Exophthalmos	Protrusion of the globe, also known as *exophthalmia* or *proptosis*, may be unilateral (e.g., orbital tumor, orbital cellulitis, or a retrobulbar hemorrhage) or bilateral (Graves disease or *hyperthyroidism*)
Conjunctivitis	Acute inflammation of palpebral and bulbar conjunctiva; etiology includes viral, bacterial, corneal abrasion, allergy, or environment irritation
Blepharitis	An acute or chronic irritation of the eyelid; may be caused by allergic conditions such as seborrhea, bacterial infections (staphylococcal), inflammation of meibomian glands, or parasites
Chalazion	A cyst in the eyelid caused by inflammation of the meibomian gland; differs from a stye or hordeolum because it is usually painless. Also known as *meibomian gland lipogranuloma*
Stye or hordeolum	An infection of the sebaceous glands of Zeis at base of eyelashes; can be external or internal. Internal stye or hordeolum is an infection of the meibomian sebaceous glands lining the inside of the eyelid
Episcleritis	Inflammation of the episclera, layer beneath conjunctiva, causing acute irritation and redness of eyes, occurs in 2%–5% of children with inflammatory bowel disease (IBD), management with topical therapies and management of IBD [10]
Pterygium	Overgrowth of conjunctival tissue extending from the lateral canthus to cornea; begins in childhood with overexposure to sun and constant dust/environmental irritants
Scleral icterus	Yellowish coloration of sclera extending to the cornea; most often first indication of systemic jaundice and liver dysfunction in neonate; often first sign of liver disease in children and adolescents
Lacrimal duct obstruction	Abnormal tearing pattern; upward pressure on lacrimal sac often yields mucoid discharge; massage of nasolacrimal duct with downward pressure on lacrimal sac may open duct to normal drainage by 6 months of age
Dacryocystitis	Inflammation of nasolacrimal sac; swelling and redness occur around lacrimal sac in area of inner canthus
Retinoblastoma	Solid intraocular tumor; presents as abnormal retinal or retinal light reflex in newborn or as white pupillary reflex in infant; can be associated with proptosis, protruding eye bulb
Congenital glaucoma	Symptoms of photophobia (sensitivity to bright light), epiphora (excessive tearing), and blepharospasm (eyelid squeezing), conjunctival injection, causes ocular enlargement and visual impairment

Summary of eye examination

- Begin by noting the symmetry of the eyes and the size and shape of the periorbital cavity.

- Perform the corneal light reflex to determine the clarity of the cornea, as well as the alignment and the position of the pupil in the visual field.
- Evaluate the extraocular muscles in the six cardinal fields of gaze.
- Perform the cover-uncover test for ocular alignment.
- Elicit the retinal light reflex or red light reflex.
- Check for pupillary accommodation.
- Perform age-appropriate visual acuity testing.
- Perform an ophthalmoscopic examination for children and adolescents.

CHARTING

3-year-old child

Eye: Vision with Allen vision cards 20/40 bilaterally. Extraocular movements intact, sclera and conjunctiva clear, corneal reflex intact bilaterally, irides brown, pupils accommodate, symmetrical red or retinal light reflex (RLR).

CHARTING

Well adolescent

Eye: Sclera and conjunctiva clear, extraocular movements normal (nl), irides brown, PERRLA (*P*upils, *E*qual, *R*ound, *R*eact to *L*ight, and *A*ccommodate), funduscopic examination—without opacities, optic disc visualized, pale yellow, disc margins clear (cl). Vessels nl, arteries/veins (A/V) ratio 2:3.

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CHAPTER 13

Ears

Patricia Jackson Allen

The ear is a sensory organ that functions as part of a complex sensory system for hearing and vestibular equilibrium. Visual inspection of the ear is only the first step in determining the normal function of this complex organ. The role of the pediatric health care provider is to maintain function of the ear to preserve hearing and balance in the child and adolescent and to detect any abnormalities early in infancy. The goal of early detection is to promote optimum development of hearing and to support normal development of speech and language.

Embryologic development

External ear

The external ear is a cartilaginous structure on the external surface of the temporal bone (Fig. 13.1). The structures of the ear evolve in the mesoderm, and development of the external ear begins during the sixth week of gestation when the six *hillocks of His* develop from the first and second branchial arches. The individual portions of the *auricle,* or flap of the ear, begin to fuse and assume the classic adult shape by the 12th week of gestation, and fusion is complete by the 20th week (Fig. 13.2). The normal auricle should be no greater than 10 degrees off vertical plane or slope, and the superior portion should be in line with the outer canthus of the eye (Fig. 13.3). Minor malformations of the auricle may be normal variants, such as

preauricular skin tags, a *preauricular sinus,* or *Darwin tubercle*, a slight thickening or nodule at the upper portion of the helix (Fig. 13.4). Malformations can also be the result of intrauterine position, exposure to certain toxins or medications such as isotretinoin (Accutane), intrauterine infection, or other intrauterine complications occurring during this early phase of fetal development. Some genetic syndromes, Down syndrome, first arch syndrome, Treacher Collins syndrome, and Nager syndrome, are associated with ear malformations, such as a small external ear, *anotia*, the total absence of the auricle with narrowing or absence of the external auditory opening, or malpositioned ears often in combination with internal ear abnormalities. Hearing loss also has viral causes (rubella, toxoplasmosis, cytomegalovirus [CMV]), and multiple genetic causes including Usher syndrome, Alport syndrome, and CHARGE (coloboma, heart defects, atresia choanae-also known as choanal atresia, growth retardation, and ear abnormalities) syndrome. Bilirubin toxicity in newborns is associated with high-frequency hearing loss.

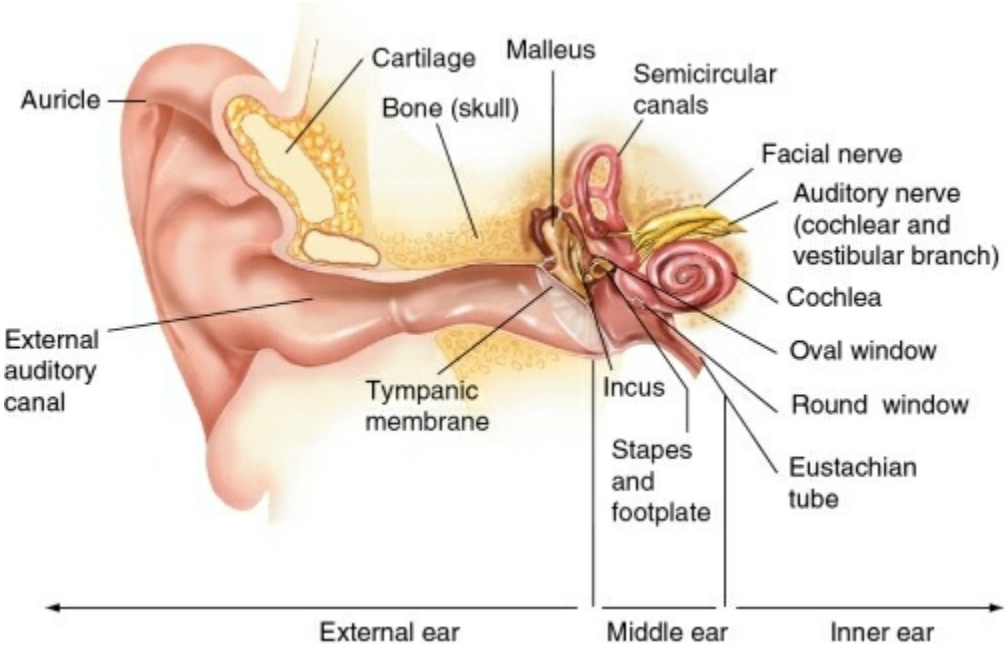

FIGURE 13.1 Anatomy of the middle and inner ear in skull.

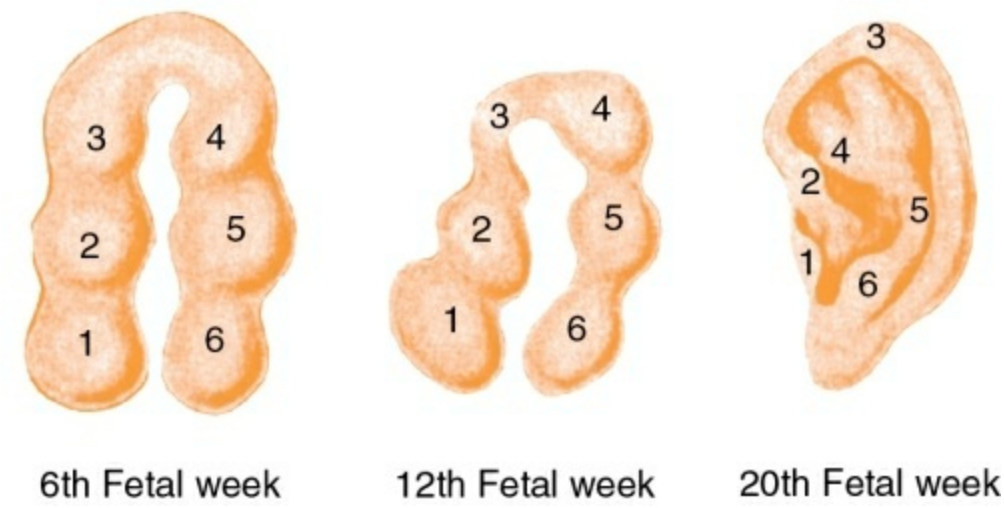

FIGURE 13.2 Auricular development in the fetus. Source: (Adapted from Flint P, Haughey B, Lund V, et al: *Cummings Otolaryngology–Head and Neck Surgery*, ed 5, Philadelphia, 2011, Mosby.)

FIGURE 13.3 The normal alignment of the ear.

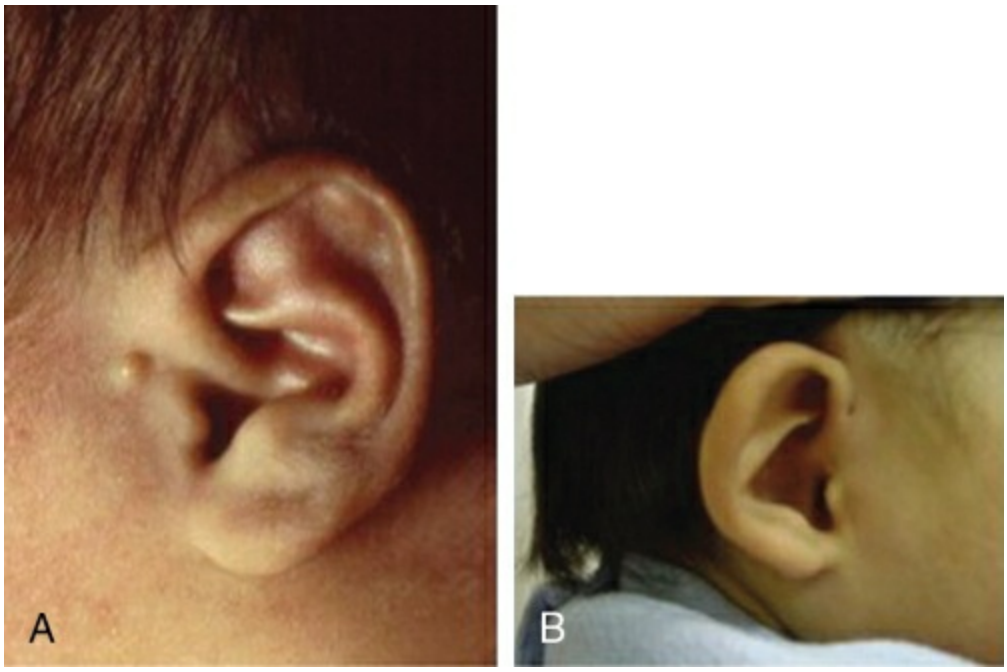

FIGURE 13.4 (A) Preauricular tag. (B) Preauricular sinus. Source: (From Jones KL, Jones MC, del Campo M: *Smith's Recognizable Patterns of Human Malformation*, ed 7, Philadelphia, 2013, Elsevier.)

Inner ear

The inner and middle ear are located in the temporal bone of the skull. Although the external ear formation coincides with the gestational formation of the internal ear structures, they develop separately. The auditory placode and the acousticofacial ganglion are present the fourth week of gestation. Over the next month the first of the three turns in the cochlea develops. Arrest in development during this phase results in a common bony abnormality of the inner ear associated with congenital sensorineural hearing loss (SNHL) known as *Mondini deformity*. The final 2.5 turns of the cochlea occur by the 12th week of gestation. The *organ of Corti* develops from the epithelium of the cochlea and is responsible for transmission of sound impulses to the eighth cranial nerve, the *acoustic nerve.* Improper development of the membranous labyrinth of the organ of Corti results in a *Scheibe deformity*, which is the most common congenital abnormality of the cochlear duct, resulting in SNHL (see later section, Hearing Loss). Alterations in development during this period due to chromosomal

abnormalities or other causes can lead to hearing loss of particular auditory tones. The semicircular canals first appear in the sixth week of gestation with differentiation of canal structures being complete by the 16th week. The sensory cells needed for equilibrium actually attain adult size by the 23rd week of gestation.

Middle ear

Simultaneous with the early development of the inner ear is the formation of the first pharyngeal pouch, Meckel cartilage, in the oropharynx. The proximal portion of the pharyngeal pouch develops into the *eustachian tube,* and the distal portion becomes the tympanic cavity and supporting structures. The eustachian tube is 17 to 18 mm long at birth and lies 10 degrees off the horizontal plane. The development of the tympanic cavity with the three ossicles, the malleus, incus, and stapes, is not complete until the eighth month of gestation, but the malleus and incus reach adult size and shape by the 18th week of gestation and the stapes by the 20th week of gestation.[1] *Meckel cartilage* forms the *incus* and *malleus* of the middle ear, and the second pharyngeal pouch, the *Reichert cartilage*, forms the *stapes*. Failure of these structures or their supporting ligaments to form properly results in a conductive hearing loss[2] (see later section, Hearing Loss). Any malformation or dysfunction of the middle ear structures can result in conductive hearing loss.

Developmental variations

Table 13.1 presents variations in the pediatric age group from the preterm infant to the school-age child.

TABLE 13.1

Developmental Variations of the Ear

Age Group	Physiologic Variations
Preterm	Vulnerable to hearing loss, particularly before 33 weeks, from noise exposure, hypoxia, ototoxic drugs, hyperbilirubinemia, persistent pulmonary hypertension
Newborn	At birth, tympanic membrane is almost adult size but lies in a more horizontal plane compared to the adult ear, which alters visual assessment

	Intrauterine positioning may result in disfiguring of the pinna, which will usually resolve after birth with proper positioning because of the elastic quality of the ear cartilage Whitish material including vernix caseosa may be present in external auditory canal Canal narrow and curved making assessment of the middle ear difficult Determining patency of canal is critical
Infancy	Fluid easily trapped in the middle ear due to eustachian tube dysfunction, particularly common in infants with Down syndrome, preterm infants, and any infant with craniofacial abnormalities
Early childhood	External auditory canal ossifies by 2 years of age, straightening the canal and improving visualization of tympanic membrane The pinna is approximately 80% of the adult size in the 4- to 5-year-old
Middle childhood	In a 9-year-old, the pinna and external auditory canal have attained adult size The canal measures 2.5 cm and has become somewhat S shaped

Anatomy and physiology

External ear (pinna)

The external ear, the *pinna,* is divided into sections: the outer portion is called the *helix,* just medial and parallel to the helix is the *antihelix,* and the *concha* is the cavity leading to the *external auditory meatus* or opening of the external canal (Fig. 13.5). A firm protuberance on the anterior portion of the ear just at the entrance to the auditory canal is the *tragus,* and across from the tragus on the border of the antihelix is the *antitragus.* Beneath the tragus is the soft fold of skin that forms the ear lobe. Although the shape of the auricle varies slightly from person to person, the ears should be comparable bilaterally in size, shape, position, and appearance.

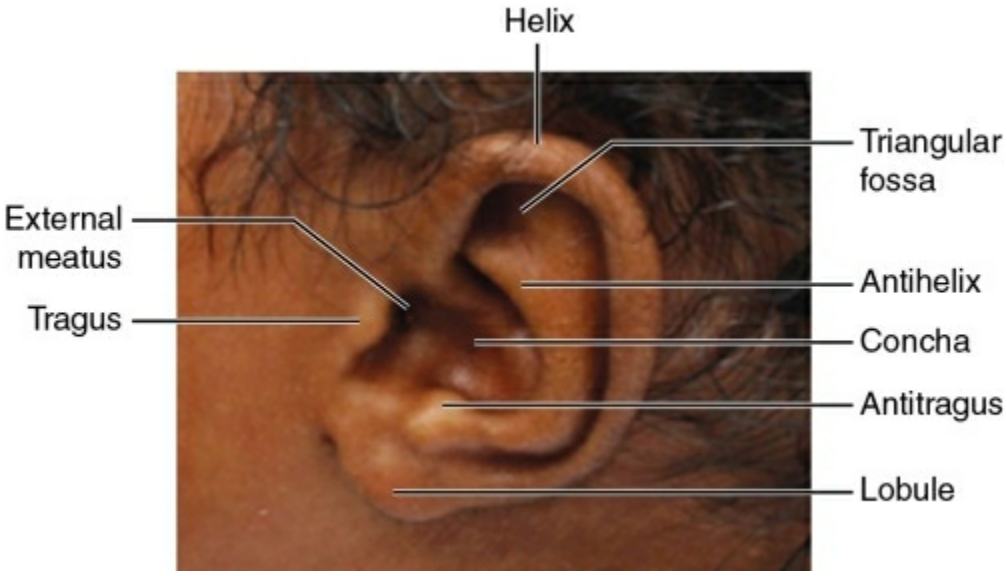

FIGURE 13.5 Anatomy of the external ear of small child labeled with anatomy.

The external auditory canal, an S-shaped canal measuring approximately 2.5 cm in length by middle school-age, connects the outer ear to the middle ear and funnels sound waves to the tympanic membrane. The auricular muscles are innervated by the seventh cranial nerve, or *facial nerve.* The medial portion of the canal is innervated by the fifth cranial nerve, or *trigeminal nerve,* and the posterior canal is innervated by the 10th cranial nerve, or *vagus nerve*. The distal third of the ear canal contains hair follicles, ceruminous glands, and sebaceous glands. The *cilia* (specialized hair) are extremely sensitive to tactile stimulation and assist in sensing sound. The *ceruminous* gland, a modified apocrine sweat gland, secretes a milky substance that forms cerumen when exposed to the secretions of the sebaceous glands and air. *Cerumen* lubricates the skin of the canal, acts as a barrier to foreign objects entering the interior canal, and has protective antibacterial properties to reduce the incidence of skin infection in the external canal. Natural lateral movement of skin and hair cells in the external canal facilitates drainage of cerumen and other debris from the external ear canal.

Middle ear

The tympanic membrane is a thin, oval-shaped layer of skin attached to the wall of the external canal and is approximately 9 to

10 mm in diameter (Fig. 13.6). It is concave being pulled in by its attachment to the malleus and sits at an oblique angle in the external canal. It is surrounded by a fibrous band called the *annulus,* which becomes sclerotic later in life, diminishing movement of the tympanic membrane and often resulting in conductive hearing loss. The tympanic membrane is attached to the malleus along the manubrium conducting sound waves to the incus, stapes, and oval window of the inner ear. The tympanic membrane has a resonance frequency of 800 to 1600 Hz, approximating the normal speech frequency of 500 to 2000 Hz found in humans. The tympanic membrane is divided into sections: (1) the *pars flaccida* is superior to the lateral process of the malleus, (2) the *pars tensa* comprises the majority of the tympanic membrane inferior to the lateral process of the malleus, and (3) *Prussak space,* which lies medial to the pars flaccida in the anterior superior quadrant of the tympanic membrane (see Fig. 13.6). *Prussak space* is the most common location of retraction pockets and congenital or acquired *cholesteatoma* (Fig. 13.7), an initially asymptomatic white mass in the middle ear that is thought to arise from repeated infections or pulling inward of the eardrum due to eustachian tube dysfunction. Over time, this mass can increase in size and destroy the delicate bones of the middle ear leading to conductive hearing loss.[1] Tympanosclerosis, thickening and scarring of the tympanic membrane, is commonly seen after chronic infections of the middle ear or traumatic injury or surgery to the tympanic membrane (Fig. 13.8).

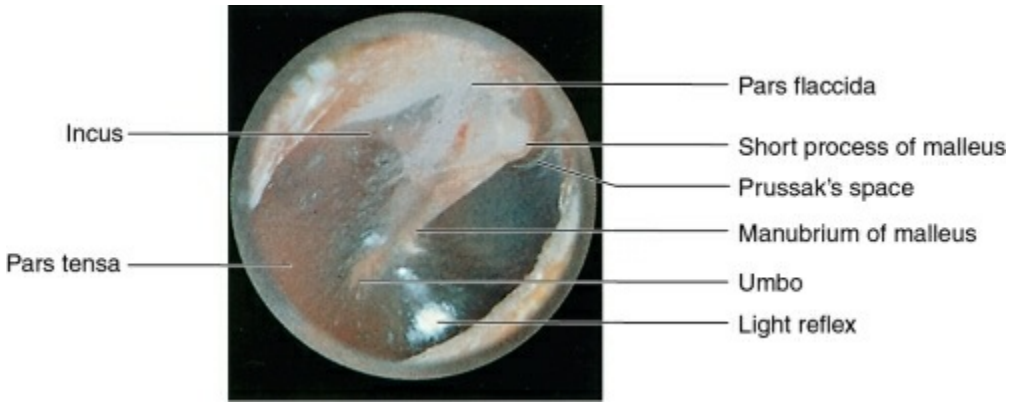

FIGURE 13.6 Normal left tympanic membrane. Source: (From Seidel HM, Ball JW, Dains JE, et al: *Mosby's Guide to Physical Examination*, ed 7, St. Louis, 2011, Mosby.)

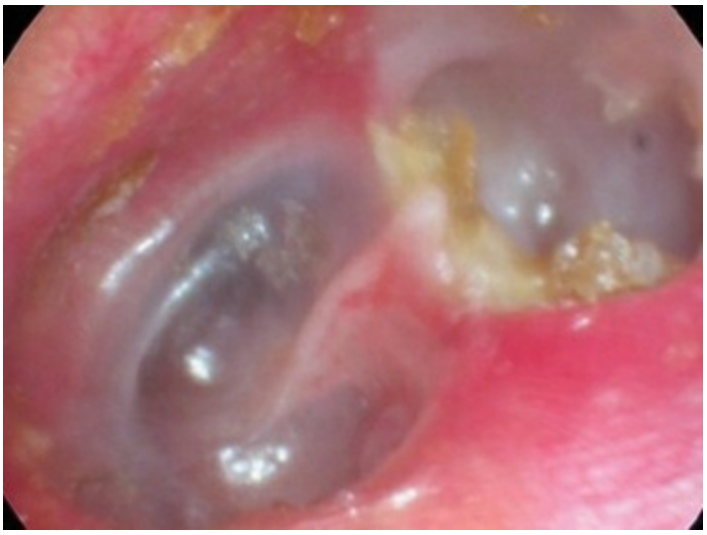

FIGURE 13.7 Cholesteatoma. Source: (*Reproduced from Nevoux J, Lenoir M, Roger G, Denoyelle F, Duccou LePointe H, Garabedian E.-N. Child-hood cholesteatoma. European Annals of Otorhinolaryngology, Head and Neck Diseases. Sept 2010; 127(4): 143-150. Copyright © 2010 Elsevier Masson SAS. All rights reserved.*)

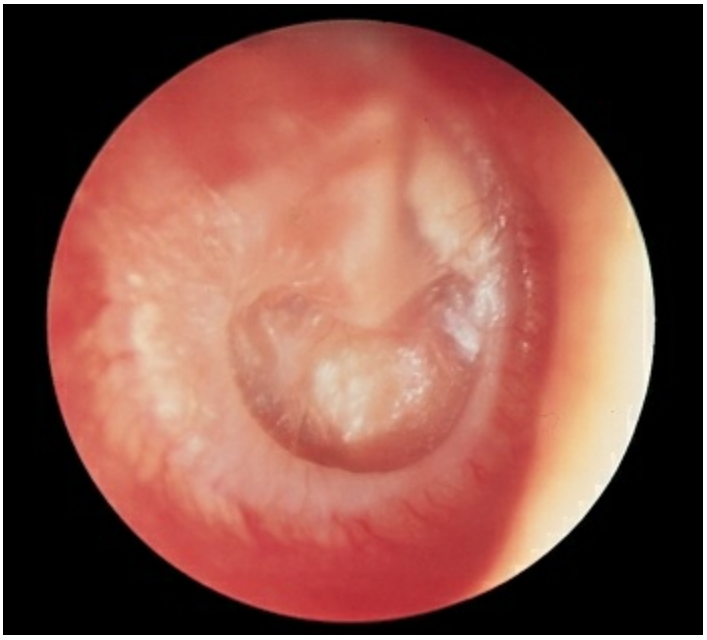

FIGURE 13.8 Scarring on the tympanic membrane. Source: (From Zitelli BJ, Davis H: *Atlas of Pediatric Physical Diagnosis*, ed 5, St. Louis, 2008, Mosby.)

The three *ossicles* of the inner ear, the smallest bones in the body, transmit the movement of the tympanic membrane to the oval window and subsequently to the vestibular and cochlear branches of the eighth cranial nerve, the *acoustic nerve* (Fig. 13.9). The head of the *malleus* articulates with the body of the *incus* at the incudomalleolar joint. The long crus or leglike structure of the incus, articulates with the head of the *stapes* at the incudostapedial joint. These joint areas are the most vascular regions of the *ossicles* and, therefore, are the most susceptible to trauma or infection. The footplate of the stapes sits upon the oval window of the inner ear at the fibrous stapediovestibular joint. Because of the mechanical function of the three ossicles and the transmission of sound waves from the larger surface area of the tympanic membrane to the smaller surface area of the oval window, there is a net increase of 22 times the sound energy radiating from the tympanic membrane to the oval window.

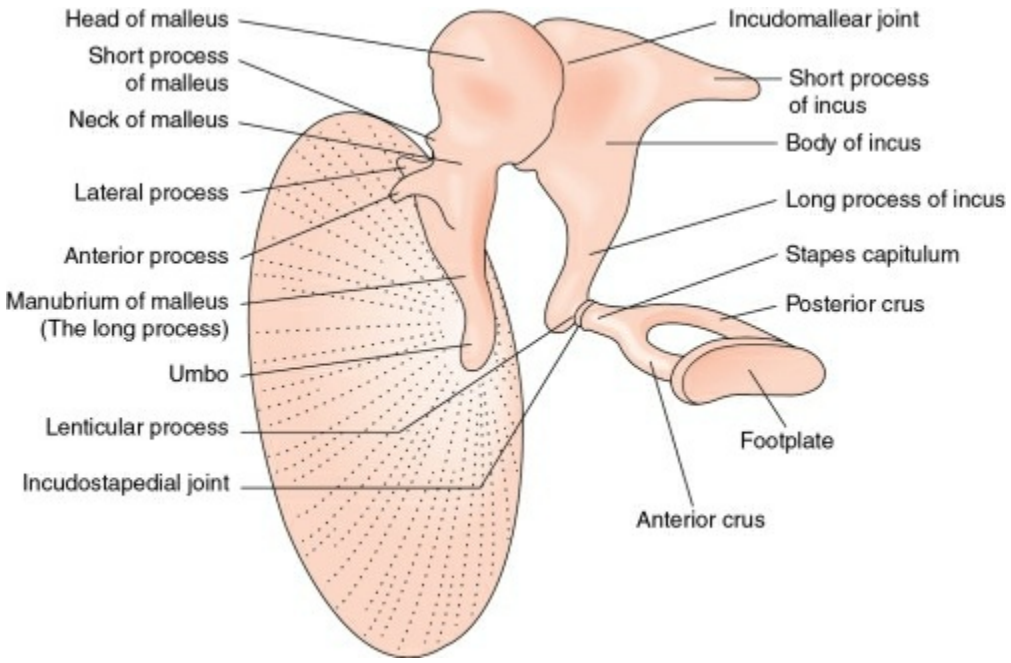

FIGURE 13.9 Anatomy of the inner ear.

The eustachian tube opens into the oropharynx just behind the nasal cavity and is the drainage and ventilatory structure for the middle ear. The eustachian tube connects the middle ear with the back of the throat. The middle ear is an air-filled space and when

patent has the same air pressure as the outside air pressure. Swallowing, yawning, and sneezing all open the eustachian tube and restore and equalize the pressure between the middle ear and the outside air pressure. Changes in altitude or ambient pressure, such as occurs on an airplane, can result in decreased air pressure in the middle ear, resulting in pain or discomfort if the eustachian is swollen or blocked. Repeated swallowing can often open the eustachian tube, equalizing pressure and reducing the associated pain. The infant who sucks on the bottle or breast can also reduce this discomfort in flight. In the adult, the eustachian tube averages 35 mm in length. In infancy, it is half that length (approximately 18 mm) and lies in a more horizontal plane than in the adult, which allows bacteria and viruses to more easily migrate from the oropharynx to the middle ear (Fig. 13.10). The musculature of the eustachian tube, which controls function, is innervated by the motor division of the fifth cranial nerve, or trigeminal nerve. In infants and children with cleft palate and other central craniofacial abnormalities such as Down syndrome, structural or functional abnormalities of the eustachian tube interfere with normal ventilation and clearance of the middle ear, increasing the incidence of otitis media (OM) in these children. Equalization of pressure in the middle ear is critical for normal sound wave vibration of the tympanic membrane.

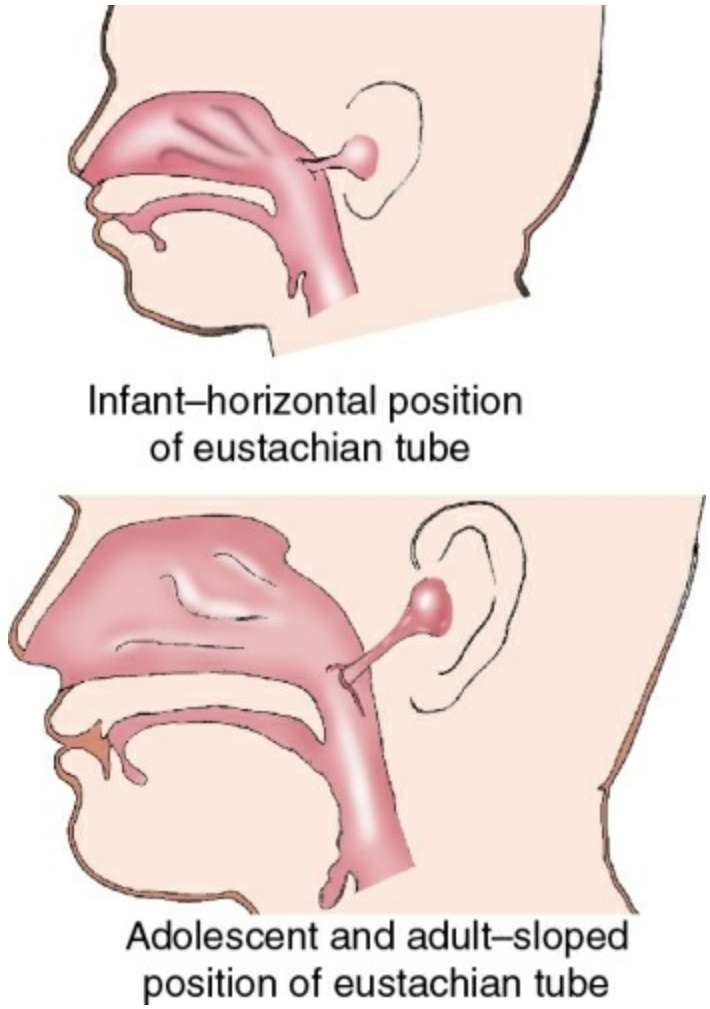

FIGURE 13.10 Position of eustachian tube in infant and in adult.

Muscle maturation, elongation of the eustachian tube, and a more vertical position all contribute to the decreased incidence of OM and middle ear effusions in middle childhood, adolescence, and young adulthood.

Inner ear

The inner ear is the sensory end organ and is directly responsible for hearing and balance. The inner ear contains the *vestibule, semicircular canals,* and *cochlea* and is bathed in fluid that facilitates the transmission of sound waves to the auditory nerve and sensations of balance in the *semicircular canals* (see Fig. 13.1). The sound waves pass over approximately 30,000 innervated hair cells in the cochlea, which are the primary receptors, transducers, and conveyers of sound energy to the brain.

Physiologic variations

In recent years, the understanding of genetics and the auditory system has increased significantly. Autosomal recessive genetic SNHL accounts for approximately 70% to 80% of all genetic forms of hearing loss in children, and autosomal dominant genetic SNHL accounts for 10% to 15%.[1] There are more than 400 known causes of genetic hearing loss. The most common form of recessive hearing loss is due to one gene, connexin 26 (abbreviated CX26). Approximately one-third of children with hearing loss have syndromic hearing loss with other clinical findings, such as Usher syndrome, Pendred syndrome, or Jervell and Lange-Nielsen syndrome. Infants with malformed external ear structures require close monitoring. Infants identified with nonsyndromic hearing loss (hearing loss without additional clinical findings) may be missed and are harder to diagnose. Many genetically determined causes of hearing loss do not present at birth and may not be identified through newborn screening. Radiographic imaging such as a computed tomography (CT) scan or magnetic resonance imaging (MRI) can identify up to 40% of middle and inner ear abnormalities, but imaging is used infrequently because of the concern of long-term risks of radiation exposure for infants and young children. Genetic testing is now often the first investigative tool to identify the cause of hearing loss.[2]

Acquired causes of hearing loss are numerous, and risk factors must be identified (Box 13.1). Prenatal risk factors include gestational diabetes in the mother, congenital infections (CMV, rubella, toxoplasmosis, herpes, syphilis, varicella), exposure to teratogens (alcohol, methyl mercury, thalidomide, cocaine), and ototoxic medications (aminoglycosides, loop diuretics, quinine). Prematurity, birth hypoxia, hyperbilirubinemia, sepsis, and administration of ototoxic medications are risk factors for acquired hearing loss in the perinatal period. Head trauma, infections (mumps, measles, varicella, meningitis, Lyme disease), recurrent OM, and excessive noise exposure are risk factors for hearing loss any time they occur.[2]

Environmental exposure to noise is a major source of hearing loss, and children and adolescents should be screened for exposure to loud sounds in their home, school, or recreational environments.

Children living in industrial areas with heavy traffic or near loud machinery are more prone to hearing loss in the decibel range associated with the environmental exposure.[2]

Family, cultural, racial, and ethnic considerations

Risk factors for OM across ethnic groups include children living in low-income households, bottle feeding verses breast feeding, unimmunized or immunizations not up–to-date and exposure to secondhand smoke.[3, 4] There is research indicating an increased incidence of OM in Native American/Alaska Native children.[3] There is a predisposition or genetic inheritance pattern for both OM and SNHL; therefore family history should be carefully evaluated when gathering a comprehensive health history, and children at risk should have more frequent screening for both OM and hearing deficits. Cerumen appears to vary in appearance and consistency by ethnic group. Dry, white, and flaky cerumen is more often found in Asians and Native Americans, and brown, wet, and oily cerumen is more predominant in whites and African Americans.

System-specific history

The Information Gathering table presents the important information to be gathered for each age group and developmental stage. Table 13.2 presents the important information for a symptom-focused history for children or adolescents presenting with ear symptoms.

Information Gathering for Ear Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant	History of maternal infection? Maternal drug use or maternal diabetes? Antibiotic treatment with aminoglycosides, other ototoxic antibiotic use, or salicylates? Is there a family history of hearing deficit or loss, congenital or acquired?
Newborn	Newborn hearing screening results? ABO incompatibility? Elevated bilirubin level >20 mg/100 dL of serum? Premature infant? History of

	anoxia, pulmonary hypertension, ECMO therapy, or meningitis? Any craniofacial abnormalities noted? Family history of hearing deficit, congenital or acquired?
Infancy	Does infant react to sound with startle response or change in activity? Turn head or body toward sound? Does the infant make cooing or babbling noises? Is the infant breastfed, up-to-date on immunizations? Does infant have frequent colds? History of recurrent ear infections or ruptured tympanic membrane? Ear drainage? Parental concerns regarding infant hearing or verbalizing? Does infant turn head towards parent when name is called? Are there any concerns regarding the child's motor development?
Early childhood	Do you have any concerns about child's ability to hear or speak? How many words does child use? Does child combine words into meaningful sentences? Does the child stutter while trying to speak? How clear is child's pronunciation? How many languages are spoken at home or by care providers? Does child play with his/her ears? Has he/she ever put small objects in his ears or nose? Has child ever had a hearing test done? Were the results normal? History of ear infection, ear pain, or ear drainage? Was it treated with antibiotics? Does child have frequent colds or respiratory allergies? Does the child attend daycare? If in daycare or preschool, do care providers have any concerns about child's hearing, speech, or balance? Is the child up-to-date with all recommended immunizations? Has the child had any serious infections, head trauma or concussion? Does the child use headphones or ear buds for activities and how is the volume monitored? Is the child exposed to loud environmental noises at home, school, in the community? Is the child walking, running, climbing as well as his/her peers? Does the child fall or appear clumsy more than other children?
Middle childhood	Does child have frequent colds or respiratory allergies? Has child had any drainage from ears? Frequent ear pain? Do you or child's teachers have any concerns about child's hearing, speech, gross motor, reasoning or learning ability? Does child have difficulty following directions in school? Has child been exposed to repetitive environmental loud noises? Does child use headphones or ear buds to listen to music, play video games? Is the volume loud? Has child ever complained of ringing in ears, dizziness? Does child spend a lot of time in the water, play water sports? Is there a history of concussion, injury/trauma to head, ears, or mouth? What type of sports does the child play and what safety precautions are used? Is the child exposed to firearm sounds? Wear protective earplugs? Is the child clumsy, have poor coordination or balance, fall frequently, or complain of dizziness? History of meningitis? History of cancer therapy?
Adolescence	History of frequent colds, nasal allergies, or ear infections or ear pain? Does adolescent use headphones/earbuds to listen to music? Is the volume audible to others? Has adolescent had frequent exposure to unusually loud music or noises?

	Has adolescent ever complained of ringing in ears, dizziness? Does adolescent spend a lot of time in the water, playing water sports? Any recreational activities potentially affecting ear (e.g., swimming, scuba diving, flying, boxing, hunting) or work activities (construction work, machinery use)? Has the adolescent complained of clumsiness, poor coordination, dizziness, poor balance, or frequent falls?
Environmental risks	Crowded living conditions? Exposure to secondhand smoke? Exposure to loud noises?

ECMO, Extracorporeal membrane oxygenation.

TABLE 13.2
Symptom-Focused History for Ear Assessment

Symptom	Questions to Ask
Ear pain	Onset, duration, and intensity of pain? Location? Unilateral or bilateral? Associated symptoms (e.g., fever, rhinorrhea, cough, drainage from eyes, ear drainage, hearing loss, vertigo, ringing in ears, swelling or redness around ear, mouth sores, dental pain, sore throat, difficulty sucking or swallowing, vomiting, neck swelling, tenderness)? Concurrent illness (e.g., upper respiratory infection, mouth infection, skin infection, conjunctivitis)? Home management of pain (e.g., medications/home remedies/complementary and alternative therapies): type, how much, how often, how effective? Changes in activities of daily living (e.g., loss of sleep, change in appetite, ability to attend daycare, school, or work)? Changes in activity level, balance, dizziness/vertigo, talking, or movement of temporomandibular joint? Change in interaction with others (e.g., playful, withdrawn, irritable)? What makes the pain feel better, worse? Others at home, daycare, school, or work with similar symptoms? What do you think might be the cause of the pain? In infancy: is infant pulling at ear, showing increased irritability, feeding poorly, or waking more frequently at night?
Ear drainage	Onset, duration, and intensity of discharge? Associated symptoms (e.g., fever, rhinorrhea, cough, ear pain, hearing loss, vertigo, ringing in ears, swelling or redness around ear, vomiting)? Concurrent illness (e.g., upper respiratory infection, mouth infection, skin infection, conjunctivitis)? Changes in activities of daily living (e.g., loss of sleep, change in appetite, ability to attend daycare, school, or work)? Changes in activity level, balance, dizziness/vertigo, interaction with others (e.g., playful, withdrawn)? Home management of drainage (e.g., medications/home remedies/complementary and alternative therapies): type, how much, how often, how effective? Injury caused by pressure or trauma (e.g., laceration or barotrauma)? Any foreign body in ear canal? Others at home, daycare, school, or work with similar symptoms? How do you care for/clean your child's ears?

	What do you think might be the cause of the ear drainage?
Hearing difficulty relevant in school-age child and adolescent	Gradual or sudden onset? Progressive? Bilateral or unilateral? Associated with other symptoms (e.g., ear pain, sense of fullness, drainage, balance problems or dizziness, systemic symptoms of illness)? Concurrent illness (e.g., otitis media, otitis media with effusion, respiratory allergies, developmental delay)? Trauma or exposure to loud noises? Changes in activities of daily living (e.g., difficulty hearing in school, at home, watching television, talking on phone. Difficulty walking)? Home management of hearing difficulty (e.g., sitting closer to television or in front of classroom, increasing visual cues for communicating)? What conditions make hearing better or worse? What do you think might be the cause of the hearing difficulty?
Dizziness or vertigo relevant in school-age child and adolescent	Gradual or sudden onset? When does it occur? With activity? Position change? Associated with other symptoms (e.g., nausea, vomiting, tinnitus, ear pain, ear drainage, hearing loss, systemic symptoms of illness)? Concurrent illness (e.g., viral illness, gastroenteritis, respiratory allergies/illness)? Use of medications or recreational drugs? Changes in activities of daily living (e.g., ability to attend school and work)? Any sports activities? Home management of dizziness? Others in home with similar symptoms? What makes dizziness better or worse? What do you think might be the cause of the dizziness?

Physical assessment

Equipment

Equipment for examining the ear includes an otoscope with halogen light and speculum, pneumatic bulb attachment, and gloves if any apparent skin infection or ear drainage is present. MacroView otoscopes (see Fig. 13.14B) offer improved images of the ear canal and tympanic membrane, and computer-aided and cell phone software is also available for advanced viewing and education in the clinical setting.

Positioning

Proper positioning of the infant and young child will ensure the least discomfort during the examination, prevent injury to the canal or tympanic membrane during examination, and ensure the health care provider has sufficient opportunity to visualize the canal and tympanic membrane. Letting the young child become familiar with

the otoscope by touching the light of the otoscope on the finger or hand often decreases the anxiety of the ear exam (Fig. 13.11A). The young infant is best positioned lying on the examination table with head securely held on either side by the examiner and the arms restrained by a comforting parent. Older infants who are able to sit securely and young children are best positioned in the parent's lap with the arm and head secured by the parent or examiner (see Fig. 13.11B). Distraction may work for some older infants and young children while examining the ears. The curve of the pediatric ear canal can be lessened by pulling the auricle inferiorly and posteriorly (down and back) in the infant and young child, as compared with superiorly and posteriorly (up and back) in middle childhood and adolescence. Be sure not to hold the auricle too firmly, causing pain when attempting to straighten the ear canal. Another examination technique that is very useful in the pediatric patient is for the examiner to position the hand above the ear, supporting the ear with the forefingers, and pulling the tragus forward or anteriorly with the thumb or forefinger (see Fig. 13.11C). This position effectively opens the external canal in young children to improve visualization of the tympanic membrane and causes less discomfort. The handle of the otoscope can be held horizontally or vertically with the examiners hand resting on the child's head when examining children to help stabilize the head and prevent movement of the otoscope during the examination (Fig. 13.12).

FIGURE 13.11 **(A)** Preparing the young child for the ear exam. **(B)** Positioning of the toddler for ear exam. **(C)** Positioning of tragus forward with hand above the ear.

FIGURE 13.12 Holding the otoscope handle in the upright position. Source: (From Wilson S, Giddens J: *Health Assessment for Nursing Practice*, ed 3, St. Louis, 2005, Mosby.)

PEDIATRIC PEARLS

The technique of pulling the tragus forward straightens the auditory canal for ease in examination and causes less discomfort than pulling on the pinna in young children.

If the child is complaining of ear pain in one ear, examine that ear last.

Do not attempt to examine the tympanic membrane unless the child can be securely positioned and held still for the examination.

External ear

Inspection

Before examining the ear, inspect the head, face, and neck for any asymmetry or indication of craniofacial abnormality, defect, or infection. The superior portion of the auricle should be equal in

height to the outer canthus of the eye and vertical with no more than a 10-degree tilt. An ear that is set lower than an imaginary horizontal line drawn from the outer canthus of the eye or tilted greater than 10 degrees may indicate chromosomal abnormality or congenital abnormalities in other body systems or structures (see Fig. 13.3).

Inspect the auricles for size, shape, deformity, placement, discharge, and color. The size and shape of the ears should be similarly symmetrical and may have familial characteristics. In the newborn the cartilage should have instant recoil, but in the premature infant the cartilage may appear flattened and have less prominent incurvings of the helix or concha. Grossly misshapen external ears are often associated with anomalies of the middle and inner ear structures and with SNHL. The placement and angle or tilt of the external ear is discussed earlier. There should be no discharge from the external ear canal, although cerumen may be evident near the opening to the canal. Serous or purulent drainage may indicate a ruptured tympanic membrane, the presence of patent myringotomy tubes, inflammatory response to a foreign object in the ear, or a cholesteatoma. A white cheesy drainage may indicate an infection in the external auditory canal. The color of the auricle should be similar to the facial skin. Redness may indicate inflammation or trauma, and bruising is of particular concern as an indication of trauma possibly associated with head injury or nonaccidental trauma in cases of child abuse.

The common normal variations of the auricle include *auricular* or *preauricular sinus, preauricular skin tags,* and *Darwin tubercle* (see Fig. 13.4). Occasionally an infection can occur in the preauricular sinus, resulting in inflammation, redness, or discharge from the sinus. Ear piercings should be examined for signs of infection, excessive scar tissue, or trauma.

Palpation

Palpate the auricle for any masses or areas of tenderness. Scar tissue may be palpable around ear piercings but is generally nontender. Sebaceous cysts may occur around the auricle or in the external canal and are often mildly inflamed and tender. If movement of the auricle results in pain, the examiner should suspect *otitis externa,* or

other inflammation of the auditory canal. A foul-smelling cheesy discharge is commonly found with otitis externa and often is caused by the bacterium *Pseudomonas*. The mastoid process, posterior to the auricle, should be assessed for swelling, redness, or pain on palpation. *Mastoiditis* is an uncommon finding in immunized children, but a serious complication of OM in the developing world. If undiagnosed and untreated, it can lead to meningitis and hearing loss.

External canal

Inspection

Inspect the external auditory canal for patency, color, discharge, odor, and foreign bodies. The largest speculum that will fit comfortably into the external canal should be used to increase the field of vision. The smallest ear speculum (2.5 mm) is often used for the infant and young child. During the initial newborn examination, patency or atresia of the external auditory canal must be determined. If the canal is not patent or is abnormally narrow or curved, additional abnormalities of the auditory system should be suspected and referral to otolaryngology and genetics for further evaluation should be made immediately. Children with Down syndrome have external canals that are narrower than normal, so the tympanic membrane may be difficult to visualize in early infancy. Vernix caseosa, a whitish cheesy debris, can often be seen in newborn ear canals and can obstruct visualization of the tympanic membrane. It also can be a contributing factor in failed newborn hearing screening or *evoked otoacoustic emission* (OAE) testing in the newborn. Because of the normally curved S shape of the canal, visualization is improved with minimal discomfort if the tragus is pulled forward to visualize the auditory canal and the tympanic membrane. The tympanic membrane of the newborn infant is thicker, grayer, and less translucent than in older children and lies on a more horizontal plane, making visualization more difficult.

Internal ear

Inspection

Inspect the tympanic membrane for contour (normally concave), intactness (no perforations, tympanostomy or myringotomy tubes), color (normally gray or silver but may be pink or red after crying), translucency (normally translucent without scarring or opacity), and presence of visible landmarks (umbo, handle of malleus, and light reflex; see Fig. 13.6). The light reflex is usually found between the 4 and 6 o'clock positions on the right tympanic membrane and 6 to 8 o'clock on the left tympanic membrane. The examiner should also look for the appearance of fluid bubbles behind the tympanic membrane or a fluid line, indicating the eustachian tube is not properly draining the middle ear (Fig. 13.13).

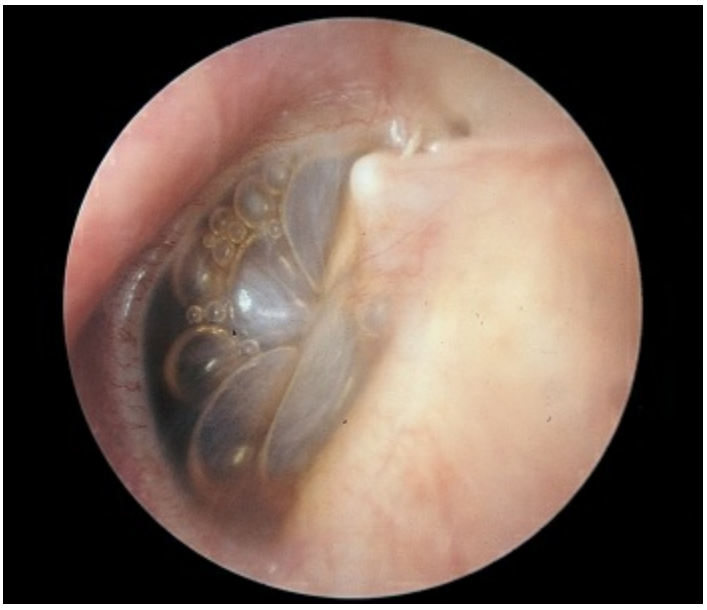

FIGURE 13.13 Middle ear with eustachian tube dysfunction and fluid bubbles. Source: (From Zitelli BJ, Davis H: *Atlas of Pediatric* Physical Diagnosis, ed 5, St. Louis, 2008, Mosby.)

PEDIATRIC PEARLS

Color of the tympanic membrane is less important in diagnosing middle ear infections than identifying bony landmarks and the movement and quality of the tympanic membrane. A red or pink tympanic membrane may occur as a result of crying, irritation, or

fever and may not be an indication of an acute otitis media.

Mobility of the tympanic membrane, an important indication of middle ear pressure, can be assessed with a pneumatic attachment to the otoscope (Fig. 13.14) or by use of a tympanometer. If the middle ear pressure is equalized, the tympanic membrane will move or flutter in response to air pressure from the *pneumatic insufflator* in the external canal. This can be visualized through the otoscope as movement of the light reflex or recorded on the tympanometer as an equal rise and fall of pressure over the normal pressure setting of zero (Fig. 13.15). Decreased or limited movement indicates either *increased negative pressure* in the middle ear, which is associated with *eustachian tube dysfunction* and *otitis media with effusion* (OME) with the tympanic membrane being retracted and taut and the bony landmarks accentuated, or decreased movement due to fluid buildup behind the membrane causing the membrane to become inflamed, convex in shape, and taut. This causes an opacity in the tympanic membrane with loss of visible bony landmarks indicating infection as in *acute otitis media* (AOM) (Fig. 13.16). A ruptured tympanic membrane, patent tympanostomy tubes, or cholesteatoma can result in discharge from the middle ear into the external canal. Ear drainage should alert the examiner to these conditions, as well as otitis externa.

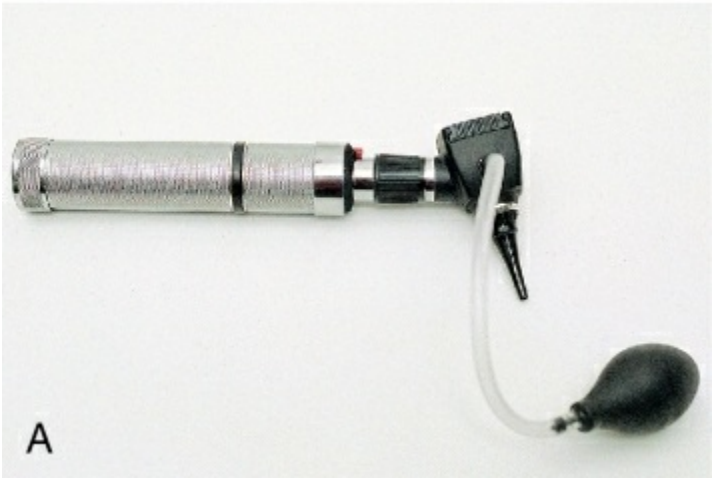

FIGURE 13.14 **(A)** Insufflator or pneumatic attachment to otoscope. **(B)** Advanced MacroView otoscope. Source: (From Wilson S, Giddens J: *Health Assessment for Nursing Practice*, ed 5, St. Louis, 2013, Mosby.)

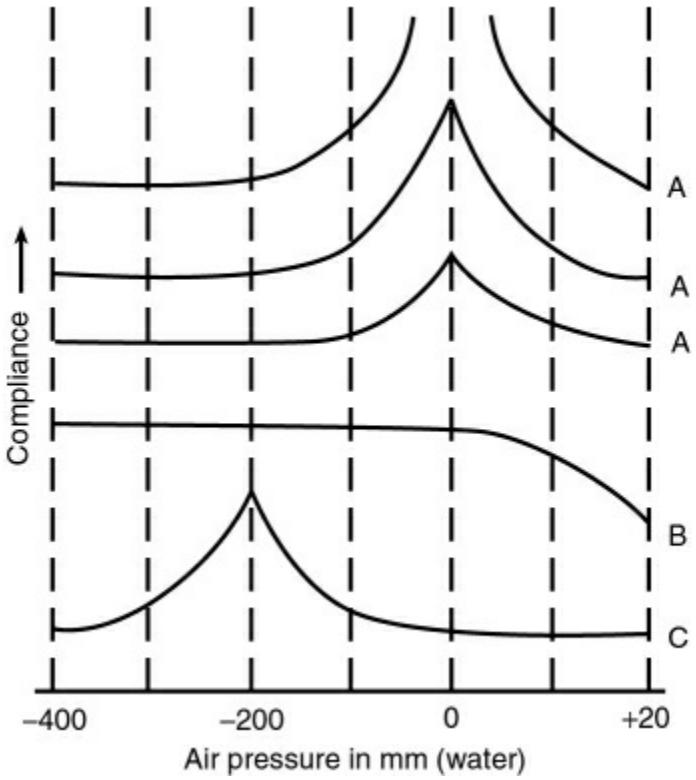

FIGURE 13.15 Tympanometry readings. Source: (From Martin R: *Introduction to Audiology,* Boston, 1991, Allyn & Bacon, © by Pearson Education. Reprinted by permission of the publisher.)

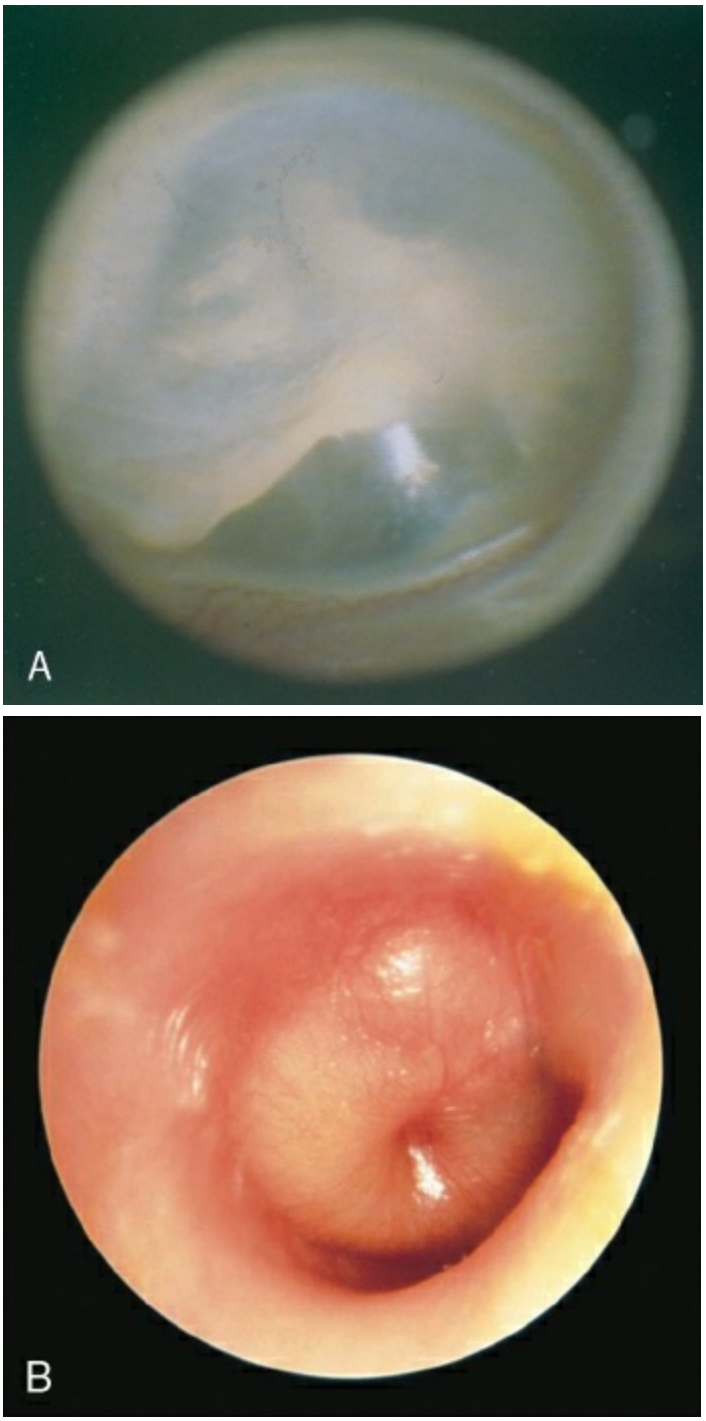

FIGURE 13.16 **(A)** Retracted tympanic membrane (TM). **(B)** TM with otitis media. Source: (From Lemmi FO, Lemmi CA: *Physical Assessment Findings Multi-User CD-ROM*, St. Louis, 2000, Saunders.)

Ear cerumen

Parents and caretakers should always be told cerumen is a normal,

protective ear secretion. Some infants and children naturally have more wax than other children, particularly children with an oily skin type. Children with allergic skin conditions often have additional cerumen, complicating the assessment of ear complaints associated with allergic symptoms or upper respiratory infections. Cerumen has two predominant types: Dry cerumen, which is gray and flaky, is found in 84% of Asians and Native Americans, and wet, honey-colored to dark brown cerumen is found in 97% of whites and 99% of African Americans.[1]

Parents should be encouraged to clean the child's ears only with warm soapy water and should not use cotton-tipped applicators, which can cause injury to the ear canal or tympanic membrane or result in further impaction of cerumen. School-age children and adolescents should also be instructed to not use cotton-tipped applicators. Removal of cerumen or debris from the ear canal may be necessary to visualize the tympanic membrane. If the child is cooperative, a plastic or metal cerumen spoon can be used for removal. Lighted devices for cerumen removal offer increased visualization for the examiner. An infant or young child must be positioned securely before attempting cerumen removal. If the child cannot cooperate for removal of cerumen when necessary, then irrigating the ear canal with warm water will usually loosen and flush out built-up cerumen. Irrigation should never be attempted if a ruptured tympanic membrane is suspected. Tympanostomy or myringotomy tubes inserted into the pars tensa area of the tympanic membrane for eustachian tube dysfunction are also a contraindication for irrigation of the external auditory canal (Fig. 13.17). If the child is asymptomatic, cerumen buildup can be reduced and the canals cleared by daily use of commercially prepared eardrops for dissolving earwax.

RED FLAG IN EAR ASSESSMENT

Irrigation of the external ear canal should not be attempted if there is a possibility of a ruptured tympanic membrane or patent myringotomy tubes.

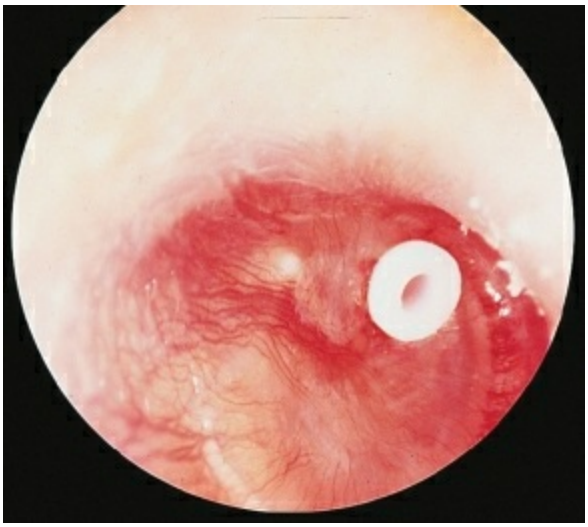

FIGURE 13.17 Tympanostomy or ventilation tubes in serous otitis media. Source: (From Zitelli BJ, Davis H: *Atlas of Pediatric Physical Diagnosis*, ed 5, St. Louis, 2008, Mosby.)

Hearing assessment

Hearing impairment in infants and children is a common disability with implications in cognitive, psychosocial, and academic development.[2] Approximately 1% to 3% of neonates have hearing loss identified in the newborn period by universal newborn hearing screening (UNHS).[5, 6] The goal of UNHS is to have all neonates screened for hearing loss before hospital discharge or by 1 month of age to ensure appropriate follow-up is obtained. Newborns who fail the newborn hearing screening and subsequent rescreening should be referred for audiologic and medical evaluations to confirm hearing loss by 3 months of age. All infants with hearing loss should begin receiving early intervention services before 6 months of age. Children who receive intervention for hearing loss by 6 months of age usually have normal language development by 5 years of age.[6] Early Hearing Detection and Intervention programs are working to ensure all newborns are screened for hearing loss, receive follow-up diagnostic testing if they fail the screening, and are enrolled in early interventions services if diagnosed with permanent hearing loss.[5] Rates of newborn screening are now greater than 96%, and enrollment in early intervention programs for infants diagnosed with hearing loss has risen from 55% to 62% in recent years.[7] The pediatric health care provider must continue to

track infants who fail the newborn screening to ensure these infants are retested and diagnosed and hearing augmentation is initiated in a timely manner.[7] The health care provider should not assume if the newborn hearing screening is normal that the growing infant or child's hearing is normal and no further hearing screening is necessary. Ten percent of childhood hearing loss is acquired after birth. It is important to routinely screen for hearing deficits and inquire about any changes noted in the home or school setting when gathering the health history.

The American Academy of Audiology recommends all children and adolescents be screened annually via conventional pure tone audiometry starting at age 3 years.[6] Any child under 3 years of age with speech and language delays, or who has a history indicating high risk for hearing loss or whose parents have concern regarding the child's hearing should be referred to an audiologist or otolaryngologist for testing (see Box 13.1). If significant concerns about hearing are present at any age, generated either from the health history or initial screening, then referral is indicated. Most primary care practices have access to conventional pure tone audiometry, but a variety of other screening tests are available for infants and young children (Table 13.3).

TABLE 13.3
Behavioral Audiometry in Infants and Young Children

Test	Age	Method
Conventional audiometry	4–5 years	Child is instructed to listen quietly for the test tone and to raise a hand or give a verbal response when it is heard
Bone-conduction testing	5 years	Calibration standards have not been established on infants and children; responses to bone-conducted stimuli may be inferred by head-turn response or as in conventional testing; young children may object to wearing oscillator
Hear test	Infants	Infant reaction to different frequency sounds is observed; elicited with standardized toys (e.g., bell, squeak toy) that make noises at different frequencies
Conditional play audiometry	2–5 years	Child performs a repetitive play task (e.g., places block in dish or peg in pegboard) in response to transmitted tone

Visual reinforcement audiometry	Developmental age 6 months–2 years	Loudspeakers, earphones, or bone-conduction oscillator is used to observe child's ability to hear and localize sound (by turning head or body); visual reward (e.g., lighted toy) provided for accurate responses
Behavioral observation audiometry	Developmental age birth to 5 months	Similar to visual reinforcement audiometry but used for infants or children unable to move head or eyes reliably; any repeatable response to sound may indicate hearing

BOX 13.1

Risk Factors for Hearing Loss in Infants and Children

- Congenital syndromes (Alport, Jervell, Lange-Nielsen, Usher, Down, Treacher Collins)
- Congenital infections: cytomegalovirus, rubella, toxoplasmosis, herpes, syphilis, and varicella
- Premature birth
- Very low birth weight (VLBW)
- Persistent pulmonary hypertension of the newborn
- History of extracorporeal membrane oxygenation (ECMO) therapy
- History of meningitis
- Exposure to ototoxic drugs (aminoglycosides, platinum-containing chemotherapy) and cranial radiation
- Cholesteatoma
- Chronic or recurrent acute otitis media (AOM) and otitis media with effusion (OME)
- Osteogenesis imperfecta

Behavioral audiometry determines the weakest intensity at which a child shows behavioral awareness of the presence of sound. Normal sound fields include 250 Hz to 6000 Hz, but hearing screening is often performed at 25 decibels between 500 Hz and 4000 Hz. Physiologic measures of hearing determine the infant's

physiologic response to stimulation of the auditory system (Table 13.4). Speech audiometry determines the child's response to speech stimuli and tests the clarity of sound received and perceived (Table 13.5).

TABLE 13.4
Physiologic Measures of Hearing

Screening Test	Response
Auditory brainstem response (ABR)	Measures electrical activity via scalp electrodes in the entire hearing pathway. Headphones or ear probes administer sounds and electrodes on the head measure the waveform response to sound. Can be used for UNHS with pass/fail reading or as diagnostic test. Results can give frequency range and decibel response level information. May be recommended after failed newborn hearing screen
Otoacoustic emissions (OAE)	Measures function of the external auditory canal, tympanic membrane, middle ear, and outer hair cells of the cochlear but not the inner hair cells or cochlear nerve. Failure may indicate a nonpatent ear canal, nonaerated middle ear, or lack of normal outer hair cell function needed for auditory nerve function.

UNHS, Universal newborn hearing screening.

TABLE 13.5
Speech Audiometry

Screening Test	Response
Speech detection threshold	Speech stimulus used to determine the ability to hear at varying decibels and frequencies via sound field, earphones, or bone conduction
Speech reception threshold	Word stimulus given and child repeats word or points to picture to indicate word heard
Central auditory processing tests	Tests evaluate school-age children with normal pure tone audiograms to determine speech perception with background noise, sounds in contralateral ear, rapid rate of presentation, or filtering

Interprofessional collaboration

Prompt referral of infants and children identified with hearing loss, consultation and collaboration with pediatric otolaryngology and

audiology specialists, and close follow-up in primary care will assure optimum development of hearing in affected children.

Weber and rinne hearing screening tests

In older children and adolescents, the *Webe*r and *Rinne* tests can be performed as additional screening tests to determine deficits in either conductive hearing or sensorineural hearing. These hearing screening tests are no longer performed in the primary care setting but may have application in the pediatric specialty setting or in health care settings in low-resource countries.

The Weber test is performed by placing a vibrating tuning fork (512 Hz) midline on the skull, making sure the examiner's hand does not touch the prongs of the tuning fork or the child's head. The school-age child or adolescent is then asked if he or she hears the sound of the tuning fork better on one side or the other, or equally well on both sides. If the child/adolescent indicates the sound is heard better on one side, this is called *lateralization* and indicates a conductive hearing deficit in the ear perceived as hearing the tuning fork better.

The Rinne test compares air conduction to bone conduction (Fig. 13.18). A vibrating tuning fork is placed on the child's mastoid bone to determine hearing via bone conduction. When the sound is no longer heard, the tuning fork should be moved to a position 1 cm to 2 cm from the external auditory canal. Sound is then being processed via air conduction in that area. Air conduction should be twice as long as bone conduction. If the bone conduction of sound is heard longer than air conduction, then a conductive hearing loss is present in the affected ear. If the ratio of air conduction to bone conduction is less than 2:1, then a SNHL is present. The Weber and Rinne tests are not reliable on children until school age.

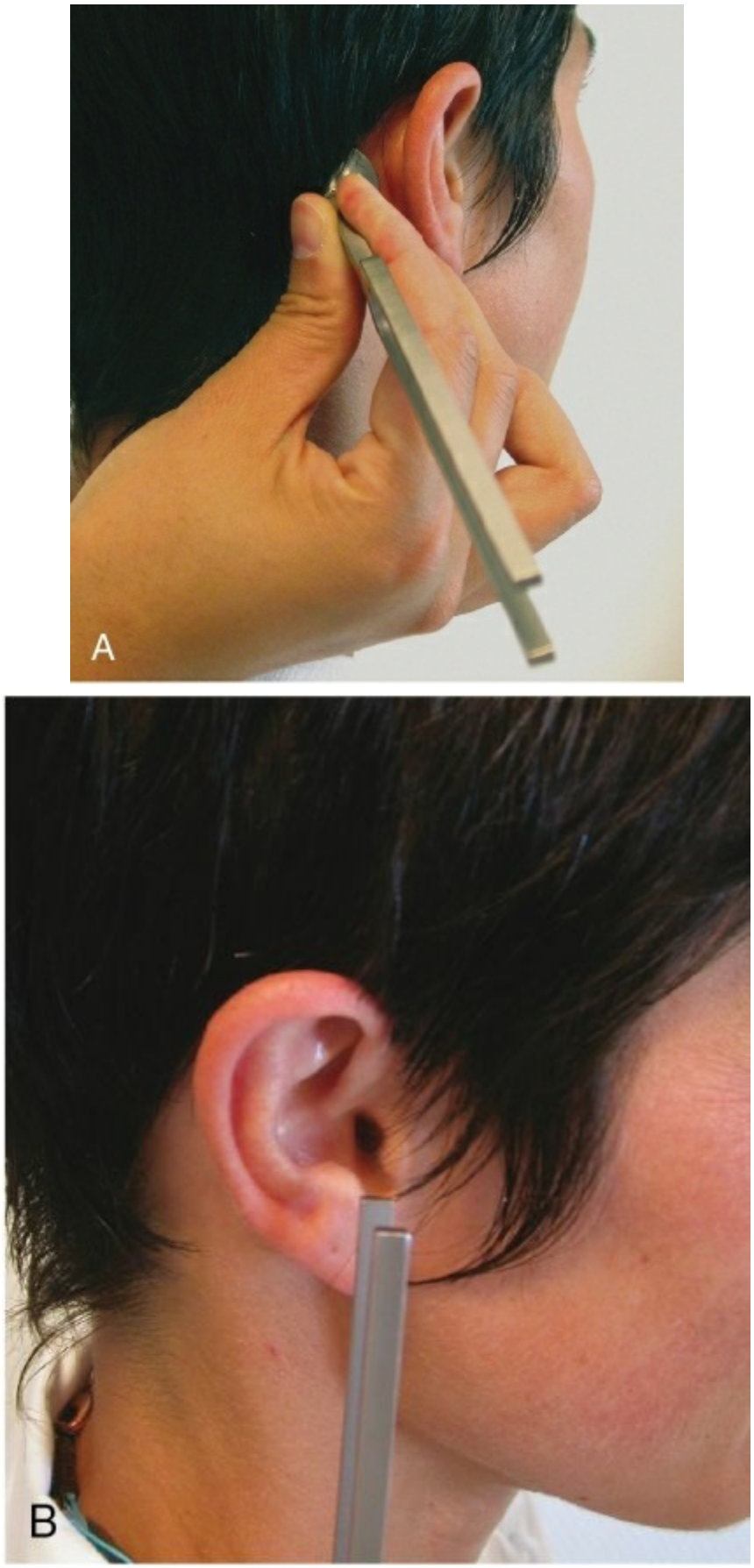

FIGURE 13.18 **(A)** Rinne test—bone conduction. **(B)**

Rinne test—air conduction. Source: (Published in Legent F, Bordure P, Calais C: *Audiologie Pratique Audiométrie*, ed 3, Paris, p. 9.

