CORE HANDBOOKS IN PEDIATRICS

PEDIATRIC PRIMARY CARE
Ill-Child Care

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Editor

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To my mother, Ruth Abigail Baker,
who taught me that learning is a lifelong process

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Preface

The precursor to this book, the *Handbook of Pediatric Primary Care,* was well received. The *Handbook* found a place in the pockets of many medical and nursing students and residents learning how to provide primary care to children as well as young doctors and nurses leaving their formal training to begin careers in pediatric primary care. Because of its utility as a resource, the editor of the handbook series at Lippincott Williams & Wilkins, Timothy Hiscock, asked me to expand the book to two separate volumes covering the well-child visit and the ill-child visit, to accommodate more easily additional topics and new information in the field. These books, with others, will be the basis for a new series, the Core Handbooks in Pediatrics.

Pediatric Primary Care: Well-Child Care will concentrate on health maintenance supervision, the foundation of pediatric primary care, and on behavioral and developmental pediatrics, integral pieces in the provision of well-child care. Several new chapters cover newborn screening, the sports physical, the adolescent visit, and injury prevention. Additional chapters address and expand on age-specific well-child visits. Furthermore, the behavioral and developmental section, significantly expanded, covers common negative behaviors that a primary care provider should be able to help parents manage as well as a chapter on child sexuality. Discussions of developmental topics of importance to the primary care provider include the approach to the developmentally delayed child, school issues, and attention deficit hyperactivity disorder. Finally, other chapters address telephone medicine, medical informatics, cultural competence, and teaching in the primary care setting.

The companion volume, *Pediatric Primary Care: Ill-Child Care,* addresses the common illnesses encountered in primary care sick or problem visits. New topics include common exanthems of childhood, acne, cardiac murmur evaluation, gastrointestinal parasites, gastroesophageal reflux disease, acute abdominal pain, diabetes, and vision and hearing abnormalities.

Medications in this handbook that are written with generic names begin with a lower case first letter. For many drugs, I also give examples of brand names, written with an upper case first letter. The brand names listed do not represent personal preferences and, for most drugs, do not represent all brand names available. Because of the variety of antimicrobials available for pediatric use and their frequency of use in the field of primary care pediatrics, I list only antimicrobials in the medication dosage guidelines in the appendix. For most other non-antimicrobial drugs suggested throughout the handbook, generic names, brand names and suggested dosages appear within the text.

Like the original book, I hope this handbook finds a special place in pediatric and family medicine continuity clinics, now such an important part of the training of primary care physicians

in this new age of ambulatory medicine. As with any handbook, my goal of allowing easy access to practical information that can be used *during* the delivery of clinical care required me to undertake the difficult task of compressing large amounts of information into a useable, readable format. I did not intend that this book definitively describe diseases and disease processes. Therefore, the end of each chapter lists up-to-date references and reviews, including internet sites, for the reader who wishes more information about any of the topics discussed.

Raymond C. Baker, M.D.

I

Introduction

1 The Effects of Illness and Hospitalization on Infants and Children

Janet R. Schultz and Raymond C. Baker

Acute illness and hospitalization are stressful experiences for children and caregivers, and their reactions to these stresses vary considerably (Table 1.1). Because health care providers spend a large part of their lives in the midst of the controlled chaos of the health care environment, they may forget that children and parents perceive the same environment as alien, frightening, and sometimes even hostile.

Parents' feelings are doubly important because parents' reactions are picked up readily by children. These reactions, positive or negative, influence how the child copes with the system and his own emotional reaction. Different aspects of the parents, child, and health care system either work together to promote or work against the child's ability to cope in the face of illness and hospitalization.

I. Characteristics that Affect Parental Coping Skills

A. Experience with the health care system. Parents who have had positive experiences of their own with the health care system or who have had experiences with their children that have afforded them some sense of mastery are more likely to manage successfully the hospitalization of their children. Sometimes young parents with little experience in child rearing of any kind, much less in caring for the ill child, do not have the skills, confidence, or maturity to face the challenges of illness and hospitalization. Their own anxieties about the illness will strongly affect, and perhaps hinder, their ability to help their child cope with the illness.

B. Attitudes toward the health care system. Parents' attitudes toward the health care system in general may positively or negatively affect the child's coping abilities. Parents who do not trust health care providers based on past experience or prejudices communicate their concerns to their children. On the other hand, parents who are empowered by being a part of the decision-making process with health care providers are more likely to model more secure and confident attitudes. They are also more likely to convey acceptance of the treatment plan.

C. Parents' emotional state. Parents of an ill child may have so much difficulty dealing with their own emotional state (fear of the death of their child, guilt, depression, grief) that they are unable to help their child cope. Since the emotional state fluctuates, some parents will display varying capacity to support their child's coping. Parents' anxieties about illness outcome, financial impact of the illness, and worries about what other people think of them (*"If I were a better parent, he wouldn't have gotten sick in the first place."*)

Table 1.1. Sources of stress to children from illness and hospitalization

- Atypical environment and unknown caregivers
- Loss of familiar routine
- Loss of control
- Painful or frightening procedures
- Separation from family and friends
- Parental distress
- Fear of death, incapacitation, loss of body part (realistic or not)
- Loss of privacy
- Physical aspects of the illness (pain, loss of function)
- Fear of the unknown

may negatively affect their ability to help their child cope with illness and hospitalization.

D. Emotional and practical support. Parents who themselves have support systems are generally more able to address the needs of the child. Practical support (e.g., care for siblings of the ill child) also directly reduces the level of stress for parents. When the child's father and mother support each other, rather than blaming or undercutting each other's authority, the experience of hospitalization is less stressful for all involved.

II. Child's Characteristics that Affect Coping Skills

A. Cognitive level. The child's cognitive level determines the level of understanding of illness and hospitalization. Preverbal children may view hospitalization as abandonment, whereas preschoolers may perceive hospitalization as punishment for bad behavior. Older children may use the illness to manipulate parents or may misinterpret routine hospital events (e.g., a team of doctors outside the door discussing the child with the parents) as sinister actions.

B. Temperament. A child with a "difficult" temperament may have more difficulty with the demands of acute illness or the unfamiliar and altered routine of hospitalization and may react by showing resistance, irritability, or negative behavior. "Slow-to-warm-up" or shy children may withdraw and seem unresponsive to the efforts of hospital staff.

C. Coping style. Some children tend to use denial or distraction as a general approach; others actively gather information by asking questions about the illness and procedures. This style may change in the course of a prolonged condition. Generally, children who cope with stressful medical situations by seeking information adapt better to hospitalization and surgery. On the other hand, no evidence shows that to insist that children acquire information when they are not interested results in anything other than distress and frustration.

D. Previous experience with medical providers. Depending on the child's cognitive age, previous experiences with health care providers may provoke fears (*"If it hurt that bad in the office, how bad will it be in the hospital?"*) or confidence. The child may also have made a connection with acute illness or hospitalization and feel certain events are inevitable (*"My grandmother died in the hospital."*). Such beliefs are common but require a little digging to unearth.

E. Knowledge and preparation. Children who understand what is happening to them and what they can expect fare better. Preparation of children by videos, doll demonstrations, books, and explanations generally reduce their distress and increase cooperation. Preparation also decreases behavior problems after discharge. This is particularly true of children experiencing hospitalization or surgery for the first time. On the other hand, children who can remember having been through the same procedures before may show more distress if preparation strategies are used.

III. Health Care System Characteristics Affecting Children's Coping

A. Developmental appropriateness. Health care systems and providers that take into account the cognitive, emotional, motor, behavioral, and social development of patients address their needs more directly and appropriately. Studies have shown that many systems and providers are not accurate in their estimation of these levels or in their responses to them. For example, the primary care physician (PCP) often falls into the trap of overestimating the capacities of younger patients and underestimating those of older ones, so that all school-aged children may be treated alike. Developmentally inappropriate communication may increase distress through misinterpretation. Maintaining a child's sense of security may include accommodations, such as allowing a transitional object or favorite toy to stay close by and keeping the child's bed a safe haven by performing potentially painful procedures elsewhere. Finally, children, especially young ones, function best with routines, such as bedtime rituals. Hospital staff should make every attempt to establish routines in the child's hospital life. Routines may include mealtimes, medication administration, trips to the playroom, and quiet time.

B. Orientation to teaching and preparation of children and families. Developmentally appropriate preparation and teaching reduces the anxiety of both children and their families, reduces distress, and improves postdischarge behavior. Many hospitals offer anticipatory tours of hospital facilities as preparation for a planned hospitalization. These tours prepare children for the medical environment and quell some of their misconceptions about hospital activities.

C. Memory prompting. Most parents and children only remember a small portion of what is told to them by health care professionals. Often, in conversations where a diagnosis

and recommendations are given, only the diagnosis is remembered clearly. In other conversations, where several recommendations are given, only a few may be recalled. Written materials, repeated explanations, opportunities for questions, and consistent responses from all the professionals involved in the care of the child increase learning and coping for both parents and children.

D. Culturally sensitive. Medical care systems and professionals must recognize that many cultural and subcultural differences in child-rearing practices and attitudes are found. These differences include beliefs about health maintenance, the etiology and treatment of disease, and the role of the sick child in his or her family as well as ways of expressing emotion. Health care professionals must be able to adapt their expectations, practices, and communication to the needs of the individual child and family members involved. Such sensitivity increases cooperation and mutual communication and reduces stress.

E. Team orientation. Optimizing a child's ability to cope with acute illness and hospitalization in order to facilitate treatment and recovery requires a team effort. This team should include not only professionals from the different disciplines involved but also the child's parents and, where possible, the child. The PCP must be the key member of the team, providing overall leadership and being involved in day-to-day decisions about the child's care with the other team members.

F. Family orientation. Separation should be eliminated or minimized whenever possible, especially in infants and younger children. Fortunately, many children's hospitals are now much more parent friendly than in the past and are well equipped with furniture that facilitates the parents' spending the night with their child. Allowing parents in the room for procedures and during sedation and setting flexible visiting hours are additional means to reduce separation. Sibling visits may reduce feelings of being "left out" as well as reassure both the patient and the sibling of their mutual well-being. Siblings need to be prepared for what they will see, especially for changes in the patient, such as having tubes in place, casts, bandages, or an altered appearance or behavior. Encouraging parents to help take care of their hospitalized child by providing them with the necessary education and hands-on demonstrations of new techniques helps to provide a sense of usefulness and maintain the typical parental role.

G. Communication. An important strategy to relieve parental anxiety and to help parents cope with their child's acute illness and/or hospitalization is communication. It is crucial that communications with child and caregivers are consistent, honest, and frequent in order to allay the anxieties associated with acute illness and hospitalization. This requires frequent interaction between the PCP and the nursing staff,

who also regularly interact with families. Communications from consultants should generally be relayed by the PCP or at least with the PCP present to avoid inconsistencies or accidental disclosures. Communication must be clear and at the level of understanding of the parents. With widespread Internet availability, it is not uncommon to find a significant degree of medical sophistication in parents. On the other hand, some parents require very basic explanations to help them participate in medical care decisions. Parental involvement in decision making is empowering when handled supportively and reduces the sense of loss of control that parents experience.

H. Attention to pain and painful procedures. Pain management has received much attention in recent years, and several strategies can be used to reduce pain and accompanying anxiety in children. Preparation of parents and children for the procedures is important. During less painful procedures, distraction or parental coaching can reduce pain and distress, but sedation is more helpful during more painful procedures. Often, having parents in the treatment room can be helpful if parents are prepared in terms of knowledge of the procedure and of behaviors expected of them. Overly sympathetic reactions by parents tend to increase children's distress.

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2 The Problem Visit: Overview

Raymond C. Baker

Sick-child or problem visits in the primary care setting are the variable feature of the daily schedule. Add-on appointments caused by illness vary widely with time of year, often peaking during the winter respiratory season. Such patients are squeezed in among previously scheduled visits for routine care. Some physicians set aside blocks of time each day for sick visits; others schedule them between other types of visits (e.g., one or two between longer well-child visits). During the busy respiratory season, the sick visits may even keep the office open in the evening and on weekends. Although most sick visits are the result of telephone triage, others come from referrals from other physicians, urgent-care centers, or emergency rooms. Many physicians also have "walk-in" times, especially during periods of high volume.

Because most pediatric sick visits are the result of infection, a separate area of the waiting room, perhaps with a separate entrance, should be set aside to accommodate sick children. The receptionist is often the traffic controller in the waiting area, making these decisions based on a few pertinent questions over the phone or at presentation. Special care should be taken to isolate children known to be particularly infectious, such as those with varicella.

Problem visits are usually brief, depending on the physician, the number of ill visits scheduled, and the complexity of the illnesses. Five to 10 minutes are usually allowed and are adequate for most sick visits. However, it only takes one child who requires an urgent procedure or a referral to an emergency department for admission or further workup to wreck the best-planned schedule.

CHECK-IN

The office nurse or other office personnel can greatly increase the efficiency of seeing sick patients by getting the patient's medical record and obtaining a chief complaint and brief history from the caregiver. This brief information will help prioritize the visits so that children with serious illnesses are seen first. The history might also suggest an intervention prior to seeing the physician. Acetaminophen or ibuprofen should be given for a significant fever to make the child more comfortable and optimize the physician-child interaction. Certain laboratory procedures may also be considered before the physician sees the child. Examples are a throat swab for streptococcal antigen in children with fever and sore throat who are more than 2 years of age or a urine sample for urinalysis and/or culture in children with dysuria. These procedures should be based on practice guidelines developed by the physician and nursing staff. Vital signs need only include weight and temperature in most circumstances, followed by putting the child into the examination room. If the waiting period will be short (and the examination room is warm enough), the child should be undressed and gowned as appropriate for age. Young children do not usually require gowns (the child's mother will know whether the child is

modest enough to need a gown) and can be simply undressed to the waist.

PHYSICIAN VISIT

I. History

The problem visit history by the physician should be focused and directed, addressing the presenting problem(s) or symptom(s). Questions should explore the presenting symptom(s) (severity, time frame, home treatment and response, associated symptoms with a brief review of systems, and exposures), rule out serious illnesses (pertinent negatives), and confirm the most likely diagnosis (pertinent positives). Much of the past medical history that might be pertinent to the present illness should be in the child's medical record, such as immunizations, medications, and chronic illnesses.

II. Physical Examination

The extent of the physical examination in a sick visit varies with the complaint. The examination for respiratory and gastrointestinal complaints should include, at a minimum, examination of the skin, head, eyes, nose, ears, throat, neck, chest, and abdomen. For injuries, a head, musculoskeletal, and neurologic examination is usually adequate, depending on the extent of the injury. Other complaints may suggest further examination, such as:

Dysuria—abdomen, percussion for costovertebral angle (CVA) tenderness, genitalia.

Constipation or altered stool color—abdomen, rectal examination (and stool screen for occult blood).

Headache—neurologic examination and eye examination with visual acuity. (Even though decreased visual acuity is a very uncommon cause of headache, parents expect an eye examination and visual acuity check.)

III. Diagnosis and Plan

Following the history and physical examination, the diagnosis and plan of treatment should be discussed with the caregiver. This may include a prescription for medication (and advice on giving or applying the medication) and home therapy (e.g., dietary manipulation). The physician should discuss the expected course of the illness, precautions to take at home (e.g., handwashing), and possible complications that would require calling the physician or returning to the office. Finally, follow-up should be arranged and perhaps a reminder of the next well-child visit.

Although the physician must be thorough enough to capture all the appropriate information and make the correct diagnosis, the realities of a busy primary care provider's office often require efficiency. A waiting room full of sick children, hospital rounds, and a pile of paperwork are common scenarios in the primary care physician's office that influence how much time can be spent with each child. When things are hectic, ways to cut corners without reducing effectiveness include the following:

A. Perform the physical examination while taking the history.

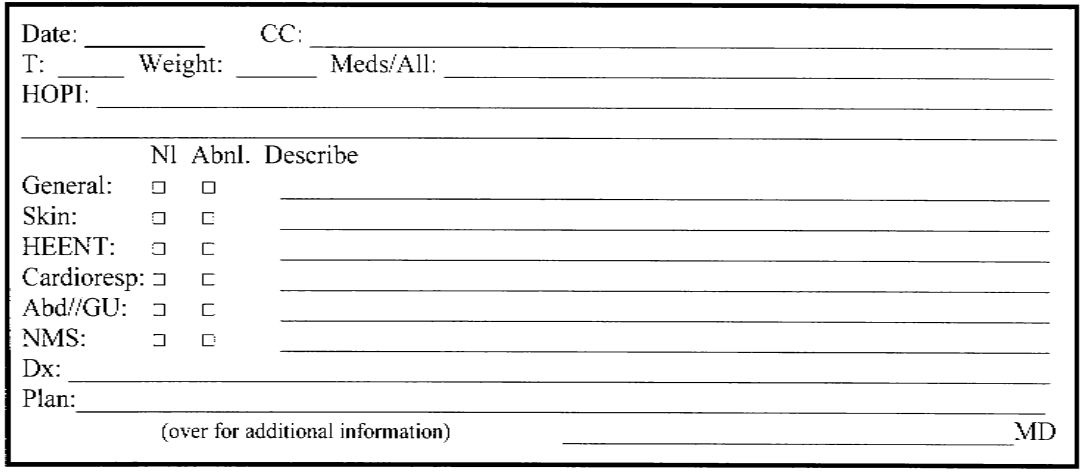

Fig. 2.1. Example of custom-designed, standardized problem visit form.

B. Don't sit down in the examining room. Although this would certainly not be appropriate during well-child care, most parents, especially those of established patients with whom a trusting relationship exists, understand the "need for speed" when they see a full waiting room and can overlook the absence of certain amenities.

C. Keep your prescription pad, tongue blades, and other disposable examining equipment in your pocket.

D. If you have enough ancillary staff, they can help by answering parents' questions, providing patient care instructions, and arranging follow-up as part of discharge procedures.

E. Write notes using small (one-third-page), custom-designed, standardized forms affixed to a page of the medical record (only top of form attached) so that they can be flipped over, allowing both sides to be viewed easily. These forms can be designed so that normal findings on the physical examination can be indicated by a simple checkmark. Abnormal findings can be briefly noted on a single line next to a box indicating "abnormal" (Fig. 2.1). Limited space is allotted to record temperature, weight, and complaint (completed by office nurse) and physician's history, physical, diagnosis, and plan. Otherwise, brief, succinct notes on the medical record can be used, keeping in mind that documentation of the visit must be adequate to be useful for future review and for legal documentation.

Even though the problem visit is usually shorter than a well-child visit and is very focused, the physician should briefly review the medical record for well-child-care information and immunizations. Children who have been noncompliant with well-child-care visits for whatever reason may be behind on important immunizations that can often be given despite a minor illness. Such visits also provide an opportunity to discuss the importance of routine well-child care with the child's caregiver.

DISCHARGE

The discharge process following the physician visit varies from practice to practice, depending on the patient volume and the number of nursing and ancillary staff. In many practices, the nurse reviews the physician plan and instructions, including medications, follow-up, and return appointments, and discusses them with the patient. With high patient volume this is an important nursing function that extends the physician visit, answers questions, and ensures that the caregiver understands all the instructions.

II

Dermatologic Disorders

3 Atopic Dermatitis and Diaper Dermatitis

Raymond C. Baker

ATOPIC DERMATITIS (ECZEMA)

Atopic dermatitis or eczema is an inherited, chronic, inflammatory skin condition of infants and children that is commonly associated with dry skin (xerosis cutis), elevated serum IgE, and eosinophilia. The diagnosis of eczema requires four of the following five diagnostic criteria:

Pruritus (necessary to make the diagnosis)
Onset between 6 weeks and 5 years of age
Typical distribution (see later)
Chronic or relapsing dermatitis
Personal or family history of atopic disease (atopic dermatitis, asthma, allergic rhinitis/conjunctivitis)

I. Description

The disease begins in the first few months of life as a red, intensely pruritic dermatitis, primarily of the cheeks, trunk, and extensor surfaces of the limbs. The diaper area is commonly spared. As the child grows older, the rash concentrates on the flexured areas of the body—antecubital fossae, popliteal fossae, posterior neck, wrists, and ankles.

Affected skin typically is erythematous and exhibits papules, crusting, and oozing (the latter two suggestive of superinfection) with secondary lesions, including excoriations, lichenification, and hyperpigmentation. The condition tends to wax and wane, but prominent features at all times are dry skin, itch, and hypersensitivity of the skin to external contacts (such as harsh soaps or an irritating cloth next to the skin). Exposure of the skin to these irritants results in itching, scratching, and finally, typical eczematous skin lesions—the "vicious cycle" of atopic dermatitis. Other skin findings that often accompany atopic dermatitis and may aid in the diagnosis are accentuated palmar creases, atopic pleats of the lower eyelids (Dennie-Morgan pleats), and dermatographism. Associated skin conditions include keratosis pilaris, pityriasis alba, and ichthyosis vulgaris. Factors that exacerbate atopic dermatitis should be sought when evaluating the atopic child and include dry skin, irritants, infection, sweating and heat, emotional stress, and sometimes exposure to specific allergens.

II. Treatment

The treatment of atopic dermatitis consists of attention to the four "I's" of eczema:

Itch
Inflammation
Infection
Immersion in water

A. Itch is a constant feature of eczema. In the young infant who is unable to scratch effectively, difficulty in sleeping or restless sleeping may be the primary indicator of itch. At all ages, excoriations and lichenification (from rubbing pruritic skin) are evidence of itch. Scratching is sometimes vigorous enough to cause bleeding. (The only other pediatric disease with equivalent itch is scabies.) Treatment of itch may require antihistamines to control it until other interventions take effect. Diphenhydramine (1.5 to 2.0 mg/kg per dose PO Q4H) or hydroxyzine (0.5 to 1.0 mg/kg per dose PO Q4H), especially at bedtime, are usually effective. The less-sedating, second-generation antihistamines, cetirizine (Zyrtec) and loratadine (Claritin), are also effective if daytime sedation from first-generation antihistamines is a problem (e.g., during school).

B. Inflammation is typically controlled with low-potency, topical steroids applied thinly and rubbed into the skin twice a day (BID) to three times a day (TID). If ineffective, moderate-potency steroids can be used for a few days until the inflammation is under control, and then the lower-potency preparation can be reinstituted for maintenance. The more potent, fluorinated steroid creams should be used cautiously in children and avoided in naturally occluded areas (groin, axilla) and face due to the increased possibility of local side effects of the steroid medication (cutaneous atrophy, steroid rosacea, striae, and telangiectasia). Low-potency steroids include hydrocortisone base or acetate, 1% and 2.5% (Hytone) and triamcinolone, 0.025% (Aristocort, Kenalog). Moderate-potency preparations are desonide, 0.05% (Tridesilon, DesOwen); fluocinolone, 0.025% (Synalar); and hydrocortisone valerate, 0.2% (Westcort). Ointments tend to be more potent than their cream counterpart with similar strength due to the occlusive nature of the vehicle.

C. Infection should be considered any time an exacerbation occurs or typical crusting and oozing develops. Superinfection with skin organisms, especially *Staphylococcus aureus,* is common and requires systemic antibiotics, such as erythromycin estolate (Ilosone).

D. Immersion in water—hydration. Perhaps the most important aspect of care is hydration of the skin. Dryness of the skin tends to produce itch and subsequent exacerbation of the dermatitis. Therefore the skin should be kept hydrated at all times. This is best accomplished by daily bathing or soaking in the bathtub or shower with lukewarm, not hot, water and using either no soap or a soap substitute such as cetyl alcohol (Cetaphil). Soaps can act as an irritant and exacerbate the condition. If dermatitis is under good control, a mild soap, such as Dove, may be used sparingly. The bath is followed immediately by a topical cream or ointment (lotion is not as effective for this purpose; ointment is most effective) to trap the water in the skin to keep it hydrated. Specific instructions to the parent are to allow the child to play/soak in the water

until the skin is thoroughly hydrated, and then, before drying, to apply the topical preparations (topical steroids first, then creams or ointments on top to maintain hydration of the skin). Sometimes the two can be combined into one if the vehicle of the steroid is appropriate, but this tends to be more expensive. In general, younger children tolerate ointments better (which are more occlusive and less expensive—Vaseline, Aquaphor), whereas older children object to the greasy feel of ointments and tolerate creams better (Eucerin, Nivea).

III. Prevention

Preventive measures in atopic dermatitis are of two types. Avoidance of factors known to exacerbate atopic dermatitis should be the mainstay, but these factors are not always known. Some factors that regularly exacerbate eczema and that should be avoided are dry skin, irritants (such as medicated soaps and creams and scratchy clothing), excessive sweating from heat, emotional stress, and exposure to specific allergens. The last may include certain foods and dust mites, which require skin testing to confirm. Cow's milk has also been indirectly implicated in the development of atopic dermatitis. Studies of breast-fed versus cow's milk–based formula-fed infants (at high risk due to a positive family history of atopy) have shown a decreased incidence of atopic dermatitis in breast-fed infants. Although soy-based formula does not appear to offer any protection, a decreased incidence of atopic dermatitis in high-risk infants fed protein hydrolysate formulas (e.g., Nutramigen, Alimentum) is recognized.

DIAPER DERMATITIS

Diaper dermatitis occurs in up to 35% of infants, peaking in the 7- to 12-month age range. Although improvements in diaper design have reduced the incidence of this condition, it continues to occur in many infants despite seemingly close attention to diaper hygiene. Effective treatments are available but require an understanding of the different etiologies of diaper dermatitis.

Care of normal infant skin in the diaper area is important and should be discussed with parents as part of routine anticipatory guidance at discharge from the birth hospital. The newer designs of paper diapers have done much to decrease urine contact because of their absorbency, but regular changing is still the mainstay of routine diaper care. Routine care should include gently wiping excess urine off with lukewarm water on a soft cloth followed by air drying. After soiling, parents should clean the diaper area similarly, perhaps using a mild soap for difficult areas, followed by air drying. Commercially available presoaked diaper wipes, despite their claims, offer no advantage and may even be irritating to inflamed skin. Although baby powders smell nice, they are unnecessary and may represent a hazard if inhaled by the infant. Corn starch, likewise, adds little since its primary functions are to absorb leftover moisture and decrease friction in the diaper area, which is unnecessary with current paper diaper designs. Once infants are sleeping through the night, there are

longer periods of time during which urine may be in contact with the skin. If, despite routine care, redness begins to appear, a thin layer of Vaseline at bedtime will act as a protective barrier overnight. Cloth diapers, despite the claims of well-meaning grandmothers everywhere, are not, in fact, superior to paper diapers. They may even be worse if rubber pants are used on top because they increase the interval between changes, prevent air from reaching the skin, and exacerbate the effects of urine/skin contact.

I. Irritant Diaper Dermatitis

Irritant diaper dermatitis is the most common form of diaper dermatitis and is due to the irritant effects of prolonged contact of the skin with urine and stool. Chronic contact of skin with wetness causes overhydration of the skin, edema of the epidermal cells, and increased permeability and susceptibility to the irritants contained in urine and stool. At the same time, fecal bacteria interact with urine to produce ammonia, which raises the pH and activates enzymes in the stool that act as irritants. Although direct invasion of the skin with bacteria is not a common event, the changes described earlier certainly increase the likelihood of infection with skin flora (*Staphylococcus, Streptococcus,* and *Candida*).

Clinically, irritant diaper dermatitis begins on the convex surfaces of the diaper area, avoiding the creases at first. With increasing inflammation, however, the creases may be invaded, making differentiation from other forms of dermatitis more difficult. Other changes that may occur with prolonged inflammation are papules, scale, maceration, intertrigo, and erosions. **Treatment** of irritant diaper dermatitis consists of frequent diaper changes and barriers to prevent further contact of skin with irritating stool and urine. Parents should avoid cleaning with irritating soaps and excessive scrubbing. Gentle cleaning with mild soaps (e.g., Cetaphil, Dove) after soiling is important to remove irritating bacteria before the application of medicaments. Barrier creams, such as A & D, Desitin, and Balmex, are effective but may be difficult to remove. For more severe inflammation, a low-potency, nonfluorinated steroid (1% hydrocortisone) applied thinly BID (with barriers then applied on top) for no more than 1 week is effective. Finally, antifungal creams (nystatin, clotrimazole) applied thinly BID (with or without steroid), also underneath the barrier, should be considered for moderate to severe diaper dermatitis that has been present for several days because superinfection with *Candida* is common with prolonged irritant diaper dermatitis.

II. Candidal (Monilial) Diaper Dermatitis

Candidiasis in the diaper area is a common complication of irritant diaper dermatitis that has been present for several days. The hallmark of candidiasis is a bright red rash with scale and red, satellite papules. Response is usually rapid when 1% clotrimazole (Lotrimin) or nystatin (Mycostatin) cream are applied thinly BID for a few days.

III. Chafing Diaper Dermatitis

Chafing diaper dermatitis is caused by the rubbing of the diaper against the skin of the diaper area. Mild erythema sparing the folds of skin and a shiny, glazed surface suggests the diagnosis. Treatment is corn starch or talcum powder applied at diaper changes to decrease the friction of the skin rubbing against the diaper, the problem that caused the rash. The physician should caution parents about leaving containers of talcum powder within reach of the infant, who may mistake it for a bottle and ingest/inhale some of the powder.

IV. Seborrheic Diaper Dermatitis

Seborrheic dermatitis of the diaper area, first appearing at 3 to 4 weeks of age, usually begins in the creases with spread to the convex surfaces. It is characterized clinically by scaly, red plaque, commonly accompanied by similar lesions in the scalp, axillae, neck folds, and postauricular area. The etiology is thought to involve the *Pityrosporum* yeast, but *Candida* may also be present, especially if the rash has been active for several days. Treatment with 1% clotrimazole cream BID, alone or alternating with a low-potency topical steroid, such as 1% hydrocortisone, is effective.

V. Atopic Dermatitis

Although atopic dermatitis usually spares the diaper area, the typical pruritic red rash of eczema may occur in the diaper area in the infant with widespread disease. Atopic dermatitis as the etiology of diaper dermatitis is usually suggested by vigorous scratching of the diaper area during diaper changes and the presence of typical atopic dermatitis elsewhere. Treatment is described earlier.

VI. Staphylococcal Pustulosis (Bullous Impetigo)

Staphylococcal pustulosis or bullous impetigo of the diaper area is characterized by crusting and pustules or bullae, which are filled with cloudy fluid or pus. If the lesions are isolated to the diaper area, they may respond to mupiricin (Bactroban) topically. However, many physicians tend to treat more conservatively with systemic antistaphylococcal antibiotics orally, such as cephalexin (Keflex) or erythromycin (Ilosone), especially in young infants and infants with similar lesions elsewhere on the body.

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4 Acne Vulgaris

Raymond C. Baker

Acne vulgaris is the most common skin disorder of adolescents, affecting 85% of those between the ages of 12 and 24 years. Unfortunately, the peak incidence of this very visible condition occurs at a time of life when appearance is so important to the self-esteem, body image, and psychosocial well-being of teenagers for socialization, formation of relationships, and even school performance. Most adults can remember the embarrassment of developing what they perceived as a very noticeable blemish just before a big date or a dance. Yet many primary care physicians may not address acne for several reasons. Adolescents do not see their primary care provider very often during the teenage years, and when they do, there always seem to be many more pressing issues to discuss. Many teenagers are embarrassed to bring up the topic and suffer in silence, relying on television advertisements for their acne advice. And finally, many parents consider acne "just one of those things all teenagers go through" and do not consider it a reason to see the physician.

I. Pathogenesis

Acne vulgaris is a disorder of the pilosebaceous unit. Under the influence of rising androgenic hormones associated with onset of puberty, the sebaceous gland increases production of sebum. At the same time, altered keratinization of the follicular canal, also influenced by androgens, results in plugging of the canal with squamous cells, obstructing the flow of sebum. This plugging produces comedonal acne with (a) **open comedones** (blackheads), in which simple obstruction occurs with dilated follicles and central black plugs, and (b) **closed comedones** (whiteheads), which are roofed. Complications of these lesions develop when an overgrowth of anaerobic organisms normally present in follicles occurs, producing inflammatory lesions (papulopustular acne).

II. History

A. Facial products currently in use, including prescription items and over-the-counter (OTC) items
B. Oral contraceptive use and menstrual history
C. Other systemic medications
D. Previous acne treatment and response
E. Known precipitants of lesions (e.g., medications, make-up, stress, menstrual cycle)

III. Physical Examination

The physical examination should include a general examination to determine the overall health of the patient and a focused skin examination to document the location of the acneiform lesions (e.g., face, back, chest) and the types of lesions present:

A. Open comedones (blackheads)
B. Closed comedones (whiteheads)
C. Inflammatory papules and pustules
D. Cysts and nodules

IV. Treatment

The treatment of acne will depend on the types of lesions that are present and the treatment history. In general, acne treatments are categorized as comedolytics, topical antibiotics, systemic antibiotics, and oral retinoids. The latter are reserved for severe cystic acne and should be prescribed by a dermatologist.

A. Routine skin care in the adolescent with acne. Once- or twice-daily gentle cleansing with mild soap with moisturizer should be routine. Other important features of routine skin care are (a) use of noncomedogenic make-up; (b) avoidance of astringents; (c) avoidance of scrubbing and picking at the face, which can lead to inflammation and pigmentary changes with healing; and (d) use of sunscreens due to the photosensitizing effects of some of the topical acne medications. Diet, bad as it may be in teenagers, has not been shown to affect acne significantly (a fact that should delight most teenagers!).

B. Comedolytic agents. Two retinoids are available and effective: tretinoin (Retin-A) and adapalene (Differin), the latter a synthetic retinoid analog. These are effective in comedonal acne alone or in combination with topical antibiotics. Teenagers with oily skin tolerate gels better, whereas those with dry, fair skin should use creams. A general rule is to start with the lowest strength, applied once a day in the evening (pea-sized amount), 20 minutes after gentle face washing. If these agents are used in combination with a topical antibiotic, each should be applied at a different time (e.g., one at night, the other in the morning, or alternating nights).

C. Topical antibacterials. Several topical antibacterials are available and effective in acne with inflammatory lesions. The mainstay of acne therapy is benzoyl peroxide. This agent is available in many formulations, including gel, lotion, and cream, and in many strengths, from 2.5% to 10% (Desquam-X, Benzagel). Creams are gentler than gels. Treatment is effective alone or in combination with a topical comedolytic. Treatment should begin with a lower strength applied once a day. Other topical antibiotics are erythromycin (available in combination with topical benzoyl peroxide as Benzamycin) and clindamycin (Cleocin T). These likewise are appropriate with inflammatory lesions.

D. Oral antibiotics. Oral tetracycline (Achromycin, Sumycin), minocycline (Minocin), doxycycline (Vibramycin), and erythromycin (EES, Ilosone) can be used in inflammatory acne that does not respond to topical antibiotics.

E. Treatment Regimens

1. *Comedonal acne.* Tretinoin cream (0.05%) or gel (0.01%) can be used on alternative nights for 2 weeks. If they are tolerated, increase to nightly use; then increase strength (up to 0.1% cream or 0.025% gel) as tolerated. Four to 6 weeks are usually needed to see significant improvement.

2. *Mild to moderate inflammatory, papulopustular acne.* Use tretinoin cream 0.05% and benzoyl peroxide gel 5% on alternate nights for 2 weeks. If these are well tolerated, benzoyl peroxide can be used in the morning and tretinoin at night. If no response is seen after 6 weeks, change to topical erythromycin or tetracycline. If there is no improvement in the inflammatory component after 6 weeks, change to oral erythromycin or tetracycline, 500 mg BID. Further therapy for recalcitrant acne requires dermatology consultation.

The treatment of acne requires ongoing counseling and visits, initially every 6 weeks, with the adolescent to maintain compliance, monitor response, and provide encouragement. Acne medications take time. If impatience pushes too rapid increase in strength of preparation, the result will be side effects that may discourage further use.

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5 Impetigo

Raymond C. Baker

Impetigo contagiosa is the most common bacterial skin dermatitis of childhood, accounting for about 10% of skin disorders presenting to the primary care physician. There are two classic forms of impetigo: nonbullous impetigo and bullous impetigo.

I. Nonbullous Impetigo

Nonbullous impetigo represents about 70% of impetigo in children. The typical lesion begins as an erythematous papule, small vesicle, or pustule and rapidly spreads to become an amber-colored crust on an erythematous base ranging in size from several millimeters to 1 to 2 cm. Impetigo commonly occurs where the integrity of the protective epidermis has been broken by the child from scratching primary lesions such as abrasions, lacerations, burns, or insect bites. The lesions occur most commonly on the extremities and around the nose and mouth. Nonbullous impetigo is seen more commonly during the summer months in preschool- and early-school-aged children. The diagnosis is made by the clinical appearance of the lesions; bacterial culture usually is unnecessary.

The **etiology** of nonbullous impetigo is *Staphylococcus aureus* (50% to 60%), *Streptococcus pyogenes* (5%), or both (35% to 45%). **Treatment** should be selected to cover both organisms. Oral antibiotics choices are erythromycin (Ilosone, EES) or cephalexin (Keflex) for 10 days. Topical therapy with mupirocin (Bactroban) applied three times per day has also been shown effective. The choice of treating orally (systemically) versus topically is influenced by the area of skin to be covered, the local prevalence of erythromycin-resistant *S. aureus,* and parental preference. Treatment of impetigo caused by *S. pyogenes* does not prevent the complication of acute poststreptococcal glomerulonephritis.

II. Bullous Impetigo

Bullous impetigo is less common than nonbullous impetigo and is due exclusively to *S. aureus.* The organism produces an exotoxin that acts locally to cause separation within the lower layers of the epidermis, encouraging bulla formation. The flaccid bullae can be filled with cloudy fluid and arise from normal-appearing skin. The lesions range in diameter from 0.5 to 3 cm. After they rupture, a thin, clear, varnishlike coating appears over the denuded area. The lesions may enlarge even after the bulla has ruptured. They are often found in small groups of three to six bullae confined to a single area. In the newborn, lesions are often found in the perineal, periumbilical, and axillary areas. In older children, they usually occur on the extremities. The diagnosis is usually made clinically; a bacterial culture is confirmatory. Treatment consists of systemic antistaphylococcal antibiotics, such as those listed earlier, given for 10 days.

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6 Lice and Scabies

Raymond C. Baker

I. Head Lice (Pediculosis Capitis)

Head lice are a significant public health issue in the United States, with an estimated 6 to 12 million cases per year, most of which occur in school-aged children. Although no diseases are carried by the head louse, considerable social stigma arises from the condition, the infestation is contagious and uncomfortable, and it results in significant exclusion from school for treatment.

A. Life cycle. The egg of the organism *Pediculus humanus capitis* incubates for 7 to 12 days before hatching in the nymph form. This form is smaller than the adult and seldom transmits. After 8 to 9 days of growth through molting, the adult form emerges and begins feeding (about five times per day) and laying eggs (about six eggs per day for 1 to 2 months). The eggs are laid close to the scalp, as they require the warmth afforded by the scalp for optimal growth. They are firmly cemented to the hair shaft and are not easily removed. Although eggs can survive for about 30 days off the host, they do not represent a mode of transmission. The adult, which is the agent of transmission, can survive only 1 to 2 days off the host.

B. Epidemiology. Girls are affected more frequently than boys, probably because they tend to demonstrate more social physical contact than boys. It is not the longer hair that increases transmission. Children are affected more frequently than adults, and whites much more frequently than blacks, probably because the female louse has greater difficulty in attaching to the more oval shape of the hair shaft in blacks. The greatest incidence of head lice occurs in the fall and winter with spread coming predominantly by direct hair-to-hair contact. Fomite-to-hair (hats, brushes, etc.) transmission is disputed and probably plays only a small role.

C. Diagnosis. The diagnosis of head lice is made by a history of itch, known exposure, and the presence of nits and adult lice in the hair. Nits appear as white or gray, smooth, oval particles firmly attached to the hair shaft close to the scalp. They fluoresce weakly with a Wood's lamp, a fact which is useful in screening large numbers of children, such as in a school setting. Excoriations, especially at the back of the neck, may be present. Particles of dandruff, which may resemble nits, are easily distinguished by easy removal from the hair shaft whereas nits are firmly attached to the hair shaft. Microscopic examination of the hair with a nit is unmistakable, if in doubt. Adult lice are seen sometimes, especially with heavy infestations, but appear less often than the nits. Studies have shown that in most cases of lice infestation, the number of adult lice in the scalp is less than 10. Itch occurs in about 35% and requires a sensitization period, similar to scabies, before symptoms develop.

D. Treatment. Several effective treatments are available for head lice, but all require nit removal to complete therapy and ensure success because of the gradual emergence of resistance. **Permethrin** (Nix) has the advantage of a residual effect and is advertised as being ovicidal. However, with the emergence of resistance to all forms of treatment, nit removal is advised following treatment. It is possible that the residual effect may, in fact, be contributing to the development of resistance. This product is applied to the hair after washing and towel drying, left on for 10 minutes, and then rinsed off with water. **Pyrethrins and piperonyl butoxide** (Rid, A-200, Pronto, R&C shampoo) are applied directly to dry hair, enough to saturate all the hair, left on for 10 minutes, then lathered with water, and rinsed off. This must be followed by careful nit removal. **Lindane** (Kwell) is no longer recommended because of decreasing effectiveness compared with other products, greater neurotoxicity, longer killing time, and need for prescription. The role of fomites in transmission is unclear and probably not of great importance. However, most physicians recommend putting clothing and bedding through a high-heat dryer cycle, followed by washing in hot water and redrying in a high-heat clothes dryer. Nonwashable items can be dry-cleaned or stored in a plastic bag for 2 weeks, which will starve the organisms.

Other treatments that have been tried and found successful anecdotally are systemic trimethoprim/sulfa, which acts by killing microflora that lice need to survive, systemic ivermectin (approved only for certain visceral parasites), 5% permethrin cream (Elimite) topically, and occlusive agents (e.g., petrolatum, mayonnaise, olive oil), which presumably act by suffocating the organism. None of these is currently recommended.

II. Pubic Lice (Pediculosis Pubis)

A. Life cycle. The life cycle of the causative organism, *Phthirus pubis,* is similar to that of the head louse.

B. Epidemiology. The peak age for transmission of pubic lice is 15 to 40 years; there is no racial predilection. The adult louse is usually transmitted during sexual intercourse. Pubic lice should be considered a sexually transmitted disease. About one-third of teens and young adults with pubic lice have another coexisting sexually transmitted disease. Pubic lice infestation as a result of sexual abuse of children has been described, but children may also acquire infestation from contact with clothing and bedding. *Phthirus pubis* infestations in prepubertal children are usually of the eyelashes and eyebrows (pediculosis palpebrarum).

C. Diagnosis. The diagnosis is made from a history of itch and exposure. Nits and adult lice are visible in the pubic hair on examination. Excoriations may be present, including characteristic sky-blue spots (maculae cerulae) in the pubic area.

Patients and sexual partners should also be tested for other sexually transmitted diseases.