Ear conditions

Hearing loss

Before the advent of UNHS, the average age of identification of congenital hearing loss was 2.5 to 3 years. Some infants with mild hearing loss will be missed in UNHS, and a proportion of children who pass the UNHS will develop acquired hearing loss. The Joint Committee on Infant Hearing has established guidelines to improve identification of children at risk for late-onset hearing impairment (see Box 13.1).[2] Ongoing regularly scheduled surveillance of developmental milestones, auditory skills, speech and language development, parental concerns regarding hearing, and objective hearing testing are needed to identify children with progressive or acquired hearing loss.[2,7]

EVIDENCE-BASED PRACTICE TIP

Genetic testing is now often the first investigative tool to identify the cause of hearing loss. [3]

Conductive hearing loss

Conductive hearing loss is caused by an abnormality in the transmission of sound waves through the ear canal, the tympanic membrane, middle ear space, or middle ear ossicles. The auditory nerve system is intact, but the sound impulses do not reach the nerve. Transient *conductive hearing loss* is common during episodes of OME or AOM. Recurrent or chronic bilateral ear effusions during the early years of rapid language, speech, and communication development may impede development. Cholesteatoma, with its associated destruction of the middle ear, is another common cause of *conductive hearing loss,* but this hearing loss will be permanent and progressive unless the cholesteatoma is surgically removed and

middle ear reconstructed. Chronic or recurrent ear infections can cause *tympanosclerosis,* visualized as white scarring and thickening of the tympanic membrane (see Fig. 13.8), but scarring alone rarely results in measurable hearing loss. Acquired ossicular fixation from chronic diseases of the ear is almost never seen in children, although it is a relatively common cause of acquired hearing loss in older adults. Children with *osteogenesis imperfecta* do develop otosclerosis and must be followed by an ear specialist.

Congenital conductive hearing loss

Congenital conductive hearing loss can occur with Down syndrome or any gestational abnormality of the craniofacial structures. Isolated malformations of the external ear or *microtia, or* malformations of the ear canal, can result in *conductive hearing loss.* Congenital stenosis, congenital atresia of the stapes known as *Treacher Collins syndrome,* or congenital fixation of the stapes in the middle ear also result in a *conductive hearing loss.*

Sensorineural hearing loss

SNHL is caused by abnormalities of the cochlea, auditory nerve, or the auditory pathways that traverse the brainstem ending in the auditory cortex of the brain. SNHL is often congenital and genetically acquired. Genetic predisposition is thought to play a role in 50% of those affected by SNHL. Genetic syndromes that are associated with SNHL are Alport, Jervell and Lange-Nielsen, and Usher syndromes.

Newborns with possible perinatally acquired infections from a variety of pathogens should be screened for SNHL based on their clinical presentation. Any newborn with a history of TORCHS (*t*oxoplasmosis and *o*ther diseases: *r*ubella, *C*MV infections, *h*erpes simplex, *s*yphilis) should also be tested and monitored for SNHL. Infants and children with symptomatic congenital CMV infection are at greater risk for hearing impairment than those with asymptomatic infection. Approximately 22% to 65% of children with symptomatic infection have subsequent SNHL compared with only 6% to 23% of the children with asymptomatic infection.[8] CMV infection at any age may result in hearing loss.

Premature infants and very low birth weight (VLBW) infants are

at increased risk for hearing loss and have a higher incidence of hearing loss than full-term infants. Neonates with a history of persistent pulmonary hypertension or extracorporeal membrane oxygenation (ECMO) therapy have a 21% incidence of SNHL.[9] Children of any age who develop meningitis must be carefully tested for hearing loss because of both the consequences of the infections and the ototoxic side effects of many antibiotics used to treat meningitis. Children treated for malignancies with platinum compounds (cisplatin or carboplatin) or who are receiving cranial radiation may develop delayed SNHL and must be followed carefully with audiometry testing.

Mixed hearing loss

Hearing loss may also be a combination of conductive hearing deficits and sensorineural hearing deficits. Children with congenital syndromes often have mixed hearing loss.

The management of hearing deficits in children has advanced with new surgical techniques and bone-anchored, bone-conduction hearing aids (Fig. 13.19). Cochlear implantation and advances in hearing aids have improved the treatment of hearing loss dramatically, providing some sound to most children with even severe hearing loss.

FIGURE 13.19 Example of a bone-anchored hearing aid. Source: (Copyright Oticon Medical. Used with permission.)

Acute otitis media and otitis media with effusion

AOM is an inflammation within the middle ear that is often acute but can be recurrent or chronic, lasting more than 3 months. AOM is one of the most common conditions seen in pediatric practice in young children between 6 months and 3 years of age.[10] Children exposed to daycare or crowded living situations are prone to upper respiratory infections that may result in inflammation in the middle ear. Exposure to secondhand smoke has also been shown to increase the risk for AOM, whereas breastfeeding has been shown to be protective.[3] There is a genetic component to OM, with a higher incidence of OM in children who have older siblings or parents with a significant history of OM.

Abnormal clearance of middle ear fluid is the cornerstone of AOM and OME. Viruses and bacteria from the nasal pharynx enter the middle ear via the eustachian tube opening in the oropharynx. Young children are more prone to AOM because of their short, horizontal, less mature eustachian tubes, and more frequent upper respiratory tract infections. Children with nasal allergy are also more prone to AOM because inflammation of the respiratory tract

associated with allergies often causes swelling and obstruction of the eustachian tube, trapping fluid. Viruses or bacteria in the middle ear from respiratory infection cause further inflammation and often obstruction. Children with craniofacial defects or immunodeficiencies are at greatest risk for ear infections, and children with placement of nasogastric tubes also have a higher susceptibility.

The majority of OM infections are presumed to be of viral etiology. *Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis* are the most common bacterial pathogens found in AOM. Expanded immunization schedules covering an increased number of phenotypes of *H. influenzae* and pneumococcal strains have resulted in decreased incidence of OM caused by these organisms.

The presenting symptoms of AOM include rapid onset of ear pain or *otalgia,* fever, irritability, disrupted sleep pattern and occasionally *otorrhea,* or drainage from the ear. Severe conjunctivitis may also be present. Physical assessment should include careful otoscopy and pneumatic otoscopy to determine inflammation in the middle ear. AOM results in moderate to marked bulging of the tympanic membrane, a purulent effusion in the middle ear, and hemorrhagic redness, making bony landmarks difficult to see (see Fig. 13.16B).[10] The light reflex becomes diffuse and abnormally positioned on the tympanic membrane or absent. With pneumatic otoscopy the normal fluttering of the membrane is not present because of the increased fluid pressure in the middle ear. The tympanic membrane is usually erythematous, often with increased vascularity; however, these findings can also be present in a child who has been crying, and color should not be used as the primary finding to diagnose AOM in young children.

OME is a more common finding than AOM and is defined as middle ear effusion without signs or symptoms of AOM. OME presents with opacity of the tympanic membrane caused by a clear or serous transparent effusion in the middle ear or a cloudy nontransparent effusion.[10] OME causes decreased movement of the tympanic membrane on pneumatic otoscopy or tympanogram, a retracted or concave membrane, and visible air bubbles or a fluid line indicating eustachian tube dysfunction. OME may occur after

AOM as the acute infection resolves but before air pressure equilibrates, or it may result from eustachian tube dysfunction without acute infection. Chronic OME, or persistent effusion, interferes with sound wave transmission into the middle ear and is a common cause of conductive hearing loss.

Diagnosing AOM or OME with certainty in infants and young children can be a challenge. Positioning and restraining an irritable child, presence of cerumen in a narrow curved ear canal, and difficulty obtaining a proper seal of the ear canal for pneumatic otoscopy or tympanostomy all make certainty of diagnosis difficult. Bilateral AOM occurs more frequently in the child who is younger than 24 months than in children with unilateral AOM. Occurrence of bilateral AOM should not be a determining criterion for treatment.[10] Treatment guidelines and algorithms for AOM and OME are well established and take into consideration the child's age, duration of symptoms, and risk factors.[3]

Vestibular disorders affecting children

There is a growing recognition of vestibular system dysfunction in children resulting in gaze instability, dizziness, and balance problems. A recent study in children from 3 to 17 years found an overall prevalence of dizziness and balance problems of 5.3% that increased with age from 4.1% for children aged 3 to 5 years to 7.5% for children aged 15 to 17 years of age.[11] Dizziness, vertigo, disequilibrium, and unsteadiness can cause life-altering disability and health care burden. Increased risk factors associated with dizziness and balance problems include children with history of LBW or VLBW, intellectual disability, developmental delay, frequent headaches or migraines, recurrent AOM or hearing loss, or history of seizures. It is important for pediatric health care providers to screen children for vestibular problems and refer children to a pediatric neurologist, otolaryngologist, or early developmental intervention program.[11]

Summary of examination

- Before examining the ear, inspect the head, face, and neck for any asymmetry. The superior portion of the auricle should be

equal in height to the outer canthus of the eye.

- Inspect the auricles for size, shape, symmetry, deformity, placement, discharge, and color.
- Inspect the tympanic membrane for contour, intactness, color, translucency, and presence of visible landmarks (umbo, handle of malleus, and light reflex).
- The light reflex is usually found between the 4 o'clock and 6 o'clock position on the right tympanic membrane and 6 o'clock to 8 o'clock on the left tympanic membrane.
- Mobility of the tympanic membrane, an important indication of middle ear pressure, can be assessed with a pneumatic attachment to the otoscope or by use of a tympanometer.
- Ear drainage in the external canal should alert the examiner to otitis externa, a ruptured tympanic membrane, patent tympanostomy tubes, or cholesteatoma.
- Ten percent of childhood hearing loss is acquired after birth. Routinely screen for hearing deficits and begin puretone audiometry at 3 years of age.
- Adhere to current guidelines for treatment of AOM and OME in infants and children.
- Vestibular disorders should be considered in children with gaze instability, dizziness, and balance problems.

DOCUMENTATION

Term newborn infant

Ears: Auricle well formed, symmetrical, with normal alignment. External canals patent with small amount of white residue. Tympanic membranes partially visible, gray, opaque, without visible light reflex or bony landmarks. Newborn hearing screening normal.

CHARTING

Adolescent

Ears: Auricles well formed, symmetrical, with two healed piercings on outer border of helix and one healed piercing center of lobe. No masses, erythema, or tenderness noted. External canals with minimal dark brown cerumen. Tympanic membranes pearly gray, concave, light reflex and bony landmarks visible. + movement with insufflation. Screening audiometry—NL. 1000–4000 frequency (Hz) at 25 decibels (dB).

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CHAPTER 14

Nose, mouth, and throat

Patricia Jackson Allen

A thorough assessment of the nose, mouth, and throat is an essential part of the pediatric physical examination. Infants and children have frequent upper respiratory and viral infections, and viewing the oropharynx is particularly important when looking for a focus of infection in a febrile child. Also, the oral health of children is key to overall health, and the pediatric health care provider is an important link in providing oral health assessments and preventing dental caries.

Embryologic development

The facial structures develop in the embryo during the first few weeks of gestation. The tongue, lips, gums, and tooth enamel all evolve from the ectoderm of the primitive mouth, the *stomodeum*, early in the fourth week. The lips are formed during the fourth to eighth weeks of gestation. The primary teeth and salivary glands are formed between the sixth and eighth weeks of fetal life. By the sixth fetal month, the ducts are hollow and begin producing saliva. Calcification of the *primary teeth* begins in the fourth month of fetal life and is complete by 1 year of age. Any insult during pregnancy to the sensitive process of tooth formation can result in an anomaly in the color, size, or shape of the primary or permanent dentition.

Early development of the nose begins during the fifth week of gestation, with development of muscle, bone, and cartilage complete by the 12th week of gestation. The *palate* evolves from fusion of the maxillary prominences during the seventh and eighth weeks of gestation and is completely formed by the 12th week of gestation during the fusion of the primary and secondary palates. Failure in fusion results in *cleft palate*. *Cleft palate* is a relatively common congenital anomaly and occurs in 1 in 690 births (Fig. 14.1B).[1] The incidence varies with race, with Native Americans having a rate of 20.5 in 10,000 births, and blacks having the lowest rate of 10.2 in 10,000 births.[1] Cleft lip can be an isolated defect or associated with cleft palate or a component of a congenital syndrome (Fig. 14.1A).[2] The etiology is multifactorial involving both genetic and environmental risk factors in both syndromic and nonsyndromic clefts. Several risk factors associated with cleft type have been identified and include maternal smoking, alcohol consumption, diabetes, maternal nutrition and folic acid intake, and maternal medications.[1] A *subcutaneous cleft* also can occur during this period with incomplete fusion of the palate; it often goes undetected in the newborn and is associated with persistent abnormal speech patterns.

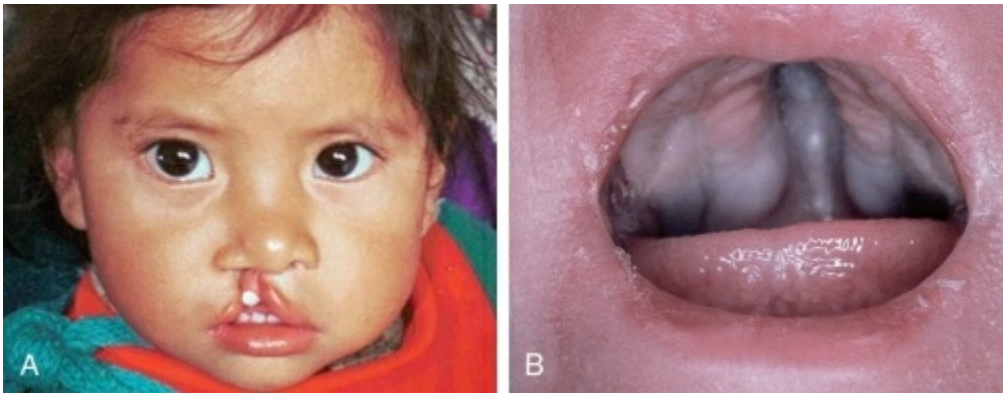


FIGURE 14.1 **(A)** Cleft lip. **(B)** Posterior cleft soft and hard palates. Source: ([B] From Chaudhry B, Harvey D: *Mosby's Color Atlas and Text of Pediatrics & Child Health,* St. Louis, 2001, Mosby.)

Developmental variations

Table 14.1 presents the physiological variations to be monitored from infancy to adolescence. Table 14.2 reviews the development of the sinuses from infancy through adolescence.

TABLE 14.1
Physiologic Variations of the Nose, Mouth, and Throat

Age-group	Physiologic Variations
Newborn	Nose cartilage is soft, malleable; deformities in external appearance from intrauterine or birth positioning usually resolve spontaneously; congenital anatomic deformities, obstructive masses, or traumatic obstruction can occlude the nasal passagesNatal teeth may be present Epstein pearls, small whitish nodules or cysts, at juncture of hard and soft palates may be visible in first month of life; Bohn nodules, or mucous gland cysts, may be present on gum surface in first 2–3 months Rooting, gag, sucking reflexes are present A short tight lingual frenulum attached to the inferior tip of the tongue may impede movement of the tongue and breastfeeding
Infancy	Anatomically small airway passagesOcclusion of nasal pathways can occur with nasal secretions Deciduous teeth appear between 6 and 24 months Rooting, sucking reflexes wane about 4–6 months Drooling increases as salivary gland production increases Anterior permanent teeth begin to calcify at 3–12 months Ethmoid and maxillary sinuses present but undeveloped
Early childhood	Tonsils, adenoids enlarge and may remain 2+ to 3+Nasal passages enlarge allowing easier airflow Maxillary and ethmoid sinuses present but sphenoid and frontal sinuses limited in size and function Sinuses not normally assessed in children until middle childhood because of their limited development Swallowing coordination improves; drooling decreases Permanent molars begin to calcify at 18 months to 3 years
Middle childhood	Tonsils and adenoids usually begin to atrophy returning to size 1+ to 2+Horizontal creases on anterior nose may develop in children with nasal rhinitis/allergies Deciduous teeth begin to shed; permanent teeth erupt causing change in facial structure, appearance Bridge of nose becomes more prominent Third molar, last permanent tooth, is formed and begins calcifying
Adolescence	All permanent teeth presentBridge of nose formed by bone creating pyramid shape Frontal and sphenoid sinuses completely formed and functioning

Anatomy and physiology

External nose

The nose of the newborn and young infant is generally flattened and malleable (Fig. 14.2). In the neonate, the septum is composed of cartilage; ossification occurs during childhood. The nose becomes pyramid-like by adolescence and develops a bony structure. It is divided into four sections: the proximal bony portion, often referred to as the *nasal bridge;* the mid cartilaginous vault; the tip, *columella,* and *nares;* and the interior *vestibule* (Fig. 14.3).

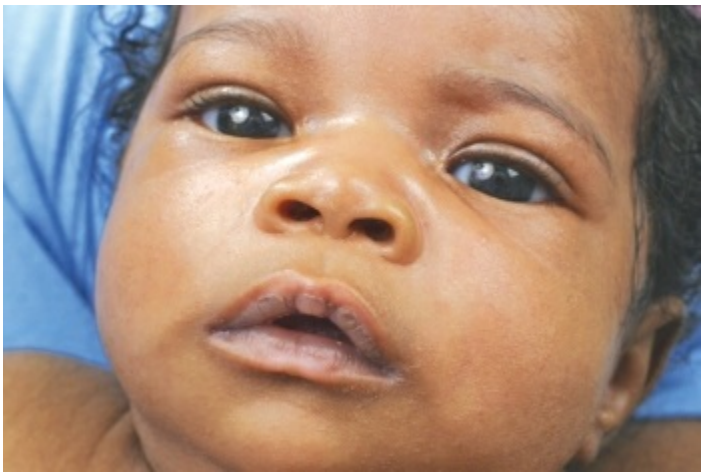


FIGURE 14.2 Flattened nasal bridge in infant.

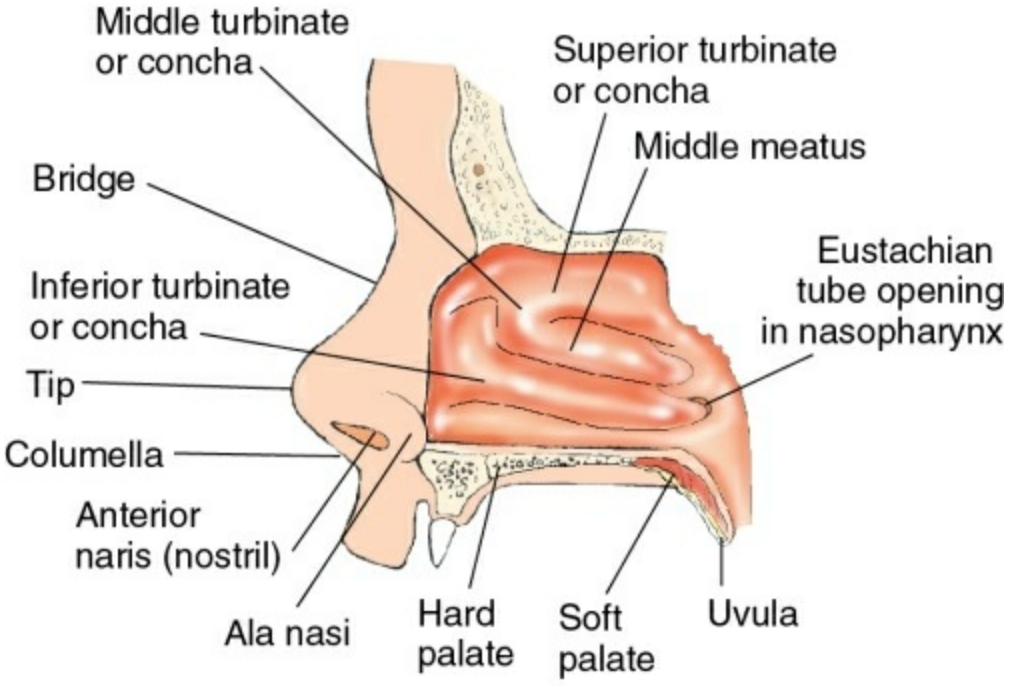


FIGURE 14.3 Anatomy of the nose.

Nasal breathing is the normal breathing pattern, and infants and young children are prone to increased airway resistance, because they have anatomically small airway passages. Nasal congestion in the neonate is a common normal finding, and newborn nurseries often discharge infants with a nasal bulb syringe to enable the gentle removal of secretions from the small narrow nasal passages to clear the airway to enhance breastfeeding and bottle-feeding. In the past, it was thought that newborns are obligatory nasal breathers for the first few months of life. However, research has demonstrated newborns have the ability to switch from nasal to mouth breathing as needed, so they are "preferential" nasal breathers.[2] Respiratory compromise or distress occurs rapidly in young infants when the nasal passages become occluded. The most common reason for occlusion is mucosal congestion or increased secretions, but congenital anatomical deformities, obstructive masses, or traumatic obstruction can occlude the nasal passages. Nasal resistance is an important factor in total airway resistance, and nasal occlusion may contribute to half of total airway resistance.

Internal nose

The internal nose, the *vestibule,* is divided by the bony and cartilaginous *nasal septum.* The septum is rarely perfectly straight, and a significant deviation of the septum at birth or resulting from the birth process or trauma must be assessed to determine whether it interferes with nasal breathing. The perpendicular plate of the nasal septum ossifies by 3 years of age. The anterior portion of the vestibule is lined with vascular squamous epithelium that has tiny hair follicles and secretes mucus. The vast majority of nose bleeds, or *epistaxis,* result from a network of small blood vessels found in the anterior superficial portion of the septal mucosa known as the *Kiesselbach plexus.* The posterior portion is lined with fragile respiratory epithelium. The lateral walls of the nose are composed of horizontal bony structures known as the *superior, middle,* and *inferior turbinates,* which mature throughout childhood and resemble those of the adult by 12 years of age (see Fig. 14.3). They are covered with vascular mucous membranes. Furrows between the bony structures provide recesses to filter air and form a nasal passage, or *meatus.* The posterior *ethmoid sinuses* drain into the superior meatus, and the *paranasal sinuses* drain into the middle meatus. Until approximately 6 years of age, the inferior meatus is nonfunctioning except that it drains the *nasolacrimal duct.* This is why the nose has increased drainage in children, particularly during periods of crying or eye irritation. The space between the posterior portion of the turbinates and the posterior wall of the nasopharynx is called the *choana* and is of little significance in children unless blocked or narrowed by a congenital abnormality such as *choanal atresia,* a bony or membranous blockage of one or both *naris* posterior to the nasal turbinates, resulting in blockage of the airway and respiratory distress in the newborn. It occurs in 1 in 5000 to 7000 births and occurs more often in females 2:1 and unilaterally 2:1. In newborns, choanal atresia can be associated with the CHARGE* syndrome.[3]

Cranial nerve I (olfactory) innervates the nasal area. The *olfactory receptor* cells line the upper reaches of the nasal cavity in the olfactory epithelium and innervate the olfactory nerve. Olfactory learning begins in utero and is well developed in the newborn. It assists newborns in recognizing the distinct smell of their mother's breast milk. Nasal congestion or mucus plugging limits airflow up

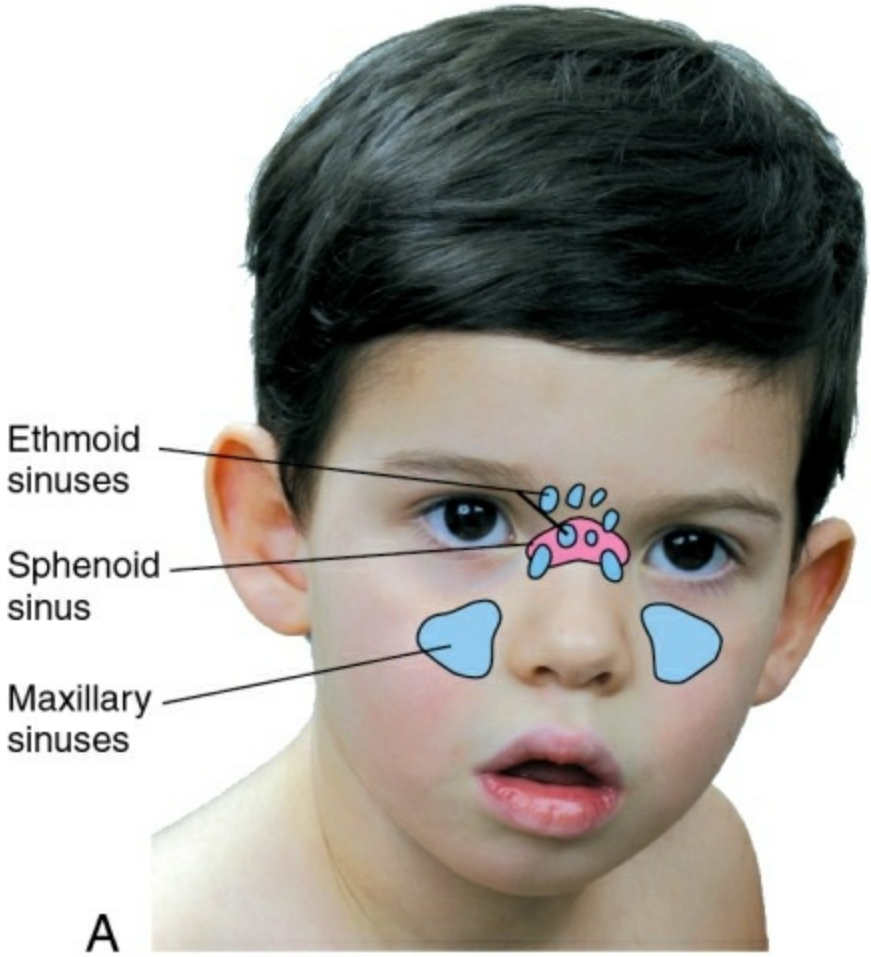
to the receptors and can block the sensation of smell.

Nasopharynx

The *nasopharynx* forms the superior portion of the pharynx. The *eustachian tube* opening is located along the lateral walls of the nasopharynx (see Fig. 14.3). Adenoidal tissue is found along the superior posterior wall of the oropharynx and is referred to as the *pharyngeal tonsils*. The inferior border of the nasopharynx is formed by the soft palate. The nasopharynx is surrounded by bone, ensuring patency unless trauma occurs.

Sinuses

The paranasal sinuses consist of paired cavities: maxillary, ethmoid, frontal, and sphenoid sinuses. The *maxillary* and *ethmoid* sinuses are present at birth but are small. The *frontal* sinuses begin to develop by 7 years of age, and the *sphenoid* sinuses develop in adolescence. As the sinuses develop, they become air-filled cavities; and when mature, they are lined with ciliated epithelium containing goblet cells and submucosal glands that produce seromucinous secretions and immune mediators. The sinuses reach their final maturity between 12 and 14 years of age (Fig. 14.4; see Table 14.2).[4]


Ethmoid sinuses
Sphenoid sinus
Maxillary sinuses
A

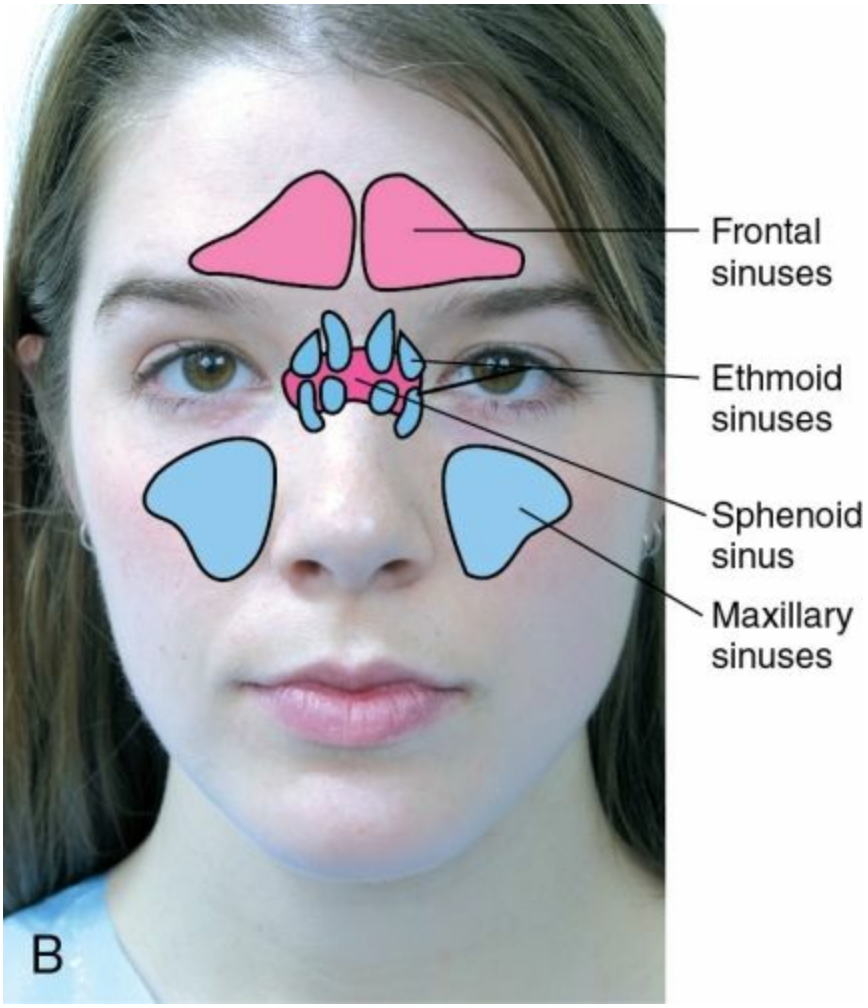


FIGURE 14.4 **(A)** Sinus development in childhood. **(B)** Sinus development in adolescence.

TABLE 14.2
Development of Sinus Cavities

Sinus Cavity	Development
Maxillary	Present at birth; first sinuses to develop significantly; can be seen radiologically at 4–5 months of age; opens beneath the middle turbinate into the middle meatus; rapid growth occurs between birth and 4 years of age and 6–12 years of age.
Frontal	Last sinuses to develop beginning 7 years of age and do not develop fully until late adolescence. Secretions drain into the middle meatus. The walls of the frontal sinus border the orbital and intracranial cavities, which can increase the risk for frontal sinus infections spreading directly into these adjacent structures.
Ethmoid	Present at birth, but not developed, grow rapidly during the first 4 years and are fully developed by 12–14 years of age; they are first seen radiologically at 1 year of age. Ethmoid sinuses are divided into anterior portion, draining into the middle meatus, and posterior portion, which drains into the superior meatus.

Sphenoid	Undeveloped at birth and do not begin to grow rapidly until 3–5 years of age; development complete between 12 and 15 years of age. They lie anterior to the pituitary fossa, and the optic nerve and carotid artery are located on the lateral wall of the sinuses, offering a potential route for spread of infection into the central nervous system.

Data from Schoem SR, Darrow DH: *Pediatric Otolaryngology*, Elk Grove, IL, 2012, American Academy of Pediatrics.

Mouth and oropharynx

The *oral cavity* is composed of the lips, cheeks, hard and soft palates, teeth, posterior pharynx, tongue (Fig. 14.5), sensory cells for taste, and the mandible that supports the lower gums and teeth. The cheeks form the lateral walls that are lined with *buccal mucosa.* Cheeks may be particularly prominent in young children because of the buccal fat pad. The cheeks and lips are innervated by *cranial nerves V (trigeminal)* and *VII (facial)*. The central nervous system controls the complex mechanisms of the mouth needed for sucking, swallowing, breathing, and vocalization. The *hard palate* is the anterior two thirds of the palate and separates the nasal and oral cavities. The posterior third of the palate is the *soft palate,* which is contiguous with the lateral pharyngeal wall. It provides a slightly mobile barrier between the nasopharynx and oropharynx and is essential for normal articulation and speech intonation.

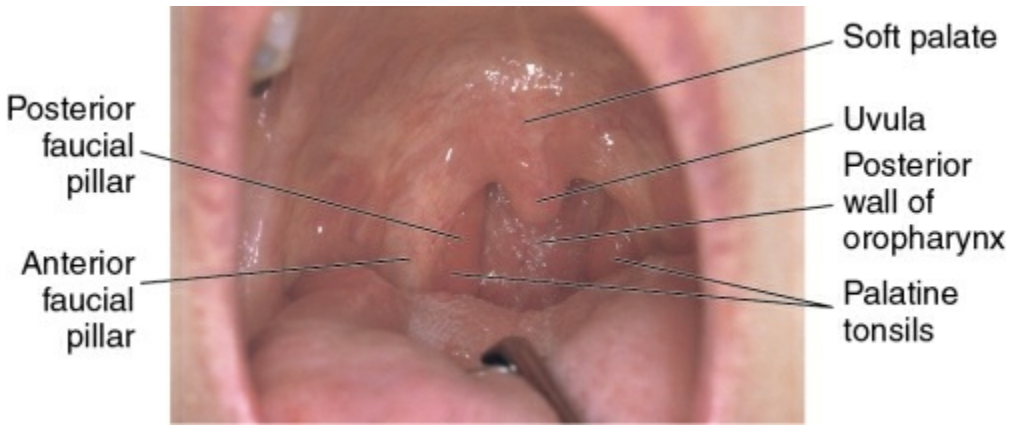


FIGURE 14.5 Anatomy of the posterior oral cavity. Source: (From Fehrenbach M, Herring S: *Illustrated Anatomy of the Head and Neck*, ed 4, St. Louis, 2012, Elsevier.)

Tonsils

The *palatine tonsils* form the anterior and posterior tonsillar pillars. Tonsillar size is graded on a scale of 1+ to 4+ (Table 14.3 and Fig. 14.6). Additional tonsillar tissues surround the posterior pharynx but are not visible on examination. The *uvula* hangs down from the middle of the soft palate in line with the anterior pillar, or *palatoglossus muscle.* A *bifid uvula,* a cleft uvula with two parts, is an anomaly that results from disruption of the palate development and may indicate a *submucosal cleft palate.* It may also be associated with nasal polyps and is also more common in children with cystic fibrosis.

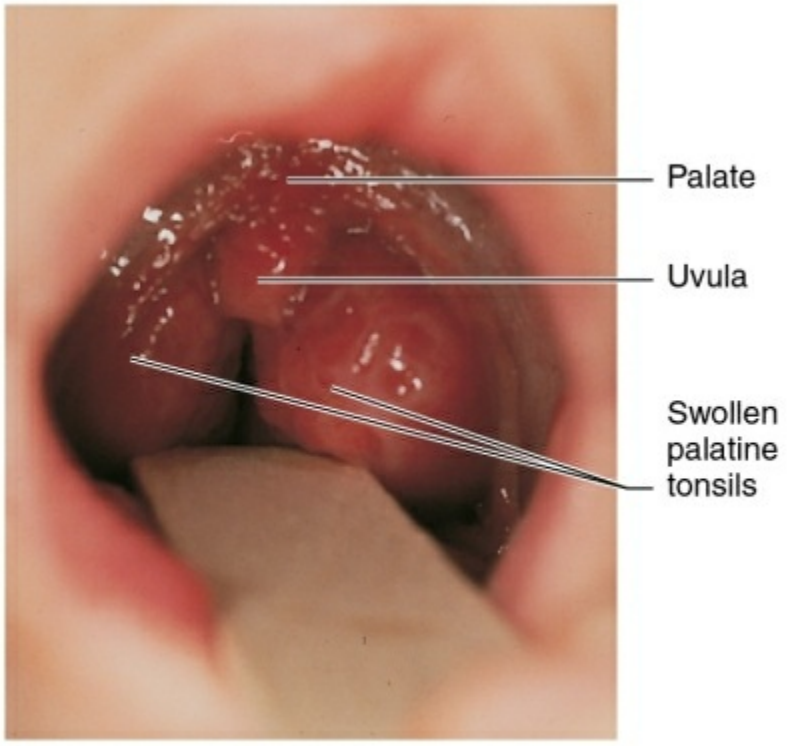


FIGURE 14.6 Enlarged tonsils in child. Source: (From Thibodeau G, Patton K: *The Human Body in Health and Disease*, ed 5, St. Louis, 2010, Mosby.)

PEDIATRIC PEARLS

In the infant, the palatine tonsils are not normally visible, but by

2 years of age, they are usually seen extending medially into the oropharynx. They generally are at their peak size between 2 and 6 years of age and then begin to atrophy or decrease in size along with other lymphatic tissue.

Enlarged tonsils and adenoids can partially occlude the posterior pharynx during sleep resulting in sleep-disordered breathing (SDB).

Teeth

The mandibular central incisors are the first to erupt in the majority of infants, followed by the maxillary central incisors, the upper and lower lateral incisors, first molars, cuspids or canine teeth, and then the second molars. Tooth *eruption*, movement of the tooth through alveolar bone and gums, normally occurs between 4 and 12 months of age for the first tooth, and occurs when about two-thirds of the root for the tooth is developed. The maxillary incisors usually erupt 1 to 2 months after the mandibular incisors. The eruption of the 20 primary teeth should be complete between 24 and 30 months of age (Fig. 14.7A, C). The timing and sequence of tooth eruption depend on genetic, nutritional, environmental, and systemic factors. Delayed eruption of the primary teeth can occur in premature infants, infants small for gestational age, infants or children with metabolic or chromosomal abnormalities, or children with severe malnutrition. A familial pattern of delayed tooth eruption can also occur across generations.

The permanent teeth begin developing in the mandible during the first 6 months of life. The period of *eruption* of the *mixed dentition* occurs between 5 and 13 years of age, beginning with the eruption of the first permanent tooth. *Exfoliation*, or loss of the primary dentition, often begins with the central incisors and follows the eruption pattern. There are 32 permanent teeth (see Fig. 14.7B, D). Low birth weight, infection, and trauma have been associated with delayed eruption of the permanent teeth. Delayed exfoliation of the primary dentition has also been associated with Down syndrome, hypothyroidism, osteogenesis imperfecta, and other congenital endocrine disorders. Dental enamel can be eroded, resulting in structurally weakened teeth in some conditions such as chronic

gastroesophageal reflux disease, bulimia, and celiac disease.

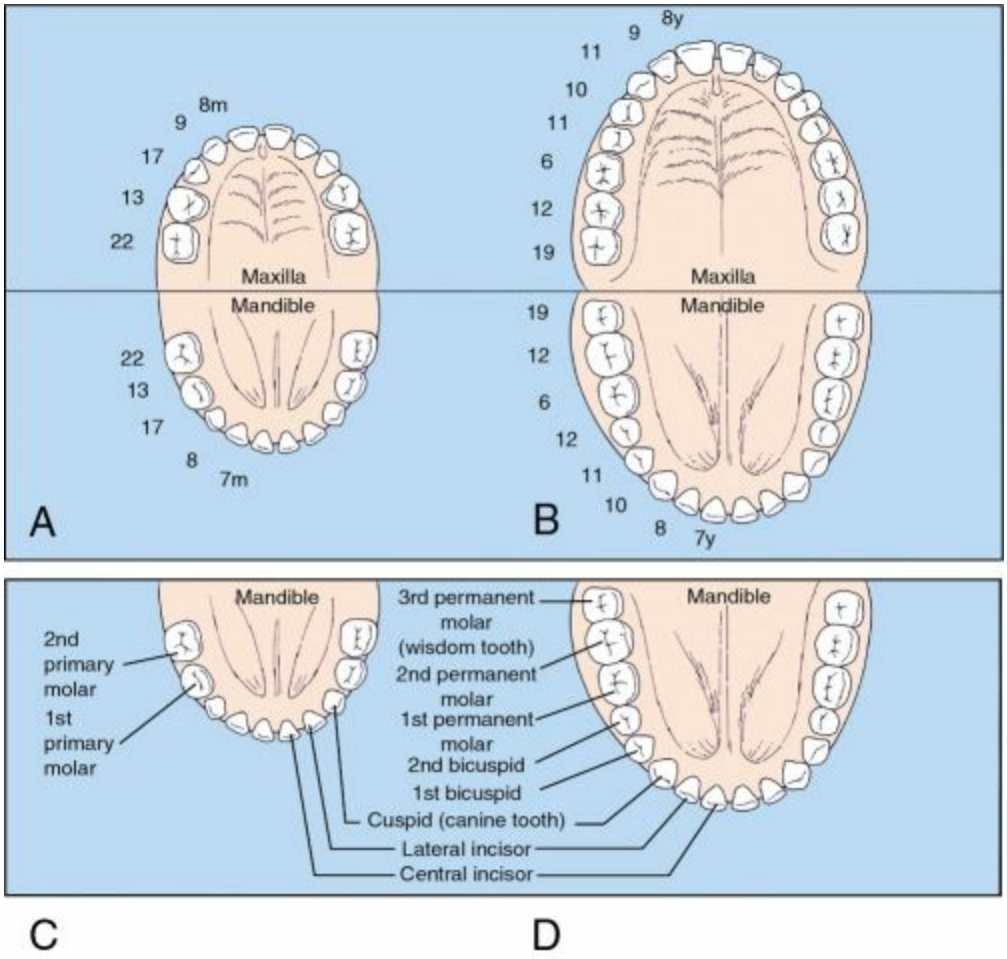


FIGURE 14.7 Ages of tooth eruption. **(A)** Primary dentition of maxilla. **(B)** Permanent dentition. **(C)** Primary dentition of mandible. **(D)** Permanent dentition of mandible. Source: (From Zitelli BJ, McIntire SC, Norwalk AJ: *Atlas of Pediatric Physical Diagnosis*, ed 6, Philadelphia, 2013, Elsevier.)

Dental caries are the most common chronic health condition in childhood.[5] The infectious process of dental decay begins early in infancy when the causative bacterium *Streptococcus mutans and lactobacilli* can be transmitted from parent or caretaker to the infant through oral contact in the first few months of life. Ingestion of a high carbohydrate diet and/or frequent dietary sugars in infants and children provides a substrate for the bacteria to flourish and alters the oral bacterial composition, enhancing the development of dental caries.[6]

Tongue

The *tongue* is a mobile muscle, with its anterior two thirds located in the oral cavity and the posterior third located in the oropharynx. The anterior dorsal surface of the tongue is composed of a thick mucous membrane lined with *filiform,* or threadlike, papillae, and the posterior dorsal surface is lined with lymphoid tissue that forms the *lingual tonsil.* The ventral surface of the tongue has a thin mucous membrane with visible vessels and is normally anchored to the floor of the mouth by the *lingual frenulum* (Fig. 14.8).

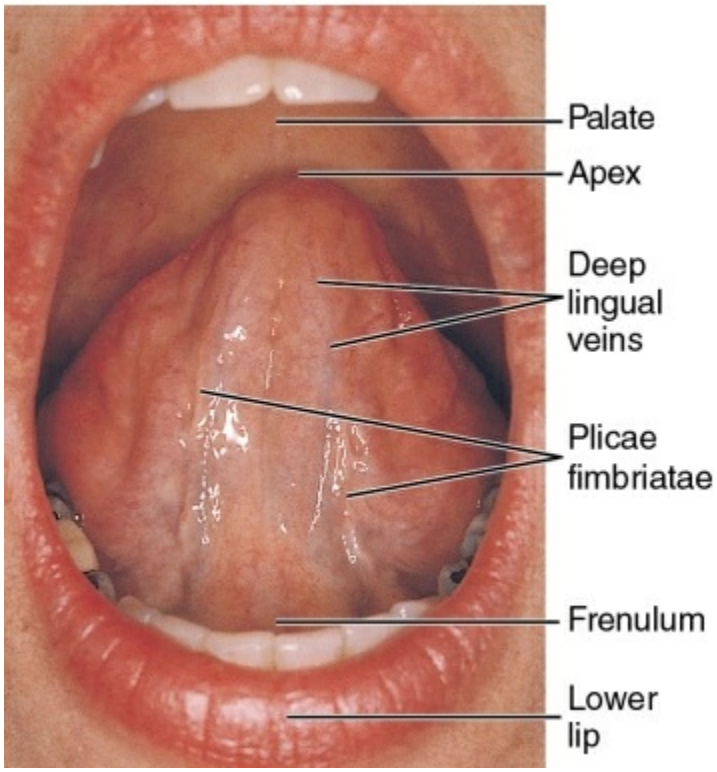


FIGURE 14.8 Ventral surface of the tongue and salivary glands. Source: (From Fehrenbach M, Herring S: *Illustrated Anatomy of the Head and Neck,* ed 4, St. Louis, 2012, Elsevier.)

Cranial nerves IX (glossopharyngeal) and *X (vagus)* innervate the tongue for sensation and taste, and *cranial nerve XII (hypoglossal)* innervates the tongue for motor function. The sensation of taste is immature at birth and not fully functional until approximately 2 years of age. Term infants also have a *tongue-thrust reflex* for the first 4 months of life that aids in breastfeeding or bottle-feeding and to protect them from choking, but it is counterproductive when trying

to feed solids by spoon. At the base of the tongue in the oropharynx lies the *epiglottis,* a glistening pink spoon-shaped appendage that helps direct the passage of food into the esophagus and away from the trachea (Fig. 14.9). Normally, it is not visible on examination, but in some children, it can be seen protruding upward from the posterior oropharynx, almost opposite from the uvula when the tongue is depressed and the child says "ah," which opens the throat area for examination.

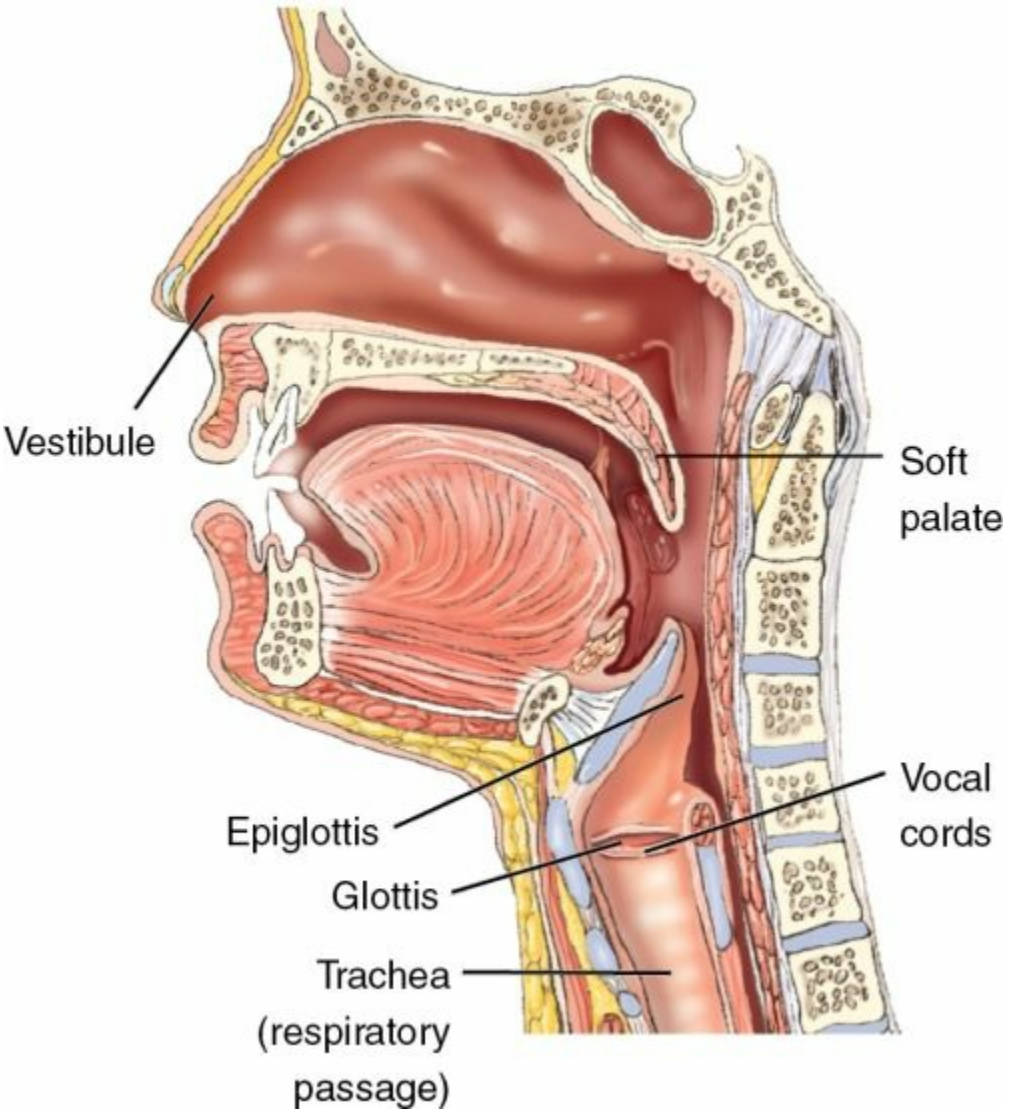


FIGURE 14.9 Sagittal view of mouth and oropharynx.

Salivary glands

The *salivary glands* are paired exocrine glands that secrete enzymes that aid in initial digestion. The *parotid glands* are the largest salivary glands and are the glands that become inflamed with *mumps,* or *parotitis.* The *parotid duct,* or *Stensen duct,* empties into the oral cavity opposite the upper second molar. The *submandibular gland* is the second largest gland and is located in the floor of the mouth. The *submandibular ducts,* or *Wharton ducts,* exit into the mouth on either side of the lingual frenulum. The third set of salivary glands is the *sublingual glands,* which release their enzymes through approximately 12 ducts located on the floor of the mouth. The sublingual glands are not visible on examination. Secretions from the salivary glands begin around 6 weeks of age in the term infant, which results in increased drooling by 3 to 4 months of age. As infants mature and become more proficient in swallowing and the lower teeth develop to create a dam, the drooling decreases even though the production of saliva increases. Hundreds of additional salivary glands line the mucous membranes of the mouth and oral pharynx by late adolescence, providing additional serous and mucous secretions.

System-specific history

The Information Gathering table presents important information to gather for different ages and developmental stages when assessing the nose, mouth, and throat.

Information Gathering for Assessing the Nose, Mouth, and Throat at Key Developmental Stages

Age Group	Questions to Ask
Preterm and newborn	History of maternal infection, TORCH infections? Maternal drug use? Perinatal exposure to infection? Any difficulty sucking, feeding? Difficulty breathing through nose? Is the frenulum attached to the anterior portion of the ventral surface of the tongue limiting movement of the tongue? Any natal teeth or lesions in mouth?
Infancy	Any nasal discharge? Any difficulty sucking, feeding, introducing solid foods?Any sores, white patches, or bleeding in mouth? Have any teeth erupted? Use of juice in bottle? Is infant on fluoride supplement or is water fluoridated? Plans for weaning from breast or bottle?

	Does infant habitually put objects in mouth? Use pacifier or suck thumb or fingers? How frequently?
Early childhood	Does child have difficulty eating solid foods? Does the child have strong food preferences or known food allergies? Does the child use a bottle for milk or juice?Does the child put objects in their mouth or nose? Are there any concerns about child's speech? Does child's speech have a nasal or congested resonance? History of frequent nasal congestion, chronic rhinorrhea, or tonsillitis? Does the child snore? Have restless sleep patterns? Complain of being tired during the day? Has child had nose injuries? Recurrent nose bleeds? Does child suck a digit or pacifier? Is child in daycare or preschool? Age at first dental visit? Recent application of fluoride varnish? Does child brush teeth with parental assistance? Does the child have any missing or injured teeth? History of trauma to mouth or gums?
Middle childhood	Any mouth or nose injuries?History of nasal congestion, chronic rhinorrhea, tonsillitis? Does child snore? Have restless or interrupted sleep pattern? Complain of being tired during the day? Any known exposure to Group A Streptococcal infection? Has child had a documented GABHS pharyngitis? Or other pharyngeal infections? Routine dental care? Does child brush and floss? Are teeth aligned properly or is a referral needed for orthodontia? Has child had any teeth extracted? Tonsils or adenoids removed? Does child participate in competitive contact sports? Wear a mouth guard?
Adolescence	Any injuries to mouth or nose? Does adolescent play competitive contact sports? Wear a mouth guard?History of nasal congestion, chronic rhinorrhea, tonsillitis? Does adolescent snore? Have restless sleep? Complain of being tired during the day? Have tonsils or adenoids been removed? Has adolescent had previous documented GABHS infections or recently been exposed to Group A Streptococcal infection? Other pharyngeal infections? History of oral sex? Are there any oral piercings? Oral lesions/sores? Routine dental care, brushing, flossing? Dental braces or orthodontic appliances? Teeth removed or lost due to injury?
Environmental risks	Exposure to household tobacco smoke? Recreational drug or tobacco use or exposure to use? Recreational activities or sports with increased risk of injury to mouth or nose?

GABHS, Group A beta-hemolytic streptococci; *TORCH,* toxoplasmosis, other diseases (syphilis), rubella, cytomegalovirus, herpes simplex virus.

Physical assessment

Equipment

Equipment needed for examination of the nose, mouth, and throat

includes an otoscope with halogen light and speculum, tongue depressor, and gloves for palpation of palate and gums if any skin lesions are present.

Positioning

Delay the examination of the mouth and nose in the infant and young child until after the "quiet" parts of the exam and after the ear examination. If the infant or young child cries, attempt to visualize the mouth and oropharynx with a tongue depressor. Infants are best positioned on the examination table with the arms secured by the sides and head stabilized to visualize the internal nose and mouth. Examination of the older infant or young child can also occur while the child is sitting in the parent's or caregiver's lap with the head secured by the examiner's hands and with the child's arms secured by the parent (Fig. 14.10). The feet may need to be secured between the parent's legs if the child is uncooperative. With the young child on the examination table, the parent may also secure the arms above the head to stabilize the head and visualize the oropharynx (Fig. 14.11).

FIGURE 14.10 Position for the oral examination in the infant and young child.

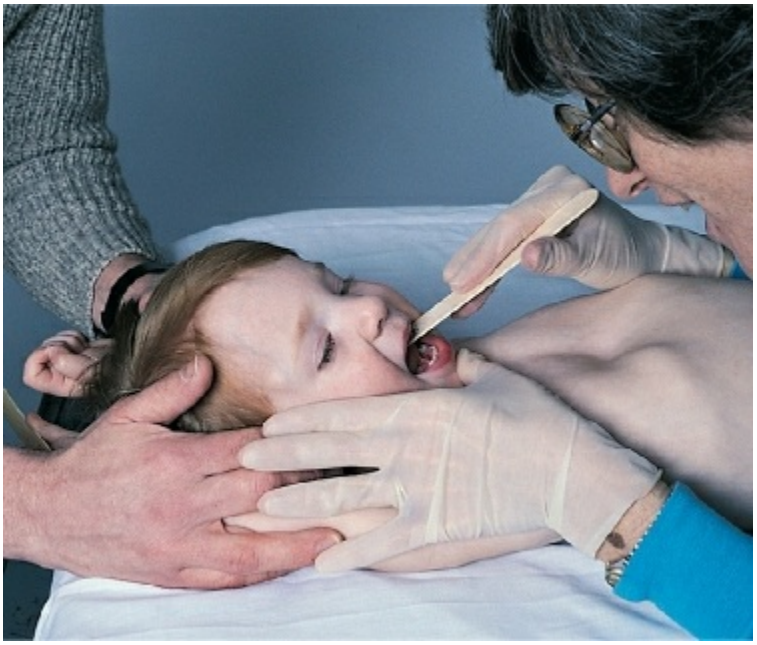


FIGURE 14.11 Position for stabilizing child's head for oral examination. Source: (From Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW: *Seidel's Guide to Physical Examination,* ed 8, St. Louis, 2015, Mosby.)

An alternative position for conducting the inspection of the teeth is to place the child in the "knee-to-knee" position. In this position, the examiner and parent sit face to face with their knees touching to make a comfortable support for the young infant, and the infant or young child's head is placed in the examiners lap to look directly into the child's mouth (Fig. 14.12). This position is best used for examining the teeth and applying fluoride varnish in the older infant and young child.

FIGURE 14.12 Knee-to-knee position for dental assessment. Source: (From Clark C: Open mouth, open mind: expanding the role of primary care nurse practitioners. *J Pediatr Health Care* 30(5):480-488, 2015.)

External nose

While the child is comfortable, note any flaring or narrowing of the nares with breathing. If an infant is feeding, watch carefully for indications of nasal obstruction requiring mouth breathing. Note the shape of the nose, any obvious deviation of the bridge or columella, the tip of the nose. In older children, a transverse crease across the nose or nasal bridge caused by repeated upward swipes with the hand due to chronic nasal drainage is known as the *allergic salute*. If there is drainage from the nose, note the color, consistency, and quantity and whether it is unilateral or bilateral. Allergic conditions and upper respiratory infections cause bilateral drainage, whereas foreign objects in the nose can cause unilateral,

purulent, malodorous discharge. *Epistaxis,* bleeding or hemorrhage from the nose, occurs from irritation of the nasal mucosa, often due to cold, dry environmental conditions, nasal allergy, or as a result of trauma. Sinus infections also may cause unilateral drainage. Swelling or dark discoloration under the eyes, often called *allergic shiners,* may occur with nasal congestion, *sinusitis,* or nasal allergy.

Palpate any areas around the nose that appear discolored or inflamed. If there is a history of facial trauma, palpate the bridge of the nose to determine tenderness or pain. If the child has a recent history of head or facial trauma and clear watery nasal discharge, a cerebrospinal fluid leak must be considered and confirmed with imaging. Traumatic fractures of the protective facial bones increase the risk of developing meningitis. Children with obvious deviation of the nose should be referred to a craniofacial specialist for evaluation. Patency of each side of the nose can be determined by gently occluding one naris at a time. In the newborn, this technique assists in diagnosis of choanal atresia.

Internal nose

To inspect the internal nose, use a penlight or otoscope with halogen light and nasal speculum while being careful not to touch the sensitive internal nasal septum. A large ear speculum can be inserted 2 to 3 mm into the nares in older children and adolescents for inspection of the nasal cavity but is not recommended in infants and young children, to avoid trauma. An otoscope with halogen light used without a speculum is effective for visualizing the nares in infants and young children who are positioned on the exam table or on the parent's lap (Fig. 14.13).

FIGURE 14.13 View of the nasal vestibule and turbinates.

The vestibule of the nose should be assessed for any blockage by foreign body, polyps, nasal secretions, mucous plugs, or dried blood. The mucosal lining should be assessed for consistency of color, abrasions, lesions, and swelling. The color is normally deep pink, and a thin layer of clear mucus gives it a shiny appearance. The nasal septum should be examined for alignment, perforations, abrasions, bleeding, or crusting. It should be relatively straight and midline in the nose. Significant deviations of the septum may interfere with breathing. The turbinates should be assessed for color and swelling. Pale, swollen mucosa and edema of the turbinates is associated with allergic rhinitis and occlusion of air passage; and inflamed, reddened mucosa and turbinates are associated with respiratory infections. Children with chronic respiratory conditions may develop polyps that appear as shiny sacs extending into the nasal vestibule.

If a foreign body is suspected, attempt to have the child blow out the object while occluding the unaffected side. If this is not successful in dislodging the object, securely position the child on the exam table for removal of the object. A gentle probe with a curette or tweezers can be attempted to dislodge the foreign body. If unsuccessful and the object has become adhered to the mucosal wall or nasal septum, refer to a pediatric otolaryngologist for removal.

Sinuses

The maxillary and frontal sinuses can be assessed by physical examination beginning around 7 years of age through inspection and palpation. The facial area over the maxillary and frontal sinuses should be evaluated for swelling, erythema, and tenderness. Percuss with the forefingers or apply mild pressure with the thumbs over the maxillary and frontal sinus area. Evaluate pain, tenderness, and increased sensation in the frontal, ethmoid, or maxillary area, especially if there is a history of prolonged upper respiratory infection (Fig. 14.14). It may be difficult for young school-age children to accurately determine increased pain or tenderness caused by sinus inflammation, therefore, relying on accurate health history and symptom assessment is important in this age group. The diagnosis of sinusitis in children is generally made clinically. Imaging studies are not recommended unless children are unresponsive to medical therapy, develop complications of rhinosinusitis (RS), or who are being considered for surgical intervention.[2]

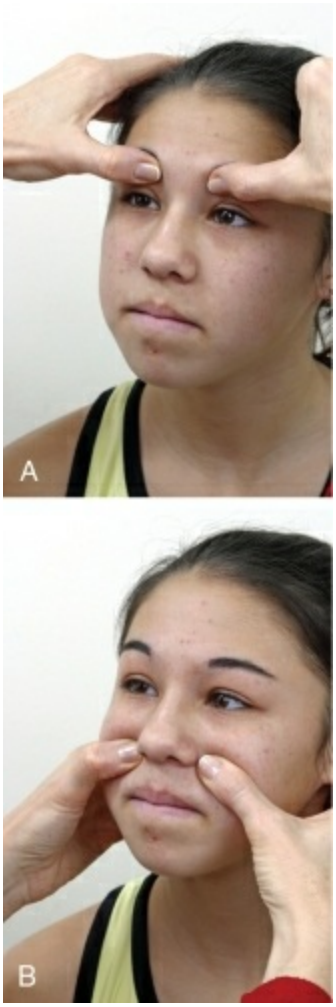


FIGURE 14.14 **(A)** Palpation of ethmoid sinuses. **(B)** Palpation of the maxillary sinuses.

Mouth

While inspecting the oral cavity, observe for the presence of any unusual odor or lesions. Inspect the lips for color, symmetry, lesions, swelling, dryness, and fissures. The color should be pink at rest and with feeding or crying. Note any asymmetry of movement or drooling that might indicate nerve impairment. Drooling during infancy from 3 to 15 months of age is normal, but drooling later may indicate nerve damage and loss of control of oral secretions. Young infants may have a callus or blister on the lip from vigorous sucking. This is particularly common in breastfed infants in the first few months of life (Fig. 14.15). Swelling of the lips may be caused by injury or allergic reaction. Cracked, dry lips can be caused by harsh weather conditions, repeated lip licking or biting, mouth

breathing due to nasal allergy, fever, or from illness or dehydration. Sores in the mouth or on the lips may indicate a viral infection such as *Coxsackie virus* or *herpes simplex virus* type 1. *Halitosis*, mouth odor or bad breath, in children may be caused by poor oral hygiene and dental caries, tonsillitis, or sinusitis. Note the frenulum of the upper lip that connects the upper lip to the gums of the maxilla at the point of the central incisors. It is prominent in the infant and disappears slowly in childhood with growth and development of the maxilla. Trauma to the upper lip and gum in the young child often includes trauma to the frenulum.

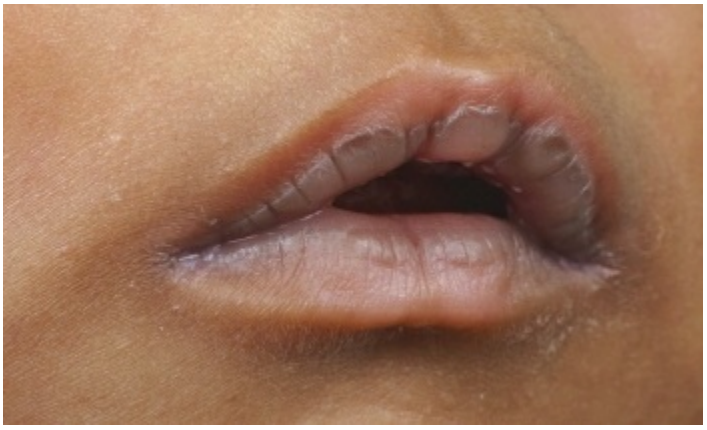


FIGURE 14.15 Sucking blister.

Inspect the buccal mucosa and gingivae with a tongue blade or tongue depressor for color, moisture, symmetry, and lesions (Fig. 14.16). The mucosa normally is shiny, smooth, and moist throughout. The oral mucosa may appear pale in children with darker pigmented skin and pink in children with lighter pigmented skin. Use a tongue depressor or a gloved finger to move the tongue and lips to ensure all surfaces of the mucosa are inspected. *Epstein pearls,* white pearly papules at the juncture of the hard and soft palates or on the anterior surface of the buccal mucosa, are common in newborns and resolve spontaneously. With a gloved finger, palpate unusual-looking areas for swelling and tenderness. If the mucosa of the gum appears inflamed or swollen, palpate for erupting teeth or hematomas. An *eruption cyst or eruption hematoma* is a bluish-purple, blister-like swelling on the gum, which may precede tooth eruption, particularly with the first and second

molars. Reddened, swollen, or friable gums can be an indication of poor oral hygiene, infection, or poor nutritional intake. Anticonvulsants may also cause hyperplasia of the gums.

FIGURE 14.16 Inspection of teeth and gums in an older child.

Candidiasis, appearing as bright white superficial lesions on the tongue and buccal mucosa of the cheeks, is often seen in the young breastfed infant or in the infant or child after use of oral antibiotics or with chronic infection (Fig. 14.17). The lesions of candidiasis can be differentiated from milk or formula residue by the bright white appearance. *Candidiasis lesions* do not scrape off the oral mucosa with the tongue depressor. *Petechiae,* pinpoint erythematous lesions, may be present on the soft palate with streptococcal infections or may be indicative of a bleeding disorder.

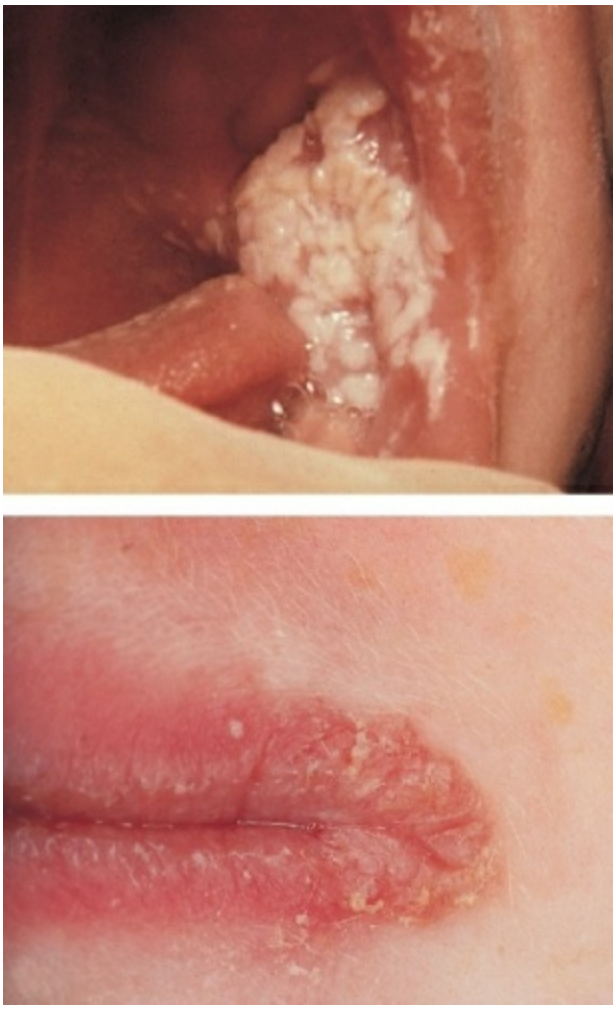


FIGURE 14.17 Candidiasis. Source: (From Zitelli BJ, McIntire SC, Norwalk AJ: *Atlas of Pediatric Physical Diagnosis,* ed 6, Philadelphia, 2013, Elsevier.)

Teeth

Inspect and note the number, color, size, and shape of the primary and permanent teeth and the pattern of eruption (Fig. 14.18). *Natal teeth* are prematurely erupted primary teeth that are present at birth. Natal teeth rarely occur in Caucasian infants, but they are a common variant in Native American infants.[7] The incidence of natal teeth is approximately 1 in 2000 births and is often seen in infants with cleft palate and other chromosomal deletion syndromes.[8] *Neonatal teeth* erupt in the first month of life, and 90% of neonatal teeth are lower primary teeth, or *mandibular incisors*.[7] Both natal and neonatal teeth are often immature caps of enamel and dentine with poorly formed roots and are unstable in the gum or attached to the

gum. Natal teeth can be a risk of aspiration in the neonatal period. If the teeth are supernumerary, very loose, or cause feeding problems, consultation with a pediatric dentist and extraction may be indicated.[7] Precocious eruption of primary teeth has been associated with precocious puberty. In older children with loose teeth, precaution with surgical procedures and anesthesia is indicated.

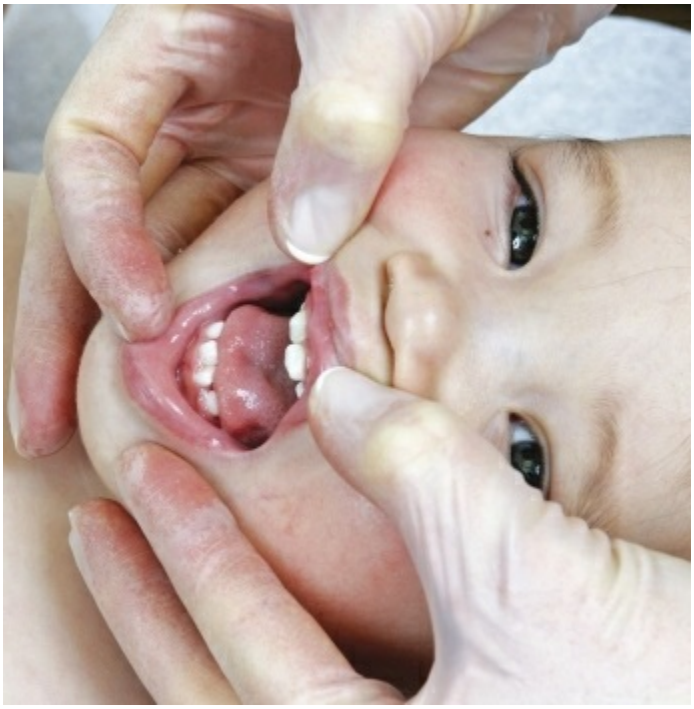


FIGURE 14.18 Inspection of primary teeth.

Inspection of the teeth in infants and young children includes identifying any presence of plaque on the teeth. Evaluate oral hygiene practices. Check the primary teeth in the infant and young child for *white spot* lesions, or decalcifications, and *brown spot* lesions, or cavitations, indicating the first sign of dental decay (Fig. 14.19). White spot lesions on the anterior tooth surfaces may be a sign of *early childhood caries.* They are caused by early transmission of the bacterium *S. mutans* from parent or caretaker to child and intake of frequent dietary sugars reducing the normal pH of oral secretions, resulting in demineralization of the tooth enamel. The mandibular incisors in infants are protected by the tongue when sucking, and they are therefore not prone to decay with prolonged

bottle-feeding or breastfeeding. Dental care should commence as soon as teeth erupt, and daily oral hygiene using a damp cloth or soft infant toothbrush to gently rub the gums and teeth of the infant can prevent plaque development. In older infants and young children, teeth should be brushed at least twice daily with fluoridated toothpaste under parental supervision.

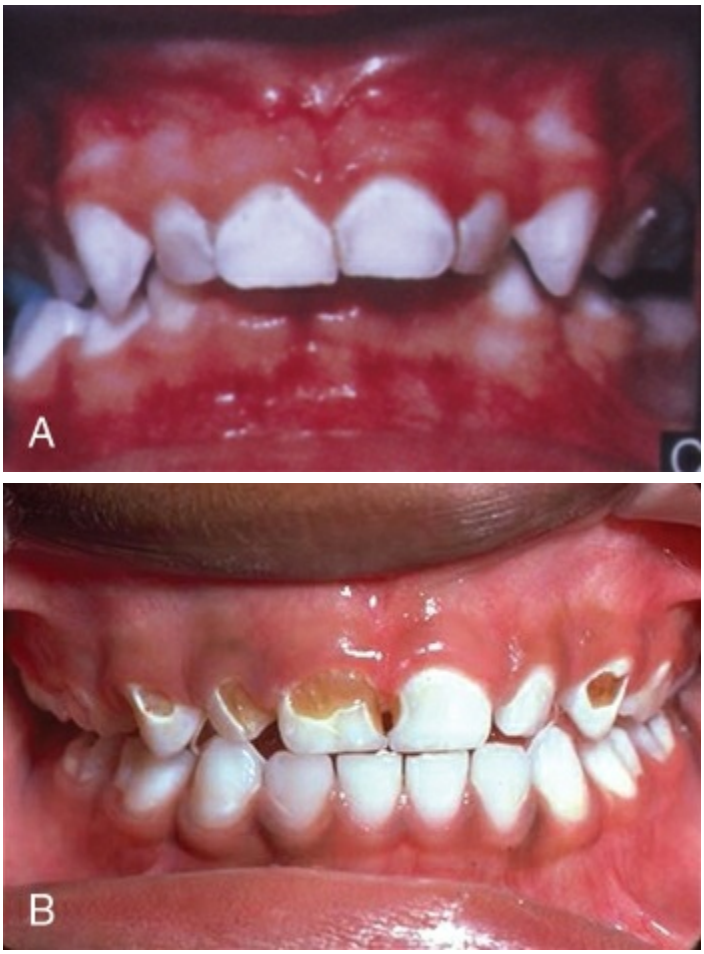


FIGURE 14.19 **(A)** White spot lesions. **(B)** Brown spot lesions. Source: (Courtesy Dr. Francisco Ramos-Gomez, University of California, Los Angeles.)

The American Academy of Pediatric Dentistry recommends establishing a dental home with a comprehensive dental assessment by 12 months of age.[9] Fluoridated drinking water and application of topical fluoride varnish by pediatric health care providers helps to reduce the incidence of early childhood caries.[9] Caries risk assessment in all children is an important component of the

assessment of the oral cavity. Box 14.1 assists health care providers in assessing children from 6 months to 3 years of age who are high risk for development of dental decay.[9] Access to a pediatric dental home and application of dental sealants also improves oral health outcomes and reduces the incidence of dental caries.

BOX 14.1

High Risk for Dental Caries 6 Months to 5 Years of Age

- Mother/primary care giver has active cavities
- Child has >3 between meal sugary snacks or sugary beverages
- Child is put to bed with bottle containing natural or added sugar
- Child with special health care needs
- Child from low-income or immigrant family

Adapted from American Academy of Pediatric Dentistry: Guidelines on caries-risk assessment and management for infants, children, and adolescents. *Pediatr Dent* 2015/2016;35(5):E157-E164.

Maxillary permanent incisors may erupt widely spaced and protruding outward, and mandibular incisors may erupt behind the primary incisors but align with normal development of the oral cavity unless there is a familial pattern of malocclusion or dental deformities. A slight overlap of the maxillary incisors to the mandibular incisors occurs with normal permanent dentition. Children with significantly misaligned teeth should be referred to a pediatric orthodontist for evaluation and treatment. *Bruxism,* or tooth grinding, which induces moderate wear on the surface of the canines and molars, may be noted on inspection. The peak incidence is during the developmental period of mixed dentition, and it rarely damages the dentition in young children but may be significant in older children or children with special health care needs. Dental hygiene in children with special health care needs is

often a challenge because of oral aversion, oral side effects of medications or special diets, and the child's ability/inability to participate in daily oral hygiene.

EVIDENCE-BASED PRACTICE TIP

Application of fluoride varnish by the pediatric health care provider to the primary teeth beginning in the first year of life is recommended to prevent dental caries. Two or more applications of fluoride varnish per year is effective in preventing caries in infants and children at high-risk for dental disease. [9]

Tongue

Inspect the tongue, noting color, size, and movement. The dorsal surface should appear slightly rough but moist and pink to pale pink. There may be variation in the papillae, giving the dorsal surface a patterned appearance. *Geographic tongue,* a benign inflammation of the dorsal surface of the tongue, causes pink areas with absent papillae and a surrounding whitish border (Fig. 14.20).

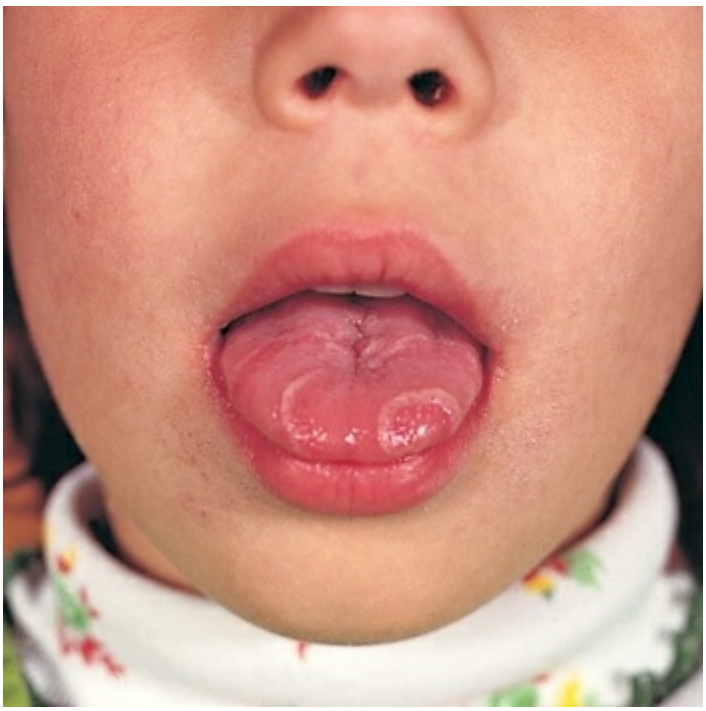


FIGURE 14.20 Geographic tongue. Source: (From Zitelli BJ,

McIntire SC, Norwalk AJ: *Atlas of Pediatric Physical Diagnosis,* ed 6, Philadelphia, 2013, Elsevier.)

The ventral surface appears thin with prominent vessels without hematomas. Connecting the ventral surface of the tongue to the floor of the mouth is the *lingual frenulum*. The lingual frenulum should allow movement of the tongue past the lips and to the roof of the palate. Movement of the tongue can be assessed through observation while an infant cries or a child vocalizes. Infants who are able to breastfeed or bottle-feed without difficulty have adequate movement of the tongue, and no further assessment of *cranial nerve XII (hypoglossal)* is needed. Newborns with significant feeding problems should be referred to an otolaryngologist and an occupational therapist trained and experienced in neonatal feeding problems. A significantly shortened lingual frenulum, *ankyloglossia,* is caused by an anterior attachment of the frenulum to the tip of the tongue (Fig. 14.21). It may in some cases interfere with adequate latch and sucking in the newborn and impair the infant's ability to breastfeed. Surgical intervention may be indicated in some infants and children. A *frenulotomy* or *frenulectomy* is the surgical procedure in which the lingual frenulum is cut. This surgical procedure remains controversial, particularly in relation to later speech development.

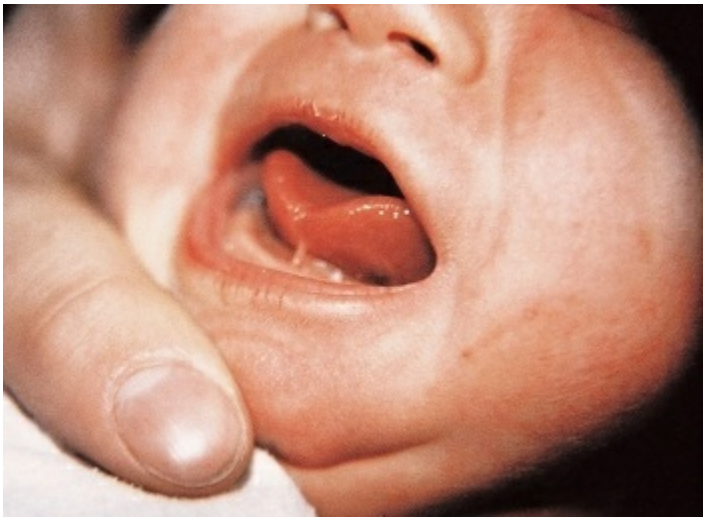


FIGURE 14.21 Ankyloglossia; short lingual frenulum. Source: (From Moore K: *The Developing Human: Clinically Oriented Embryology*, ed 8, Philadelphia, 2008, Elsevier. Courtesy Dr.

Macroglossia, enlarged tongue, can be congenital or acquired and is associated with hypothyroidism, Down syndrome, and other congenital anomalies. *Pierre Robin* syndrome is associated with a malpositioned tongue, feeding and breathing difficulty, and a high arched or cleft palate. A small jaw, or mandible, can be associated with congenital craniofacial anomalies, genetic conditions, or small for gestational age infant and may be associated with feeding problems, choking, or gagging.

In the older child and adolescent, ask the child to stick the tongue out past the lips and move the tongue from side to side to test *cranial nerve XII (hypoglossal).* These maneuvers should be easy to perform without fasciculation of the tongue. The ability to curl the tongue is a hereditary trait in some children. Any lesions, areas of tenderness, or swelling should be palpated to determine the size and depth.

Palate

Inspect the hard palate for patency or lesions. It should appear dome shaped but not deeply indented, lighter in appearance than the skin on the buccal mucosa and soft palate, and have transverse firm ridges. In a newborn infant with jaundice, the hard palate appears yellowish. In darker pigmented infants and children, it is helpful to inspect the hard palate and sclera to assess for jaundice. The hard palate is contiguous with the soft palate and extends to the anterior pillars and the uvula. The soft palate should appear intact and rise symmetrically along with the uvula when the child vocalizes or says "ah." This movement tests for *cranial nerve X (vagus).* Movement of the soft palate is necessary for the development of normal speech and articulation.

The hard and soft palates should always be palpated in the newborn to determine whether there is any submucosal cleft not visible on inspection or congenital anomalies associated with cleft palate. A gloved finger can be placed on the infant's palate to determine whether the palate is intact. As the infant sucks, evaluate the strength of the suck reflex and the palate surface. After the newborn period, palpation of the palate is not usually performed

unless lesions, swelling, or erythema is noted.

Tonsils

Inspect the palatine tonsils for size, color, exudates, pitting or enlarged crypts, or membranous covering. The tonsils are normally the color of the buccal mucosa or slightly lighter, should appear equal in size and position, and should be rated on a scale of 1+ to 4+ during well visits and during periods of illness to evaluate change (see Table 14.3). Tonsils that are larger than normal may indicate chronic respiratory allergies and if reddened indicate infection. White or yellowish exudate in the crypts of the tonsils is often associated with bacterial *tonsillitis* or *infectious mononucleosis* and requires further diagnosis and treatment if indicated. Unequal size tonsils and color may indicate a *peritonsillar abscess,* requiring further diagnostics, antibacterial therapy, and possible hospitalization. Pitting or enlarged crypts of the tonsils is often seen in children with a history of recurrent throat infections or chronic respiratory allergies (Fig. 14.22).

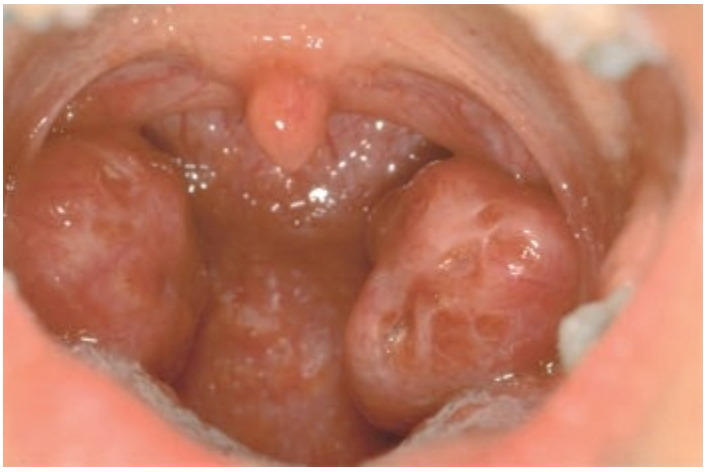


FIGURE 14.22 Large cryptic tonsils. Source: (From Lemmi FO, Lemmi CAE: *Physical Assessment Findings Multiuser CD-ROM,* St. Louis, 2000, Saunders.)

TABLE 14.3
Tonsillar Size

Size	Description
1+	Tonsils visible slightly beyond tonsillar pillars
2+	Tonsils visible midway between tonsillar pillars and uvula
3+	Tonsils nearly touching the uvula
4+	Tonsils touching at midline occluding the oropharynx

Some children and adolescents have chronically enlarged tonsils and adenoids that partially block air passage in and out of the oropharynx, requiring them to breathe with their mouth open to enlarge the air passageway. Dry, cracked lips are a hallmark sign of chronically enlarged tonsils in children. During sleep, relaxation of the pharyngeal musculature exacerbates the occlusion of the air passages, resulting in obstruction of airflow and *sleep-disordered breathing* and may result in periodic *sleep apnea. Sleep-related breathing disorders,* referring to the duration and quality of sleep, have been associated with nasal allergy, tonsillar hypertrophy related to allergies or recurrent viral or bacterial infections, and childhood obesity. They may impact school performance because of fatigue and inattention and have been associated with hyperactivity and behavioral problems.[10] The pediatric health care provider should obtain a health history regarding the child's sleep, sleep pattern, and the occurrence of snoring. Referral of the child to pediatric pulmonology to determine the need for a sleep study and follow up with a pediatric otolaryngology specialist to evaluate the oropharynx is often warranted. Adenotonsillectomy is one of the most commonly performed surgical procedures in children. The two leading indications for adenotonsillectomy are sleep-disordered breathing and recurrent throat infections.[10] Obstruction of the airway, rather than infection, has become a primary indication for tonsillectomy or adenotonsillectomy in younger children. Infection becomes a more prominent indication as age increases.[4]

Vocalization

Vocalization and speech patterns in infants and children should also be assessed. A high-pitched cry in the newborn or young infant may indicate increased intracranial pressure, and a hoarse cry in infants with upper respiratory infection may indicate *croup* or

laryngitis. Hoarseness in the neonatal period may be associated with congenital anomalies or a laryngeal hemangioma. In children, prolonged hoarseness should be investigated to determine the etiology that includes acute or chronic infection, benign lesions of the vocal cord folds, gastroesophageal reflux disease, and psychogenic causes. Prolonged unintelligible speech may indicate a speech articulation problem, expressive language delay, cognitive delay, or a hearing problem and should be promptly evaluated and referred for early intervention. Intelligible speech is critical for early success in school, and evaluation of speech and language delay and initiating speech therapy should not be delayed in hopes that the child will outgrow the problem. See Chapter 3 for further discussion on important developmental milestones for speech and language development and Chapter 13 for hearing assessment.

Nose, mouth, and throat conditions

Table 14.4 presents abnormal infectious conditions of the mouth and throat in infants, children, and adolescents.

TABLE 14.4

Conditions of the Mouth and Throat

Condition	Descriptions
Aphthous ulcers	Round to oval lesions with an erythematous halo appearing on buccal mucosa of cheeks, also known as "canker sores." May be painful and associated with fever and pharyngitis. Often resolve spontaneously within 1 week.
Epiglottitis	Edema and inflammation of epiglottis resulting in occlusion of trachea and acute respiratory distress; a medical emergency that may require intubation and radiographs for confirmation of diagnosis; avoid exam of oropharynx; incidence has decreased 80%–90% in children due to vaccine coverage with *Haemophilus influenzae* vaccine.
Gingivostomatitis	Vesicular lesions of lips, tongue, gingivae, oral mucosa resulting in swollen, painful, friable gums; common between 6 months and 3 years of age preceded by fever and irritability. Most often associated with herpes simplex type 1 or post coxsackievirus. Treatment focused on symptom management and prevention of dehydration.
Herpangina (coxsackievirus Groups A and B)	Small vesicles on posterior pharynx, tonsils, soft palate that rupture to form ulcers; occurs in young children with onset of sore throat, fever, malaise. Caused by coxsackievirus Group A. Treatment focused on symptom management and prevention of dehydration.

Mononucleosis	Enlarged tonsils, general malaise, fatigue, lymphadenopathy, splenomegaly usually caused by Epstein-Barr virus (EBV) but can be caused by other organisms such as cytomegalovirus (CMV); confirmed by EBV antibody testing.
Mucoceles	Fluid-filled cavities extending from mucous glands lining the epithelium of oral mucosa. May be associated with mild oral trauma frequently on lower lip. Spontaneous rupture usually occurs with resolution of lesion.
Streptococcal pharyngitis	GABHS pharyngotonsillitis is acute in onset and characterized by high fever, palpable cervical lymphadenopathy, sore throat, headache, and abdominal pain. Positive rapid streptococcal antigen test (RAST) indicates necessity to treat with penicillin or amoxicillin as preferred antibiotic to prevent sequelae.

GABHS, Group A beta-hemolytic streptococci.

Rhinosinusitis (RS) is one of the most prevalent diseases in childhood. Children average 6 to 8 upper respiratory tract infections per year, and the vast majority are caused by viruses with only 0.5% to 5% progressing to acute sinusitis.[2] Symptoms include nasal congestion, nasal discharge that can be clear, mucoid, thick or thin, low-grade fever, irritability, cough, halitosis, and rarely headache, a cardinal sign of sinusitis in adults. Acute bacterial RS should be suspected when symptoms persist beyond 10 days or if there is purulent rhinorrhea for 3 to 4 consecutive days with fever of 39°C or above. Since RS usually has a viral origin, no antibiotics should be prescribed, and rarely are nasal decongestants indicated. There is no consensus on management of acute bacterial RS in children as compared to adults, with limited indications for use of antibiotics.

Group A beta-hemolytic streptococci (GABHS) is the most common bacteria associated with pharyngotonsillitis in children and the only "sore throat" treated with antibiotics. "Strep throat," the common term used for GABHS pharyngitis, peaks in winter and spring, and transmission occurs through spread of droplets. Symptom history may reveal a household contact with similar symptoms or a diagnosed strep throat. Children and adolescents with prior history of GABHS are more susceptible to repeat infection. Signs and symptoms of GABHS pharyngitis are acute in onset and characterized by high fever, palpable cervical lymphadenopathy, sore throat, headache, and abdominal pain, sometimes with nausea and vomiting. Pharyngeal and tonsillar

mucosa are typically erythematous with exudate present in 50% to 90% of cases.[2] A rapid antigen detection test (RADT) with confirmative throat culture is necessary to accurately diagnose GABHS.

Although symptoms will resolve within a few days without treatment, early treatment with penicillin (preferred) or amoxicillin, cephalosporins, macrolides, or clindamycin has effectively eradicated the infection and are presumed to prevent sequelae, including rheumatic fever. The incidence of rheumatic carditis is 0.3% in endemic environments. Acute glomerulonephritis is a sequela of a specific nephritogenic strain of GABHS, and 10% to 15% of people infected with this strain of GABHS will develop acute glomerulonephritis regardless of treatment.[2]

Summary of examination

- In the infant, inspect for patency of nares and note any flaring or narrowing of the nares with breathing.
- An otoscope with halogen light is often used without a speculum to externally visualize the nares in infants and young children.
- Maxillary and ethmoid sinuses are present at birth but are small; the frontal sinuses begin to develop by 7 years of age and the sphenoid sinuses develop in adolescence.
- Percuss with the forefingers or apply mild pressure with the thumbs over the maxillary and frontal sinus area to evaluate tenderness.
- The cheeks and lips are innervated by cranial nerves V (trigeminal) and VII (facial).
- Inspect the lips for color, symmetry, lesions, swelling, dryness, and fissures.
- Delay the examination of the mouth and nose in the infant and young child until after the "quiet" parts of the exam and after the ear examination.
- Inspect the buccal mucosa and gingivae with a tongue depressor for color, moisture, symmetry, and lesions.
- Inspect for tonsillar size and quality. Tonsillar size is graded on a scale of 1+ to 4+.

- Tooth *eruption* normally occurs between 4 and 12 months of age for the first tooth, and eruption of the 20 primary teeth should be complete between 24 and 30 months of age.
- Check the primary teeth in the infant and young child for "white spot" lesions, or decalcifications, and "brown spot" lesions, or cavitations, indicating the first sign of dental decay

DOCUMENTATION

Healthy newborn

Nose, mouth, and throat: Nares patent bilaterally without flaring, clear nasal discharge. Strong suck. Mucous membranes pink, moist without lesions. Soft and hard palate intact. Uvula and tongue midline, nonprotuberant, gag response intact, without natal teeth.

DOCUMENTATION

Adolescent

Nose, mouth, and throat: No nasal discharge, nasal septum midline, turbinates pink, moist. No facial swelling or tenderness over sinuses. Buccal mucous pink and moist without lesions. Gums pink, firm without bleeding. Thirty-two teeth present in good repair without evidence of active decay. Pharynx pink, tonsils 1+ without exudate or pitting, uvula midline, sensitive gag response.

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*CHARGE syndrome associated anomalies-coloboma, congenital heart disease, choanal atresia, growth retardation and cognitive delays, genital hypoplasia, and ear deformities.

CHAPTER 15

Abdomen and rectum

Victoria F. Keeton

Assessment of the abdomen and rectum involves the evaluation of multiple organ systems and functions, including the gastrointestinal, renal, vascular, endocrine, immune, and female reproductive systems. The health care provider should always maintain a holistic view of the child, adolescent, and family during the assessment, which may help distinguish clinical symptoms from psychosomatic complaints in the pediatric population and help focus the abdominal examination.

Embryologic development

The primitive gut forms during the fourth week of gestation from the dorsal section of the yolk sac. It begins as a hollow tube arising from the endoderm, which then forms the *foregut, midgut,* and *hindgut.* The *foregut* develops into the esophagus, stomach, upper portion of the duodenum (bile duct entrance), liver, biliary system, and pancreas. It is perfused by the celiac artery. The *midgut* develops into the distal duodenum and the remainder of the small intestine, cecum, appendix, ascending colon, and most of the proximal portion of the transverse colon; it is perfused by the superior mesenteric artery. The hindgut develops into the remaining transverse colon, descending colon, sigmoid colon, rectum, and superior portion of the anal canal; it is perfused by the inferior mesenteric artery.

By the end of the sixth week of gestation, the gut herniates

outside of the abdominal cavity, where it rotates 90 degrees counterclockwise and continues to elongate. By the 10th week of gestation, the gut returns to the abdominal cavity and rotates another 180 degrees counterclockwise. With the normal intestinal rotation, the stomach and pancreas rotate into the left upper quadrant (LUQ) and are pressed against the dorsal abdominal wall to fuse into position.

The pancreas arises from ectodermal cells from the most caudal part of the foregut and develops into dorsal and ventral buds. The dorsal bud is larger and becomes the major portion of the pancreas. The dorsal and ventral buds fuse to form the main pancreatic duct. Secretion of insulin begins around the 20th week of gestation. Up until the 14th week, the spleen is only a hematopoietic organ. Between weeks 15 and 18, the spleen then loses its hematopoietic function and transforms into an organ of the immune system.

The liver begins as a bud that develops on the distal part of the foregut and grows into the *septum transversum,* where it divides into two parts. The larger part develops into the right and left lobes of the liver, and the second smaller division of the hepatic bud develops into the biliary system. Hematopoiesis begins at the sixth week of gestation and is responsible for the large size of the liver. It is approximately 10% of the total weight of the fetus. Bile begins to form at 16 weeks of gestation, giving meconium its dark green color.

Development of the kidney begins with a primitive, transitory structure called the *pronephros,* or forekidney, which arises near the segments of the spinal cord. These segments appear early in the fourth week of gestation on either side of the nephrogenic cord. The pronephros itself soon degenerates but leaves behind its ducts for the next kidney formation, the *mesonephros,* or midkidney, to utilize. In the fifth week, the *metanephros,* or hindkidney, begins to develop and becomes the permanent kidney. By the eighth week, the hindkidney begins to produce urine and continues to do so throughout the fetal period.

The adrenal glands develop from the medulla, which originates from the neuroectoderm. At the seventh week of gestation, the medulla attaches to the fetal cortex, which develops from the mesoderm, and by the eighth week, the fetal cortex begins to

encapsulate the medulla. The fetal adrenal gland is 20 times larger than the adult adrenal and is large compared with the kidneys. However, the adrenals rapidly decrease in size as the fetal cortex regresses and completely disappear by 4 years of age, when they are replaced by the adult cortex.

Anatomy and physiology

The abdomen is the area of the torso from the diaphragm to the pelvic floor and is a three-dimensional space within which many organs overlap one another (Fig. 15.1). It is lined by the *peritoneum*, a serous membrane covering the abdominal viscera. The membrane of the peritoneum creates a smooth, moist surface that allows the abdominal viscera to glide freely within the confines of the abdominal wall and also provides protection.

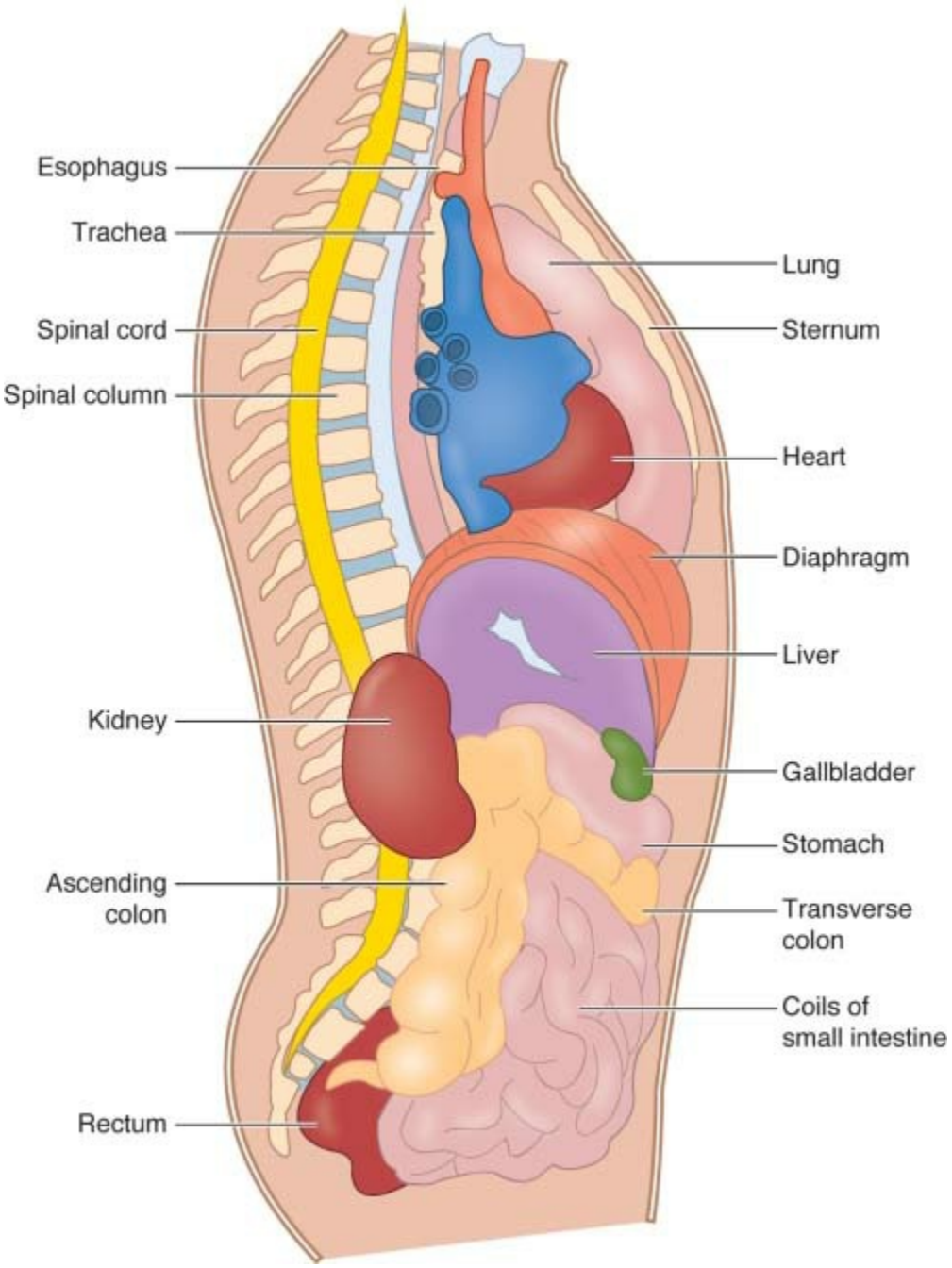


FIGURE 15.1 Side view of the torso and abdomen.

The *liver* lies immediately below the right diaphragm and is the largest and heaviest organ in the body. It is composed of the right and left hepatic lobes and is an extremely vascular organ. The liver is perfused by the hepatic artery, which arises from the *abdominal aorta*, and by the portal vein, which delivers blood from the spleen, pancreas, and intestines. The liver is responsible for metabolizing carbohydrates, fats, and proteins. It also breaks down toxic substances and drugs; stores vitamins and iron; produces

antibodies, bile, prothrombin, and fibrinogen for coagulation; and excretes waste products. The hepatic veins return blood to the vena cava. Within the inferior surface of the liver lies the *gallbladder,* a sac-like organ. The liver excretes bile into the hepatic duct, which is collected and stored in the gallbladder. Bile is secreted into the duodenum via the cystic duct and the common bile duct to aid in the digestion of fats.

Below the left diaphragm, from posterior to anterior, respectively, lie the spleen, pancreas, and stomach. The *spleen* is a concave organ made mostly of lymphoid tissue that lies around the posterior fundus of the stomach. The spleen filters and breaks down red blood cells and produces white blood cells (lymphocytes and monocytes). It also stores blood, which can be released into the vascular system during an acute blood loss. The *pancreas* is nestled between the spleen and stomach and crosses the midline over the major vessels. The pancreatic head extends to the duodenum and the tail reaches almost to the spleen. It is responsible for producing enzymes needed for the metabolism of proteins, fats, and carbohydrates; these enzymes are excreted into the duodenum via the pancreatic duct and aid digestion. The pancreas also produces insulin and glucagon, which are secreted directly into the bloodstream, helping regulate blood glucose levels and endocrine function. The *stomach* is the most anterior organ in the LUQ of the abdomen. It is connected proximally to the esophagus, which enters through the diaphragm at the *esophageal hiatus.* The stomach receives food from the esophagus through the lower esophageal sphincter. It secretes hydrochloric acid and digestive enzymes, which are used to metabolize proteins and fats. When the stomach is distended, it is stimulated to contract and expel its contents through the pyloric sphincter into the *duodenum,* the first portion of the small intestine.

The *duodenum* is C-shaped and curls around the head of the pancreas. The pancreatic and bile ducts empty into the upper portion of the duodenum. The duodenum transitions to the *jejunum,* which is responsible for the majority of the absorption of water, proteins, carbohydrates, and vitamins. The *ileum* composes the last and longest part of the small intestine and absorbs bile salts, vitamins C and B_{12}, and chloride. Intestinal contents leave the ileum

through the *ileocecal valve* and empty into the *cecum,* located in the right lower quadrant of the abdomen, which is the beginning of the large intestine (Fig. 15.2). The *appendix,* a long, narrow, tubular structure, arises from the base of the cecum. The large intestine lies anteriorly over the small intestine, ascends along the right anterior abdominal wall, and forms the *ascending colon*; it traverses the abdomen to the splenic flexure, forming the *transverse colon,* and descends along the left lateral abdomen wall as the *descending colon*. At the level of the iliac crest, the colon becomes the S-shaped *sigmoid* colon. It descends into the pelvic cavity and turns medially to form a loop at the level of the midsacrum. The sigmoid colon connects to the *rectum,* which lies behind the bladder in males and the uterus in females. It stores feces until it is expelled through the *anal canal* and out the *anus,* which is located within a ring of nerves and muscle fibers midway between the tip of the *coccyx* and the *scrotum* or *vaginal fourchette*. The anal canal and anus remain closed involuntarily by way of a ring of smooth muscle, the *internal anal sphincter,* and voluntarily by a ring of skeletal muscle, the *external anal sphincter.*

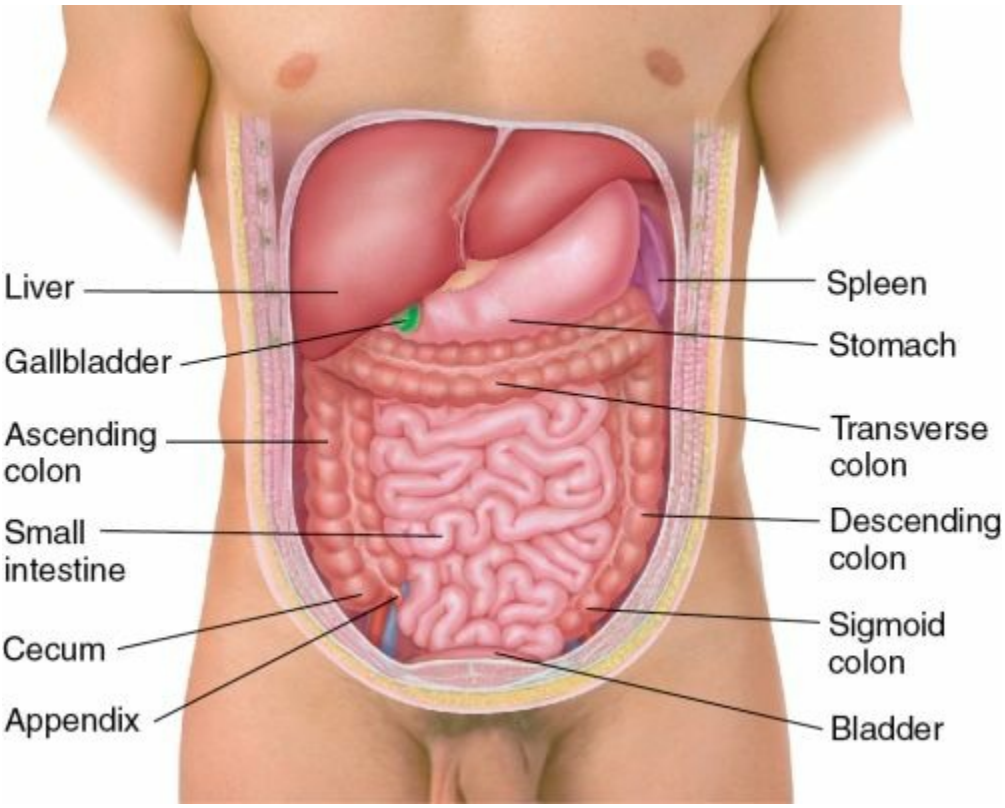


FIGURE 15.2 Anatomic structures of the abdominal cavity in an adolescent male. Source: (From Seidel HM, Ball JW, Dains JE, et al: *Mosby's Guide to Physical Examination,* ed 7, St. Louis, 2011, Mosby.)

The *kidneys* lie on either side of the vertebral column in the retroperitoneal space below the liver and spleen. The right kidney tends to be lower than the left because it lies below the right lobe of the liver. Kidneys have a lobulated appearance at birth, which disappears with the development of the glomeruli and tubules in the first year of life. The kidneys are perfused by the renal arteries and filter and reabsorb water, electrolytes, glucose, and some proteins. They regulate blood pressure, electrolytes, and the acid-base composition of blood and other body fluids; actively excrete metabolic waste products; and produce urine. The kidneys are capped by the adrenal glands, pyramid-shaped organs that synthesize, store, and secrete epinephrine and norepinephrine in response to stress. The adrenals also produce the corticosteroids, which affect glucose metabolism, electrolyte and fluid balance, and immune system function.

Urine is excreted from the kidney into the *ureters,* long, thin muscular tubules that transport urine to the bladder. The *ureters* connect to the superior pole of the renal pelvis. They descend posteriorly to the peritoneum and slightly medially in front of the psoas major muscle into the pelvic cavity, implanting into the superior posterior wall of the *urinary bladder.* The oblique insertion of the ureters through the bladder wall creates a one-way valvular mechanism that prevents the reflux of urine. The urinary bladder lies anterior to the uterus in females and anterior to the rectum in males. When filled to its capacity, the bladder then contracts and releases urine through the bladder neck and out the *urethra.* The urethra is normally located at the tip of the penis in males and between the clitoris and vagina in females. In nonpregnant females, the reproductive organs lie within the pelvis between the bladder (anterior pelvis) and the rectum (posterior pelvis). They include the *ovaries, uterine* or *fallopian tubes,* and *uterus.* These organs descend into the pelvic cavity during normal growth and development and ascend into the abdominal cavity during pregnancy or with ovarian cysts or other abnormalities of the female reproductive system. See

Chapter 18 for further discussion of the female genitourinary system.

Finally, a layer of fascia and then muscle covers the anterior abdomen. The *rectus abdominis* muscle extends the entire length of the front of the abdomen and is separated by the *linea alba* in the midline. The *transverse abdominis* and *internal* and *external oblique muscles* cover the lateral abdomen. The *umbilicus* lies in the midline usually below the midpoint of the abdomen (Fig. 15.3).

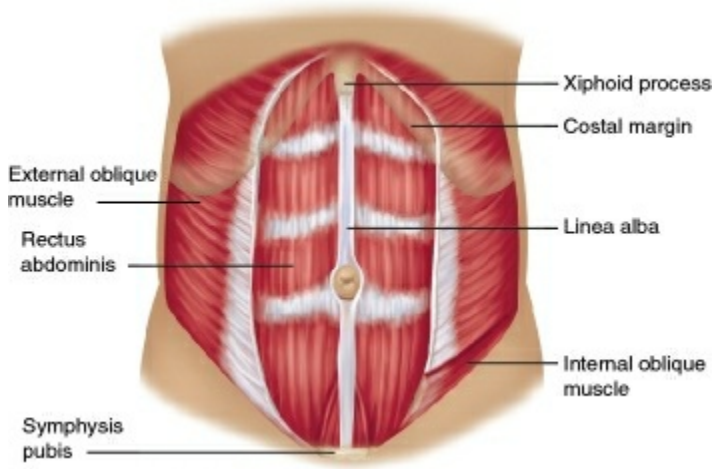


FIGURE 15.3 Abdominal musculature in adolescent.

Physiologic variations

Many aspects of the development of the organs and structures in the abdomen continue into the first few years of life. The muscle tone of the lower esophageal sphincter is not fully developed until 1 month of age and may remain slightly weak for the first year. The stomach is round until approximately 2 years of age and then elongates into its adult shape and position by about 7 years of age. Stomach capacity is also smaller in the infant, whereas emptying time is faster, which results in the pattern of small and frequent feedings. The small bowel grows from approximately 270 cm at birth to up to 550 cm by 4 years of age. The liver constitutes 5% of the term neonate's body weight, versus 2% in the adult. The kidneys also remain relatively large for the size of the abdomen until adolescence.

System-specific history

Information gathering for the assessment of the abdomen should include questions regarding diet, elimination, medications, environmental exposures, and a thorough psychosocial history (see Information Gathering table). Evaluation of abdominal symptoms that may be related to the genitourinary tract are discussed in more depth in Chapters 16 and 18. An assessment of the menstrual cycle in the female and history of sexual activity in adolescents is also essential.

Information Gathering at Key Developmental Stages

Age Group	Topics to Address
Preterm and newborn	Maternal drug use, infection, family history of GI conditions Birth weight and gestational age First meconium and stooling/voiding patterns Amount and type of feedings Spitting up or vomiting Jaundice Any abnormal prenatal ultrasound findings, results of amniocentesis, genetic workup (polyhydramnios, gastric bubble, intestine location, hydronephrosis)
Infancy	Weight gain and growth pattern Amount and type of feedings Food allergies Stooling/voiding patterns Spitting up or vomiting Family history for chronic constipation
Early childhood	Weight gain and growth pattern, including BMI Diet history Ingestion of nonnutritive substances (pica) Presence and patterns of abdominal pain Stooling/voiding patterns, including Toilet training Constipation or stool withholding Enuresis or encopresis Blood or mucus in stool Symptoms of UTI (abdominal pain, dysuria, enuresis) Recent illnesses Psychosocial stressors including transition to child care or preschool and any family dysfunction
Middle childhood	Weight gain and growth pattern, including BMI Diet history Presence and patterns of abdominal pain Stooling/voiding patterns, including Constipation or stool withholding Enuresis or encopresis

	Blood or mucus in stool Symptoms of UTI (frequency, dysuria, urgency) Recent illnesses Psychosocial stressors including bullying and any family dysfunction
Adolescence	Weight gain and growth pattern, including BMI Diet history and fluid intake Body image and risk for disordered eating/purging Presence and patterns of abdominal pain Stooling and voiding patterns Symptoms or history of UTI Menstrual history (females) Sexual activity and use of contraception and barrier methods Symptoms or history of STI Psychosocial stressors including bullying and intimate partner violence
Environmental risks (all ages)	Home, child-care or school exposures to infectious disease Family members with chronic abdominal pain or diarrhea Cultural practices for feeding or complementary/alternative healing Recent travel to areas with endemic food- or water-borne illness Recent backpacking or camping Exposure to contaminated foods Environmental lead exposure Low socioeconomic status Parental mental health illness Sibling with chronic disease/health of family members

BMI, Body mass index; *GI*, gastrointestinal; *STI*, sexually transmitted infection; *UTI*, urinary tract infection.

Abdominal pain

When a complaint of abdominal pain is reported, the examiner must elicit a detailed history of the pain. Information regarding the character and severity of the pain, onset and duration, location or radiation, position of comfort, things that alleviate or worsen the pain, history of trauma, and any associated symptoms of fever, vomiting, anorexia, constipation, diarrhea, or frequent stooling with or without noting blood is important in narrowing the scope of the differential diagnosis. A complaint of recurrent pain related to meals or after meals should be elicited in the history. A detailed history can help in determining whether the abdominal pain is acute or chronic. Remember that abdominal pain can be referred from an extra-abdominal source or can be a condition associated with systemic disease. For example, abdominal pain is common in children with beta-streptococcal pharyngitis, lower lobe pneumonia, sickle cell anemia, cystic fibrosis, Henoch-Schönlein purpura, and many other conditions. See Box 15.2 for a differential diagnosis of symptoms or conditions related to different abdominal

regions.

EVIDENCE-BASED PRACTICE TIP

When a child presents with chronic abdominal pain, a primary goal is to determine whether the pain has a functional or organic cause to create an effective management plan. However, confirming the presence or absence of an organic disease is not enough to determine the degree of impairment a child may experience related to chronic abdominal pain. Evidence shows that children with functional abdominal pain (FAP) experience a significant decrease in quality of life and are more prone to impairment in social, psychological, and academic functioning. [1] Therefore it is imperative to include an evaluation of functional impairment for any child with chronic abdominal pain.

Red Flag In Abdominal Assessment

Nocturnal awakening with pain and/or stooling may be a red flag in the school-age child and requires further assessment and diagnostic workup as indicated.

Physical assessment

Equipment

In performing an abdominal exam in an infant or child, good lighting, warm hands, and a stethoscope may be all the equipment necessary. Diagnostic imaging can play an important complementary role in the assessment of some abdominal complaints, but it is essential that the decision to use such resources be warranted and evidence-based.[2] A proper physical examination by the health care provider remains essential to determining the best course of action and the level of acuity of the presenting condition. If the assessment reveals abdominal pain accompanied

by any red flags (Box 15.1), further evaluation through laboratory analysis and/or diagnostic imaging may be warranted.[1-3] See later section, Diagnostic Procedures, for further discussion.

BOX 15.1

Red Flag Symptoms and Signs in Children with Recurrent Abdominal Pain

Symptoms and key features in the history

Involuntary weight loss
Chronic severe diarrhea
Gastrointestinal blood loss
Gynecologic symptoms
Family history of IBD/celiac disease
Nighttime waking
Significant vomiting (especially if bilious)
Urinary symptoms
Joint pains, mouth sores, skin rash, unexplained fevers—extraintestinal manifestations of IBD

Red flag signs of gastrointestinal disease

Slowing of linear growth
Clubbing
Mouth ulcers
Abdominal masses
Pain radiating through to the back (pancreatitis) or loins (renal pain)
Anorexia/delayed puberty
Hypertension/tachycardia
Perineal changes (tags/fistulas)

IBD, Inflammatory bowel syndrome.
Data from Brown LK, Beattie RM, Tighe MP: Practical management of functional abdominal pain in children. *Archiv Dis Child* 101:677-683, 2016.

Preparation and positioning

Infants and toddlers can initially sit comfortably on the parent's lap directly facing the examiner. Inspection and auscultation of the abdomen can be done with the child sitting upright. For light and deep palpation, the examiner should be seated in a knee-to-knee position with the parent and with the child lying with head and torso in the parent's lap and the hips and legs in the examiner's lap (Fig. 15.4). The initial examination can occur with the child partially dressed and the diaper can be unfastened or pants and underwear pulled below the groin area. For older children and adolescents, the abdomen should be assessed on the examination table. It is important for the examiner to consistently place the older child or adolescent in the same position when preparing for the abdominal examination—ideally with the head on the examiner's left and the right side of the child's body in front of the examiner. This will aid in accurate anatomic findings and accurate diagnosis of abdominal symptoms and conditions.

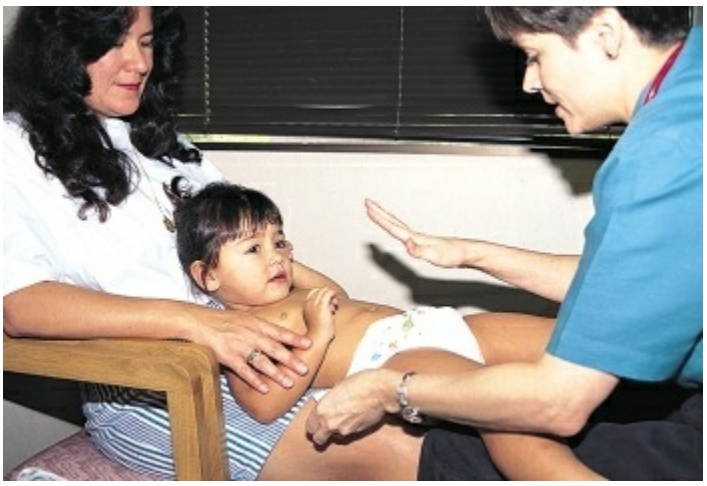


FIGURE 15.4 Knee-to-knee position for examining abdomen in infancy and early childhood. Source: (From James SR, Ashwill JW: *Nursing Care of Children,* ed 2, St. Louis, 2003, Saunders.)

The abdomen is divided into four equal quadrants, with the transverse and midsagittal planes intersecting at the umbilicus (Fig. 15.5A). For a child or adolescent who presents with abdominal pain or for other abdominal conditions, use a mapping technique with

nine sections to accurately describe findings and for purposes of charting in the electronic health record (see Fig. 15.5B). Mentally visualizing the anatomic location of the organs and adopting a mapping technique for abdominal assessment is key to an accurate and informative examination. There may be some variation in the anatomic positions of the organs in children and adolescents depending on the body type, respiratory phase at the time of the exam, the amount of contents within the stomach or bladder, and the amount of palpable stool in the abdomen.

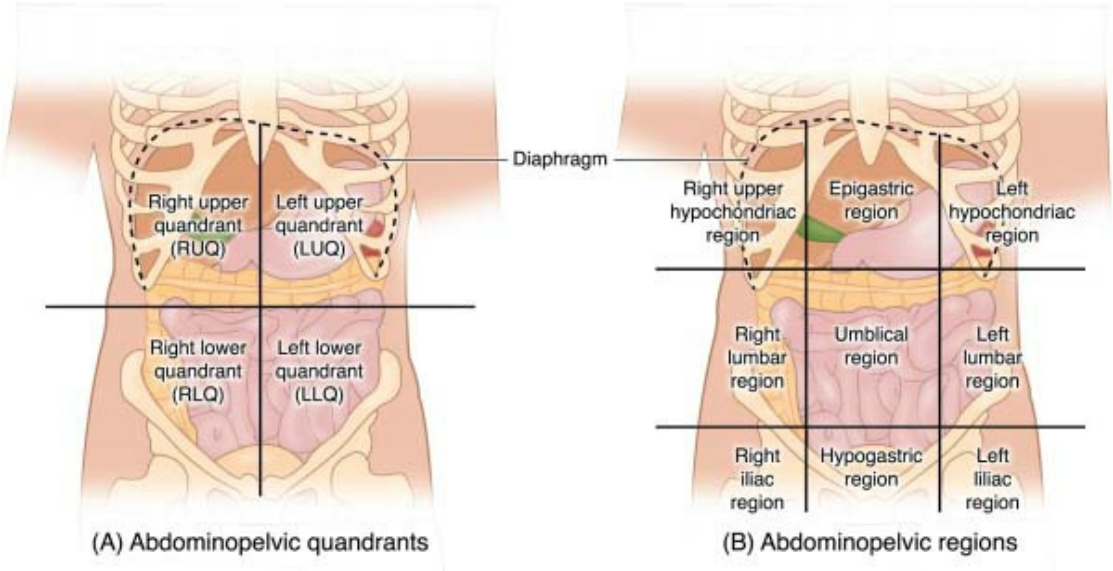


FIGURE 15.5 **(A)** Four abdominopelvic quadrants. **(B)** Nine abdominopelvic regions.

Inspection

Examination of the abdomen begins with the initial inspection of the child or adolescent's facial expression and color, attitude, activity level, and level of comfort to determine any distress. Observe whether the child has difficulty walking or climbing onto or off of the exam table. Children with peritoneal irritation often walk cautiously and resist lying on the exam table. Ask the child to hop or jump and note whether these movements elicit pain. If the child is guarding the abdomen, prefers to keep the legs flexed, or if the abdomen is rigid, this can indicate diffuse peritonitis.

Begin the abdominal assessment by extending each leg and note

if pain is elicited and its location. Inspect the abdomen by noting its contour, symmetry, skin texture, color, and integrity. Note any lesions, rashes, pigment variations, piercings, gastrostomy tubes, or scars. Scars can indicate previous abdominal surgery and should always be explored during history taking. View the abdomen from the side and note the shape and anteroposterior dimension. Infants and young children have less developed abdominal musculature, so the abdomen is more protuberant and round. Young children to 4 years of age will have a potbellied appearance while supine or standing (Fig. 15.6). If the abdomen is scaphoid at any age, it can indicate malnutrition or displaced abdominal organs, as with a diaphragmatic hernia or intestinal atresia in a newborn. If the abdominal contour is distended, it can indicate an intestinal obstruction, a mass, organomegaly, or ascites. Fullness over the symphysis pubis can be seen in a thin child with a full bladder. Asymmetry of the abdomen may indicate a mass, organomegaly, hollow organ distention, or curvature of the spine.

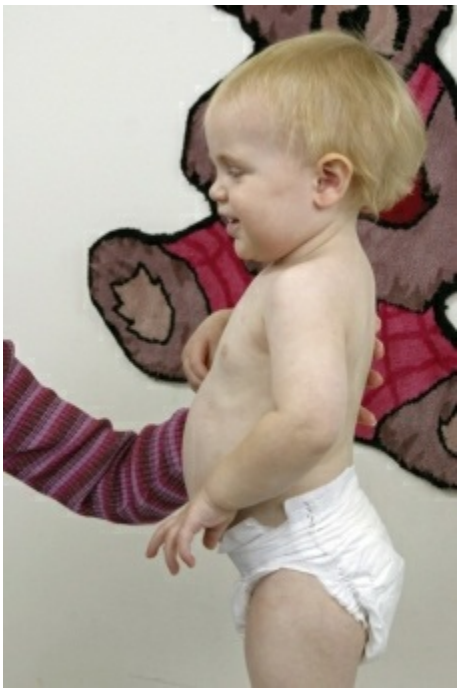


FIGURE 15.6 Potbelly stance of the toddler.

Observe for any pulsations. It is normal to see pulsations in the epigastric area of a young infant or very thin child. Distended veins in the abdominal integument could indicate vascular compression

or obstruction, hypertension, or intestinal obstruction. If an intestinal obstruction is suspected, note any obvious loops of bowel and observe for peristaltic waves on the surface by viewing the abdomen at eye level.

Inspect the umbilicus for any signs of drainage, infection, hernia, or mass. In the initial newborn exam, inspect the cord for the umbilical vessels, two arteries, and a single vein. The arteries are smaller and have a thicker vessel wall. Infants with a two-vessel cord may have congenital anomalies and should be referred for further diagnosis and evaluation. The majority of umbilical cord remnants detach by the 10th day of life but can take up to 3 weeks to slough. Once the cord has detached, the stump should dry and heal within a few days. Occasionally, *umbilical granulomas* or granular tissue at the base of the umbilicus can be present and drain serous or seropurulent fluid or occasionally blood (Fig. 15.7). For persistent umbilical granulomas, cauterization or surgical ligation of the stump may be needed. Any prolonged drainage should be investigated for presence of a *urachal remnant* or *cyst*. If stool is noted coming from the umbilicus, an *omphalomesenteric duct remnant* is present, and the infant should be referred immediately to a pediatric surgeon. Any sign of infection in the umbilicus should be aggressively treated in the neonate. *Neonatal omphalitis* is a rapidly progressing, acute, and potentially fatal infection of the abdominal wall caused by a bacterial pathogen. Any infant with purulent discharge or sign of cellulitis should be treated with systemic antibiotics and referred immediately if the infection progresses.

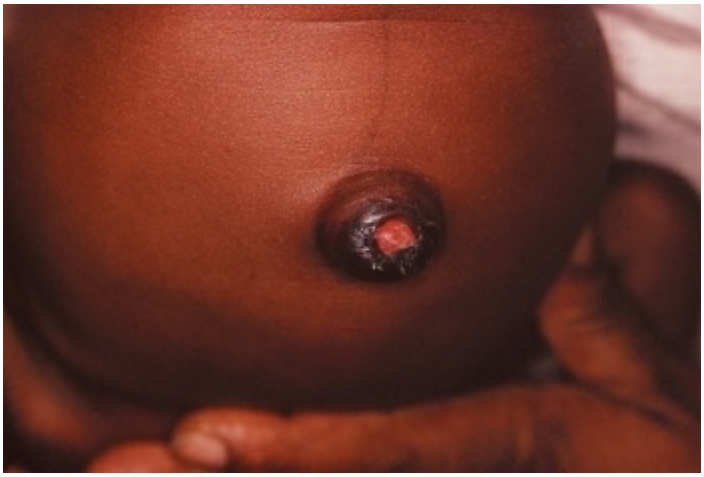


FIGURE 15.7 Umbilical granuloma. Source: (From Clark DA: *Atlas of Neonatology,* ed 7, Philadelphia, 2000, Saunders.)

Note any protrusion or mass in the umbilicus. Observe the midline of the abdomen of the infant or young child when he or she is reclining or sitting. A wide bulging superior to the umbilicus is likely *diastasis recti abdominis* (Fig. 15.8), a common finding in children when the rectus abdominis muscle does not meet in the midline. Diastasis recti abdominis does not create any functional problem during infancy and early childhood and usually diminishes or resolves as the child grows and the abdominal muscles strengthen.

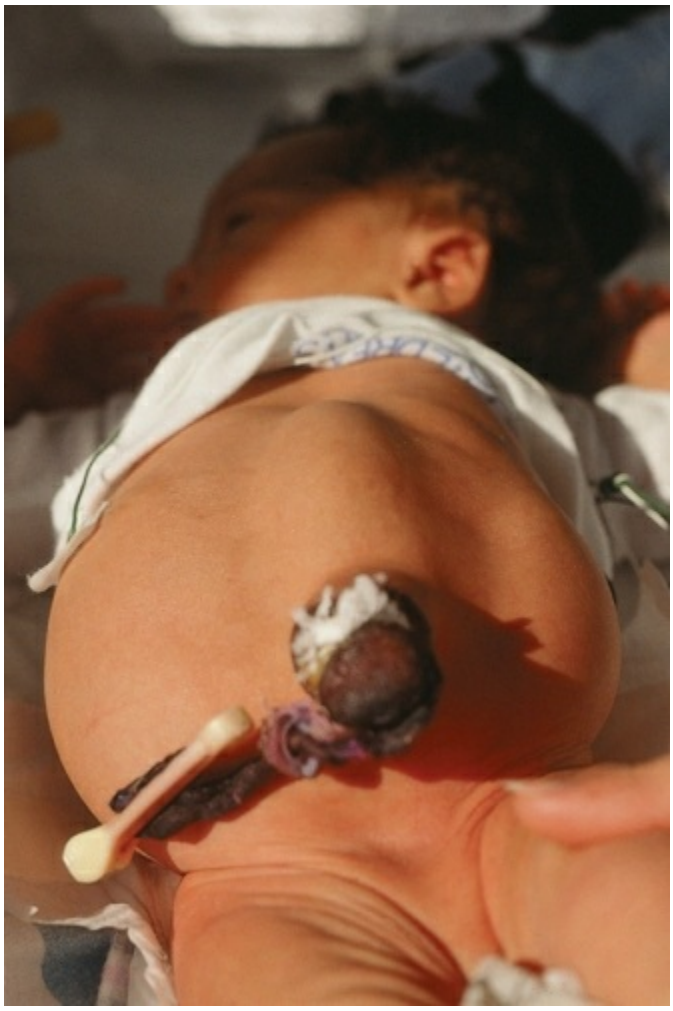


FIGURE 15.8 Diastasis recti. Source: (From Clark DA: *Atlas of Neonatology,* ed 7, Philadelphia, 2000, Saunders.)

Auscultation

Auscultation of the abdomen should be done before palpation or percussion so as to prevent any alteration in bowel sounds that may occur from manipulation of the area. Systematically listen to each quadrant or section of the abdomen with a stethoscope. In the infant and young child, auscultate the abdomen when you are completing the quiet parts of the physical exam (i.e., the cardiac and respiratory assessment). Bowel sounds can be heard within the first few hours of life and indicate peristalsis and movement of contents through the bowels. Normal bowel sounds are heard every 10 to 20 seconds, and frequency of bowel sounds may be approximately 5 to 30 in 1 minute. Hypoactive (more than 30 seconds apart) or absent bowel sounds (none heard within 3 to 5 minutes) may indicate a *paralytic ileus,* or inactivity of the intestines, as in constipation. Hyperactive bowel sounds indicate rapid movement through the intestines, usually associated with diarrhea or a mechanical obstruction. If the child is hypertensive, auscultate for bruits using the bell of the stethoscope to assess for signs of renovascular disease. A newborn with a *scaphoid* abdomen or signs of respiratory distress should be carefully evaluated for bowel sounds or decreased breath sounds in the chest, which may indicate a *diaphragmatic hernia.* During auscultation of the abdomen, the examiner can carefully watch the child's facial expression to identify areas of tenderness while lightly pressing the stethoscope against each section of the abdomen.

Palpation

Palpation is the most important technique in assessing the abdomen and identifies areas of tenderness, masses, organomegaly, ascites, and signs of inflammation or peritonitis. It should not be omitted even with a child who is having difficulty cooperating with the exam. The ideal position for palpation of the abdomen is to have the child lie supine with the knees and hips gently flexed to relax the abdominal wall.

Begin with light palpation using the pads of the forefingers to assess the four quadrants of the abdomen. Use a firm therapeutic touch for palpation. Massaging the abdomen should be avoided. Observe the child's facial expression to note any signs of pain,

discomfort, or areas of tenderness. If areas of tenderness are detected with light palpation, examine those areas last when performing deep palpation.

PEDIATRIC PEARLS

Give infants a pacifier to suck, or distract the young child in the parent's lap with a toy or favorite stuffed animal, so as to help relax the abdomen during the abdominal exam. If a child is ticklish during palpation, place his or her hand under your hand with your fingers interlaced and palpate the abdomen together. Distraction with conversation is also effective in eliciting cooperation throughout the exam.

Deep palpation requires a firmer therapeutic touch, and the child may resist palpation because of fear or pain over areas of inflammation. It may be helpful to use two-handed palpation with the nondominant hand to exert pressure over the examining hand when evaluating the abdomen in obese children and adolescents. *McBurney's sign,* tenderness with deep palpation of the right lower quadrant, or referred right lower quadrant pain with deep palpation of the left lower quadrant, *Rovsing's sign*, could be suspicious for *appendicitis* and should be further evaluated and referred. With deep palpation, pain can also be assessed with *rebound tenderness.* To produce *rebound tenderness,* place fingertips at a 90-degree angle against the abdomen and gently but firmly press into the abdomen. Quickly lift the hand off the abdomen and note if any pain is elicited. The child with *rebound tenderness* will have more pain when the examiner's hand is lifted from the abdomen than with deep palpation. Pain with rebound of the abdomen may be a sign of inflammation or *peritonitis. Rigidity* of the abdomen is the involuntary tightening of the abdominal musculature that occurs in response to underlying inflammation in the abdomen. *Peritonitis* may be indicated if pain is worse when the examiner lifts the hand off the abdomen when testing for *rebound tenderness. Guarding* is the voluntary contraction of the abdominal wall musculature to prevent pain and may subside if the child relaxes.

To palpate the liver, place the fingertips at the right midclavicular line a few centimeters below the rib cage at the costal margin. Move the fingers slightly up and inward and feel for a firm nudge by the liver tip on inspiration. Note the distance between the location of the costal margin and liver tip. The liver may be palpable in an infant or toddler 1 to 2 cm below the right costal margin. Hepatomegaly is suspected in any child whose liver is palpable more than 3 cm below the costal margin. In the obese child or adolescent, hepatomegaly is highly suspect for nonalcoholic fatty liver disease (NAFLD) and should prompt further evaluation through laboratory and imaging studies[4]; however, other causes of an enlarged liver should be considered; these include systemic infection, tumors, hepatic storage disorders, biliary masses, intrahepatic vascular disease, and cardiac disease.

To palpate the spleen, position the fingertips in the left midclavicular line below the costal margin and feel for firmness on inspiration (Fig. 15.9A and B). An alternative technique in infants is to palpate the spleen between the thumb and forefinger of the right hand. The spleen can be felt in about 5% to 10% of children and should be slightly mobile. If splenomegaly is suspected, ultrasound examination can differentiate spleen enlargement from other masses that may arise in the LUQ. Splenomegaly can be caused by infection, inflammation, blood dyscrasias, a mass, or vascular and oncologic conditions. The spleen is a very vascular organ and should be *gently* palpated if injury/trauma is suspected. If blunt trauma to the abdomen is suspected, avoid deep palpation of the spleen or liver. If the liver or spleen is lacerated, a clot *tamponading* the laceration could be dislodged, causing further bleeding with deep palpation.

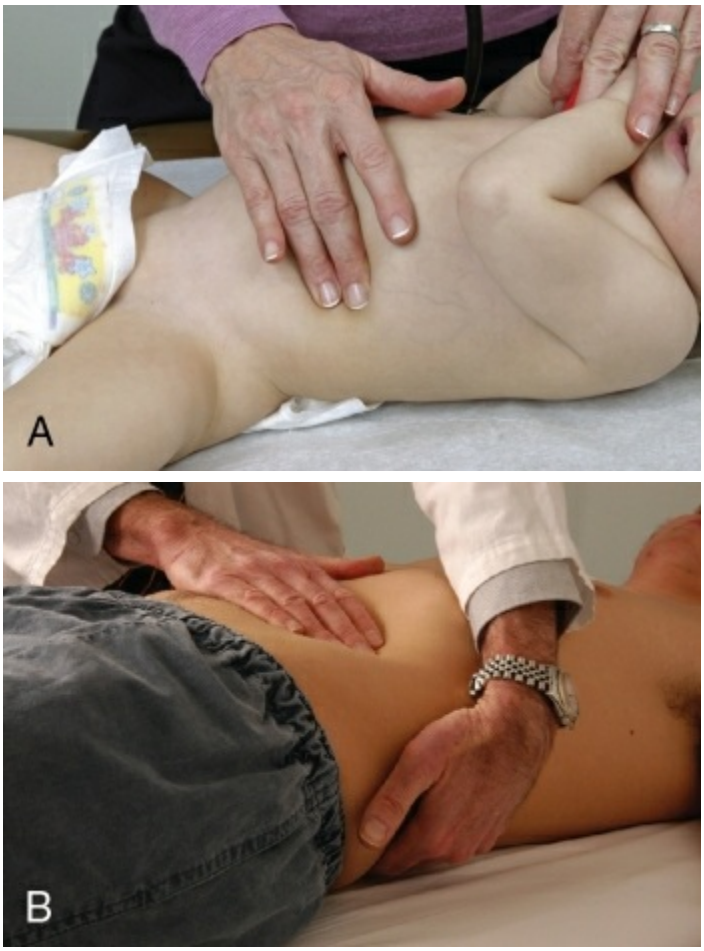


FIGURE 15.9 **(A)** Palpating the spleen in the toddler. **(B)** Two-handed palpation of the spleen in an adolescent. Source: ([B] From Talley N: *Clinical Examination: A Systematic Guide to Physical Diagnosis,* ed 6, Sydney, 2010, Churchill Livingstone.)

The kidneys can sometimes be palpated in infants. Place the left hand behind the right flank of the infant and, using the fingertips of the right hand, palpate deeply in the right upper quadrant (RUQ), to the right of the midline. The right kidney may be "trapped" between the hands. Repeat the technique, placing the right hand behind the infant's left flank, and use the left fingertips to palpate the left kidney in the LUQ. The kidneys should be round, smooth, and firm. A distended bladder may be palpated in the midline above the symphysis pubis, which may indicate a vesicoureteral or bladder neck obstruction, acute bladder retention, or neurogenic bladder.

Palpate the umbilicus for masses or herniation. The most common umbilical disorder is an *umbilical hernia* in which the

intestine protrudes through the abdominal fascia, or *linea alba* (Fig. 15.10). The umbilicus appears to protrude especially when the child is crying, stooling, or coughing, but generally it can be easily reduced when the examiner applies light pressure with the fingertips to the umbilicus. Palpate the fascia below the umbilicus with the fingertips to determine the size of the defect. If the opening is larger than the width of two fingers or the child is older than 3 years of age, surgical closure may be necessary. Incarceration of an umbilical hernia is very rare. Most will spontaneously close by the time the child is 3 or 4 years of age. The most common umbilical mass is a *dermoid cyst.* It appears as a firm, skin-covered, nonreducible mass within the umbilicus that may have a slight discoloration and can be lobulated. Other cysts or an *umbilical polyp* should be referred to a pediatric surgeon for evaluation. *Diastasis recti abdominis*, or separation of the abdominal wall musculature, can be noted with light palpation over the midquadrant. An infant between the ages of 3 and 8 weeks of age with projectile, nonbilious vomiting may have *pyloric stenosis.* Examine the infant while the abdomen is relaxed and palpate in the upper abdomen, slightly right of the midline for a firm, olive-shaped mass, which is highly suspicious for a hypertrophic pylorus. If a child presents with constipation, a sausage-shaped mass of stool may be palpated in the left lower quadrant (LLQ) or in the midline below the umbilicus or *rectosigmoid* colon. The palpation of stool throughout the abdomen may indicate fecal impaction.

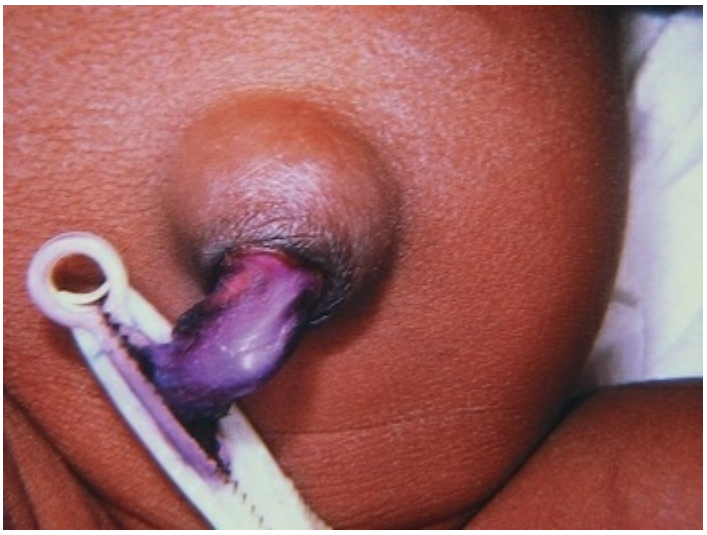


FIGURE 15.10 Umbilical hernia. Source: (From Clark DA: *Atlas of Neonatology*, ed 7, Philadelphia, 2000, Saunders.)

Palpate the groin bilaterally for femoral arterial pulses and the presence of lymph nodes (Fig. 15.11). Check the groin area for a mass or *inguinal hernia.* Note any persistent bulging in the groin. Palpate to determine size and reducibility. If the inguinal bulge or *hernia* can be reduced, referral is indicated for elective surgery and repair. An irreducible or incarcerated hernia is a surgical emergency. Scars from previous surgical hernia repairs should also be evaluated. An incisional hernia may be present and should be evaluated and referred.

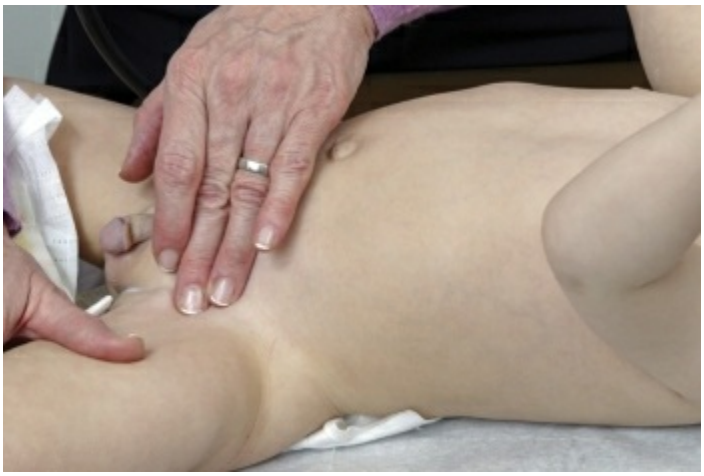


FIGURE 15.11 Palpating femoral pulses and lymph nodes.

Percussion

Percussion of the abdomen can help to identify whether distention of the abdomen is caused by air, a mass, or fluid (Fig. 15.12). When percussing, place your nondominant hand firmly against the abdominal wall so that only your middle finger is resting on the skin. With a relaxed wrist, tap on the distal interphalangeal joint of your middle finger two or three times with the tip of the middle finger of your dominant hand. Air or gas in the abdominal cavity creates a hollow, drum-like sound, or *tympany,* in tapping firmly over the area. Tympany is common in infants and young children

because they swallow air while feeding or crying. Percussion can also delineate rough dimensions of solid masses and organs, although in most settings diagnostic imaging is used to confirm the accuracy of any abdominal findings. To measure liver size, percuss superiorly between the ribs until no dullness is noted. The upper edge of the liver should be detected at the right midclavicular line near the fifth intercostal space. Mark this point and measure its distance from the lower edge of the liver. The lower edge of the liver should not extend more than 1 to 2 cm below the costal margin. This technique identifies only the anterior surface of the liver and not the anteroposterior dimension. Fluid in the abdomen creates a dull sound when percussing over the area and may indicate the presence of *ascites* in the abdominal cavity.

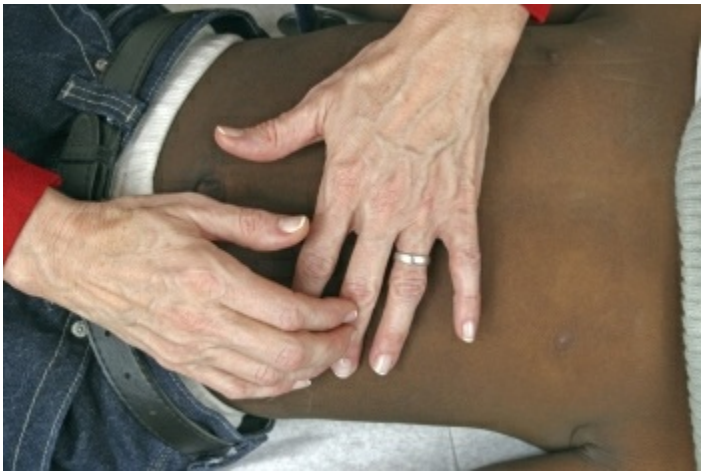


FIGURE 15.12 Percussion of the abdomen.

Rectal examination

A rectal exam should be approached carefully with informed consent of child and parent, and it should be performed if there is concern regarding anal or rectal patency, anal discomfort, constipation or sphincter tone, fissures, hemorrhoids, or rectal polyps.[5] Abdominal pain may be an indication for a rectal exam depending on the child's or adolescent's health history.

If the abdominal exam and health history are unclear or the information obtained from a rectal exam would assist in the diagnosis, explain clearly to the parent and child or adolescent the

need for the rectal exam. Keep in mind that a rectal exam is physically invasive, and many children will no longer cooperate with a physical exam after a rectal exam has been performed. If it is necessary to perform a rectal exam, do so at the very end of the physical examination, using the talk-through approach as presented in Chapter 1.