D. Treatment. Treatment is similar to that for head lice, using either permethrin (Nix) or pyrethrins with piperonyl butoxide (Rid, A-200). The pediculicide is applied to all the pubic hair and surrounding abdominal, leg, and perianal hair and is left on for 10 minutes, after which it is rinsed off with water. Nits require removal with a fine-tooth comb. Sexual partners should be treated also.

E. Pediculosis palpebrarum. Infestation of the eyelashes/lids and eyebrows with pubic lice can occur as indicated earlier. Preferred treatment is petrolatum applied four or five times per day followed by the mechanical removal of nits with forceps. Because pediculosis palpebrarum is virtually always caused by pubic lice, sexual abuse should be considered when this disorder is identified in prepubertal children and should prompt further investigation.

III. Scabies

A. Life cycle. The impregnated female *Sarcoptes scabiei* burrows superficially into the epidermis, forming a burrow at a rate of 1 to 5 mm/day, laying eggs at a rate of about two or three per day. The adult life span is 15 to 30 days. Eggs hatch in 3 to 5 days and mature in 8 to 17 days, ready for impregnation. Males do not burrow, and die on the surface of the skin shortly after copulation with the newly matured, adult female.

B. Demographic profile. Men are affected more than women; girls, more than boys. The most common age is 15 to 40 years, although scabies is seen fairly commonly in children. Spread is by skin-to-skin contact, as occurs during sexual intercourse or mother/child contact. Fomites are of little importance in transmitting the organism.

C. Pathogenesis. The symptoms in scabies are most likely the result of allergic sensitization to the mite and its products. Evidence of an allergic pathogenesis includes (a) the observation that symptoms are delayed until 30 days after first infestation yet develop within 24 hours after reinfestation; (b) reinfestation is uncommon, suggesting that the allergic inflammatory response kills or attenuates the organism; (c) positive intracutaneous skin test with crude extract (immediate reaction); and (d) positive Prausnitz-Kustner passive transfer test. Although the itch is often widespread, there are actually relatively few organisms (10 to 20) in most patients (in immunocompetent individuals).

D. History. The characteristic history in scabies is itch, itch, itch! The itch of scabies typically has a gradual onset, is progressive, and is usually worse at night. The scratching it evokes is severe enough to cause bleeding, one of only two common pediatric conditions to do so (the other is atopic dermatitis). There is almost always someone else in the family that is infested with whom the affected child sleeps (siblings or parents).

E. Physical examination. Physical examination reveals a characteristic distribution that includes the flexured areas of the body—the wrists, ankles, elbows, axillae, umbilicus, interdigital web spaces, penis, and nipples. In young children, the palms and soles are commonly affected. The primary lesions are burrows, papules, vesicles, and pustules. Secondary lesions are excoriations, impetiginization, urticaria, and nodules. A common feature of the disease that aids in diagnosis is the presence of several types of primary lesions in association with severe itching.

F. Diagnosis. The diagnosis is made by the combination of a characteristic history, characteristic distribution and appearance of the lesions, identification of burrows, and demonstration of the mite on skin scraping. The latter is unnecessary if burrows are identified. Skin scrapings are performed by placing a drop of mineral oil or immersion oil on the suspicious lesion, gently scraping back and forth with a scalpel blade held at 90 degrees from the skin, then placing the scraped material onto a microscope slide for viewing under low dry power. A positive scraping is present if any of the following are seen in the scraping: scabies mite, egg, egg casing, or fecal pellet (scybalum).

G. Treatment. The preferred treatment of scabies is topical permethrin (Elimite) cream applied and left on overnight (adults and children). It is important to stress to the parent that the entire body up to the hairline must be covered, including ears and face (being cautious around the eyes). Adjunctive therapy may include oral antihistamines for itch, topical steroids for inflammation, and oral antibiotics (to cover *Staphylococcus* and *Streptococcus*) for superinfection (e.g., erythromycin, cephalexin, Augmentin).

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7 Cutaneous Dermatophytic Infections: Tinea Capitis, Tinea Corporis, Tinea Pedis, and Tinea Cruris

Christine L. McHenry

I. Tinea Capitis

Tinea capitis (ringworm of the scalp) is a dermatophytic scalp infection seen most frequently in toddlers and young school-aged children. However, infants as young as 6 days and adults as old as 70 years have been known to be infected. Three organisms are primarily responsible for disease in the United States: *Trichophyton tonsurans* (>90%), *Microsporum audouinii,* and *Microsporum canis.*

A. Clinical presentation. *T. tonsurans* is transmitted between humans and is more common in African-American children. It can cause several different types of infection: (a) seborrheic, with diffuse scaling and pruritus; (b) "black dot," with alopecia and hair remnants within the follicular orifice, giving the appearance of black dots on the scalp; (c) kerion, which represents an immune response to the dermatophyte and presents as an erythematous, boggy, tender mass that may be accompanied by systemic symptoms such as fever and lymphadenopathy; and (d) pustules or scabbed areas without scaling or significant alopecia. Major complications of tinea capitis include tinea corporis; secondary bacterial infection, usually with *Staphylococcus aureus* (uncommon); permanent alopecia; and trichophytid reactions ("id" reactions).

B. Differential diagnosis. The differential diagnosis for a child with a scaling scalp and/or alopecia includes psoriasis, seborrheic/atopic dermatitis, alopecia areata, bacterial folliculitis, trichotillomania, and traction folliculitis.

II. Diagnosis

Because many children with tinea capitis do not have areas of alopecia and broken hairs, they frequently are not diagnosed initially. A definitive diagnosis can be made only by fungal culture, which is usually positive within 1 to 2 weeks. Two other tests can be performed at the time of presentation. A Wood's lamp examination may be helpful if the infection is secondary to an ectothrix organism, such as *Microsporum* species, in which the hyphae and spores are on the surface of the hair shaft. This will cause a characteristic fluorescence under the Wood's lamp. *T. tonsurans,* which is the more common etiology, however, causes an endothrix infection, with hyphae and spores within the hair shaft, and therefore does not fluoresce. A potassium hydroxide (KOH) prep of either involved hairs or the scales may reveal the characteristic hyphal and spore appearance.

III. Management

"Tolerance" to griseofulvin has been reported for several species of *Trichophyton* and for *Microsporum canis,* resulting in the need to use higher doses of griseofulvin for longer periods of time. Currently, first-line treatment consists of oral microcrystalline griseofulvin at 20 to 25 mg/kg per dose daily given with a meal (fatty food in the meal enhances absorption). If ultramicrocrystalline griseofulvin is used, the dosage is reduced by one-half. Grifulvin V (125 mg/5 mL) is the only form of griseofulvin available as an oral suspension. A short course of oral prednisone (5 to 14 days, 1 to 2 mg/kg per day with a taper if used for more than 7 days) may be considered if a large, symptomatic kerion is present. If a superimposed *S. aureus* infection is present, the appropriate antistaphylococcal antibiotic should be added. The child should be seen at 4-week intervals to evaluate progress, encourage compliance, and provide an opportunity for reculture. Treatment with griseofulvin should be continued until a negative culture is obtained (usually 6 to 8 weeks) and clinical infection has resolved. Since griseofulvin is derived from a species of *Penicillium,* a penicillin-sensitive individual might react to griseofulvin. Reported side effects of griseofulvin include leukopenia, aplastic anemia, hypersensitivity, photosensitivity, gastrointestinal disturbance, and paresthesias. Monitoring blood counts while the child is on a 6- to 8-week course of griseofulvin is probably unnecessary. Shampooing twice weekly with selenium sulfide 1% (sold over the counter as Selsun Blue) or 2.5% (prescription-strength Selsun) will decrease spore shedding and help prevent spread of the infection. Combs, brushes, ribbons, and so on should not be shared, and clothing and bed linens should be laundered in a hot-cycle washer and dryer. Since almost half of asymptomatic family members may be carriers, some recommend the use of selenium sulfide shampoo on family members at the same time the index child is being treated. If the offending organism is *Microsporum canis,* the household dog or cat requires evaluation for infection. Children can return to school once griseofulvin therapy is started. If the physician suspects tinea capitis but elects not to begin griseofulvin until culture results are back, the child should shampoo twice weekly with selenium sulfide so spore dissemination will not occur in the school setting. For "griseofulvin-tolerant" tinea capitis, itraconazole (Sporanox) at 3 to 5 mg/kg per day for 4 weeks, fluconazole (Diflucan) at 6 mg/kg per day for 3 to 6 weeks, or terbinafine (Lamisil) at 3 to 6 mg/kg per day for 4 weeks may be considered. Since none of these drugs is approved by the Food and Drug Administration for tinea capitis in pediatrics, a thorough discussion with the parents and older child of the benefits and risks must occur before use.

IV. Tinea Corporis

Tinea corporis (common ringworm) is a common superficial dermatophytic skin infection of children most commonly caused by *Trichophyton rubrum*, *mentagrophytes*, and *Microsporum canis. Trichophyton* infections are acquired through contact with an infected human or inanimate fomite; *Microsporum*,

through contact with an infected cat or dog. These infections present as pruritic annular, red, scaly lesions, often with central clearing and slightly raised borders. They are most commonly single lesions and occur almost anywhere on the body, including the trunk, extremities, and face. Although these lesions are usually recognized clinically, they may resemble the herald patch of pityriasis rosea, granuloma annulare, and other circumscribed dermatitides. Many effective topical treatment options are available, including clotrimazole (Lotrimin) and miconazole (Micatin), both of which are sold over the counter. These should be applied twice a day for 2 to 4 weeks until the lesions have cleared.

V. Tinea Pedis

Tinea pedis (athlete's foot) is a superficial dermatophytic skin infection seen in teenagers and adults and is caused by *Trichophyton rubrum* and *Epidermophyton floccosum.* These infections are acquired most commonly by walking on infected surfaces. Tinea pedis has three common clinical appearances: (a) **interdigital,** with pruritic fissuring and maceration between the toes (most commonly the third, fourth, and fifth); (b) **moccasin type,** with a dry, scaly, mildly inflammatory dermatic rash of the entire sole of the foot; and (c) **inflammatory,** with pruritic vesicles, inflammation, and erosions of the instep. Tinea pedis is usually a clinical diagnosis, but it may resemble dyshidrotic eczema and contact dermatitis. Treatment is topical antifungals as given earlier.

VI. Tinea Cruris

Tinea cruris (jock itch) is a dermatophytic skin infection seen in teenagers and adults (mostly male) caused by *Trichophyton rubrum*, *mentagrophytes*, and *Epidermophyton floccosum.* It occurs most commonly in humid environments, especially with obesity and occlusive clothing, such as athletic supporters and swim suits. Lesions are located on the inner thighs and inguinal creases; they appear as pruritic, erythematous plaques spreading along the inner thighs and buttocks, usually sparing the scrotum and penis. Tinea cruris is usually diagnosed clinically, but seborrheic dermatitis and psoriasis may resemble this infection. Treatment is topical antifungals as given earlier.

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8 Acute, Febrile Exanthems of Childhood

Raymond C. Baker

As many as 10% to 20% of pediatric patients seen for a problem visit in a pediatric practice, urgent-care center, or emergency department have a rash, either as the presenting complaint or as an associated feature of their illness. Some exanthems are distinctive enough in their appearance that a diagnosis can be made by visual inspection alone. In most patients, however, the physician must use the rash as a clue to the underlying diagnosis and obtain historical, physical, and occasionally, laboratory information to make a diagnosis.

I. History

A. Chronology of the rash. The initial location and the changing appearance over time.

B. Associated nondermatological symptoms. Fever (height of fever and length of time between fever and onset of rash), gastrointestinal symptoms (vomiting, diarrhea, abdominal pain), respiratory symptoms (cough, rhinorrhea, sore throat), and neurologic symptoms (headache, malaise, weakness).

C. Symptoms of the rash. Symptoms of the rash include pruritus (pruritus in infants is suggested by observed itching and restless sleep), pain, or tenderness.

D. Treatment and response to treatment. Treatment the parent has tried at home, including over-the-counter medications and topical medications.

E. Past medical history. Exposures, medications, immunizations (past and recent), allergies, previous exanthematous illnesses (especially common childhood illnesses associated with rash).

II. Physical Examination

Physical examination should include a general physical examination, plus a specific complete skin assessment with attention to skin, hair, nails, and mucous membranes (looking for associated enanthems) and description of the rash:

A. Terminology. Primary lesions.

1. *Macule.* Flat lesion < 0.5 cm in diameter.

2. *Patch.* Flat lesion > 0.5 cm in diameter.

3. *Papule.* Raised, palpable, superficial lesion < 0.5 cm in diameter.

4. *Plaque.* Raised, palpable, superficial lesion > 0.5 cm in diameter.

5. *Nodule.* Three-dimensional enlargement of a papule.

6. *Vesicle.* Loculated, fluid-filled papule < 0.5 cm in diameter.

7. *Bulla.* Loculated, fluid-filled papule > 0.5 cm in diameter.

8. *Pustule.* Vesicle or bulla filled with purulent material.

B. Terminology. Secondary lesions.
1. *Excoriations.* Linear abrasions from scratching.
2. *Scale.* Adherent (shiny) or loose (gray to skin colored).
3. *Crusting/oozing.* Dry/wet products of exudation.
4. *Hypopigmentation or hyperpigmentation.* Evidence of recent resolved inflammation.

C. Distribution of the lesions. Generalized, acral, truncal, symmetric.

D. Arrangement of lesions. Annular, linear, grouped, random.

E. Color. Red, white, depigmented, pigmented, skin colored, fluid filled.

F. Manipulations. Blanch with pressure, Nikolsky's sign.

III. Laboratory Examination

A. Gram stain, fluorescent antibody, and other specialized stains

B. Tzanck, KOH, oil preparations

C. Culture, serologies

D. Complete blood cell count, body fluid cultures, lumbar puncture

RED, MACULOPAPULAR EXANTHEMS

I. Rubeola (Measles)

A. Prodrome. Fever, malaise, coryza, conjunctivitis (nonexudative), photophobia, toxicity.

B. Description/chronology of rash. Koplik spots (enanthem) appear shortly before the onset of the exanthem as white papules on an erythematous base located on the buccal mucosa opposite the upper molars. The rash is a red maculopapular eruption beginning on the face/hairline and spreading downward, eventually becoming generalized.

C. Course. The rash appears 3 to 4 days after onset of prodrome and worsens for 3 days, becoming confluent on face, trunk, and extremities; it then clears over a 3-day period as the child defervesces. Recovery occurs within 10 days, although the rash may desquamate 1 to 2 weeks after the rash. The child is contagious until 4 days after onset of the rash. The diagnosis of measles is clinical; however, because of the public health implications of this diagnosis, serologic confirmation is suggested.

D. Treatment. Antipyretics for comfort, fluids, observation. Vitamin A has been shown to be reduce morbidity and mortality in underdeveloped countries.

E. Etiology. Measles virus.

II. Rubella (German, 3-Day Measles)

A. Prodrome. Low-grade fever, minor cold symptoms.

B. Description/chronology of rash. The rash is red and maculopapular without enanthem, beginning on the face/hairline and spreading downward. The upper body rash fades as spread downward occurs, with resolution of the rash by 3 days. Suboccipital, postauricular, and posterior cervical adenopathy are common.

C. Laboratory. Because this illness has become so rare with universal immunization, this disease cannot be diagnosed clinically. Serologic diagnosis is required.

D. Course. The illness is mild, with complete resolution in about 7 days. Complications are few except for monarticular arthritis in adolescent girls. The child is contagious for 5 to 7 days after the onset of the rash.

E. Treatment. Antipyretics for comfort, observation.

F. Etiology. Rubella virus.

III. Roseola Infantum (Exanthem Subitum)

A. Prodrome. High fever, occasional febrile seizure.

B. Description/chronology of rash. The rash is a red to pink maculopapular exanthem that begins abruptly (*subitum*—Latin for "suddenly") within 24 hours of defervescence after 3 to 4 days of fever. The rash is generalized from the onset and often has associated suboccipital adenopathy.

C. Course. The illness occurs in infants from 6 to 24 months of age. Infants are usually nontoxic in appearance, even during the febrile period, and the onset of the rash heralds the end of the disease. The infant is contagious until 24 hours after defervescence. The diagnosis of roseola is clinical; laboratory confirmation is not required.

D. Treatment. Antipyretics for comfort, fluids, observation.

E. Etiology. Human herpesvirus 6 (most common), human herpesvirus 7 (uncommon).

IV. Erythema Infectiosum (Fifth Disease)

A. Prodrome. Minor cold symptoms; fever may be low grade or absent.

B. Description/chronology of rash. The rash begins as a confluent red macular exanthem on the cheeks ("slapped cheek appearance"), followed by a red, maculopapular, lacy, reticular exanthem on the shoulders and proximal extremities, spreading to the trunk.

C. Course. This illness is most commonly seen in school-aged children, 3 to 12 years old. Systemic symptoms are usually absent. The diagnosis can be made clinically without laboratory confirmation. Isolation is not needed because the contagious period is commonly over when the diagnosis is made.

D. Treatment. None.

E. Etiology. Human parvovirus B19.

V. Scarlet Fever

A. Prodrome. High fever, sore throat, headache, abdominal pain.

B. Description/chronology of rash. The enanthem of scarlet fever (streptococcal pharyngitis) begins 1 to 2 days before the exanthem as an injected pharynx with enlarged, red, exudative tonsils; a strawberry tongue; and palatal petechiae. The exanthem of scarlet fever is a finely papular, sandpaper-like, red rash beginning in intertriginous areas (neck, axillary, groin, antecubital—Pastia's lines) before

becoming generalized the following 1 to 2 days. Circumoral sparing is common. Petechiae may be present with the exanthem, mostly above the nipple line. Tender anterior cervical adenopathy is usual.

C. Course. Scarlet fever is seen most commonly in children more than 2 years of age. Systemic toxicity resolves within 24 to 48 hours of antibiotic therapy; pharyngeal pain ends shortly thereafter. The rash lasts 7 to 10 days with desquamation of the rash 1 to 2 weeks after onset, especially on the palms and soles of the feet. The diagnosis should be confirmed with a streptococcal antigen or throat culture. The child is contagious until 24 hours after antibiotic treatment.

D. Treatment. The treatment of scarlet fever is the same as that for streptococcal pharyngitis: benzathine penicillin G, 600,000 U (child's weight <60 pounds or 27 kg) or 1,200,000 U (>60 pounds) intramuscularly as a single injection; penicillin V, 250 mg BID (children) or 500 mg BID (adolescents) for 10 days; amoxicillin, 20 mg/kg per dose BID for 10 days (better taste than most oral penicillins); erythromycin 20 mg/kg per dose BID for 10 days for penicillin-allergic children.

E. Etiology. *Streptococcus pyogenes* (group A streptococcus). Some strains of *Staphylococcus aureus* that produce an exfoliative toxin also cause a disease resembling scarlet fever. Staphylococcal scarlet fever is differentiated from streptococcal scarlet fever by the absence of pharyngitis, tenderness of the rash, and early desquamation (2 to 3 days).

VI. Enteroviral Exanthems

Enteroviral exanthems are common during the summer months. Because of the large number of enteroviruses that cause clinical illness in children, including coxsackieviruses and echoviruses, the exanthematous manifestations are many and varied. Commonly, an exposure history, a febrile prodrome of 1 to 3 days, and associated gastrointestinal or respiratory symptoms are present. The rash is most commonly red, maculopapular, or morbilliform in appearance, but a vesiculopapular exanthem is also seen with some types. Petechiae may be seen but are not the predominant lesion. Most children are nontoxic with the illness, and the rash and symptoms resolve in 7 to 10 days without treatment. Antipyretics for comfort and fluids (especially with gastrointestinal symptoms) to prevent dehydration are the only treatment modalities needed. Because of the benign nature of most of the enteroviral exanthems (associated aseptic meningitis is the exception), it is usually unnecessary to confirm the diagnosis with culture or serologic tests, and the diagnosis is largely one of exclusion.

Some of the enteroviruses cause a distinctive clinical illness, which permits a more exact diagnosis:

A. Hand/foot/mouth disease. The predominantly vesicular rash is located on the feet, hands (mostly ventral surfaces), and buttocks, with an associated enanthem (herpetic-like stomatitis, but sparing the gingivae). Treatment is unnecessary,

and the disease resolves in 7 to 10 days. Isolation until 24 hours after defervescence prevents spread to other children.

B. Herpangina. This illness presents with fever and sore throat. An exanthem is usually absent; the physical findings consist of an enanthem with vesicles and ulcers located posteriorly in the mouth and on the tonsils, tonsillar pillars, posterior soft palate, and posterior pharynx. Sore throat symptoms may be alleviated with cold liquids and soft solids (e.g., cold pudding, ice cream) and ibuprofen. Topical anesthetics may also alleviate sore throat pain but are difficult to apply to the affected area.

VII. KAWASAKI DISEASE

A. Diagnostic criteria.

1. *Fever persistent for 5 or more days* that is poorly responsive to antipyretics.
2. *Nonexudative conjunctivitis* (primary bulbar).
3. *Nonsuppurative cervical adenopathy.*
4. *Erythema and edema of palms/soles.*
5. *Enanthem:* red, cracked lips, strawberry tongue, nonexudative pharyngitis/tonsillitis.
6. *Rash:* red, maculopapular exanthem, primarily of trunk. In infants still in diapers, scarlatiniform rash in diaper area. Desquamation 10 to 20 days following rash, especially involving fingers and toes.

B. Laboratory. Elevated acute-phase reactants, including platelet count, elevated liver enzymes with cholestatic pattern of elevated bilirubin and alkaline phosphatase, sterile pyuria. Streptococcal and staphylococcal disease must be ruled out.

C. Course. The acute phase lasts 10 to 14 days followed by the subacute phase of 10 to 14 days, during which symptoms of fever and rash resolve. Cardiac manifestations occur during the latter phase. The convalescent phase may last several months as evidence of the disease process resolves.

D. Treatment. The acute treatment is intravenous immune globulin (IVIG) 2 g/kg and aspirin 80 to 100 mg/kg per day divided four times a day, followed by low-dose aspirin. Careful follow-up is necessary to diagnose cardiac pathology (myocarditis, coronary dilatation, and aneurysms).

E. Etiology. Unknown.

VIII. Rocky Mountain Spotted Fever

A. Prodrome. Rapid fever rise, headache, myalgia, tick exposure (often from endemic area).

B. Description/chronology of rash. The rash of Rocky Mountain spotted fever (RMSF) is a red, maculopapular rash beginning peripherally (on the hands, feet, wrists, and ankles) and spreading inward. At any stage of the illness, the rash is most concentrated peripherally. Untreated, the rash may progress to a petechial and hemorrhagic rash.

C. Laboratory. Tendency to thrombocytopenia (mild) and hyponatremia. Specific serologies are diagnostic.

D. Course. RMSF presents with increasing fever and toxicity, headache, and myalgia. Untreated, the rash and symptoms progress to a severe illness with prominent central nervous system disease and multisystems disease, disseminated intravascular coagulation (DIC), shock, and death in 10% to 40% of those infected. With early treatment, prognosis for full recovery is excellent, especially in children. The diagnosis of RMSF is usually clinical, especially in areas of endemic disease. Serologies are confirmatory.

E. Treatment. Doxycycline for 7 to 10 days until patient has been afebrile for 3 days. Despite the possibility of dental staining in children, doxycycline is the drug of choice because it also covers Ehrlichiosis, which may resemble RMSF, and because a single course of doxycycline is unlikely to cause dental staining.

F. Etiology. *Rickettsia rickettsii.*

VESICULOBULLOUS EXANTHEMS

I. Varicella (Chicken Pox)

A. Prodrome. Fever, anorexia, malaise, headache.

B. Description/chronology of rash. The primary lesion of varicella is a pruritic vesicle on an erythematous base progressing to a pustule, an umbilicated pustule, then to a crust. The rash begins on the face and trunk and spreads to all parts of the body, including the scalp and mucous membranes (enanthem) of the mouth, eyes, and genitalia. The presence of pruritic vesicles or ruptured vesicles in the scalp is diagnostic.

C. Course. Varicella can be seen at any age, including the neonatal period. The severity of varicella varies from mild to severe illness. In general, adolescents and adults tend to have more severe disease. Fever may persist 3 to 5 days after the appearance of rash. The child is no longer contagious after all lesions have crusted (5 to 7 days after onset of rash).

D. Treatment. Acetaminophen for comfort (ibuprofen contraindicated because of possible association with necrotizing fasciitis; aspirin contraindicated because of possible association with Reye syndrome), fluids, antihistamines for pruritus, observation for complications (especially group A streptococcal secondary infection). Acyclovir, 20 mg/kg per dose four times per day for 5 days (maximum dose 3,200 mg/day) given within 24 hours of onset of rash results in a modest decrease in the morbidity of the disease. It is not recommended routinely but should be considered for adolescents, immunosuppressed children (given parenterally; see *2000 Red Book*), and children with underlying chronic skin (severe eczema) and pulmonary disorders.

E. Etiology. Varicella zoster virus.

II. Zoster

Zoster (shingles) results from reactivation of dormant varicella zoster virus from sensory root ganglia. The rash most commonly affects the head, neck, and thoracic nerves; systemic

symptoms are usually absent. The rash begins with a prodrome of itching or burning followed by the appearance of papulovesicles in a dermatome (often incomplete dermatome) distribution. The rash is usually fully developed by 3 to 5 days and resolves in 1 to 2 weeks with the development of crusts much like varicella.

Although zoster is seen more commonly in immunosuppressed children, it can also occur in healthy children, particularly those whose primary varicella occurred in the first 2 to 3 months of life. Zoster is contagious (will cause varicella in exposed, nonimmune, unimmunized children) until crusting occurs, but isolation is probably unnecessary if the lesions are covered by clothing. Treatment is largely symptomatic, as in varicella, except in immunocompromised children who benefit from acyclovir. Topical acyclovir is not effective.

III. Primary Herpes Simplex Infection

A. Prodrome. Fever, irritability.

B. Description/chronology of rash. Clusters of red papules that evolve into vesicles and pustules over a 5- to 7-day period. Location depends on inoculum site (self-inoculation from oral herpes of a previously injured site). Common cutaneous sites are the fingers (herpetic whitlow), periorbital, perioral, and genital. The latter in children are most commonly the result of self-inoculation with HSV type 1 (HSV-1), but sexual abuse and infection with HSV-2 should be considered.

The most common type of primary herpes (HSV-1) in children is acute, primary herpetic gingivostomatitis. In this acute, febrile illness, the young child (usually under 4 to 5 years of age) presents with fever, irritability, cervical adenopathy, and oral lesions. The oral manifestations of primary herpes are gingivitis, with red, swollen, friable gums, and herpetic vesicles and erosions of the oral mucous membranes, including buccal mucosa, gums, palate, tongue, and posterior pharynx. Occasionally, herpetic lesions are found scattered on the lips and perioral area, face, and upper trunk. The child is miserable and does not eat well because of the pain and irritation of the oral lesions that is caused by food and chewing. Occasionally, dehydration results from the lack of oral intake. Enteroviral stomatitis is differentiated from herpetic stomatitis by the absence of gingival involvement.

In the neonate, herpes infection (predominantly HSV-2 acquired during delivery) involving the eye (keratitis), skin, and mucous membranes (vesicles) may be localized to these sites (about one-third) or evident at these sites as a part of a disseminated infection (one-third). Disseminated disease has a considerably poorer prognosis and must be treated aggressively with parenteral acyclovir and supportive therapy.

C. Course. Primary herpes infections can cause significant fever and irritability because of the tenderness of the lesions and irritating effects of food and chewing. Lesions resolve in 10 to 14 days but may serve as a site for recurrent herpes. Recurrent herpes is not associated with fever and has

a shorter duration (7 days). The most common site of recurrent herpes in children and adolescents is the lips.

D. Treatment. In previously healthy children with gingivostomatitis, no treatment is usually needed other than symptomatic. Cutaneous lesions should be kept clean to prevent bacterial superinfection, and proper hygiene measures should be instituted to prevent spread. Although some physicians recommend topical anesthetics for the painful oral lesions of primary stomatitis, many young children do not tolerate them. Topical cold applications in the form of popsicles, cold puddings, and ice cream are usually better tolerated, are effective, and have the added benefit of calories and liquid in the child's sparse diet. Herpetic infections elsewhere (whitlow, periorbital, perioral, genital) in children likewise usually require only symptomatic care. It is important to recognize a herpetic whitlow, with its confluent clusters of vesicles, to avoid lancing it, which may introduce bacteria and result in superinfection.

E. Etiology. HSV-1.

IV. Hand/Foot/Mouth Disease

See "Enteroviral Exanthems," earlier.

V. Bullous Impetigo and Staphylococcal Scalded Skin Syndrome

Exfoliatin-producing strains of *Staphylococcus aureus* produce two characteristic skin syndromes, both mediated by the exfoliative toxin. **Bullous impetigo** results from the local action of the exfoliatin toxin following invasion of the skin by the organism, usually through a trauma-induced break in the integrity of the epidermis. Bullous lesions result from separation within the granular layer of the epidermis to produce characteristic fluid-filled lesions. They often rupture, leaving a denuded patch that may continue to enlarge. The face and extremities are common sites for bullous impetigo in older children. The diaper area is a common site in infants. *Staphylococcus aureus* can be cultured from the lesions, but culture is unnecessary as the diagnosis can usually be made clinically. Treatment is systemic antibiotics, with oral first- or second-generation cephalosporins, for 7 to 10 days, supplemented with local hygiene measures. Erythromycin is not indicated because of increasing resistance among staphylococcal organisms.

Staphylococcal scalded skin syndrome (SSSS) results from the hematogenous spread of the exfoliatin toxin resulting in widespread toxin effect with fever, skin tenderness and erythema, and fluid-filled, thin-walled bullae. Nikolsky's sign may be present (gentle friction of the skin resulting in sloughing of superficial sheets of skin). Organisms usually cannot be recovered from individual bullae; the primary infection site is most commonly the respiratory tract (conjunctivae or nasopharynx). SSSS is seen predominantly in infants and young children less than 5 years of age. Infants with the disease are irritable because of the skin tenderness and are often febrile. Bacteremia may be

present, especially in febrile infants. Treatment is systemic antibiotics, oral or parenteral, depending on the clinical appearance, presence of significant fever, and presence of bacteremia. First- or second-generation cephalosporins are effective.

HEMORRHAGIC EXANTHEMS

I. Acute Meningococcemia and Acute Meningococcal Meningitis

The hemorrhagic exanthems refer to those exanthems with nonblanching red to purple petechiae, purpurae, and/or ecchymoses either as the predominant lesion or in combination with other types of lesions. **Acute meningococcemia and acute meningococcal meningitis** often present with fever and a progressive hemorrhagic rash (50% to 70%). The disease tends to develop rapidly with fever, toxicity, and petechiae progressing to ecchymoses as DIC develops. Occasionally, the rash begins as an urticarial or red, maculopapular rash before the development of the hemorrhagic lesions. The areas of predilection are the lower extremities and trunk, although lesions can occur elsewhere, including mucous membranes. Because meningococcemia can be a rapidly progressive illness, treatment should be instituted as soon as possible. Patients presenting with a high fever and a toxic appearance should be thoroughly examined for hemorrhagic lesions. Their presence should prompt a quick evaluation for sepsis with blood and cerebrospinal fluid cultures and initiation of antibiotic therapy. Because bacterial infections other then *Neisseria meningitidis* can also cause a hemorrhagic rash, broad-spectrum antibiotics should be used, such as the combination of ampicillin and a third-generation cephalosporin.

II. Scarlet Fever or Streptococcal Pharyngitis

Scarlet fever or streptococcal pharyngitis (without the scarlatiniform rash) can also present with a few petechiae located primarily above the nipple line. The diagnosis should be suspected by the presence of associated exudative pharyngitis (as earlier). In older children (more than 2 years of age), the streptococcal antigen test can confirm the presence of group A streptococci and allow outpatient management without further workup. The younger child who appears toxic and has a hemorrhagic rash requires evaluation for sepsis and meningitis as stated earlier.

III. Enteroviral Illness and Rocky Mountain Spotted Fever

Two other etiologies of acute, febrile exanthems may also have a hemorrhagic component to the exanthem—enteroviral illness and Rocky Mountain spotted fever, both discussed earlier.

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III

Head, Ears, Eyes, Nose, and Throat Disorders

9 Acute Otitis Media

Raymond C. Baker

Acute otitis media (AOM) is the most common bacterial infection in pediatrics, accounting for as many as 40% of all illness visits to the primary care physician. At least two-thirds of children experience one or more episodes of AOM during the first year of life and half have had at least three episodes by 3 years of age.

I. Epidemiology

The highest incidence of AOM is in the first 2 years of life due to several factors. Infants have increased susceptibility to upper respiratory tract infections; they have more obstruction of the auditory tube due to relatively larger amounts of nasopharyngeal lymphoid tissue; and the auditory tube is less competent. Risk factors that have been reported to be associated with an increased incidence of AOM during infancy and early childhood are the following:

A. Smoking in the home
B. Attendance at day care
C. Family history of recurrent AOM
D. Presence of siblings in the home
E. Cow's milk formula
F. American Indian or Eskimo ethnic background
G. Immunodeficiency
H. Lower socioeconomic status
I. Pacifier use
J. Male gender
K. Nasopharyngeal colonization with otitis media pathogens
L. Presence of cleft palate (virtually 100% incidence of recurrent AOM)
M. Down syndrome

II. Pathogenesis

The normal functions of the auditory tube are (a) ventilation of middle ear space, (b) protection from the nasopharynx, and (c) clearance of secretions from the middle ear space. In the presence of an upper respiratory viral infection, the mucous membranes of the auditory tube, middle ear (including the tympanic membrane), and lymphatic tissue in the area become inflamed and swollen. The inflammation results in the obstruction of the tube to the egress of secretions from the middle ear space and to the ingress of air to equalize pressure in the middle ear space. The result is negative pressure in the middle ear space, which encourages entry of pathogenic organisms from the nasopharynx through the auditory tube and infection with pus formation (under pressure) in the middle ear. Clinically, this manifests as fever, tympanic membrane inflammation (viral effect), followed by pus under pressure in the middle ear space (bacterial effect) with outward pressure on the tympanic membrane, and pain from the distortion, inflammation, and edema of the tympanic membrane. Although the most common cause of obstruction of the auditory

tube that predisposes to middle ear infection is swelling secondary to viral infection, other causes are hypertrophied lymphoid tissue (adenoids) or tumor, allergic inflammatory edema, impairment of the opening mechanism from muscular dysfunction (tensor veli palatini muscle), and excessive tubal wall compliance.

III. Diagnosis

In the very young infant, symptoms of AOM are nonspecific, as in most other illnesses at this age, and may include irritability, rhinorrhea, poor feeding, diarrhea, cough, and fever. In the older child, symptoms are otalgia, fever, rubbing or pulling at the ears and poor sleeping (in the preverbal infant), otorrhea from a ruptured tympanic membrane (TM), and decreased hearing.

The diagnosis is most accurately made with the pneumatic otoscope, which may reveal a bulging TM secondary to pus under pressure in the middle ear canal; immobility or decreased mobility of the TM due to middle ear fluid or pus; opacity, thickness, or erythema (due to tympanitis) of the TM; pus visible through the TM; or perforation of the TM with pus exuding. Sometimes, especially during the viral phase of AOM (before the development of pus in the middle ear space), TM changes of erythema/inflammation due to tympanitis may be the only abnormality seen on otoscopy. Since crying may also cause TM erythema, this finding must be associated with other symptoms or signs suggestive of a respiratory infection to suggest true pathology.

IV. Etiology of AOM

The etiology of AOM in otherwise healthy infants and children is the same regardless of age. The causative organisms of acute otitis media, in decreasing order (based on tympanocentesis findings), are *Streptococcus pneumoniae,* nontypable *Haemophilus influenzae,* nontypable agents, *Moraxella catarrhalis, Streptococcus pyogenes,* and *Staphylococcus aureus.* As many as 25% of cultures of middle ear fluid during an episode of AOM are sterile, and 10% will grow only respiratory viruses.

Another category of organisms that has become increasingly important to the clinician, especially when making treatment decisions, is the growing number of antibiotic-resistant organisms. The first type of resistance that became particularly relevant in AOM therapy was the development of beta-lactamase-producing nontypable *Haemophilus influenzae,* which appeared in the 1980s. As the second most common etiology of AOM, the prevalence of resistant *H. influenzae* in some communities had a considerable impact on treatment decisions and the use of the old stand-by antibiotic, amoxicillin. The result has been the increasing use of other classes of antibiotics that tend to be broader spectrum (and more expensive). Other pathogenic organisms in AOM have a similar resistance mechanism (*Moraxella catarrhalis* and *Staphylococcus aureus*) but are less common etiologies of AOM.

The second type of resistance, which emerged in the 1990s, is penicillin-resistant *Streptococcus pneumoniae.* Resistance of this organism is based on altered penicillin-binding proteins. To date, most penicillin resistance in noninvasive disease, such as AOM,

has been in the intermediate resistant category, which can be largely overcome by the use of higher doses of amoxicillin or other classes of antibiotics.

A third resistance pattern that has affected the treatment of AOM is the resistance of several pathogenic organisms of importance in AOM to trimethoprim/sulfamethoxazole. The mechanism of resistance organisms have developed for this antibiotic is reduced inhibition of the enzyme dihydrofolic acid reductase. The prevalence of this mode of resistance has all but eliminated the usefulness of this antibiotic in AOM.

V. Treatment

The choice of antibiotic should be based on regional prevalence of organisms likely to cause AOM, regional prevalence of antibiotic resistance of these organisms, and concentrations of antibiotic that can be obtained in the middle ear. Other factors that influence antibiotic choice are compliance (taste, frequency of dosing) and cost. Because of the emergence of resistant organisms and the overuse of antibiotics that many experts argue has contributed to the development of resistance, the decision to treat AOM is changing. Whereas the provider previously treated patients at first visit with any evidence of AOM, many experts now suggest that treatment at first visit be reserved for patients with significant findings consistent with AOM with pus in the middle ear space—fever, bulging and immobile TM, pus visible through the TM, perforation with pus exuding. For patients with less convincing findings, treatment with analgesics for pain and fever and follow-up in 24 to 48 hours (perhaps by telephone) might eliminate the need for antibiotics in some patients. This approach is especially appealing since AOM outcome studies have demonstrated resolution without antibiotics in 70% to 90% of patients. Furthermore, no research evidence demonstrates the long-term efficacy of antibiotics in reducing compromises in hearing and speech or in the reduction of the need for surgical intervention.

Antibiotics may be divided into first line (new-onset AOM) and second line (treatment failure, known organism sensitivities). In general, second-line antibiotics are more expensive and have a broader spectrum of activity, especially against beta-lactamase-producing organisms. Although the time-honored length of therapy is 10 days, recent studies suggest that a 5-day course is sufficient for uncomplicated AOM in children more than 24 months of age. Single-dose, intramuscular ceftriaxone has also been shown effective in uncomplicated AOM, but because of the very broad spectrum of its activity and the need for injection, this treatment should be reserved for patients in whom compliance is an issue, patients with significant vomiting, and patients with coexisting conditions requiring ceftriaxone.

A. First line. Amoxicillin, 40 to 45 mg/kg PO BID (total daily dose 80 to 90 mg/kg)

B. Second line

1. Cefprozil, 15 mg/kg PO BID
2. Amoxicillin/clavulanate, 20 to 22 mg/kg PO BID

3. Cefixime, 8 mg/kg PO QD
4. Loracarbef, 15 mg/kg PO BID
5. Ceftriaxone, 50 mg/kg (maximum dose 1,000 mg) IM × 1.
6. Clarithromycin, 7.5 mg/kg PO BID
7. Azithromycin, 10 mg/kg QD on day 1, followed by 5 mg/kg QD on days 2 to 5

C. Adjunctive treatment. Adjunctive treatment for AOM includes acetaminophen or ibuprofen for fever and pain (ibuprofen has the advantage of greater analgesic effect and longer antipyretic effect) and topical antibiotics (if TM is perforated). Decongestants or decongestant/antihistamine combinations have not proved effective in preventing either the development of AOM during a viral illness or complications of AOM (chronic effusion).

D. Treatment. Treatment failures may be due to noncompliance with the antibiotic regimen (not given, spit out by the child, vomited), a resistant organism, or an adverse (real or imagined) reaction to the antibiotic prompting the caregiver to discontinue it. If a patient demonstrates evidence of continued acute infection (as outlined earlier) after 2 to 4 days despite first-line therapy, a second course of antibiotics is appropriate. Depending on the clinical setting, some consideration should be given to a tympanocentesis and culture.

VI. Resolution of Tympanic Membrane Abnormalities

Follow-up of AOM should occur 4 to 6 weeks after infection, at which time the clinical examination should have returned to normal. For uncomplicated AOM (first episode or significant interval since previous infection) with prompt resolution of all symptoms, no follow-up is necessary except at the next well-child visit. Early follow-up by telephone at 48 to 72 hours to determine response to the antibiotic has the effect of promoting the provider/patient bond, encouraging compliance with the therapeutic regimen, and assessing response to therapy. Defervescence and significant pain resolution should occur by this time.

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10 Acute, Subacute, and Chronic Sinusitis

Christine L. McHenry

Sinusitis is an inflammation of the mucosal lining of one or more of the paranasal sinuses. Sinusitis can be classified according to duration of symptoms as acute (<30 days), subacute (30 to 120 days), and chronic (>120 days). Obstruction of sinus ostia, impaired ciliary function, and/or overproduction or increased viscosity of secretions will lead to retention of secretions in the paranasal sinuses and, sometimes, to bacterial superinfection. Upper respiratory tract infections and allergies are the most common predisposing factors in both acute and chronic sinusitis. Clinically, most cases of sinusitis in children involve the maxillary and ethmoid sinuses, both of which are present and clinically significant at birth. The frontal and sphenoid sinuses are developed by 10 to 12 years of age. Isolated sphenoid sinusitis is rare. If sphenoid sinusitis is present, it is usually part of a pansinusitis picture.

I. Acute Sinusitis

A. Clinical presentation. Common symptoms of acute sinusitis in adolescents and adults include headache, fever, and facial pain; however, children may have fairly nonspecific complaints. Sinusitis should be suspected if (a) there is persistence of upper respiratory tract symptoms beyond 10 to 14 days without improvement, especially with persistent cough; (b) the child with an upper respiratory tract infection seems sicker than usual with high fever and purulent nasal drainage for at least 4 consecutive days; and/or (c) the allergic child has an acute exacerbation of respiratory symptoms, or the respiratory symptoms are difficult to control with usual management. Findings on the physical examination that would suggest sinusitis include mucopurulent discharge from the nose, erythematous nasal mucosa, otitis media, facial tenderness, and periorbital edema.

B. Infectious agents. Acute sinusitis may be a viral, bacterial, or mixed infection. The most common bacterial pathogens isolated are nontypable *Haemophilus influenzae, Streptococcus pneumoniae,* and *Moraxella catarrhalis.* Anaerobes are recovered about 10% of the time. Viral pathogens include rhinovirus, adenovirus, influenza, and parainfluenza.