Position the child on the side with knees flexed (fetal position). An infant or toddler can lie supine with the hips and knees flexed. Assess the sacrum for dimples, sinuses, or tufts of hair. Note the location of the anus; if displaced anteriorly, it could predispose a child to issues such as constipation. Inspect the anus for rashes, fissures, skin tags, or discoloration. Contraction of the external anal sphincter (anal wink reflex) is a normal response to stroking of the skin in the perineum. Gently insert a gloved, lubricated finger (usually the smallest finger in infants; the index finger in an older child) into the rectal vault and feel for any narrowing. Assess sphincter tones. Feel for any masses or polyps or pressure compressing the lumen of the rectum. Note whether there is stool in the rectum and whether it is hard or soft. If an explosive stool is elicited with the rectal exam, it may be a sign of a rectal obstruction, such as *Hirschsprung disease*. Gently press toward the right lower quadrant and observe whether this elicits a pain response, which can support a diagnosis of appendicitis. Alternately press in all directions and note any pain. If any stool is retrieved from the exam, perform a guaiac test on the specimen if there is concern for occult blood in the stool, keeping in mind that trauma during a rectal exam may affect the result.

Diagnostic procedures

A number of diagnostic procedures may be useful to aid in the assessment of abdominal and rectal complaints.[6] Guaiac stool testing or a urine dipstick may be easily performed in the clinic setting or office. Laboratory analyses that could be useful include the evaluation of liver and kidney function, analysis of urine or stool, monitoring of electrolytes, or checking for antibody markers, including for *Helicobacter pylori* or celiac disease. Abdominal ultrasound is often the first step in diagnostic imaging, depending

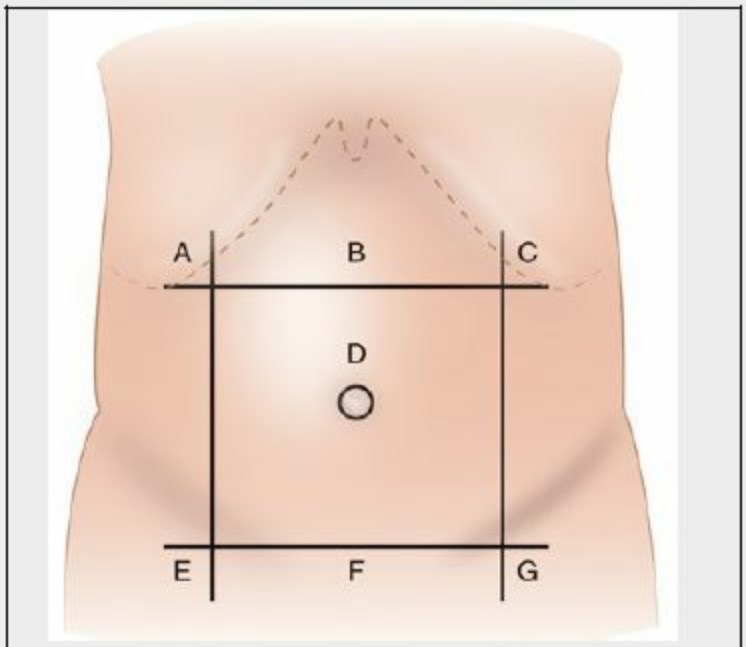
on the child's health history and physical examination findings. Proceed with radiographic procedures such as a kidneys-ureters-bladder (KUB) x-ray or magnetic resonance imaging (MRI), if indicated, to locate and measure organs or masses or detect intestinal inflammation, obstruction, or perforation. A computed tomography (CT) scan would be performed with caution in consultation with pediatric surgery or gastroenterology when indicated. Endoscopy of the upper or lower gastrointestinal (GI) tract may be useful to visualize anatomy, lesions, and obtain biopsies; for example, to evaluate for inflammation of the esophagus due to gastroesophageal reflux disease.

Abdominal conditions

Box 15.2 presents conditions of the abdomen according to where the signs or symptoms may occur. Keep in mind that many conditions involve diffuse abdominal pain, and children often have a difficult time localizing pain to a specific region.

BOX 15.2

Abnormal Conditions of the Abdomen



A, Right Upper Quadrant	B, Epigastric	C, Left Upper Quadrant
Hepatitis Cholecystitis Liver, renal, or adrenal neoplasm Hydronephrosis Pyloric stenosis Pancreatitis High fecal impaction Intussusception	Gastroesophageal reflux Cholecystitis Hepatitis Pancreatitis **D, Periumbilical** Acute gastroenteritis Constipation Functional abdominal pain Appendicitis (referred) Umbilical hernia	Renal or adrenal neoplasm Hydronephrosis Pancreatitis Splenic mass Mononucleosis High fecal impaction
E, Right Lower Quadrant	**F, Suprapubic**	**G, Left Lower Quadrant**
Appendicitis Intestinal obstruction Ovarian torsion or cyst Mittelschmerz Dysmenorrhea Ectopic pregnancy Mesenteric adenitis Incarcerated hernia Lymphoma Pyelonephritis Renal neoplasm	Bladder distention Urinary tract infection Bladder neoplasm Ovarian torsion or cyst Mittelschmerz Dysmenorrhea Ectopic pregnancy Pelvic inflammatory disease	Constipation Intestinal obstruction Hirschsprung disease Ovarian torsion or cyst Mittelschmerz Dysmenorrhea Ectopic pregnancy Incarcerated hernia Lymphoma Pyelonephritis Renal neoplasm

Data from Rakel RE, Rakel DP: *Textbook of Family Medicine*, ed 8, Philadelphia, 2011, Saunders.

Summary of examination

- A thorough and detailed history is an essential part of the abdominal assessment and should include the medical and family health history, nutrition and elimination habits, a psychosocial evaluation, and a review of pain or other

presenting symptoms.

- Infants and young children may be more comfortably assessed in the lap of the parent or caregiver.
- Abdominal assessment should be performed using a mapping technique by mentally dividing the abdomen into four quadrants or nine regions.
- Begin inspection of the abdomen by observing the child or adolescent's posture, behavior, and activity level; then inspect the abdomen for size, shape, and pulsations.
- Auscultate each quadrant of the abdomen before palpation and note the quality and frequency of bowel sounds.
- Use light palpation to identify areas of tenderness before deep palpation.
- During palpation, note size of organs if palpable, presence of tenderness or pain on palpation, any guarding or rigidity in the abdomen, and the size and quality of any masses.
- Perform percussion to identify the presence of air or gas and to estimate the size of solid organs or masses.
- A rectal examination should be performed only when applicable and, if so, should be done at the end of the physical exam.

DOCUMENTATION

1-month-old term infant

Abdomen: Abdomen symmetrical, soft, round, nontender without masses or organomegaly. Liver palpated 2 cm below right costal margin. Normal bowel sounds over all quadrants. Umbilicus clean, dry without hernia, mass, inflammation, or discharge. Epigastric pulsations noted. Percussion tones tympanic.

12-year-old female with abdominal pain

Abdomen: Abdomen symmetrical, flat, tender with guarding throughout and rebound tenderness noted in RLQ. Bowel sounds hypoactive, especially in the lower quadrants. No masses or organomegaly.

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CHAPTER 16

Male genitalia

Angel K. Chen

The pediatric physical examination is not complete without a thorough evaluation of the developing genitalia. Routine surveillance of the genitourinary system is equally important in all patient encounters, and when assessment of the genitalia is performed routinely, abnormalities can be identified and promptly treated. Many parents, children, and adolescents are concerned and anxious about the genitalia, yet they may not feel comfortable expressing those concerns initially. A review of systems in the health history and the routine genital exam offers an opportunity for the parent, child, and/or adolescent to voice concerns and for the provider to offer reassurance and education on normal development. The provider has a role in fostering dialogue between the parent and child regarding reproductive health, and establishing this dialogue early with families when gathering the health history forms the basis for discussions on reproductive health during puberty and in the developing adolescent.

Embryologic development

The differentiation of the sexual organs begins as early as the third week of embryonic development, dictated by the sex chromosomes at the time of fertilization.[1] The *mesoderm* is the embryonic layer that becomes smooth and striated muscle tissue, connective tissue, blood vessels, bone marrow, skeletal tissue, and the reproductive and excretory organs. As proliferation of the embryonic layers

continues, maturation of the external genitalia in the fetus is established by the 12th week.[1] The sex-determining region of the Y chromosome (*SRY* gene) activates the differentiation of the embryonic gonad into a testis, without which, the gonad would become an ovary. The antimüllerian hormone (AMH) prevents development of the uterus and fallopian tubes, whereas testosterone stimulates the wolffian ducts to develop into the male reproductive structures, including the *epididymis, vas deferens,* and *seminal vesicles.* Testosterone is also the precursor to dihydrotestosterone (DHT), which stimulates the formation of the male *urethra, prostate,* and *external genitalia.*[1]

Urine production begins between the 9th and 12th weeks and is excreted into the amniotic cavity through the *urethral meatus.* During development, the fetus continues the production and excretion of urine, which forms the amniotic fluid. Between the 17th and 20th weeks, the *testes,* which develop in the abdominal cavity, begin to descend along the inguinal canal into the scrotum, bringing the arteries, veins, lymphatics, and nerves that are encased within the cremaster muscle and spermatic cord.[1] The inguinal canal closes after testicular descent. Incomplete or abnormal embryonic development of the inguinal canal predisposes the newborn to the formation of hydroceles or hernias. Preterm infants are at increased risk for these conditions. At birth, the infant's genitourinary system is functionally immature with limited bladder capacity, inability to concentrate urine sufficiently, and frequent voiding.

Growth of the fetus and differentiation of the sexual organs can be affected by placental function, the hormonal environment during pregnancy, maternal nutrition, maternal infection, and genetic factors or chromosomal abnormalities. A fetal insult from intrinsic or extrinsic factors during the eighth or ninth week of gestation may lead to major abnormalities of the developing external genitalia. The data on impact from chemical exposure is mixed and inconclusive;[2] however, endocrine-disrupting chemicals do play a role in the development of the endocrine and genitourinary system, which is further reviewed in Chapter 5.

Anatomy and physiology

Penis

The *penis* consists of the shaft, glans, corona, meatus, and prepuce. The *shaft* is composed of erectile tissue called *corpora cavernosa* (two lateral columns) and *corpus spongiosum* (ventral column). The anterior portion of the corpus spongiosum forms the *glans penis,* the border or edge of which is called the *corona.* The urethra is within the corpus spongiosum, and the orifice, or *urethral meatus,* is the slitlike opening just ventral to the tip of the penis (Fig. 16.1).

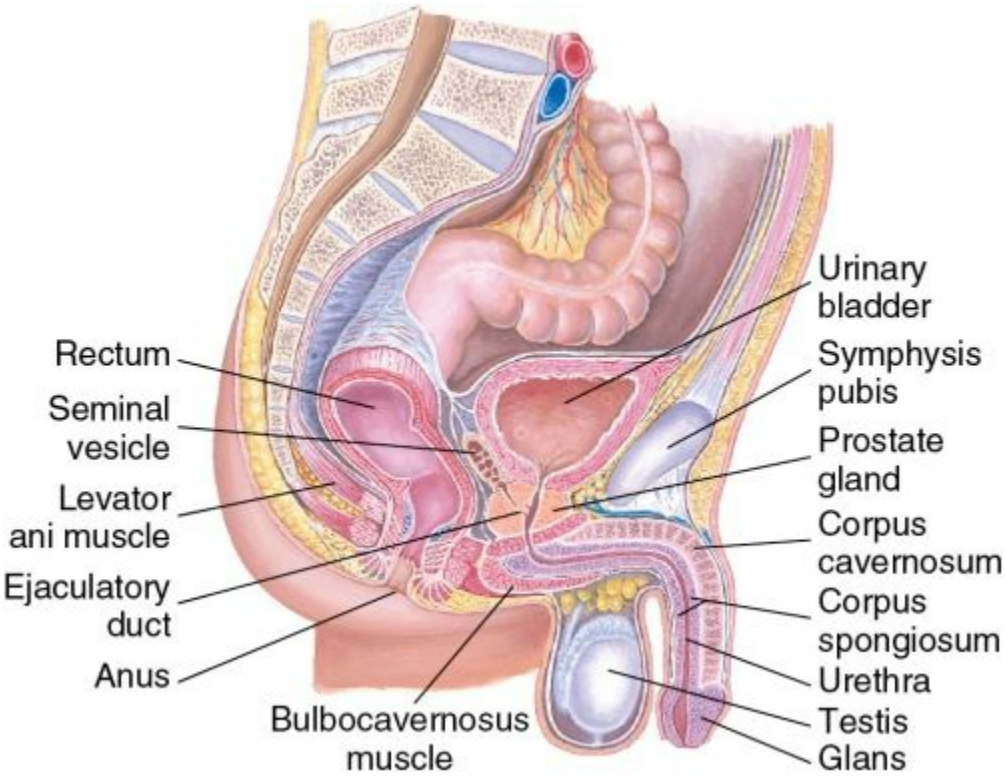


FIGURE 16.1 Internal and external genitalia. Source: (From Seidel HM, Ball JW, Dains JE, et al: *Mosby's Guide to Physical Examination,* ed 7, St. Louis, 2011, Mosby.)

The *prepuce,* or *foreskin,* is the fold of skin at the tip of the penis that covers the glans penis in the uncircumcised male and forms the secondary fold of skin from the urethral meatus to the coronal region of the penis called the *frenulum.* The skin of the penis is thin, does not contain subcutaneous fat, and is loosely tied to the deeper layer of the dermis and fascia. Often, the skin of the shaft is more darkly pigmented, particularly in children or adolescents with darker pigmentation or darker skin. The skin of the *glans penis* does

not contain hair follicles but does have small glands and papillae that form in the epithelial cells to produce *smegma,* the white oily material made up of desquamated epithelial cells trapped under the foreskin, which is normal and often mistaken as pathologic (Fig. 16.2). The foreskin is generally nonretractable at birth because of adherence of the inner epithelial lining of the foreskin and glans.[3, 4] Desquamation of the tissue layers continues until the separation of the prepuce and glans penis is complete secondary to intermittent erections and keratinization of the inner epithelium, generally by 6 years of age. Partial adhesions of the foreskin to the prepuce may produce smegma along the coronal region, which often persists throughout childhood.

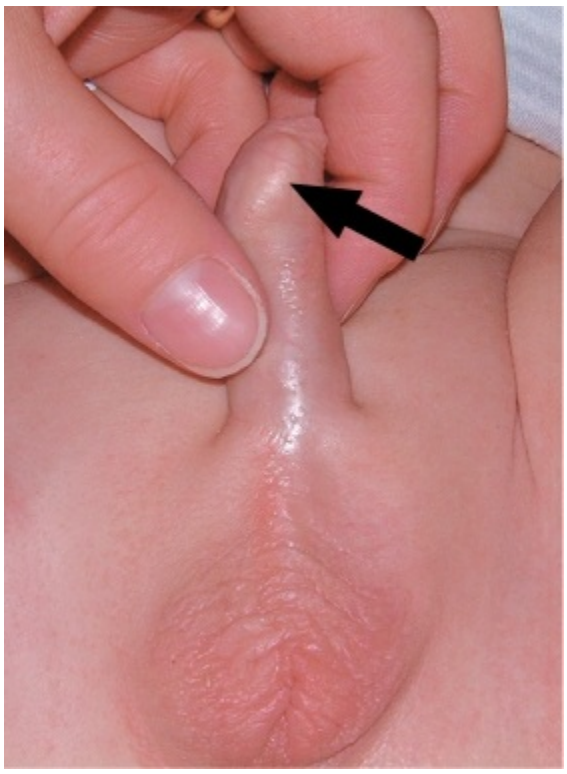


FIGURE 16.2 Smegma. Source: (From Godbole P: General paediatric urology. *Surgery (Oxford)* 26(5):223–227, 2008.)

Scrotum and testes

The *scrotum* is made up of a thin layer of skin that forms *rugae* (folds) and contains the *testes*. The *epididymis* is the thin palpable structure along the lateral edge of the posterior side of the testes.

Sperm production occurs within the *seminiferous tubules* in the testes, which connect to the *epididymis.* The *vas deferens* is the cordlike structure that is continuous with the base of the epididymis and stores sperm. The *spermatic cord* extends from the testes to the *inguinal ring* and is felt only on deep palpation of the *testicular sac.* The right spermatic cord is shorter in some males, which causes the right testis to hang higher than the left.

The male accessory organs include the *seminal vesicles,* which lay deep in the abdominal cavity alongside the vas deferens and secrete liquid into the *semen* as it passes from the testes. The opening of the seminal vesicles joins the vas deferens to form *ejaculatory ducts* and drains to the posterior urethra. The *Cowper glands,* or bulbourethral glands, lie along the urethra in the male genitalia. These are exocrine glands that produce a viscous secretion that lubricates the urethra for the passage of sperm. It also neutralizes the acidic urethral secretions that could otherwise damage the spermatozoa in the *semen.* The *prostate gland* is the small, firm mass that lies within the pelvic cavity. It first becomes palpable on manual examination in the adolescent male. The prostate gland surrounds the posterior urethra and secrets an alkaline secretion during ejaculation to promote fertilization. The smooth muscle within the prostate gland assists with urinary elimination. An enlarged prostate gland may cause urinary obstruction.

Physiologic variations

Table 16.1 presents the physiologic variations of the male genitalia in the pediatric age-group from infancy to early childhood.

TABLE 16.1

Physiologic Variations of the Male Genitalia

Age Group	Variation
Preterm infant	Rugae absent on scrotum in low-birth-weight infantTestes undescended in ~20% of infants weighing <2500 g[a]
Newborn	Rugae present from 37 weeks' gestationNote and evaluate any discoloration of scrotum at birth Testis: volume 1–2 mL Length of penis in term infant: 2.5–3.5 cm; size should be palpated during exam

	Shaft may appear short or retracted in infants with significant suprapubic fat pad
Infancy/early childhood	Foreskin partially retractable over glans penisTestes present in scrotal sac Initial sexual arousal and erection occur with normal sexual exploration or exposure of genitalia

[a]Data from Cavaliere TA: Genitourinary assessment. In Tappero EP, Honeyfield ME, editors: *Physical Assessment of the Newborn*, ed 5, Petaluma, CA, 2015, NICU INK, pp 121–137.

Physiologic changes in puberty

Puberty has a gradual onset, and although it is marked by a growth spurt, it may be difficult to ascertain when the first changes in the male begin in the transition to secondary sexual characteristics (Fig. 16.3). In males, sexual development, or true gonadal activation, begins with an increase in size of the testicles, which is often difficult to determine. Testicular volume should be evaluated and is the most accurate measurement of the progression of puberty in the developing male. In some boys, this begins around 10 years of age and is complete in about 3 years, with the normal range extending to 5 years.[5] The sexual maturity rating (SMR) scale, also known as Tanner stages, helps to classify physical pubertal maturation (Fig. 16.4 and Table 16.2).[5] Genital development and pubic hair may develop at different rates. Ejaculation usually occurs at SMR 3 during the midpoint of sexual development. Fertility is established by SMR 4, although sperm are present in some quantity with ejaculation during SMR 3.

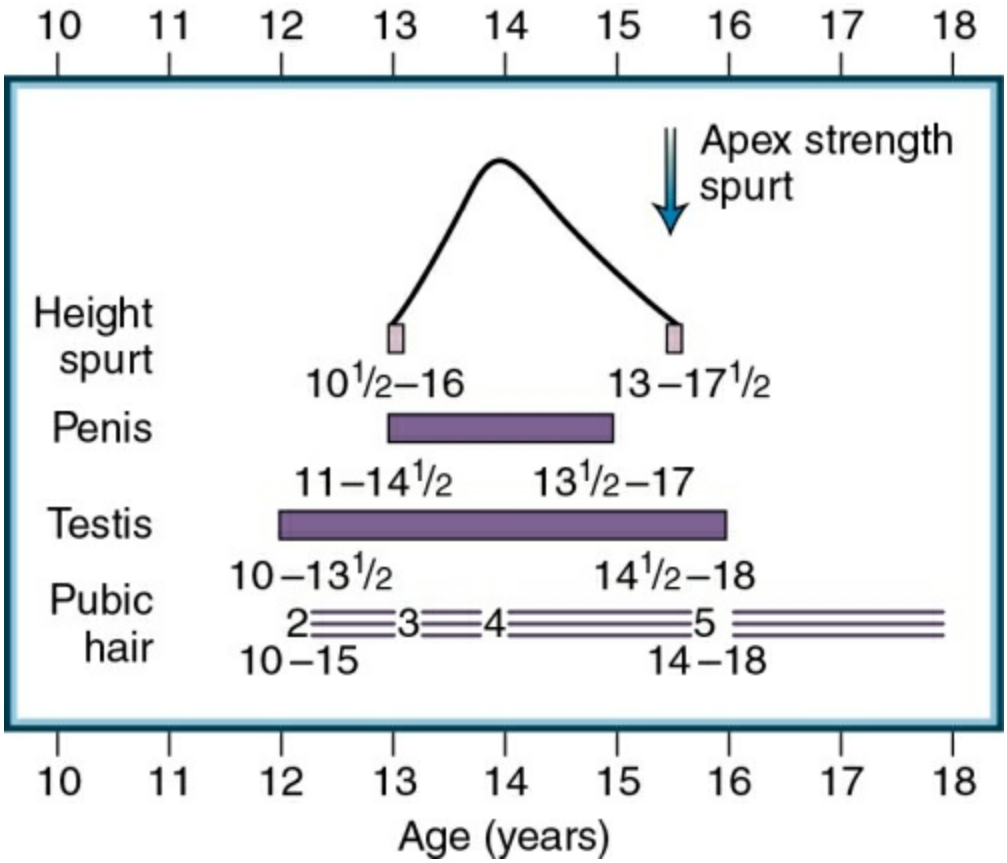


FIGURE 16.3 Pubertal changes. Source: (From Marshall WA, Tanner JM: Variations in the pattern of pubertal changes in boys. *Arch Dis Child* 45:13–23, 1970.)

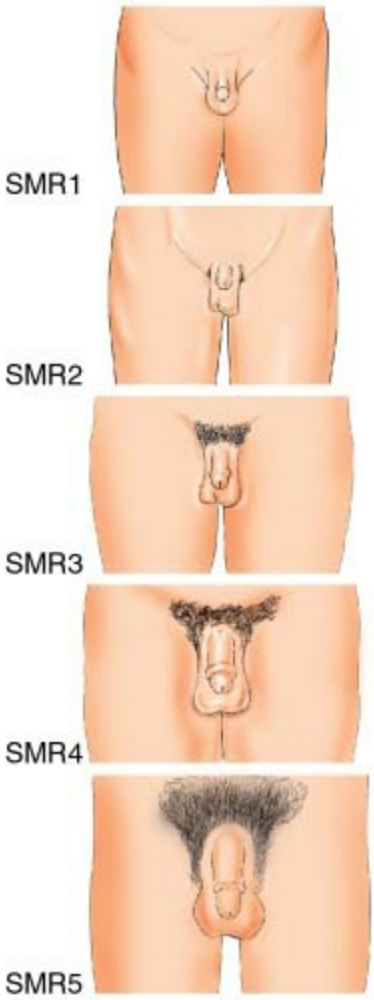


FIGURE 16.4 Sexual maturity rating or Tanner staging.

TABLE 16.2
Developmental Changes in Puberty

	Developmental Changes			
Tanner Stage/Sexual Maturity Rating (Smr)	Pubic Hair	Penis	Scrotum	Testicular Volume
Tanner stage 1SMR 1	None	Unchanged from early childhood	Unchanged from early childhood	<1.5 mL
Tanner stage 2SMR 2	Small amount of light, downy hair along base of penis	Unchanged or slightly enlarged	Enlarged and reddened pigmentation; skin taut and thinner; increased rugation	1.6–6 mL
Tanner stage 3SMR 3	Moderate amount of curly, pigmented coarse pubic hair extending laterally over symphysis pubis	↑ Length and circumference	Further enlargement	6–12 mL
Tanner stage 4SMR 4	Abundant adult-like quality	Glans penis broader and larger; penis ↑ length and circumference	Further enlargement and darker pigmentation	12–20 mL
Tanner stage 5SMR 5	Extends to medial surface of the thighs	Adult size	Adult size	>20 mL

Data from Neinstein LS, Katzman DK, Callahan ST, Gordon CM, Joffe A, Rickert VI: *Neinstein's Adolescent and Young Adult Health Care: A Practical Guide,* ed 6,

Philadelphia, 2016, Wolters Kluwer.

In male adolescents, it is the tempo of growth and maturation that is distinctly different than that in females. At mid-puberty, with rising testosterone concentrations, most of the rapid linear growth occurs. Recent studies have provided evidence of earlier onset puberty in males, particularly in African Americans.[5] The trend is an overall earlier start of puberty but prolonged progression to genital maturity.

Family, cultural, racial, ethnic, and other considerations

Understanding and respecting cultural differences is an important part of the role of the health care provider, particularly in relation to the assessment of the genitourinary system. Identifying which family members may be a part of the discussion and/or be present during the examination of the genitalia and how to approach the exam are the first steps in developing a trusting relationship with the family. Parental and family attitudes about sexuality vary widely both within cultures and among cultures. Sexual awakening first occurs in the young child with the discovery of the genitalia as a sensual organ. This initial experience and the parental attitude toward normal fondling and exploration may set the stage for either normal sexual development in a child or a feeling of shamefulness and an attitude in the child that fosters abnormal functioning throughout life. Often, cultural attitudes concerning sexuality and reproduction form the basis of conflict between the parent and young child or maturing adolescent. Mediating this difference between the adolescent and the parent may be the most important and challenging role for the health care provider. In addition, documentation in the health care record provides opportunity to communicate these attitudes to the next provider to ensure consistent delivery of care.

System-specific history

Important information for the assessment of the male genitalia at key developmental stages and ages is presented in the Information

Gathering table as follows.

Information Gathering for Male Genitalia Assessment at Key Developmental Stages

Age Group	Questions to Ask
For all age groups	Gestational and birth historyHistory of maternal hormone ingestion during pregnancy Maternal alcohol and/or drug use Maternal exposure to hazardous chemicals and/or pesticides Family history of GU abnormalities Current medication Circumcision status Scrotal or inguinal mass; ± pain Voiding history (include voiding frequency, voiding stream, ballooning of foreskin [if applicable]) Elimination history (frequency and consistency)
Preterm, newborn, and infant	History of maternal infectionHistory of significant neonatal infections Presence of testes in scrotum at birth
Early childhood, middle childhood	Toilet training processAdditional voiding history (history of urinary frequency, postponing behavior, daytime and/or nighttime wetting, difficulty, or pain on urination) History of balanitis and/or (documented) urinary tract infections Physiologic phimosis
Adolescence	Family history of delayed pubertyOnset of growth spurt and puberty Painful erection History of balanitis and/or (documented) urinary tract infections Testicular pain/swelling Penile discharge Sexual identity and gender expression Sexual debut: With males, females, or both Number of partners Types of sexual activity (vaginal, oral, and/or anal intercourse) Condom use STI exposure/testing/results History of coerced sexual contact (molestation/rape) Inability to achieve erection and/or to ejaculate

GU, Genitourinary; *STI*, sexually transmitted infection.

Physical assessment

Preparation

Although there is a tendency toward skipping assessment of the genitalia in the course of performing a physical examination, the

provider should be mindful that it is a critical part of evaluating a growing and developing body. If performed regularly by the provider during pediatric well visits, the genital examination then becomes simply an expected part of the routine physical assessment at all ages, including through adolescence. Reticence on the part of the child, adolescent, parent, or even the provider is not a reason for omitting the examination of the genitalia. The provider should proceed as with the rest of the physical examination, maintaining a matter-of-fact approach and using a purposeful technique that is not traumatic for the child or adolescent.

Before the start of the genitourinary exam, the provider should discuss the rationale for performing this exam as well as what to expect during the exam. Anticipatory guidance is of utmost importance for all age groups beyond the infant and young child. Parents of adolescents should be prepared to leave the exam room for the physical examination of the genitalia after bringing up any concerns during the health history. This gives the provider an opportunity to discuss with the adolescent the consent to confidential reproductive health services and the limits to confidentiality, which differ from state to state (see Chapter 4).

PEDIATRIC PEARLS

In early childhood, the provider should emphasize that the exam is only to be done when a parent or guardian is present but not with strangers or other adolescents or adults. In middle childhood, the genital exam is a chance to reinforce normal development, and the exam prepares the child for what to expect as an adolescent. In adolescence, the exam is a chance to be alone with the provider to discuss questions and/or concerns about their genitalia and sexual development.

As with all parts of the physical examination in pediatrics, it is important to *talk through* the assessment of the male genitalia and reassure the adolescent of normal findings as well as discuss any concerns when completing the examination. This is also an opportunity to discuss normal growth and development, hygiene

issues, and anticipatory guidance. With the adolescent male, the talk-through exam provides the opportunity to establish rapport and validates feelings and concerns during and after the examination. There is often a hidden agenda with adolescents around issues of normalcy or reproductive health. The pediatric health care provider must be ready to discuss any issues involving reproductive health in a supportive and professional manner.

Positioning

During the first 4 to 6 months of life, infants are best positioned on the examination table for assessment of the male genitalia. Beyond early infancy, the child may be lying on the parent's lap in a knee-to-knee position with the provider, or continue to be examined on the exam table. The young child may be lying down or sitting in the tailor position (cross-legged) on the exam table (Fig. 16.5). The middle-age child may be examined lying supine on the exam table or sitting or standing to enhance gravity of the testicles, as in the adolescent and young adult male.[5, 6] It is important to have draping available and only expose the child as long as needed for inspection, palpation, and/or education. Lengthy discussions can be done once the child is dressed. Respecting the need for privacy, particularly in the young child and the developing adolescent, establishes a good patient-provider relationship.

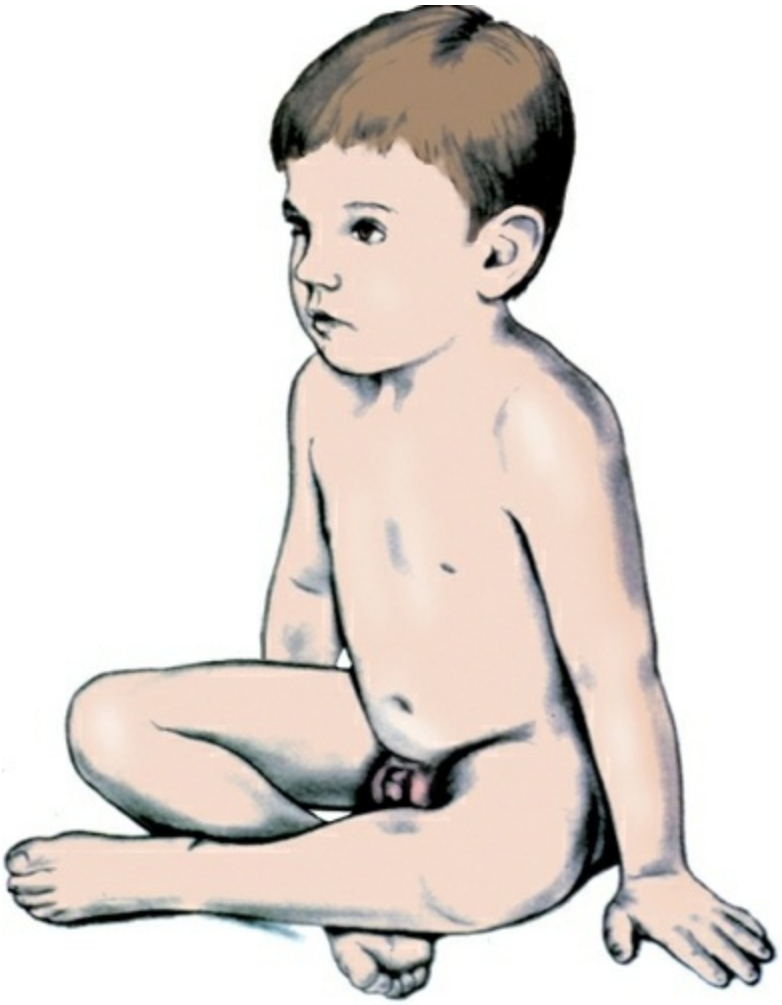


FIGURE 16.5 Young child sitting tailor style (cross-legged) in preparation for testicular exam.

Inspection and palpation

Assessment of the male genitalia includes inspection and palpation of the penis, foreskin, urethral meatus, scrotum, testes, rectal sphincter, and perineal skin status. Always inform the child or adolescent before beginning inspection and palpation. *Physiologic phimosis*, when the foreskin cannot be retracted over the glans penis, is normal before 6 years of age (Fig. 16.6). Forcible retraction of the foreskin may cause trauma, with increased risk for infection and/or scarring, leading to pathologic phimosis (see Table 16.4). Good hygiene of the penis and foreskin can decrease the risk of developing infection or adhesions of the foreskin to the glans penis and should be reviewed regularly at well-child visits during childhood.

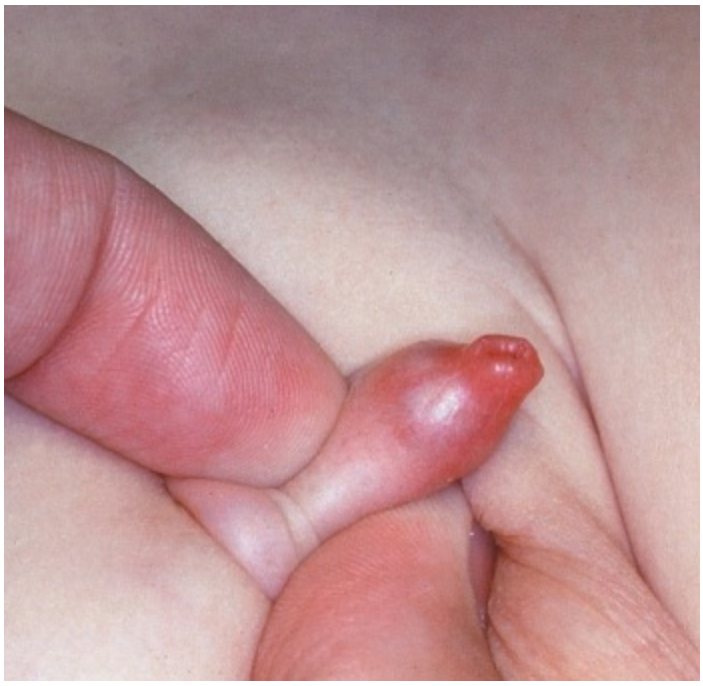


FIGURE 16.6 Physiologic phimosis. Source: (From Gearhart J, Rink R, Mouriquand P: *Pediatric Urology*, ed 2, Philadelphia, 2010, Saunders.)

EVIDENCE-BASED PRACTICE TIP

Do not forcibly retract the foreskin. If indicated, treat with steroid ointment (betamethasone 0.05% or triamcinolone 0.1% ointment), applied 3 times/day to retracted foreskin for 4–6 weeks (see Fig. 16.6). Surgical intervention is rarely indicated in physiologic phimosis. [7, 8]

EVIDENCE-BASED PRACTICE TIP

Care of the uncircumcised penis: Rinse penis and foreskin with water and soap during bathing. Always return foreskin over glans following retraction. For older boys, instruct the child to gently retract the foreskin during voiding. Refer parents to the American Academy of Pediatrics website for more information on caring for a child's penis. Available at

http://www.healthychildren.org/English/ages-stages/baby/bathing-skin-care/Pages/Caring-For-Your-Sons-Penis.aspx

In the newborn, the urethral meatus is normally on the tip of the glans penis. In middle childhood and adolescence, the urethral meatus is a slitlike opening on the ventral side of the glans penis. Examination in the adolescent male includes an inspection for erythema or discharge (Fig. 16.7).

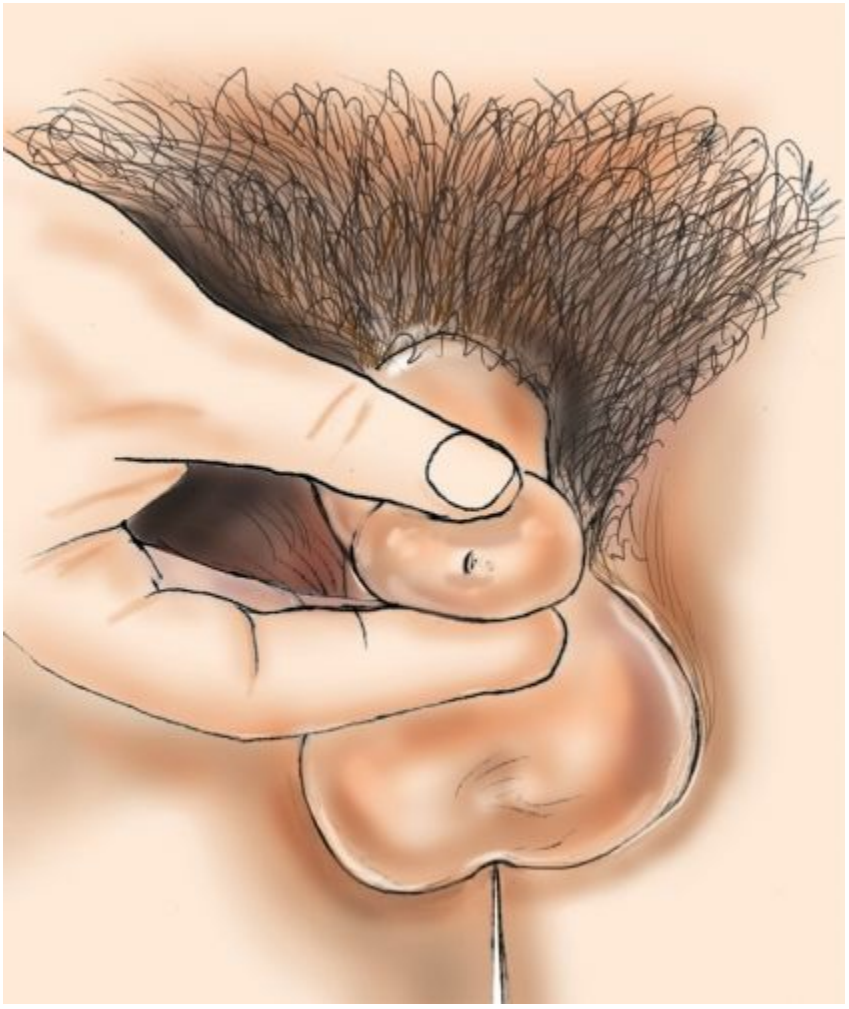


FIGURE 16.7 Examination of urethral meatus in adolescent.

Inspection of the scrotum includes assessing the fullness of the scrotum, color, median raphe, and rugae, as well as determining SMR or Tanner stage. Before palpation of the scrotum in the infant

and prepubertal male, the provider should place a hand firmly above the inguinal canal, near the superior anterior iliac crest, milk down the testis along the inguinal canal, and trap the testis in the scrotum with the thumb and index finger of the opposite hand to prevent retraction of the testes (Fig. 16.8). Isolate the testis between the thumb and forefinger, and roll within the pads of the fingers to assess for testicular size, shape, consistency, and any point tenderness (Fig. 16.9). The *cremasteric reflex* causes retraction of the testes into the inguinal canal/abdominal cavity and can be activated by cold, touch, or emotion, particularly in infancy and early childhood. *Retractile testis* is a testis that may retract on exam but may be normally positioned in the scrotum. This may confuse the findings of the exam, prompting unnecessary referral in a normal male child.

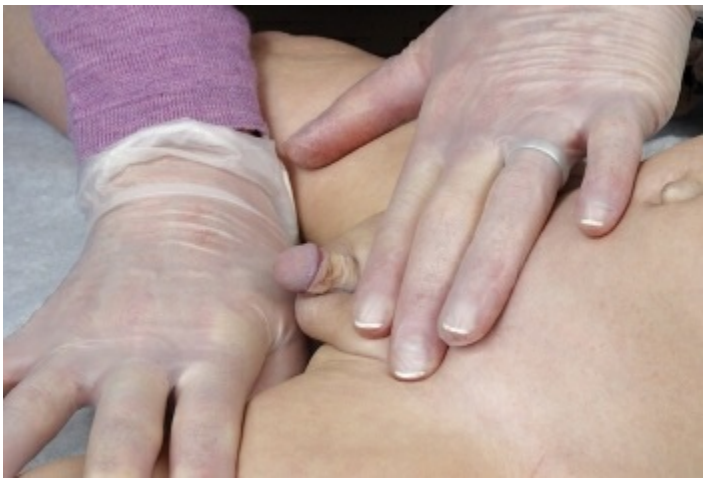


FIGURE 16.8 Trapping of testis in scrotum for examination.

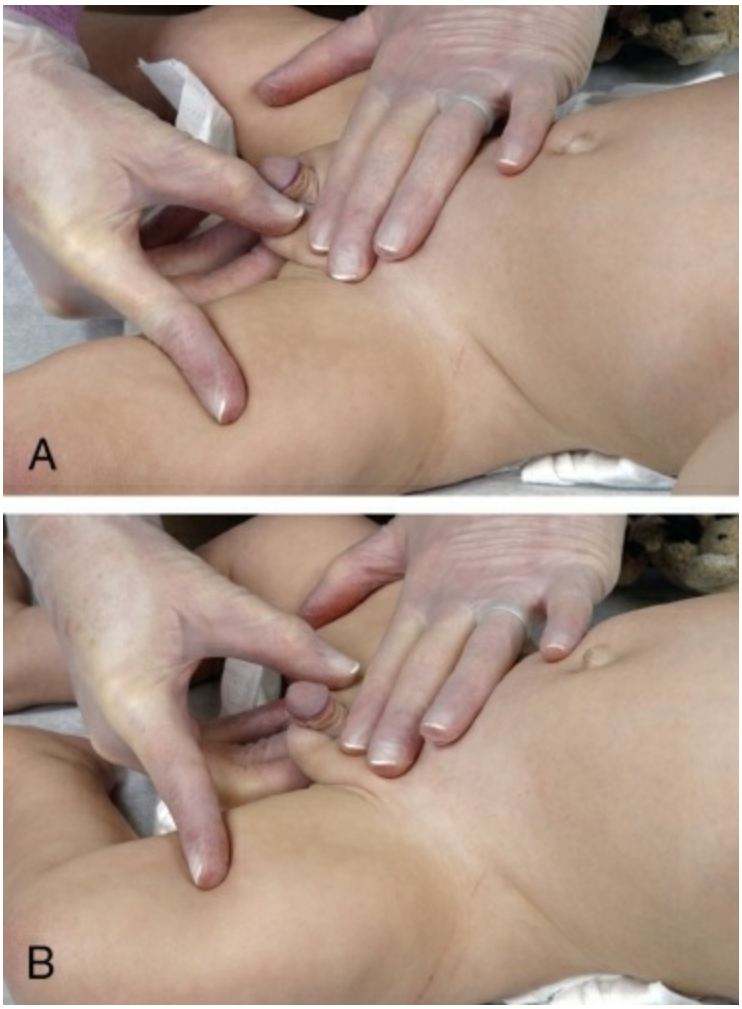


FIGURE 16.9 Palpation of scrotal sac. **(A)** Left testicle. **(B)** Right testicle.

Compare the testes bilaterally for any significant difference in size. Testicular size discrepancy of greater than 15% should be noted for further evaluation.[6] An *orchidometer* may be used to screen testicular size in the growing and developing male (Fig. 16.10), although scrotal ultrasound is used as a more accurate measurement of testicular volume.[9] If the testis is not palpable in the scrotum in the young child, often a positional change to sitting with legs crossed (tailor style) will facilitate relaxation of the cremasteric muscle and allow for the testes to descend to the scrotal sac (see Fig.16.5). Careful inspection and palpation of the inguinal, suprapubic, scrotal, and perineal region may reveal an *ectopic* testis.[10]

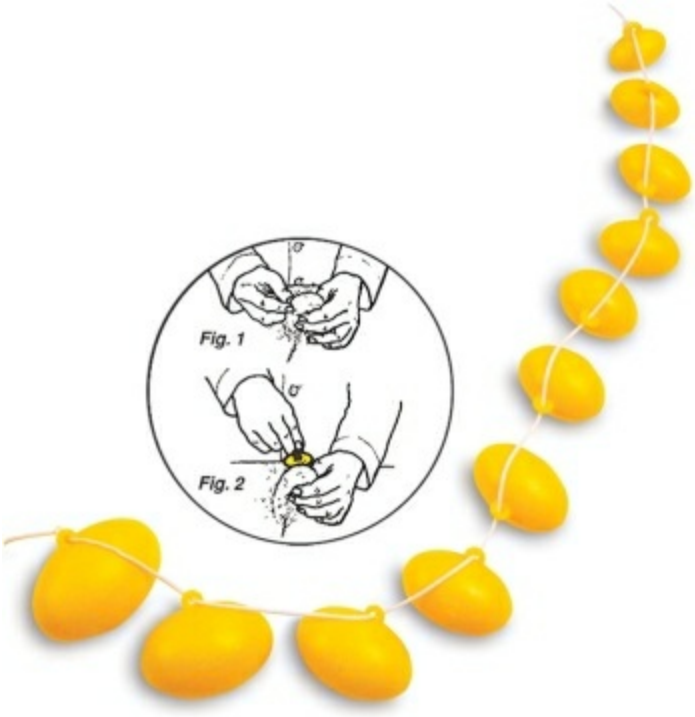


FIGURE 16.10 Orchidometer to evaluate testicular size. Source: (Courtesy Accurate Surgical & Scientific Instruments Corp, Westbury, NY.)

EVIDENCE-BASED PRACTICE TIP

Cryptorchidism, or undescended testicle, is best evaluated by history and physical examinations initially. Radiologic imaging with ultrasounds offer poor diagnostic value and increase the cost of health care. Referral to pediatric urologists *without* first obtaining ultrasounds for evaluation and management is indicated. [10, 11] Generally, if testes were detected in the normally formed scrotum in the first 6 months of life but are now present as undescended, they are most likely *retractile* in nature. Acquired cryptorchidism, or ascent of a previously descended testis, may occur in 1%–7% of the boys and peak at 8 years of age. [10] Acquired cryptorchidism would require further evaluation by a pediatric urologist.

In the adolescent male, the examiner should palpate for the *epididymis* on the posterior surface. It can be felt on the lateral

surface of the testes as smooth, discreet, and slightly irregular. The *vas deferens* can be palpated from the testes to the inguinal ring. Any testicular nodules should be referred for further evaluation to rule out malignancy. The adolescent male should be educated about *testicular self-examination* (TSE) along with a return demonstration. Although there is controversy among organizations regarding the effectiveness of routine TSE for testicular cancer screening,[12-15] the Society for Adolescent Health and Medicine (SAHM) does recommend TSE as a way for adolescents to detect abnormalities that may lead to testicular cancer or infertility.[16]

EVIDENCE-BASED PRACTICE TIP

Some helpful testicular self-examination resources for teens include the following:

Kids Health: http://kidshealth.org/teen/sexual_health/guys/tse.html

MedlinePlus: http://www.nlm.nih.gov/medlineplus/ency/article/003909.htm

American Cancer Society: http://www.cancer.org/cancer/testicularcancer/moreinformation/doihavetesticularcancer/do-i-have-testicular-cancer-self-exam

For young adolescent males with SMR or Tanner stage 3 and above, another essential part of the genitourinary exam is the inguinal hernia exam. This is a routine part of the preparticipation sports physical assessment. To examine the inguinal ring, invaginate the skin from the scrotal sac with index finger and advance into the slitlike opening of the inguinal ring (Fig. 16.11). Ask him to bear down or cough so any protrusion/masses may be noted. If an inguinal mass is noted, auscultation may occasionally be used to ascertain whether bowel sounds are present, although this technique has been replaced by using ultrasound in most instances.

FIGURE 16.11 Examination for inguinal hernia.

Rectal examination

Rectal examination in the infant and young child is indicated to assess tone, constipation, rectal bleeding, fissures, hemorrhoids or other gastrointestinal concerns, and history of sexual abuse when indicated. The rectal examination may include an external inspection, and/or a digital rectal exam. It is performed in a young child in a supine position with the legs flexed. Inspect the anal area for location of the anus, fissures, rectal tears, hemorrhoids, any lesions, or inflammation and placement. A discoloration around the anus may indicate heavy metal toxins. Note any sacral sinus, tufts of hair, or dimpling on the buttock. Use the index finger, or the fifth digit for an infant, gloved and lubricated with a small amount of gel. Press the pad of the finger against the anus. The anal sphincter should relax (this is called the anal wink) and allow the small digit

to slip into the anal canal. Digital exploration of the rectal vault requires a gentle approach. During digital exam assess tone, presence and characteristics of stool, feel for polyp, sweep finger all around anal canal. On any stool obtained on exam test for blood using office fecal occult blood testing materials. False positive occult blood test result may be related to trauma during the exam. The digital rectal exam is not performed routinely unless symptoms or history warrants the examination.

In the adolescent male, digital manipulation is performed with the adolescent positioned on his left side with knees flexed or standing with hips flexed and upper body supported with hands on the examining table. The index finger, gloved and lubricated, is used for the exam, as previously described. Rotate the finger and palpate the anterior rectal wall to feel the prostate gland, which in the adolescent and young adult male should be firm and smooth and measure about 2 to 3 cm (Fig. 16.12). The rectal exam in the adolescent requires careful explanation of the purpose of the exam beforehand to avoid inflicting physical or psychological trauma.

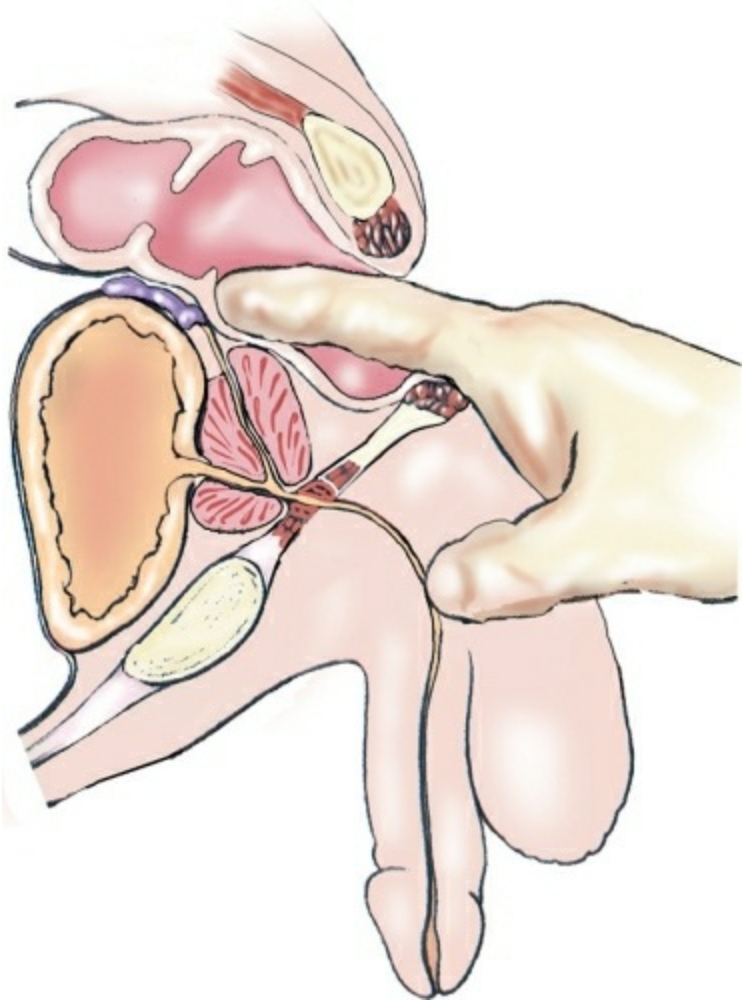


FIGURE 16.12 Rectal examination technique in adolescent.

Circumcision

Circumcision is the excision of the *foreskin*. In the newborn, the procedure is a matter of parental choice in consultation with the health care provider, taking into consideration the cultural, religious, and ethnic traditions along with medical factors. The American Academy of Pediatrics (AAP) formed a multidisciplinary task force that concluded the health benefits of newborn circumcision "outweigh the risks of the procedure," and thus families should have *access* to circumcision should they choose to have it done.[17] Benefits of circumcision may include the decrease in risk of urinary tract infections, which is most beneficial in high-risk males, as well as potential decrease in risk of transmission of some

sexually transmitted infections (STIs), including HIV, and development of penile carcinoma.[17] However, evidence is lacking for recommendation of routine newborn circumcision for all males.[17] Parents should be given accurate and unbiased information and be provided opportunity to discuss and make the decision regarding circumcision. Newborns who are circumcised require procedural analgesia for pain control, and the risks of analgesia should be reviewed with parents.

Sexual abuse

A complete physical examination, including thorough evaluation of the genitourinary system, is warranted with any indication or revelation of sexual misconduct on history taken either from parent or child or adolescent. Although in 95% of sexual assault cases there is no apparent visible physical evidence, children and adolescents who are known or suspected victims of sexual assault require careful evaluation for any evidence that may be present.[18] The history must be obtained in a safe and private environment, while using open-ended and nonleading, developmentally appropriate questions. Examination of the oral cavity as well as the genitals and anus requires careful inspection. Penis and scrotum should be evaluated for bruising or signs of external trauma, redness, swelling, or discharge. The anus and rectum should be inspected for hemorrhoids, warts, rectal dilatation, rectal fissures, or fistulas that would indicate trauma incurred as a result of intercourse. A digital exam should be performed with a gloved, lubricated finger to evaluate the sphincter tone and the rectal vault. For a complete explanation of findings to the child or adolescent and family, referral to Child Protective Services (CPS), consultation with child abuse experts, referral to additional resources, and follow-up are indicated.

Disorder of sexual differentiation

The first step in diagnosing the infant or child with disorder of sexual differentiation (DSD), including those with disorder of chromosomal sex, disorders of gonadal sex, or disorder of phenotypic sex including ambiguous genitalia, starts with obtaining

a detailed history. Family history of previous spontaneous abortion, stillbirth, or any neonatal death of a male sibling that could be related should be noted; family history of excess androgen and congenital adrenal hyperplasia or an autosomal recessive genotype should also be carefully reviewed. Afterwards, a careful inspection of any dysmorphic features and examination of the abdominal and genital area is performed. The key finding of the genital exam is the presence or absence of gonadal or testicular tissue palpable in the scrotum, labioscrotal folds, or inguinal canal (Fig. 16.13).[19] Palpable gonads lead to the high probability that the infant is an XY male, because ovaries do not descend. Document size of penis, location of meatus, and any hyperpigmentation of the labioscrotal folds. For newborn males with bilaterally impalpable testes yet phenotypically appear to be male with formed phallus, an immediate consult to pediatric urology and pediatric endocrinology is warranted to determine a possible diagnosis of DSD. This can be a potential life threatening condition such as congenital adrenal hyperplasia if the infant if not diagnosed and treated.[6, 10]

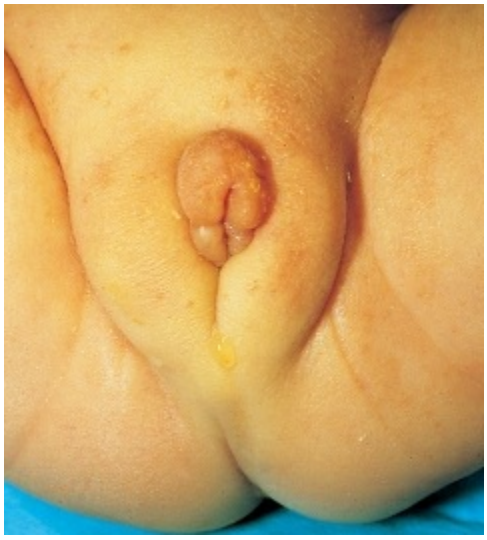


FIGURE 16.13 Ambiguous genitalia. Source: (From Zitelli BJ, Davis, HW: *Atlas of Pediatric Physical Diagnosis,* ed 4, St. Louis, 2002, Mosby.)

Research continues to define new and multigenetic factors involved in the development of DSD, but there is still insufficient information to develop practice guidelines.[20] Acute conditions

warrant immediate management. Genetic karyotyping and additional laboratory tests are required to make a sex determination, and immediate referral to a multidisciplinary team, including neonatologist, pediatrician/pediatric nurse practitioner, pediatric endocrinologist, pediatric urologist, geneticist, psychologist, social worker, and ethicist, is indicated.[21]

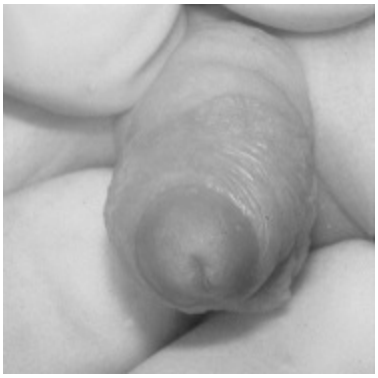
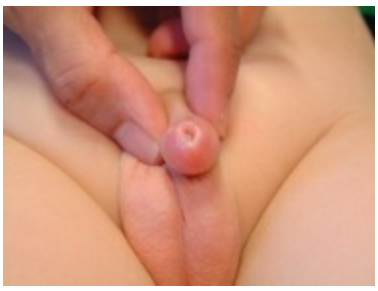
Male genital conditions

Table 16.3 summarizes findings that require a referral to a pediatric urologist, and Table 16.4 presents abnormal conditions of the male genitalia in the infant, child, and adolescent.

TABLE 16.3
Abnormal Findings of Male Genitalia

Area	Abnormal Findings and Referral Criteria
Penis	Abnormal position of urethral meatus, ventral or dorsal to tip of penisAbnormal curvature of penis Micropenis: associated with syndromes and organ abnormalities Abnormal discharge, erythema (redness), swelling Thickened/scarred foreskin Foreskin unable to be retracted (>6 years of age) Foreskin unable to return to cover glans (see paraphimosis) (urologic emergency)
Scrotum	Painful or red/swollen testicle/scrotum **(urologic emergency)**Undescended or absent testes Communicative hydrocele (changes in size) Hydroceles that prevents assessment of testes (via manual exam or transillumination) Hernia Onset of puberty before 8 years of age Delayed or absent pubertal development Presence of skin lesions, vesicles, chancre, or lice Testicular size discrepancy of >15% Isolated right-sided varicocele
Inguinal region	Inguinal mass or herniaTender lymph nodes
Anus/rectum	Abnormal distance from scrotum to anusAbnormal rectal patency Rectal polyps, hemorrhoids (adolescents)

TABLE 16.4
Male Genital Conditions

Condition	Image	Descrip
Adhesions (penile)		Remna bet pre sme unc ado
Balanoposthitis/balanitis		Superfi pen swe ten and yell be p may and phi
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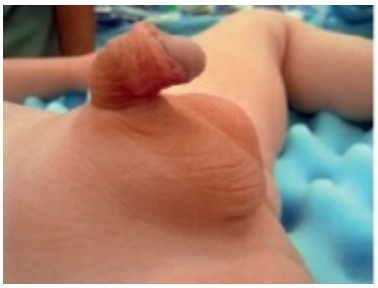
Chordee		Ventra cur fibr an i ofte
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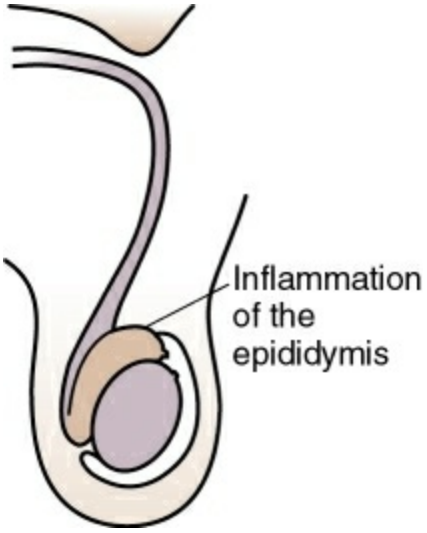
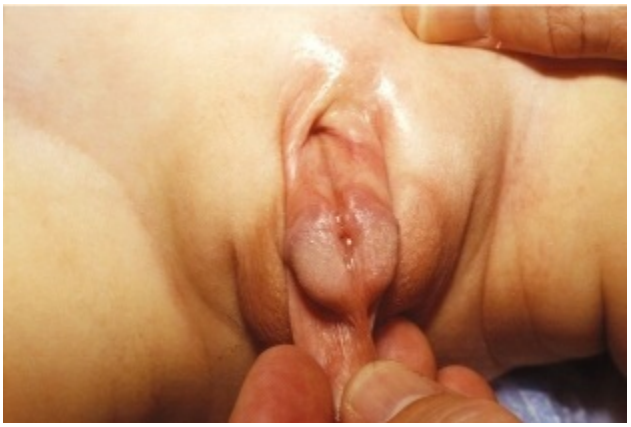
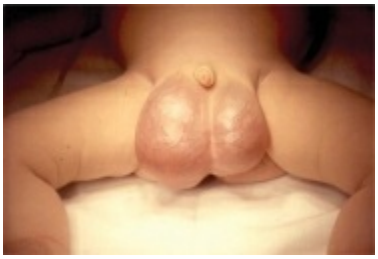
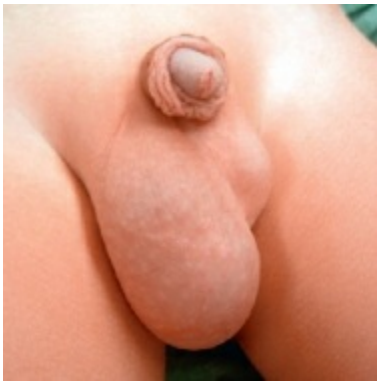
		Prel witl scr
Epispadias		Abnor ure surf
Hernia		Inguin of t thr to p vag wit The and pre imr imr bec for
Hydrocele		Enlarg due bet visc vag sur may non Tra clea test *Nor*

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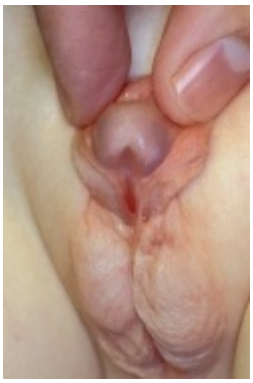
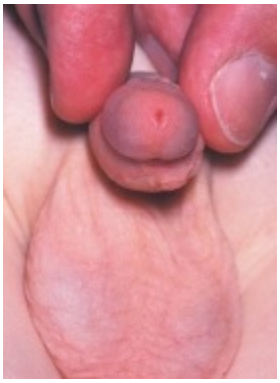
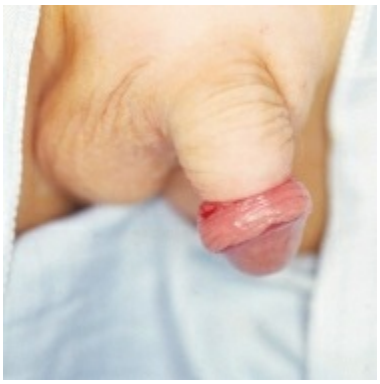
Hypospadias		Congen inc of a cor fore abn the the pen glan pen junc Ger on exc dor
Meatal stenosis		Recurr mea exp env ure trau fro or u
Paraphimosis		Urolog For glan pro swe rest Me mar aro pen foll the dov the glan sur inte

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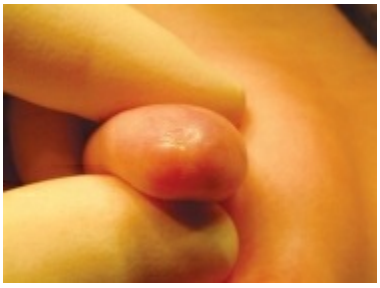
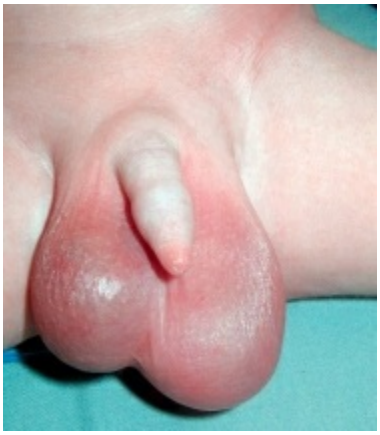
Phimosis		Delaye pre the the inc the afte Fig. chr cau phi
Testicular torsion		Urolog Twi spe **sud uni scr** swe of t obs org lea **inf** ass and sho refl (pa of t lie i pot Occ old Ma new pre non by u ute to p ind
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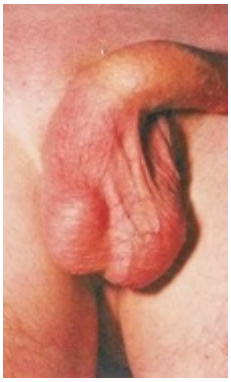
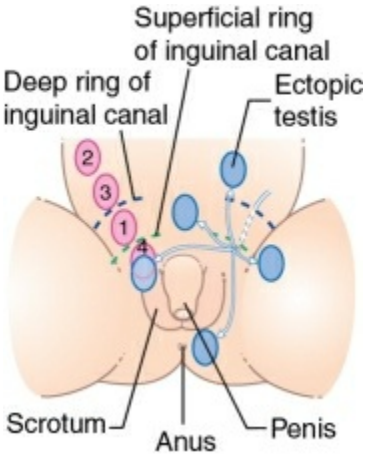
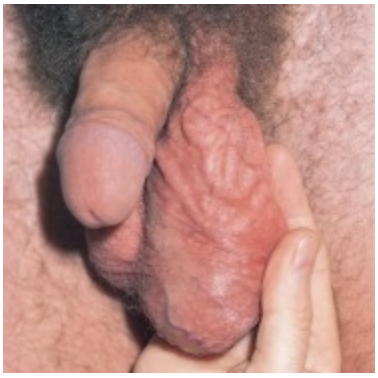
		area lum trar Ger gen inci can in 1 con 15–; 4.5 in v Am test tim hist
Undescended testis/cryptorchidism		Testis i be r sac; the patl ring *retr* *ecto* *con* *fem* eva age 16.3 gen intr Bila in a con of s diff adr Acquir wh pre the upv be b scr
Varicocele		Dilatio vei wit pre spe wo ass con mo stra con patl

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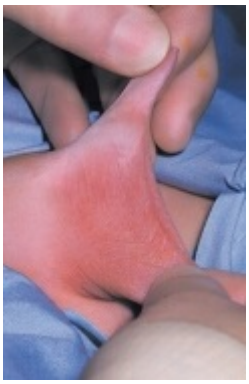
Webbed penis		Congen invo obs ang nor pen by a scro to p

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EVIDENCE-BASED PRACTICE TIP

Acute onset of scrotal pain is presumed to be testicular torsion until proven otherwise and is a true urologic emergency. Ischemia longer than 8 hours will attribute to testicular atrophy; thus, the best course of action is to restore testicular blood flow through manual detorsion (temporary relief) or surgical exploration. [22, 23]

Summary of examination

- Routine examination of the genitourinary system is expected at all routine well visits. It is an opportunity to discuss age-appropriate normal growth and development, provide anticipatory guidance, and identify any abnormality or pathology early in development. Be familiar with the wide variations of "normal."
- Be mindful and respectful of cultural variations in discussion of the genitourinary development and examination. Ask permission to discuss and examine.
- Prepare the child/adolescent by discussing what the exam will entail and provide proper draping as appropriate; be mindful of exposing child/adolescent longer than necessary. Talk through the exam and provide reassurance of normal development.
- Adolescents will be given the opportunity to have the exam performed without parents in the room, and informed consent/confidentiality policy will be discussed.
- Obtain a thorough history including gestational history, birth history, family history, and voiding/elimination history (see Information Gathering table).
- Assess penis and note foreskin status, penile size, curvature, and meatal location.
- Physiologic phimosis is expected until about 6 years of age. Avoid forceful retraction of foreskin. Discuss care of the uncircumcised penis with the family. Refer to pediatric urology if pathologic phimosis is unresponsive to steroid ointment trial.

- Assess scrotum and testes at every routine well visit. Determine if palpable testes are descended, retractile, undescended, ascending, or ectopic. Assess testicular size, shape, and consistency and note the presence of any masses or pain. Refer to pediatric urology if testes are undescended by 9 months of age or impalpable at any age.
- Perform a detailed scrotal and testicular examination for any scrotal mass or pain.
- Evaluate rectum, anus, and lumbar sacral region for dimples, clefts, or tufts of hair.
- Report and follow up on any suspicion of sexual abuse from history or physical exam.

DOCUMENTATION

Term infant at 1 month of age

External genitalia: nl uncircumcised male, foreskin partially retractable revealing meatus at tip of penis, ∅ discharge noted, nl urinary stream, testes ↓↓×2 of nl size/shape/consistency, ∅ hernia/hydrocele, back nl w/o pits/dimples, rectal sphincter nl.

nl, Normal; ↓↓, descended bilaterally.

DOCUMENTATION

Healthy 12-year-old male

External genitalia: nl Tanner stage 2 circumcised male, urethral meatus at tip, ∅ discharge noted, normal scrotum with testes ↓↓×2 of nl size/shape/consistency, ∅ masses palpated, (–) inguinal hernia exam, back nl w/o pits/dimples, rectal sphincter nl.

nl, Normal; ↓↓, descended bilaterally.

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CHAPTER 17

Male and female breast

Erica Bisgyer Monasterio, Naomi A. Schapiro

Male and female breast development is similar until puberty; therefore the examination of both boys and girls will be reviewed in this chapter, along with pubertal changes expected for both genders. The primary intention of the breast exam in children and adolescents is to recognize normal variants, monitor development, and identify nonmalignant pathology. Making assessment of the breast part of the routine physical examination in preadolescents gives the parent or caregiver and child an opportunity to voice any concerns and fosters a dialogue about reproductive health and puberty. It is important to respect privacy in performing the examination of the breast in all children and adolescents. Current evidence and recommendations related to the efficacy of breast examination focus on the detection of breast cancer, which is an exceedingly rare diagnosis in adolescents; therefore, child and adolescent breast exam recommendations are based on expert practice rather than evidence.[1]

TABLE 17.1

Female Breast Development Sexual Maturity Rating

Sexual Maturity	Breast Findings	Areola and Nipple Findings

Rating (SMR)	Breast Findings	Areola and Nipple Findings
SMR stage 1	Prepubertal; no glandular tissue	Conforms to general chest line
SMR stage 2	Breast bud; small amount of glandular tissue	Areola widens
SMR stage 3	Larger and more elevation; extends beyond areolar parameter	Areola continues to enlarge but remains in contour with breast
SMR stage 4	Larger and more elevation	Areola and papilla form a mound projecting from breast contour (half of teens; in some cases persists into adulthood)
SMR stage 5	Adult size (variable)	Areola part of breast contour, nipple projecting above areola

Data from Neinstein LS: *Adolescent Health Care: A Practical Guide*, ed 5, Philadelphia, 2008, Lippincott Williams & Wilkins.

Embryologic development

A mammary ridge forms from the ectodermal layer on day 20 of embryonic life and extends from the forelimb to the hind limb. In the sixth week of fetal life the nipple and areola form over a bud of breast tissue that is composed of the primary mammary ducts and a loose fibrous tissue or stroma. Fifteen to twenty-five secondary buds then develop and bifurcate into tubules, forming the basis of the duct system.[2] Each duct, as it develops, opens separately into the nipple.

Physiologic variations

At birth, the breasts of both male and female infants may be swollen because of the maternal estrogen effect (Fig. 17.1). An unusual but normal finding in the newborn is the secretion of a milklike substance, also known as "witch's milk," for 1 to 2 weeks (Fig. 17.2).[3] Male and female breast tissue is similar until puberty. Occasionally, a prepubertal male or female develops an enlargement of one or both breasts, which involves a soft, mobile, subareolar nodule of uniform consistency.[2] In general, the nipple and areola are not developed or pigmented with such an

condition usually regresses spontaneously within weeks to months, and in the absence of other secondary sexual characteristics, a biopsy is not indicated.[2] If other signs of puberty appear, then these changes could be the first sign of *precocious puberty,* in which case referral and further diagnostic work-up are indicated.

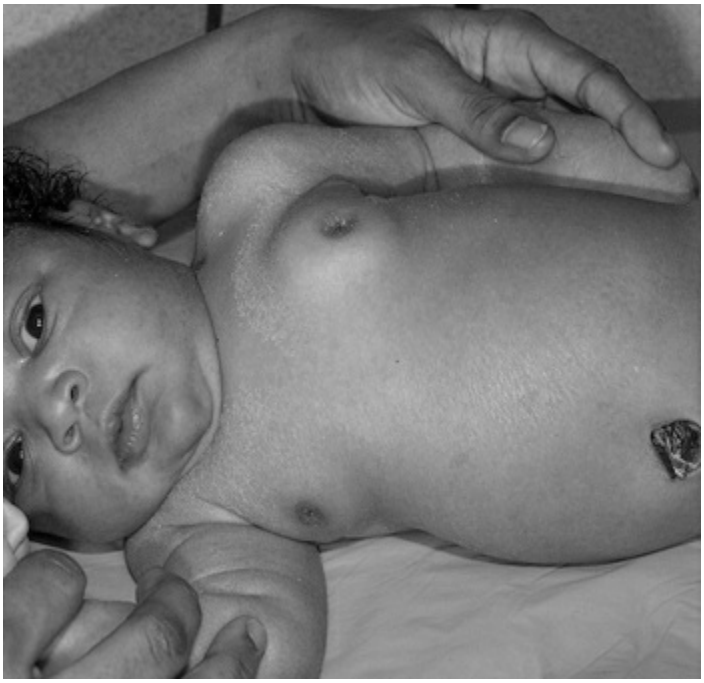

FIGURE 17.1 Estrogen effect in the newborn. Source: (From Shah BR, Laude TA: *Atlas of Pediatric Clinical Diagnosis,* Philadelphia, 2000, Saunders.)

FIGURE 17.2 Witch's milk. Source: (From Clark DA: *Atlas of Neonatology*, ed 7, Philadelphia, 2000, Saunders.)

Supernumerary nipples may arise from the mammary ridge and be present at birth (Fig. 17.3A). They are often raised, generally require no treatment, and become imperceptible over time. There is a weak association between supernumerary nipples and renal and cardiovascular abnormalities in white newborns.[3] In females, supernumerary nipples will on rare occasion develop a small amount of breast tissue during puberty (see *polymastia* in Table 17.2; see Fig. 17.3B). The adolescent may elect to have cosmetic surgery for removal of the supernumerary breast tissue. Widespread nipples are defined as a nipple spread of greater than 25% of the chest circumference and may be associated with congenital disorders such as Turner syndrome.[3]

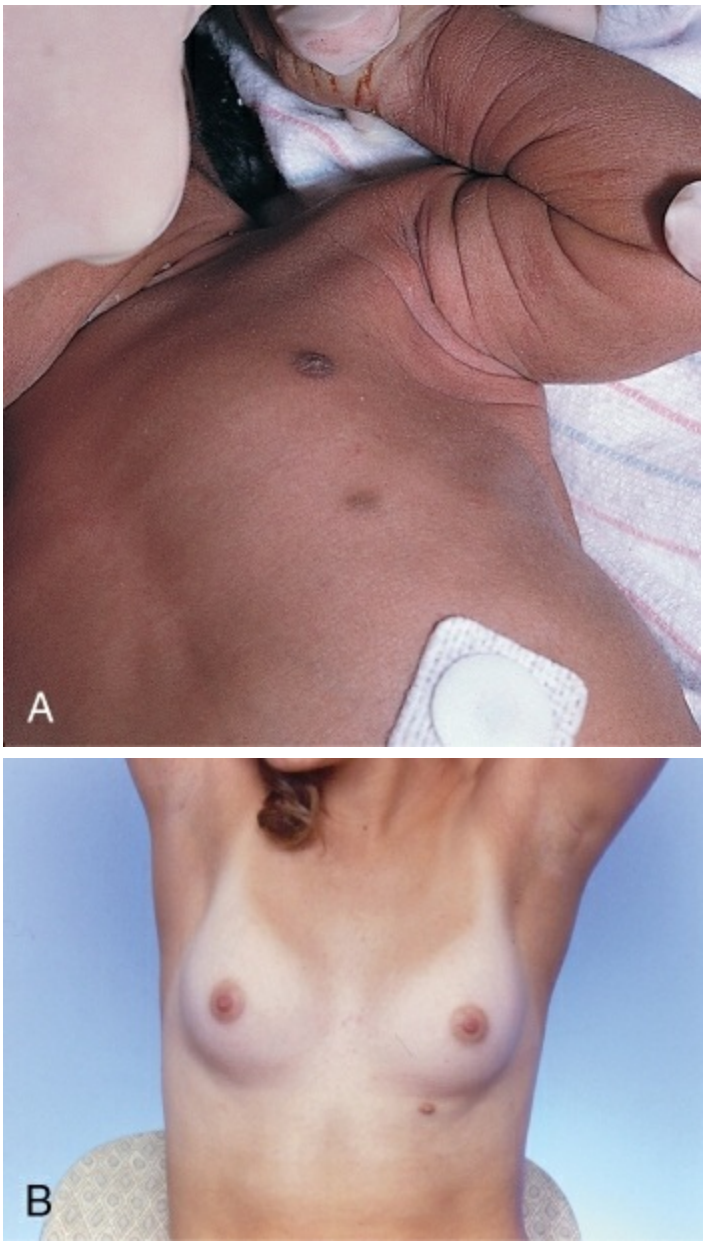

FIGURE 17.3 (A) Supernumerary nipple. (B) Supernumerary nipple located in the inframammary fold of the left breast of an adolescent female. Source: ([A] From Eichenfield L, Frieden IJ, Zaenglein AL: *Neonatal Dermatology,* ed 2, Philadelphia, 2009, Saunders; [B] From van Aalst JA, Sadove M: Treatment of pediatric breast problems. *Clin Plast Surg* 32(1):65-78, 2005.)

TABLE 17.2
Conditions of the Male and Female Breast

Condition	Description

Polymastia	Breast tissue that develops around supernumerary nipples; although clinically insignificant, this tissue may become uncomfortably engorged postpartum
Fibroadenoma	Most common benign breast tumor excised during adolescence; firm, rubbery, mobile, nontender; ranges in size from <1 cm to 10 cm; usually discovered by the adolescent
Amastia	Absence of breast tissue. *Athelia,* absence of nipple; often connected to a chest wall defect or more extensive congenital anomalies
Breast atrophy	Most commonly caused by significant loss of fat and glandular tissue as a result of eating disorders but also may occur in premature ovarian failure, androgen excess (tumors or ingested steroids), and chronic diseases that lead to significant weight loss
Macromastia	Exceptionally large breasts (Fig. 17.11); definition of "normal" breast size is difficult to determine, but this condition is associated with obesity and a strong familial incidence; 80% of cases begin in adolescence with teens complaining of psychologic effects as compared with the more common complaints of breast, shoulder, and back pain among adults
Virginal or juvenile hypertrophy	May be an abnormal response of the breast to normal estrogen levels; occurs perimenarchally; breasts may enlarge as much as 30–50 lbs.
Nipple discharge	Multicolored or sticky discharge may be caused by duct ectasia Serous or serosanguineous discharge may be caused by intraductal papilloma, benign changes, duct ectasia, or rarely cancer Milky discharge (galactorrhea) may be the result of hormonal imbalance, pregnancy, or past abortion; medications such as combination oral contraceptive, antipsychotics and some selective serotonin uptake inhibitors (SSRIs) or illicit drugs such as marijuana; pituitary tumors; or it may result from stimulation by sexual partners Purulent discharge due to mastitis Watery discharge due to papilloma or cancer

Physiologic variations in the male breast

At puberty the ductal and periductal mesenchymal breast tissue of boys proliferates under the influence of estrogens, with later involution as testicular androgens rise to adult levels.[2] Male estradiol levels triple during puberty, and androgens ultimately increase 30 times. Until recently, it was believed that peak estrogen levels occurring prior to peak androgen levels resulted in *physiologic gynecomastia,* a benign increase in glandular and stromal breast tissue in pubertal males (Fig. 17.4). However, the current understanding identifies leptin, a hormone made by adipose cells to regulate energy balance, as the hormone most likely responsible for pubertal gynecomastia.[2] Most circulating estrogens are produced outside of the testes, and an increase in fatty tissue, as in obesity, may lead to a higher incidence of *gynecomastia* (Box 17.1).

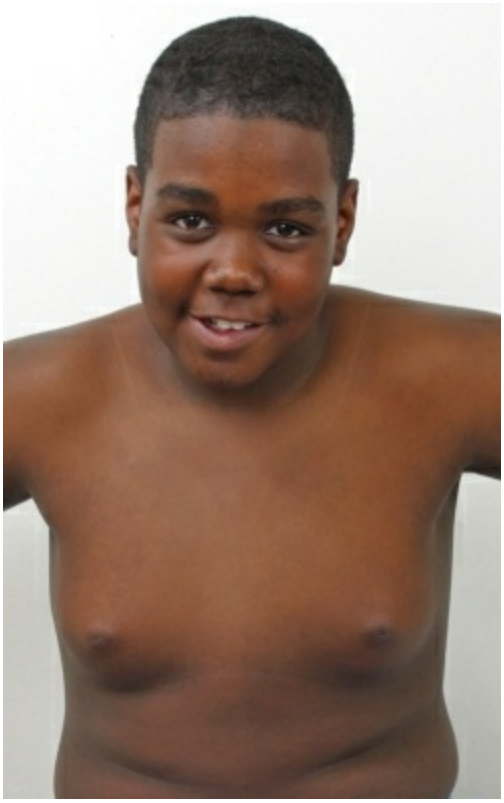

FIGURE 17.4 Gynecomastia.

BOX 17.1

Classifications of Gynecomastia

Type I: One or more subareolar nodules, freely movable
Type II: Breast nodules beneath areola but also extending beyond the areolar perimeter
Type III: Resembles female breast development stage 3 (see Table 17.1)

Data from Neinstein LS: *Adolescent Health Care: A Practical Guide*, ed 4, Philadelphia, 2002, Lippincott Williams & Wilkins.

At 14 years of age, up to 64% of adolescent males have some degree of gynecomastia, with only 4% of adolescent males having severe gynecomastia that persists into adulthood.[2] Approximately 50% of males experience the onset of gynecomastia at sexual

maturity rating (SMR) stage 2 of male genital development, another 20% at SMR stage 1, 20% at SMR stage 3 of male genital development, and 10% beginning at SMR stage 4 (see Chapter 16). Recent research shows an association between pubertal height velocity and the development of benign pubertal gynecomastia in a cohort of boys with delays in development, suggesting a causal relationship between acceleration of growth and development of gynecomastia in peripubertal males.[4] In general, adolescent males can be reassured that most cases of *physiologic gynecomastia* will resolve spontaneously within 12 to 18 months, although it can rarely persist after sexual maturity.[3] Treatment alternatives for persistent *gynecomastia,* including pharmacologic and surgical options, may be appropriate to discuss with youth and families when this condition does not resolve or is particularly distressing.

Gynecomastia, although a normal variant, is also present in relation to hormone imbalances due to thyrotoxicosis, cirrhosis, adrenal and testicular neoplasm, primary hypogonadism, chromosome abnormalities such as Klinefelter syndrome, and severe malnutrition.[2] In addition, prescription drugs such as ranitidine, omeprazole, ketoconazole, metronidazole, antiretrovirals, digoxin, spironolactone, phenothiazines, and some illicit drugs (e.g., marijuana, anabolic steroids, amphetamines, and alcohol) can cause gynecomastia.[3]

Breast development, an initial sign of secondary sexual development in males and females, is readily apparent to family members and peers, often eliciting unwanted comments as to presence or absence and size of breast tissue. Breast development is a common issue for teasing, bullying, and sexual harassment in middle school. The health care provider should be mindful that sexual harassment is a major issue for teens, often jeopardizing a teen's mental health and school performance. If the preteen or young adolescent seems particularly concerned or uncomfortable with the health history about breast development or the breast examination, gentle exploration of the issue by the provider, including queries as to teasing or bullying, or unwanted attention from family members, should be explored, and the adolescent should be referred for counseling if indicated.

Physiologic variations in the female breast

Thelarche, or the beginning of female breast development, is usually the first sign of puberty in girls and occurs between 8 and 13 years of age, on average at the age of 11.2 years (Table 17.1 and Fig. 17.5).[1] Full breast development at SMR 5 signals the end of puberty in females. Breast development during puberty involves both multiple hormones and the binding of hormones to breast tissue. Estrogen, especially estradiol, influences ductal development, whereas progesterone influences additional lobular alveolar development.[2] Thyroxine and corticosteroids are also involved.

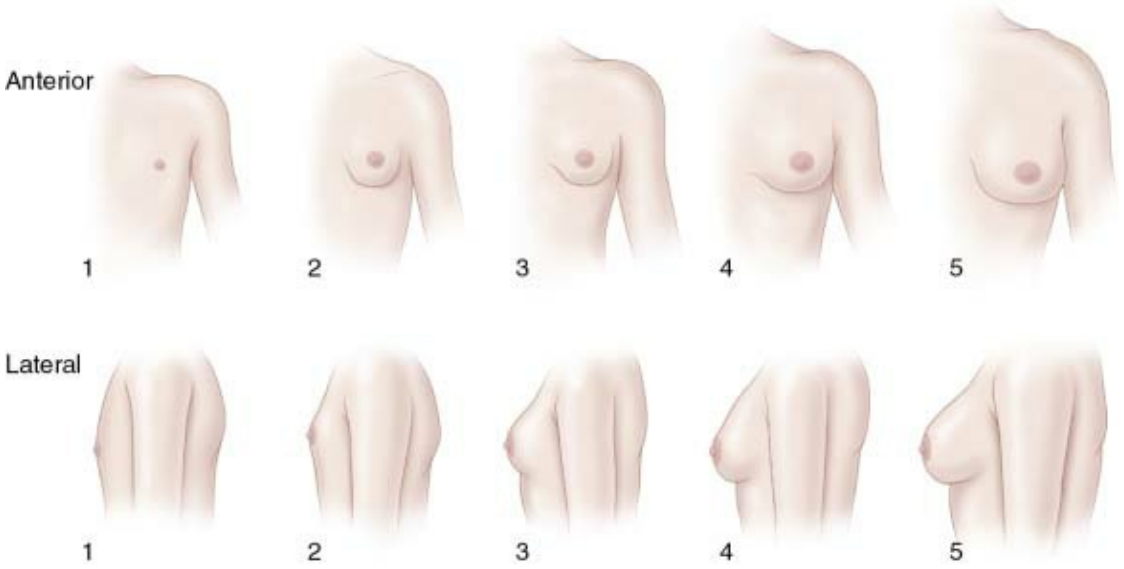

FIGURE 17.5 Normal female breast development, from Tanner stage 1 (prepubertal) to Tanner stage 5 (adult breast). Source: (From Herring J: *Tachdjian's Pediatric Orthopaedics,* ed 4, Philadelphia, 2008, Saunders.)

Up to 90% of newborns of both sexes commonly have a palpable nodule under the nipple and areola, which is thought to be related to exposure to estrogens through the placenta, and which generally regresses within 6 to 12 months.[2] *Premature thelarche* is defined as breast development without other signs of puberty, occurring before 7.5 years. Although isolated *premature thelarche* is usually benign and self-limited, the examiner should be sure to evaluate for other signs of precocious puberty. (See Chapter 5 for a discussion on endocrine-disrupting chemicals.)[2]

Anatomy and physiology

Mature female breast tissue at SMR stage 5 extends from the second or third rib to the sixth or seventh rib, and from the sternal margin to the midaxillary area with the nipple located centrally, surrounded by the areola. The breast is composed of glandular and fibrous tissue and subcutaneous and retromammary fat (Fig. 17.6). Fifteen to twenty lobes radiate around the nipple, and each lobe is divided into 20 to 40 lobules of milk-producing acini cells that empty into lactiferous ducts.[2] These cells are small and inconspicuous in the nonpregnant, nonlactating woman. A layer of subcutaneous fibrous tissue provides support for the breast, as do the suspensory ligaments. The muscles forming the floor of the breast are pectoralis major, pectoralis minor, serratus anterior, latissimus dorsi, subscapularis, external oblique, and rectus abdominis. Vascular supply comes from the internal mammary artery and the lateral thoracic artery. The lymph system drains to the anterior axillary, subscapular, and supraclavicular nodes.

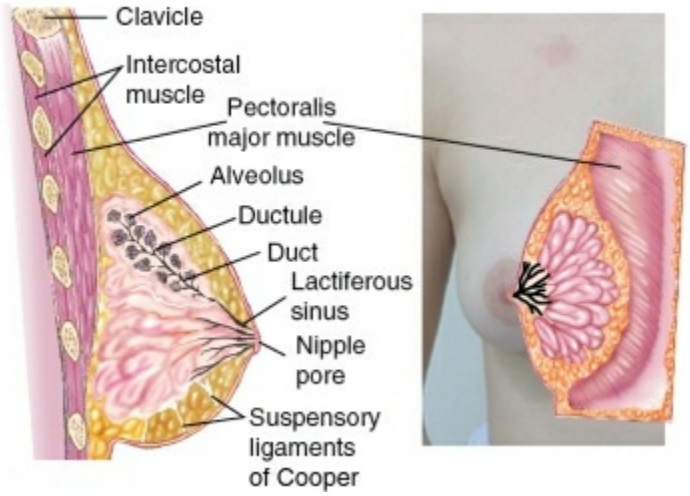

FIGURE 17.6 Anatomy of the breast.

Family, cultural, racial, ethnic, and other considerations

Cross-sectional studies in the past 20 years have found variations in

the onset of puberty by race, ethnicity, and weight. A recent longitudinal study of breast development in more than 1200 girls (childhood to early adolescent) has confirmed these observations, finding variation in thelarche by both race/ethnicity and body mass index (BMI).[5] Biro et al. found that thelarche, as defined by the age at which girls reach SMR stage 2 of breast development, occurs at an average age of 8.8 years in African American girls, 9.3 years in Hispanic girls, and 9.7 years in non-Hispanic white and Asian girls.[5] Development was also influenced significantly by BMI, with girls above the 50th percentile reaching SMR 2 at an earlier age. Regardless of race/ethnicity, a BMI at or above the 85th percentile was associated with earlier breast development in all groups and was found to be the strongest predictor of earlier age of thelarche.

System-specific history

The Information Gathering table reviews the important information to be gathered for male and female breast assessment at key developmental stages, and for adolescent females presenting with breast symptoms or concerns.

Information Gathering for Male and Female Breast Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preadolescent girls and boys	Breast tenderness or breast buds noted? At what age did breast development begin? Any pain in breast? Other signs of puberty noted? Axillary or pubic hair? Concern about breast development?
Female adolescents	Any concerns about breast health, breast development or breast size? Last menstrual period? Contraceptive use? Breast tenderness or pain noted? Relationship of breast discomfort or pain to menstrual cycle? For female adolescents with large breasts: any back or shoulder pain? Previous pregnancy? Any discharge from nipple? Any lump noted in breast or axilla? Redness or irritation on skin?

Physical assessment

Inspection and palpation

Prepubertal breast

Prepubertal breasts are easily inspected and palpated while examining the chest to assess cardiovascular and respiratory status. The health care provider should note any masses or pain, nipple discharge, or signs of premature thelarche, or breast development.

Adolescent male

Pubertal breasts are inspected with the male adolescent supine with his hands behind his head. Place the pads of the three middle fingers at the margins of the breast. Palpate the breast bilaterally for the presence of adipose tissue or breast buds if evaluating for gynecomastia. Note any nodular tissue or firm rubbery masses. In conditions such as a *lipoma* or *dermoid cyst*, the mass is usually noted to one side of the areola.

Adolescent female

There is controversy about the age at which health care providers should perform routine breast exams in women, or whether they should be performed as part of a preventative visit at all. The American Cancer Society (ACS) does not recommend the clinical breast examination (CBE) for women of average risk at any age.[6] The American Congress of Obstetricians and Gynecologists (ACOG) are under review but currently recommend including CBE as part of a routine physical exam every 1 to 3 years for women age 20 to 39 years and annually after age 40 years.[7] The US Preventive Services Task Force (USPSTF) updated recommendations for mammography and retained the recommendation of the CBE to begin at 40 years of age.[8]

Although malignancies in adolescents are rare and more often due to metastatic disease than to a primary breast malignancy, adolescents and young adults do present with breast concerns that must be addressed. Discussion about breast development in early to middle female adolescents provides an opportunity for reassurance and education about normal breast findings and variations of normal. Most adolescent breast complaints are related to normal developmental changes and can be evaluated through obtaining an adequate history and physical exam. When imaging is indicated, ultrasound is the primary modality because adolescent breast tissue

tends to be too dense for mammography.

Red Flag in Breast Assessment

Concerns requiring a CBE with or without imaging include complaints of breast masses, breast pain, and/or nipple discharge.

For adolescents with a family history consistent with hereditary breast cancer, CBE paired with screening ultrasound beginning at 25 years old is recommended. Screening mammography and/or magnetic resonance imaging (MRI) is recommended for women beginning at 30 to 35 years of age depending on the earliest onset of known breast cancer in the family.[9] Young women with a history of irradiation to the chest (e.g., treatment of Hodgkin disease) have a breast cancer risk comparable to those with hereditary breast cancer risk. For these young women, annual mammography with MRI is recommended starting at 25 years of age or 8 to 10 years after completion of radiation therapy.[10] Some authors recommend performing and teaching about *breast self-exam (BSE)* as a way of increasing young women's investment in their own health care,[3] and experienced adolescent providers believe there is a value in teaching adolescents about the texture of glandular breast tissue and normal findings around and under breast tissue, such as ribs and ligaments, which may increase comfort with their own developing bodies. However, there is limited evidence about the best time in adolescent or young adult development to teach breast self-awareness.

Clinical breast examination

In a full adolescent or young adult breast exam, the breasts are initially inspected with the patient sitting, disrobed to the waist, in the following positions: arms extended overhead, hands pressed against hips or against each other, and leaning forward from the waist (Fig. 17.7). *These three positions are most helpful if examining for a lump or mass.*[3] The provider will have to weigh the benefits of full breast inspection in young adult women who are at low risk for breast cancer against the aversion most teens or young adults have

to being undressed in front of a provider.

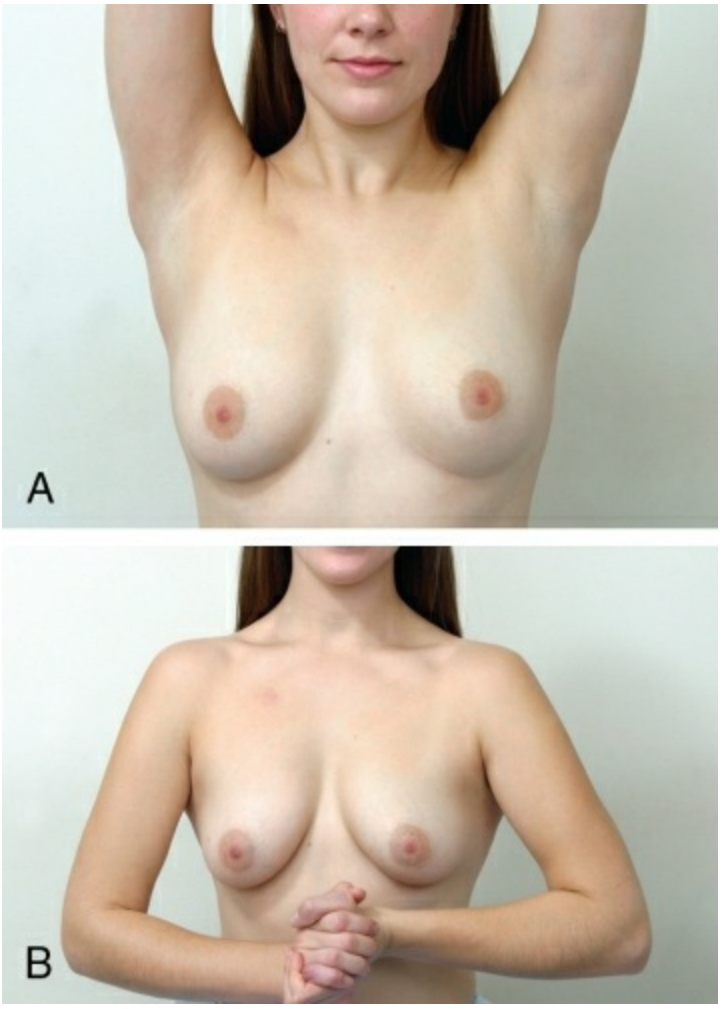

FIGURE 17.7 Inspection of the breast. (A) Arms extended overhead. (B) Hands pressed together.

Although the breasts can be palpated sitting and supine, the preferred position for the adolescent is supine, with one arm under her head. With the adolescent supine, the breasts are in a more stable position, and touching of the breast with the nondominant hand of the examiner can be minimized. For purposes of examination, the breast is divided into four quadrants and a tail that extends into the axilla (Fig. 17.8).

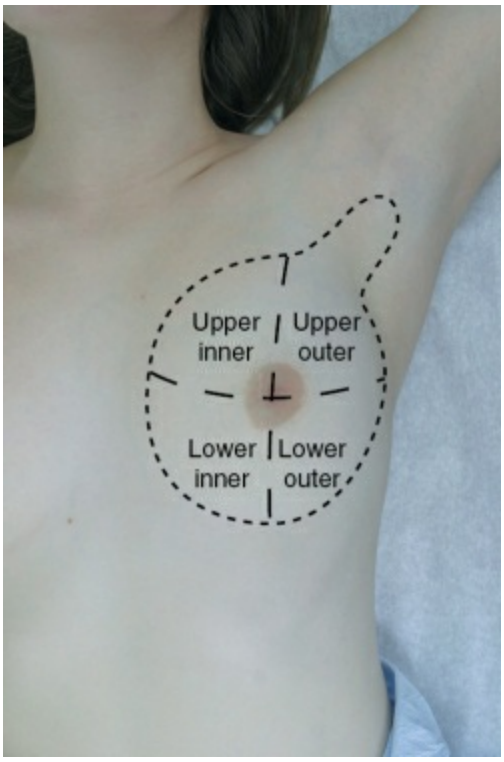

FIGURE 17.8 Quadrants of the left breast, tail of Spence.

The breasts should be palpated with the pads (not tips) of the middle three fingers with the fingers palpating over the breast tissue from the sternal margin to the midaxillary area and from the second or third rib to the sixth or seventh rib at the base of the breast. Palpation proceeds stepwise with the fingers to ensure a thorough assessment of the breast tissue. The preferred methods for palpation of the breast are illustrated in Fig. 17.9. The examiner should include palpation of the *tail of Spence* in the axilla in addition to the supraclavicular and infraclavicular regions. When palpating the breast, the examiner should use the dominant hand and stabilize the breast, if necessary, with the nondominant hand. Place the pads of the middle fingers at the margins of the breast to begin palpation, and note any masses, pain, nipple discharge, or signs of premature breast development. If firm, rubbery tissue or a nodular mass is noted, fine palpation is required for accurate sizing and to distinguish among fatty tissue, cysts, and other nodular masses.

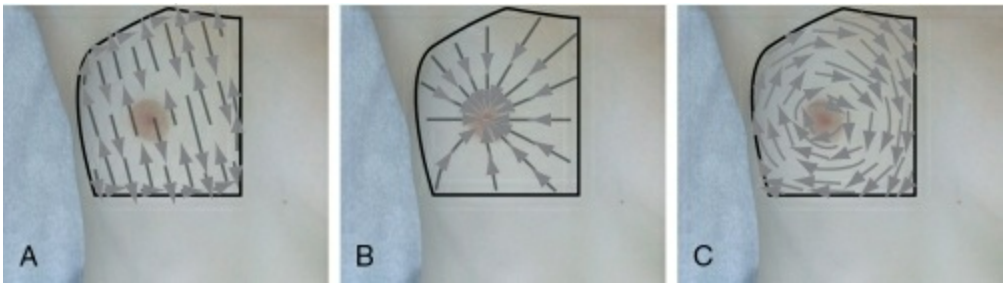

FIGURE 17.9 Methods of breast palpation. (A) Palpation strip method. (B) Palpation wedge method. (C) Palpation circle method.

It is important to incorporate patient education into the breast examination for the young female adult. During the examination, ask permission to guide the adolescent's hand onto her breast, pointing out landmarks and normal findings in the developing breast, such as the thicker ridge of breast tissue often found in the inferior aspect of the breast and the nodularity of normal adolescent breast tissue especially in the upper outer quadrant. Have the adolescent also feel the ribs underneath the medial border of the breast because this normal finding is often mistaken by adolescents for a breast lump.

PEDIATRIC PEARLS

Asymmetrical breasts are common early in pubertal development in both adolescent males and females (Fig. 17.10).

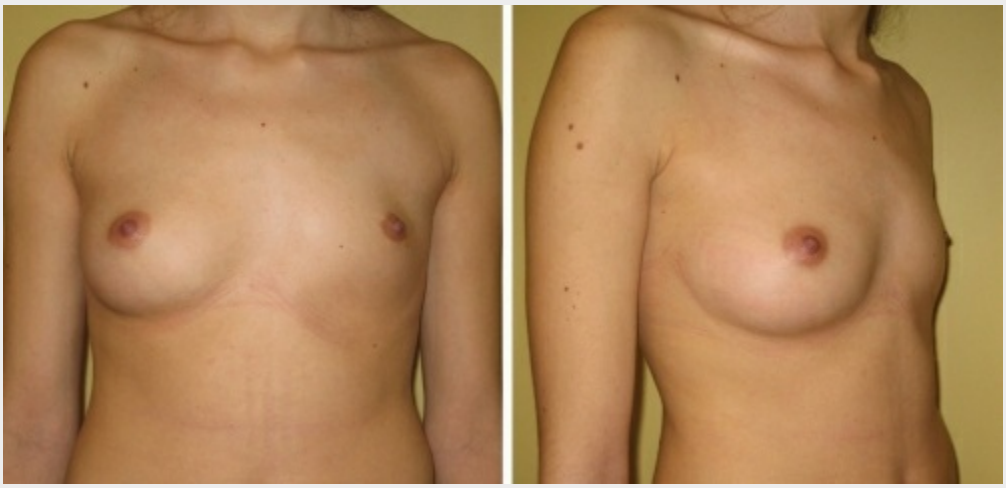

FIGURE 17.10 Asymmetrical breasts. Source: (From Khouri

R, Del Vecchio D: Breast reconstruction and augmentation using pre-expansion and autologous fat transplantation. *Clin Plast Surg* 36(2):269-280, viii, 2009.)

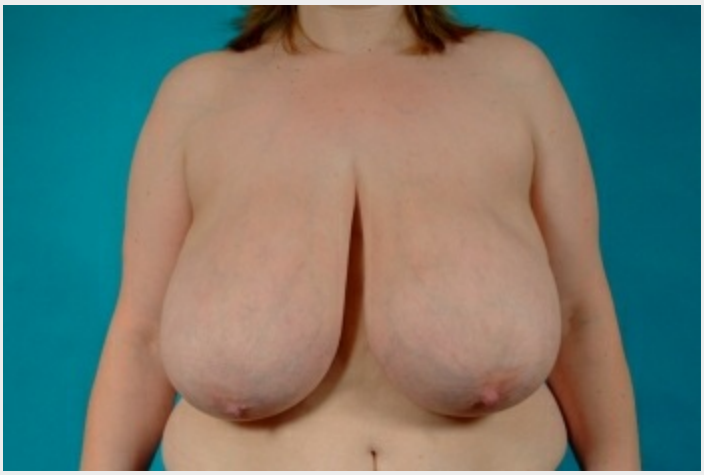

FIGURE 17.11 Profound macromastia. Source: (From Hammond D: *Atlas of Aesthetic Breast Surgery,* London, 2009, Saunders.)

Breast self-awareness

Teaching *BSE* has become replaced by the concept of teaching breast self-awareness.[7] Breast self-awareness may assist the adolescent in accepting her body, increase comfort with the breast examination, and provide an opportunity to reinforce or correct information the adolescent may have received from health classes, teen magazines, or female relatives about breast health. However, the USPSTF recommends *against* teaching BSE for adolescents or women of any age.[11] Both the ACS and ACOG recommend teaching *breast self-awareness* to women age 20 years and older.[7, 12] This includes assisting women to become familiar with their breasts' normal feel and appearance, focusing on the importance of self-detection and early assessment of symptoms. As noted previously in the section on clinical breast exams, there is currently no consensus or evidence-based recommendation on teaching such awareness to younger adolescents.

Breast conditions

The following are variations of normal that occur with stages of breast development:

- **Physiologic swelling and tenderness:** Breast lobules undergo proliferative changes due to the normal menstrual cycle, leading to pain and discomfort, swelling, and distinct masses that recede after menses.
- **Asymmetry:** In most women, one breast is slightly larger than the other, and this difference may be accentuated by asymmetrical breast development during puberty, which usually corrects by adulthood.
- **Proliferative breast changes:** Formerly called *fibrocystic disease,* many authorities now consider increased nodularity to be a variation of normal, occurring in more than 50% of women of reproductive age. Adolescents often have painless lumps, which may become tender 1 week before menses. Areas of nodularity may be a few millimeters to 1 cm in diameter.
- **Cysts:** Usually associated with few symptoms, cysts are well circumscribed, small, and freely movable masses, commonly less than 1 cm in adolescents.
- **Montgomery tubercles:** These tubercles arise from sebaceous glands associated with a lactiferous duct. They present as small, soft papules around the areola, with occasional thin, clear to brown discharge and possibly a small lump under the areola. The condition usually resolves without intervention.

Breast cancer in adolescents is extremely rare. Malignant lesions comprise only 0.9% of all surgically excised lesions in the adolescent and young adult age group. Breast variations and benign tumors are presented in Table 17.2.

Summary of examination

- Prepubertal breast examination in both male and female children is focused on the identification of normal variants and nonmalignant pathology and monitoring development.
- Focus on the developing breast may be embarrassing and uncomfortable for pubertal children and early adolescents.
- Pubertal gynecomastia is common in approximately 50% of

developing male adolescents and may be a source of concern for youths and families.

- Thelarche is the first sign of puberty in developing female adolescents.
- Breast development is often a common issue for teasing, bullying, and sexual harassment in middle school.
- There is insufficient evidence to formulate recommendations for BSE and CBE in adolescents and young adults.
- Young adult females may benefit from patient teaching on breast self-awareness, which includes the normal feel and appearance of the breasts, focusing on the importance of self-detection, and early assessment of symptoms.

DOCUMENTATION

9-year-old female

Chest: Symmetrical, lungs clear to auscultation bilaterally, breasts SMR 2, breast bud noted under R areola, warm and tender to touch, nonerythematous, L breast without swelling or tenderness.

DOCUMENTATION

14-year-old male

Chest: Symmetrical, lungs clear to auscultation bilaterally, breast SMR 2, breast buds noted under L and R areola, warm and tender on palpation.

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CHAPTER 18

Female genitalia

Erica Bisgyer Monasterio, Naomi A. Schapiro

The female genital examination is a recommended yet an underperformed part of the routine physical for pediatric and adolescent girls. Routine performance of the female genital exam allows the health care provider to build skills and familiarity with normal variants in the genitourinary system and establish a baseline from which to monitor individual development and provide information and reassurance to children, youth, and parents or caregivers. However, many health care providers are underprepared to recognize normal genital findings in the prepubescent girl and are unfamiliar with common variations.[1]

With knowledge of normal development of the female reproductive anatomy, the health care provider can incorporate the routine examination of the genitalia into well-child care. The review of systems and genital exam offer an opportunity to foster a dialogue between parent and child concerning reproductive health. For religious and cultural reasons or personal preference, parents, children, and adolescents may be more likely to request female providers for the breast and female genital exam. In nonemergent situations, it is important to honor this request, either within the practice setting or by referral, and to respect privacy and confidentiality in performing the examination and discussing findings.

Embryologic development

At 5 to 6 weeks of gestation, fetal gonads are bipotential, capable of differentiating into either a testis or an ovary. Both male and female embryos have one pair of primary sex organs, or gonads, and two pairs of ducts, *wolffian* ducts and *müllerian* ducts. During the sixth week the primordial germ cells migrate into the primary sex cords and begin to differentiate. Leydig and Sertoli cells appear in male embryos, producing testosterone and anti-müllerian hormone. In female embryos the gonads do not produce testosterone, and the gonads develop into ovaries. The wolffian ducts deteriorate, and the müllerian ducts develop into the uterus, upper vaginal tract, and fallopian tubes. The external genitalia differentiate at between 8 and 12 weeks of gestation (Fig. 18.1). Active mitosis continues and thousands of germ cells, *oocytes,* are produced. A newborn female may have 2 million primary oocytes at birth. However, after birth, no further oogonia occurs.[2]

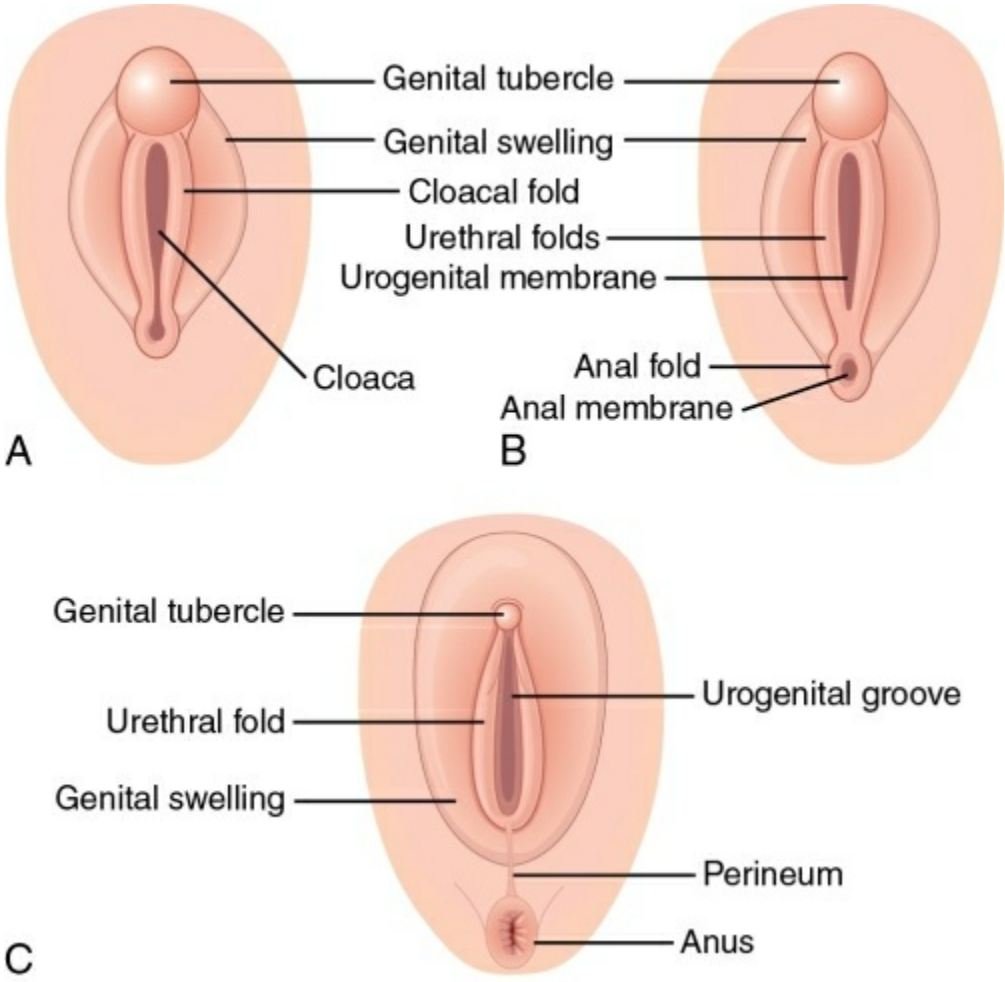


FIGURE 18.1 Development of the external genitalia. **(A)** Early development before completion of the urorectal septum. **(B)** Separation of the anus from the urogenital sinus lined by the urethral folds. **(C)** Development of the genital swelling. Source: (From Crum C, Nucci M, Lee K: *Diagnostic Gynecologic and Obstetric Pathology*, ed 2, Philadelphia, 2012, Saunders.)

Developmental and physiologic variations

In preterm neonates the *labia majora* may not cover the *labia minora,* and the *clitoris* will be prominent. Term newborns will have enlarged labia majora, which usually cover other external structures, a relatively large clitoris, and labia minora with dull pink epithelium, because of maternal estrogen effects (Fig. 18.2). A creamy white or slightly blood-tinged discharge is normal for up to 10 days after birth. The *hymen* is relatively thicker, pink-white, and redundant and may remain so up until 2 to 4 years of age (Fig. 18.3).[3]

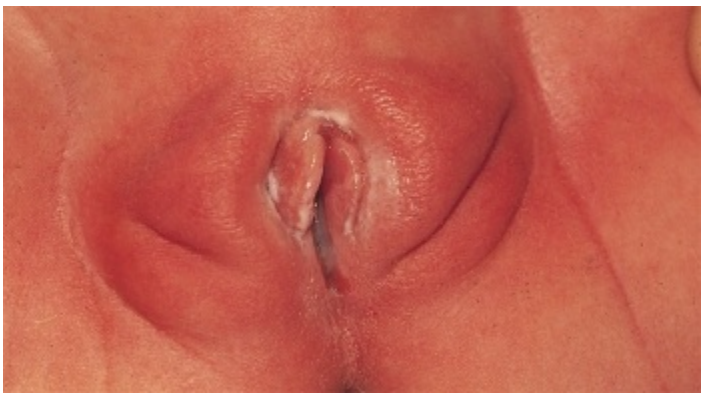


FIGURE 18.2 Newborn with estrogen effect. Source: (From Zitelli BJ, Davis HW: *Atlas of Pediatric Physical Diagnosis*, ed 4, St. Louis, 2002, Mosby. Courtesy Ian Holzman, MD, Mt. Sinai Medical Center, New York, NY.)

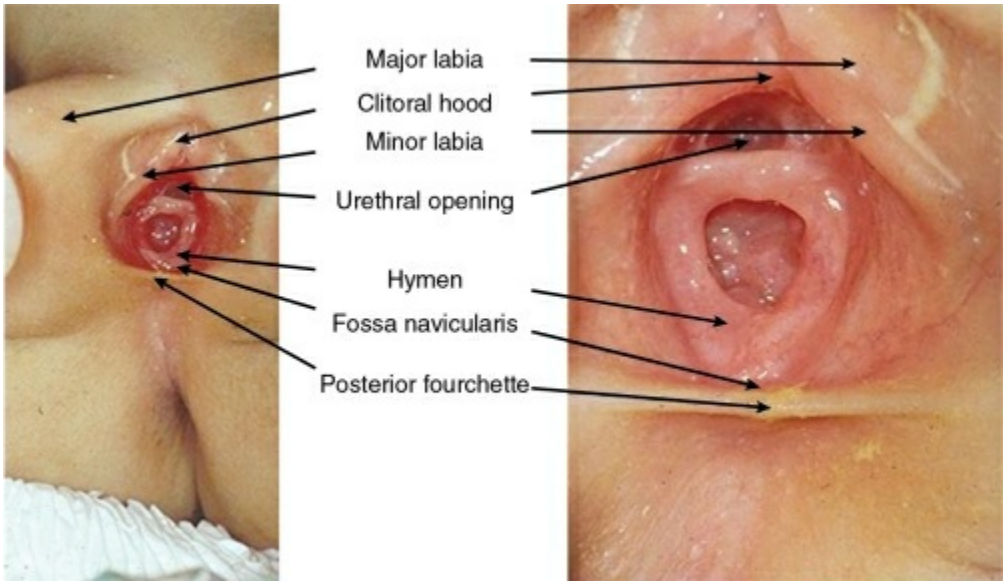


FIGURE 18.3 External genitalia of the prepubertal child. Source: (From Baren J, Rothrock S, Brennan J, et al: *Pediatric Emergency Medicine*, Philadelphia, 2008, Saunders.)

Disorders of sexual differentiation (DSD) have their genesis in early fetal development and result from developmental variations in one or more of the three components of sex determination and differentiation: chromosomal sex, gonadal sex, and/or phenotypic sex. Manifestations of some types of DSD are evident at birth in the newborn with ambiguous genitalia, and infants with ambiguous genitalia should be referred in the neonatal period to a tertiary center with a comprehensive multidisciplinary team with an ethicist.[4] Other types of DSD may become evident only in early adolescence with variations in secondary sexual development (see Chapter 16 for further discussion).

In the absence of congenital anomalies, all female infants are born with a hymen, which can present in a variety of configurations. Commonly, the hymen is *fimbriated*, *annular*, or *crescentic* (Fig. 18.4). Annular hymens are more common at birth, whereas crescentic hymens are more common in girls older than 3 years. Fig. 18.5 illustrates hymen types that are rare—*septate*, *cribriform*, and *imperforate*. Table 18.1 presents congenital anomalies in development of the female genitalia.

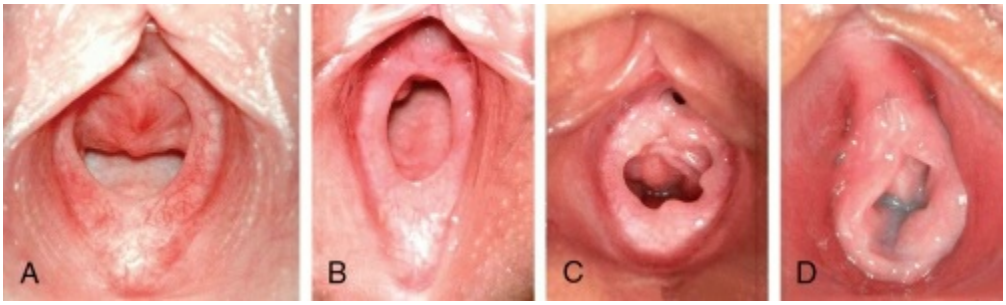


FIGURE 18.4 Variations in normal hymens. (A) Crescentic. **(B)** Annular. **(C)** Fimbriated. **(D)** Redundant. Source: (From McCann JJ, Kerns DL: *The Anatomy of Child and Adolescent Sexual Abuse: a CD-ROM Atlas/Reference*, St. Louis, 1999, Intercorp Inc.)

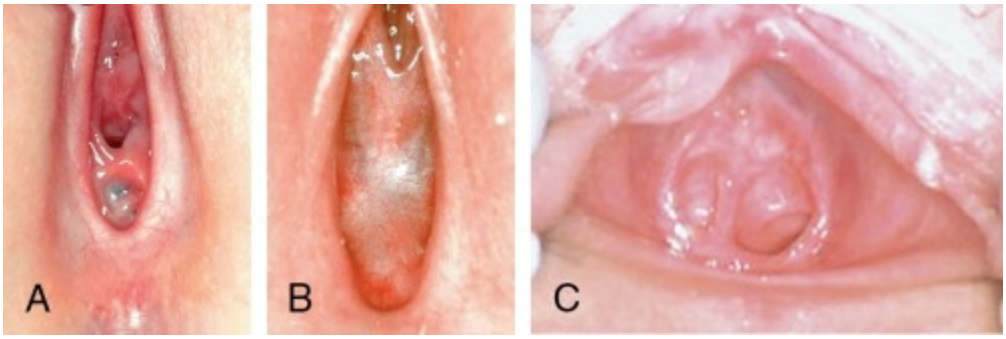


FIGURE 18.5 Abnormal hymens. (A) Cribriform. **(B)** Imperforate. **(C)** Septate. Source: (**[A** and **B]** From McCann JJ, Kerns DL: *The Anatomy of Child and Adolescent Sexual Abuse: a CD-ROM Atlas/Reference*, St. Louis, 1999, Intercorp Inc.; **[C]** From Lahoti SL, McClain N, Girardet R, et al: Evaluating the child for sexual abuse. *Am Fam Physician* 63[5]:883-892, 2001.)

TABLE 18.1
Congenital Anomalies in the Development of Female Genitalia

Condition	Description
Ambiguous genitalia	Partially fused labia, enlarged clitoris; hypospadias, bilateral cryptorchidism in an apparent male are signs of a possible intersex condition (see Chapter 16)
Hydrocolpos	Vaginal secretions collecting behind an imperforate hymen at birth may appear as small cystic mass between labia
Vaginal agenesis	Congenital absence of or hypoplasia of fallopian tubes, uterine corpus, uterine cervix, and proximal portion of vagina; occurring in

	approximately 1 in 5000 births; in approximately 10% of these births, there is a rudimentary or functional uterus Commonly diagnosed at puberty when child presents with primary amenorrhea; however, can be recognized at birth; should be diagnosed by vaginal inspection before onset of puberty

Anatomy and physiology

After the newborn period and before menarche, the clitoris is approximately 3 mm in length and 3 mm in transverse diameter. The effects of estrogen can be seen in infants, with pinker and more redundant, or wavy hymens. In prepubertal preschool- and school-age girls, hymens are thinner, redder and more sensitive to touch. The prepubertal *vagina* is rigid, nonelastic, and thin-walled, lined by columnar epithelium, which normally appears redder than the squamous epithelium lining the vagina of pubertal adolescents and adult women. During early puberty, the effects of increased circulating estrogens are seen on the vulva, including the hymen, as labia minora develop more fully and the hymen becomes thicker, pinker and more redundant.[3] Table 18.2 presents pubertal changes of the vagina.

TABLE 18.2

Pubertal Changes of the Vagina

Vaginal Changes	Prepubertal Girls	Pubertal Adolescents
Vaginal pH	6.5–7.5 (alkaline)	<4.5
Vaginal mucosa	Columnar epithelium (red)	Stratified squamous epithelium (pink); presence of columnar epithelium on ectocervix, surrounding the os
Vaginal mucous glands, discharge	Absent	Present; physiological leukorrhea usually begins at SMR 2–3
Normal vaginal flora	Gram-positive cocci and anaerobic gram-negative bacteria; gonorrhea and chlamydia focally infect vagina	Lactobacilli; yeast part of normal flora; gonorrhea and chlamydia commonly infect cervix
Vaginal length	4–5 cm	11–12 cm
External genitalia	Thin labia, rigid, nonelastic, thin-walled vagina	Thicker labia; thicker, more elastic, wavy, or redundant hymen; more

		elastic vagina

Data from Jenny C: Sexually transmitted diseases and child abuse, *Pediatr Ann* 21(8):497-503, 1992; Emans SJ, Laufer, MR, editors: *Pediatric and Adolescent Gynecology*, 6 ed. Philadelphia, 2012, Lippincott Williams & Wilkins.

SMR, Sexual maturity rating.

Adrenarche, the development of pubic and axillary hair, occurs at approximately the same time as *thelarche,* the development of the breast. The mean age of adrenarche in girls is 9 to 10 years of age but may occur as early as 7 to 8 years of age, particularly in African American girls (Fig. 18.6). Table 18.3 correlates development and *sexual maturity rating* (SMR), which includes breast development and pubic hair distribution in girls. The external genitalia and internal structures—labia majora, labia minora, hymen, vagina, ovaries, uterus—are developing under the influence of increasing estrogens, but they are not included as part of the SMR.

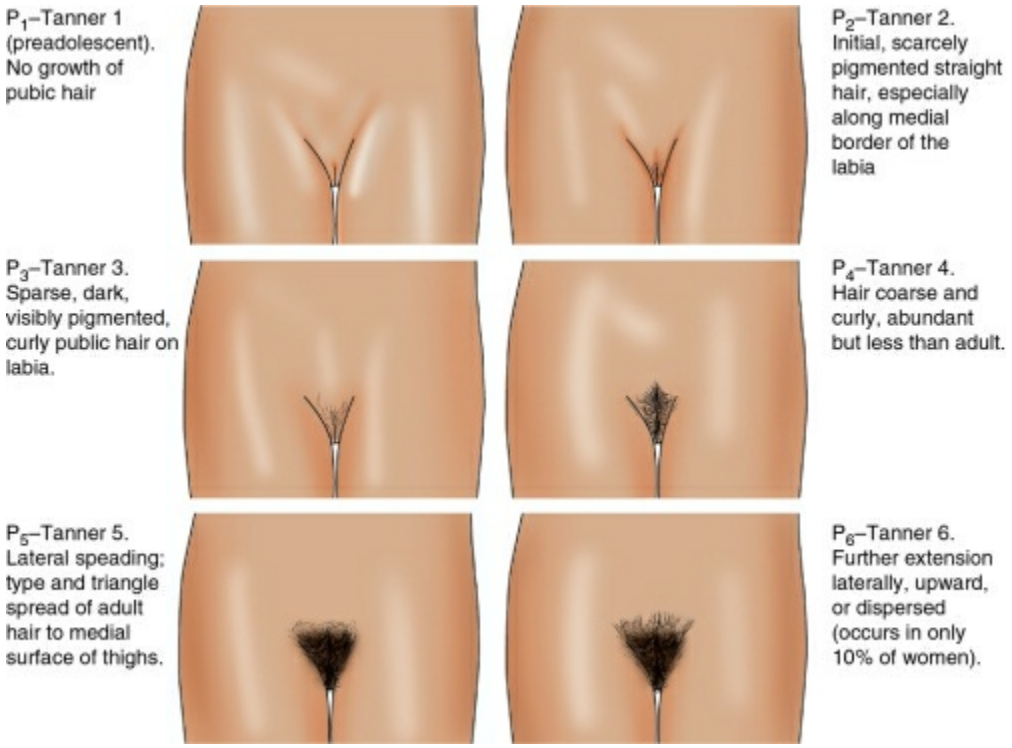


FIGURE 18.6 Sexual maturity rating in females.

TABLE 18.3
Sexual Maturity Rating

Sexual Maturity Rating/Tanner Stage	Pubic Hair Development	Genital Changes Due to Estrogenization [a]
1	No growth of pubic hair	Thicker, more elastic labia minora and hymen, change from columnar (red) to squamous (pink) epithelium in vagina (SMR 1–2)
2	Downy, scarcely pigmented straight hair, especially along medial border of labia majora	Physiologic leukorrhea (SMR 2–3)
3	Sparse, visibly pigmented curly (coarse and straight in Asian and American Indian girls) pubic hair on labia	Physiologic leukorrhea (SMR 2–3)Onset of menses (SMR 3–4)
4	Hair coarse, curly (coarse and straight in Asian and Native American girls), abundant, but less so than adult; no extension onto medial thighs	Onset of menses (SMR 3–4)
5	Lateral spreading, triangle distribution with some spread onto medial thighs	—

[a]Not part of classic SMR, but important to note in exam findings and for anticipatory guidance.

Data from Neinstein LS, et al, eds: *Neinstein's Adolescent and Young Adult Health Care: a Practical Guide,* ed 6, Philadelphia, 2016, Wolters Kluwer; Jenny C: Sexually transmitted diseases and child abuse, *Pediatr Ann* 21(8):497-503, 1992; Emans SJ, Laufer MR, eds: *Pediatric and Adolescent Gynecology*, ed 6, Philadelphia, 2012, Lippincott Williams & Wilkins.

SMR, Sexual maturity rating.

Family, cultural, racial, ethnic, and other considerations

Development and amount of pubic and general body hair varies greatly with race/ethnicity. Young women of Asian or indigenous descent tend to have less body hair than young women of European or African descent, and pubic hair development may not correlate well with sexual maturity. Many young women remove pubic hair through shaving or waxing, which may make the determination of SMR based on pubic hair distribution challenging. Genital piercings are also an increasingly common practice among adolescents.

Female circumcision, or *female genital mutilation*, is prevalent in many parts of the world, particularly in some African and Middle Eastern countries, and is considered a rite of passage and a prerequisite for marriage in some cultures. The procedure is not legal in the United States or Canada, but immigrant girls and adolescents may have had the procedure performed before migration or during a visit back to their country of origin.[5, 6] It is estimated that as many as 513,000 women in the United States may have had this procedure performed.[6] Complications include infection, hemorrhage, tetanus, difficulty in urination, sexual dysfunction, childbirth complications, and infertility.[7] There is evidence the acceptability of female genital mutilation is decreasing in some countries and with immigrant populations in the United States; however, second-generation girls and adolescents may be at risk.[6]

System-specific history

The Information Gathering table reviews information gathering on preadolescent and adolescent menstrual history and adolescent sexual history. For approach to adolescent information gathering and obtaining sensitive health information, see Chapter 4.

Information Gathering for Female Genitalia at Key Developmental Stages

Age Group	Questions to Ask
Preadolescent and adolescent	*Menstrual history:* Age at menarche, regularity of cycles, any spotting or bleeding between cycles, dysmenorrhea, family history of menstrual problems?For primary amenorrhea: Age of thelarche and adrenarche, presence or absence of secondary sex characteristics? For secondary amenorrhea: Any weight loss attempts, including restriction, binging, purging? Significant physical or emotional stress? Other chronic health conditions?
Adolescent	*Sexual history:* Any prior sexual activity, including number and gender of partners, types of activity, use of contraception and/or barrier protection? Coerced or unwanted sexual activity?History of prior examinations, prior infections or prior vaginal procedures or surgery? Other prescription medications? Use of over-the-counter medications and cosmetic products or douches? Piercing, shaving, waxing or use of depilatories for pubic and thigh hair?

Physical assessment

Examination of the newborn

Inspection and palpation

In the newborn, assess presence and size of the clitoris, patency of the vaginal orifice, presence and location of urethra, and distance between the posterior fourchette and the anus. The labia majora should be palpated for the presence of gonads or hernias, even in a normal-appearing female. Any palpable gonads are likely to be testes because ovaries rarely descend below the inguinal ring. Reassurance of a normal vaginal examination or prompt communication of any abnormal or concerning physical findings is an important part of building a trust relationship between the parent or caregiver and pediatric health care provider.

Examination of prepubertal girls

Positioning

Most young children can be examined in the *frog-leg position:* supine, with knees apart and feet touching in the midline (Fig. 18.7). For an apprehensive young child, the parent or caretaker can sit in a chair or on the examination table in a semireclined position (feet in or out of stirrups) with the child's legs straddling her thighs. Older children can be placed in adjustable stirrups. In cases of suspected trauma or abuse, a foreign body in the vagina, or other suspected structural abnormalities, *knee-chest position* can be used in a child older than 2 years of age (Fig. 18.8). Have the child rest her chest on the exam table and support her weight on bent knees, which are positioned 6 to 8 inches apart. Her buttocks will be held up in the air, and her back and abdomen will fall downward. In this position, using a penlight or an otoscope head for magnification and light, the examiner can visualize the lower vagina, and in prepubertal girls often the upper vagina. Lateral separation of the labia will be required to visualize the hymen (Fig. 18.9).

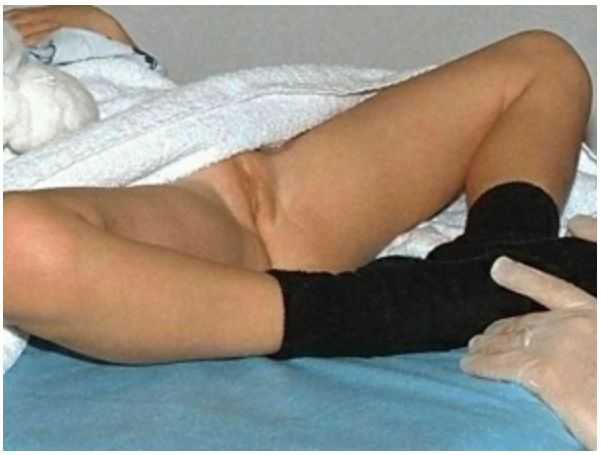


FIGURE 18.7 Frog-leg positioning. Source: (From McCann JJ, Kerns DL: *The Anatomy of Child and Adolescent Sexual abuse: a CD-ROM Atlas/Reference*, St. Louis, 1999, Intercorp Inc.)

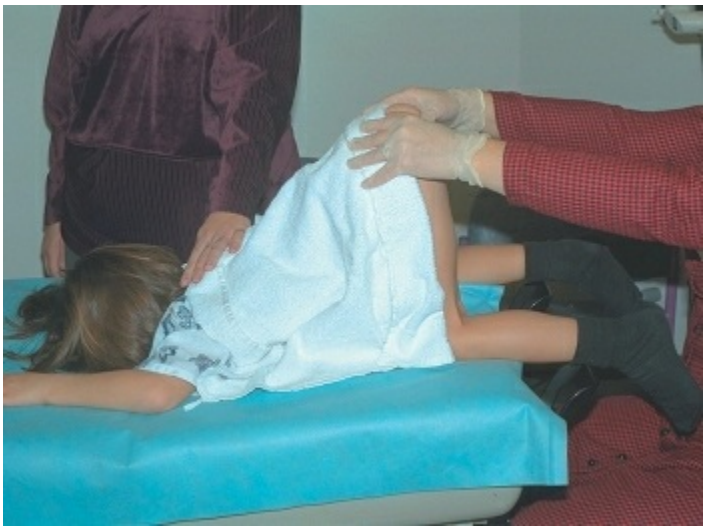


FIGURE 18.8 Knee-chest positioning. Source: (From Gall JA, Boos SC, Payne-James JJ, et al: *Forensic Medicine*, London, 2003, Churchill Livingstone.)

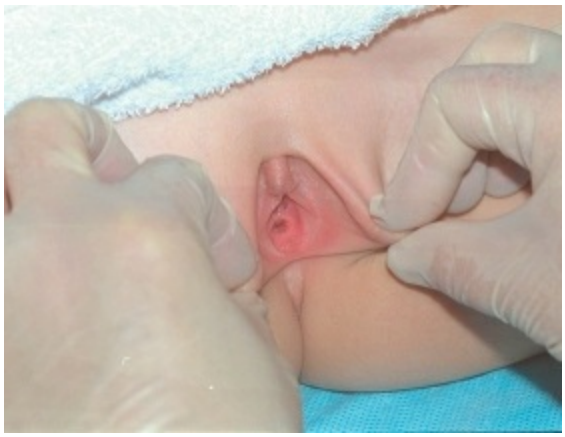


FIGURE 18.9 Anterior labial retraction. Source: (From Gall JA, Boos SC, Payne-James JJ et al: *Forensic Medicine*, London, 2003, Churchill Livingstone.)

Touching the hymen in prepubertal girls causes pain. Discharge for wet mounts, potassium hydroxide (KOH) exams, Gram stains, or culture should be collected with a small Dacron-tipped swab moistened with saline. The child can be asked to take a deep breath for distraction and to open the hymen. If the examiner avoids touching the hymen, this procedure can be painless. Rarely, in cases of suspected abnormalities, a rectoabdominal examination may be performed following inspection of the vaginal area by placing the gloved and lubricated index finger or little finger of one hand into the rectum and placing the other hand on the abdomen. The cervix and uterus may be felt as a "button," and ovaries are not palpable. As the examiner withdraws the finger, the vagina can be gently milked to elicit discharge, a foreign body, or in rare cases, a polypoid tumor.

Young preschool girls are curious about their bodies and often explore the vaginal area. It is not uncommon for young girls to insert foreign objects—crayons, beads, coins, batteries, small parts of toys, and other small objects—into their vagina. It is also common for toilet tissue to ball up and enter the vagina. Children with developmental delay who are diapered may have stool pushed into the vaginal vault. Foreign bodies can remain in the vagina for an extended period and cause inflammation and vaginal discharge, often foul smelling, or bleeding. A foreign body should always be considered when a young preschool girl presents with vaginal or urinary symptoms. If the examiner sees a foreign body, it

may be removed by using a moistened cotton swab or by gently irrigating the vagina with normal saline, or consider referral for removal under anesthesia.

There is no indication for a speculum exam in the prepubertal child. Any invasive exams or procedures should be performed under anesthesia by a pediatric gynecologic specialist.

Examination of the adolescent

There are currently few indications for a pelvic exam in an adolescent. Regardless of history of sexual activity, only the symptomatic young woman may be a potential candidate for a pelvic exam until age 21 when a *Papanicolaou* (Pap) *smear* is indicated.[8] Many examiners will consider an initial evaluation of vaginal complaints in adolescents with careful inspection of the external genitalia and specimen collection of vaginal discharge from the introitus and a vaginal swab for *sexually transmitted infection* (STI) testing if the patient reports sexual activity. The vaginal specimen can be obtained through the patient obtaining a self-swab or can be collected by the examiner. This preliminary process can be followed by a full pelvic exam if indicated. The presence of trichomonas, a vaginal STI, on wet mount indicates the need for a full pelvic exam. In the case of *primary amenorrhea* or absence of the onset of menstrual periods, abnormal vaginal bleeding, abnormal vaginal discharge or lower abdominal pain, a pelvic exam and/or ultrasound imaging may be appropriate.[9]

Table 18.4 reviews current indications for pelvic exams and alternatives to the recommended exam for adolescents who refuse or are unable to tolerate the pelvic examination.

TABLE 18.4

Indications for Pelvic Exams and Alternatives

Indications	Recommended Exam	Alternatives
Sexually active adolescent, asymptomatic, ≤21 years of age	Pelvic exam and Pap smear at age 21, then every 3 yearsUrine or high vaginal specimen for gonorrhea and chlamydia screen using NAAT technology annually	Pap smear not indicated <21 yearsUrine or high vaginal specimen for gonorrhea and chlamydia screen using NAAT technology annually

Adolescent at age 21, asymptomatic, no history of sexual activity	Pelvic exam and Pap smear	Pap smear using a "blind technique" for youth whose disability precludes performing a speculum exam without sedation
Sexually active adolescent, asymptomatic, desires start of hormonal contraception	Urine or high vaginal swab for NAAT STI screen, urine β-hCG if indicated, history, weight and BP check	May defer STI screen and urine β-hCG if menstruating at time of visit, schedule for STI screening after menses
Adolescent desires start of hormonal contraception, no history of sexual activity	History, urine β-hCG, weight and BP check	—
Vaginal discharge without abdominal pain, no history of sexual activity	Vaginal swab for wet mount, KOH prep, STI screen if indicated by microscopic exam	Teen may insert vaginal swab, place in test tube with saline; wet mount, KOH prep
Vaginal discharge without abdominal pain, sexually active adolescent	Urine or high vaginal swab for NAAT STI screen, vaginal swab for wet mount, KOH prep; if wet mount findings consistent with STI, perform pelvic exam, Pap smear if due	Teen may insert vaginal swabs for STI screen and wet mounts
Adolescent with lower abdominal/pelvic pain; no history of sexual activity	Urine β-hCG, bimanual exam, speculum exam if indicated by history or presence of discharge or bleeding; ultrasound if results unclear	Urinalysis, urine β-hCG, urgent ultrasound
Sexually active adolescent with lower abdominal/pelvic pain	Pelvic exam, including speculum exam and bimanual, wet mount/KOH prep, and STI screening	Full exam essential; however, adolescent should not be examined against her willAbdominal exam and U/S imaging may be substituted in patient unable to tolerate pelvic exam

Data from: Practice Bulletin No. 168: Cervical Cancer Screening and Prevention, *Obstet Gynecol* 128(4):e111-e130, 2016; Emans SJ: Office evaluation of the child and adolescent. In: Emans SJ, Laufer, MR, eds: *Pediatric and Adolescent Gynecology,* ed 6, Philadelphia, 2012, Lippincott Williams & Wilkins; Shafer MA: Annual pelvic examination in the sexually active adolescent female: what are we doing and why are we doing it? *J Adolesc Health* 23(2):68-73, 1998; Abells D, Kirkham YA, Ornstein MP: Review of gynecologic and reproductive care for women with developmental disabilities, *Cur Opin Obstet Gynecol* 28(5):350-358, 2016.

β-hCG, Beta human chorionic gonadotropin; *BP*, blood pressure; *KOH*, potassium hydroxide; *NAAT*, nucleic acid amplification test; *STI*, sexually transmitted infection; *U/S*, ultrasound.

The following description of the pelvic examination should be considered a supplement to careful supervision and mentoring of the novice examiner by the more experienced health care provider. Proper clean technique is more easily demonstrated, and specific procedures for collecting specimens for the *Pap smear* and testing of STIs vary among clinics and laboratories.

EVIDENCE-BASED PRACTICE TIP

Cervical cancer screening (Pap smear) should not begin until age 21 years, regardless of age of onset of sexual activity in immunocompetent young women. [8]

Preparing for the exam

Explain the pelvic exam carefully, using a plastic pelvic model, diagram, or internet module and video (see http://www.sexualityandu.ca/sexual-health/going-to-doctor/first-pelvic-exam) that enables the adolescent to gain a concrete understanding of the examination process. It is useful to show the adolescent the speculum and specimen collection implements in advance to dispel any anxiety or fear of the unknown. Clearly define for the patient whether the exam must be completed with some urgency (e.g., to rule out pelvic inflammatory disease) or can be performed when the youth feels prepared (e.g., as part of an evaluation of primary amenorrhea) and has brought her support person of choice to accompany her. Some examiners provide the adolescent with a mirror, if desired, to observe the examination in progress. The adolescent should be encouraged to empty her bladder before the exam. If any specimens are needed, such as for urine pregnancy test, urinalysis, or urine for STI screening tests, they can be collected at this time. The adolescent should be informed that she can change her mind about the exam at any point, and the exam should never be forced or coerced. Some adolescents may want to have a parent, friend, or partner present for the pelvic examination to provide support. With sufficient preparation and explanations, most adolescents are able to tolerate the examination well. However, adolescents who have been

sexually abused, have suffered other trauma, or who are particularly anxious may be helped by specific visualization and relaxation techniques.[10]

Positioning

Adjust stirrups appropriately for the leg length. The adolescent can leave socks on to make the stirrups more comfortable. Ask the youth to move her buttocks forward to the very edge of the exam table, while the feet are in the stirrups. Although many adolescents are uncomfortable with this position, it is necessary for proper visualization and manipulation of the speculum. Avoid touching or pulling the teen; encourage her to move on her own. Most adolescents prefer to have a sheet draped over their abdomen and thighs, but the drape should be positioned so that the examiner can maintain eye contact with the adolescent. The knees should be abducted as far as possible. To have the teen's active participation, encourage her to push her knees apart as if she were doing an exercise in stretching to avoid any references that could remind her of unwanted sexual activity. The adolescent should be encouraged to take slow, even breaths, to avoid tensing her abdominal muscles, and to keep her buttocks down on the exam table.

Inspection

Note the condition of the clitoris, urethra, labia majora, labia minora, hymen, and introitus (Fig. 18.10). Inspect for swelling or lesions such as inflamed pubic hair follicles; *condyloma,* or venereal warts; any clitoral hypertrophy; presence or absence of estrogenization; and any inflammation or discharge. The external structures can be visualized more completely if the examiner gently separates the labia majora to note the vaginal structures and anal area.

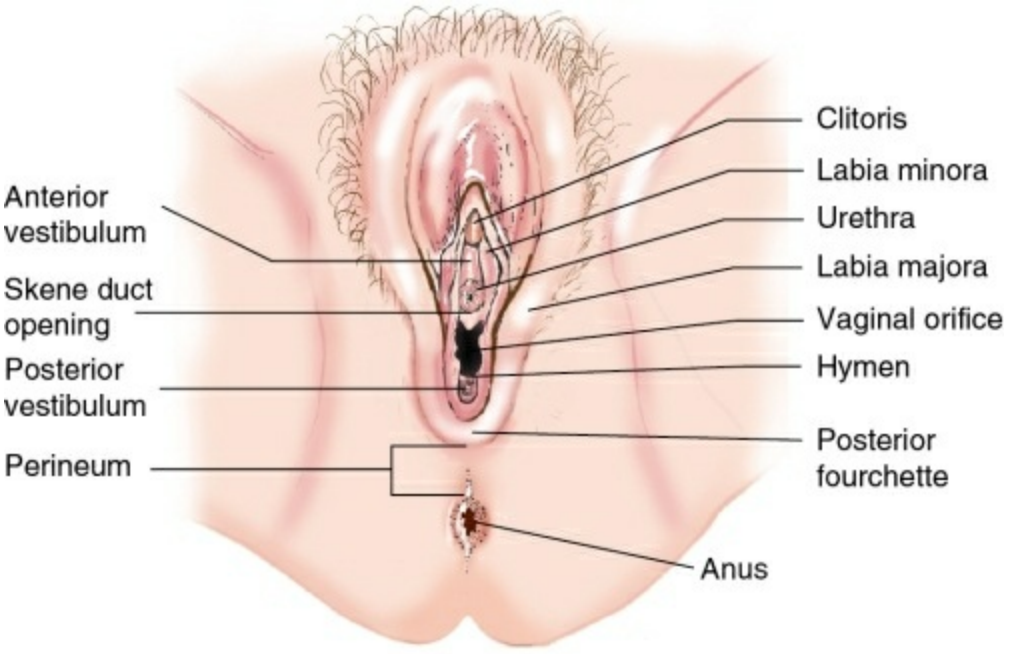


FIGURE 18.10 External female genitalia.

Palpation

Palpation of the *Skene glands* and *Bartholin glands* is usually avoided in the adolescent, unless the adolescent presents with a complaint of pain or swelling in the labial or vaginal area or the examiner notes abnormalities upon inspection (Fig. 18.11).