C. Management. For uncomplicated, acute sinusitis in a child who has not had a recent course of antibiotics (<1 month), amoxicillin 60 to 90 mg/kg per day is an appropriate initial antibiotic, given amoxicillin's safety profile and the high spontaneous cure rate of acute sinusitis. If the child does not improve or worsens over the first 48 hours of antibiotics, changing antibiotics to a beta-lactamase-resistant drug, such as erythromycin/sulfisoxazole (Pediazole) 1.0 mL/kg per day divided TID/QID, amoxicillin/clavulanate (Augmen-

tin) 45/10 mg/kg per day plus amoxicillin 45 mg/kg per day divided BID, cefuroxime axetil (Ceftin) 30 mg/kg per day divided BID, or cefpodoxime proxetil (Vantin) 10 mg/kg per day divided BID may be beneficial. For those children who respond rapidly (within 3 to 4 days) to antibiotic treatment with dramatic improvement in symptoms, 10 days of antibiotic treatment is sufficient. For those with a slower response, antibiotics should be continued until the child is asymptomatic plus an additional 7 days. Topical decongestants and steroids may provide some symptomatic relief. To avoid rebound mucosal edema, topical decongestants should not be used longer than 5 days. Saline nasal washes also are helpful for removing intranasal crusts and possibly decreasing mucosal edema. Systemic decongestants and antihistamines generally are not recommended because of their drying effect and their negative effect on mucociliary clearance.

D. Complications. Complications of sinusitis include orbital cellulitis/abscess, osteomyelitis, epidural/subdural abscess, cavernous sinus thrombosis, meningitis, and brain abscess. Obviously, such complications require hospitalization with intravenous antibiotics and appropriate subspecialty consultation. Because of a higher incidence of intracranial complications with frontal and sphenoid sinusitis, initial management of these patients requires hospitalization.

E. Imaging. Plain sinus radiographs lack sensitivity and specificity, especially in the young child, and are not helpful in diagnosing acute sinusitis. CT scans should be reserved for those who have complicated disease, frequent recurrences, or protracted symptoms not responsive to therapy.

II. Subacute and Chronic Sinusitis

A. Clinical presentation. Children with subacute and chronic sinusitis may present with mucopurulent nasal discharge, nasal congestion (obstruction), cough (especially at night), sore throat, snoring, sleep disturbance, intermittent fevers, chronic headache (rare), and malodorous breath. Findings on physical examination may include mucopurulent nasal discharge, erythematous nasal mucosa, injected pharynx, and acute or serous otitis media.

B. Infectious agents. Nontypable *Hemophilus influenzae, Streptococcus pneumoniae,* and *Moraxella catarrhalis* are the most common bacterial agents recovered. Anaerobes, including *Bacteroides* species and fusobacteria, and aerobes such as alpha-hemolytic streptococci and *Staphylococcus aureus* appear to play a lesser role in subacute and chronic sinusitis in childhood.

C. Management. Amoxicillin/clavulanate is an ideal agent to cover most suspected pathogens. Reasonable alternatives include erythromycin/sulfisoxazole, cefuroxime axetil, and cefpodoxime proxetil. Antibiotics should be continued until the child is symptom-free plus an additional 7 days. Adjunctive topical therapy listed earlier also may be beneficial. If

the child does not respond to appropriate antibiotic management by 7 days, it is unlikely that infection is a major component of the persistent symptoms. Other etiologies for chronic nasal symptoms and/or cough such as reactive airway disease, allergic rhinitis, immunodeficiency, foreign body, nasal polyp, deviated nasal septum, and choanal atresia should be considered. If an anatomic problem is suspected, a CT scan is the imaging modality of choice.

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11 Conjunctivitis and Hordeolum

Raymond C. Baker

Conjunctivitis is a common eye condition in infants and children that is characterized by inflammation, discharge, and itch or foreign-body sensation. The most common causes of isolated conjunctivitis in children are infections and allergies, but in the infant or child presenting with a red eye, injury (including chemical), corneal abrasion, iritis/iridocyclitis, foreign body, and nasolacrimal duct obstruction need to be considered in the differential diagnosis.

INFECTIOUS CONJUNCTIVITIS

I. Neonatal Conjunctivitis

Neonatal conjunctivitis (ophthalmia neonatorum) is usually related to pathogens acquired during the infants's passage through the birth canal. The most common organism is *Chlamydia trachomatis; Neisseria gonorrhoeae* and herpes simplex virus are less common etiologies.

Conjunctivitis occurring in the first 3 weeks of life should be evaluated with a Gram stain and culture (Thayer-Martin medium for gonococci, viral/chlamydial tissue culture, and routine bacterial culture). When culturing for *Chlamydia,* the lower palpebral conjunctiva is swabbed and placed into transport medium. The nasopharynx should also be swabbed, and the swab should be placed in the same transport medium as the conjunctival swab, thereby saving the cost of one tissue culture. From a treatment perspective, it is not important whether the organism grows from the conjunctivae or nasopharynx because either should be treated systemically.

A. Chemical conjunctivitis. Chemical conjunctivitis is the most common etiology of neonatal conjunctivitis and manifests with hyperemia, watery discharge, and chemosis within hours of silver nitrate instillation in the neonate. It resolves spontaneously within 24 to 48 hours.

B. *Chlamydia trachomatis.* *Chlamydia trachomatis* conjunctivitis is the most common infectious etiology of neonatal conjunctivitis, usually developing within 5 to 14 days of birth. This infection requires systemic treatment with erythromycin estolate (Ilosone) 30 to 40 mg/kg per day PO divided TID/QID for 14 days to eliminate the organism from the primary site, as well as from the remainder of the respiratory tract.

C. Gonococcal conjunctivitis. Gram stain evidence of gram-negative, intracellular diplococci requires prompt treatment for presumptive *N. gonorrhoeae* infection. Because of the aggressive nature of gonococcal conjunctivitis, delay in treatment can result in corneal ulceration or even perforation. Isolated conjunctivitis should be treated with ceftriaxone 50 mg/kg (maximum 125 mg) intramuscularly as single-

dose therapy. Frequent saline irrigations are also needed for the accompanying copious discharge.

D. Nasolacrimal duct obstruction. Nasolacrimal duct obstruction (dacryostenosis) is a congenital condition in which the lower end of the nasolacrimal duct is obstructed by a membranous fold. The result clinically is a persistently wet eye and intermittent mucopurulent discharge of varying degrees in infants beginning shortly after birth. About 95% of cases resolve spontaneously by 12 months of age. If the condition has not resolved spontaneously by 1 year of age, referral to ophthalmology is necessary for possible probing. If recurrent mucopurulent discharge occurs (reflux from the obstructed duct or recurrent conjunctivitis), topical ophthalmic antibiotics are indicated. Resolution of the obstruction may be hastened by massaging the lacrimal sac, which parents can be taught to do at home. Parents should be shown how to place their fingertip in the corner of the eye at the inner canthus over the lacrimal sac and apply pressure in an up-and-down motion.

II. Conjunctivitis in Older Infants and Children

A. Common infectious. Most infectious conjunctivitis beyond the neonatal period is either bacterial (nontypable *Haemophilus influenzae* or *Streptococcus pneumoniae*) or viral (adenovirus). Treatment of conjunctivitis out of the neonatal period is often empiric because it is usually not possible clinically to distinguish viral from bacterial disease reliably. Because the infection is usually benign and responds to (or in spite of) topical antibiotic drops, most clinicians do not culture except in the neonate. They make the decision of whether to use topical antibiotics based on their clinical suspicion of bacterial disease. General guidelines that help one distinguish between bacterial and viral conjunctivitis and influence the decision to use topical antibiotics appear in Table 11.1.

Table 11.1. Guidelines to distinguish between bacterial and viral conjunctivitis

	Bacterial	Viral
Discharge type	Purulent	Seromucous
Discharge quantity	++/+++	+ ++
Concurrent otitis	*H. influenzae*	
Concurrent pharyngitis/ upper respiratory infection		*Adenovirus*
Season	Winter (*H. influenzae*)	Fall (*Adenovirus*)
Preauricular adenopathy	Uncommon	Common

If the decision is to use topical antibiotic drops, several factors will influence which of the many drops and ointments available is most appropriate. In general, ointments last a little longer, so that TID application is effective, but they tend to blur the vision and require more cooperation on the part of the child to apply. Drops require at least QID application to be effective, but are easier to apply to the uncooperative child (see later). Finally, some drops sting somewhat upon application (especially sulfacetamide drops) and are less useful in the younger child.

In the young, uncooperative patient, who typically resists by squeezing the eyes shut, the following technique can be used with ophthalmic drops: The parent sits on the floor with legs slightly abducted and extended, forming a "V." The infant is positioned supine on the floor with the upper extremities trapped beneath the parent's legs, child's head held by the parent's upper legs at the point of the "V." The drops are then placed at the inner canthus of the eye (which is usually tightly closed!). The parent then sets the eyedrop bottle down and with the thumb and index finger pulls the lids apart gently, allowing the drops to enter the eye.

B. Herpes simplex conjunctivitis. Herpesvirus conjunctivitis is seen less commonly than the other types of infectious conjunctivitis but is more serious and requires specific therapy. Vesicles in the periorbital area, if present, suggest the diagnosis. Otherwise the appearance is similar to other etiologies. A history of exposure may also be helpful. The clinical presentation is redness, severe pain, and the sensation of abrasion or foreign body—and is almost always unilateral. The hallmark of herpesvirus keratoconjunctivitis is the dendritic ulcer visible with fluorescein. An irregular and hazy appearance to the cornea may also be present. Treatment should be with ophthalmologic consultation and consists of trifluridine (Viroptic) or vidarabine (ViraA) drops. Steroid drops should never be used, as they worsen the dendritic lesions leading to an irreversibly opaque cornea.

III. Allergic Conjunctivitis

The typical presentation of allergic conjunctivitis is watery discharge, itchy eyes, tearing, and edema of the conjunctiva (chemosis) and lids. Symptoms are often seasonal with identifiable allergens (e.g., pollens, molds); and a history of other atopic disease (especially allergic rhinitis) is common. The disorder is most commonly bilateral, although unilateral disease can occur from inoculation with animal dander or hair. Effective treatment consists of topical decongestants along with an antihistamine (e.g., Visine or OcuHist ophthalmic solution). Cromolyn sodium 4% solution (Opticrom) also may be used prophylactically for severe allergic conjunctivitis. Oral antihistamines, especially the second-generation nonsedating products, may be used alone or in conjunction with topical therapy. Topical steroids are effective for allergic conjunctivitis, but their use should be with ophthalmologic

consultation (because of potential exacerbation of unrecognized herpetic conjunctivitis). Finally, topical nonsteroidal antiinflammatory drugs are available, but many sting upon application.

IV. Control Measures

Hand washing is the most important control measure to prevent the spread of infectious conjunctivitis. Some school systems require a physician's note before the child is allowed back in school, even though isolated conjunctivitis is not a medical reason for exclusion from school. In day care facilities, the concern of spread is greater, and some day care centers allow the child to return only after being seen by a physician.

V. Hordeolum (Stye)

A hordeolum, or stye, is an acute, bacterial (most commonly, *Staphylococcus aureus*) abscess of glands of the eyelid. Hordeola may be internal, on the mucosal side of the lid (Meibomian gland infection) or external, at the lid margin (sebaceous gland infection). Internal hordeola are larger and deeper in the lid. Both present as tender, red nodules of the eyelid. External hordeola are easily visible; internal require partial lid eversion to appreciate the inflammatory component. Internal hordeola may rupture, spilling their contents onto conjunctival surfaces and resulting in conjunctivitis. The treatment for both internal and external hordeola is the same—warm compresses and topical antibiotics QID until resolution.

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12 Pharyngitis and Tonsillitis

Paul S. Bellet

Pharyngitis, an inflammation of the mucous membranes and underlying structures of the throat, may be divided into two categories: illness with nasal involvement (nasopharyngitis) and illness without nasal involvement (pharyngitis, tonsillitis, or tonsillopharyngitis).

I. Nasopharyngitis

Viruses nearly always cause nasopharyngitis (adenoviruses, parainfluenza viruses, influenza viruses, and less commonly, rhinoviruses and respiratory syncytial virus). Nasopharyngitis also may occur in children with rotavirus gastroenteritis. The clinical findings in nasopharyngitis include rhinorrhea, nasal mucosal edema, mild pharyngeal erythema, and usually fever. This is an acute, self-limited illness lasting from 4 to 10 days. The treatment of nasal symptoms is the same as for the common cold. Symptomatic treatment for sore throat is discussed later.

II. Pharyngitis

Pharyngitis without nasal involvement is usually due to viruses (adenoviruses, parainfluenza viruses, influenza viruses, Epstein-Barr virus, enteroviruses) and bacteria (group A *Streptococcus*). Less common causes include herpes simplex virus, *Mycoplasma pneumoniae,* other streptococci (groups B, C, and G), anaerobes (*Bacteroides* species, *Fusobacterium* species, *Peptostreptococcus*), *Neisseria gonorrheae* (sexually active adolescents or exposed children and adolescents), and *Arcanobacterium haemolyticum.* The onset of illness is usually acute, with sore throat and fever. The pharynx and/or tonsils are inflamed with erythema, exudate, ulceration, and/or vesicles. The cervical lymph nodes may be enlarged and tender. Exudate on the pharynx and petechiae on the soft palate are seen most frequently with group A *Streptococcus* and infectious mononucleosis. Ulcerative and vesicular lesions are seen most frequently with enteroviral infections (herpangina) and herpes simplex virus.

III. Diagnosis

Children with nasopharyngitis, herpangina, or pharyngoconjunctival fever have a viral disease and do not require culture or antibiotic therapy. The child with acute pharyngitis and exudate suggests group A streptococcal infection, which is confirmed with a rapid streptococcal antigen test (with culture back-up, if negative). However, many children fall between these extremes, and a throat swab for a rapid streptococcal antigen test and/or throat culture should be performed, especially if there is a positive exposure history.

IV. Treatment

Effective treatment for group A streptococcal infection includes several options:

A. Penicillin V, 250 mg (<12 years of age), 500 mg (>12 years) PO BID for 10 days is the drug of choice for streptococcal pharyngitis.

B. Intramuscular benzathine penicillin G (600,000 U for children less than 60 pounds or 1,200,000 U for those more than 60 pounds) as a single injection is also appropriate therapy.

C. For young children who require liquid medications, a better-tasting alternative to penicillin V is amoxicillin; however, amoxicillin has no microbiologic advantage over penicillin V. Preliminary data suggest that oral amoxicillin given in a single daily dose for 10 days is as effective as penicillin V given TID for 10 days.

D. In patients allergic to penicillin, erythromycin estolate 20 to 40 mg/kg per day PO divided BID for 10 days (maximum dose, 1,000 mg/day). Alternative antimicrobials in penicillin-allergic patients are azithromycin for 5 days and clarithromycin for 10 days. However, these alternatives offer no advantage over erythromycin and are more expensive.

E. Symptomatic treatment of sore throat includes acetaminophen or ibuprofen orally plus cold liquids, popsicles, and ice cream in younger children. Older children and adolescents may find relief from gargling with warm saline solution.

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13 Common Oral Conditions in Infants and Children

James F. Steiner

Parents commonly bring their infants and children to the physician with problems and concerns about the mouth and teeth. The primary care physician needs a working knowledge of common oral conditions to recognize and to manage the problem, to reassure the parents if the condition is benign and self-limiting, or to make a pediatric dental referral. The following are examples of typical oral problems that the physician might see as part of routine primary care. They are predominantly clinical conditions that present during the child's first 24 months or situations that appear later but begin in this period.

I. Epstein's Pearls

Found in more than half of newborns, Epstein's pearls are white, raised, circular, 1-mm epithelial inclusion cysts in the midline of the hard palate. These resolve without treatment.

II. Tongue Tie

The lingual frenum, a band of tissue that courses from the ventral surface of the tongue to the floor of the mouth, may occasionally limit movement when the tongue is protruded. Treatment is rarely indicated, as this condition alone usually does not affect speech. Before referring for frenectomy, also known as tongue clipping, a speech pathologist should be consulted for recommendations.

III. Eruption Conditions

The first primary tooth erupts at about 6 months, and all 20 primary teeth are usually in place by 36 months of age.

A. Natal and neonatal teeth. Teeth present at birth are natal teeth, and those erupting during the first month are neonatal teeth. Eighty-five percent of both natal and neonatal teeth are the normal primary incisors and not supernumerary teeth. If they are quite loose, consideration should be given to removing them. An associated, rare condition, Riga-Fedes disease, is a traumatic ulcer on the ventral surface of the infant's tongue caused by a sharp spot on the natal or neonatal tooth. Pain from the ulcer may prevent feeding. When the sharp spot is smoothed, the ulcer will heal and feeding will resume. A pediatric dentist should be consulted if smoothing of the incisor is indicated.

B. Teething. Many symptoms are attributed to teething. Those that probably are associated with teething are drooling, irritability, and occasional low-grade fever. Fever over 101 should not be attributed to teething, and a source for the fever should be sought. Folk remedies associated with teething abound, but none offer consistent results. A cool, water-filled teething ring and oral acetaminophen often provide relief.

C. Eruption hematoma. As primary and permanent teeth move toward eruption, a blue, blood-filled swelling may

surround the emerging tooth. This so-called eruption hematoma usually requires no treatment as it ruptures and resolves when the tooth penetrates the gingiva.

D. Delayed eruption. When no teeth have erupted by 12 to 14 months in a well baby, the physician should refer the patient to a pediatric dentist to rule out missing teeth. If radiographs reveal teeth, continued watchful waiting is appropriate. Some conditions that may delay eruption are hypothyroidism; fibromatosis gingivae, a rare genetic condition in which dense gingivae prevent eruption; and dilantin and cyclosporine gingival hyperplasia.

E. Ectopic eruption. Ectopic tooth eruption is commonly seen when permanent teeth begin replacing primary teeth around 6 years of age. They present most frequently as a "double row of teeth" in the mandibular anterior area. The permanent incisors erupt behind the primary incisors. If the primary incisor is loose, parents should encourage the child to manipulate the loose tooth manually to encourage exfoliation. If the primary tooth is not loose, the patient should be referred to the pediatric dentist for removal of the primary incisor.

IV. Discoloration

Discoloration presents as extrinsic, easily removed surface stains or intrinsic, subsurface discoloration incorporated within the enamel and dentin of the tooth crown that cannot be removed.

A. Iron stain. A gray to black, extrinsic stain may be seen in infants on iron drops. This staining can be removed by the pediatric dentist. Changing to the chewable or tablet form of iron, if appropriate to age and ability, will prevent further staining.

B. Trauma. When primary teeth are concussed, the dental nerve, known as the pulp, may hemorrhage into the dentin, resulting in light to dark gray discoloration of the crown. A stronger force may sever the neurovascular bundle supplying the pulp and result in eventual pulp necrosis and a similar crown discoloration. Watchful waiting is appropriate. If pain, swelling, or a draining fistula through the gingiva develop, referral to a pediatric dentist is required.

C. Fluorosis. Mild fluorosis, a lacy, white, intrinsic color change observed in the enamel of permanent teeth, results from ingestion of greater-than-optimal amounts of fluoride during enamel formation. Young children who drink optimally fluoridated water or who, in nonfluoridated areas, are on systemic fluoride supplements are at risk for fluorosis if they swallow toothpaste while brushing. To reduce the risk of fluorosis, toothbrushing should be supervised by an adult until 5 or 6 years of age, and a pea-sized portion of paste should be placed on the brush. Fluoride toothpaste should not be used in children under 24 months of age.

D. Tetracycline. Tetracycline stain presents as a gray to brown to yellow intrinsic stain of permanent teeth caused by

systemic tetracycline prescribed during tooth formation, a process that begins early in the first year of life. The physician should defer long courses of systemic tetracycline until age 6 years. However, infrequent, short, 7-day courses before 6 years of age are not likely to cause staining.

V. Dental Caries

Dental caries is a preventable condition. Daily guardian-supervised brushing using a fluoride toothpaste and regular mealtimes plus two snacks per day will minimize the risk of caries. Sleeping with the nursing bottle or, if breast-fed, with the mother is high-risk behavior for early childhood caries and should be discouraged.

A. Early recognition. White, chalky lines in enamel near the gingiva in primary teeth represent early, reversible dental caries. If plaque has accumulated in this area, wipe it away and examine for the presence of chalky white lines suggestive of early caries. If chalky white lines are observed, the physician should suspect a bedtime bottle, nighttime breastfeeding, and frequent eating and drinking and should inquire about feeding practices. Parents should be reminded about twice-daily brushing to remove plaque.

B. Advanced caries. Advanced caries occurs as early as 12 months of age, when either high-risk feeding behaviors are not recognized by health care providers or oral health recommendations concerning low-caries-risk feeding are not complied with by the caretaker. Advanced caries results when the demineralized chalky white lines cavitate. It presents as tan-colored holes in the enamel surface. Referral to a pediatric dentist is indicated.

C. Toothache. When dental caries goes unrecognized or untreated, the pulp becomes infected and painful. Treatment with pain medication and emergent dental referral to a pediatric dentist is indicated. A complication of this process occurs when the pulpal infection breaks through into the surrounding gingiva, forming a dental abscess ("gum boil"), which likewise requires emergent referral to a pediatric dentist for treatment.

D. Facial cellulitis. Facial cellulitis results when pulpal infection spreads from the tooth, through surrounding bone, and into the facial tissues. The patient presents with facial swelling and may appear quite ill. Facial cellulitis secondary to an infected tooth should be treated with antibiotics (usually penicillin or amoxicillin alone) and pain medication and should be referred immediately to a pediatric dentist. Facial cellulitis of dental origin must be differentiated from primary bacteremic facial (buccal) cellulitis seen in infants and young children (3 months to 3 years of age), which requires different evaluation and management.

VI. Trauma

When infants begin the process of learning to walk, they are at risk for accidental oral trauma. The clinician must be alert

to physical abuse as part of the differential diagnosis, since oral injuries are seen in half of physically abused children. Principles of oral trauma management include evaluation and treatment as soon as possible.

A. Loose teeth. A loose tooth is the most frequent injury of the primary dentition. Patients with loose teeth should be referred to a pediatric dentist for evaluation and long-term follow-up.

B. Intruded teeth. The traumatic force pushes the tooth deeper into the tooth socket. In some instances, the tooth is pushed so deep that the crown is no longer visible. If the crown is not visible, the clinician must always rule out ingestion or aspiration. Dental referral is indicated.

C. Fractured teeth. Teeth that are fractured should be evaluated by a pediatric dentist within a day of the injury. Lip lacerations, frequently associated with crown fractures, may contain tooth fragments, which should be removed before suturing.

D. Avulsed teeth. Replacing avulsed primary teeth is generally not recommended. Avulsed permanent teeth, however, should be replaced as soon as possible, because the prognosis is best when the tooth is replaced quickly. The avulsed tooth should be held by the crown and any foreign material should be rinsed from the root. The tooth should then be returned to the empty socket. If the avulsed permanent tooth cannot be replaced, it should be transported to the dentist in milk. If the avulsed tooth cannot be found, aspiration, ingestion, or intrusion should be considered.

E. Mandibular fracture. Children with chin tip trauma are at risk for unilateral or bilateral subcondylar fracture. They may present with pain and swelling anterior to the tragus, pain on swallowing, and drooling onto the chin and chest because the child cannot swallow secondary to condylar pain. In unilateral subcondylar fracture, the mandible will deviate to the side of the fracture. If both condyles are fractured, an anterior open bite is often seen. A panoramic dental film and Towns view of the mandible are helpful in confirming the diagnosis.

Mandibular body fractures are always accompanied by a hematoma in the floor of the mouth. Frequently seen are vertical tears in the gingiva and, upon palpation of the inferior border of the mandible, a bony step defect. Emergent referral to an oral surgeon is recommended.

VII. Acute Herpetic Gingivostomatitis

Acute herpetic gingivostomatitis is seen predominantly in children under 6 years of age and is caused by Herpes simplex virus (*Herpes hominis,* type I). The initial symptoms are fever, fretfulness, cervical lymphadenopathy, and refusal to eat. Several days later, the gingiva becomes red and painful. Mucosal surface vesicles develop on and around the lips, the gingiva, the tongue, and the hard palate. The vesicles ulcerate and form lesions 1 to

3 mm in diameter that may coalesce to form larger ulcers. Therapy is symptomatic, with cold liquids, ice cream, cold pudding, popsicles, and so on, and acetaminophen or ibuprofen. Children with gingivostomatitis may occasionally have severe enough pain that they are unable to keep themselves hydrated orally, thus requiring other methods of hydration. The ulcerations usually resolve within 1 or 2 weeks. Acute herpetic gingivostomatitis is contagious, so careful hygiene measures and isolation are recommended.

VII. Smokeless Tobacco Use

Children and adolescents may use smokeless tobacco products that cause irritation of oral soft tissues and white, leukoplakia lesions. When the patient's history elicits use of these products, the oral tissues should be examined for the presence of white, soft-tissue leukoplakia. If leukoplakia is observed, the patient and guardian should be counseled on discontinuing the habit; and the patient should return in 6 weeks. If tobacco use is discontinued, the white spot will resolve. All patients should be counseled on the health effects of smokeless tobacco and the benefits of cessation.

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Web Sites

http://www.aapd.org (American Academy of Pediatric Dentistry web site)

14 Upper Respiratory Tract Infection (the Common Cold)

Paul S. Bellet

An upper respiratory tract infection (the common cold) is an acute, communicable viral infection. Viruses that usually cause the common cold include rhinoviruses, parainfluenza viruses, respiratory syncytial virus, and coronaviruses. Other agents that occasionally cause the common cold include adenoviruses, enteroviruses, influenza viruses, reoviruses, and *Mycoplasma pneumoniae.* Children have an average of three to eight colds per year.

I. Diagnosis

The common cold is characterized by nasal stuffiness, sneezing, coryza, throat irritation, and low-grade or no fever. Other manifestations include cough, malaise, headache, muscle ache, vomiting, and diarrhea. The usual duration of the illness is about a week, but cough and nasal discharge may persist for 2 weeks or more. The physical examination reveals inflamed, swollen turbinates with clear or mucopurulent discharge. The diagnosis is clinical, but a specific diagnosis can be made by virus isolation from nasal secretions (nasal wash technique or nasopharyngeal swab). The primary disorder to consider in the differential diagnosis is allergic rhinitis (seasonal or perennial). The most common complications are otitis media, sinusitis, and pneumonia.

II. Treatment

No therapy is necessary in most cases of the common cold in children. Symptomatic care can be considered in the individual case, when needed. Relief of nasal obstruction is most important in infants, so they can feed and sleep more comfortably. Isotonic saline drops and gentle nasal aspiration usually are effective. In children and infants older than 6 months of age, oral decongestants, such as pseudoephedrine (Sudafed), may be used. Topical decongestant nose drops and sprays can be useful, but persistent use for more than 3 to 5 days may lead to rebound obstruction. In infants 6 to 24 months of age, phenylephrine (Neosynephrine) 0.125% nose drops may be used as often as every 4 to 6 hours but are recommended only at bedtime and before naps to help sleep. In children 2 to 6 years of age, xylometazoline (Otrivin) pediatric nose drops 0.05% may be used every 8 to 12 hours as a longer-acting alternative to phenylephrine. In children older than 6 years, oxymetazoline adult nose drops or spray 0.05% or xylometazoline 0.1% may be used every 8 to 12 hours. Antihistamines have no place in the routine therapy of the common cold, although they are included in many over-the-counter cold medicines. If allergic rhinitis is possible, an oral decongestant/antihistamine combination can be used. Examples of over-the-counter decongestant/antihistamine combinations are Novahistine, Triaminic, and Actifed. Cough suppressants are often included in over-the-counter cold medications, but their effectiveness in children has not been well

documented, and they are contraindicated in infants less than 12 months of age due to possible respiratory depression.

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IV

Cardiorespiratory Disorders

15 Asthma

Christine L. McHenry

Asthma affects 4% to 7% of American children and is responsible for more days of missed school and of restricted activity than any other chronic childhood disease. The number of children affected and the severity of the disease have increased over the last years. Risk factors for asthma include male gender, African-American and Hispanic-American races, environmental factors (crowding, poverty, air pollution, tobacco smoke), prematurity, and a positive family history of allergy. Asthma is a chronic inflammatory disease of the airways that also involves airway hyperresponsiveness and complete or partial airway obstruction. When a child is exposed to an environmental trigger, an increase in mucous production and airway inflammation is followed by airway obstruction that is responsible for clinical symptoms, including dyspnea and wheezing. An asthma exacerbation has two phases. The **first phase** involves the release of mediators from bronchial mast cells, macrophages, and eosinophils that produce immediate smooth muscle contraction along with inflammation and recruitment of granulocytes. These granulocytes (basophils, neutrophils, and eosinophils) mediate the **second phase** by secreting inflammatory mediators 4 to 12 hours after the initial environmental exposure, causing airway edema, mucous secretion, disruption of the integrity of the respiratory epithelium, and increased bronchial hyper responsiveness.

I. History

In assessing a patient with asthma, current symptoms and medications, precipitating factors, past medical history, and family history are important areas to explore. In addition, the number of days of missed school may indicate either the severity of the disease and/or how well the disease is controlled. Finally, the impact of the patient's disease on the family should be evaluated.

A. Current symptoms and medications. Describe the onset of symptoms, including cough, wheezing, dyspnea, chest tightness, and/or pain; list the medications used, including doses and frequency, most recent dose, and recent systemic steroid use.

B. Precipitating factors. Respiratory tract infection, tobacco smoke, changes in the weather, exercise, emotions, drugs (e.g., aspirin, NSAIDs) or foods, animals, seasonal and perennial allergies.

C. Past medical history. Emergency room visits and hospitalizations for asthma, including intensive care unit admissions; history of bronchiolitis, bronchopulmonary dysplasia, or gastroesophageal reflux; history of recurrent otitis media, chronic or seasonal rhinitis, or sinusitis.

D. Family history. Asthma, allergies, eczema, hay fever, chronic sinusitis.

II. Physical Examination

The physical examination begins with a general assessment of the patient; for example, is the patient in severe respiratory distress, using accessory muscles to breathe and dyspneic when talking, or is he or she, when sitting comfortably on the examination table, able to complete full sentences without becoming short of breath? The chest examination begins with observation for increased anteroposterior (AP) diameter, accessory muscle use, flaring of the alae nasi, and skin color. On auscultation, one should listen for the character (normal, decreased) of the breath sounds, wheeze (expiratory, inspiratory, both), inspiratory to expiratory ratio (I:E ratio), crackles, and focality of findings. If the patient is old enough to cooperate, a peak flow measurement compared with the standard table or the patient's own best peak flow when not symptomatic is a much better indicator of airway obstruction than are physical findings, especially in mild to moderate obstruction. Other evidence of allergy and/or infection, such as eczema, rhinitis, or sinusitis as well as vital signs, also should be noted.

III. Outpatient Management

A. Crisis management. Mild to moderate exacerbations of asthma often can be managed at home or in the physician's office. Parents and patients should be instructed as part of the routine care of the child in how to assess the severity of airway obstruction, ideally including some quantitative measurement, such as peak flow (PF) if the child is old enough. The physician should provide the patient or parent with some general guidelines regarding when to call the physician and when to go directly to the emergency room for acute care.

1. *Mild exacerbations.* With a mild exacerbation (PF 70% to 90% of baseline, minimal accessory muscle use, mild dyspnea, normal speech, good color, expiratory wheeze only) the patient should begin at home with a short-acting inhaled beta-2 agonist such as albuterol (Ventolin, Proventil) delivered by a meter dose inhaler or nebulizer, if so equipped. This can be given every 15 to 20 minutes up to three doses. If this fails, then the physician should be contacted to determine the next step.

2. *Moderate exacerbations.* In moderate exacerbations (PF 50% to 70% of baseline, moderate accessory muscle use, moderate dyspnea, speaks in phrases, color good to pale, inspiratory and expiratory wheezing) the patient should be given a single inhaled treatment of a short-acting beta-2 agonist, and the physician should be contacted to determine where the patient should be seen. Treatment in the office or the emergency room should include nebulized albuterol every 10 to 15 minutes until improved, oxygen to keep the oxygen saturation above 93%, and systemic prednisone at 2 mg/kg. If the patient has a good response that is sustained for 1 hour after the last nebulized treatment, he or she may be discharged to go home on a short-acting inhaled beta-2 agonist every 4 hours and systemic steroids with

close follow-up. If the patient does not have a sustained response after the above treatment, then albuterol treatments should be continued every 15 to 20 minutes and ipratropium bromide (Atrovent) added. Depending on the subsequent response, the patient may require hospitalization.

3. *Severe exacerbations.* In severe exacerbations (PF < 50% of baseline, accessory muscle use, nasal flaring, severe dyspnea, single-word speech, pale color to cyanotic, diminished to inaudible breath sounds) the patient should be given one or more short-acting inhaled beta-2-agonist treatments and immediately transported to the emergency department for further care.

B. Chronic asthma management. Education is a vital component of the outpatient management of chronic asthma. The patient and family should know what the patient's asthma triggers are and avoid those triggers as much as possible. Older patients should know the proper technique for a PF measurement as well as when to step up therapy using the PF as a guide. In addition, patients should be instructed in inhaler technique to ensure proper delivery of medication. A daily diary of symptoms and response to treatment will be useful in modifying therapy.

1. *Mild intermittent asthma.* Mild intermittent asthma is defined as symptoms no more than twice per week and nighttime symptoms no more than twice per month with normal PF between exacerbations. Exacerbations last a few hours to a few days. A short-acting inhaled beta-2 agonist such as albuterol should be used for acute symptoms.

2. *Mild persistent asthma.* Mild persistent asthma is defined as symptoms more than twice a week that may last for days and that affect activity. Nighttime symptoms occur more than twice per month. Daily medication for long-term control should be used and should include mast cell inhibitors, such as cromolyn sodium (Intal) TID/QID and nedocrimil (Tilade) BID/QID. Alternatively, low-dose inhaled steroids [beclomethasone dipropionate (Beclovent, Vanceril), budesonide (Pulmicort), fluticasone propionate (Flovent), flunisolide (Aerobid), triamcinalone acetonide (Azmacort) BID/QID], depending on the steroid, could be used. Other medications to consider include methylxanthines, such as sustained-release theophylline (Slobid, Theodur), and leukotriene modifiers, such as montelukast sodium (Singulair). Singulair is the only leukotriene modifier approved for children 2 years of age and older. A short-acting beta-2 agonist such as albuterol may be used as required.

3. *Moderate persistent asthma.* Moderate persistent asthma is defined as daily symptoms with two or more exacerbations per week that affect activity. Nighttime symptoms occur two or more times per week. Therapy consists of medium-dose inhaled steroids *or* low- to medium-dose inhaled steroids plus a long-acting inhaled beta-2 agonist

such as salmeterol (Serevent) or a methylxanthine, a leukotriene modifier, or cromolyn sodium. A short-acting inhaled beta-2 agonist may be used as required.

4. *Severe persistent asthma.* Severe persistent asthma is defined as continual symptoms with frequent exacerbations, frequent nighttime symptoms, and limited physical activity. Therapy consists of high-dose inhaled steroids plus a long-acting inhaled beta-2 agonist or a methylxanthine or a leukotriene modifier or a mast cell stabilizer. Daily ipratropium bromide and daily or every-other-day systemic steroids should be considered.

C. Exercise-induced asthma. Approximately 35% to 40% of patients with allergic rhinitis and up to 90% of patients with asthma will have bronchoconstriction with exercise. Exercised-induced asthma usually is easily controlled by using a short-acting inhaled beta-2 agonist such as albuterol (two puffs) or a mast cell stabilizer such as cromolyn sodium (two puffs) before exercise, followed by use as required. If the patient still wheezes with the preceding regime, then four puffs of albuterol or cromolyn sodium, or two puffs of each can be used before exercise.

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Web Site

http://www.ama-assn.org/special/asthma/treatmnt/guide/guidelin/guidelin.htm (Expert Panel Report II: Guidelines for the Diagnosis and Management of Asthma)

16 Bronchiolitis

Robert M. Siegel

Bronchiolitis is an acute, febrile, clinical syndrome of infants and young children associated with a lower respiratory tract infection and characterized by cough and wheeze.

I. Etiology and Pathophysiology

The most common cause of bronchiolitis is respiratory syncytial virus (RSV). Other pathogens associated with the disease are parainfluenza and influenza viruses, rhinovirus, adenovirus, and *Mycoplasma pneumoniae.* Bronchiolitis occurs most often during the winter months in infants under 12 months of age (80%). The natural course of the disease is an upper respiratory tract infection with fever and rhinorrhea that may progress over 2 to 5 days to lower tract involvement with wheezing and some degree of respiratory distress accompanied by tachypnea, nasal flaring, and retractions. In RSV infection the virus invades the epithelial cells of the respiratory tract, moving from upper tract to lower by cell-to-cell transfer. The result is necrosis of the epithelium with subsequent sloughing and obstruction of the small airways. This leads to the characteristic findings of wheezing and hyperinflation.

II. Clinical Course

Two-thirds of children with bronchiolitis will have fever. Physical examination often reveals an irritable infant with tachypnea and tachycardia. Wheezing is the hallmark of the disease. Affected infants may be unable to take adequate oral fluids and become dehydrated. Other complications of bronchiolitis are hypoxemia, respiratory failure, apnea, and bacterial superinfection. Apnea can be a presentation in infants less than 6 months of age. Some of the factors associated with more severe disease include a history of prematurity, young chronologic age, chronic lung disease (especially bronchopulmonary dysplasia), and congenital heart disease. Ten to 20% of infants with bronchiolitis will develop significant respiratory compromise, necessitating admission to the hospital. The mortality for bronchiolitis caused by RSV is 0.5% to 1.5% for hospitalized patients (as high as 37% for infants with congenital heart disease).

III. Evaluation

The outpatient evaluation should begin with a thorough history and physical examination, since bronchiolitis is, for the most part, a clinical diagnosis. A chest x-ray (CXR) is not routinely recommended but should be considered in select cases to rule out other causes of wheezing in this age group, such as foreign-body aspiration, congenital anomalies, or bacterial pneumonia. The CXR will usually show hyperinflation and peribronchial thickening. The presence of atelectasis may indicate a more severe course. Pulse oximetry determination of oxygen saturation may reveal hypoxemia, which is common, even in the absence of clinically detectable cyanosis. If the infant is extremely tachypneic or in moderate respiratory distress, a capillary blood gas

should also be drawn to rule out carbon dioxide retention, a sign of respiratory failure. A complete blood count (CBC) is generally not helpful.

IV. Diagnosis

Although bronchiolitis is a clinical diagnosis, a specific etiology can be determined by obtaining a nasal wash from the child for culture and RSV antigen.

V. Treatment

Treatment for most children is largely supportive. Oxygen should be given to those with hypoxia and intravenous fluids to those with inadequate oral intake. The decision to admit a child with bronchiolitis is complex and multifactorial. It should be based on (a) general appearance (whether a child looks toxic or appears happy and playful), (b) age (infants less than 3 months of age or with a history of prematurity with gestational age of less than 34 weeks are at greater risk for apnea and should be closely observed in the hospital), (c) tachypnea (an infant consistently breathing more than 60 to 70 times/minute is likely to tire and needs monitoring), (d) oral intake (if an infant is unable to take adequate oral fluids because of tachypnea or respiratory distress, IV hydration is necessary), and (e) hypoxemia. (Resting oxygen saturation of less than 95% indicates hypoxemia; saturation is likely to decrease with crying or sucking, and such infants should be admitted with supplemental oxygen.)

The efficacy of bronchodilator therapy in bronchiolitis is questionable and depends on the degree of bronchospasm that is present. Some infants with a positive family history for atopic disease may have a significant degree of bronchospasm present, and the current illness represents the first episode of reactive airways disease secondary to lower respiratory tract infection. This cannot be distinguished clinically from bronchiolitis. Other infants have a predominantly viral lower respiratory tract infection component to the disease with little to no bronchospasm present. The latter commonly have significant crackles present on physical examination. Beta-agonist aerosols may be tried and continued if there is improvement with the therapy. Finally, aerosolized racemic epinephrine may have some value, and a trial may be considered in select patients.

Ribavirin is a broad-spectrum antiviral agent that has been used for the treatment of RSV infection in more than 100,000 patients. The drug proved effective in some early studies, but more recent trials question its efficacy. Because ribavirin is expensive and many experts question its efficacy, its use should be limited to children at high risk for severe disease and should be decided clinically on a case-by-case basis. The drug is administered by a small particle generator and may be given over 12 hours per day.

VI. Prevention

RSV has a high potential for spread in the day care and hospital settings. Since the infection is spread from person to per-

son by large droplets of respiratory secretions, the mainstays of prevention are good hand washing and protective masks. The hospitalized patient should be put in contact isolation. The use of goggles and gloves may also help prevent the spread of this agent. Recently, passive immunoprophylaxis with monoclonal antibodies has become available. This is available in an intravenous form, RSV-IGIV, and as an intramuscular preparation, palivizumab. Both preparations have been approved for use in children under 2 years of age for the prevention of RSV disease and are given on a monthly basis during RSV season. Unfortunately, both preparations are extremely expensive and are recommended only to a select group of patients. The AAP recommendations for the use of palivizumab and RSV-IGIV are summarized in Table 16.1.

Table 16.1. Summary of AAP recommendations for palivizumab and RSV-IGIV use

1. Consider prophylaxis in children younger than 24 months of age with chronic lung disease (formerly bronchopulmonary dysplasia) or those who have required therapy for CLD within 6 months before the onset of RSV season.
2. Consider prophylaxis in infants without CLD born at 32 weeks gestation or earlier. Risk factors to consider are gestational age and chronologic age at the time of onset of RSV season.
3. Consider prophylaxis in infants of 32 to 35 weeks gestation with risk factors that may predispose to respiratory complications, such as neurologic disease, multiple siblings, day care center attendance, smoke exposure, and anticipated cardiac surgery.
4. Palivizumab and RSV-IGIV are not approved for use in patients with congenital heart disease, and data suggest that RSV-IGIV should should not be used in those with cyanotic heart disease. Those who meet criteria 1 or 2 and have asymptomatic acyanotic heart disease may benefit by prophylaxis.
5. There are no specific recommendations for immune-compromised children. Those with severe compromise may benefit from prophylaxis.
6. RSV prophylaxis should begin at the beginning of RSV season and terminate at the end of RSV season.
7. The primary means to prevent RSV infection in the hospital setting remains strict observance of infection control practices.
8. If RSV-IGIV is used, MMR and varicella vaccines should be deferred for 9 months after the last dose. Palivizumab, however, does not interfere with immunization response.

Adapted from American Academy of Pediatrics Committee on Infectious Diseases and Committee on Fetus and Newborn. Prevention of respiratory syncytial virus infections: indications for the use of palivizumab and update on the use of RSV-IGIV. *Pediatrics* 1998;102:1211–1216.