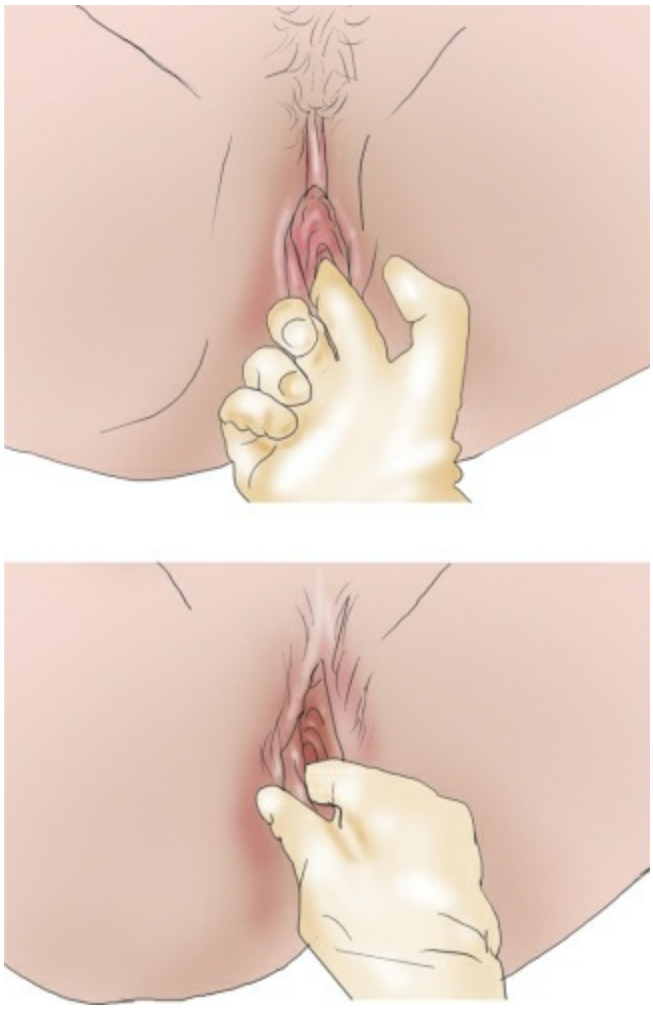


FIGURE 18.11 Examination of the Skene and Bartholin glands.

Speculum exam

Choosing the speculum

The correct size speculum should be selected, and the speculum warmed, if possible, before insertion. Although many clinics use plastic specula (Fig. 18.12), the range of available sizes is limited and the option to use metal specula is more desirable if the capacity to clean and sterilize them is available.

- A Huffman (Huffman-Graves) speculum, (½ × 4½ inches) should be used if the hymenal opening is small, as in virginal teens.
- A pediatric or child speculum (5⁄8– 7⁄8 × 3 inches) should not be used in the postpubertal adolescent because of the excessive width and inadequate length.

- A Pedersen speculum (7⁄8 × 4½ inches) and rarely a Graves speculum (1¾ × 3¾ inches) are used for a sexually active teen.
- *Light source:* Some plastic specula have a built-in light source. Otherwise, angle the light over the examiner's shoulder to illuminate the introitus. Warn the patient the light may feel warm. If the lamp needs readjustment after speculum insertion, remember to change gloves before touching the neck of the light.
- *Inserting the speculum:* The examiner should develop an approach that works for him or her to maintain clean technique. Some examiners double glove, others change gloves after inspection of the external genitalia and insertion of the speculum. Using the fingers of one hand to separate the labia, one finger, lubricated with water or a scant amount of lubricating jelly, can be inserted into the introitus, pressing down on the posterior fourchette to facilitate easy insertion of the speculum. The examiner should use the other hand to gently insert the speculum, angled toward the sacrum, with slight downward pressure to avoid irritating the urethra. Be careful not to pinch the labia minora or to catch pubic hair in the speculum bills. Fig. 18.13 illustrates the steps of speculum insertion.
- Avoid putting pressure on the hymen in virginal teens.
- An alternate method of speculum insertion is to use the second and middle fingers to separate the labia, then press in gently on either side of the lower introitus to relax the fourchette tissues without direct pressure.
- Have the teen take in a deep breath or perform a *Valsalva maneuver* to help relax the introitus to insert the speculum.
- To locate the cervix, use a slight side-to-side motion; if the cervix does not come into view, it may cause less discomfort to withdraw the speculum and use one finger to locate the cervix rather than to move the speculum excessively.
- After the speculum is in place and the cervix is in view, secure the speculum in the open position, and remove the first glove on the hand that separated the labia (if double-gloved) or change the glove on the hand that contacted the labia/introitus; then proceed with specimen collection.

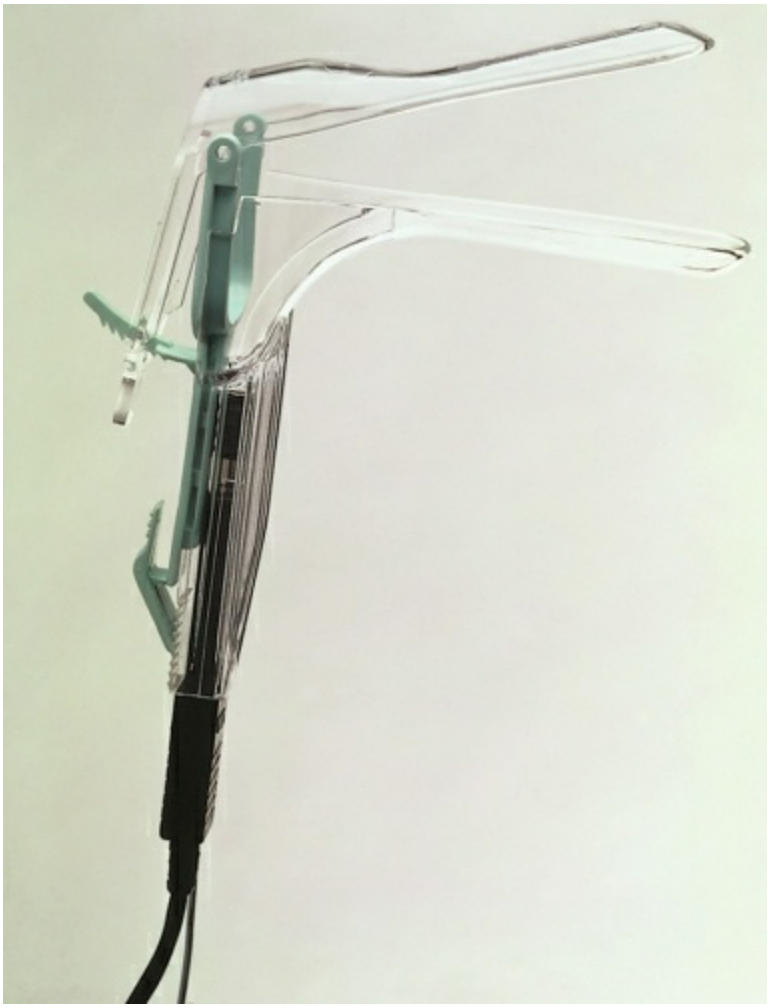
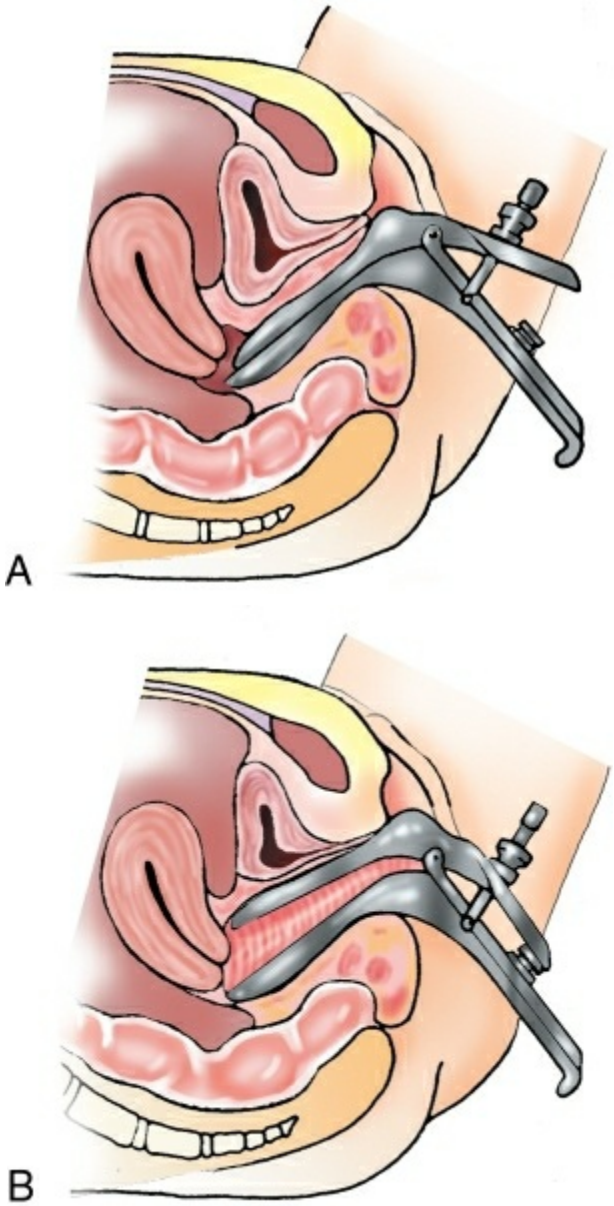


FIGURE 18.12 A vaginal speculum. Source: (From Harkreader H, Hogan MA, Thobaben M: *Fundamentals of Nursing: Caring and Clinical Judgment*, ed 3, St. Louis, 2008, Saunders.)


A
B

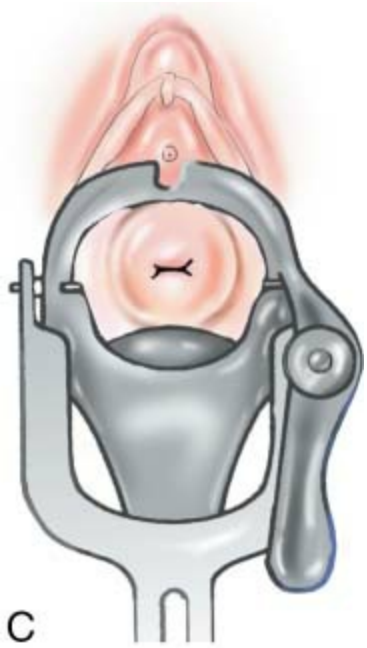


FIGURE 18.13 Female genital examination. **A,** Insertion of closed speculum. **B,** Open speculum and locate cervix. **C,** When cervix is in view, secure speculum.

Inspection of the vagina and cervix

Inspect the walls of the vagina, and note the presence, color, and consistency of any discharge. Carefully inspect the cervix for color, lesions, and any discharge at the *cervical os.* The *cervical os,* the opening of the cervix into the endometrial canal, is round in a *nulliparous* woman, but may be slitlike in a *parous* adolescent. Physiologic variations of the cervix include *nabothian cysts,* which are small, white or yellow, raised, round areas on the cervix; and an *ectropion cervix,* a visible ring of redder, glossy columnar epithelium protruding out and surrounding the os of the cervix. Table 18.5 presents abnormal findings of the female genitalia.

TABLE 18.5

Abnormal Findings of Female Genitalia on Pelvic Exam

Findings	Descriptions
Vulvar and vaginal abnormalities	Imperforate hymen, hematocolpos, vaginal agenesis or signs of a transverse vaginal septum (caused by congenital incomplete fusion of upper and lower vagina), external condyloma, signs of trauma, erythema, and discharge

Cervical abnormalities	Reddened, friable (bleeds with insertion of cotton swab into os), visible condyloma, petechiae ("strawberry spots" rarely seen with trichomoniasis), erosions, double cervix (associated with uterus didelphys), absence of cervix
Pelvic masses	Masses resulting from intrauterine or ectopic pregnancy or from pelvic infection (salpingitis or tuboovarian abscess); adnexal torsion secondary to cyst (which presents with twisting sensation, intermittent bouts of severe pain separated by generalized aching, can be surgical emergency); functional, corpus luteal or dermoid, or other complex ovarian cysts; endometriosisUterine tumors (such as leiomyomas) are rare in the adolescent; ovarian cancers comprise approximately 1% of all childhood cancers, but they are the most common genital tract cancer in adolescents All adnexal masses should be imaged, first with ultrasound, then (if needed) with computed tomography or magnetic resonance imaging

Specimen collection

The recommended order of specimen collection is to start with the Pap smear then follow with the collection of specimens for STI screening. It may be reasonable to collect STI specimens first in some adolescents if there is a copious amount of discharge or purulent discharge from the cervical os, because the exudate needs to be removed before specimen collection for the Pap smear.

- Use a spatula and cytobrush for Pap smear collection. Follow laboratory instructions for either a conventional Pap smear or the newer liquid cytology, in which the cells are collected and placed in a liquid medium. Be sure to sample the squamocolumnar junction, which may be on the *ectocervix*, or *external os*, in some adolescents. In some young women with disabilities, collection of the Pap smear by means of a speculum exam may not be a viable option due to spasticity or other conditions that preclude a standard pelvic exam. The option to obtain a Pap smear using a "blind technique" (without using a speculum, palpate the cervical os using a finger then slide the cytobrush over the finger to obtain the endocervical sample) is an acceptable alternative to ensure appropriate and timely cancer screening.[11]
- Next collect vaginal swabs for chlamydia and gonorrhea—the preferred specimen source due to the highest sensitivity of nucleic acid amplification test (NAAT),[12] unless using urine for STI screening.
- Use a moistened cotton-tipped swab or plastic or wooden

spatula to collect some vaginal secretions for wet mount and KOH preparations from both the vaginal pool and the vaginal wall, unless there is significant blood present.

Make sure to release the locking mechanism of the speculum before withdrawal to avoid pinching the cervix when closing the speculum bills. Do not force the speculum bills closed because this may cause the cervix or vaginal walls to be pinched. Testing vaginal pH is a valuable adjunct to the wet mount and KOH prep exams, and vaginal discharge adhering to the speculum bills can be collected for pH testing before the speculum is discarded or placed in cleaning solution. Alternatively, a swab of vaginal secretions can be applied to a pH tape or strip.

The bimanual exam

The purpose of the bimanual exam is to assess the cervix, the corpus of the uterus, and the adnexa. The adolescent should be encouraged to relax the abdominal muscles by breathing slowly and steadily, or performing a Valsalva maneuver, which may facilitate insertion of the examiner's fingers. The examiner applies lubricant to the fingers and then inserts the middle and index fingers of a gloved hand into the vagina, keeping the pressure against the posterior fourchette and the pubococcygeal muscle and away from the delicate anterior structures of the clitoris and urethra (Fig. 18.14). In virginal adolescents, it may be possible to insert only one finger. If the need for a bimanual exam is indicated, the examiner should consider the alternative option of a *rectoabdominal exam* for adolescents who request to avoid vaginal penetration for cultural or religious reasons.

- **Palpate the cervix:** Note consistency of the cervix, normally firm and shaped like the tip of a nose. Note presence of any bumps on the cervix, such as nabothian cysts, 3- to 8-mm smooth, nontender, firm lumps, which result from blockage of endocervical gland ducts.
- **Palpate the uterus:** Press down on the abdomen with the other hand while supporting the cervix. Assess the size, shape, consistency, mobility, and tenderness of the uterus and any palpable masses. The position of the uterus varies from anteverted, anteflexed, midposition, and retroverted to

retroflexed (Fig. 18.15). The normal uterus is shaped like an upside-down small pear and is approximately 7.5 cm long and 2.5 cm thick.

- **Assess for cervical motion tenderness:** The examiner wiggles the cervix side-to-side and forward and backward between the palpating fingers. In a normal exam the movement of the uterus creates an unusual sensation in the pelvis for an adolescent. Ask the adolescent to distinguish between pain and discomfort or an unusual feeling. Tenderness or pain on exam is a symptom of pelvic infection.
- **Assess the adnexae and ovaries:** Place the examining fingers in the vagina first at the right lateral fornix then at the left lateral fornix, posteriorly and high. Begin with the opposite hand on the abdomen just below and medial to the iliac crest and move diagonally toward the symphysis pubis. Apply a firm, steady sweeping motion with the hand on the abdomen as you palpate briefly. Normal ovaries are smooth and almond shaped, slightly tender to deep palpation, and approximately 3 × 1.5 × 1 cm. Tenderness or fullness of the adnexae is abnormal and may indicate infection, ectopic pregnancy, or endometriosis.
- **Rectovaginal exams:** This exam is usually omitted in adolescents, unless there is a suspicion of abnormality or an extremely retroflexed uterus, or the hymenal ring is too tight to adequately assess for the cervix and uterus. It is performed with the index finger in the vagina, the middle finger in the rectum, and the opposite hand on the abdomen. The *rectovaginal septum* should be thin, pliable, and free of masses. The teen should be reassured that although she may feel the urge to defecate, she will not.
- **Sizing an intrauterine pregnancy:** Recognizing a pregnancy in the adolescent can aid in the swift diagnosis of the cause of complaints ranging from fatigue and nausea to secondary amenorrhea, abdominal fullness, or a mass. The health care provider should maintain a high index of suspicion for pregnancy, even in the youngest adolescents who deny a history of sexual activity. Adolescents may be reluctant to disclose early or unwanted sexual activity. Point of care urine testing for β-hCG is invaluable. The weeks of pregnancy are counted from

the first day of the last menstrual period, even though ovulation generally occurs 2 weeks after the beginning of menses (Box 18.1). Fundal height provides a guide for estimating uterine size (Fig. 18.16).

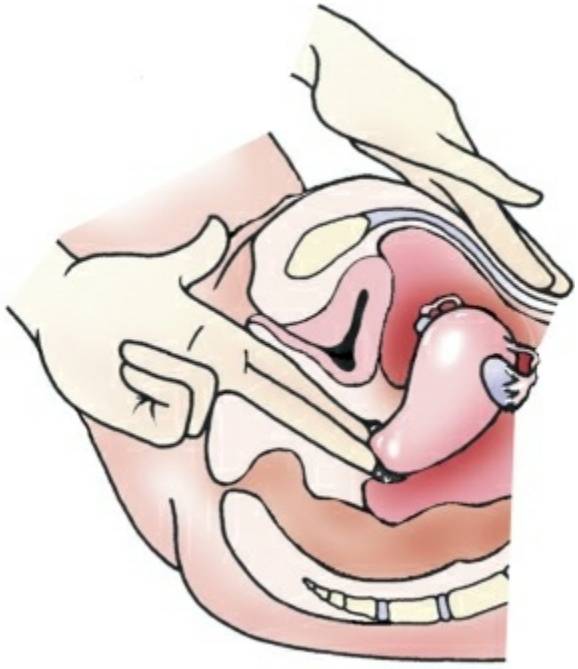


FIGURE 18.14 Bimanual exam.

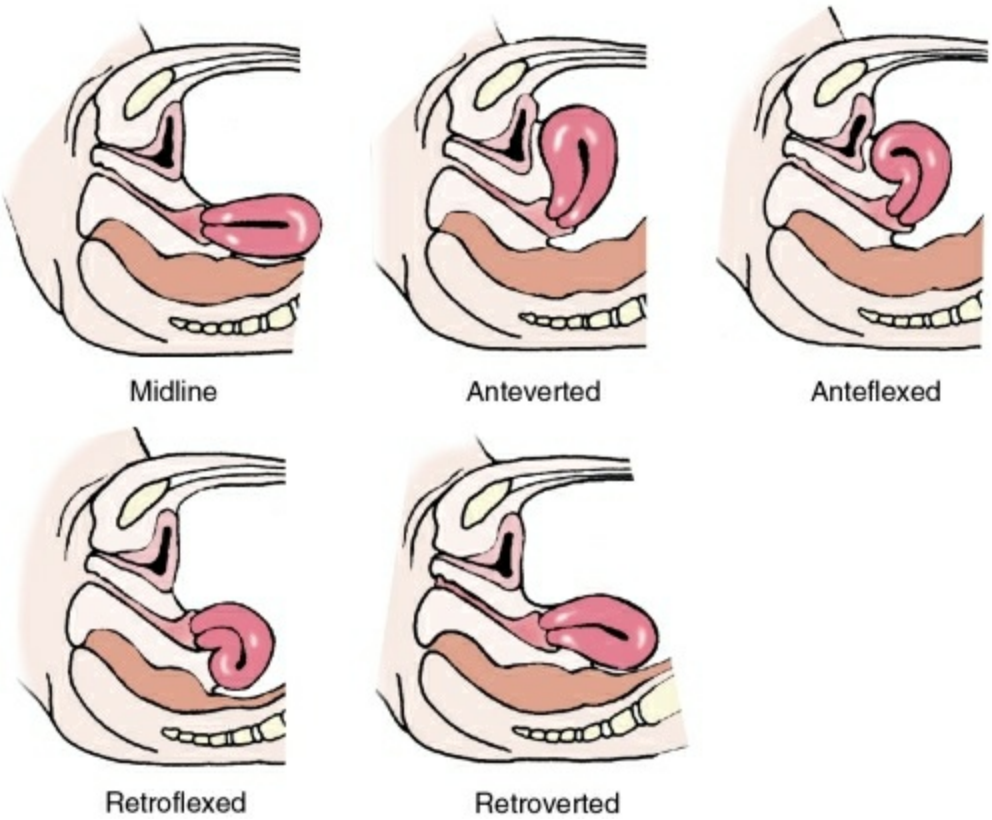


FIGURE 18.15 Various positions of the uterus.

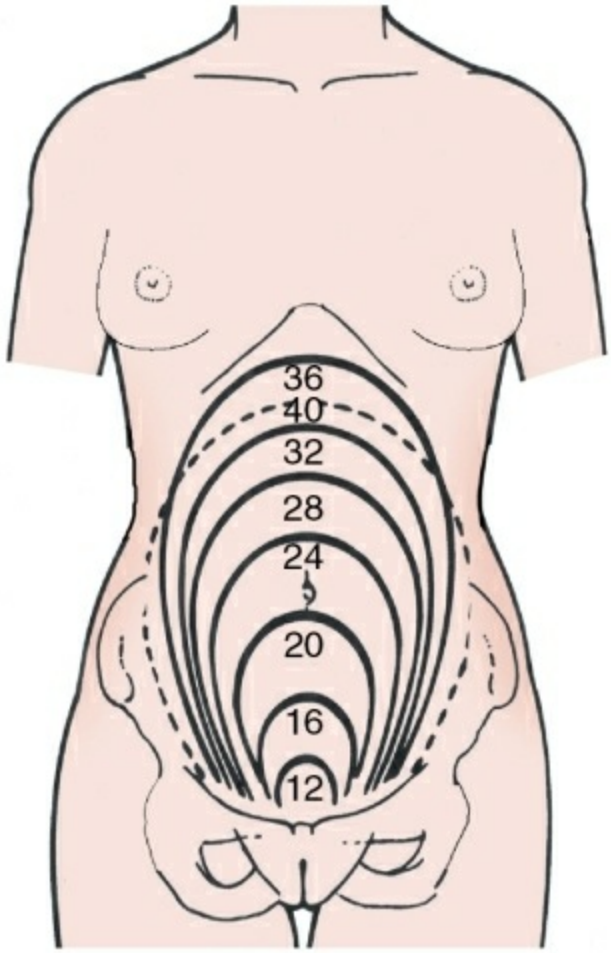


FIGURE 18.16 Fundal height.

BOX 18.1

Sizing an Intrauterine Pregnancy

Nulliparous uterus: 7.5 × 2.5 cm (size of small lime on bimanual exam)
8-week uterus: 9–11 cm × 5 cm (size of orange on bimanual exam)
12-week uterus: 12–14 cm × 7 cm (size of grapefruit, very soft, sometimes difficult to palpate on bimanual exam); just palpable, abdominally at level of symphysis pubis
16-week uterus: Palpate abdomen, felt halfway between symphysis pubis and umbilicus
20-week uterus: Palpate abdomen, felt at umbilicus

More than 20 weeks: Measure from symphysis pubis to height of fundus (number of centimeters − approximate number of weeks)

Wrapping up the examination

The adolescent should be handed some tissues, so she may wipe off the lubricant used. The examiner should stay in the room long enough to ensure that the adolescent does not feel dizzy upon sitting up and then leave while she dresses. Results of the exam should be given while the teen is dressed, and any abnormalities should be thoroughly explained. To ensure confidentiality, present and discuss results with the adolescent alone.

Suspicion of sexual abuse

In cases of suspected child sexual abuse the history and behavioral observations are crucial. Any history of abuse disclosed to the health care provider that occurred within the previous 72 hours (up to 120 hours in some states) requires immediate referral for a forensic examination with possible collection of evidence. Guidelines recommend collection of DNA evidence within 24 hours for prepubertal children and 72 hours for adolescents; however, some regions using DNA amplification will collect DNA evidence over a longer time span, and providers should be familiar with local resources and recommendations.[13] Most sexually abused prepubertal girls will have normal physical exams because of the following:

- Delays in disclosure
- Rapid healing of genital and anal tissue in prepubertal children
- Predominance in this age group of sexual abuse involving fondling or oral-genital contact, which does not leave physical signs

It is important to remember that the vast majority of children reporting sexual abuse will have a normal genital exam.[1] The following physical findings in prepubertal girls should raise suspicion of sexual abuse and prompt referral for an exam by specially trained practitioners[13]:

- Hymenal irregularities or absence of hymen, including notches,

transections, or thin, rounded edges, posterior from the 4- to 8-o'clock position with the child supine; should be confirmed in knee-chest position, because an apparent notch may be a fold in a redundant hymen

- Acute trauma to the hymen or posterior fourchette, or laceration of vaginal mucosa, particularly extending to the rectal mucosa
- Confirmed STI, including human papillomavirus (HPV) infection in a child ≥2 years old

Other possible findings include increased erythema, irritation, and vaginal discharge, which may or may not be signs of sexual abuse. Failure or delay of fusion of the median *raphe* between the posterior fourchette and the anus is often mistaken for trauma or sexual abuse. However, it is essential for health care providers to keep in mind they should not solely rely on physical findings when considering a diagnosis of sexual abuse. The examiner should keep the possibility of sexual abuse in mind whenever other nonvenereal infections, chemical irritation, foreign bodies, and/or poor hygiene are included in the differential diagnosis.

Female genital conditions

Table 18.6 presents common conditions and findings of the genitalia in the prepubertal and pubertal female. The prepubertal vagina is hostile to yeast, and vulvovaginal candidiasis is rare except in cases of diabetes mellitus, recent antibiotic usage, compromised immune system, or diaper use in infants or children with special health care needs. Sexually transmitted pathogens, such as *Neisseria gonorrhoeae* and *Chlamydia trachomatis,* infect the columnar epithelium of the vagina rather than the *cervix* in sexually active adolescents. Bacterial infections, such as *Streptococcus* and *Shigella,* can cause purulent or even bloody vaginal discharge in infected individuals.

TABLE 18.6

Female Genital Conditions

Condition	Description
Labial	Adherence of labia minora or majora, primarily seen in girls 3 months to 6

adhesions	years of age; adhesion may persist until puberty, or may separate spontaneously; treatment is controversial if opening is large enough for normal urinary flow and vaginal drainage
Labial abscesses	Usually caused by *Staphylococcus aureus* and *Streptococcus pyogenes*
Labial lipoma	May be initially mistaken for a hernia
Clitoral lesions	Edema of clitoris, with hypoproteinemia, in conditions such as nephrotic syndrome Hypertrophy of clitoral hood and clitoris, caused by neurofibromatosis, rhabdomyosarcoma, increased androgens Hemorrhages around clitoris caused by lichen sclerosis or trauma (see later)
Urethral prolapse	Presents with bleeding and friable, red-blue doughnutlike annular mass that may be visible in perineum ("hemorrhagic cranberry")
Lichen sclerosis	Uncommon; presents with atrophic, hypopigmented, parchmentlike friable skin around vulva and anus, often in an "hourglass" configuration, with inflammation and subepithelial hemorrhages
Vulvar irritation	Often results from irritants such as bubble bath, poor hygiene, candidal overgrowth caused by antibiotics or diaper occlusion; rarely scratching can occur secondary to pruritus from a pinworm infestation
Vulvovaginitis	Vaginal discharge may be caused by bacteria (e.g., *Streptococcus, Shigella*) or overgrowth of normal flora; foreign body in vagina (typically toilet paper); poor hygiene; sexually transmitted infection (STI)
Straddle injuries to vulva	Straddle injuries (as in playground falls) generally cause trauma to anterior vulvar structures and rarely cause trauma to posterior portion of hymen or posterior fourchette; in addition, injury is usually somewhat asymmetrical and not penetrating in nature

Summary of examination

- The genital exam in the child and asymptomatic adolescent includes an assessment of the external genitalia only.
- SMR in girls is based on breast development and pubic hair distribution.
- Secondary sexual development must be assessed in the context of overall growth and development.
- The norms for SMR are based on data collected on white children. Children of Asian or Native American descent may not have pubic hair development that matches SMR. Pubic hair development may not correlate well with sexual maturity.
- There are currently few indications for a pelvic exam in an adolescent.
- A pelvic exam is not necessary for recommended STI screening in asymptomatic, sexually active adolescents.

- Ninety-five percent of children reporting sexual abuse will have a normal genital exam.
- Variations in external genitalia may be due to congenital disorders or cultural practices, such as female circumcision.

DOCUMENTATION

Documenting exam on a sexually active female teen

External Genitalia: Pink, no discharge noted, without lesions. SMR stage 4 to 5 pubic hair distribution.

Vaginal Exam: Cervix pink without lesions, scant blood at os from menses, scant mucoid discharge in vault.

Bimanual: No pain on deep palpation or with movement of cervix. Uterus midposition. Adnexae nontender, nonthickened.

Rectal: No fissures, lesions, or hemorrhoids normal placement and tone; stool soft brown, guaiac neg.

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CHAPTER 19

Musculoskeletal assessment

Karen G. Duderstadt, Naomi A. Schapiro

Assessing the musculoskeletal system is challenging for pediatric health care providers due to the variations in growth and development and the normal range of rotational changes in the extremities in children from birth to young adulthood. Increasing knowledge about the normal developmental changes in the musculoskeletal system is key to accurate assessment and limiting overreferral. Performing appropriate physical assessment techniques assists in diagnosis of common orthopedic conditions and prompt referral when indicated for children with intentional or unintentional injuries.

Embryologic development

The rudimentary skeletal system forms as early as the fourth week of gestation, when the development of the vertebrae begins and the upper extremities begin as buds on the fetus. As early as the seventh week of gestation, embryonic vascular growth progresses toward the center of the *osteoblasts* in the long bone, which forms cartilage. During the ninth week of gestation, *ossification*, or bone growth, begins in the ossification centers in the lower thoracic and upper lumbar vertebrae, and ossification continues in the femur. The hand pads develop from the extremity buds by days 33 to 36,

and the finger rays begin to form on days 41 to 43.[1] It is during this period of gestation that *polydactyly,* the presence of extra digits, or *syndactyly,* the webbing or fusing of the digits, occurs along with other deformities of the extremities. The *calcaneus,* the largest tarsal bone of the foot, ossifies at the sixth month of fetal life. The position of the fetus in utero has the greatest impact on the skeletal system, and the most common variants present at birth.

Muscle structures, including the tendons, ligaments, cartilage, and joints, originate from the embryonic *mesoderm.* Muscle fibers are developed by the fourth or fifth month of gestation. They increase in size along with changes in muscle and fat proportions throughout childhood and adolescence.

Developmental variations

At birth, the *epiphyses* of the long bones are composed of hyaline cartilage. Shortly after birth, the secondary ossification centers begin to replace the cartilage along the *mat plate.* During early periods of rapid growth, ossification occurs in secondary sites throughout the body: the ends of the long bones, the vertebrae, the flat bones in the clavicle, and the skull. The replacement of cartilage by bone is known as *endochondral ossification.* The epiphyseal plate ossifies, becoming the *diaphysis,* the shaft of the long bone; and cartilage at the metaphyseal plate replaces bone cells (Fig. 19.1).

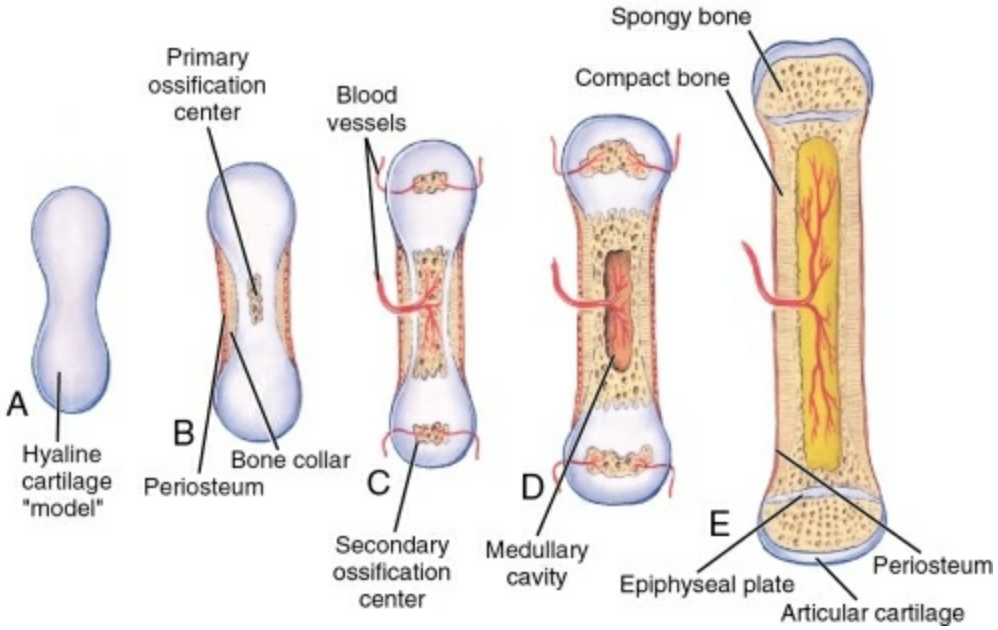


FIGURE 19.1 Growth plates and transition from cartilage to bone at the epiphyseal plate. (A) Hyaline cartilage "model." **(B)** Periosteum and bone collar form. **(C)** Blood vessels and osteoblasts infiltrate primary ossification centers. **(D)** Osteoclasts form medullary cavity. **(E)** Ossification is complete. Hyaline cartilage remains as articular cartilage and in the epiphyseal plate. Source: (From Applegate E: *The Anatomy and Physiology Learning System*, ed 4, St. Louis, 2011, Saunders.)

Bone growth continues in infants and children along the *epiphysis* as new bone is added to the outer surface of existing bone. Growth along the epiphyseal plate continues until the cells in the growth plate mature and stop dividing in puberty, and closure of the growth plate occurs in young adulthood when the metaphysis and the epiphysis fuse.

Several factors influence the healthy development of the skeletal system and normal growth at the epiphyseal plate. Trauma during childhood can cause separation of the epiphysis and the blood vessels to rupture, resulting in cessation of bone growth and a shortened extremity.[1] Nutritional factors, such as adequate protein in the diet, the amount of calcium intake daily, and adequate intake of vitamin D, which regulates the adsorption of calcium and phosphorus in the intestines, impact bone growth. Adequate levels of vitamin D, particularly important in the breastfed infant, may also have a role in improving muscle and immune function in

childhood. Parents are advised to apply sunscreen and limit direct sun exposure for infants and children due to long-term risks of skin cancer, therefore reducing vitamin D synthesis from the skin. Balancing safe sun exposure and maintaining adequate levels of vitamin D in infants and children is important to maintaining bone strength. *Rickets* is a deficiency of vitamin D in which the growth plate is impaired by calcification of newly formed bone on the metaphyseal plate. Alterations in thyroid or growth hormones can also impact normal patterns of growth in childhood.[1]

Anatomy and physiology

Head and neck

Assessment of the head and neck area is reviewed in Chapter 10.

Upper extremities and torso

The *clavicle* lies in a horizontal plane above the first rib and rotates between the sternum and the superior surface of the *scapula* (Fig. 19.2). The *scapula,* a large, flat triangular bone, lies posterior on the upper back and forms the posterior portion of the upper extremity, and the superior, lateral angle articulates with the rounded head or capsule of the *humerus,* the large bone in the upper arm. The distal end of the *humerus* forms the *condyle* and is divided into the *medial epicondyle* and the *lateral epicondyle.* The proximal end of the *ulna* attaches at the articulating surface of the humerus and forms one of two long bones in the forearm. The *olecranon* is the outer, rounded distal surface of the *ulna* and forms the major portion of the elbow. The distal end of the ulna is small and articulates with the wrist bones. The *radius* is along the lateral side or thumb side of the forearm, and the distal end of the *radius* forms a large portion of the wrist bones.

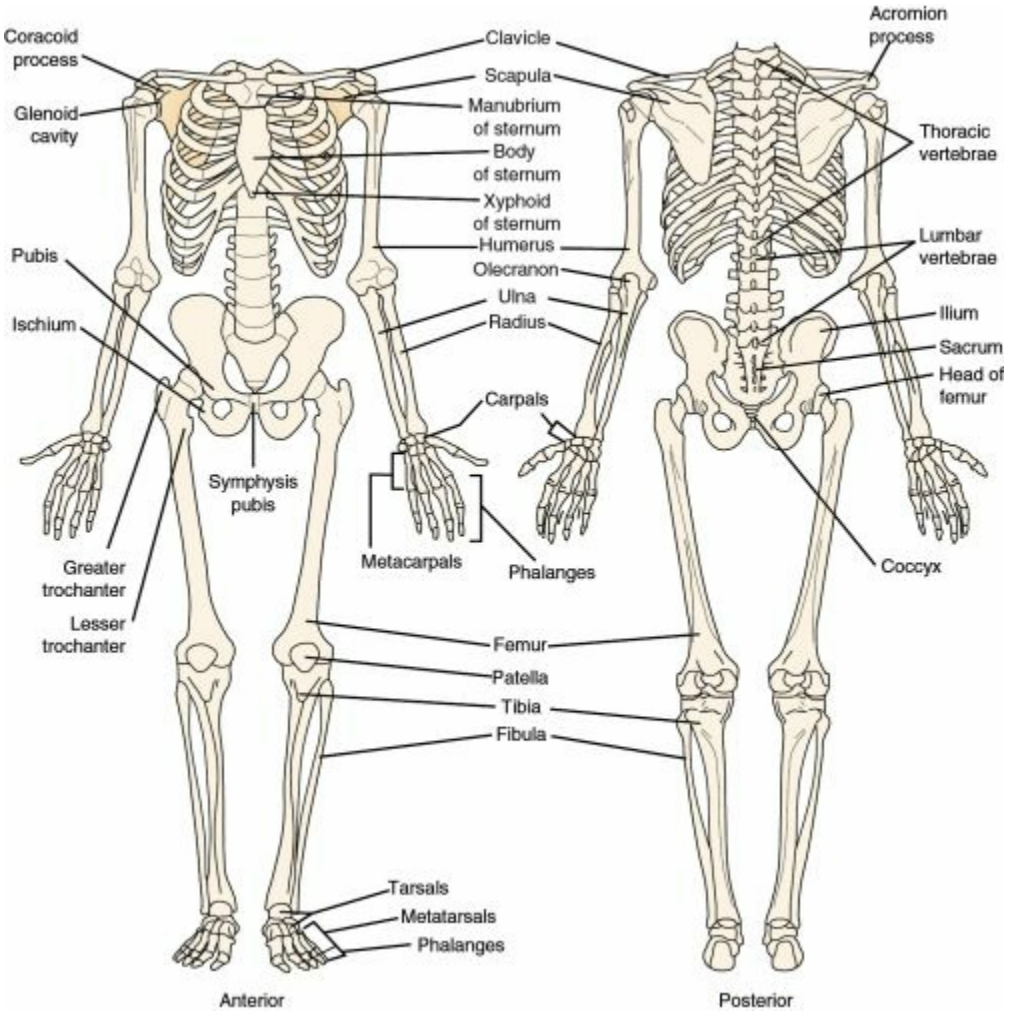


FIGURE 19.2 Mature skeleton.

The hand consists of eight carpal and five metacarpal bones. Many of the carpal bones are cartilaginous at birth. A radiograph of a child's hand at 2.5 years of age illustrates ossification of only the *capitate* and *hamate* bones in the midhand. Development and ossification of the hand continues until 11 years of age, when all of the carpal bones are ossified except for the small *pisiform* bone, which develops by 12 years of age. Abnormalities of the *phalanges,* or fingers, such as *syndactyly,* webbing, or fusion between adjacent digits of the hands or feet, may be an isolated anomaly or indicative of profound developmental delay (Fig. 19.3). *Polydactyly,* the presence of supernumerary digits, is a more common variant and is not usually associated with other congenital anomalies.

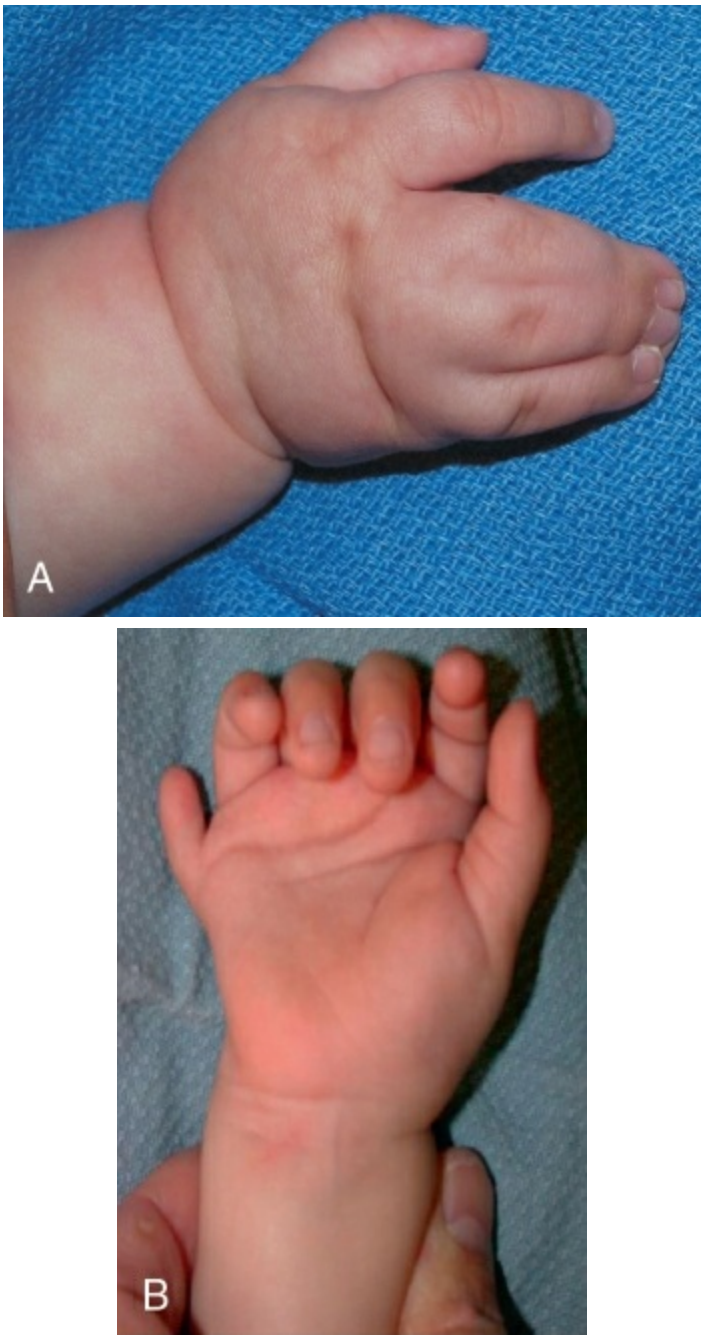


FIGURE 19.3 **(A)** Syndactyly. **(B)** Polydactyly. Source: (**[A]** From Davis P, Cladis F, Motoyana E: *Smith's Anesthesia for Infants and Children*, ed 8, Philadelphia, 2012, Mosby. **[B]** Chung, KC: Hand and Upper Extremity Reconstruction with DVD: a Volume in the Procedures in Reconstructive Surgery Series. Philadelphia, 2009, Saunders.)

Spine

The infant has a C-shaped spinal curve at birth in comparison with the double S curvature that is present in late adolescence (Fig. 19.4).

Thoracic and pelvic curves are present at birth, and the secondary cervical curve is present by 3 to 4 months of age, when the infant begins to hold up his or her head. The lumbar curvature begins to form as the infant bears weight and begins to walk. The young child often has an exaggerated thoracic-lumbar curvature and a protuberant abdomen until 3 years of age, when gait and balance become more normal (Fig. 19.5). The *sacrum* is composed of five separate bones at birth and by 18 to 20 years of age is fused into one large bone. The *coccyx* consists of three or four small bones that begin to ossify between the first and fourth years of life and fuses into one bone by 25 years of age.

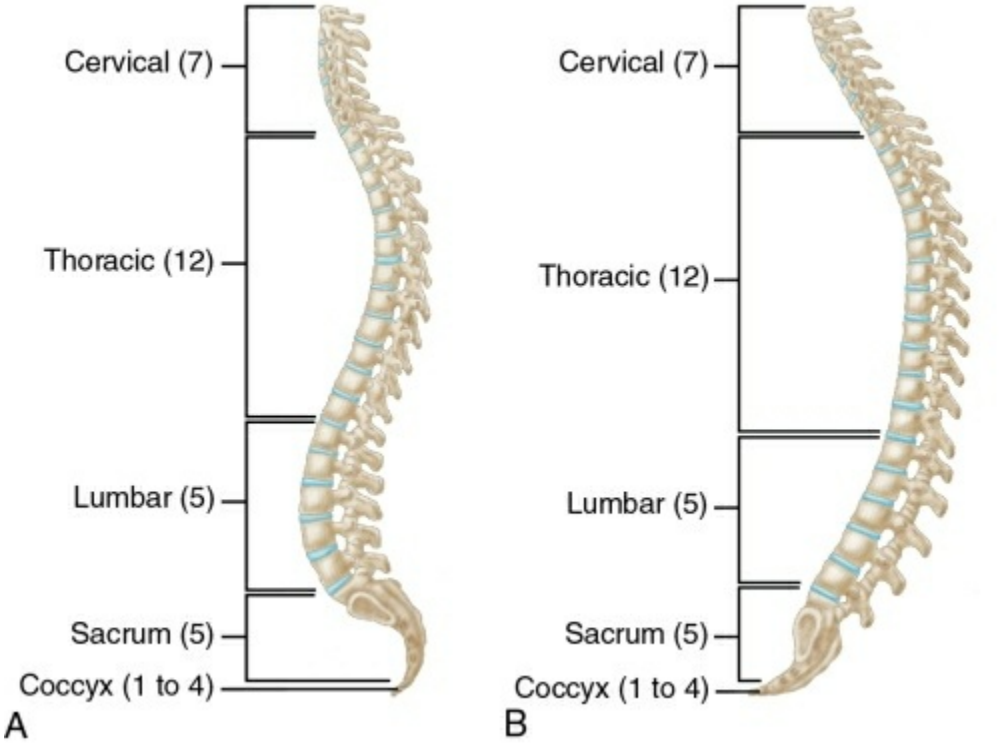


FIGURE 19.4 **(A)** Mature spinal column. **(B)** Immature spinal column.

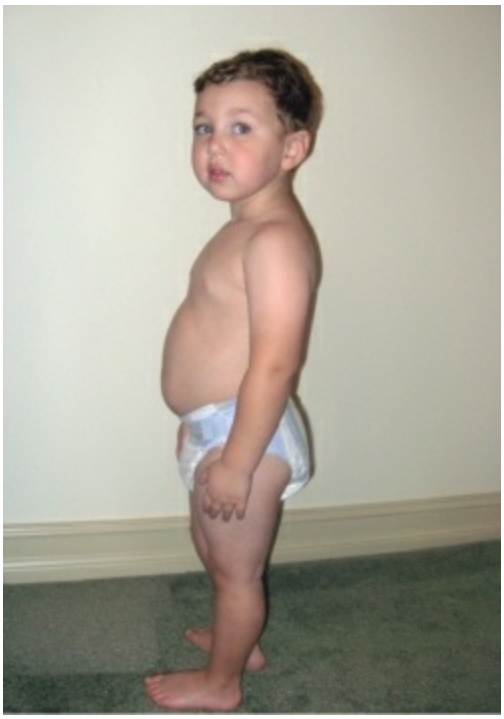


FIGURE 19.5 Protuberant abdomen of the young child. Source: (From Seidel H, Ball J, Dains J, et al: *Mosby's Guide to Physical Examination*, ed 7, St. Louis, 2011, Mosby.)

Pelvis

The hip bone has three distinct parts in childhood, which are later fused in the adult. The *ilium* is the superior, broad, flat surface of the hip bone. The *ischium* is the strongest portion of the hip bone and contains a portion of the *acetabulum,* which forms the attachment at the hip bone for the femur and the opening or *foramen* (see Fig. 19.2). The *pubis* contains the medial portion of the acetabulum and joins medially to form the complete pelvic girdle.

Lower extremities and feet

The *femur* is the longest and strongest bone in the body. The head of the femur becomes ossified during the first year of life, but the shaft of the femur does not become completely ossified until 14 years of age. The long bones of the extremities continue to grow at the site of the epiphyseal plate throughout childhood and adolescence; peak bone mass is achieved by young adulthood. In the lower leg, the larger bone is the *tibia,* the proximal end articulates with the femur and the distal end forms the *medial malleolus* of the ankle. The *fibula*

is the smaller bone in the lower leg and lateral to the tibia. The distal end of the fibula forms the *lateral malleolus* of the ankle.

The *patella* is the small triangular bone that forms the kneecap and lies over the junction of the *femur* and the *tibia.* The health of the patella during growth and development depends not only on strong bone growth but also on the strength of the ligaments and tendons supporting the patella.

The seven tarsal bones and five metatarsal bones of the foot undergo dramatic ossification during the first year of life. The ossification of the bones in the foot follows the normal development of the gross motor milestones. The *talus* is ossified by the seventh month of life to form the ankle, and the remaining *metatarsal* bones continue to form during the latter half of the first year of life. The *phalanges* continue the ossification process through adolescence.

The lower limbs go through a continuous process of growth and rotational change until approximately 8 years of age. The torsional development of the skeletal system is a process that begins in infancy and progresses from sitting to crawling, then standing, cruising, and finally walking with an exaggerated gait and a wide base of support. The laxity in the ligaments and fetal positioning in the intrauterine environment, as well as sitting and sleeping patterns in the early years, contribute to the normal torsional variations in the lower extremities during childhood. Torsional variations and musculoskeletal abnormalities are hereditarily linked and often show a strong family tendency.

Monitoring growth

During growth, bone has the remarkable capacity to remodel itself. Bone is deposited in areas subjected to stress and reabsorbed in areas where there is little stress. This physiologic process explains the ability of children to remodel residual deformity after fractures and demonstrates the biologic plasticity of growing bones. The rate of bone growth is greatest in the lower extremities before the onset of puberty, and the *distal* extremities reach adult size in early puberty. This is often reflected in shoe size in preadolescents. The trunk and *proximal* extremities exhibit the dominant growth during puberty and into young adulthood. Bone growth is completed at age 20 years, and peak bone mass is not achieved until 35 years of

age.

Monitoring the velocity of growth is the key focus of routine well-child visits and when following children with chronic health conditions, particularly cardiac conditions. For the child with slow growth or delayed onset of puberty, obtaining past growth charts and evaluating the progression of the growth curve along with familial patterns of growth and pubertal development is critical to assessing an abnormal growth pattern.

Muscle development

The rate of muscle growth and development increases rapidly beginning at age 2 years. Early muscle growth is balanced by a normal decrease in adipose tissue in early childhood.[1] The growth and maturation of the muscles continues along with the ossification of the skeletal system (Fig. 19.6). *Muscle tone* is the state of normal tension of the muscles, and term infants normally have strong muscle tone at birth. Abnormalities in muscle tone in an infant may be noted shortly after birth when an infant is floppy. Less than normal muscle tone is described as *hypotonia; rigidity* or increased muscle tone is known as *hypertonia;* and *spasticity* is resistance to the range of motion of the muscles, including flexion and extension. Musculoskeletal terminology is presented in Box 19.1. Normal muscle function requires the normal function of the *lower motor neurons* (LMNs) in the spinal cord and the normal reflexivity of the muscle fibers. Normal *muscle tone* requires the normal function of the spinal cord stretch reflex and the balance of the *upper motor neurons* (UMNs) and LMN function. Lesions on the UMNs result in increased tone, and LMN lesions result in decreased tone.

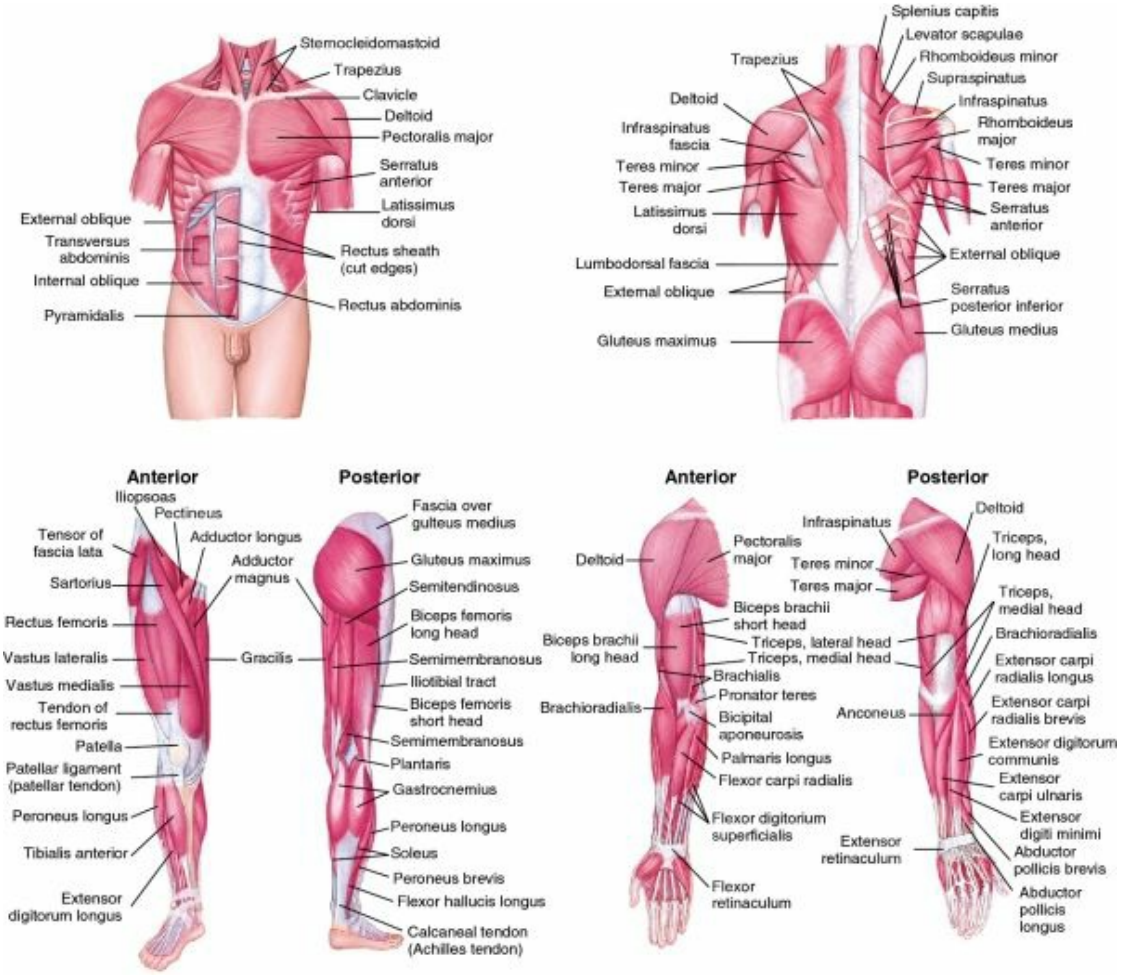


FIGURE 19.6 Muscular system, front and back. Source: (From Mourad LA: *Orthopedic Disorders*, St. Louis, 1991, Mosby.)

Box 19.1

Musculoskeletal Terminology

Distal	Farthest from the point of attachment
Proximal	Nearest to the point of attachment
Flexion	A decrease in the angle of the resting joint in the upper or lower extremities
Extension	An increase in the joint angle
Hyperextension	An increase in the angle of the joint beyond the usual arc
Abduction	Movement away from the midline
Adduction	Movement toward the midline

Rotation	Movement around a central axis
Circumduction	Rotation or circular movement of the limbs
Dorsiflexion	Backward rotation
Supine/supination	Lying on back facing upward, palmar surface facing upward
Prone/pronation	Lying face downward/palmar surface facing downward
Inversion	Turning inward/movement toward the body
Subluxation	Partial or incomplete dislocation
Eversion	Turning outward/movement away from body
Varus	Toward midline of body
Valgus	Away from midline of body
Dorsiflexion	Movement of the hands and feet upward

The greatest increase in muscle size occurs during puberty when boys' muscle mass and muscle cell size begin to exceed that in girls. In early and middle childhood, girls' muscle size increases at a greater rate than in boys up until puberty.

Physiologic variations

Table 19.1 presents physiologic and developmental changes in the musculoskeletal system from the newborn through adolescence.

TABLE 19.1

Physiologic Variations of the Musculoskeletal System at Key Developmental Stages

Age Group	Variation
Preterm infant and newborn	Lower extremities in external rotation and flexion at hips; upper femur is anteverted and knees are flexed; tibias are internally rotated; feet are dorsiflexed
Infancy	Tibias gradually rotate externally to approximately 20 degrees toward midline by 12 months of age; flat feet and bowed legs until walking is firmly established
Early childhood	Stance with wide base of support, hyperflexion of hips and knees with disjointed (toddling) pattern when walking; arms held abducted and elbows extended; intoeing is common beginning at 15 months; normal arm swing and heel-toe walking generally begin by 18 months of age; longitudinal arch not present in infant but begins to develop by 2.5 years of

	age. At 3 years of age, children exhibit mature pattern of motion and muscle action; resolution of intoeing and marked torsion of lower extremities normally disappear by school entry
Middle childhood	*Knock-knee* is present until 7 years of age; by 8–10 years of age, femur rotates to position of about 14 degrees toward midline from average of 45 degrees at birth
Adolescence	Hormonal changes impact ligaments and tendons; laxity of knees is particularly common in adolescent females, making them vulnerable to injury

Family, cultural, racial, and ethnic considerations

African American infants often have advanced musculoskeletal development and achieve developmental milestones earlier. Asian American infants often have increased hypotonia at birth due to increased laxity in the ligaments of the muscle, and they may also have an increased abduction of the hips bilaterally on examination in early infancy.

EVIDENCE-BASED PRACTICE TIP

Racial and ethnic diversity may impact the rate of bone growth and must be considered when evaluating growth curves in the primary care setting. A growth parameter that falls outside of the accepted two standard deviations (SDs) above or below the mean may be normal when the parental stature is considered.

System-specific history

Obtaining a complete history or a symptom-related history of the musculoskeletal system is key to accurate assessment in children and adolescents. The Information Gathering table reviews the pertinent areas for each age group and developmental stage of childhood.

Information Gathering for Assessment of the Musculoskeletal System at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant	History of lack of oxygen (hypoxia) in early neonatal period? Any history of bleeding in the brain, seizures, or injuries to the brain (intraventricular insult)? Did the mother of the child drink alcohol or use drugs while pregnant? When did prenatal care begin?
Newborn	History of chronic health conditions in mother or infection while pregnant? Any trauma sustained at birth? Need for resuscitation or ventilation in immediate newborn period? Presentation at birth? Breech or shoulder? Family history of skeletal deformities or genetic disorders?
Infancy	Family history of bone or joint disorders? Any delay in achieving gross motor milestones? Does infant roll over? Sit without support? Crawl? Stand alone? Walk without support? Does the child tend to walk on their toes?
Early childhood	Do you think your child is clumsier or falls more often than other toddlers? Is the child's gait normal? Is there a family history of feet turning in when walking?
Middle childhood	Involved in organized/competitive sports? Any complaint of pain when walking/running or pain that awakens child at night? History of joint stiffness or swelling? Any complaint of back pain? Does the child carry a heavy backpack? How do they carry their backpack? History of prolonged steroid use with chronic conditions?
Adolescence	Involved in organized/competitive sports? Involved in the same sport year-round? Any limited range of motion of joints? History of fractures, sprains, or trauma? Any complaint of back pain? Is posture normal when standing? Does adolescent carry a heavy backpack? Family history of skeletal deformities? Start of menstrual periods?
Environmental risks	Contact with chemical cleaning agents, hazardous smoke, or chemicals? Exposure to toxic pesticides? History of elevated lead level?

Physical assessment

Preparation for the examination

For a thorough and complete assessment of the musculoskeletal system, infants must be undressed except for the diaper, and children must be undressed except for underwear. A young child cannot be thoroughly assessed when wearing socks and shoes or when gowned. In preadolescence and adolescence, maintaining modesty while assessing the spine can be challenging, particularly in girls. Maturing females must be in underwear and gown to obtain a thorough examination of the spine. The skeletal positions are presented in Fig. 19.7.

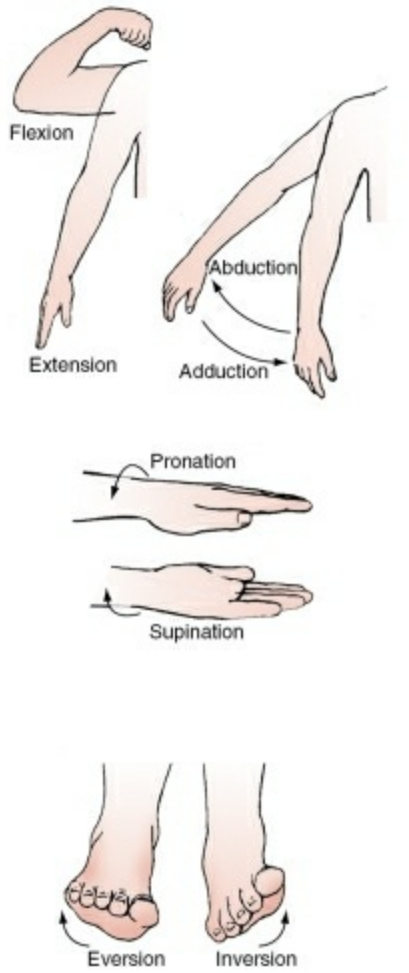


FIGURE 19.7 Skeletal positions.

Inspection and palpation

Initial inspection of the musculoskeletal system in the child from birth to adolescence begins with inspection of skin for color and temperature; inspection of palmar creases; and noting of scars, unusual pigmentation or lesions, swelling, and any bruising. Erythema, swelling, tenderness, or temperature changes should also be noted over the joints. Bruising is the most common sign of physical abuse and intentional injury in children. Bruising over bony prominences such as the shins, knees, and forehead are common in children and usually the result of active play. However, bruising seen on the buttocks, neck, face, and earlobes should be considered suspect and require further information gathering and assessment for signs of abuse (Fig. 19.8).[2]

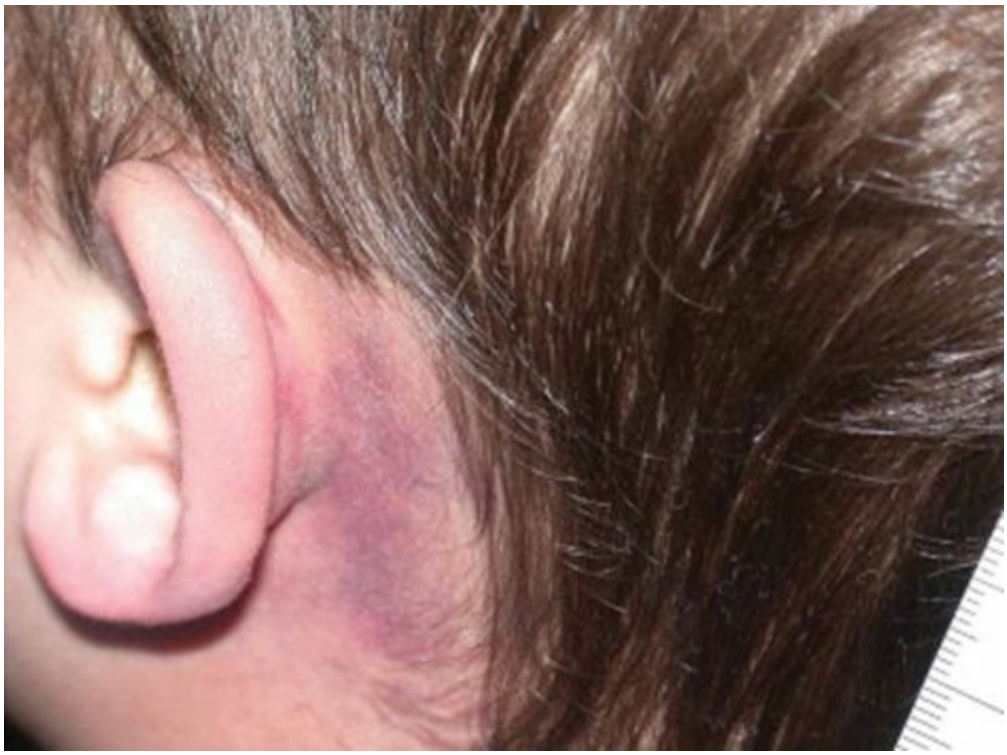


FIGURE 19.8 Skeletal bruising concerning for abuse. Source: (From Hornor, G. Medical evaluation for child physical abuse: what the PNP needs to know, *J Pediatr Health Care* 26:163-170, 2012.)

Observation of posture when standing and sitting, assessing the proportion of upper extremities to lower extremities, and noting any obvious gait abnormalities are part of a complete musculoskeletal assessment. Palpation for bone or joint tenderness, and any unusual prominence, thickening, and/or indentations in the bony skeleton should be noted. Muscle tone, muscle strength, and symmetry should be evaluated. Assessment of the mobility of the spine and range of motion of the joints and extremities are included in a thorough and complete assessment of the musculoskeletal system.

Joints

Joints in the body have a slightly moveable to freely moveable motion throughout the period of growth and development. The *hinge joint*, between the humerus and the ulna, permits motion in one plane, whereas the *pivotal joint*, between the radial-ulnar joint, allows rotation only (Fig. 19.9). The *condyloid joint* in the wrist

allows flexion, extension, adduction, abduction, and circumduction. *Saddle joints*, such as in the thumb, have a similar motion to the condyloid joint in the wrist, except that the joint forms a concave-convex fit to achieve motion. Note any widening of wrist joints, which can indicate decreased bone mineralization at the growth plates, common in children with rickets. The hip and shoulder joints are examples of *ball-and-socket joints*. Finally, the *gliding joints*, also known as *planar joints*, allow a gliding motion between two flat or nearly flat surfaces, such as in the vertebrae and between the carpal and metacarpal bones in the hand and the tarsal and metatarsal bones in the feet.

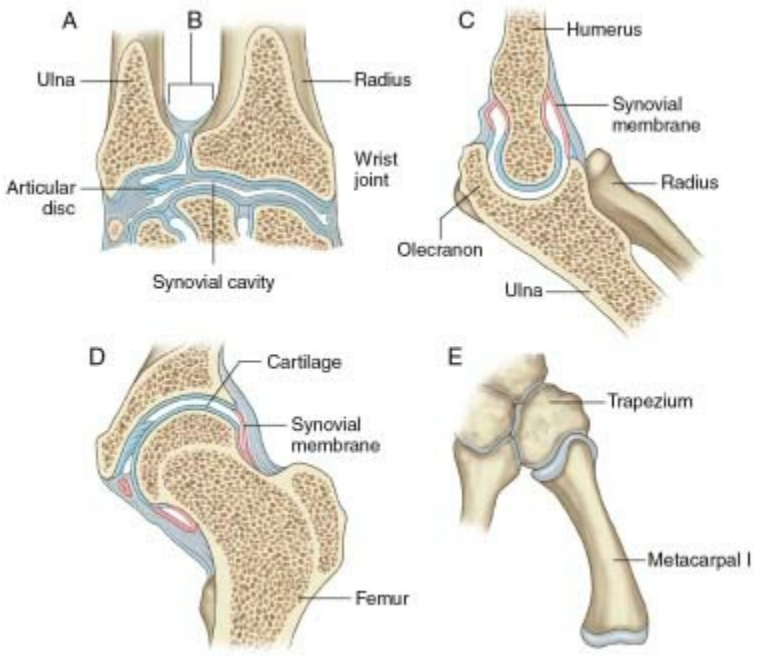


FIGURE 19.9 Skeletal joints. (A) Condyloid (wrist). **(B)** Pivotal (radioulnar). **(C)** Hinge or ginglymus (elbow). **(D)** Ball and socket (hip). **(E)** Saddle (carpometacarpal of thumb). Source: (From Drake R, Vogl AW, Mitchell A: *Gray's Anatomy for Students*, ed 2, Philadelphia, 2010, Churchill Livingstone.)

Head and neck

Assessment of the head and neck is presented in Chapter 10. Children presenting with head and neck pain should be evaluated for *nuchal rigidity* to determine if they have pain or resistance when

neck is flexed forward when lying supine. See Chapter 10 for further musculoskeletal examination techniques to evaluate head and neck pain in children.

Upper extremities and clavicle

In the newborn infant the clavicles should be fully palpated to detect a possible fracture often associated with a traumatic delivery. Localized tenderness at the proximal end of the clavicle shortly after birth often leads to a palpable bony prominence as the fracture heals. A full range of motion of the upper extremities, including the elbows and wrists, must be evaluated to determine any trauma that may have affected the clavicles or shoulders during the birth process. In *Erb palsy,* injury to the fifth and sixth cranial nerves, no spontaneous abduction of the shoulder muscles or flexion of the elbow is noted on examination (Fig. 19.10). The arm is adducted and internally rotated, but normal grip in the hand is present.

Range of motion should be passively evaluated in infants and young children and assessed actively when children are cooperative with examination. Any limitation of range of motion in joints and extremities or asymmetrical response should be further investigated. Muscle tone and strength should also be evaluated. In early childhood the radial-ulnar joint is particularly vulnerable. *Subluxation,* or partial dislocation, of the radius from the humerus, often referred to as *nursemaid's elbow,* is common in children from 2 to 4 years of age. Often there is no clear history of trauma, but laxity of the ligaments in the young child predisposes them to injury. In cases of a subluxation the child usually refuses to use the affected arm. Passive range of motion is possible except for *supination* (palm facing upward). Dislocation of the shoulder can also occur in the young child and is marked by swelling, pain, and a limp arm.

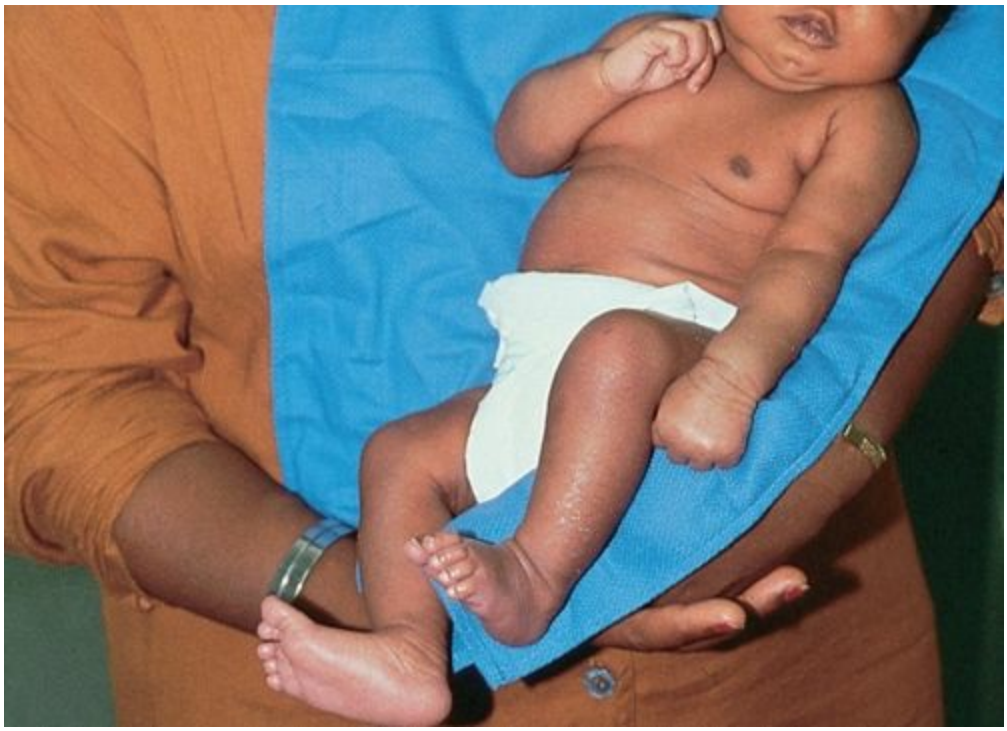


FIGURE 19.10 Erb Palsy. Source: (From Hockenberry MJ, Wilson D: *Wong's Nursing Care of Infants and Children*, ed 10, St. Louis, 2015, Mosby.)