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17 Cardiac Murmurs: Functional and Pathologic

Thomas R. Kimball

World-renowned auscultatory pioneer Peter Latham stated, *"Men are as often deceived by their ears as by their eyes, and they may hear ghosts as well as see them. The ear must be a well-educated and well-practiced ear, or it is not a trustworthy witness."* This warning is particularly relevant to today's primary care physician who was trained during an era of robust developments in cardiovascular technology when auscultatory training was less emphasized but who now must rely more heavily on auscultatory findings because of technologic rationing in an era of managed health care. Therefore distinguishing functional or innocent murmurs from pathologic ones and investigating for the presence or absence of cardiovascular disease in children is a pressing issue for today's health care provider.

Adding to the problem is that the diagnostic standard has shifted over time. In the nineteenth century, pathologic examination was the only means to determine if a murmur was innocent. As more sensitive techniques were developed (first catheterization, then echocardiography), the significance of auscultatory findings has changed. For example, the systolic click of mitral valve prolapse was initially felt to be innocent until echocardiography demonstrated its origin.

The prevalence of murmurs in children is very high (up to 90% in some series). However, only 2% to 7% of murmurs are pathologic, so that the primary care physician has the difficult task of selecting the very few patients with significant disease from a very large number of normal children.

INNOCENT MURMURS

Innocent murmurs may be organized by their timing within the cardiac cycle (i.e., systole, diastole, and continuous) as in Table 17.1.

I. Systolic Murmurs

A. Still's murmur. Still's murmur occurs most frequently in late-preschool- and early-school-aged children. It is a low-pitched, systolic ejection (crescendo/decrescendo, diamond-shaped) murmur usually of I to III/VI intensity. Its quality is described as buzzing, vibratory, or musical. It is best heard between the lower sternal border and the apex with limited radiation. The murmur characteristically diminishes with standing. It may be mistaken for the murmurs of a small ventricular septal defect or left ventricular outflow tract obstruction in hypertrophic cardiomyopathy. However, these pathologic murmurs are usually of higher intensity and do not decrease with standing.

B. Innocent pulmonary flow murmur. Innocent pulmonary flow murmur represents 15% of all innocent murmurs. It is a medium-pitched, systolic ejection murmur of I to III/VI

Table 17.1. Typical characteristics of innocent (functional) heart murmurs

	Pitch	Timing	Intensity	Quality	Location	Diminished by:	DDx
Systolic							
Still's murmur	L	Ejec	I–III	Vib	LLSB	Stand	VSD, HOCM
Pulmonary flow murmur	M	Ejec	I–III	Blow	LUSB	Stand, Insp	ASD, PS
Supraclavicular bruit	H	Ejec	I–III	Harsh	RSCA	Shoulder move	AS
Pulmonary branch murmur	M–H	Ejec	I–III	Blow	LUSB	Low output	PS
Split first heart sound	L	NA	I	Vib	LLSB	NA	S4, AS, PS, MVP
Diastolic							
Third heart sound	L	Mid	I	Vib	LSB	Sitting	MS, rumble
Fourth heart sound	L	Late	I	Vib	LSB	NA	Rumble
Continuous							
Venous hum	M–H	Con	I–III	Blow	RUSB	Head move	PDA, AVM
Mammary souffle	M	Con	I–III	Blow	RUSB	Supine	PDA, AVM

Abbreviations: AVM, arteriovenous malformation; AS, aortic stenosis; ASD, atrial septal defect; Con, throughout the cardiac cycle; Ejec, ejection; H, high; HOCM, hypertrophic obstructive cardiomyopathy; Insp, inspiration; L, low; LLSB, left lower sternal border; LSB, left sternal border; LUSB, left upper sternal border; M, medium; MS, mitral stenosis; MVP, mitral valve prolapse; NA, not applicable; PDA, patent ductus arteriosus; PS, pulmonic stenosis; RSCA, right supraclavicular area; Vib, vibratory; VSD, ventricular septal defect.

intensity and is distinguished by its blowing quality. It is best heard at the left upper sternal border with radiation to the axillae and diminishes with standing or inspiration. This murmur may be confused with the murmurs of an atrial septal defect and pulmonary stenosis.

C. Supraclavicular bruit. Supraclavicular bruit is due to turbulence at the origins of the brachiocephalic vessels and is most common in middle childhood. It is a high-pitched, systolic ejection murmur of I to III/VI intensity, harsher in quality than the pulmonary flow murmur. It is best heard in the right supraclavicular fossa and radiates into the neck and is attenuated by shoulder hyperextension (i.e., raise chin and throw back shoulders). The supraclavicular bruit must be distinguished from aortic stenosis.

D. Pulmonary branch murmur. Pulmonary branch murmur is caused by turbulence produced at the relatively acute angle of the bifurcation of the pulmonary artery and is most common in neonates, particularly premature babies. It usually disappears by 1 to 8 weeks of age. It is a medium- to high-pitched systolic ejection murmur of I and III/VI intensity, blowing and squirting in quality. It is best heard at the left upper sternal border and radiates into the axillae and back. It is increased by conditions that raise cardiac output. The pulmonary branch murmur may be confused with valvar and true branch pulmonary stenosis.

E. Split first heart sound. Split first heart sound can be heard at the lower left sternal border and the apical area. It is most common in late childhood and adolescence and is due to the slight difference in timing between mitral and tricuspid closures. A split first sound may be confused with a fourth heart sound (S4).

II. Diastolic

Murmurs occurring in diastole are rarely innocent. In the newborn, a closing patent ductus arteriosus seldom results in a transient diastolic murmur. Although a third or fourth heart sound is usually a sign of pathology, each sound may be a normal diastolic event. However, a diastolic sound or murmur should most often be considered abnormal.

III. Continuous

A. Venous hum. Venous hum is most often heard in the infraclavicular area or along the right sternal border. It is of medium to high pitch and of I to III/VI intensity. The venous hum is usually continuous and has the quality of a blowing murmur or, sometimes, a machinery murmur. Most important, its intensity is diminished by firm stethoscope pressure or head rotation. The differential diagnosis includes a patent ductus arteriosus (PDA), cervical arteriovenous malformation, or goiter bruit.

B. Mammary souffle. Mammary souffle is a continuous murmur of medium intensity and pitch. It is best heard along the right upper sternal border. Supine position or applying jugular pressure diminishes the murmur. It is common in

adolescent or young women who are pregnant or lactating and is felt to be due to turbulent flow through the large arteries or vein to/from the breasts. The differential diagnosis includes a PDA or a cervical arteriovenous malformation.

THE PEDIATRIC CARDIOVASCULAR EXAMINATION

A careful and deliberate physical examination will help narrow the differential diagnosis of the patient with suspected cardiovascular disease.

I. Vital Signs

The **vital signs** are important for assessing general cardiovascular well-being. The neonatal patient has a limited ability to increase cardiac output by increasing contractility or preload or by decreasing afterload, so increasing the heart rate is the primary means of increasing cardiac output. Therefore an elevated pulse may be indicative of hemodynamic derangement. Tachypnea can be a sign of left ventricular failure or pulmonary overcirculation.

II. Blood Pressure

The blood pressure should *always* be taken in both arms and in at least one leg. A higher blood pressure in the right arm (compared with the leg or left arm) is usually diagnostic for coarctation of the aorta. In patients with the relatively uncommon lesion of supravalvar aortic stenosis, the streaming effect of the stenotic jet into the right brachiocephalic artery can also cause right upper extremity hypertension.

III. General Appearance

Certain syndromes are evident on **general appearance** that may indicate underlying cardiovascular disease. Trisomy 21 (Down syndrome) has a high association with atrioventricular septal defects. Williams syndrome patients may have supravalvar aortic stenosis and/or hypoplasia of other arteries. Turner syndrome has a high association with coarctation of the aorta. Patients with Noonan syndrome may have valvar pulmonary stenosis, hypertrophic cardiomyopathy, or partial atrioventricular septal defect with left ventricular outflow tract obstruction. Marfan syndrome can be associated with aortic dilatation, aortic insufficiency, aortic dissection, and mitral valve prolapse.

IV. Cyanosis

Cyanosis is less easily recognized in the newborn because of the presence of fetal hemoglobin, which has a high affinity for oxygen. In addition, central cyanosis must be differentiated from peripheral or acrocyanosis, which is a normal finding in the newborn because immaturity of the neuromuscular control of the peripheral venous system leads to venous pooling. This is evident clinically as a bluish discoloration of the hands and feet and the skin around the mouth. Central cyanosis is evident clinically in the nailbeds, conjunctivae, and mucous membranes.

V. Respiratory

The respiratory pattern of hyperpnea is often a sign of hypoxemia and may be found in cyanotic heart disease. Retractions, nasal flaring, and grunting are indicative of respiratory distress that may be due to left ventricular failure or pulmonary overcirculation. Rales and crackles are often not evident in the newborn with congestive heart failure and are more likely due to primary pulmonary disease.

VI. Cardiac Examination

The cardiac examination should begin by palpation of the **precordial impulse.** A right ventricular heave, possibly evident over the sternum, indicates right ventricular overload. A left ventricular thrust, evident in the left axilla, indicates left ventricular overload. Increased precordial activity is often the only cardiovascular sign, even in the presence of significant heart disease (e.g., hypoplastic left heart syndrome).

The **second heart sound** is of particular importance. Normally, the second heart sound splits into two audible components (aortic and pulmonic) during inspiration because of delayed pulmonary valve closure due to the acute increase in right ventricular volume. A *single* second heart sound is indicative of the absence of one semilunar valve (i.e., aortic or pulmonary atresia) or, in some instances, transposition of the great vessels. Persistent splitting of the second heart sound throughout the respiratory cycle is the diagnostic and usually the only finding of an atrial septal defect. (In a postoperative cardiac patient, persistent splitting of the second heart sound is often due to right bundle branch block). A particularly loud second heart sound is usually indicative of pulmonary hypertension and is particularly important to characterize in patients with large left-to-right shunts.

Clicks are usually systolic in timing. An **early** systolic click is best heard toward the apex and is due to the opening of a stenotic pulmonic or aortic valve. A **mid-systolic** click is usually due to mitral valve prolapse. This click becomes more prominent and occurs earlier when the patient assumes an upright position or performs other maneuvers resulting in reduced left ventricular volume.

Gallop rhythms can be normal in children. However, they may also be due to abnormalities during ventricular diastole—either during rapid ventricular filling (third heart sound) or atrial contraction (fourth heart sound).

The various murmurs have already been discussed. However, the examination should focus on **six main aspects of the murmur:**

A. Location

1. *The apex,* where problems of the mitral valve are usually heard.
2. *The left lower sternal border,* where problems associated with the tricuspid valve and ventricular septum are best heard.

3. *The left upper sternal border*, where problems of the pulmonic valve are appreciated.
4. *The right upper sternal border*, where problems of the aortic valve are heard.

B. Timing
C. Intensity
D. Radiation pattern

There are **three radiation patterns** of interest. Radiation of a murmur into the neck is usually due to aortic stenosis caused by the stenotic jet coursing into the ascending aorta and the carotid arteries. Radiation of a murmur to the axillae and/or back is usually due to pulmonary stenosis caused by the posterior course of the main pulmonary artery and the peripheral courses of the branch pulmonary arteries. Radiation of a murmur into the left axilla alone is usually indicative of mitral insufficiency due to the insufficiency of the jet coursing into the left atrium.

E. Quality
F. Changes elicited by physical examination maneuvers

The murmur should be localized to one of the **four auscultatory areas:**

VII. Abdominal Examination

Hepatosplenomegaly can be a sign of right heart failure or back failure from left ventricular dysfunction.

VIII. Extremities

Examination of the extremities can be very enlightening. The strength of the *pulses* can be a sign of general cardiovascular well-being and cardiac output. Particularly prominent pulses may be indicative of lesions producing diastolic runoff (e.g., aortic insufficiency, arteriovenous malformation, patent ductus arteriosus, surgical systemic-to-pulmonary arterial shunt). Diffusely diminished pulses are indicative of poor left ventricular output due to left ventricular dysfunction or obstruction to left ventricular outflow (e.g., hypoplastic left heart, critical aortic stenosis). Differential pulses with those in the upper extremities being stronger than those in the lower extremities are indicative of coarctation of the aorta. Alternatively, coarctation may present with a delay in the pulse in the lower extremities.

The **perfusion** of a patient is an index of general cardiac output and/or degree of systemic vascular resistance and can be assessed by judging capillary refill time in the toes or fingers or the general warmth of the extremities.

Clubbing of the nailbeds is a sign of chronic cyanosis and is usually seen in older patients with unrepaired congenital heart disease (e.g., tetralogy of Fallot, double outlet right ventricle with pulmonary stenosis, atrioventricular septal defect with Eisenmenger syndrome), primary pulmonary hypertension, or repaired or palliated lesions with residual problems (e.g., single ventricle with pulmonary artery band).

IX. Bruits

The presence of **bruits** should be sought in the head, in the abdomen, and in the neck and can be indicative of arteriovenous malformations.

THE PEDIATRIC CARDIOVASCULAR LABORATORY WORK-UP

I. Oxygen Saturation

The oxygen saturation allows definitive differentiation of peripheral acrocyanosis from central cyanosis due to hypoxemia. In addition, the response of the oxygen saturation to inspired supplemental oxygen can help differentiate hypoxemia due to primary pulmonary disease (saturation will increase) and that due to right-to-left intracardiac shunting (little change in saturation). Finally, simultaneous saturation measurements in the right upper extremity and the lower extremities can help diagnose a right-to-left ductal shunt (right upper extremity saturation > lower extremity saturation).

II. Electrocardiogram

The electrocardiogram is helpful in determining the presence of **atrial enlargement** or **ventricular hypertrophy.** In addition, the presence of left axis deviation is usually very specific for two particular lesions—atrioventricular septal defect or tricuspid atresia with normally related great vessels.

III. Chest x-ray

The chest x-ray is helpful in determining **overall cardiac and individual chamber sizes.** Right atrial enlargement is usually evident on the posteroanterior (PA) projection with a rightward bulge of the cardiac silhouette. Right ventricular enlargement is evident on the lateral projection with filling of the retrosternal space and on the PA projection as an upturned apex. Main pulmonary artery dilatation is evident on the PA projection as a slight bulge along the left border of the cardiac silhouette. Left atrial dilatation is evident on the lateral projection as a posterior bulge. Left ventricular dilatation is evident on the PA projection as an inferior, leftward appearance of the cardiac apex. Aortic dilatation is evident on the PA projection as prominence of the right portion of the cardiac silhouette.

The degree of **pulmonary vascularity** can be assessed as normal, increased, or decreased. Thoracoabdominal situs can be determined. The combinations of abdominal situs inversus with levocardia (or vice versa—abdominal situs solitus with dextrocardia) are usually associated with complex congenital heart disease.

A **right-sided aortic arch** can be determined by chest x-ray and, if found, is usually associated with one of only two conditions—tetralogy of Fallot or truncus arteriosus. These two conditions can usually be distinguished on the chest x-ray by examining pulmonary vascularity. (Tetralogy of Fallot usually has decreased vascularity whereas truncus arteriosus usually has increased vascularity.)

IV. Echocardiogram

The definitive diagnostic laboratory test is the echocardiogram, which is noninvasive, portable, and painless. In children less than 4 years of age, it usually requires sedation since the patient must lie still for approximately 45 minutes. The echocardiogram consists of performing ultrasound from four windows (along the left parasternal area, at the cardiac apex, below the xiphoid process, and in the suprasternal notch). Usually, a complete delineation of cardiac anatomy can be obtained. The only significant limitations of the test are visualizing the branch pulmonary arteries, their more distal branches, and the distal coronary arteries.

Often an echocardiogram cannot be ordered without first obtaining a cardiac consultation. However, increasingly primary care physicians are gaining unrestricted access to the echocardiography laboratory. Such a privilege necessitates responsible use. An echocardiogram is usually indicated for most pathologic murmurs (although some murmurs [e.g., those due to a small ventricular septal defect (VSD)] are so diagnostic that an echocardiogram is often unnecessary). Clues that a murmur may be pathologic are outlined in Table 17.2.

An echocardiogram may also be indicated to distinguish an innocent murmur from a pathologic murmur. In this instance, the echocardiogram can definitively establish the diagnosis, even if the heart is structurally normal, thereby relieving parental anxiety. There are some conditions in which an echocardiogram is often indicated even in the absence of a murmur (e.g., a neonate with trisomy 21). Other clinical scenarios in which an echocardiogram is indicated are outlined in Table 17.3. In general, an echocardiogram should be considered in populations where clinical accuracy is lower and incidence of disease is higher (e.g., congenital syndromes or younger patients [<4 years of age]. Potentially low-yield conditions in which an echocardiogram is likely to be negative are (a) suspicion of endocarditis, (b) evaluation of nonexertional chest pain, and (c) evaluation of syncope.

Table 17.2. Clues that a murmur may be pathologic

Historical	**Physical Examination**
Genetic syndrome	Pulse or blood pressure discrepancy
Neonatal age	Loud murmur
Failure to thrive	Diastolic murmur
	Abnormal second heart sound
Laboratory	Presence of a click or gallop
Abnormal chest x-ray	Poor perfusion, hepatosplenomegaly, or tachypnea
Abnormal electrocardiogram	
Decreased oxygen saturation	Central cyanosis

Table 17.3. Clues to narrow the differential diagnosis of congenital heart disease

Clinical Finding	Suggested Diagnosis
Left axis deviation on electrocardiogram	Atrioventricular septal defect or tricuspid atresia with normally related great vessels
Trisomy 21 (Down syndrome)	Atrioventricular septal defect
Turner syndrome	Coarctation of the aorta
William syndrome	Supravalvar aortic (and/or pulmonic) stenosis
Noonan syndrome	Pulmonary stenosis or hypertrophic cardiomyopathy
Right arm systolic blood pressure greater than lower-extremity blood pressure	Coarctation on the aorta with closing (closed) posterior descending artery (PDA)
Right upper-extremity oxygen saturation greater than lower-extremity oxygen saturation	Coarctation of the aorta, interrupted aortic arch, persistent pulmonary hypertension of the newborn
Multiple clicks	Ebstein's anomaly
Supraventricular tachycardia	Ebstein's anomaly, congenitally corrected transposition of the great vessels (L-TGV)
Complete heart block	Congenitally corrected transposition of the great vessels (L-TGV)
Right aortic arch on chest x-ray	Tetralogy of Fallot (if normal or decreased pulmonary blood flow) or truncus arteriosus (if normal or increased pulmonary blood flow)

ARRIVING AT A DIFFERENTIAL DIAGNOSIS

The final process in delineating a differential diagnosis is developing a scheme of congenital heart disease to facilitate organization of positive historical, physical, and laboratory data into one of the broad areas of disease, as in Table 17.4. There are two main types of congenital heart disease—acyanotic and cyanotic. The acyanotic diseases consist of shunt lesions (e.g., VSD) and obstructive lesions (e.g., aortic stenosis). Cyanosis can be due to two main physiologic phenomena: (a) decreased pulmonary blood flow (e.g., tetralogy of Fallot) or (b) mixing of systemic and pulmonary venous blood (e.g., truncus arteriosus).

Table 17.4. Schema for congenital heart disease classification

Acyanotic Heart Disease

Shunt lesions
- Atrial septal defect
- Atrioventricular septal defect
- Ventricular septal defect
- Aortopulmonary window
- Patent ductus arteriosus

Obstructive lesions
- Pulmonary valve stenosis
- Aortic valve stenosis
- Coarctation of the aorta
- Hypoplastic left heart

Cyanotic Heart Disease

Decreased pulmonary blood flow
- Pulmonary atresia/intact ventricular septum (hypoplastic right heart)
- Ebstein's anomaly
- Tricuspic atresia
- Tetralogy of Fallot

Increased pulmonary blood flow or mixing lesions
- Transposition of the great vessels
- Truncus arteriosus
- Total anomalous pulmonary venous return
- Single-ventricle and other complex disease

OVERVIEW OF CONGENITAL HEART DISEASE

I. Normal

Deoxygenated blood from the systemic circulation returns to the heart via the superior vena cava (SVC) and inferior vena cava (IVC). The blood flows through the tricuspid valve into the right ventricle (RV). It then courses across the pulmonary valve into the main pulmonary artery (MPA), which branches into the right and left pulmonary arteries (RPA, LPA) and into the lungs to become oxygenated.

The oxygenated pulmonary venous return enters the left atrium (LA) through the pulmonary veins. The blood flows through the mitral valve into the left ventricle (LV). It then courses across the aortic valve into the aorta (Ao) and out to deliver oxygen to the body (Fig. 17.1).

II. Atrial Septal Defect

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries into the lungs. Oxygenated blood passes into the LA. From here the oxygenated blood can pass across the atrial septal defect as a left-to-right shunt into the RA and then to the RV, MPA, and lungs, causing dilatation of these structures. Alternatively, the blood may pass from the LA through the mitral valve, LV, Ao, and body (Fig. 17.2).

III. Ventricular Septal Defect

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries into the lungs.

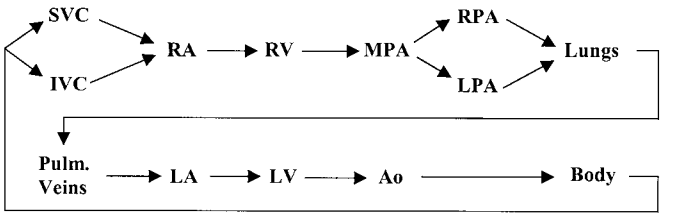

Fig. 17.1. Normal circulation.

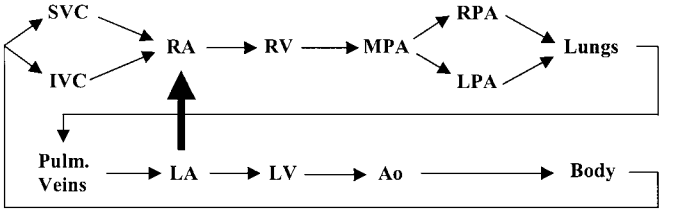

Fig. 17.2. Atrial septal defect.

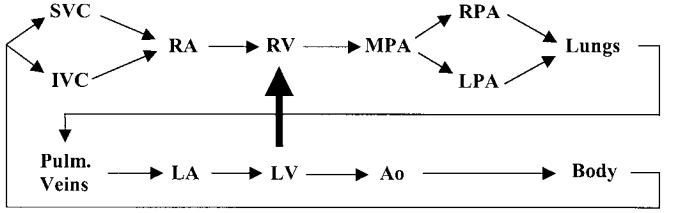

Fig. 17.3. Ventricular septal defect.

Oxygenated blood passes into the LA and LV. From here the blood can pass across the ventricular septal defect as a left-to-right shunt into the RV and MPA and lungs. Since this event occurs during systole, the LV ejects the blood into the MPA through the RV. LV and LA dilatation ensue. Alternatively, blood in the LV can course normally through the Ao into the body (Fig. 17.3).

IV. Atrioventricular Septal Defect

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries into the lungs. Oxygenated blood passes into the LA. Left-to-right shunting may occur from LA to RA, from LV to RV, or from LV to RA. Sometimes

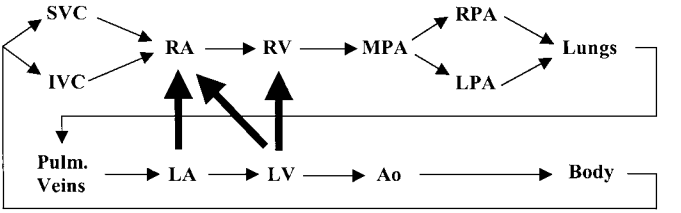

Fig. 17.4. Atrioventricular septal defect.

pulmonary artery pressures may be high enough that a right-to-left shunt can occur from RA to LA or from RV to LV. Blood may also pass normally through the LV into the Ao to the body (Fig. 17.4).

V. Patent Ductus Arteriosus or Aortopulmonary Window

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries into the lungs. Oxygenated blood passes into the LA, LV, and Ao. Some oxygenated blood can flow from the Ao into the MPA through the PDA (or AP window) as a left-to-right shunt, eventually flowing back through the LA and LV and causing dilatation of those chambers. Blood may also flow normally through the Ao to the body (Fig. 17.5).

VI. Hypoplastic Left Heart

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries into the lungs. Oxygenated blood passes into the LA. The mitral valve is at the least stenotic and is frequently atretic so that there is a left-to-right atrial shunt of oxygenated blood from the LA to the RA, which then courses to the RV and MPA, causing RA, RV, and MPA dilatation. There must be a right-to-left shunt through a PDA to supply blood to the body (Fig. 17.6).

VII. Tetralogy of Fallot

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries into the lungs. However, depending on the degree of right ventricular outflow

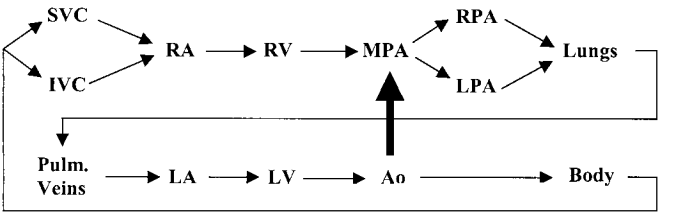

Fig. 17.5. Patent ductus arteriosus.

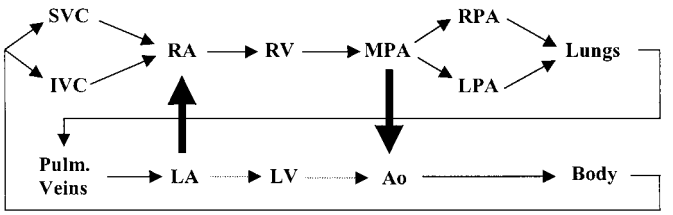

Fig. 17.6. Hypoplastic left heart.

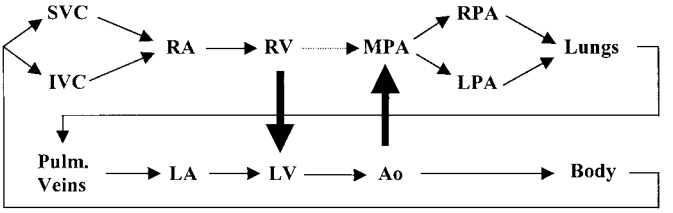

Fig. 17.7. Tetralogy of Fallot.

obstruction (subvalvar, valvar, and/or supravalvar), there will be right-to-left ventricular shunting of deoxygenated blood through the VSD. Oxygenated blood passes into the LA, LV, and Ao. If right ventricular outflow obstruction is severe enough that antegrade pulmonary blood flow is very diminished, there may be a need for a PDA to provide pulmonary blood flow via a left-to-right ductal shunt (Fig. 17.7).

VIII. Pulmonary Atresia With Intact Ventricular Septal Defect (Hypoplastic Right Heart)

Deoxygenated blood passes from the SVC and IVC to the RA and RV. However, since there is no easy egress from the RV, it becomes hypertensive (usually suprasystemic in pressure). Blood usually exits the RV as severe tricuspid insufficiency into the RA. Alternatively, a coronary/cameral fistula may develop between the body of the RV and the coronary arteries so that blood flows retrograde from the RV through the coronary arteries, exiting out the coronary ostia into the Ao.

From the RA, most blood avoids the high-pressure RV and courses across an ASD to the LA as a right-to-left shunt. From here the blood flows into the LV and Ao. To supply blood to the lungs, a PDA is necessary so that there is left-to-right shunting from Ao to MPA (Fig. 17.8).

IX. Tricuspid Atresia/Normally Related Great Vessels

Deoxygenated blood passes from the SVC and IVC to the RA. Since the tricuspid valve is atretic, the only egress from the

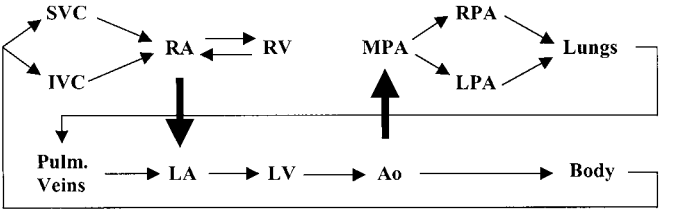

Fig. 17.8. Hypoplastic right heart.

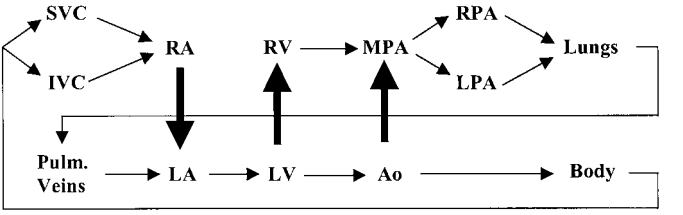

Fig. 17.9. Tricuspic atresia.

RA is through an ASD, which is required to supply blood to both the body and the lungs. Blood flows normally into the LV. Depending on the size of the associated VSD, there will be some blood flow from LV to RV as a left-to-right shunt. Therefore the size of the VSD also dictates the degree of associated pulmonary stenosis. Some blood may course antegrade from the LV to the RV and MPA, but the patient may be in need of a PDA to supply pulmonary blood flow if the VSD is small and the pulmonary stenosis is severe (Fig. 17.9).

X. Tricuspid Atresia/Transposition of the Great Vessels

Deoxygenated blood passes from the SVC and IVC to the RA. Since the tricuspid valve is atretic, the only egress from the RA is through an ASD, which is required to supply blood to both the body and the lungs. Blood flows normally into the LV. Depending on the size of the associated VSD, there will be some blood flow from LV to RV as a left-to-right shunt. Therefore the size of the VSD also dictates the degree of associated aortic valve stenosis and arch hypoplasia. Some blood may course antegrade from the LV to the RV and Ao, but the patient may be in need of a PDA to supply blood flow to the body if the VSD is small and the aorta is hypoplastic (Fig. 17.10).

XI. Transposition of the Great Vessels

Deoxygenated blood enters the RA through the SVC and IVC. The blood courses through the RV and, because the great ves-

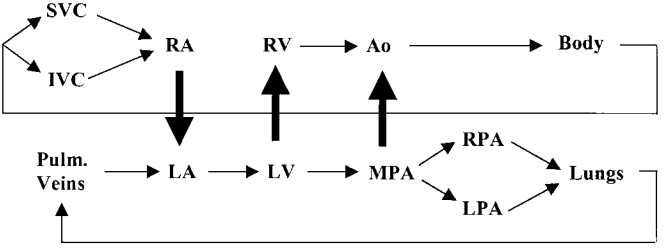

Fig. 17.10. Tricuspid atresia/transposition of the great vessels.

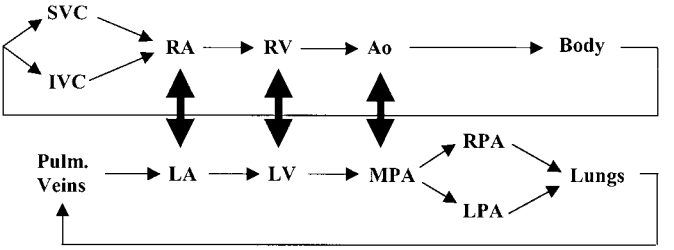

Fig. 17.11. Transposition of the great vessels.

sels are transposed, into the Ao to the body. This blood returns to the SVC and IVC, where the same circuit of blood flow is repeated.

Oxygenated blood enters the LA through the pulmonary veins. The blood courses through the LV into the pulmonary artery. The blood returns to the pulmonary veins, where the same circuit of blood flow is repeated.

The two circulations exist in parallel rather than in series. There must be at least one communication between the circulations if there is survival. This communication may exist as an ASD, a VSD, and/or a PDA (Fig. 17.11).

XII. Ebstein's Anomaly

Deoxygenated blood returns to the RA via the IVC and SVC. Blood may flow antegrade through the Ebsteinoid tricuspid valve into the RV and MPA. But because there is usually tricuspid insufficiency or right ventricular outflow obstruction from the displaced tricuspid anterior leaflet or RV hypoplasia, blood can also course across an ASD as a right-to-left shunt into the LA. From here blood flows into the LV and Ao. Depending on the degree of antegrade blood flow through the right heart into the MPA, the patient may require a PDA for left-to-right shunting to augment pulmonary blood flow (Fig. 17.12).

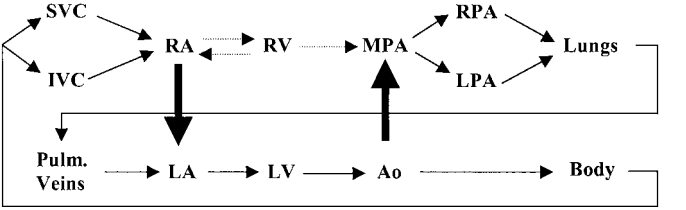

Fig. 17.12. Ebstein's anomaly.

XIII. Truncus Arteriosus

Deoxygenated blood passes from the SVC and IVC to the RA and RV. Since the truncus overrides the ventricular septum through a VSD, blood exits the RV through the truncus. In the truncus, blood may flow through the aortic arch to supply the body. Alternatively, blood may enter the pulmonary arteries from the truncus to supply blood to the lungs. Oxygenated blood flows normally back to the LA (Fig. 17.13).

XIV. Total Anomalous Pulmonary Venous Return

Deoxygenated blood passes from the SVC and IVC to the RA, RV, MPA, and branch pulmonary arteries. Oxygenated blood enters the pulmonary veins, which drain anomalously to either the hepatic and portal venous systems inferior to the diaphragm, to the IVC, to the RA, to the SVC, or to the coronary sinus. RA, RV, and MPA dilatation ensues. An ASD is necessary to ensure adequate perfusion through the left heart to the body (Fig. 17.14).

XV. Single Ventricle

Deoxygenated blood returns to the RA via the SVC and IVC. At the atrial level there is frequently mixing of oxygenated and deoxygenated blood if one of the atrioventricular valves is atretic or hypoplastic. Blood then enters the single ventricle (which

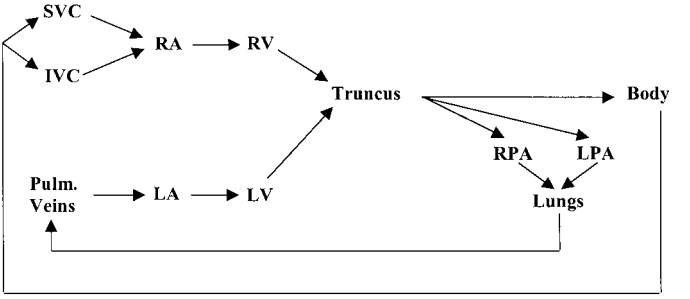

Fig. 17.13. Truncus arteriosus.

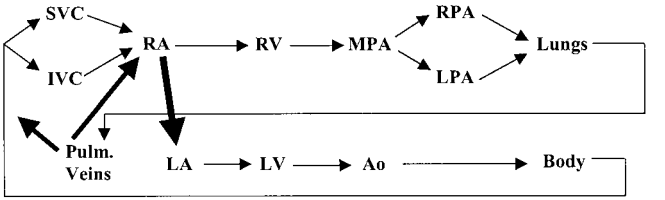

Fig. 17.14. Total anomalous pulmonary venous return.

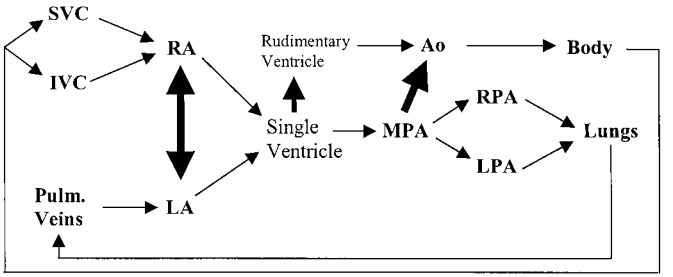

Fig. 17.15. Single ventricle.

is usually of LV morphology) through one or both of the atrioventricular valves. There is almost always a rudimentary ventricular chamber that usually sits leftward and anterior to the main ventricular chamber to which it is connected by a VSD (also called the *bulboventricular foramen*). The size of the VSD dictates the size of the great vessel (usually the aorta), which arises from the rudimentary chamber. So some blood will course across the VSD into the rudimentary chamber and out into the aorta. However, most blood will exit the single ventricle via the MPA, especially if the VSD is small. In these instances, there may be associated aortic stenosis, aortic arch hypoplasia, and coarctation of the aorta. Frequently a PDA is necessary in these circumstances to maintain adequate perfusion to the body. Oxygenated blood returns via the pulmonary veins to the LA (Fig. 17.15).

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(University of Kansas Medical Center)
http://www.rchc.rush.edu (Rush Children's Heart Center Home Page)

Web Sites for Families

http://sln.fi.edu/biosci/biosci.html (The Heart: An Online Exploration)
http://www.tchin.org (Children's Health Information Network)
http://www.csun.edu/hfmth006/chaser
(CHASER [Congenital Heart Anomalies, Support, Education and Resources])

18 Croup: Acute Viral Laryngotracheobronchitis

Raymond C. Baker

I. Description

Croup (acute viral laryngotracheobronchitis) is an acute lower respiratory tract infection caused primarily by parainfluenza viruses types 1, 2, and 3. Other less common viral etiologies are respiratory syncytial virus, adenovirus, and influenza viruses. It is seen more commonly in young children from 6 months to 3 years of age during the fall and winter months of the year. Croup begins with viral upper respiratory infection (URI) symptoms of fever, runny nose, and cough in a nontoxic-appearing child. These symptoms are followed in 1 to 4 days by hoarseness; a resounding, seal-like, barking cough; inspiratory stridor; intercostal retractions; and, in unusually severe cases, hypoxia progressing to cyanosis.

The laboratory is rarely helpful in the diagnosis; the diagnosis is made almost exclusively on clinical grounds. Airway films reveal subglottic stenosis on anteroposterior (AP) view (steeple sign) but are not usually necessary to make the diagnosis.

Viral croup must be differentiated from two other serious illnesses that may present similarly—**epiglottitis** and **bacterial croup** (also called *membranous croup* or *bacterial tracheitis*). Features that distinguish epiglottitis are age (usually older—in the 3 to 7 years of age range), the absence of cough as a significant symptom, the presence of a severe sore throat, very acute onset with septic appearance and rapid progression, and a muffled voice. Respiratory distress is a sign of advanced disease that requires immediate airway management. As this disease has a predominantly *Haemophilus influenzae* type b etiology, it is seldom seen since the advent of universal immunization with conjugate *H. influenzae* vaccine.

Bacterial croup probably begins as a viral, crouplike illness with similar symptoms, but bacterial superinfection of the laryngotracheal mucous membranes develops, causing acute exacerbation of symptoms. The child develops increasing toxicity and respiratory distress as the airway becomes occluded with a pseudomembrane of pus and nonviable tissue. The most common superinfecting bacterial organism is *Staphylococcus aureus;* other, less common organisms are *Haemophilus influenzae,* streptococci, and *Neisseria* species.

II. Treatment

The treatment of mild croup is largely supportive. A cool mist humidifier that has been advocated in the past probably offers little help other than giving parents something to do. Many practitioners also instruct parents to allow the child to breathe cold air "to break the attack," but this has not been studied for efficacy and safety. Oral antipyretics are effective in reducing significant fever, if present, which is desirable to decrease oxygen consumption and decrease pulmonary effort. The child should be

Table 18.1. Croup score

	0	1	2	3
Stridor	None	Only with agitation	Mild at rest	Severe at rest
Retractions	None	Mild	Moderate	Severe
Color	None			Cyanotic
Level of consciousness	Normal	Restless when disturbed	Restless when undisturbed	Lethargic
Key to Croup Score				
Total score	≤ 4	5–6	7–8	≥ 9
Severity	Mild	Mild to moderate	Moderate	Severe

Adapted from Fleisher GR, Ludwig S, eds. *Textbook of pediatric emergency medicine*, 3rd ed. Baltimore: Williams & Wilkins, 1993.

closely observed for progression of symptoms that might require hospitalization.

In more severe croup, hospitalization or treatment in an emergency setting may be necessary for closer observation, oxygen, and other therapies directed at reducing airway edema if symptoms progress. Racemic epinephrine by aerosol is effective in rapidly reducing airway edema and decreasing symptoms, but the effects are short-lived (<2 hours), requiring an additional therapy for longer effect. Corticosteroids given with the racemic epinephrine provide longer-term airway edema reduction. Dexamethasone, given either intramuscularly or orally as a single dose of 0.15 to 0.6 mg/kg, is the preferred steroid. This combination has proved effective in reducing symptoms and the need for additional racemic epinephrine aerosols, hospitalization, intensive care unit management, and intubation. Steroids (budesonide, 2 mg) given by aerosol are also effective but cost more than either oral or parenteral dexamethasone.

Determining the severity of croup and the need for aggressive therapy and hospitalization is almost entirely clinical. Factors that influence this decision include (a) age less than 12 months, (b) a second unplanned visit to the physician or emergency room, (c) questionable compliance or follow-up, (d) greater distance to the hospital, and (e) presence of any of the following: somnolence, poor oral intake, cyanosis, stridor at rest, dehydration from refusal of oral liquids, and croup score over 7 (Table 18.1).

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19 Community-Acquired Pneumonia

Raymond C. Baker

Pneumonia in infants and children has multiple infectious etiologies, but because there is no simple and reliable way of establishing a specific etiology, the clinician must make treatment decisions based on age, clinical markers, associated findings, and, sometimes, nonspecific laboratory tests, such as the white blood cell count. The chest x-ray, although the gold standard for making the diagnosis (in combination with clinical findings), seldom pinpoints a specific etiology. Viruses are the most common etiology at almost any age, with respiratory syncytial virus (RSV) most common, and parainfluenza virus, rhinovirus, adenovirus, and influenza virus predominating. Bacterial pneumonia is a less common cause of community-acquired pneumonia but usually causes more significant illness. Bacterial etiologies in immunocompetent infants and children include *Streptococcus pneumoniae* (most common), *Haemophilus influenzae* type b (rare since the introduction of the *H. influenzae* vaccine), *Staphylococcus aureus, Streptococcus pyogenes,* group B streptococcus (newborn), gram-negatives (newborn), *Bordetella pertussis,* and *Mycobacterium tuberculosis.* Miscellaneous infectious agents include *Mycoplasma pneumoniae* (common) and *Chlamydia* (*trachomatis* and *pneumoniae*). Less common infectious agents include various fungal and parasitic organisms, including *Pneumocystis carinii.*

I. Symptoms

The symptoms of pneumonia vary from fever alone to a combination of symptoms that might include fever, chills, cough, vomiting, abdominal pain, pleuritic chest pain, decreased activity, and decreased appetite.

II. Physical Examination

On physical examination, fever is usually present in association with any combination of tachypnea, grunting respirations, retractions, crackles, wheeze, decreased or bronchial breath sounds, and dullness to percussion. Compared with adults, children may show little to suggest pneumonia on physical examination other than fever and tachypnea. The presence of fever and abdominal pain, with a normal abdominal examination, is sometimes suggestive of lower lobe pneumonia in the young child.