Trauma to the upper extremities is common in children and adolescents. Strains, sprains, or fractures can occur with strenuous activity, falls, motor vehicle or pedestrian injuries, or participation in competitive sports. Careful assessment is warranted in the growing child when any history of trauma is obtained. Fractures that are highly suggestive of intentional injury or abuse in children are rib fractures, especially posterior rib fractures, scapular fractures, sternal fractures, fractures of the spinous processes, and metaphyseal lesions.[2] Clavicular fractures and long bone fractures in children older than 1 year of age are common traumatic injuries of childhood.

Lower extremities and feet

Assessment of the lower extremities includes evaluating flexion/extension, adduction/abduction, and internal/external rotation. Muscle tone, muscle strength, and symmetry should be evaluated with the child standing and while observing gait. Leg length is evaluated with the infant or child supine, with knee and hip joints extended and legs aligned. A discrepancy in length or

asymmetrical appearance may indicate an abnormality in hips, long bones, or knees. In infants, leg length discrepancy may indicate congenital or developmental hip dislocation. Leg length in children can be evaluated by measuring from the anterior superior iliac spine to the medial malleolus. Abnormal leg length should be evaluated by bone scan and referred to pediatric orthopedics for further evaluation.

Inspect the *malleoli* to evaluate the presence of torsion in the lower extremities. The medial *malleolus* lies at the distal end of the *tibia*, forming the medial ankle, and the lateral *malleolus* forms the lateral ankle at the distal end of the *fibula*. In the infant the medial and lateral malleoli are parallel when examining the infant supine. A rotation of up to 20 degrees posteriorly occurs in the lateral malleoli during the normal growth and development of the musculoskeletal system.

At birth, the infant has significant torsion in the lower extremities. Intrauterine positioning may be a contributing factor to the degree of bowleggedness or *genu varum* in the infant and young child. Genu varum is a normal condition until 2.5 to 3 years of age (Fig. 19.11). It is the most common cause of intoeing in children less than 3 years of age and normally resolves with growth. Standing behind the young child, observe the child walk while wearing only a diaper or underwear. Observation of gait is important in evaluating the impact of genu varum on motor development and performance. Genu varum or bowing after 2.5 to 3 years of age that is severe can be the result of nutritional deficiencies or obesity in young children and may require further evaluation.

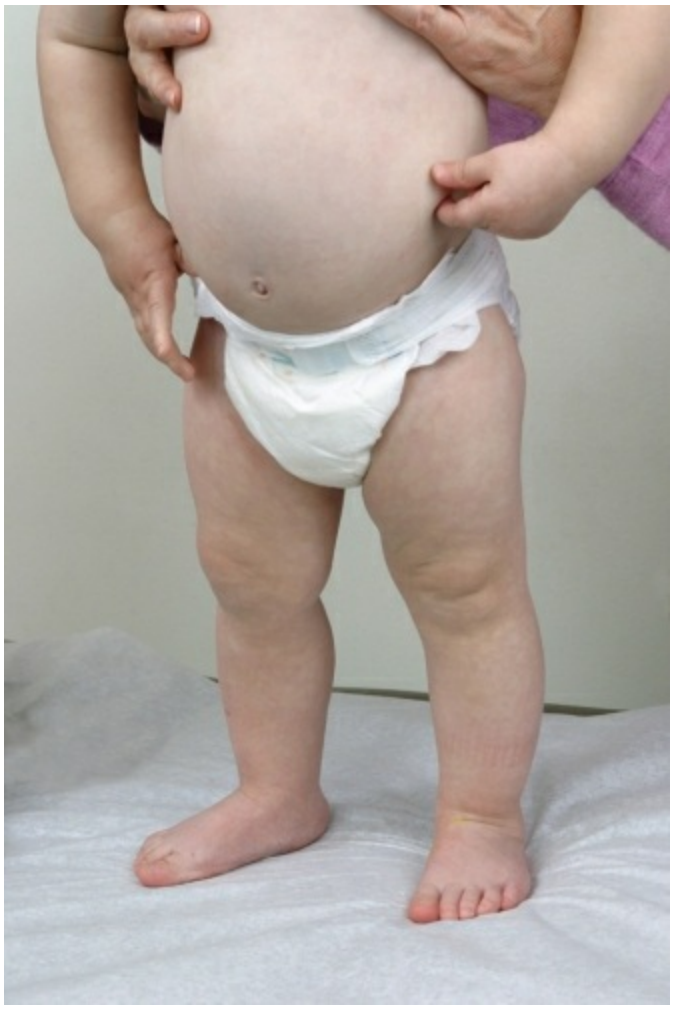


FIGURE 19.11 Genu varum (bowleggedness).

Tibial torsion, a curvature or twisting of the tibia also often referred to as *bowleggedness*, is a common finding in young children and is the most common cause of intoeing. The child should be examined wearing only underwear and be seated with the legs dangling freely from a chair or the exam table (Fig. 19.12). The provider places a thumb and forefinger on the lateral and medial malleoli with the knee facing forward to determine the degree of rotation and flexibility of the tibia. The forefoot and hindfoot should be in line with the knees. On inspection, only the anterior edge of the lateral malleolus should be in the midline. Tibial torsion generally resolves with growth and is common until 4 or 5 years of age. Therefore reassuring the parent is an important component of anticipatory guidance.

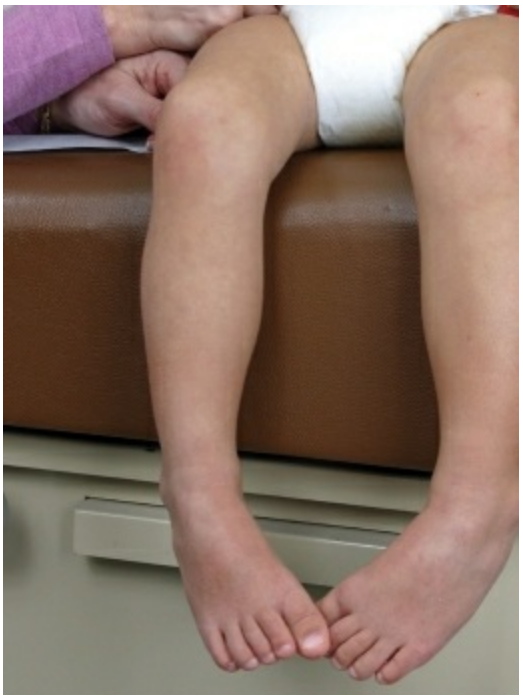


FIGURE 19.12 Tibial torsion.

Genu valgum, or knock-knee, should be evaluated with the child standing and is present if the medial malleoli are more than an inch apart when the knees are touching. Genu valgum is normal until 7 years of age and resolves in middle childhood with the rotational development of the lower extremities (Fig. 19.13). Persistence of genu valgum may be familial or the result of childhood obesity and, if persistent, may require further evaluation and referral.

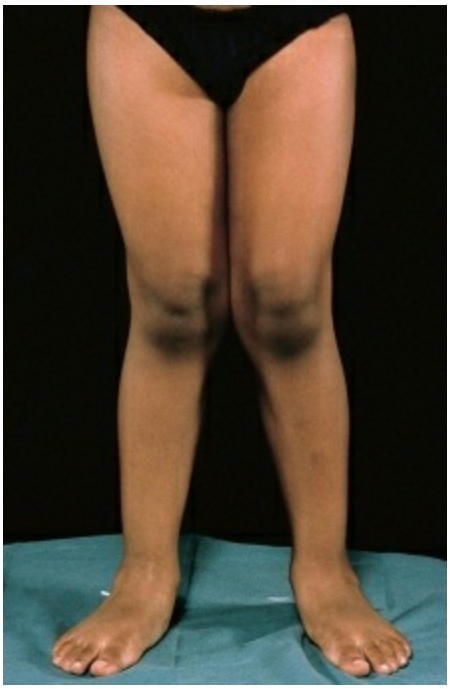


FIGURE 19.13 Genu valgum (knock-knee). Source: (From Chaudhry B, Harvey D: *Mosby's Color Atlas and Text of Pediatrics and Child Health*, St. Louis, 2001, Mosby.)

Gower sign is a screening test for muscle weakness in children and is an early sign of a neuromuscular abnormality often associated with *Duchenne muscular dystrophy*. Ask the child to stand from a sitting or supine position. The child with muscle weakness will move from a supine position to standing by facing prone and then grasping on the legs and hips with the hands to push the trunk up until they are erect. A child with good muscle strength will rise to a standing position without using the arms for leverage.[3]

Assessment of the feet in the infant includes the position and alignment of the forefoot and heels and the range of motion of the ankle and plantar arch. Limited dorsiflexion, or a fixed position of the hindfoot, is abnormal in the newborn, as is adduction of the forefoot and may be diagnostic of *clubfoot*. Decreased range of motion or pain should be noted on exam and referred for further evaluation. With the infant supine and the knees flexed to 90 degrees, inspect the thigh-foot angle and evaluate any adduction of the forefoot past the midline, which may be diagnostic for *metatarsus adductus* (Figs. 19.14 and 19.15). A foot that is rigid on range of motion requires further evaluation. A mild deformity with a flexible foot requires passive stretching, which consists of

supporting the heel at a right angle to the leg and rotating and stretching the forefoot laterally.

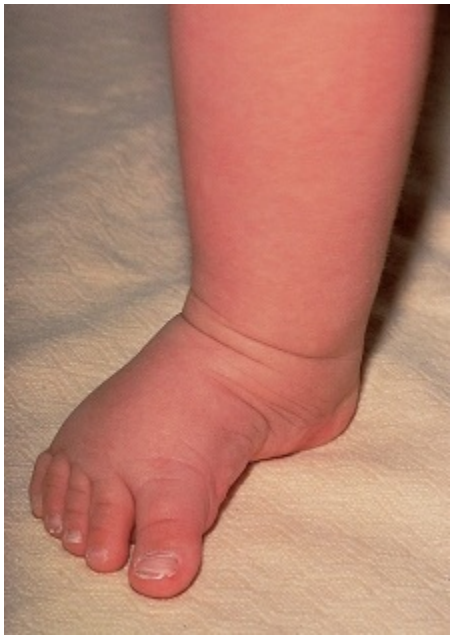


FIGURE 19.14 Metatarsus varus (adduction of the forefoot). Source: (From Chaudhry B, Harvey D: *Mosby's Color Atlas and Text of Pediatrics and Child Health*, St. Louis, 2001, Mosby.)

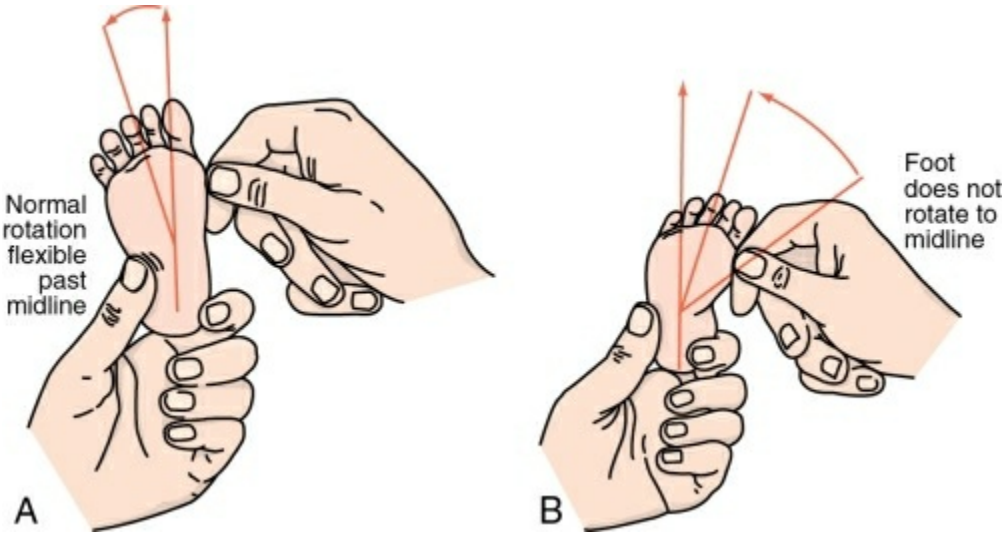


FIGURE 19.15 Examination of the forefoot. **A,** Normal rotation of foot. **B,** Fixed position of forefoot.

Infants and toddlers do not develop a longitudinal arch until the

second or third year of life. Lack of development of a longitudinal arch in the young child may indicate a generalized laxity of the ligaments or flat foot, *pes planus,* and a thorough assessment of muscle tone is warranted. Pes planus is hereditary, and use of orthotics and referral is required for further evaluation if symptomatic. Examine the feet with the child standing erect and on tiptoe to determine the longitudinal arch of the foot (Fig. 19.16). Older children may have physiologic flat feet due to laxity of the ligaments with rapid growth, which is a normal variant and usually improves with age.

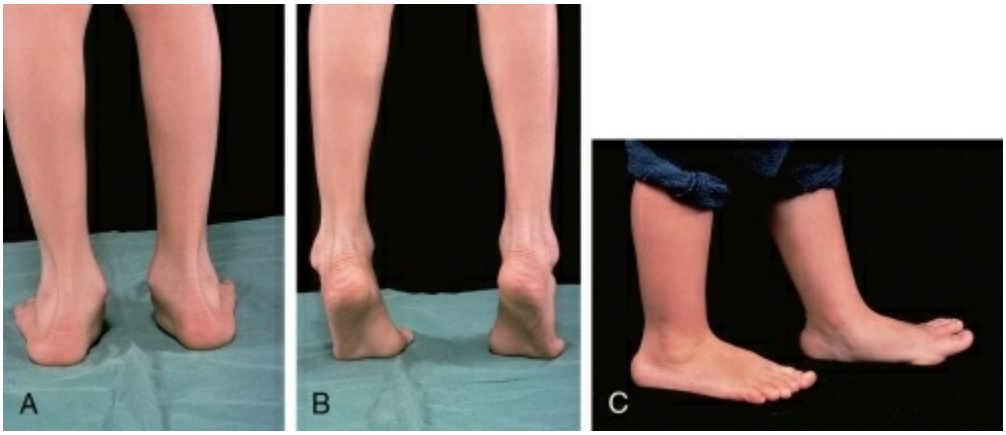
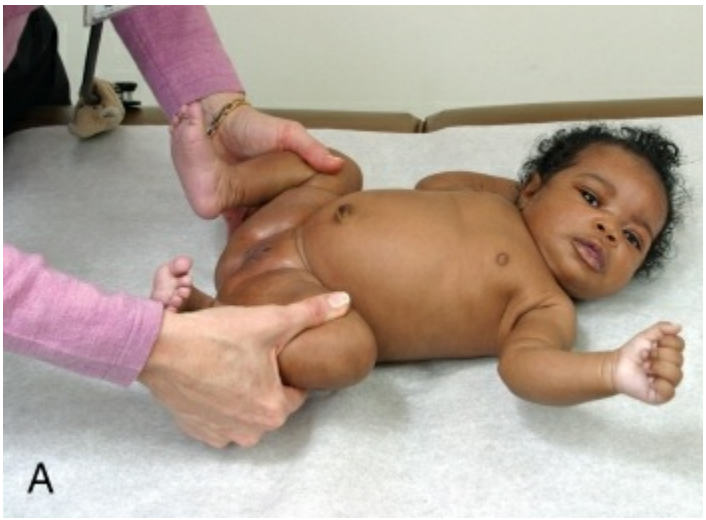


FIGURE 19.16 **(A)** Physiologic flat feet. **(B)** Normal arch on tiptoe. **(C)** Pes planus (flat foot). Source: (From Chaudhry B, Harvey D: *Mosby's Color Atlas and Text of Pediatrics and Child Health*, St. Louis, 2001, Mosby.)

Hip

The examination of the hip in the newborn and growing infant is performed with the infant in the supine position with the knees flexed bilaterally and supported by the thumb and forefinger of the examiner, with the pad of the second finger on the bony prominence of the greater trochanter and the thumb near the lesser trochanter (Fig. 19.17A). It is best to have the diaper open when examining the hips. Fully abduct the thighs to the examining table; mild pressure to the greater trochanter may cause the unstable hip to partially dislocate, *subluxation,* or dislocate. *Ortolani sign*, the presence of a *clunk* during the maneuver, is a positive sign for

dislocation of the hip. *Barlow test* also assists in detecting the unstable or dislocatable hip. With the infant supine and the knees flexed, the thigh is grasped and adducted while applying downward pressure (see Fig. 19.17B). With pressure on the acetabulum, the hip goes from a reduced position to a dislocated position. A dislocation of the femoral head is a positive Barlow. A *click* can occur during the maneuvers that may radiate from the knee and is often associated with crepitus of the joint, which is a normal finding. *Galeazzi* or *Allis sign* is also used to detect unequal leg length or hip dislocation. With the knees in the infant flexed to 90 degrees and the feet stabilized on the examining table, inspect for symmetry of height of knees. The knees should be equally aligned. Any asymmetry of the knees is abnormal and may indicate a subluxated or dislocated hip that requires further evaluation. With the infant prone, inspect for the symmetry of the thigh folds (see Fig. 19.17C). Asymmetry should be noted, but it is not highly correlated with hip abnormality. Examination of the hips is indicated until an infant is walking independently without support and gait is normal.



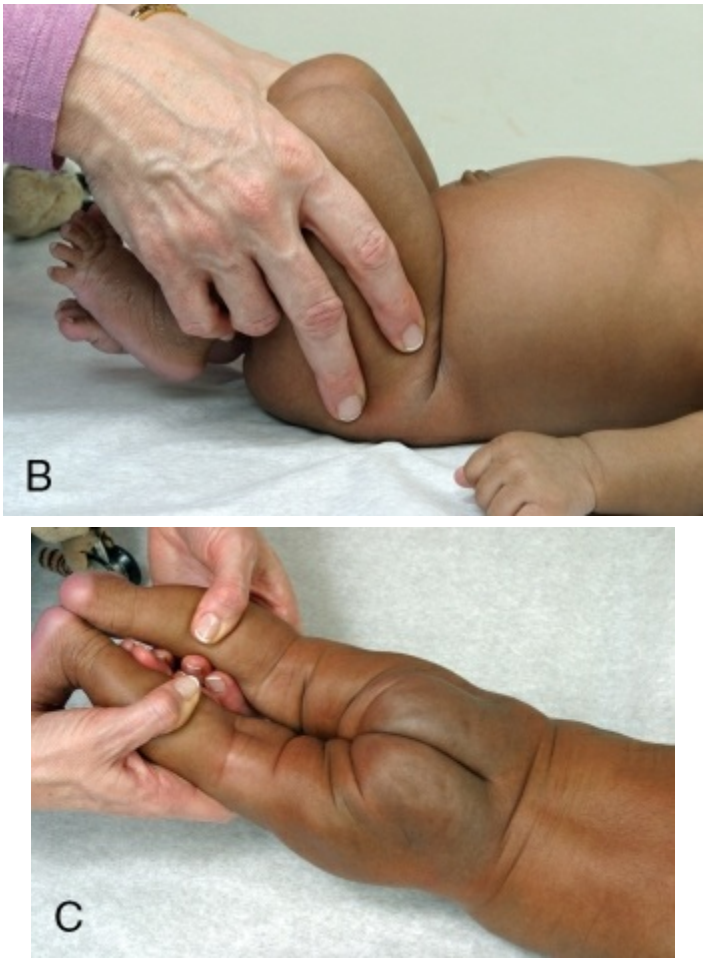


FIGURE 19.17 **(A)** Ortolani sign. **(B)** Barlow sign. **(C)** Inspecting thigh folds.

Developmental dysplasia of the hip (DDH) is a congenital or acquired condition involving an improper alignment of the radial head of the femur and the acetabulum. DDH is associated with a range of conditions. The incidence of DDH in infants remains controversial due to the changing development of the femoral head in the acetabulum. The incidence of DDH with dislocation or hips with persistent dysplasia is 3 to 5 per 1000 infants. The overall incidence of DDH including infants with a partial dislocation or subluxation of the hip or an unstable hip is higher, at 1.4 to 35 per 1000 infants.[4] In infants with a positive clinical examination in the early neonatal period, DDH resolves in 60% to 80% of infants by 2 to 8 weeks of age.[5] Screening for DDH leads to earlier detection in affected infants but can lead to overtreatment. Therefore the recommendation for routine clinical examination and screening remains controversial.[6] Infants born in breech presentation, female

infants, and infants with a positive family history of DDH are at higher risk, and ultrasound and referral is recommended.[4] Clinical examination and observation of an unstable hip by the pediatric health care provider can limit unnecessary imaging and overreferral in the first 2 months of life. Nine percent of all primary hip replacements and up to 29% of hip replacement in adults up to 60 years of age are related to DDH, so detecting and referring the infant with persistent misalignment of the femoral head in the acetabulum remains important to prevent premature degenerative joint disease in adulthood.[7]

Examination of the hip in middle childhood and adolescence begins with observation of the standing posture. The height of the iliac crests should be level or equal, and the child should be able to stand on one foot without any tilting of the pelvis.[8] A positive *Trendelenburg sign* is noted when the iliac crest drops, indicating weak hip abductor muscles on the weight-bearing side. The range of motion of the hips should be evaluated with the child or adolescent lying supine. With the hip and knee fully flexed, rotate the hip joint medially and laterally and note any pain or asymmetry in range of motion. Any child with abnormal findings, pain on examination, and/or presenting with a limp should be further investigated and referred if indicated.

Knee

The knee is a common site for both overuse and injury in middle childhood and adolescence. The symmetry and range of motion of the knee should be assessed initially with the child standing. Note any swelling in the knee and pain with weight bearing. The remainder of the knee examination should be performed with the child lying supine on the exam table. The *patella* should be evaluated for any abnormal tilt or alignment from the midline and for any evidence of effusion in the joint, or *ballotment*. Joint line tenderness or pain may indicate a *meniscal tear*.[9] Inspect and palpate knee for any swelling, obvious deformity, and incomplete range of motion on medial or lateral rotation, as well as signs of *ligamentous* laxity. Children and youth with a history of knee pain or laxity, as well as those who are unable to fully squat and perform a duck walk, require further evaluation and referral.

A common complaint of adolescent athletes is medial or anterior patellar pain with exercise or prolonged sitting, and pain or buckling when ascending or descending the stairs. Pain may be elicited on exam; however, findings may also be relatively normal. Sudden traumatic injury of the knee in the young athlete is common, particularly in adolescent females. Box 19.2 presents the physical examination of the knee and assists with decision-making regarding the need for imaging and referral. The knee maneuvers should be performed bilaterally beginning with the noninjured knee with the child lying supine on the examining table. Some variation in the flexibility and laxity of the joints is within normal.

BOX 19.2

Examination of the Knee

Lachman test

- To assess anterior cruciate ligament (ACL), knee should be flexed 20–30 degrees with child or adolescent lying supine; stabilize the femur with one hand while pulling the tibia anteriorly with the dominant hand; feel resistance at firm endpoint and note how far tibial tubercle moves anteriorly compared with the unaffected knee.
- **Positive finding:** Excessive movement of tibial tubercle anteriorly or forward from neutral position indicates partial or complete tear of the ACL.[6]

Anterior/posterior drawer test

- To assess the ACL and posterior cruciate ligament (PCL) for injury, hips should be flexed to 45 degrees and knees flexed to 90 degrees with child lying supine; foot should be stabilized on exam table in neutral position with examiner sitting on the child's foot. With the hamstrings relaxed, pull the tibia anteriorly (anterior drawer test) and posteriorly (posterior drawer test) from neutral position (Fig. B19.2A).

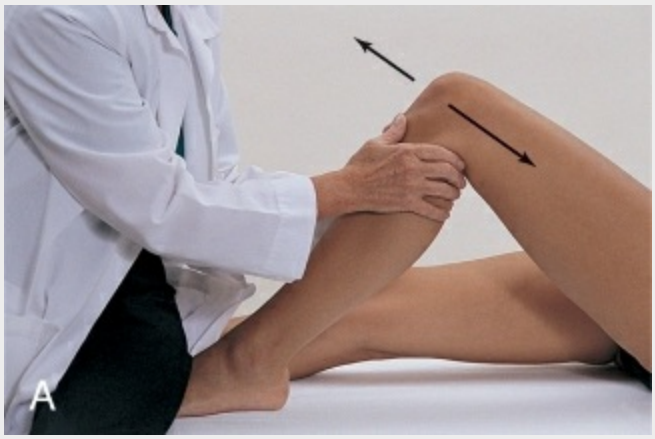


FIGURE B19.2A Drawer test

- **Positive finding:** Excessive movement of tibia anteriorly from neutral position or lack of firm endpoint indicates ACL insufficiency. Excessive posterior tibial sag suggests PCL insufficiency. [6] The PCL is normally placed anterior to femur with knee flexed to 90 degrees. Significant laxity on exam suggests tear of ACL or PCL.

Varus/valgus stress test

- To assess the stability of medial collateral ligament (MCL) and lateral collateral ligament (LCL), with the child lying supine, leg should be extended without flexion in knee with femur and ankle stable on exam table. With examiner's hand stabilizing knee joint anteriorly, lift knee to 20–30 degrees and apply varus stress to tibia; move hand to posterior side of knee and apply valgus stress to knee (see Fig. B19.2B).

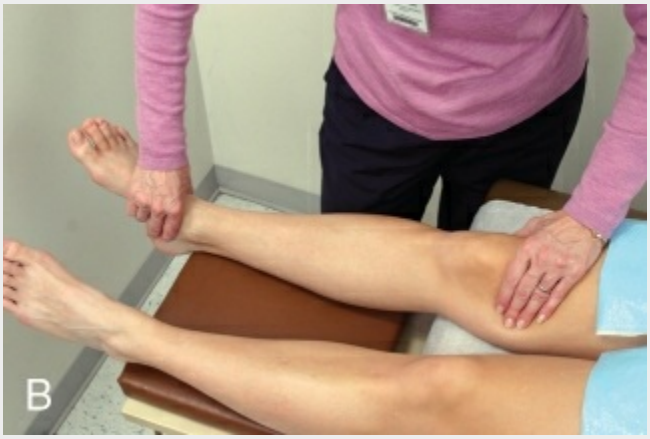


FIGURE B19.2B Varus/valgus stress test with knee

extended

- Note: For valgus stress, push on the lateral knee, stabilizing at medial malleolus. For the varus test, push on medial knee while stabilizing lateral malleolus.
- **Positive finding**: Lateral knee pain or excessive varus movement of tibia indicates injury to LCL. Medial knee pain or increased valgus movement with lack of a firm endpoint indicates injury to MCL. Laxity or opening in the joint space suggests tear of MCL or LCL.

Mcmurray test

- Assess the medial and lateral meniscus with child lying supine, flex knee to 90 degrees while supporting foot and lower leg. Stabilize medial and lateral joint lines of knee with thumb and forefinger (see Fig. B19.2C). Using other hand, rotate tibia medially (valgus force) to test medial meniscus and laterally (varus force) to test lateral meniscus while extending knee to 90 degrees.

FIGURE B19.2C McMurray test

- **Positive finding:** Any clicking or pain along medial joint line indicates a medial meniscus tear. A click or pain along lateral joint indicates a lateral meniscus tear.

Ballottement of patella test

- Ballottement sign: With child lying supine and knee fully

extended, examiner applies downward pressure on suprapatellar pouch to force fluid between patella and femur; then push the patella downward against the femur (see Fig. B19.2D).

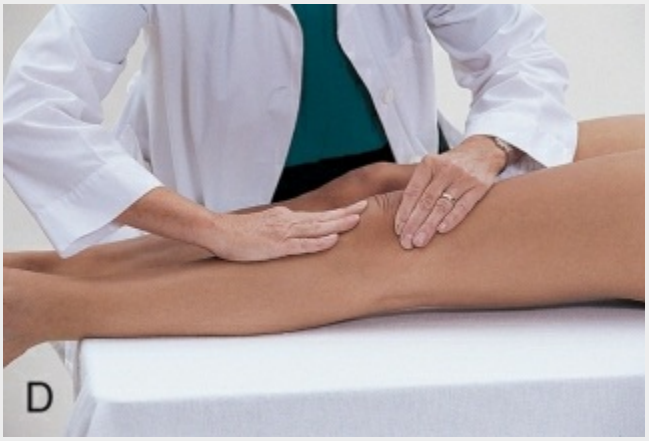


FIGURE B19.2D Ballottement/bulget test

- **Positive finding:** If patella floats back to neutral position, fluid is present. A palpable click of patella striking femur on downward pressure is positive for knee joint effusion.

Figures B19.2A and B19.2D, From Wilson S, Giddens J: Health assessment for nursing practice, ed 4, St. Louis, 2009, Mosby.

Spine

The spine should be inspected and palpated in infancy to note any congenital abnormalities, such as hair tufts, dimples, a sacral sinus, or hemangiomas, that could indicate spinal abnormalities. Young children develop a normal curvature of the spine with *lordosis* of the neck and lumbar region and *kyphosis* of the thorax. An exaggerated *lordosis* is normal in the young child. Viewing the child or adolescent from the side allows the examiner to evaluate normal alignment. Inspection of the spine in the child and adolescent begins with the examiner behind the child. Assess for the contour of the back, the symmetry of the shoulders, the symmetry and/or prominence/shape of the scapula and ribs, and the symmetry of the waistline and the iliac crests (Fig. 19.18A–C). The head should be

aligned directly over the sacrum because any deviation from the midline may indicate scoliosis or a spinal deformity. Range of motion of the spine is evaluated by asking the child or adolescent to bend to the side, flex, and extend. An elevated scapula, uneven iliac crests, or an uneven waistline, or the presence of a rib hump indicates a positive finding (Fig. 19.19). The *Adam's forward bend test* begins with the child or adolescent standing with knees straight and feet together. Ask the child to bend forward and to touch the toes with hands dangling or in a diving position (see Fig. 19.18D). Observe the spine for alignment, any curvature, asymmetry, or rib hump from the rear and sides. A *scoliometer* can be used to assess the angle of trunk rotation (Fig. 19.20). Place the scoliometer on the trunk at the peak of the curvature to evaluate alignment. A suspected curve or rotation of 10 degrees or greater requires further evaluation and referral.

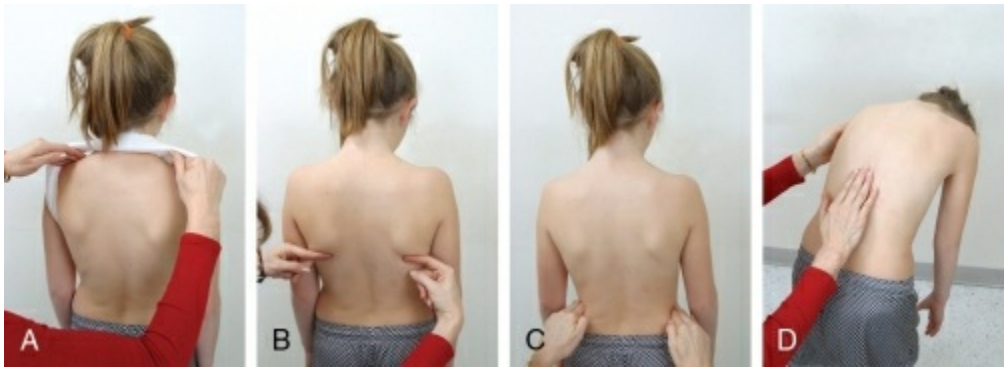


FIGURE 19.18 Assessment of the spine. (A) Testing shoulder symmetry. **(B)** Scapular symmetry. **(C)** Iliac crest symmetry. **(D)** Beginning Adams forward bend test.

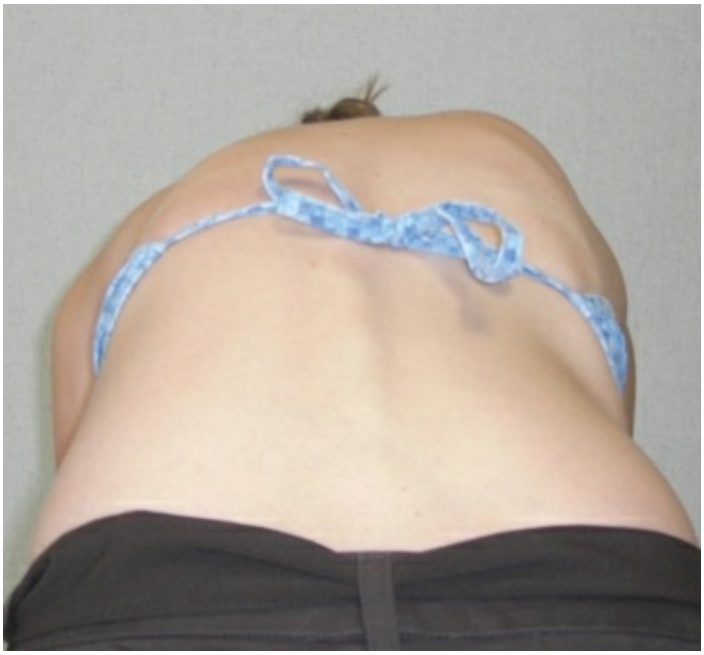


FIGURE 19.19 Positive rib hump. Source: (From Skirven T, Osterman A, Fedorczyk J, et al: *Rehabilitation of the Hand and Upper Extremity*, ed 6, Philadelphia, 2012, Mosby.)

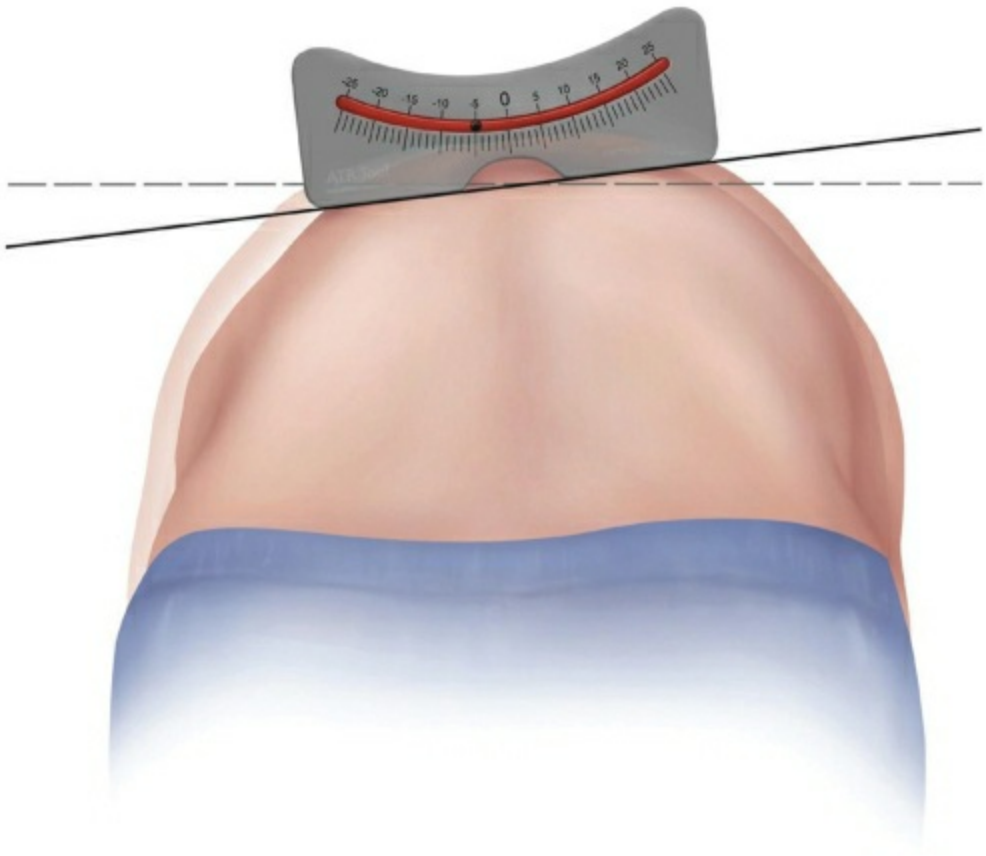


FIGURE 19.20 Scoliometer measuring inclination angle of thoracic spine. Source: (From Horne JP, Flannery R, Usman S. Adolescent idiopathic scoliosis: diagnosis and management, *Am Fam Physician* 89(3);193-198, 2014.)

Routine screening for scoliosis remains controversial, and the US Preventive Services Task Force no longer recommends routine school screenings because the benefits do not outweigh the risk of overreferral and unnecessary imaging. The American Academy of Pediatrics does recommend that pediatric health care providers screen for scoliosis and judiciously manage positive screening results and the need for further evaluation or referral. Adolescents who are in a period of rapid growth, have a positive family history, and have a more notable curve on presentation are more likely to progress and require imaging and referral. Females have up to a 10-fold greater risk of curve progression. Evaluation of *sexual maturity rating (SMR) or Tanner stage* is key to determining skeletal and spinal maturity when diagnosing scoliosis in adolescents.

Diagnostic procedures

Concern over the relationship of frequent cumulative exposure to radiologic diagnostic procedures, particularly computed tomography (CT) scans, and later health effects including cancer in children and adults remains. Pediatric health care providers have continued to adhere to the As Low As Reasonably Achievable (ALARA) policy and an Image Gently in most pediatric health care settings.[10]

Ultrasound is used to evaluate soft tissue masses and to diagnose DDH until 4 to 6 months of age, due to incomplete ossification of the femoral heads in early infancy.[11] Magnetic resonance imaging (MRI) is often used for higher anatomic detail for soft tissue masses, but sedation is frequently required.[11] *Bone age* radiographs of the secondary ossification centers at the end of the long bones are used to assess growth rate in children who lag behind in height velocity compared with the norm or who have delayed onset of puberty. A skeletal series of radiographs is indicated to detect occult fractures when serious physical abuse is suspected, especially in children under 2 years of age.[2] A *bone scan* or *CT scanogram* is used to diagnose and evaluate unequal leg length in childhood.[11]

Preparticipation sports physical

Preparticipation sports history

The primary purpose of the preparticipation sports physical exam is to exclude or restrict the young athlete with a temporary or permanent contraindication from participation in competitive sports. In addition, the pediatric health care provider can help to match the athlete to the appropriate type and level of sport and can help to prevent injury by recommending the appropriate stretching or strengthening exercises. The *preparticipation physical evaluation (PPE)* is often the healthy adolescent's only contact with a pediatric health care provider during an important period of physical and psychosocial growth and development. Therefore, the PPE should incorporate routine health maintenance and confidential psychosocial screening. It is important to evaluate any risks related to sports participation and use the encounter as an opportunity for

anticipatory guidance regarding reproductive health and in encouraging positive health behaviors.[12]

The PPE is also an opportunity for the pediatric health care provider to evaluate risky health practices related to participation in sports, particularly unhealthy weight loss or weight gain (Box 19.3). Sports such as wrestling, boxing, and martial arts are graded by weight class, which creates significant pressure on the young athlete to maximize body strength and bulk. There are equally powerful pressures on dancers, distance runners, and gymnasts to maintain a low weight. By contrast, youth playing positions such as linebacker in football may want to increase weight gain, even if their body mass index (BMI) is greater than the 95th percentile for age. Information gathering during the PPE should include satisfaction with or concerns about weight and body image. Athletes should also be questioned about such practices as severe food restriction, binging or purging, weight loss, or muscle-enhancing supplements, including medications containing *ephedra*, *creatine*, and anabolic steroids or their analogs. Tables 19.2 and 19.3 present a review of systems and medical conditions related to participation in competitive sports.

TABLE 19.2

Review of Systems for Preparticipation Physical Evaluation for Middle Childhood and Adolescence

System	Disorders
HEENT	Otitis externa, frequent otitis media (OM), allergic rhinitis?
Respiratory	Shortness of breath (SOB), wheezing with exercise?
Cardiovascular	SOB, chest pain, dizziness, syncope with exertion, palpitations with exertion?
Gastrointestinal	Pain or reflux with exercise? Current/chronic problems with diarrhea, constipation?
Genitourinary	*For males:* History or symptoms of hernias, lumps or masses in groin or testicles? *For females:* Menstrual history (menarche, length, regularity of cycles, missed cycles during sports)? Last menstrual period (LMP)?
Musculoskeletal	Instability of any joints (especially shoulder, knee, ankle)? Leg or foot pain with exercise? Swelling of joints? Any weakness?
Neurologic	Headaches, dizziness, seizures, recent concussions, recent injuries, weakness, difficulties with sleep, cognitive function, emotional regulation, balance or gait?

Dermatologic	*Lesions:* Recent history of herpes, fungal infections, bacterial infections? Extent of acne and use if retinoids? Sunscreen protection? Eczema, reaction of skin to perspiration or athletic equipment?

HEENT, Head, eyes, ears, nose, and throat.

TABLE 19.3
Medical Conditions and Level of Sports Participation

Condition	Level of Sports Participation and Rationale
Bleeding disorder	Qualified yes [a]
Cardiovascular disease Carditis (inflammation of the heart) Hypertension (high blood pressure) Congenital heart disease (structural heart defects present at birth) Dysrhythmia (irregular heart rhythm) Heart murmur	No May result in sudden death with exertion Qualified yes [a] Qualified yes [a] Those with mild forms may participate fully; those with moderate or severe forms or those who have undergone surgery need evaluation Qualified yes [a] Those with symptoms (chest pain, syncope, dizziness, shortness of breath) or evidence of mitral valve regurgitation (leaking) need evaluation; all others may participate fully Qualified yes [a] If innocent, full participation is permitted; otherwise, the athlete needs evaluation
Cerebral palsy	Qualified yes [a]
Diabetes mellitus	Yes Blood glucose concentration should be monitored every 30 min during continuous exercise and 15 min after completion of exercise
Diarrhea	Qualified no [a] Unless mild, no participation is permitted, may increase risk of dehydration
Eating disorders Anorexia nervosa Bulimia nervosa	Qualified yes [a] Qualified yes [a]
Fever	No Increases cardiopulmonary effort, reduces maximum exercise capacity, makes heat illness more likely, and increases orthostatic hypertension during exercise
Hepatitis/HIV	Yes All sports may be played that athlete's state of health allows; skin lesions should be covered properly, and universal precautions used when handling blood or body fluids with visible blood
Musculoskeletal disorders	Qualified yes [a]

Neurologic disorders History of serious head or spine trauma, severe or repeated concussions, or craniotomy	Qualified yes [a] Research supports conservative approach to management of concussion, with gradual return to play only when asymptomatic. Athletes with multiple concussions may need an extended time away from sports
Seizure disorder *Well controlled* *Poorly controlled*	Yes Risk of seizure during participation is minimal Qualified yes [a] Archery, riflery, swimming, weight or power lifting, strength training, or sports involving heights should be avoided because occurrence of a seizure may pose risk to self or others
Obesity	Qualified yes [a]
Organ transplant recipient	Qualified yes [a]
Respiratory conditions Pulmonary compromise, including cystic fibrosis Asthma Acute upper respiratory infection	Qualified yes [a] Yes Only most severe asthma requires modified participation Qualified yes [a] Individual assessment required for all but mild disease
Sickle cell disease or trait	Qualified yes [a] Carefully condition, acclimatize, and hydrate to reduce any possible risk
Skin disorders Boils, herpes simplex, impetigo, scabies, molluscum contagiosum	Qualified yes [a] While contagious, participation in gymnastics with mats; martial arts; wrestling; or other collision, contact, or limited-contact sports is not allowed

[a]Patient needs evaluation.

Adapted from Rice SG: Medical conditions affecting sports participation, *Pediatrics* 121(4):841-848, 2008; Halstead ME, Walter KD: American academy of pediatrics. Clinical report—sport-related concussion in children and adolescents, *Pediatrics* 126(3):597-615, 2010.

HIV, Human immunodeficiency virus.

BOX 19.3

Focused History for Preparticipation Physical Evaluation for the Middle Childhood and Adolescence

Current Medical History	Past Medical History	Family History	Dietary History
• Sport youth intends to play, including position and weight class if relevant • Any medications used by the youth, including diet supplements, creatine, anabolic steroids and their analogs, *macrolide* antibiotics, *tricyclic* antidepressants, *neuroleptics* • Any chronic illnesses, including asthma, how well controlled, and any exacerbations during exercise	• Any previous experience in the same or other sports, including previous injuries, especially injuries requiring exclusion for >1 week • Any history of shortness of breath, syncope, or chest pain during exertion • History of seizures, including type, frequency, controlling medication, and past complications • *For young women:* Age of menarche, regularity of menses, and any prior disturbances of menses during sports	• Family history of cerebrovascular accident (CVA) or myocardial infarction (MI) before age 50 years • Any family history of sudden, unexplained death in adolescent or young adult relative • Family history of hypertrophic cardiomyopathy (HCM), prolonged QT syndrome, Marfan syndrome, or other cardiac/circulatory abnormalities	Are you happy with current weight? Want to gain or lose for sport? • *Weight loss methods:* Ask explicitly: any bingeing, purging, restricting? • Fruits/vegetables daily? Milk or calcium intake? • Fluids for hydration (water, sports drinks, sodas, caffeine intake)? • Supplements, medications for weight loss, weight gain, muscle gain?

To perform the PPE, the young pubertal female athlete should be examined in gym shorts and sports bra, and young males should be in gym shorts to ensure a thorough assessment of the joints and muscles. A 14-step orthopedic exam is included in the PPE to assess the athlete's musculoskeletal health (Fig. 19.21). The components of the 14-point orthopedic examination are as follows:

1. With the young athlete standing, assess for frontal symmetry of trunk, shoulders, and extremities.
2. Assess neck flexion, extension, lateral flexion side to side, and rotation to evaluate range of motion of cervical spine.
3. Assess trapezius strength by having the young athlete shrug shoulders against resistance from the practitioner.
4. Assess deltoid strength by having the young athlete abduct the shoulders against resistance from the practitioner.
5. Assess internal and external rotation of shoulder to evaluate range of motion of the glenohumeral joints.
6. Assess range of motion of the elbows by having young athlete perform flexion and extension of the arms.
7. Assess range of motion of the wrists and elbows by observing pronation and supination of the forearm.
8. Assess range of motion of the hands and fingers by having the young athlete clench the fist and spread the fingers.
9. Assess symmetry of the posterior body with the young athlete standing.
10. Have the young athlete stand with knees straight and then flex forward and bend backward to assess any discomfort of the lumbar spine.
11. Perform Adam's forward bend test by having the young athlete bend forward and touch the toes if possible with hands dangling

or in a diving position. Assess for rib hump or asymmetry.
12. To assess for symmetry of leg musculature, have the young athlete stand facing the practitioner with quadriceps flexed.
13. Assess hip, knee, and ankle range of motion, strength, and balance by having the young athlete duck walk four steps.
14. Assess calf strength, symmetry, and balance by having the young athlete stand on heels and then toes.

FIGURE 19.21 Fourteen-step orthopedic examination. Source: (Modified from American Academy of Family Physicians, American Academy of Pediatrics, American College of Sports Medicine, American Medical Society for Sports Medicine, American Orthopedic Society for Sports Medicine, American Osteopathic Academy of Sports Medicine: *PPE: Preparticipation Physical Evaluation*, ed 4, Elk

Grove, IL, 2010, American Academy of Pediatrics.)

Cardiac preparticipation physical evaluation

The American Heart Association (AHA) recommends the PPE include (1) auscultation for heart murmurs, (2) palpation for femoral pulses to assess for coarctation of the aorta, (3) evaluation for the physical findings of *Marfan syndrome,* and (4) blood pressure evaluation of the brachial artery in sitting position.[13] The AHA also recommends gathering a detailed family history and personal history to identify asymptomatic young athletes with underlying cardiac disease (Box 19.4).[13]

BOX 19.4

American Heart Association Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes

Personal history

1. Exertional chest pain/discomfort
2. Unexplained syncope/near-syncope[a]
3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure

Family history

6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease, in ≥1 relative
7. Disability from heart disease in a close relative <50 years of age
8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome,

or clinically important arrhythmias

Physical examination

9. Heart murmur[b]
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure (sitting position)[c]

[a]Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion.
[b]Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.
[c]Preferably taken in both arms.
Parental verification is recommended for high school and middle school athletes.
Adapted from Maron BJ, Friedman RA, Kligfield P, et al. "Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12-25 years of age): a scientific statement from the American Heart Association and the American College of Cardiology," Circulation 130(15):1303-1334, 2014.

In young athletes, *hypertrophic cardiomyopathy,* a cardiovascular condition caused by an asymmetrical left ventricular hypertrophy and a nondilated left ventricle with impaired diastolic function, is the leading cause of sudden cardiac death. *Sudden cardiac death* is defined as a nontraumatic death by cardiac arrest within 6 hours of a previous state of good health.[14] The incidence is thought to be 1 in 200,000 high school athletes and 1 in 65,000 college athletes,[14] although the incidence is higher in African American athletes compared with white athletes and to young men compared with young women.[15] In general, no evidence of cardiac disease is present before sudden cardiac death. Other cardiac conditions implicated in sudden cardiac death include anomalous coronary arteries, Marfan syndrome, prolonged QT interval, and cardiac *dysrhythmias.* Following the example of countries such as Italy, some US experts recommend a screening ECG for all adolescent athletes.[15] However, the AHA has recently affirmed its stance against universal ECG screening. Instead, they recommend that any concerning history elements and any findings on physical examination, which include hypertension, absence of femoral pulses, signs of stigmata of Marfan syndrome, or auscultation of

heart murmur, would indicate the need for referral to pediatric cardiovascular specialists.[13]

Sports injuries

Sports injuries can be classified as traumatic *(macrotrauma)*, usually from collision or contact sports, such as basketball, football, or soccer, or as overuse *(microtrauma)*. The pediatric health care provider should assess the level of sports participation, recreational versus competitive sports, that young athletes are involved in when gathering history about injuries. Young athletes who play high-contact sports such as football, basketball, and soccer should be screened for history of previous concussion, fractures, and ligament damage. Athletes who participate in lower-contact sports, such as running or swimming, should be screened for repetitive motion and overuse injuries to the shoulder, knee, elbow, or shin (tibia).

Children who are overweight or obese have a higher number of reported fractures, more musculoskeletal discomfort, and impaired mobility.[16, 17] The incidence of lower extremity malalignment predisposing children and adolescents to injury are also more prevalent in overweight children and adolescents. Studies show young female athletes are at greater risk than young male athletes for stress fractures, particularly tibial stress fractures.[18] In addition, young female athletes who are runners sustain an overall higher injury rate than young male runners.[18] A young athlete's SMR is also relevant to injury and the mechanism for injury. Rapid growth during adolescence results in decreased strength in the growth plates and greater strength in ligaments compared with bones, leading to greater potential for fractures relative to older adolescents and adults.[19] Rapidly growing athletes with relative good muscle strength yet incomplete ossification of the growth plate are predisposed to physeal (growth plate) fractures. Because injuries may recur from season to season, young athletes with a history of repetitive injuries such as frequent ankle sprain should be given strengthening exercises at the time of the PPE to help prevent future injury. Early sports specialization may lead to overuse injuries, and cross training in young athletes is recommended.[20] For most adolescents the physical and social benefits of carefully

monitored sports participation outweigh the risks, including adolescents with chronic health conditions and special health care needs.

PEDIATRIC PEARLS

Ligaments are stronger than bones until adolescence; therefore injuries to long bones and joints in the preadolescent are more likely to cause fractures rather than sprains.

Sports-related concussions

Concussions are common, often unrecognized, and usually do not involve loss of consciousness.[6] *Concussion* is defined as an impact to the brain, face, neck, or elsewhere on the body with an "impulsive force" transmitted to the head, resulting in rapid onset of neurologic symptoms, with or without loss of consciousness, with spontaneous (though often delayed) resolution.[21] There has been an increased focus on the immediate and long-term sequelae of concussion as a subset of *traumatic brain injury* (TBI) in the pediatric and adolescent athlete that is due to their increased in participation in high-impact competitive sports.[21, 22] It has been estimated that by the time a young athlete reaches high school, 53% will have reported a history of concussion.[23] Athletes younger than 18 years take longer to recover from concussion than older athletes. The short sequelae of concussion may include *postconcussion syndrome,* which can last for weeks to months, and *second impact syndrome* can occur if a second head injury occurs during the postconcussion period. The long-term sequelae of concussion include permanent neurologic deficits, such as decreased cognitive functioning and limiting cognitive potential.[21]

Current guidelines for concussion recommend cognitive rest, in addition to exclusion from play until completely asymptomatic, with a gradual return to play after symptoms resolve. In no cases should the athlete return to play on the same day as the injury.[21] Information gathered during the PPE should include screening questions related to prior concussions and related symptoms, including any headaches or cognitive, emotional, or sleep

impairment. See Box 19.5 for signs and symptoms of concussion. The *Sports Concussion Assessment Tool 3 (SCAT3)* for evaluation of young athletes 13 years of age and older following history of head injury or concussion, and a Child SCAT3 for children 5 to 12 years of age and be obtained at http://bjsm.bmj.com/content/47/5/263.full.pdf.

Box 19.5

Signs and Symptoms of Concussion

Cognitive symptoms

- Confusion
- Posttraumatic amnesia
- Disorientation
- Difficulty focusing
- Excessive drowsiness
- Delayed verbal or motor responses
- Slurred or incoherent speech
- Vacant stare
- Loss of consciousness

Physical symptoms

- Headache
- Fatigue
- Nausea and vomiting
- Visual disturbance
- Phonophobia

Affective symptoms

- Irritability
- Emotionally labile

Data from Bernhardt DT, Roberts WO: *PPE: Preparticipation Physical Examination*, ed 4, Elk Grove, IL, 2010, American Academy of Pediatrics.

The female athlete

Female athletes are at higher risk than male athletes for certain musculoskeletal injuries, such as noncontact anterior cruciate ligament (ACL) injuries and dislocation of the patella. The lowest intake of vitamin D and calcium are in female adolescents and female young adults. Eating disorders are also more common in female adolescents than male adolescents. *Patellofemoral pain syndrome* is also more common in female athletes, and it often presents as complaints of anterior knee pain radiating from the posterior patella and biomechanical or physical changes in the patellofemoral joint. It is an overuse or overload injury occurring from repeated weight-bearing impact and may be managed with rest and quadriceps strengthening exercises.

Undernutrition in female adolescents can impair skeletal health and impact bone mineral density (BMD) in adulthood. The *female athlete triad* comprises menstrual function, BMD, and energy availability and is more common in sports that emphasize endurance and a lean body.[24] Although previously defined as amenorrhea, osteopenia, and disordered eating, the current definitions allow for conceptualization of each element of the triad on a spectrum from health to severe dysfunction.[24] Furthermore, studies have shown that inadequate energy intake may be unintentional, rather than a sign of an eating disorder. It is thought that between 4% and 18% of female high school athletes have two of the triad components concurrently and the prevalence of one component is between 16% and 54%. Casual comments by coaches or family members about the adolescent's need to lose weight can be triggers for disordered eating.[25] Whether intentional or not, prolonged inadequate energy availability affects bone density accumulation and puts athletes at increased risk for stress fractures, with greater menstrual disruption and injury seen in those teens who do intentionally restrict their energy intake.[24]

Musculoskeletal conditions

Table 19.4 presents the most common abnormal orthopedic conditions seen in infants, children, and adolescents by the

pediatric health care provider.

TABLE 19.4
Musculoskeletal Conditions

Condition	Description
Blount disease	Growth disorder of the tibia abnormal ossification of the proximal tibia, also known as *tibia vara*
Clubfoot	Rigidity of foot and inability of foot to right itself from fixed medial position
Talipes equinovarus	Inversion of forefoot, plantar flexion, and heel inversion
Talipes calcaneovalgus	Eversion and dorsiflexion of forefoot
Metatarsus adductus	*Varus* abnormality of forefoot at the tarsometatarsal junction; ankle and hindfoot are normal; lateral border of foot is curved rather than straight, usually shaped like a "kidney bean"; line drawn medially from heel often intersects third toe
Pes planus (flat feet)	Flattening of longitudinal arch in school-age child when standing erect with full weight bearing on feet bilaterally; flat feet are developmentally normal until 3 years of age and often are accentuated by a fat pad on ventral surface of foot
Tibial torsion	Inward twisting or bowing of tibia and fibula, often a variation of normal rotational development; intrauterine position may be contributing factor; most common cause of intoeing children <3 years of age; resolves with normal growth; continued reassurance to parent is important
Slipped capital femoral epiphysis (SCFE)	Displacement of the capital or proximal femoral epiphysis from the femoral neck through the physeal plate; presenting symptoms are limp, knee or hip pain, particularly with strenuous activity; common presentation in obese child in early puberty during rapid growth period, male-to-female ratio 1:5–1; requires urgent orthopedic consultation and referral
Legg-Calvé-Perthes	Blood supply to femoral capital epiphysis is disturbed and produces avascular necrosis of femoral head; affects children 3–12 years of age, with peak incidence in males 5–7 years of age; male-to-female ratio 4:1; children may have history of intermittent limp and present with diffuse pain in hip, knee, or the upper thigh; on examination, child has limited internal rotation of effected hip; a high index of suspicion and early diagnosis and management are key to preventing long-term sequelae
Femoral anteversion	Increased internal rotation of femoral head at the hip; common cause of intoeing after 3 years of age; female-to-male ratio 2:1; evaluate child when undressed and lying in prone position; note internal rotation of hips bilaterally; resolution usually occurs by 11 years of age
Osgood-Schlatter syndrome	Inflammation of proximal tibia causing swelling and tenderness at insertion of infrapatellar tendon into tibial tubercle; presents with pain which increases after vigorous physical activity and is relieved with

	rest; common finding in adolescent athletes with peak occurrence from 11–14 years of age
Osteogenesis inperfecta	An inherited connective tissue disorder and deficiency of type I collagen but has multiple phenotypes resulting in brittleness of bones characterized by spontaneous fractures, and short, bowed extremities; associated with scoliosis, bluish sclera, and hearing loss
Marfan syndrome	Inherited connective tissue disorder characterized by excess linear growth of the long bones and hypermobility of joints, arachnodactyly (long/slender fingers/toes), pectus deformities, hindfoot valgus, longer extremities in proportion to trunk; may be associated with serious cardiac manifestations (increased risk of aortic enlargement and mitral valve prolapse)

Summary of examination

- Nutritional factors, such as adequate protein in the diet, the amount of calcium intake daily, and adequate intake of vitamin D, which regulates the absorption of calcium and phosphorus in the intestines, impact bone growth.
- Growth along the epiphyseal plate continues until the cells in the growth plate mature and stop dividing in puberty, and closure of the growth plate occurs in young adulthood.
- Evaluating progression of the height growth curve is critical to assessing normal and abnormal growth patterns, along with familial patterns of growth and pubertal development.
- Note any obvious gait abnormalities; observe posture when standing and sitting; assess proportion of upper extremities to lower extremities. Standing behind the young child, observe the child walk to evaluating torsional variations of *genu varum* and *genu valgum.*
- Evaluate range of motion of joints and assess muscle tone of trunk and extremities.
- Evaluation of the lower extremities includes assessment of flexion/extension, adduction/abduction, and internal/external rotation.
- Assessment of the hip in the young infant is performed in supine position with the knees flexed bilaterally and thumb and forefinger of examiner on the bony prominence of greater trochanter. *Ortolani sign* tests for dislocation of hip and *Barlow test* evaluates stability of hip.

- Knee assessment maneuvers should be performed bilaterally beginning with the noninjured knee. Some variation in the flexibility and laxity of knee joints is normal.
- Assess for contour of back, symmetry of shoulders, shape and/or prominence of scapula and ribs, and symmetry of iliac crests.
- Observe for alignment of spine with child or adolescent in forward bend position. Note any curvature, asymmetry, or rib hump from the rear and sides of the back.
- To perform the PPE, a 14-step orthopedic exam is included to assess the athlete's musculoskeletal health.
- The AHA recommends the PPE include (1) auscultation for heart murmurs; (2) palpation of femoral pulses; (3) evaluation for *Marfan syndrome;* and (4) blood pressure evaluation.

DOCUMENTATION

A healthy young child

Extremities and Back: nL ROM (range of motion), mild tibial torsion and intoeing bilaterally R > L, spine straight with mild lordosis.

DOCUMENTATION

14 year old PPE

Extremities and Back: nL ROM (range of motion) & strength bilaterally, 14 pt. exam nL, spine straight, no scoliosis noted.

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CHAPTER 20

Neurologic assessment

Karen G. Duderstadt

The normal development of the nervous system provides the vital motor, sensory, and cognitive functions that sustain human life and comprise human behavior. For the pediatric health care provider, surveillance and monitoring the development of the nervous system is one of the most important aspects of assessment in infants and children. The nervous system contains the most complex and delicate pathways in the body and is the center of all vital bodily functions. The challenge is to assess not only the progress of gross motor and fine motor skills in infants and children, but also to assess cognitive development and subtle deficits in attention and information processing that may impact learning behavior.

Embryologic development

The formation of the nervous system begins very early during the third week of embryonic life. Cell differentiation when the ectoderm, mesoderm and endoderm split to begin forming tubes which support organ growth. The ectoderm forms skin, hair, nails, brain, spinal cord and peripheral nervous system.[1] The *notochord*, which becomes the spinal column, develops during this period and forms the neural plate and neural folds. Closure of the neural plate is complete by the fourth week of embryonic development, and the

neural tube is formed. Any fetal insults that occur during this period can result in defects in the brain and spinal cord, such as anencephaly or *spina bifida* defects, which occur in 1 of 1000 live births. During the fifth week of fetal development, the anterior portion of the neural tube enlarges to form the segments of the brain. Brain growth proceeds, with the most rapid brain growth occurring between 15 and 20 weeks of gestation.

By 24 weeks of gestation, the fetus has developed most of the nerve cells, or neurons, needed for the formation of the neural pathways. The germinal cell matrix is highly vascular and is prone to hemorrhage in the preterm infant.

The *neuron* is the basic unit of the nervous system, and each neuron contains numerous dendrites and one axon (Fig. 20.1). Dendrites are the protoplasmic branches of the cell body. Neural impulses enter the cell body through the dendrites and leave through the single axon. They then connect by a series of synapses with another dendrite of the next axon. At the synaptic junction, neurons also release specific neurotransmitters (such as GABA, glutamate, acetylcholine, norepinephrine, dopamine, serotonin and histamine) that regulate brain and body functions. *Myelin,* a lipoid material surrounding cell fibers, covers the axons and allows for rapid and smooth transmission of nerve impulses. However, only a portion of the axons contain myelin at birth.

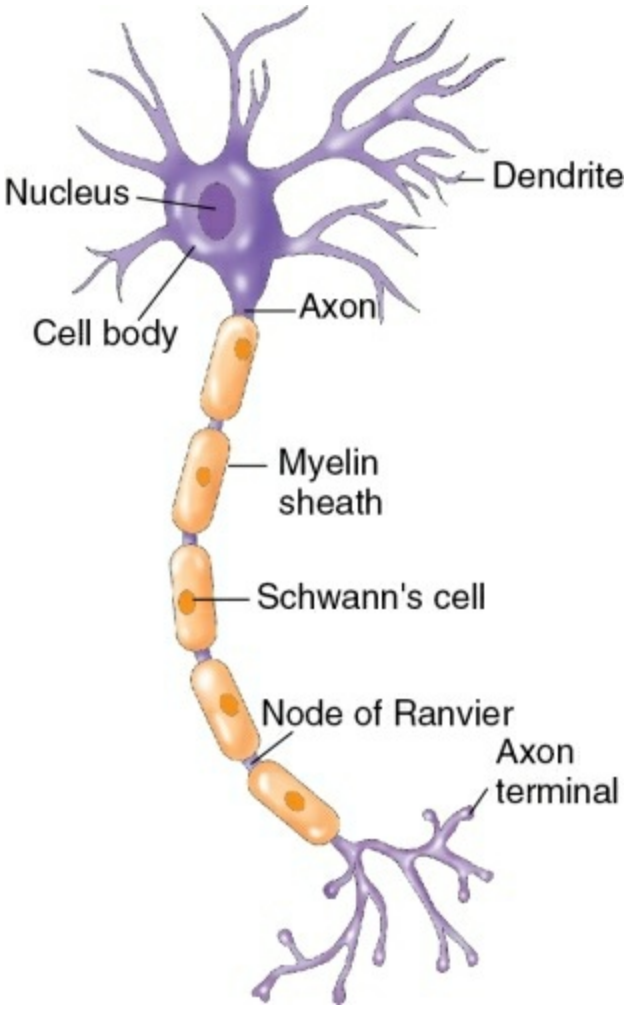

FIGURE 20.1 The neuron.

Developmental variations

Brain growth is rapid after birth, with 50% of postnatal brain growth achieved by 1 year of age. By 2 to 3 years of age, the brain is 80% of adult size.[1] Head circumference increases sixfold in the first year of life and is the best indication of normal brain growth. *Myelination,* the deposit of the protective fatty substance around the axons, continues in the brain throughout the first 2 years of life, and in the preterm infant continues into the third year of life. Myelination proceeds from head to toe, *cephalocaudal,* beginning with the spinal cord and cranial nerves and then from midline to fingertips, *proximodistal,* following with the brainstem, corticospinal tracts, and sensory pathways. Preterm or very low birth weight (VLBW) infants may develop from toe to head, which is associated

with persistent hypertonia in the lower extremities. The control of motor functions in infancy and early childhood is closely associated with the normal myelination of the nerve fibers.[1] The development of intelligence in infants depends on the normal progression through two primary developmental domains—problem solving and language.

The rapid increase in myelination in the brain in the first 2 years of life is followed by a decrease in brain growth and maturation during early childhood. The growth of the gray matter in the brain continues throughout childhood into adolescence and early adulthood in a nonlinear pattern.[2] Lifestyle habits such as sleep and adequate nutrition, as well as genetic factors, affect the volume of gray matter and brain growth.[2]

Anatomy and physiology

Central nervous system

Cerebrum

The outermost part of the brain is the *cerebral cortex,* which is often referred to as the gray matter because the neurons are unmyelinated and give a gray rather than a white appearance. The outer layer of the brain is composed of fissures and grooves, or *sulci,* and the ridge between grooves is the *gyrus.* The newborn infant has fewer convolutional surfaces, or sulci, in the cerebral cortex and more pliable skull bones, which decrease the incidence of bruising and tearing of the cerebral cortex in the developing infant with minor head trauma. The sulci of the brain deepen throughout childhood and continue to mature into young adulthood.

The *cerebrum* is the largest part of the brain and is covered by the cerebral cortex. The cerebrum is divided into two hemispheres, the left and right hemispheres. The right hemisphere controls the functions of the left side of the body, and the left hemisphere controls the functions of the right side of the body. Depending on hand preference, one hemisphere is considered dominant due to the location of important speech and language centers. For right-

handed people, the left hemisphere is dominant, and for left-handed people, the right hemisphere or both hemispheres may be dominant. The hemispheres are connected by a bridge of myelinated axons, the *corpus callosum,* which lies between the fissures of the left and right hemispheres.[1] The corpus callosum controls and integrates motor, sensory, language, and higher intellectual functions. The right and left hemispheres are divided into four lobes—*frontal, parietal, temporal, and occipital*—with arbitrary borders. Each lobe controls particular bodily functions and behaviors (Fig. 20.2).

- **Frontal lobe:** Considered the "personality center" of the brain and is responsible for higher cognitive functions. Initiates movement control of the flexor muscles of hands and feet. *Broca area* in the frontal lobe controls the ability to articulate speech. Prefrontal area controls thought processes for anticipation and prediction of behavior, and the frontal region is involved in complex learning movement patterns and writing. Damage to the frontal region causes *expressive aphasia,* motor weakness or personality changes.
- **Parietal lobe:** Controls processing and interpretation of sensory input—visual, auditory, smell, taste, and touch sensations, including pain and temperature; perceives where a stimulus or pressure is and on which part of the body, and provides *proprioception,* the sense of the position of the limbs of the body. It has an important role in language processing and symbol interpretation. Damage to the parietal region results in sensory loss, spatial disorientation, or receptive language deficits like *agnosia,* an inability to recognize or perceive the meaningfulness of an object, persons, sounds, shapes, or smells.
- **Temporal lobe:** Primary center for the perception and interpretation of sounds and auditory association and perception, and memory recall; also involved in integration of taste, smell, and balance. *Wernicke area* in the temporal lobe is related to spoken words and language comprehension. The temporal lobe also has a role with mood and emotional control.
- **Occipital lobe:** Primary visual cortex in the brain and is the center for receiving and interpreting visual data and depth perception.

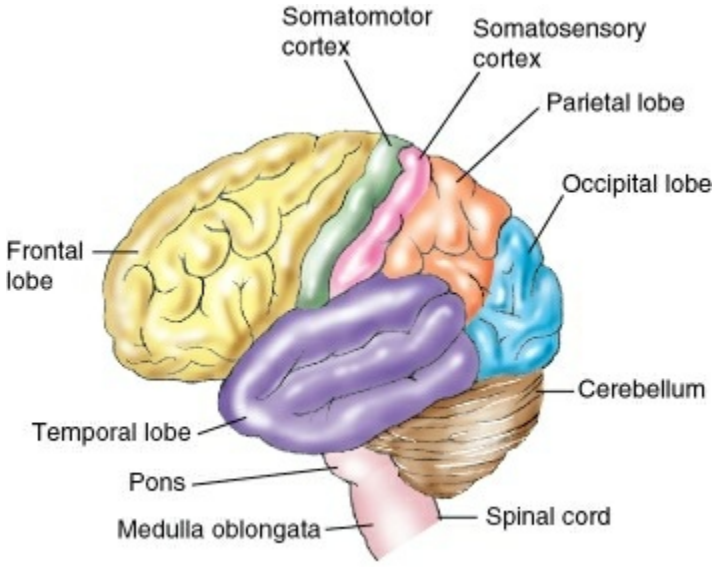

FIGURE 20.2 Regions of the cerebral cortex.

The *basal ganglia* are a cluster of neurons deep in the subcortical layer that process connections between the cerebral motor cortex and the upper brainstem. The maturation of the basal ganglia occurs in early childhood with the development of fine motor skills and, in childhood, process movement functions such as arm swinging during walking and running and throwing a ball overhand. The basal ganglia also have a role with behavioral control and are associated with disorders such as Tourette's syndrome, obsessive-compulsive disorder and addiction. Dopamine plays an important role in basal ganglia function. Dysfunction of the basal ganglia results in abnormal postural movement patterns as in *cerebral palsy* (CP), Parkinson's disease, and *Huntington chorea*.

Cerebellum

The *cerebellum* is located in the posterior cranium and maintains the body's equilibrium and coordinates both voluntary and involuntary movements of the limbs, trunk, head, larynx, and eyes.[1] The motor cortex in the cerebrum relays signals to the cerebellum, which results in the precise muscle movements requiring a high level of fine motor dexterity. It is the portion of the brain that processes the sensory input from the musculoskeletal system, as well as from the visual, auditory, and touch receptors, and it transmits signals to the motor system to direct reflexes, muscle tone, posture, and balance.

The cerebellum also has some role with cognitive functions, emotional control and speech. Damage to the cerebellum can cause *ataxia*, loss of coordination of motor movement, inability to perform rapid alternating movements, and a wide-based gait. Abnormal development or dysfunction of the cerebellum in preterm infants can result in hypotonia, nystagmus, and language deficits.