III. Laboratory

The laboratory is often not helpful in establishing the diagnosis and etiology, with the exception of culture when a specimen is available (e.g., pleural fluid, blood culture). Laboratory work-up may include a complete blood count (CBC), blood culture, and chest x-ray. The degree of illness may dictate other laboratory parameters to determine acuity, such as arterial blood gases and/or oxygen saturation. When a specific etiology is needed (e.g., immunosuppression, failure of first-line therapy, chronic or recurrent pneumonia), more aggressive laboratory

methods may be used, such as culture of fluid obtained by thoracentesis, serologic testing, immunofluorescence studies, or polymerase chain reaction (PCR).

IV. Radiology

Chest x-ray is the gold standard for the diagnosis of pneumonia. However, radiologic findings often lag behind clinical findings, especially in the presence of dehydration. Many clinicians make a diagnosis of "clinical pneumonia" on the basis of history and crackles on examination and treat without obtaining a chest x-ray. The chest x-ray may be helpful in establishing a cause but often does not add significantly to the management (contrary to what many radiologists may declare). However, there are some clinical settings in which the chest x-ray should be obtained in a patient with clinical pneumonia: (a) first-time wheezing episode associated with clinical findings of pneumonia, (b) immunosuppressed patient, (c) chronic or recurrent pneumonia, (d) suspected pleural effusion or pneumothorax, (e) concurrent cardiac disease, (f) septic-appearing infant/child, (g) suspected congenital anomaly or foreign body predisposing to pneumonia, or (h) suspected staphylococcal pneumonia.

V. Etiology

The combination of age, history, examination, and CBC with differential and chest x-ray of the patient may sometimes suggest a cause of the pneumonia that is helpful for directing therapy:

A. Bacterial. Children with bacterial pneumonia usually have an abrupt onset of symptoms, appear toxic with tachypnea and high fever, have an elevated white blood cell count, and demonstrate chest x-ray findings of unilobar disease, alveolar infiltrate, effusion, or pneumatoceles. The latter is especially associated with *Staphylococcus aureus.* Other clues to bacterial pneumonia are neonatal age range (group B *Streptococcus* and gram-negatives) and underlying immunosuppression or debilitation (*Staphylococcus aureus,* nonpathogenic, saprophytic organisms).

B. Viral. Infants and children with pneumonia of viral origin tend to have a more gradual onset, appear less ill, wheeze on clinical examination, and have multilobar disease, pneumonitis, and/or hyperinflation on chest x-ray.

C. Mycoplasma. In the older child and adolescent, headache, sore throat, wheeze, and a history of other family members with a similar illness suggests *Mycoplasma pneumoniae* ("walking pneumonia").

D. *Chlamydia.* Chlamydial pneumonia (*Chlamydia trachomatis*) is seen in the young infant who is afebrile, is mildly to moderately ill, and has a staccato cough. There may be conjunctivitis on examination (or a history of conjunctivitis). The chest examination (crackles) usually sounds worse than the child appears; the chest x-ray is hyperinflated, with a diffuse, reticulonodular appearance. The CBC may show eosinophilia. *Chlamydia pneumoniae* is a relatively common

cause of pneumonia in preschoolers, children of school age, and adolescents. The disease is not distinguishable from viral pneumonia and tends to be a milder disease. It commonly presents as fever, malaise, sore throat, and cough. In known asthmatics, chlamydial pneumonia commonly causes exacerbation of asthma symptoms.

E. Pertussis. Paroxysms of cough with color change and the presence of inspiratory stridor (whoop) are characteristic of pertussis. The white blood cell count characteristically is elevated with a pronounced lymphocytosis.

VI. Admission to Hospital

Several clinical situations might suggest a need for hospitalization:

A. Children who are vomiting significantly and would be unable to take oral medications may require a brief period of parenteral therapy.

B. Young infants with a presumed bacterial etiology usually require parenteral antibiotics.

C. Young infants with probable pertussis should be hospitalized initially because of the significant complications in this age range.

D. Very young infants in whom sepsis cannot be ruled out should be hospitalized. Clinically and immunocompromised children are usually hospitalized pending cultures.

E. Infants and children with chronic or recurrent pneumonias may require hospitalization for evaluation.

F. Other indications for hospitalization might include septic-appearing infants, a progressive course, pyogenic complications, significant pleural effusion, and significant respiratory distress or impending respiratory failure.

VII. Treatment

Because the specific etiology of pneumonia is usually unknown at the initial encounter and prompt antibiotic therapy can be crucial in some clinical settings, antibiotics are commonly prescribed empirically in patients with clinical pneumonia. The choice of antibiotics depends on the most likely, treatable etiology based on the epidemiologic and clinical factors outlined earlier. Some general guidelines for antibiotic therapy follow.

A. Outpatient

1. *No antibiotic therapy may be considered in the infant with typical bronchiolitis* during a period of RSV prevalence.
2. *In older infants and children with mild to moderate symptoms in whom bacterial disease is unlikely*, oral erythromycin estolate for 10 days is reasonable empiric therapy. This provides adequate coverage for *Mycoplasma* and *Chlamydia* as treatable causes of pneumonia, as well as the occasional pneumococcal pneumonia that was unsuspected based on clinical grounds. The other macrolides, clarithromycin and azithromycin, are alternatives but offer no particular advantage over erythromycin other than frequency of dosage.

3. *In older infants and children with suspected bacterial pneumonia*, amoxicillin provides adequate coverage of pneumococcal pneumonia (in the higher dosage range to cover organisms with intermediate resistance) but is not adequate for *Mycoplasma* or *Chlamydia* or most other bacterial etiologies, particularly beta-lactamase-producing organisms. Some clinicians prefer to initiate therapy with a single injection of a parenteral antibiotic such as procaine penicillin or ceftriaxone, especially if the patient is vomiting. First- or second-generation cephalosporins are alternatives in suspected bacterial pneumonia but have little to no activity against *Mycoplasma* or *Chlamydia.*

B. Inpatient

1. *Under 3 months of age.* In this age range, broader coverage is desirable to cover gram-negatives and group B streptococcus. A reasonable combination of antibiotics initially might include IV ampicillin and gentamicin (under 4 to 6 weeks of age) or a third generation cephalosporin (6 to 12 weeks); intravenous nafcillin may be substituted for ampicillin if *S. aureus* is suspected. Oral erythromycin may be added or substituted if *Chlamydia* or pertussis is suspected.
2. *Three to 6 months of age.* Supportive therapy plus intravenous ampicillin alone. Oral erythromycin may be substituted or added if pertussis is suspected. Cefuroxime has broader coverage and should be used if *Haemophilus* or *S. aureus* is suspected or if the infant appears septic.
3. *Six months to 6 years of age.* Ampicillin or cefuroxime alone. Oral erythromycin may be added if pertussis, *Mycoplasma,* or *Chlamydia* is suspected; cefuroxime, if *Haemophilus* or *S. aureus* is suspected.
4. *More than 6 years.* Penicillin G, ampicillin, or cefuroxime alone. Oral erythromycin may be added if *Mycoplasma* is suspected.

C. Other therapy. Bronchodilators may also be indicated, especially if asthma is suggested by family history or past medical history. Supportive therapy with humidified oxygen and hydration may also be needed, depending on the child's clinical appearance and oxygen saturation or blood gas analysis.

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20 Pertussis

Raymond C. Baker

Pertussis is an acute infection of the respiratory tract caused by *Bordetella pertussis,* a small gram-negative, pleomorphic rod. A syndrome resembling pertussis may also be caused by *Bordetella parapertussis, Mycoplasma pneumoniae, Chlamydia trachomatis/pneumoniae, Bordetella bronchiseptica,* and certain adenoviruses.

I. Epidemiology

Pertussis, especially during the early, catarrhal stage, is extremely contagious, with transmission by the respiratory route; up to 90% of nonimmune household contacts become infected. It occurs during all seasons of the year but tends to occur in sporadic endemics. All ages can be infected and symptomatic, but the greatest incidence and highest morbidity and mortality occur in infants and children. The mortality is as high as 1.3% in infants less than 1 month of age and 0.3% in those from 1 month to 1 year. Complications in young infants include pneumonia in 20%, seizures in 3%, and encephalopathy in about 1%. The incidence of pertussis declined dramatically with the introduction of a vaccine, but several thousand cases still occur each year in the United States, with a significant increase in the last few years.

II. Description

Following an incubation period of 6 to 20 days (mean, 7 days), the disease usually runs a course characterized by three stages:

A. Catarrhal. The catarrhal stage lasts 1 to 2 weeks, with mucous rhinorrhea, mild cough, conjunctival injection, and low-grade fever. The mucus produced during this stage is characteristically stringy and viscous, resulting in young infants producing "nose bubbles" and ropy strings of mucus.

B. Paroxysmal. In the paroxysmal stage, which lasts 2 to 4 weeks, the cough becomes more frequent and severe, occurring in paroxysms that cannot be suppressed. The classic pertussis cough is a series of staccato-like coughs followed by a sudden forceful inspiration that produces a characteristic whoop (inspiratory stridor). Posttussive vomiting is common. Infants less than 6 months of age may have a severe, persistent cough with post-tussive vomiting without the whoop. Apnea is also a common manifestation of pertussis in this younger population.

C. Convalescent. The convalescent stage consists of episodes of coughing and vomiting, which gradually diminish over 1 to 2 weeks, although the cough may persist for months. Physical findings are nonspecific during this stage.

III. Laboratory

The white blood cell count is commonly elevated (between 20,000 and 50,000/mm^3) with a marked predominance of lymphocytes. The chest radiograph may show perihilar infiltrates and/or atelectasis ("shaggy heart"). A positive fluorescent anti-

body test of nasopharyngeal secretions suggests the diagnosis, but false-positives are frequent. A positive nasopharyngeal culture on Bordet-Gengou medium confirms the diagnosis (80% positive). The yield is greatest during the catarrhal phase, which corresponds with the period of greatest infectivity.

IV. Treatment

The treatment of pertussis is erythromycin estolate (30 to 50 mg/kg per day in four divided doses for 14 days, maximum, 2 g/day), which may modify the illness if administered during the catarrhal stage. Newer macrolide antibiotics, clarithromycin and azithromycin, are also effective for patients who do not tolerate erythromycin. Antibiotics have no effect on the course of illness once paroxysms are established but are recommended to limit the spread of the organism. Otherwise, treatment is supportive. Young infants, especially those under 6 months of age and those with potentially severe disease, should be hospitalized to anticipate and monitor for complications such as apnea, life-threatening hypoxia, seizures, encephalopathy, and pneumonia. Respiratory isolation should be continued until 5 days of antibiotics are completed or until 3 weeks after the onset of paroxysms in the untreated child.

Close contacts, including household contacts and day care contacts, should receive erythromycin or other macrolide antibiotic in appropriate dosage as prophylaxis, regardless of vaccination status. Previously immunized contacts less than 7 years of age who have not had a booster dose within the previous 3 years should receive a booster dose of pertussis vaccine. Unimmunized contacts less than 7 years of age should be started on an immunization schedule. The current acellular vaccine provides protection in 50% to 90% of children who have received at least three doses of vaccine. Immunized children who contract the disease from household contacts often have mild disease.

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V

Gastrointestinal Disorders

21 Acute Abdominal Pain

Omer G. Berger

After 40 years of extensive experience, I still approach the acutely painful abdomen of a child with much apprehension and a greater feeling of uncertainty than any other domain of childhood.
—Joseph Brennemann, M.D.

Acute abdominal pain is a common presenting symptom of children seen in pediatric emergency rooms. The differential diagnosis is extensive and varies with the age of the child. In young children, medical etiologies are most common, especially acute gastroenteritis, whereas in children more than 2 years of age, appendicitis is the most common cause of acute abdominal pain and emergency abdominal surgery. The following information addresses common causes of acute abdominal pain, particularly those considered to be medical and surgical emergencies, and focuses on acute appendicitis as the most common cause of the acute surgical abdomen in childhood.

I. History

Periumbilical pain, especially if intermittent, is most likely functional or is a benign process. A general rule is that the more distant from the midline, the more likely the pain represents an organic problem. Acute pain of surgical origin generally precedes other gastrointestinal symptoms, such as vomiting and diarrhea, as opposed to the abdominal pain of acute gastroenteritis, in which vomiting and diarrhea commonly precede the abdominal pain. Acute abdominal pain lasting more than 3 hours most likely represents an acute, emergency condition. Fever and decreased activity (secondary to pain) suggest peritoneal inflammation.

II. Physical Examination

The physical examination of the child begins as one enters the examining room and observes the child's interactions with parents and the environment. For example, the child may exhibit respiratory distress, tachypnea, cough, or a toxic appearance or may be lying very still, with the anxious look of pain apparent. The skin is examined for rashes, especially hemorrhagic rashes, and hydration. Lymph node enlargement and tenderness are noted that might suggest infection or malignancy. The chest is examined for crackles, bronchial breathing, and dullness suggestive of pneumonia. The abdomen should be inspected for distention and ausculted for bowel sounds. Increased bowel sounds suggest acute gastroenteritis, whereas a quiet abdomen is more consistent with peritonitis. Abdominal palpation should begin lightly and progress to deeper palpation, looking for guarding, tenderness (including rebound), masses, or organomegaly. The physician should distract the child's attention away from the abdominal examination and watch the child's face for signs of pain in response to palpation. A rectal examination should be performed to detect constipation, an inflamed retrocecal appendix, or other intraabdominal pathology. In postpubertal females,

a pelvic examination is usually indicated to assess for pain of pelvic origin.

III. Differential Diagnosis of Acute Abdominal Pain

A. Differential diagnosis of acute abdominal pain in infants and young children (0 to 24 months of age). See Table 21.1.

B. Differential diagnosis of acute abdominal pain in children (2 to 12 years of age). See Table 21.2.

C. Differential diagnosis of acute abdominal pain in adolescents and young adults (13 to 21 years of age). See Table 21.3.

IV. Laboratory Investigation

Laboratory investigation generally includes a complete blood count and urinalysis. A pregnancy test may be indicated in adolescent females. An abdominal ultrasound may confirm the diagnosis of appendicitis or ovarian cysts when skilled ultrasonographers are available. The CT scan is becoming much more useful than the traditional plain film of the abdomen in the diagnosis of appendicitis and other surgical conditions.

Table 21.1. Differential diagnosis of acute abdominal pain (0–24 months)

Diagnosis	Suggested by
Viral illness	Fever, nonfocal abdominal examination, complete blood cell count
Constipation	Rectal examination, plain abdominal x-ray
Colic	Nonfocal abdominal examination, hearty appetite
Intussusception	Blood in stool ("current jelly stool"), intermittent nature of pain, abdominal mass
Incarcerated or strangulated hernia	Inguinal examination
Milk protein sensitivity	Blood in stools, diarrhea, nonfocal abdominal examination
Bacterial gastroenteritis	Fever, blood/mucus in diarrheal stools, exposure or family history, increased bowel sounds (BS), vomiting preceding pain
Less Common Causes of Acute Abdominal Pain in This Age Range	
Appendicitis	Malabsorption
Hirschsprung disease	Pneumonia
Congenital anomaly (with obstruction)	Neoplasm
	Sepsis

Table 21.2. Differential diagnosis of acute abdominal pain (2–12 years)

Diagnosis	Suggested by
Appendicitis	See below.
Mesenteric adenitis	Nonfocal abdominal examination, US or CT scan
Streptococcal pharyngitis	Sore throat, pharyngeal examination, positive streptococcal antigen
Gastroenteritis	Fever, diarrheal stools, exposure/family history, increased bowel sounds (BS), vomiting preceding pain
Constipation	Rectal examination, stool palpable on abdominal examination, plain abdominal x-ray
Pneumonia	Fever, cough, tachypnea, auscultation of lungs, chest x-ray
Toxic ingestion	History, toxicology screen
Urinary tract infection	Dysuria, frequency, urinalysis and culture
Less Common Causes of Acute Abdominal Pain in This Age Range	
Inflammatory bowel disease	Occult trauma
Meckel's diverticulum	Ovarian torsion
	Pancreatitis

V. Acute Appendicitis

Acute appendicitis is the most common cause of an acute abdomen in childhood. In older children, appendicitis presents with colicky or persistent pain that shifts from the periumbilical area to the right lower quadrant, where it becomes constant and increasingly severe. The pain is often followed by nausea, vomiting, and low-grade fever. In preverbal children, the history of pain may be difficult to obtain, and anorexia and inactivity (from pain with movement) may be the only symptoms.

On examination, maintaining cooperation is essential, so the abdominal examination of infants and young children may be done in the parent's arms for optimal effectiveness. Observation, auscultation, and palpation are performed in a search for localized guarding and rebound tenderness or other signs of peritonitis. Rectal examination is always indicated because the appendix may be in an atypical location.

Surgical opinion must be obtained any time a diagnosis of appendicitis or other surgical etiology is considered. When the diagnosis is uncertain, repeat examinations should be performed to look for progression of the disease process. As the incidence of

Table 21.3. Differential diagnosis of acute abdominal pain (13–21 years)

Diagnosis	Suggested by
Appendicitis	See below.
Gastroenteritis	Fever, diarrheal stools, exposure/family history, increased bowel sounds (BS), vomiting preceding pain
Constipation	Rectal examination, stool palpable on abdominal examination, plain abdominal x-ray
Dysmenorrhea	Association with menstrual period, pelvic examination
Pelvic infection	History of unprotected sexual activity, pelvic examination, vaginal discharge microscopic and culture
Mittelschmerz	Brief, sharp adnexal pain, timing in menstrual cycle
Pregnancy	History of unprotected sexual activity, pelvic examination, pregnancy test
Ovarian cyst	Timing in menstrual cycle, pelvic examination
Less Common Causes of Acute Abdominal Pain in This Age Range	
Hepatitis	Occult trauma
Ovarian torsion	Epididymitis
Ectopic pregnancy	Peptic ulcer disease
Endometriosis	Pneumonia
Cholecystitis	Peritonitis
Inflammatory bowel disease	Torsion of the testis

perforation is 40% in infants and children, a high index of suspicion is very important in this age group.

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22 Acute Gastroenteritis

Raymond C. Baker

I. Etiology

Acute gastroenteritis (AGE) with diarrhea, vomiting, and fever is a common illness of infants and children presenting to the primary care physician. Multiple etiologies exist for infectious enteritis, but a relatively small number of organisms account for the majority of cases. Among viral etiologies, rotavirus and Norwalk-like viruses predominate during cold winter months; the other important viral etiology is enteric adenovirus. Bacterial etiologies are less common but tend to be more severe and have more complications. These include *Salmonella, Escherichia coli, Shigella, Campylobacter, Yersinia,* and *Aeromonas. Giardia* is the only common parasitic cause of AGE, although *Cryptosporidium* is seen in immunosuppressed individuals.

II. Evaluation

The evaluation of infants and children with AGE begins with a history that might suggest an etiology (food-borne illness, exposure, time of year, underlying illness, associated symptoms) and a historical assessment of the state of hydration, including urine output; stool quality, quantity, and frequency; and the ability to maintain hydration. The physical examination should focus on the state of hydration (Table 22.1), degree of illness, and physical features that might suggest an etiology (e.g., associated rash).

In most cases of AGE, no laboratory work-up is needed and the disease is treated supportively with appropriate oral rehydration fluids and dietary manipulation. Stool cultures should be considered in the following clinical settings: (a) blood or mucus in the stool, (b) significant fever, (c) sudden onset of diarrhea, (d) minimal vomiting or vomiting that came on after the diarrhea, and/or (e) positive exposure history. Specific diagnosis (and therefore the need for stool culture) is more critical if the history reveals a caregiver who works in the food industry. In febrile infants under 6 months of age in whom a bacterial etiology is suspected, a blood culture should also be obtained, since bacteremia commonly accompanies AGE caused by *Salmonella* species in this age range.

III. General Treatment Guidelines

In mild diarrhea (≤5% dehydration) of short duration (1 to 2 days), no treatment is necessary other than increasing fluid intake. The electrolyte content of the fluids in mild diarrhea of short duration is not important. Routine feedings should be continued, including cow's milk–based formula and breast-feeding. Added fluids might include commercially available oral rehydration solutions (ORS) with 40 to 60 mEq/L sodium (e.g., Pedialyte, Infalyte). Follow-up examination, other than by telephone, is usually unnecessary.

In more significant diarrhea, attention should be paid to electrolyte replacement, and commercially available ORS with 75 to 90 mEq/L of sodium (Appendix B) should be used for an initial rehydration period. The volume of rehydration fluids is dependent on the degree of dehydration present and can be estimated

Table 22.1. Physical signs of dehydration

	Mild (3–5%) (<50 mL/kg deficit)	Moderate (5–10%) (50–100 mL/kg deficit)	Severe (>10%) (>100 mL/kg deficit)
Mental status	Well, alert	Restless, irritable	Lethargic, unconscious
Urine output	Slightly ↓	Moderately ↓	Absent
Mouth and tongue	Moist	Sticky	Dry
Thirst	Normal	Thirsty, drinks eagerly	Drinks poorly, unable to drink
Eyes	Normal	Sunken	Very sunken/dry
Tears	Present	Absent	Absent
Fontanelle	Normal	Sunken	Sunken
Skin retraction following pinch	Immediate	Slow	Very slow
Extremities	Warm, normal capillary refill	Delayed capillary refill (>2 seconds)	Cool, mottled
Heart rate	Normal	Somewhat increased	Increased
Blood pressure	Normal	Normal	Decreased

Adapted from Gastanaduy AS, Begue RE. Acute gastroenteritis. *Clin Pediatr* 1999;38:1–12.

clinically using Table 22.1. Oral rehydration can be initiated in an emergency department or hospital setting, making intravenous resuscitation unnecessary except in more severe dehydration with hemodynamic instability. The duration of initial oral rehydration is 4 to 6 hours, followed by refeeding and ORS for maintenance fluids and replacement of ongoing losses. In infants, breast or formula should be begun after rehydration plus supplements of ORS with 40 to 60 mEq/L sodium to replace ongoing losses. Ongoing losses can be estimated, assuming a diarrheal stool is about 10 mL/kg and emesis is 2 mL/kg. Older infants and children tolerate foods with complex carbohydrates (rice, wheat, potatoes, bread, cereal), lean meats, yogurt, fruits, and vegetables. Follow-up is necessary to assure compliance and resolution of the illness as well as attention to public health issues in patients with bacterial enteritis. In patients with severe dehydration, intravenous rehydration with monitoring of electrolytes and renal function is necessary until the child is able to tolerate oral liquids.

Another important feature of the treatment of AGE is advice to parents concerning infection control measures. Frequent hand washing, especially after diaper changes, will hinder spread of infection within the household. The primary care physician should also discuss such hygiene measures as part of anticipatory guidance during well-child care visits. Proper food handling with frequent hand washing and care to avoid contamination of foods for the table, eating utensils, and serving dishes with raw meat and meat products should be stressed to caregivers. Many of the bacterial enteritides are related to improperly handled and cooked foods, especially meats.

Antimotility drugs, such as loperamide (e.g., Immodium), are not indicated in infants and young children with common diarrhea. Diarrheal illness is usually self-limiting and well tolerated in this age range, and toxicities can occur from their use, especially in infants and young children. They are specifically contraindicated in bacterial gastroenteritis, as they may prolong the illness and encourage invasive complications (such as hemolytic uremic syndrome in *E. coli* O157:H7 infections).

IV. Antibiotic Treatment of Bacterial Gastroenteritis

The decision to treat the bacterial enteritides depends on several factors: size of inoculum required to transmit the disease (e.g., high for *Salmonella,* low for *Shigella*), antibiotic sensitivities, patient-related public health issues (e.g., day care, restaurant worker), incidence of treatable complications, and ability of antibiotic therapy to shorten the course (and contagious period) of the illness.

A. Salmonella. In infants under 6 months of age, further evaluation for sepsis may be indicated in documented *Salmonella* gastroenteritis due to the high incidence of associated bacteremia in this age group (5% to 40%). Many experts advocate treatment with parenteral antibiotics in patients at increased risk for invasive disease, such as in infants under 3 months of age, immunosuppressed patients (e.g., HIV infection and chemotherapy), patients with hemoglobinopathies (e.g., sickle cell disease), and patients with severe colitis. In older infants and children, antibiotic therapy is not indicated. Contagion is by the fecal/oral route and by ingestion of contaminated eggs, dairy products, and meats. Close attention to infection control measures should be instituted, especially if the infant is in a day care setting or if any of the caregivers are foodhandlers. Children with *Salmonella* infection may excrete organisms and remain contagious for up to 1 year following initial infection. This period of excretion is prolonged with antibiotic therapy.

B. *Shigella.* *Shigella* gastroenteritis is suggested by abrupt onset of diarrhea, crampy abdominal pain, fever and toxicity, and tenesmus. Stools, initially watery and high volume, progress to bloody, mucoid stools with painful defecation. Neurologic complications are common (headache, seizures, lethargy). Contagion is by the fecal/oral route. The peripheral white

blood count is typically modestly elevated with a marked left shift. All infants and children with documented *Shigella* AGE should be treated with antibiotics to shorten the course of the illness and to prevent person-to-person spread, since a very small inoculum of *Shigella* can cause infection (as few as 10 organisms). Oral trimethoprim/sulfa, 0.5 mL/kg PO BID for 5 days is usually effective.

C. *Campylobacter.* *Campylobacter* gastroenteritis presents with fever, abdominal pain, and diarrhea. Stools are commonly secretory with or without blood and mucus. Contagion is by the fecal/oral route and by ingestion of contaminated meat, especially chicken. If begun early in the course of the illness (within 4 days), treatment with erythromycin estolate, 10 mg/kg PO TID, may shorten the course of the illness and the period of excretion. This is especially appropriate if the child is in day care or if members of the family are involved with public foodhandling.

D. *Yersinia.* *Yersinia enterocolitica* causes abdominal pain, fever, and vomiting. The pain often localizes to the right lower quadrant, making differentiation from appendicitis difficult. Diarrhea may be minimal to absent (50%). Contagion is by the fecal/oral route and by ingestion of contaminated meats (especially pork), dairy products, water, and vegetables. Antibiotic treatment has not been shown effective in altering the course of uncomplicated *Yersinia* enterocolitis and should be used only for systemic or extraintestinal infection, which occurs infrequently.

E. *Escherichia coli.* Five types of diarrheogenic *Escherichia coli* have been defined by their virulence characteristics: enteropathogenic (EPEC), *Shiga*-toxin-producing (STEC), enterotoxigenic (ETEC), enteroinvasive (EIEC), and enteroaggregative (EAEC). All produce varying degrees of fever, vomiting, and diarrhea. Depending on the mechanism of action, the diarrhea varies from self-limiting to severe and from watery to bloody/mucoid. Children are particularly at risk for the development of hemolytic uremic syndrome (HUS) with STEC (*E. coli* O157:H7). About 6% to 8% of children infected with this strain develop thrombocytopenia, acute renal injury, and microangiopathic hemolytic anemia, which are characteristic of HUS. The STEC strain has been associated with contaminated meats, unpasteurized milk, and products contaminated with cow feces. Routine laboratory procedures do not distinguish among the different strains of *E. coli,* with the exception of O157:H7, which can be distinguished by its inability to ferment sorbital. In general, antibiotic treatment is not indicated except in systemic infection. Antibiotic prophylaxis for traveler's diarrhea (ETEC) has not been studied.

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23 Chronic Abdominal Pain in Children

Michael K. Farrell

I. Introduction

Chronic abdominal pain is a frequent problem encountered by the primary care physician. Approximately 10% to 15% of the school-aged population is affected. About 20% of children will have complained of abdominal pain in the past month, and 25% will seek medical attention. A reasonable definition of chronic abdominal pain is one proposed by Apley: three or more episodes of abdominal pain within 3 months severe enough to interfere with the child's normal activities. Evaluation of the child with chronic abdominal pain requires time and patience; it cannot be done in a brief office visit. The initial visit should reassure the physician and family that no serious life-threatening condition is present; an extended visit should then be scheduled for further evaluation. The purpose of the evaluation is to exclude serious disorders and to explain the natural history of chronic abdominal pain to the child and family. Specific appropriate therapy may then be instituted.

There are three distinct patterns of chronic abdominal pain: dyspepsia, paroxysmal periumbilical pain, and crampy lower abdominal pain associated with altered defecation. Each pattern occurs under different clinical circumstances and has different etiologies. Differentiating the type and pattern of pain early in the evaluation is important. The vast majority of chronic abdominal pain in children is functional and is exacerbated by stress. The physician should mention this early in the evaluation, and the evaluation should proceed along psychologic and physical lines simultaneously.

Dyspepsia is a vague epigastric pain often associated with meals and is seen more commonly in older children and adolescents. Related gastrointestinal symptoms, such as nausea, bloating, early satiety, and flatulence, are common. The **paroxysmal periumbilical abdominal pain** pattern is the most common type in school-aged children and is usually associated with stress (functional). The **crampy lower abdominal discomfort** associated with altered bowel habits is more common in adolescents and is equivalent to the irritable bowel syndrome.

II. Etiology

The specific etiology of chronic abdominal pain is usually unknown. In multiple series, no organic cause for the pain can be found in 90% to 95% of cases. Current theory is that the pain results from a gastrointestinal stimulus such as peristalsis that is perceived as pain in a sensitized host. The response to stress as a sensitizing factor plays a critical role in the syndrome. Dyspepsia may be due to *Helicobacter pylori* infection, NSAID gastropathy, gallbladder disease, or an upper gastrointestinal motility disturbance. Irritable bowel syndrome, characterized by pain,

constipation, diarrhea, or alternating diarrhea and constipation, is common in adolescents.

III. History

History is key to the diagnosis and management of chronic abdominal pain. The history should be obtained whenever possible from both the child and the parents in an unhurried manner that encourages sharing of psychosocial issues. The pattern of the painful episodes, associated symptoms, relationship to foods, and life events must be carefully examined. Time must be spent discussing the child's personality and response to stressors. The two most common stresses in a child's life are the family and school; they should be explored in detail. Parents frequently deny stress in their children; however, they often do not look at the situation from the child's perspective. Children with chronic abdominal pain tend to be high-strung, perfectionistic, and "worriers." In Apley's words, "They take the little issues of life too much to heart." The parents' and child's concerns should be discussed, so their fears and ideas can be explored. Two common issues are the fear of cancer and concern about appendicitis or ulcers. The family history is useful both in excluding serious gastrointestinal disease in the family and in eliciting a positive history of stress-related disease. "Red flags" in the history that suggest organic disease are listed in Table 23.1. Particularly important are any systemic or nocturnal symptoms, poor growth, and/or weight gain. Potential secondary gain that the child is receiving from the chronic abdominal pain (time off from school, more contact with an absent parent) must be sought. Depression may present as abdominal pain, particularly in adolescents, and should be considered in the differential diagnosis.

IV. Physical Examination

Growth parameters, including the stage of pubertal development, must be assessed and recorded. Growth velocity plotted on standard curves is the most sensitive indicator of growth failure. Any deviation from previously established growth patterns or delay in maturation is cause for alarm. A meticulous physical examination must be completed and discussed with the family and child. It must include inspection of the perianal area and a

Table 23.1. "Red flags" suggesting organic etiology

Pain is not central
Pain is nocturnal
Diarrhea (with or without occult blood)
Hematemesis
Hematochezia
Weight loss or growth failure
Delayed sexual maturation
Perianal lesions, recurrent oral aphthous ulcers, arthralgias, erythema nodosum (all suggestive of inflammatory bowel disease)

digital rectal examination to exclude constipation, often a great masquerader, especially in the school-aged child.

Physical findings that suggest organic disease include epigastric pain, fullness or mass in the right lower abdominal quadrant, recurrent mouth ulcerations, digital clubbing, perianal disease, and objective joint findings. Any concerns about perianal conditions such as "hemorrhoids" mandates close evaluation because children rarely develop hemorrhoids. The most common causes for perianal lesions are chronic constipation, inflammatory bowel disease, and abuse. It is often useful to ask the family to return when the child is having pain, so that an examination may be performed during an episode, thus reassuring the family and child. The physician should always acknowledge that the pain is real. Even though the pain may result from an emotional cause, it is very real to the child.

V. Laboratory and Radiologic Evaluation

Following the clinical evaluation, certain baseline laboratory studies may be indicated. The most common are a complete blood count, sedimentation rate, urinalysis and culture, and stool for occult blood. If these with the history and physical examination are normal, little likelihood of serious abdominal pathology exists. Radiographic studies are rarely helpful. The upper gastrointestinal series best identifies anatomic lesions but often misses superficial mucosal lesions and does not detect *Helicobacter pylori* disease. The barium enema is insensitive for mucosal disease and results in significant gonadal radiation. The abdominal and pelvic ultrasound, though rarely positive, does view the genitourinary and biliary systems and may occasionally be indicated to rule out disease in these organ systems. Table 23.2 lists the most frequent organic causes of chronic abdominal pain and also clues for suggesting the diagnosis.

Helicobacter pylori infection should be suspected if there are nocturnal symptoms, epigastric pain and tenderness, and a positive family history of peptic ulcer disease. Infection with *Helicobacter* occurs in childhood, but most infections are asymptomatic. Considerable controversy exists over which symptoms may be attributed to *Helicobacter*. A recent metaanalysis suggests that duodenal and gastric ulcers are frequently related to *Helicobacter* infection, but that chronic periumbilical abdominal pain or the irritable bowel syndromes are not. If *Helicobacter* is suspected, the best test is a ^{13}C or ^{14}C urea breath test; serologic studies are unreliable, especially in younger children. Endoscopy allows the pathologic examination of mucosal biopsies and the performance of the rapid urease test for *Helicobacter*. However, it is important to note that *Helicobacter* is not considered a cause of chronic abdominal pain and that routine testing is not indicated. Endoscopy should be reserved for children with suspected peptic disease.

Lactose intolerance, if suspected by the clinical history, can be confirmed with elimination and rechallenge or a breath hydrogen test following ingestion of an appropriate dose of lac-

Table 23.2. Most common organic etiologies of recurrent abdominal pain

Disease	Suggested by
Urinary tract infection	Vomiting, flank pain
Inflammatory bowel disease	Poor growth and/or delayed sexual maturation, perianal disease, systemic symptoms, anemia, lower abdominal pain/tenderness/mass
Peptic disease	Family history of peptic disease, nocturnal pain, epigastric tenderness
Pancreatitis	Radiation of pain to back, vomiting, family history
Constipation	Left lower quadrant pain, palpable stool, symptoms relieved by defecation, encopresis
Esophagitis	Burning epigastric pain, substernal pain, odynophagia
Giardiasis	Diarrhea, distention, flatulence
Lactose intolerance	Diarrhea, distention, symptoms related to meals (also seen with excessive sorbitol/fructose ingestion)

tose. Gallbladder disease is uncommon in children unless there are underlying conditions that predispose to cholelithiasis, such as hemolytic anemia and ileal resection. Recently, there has been interest in biliary dyskinesia as a cause of chronic dyspeptic symptoms in children; however, this is controversial at best. The diagnosis is made by administration of a radionucleotide and measuring the rate of gallbladder emptying. Neither normals for children nor the best protocol for administering the test have been established. Referral to an experienced pediatric gastroenterologist or surgeon is indicated since the treatment is cholecystectomy.

VI. Treatment

The treatment of chronic abdominal pain begins with explanation and reassurance. The fact that serious organic disease has been eliminated must be emphasized. The presence of stress in the child's life should be acknowledged, and the family and child should be educated about the physiologic response to stress. The pain again must be acknowledged as real and not "in the head." Pertinent examples relevant to the family's and child's experiences may be helpful. For example, most children and adults have experienced abdominal discomfort ("butterflies in the stomach") before an important examination or before participation in a pivotal sporting event.

The clinician should be confident and avoid the "one more test" trap that undermines the family's confidence. Specific stressors that have been identified should be discussed, and occasionally psychologic intervention may be indicated. Stress reduction techniques are useful.

Few data exist that support a pharmacologic approach to chronic abdominal pain. H_2 blockers and proton pump inhibitors are useful only in acid peptic disease. If evidence of *Helicobacter pylori* infection is present, it should be treated appropriately. One study has suggested that increasing dietary fiber may be helpful. Constipation must be treated vigorously.

In a few cases of severe long-standing pain, the syndrome takes on a life of its own. A state of "visceral hyperalgesia" develops, and normal stimuli are interpreted as intense pain. Treatment of this syndrome requires a multidisciplinary team. The child must be encouraged to return to normal activities, and antidepressants such as amitriptyline may be helpful.

If the family's and child's concerns are recognized, a complete and thorough evaluation done, and an honest discussion of the findings offered, most children respond well. The physician should emphasize to the family his or her availability and should schedule frequent follow-up appointments to check on the progress of the child.

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24 Encopresis

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Encopresis (fecal hoarding) is a syndrome of chronic, functional, retentive constipation and fecal incontinence in young school-aged children. The incidence of encopresis ranges from 3% to 4% of 4-year-old children to 1.6% of 10-year-olds. Boys are affected three to six times more commonly than girls. The disorder typically begins after stool continence has been achieved for a period of 1 or more years (secondary encopresis). Multiple causes of the initial motivation to fecal retention lead to the syndrome of fecal soiling. Psychologic issues may play a role, with voluntary retention emerging as a control factor in the toileting process; voluntary retention may be a means of gaining attention as a reaction to a new sibling; or the child may simply be too distracted by other life events to take the time (e.g., attention deficit hyperactivity disorder [ADHD]). A period of painful defecation (rectal fissures, perianal abscess, large-caliber stool) may initiate voluntary retention. Regardless of the etiology of the initial retention, a pattern of chronic retention develops that leads to the symptoms of the syndrome of fecal soiling. The chronic withholding of stool passage, over time, causes distention of the colon, with hard stool, loss of tone and a sensation of fullness of the colon, and finally leakage of liquid stool around the impaction through a stretched anal sphincter.

I. History

The typical presenting complaint of the parent of a child with encopresis is frequent fecal soiling of small amounts of soft stool. Further questioning will commonly uncover additional information that suggests significant constipation and fecal retention—ritual gluteal tightening and posturing, decreased food intake, irritability, and intermittent abdominal pain. Following a 2- to 4-week period of such symptoms, a painful defecation of a large quantity of large-caliber stool occurs, sometimes accompanied by traumatic internal anal fissures. Parents may report that the quantity and size of the stool stops up the toilet and has to be broken up to flush it down. After the passage of an extremely large stool, a short period of decreased symptoms usually follows, and then the cycle repeats. Children may exhibit denial of the process and may even hide soiled underwear from parents and siblings. Secondary psychologic problems commonly result from the intolerance (and derision) of siblings and schoolmates to the obvious odor created by feces in underwear for several hours. Intermittent or regular incontinence of large amounts of normal-appearing stool is atypical for encopresis and suggests a psychobehavioral problem.

II. Physical Examination

The physical examination is usually normal except for a stretched anal sphincter, secondary to a large quantity of hard stool in the ampulla. Hard stool may also be palpable on abdominal examination. The primary differential is Hirschsprung disease and psychobehavioral encopresis. In the former disorder an

aganglionic segment of distal colon functionally obstructs passage of stool. Children with Hirschsprung disease tend to be small, pass small-caliber stools, and have an empty ampulla on examination. Most have had abnormal stool patterns from birth. In fecal incontinence of psychobehavioral origin, the tendency is to be periodically incontinent of larger quantities of normal-appearing stool.

III. Treatment

The treatment of encopresis requires the physician to explain to the parents the pathogenesis of the condition, especially since the treatment (laxatives) may seem paradoxical to parents whose perception is that their child has frequent liquid or very soft stools. Bringing the parents to an understanding that the end result of encopresis—namely, incontinence—is involuntary is important. Treatment of encopresis takes time (often months) and patience. The child must relearn normal bowel function through behavioral modification techniques that reward the child for both an effort and the result, and this learning process cannot be considered successful until the child's every bowel movement is painless.

The principles of treatment of encopresis are (a) initial complete evacuation of the bowel, (b) maintenance of normal soft stool content of the bowel, and (c) retraining in normal bowel function. At the initial visit, the bowel needs to be emptied—either as part of the visit (with enemas) or with careful instructions to the parent for carrying the procedures out at home. Colonic evacuation may be accomplished with a combination of enemas (e.g., mixture of 3 ounces milk and 3 ounces molasses QD or BID for an average-sized child) possibly in combination with an osmotic laxative (e.g., milk of magnesia). An alternative method is oral mineral oil, 1 ounce per year of age up to 8 ounces given QD or BID for 3 days. Mineral oil may be mixed with juice or taken with a straw (bypassing the taste buds) to help with compliance. Follow-up by telephone is necessary to assure success.

Subsequently, the child should be started on a combination of (a) regular stool softeners and stimulants to maintain very soft stools, which are passed easily and without pain at least daily, plus (b) defecation retraining.

A. Stool softeners. In the cooperative older child, mineral oil is very effective and inexpensive (available over the counter). It should be given in progressively larger doses on a regular basis until the desired very soft consistency of the stool is achieved. Then that dose is given regularly for a period of several months as necessary to maintain stool softness. The dose is usually in the range of 1 to 2 ounces QD to BID. Since mineral oil may decrease absorption of fat-soluble vitamins in food, it should not be given at mealtime. Mineral oil can be made more palatable to some children by mixing it with oatmeal or orange juice. Some children may also require a mild stimulant laxative, such as Senokot syrup or granules, to aid evacuation during the retraining period. (See the fol-

lowing discussion.) Another effective stool softener is lactulose (requires a prescription) given regularly in increasing doses of 0.5 to 1 mL/kg PO BID (maximum dose 45 mL BID) until achieving the desired stool consistency. The starting dose for adolescents is 15 mL PO BID. The dose is titrated to maintain a very soft consistency to the stools. Adding lactulose, which has a sweet taste, to Kool-Aid, oatmeal, or chocolate milk may help children tolerate the medication better.

B. Defecation retraining. Children with encopresis often have long-standing habits of voluntary fecal retention with resulting loss of normal colonic tone that may require extensive retraining in new defecation habits to allow the regular passage of stool. Retraining consists of taking advantage of the gastrocolic reflex by having the child sit on the toilet for 5 to 10 minutes after meals and encouraging him/her to try to have a bowel movement. Parents should make clear to the child that sitting on the toilet is mandatory, but this should not be presented in a negative or disciplinary manner. The parent may want to give the child something to do while he sits on the toilet, such as reading to him or providing him with toys or books. The child should be rewarded both for sitting and for a successful bowel movement. Over a period of days, a pattern will develop in which the child defecates regularly after one or two meals.

At the same time the stool softeners are added, the parent should add fiber to the child's diet using fiber supplements and foods high in fiber, such as popcorn, grains, fruits, and vegetables. Stool softeners should be continued for several months, until regular stooling is established. A very important aspect of the retraining program is to provide positive reinforcement for sitting on the toilet, passing stools in the toilet, and lack of soiling. This may be in the form of new underwear, especially brightly colored, figured underwear; a trip to the local fast-food restaurant; and so on. Parents should be educated about the possibility of recurrence of impaction and should intervene vigorously with laxatives if the child goes more than 48 to 72 hours without a bowel movement.

C. Psychologic referral. Many children with longstanding encopresis develop low self-esteem, regression and loss of coping skills, school avoidance and truancy, and affective disturbances. The physician needs to keep abreast of these problems through a careful initial history and ongoing questions about school, friends, and interactions in the home. Although these issues resolve with appropriate medical management of the underlying problem in many children, some require psychologic intervention and support from a clinical psychologist. This important aspect of care, if overlooked, can counteract the best intentions and medical therapy.

D. Follow-up. A most important aspect of therapy is frequent follow-up, some of which may be by telephone. Regular

visits to document progress and to reinforce the regimen are needed to ensure compliance and success. As a key part of follow-up, the physician should talk directly to the child, praising for successes and empathizing with relapses with carefully worded, understandable aids.

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25 Common Intestinal Parasites

Omer G. Berger

Intestinal parasites are common in North American children but result in asymptomatic or mild illness in virtually all cases, making routine screening for parasitic infection unnecessary. Because the risk of parasitic infection is higher in underdeveloped countries, refugees or migrants from endemic areas who are being seen for an initial evaluation in a primary care setting for well-child care should routinely be evaluated by obtaining a stool specimen for ova and parasites. Parasites not seen in the United States often show up in this setting, usually sending the primary care physician scrambling for the *Red Book* to look up the treatment. The following information focuses on the three most common gastrointestinal parasites seen regularly in North America.

I. Pinworms

Pinworms (*Enterobius vermicularis*) affect as many as 20% of young children and cause perianal itching or "pain" as the 5- to 10-mm gray to brown roundworms deposit eggs on perianal skin (usually at night). Spread is by the fecal/oral route, with worm eggs transmitted by fingers from shared toys, bedding, clothing, and so on. The diagnosis may be made by the following:

A. A history of parents seeing the threadlike parasite on stool or around the anus.

B. A microscopic examination of clear cellophane tape applied to perianal skin revealing characteristic ova.

C. A history of nocturnal perianal itching.

Mebendazole (e.g., Vermox) is the drug of choice; 100-mg chewable tablets are given as a single dose that is repeated after 2 weeks to eliminate newly hatched worms.

As recurrence is common, hygiene, trimming fingernails, and frequent changing of sheets are important. Consideration should be given to treating other family members if infection recurs.

II. Giardia Lamblia

Giardia lamblia is the most common protozoal parasite, causing recurrent foul-smelling diarrhea, anorexia, abdominal distention, and flatulence. Toddlers may exhibit poor weight gain and vomiting during sleep. As many as 25% of infected children may be asymptomatic. Vitamin deficiencies and intestinal enzyme depletion may result from chronic disease and malabsorption.

Although long associated with drinking water contaminated by the stool of infected animals, giardiasis has recently been reported in up to 50% of children in some day care units, suggesting easy passage from child to child via the fecal/oral route.

The diagnosis of giardiasis is made by finding the organism, cysts, or the antigen (e.g., enzyme immunoassay or direct immunofluorescence) in stool samples. Specimens should be examined immediately, or stool collection kits containing preservatives can be given to the parent to collect stool specimens at home.

The treatment of choice is metronidazole (e.g., Flagyl) 5 mg/kg TID for 5 days (maximum dose 250 mg TID). If the local

pharmacy is unable to make a suspension of metronidazole, furazolidone (e.g., Furoxone), which is available as a liquid, may be used in a dose of 5 mg/kg per day QID for 7 to 10 days (maximum dose, 100 mg QID). Preventive measures include improved hygiene in day care centers, filtration of city water supplies, and boiling water obtained directly from streams.

III. Toxocariasis

Toxocariasis is caused by dog and cat roundworms (*Toxocara canis* and *Toxocara cati*), which have an incomplete life cycle in humans. Infection is common, ranging from 3% in urban children to 50% in rural areas with poor sanitation. Heavy infestation results in viscera larval migrans with hypergammaglobulinemia, hepatomegaly, and marked eosinophilia. Ocular involvement usually occurs in the absence of eosinophilia and visual signs.

Fortunately, most children have light infestations and have only eosinophilia and no symptoms of disease. The diagnosis may be confirmed by serologic examination or biopsy. Treatment is not indicated for children without symptoms. Prevention requires proper disposal of feces and periodic deworming of puppies and kittens.

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26 Gastroesophageal Reflux in Infants

Omer G. Berger

I. Uncomplicated Gastroesophageal Reflux (GER)

Reflux of gastric contents into the esophagus occurs periodically throughout the day in infants, children, and adults. Approximately half of normal infants in the first 3 months of life manifest regurgitation, defined as the presence of gastric contents in the mouth. A smaller percentage of children will demonstrate vomiting, the forceful expulsion of gastric contents through the mouth. Infants with uncomplicated regurgitation are referred to as "happy spitters" and require no specific treatment. The following conservative nonspecific management strategies are frequently employed:

A. Formula thickened with rice cereal (1 tablespoon per 1 to 2 ounces) may result in decreased vomiting. This tactic does not reduce reflux into the esophagus, but reduces soiled clothing and time in the laundry room.

B. A trial of elemental formula may be appropriate, as milk protein sensitivity may present as GER.

C. The prone position results in less reflux than the supine position or the semisupine position in an infant seat. However, the risk of SIDS precludes the use of the prone position except when, in the practitioner's opinion, the supine position places the infant at greater risk of aspiration and apparent life-threatening events (ALTE).

D. Avoidance of tobacco smoke.

Antacids and pharmacologic therapy do not appear to affect the natural history of uncomplicated reflux in infants, which usually resolves by 12 to 15 months of age. If symptoms do not resolve by this age, the health care provider should consider an upper gastrointestinal radiographic study (upper GI series) and consultation with a pediatric gastroenterologist.

II. Complicated Gastroesophageal Reflux or Gastroesophageal Reflux Disease (GERD)

GERD is characterized by respiratory or GI signs and symptoms, such as the following:

A. Poor weight gain.

B. Esophagitis, as manifested by neck extension after feeding, pain (irritability), or evidence of bleeding, anemia, or esophageal stricture.

C. Aspiration, as suggested by cough and wheezing, especially after feeding.

D. Pneumonia.

E. An apparent life-threatening event (ALTE).

Diagnostic evaluations to assess other potential causes of vomiting in the infant with suspected GERD may include blood lead, electrolytes, liver enzymes, ammonia levels, urinalysis, and a review of newborn screening test results. An upper GI series to

evaluate anatomic reasons for vomiting is generally advisable; further diagnostic studies of GERD, including esophageal pH probe and endoscopy, are generally planned with the assistance of a pediatric gastroenterologist.

III. Management of GERD

Infants with evidence of GERD and poor weight gain must be evaluated to ensure that adequate caloric intake is provided. Thickening formula with cereal may increase the total intake of calories but does not significantly reduce reflux, as shown by pH studies. A trial of an elemental formula should be considered. Hospitalization may be indicated in severe cases in early infancy. Acid suppression with ranitidine (e.g., Zantac), 5 to 8 mg/kg per day PO divided BID, may be helpful when esophagitis is suspected. Rule out other causes, such as aspiration during swallowing or congenital anomalies. The relationship between ALTE and GER is unclear, and there are no studies of the treatment of GER relative to ALTE. Recurrent pneumonia due to GER is thought rare.

Gastroesophageal reflux is very common in infants and children. Primary care practitioners must be aware of the natural history of GER and the possible range of complications. Asking about reflux and regurgitation should be part of well-child interviews, especially in early infancy. A conservative course of treatment is recommended before ordering laboratory and radiographic investigation.

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27 Hyperbilirubinemia in the Term Infant

Christine L. McHenry

Hyperbilirubinemia is present in all newborns early in the neonatal period, with 60% of newborns showing clinical signs of jaundice. The primary care physician must determine whether the jaundice is secondary to physiologic jaundice of the newborn or whether it signals a pathologic process. This task is somewhat more difficult with early discharge programs from the newborn nurseries. Any infant discharged from the nursery in less than 48 hours must be evaluated by a health care professional (home health nurse, physician) within 48 hours of discharge for evidence of jaundice.

I. History and Physical Examination

A. Maternal history

1. *Obstetric history.* Maternal diabetes as well as certain congenital infections such as rubella, toxoplasmosis, cytomegalovirus (CMV), and syphilis are associated with hyperbilirubinemia. In addition, acute infections with organisms such as group B *Streptococcus* are associated with elevated bilirubin levels.
2. *Race and ethnic background.* East-Asian infants are much more likely to become jaundiced than Caucasian infants, and Caucasian infants are more likely to become jaundiced than African-American infants. Individuals from the Mediterranean, China, and Nigeria or those of Sephardic Jewish heritage have a higher incidence of G-6-PD deficiency.
3. *Blood type.* If the mother is blood type O and the infant is A (most common) or B or if the mother is Rh negative and the infant is Rh positive, isoimmune hemolytic disease is possible.
4. *Family history.* Neonatal jaundice tends to run in families.

B. Delivery history

1. *Oxytocin.* The use of oxytocin during labor is associated with slightly higher bilirubin levels.
2. *Vacuum extraction.* Vacuum extraction may produce a cephalohematoma that will be a source of bilirubin as the blood is mobilized and broken down.
3. *Premature rupture of membranes.* The incidence of neonatal sepsis rises dramatically with the length of time (over 12 hours) that membranes are ruptured. Sepsis may be associated with both indirect (unconjugated) hyperbilirubinemia and direct (conjugated) hyperbilirubinemia.
4. *Apgar score.* Hypoxia is associated with higher bilirubin levels.

C. Infant history

1. *Age of onset of jaundice.* Infants who are jaundiced in the first 24 hours of life do not fit the parameters of

physiologic jaundice; therefore, other causes of the jaundice must be sought.

2. *Feeding.* Breast feeding, especially if ineffective and infrequent, will cause elevated bilirubin levels in the first several days of life. A late-onset, prolonged indirect (unconjugated) hyperbilirubinemia also is seen with breast feeding. Vomiting may indicate intestinal obstruction that will increase the enterohepatic circulation and thus the bilirubin load presented to the circulation. If the infant is formula fed, knowing the type of sugar in the formula is important. Certain metabolic abnormalities, such as fructosemia, can cause a direct (conjugated) hyperbilirubinemia.

3. *Stools.* Delayed passage of the first meconium stool may indicate an intestinal motility problem such as Hirschsprung disease or meconium ileus, which can be seen in cystic fibrosis. Acholic stools indicate a problem with conjugation of the bilirubin.

4. *Fever, irritability, or lethargy.* Fever, irritability, or lethargy suggest sepsis, which may be associated with unconjugated hyperbilirubinemia.

D. Physical examination

1. *General impression.* Is the infant awake and alert, or is the infant irritable or lethargic?

2. *Skin.* Infants with indirect (unconjugated) hyperbilirubinemia appear yellow, in contrast to infants with direct (conjugated) hyperbilirubinemia, who appear greenish yellow. The presence of yellow jaundice on the hands and feet suggests severe indirect (unconjugated) hyperbilirubinemia.

3. *Signs of congenital infection.* Depending on the congenital infection, infants may present with a variety of findings, including "blueberry muffin" spots, hepatosplenomegaly, cardiac defects, and eye defects.

4. *Congenital anomalies.* Midfacial anomalies are associated with pituitary abnormalities. Decreased bilirubin clearance can be seen with hypothyroidism and hypopituitarism.

5. *Enclosed hemorrhage.* A cephalohematoma is a source of bilirubin as the blood is metabolized and broken down. As a result, a cephalohematoma can cause prolonged (>7 days) indirect hyperbilirubinemia.

6. *Abdominal distention.* Abdominal distention will be seen in intestinal obstruction.

7. *Signs and symptoms of acute infection.* Such signs and symptoms may include hypothermia or hyperthermia, irritability or lethargy, poor capillary refill, bulging fontanelle, tachypnea, and tachycardia.

II. Classification of Hyperbilirubinemia

A. Classification. Hyperbilirubinemia can be classified as indirect, or unconjugated, when the direct or conjugated component is less than 15% of the total; as direct, or conjugated, when the direct component is more than 30% of the total; and

as indeterminate when the direct component is between 15% and 30%.

B. Criteria for laboratory investigation:

1. *Infants who are jaundiced within the first 24 hours of life.*
2. *Infants who have a rapid rise in total bilirubin* (>5 mg/dL day).
3. *Full-term infants with a total bilirubin more than 13 mg/dL.*
4. *Infants who are jaundiced after 7 days of age.*
5. *Infants who have direct (conjugated) hyperbilirubinemia.*

III. Indirect (Unconjugated) Hyperbilirubinemia

A. Differential diagnosis

1. Isoimmune hemolysis (ABO and Rh incompatibility).
2. Nonisoimmune hemolysis (G-6-PD deficiency, pyruvate kinase deficiency, spherocytosis, elliptocytosis).
3. Polycythemia (from delayed cord clamping, small-for-gestational-age infant, infant of a diabetic mother).
4. Enclosed hemorrhage.
5. Swallowed maternal blood.
6. Increased enterohepatic circulation from small bowel obstruction.
7. Asphyxia.
8. Sepsis.
9. Hypothyroidism.
10. Crigler-Najjar syndrome.
11. Physiologic jaundice.
12. Breast feeding/milk jaundice.

B. Evaluation. The evaluation of "clinically significant" indirect (unconjugated) hyperbilirubinemia in an otherwise healthy full-term infant should consist of (a) knowledge of mother's blood type and Rh, (b) direct Coombs' test on the infant blood, and (c) infant's total serum bilirubin and direct bilirubin fraction. Additional tests to consider if the history and physical examination are suggestive include a sepsis work-up, blood count with a platelet count, reticulocyte count, G-6-PD screen, thyroid function studies, and abdominal film or upper GI study.

C. Treatment. The level of total serum bilirubin at which phototherapy or exchange transfusion should be considered will vary somewhat with the age of the infant. At 24 to 48 hours of age, phototherapy should be started at a bilirubin of ≥15 mg/dL; at a bilirubin of ≥25 mg/dL, an exchange transfusion plus intensive phototherapy should be performed. At 49 to 72 hours of age, phototherapy should be started at a bilirubin of ≥18 mg/dL; exchange transfusion plus intensive phototherapy should be initiated at a bilirubin of ≥30 mg/dL. After 72 hours of age, phototherapy should be started at a bilirubin of ≥20 mg/dL; exchange transfusion plus intensive phototherapy are started at a bilirubin of ≥30 mg/dL.

IV. Physiologic Jaundice of the Newborn

A. Definition and etiology. Physiologic jaundice of the newborn, a transient elevation of indirect (unconjugated) bilirubin, usually is evident between 48 and 72 hours of age and resolves by 1 week of age. The etiology of physiologic jaundice is a complex interplay among several factors, including (a) increased bilirubin load from decreased red cell survival, larger blood volume per unit body weight, and higher hemoglobin concentration; (b) decreased bilirubin uptake by the hepatocyte; (c) decreased glucuronyl transferase activity; and (d) increased enterohepatic circulation.

B. Treatment. The use of phototherapy and exchange transfusion for physiologic hyperbilirubinemia is controversial. Most studies of hyperbilirubinemia and kernicterus have included only babies with hemolytic disease, particularly Rh incompatibility. Studies of hyperbilirubinemia and other neurologic problems have shown mixed results. Reasonable recommendations for the full-term infant with hyperbilirubinemia secondary to physiologic jaundice are to institute phototherapy at a bilirubin level of 18 to 20 mg/dL and to consider an exchange transfusion at more than 25 mg/dL if phototherapy fails. The major question is whether or not phototherapy and/or exchange transfusion is of greater benefit than risk to the infant with physiologic hyperbilirubinemia.

V. Jaundice Associated with Breast Feeding

A. Breast-feeding jaundice. Early-onset jaundice (first several days of life) in breast-fed infants is the equivalent to adult starvation jaundice secondary to increased enterohepatic circulation of bilirubin. Inadequate frequency of breast feeding results in decreased volume of breast milk, decreased stooling, and weight loss. Mothers should be encouraged from the beginning to breast-feed 8 to 10 times per day. Supplemental water or glucose water has not been shown to decrease bilirubin levels and should be discouraged.

B. Breast milk jaundice. Late-onset jaundice in breast-fed infants peaks between 2 and 3 weeks of age and is probably secondary to an as-yet-unidentified factor in breast milk that increases the enterohepatic circulation of bilirubin. Most infants are no longer jaundiced by 4 to 6 weeks of age. The differential diagnosis of prolonged indirect (unconjugated) hyperbilirubinemia includes chronic hemolysis, such as ABO incompatibility, hypothyroidism, Crigler-Najjar syndrome, and breast milk.

VI. Direct (Conjugated) Hyperbilirubinemia

A. Differential diagnosis. Direct (conjugated) hyperbilirubinemia is always pathologic and always requires an investigation into the etiology. The differential diagnosis includes the following:

1. *Extrahepatic obstruction* (extrahepatic biliary atresia, choledochal cyst).
2. *Intrahepatic cholestasis* (intrahepatic biliary atresia, drugs).

3. *Neonatal hepatitis and infection* (hepatitis B, syphilis, toxoplasmosis, rubella, cytomegalovirus, herpes, echovirus, coxsackievirus, bacterial sepsis).
4. *Genetic and metabolic disorders* (cystic fibrosis, alpha-1-antitrypsin deficiency, galactosemia, fructosemia, tyrosinemia, Gaucher disease, hypothyroidism, hypopituitarism).
5. *Dubin-Johnson and Rotor syndromes.*

B. Evaluation. The initial evaluation of an infant with direct (conjugated) hyperbilirubinemia should include a fractionated serum bilirubin, serum transaminases and alkaline phosphatase, prothrombin time, congenital infection evaluation (TORCH titers), and urine for reducing substances. It also is important to look at the stool color to see if it is pigmented. Most, if not all, infants with direct (conjugated) hyperbilirubinemia will need to be evaluated by a gastroenterologist for a more detailed diagnostic evaluation and therapeutic intervention.

C. Treatment. Treatment consists of supportive measures plus specific treatment directed at the underlying etiology of the direct (conjugated) hyperbilirubinemia.

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28 Infantile Colic

Raymond C. Baker

Infantile colic is a difficult management problem without easy solutions for the primary care physician. A colicky infant in the home can be very stressful even in the most stable of families. When the caregivers are young parents, a single parent, parents with a large family with several young children, parents with poor coping skills, or parents without outside support, the stress can create significant dysfunction and angst. With the focus of more attention on the problem of colic in recent years, a clearer understanding of the disorder and its behavioral/temperamental underpinning has resulted. Other terms for colic that are somewhat more descriptive of the condition are *paroxysmal fussing* and *primary excessive crying*.

I. Definition

To define *colic* one must first define normal crying behavior is in infants. Brazelton studied 80 middle-class normal infants and found that, on average, 2-week-old infants spent 1.75 hours per day crying; 4-week-olds, 2 hours; 6-week-olds, 2.75 hours; 8-week-olds, 2 hours; 10-week-olds, 1.5 hours; and 12-week-olds, 1 hour. In the same study, he noted that the majority of crying at all ages occurred during the early evening hours.

Abnormal crying behavior, or colic, may therefore be defined as an exaggeration of the normal crying time, usually peaking during the early evening hours. Practically speaking, however, the physician must also accept that the *perception of abnormal* depends somewhat on the expectations of the caregiver and on the caregiver's tolerance of and reaction to crying behavior. What constitutes colic and intolerable crying to one parent may be viewed as merely a fussy infant requiring extra cuddling by another.

Illingworth defined *colic* as rhythmic attacks of screaming during the first 3 months of life, usually in the evenings, that cannot be explained by any known cause of crying. Adams described colic as crying time greater than the 75th percentile above "normal" crying time. Carey defined the colicky infant as one crying "full force" (not just fussing) for greater than 3 hours per day for at least 4 days per week.

Although a minority of infants with colicky symptoms may indeed have an underlying medical diagnosis such as true formula intolerance (e.g., cow's milk allergy or lactose intolerance), these are usually suggested by the infant's history and a trial of non-cow's-milk-based formula. Because the majority of infants with colic have no medical cause for their prolonged crying, infant temperament appears a likely explanation. Infants with colic tend to have heightened responses to external stimuli, especially noxious stimuli, and underdeveloped coping skills for reacting to these stimuli in any way except crying. They seem to lack the skills to make smooth transitions from one state of consciousness to another and to interact effectively with caregivers. Likewise, parents of babies who become labeled as colicky often have less

effective skills at reading their infant's needs and reacting appropriately. Carey summarizes the underlying problem in colic as "a temporarily unsatisfactory interaction between a sensitive, irritable infant and parents who have not yet learned to read the infant's needs correctly and respond to them effectively." Many colicky babies fit into the general temperament category of "difficult."

II. Natural History of Colic

The natural history of colic is that of a term infant, following a normal pregnancy, labor, and delivery, who develops symptoms at 2 to 3 weeks of age, peaking at 1 to 2 months, and resolving by 3 to 4 months. Colicky infants tend to be overweight, rather than small, as a result of the parents' frequent feeding in an attempt to decrease the crying (misinterpreting the crying as hunger). Resolution corresponds with (a) the normal development of the infant's ability to self-stimulate, (b) increased interaction with environment and parents/siblings (smiling, cooing), and (c) increased maturation of self-regulating mechanisms (dampening responses to external stimuli).

III. Evaluation and Treatment

The evaluation (and treatment) of colic begins with a careful history looking for a medical or physical explanation for crying and includes accurate measurements of height, weight, and head circumference as an assessment of adequate growth. Performing a careful physical examination *in front of the parents* to assure them that you are taking their complaints seriously is important. In classic colic, the physical examination is entirely normal except for a tendency to be overweight.

The physician treats colic mostly by listening to the parents' concerns, showing empathy for their distress, and counseling regarding the self-limiting nature of the problem. Several important points should be included in the health care provider's discussion with the parents about their infant's health:

A. The infant is physically healthy (the baby is not crying from pain).

B. The parents are "not doing anything wrong."

C. The infant has normal weight gain (usually excessive), which implies that the formula "agrees with the baby." Infants do not gain weight on formulas to which they are intolerant, whether from lactose intolerance, protein intolerance, or milk allergy.

An explanation of infant temperament and differing reactions to noxious stimuli (such as an overfull stomach from too frequent feeding), the development of coping skills, and how to read the infant's needs and respond appropriately should follow. Presenting a scenario of the "colicky family" often brings nods of agreement from parents as they hear a description of the disruption in family life a colicky infant can cause.

IV. Specific Treatment Guidelines

Some specific suggestions for managing the colicky infant may include the following:

A. Limit external stimuli to the infant (e.g., quiet area to feed the infant, adherence to a feeding schedule rather than

allowing demand feeding to avoid overfeeding and abdominal distention).

B. Check the infant for avoidable causes of crying (e.g., wet diaper, hunger, too hot or cold, bored [needs cuddling]).

C. Acknowledge the impact of colic on the family and the stresses it can produce.

D. Discuss possible soothing techniques with parents:

1. Rocking
2. Patting of the back
3. Swaddling
4. Background noise/music (e.g., radio)
5. Rhythmic motions (e.g., infant swing, bounding infant seat, back-and-forth movements in a stroller, car rides)

E. Increase nonnutritive sucking (pacifier).

F. Increase carrying time (e.g., by the use of slings).

G. Discuss (recommend) babysitting with parents and the need to get away from the baby once in a while, especially in single-parent households with a sole primary caregiver.

H. Assure the parents that there is light at the end of the tunnel. Colic usually subsides around 3 months of age when the infant attains the ability to self-stimulate and interact with his environment.

I. Consider formula changes only if weight gain has been inadequate or if the family insists on a trial of another formula. In this circumstance, a trial of a soy-based formula may be appropriate.

The key to successful treatment of colic is frequent follow-up either by office visits or by telephone. The physician must be empathetic, positive, and reassuring in comments to the family. Drug therapy should not be discussed at the first visit and seldom plays an important role once appropriate counseling has been performed. If needed, simethicone may be used; its placebo effect may be helpful in some situations.

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VI

Genitourinary Disorders

29 Prepubertal Vaginitis

Julie A. Jaskiewicz

Vaginitis (inflammation of the vagina) with vaginal discharge is the most frequent gynecologic complaint of young girls. Vulvovaginitis occurs when vaginal discharge and inflammation are accompanied by symptoms of dysuria, pruritus, and/or vulvar erythema. Prepubertal girls are particularly vulnerable to these conditions because of anatomic and developmental aspects unique to the premenarchal genital tract. These include (a) nonestrogenized vaginal mucosa; (b) immature labial development and lack of pubic hair, which leave the sensitive vulvar skin unprotected; (c) close proximity of the vagina and anus; and (d) less than optimal genital hygiene. With the onset of estrogenization at puberty, the vaginal mucosa thickens, allowing for the proliferation of lactobacilli that serve as a barrier between the vagina and possible infections, thus decreasing the incidence of recurrent vaginitis in postmenarchal women.

I. Evaluation

A complete history, physical examination, and judicious use of laboratory studies will usually allow an accurate assessment of vaginitis by the health care provider in the office setting.

A. History. The clinician should inquire in detail about (a) associated symptoms, including pruritus, dysuria, vulvar erythema, and anal irritation; (b) color, odor, duration, quantity, and consistency of the vaginal discharge; (c) perineal hygiene methods, including the use of bar soaps and bubble bath products; (d) recent infections; and (e) medications, including topical and systemic antibiotics. In addition, both the child and the guardian should be questioned about the possibility of trauma and sexual abuse. Parents of girls with vaginal discharge are often worried about abuse and may not feel comfortable voicing their concern directly.

B. Physical examination. Most girls with a negative history of associated symptoms and a complaint of mild vaginal discharge will require only a simple external examination in the "frog-leg" supine position. Often in these cases, the clinician will note scant, pale mucoid discharge with minimal erythema, and further examination will not be required. However, with a history of a prolonged or thick, copious discharge, a full gynecologic examination, with the child in the knee/chest position, should be done to allow for visualization of the vagina, hymen, and cervix. Careful inspection of the hymenal contour for abnormalities, including tears and scarring; the vagina, for bruising, foreign body, and type of discharge; and the anus, for fissures, tears, or laxity, is important. On rare occasions it may be necessary to perform an examination under anesthesia if the history raises concerns and/or the child is uncooperative.

C. Laboratory. All prepubertal girls who have a thick or purulent vaginal discharge visible on physical examination

or who have recurrent or persistent vaginal discharge should have swabs of the discharge obtained for Gram stain and bacterial culture, including culture for *Neisseria gonorrhoeae.* A wet prep analysis is also recommended, as *Trichomonas* has been identified as a cause of vaginitis in some young girls. Although most cases of prepubertal vaginitis are not due to sexual molestation/abuse, further investigation for abuse is necessary if an organism commonly associated with sexual transmission is isolated. Urine for routine urinalysis and urine culture should also be considered to rule out urinary tract infection. It is important, however, to obtain the urine specimen after the examination and vaginal cultures since the cleansing process for a clean-catch urine specimen is likely to remove much of the vaginal discharge and make visual inspection of the discharge inaccurate.

II. Differential Diagnosis

Table 29.1 lists common and uncommon etiologies for vaginitis in the prepubertal girl.

A. Nonspecific vaginitis. Nonspecific vaginitis accounts for up to 75% of all cases of prepubertal vaginitis. The discharge can be copious, foul-smelling, and brown or green and may be associated with pruritus. Examination of the vaginal smear frequently reveals leukocytes and bacteria. The most common organisms associated with nonspecific vaginitis are coliform bacteria, including *Escherichia coli* and enterococcus, diphtheroids, and anaerobic bacteria, suggesting fecal contamination. Most experts agree that inadequate hygiene and tight-fitting clothing (e.g., nylon underpants, tights, and plastic-covered paper diapers) may contribute to the development of nonspecific vaginitis. Another common contributing factor, exposure to irritants such as perfumed bath and laundry products and bar soap (from vigorous washing of the genital area), introduces small amounts of chemical irritants into the introitus, causing a chemical/irritant vaginitis that becomes superinfected from local organisms.

Table 29.1. Etiologies of prepubertal vaginitis

Common	Less Common	Rare
Nonspecific vaginitis	*Shigella* sp.	Congenital anomalies
Streptococcus pyogenes	*Giardia*	Double vagina with fistula
Coagulase-positive *Staphylococcus*	*Enterobius vermicularis*	Ectopic ureter
Foreign body	*Candida albicans*	Pelvic fistula
Physiologic leukorrhea	Systemic disease	Vaginal and cervical polyps/tumors
	Sexual abuse	Urethral prolapse

B. ***Streptococcus pyogenes*** **(group A beta-hemolytic** ***Streptococcus*****) and coagulase-positive** ***Staphylococcus.*** These are the second most common etiologies associated with prepubertal vaginitis. The signs and symptoms of vaginitis caused by these bacteria are similar to those seen in nonspecific vaginitis and are felt to be the result of direct transfer of the organisms from the nasopharynx to the genital tract.

C. Foreign body. A foreign body in the vagina can cause a persistent, foul-smelling, purulent dark-brown discharge, often with vulvar erythema and irritation. Vaginal smear reveals leukocytes, bacteria, and epithelial cells. In prepubertal girls, the most frequently recovered vaginal foreign body is toilet paper (usually from vigorous wiping). Treatment requires removal of the foreign body and warm water irrigation of the vagina for comfort.

D. Physiologic leukorrhea. Physiologic leukorrhea is a benign condition causing vaginal discharge without associated symptoms in the older premenarchal girl. Characterized by a moderate, odorless, clear to white discharge beginning within 1 year of menarche, it indicates a normal response of increased endocervical mucus production following an increase in estrogen secretion. No specific treatment is indicated, and the young woman should be reassured that this discharge represents normal physiologic maturation.

E. Other causes. Less common but important causes of vaginitis in this age group include *Shigella* sp, which causes a mucopurulent, often bloody discharge, and *Giardia.* A careful history and vaginal cultures should identify these organisms. *Enterobius vermicularis* (pinworm) infestation should be considered, particularly in the child with anal pruritus and erythema. (Fecal organisms coating the pinworm cause local infection.) *Candida albicans* vaginitis is characterized by severe pruritus associated with a white or yellow thick discharge and significant inflammation on examination. It is an uncommon cause of prepubertal vaginitis but should be considered in a young girl with these classic symptoms and a history of diabetes mellitus, recent broad-spectrum antibiotic use, or an immunocompromised state. In the absence of these conditions, empiric treatment for fungal vaginitis should be avoided.

F. Systemic disease. Vaginitis may also occur as a manifestation of systemic disease, most notably scarlet fever, Kawasaki disease, and Crohn disease.

G. Rare causes. The rare causes of prepubertal vaginitis listed in Table 29.1 are usually suggested by history and/or physical examination and should be considered in persistent cases of vaginitis that do not respond to conventional therapy. In some cases, referral to a pediatric gynecologist for anatomic diagnosis and repair is necessary.

III. Treatment

The best management of vaginitis in the young child includes accurate identification of the etiology and treatment spe-

cific for that cause. Most cases of nonspecific vaginitis respond to improved perineal hygiene alone. The clinician should suggest (a) use of only cotton underpants, changed daily; (b) avoidance of nylon underpants, nylon tights, and other tight-fitting clothes; (c) appropriate cleansing of the genital area (avoiding bar soaps and bubble baths; using tepid water alone in vaginal area); (d) emphasis on good hand washing (albeit not so easy in the young toddler); (e) encouragement of front-to-back wiping after toileting; and (f) maintenance of a dry vulvar area after bathing. Sitz baths can alleviate discomfort, and barrier creams (e.g., A&D ointment, Vaseline, or Desitin) applied to irritated perivaginal areas can ease pain during urination and wiping. For persistent vaginitis after 4 weeks of improved hygiene, a 10-day course of oral antibiotics may be curative. Amoxicillin or a cephalosporin are appropriate antimicrobial choices.

For cases other than nonspecific vaginitis, treatment will depend upon the organism cultured from the vagina. Intramuscular benzathine penicillin or oral penicillin V (or amoxicillin) is curative for group A β-streptococcus, given as if for streptococcal pharyngitis. Cephalexin or amoxicillin clavulanate can be given for *Staphylococcus* sp. Pinworm infection is treated with Mebendazole 1 chewable 100-mg tablet, repeated in 2 weeks. Treatment of *Candida* vaginitis with topical clotrimazole cream is often effective, although prepubertal girls with *Candida* vaginitis will require a careful history to identify underlying precipitating factors. A positive vaginal culture for *Neisseria gonorrhoeae* in any prepubertal child should be treated with intramuscular injection of a third-generation cephalosporin and referral to child protective services for evaluation for sexual abuse.

IV. Summary

Vaginitis is common in prepubertal children. The clinician who understands the normal anatomy and physiology of the prepubertal genital tract, obtains a complete and pertinent history, and performs an appropriate genital examination will identify the etiology in most cases. Most episodes of prepubertal vaginitis are not associated with sexual abuse, and many cases can be adequately managed with proper perineal hygiene alone. The clinician should be proficient in obtaining vaginal specimens for culture, familiar with the most common infectious causes and treatment of vaginitis in children, and aware of conditions that require referral to a pediatric gynecologic specialist.

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30 Primary Nocturnal Enuresis

Raymond C. Baker

Primary nocturnal enuresis (PNE) is nighttime urinary incontinence in the absence of urinary pathology, which occurs continuously from birth to beyond the age at which the majority of children achieve nocturnal continence. The definition must be vague regarding age because the age of achieving nighttime urinary continence varies. By the age of 5 years, 67% of children are continent; at 8 years of age, 72%; and at 11 years of age, 93%. At age 17 years, 0.7% still have occasional nighttime urinary incontinence. At all ages, boys are affected more frequently than girls.

I. Etiology

The etiology of primary nocturnal enuresis is probably multifactorial, and subcategories of the condition possibly exist that will eventually be shown to match different etiologies. **Organic/structural factors** are uncommon, and many clinicians would exclude children with structural abnormalities from the diagnosis of PNE. A **familial tendency** has been well recognized, with 77% of children affected if both parents have a history of PNE; 44% with one parent; and 15% with a negative family history of PNE. A **maturational/developmental** influence is suggested by the finding that various indices of growth are delayed in children with PNE, such as Tanner stage of sexual development, bone growth, and height. **Sleep factors** have been studied in children with PNE, and results suggest that sleep stage (enuresis occurs during non-REM sleep) may play a role. This is somewhat confirmed by the fact that 60% of parents of children with PNE report that their children are hard to awaken from sleep, compared with just under 4% of nonaffected children. **ADH/renal effects** have been implicated in some children who exhibited increased nocturnal urine output. Some of these children were shown to have insufficient ADH production; others showed impaired renal sensitivity to arginine vasopressin and desmopressin. Logic would suggest that **bladder capacity** would play a role. A decreased functional bladder capacity has been found in as many as 85% of children with PNE. Finally, **behavioral factors** have been suggested as playing a role, although results of studies of associated behavioral problems in children with nighttime incontinence are conflicting. In 1966, Byrd et al. found an increased incidence of behavior problems in a study of almost 11,000 children 5 through 17 years of age whose parents completed the Behavior Problem Index developed by Zill. In a study of 92 children referred for nocturnal enuresis tested with the Eyberg Child Behavior Inventory, Friman found no significant behavioral comorbidity.

II. History

Most physicians would not consider nocturnal incontinence "abnormal" or suggest medical evaluation until about the age of 6 years. The evaluation should begin with a complete history to rule out anatomic or infectious abnormalities. Specific symptoms that suggest infection are dysuria, frequency, urgency,

daytime incontinence, and dribbling. Additional history that might suggest an etiology are associated congenital anomalies in the patient or family, incontinence in other family members, a history of constipation, psychologic problems, social problems, and a history of sexual abuse. The physician should ask what efforts have been made by the parents or other health care providers to manage the incontinence. Obvious causes of incontinence should be sought, such as fear of a dark room or inability to manipulate the pajamas.

III. Physical Examination and Laboratory Evaluation

The physical should specifically include blood pressure and growth parameters in a general examination in addition to paying special attention to the abdominal, genital, rectal, and neurologic components of the examination. From a laboratory perspective, in most circumstances only a urinalysis and urine culture are indicated unless the history and physical suggest other etiologies for the enuresis. Determination of bladder capacity is also important, since a decreased capacity portends more difficulty with treatment. At the same time however, identification of a small bladder capacity is often a relief to children and parents when they realize that the enuresis has a physical explanation. As Schmitt says, "They can start blaming the bladder rather than the child." Bladder capacity can be estimated by having the child void into a measuring device on three separate occasions and reporting the results to the physician. Normal bladder capacity in ounces can be calculated by adding 2 to the age of the child in years.

IV. Treatment

The treatment of primary nocturnal enuresis is variable according to the child's age and cognitive abilities. Before the age of 6 years, no treatment is recommended other than counseling regarding normal development of the urinary tract and continence. The physician should specifically make the parent aware that the child is not bedwetting consciously and that punishment is therefore not indicated. A discussion of practical issues such as the use of plastic sheets (or plastic garbage bags) beneath the sheets may help the parent cope with the inconvenience caused by the enuretic child. Prolonged use of diapers or pull-ups beyond bowel incontinence is usually not recommended, especially if it encourages teasing by siblings and peers. Several principles of treatment (Schmitt) should be discussed with the parents and child before a specific plan is developed.

A. Set a goal. The goal of therapy is for the child to get up at night and use the bathroom.

B. Make access to the toilet easy. This may mean leaving a light on in the bathroom, putting a portable toilet in the child's room if the bathroom is distant from the child's room, and making sure the child's pajamas can be manipulated for easy toileting.

C. Remind the child that it is his/her responsibility to learn to be dry at night.

D. Avoid excessive fluids within 2 hours of bedtime and empty the bladder at bedtime.

E. Have the child help remove wet sheets in the morning, put them in the clothes hamper, and help remake the bed. Also, the child needs to wash in the morning to remove the smell of urine. These tasks should be presented as a routine part of "what we do on mornings the bed is wet" rather than as a disciplinary action.

F. Protect the child's self-esteem. Enlist the aid of siblings to be supportive rather than derisive.

G. Provide handouts for parents outlining the plan.

H. Ask the parents to purchase a calendar (with the child) and a box of gold stars to monitor mornings the bed is dry and also nights the child wakened to use the bathroom. Incentives for several gold stars might include hugs, special privileges (an extra half-hour of book reading, game playing, or TV), a trip to the local fast-food restaurant, and so on.

I. Follow-up

1. *Behavioral treatment.* This approach is most likely to work with children with normal bladder capacities and normal sleep patterns. Several techniques work, probably primarily by raising to a higher level of the subconscious the issues of enuresis and nighttime waking to use the bathroom. One technique is to teach the child self-awakening by rehearsing the process of going to the bathroom at night (supervised). The child should practice two or three times before bedtime a ritual of lying in bed and pretending he/she feels the need to urinate. Then the child pretends to wake up, runs to the bathroom, and sits on the toilet. The same rehearsal technique can be practiced during the daytime by having the child go lie in bed and pretend to sleep when he/she feels the urge to void. Then, after a minute or two, the child should run to the bathroom to urinate.

   Alternatively, the parents can waken the child to go to the bathroom in the night, especially when the parents go to bed. It is important that the parent waken the child and make him/her walk to the bathroom rather than carrying or dragging the child. After this task is easily accomplished, the parent can gradually reduce the stimulus needed to waken the child so that eventually a pattern emerges with the child needing only gentle techniques to waken. At this point, the child is usually able to waken on his own or is ready for an enuresis alarm.

2. *Enuresis alarms.* These devices have the highest cure rate of any treatment for enuresis. Several alarms are available (Table 30.1), costing from $50 to $70 (see later). The stimulus to waken the child is either an alarm or a vibration. For the enuresis alarm to be effective, however, the child must be able to waken to the sound of an alarm and go to the

Table 30.1. Enuresis alarm ordering information

Palco Labs (Wet-Stop) 8030 Soquel Ave. Santa Cruz, California 95062 800-346-4488 $65.00	Nytone Medical Products (Nytone Enuretic Alarm) 2424 South 900 West Salt Lake City, Utah 84119 800-497-6573 $56.50	Ideas for Living, Inc. (Potty Pager) 1285 N. Cedarbrook Rd. Boulder, Colorado 80304 801-973-4090 $51.00

bathroom. Parents should practice with an alarm clock before purchasing the enuresis alarm to make sure the child will not sleep through the stimulus. Also, parents must supervise the child practicing with the alarm so that the child develops a ritual of wakening and getting to the bathroom, similar to the behavioral techniques described earlier. The success rate of enuresis alarms is 70% to 80%.

3. *Drug therapy.* Two drugs are used for enuresis. *Imipramine* was the first drug used in enuresis. In doses of 25 to 50 mg (for those 8 to 12 years of age) or 75 mg (for those more than 12 years of age) orally at bedtime, the cure rate is 10% to 60%. However, the relapse rate after stopping the drug is high (90%), and toxicities are significant with accidental ingestion. The drug must be kept well out of reach of younger children. Furthermore, long-term use is not recommended, which has limited the use of this drug in enuresis. Because of the problems with imipramine, *desmopressin* (DDAVP) was welcomed when it was approved for use in enuresis. Given as a nasal spray in a dosage of 20 to 40 μg, desmopressin is completely effective in about 25% of patients, but it also has a 90% relapse rate. It is also considerably more expensive, costing as much as $200 per month.

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31 Sexually Transmitted Diseases

Robert M. Siegel

The incidence of sexually transmitted diseases (STDs) continues to rise in children and adolescents. As many as half of adolescents are sexually active by 16 years of age and are at risk for STDs. Groups at particularly high risk include (a) those beginning sexual activity younger than 16 years of age, (b) those abusing alcohol or other street drugs, (c) children who have been sexually abused, (d) adolescents with multiple partners, (e) adolescents with a previous history of an STD, and (f) adolescents who are practicing survival sex (e.g., runaways, street children, and prostitutes).

The subject of sexual abuse in children is addressed in Chapter 52. It should be remembered that about half of all rapes occur to teenagers. All teens who are evaluated for STDs should be questioned as to whether their sexual activity was consensual.

Symptoms of STDs are quite variable, depending on the infectious agent, stage of the infection, and host. In general, an STD evaluation should be considered in a child or adolescent if the patient (a) complains of a genital discharge or dysuria; (b) has a history of sexual activity, particularly with multiple partners; (c) has a history of STD exposure; or (d) is pregnant.

I. Gonococcal Infection in Adolescents

Gonorrhea may present as asymptomatic infection, cervicitis, urethritis, pelvic inflammatory disease, or disseminated disease. The diagnosis is made by culturing the organism on chocolate agar (Thayer-Martin) media. Newer, more sensitive and specific technology for diagnosis, such as DNA probes, may be used in the evaluation of adolescents. Standard cultures, however, are the only legally accepted standard in evaluating children in whom child abuse is suspected. Treatment for gonorrhea depends on the site of infection and clinical syndrome.