Brainstem and cranial nerves

The *brainstem* is in the central core of the brain and includes the *pons, medulla oblongata,* and *midbrain* (Fig. 20.3). The brainstem contains the sensory and motor tracts that connect the brain and spinal cord and is the location for most of the twelve cranial nerves. The *pons* acts as the neural transmission center from all parts of the central cortex and supports ascending and descending nerve fibers. It controls basic bodily functions—breathing, eating, and motor functions. Cranial nerve V *(trigeminal nerve)*, cranial nerve VI *(abducens nerve)* and cranial nerve VII *(facial nerve)* arise from the pons. Dysfunction or damage of the peripheral pons is associated with a loss of outward or lateral motion of the eye muscles causing *strabismus* and may also cause a loss of sensory or motor function of the facial and mouth area. The *medulla oblongata,* which lies between the pons and the cerebellum, is a continuation of the spinal cord. The medulla oblongata processes impulses from the hypoglossal, vagal, spinal accessory, glossophyarngeal, and the vestibular and acoustic cranial nerves. It also aids in the life functions of respiration and circulation, and controls involuntary reflexes such as coughing, sneezing, and yawning. Dysfunction or damage of the medulla causes weakness in the shoulder muscles, affects tongue muscles and salivary function, decreases gastrointestinal motility, alters swallowing and speech functions, causes nerve deafness, and diminishes cardiovascular and respiratory functions. The *midbrain* controls the integration of basic bodily functions, and contains the neural fibers of cranial nerve III *(oculomotor)* and cranial nerve IV *(trochlear)* that come from the spinal cord and merge into the thalamus and hypothalamus. The midbrain also contains the reticular activating system which regulates wakefulness and arousability.

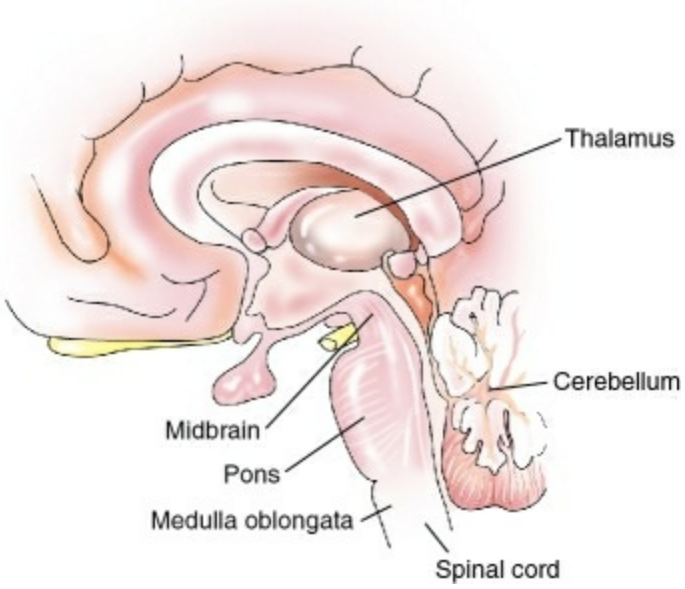

FIGURE 20.3 Brain and brainstem.

The *diencephalon* (Fig. 20.4) is the extension of the brainstem and lies embedded in the cerebral cortex. It contains the *thalamus, hypothalamus, pituitary gland,* and the *pineal gland*—an endocrine gland that produces melatonin, a hormone that regulates sleep-wake cycle. Parts of the third ventricle and the nuclei of the cranial nerve II *(optic nerve)* and retina arise from the diencephalon.

- **Thalamus:** Acts as the brain's relay station and receives input from the sensory and motor systems of the body and dispatches input from the pons and cerebellum to the cerebral cortex.
- **Hypothalamus:** Controls and regulates the body's internal environment—body temperature, metabolic processes, and involuntary response activity.
- **Pituitary gland:** Responsible for hormonal control of growth and reproductive function and assists in regulating thyroid gland function, lactation, and metabolism. The posterior pituitary produces vasopressin, which regulates the balance of water and sodium in the body.

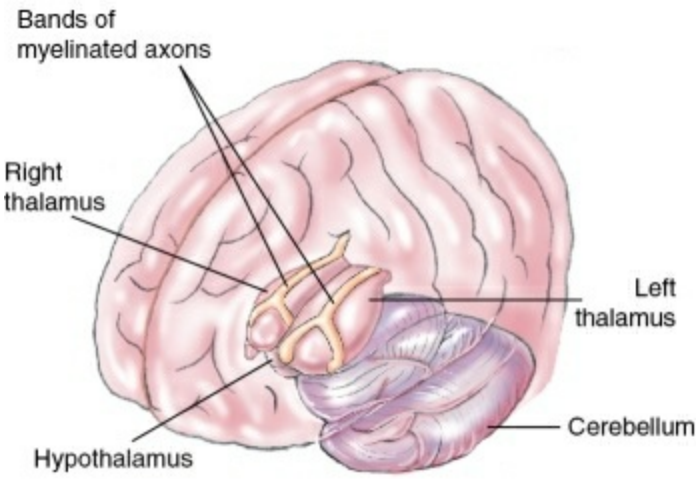

FIGURE 20.4 The diencephalon.

The *limbic system* is the group of subcortical structures in the diencephalon including the *hypothalamus* and *hippocampus.* This system regulates emotion and motivation and organizes memories. Aggression and fear also are regulated by the limbic system. Disruption that occurs during the development of the limbic system causes distorted perceptions and aggressive behavior. Maternal substance abuse early in pregnancy can disrupt the migration of neuron activity in the cerebral cortex, and later prenatal exposure disrupts neuronal synapses. Infants exposed to illicit drug use in utero often have disturbances in memory, learning, attention span, and oppositional behavior disorders during childhood.

Ventricular nervous system

The brain and spinal cord are lubricated by *cerebrospinal fluid (CSF),* which provides support and protection for the brain and allows for the removal of waste products. CSF is produced by specialized tissue called choroid plexus, which line the ventricles of the brain and spinal cord. It flows from the lateral ventricles to the third and fourth ventricles through a series of foramens. The fourth ventricle, which lies in the medulla, contains three openings that allow the cerebrospinal fluid to pass into the subarachnoid space. Approximately 500 mL of cerebrospinal fluid is produced daily in adolescence and young adults and is continually being reabsorbed in the brain.[1] Any disruption in the circulation or absorption of CSF

causes a build-up in the ventricles, resulting in hydrocephalus.

The brain is covered by protective layers that cushion and lubricate the outer surface (Fig. 20.5). The *dura mater* lies just beneath the skull bone and periosteum and consists of layers of fibrous connective tissue. Adjacent to the dura mater is the *arachnoid,* the avascular, weblike membrane that cushions the cortex. The dura mater is separated from the arachnoid by the *subdural* space. The *pia mater* is the highly vascular area of the cortex that attaches directly to the gray matter or irregular surface of the brain. The subarachnoid area and a cushion of cerebrospinal fluid separate the arachnoid from the pia mater.

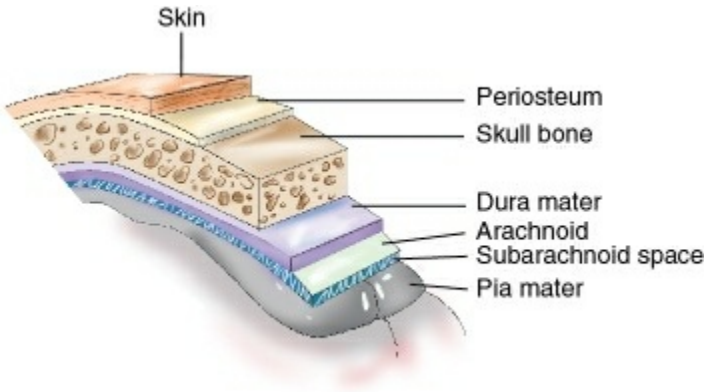

FIGURE 20.5 The meningeal layers.

Spinal nerves

There are 31 pairs of *spinal nerves* that innervate the upper and lower torso, extremities, skin, and muscles—8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal (Fig. 20.6).[1] These spinal nerves form complex nerve networks called *plexuses.* There are four major plexuses in the peripheral nervous system—the cervical, brachial, lumbar, and sacral plexuses—and the body surface that is innervated by the plexus of a spinal nerve is called a *dermatome.* Although dermatomes map specific segments of the body surface, spinal nerve sensation can be transmitted to adjacent dermatomes (see Fig. 20.6). The sensory pathways of the spinal nerves carry sensations of touch, temperature, and pain; the motor fibers activate reflexes and impulses that control skeletal muscles and the involuntary muscles of the viscera. The spinal nerves function as

part of the lower motor neurons and become dysfunctional in the presence of spinal cord lesions.

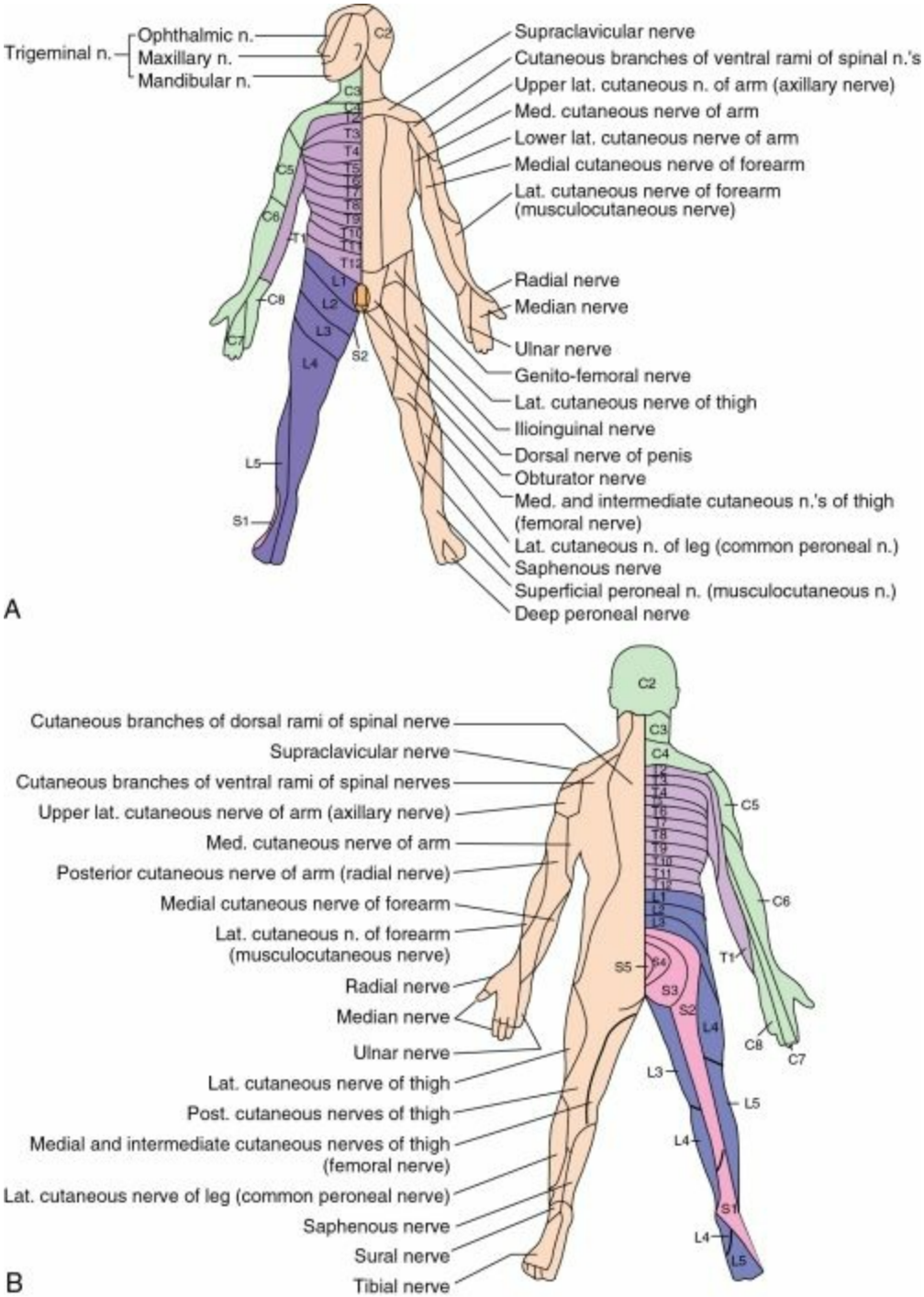

FIGURE 20.6 The spinal nerves and dermatomes **(A)** Ventral view **(B)** Dorsal view.

Spinal cord

The spinal cord is an extension of the medulla oblongata and is composed of gray and white matter extending to the lumbar region. The gray matter runs laterally along the spinal cord and protects the myelinated and unmyelinated fibers of the white matter. *Proprioceptors,* the specialized nerve endings in muscles, tendons, and joints, are located in the white matter and are sensitive to changes in the tension of muscles and tendons. Temperature, pain, touch, and equilibrium are transmitted through the proprioceptors to the brainstem.

Peripheral nervous system

The *spinal nerves* originate in the spinal cord and exit from the intervertebral spaces. They contain sensory and motor fibers and with the cranial nerves and visceral fibers of the *autonomic nervous system* compose the pathways of the *peripheral nervous system*. The autonomic nervous system carries impulses to and from the CNS. It is divided into the *sympathetic* and *parasympathetic nervous systems* and is made up of unmyelinated nerves. The sympathetic nervous system activates in times of stress and provides increased energy for needed bursts of activity. The parasympathetic nervous system balances the activities of the sympathetic nervous system by restoring stability and maintaining reserve energy for daily bodily functions such as digestion and elimination.

Upper and lower motor neurons

Upper motor neurons are located within the central nervous system (CNS) and convey impulses from the motor areas of the cerebral cortex to the *lower motor neurons* in the spinal cord. They can influence the function of the lower motor neurons, as evidenced in conditions such as *CP*. The lower motor neurons are located primarily in the peripheral nervous system and provide pathways for nerve fibers to translate movement of the muscles into action. Muscle wasting can be the result of dysfunction in the anterior horn cells of the upper motor neurons. Dysfunction in the lower motor neurons can cause wasting of localized muscle groups and a soft rather than firm tone to the muscle mass. Acquired atrophy of the muscles accompanied by a wide-based gait and muscle weakness when arising from a sitting position is characteristic of *muscular*

dystrophy, a developmental muscle wasting condition with onset in early childhood.

Spinal reflexes

Reflex behavior provides the major assessment of brainstem function. A *reflex* is an expected response between a stimulus and an elicited motor response. The *reflex arc* operates outside the level of conscious control and is the basic defense mechanism of the nervous system. Reflexes help the body maintain appropriate muscle tension and react to painful or harmful stimuli. A stimulus creates an impulse that is transmitted instantaneously outward by the motor neurons of the spinal cord via the spinal nerve and peripheral nervous system to produce a brisk muscle contraction (Fig. 20.7).

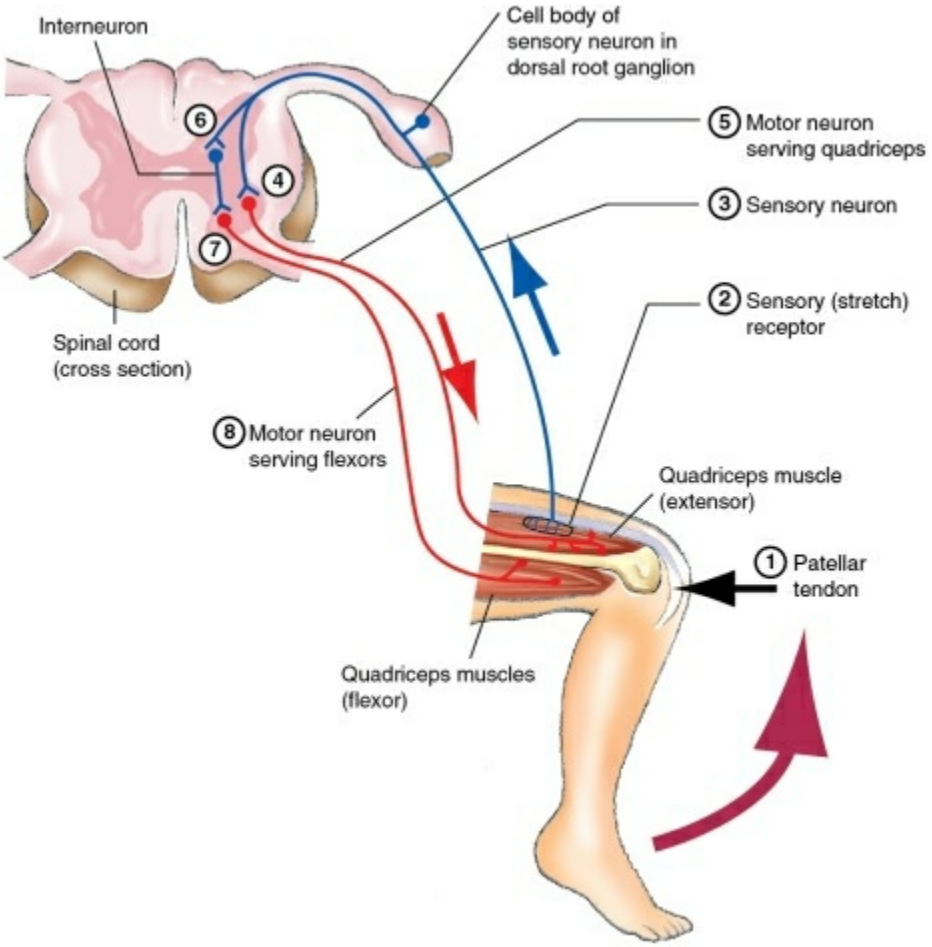

FIGURE 20.7 Reflex arc.

Physiological variations

Table 20.1 reviews the physical, developmental, and cognitive variations that occur during the development and maturation of the nervous system in infancy and childhood.

TABLE 20.1

Physiologic Variations During Development

Age Group	Physical Variations	Developmental Variations	Cognitive Variations
Preterm infant	Head lag persists for 6 months; myelination of brain continues until 3 years of age	Increased extensor tone in lower extremities; with marked stiffening and toe-pointing	Sensorimotor reflexive response to stimulus
Newborn	Decreased gyral development at birth; cerebral cortex is half the adult thickness	Exhibits primitive reflexes until 3 to 4 months; requires strong stimulus to elicit response	Innate knowledge of environment, evokes survival response such as sucking
Infancy	Head circumference increases sixfold in first year; in term infant, important myelination of brain continues until 2 years of age	*Plagiocephaly*, asymmetrical head, shape, is common; most often related to sleeping position; normally, resolves by 3 months	Develops mental image of hidden object; imitates sounds by 6 months
Early childhood (1–3 years of age)	Proprioception, awareness of spatial/body positions begins; increased movement and sphincter control	Develops basic self-control and ability to separate from attachment person; body exploration and realization of sexuality	Parallel play; independence increases; attention span develops
Early childhood (3–5 years of age)	Slowed growth, minimal change in head circumference; increased connectivity between neurons to initiate complex thought	Hand preference established; rapid fine motor development; gross motor athletic ability	Egocentric; aggressive behavior; one-dimensional understanding; magical thinking
Middle childhood	Brain reaches 90% of adult size by 5–7 years of age; deepening of sulci in brain, increasing complex thought	Transmission of nerve impulses improves, thereby enhancing fine motor and gross motor development	Consequential understanding; concrete operations and objective thought
Adolescence	Neurologic development continues into adolescence	Defines self-concept; role diffusion causes conflict	Develops abstract thinking ability

Family, cultural, racial, and ethnic considerations

Cultural practices may affect the progress of developmental milestones. Infants who are frequently swaddled on a mother's back for the first year may stand and begin walking at a later age. Increased muscle tone is often seen in African-American infants, with an equal balance between increased flexor and extensor tone. Decreased tone is more common in Asian infants and is usually unbalanced with more extensor tone throughout movement.

System-specific history

The Information Gathering table reviews the pertinent areas for the neurologic system for each age group and developmental stage of

childhood. Obtaining a complete history of gross motor and fine motor milestones in infancy, assessing speech and language development, and assessing learning including auditory and visual processing are key to early identification of insults to the nervous system.

Information Gathering for Neurologic Assessment at Key Developmental Stages

Age Group	Questions to Ask
Preterm infant	History of hypoxia in early neonatal period? Intraventricular hemorrhage? Maternal alcohol/illicit drug/substance abuse? Exposure to perinatally acquired infections (syphilis, HSV, cytomegalovirus [CMV], varicella, parvovirusB19, coxsackie virus, and others)?
Newborn	Vaginal or cesarean birth? History of birth injury? Shoulder presentation? Need for resuscitation/ventilation in immediate newborn period? Maternal infection? Perinatally acquired infection? Fetal movement during pregnancy? Age of mother and father at time of infant's birth? Large or small for gestational age? Apgar scores (if known)? Jaundice? Neonatal infections? Congenital abnormalities? Newborn screening results?
Infancy	Difficulty feeding? Protuberant tongue or tongue thrust? Any delay in achieving gross motor milestones? At what age did infant roll over? Sit without support? Crawl? Stand alone? Walk without support? Any evidence of toe-walking? Cooing, babbling? Any loss of developmental milestones?
Early childhood	Hand dominance? Feeds self? Any loss of developmental milestones? Any stumbling, limping, poor coordination? History of seizures/spasms or staring spells? Speech and language development? Attention span? Able to complete tasks? Ability to dress independently? Independent toileting achieved?
Middle childhood	History of headaches? Learning differences, difficulties or delayed in reading, math? Visual perception and auditory processing problems?
Adolescence	Headache history? Sports-related concussions?
Environmental risks	Maternal exposure to potential chemical or environmental irritants? Location of housing in relation to hazardous exposures? History of elevated lead level? Contact with chemical cleaning agents, hazardous chemicals or smoke? Pesticide exposures?

Physical assessment

The comprehensive assessment of the pediatric neurologic system includes evaluation of mental status, cranial nerves, sensory and motor function, muscle strength and tone; observation of balance, coordination and gait; and age-appropriate assessment of reflexes, speech and language development, learning and cognition. An infant or child presenting with a developmental delay or neurologic symptoms requires a more focused neurologic examination targeted to the appropriate age and developmental stage, performing the most invasive aspect of the examination, the gag reflex, last. In early childhood, it is important to remember to assess balance, gait, and agility as the child walks, pivots, and turns while holding onto the parent's hand. If the young child is fearful or refuses to walk as requested, gently pick up the child and direct him or her toward the parent and observe the gait from behind with the child undressed except for a diaper. Observing the gait will assess any abnormalities and assist the pediatric health care provider with parental anticipatory guidance about the normal neuromuscular developmental process in early childhood. Box 20.1 presents the basics of the neurologic examination in infants and young children.

BOX 20.1

Pediatric Neurologic Assessment Checklist

- Infant reflexes at <1 year of age
- Fontanels at <18 months
- Level of alertness or consciousness
- Motor and sensory function
- Cranial nerves
- Deep and superficial reflexes
- Coordination
- Balance and gait

Evaluation of motor function

Muscle tone, the normal degree of tension maintained by muscles while at rest, changes during the first 2 years of life as myelination of the neuronal pathways proceed, the *primitive reflexes* disappear, and the cerebral cortex begins to control motor functions. The assessment of muscle tone begins in the young infant by observing the resting posture. In the term infant at birth, arms and legs are in a semiflexed position with the hips slightly abducted (Fig. 20.8). At the 2-month-old visit, tone in the neck and trunk is evaluated by *gently* pulling the infant upward from the exam table, grasping the hands and forearms. Significant head lag or inability of the infant to exhibit strength in the neck and shoulders when pulled to sitting may indicate *hypotonia,* a decrease in the normal resting tension in the muscle. In the term infant 4 to 6 months of age, strength in the extremities and trunk is evaluated by *gently* pulling the infant to a sitting position; muscle tone can also be evaluated in the standing position while supporting the trunk to assess the strength of the lower extremities. At 9 months of age, gently pull the infant with both arms from a sitting position to standing to evaluate muscle tone and strength.

FIGURE 20.8 Normal flexion in term infant.

Abnormal muscle tone and abnormal positioning or posturing of the extremities in the newborn and young infant may indicate neurological dysfunction. Hips positioned in external rotation or in a "frog-leg" position in the term newborn indicates abnormal muscle tone. *Hypotonia*, low muscle tone, most commonly presents

in the newborn or infant as *floppy infant syndrome* which includes limited muscle tone and strength in the extremities, arms are often straight at the infant's side, and hips are abducted with lower extremities abducted on the exam table. When placed in ventral suspension, the infant's head and extremities will hang or drape over the examiner's hand with inability to maintain or move to a horizontal position due to hypotonia of trunk muscles. Infants with hypotonia can actively move all extremities through normal range of motion, but spontaneous movements may be less frequent than in infants with normal muscle tone.

Neurological disorders often present in early infancy with hypotonia and muscle weakness. Hypotonia may be caused by lesions in the central or peripheral nervous system, as well as neuromuscular disorders, sepsis, organ failure, and metabolic dysfunction.[3] Infants and children with a CNS etiology may have a history of neonatal intraventricular hemorrhage, hypoxia, perinatally acquired infection, maternal substance abuse or drug exposure, and or exposure to environmental toxins. It is important to obtain a family history, if known, of any neurological disorders such as congenital myotonic dystrophy, spinal muscular atrophy, or inherited metabolic disorders.[3]

Preterm infants may have *hypertonia,* or increased extensor tone in their lower extremities, during the first year of life, as well as decreased truncal tone. Infants who have hypotonia initially may later develop hypertonia or spasticity as the cerebral cortex matures. Asymmetry in muscle tone may not be identified early in infancy due to the primitive reflexes and lack of voluntary control of the musculoskeletal system from the cerebral cortex. Transient hypertonia may be noted in drug-exposed infants in the neonatal period. It generally diminishes during the first year of life and disappears by 2 years of age. If the infant exhibits *opisthotonos,* a persistent arching of the back and extension of the neck, this indicates serious neurologic compromise and needs urgent referral.

Developmental delays in preterm and term infants may occur in any area of development—gross motor, fine motor, language, social/emotional, and/or cognitive development—but delays are usually first noted in gross motor skills and abilities. Assessing the primitive and postural reflexes will assist the pediatric health care

provider to detect any evidence of delayed maturation of sensory and motor function and prompt earlier referral for evaluation and earlier intervention services. Table 20.2 presents red flags and indicators for delayed cognitive development in an infant with developmental delay.

TABLE 20.2
Cognitive Red Flags

Age	Red Flag
2 months	Lack of fixation
4 months	Lack of visual tracking
6 months	Failure to turn to sound or voice
9 months	Lack of babbling consonant sounds
24 months	Failure to use single words
36 months	Failure to speak in three-word sentences

From Wilks T, Gerber RJ, Erdie-Lalena C: Developmental milestones: cognitive development, *Pediatr Rev* 31(9):364-367, 2010.

Primitive reflexes

The *primitive reflexes* appear as early as 25 weeks' gestation. They are involuntary and controlled at birth by the brainstem. Spontaneous movement is dominated by a primitive grasp reflex at birth.[4] The primitive reflexes should always be symmetrical and are considered abnormal if asymmetrical or absent at birth (Table 20.3; Figs. 20.9 through 20.12). In the first 3 months after birth, term infants search out objects with their eyes rather than their hands and visually fixate on objects and faces by tracking with their eyes. As the primitive reflexes diminish, infants start to voluntarily grasp with their hands.[4] The primitive reflexes normally diminish by 3 to 4 months of age as the cerebral cortex matures during the first year of life. They disappear altogether in the normal term newborn by 4 to 6 months of age and are considered abnormal if persistent after 6 months of age. The primitive reflexes provide the earliest indication of CNS dysfunction. If an infant is very sleepy, irritable, or satiated after feeds, the primitive reflexes will be diminished and should be reevaluated when the infant is alert between feedings.

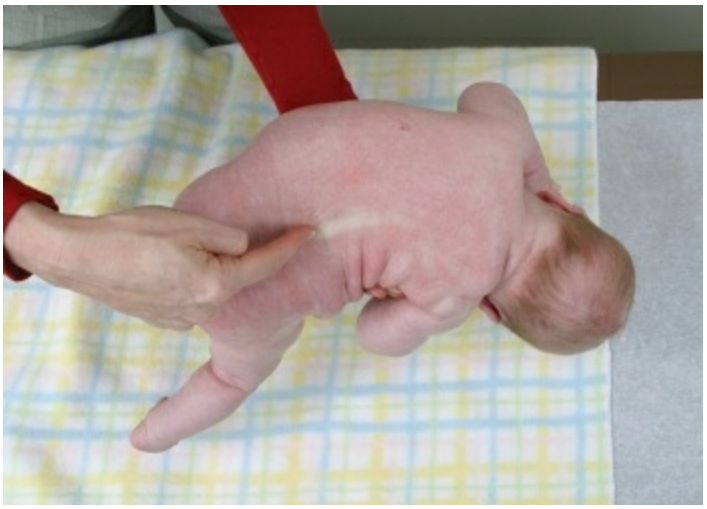

FIGURE 20.9 Truncal incurvation reflex.

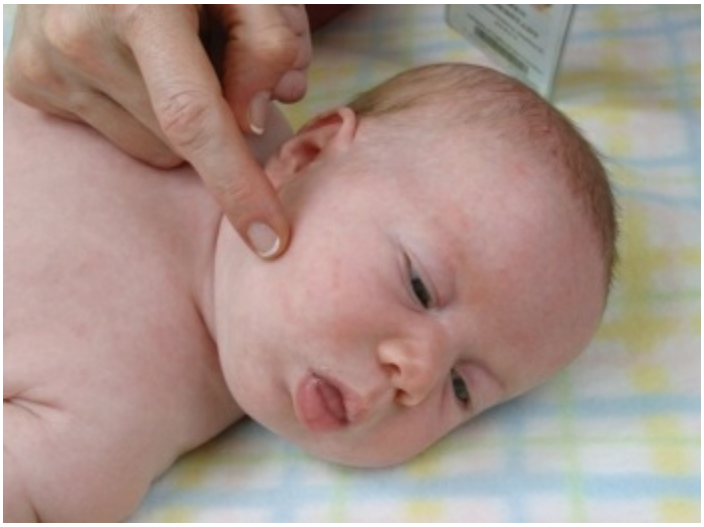

FIGURE 20.10 Rooting reflex.

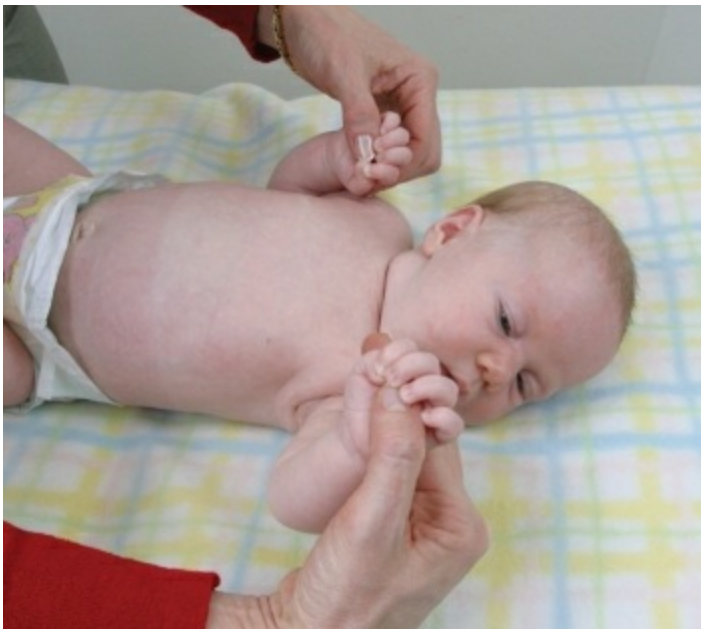

FIGURE 20.11 Palmar grasp reflex.

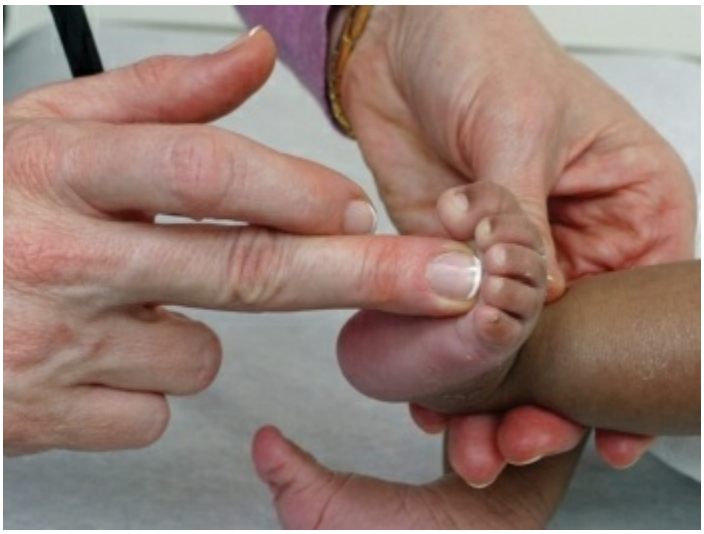

FIGURE 20.12 Plantar grasp reflex.

TABLE 20.3
Primitive Reflexes

Reflex	How Initiated	Response
Asymmetrical tonic neck	With infant on flat surface turn head 90 degrees to surface	Arm and leg extend on same side infant is turned toward, arm and leg on opposite

		side flex
Moro	Support infant at 30-degree angle above flat surface with examiner's hand; allow head and trunk to drop back to surface supported by examiner's hand; or pull infant up by hands to 30-degree angle above examining table; gently drop infant back to surface quickly and release arms	Arms extend and abduct, hands open, fingers fan out, thumb and forefinger form a C; then arms flex and adduct, knees clench, hips flex, eyes open, infant may cry
Palmar grasp	With infant's head midline, touch palm of infant's hand on ulnar surface with examiner's thumb	Fingers clasp examiner's thumb
Placing	Hold infant upright under arms over edge of table; touch dorsal surface of foot to table edge	Flexion of knees/hips, foot lifts as if stepping up on table
Plantar grasp	Touch infant on plantar surface of foot at base of toes	Toes curl downward
Rooting	Touch or stroke cheek	Infant's head turns toward stimulus and mouth should open
Stepping	Hold infant upright under the arms above exam table; palmar surface of feet should be allowed to just touch table surface	Stepping-like motion with alternate flexion and extension of legs
Sucking	Gently stroke the lips	Infant's mouth opens, sucking begins; gloved finger inserted into mouth evaluates strength of suck reflex
Truncal incurvation or Galant reflex	Hold infant firmly suspended in prone position with examiner's hand supporting chest; with opposite hand, stroke along spine lightly with fingernail just adjacent to vertebrae from shoulders to coccyx	Hips and buttocks curve/turn toward stimulus side

Postural reflexes

The appearance of the *postural reflexes* predicts normal development (Table 20.4). The postural reflexes appear between 5 and 6 months in the term infant and progress in a cephalocaudal direction beginning with head control to grasping objects. If postural reflexes do not appear by 8 to 9 months of age, it is considered an abnormal finding. When evaluating tone in early infancy, the infant's head must be kept in the midline position when supine to eliminate eliciting the *asymmetrical tonic neck reflex* (Fig. 20.13). When holding the infant firmly suspended prone in the examiner's hand, the infant should lift the head and extend the spine and lower

extremities (Fig. 20.14). While supporting the trunk, hold the infant just above the examination table to elicit the *positive support reflex* by lowering the infant to touch the feet gently on the surface of the table to elicit extension of the legs and partial weight bearing (Fig. 20.15). The normal development of the postural reflexes makes upper posture possible. Remnants of the postural reflexes persist throughout life.

FIGURE 20.13 Asymmetrical tonic neck reflex.

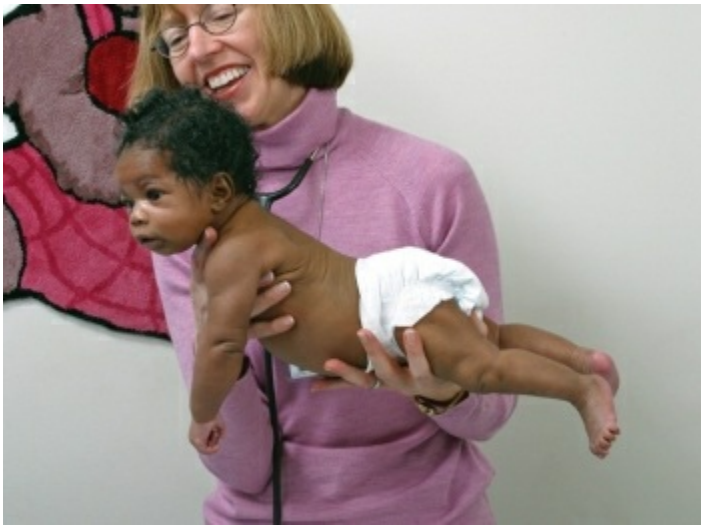

FIGURE 20.14 Landau reflex.

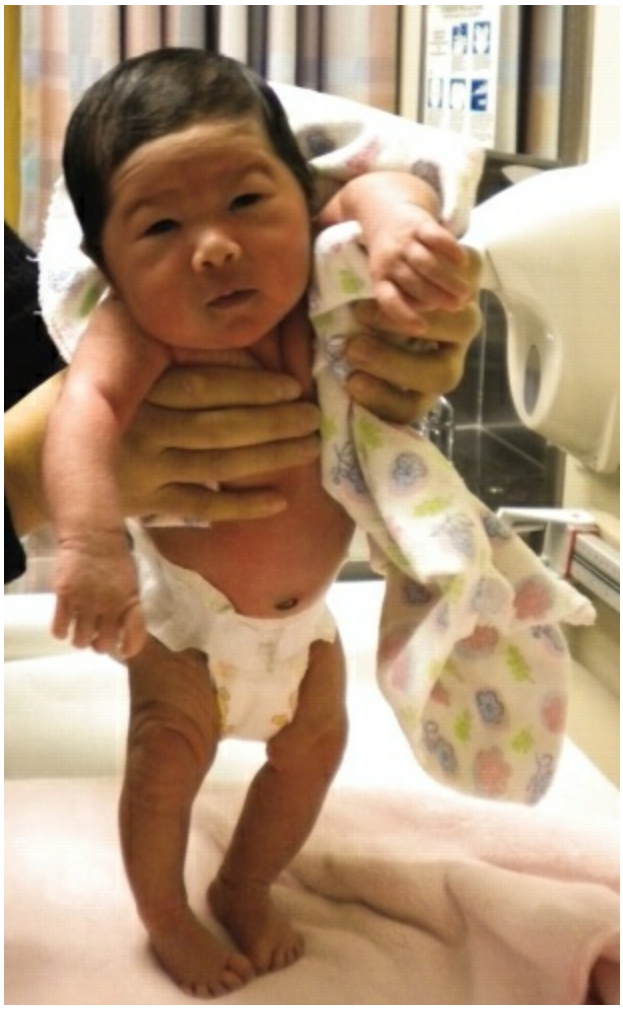

FIGURE 20.15 Positive support reflex. Source: (From Gerber RJ, Wilks T, Erdie-Lalena C: Developmental milestones: motor development, *Pediatr Rev* 31(7):267-277, 2010.)

TABLE 20.4
Postural Reflexes

Reflex	How Initiated	Response
Neck righting	Infant's head is turned to the right or left from the midline 90 degrees to the examination table	Rotation of the trunk in the direction in which the head of the supine infant is turned; this reflex is absent or decreased in infants with spasticity
Landau	Hold infant firmly suspended in prone position with examiner's hand supporting abdomen and head; legs should extend over hand	Infant should lift head, extend spine/lower extremities
Lateral parachute	Assessed at 5–7 months of age in term infant. Hold infant prone and	Observe symmetry of hand opening; infant should try to protect self by

	firmly supported; slowly lower infant toward flat surface	extending arms/legs
Forward parachute	Assessed at 7–9 months of age in term infant. Suspend infant in prone position with arms/legs extended, support with both hands over flat surface	Observe symmetry of hand opening; infant will lift head and extend spine along horizontal plane
Positive support	Hold infant upright and firmly supported under arms while over exam table; touch infant's feet to surface	Infant should extend legs and bear some weight

Involuntary motor function

Tremors, a rhythmic and oscillatory movement of a part of the body with more or less constant frequency and variable intensity,[5] can be observed intermittently in the term newborn in the first few days after birth and they are generally considered within normal limits. Persistent tremors beyond the newborn period are considered abnormal and require referral and further diagnostic evaluation by a pediatric specialist.[5]

Clonus, rhythmic tonic-clonic movements of the foot elicited by stimulus, can be a normal finding in the newborn. Clonus can be elicited by the examiner's firm touch on the sole of the foot with the finger. In infancy, clonus may indicate an upper motor neuron dysfunction and is considered abnormal as well as exaggerated or asymmetric tendon reflexes which require further diagnostic evaluation and referral if indicated.

Movement disorders in children are characterized by abnormal involuntary movement.[6] *Tics* are the most common movement disorder in childhood after 7 years of age and may be motor or vocal. If both vocal and motor tics are present, there may be a need for evaluation for *Tourette syndrome*. Note that tics can also be simple, involving one body part, or complex, involving several body parts. Children with language or learning difficulties, severe anxiety, and psychiatric disorders such as obsessive compulsive disorders may have associated tic disorders. Simple tics include blinking, facial grimacing, shoulder shrugging, and hand or head jerking. Transient tic disorders, lasting less than 3 months, occur in up to 25% of normal children, and they cease during sleep.[6] *Chorea* is the slow, involuntary, random, twisting movement that can be

seen in children with *cerebral palsy* and *Huntington disease*, and *Huntington disease* is the most frequent cause of hereditary chorea.

Infantile spasms involve the muscles of the neck, trunk, and extremities.[7] Infantile spasms is an epileptic disorder of infancy and early childhood that presents as repetitive flexor/extensor movements with head nod, mixed movements, or as myoclonic-tonic spasms or seizures. The peak incidence of onset is between 3 and 7 months of age, and 90% of affected children present with symptoms by 1 year of age.[7] The spasms may be subtle, brief, and sudden and may be associated with an underlying disorder. Infantile spasms can cause irreversible brain damage, therefore, prompt referral to a specialist is indicated if infantile spasms are suspected. The diagnostic work up includes electroencephalography (EEG), MRI of the brain, and evaluation by a pediatric neurologist.

Evaluation of sensory function

Touch, deep pressure, pain, temperature, and vibration are all characteristics assessed in sensory function. Tactile sensation can be tested in the verbal child by gently touching different areas of the body with a cotton swab when the eyes are closed. The child should be able to identify the spot by pointing to the area of the body. Pain sensation can be tested similarly in the verbal child by touching the body with the sharp and dull ends of a reflex hammer. Temperature and vibration are sensations not usually elicited in early childhood. Discrimination sensation can be assessed in the children older than 5 to 6 years of age using the following tests:

- **Stereognosis:** The ability to recognize an object by its feel. With eyes closed, ask the child to identify small familiar objects placed in the palm, such as a key or a coin. If you are not testing expressive language, have the child point to the correct object when eyes are open. Children with *CP* are generally unable to identify the object.
- **Graphesthesia:** The ability to identify shapes traced on the palm. Younger children are usually tested with shapes and older children with numbers. May be repeated in each palm to ensure accuracy in testing. Children with spatial and proprioceptive dysfunction will not be able to discriminate shapes or numbers.

- **Two-point discrimination:** A test of spatial discrimination of the body. With the child's eyes closed, touch lightly on the skin with two points in close proximity on the body, and then follow with touching the child with one point. Ask whether the child felt one or two points.

Loss of sensation can reflect impairment in the peripheral nervous system, spinal column, brainstem, or cerebral cortex. Children under 5 years of age often have difficulty with sensory testing and comparing touch points.

Cranial nerves

Assessment of the cranial nerves is performed as part of a comprehensive physical examination. Cooperative young children and school-age children usually delight in the activity of testing the cranial nerves. Tables 20.5 and 20.6 summarize the assessment of cranial nerves in infancy and early and middle childhood. Cranial nerve testing may be difficult to assess in infants if they are drowsy, crying, or satiated after feeding and in young children if they are fearful or irritable during the physical examination.

TABLE 20.5
Cranial Nerve Testing in the Newborn and Infant

Cranial Nerve	Test	Response
Cranial nerve I, olfactory	Pass strong-smelling substance (e.g., cloves, peppermint, anise oil) under nose (not often tested in newborns)	Observe for startle response, grimace, sniffing
Cranial nerve II, optic	Light source/ophthalmoscope on medium/large aperture	Pupils constrict in response to light, able to fix on object and follow for 60–90 degrees, blink to light
Cranial nerve III, oculomotor Cranial nerve IV, trochlear Cranial nerve VI, abducens	Elicit pupillary response to test optic nerve by shining pen light toward pupil, track and follow light "Doll's eye" or oculovestibular maneuver—rotate head and body from side to side, observe	Evaluate shape, size, symmetry of extraocular movements of pupil Eyes should deviate left when turning head right; if eyes remain fixed or do not track in opposite direction, suspect brainstem dysfunction

	eyes moving away from direction of rotation	
Cranial nerve V, trigeminal	Touch infant's cheek area Test jaw muscles by placing gloved finger in infant's mouth	Infant turns cheek toward touch stimulus Infant should bite down on gloved finger and begin sucking
Cranial nerve VII, facial	Observe infant's face for symmetry of facial movements and observe when crying	Asymmetrical nasolabial folds/asymmetrical facial expression may indicate nerve palsy
Cranial nerve VIII, acoustic	With infant lying supine, ring bell sharply within a few inches of infant's ears	Observe for response to sound stimulus, such as mild startle/blink reflex note: Auditory-evoked response required in many states evaluates acoustic nerve function and has replaced rough assessment of acoustic nerve and acoustic blink response to loud clap
Cranial nerve IX, glossopharyngeal	Use tongue blade to apply pressure on midtongue area to overcome tongue thrust	Elicit gag reflex; observe tongue movement, strength
Cranial nerve X, vagus	Observe infant while crying	Evaluate pitch of cry and assess for hoarseness, stridor; normal cry is loud and angry; shrill, penetrating cry indicates intracranial hemorrhage; whiny, high-pitched cry indicates central nervous system dysfunction
Cranial nerve XI, accessory	With infant lying supine, turn infant's head to one side	Infant should work to bring head to midline
Cranial nerve XII, hypoglossal	Observe infant when feeding	Sucking, swallowing should be efficient, coordinated

Data from Thureen PJ, Deacon J, Hernandez J, et al: *Assessment and care of the well newborn,* ed 2, Philadelphia, 2005, Saunders.

TABLE 20.6
Cranial Nerve Testing in Early to Middle Childhood

Cranial Nerve	Test
Cranial nerve II, optic	• Allen vision cards, tumbling E, or Snellen chart for visual acuity testing
Cranial nerve III, oculomotor Cranial nerve IV, trochlear Cranial nerve VI, abducens	• Use ophthalmoscope or light source to test direct and consensual pupillary response to light • With examiner's hand under chin, have child follow toy, light source, or index finger through six cardinal fields of gaze to test eye movement

Cranial nerve V, trigeminal	• Observe child chewing and swallowing to test normal jaw strength • Touch facial area with cotton swab and observe child move away from stimulus
Cranial nerve VII, facial	• Ask child to smile, frown, and puff cheeks, observe for symmetrical facial expressions
Cranial nerve VIII, acoustic	• Perform audiometric testing to evaluate range of hearing • Test vestibular balance with eyes closed
Cranial nerve IX, glossopharyngeal, Cranial nerve X, vagus	• Observe tongue strength and movement and elicit gag reflex with tongue blade • Child is able to swallow without difficulty • Voice quality and sound is normal and intact
Cranial nerve XI, accessory Cranial nerve XII, hypoglossal	• Have child stick tongue out and push tongue against tongue blade • Shrug shoulders to assess trapezius muscle strength • Turn head from side to side against resistance to test sternocleidomastoid muscle strength

Deep tendon reflexes

The reflex arc of the deep tendon reflexes is a complex function of the musculoskeletal and nervous systems and requires an intact sensory neuron, a functional synapse in the spinal cord, an intact motor neuron, a functional neuromuscular junction, and a competent muscle (Table 20.7; Figs. 20.16 through 20.22). Reflex testing is useful in young children because it provides information on the normal development and maturation of the neuromuscular system. Assessment of the deep tendon reflexes may help to distinguish between upper and lower motor neuron lesions. Abnormally brisk reflexes with clonus may suggest upper motor tract involvement. Absent reflexes are consistent with a neuropathic lesion, an inflammatory demyelinating disorder such as *Guillian Barre* syndrome, or severe myopathy.[8] Box 20.2 shows assessment of the response to performing deep tendon reflexes bilaterally. Further discussion of deep tendon reflexes is presented in Chapter 19.

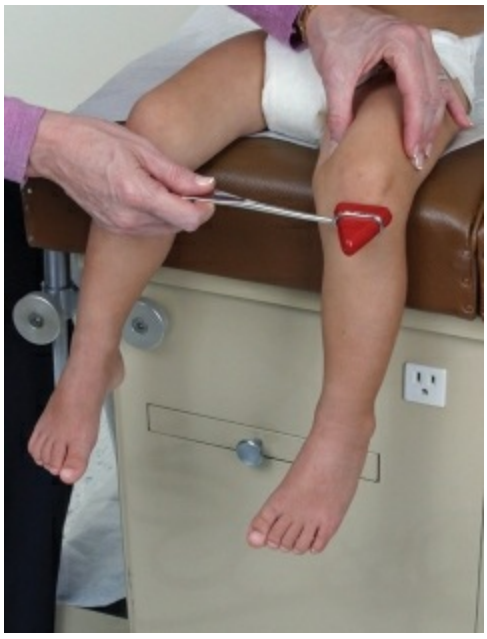

FIGURE 20.16 Patellar reflex.

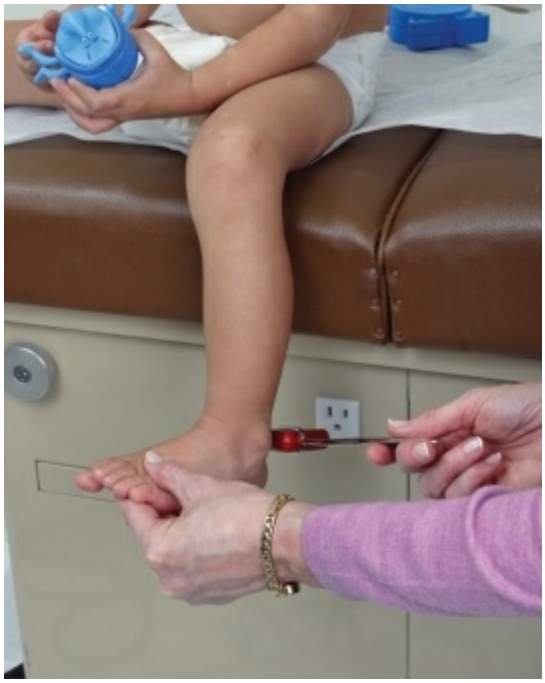

FIGURE 20.17 Achilles tendon reflex.

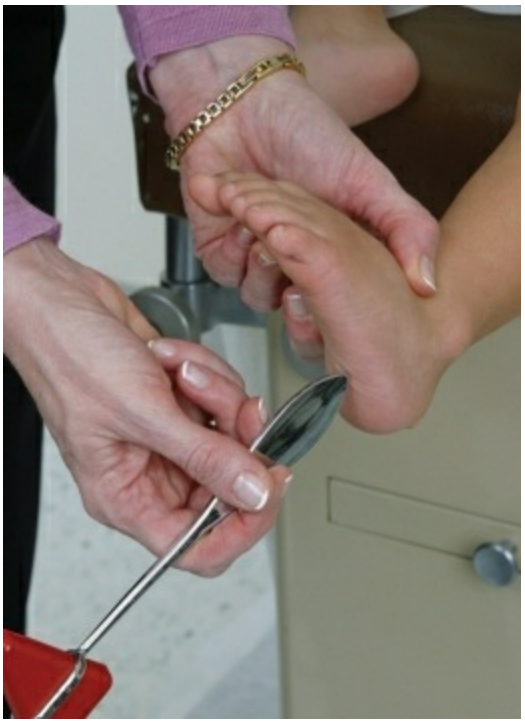

FIGURE 20.18 Plantar reflex.

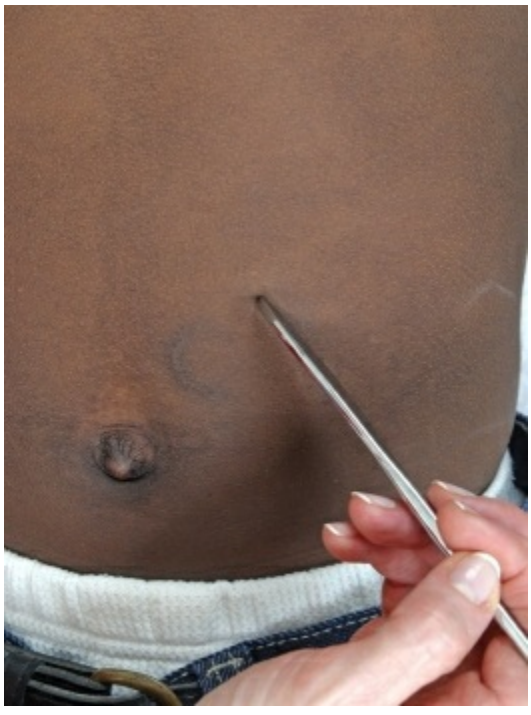

FIGURE 20.19 Abdominal reflex.

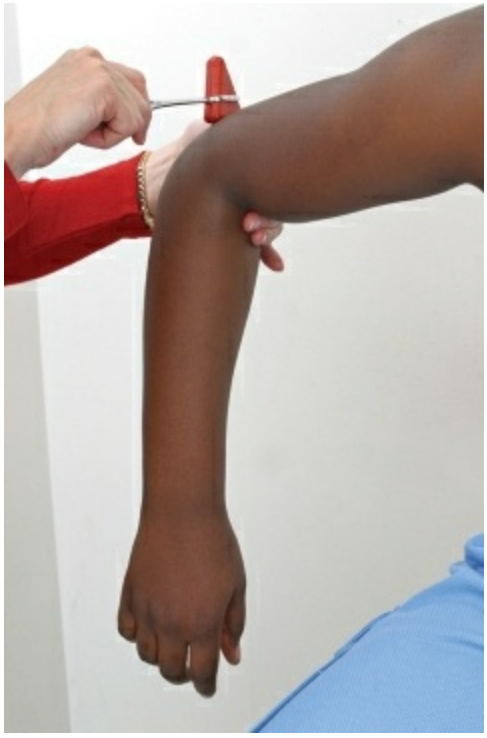

FIGURE 20.20 Triceps reflex.

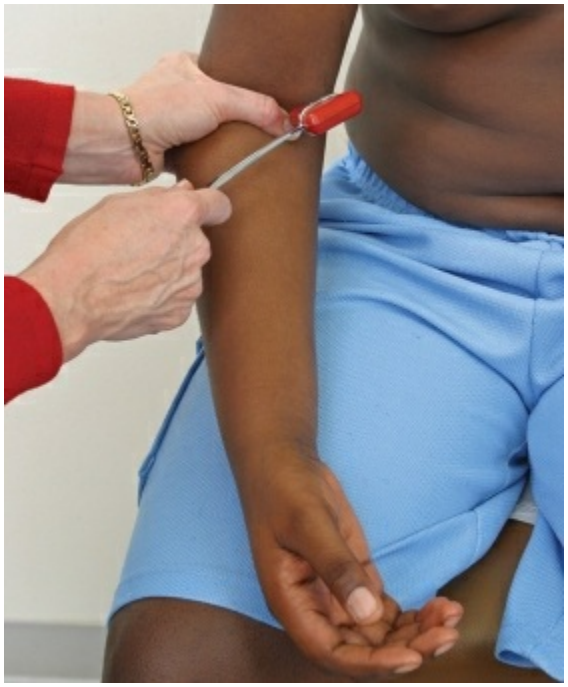

FIGURE 20.21 Biceps reflex.

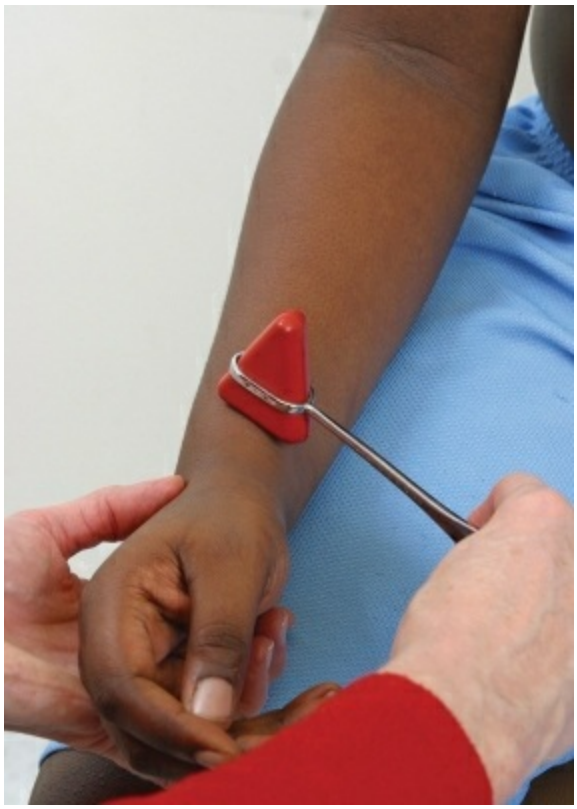

FIGURE 20.22 Brachioradialis reflex.

TABLE 20.7
Deep Tendon And Superficial Reflexes

Reflex	Test	Response
Deep Tendon Reflexes		
Biceps reflex	With examiner's thumb pressed against biceps tendon in antecubital space, support arm with palm prone; tap thumb briskly; tendon should respond by tightening	Flexion of forearm
Triceps reflex	Hold arm in flexed position slightly forward toward chest with forearm dangling downward, tap directly behind elbow on triceps tendon	Contraction of triceps and elbow should extend slightly
Brachioradialis reflex	Support child's forearm with palm resting down; tap briskly on radius approximately 2 inches above wrist	Flexion of elbow and pronation of forearm
Patellar reflex	Palpate patellar tendon just below patella and tap briskly with leg dangling; having child lock fingers and pull hard in outward direction can help elicit reflex	Contraction of quadriceps and extension of knee
Achilles tendon reflex	Support foot with ankle slightly flexed and leg relaxed, tap above heel; vary degree of flexion of foot to assist in eliciting reflex	Observe plantar flexion
Clonus	In infant, knee should be slightly flexed with infant in supine position; apply pressure to sole of foot to bring ankle into dorsiflexion	In newborn, rapid tonic-clonic movement of 4–5 beats is normal response; beyond newborn period, no rhythmic movements are expected
Superficial Reflexes		
Plantar reflex	Stroke sole of foot from heel to ball of foot curving medially with flat object	Movement of toes
Abdominal reflex	Stroke briskly above and below umbilicus	Abdominal muscles contract and umbilicus deviates toward the stimulus
Cremasteric reflex	In male, lightly scratch upper inner thigh	Testicle will elevate slightly on stimulated side
Anal reflex	Gently stroke anal area to test sphincter tone	Quick contraction of sphincter

BOX 20.2

Assessment of Deep Tendon Reflex Response

- 4+: Brisk, markedly hyperactive with clonus
- 3+: Active, brisker than normal
- 2+: Normal response
- 1+: Diminished response, low normal
- 0: Absent or no response

Evaluation of cerebellar function

Assessment of cerebellar function evaluates balance, coordination, cognitive processing, and input from visual, auditory, and touch receptors. The following simple tests can be performed in children during the physical examination and is fun and effective in assessing cerebellar function:

- **Romberg test:** Assesses balance and equilibrium. This test can be performed in the cooperative 3- or 4-year-old child. Ask the child to stand erect with eyes closed and hands touching the sides. Observe the child's balance for several seconds while monitoring the child closely. Lesions in the cerebellum can cause the child to stagger or fall.
- **Finger-to-thumb test:** Assesses cerebellar function, coordination, and cognitive processing disorders in middle childhood. Ask the child to touch each finger to the thumb in rapid succession. Test can be performed with one hand at a time and then repeated with both hands.
- **Hopping in place:** Ask the child to alternately hop on one foot and then the other. Tests for balance, cerebellar function, intact motor function, and spatial sense. The child should be able to maintain balance on one leg by 4 years of age.
- **Heel-to-toe walking or tandem walking:** Assesses balance and coordination. Ask the child in the kindergarten physical exam to

walk heel to toe in a straight line. This requires a high level of neuromuscular coordination and is often not accomplished until 6 years of age.

- **Rapid alternating movements test:** Assesses for cerebellar function, coordination, and cognitive processing disorders in the middle childhood. Ask the child to place his or her hands palm down on the thighs. Then demonstrate the rapid, rhythmic movement of the hands back and forth, asking the child to repeat the movement rapidly for 10 seconds. Next ask the child to perform the rapidly alternating movement with one hand only. Inability to perform the task may indicate cognitive or behavioral dysfunction and requires further evaluation. The child should be able to perform by 8 to 9 years of age. Findings of *motor overflow,* noted when one hand performs a motor task and the other hand mirrors the movement, may indicate a lag in the normal progression of fine motor development in children.
- **Finger-to-nose test:** Ask the child to close his or her eyes and then touch his or her nose with the first finger of one hand and then with first finger of the other hand. Then, with eyes open, have the child touch their nose with first finger and then touch the examiner's finger held approximately 18 inches in front of the child. Repeat with both hands. Then increase speed of movements with examiner's finger changing position. Consistent failure of child to point at the finger indicates dysfunction in spatial perception and coordination. The child should be able to perform this by 8 to 9 years of age. Inability to perform task may indicate ataxia, lack of coordination caused by cerebellar dysfunction, requiring referral and further diagnostic evaluation.
- **Heel to shin test:** Assesses coordination and balance in middle childhood. Ask the child to stand and place the right heel on the left shin below the knee and then slide the heel down the shin to the foot. Repeat with the left leg. The test may be performed with the child lying down or sitting. Inability to perform the task or maintain balance may indicate decreased motor strength, a cerebellar dysfunction, or an alteration in proprioception.

Evaluation of cerebral function

Assessing general cerebral function in children requires evaluating the level of consciousness, mood and affect, thought, memory, judgment, and communication. In the developing child, cerebral function is more challenging to assess than in adults. In the child with head trauma, the basic components of the initial neurologic examination include (1) pupil reactivity, (2) assessing level of consciousness, (3) sensory function, and (4) motor function.

The *Pediatric Glasgow Coma Scale* (GCS) is an adapted assessment tool that assesses the level of consciousness for children with blunt head trauma.[9] For both preverbal (<2 years of age) and verbal (>2 years of age) children, the Pediatric GCS correlates with the presence of traumatic brain injury (TBI).[10] For infants and young children, the Pediatric GCS assess visual acuity with the ability to open the eye spontaneously (response to speech, to pain, or no response); verbal cues (coos and babbles appropriately, irritable cries inconsolable, cries or screams persistently to pain, grunts or moans to pain, or no response); and motor function (normal spontaneous movements, withdraws to touch, withdraws to pain, abnormal flexion, abnormal extension, or no response) which reflect the functional integrity of the major CNS pathways (Table 20.8).

TABLE 20.8

Pediatric Glasgow Coma Scale

Sign	Glasgow Coma Scale (GCS)	Pediatric Glasgow Coma Scale (GCS) [a]	Score
Eye Opening	Spontaneous To command To pain No response	Spontaneously To sound To verbal command To pain No response	4 3 2 1
Motor Response	Spontaneous responses Follows commands Localizes pain Withdrawal to pain Abnormal flexion to pain Abnormal extension to pain No response	Normal Spontaneous movements Obeys verbal commands Withdraws to touch Localizes pain Withdrawal to pain Involuntary flexion Abnormal flexion to pain Involuntary extension Abnormal extension to pain No response	6 5 4 3 2 1
Verbal Response	Oriented Confused, disoriented Inappropriate words Incomprehensible sounds No response	Vocalizes for age (coos, babbles) Interacts, oriented to sound Irritable, Cries Incoherent words Cries or screams to pain Grunts, moans to pain No response	5 4 3 2 1
Severity of Injury	Mild head injury Moderate head injury Severe head injury		13–15 9–12 <8

[a]Calculate Pediatric Glasgow Coma Scale score by assessing eye opening, best motor and verbal response, and assign a score for each area. Total score indicates severity of the head injury.

Data from Holmes JF, Palchak MJ, MacFarlane T, Kuppermann N: Performance of the pediatric Glasgow coma scale in children with blunt head trauma, *Academic Emergency Medicine* 12:814-819, 2005 and Brazelton T, Gosain A. Classification of trauma in children. 2017; https://www.uptodate.com/contents/classification-of-trauma-in-children. Accessed May 23, 2017.

Diagnostic procedures

Pediatric health care providers in most pediatric health care settings have adopted an Image Gently policy and an *As Low As Reasonably Achievable* (ALARA) policy to limit frequent cumulative exposure to radiologic diagnostic procedures.[11] Magnetic resonance imaging (MRI) is more often used to evaluate neurologic injury or a cerebral abnormality related to developmental delay. Computed tomography (CT) is the most common diagnostic tool for evaluation of *traumatic brain injury* (TBI) or cranial abnormalities such as a skull fracture or acute hemorrhage. Indications for CT in a child who presents with blunt head trauma are presented in Box 20.3. Clinical assessment is key in influencing provider choice to image, as well as access to timely imaging and need for conscious sedation for the young child.

BOX 20.3

Red Flag Indications for Head Computed Tomography for Child with Blunt Head Trauma

High risk with one of the following:

- Suspicion of child abuse
- Focal neurologic findings
- Acute skull fracture, including depressed or basilar fracture
- Altered mental status (lethargy or irritability)
- Bulging fontanel
- Persistent vomiting
- Seizure following injury
- Definite loss of consciousness if longer than a few seconds and especially if associated with other neurologic findings

Adapted from Schutzman S. Minor head trauma in infants and chlidren: Evaluation. *UpToDate* 2017; https://www.uptodate.com/contents/minor-head-trauma-in-infants-and-children-evaluation. Accessed May, 2017.

For infants or children presenting with history of spasms or seizures, EEG is used as the diagnostic tool. Ultrasound may be used in evaluating neonates and infants with an open anterior fontanel for hydrocephalus or intraventricular hemorrhages.

Neurologic conditions

Table 20.9 presents the most common neurologic conditions occurring in infants, children, and adolescents. TBI and concussion are also addressed further in Chapter 19.

TABLE 20.9

Neurologic Conditions in Infants, Children, and Adolescents

Condition	Description
Abusive head trauma	Characterized by severe trauma or shaking in small infants causing acceleration and deceleration of the delicate brain tissue within the periosteum, resulting in retinal hemorrhages and subarachnoid and subdural hematomas. *Shaken baby syndrome* can also result in epidural hematoma. Severe trauma can be fatal.
Prader-Willi syndrome	A rare chromosomal disorder characterized by hypotonia, insatiable appetite, obesity and uncontrolled appetite, hypogonadism, incomplete sexual development, and developmental delay.
Neurofibromatosis Type 1 and 2	Characterized by six or more café au lait macules over 5 mm in diameter, axillary or inguinal freckling noted in infancy or early childhood, subcutaneous neurofibromas or optic nerve gliomas. In NF-2, children present with acoustic nerve tumors.
Migraine headache in children	Characterized by pulsating pain and high pain intensity. May be associated with chronic daily headaches in up to 53% of children presenting with migraine headaches.[8]
Tension-type headaches in children	Characterized by pressing, mild pain and described as less intense than migraine headaches.

Cerebral palsy

CP is a permanent, nonprogressive central motor dysfunction disorder that affects muscle tone, posture, and movement.[12] The overall prevalence of CP is 2 per 1000 live births, and the risk for CP increases in preterm births, with a prevalence of 6.8 to 1000 live births in infants 32 to 36 weeks gestation.[13] The etiology is multifactorial often with intraventricular hemorrhage or a brain insult or injury to the CNS as a primary contributing factor in preterm infants. There are numerous prenatal, perinatal, or postnatal risk factors for the development of CP, but the most common risk factors are perinatal hypoxia and neonatal asphyxia. Intrauterine growth retardation, intrauterine infection, and multiple pregnancies also increase the risk of CP in preterm and term infants.[14] CP is often referred to as *static encephalopathy* with a delayed developmental presentation. Although CP is a motor disorder, it can also be associated with additional developmental disabilities and cognitive impairment depending on the degree of brain injury. CP may be classified as a *pyramidal* (spastic) or an *extrapyramidal* (nonspastic) disorder, indicating the area of the brain that has been affected and the predominant disability. Children with CP may present with a wide range of abilities and may have asymmetric motor dysfunction and normal cognition or severe

motor dysfunction and delayed cognitive abilities.

Interprofessional collaboration

Interprofessional collaboration is paramount for children and adolescents with neurological disorders. It is important for the pediatric health care providers to provide team-based collaborative care with pediatric neurological and developmental specialists, occupational and physical therapists, behavioral health, and learning specialists to assess cognitive and intellectual function, and gross and fine motor skills in children with CP. This will ensure prompt early interventions that foster the child's full developmental and intellectual potential.

Down syndrome

Down syndrome, or *trisomy 21,* is the most common chromosomal abnormality in children. The prevalence of Down syndrome is 1.1 out of 1000 live births. Common characteristics of Down syndrome include facies with upward slanting palpebral fissures and inner epicanthal folds, flattened nasal area, brachydactyly or abnormal shortening of fingers and toes with wide-spaced first and second toes, simian crease, hypotonia, macroglossia, and mental retardation. Infants and children with Down syndrome are at increased risk for congenital cardiac defects, duodenal atresia, leukemia, thyroid dysfunction, visual and hearing defects, obesity, atlanto-occipital joint instability that impacts extension and flexion movements, and delayed sexual development. Strabismus and refractive errors are also common. Approximately 5% to 10% of children with Down syndrome have autism spectrum disorder. Careful assessment of cognitive and intellectual function by the pediatric health care provider as well as developmental milestones is important to assist the child in developing to his or her full potential.

Summary of examination

- Assessment of muscle tone and strength is a key component of evaluating the development of the neurologic system. Evaluate

tone and strength in the young infant by observing the resting posture and assessing movement of extremities.
- Assess balance, gait, and agility as the older infant walks, pivots, and turns while holding on to the parent's hand.
- Development delays in preterm and term infants can occur in any area of gross motor, fine motor, language, social/emotional, or cognitive skills, but delays are usually first noted in gross motor skills and abilities.
- Evaluating primitive and postural reflexes assesses sensory and motor function in the young infant.
- Cranial nerve testing and deep tendon reflex testing is performed in young children to assess normal development and maturation of the neuromuscular system.
- The Pediatric GCS assesses cerebellar and CNS function by evaluating level of consciousness, pupil reactivity, motor function, and verbal response

Documentation

18-month-old

Neurologic: Alert, active, follows simple directions; gait, balance, and coordination normal for age, DTRs intact +2 bilaterally, muscle strength equal and symmetrical, cranial nerves II-XII grossly intact, ASQ normal for age.

DTR, Deep tendon reflex.

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Average weight, height, and head circumference gains in infancy through adolescence

Average Weight, Height, And Head Circumference Gains in Infancy Through Adolescence

Age	Weight	Height	Head Circumference	Comments
	Average Weekly Gain	Average Monthly Gain	Average Monthly Gain	
0-3 months	210 g (8 oz)	3.5 cm	2.0 cm	Regain or exceed birth weight by 2 weeks
3-6 months	140 g (5 oz)	2.0 cm	1.0 cm	Birth weight doubles by 4-6 months
6-12 months	85-105 g (3-4 oz)	1.2-1.5 cm	0.5 cm	
	Average Yearly Gain	Average Yearly Gain	Average Yearly Gain	
1-3 years	2-3 kg (4.4-6.6 lb)	12 cm	3.0 cm	Height at 2 years approximately half of adult height
3-6 years	2 kg (4.5 lb)	3-7 cm	1.0 cm	
6-12 years	3-3.5 kg (7 lb)	6-7 cm	2-3 cm during entire period	Growth is discontinuous, in spurts lasting about 8 weeks, occurring 3-6 times a year
	Average Total Gain	Average Yearly Gain		
Girl, 9.7-13.5 years	17.7 kg (39 lb)	8-14 cm/yr		95% linear growth achieved by onset of menarche
Boy, 11.7-15.3 years	22.2 kg (50 lb)	8-14 cm/yr		95% linear growth achieved by 15 years Weight gain follows linear growth, with several months delay; adolescents first grow taller, then fill out

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