A. Uncomplicated gonorrhea. Any one of the following single-dose treatment regimens is adequate in adolescents for uncomplicated disease:

1. Ceftriaxone 125 mg IM *or*
2. Cefixime 400 mg PO *or*
3. Ciprofloxacin 500 mg PO *or*
4. Ofloxacin 400 mg PO *or*
5. Spectinomycin 2 g IM *plus* doxycycline 100 mg PO BID × 7 days *or* Azithromycin 1 g PO as a single dose (coverage for coexistent *Chlamydia*).

All adolescents with gonorrhea should also be evaluated for *Chlamydia* infection and syphilis. It should be noted that quinolones have no activity against syphilis and that no definitive data exists on the effectiveness of cefixime on incubating syphilis.

B. Disseminated gonorrhea. This syndrome results from gonococcal bacteremia and can yield petechial skin lesions,

arthralgias, tenosynovitis, or septic arthritis. Treatment is for a minimum of 7 days. Initial therapy should be

1. Ceftriaxone 1 g intramuscularly (IM) every 24 hours *or*
2. Cefotaxime 1 g intravenously (IV) every 8 hours *or*
3. Ceftizoxime 1 g IV every 8 hours *or*
4. Spectinomycin 2 g IM every 12 hours *plus* doxycycline *or* azithromycin as earlier (coverage for coexistent *Chlamydia*).

This regimen is continued until there is clinical improvement (usually 24 to 48 hours) and then is changed to oral therapy as follows:

- Cefixime 400 mg PO BID *or*
- Ciprofloxacin 500 mg PO BID.

C. Gonococcal infections in children. Any child outside of the newborn period with gonorrhea should be evaluated for sexual abuse. Treatment of uncomplicated disease is similar to that for adults except that quinolones should not be used in those less than 16 years of age. The efficacy of cefixime has not been proved in clinical trials. Ceftriaxone 125 IM as a single dose may be given in children. Assuming that cefixime in a dose of 8 mg/kg (maximum 400 mg) is also effective is reasonable. In infants with ophthalmia neonatorum, ceftriaxone, at a dose of 25 to 50 mg/kg IM or IV, not to exceed 125 mg, given once (with additional saline lavage) is effective. Infants with disseminated disease should be given ceftriaxone at 25 to 50 mg/kg IM or IV once daily for 7 days; and for 10 to 14 days, if meningitis is documented.

II. Chlamydial Infection in Adolescents

Chlamydia trachomatis is the most common cause of nongonococcal urethritis and cervicitis. It is also a frequent copathogen in pelvic inflammatory disease. Diagnosis can be made by culturing the suspected site or by a rapid antigen test. (NB: Rapid antigen tests are not accurate enough for sexual abuse evaluation.) Such a high prevalence of *C. trachomatis* coinfection in individuals with gonococcal infection has been established that those treated for gonorrhea should also be treated for *C. trachomatis.* First-line treatment in adolescents is

A. Doxycyline 100 mg PO BID for 7 days *or*
B. Azithromycin 1 g PO, single dose *or*
C. Alternative regimes, which include

1. Erythromycin base 500 mg PO QID for 7 days *or*
2. Erythromycin ethylsuccinate 800 mg PO QID for 7 days *or*
3. Sulfisoxazole 500 mg PO BID for 10 days *or*
4. Amoxicillin 500 mg PO TID for 7 to 10 days (in pregnant teens).

III. Chlamydial Infections in Children

Chlamydial infection from maternal infant transmission is well described. Most experts agree that this is an unlikely route beyond 3 years of age and that any infection with *C. trachomatis*

beyond this age is likely to be from sexual abuse. Culture is the only test for *C. trachomatis* that is accurate enough for a sexual abuse evaluation. Perinatal transmission of the organism from mother to infant can lead to conjunctivitis or pneumonitis. Treatment in infants and children is erythromycin 50 mg/kg PO divided QID for 10 to 14 days.

IV. Syphilis

The diagnosis and treatment of syphilis are dependent on the age of the patient and the stage of the disease. A definitive diagnosis can be made by identifying spirochetes on dark-field microscopy or by doing a direct fluorescent antibody test of a specimen. The diagnosis, however, is usually made by serology either with a nontreponemal test such as VDRL or RPR or with a specific treponemal test such as the fluorescent treponemal antibody absorption (FTA-ABS) test.

A. Congenital syphilis. All pregnant women should be screened with a nontreponemal test before delivery. Following delivery, the newborn infant should be evaluated for congenital syphilis if the infant's mother:

1. *Had untreated syphilis.*
2. *Had syphilis and was treated with erythromycin.*
3. *Was treated for syphilis within 1 month of delivery.*
4. *Was treated for syphilis and did not demonstrate an adequate reduction in nontreponemal antibody titer.*
5. *Had syphilis and did not have a well-documented history of treatment.*

Evaluation should include serologic studies, long-bone x-ray for evidence of metaphysitis, and VDRL and cytologic examination of cerebrospinal fluid . Chest x-ray and liver function tests may also be indicated.

Treatment for congenital syphilis consists of aqueous crystalline penicillin (PCN) G, 100,000 to 150,000 U/kg per day IV divided Q 8 to 12H during the first 7 days of life then Q8H afterward for a total course of 10 to 14 days. If more than 1 day of therapy is missed, the entire course should be restarted. If the diagnosis is delayed and the child is treated after 4 weeks of age, treatment consists of aqueous crystalline PCN G, 200,000 to 300,000 U/kg per day IV divided Q6H for 10 to 14 days. For PCN-allergic patients, doxycycline 100 mg BID for 4 weeks is recommended.

B. Syphilis in teenagers

1. *Early-acquired syphilis.* Syphilis of less than 1 year's duration (primary, secondary, or early latent) should be treated with benzathine PCN G 2.4 million U IM in a single dose. For those who are PCN allergic, doxycycline 100 mg PO BID, or tetracycline 500 mg PO QID for 2 weeks are alternatives.
2. *Syphilis of greater than 1 year's duration* (or if the duration is unknown) should be treated with benzathine PCN 2.4 million U IM weekly for 3 consecutive weeks.

3. *Neurosyphilis.* Central nervous system (CNS) involvement with syphilis may occur at any stage of illness. Any patient with CNS symptoms should have an examination of the cerebrospinal fluid. The recommended regimen is aqueous PCN G 12 to 24 million U daily divided Q4H for 10 to 14 days. Procaine PCN 2.4 million U IM daily plus probenecid 500 mg PO QID for 10 to 14 days is an alternative.

V. Other Sexually Transmitted Diseases in Teenagers

A. Trichomoniasis. Infection with *Trichomonas vaginalis* is frequently asymptomatic, particularly in males. Symptoms in women are often most severe just before menstruation. The diagnosis is made by examination of a wet-mount preparation of vaginal discharge or by appearance of the organism on microscopic examination of the urine. Treatment is metronidazole 15 mg/kg per day divided TID with a maximum of 250 mg per dose. Alternative regimens in adolescents are 2 g of metronidazole as a single oral dose or 500 mg BID for 7 days. Some clinicians empirically treat individuals for *Trichomonas* when treating for suspected gonorrhea (GC) or *Chlamydia.*

B. Genital herpes simplex virus. Approximately 30 million Americans have genital herpes infection. Primary disease, often the most severe, may even require hospitalization. Painful recurrences may occur. Although there is no cure, antiviral therapy can reduce the severity of illness. For primary illness, the therapy is acyclovir 200 mg PO five times a day for 7 to 10 days. For those requiring hospitalization, IV acyclovir at 5 to 10 mg/kg Q8H for 5 to 7 days may be required. Treatment regimens for recurrences are acyclovir at 200 mg PO five times a day or 400 mg TID or 800 mg BID for 5 days.

C. Bacterial vaginosis. Bacterial vaginosis (BV) is caused by the replacement of *Lactobacillus* species by anaerobic bacteria, *Gardnerella vaginalis,* and *Mycoplasma hominis.* BV gives a white homogeneous discharge. The diagnosis is made from the presence of clue cells on a wet mount of vaginal discharge or by a fishy odor with the addition of 10% potassium hydroxide (KOH) to the discharge. Treatment regimens are metronidazole 500 mg PO BID for 7 days and clindamycin cream 2% one full applicator (5 g) intravaginally every night for 7 days or metronidazole gel 0.75% one full applicator (5 g) intravaginally BID for 5 days. Alternative, but less efficacious, regimens are metronidazole 2 g PO once or clindamycin 300 mg PO BID for 7 days.

D. Pelvic inflammatory disease. Pelvic inflammatory disease (PID), infection of the upper genital tract, is most frequently caused by *N. gonorrhoeae, C. trachomatis,* and vaginal flora such as anaerobic bacteria. Empiric treatment for PID should be initiated if the following criteria are met in the absence of another explanation: (a) lower abdominal pain, (b) adnexal tenderness, and (c) cervical motion tenderness. Some experts suggest that all teenagers with PID should be

hospitalized to ensure compliance and minimize the risk of complications, such as infertility. Hospitalization is clearly indicated if any of the following factors exist: (a) pelvic abscess, (b) peritonitis, (c) pregnancy, (d) an intrauterine device in place, (e) patient's inability to tolerate oral therapy, or (f) lack of response to outpatient therapy after 48 hours. The recommended CDC initial regimens are as follows:

1. *Inpatient:*
   Cefoxitin 2 g IV Q6H (or cefotetan 2 g IV Q12H) *plus* doxycyline 100 mg IV or PO Q12H *or*
   Clindamycin 900 mg IV Q8H *plus* gentamicin loading dose of 2 mg/kg IV followed by 1.5 mg/kg IV Q8H
2. *Outpatient:*
   Cefoxitin 2 g IV plus probenecid 1 gram PO or ceftriaxone 250 mg IM *plus* doxycycline 100 mg PO BID *or*
   Ofloxacin 400 mg PO BID *plus* either clindamycin 450 mg PO QID or metronidazole 500 mg PO BID.

Therapy, regardless of the regimen, should be for 14 days.

E. General considerations. Any adolescent with an STD is engaging in high-risk sexually activity and is at risk for both pregnancy and other STDs. All teens who are treated should be counseled on risk reduction. HIV testing should also be considered in those who are diagnosed with an STD. If possible, the patient's sexual contacts should also be evaluated and treated.

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Web Site

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32 Urinary Tract Infections in Children

Robert M. Siegel

Urinary tract infection (UTI) occurs in about 1% of boys and 3% of girls. Because the symptoms can be nonspecific in young children, the diagnosis can be elusive and should be considered in all young children with fever without a source.

I. Pathophysiology

The most common predisposing factor for UTI in children is an underlying anatomic abnormality. Obstructive lesions such as posterior urethral valves are common in boys, and vesicoureteral reflux is common in girls. Poor hygiene and colonization with aggressive coliform organisms may play a role in UTI. Not being circumcised increases an infant boy's chances of UTI more than 30-fold. The most common organism isolated in males and females is *Escherichia coli,* although other species such as *Klebsiella, Enterobacter, Pseudomonas,* and *Proteus* may be isolated, particularly in those with obstructive lesions, recurrent disease, or a history of instrumentation.

II. Clinical Presentation

The signs and symptoms of UTI in the older school-aged child are similar to those of adults—dysuria and increased urinary frequency. Fever and flank pain may be present with upper tract disease. In the younger child however, the signs are nonspecific and may include fever, poor feeding, vomiting, and irritability. At the same time, no reliable clinical indicators differentiate between lower and upper tract disease in the younger child.

III. Evaluation

A. History. The history should be directed toward questioning that suggests UTI (dysuria, frequency, enuresis, previous history of UTI, family history of UTI); questions regarding nonspecific symptoms (fever, irritability, poor feeding, vomiting); and questions suggesting other etiologies, such as the use of products that can cause urethral irritation (bubble bath), sexual activity (STDs), genitourinary hygiene, and sexual abuse (post-sexual abuse syndrome).

B. Physical examination. The child's temperature, blood pressure, weight, and state of hydration should be recorded. Signs of upper tract disease may be indicated by focal abdominal pain/tenderness or flank tenderness. Genitalia should be examined for any abnormalities, including increased erythema, discharge, foreign body, or genital injury.

C. Laboratory tests. The definitive diagnosis of UTI is made by urine culture. Although a urinalysis with microscopic examination may be helpful, the presence of pyuria is neither sensitive nor specific. A positive nitrite test as part of the urinalysis is 75% to 85% specific and 31% sensitive for UTI. Leukocyte esterase on the urine dipstick has a sensitivity of about 50%. In freshly obtained urine, more than per bacterium/

high power field (HPF) in unspun urine also correlates with UTI. However, half of infants under 2 months of age with UTI will have a normal urinalysis.

Opinion on the best way to collect the urine specimen varies. Suprapubic aspiration remains the gold standard, and any growth represents infection regardless of colony count. Many practitioners, however, find the technique too invasive and prefer bladder catheterization. Colony counts of more than 10^3 colonies/mL represent infection. Clean-catch midstream collection in the continent, older child is an acceptable specimen for urine culture, particularly in males in whom the result is equivalent to catheterization. In females the risk of contamination is greater, but this risk can be minimized if the child is cleaned well and the specimen is obtained with the child sitting backward on the toilet, which causes the labia to separate. If this is not possible, then two specimens from two separate voids are satisfactory alternatives. Colony counts of more than 10^5/mL of one organism suggest a true infection. Bag specimens of both boys and girls are too likely to represent contamination and are not recommended except as a screen.

Routine laboratory tests cannot distinguish between cystitis and pyelonephritis. Therefore in the febrile younger infant, a blood culture should be considered to rule out urosepsis.

IV. Treatment

A. Supportive. The decision to admit a child with UTI to the hospital will depend on age and clinical status. In the older child who is not vomiting, not dehydrated, and likely to be compliant, outpatient management is a reasonable choice. On the other hand, several factors may suggest hospitalization: (a) infants under 4 months of age; (b) infants and children with significant systemic symptoms of fever, toxicity, and flank tenderness suggestive of pyelonephritis; (c) children requiring intravenous fluids secondary to poor intake and vomiting; and (d) children unable to take oral medications.

B. Antibiotics. Antibiotic therapy should be directed toward the most likely causative organisms, pending culture and sensitivity results. For outpatient therapy, trimethoprim/sulfamethoxazole or a third-generation cephalosporin is a good first-line oral agent. For the hospitalized patient, ampicillin combined with gentamicin should be given until the organism is identified. A broad-spectrum cephalosporin is a reasonable intravenous alternative. Duration of therapy is 10 to 14 days. Short-course therapy in children is not recommended.

V. Follow-up

A. Laboratory tests. A urine culture should be obtained after treatment to document urine sterility. Ideally, a urine culture after 48 hours of therapy would be preferable. However, in most circumstances this is not feasible, and telephone contact at 48 to 72 hours to document disappearance

of symptoms is sufficient. Children are at risk for relapsing with 75% of recurrences occurring within 1 year of infection. Although no convincing efficacy of routine screening of urine after infection is documented, it is reasonable to obtain a follow-up urine culture 1 week after stopping therapy. Following a UTI, the practitioner should aggressively seek the diagnosis of UTI if the child develops any symptoms.

B. Radiologic evaluation. Girls under 6 years of age and boys of any age with a UTI should be evaluated for the presence of an underlying real abnormality. Other indications for evaluation include any child thought to have pyelonephritis or a second UTI. The initial evaluation can be a renal ultrasound and voiding cystourethrogram (VCUG). A nuclear cystogram may be substituted for the traditional VCUG in girls because they are at low risk for posterior urethral valves and the procedure involves less radiation exposure. Both the ultrasound and the VCUG may be done during the acute illness. A dimercaptosuccinic acid (DMSA) scan detects nonfunctioning and poorly functioning renal tissue such as that seen with pyelonephritis and may be appropriate in certain circumstances, although it is not routinely recommended.

C. Chemoprophylaxis. Recurrent UTIs are common (30% experience a recurrence after the first UTI). In those patients who are at risk for renal damage secondary to reflux of bacteria (significant reflux present on cystography), prophylactic antibiotic therapy (nitrofurantoin, trimethoprim/sulfamethoxazole) should be considered. Recent guidelines on UTI management by the American Academy of Pediatrics suggest that children diagnosed with UTI should be placed on prophylactic antibiotics at least until their radiologic imaging is completed. Ongoing prophylaxis should then be directed by pathology detected by the imaging procedure(s) since prevention of UTI recurrences may limit progression of renal scarring. Urologic referral may be considered for children with recurrent UTIs and for children with anatomic abnormalities including grade III, IV, or V reflux. Siblings of children with renal abnormalities, including vesicoureteral reflux, are also at risk for urinary tract abnormalities and should be watched for symptoms of UTI.

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VII

Endocrine/ Growth Disorders

33 Assessment of Variations of Pubertal Development

Anita Cavallo

Puberty is dependent on the activation of the hypothalamic/pituitary/gonadal axis, with resultant maturation of the gonads and increased steroidogenesis. The increased secretion of sex steroids causes the development of the secondary sexual characteristics. A disturbance in the hypothalamus, pituitary, or gonad can alter the onset and cadence of puberty. **Pubarche** is the development of pubic and axillary hair, normally dependent on the activation of the pituitary/adrenal axis (**adrenarche**), with increased secretion of a weak adrenal androgen, dehydroepiandrosterone sulfate (DHEAS). Although puberty and adrenarche occur in close temporal proximity during normal development, they may be dissociated when there is an abnormality in either axis. The adrenal also has the capability to produce other androgens and estrogens. Thus abnormal sex steroid production by the adrenal gland may result in the clinical signs of puberty.

I. Definitions

A. Precocious puberty is the onset of puberty before the ages of 6 years in black girls, 7 years in white girls, and 9 years in boys.

B. Premature thelarche is the isolated appearance of breast development before the ages of 6 years in black girls and 7 years in white girls.

C. Premature pubarche is the onset of pubic hair before the ages of 6 years in black girls, 7 years in white girls, and 9 years in boys.

D. Gynecomastia is the presence of glandular breast tissue in the male.

E. Menarche is the onset of menses, which is usually preceded by breast development but may be the first manifestation of puberty in a girl.

F. Delayed puberty is the lack of signs of puberty in girls ≥13 years of age or boys ≥14 years of age.

G. Virilization is the abnormal appearance of signs of androgen excess in either sex, characterized by acceleration of linear growth, increased muscle mass, advanced bone maturation, acne, hirsutism, pubic hair, and clitoral or penile enlargement.

II. Normal Puberty

A. Sequence of physical changes of puberty. The first sign of puberty is breast enlargement in the girl and testicular enlargement in the boy (>2.5 cm in length). The Tanner staging of puberty describes the progression of these changes. (See Appendix F.) Pubarche generally begins after onset of breast or testicular development. (See Appendix G for the normal sequence of physical maturation.)

B. Common misconceptions by patients/parents.

1. *Pubertal onset.* Most lay people relate puberty to the appearance of pubic hair, acne, facial hair, or change in voice in boys and to the appearance of pubic hair or onset of menses in girls. Hence puberty may be quite advanced without the individual's awareness.
2. *Unilateral breast development.* The initial breast development may be unilateral, causing great anxiety in the child and parent.
3. *Subareolar mass.* The initial breast development may be rather firm, described as a hard lump, which is often feared to be a tumor or an abscess. Referral for biopsy or excision is not indicated.
4. *Delayed puberty.* Pubarche in boys generally does not occur until genital development is about stage 3, and there may be unfounded concern about delayed puberty.
5. *Pubic hair.* Frequently, normal body hair in the pubic area is mistaken for pubic hair. The physician should compare the texture and density of the hair in the pubic area with that of other body parts, mainly the lumbosacral area and the legs. Pubic hair is thicker and darker than body hair. Hypertrichosis (excess body hair) is not related to abnormal sex steroid production.

C. Physiologic pubertal gynecomastia. During midpuberty about two-thirds of normal males will develop some degree of subareolar breast tissue, which may be transiently tender. The tissue may be unilateral or bilateral and resolves without intervention within months to 1 to 2 years. The patient and his parents should be advised of the physiologic and transient nature of the condition and counseled that most boys at similar ages and pubertal stages are experiencing the same condition.

III. Assessment of Premature Thelarche

Early onset of breast development may be the first sign of central precocious puberty. The approach and extent of evaluation will depend on the presence or absence of associated findings (vaginal bleeding, pubic hair, virilization, cushingoid features). When any of these findings is present, the primary care provider should consider an urgent referral to a pediatric endocrinologist. The approach to isolated premature thelarche depends on the child's age, as follows.

A. Isolated premature thelarche of infancy (Fig. 33.1). Transient breast development that may be present over several months is frequently observed in infants. Generally, it becomes more evident when the infant slims down during the second year of life. One must search for exposure to exogenous sources of estrogen, such as oral contraceptives, cosmetic creams, vitamins, other "nutritional supplements," or the use of estrogen/progestin-type oral contraceptive in a nursing mother. Note that oral contraceptives are generally not indicated for the nursing mother, and, if used, a progestin alone

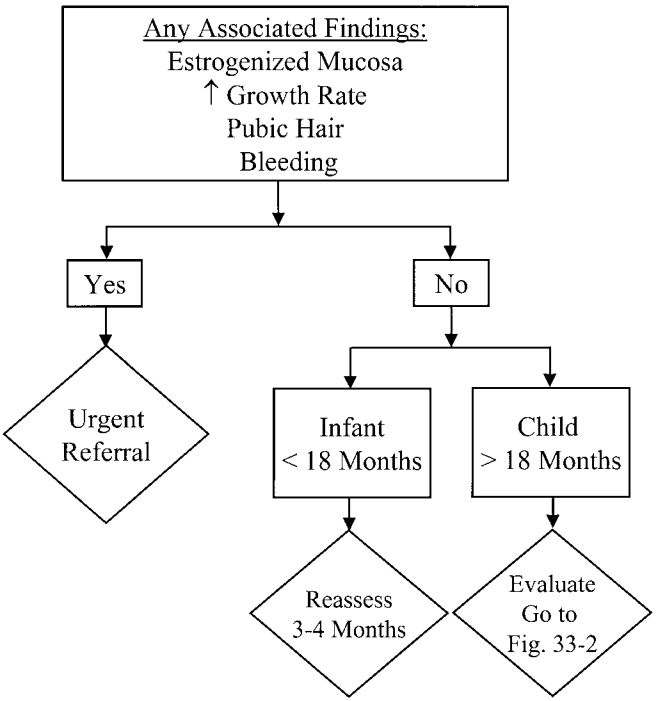

Fig. 33.1. Premature thelarche.

is preferred over a combination product. The physician should determine the growth velocity and examine the infant for associated findings, mainly pubic hair, and virilization or cushingoid features. The external genitalia should be examined for estrogenization. The typical infant with idiopathic premature thelarche will have normal growth rate, breast budding that may be sizable, but no areolar changes, no associated abnormal features, and immature vaginal epithelium. In such cases, no additional evaluations are necessary at the initial presentation, but the infant should be reexamined in 3 to 4 months. The physician should instruct the parent to return the infant for another check-up if the breast size enlarges rapidly or if there is development of pubic hair, vaginal discharge, spotting, or bleeding.

B. Isolated premature thelarche of childhood (Fig. 33.2). Beyond infancy, the appearance of isolated breast

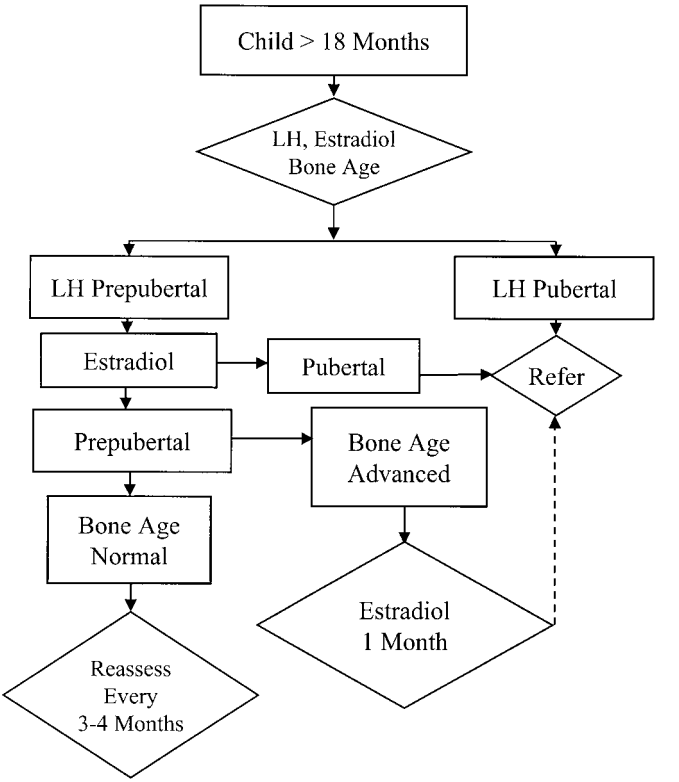

Fig. 33.2. Isolated premature thelarche.

development must be evaluated thoroughly. The initial assessment should include determination of serum estradiol and gonadotropin levels and a bone age (see Section V.C., later). Pubertal levels of estradiol or gonadotropins or advanced bone age require further evaluation immediately, which is generally done by consultation with an endocrinologist. In the absence of rapid progression and with normal bone age and prepubertal levels of estradiol and gonadotropins, reexaminations at 4- to 6-month intervals are recommended, until the child is close to the normal age of pubertal onset. Acceleration in growth rate and rapidity of progression of sexual maturation and of bone age will dictate the need to repeat the laboratory evaluations or to refer for consultation.

IV. Assessment of Precocious Pubarche

Premature pubarche may be secondary to premature activation of the pituitary/adrenal axis with increased secretion of DHEAS (premature adrenarche) or to the presence of increased secretion of androgens by either the adrenal or the gonad (virilization). See Figs. 33.3 and 33.4.

A. Age. Onset of pubic hair development in infancy is always abnormal and requires prompt and thorough investigation, which is best done by referral to an endocrinologist.

B. Associated findings. The child's evaluation will depend on the presence or absence of signs of virilization in either sex, precocious puberty, or cushingoid features.

1. *No associated findings.* The physician should obtain a bone age and serum DHEAS level. Precocious adrenarche

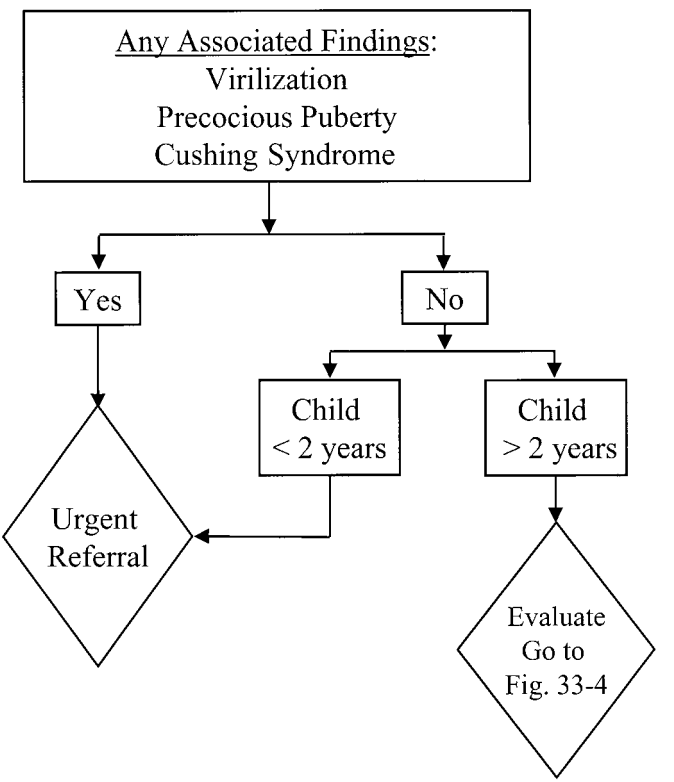

Fig. 33.3. Premature pubarche (adrenarche).

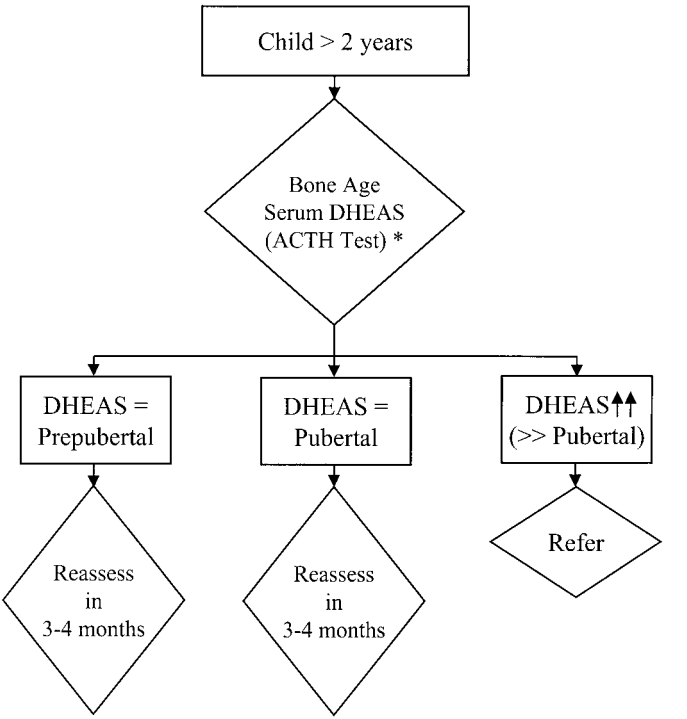

Fig. 33.4. Isolated premature pubarche.

*rarely indicated as a screening test.

usually is accompanied by slight acceleration in both growth velocity and bone maturation. The bone age will not be advanced more than a year, and the DHEAS level will be pubertal if the child has idiopathic precocious adrenarche. The child should be reexamined in 3 to 4 months to determine rapidity of progression and to monitor for associated signs of puberty, virilization, or cushingoid features.

2. *Virilization.* The presence of pubic hair in a young child along with signs of virilization requires immediate and thorough evaluation and referral. The physician should look for discrepancy between penile development and genital size. Pubic hair in a prepubertal-aged boy accompanied by testicular enlargement suggests central precocious puberty. In contrast, pubic hair in a prepubertal-aged boy accompanied by enlarged penis but prepubertal testis suggests a

virilizing disorder. Pubic hair and virilization in either sex could be related to abnormal sex steroid production by the gonad (caused by tumor) or the adrenal gland (caused by tumor, hyperplasia, or congenital adrenal hyperplasia). Serum testosterone, DHEAS, 17OH progesterone, and an adrenocorticotropic hormone (ACTH) will help in the differential diagnosis.

3. *Cushingoid features.* The presence of associated obesity with round facies, hypertrichosis, hypertension, and virilization in infancy or childhood requires urgent evaluation for adrenal hyperplasia or tumor. In addition to the laboratory tests listed earlier, serum cortisol, 24-hour urinary cortisol, and dexamethasone suppression tests should be performed, usually with an endocrinology consultation.

4. *ACTH stimulation test.* There is no consensus about the indications of the ACTH stimulation test in children with premature pubarche. This test is often performed to search for a late-onset form of congenital adrenal hyperplasia. A good physical examination and a thorough search for signs of virilization (Section I.G in this chapter) along with careful follow-up is generally the preferred approach.

5. *Insulin resistance.* Insulin resistance may accompany premature adrenarche, but the frequency of this association has not been determined. Until more data become available, the practitioner may consider obtaining a fasting serum insulin level. Monitoring children with premature adrenarche clinically and remaining vigilant about other risk factors of hyperinsulinemia, particularly obesity and acanthosis nigricans, might be more prudent.

V. Assessment of Precocious Puberty

Precocious puberty results from premature activation of the hypothalamic/pituitary/gonadal axis (**central precocious puberty**) or from increased sex steroid production without activation of the hypothalamic/pituitary/gonadal axis ("**pseudopuberty**"). Pseudopuberty may be related to exogenous sources of sex steroids (e.g., ingestion of oral contraceptives) or to a gonadal or adrenal lesion (e.g., tumor, congenital adrenal hyperplasia). In addition, precocious puberty may occur because of gonadal maturation independent of the hypothalamic/pituitary axis, such as found in the McCune-Albright syndrome (polyostotic fibrous dysplasia, abnormal pigmented skin lesions, and precocious puberty), familial male precocious puberty (testotoxicosis), and human chorionic gonadotropin–secreting tumors. Rapid progression of precocious pubertal maturation is accompanied by considerable advancement in bone maturation and significant compromise of adult height in boys and girls. Noncentral causes of precocious puberty (e.g., an adrenal or ovarian tumor) often need immediate referral for surgery. Hence the diagnostic approach is aimed first at the distinction between central and peripheral causes of sexual precocity or at the characterization of gonadotropin-independent precocious puberty (Fig. 33.5).

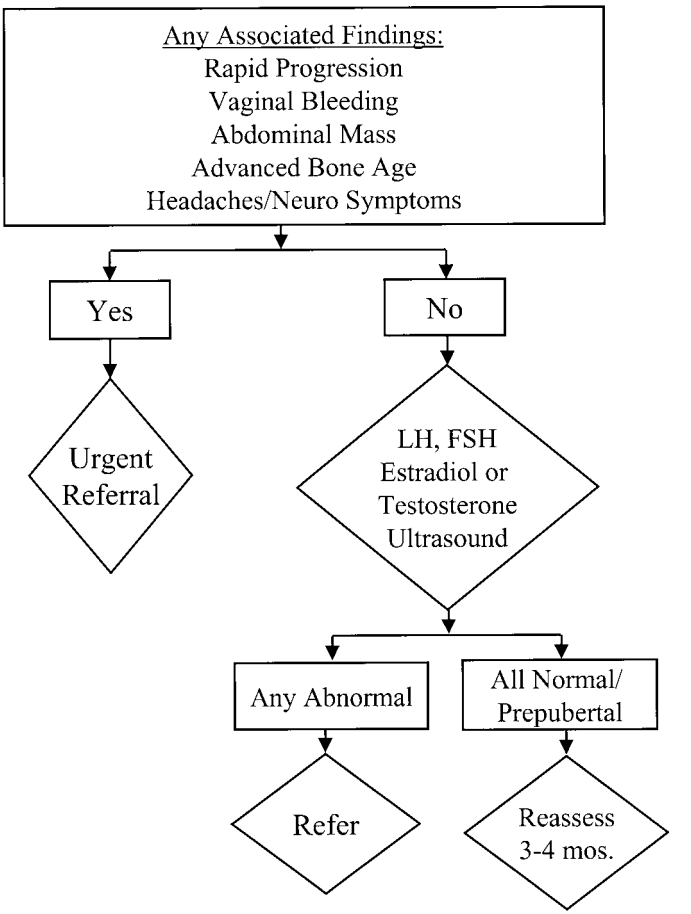

Fig. 33.5. Precocious puberty.

A. History. Important information includes age of onset, rapidity of changes, associated symptoms (e.g., headaches, abdominal symptoms, etc.). The family history is important to search for early maturers, neurofibromatosis, familial testotoxicosis, and congenital adrenal hyperplasia.

B. Physical examination. At each visit, the physical examination should include calculation of the growth rate (Chapter 34); staging of genital or breast development and

pubic hair development; a search for signs of virilization (clitoral enlargement, facial hair, acne, hirsutism); and description of the appearance of the vaginal mucosa. The immature mucosa is thin and bright red; the estrogenized mucosa is thicker and pale pink with a pearly appearance). Optic disks and visual fields must be examined. The skin should be examined for abnormal pigmentation (McCune-Albright syndrome, neurofibromatosis). In boys with central precocious puberty or familial testotoxicosis, the testicular enlargement is bilateral; unilateral enlargement should be evaluated for an androgen-secreting tumor.

C. Laboratory and other tests. The initial history and physical examination will determine the need and scope of additional evaluations.

1. *Bone age.* A baseline bone age is helpful in most cases. An advancement of bone age of more than 1.5 years over the chronological age at the initial evaluation will dictate more extensive laboratory evaluation immediately.
2. *Sex steroid and gonadotropin levels.* Significant variability in the quality of assays for gonadotropins and sex steroids still exists. Having a reliable laboratory set up for pediatric ranges of these hormones is important. Pubertal levels of estradiol or testosterone are important diagnostic findings, but they do not localize the source of the abnormality (central versus gonadal or adrenal abnormality). Pubertal levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are indicative of central precocious puberty. However, prepubertal levels do not exclude central precocious puberty, and a luteinizing hormone-releasing hormone (LHRH) stimulation test may be necessary.
3. *Pelvic and abdominal ultrasound.* A pelvic ultrasound in the girl will show uterine development (fundus, cervix, endometrium) and the ovaries. A pubertal-appearing uterus with bilateral ovarian enlargement is indicative of central precocious puberty or gonadotropin-independent precocious puberty. Ovarian cysts are a nonspecific finding, since they may be found in normal girls. Large ovarian cysts should not be referred to surgery; instead, observation and frequent reevaluation are indicated. An ovarian tumor can be detected by ultrasound and should be referred immediately for surgery. An abdominal ultrasound also may detect an adrenal mass, which requires immediate referral to surgery and endocrinology.
4. *Brain imaging.* A CT scan of the head or an MRI should be ordered immediately if the child has clear neurologic signs or neurofibromatosis. Otherwise, a decision for expensive imaging studies should be left to the discretion of a consulting endocrinologist.

C. Referral. A significant advancement in bone age, pubertal levels of sex steroids or gonadotropins, or rapid

acceleration of growth should prompt immediate referral to an endocrinologist.

VI. Assessment of Delayed Puberty

Pubertal delay is defined as lack of breast development by 13 years of age or of menses by 15 years of age in girls or as lack of testicular enlargement by 14 years of age in boys, irrespective of presence or absence of pubic hair. Similar to precocious puberty, the initial task is to distinguish between central and peripheral causes. The former include constitutional delay and hypogonadotropic hypogonadism; the latter include all forms of primary gonadal failure (Figs. 33.6 and 33.7).

A. History. Important information includes nutrition, body image, general well-being, ability to smell, headaches, visual problems, gastrointestinal symptoms, excessive exercise, pre-

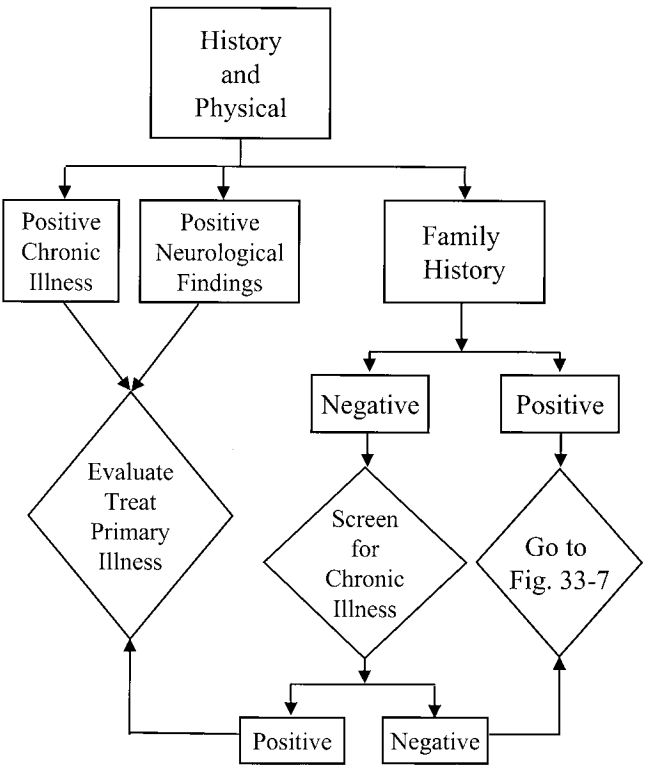

Fig. 33.6. Delayed puberty.

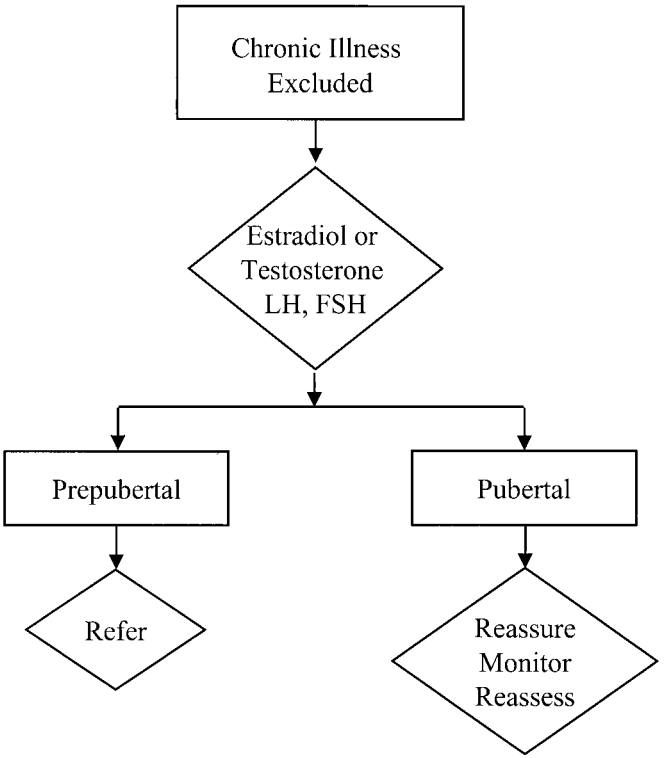

Fig. 33.7. Delayed puberty/constitutional delay.

vious heights and weights, and growth rate. The family history should focus on delayed puberty and chronic illness. The family history is usually positive for delayed puberty in cases of constitutional delay.

B. Physical examination. The physician should in particular establish the growth rate and weight gain, determine body proportions (arm span, upper/lower ratio), stage breast or genital development and pubic hair development, and examine optic discs and visual fields.

C. Laboratory and other tests.

1. *Bone age.* A baseline bone age is helpful in the diagnostic evaluation and in estimation of final adult height. In constitutional delay the bone age is usually 2 or more years delayed, relative to the chronological age.

2. *Sex steroids, gonadotropins, and other hormonal levels.* The hormonal events of puberty precede the physical changes. Hence pubertal levels of testosterone or estradiol and of LH and FSH may be found in a child who is clinically prepubertal. Prepubertal levels, however, are not diagnostic and may require a follow-up LHRH test to determine whether the hypothalamic/pituitary axis is activated to pubertal levels. In contrast, LH and FSH levels above the normal range in a pubertal-aged child without signs of sexual maturation are indicative of primary gonadal failure (e.g., Turner syndrome, anorchia, Klinefelter syndrome). Serum prolactin, thyroxine, and TSH levels may be needed when there is concern about acquired hypogonadotropic hypogonadism.

3. *Other studies.* Complete blood count with differential, erythromycin sedimentation rate (ESR), and a renal panel should be obtained to investigate possible chronic illness, such as inflammatory bowel disease or other causes of malabsorption, or chronic renal failure. Serum thyroxine and thyroid stimulating hormone (TSH) may be indicated if there is diminished growth rate or other signs of hypothyroidism. CT or MRI of the brain may be necessary when there are associated neurologic symptoms, such as headaches and visual field defects, and in cases of possible acquired hypogonadotropic hypogonadism.

D. Constitutional delay of puberty. Constitutional delay may be indistinguishable from hypogonadotropic hypogonadism and primary gonadal failure. A careful history should be obtained, including the presence of headaches, visual problems, anosmia, chemotherapy, or radiation therapy. Chronic illness, such as regional ileitis or chronic renal failure, must also be excluded before constitutional delay of puberty is considered. In children with constitutional delay of puberty, short stature is often the presenting complaint in the prepubertal years. Typically, the growth rate is normal for the first 2 to 3 years of life, then growth crosses percentiles and follows a curve that is below that expected for midparental height. Moreover, the pubertal growth spurt fails to occur at the expected age. The family history is usually positive for delayed puberty. All screening laboratory tests are normal. The bone age is delayed by 1.5 or more years, and serum testosterone or estradiol, LH, and FSH are all prepubertal. The approach may then be expectant, depending on the child's height, age, and level of anxiety. The growth rate and pubertal progression should be monitored every 4 to 6 months. Constitutional delay commonly causes social adjustment problems in boys, and testosterone treatment may be indicated. In such cases, a referral to a pediatric endocrinologist should be made.

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34 Assessment of Abnormal Linear Growth

Anita Cavallo

The assessment of growth at each pediatric visit provides important information about the child's well-being. Generally, a normal velocity of linear growth and weight gain is indicative of a healthy child. Conversely, deviations from normal velocities of either linear growth, weight gain, or both should alert the primary care physician to a possible medical problem, such as nutritional deficiencies, recurrent or chronic illness, or endocrine disorder. Deviation from normal growth may be the only manifestation of an underlying medical problem in a healthy-appearing child. In contrast, however, crossing growth channels at certain ages may be normal. In addition to plotting the growth parameters on the appropriate growth chart, it is important to determine whether (a) the growth velocity is normal and (b) the height is appropriate for the genetic potential (Fig. 34.1).

I. Normal Growth

A. Growth velocity. Generally, an interval of 3 to 4 months between two measurements permits the estimation of the annualized growth velocity. In the evaluation of possible growth disorders, the physician should consult the gender-specific growth velocity charts for boys and girls (Appendix H).

B. Target height. The genetic potential or target height must be considered even when the child's growth points are in the normal range for age (Fig. 34.1). Target height can be calculated using the following formula:

Girls: (Father's height in cm + Mother's height in cm − 13)/2 = Target height ± 5 cm

Boys: (Father's height in cm + Mother's height in cm + 13)/2 = Target height ± 5 cm

Children whose growth channel differs from the target height need to be monitored closely (Fig. 34.2). Growing along a normal channel does not exclude a problem. For example, a prepubertal girl who has a normal growth velocity but who is too short for her genetic potential (not necessarily below the fifth percentile on the height curve) should be evaluated for Turner syndrome, despite the absence of obvious stigmata.

II. Charting and Interpreting Growth Data

A. Standard growth charts. These are gender specific, based on specific measurement techniques: the charts labeled "birth to 36 months" are for supine-length measurements up to 3 years of age, and the charts labeled "2 to 20 years" are for standing height (Appendix H). Note that either technique can be chosen for children between 2 and 3 years of age.

B. Growth velocity charts. These are also gender specific (Appendix G).

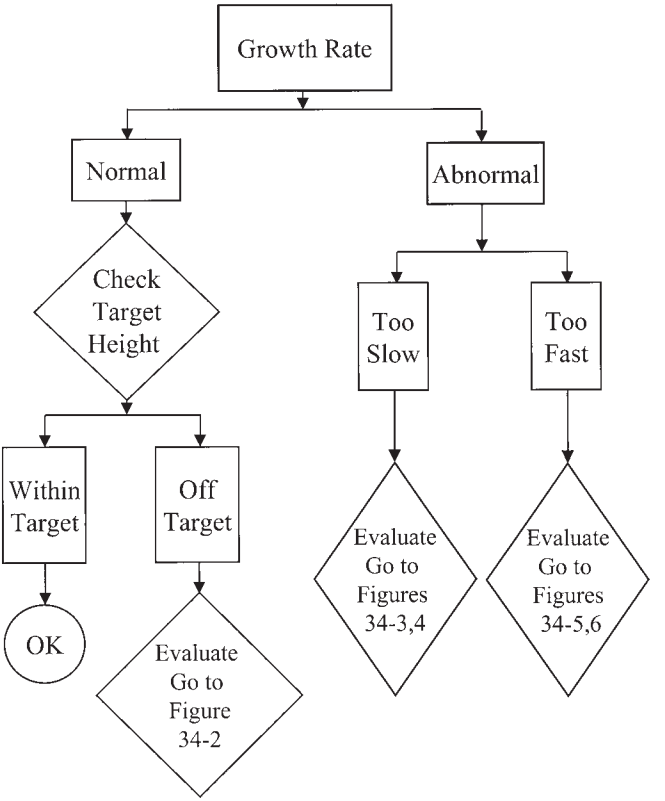

Fig. 34.1. Evaluation of growth.

C. Common pitfalls in assessment of growth. Accuracy of measurement technique and use of the appropriate chart are essential for interpretation of growth charts. Common pitfalls are the following:

1. *Poor technical skills in measuring,* such as failing to position the child properly or leaving the shoes on during the procedure.
2. *Measuring standing height and plotting it on the length chart*. There may be a loss of about 2 cm from the supine to the upright measurement in a young child, and the growth channel for the measurement may differ between the length and the height charts. Hence both the

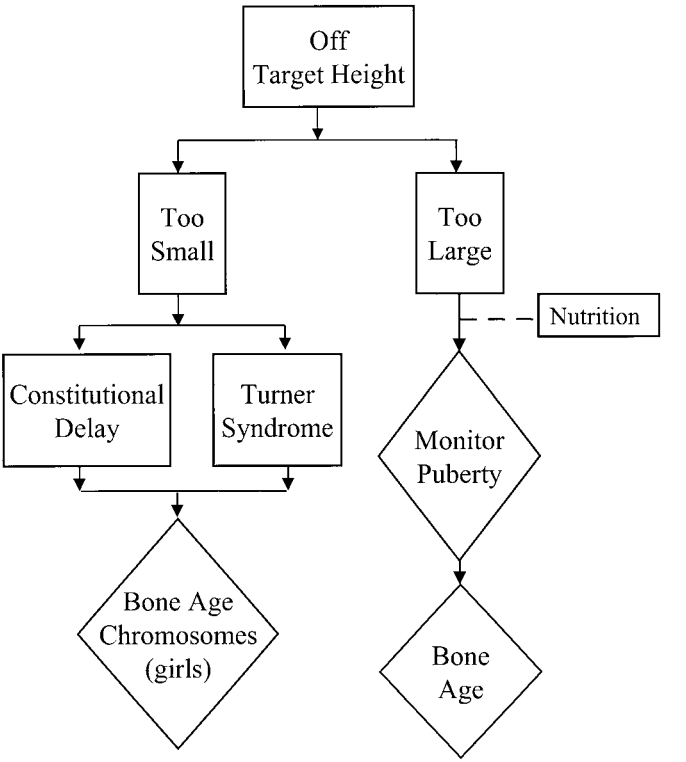

Fig. 34.2. Normal growth rate/off target height.

appropriate technique and the correct chart must be used.

3. *Plotting growth on the chart for the opposite gender.*

D. Commonsense rules for plotting growth charts. Errors in measuring or plotting occur frequently, but a commonsense approach prevents anxiety and expensive evaluations resulting from errors in the documentation of the child's growth.

1. *Plot before seeing the child,* to direct the visit according to results of the growth chart.
2. *Replot the current point* if it is off.
3. *Remeasure the child* if the replotted point remains off.
4. *Review the accuracy of previous entries.*

III. Shifting Growth During Infancy

The correlation between birth size and ultimate mature size is notoriously poor. Crossing percentiles on a growth chart during infancy may be normal when the infant shifts growth curves based on genetic potential.

A. Catch-up growth begins shortly after birth (e.g., infant born small for gestational age).

B. Slow down in growth tends to begin between 3 and 6 months of age.

C. Final growth channel is reached by 2 to 3 years of age and should be consistent with the genetic potential.

IV. Decreased Growth Velocity

A. Common causes of decreased growth velocity

1. *Genetic potential.* As noted in Section III earlier, decreased growth velocity in infancy may be linked to downward adjustment toward a growth channel consistent with the genetic potential. This conclusion should be derived with caution, after reviewing the history, performing a thorough physical examination, and reassessing the infant in 3 to 4 months. The child with genetic or familial short stature will maintain a steady growth channel after 3 years of age that is consistent with the projected target height. The bone age in infancy is of little use; in the school-aged child, it will be consistent with the chronological age.
2. *Nutritional and psychosocial problems.* Inadequate nutrition and psychosocial problems are frequent causes of poor growth and poor weight gain in infants. The preteen or teenager with inadequate nutrition or psychosocial problems may not have dramatic weight loss, but rather may fail to gain weight adequately and may experience deceleration of linear growth. Other less obvious causes in this category include the effort to maintain a lower weight for certain activities (e.g., wrestling and ballet dancing).
3. *Chronic illness.* Examples are chronic renal failure, malabsorption, and cystic fibrosis. Recurrent acute illnesses also may cause significant linear growth delay, particularly in infants. Generally, weight gain in these cases is also compromised, and the child tends to be underweight for height.
4. *Chromosome abnormalities, prenatal infections, and inborn errors of metabolism*. These are often associated with poor growth and short stature.
5. *Endocrine disorders.* These are associated with poor growth and generally are accompanied by increased weight for height (e.g., hypothyroidism, growth hormone deficiency, and Cushing syndrome). Long-term or frequent use of glucocorticosteroids, however, may cause a decrease in growth velocity without the obvious clinical signs of Cushing syndrome.

6. *Constitutional delay.* Children with constitutional delay have normal growth velocity after 3 years of age, and their height channel remains either below the fifth percentile or within the normal range for age but below the projected height based on mid–parental height. At the usual age of puberty, their height falls off the previous growth channel because they maintain a prepubertal growth velocity, whereas for normal children the height channels slope upward because of the pubertal growth spurt. The bone age will be delayed in relation to the chronological age and will be close to the height age.
7. *Rigorous physical training.* It has been documented that strict training in gymnastics for more than 18 hours weekly beginning before puberty may decrease growth velocity to the extent that the expected height may not be reached. The impact of other modalities of vigorous physical training on growth is not well documented.

B. General approach to a child with decreased growth velocity (Figs. 34.3 and 34.4).

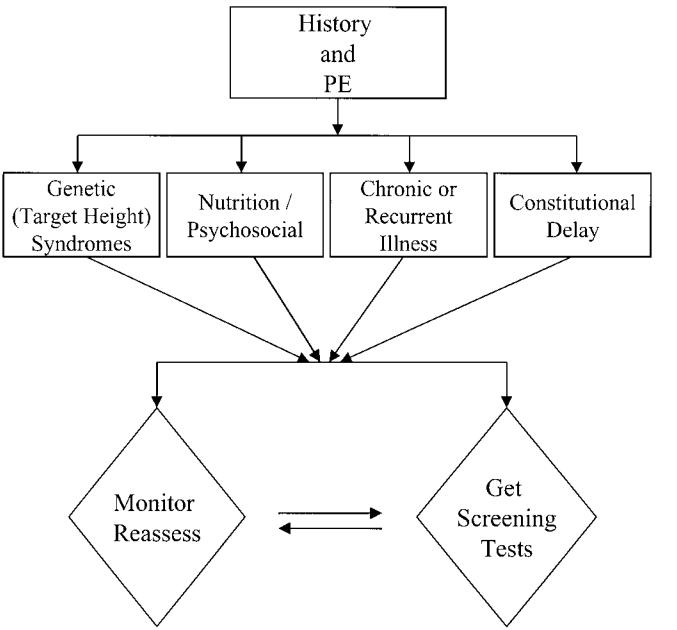

Fig. 34.3. Slow growth under 3 years.

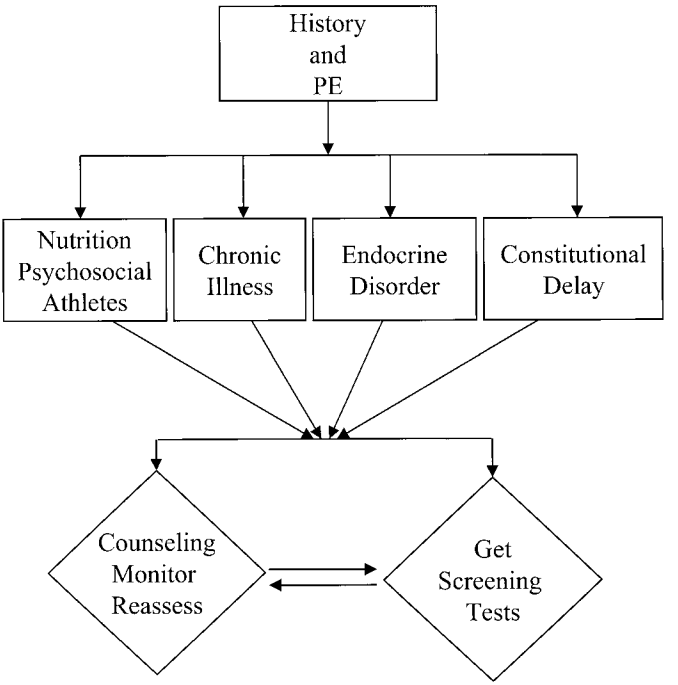

Fig. 34.4. Slow growth 3 years—adolescence.

1. *History.* A thorough medical history is essential to screen for chronic illness, feeding or nutritional problems, psychosocial issues, and so on. The family history should include questions about delayed puberty, short stature, endocrine disorders, chronic illness, and the biologic parents' height for determination of the child's target height.
2. *Physical examination.* The physical examination must be thorough, including a search for dysmorphic features and abnormal body proportions, presence or absence of signs of puberty or virilization, and a neurologic examination with assessment of visual fields and inspection of the optic discs.
3. *Screening tests.* Often the information obtained from history, physical examination, and growth chart will enable the primary care provider to decide whether further testing is necessary. In fact, when the history and physical examination do not uncover any concerns that would require

prompt investigation, monitoring the child's growth and reassessing in 3 or 4 months without obtaining any tests is appropriate. Generally, useful screening tests are a complete blood cell count with differential, erythrocyte sedimentation rate, serum chemistries (for renal and hepatic function), and serum thyroxine and thyroid-stimulating hormone (TSH) determinations. A baseline bone age is often helpful to determine the current status of bone maturation and to predict the mature height. Other tests, such as metabolic screening tests, cardiac evaluation, sweat test, and tests for malabsorption, will depend on each individual case. Presently, screening tests of growth hormone deficiency, such as insulin-like growth factor I (IGF-1) or IGF-binding proteins, are not sufficiently standardized to be valuable for use by the generalist.

4. *Reassessment.* If the history, physical examination, and screening tests cannot identify a cause for the child's decreased growth velocity and if the child is healthy, reassessing the child's growth in 3 to 4 months is appropriate. At that time, a thorough review of the history and a complete review of systems is appropriate. Referral to an endocrinologist should be considered with normal screening tests but persistent slow growth.

V. Accelerated Growth Velocity

A. Common causes of increased growth velocity

1. *Genetic potential.* As noted in Section III, growth acceleration in infancy may be linked to catch-up growth to reach the growth channel consistent with the genetic potential. This conclusion should be derived with caution, after reviewing the history and performing a thorough physical examination. Often no obvious cause is found for the growth acceleration at the initial observation, and repeated reassessment is necessary.

2. *Overfeeding.* Overfeeding in infancy may cause acceleration in linear growth in addition to excessive weight gain. Although most infants will grow appropriately with the approach of "feeding on demand," this feeding technique is often misinterpreted by parents and health care providers. When a bottle of formula or the breast is offered routinely to a crying infant in place of other measures of comfort, the baby may feed even when not hungry, with resultant obesity. Continued excessive weight gain beyond infancy may lead to tall stature in the school-aged child. Concurrent advancement of bone maturation and earlier onset of puberty, however, preclude a gain in final height.

3. *Cerebral gigantism.* Children with this syndrome (Soto syndrome) have rapid growth in infancy and normal growth velocity after 3 years of age.

4. *Endocrine disorders.* Acceleration of linear growth without excessive weight gain should alert the physician to endocrine disorders:

a. ***Gigantism*** due to excess growth hormone production is rare in the young child but should be considered in the older child or adolescent with abnormally rapid growth.

b. ***Hyperthyroidism*** of long duration may cause growth acceleration, and the symptoms and signs may be missed unless this diagnosis is being considered.

c. ***Excess of sex steroid secretion*** is more commonly implicated in growth acceleration. For example, in both boys and girls, excess androgens and estrogens may be present due to premature adrenarche, precocious puberty, congenital adrenal hyperplasia, or a tumor in the adrenal, testis, or ovary.

d. ***Precocious puberty*** should be considered in the differential diagnosis of accelerated growth with or without signs of pubertal development because the growth acceleration may precede the appearance of physical signs by several months (Chapter 33).

B. General approach to a child with increased growth velocity (Figs. 34.5 and 34.6).

1. *History.* A thorough **medical history** is essential to screen for intake of exogenous steroids, growth-promoting or body-building substances, symptoms of hyperthyroidism, and so on. The **family history** should include questions about early puberty, neurofibromatosis, tall stature, and height of the biologic parents for determination of **target height**.

2. *Physical examination.* The physical examination should include a neurologic examination, inspection of the optic fundi and assessment of the visual fields, palpation of the thyroid, a careful cardiovascular examination, and a search for signs of puberty or virilization (Chapter 33).

3. *Screening tests.* A baseline bone age is often helpful to determine the current status and to assess the child's progress. Advanced bone age in relation to chronologic age, consistent with the child's height age, may be seen in obese children with accelerated growth in early life and in the early stages of precocious puberty preceding the typical physical signs of puberty. Serum TSH and thyroxine are diagnostic for hyperthyroidism. Screening tests for growth hormone excess (gigantism), such as IGF-1 and IGF-binding proteins, may be helpful, although they are not diagnostic and should be considered only when other causes for accelerated growth have not been found.

4. *Reassessment.* If the history, physical examination, and screening tests cannot identify a cause for the child's increased growth velocity and the child is healthy, reassess the child's growth in 3 to 4 months. Referral to an endocrinologist should be considered for persistent acceleration of growth rate without an apparent cause.

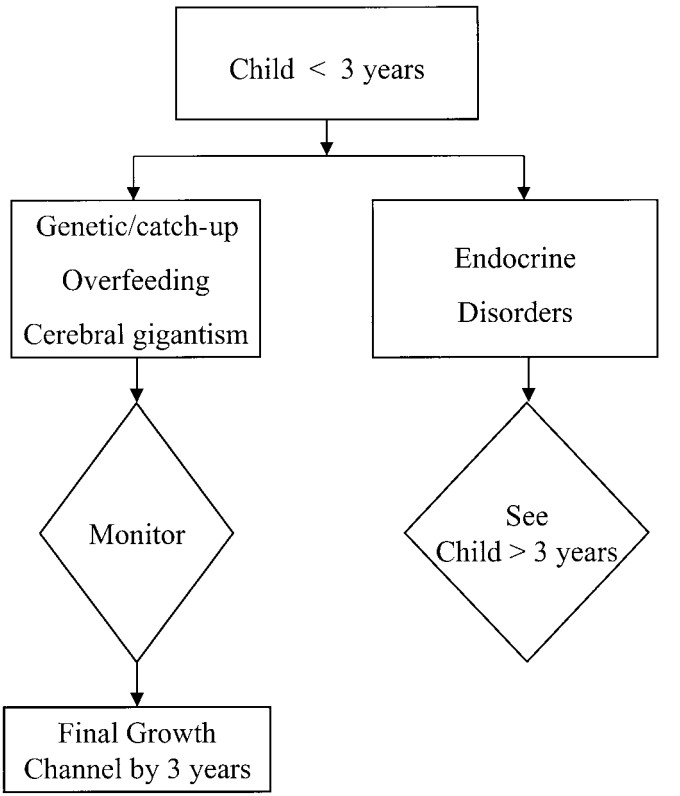

Fig. 34.5. Accelerated growth in the child under 3 years of age.

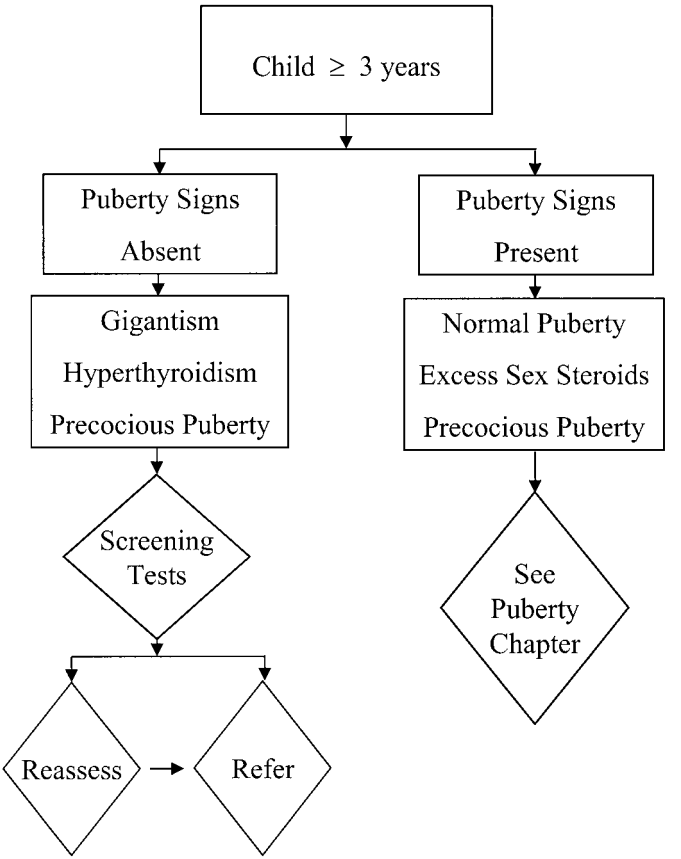

Fig. 34.6. Accelerated growth in the child over 3 years of age.

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35 Failure to Thrive

Julie A. Jaskiewicz

Failure to thrive is a condition of growth deficiency secondary to inadequate nutrition. Determining the exact cause is often complicated, and treatment poses a challenge to the primary care physician. It has been reported that 3% to 5% of pediatric hospitalizations are related to failure to thrive. Recent national and state surveys show that up to 10% of children who present for primary care have evidence of growth deficiency. Most cases of growth deficiency occur in children less than 18 months of age, with a slightly higher incidence in lower socioeconomic groups.

I. Definition

Using the standard growth charts of the National Center of Health Statistics (Appendix H), three criteria describe a child less than 2 years of age with growth deficiency: (a) weight less than the third or fifth percentile for age on more than one occasion; (b) weight less than 80% of ideal weight for age; and (c) weight drop across two major growth percentiles. A helpful way to express growth parameters is as a percentage of the median weight for age (e.g., 50th percentile for a 24-month old). Table 35.1 relates the degree of malnutrition to the percent median weight for age.

II. Differential Diagnosis

It is important to distinguish true growth deficiency from other conditions associated with "being small."

A. Constitutional short stature. This condition is associated with a weight for height usually average or above average and may reflect a genetic tendency to be short for age. Assessment of mean parental height may be useful in distinguishing this condition from true growth deficiency. However, parental height may reflect the nutritional status of the parents who may not have reached their own full genetic growth potential.

B. Prematurity. Premature infants may be well below the third percentile for all three growth parameters and should be plotted on a special growth chart for premature infants (Appendix H).

C. Congenital syndromes. Some children will have limited growth potential associated with particular syndromes, including congenital cytomegalovirus (CMV) infection, Down syndrome, and fetal alcohol syndrome. These children may never "fit" on the standard growth curve, even though they may not be truly growth deficient.

III. Pathogenesis

Failure to thrive is a complex and interactive problem involving the biomedical, environmental, and psychosocial features of a child's experience. Most cases of growth deficiency encountered in the outpatient setting are primarily related to problems in the home and psychosocial environment, but rarely is the process simply a matter of parental deprivation alone. Some infants and children without an overt medical illness may have subtle

Table 35.1. Classification of growth failure

Degree of Malnutrition	Median Weight for Age
Mild	75–85% of 50th percentile
Moderate	60–74% of 50th percentile
Severe	<60% of 50th percentile

Adapted from Bithoney WG, Dubowitz H, Egan H. Failure to thrive/growth deficiency. *Pediatr Rev* 1992;13:453–459.

neurologic conditions or behaviors that render them particularly difficult to feed. If these children are cared for in a disorganized environment by a stressed or psychosocially limited caregiver, the potential for growth deficiency will be increased. Growth deficiency results from insufficient calories to meet the body's needs and may occur by several mechanisms.

A. Inadequate intake of calories

1. *Insufficient calories provided.* Reasons include formula mixing errors, inadequate feeding frequency, lactation problems, parental neglect, and extreme poverty.
2. *Insufficient caloric intake.* Reasons include anorexia and oral-motor dysfunction (e.g., cleft palate, cerebral palsy).

B. Excessive caloric loss. This situation is usually associated with gastrointestinal abnormalities, such as vomiting, reflux, rumination, malabsorption, or diarrhea.

C. Increased caloric requirements. This condition may be associated with chronic infections, chronic respiratory insufficiency, congenital heart disease, chronic anemia, and toxin exposure.

D. Inadequate caloric utilization. Metabolic disorders, including insulin-dependent diabetes mellitus and renal tubular acidosis, are common examples.

IV. Risk Factors

Neither the child nor the environment alone determines the child's growth outcome; rather, each affects and is affected by the other. Most cases of growth deficiency involve the relationship between the demands of the child and the capabilities of the caregiver for that child. Sorting out the relative contributions of each can be very difficult. Potential risk factors for growth deficiency include the following:

A. Biologic. Biologic conditions that may place a child at risk for growth deficiency include congenital anomalies (e.g., cleft palate, microcephaly), prenatal and postnatal undernutrition syndromes (e.g., fetal alcohol syndrome, congenital infections), medically compromised prematurity (e.g., congenital heart disease, bronchopulmonary dysplasia), ongoing medical conditions (e.g., cardiac, respiratory, HIV infection), and behavioral or neurologic problems (e.g., attention deficit/hyperactivity disorder[ADHD]).

B. Environmental. Features of the environment that increase a child's risk for growth deficiency include both child issues (difficult temperament—an irritable, a fussy, or a withdrawn and passive infant) and caregiver issues (easily frustrated and anxious caregiver); disturbed interaction between child and caregiver (both show withdrawal and/or negative, demanding behaviors toward one another); abnormal feeding behaviors in the child (disinterest; bizarre maneuvers with food); and psychosocial stressors in the family (frequent moves, single-parent family, isolation, recent traumatic events, poor marital relationship).

V. Diagnostic Approach

The evaluation of a child suspected of having inadequate growth begins by determining whether the child is really growth deficient or "just small." The physician should remember that 3% of all children will have a weight below the third percentile (by definition). A diagnosis of growth deficiency should be based upon positive historic and physical findings and careful clinical assessment and observation; growth deficiency secondary to nonmedical causes is not a diagnosis of exclusion. Both biologic and environmental factors need to be addressed at the same time. The primary caregivers are key to providing an accurate history and to assuring successful long-term management. They must be approached in a caring and nonjudgmental manner to assure their complete cooperation in the assessment of their child's nutritional state and to avoid giving them the impression that they are responsible.

VI. Clinical Assessment

A. Age. Feeding behaviors of children are closely related to their developmental abilities, including motor, cognitive, and psychosocial skills. Problems with attachment, separation, and individuation may lead to maladaptive feeding and abnormal interactive behaviors between child and caregiver, which may exacerbate poor nutrition. By knowing when a child began to demonstrate growth deficiency, the stage of psychosocial development for that child may be determined. Recognizing the developmental stage of a child with growth deficiency is useful in determining appropriate intervention.

B. History. The single most important "diagnostic procedure" in determining the etiology of growth deficiency in a child is the history. Having both parents present, if possible, is optimal because of the opportunity to observe their attitudes and interactions with each other, both verbal and nonverbal (supportive versus detached, angry, impersonal). One should observe the interactions of each parent with the child (affectionate and encouraging versus negative or demanding) and look for symptoms in the child that are often associated with family dysfunction (irritability, excessive crying, sleep or discipline problems). The physician should pay attention to the parents' attitudes toward the history-taking process (tense, hostile, depressed, in a hurry to leave). A second, third, or fourth history may be more revealing than the first, especially

if some trust has been established. The following are important components of the history.

1. *Age of onset of growth decline* by parental history or growth chart.
2. *Birth history.* Maternal age, health, prenatal or postnatal depression, prenatal care, drugs, alcohol, tobacco, length of gestation, birth weight, initial feeding pattern, stool pattern, early weight gain.
3. *Development.* Milestones, delays, previous interventions.
4. *Medical history of previous illnesses,* hospitalizations, chronic illnesses (e.g., otitis media, pneumonia, diarrhea).
5. *Review of systems,* especially gastrointestinal symptoms and central nervous systems.
6. *Family history.* Parents' and siblings' growth, familial disease (especially endocrine), congenital abnormalities.
7. *Psychosocial information.* Family constellation, finances, extended family, social supports, substance abuse, physical or sexual abuse, recent changes in lifestyle, death of a family member, divorce, recent move, job change.
8. *Dietary information.* Caloric intake, 24-hour dietary recall, 3-day food diary, bizarre or fad diets, parental (mis)perceptions of child's needs, formula preparation, frequency of feedings, who feeds and how the infant feeds, bottle propping, parental anxiety, force feeding, consistency between the child's age and feeding behavior.

C. Physical examination. A thorough general physical examination is crucial. Signs and symptoms of vitamin and/or protein-calorie deficiencies or a specific medical condition may be present.

1. *General appearance.* Acutely versus chronically ill appearing, signs of poor hygiene or inflicted trauma, disturbed affect (apathetic or scared), dysmorphic features.
2. *Growth.* Weight (unclothed), height, head circumference, and weight for height plotted on the appropriate growth charts, and triceps skinfold thickness.
3. *Development.* Developmental assessment (e.g., DDST-II). Should be delayed if the child is acutely ill or dehydrated.

D. Observation. Careful observation of the child's behaviors, especially around feeding, and the interactions of the child with caregivers, other children, and adults can often provide clues to interaction difficulties between the child and his/her environment that contribute to the growth deficiency. Behaviors felt to be associated with disturbed psychosocial functioning as the primary etiology of growth deficiency include "radarlike gaze," "frozen watchfulness," minimal smiling, decreased vocalization, resistance to being held, tonic immobility of the arms (arms up and back with elbows flexed), and self-stimulating rhythmic movements.

E. Laboratory. If the history and physical examination do not suggest an underlying medical illness or organic cause of growth deficiency, no rationale supports doing an extensive

laboratory assessment. A limited number of laboratory tests may be appropriate in certain circumstances, including a screen for iron deficiency; a screen for lead (especially if there is a history of pica or the child lives in a high-risk area); urinalysis; tuberculin skin testing; HIV testing, if suggested by history or high risk; and thyroid function, if length is primarily affected. A normal bone age would be inconsistent with a diagnosis of an underlying systemic chronic disease or hormonal abnormality as the etiology of the growth deficiency. All other laboratory tests should be done only if suggested by history and/or physical examination.

VII. Management

The primary goal of management of the growth-deficient child is to restore adequate nutrition. Nutritional rehabilitation involves several concurrent processes.

A. Caloric requirements. To determine the child's caloric requirements to restore growth, the child's length is measured and ideal weight is determined for that height (fiftieth percentile for that height). Then the normal daily caloric intake needed to maintain that weight is determined and is increased by 50% to provide for an accelerated rate of catch-up growth.

B. Dietary counseling. Once the caloric requirements are established, the physician and/or nutritionist should counsel caregivers about the appropriate dietary intake to meet those needs, particularly the quality and quantity of foods. Often the parent can provide the necessary calories by increasing the caloric density of the liquids and foods offered (e.g., high-calorie supplemental drinks, increased strength of formula, high-calorie formula, such as Pediasure, substituted for regular formula), rather than increasing the volume of liquids. If the parent and child are not already participants in the Women, Infants, and Children (WIC) program and qualify, they should be referred for enrollment to help supply the nutrition required.

C. Management of medical illness. Underlying medical disease, such as infection or lead toxicity, should be addressed concurrently with nutritional support.

D. Family support. The primary care physician is often a "case manager" and can serve as a liaison between the family and other professionals, such as community health nurse, social worker, dietitian, and mental health worker. The physician should provide guidance for parents concerning appropriate developmental tasks for age, especially as they relate to feeding. Successful management of any child with growth deficiency means sustained weight gain. Frequent follow-up, often every week at the beginning, is essential. Frequent weight checks are helpful to document progress, but they are just as important as an opportunity for the physician or home health nurse to provide ongoing support and guidance for the family and to build a relationship of trust.

E. Hospitalization. The decision to hospitalize a child with growth deficiency is not an easy one. Sometimes even a child from a disturbed psychosocial environment may fail to gain weight in the hospital, away from familiar surroundings.

1. *Recommendations.* Hospitalization should be strongly considered when concerned about active abuse or neglect, severe (grade II or III) malnourishment (weight less than 60% of median weight for age), the presence of a significant medical illness that requires intensive inpatient management, the failure of intensive outpatient management to provide adequate catch-up growth over a 6-month period, the unreliability of the family to follow through with outpatient plans, or the child becoming lost to follow-up.
2. *Goals.* Goals of hospitalization include providing adequate calories for catch-up growth, documenting sustained weight gain, providing better observation of child and parent/child behaviors that may contribute to growth deficiency, providing an organized program of stimulation for the child, and providing interdisciplinary evaluation with nutrition, social services, nursing, and child life services.
3. *Expectations.* Documented weight gain in the hospital is not conclusive evidence that psychosocial problems alone are the cause of the poor growth. Children with and without specific medical illness have been shown to gain weight in the hospital, though weight gain is often not immediate. Parents need to be cautioned against expecting an overnight solution. Often infants require a few days to a week before they establish a satisfactory weight gain.

VIII. Prognosis

Although most children with growth deficiency show a gradual improvement in somatic growth during the preschool years, recent studies show that many of these children remain underweight, suggesting a chronic pattern of undereating. The effect of early childhood growth deficiency on subsequent cognitive functioning is not known for certain. Many children demonstrate delayed acquisition of language skills, for example, at the time of diagnosis of growth deficiency. Yet two recent population-based studies have shown that with treatment for growth deficiency, "catch-up" cognitive development by 6 years of age is possible in most children.

IX. Prevention

Ideally, physicians can prevent growth deficiency in children by identifying risk factors early. This requires ongoing anticipatory guidance for families of infants and children concerning development, behavior, and feeding practices. The physician should ask about biologic and environmental stressors at each regular well-child care visit. Parents should be counseled at each visit about appropriate feeding for their infant or child for the interim period until their next visit. They should learn not only what the child should be eating, but also how the child should be eating (e.g., a 1-year-old to try to feed him/herself and

make a mess is appropriate). The physician should continually monitor the psychosocial attachment between parent and child, especially as it relates to feeding, and keep abreast of new changes in the family (new baby on the way, recent separation of parents) as red flags for potential new problems with feeding and mealtime behaviors.

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36 Obesity

Julie A. Jaskiewicz

Childhood obesity is a complex and increasingly common problem. Approximately 22% of children in the United States between the ages of 6 and 19 years are overweight, a 15% increase from a decade ago. Multiple etiologies have been suggested, and experts still disagree about the magnitude of influence of the many genetic and environmental contributors. Unfortunate misconceptions about the etiology and natural history of obesity exist. Many physicians erroneously assume that "nothing can be done" for the obese child.

I. Measures of Body Fat

Obesity is defined by an excess of body fat (>95% body mass index). Evidence in adults suggests that the predominant distribution of body fat (abdominal versus pelvic) may be more important in predicting obesity-related morbidity than simply the degree of fat excess.

A. Weight. Weight alone is not a good measure of body fat. Weight is an easy and reproducible measure that can be compared with population standards, but weight gives no indication of the distribution of body fat. Using weight alone, a very muscular child may be mislabeled as obese.

B. Skinfold thickness (SFT). Using skinfold calipers, a direct measure of subcutaneous fat mass (usually triceps or subscapular skinfolds) can be obtained. The measurements can be compared with population standards and can indicate distribution of body fat but may be difficult to interpret, given the inherent error in measurement in very overweight individuals.

C. Body mass index (BMI). The BMI (wt/ht^2) is better than weight alone for measuring body fat content (Appendix E). However, although BMI can be compared with population standards for adults, using BMI to monitor weight in children requires age-dependent and gender-dependent reference graphs.

II. Classification of Obesity

A. Exogenous. Exogenous obesity represents most cases of childhood obesity. Children with exogenous obesity are tall for their age (usually height above the fiftieth percentile for age) and have a normal to advanced bone age, normal mentality, no physical abnormalities, and often, a family history of obesity.

B. Endocrine/genetic syndromes. Less than 1% of childhood obesity is associated with an endocrine abnormality or genetic syndrome. These children are typically short for their age, have a delayed bone age, may be mentally limited, often have associated physical abnormalities, and rarely have a family history of obesity.

1. *Endocrine*
   a. ***Hypercortisolism*** (Cushing syndrome).
   b. ***Hypothyroidism.***
   c. ***Primary hyperinsulinism.***

2. *Genetic syndromes*

a. ***Prader-Willi.*** This syndrome is characterized by severe hyperphagia, hypotonia, mental retardation, and occasionally the Pickwickian syndrome.

b. ***Bardet-Biedl.*** This autosomal recessive syndrome shows predominantly truncal and proximal limb obesity, polydactyly, and genital hypoplasia.

c. ***Cohen.*** This syndrome is characterized by mid-childhood onset of obesity, dysmorphic facial features, and mental retardation.

d. ***Alstrom.*** Similar to the Prader-Willi syndrome but with insulin-resistant diabetes beginning during puberty.

III. Etiology of Exogenous Obesity

A. Energy homeostasis. Obesity results from excess energy stored in the adipose tissue as fat and represents an imbalance between excess energy and energy expenditure. Obesity is a problem of energy homeostasis rather than a primary abnormality of the fat itself. Obese people do not have a different kind of fat from that of lean individuals.

B. Genetics versus environment. Recent studies have shown that a child's risk for obesity is related to parental obesity. If both parents are obese, two-thirds of their children will be obese. If only one parent is obese, 50% of their children will be obese. Only 9% of children born to parents of normal size will become obese. Further evidence for a genetic influence on the development of obesity came in 1994, when the first rodent gene for obesity was identified. This discovery was followed by the identification of four additional rodent genes and later by the identification of the human locus of each gene. Gene products for most genes have been described, and evidence now suggests that appetite set point and metabolic rate are encoded in these genes. This discovery supports the belief that the problem of energy homeostasis in obesity is more complex than simply "too much food" with "too little activity." Diet, exercise, and behavior modification are all essential elements of obesity management.

IV. Natural History

Most obese children will not become obese adults, but the persistence of childhood obesity into late childhood and adolescence increases the likelihood that obesity will continue into adulthood. Children who are obese at 6 years of age have a 25% chance of being obese as adults. Obesity at 12 years of age gives a 75% chance of continued obesity in adulthood.

V. Consequences

Obese children experience many of the same morbidities associated with excess fat stores as do obese adults. Almost all these morbidities can be reversed with weight loss.

A. Cardiovascular. Obesity is the most common identifiable cause of hypertension in children.

B. Pulmonary. Abnormalities include the Pickwickian syndrome (hypoventilation, obesity, and congestive heart failure) and sleep apnea.

C. Orthopedic. Common abnormalities are slipped capital femoral epiphysis, aseptic necrosis of the hip, and varus and valgus deformities.

D. Endocrine. Hyperinsulinemia and resistance to insulin-mediated glucose transport are common, although chemical diabetes is rare. Hyperlipidemia, increased adrenocorticotropic hormone resulting in premature adrenarche, and an accelerated bone age from an increase in somatomedin may occur.

E. Integument. Overlapping skinfolds are frequent sites for intertrigo and furunculosis.

F. Psychiatric. Evidence suggests that some obese children have low self-esteem, and a small percent may suffer from clinical depression. Whether or not obese children are discriminated against by their peers, teachers, or other adults is not entirely known and is a source of great controversy regarding their psychosocial well-being.

VI. Outpatient Approach

Physicians should identify obese children and children at greatest risk for becoming obese so that timely intervention can be initiated.

A. History

1. *Medical.* Complete past medical history, including birth weight, weight gain pattern in infancy and early childhood, height pattern, developmental delays, and chronic conditions.
2. *Family.* Family history may help identify exogenous obesity: weight and height of parents and siblings and cardiovascular risk factors and morbidities in parents, especially if parents are obese.
3. *Nutrition.* Dietary history, including intake, type of foods, mealtime behavior and structure, snacking habits, and parental/child concerns about weight (worried or nonchalant).
4. *Psychosocial.* Evidence that parents use food as a reward or to control the child's behavior; signs and symptoms of depression, anxiety, or boredom.

B. Physical examination

1. *Growth.* Weight, height, and weight for height on the appropriate growth charts (routine); distribution of body fat, skinfold thickness and BMI calculation for children identified as overweight on standard growth charts (weight for height).
2. *Development.* Stigmata associated with endocrine or genetic obesity, particularly mental deficiency, genital abnormalities, and dysmorphic features.
3. *Blood pressure measurement.*

C. Laboratory

1. *Urinalysis.* Glycosuria is the most common abnormality.
2. *Nutritional indices.* Cholesterol, triglycerides, LDL, and HDL. Abnormalities will require further testing.

3. *Other.* Additional studies should be done if indicated by the history or physical examination. Examples might include fasting blood sugar, thyroid studies, or karyotype.

D. Classification. Exogenous versus endocrine/genetic obesity.

E. Co-morbidities. Identify and document.

VII. Management

The primary goal of management of the child with exogenous obesity is to minimize morbidity without causing untoward effects from the treatment. Most children will require a combination of dietary, exercise, and behavior modification interventions, but severe measures for weight control in children, such as surgery or drug therapy, are not recommended.

A. Morbidities. The first priority of management of the obese child is to identify and treat any morbidity associated with the condition. Hypertension is usually mild and should respond to dietary changes and weight reduction. Significant hypertension should be treated. Orthopedic problems, especially slipped capital femoral epiphysis, and sleep apnea require immediate attention.

B. Diet. The type of diet will vary according to the age of the child and the severity of the obesity. The diet should be designed to provide sufficient quantities of carbohydrates, fat (especially essential fatty acids), protein, minerals, and vitamins to meet fully lean body mass and linear growth requirements. Protein-sparing diets that are very low in calories and carbohydrate-free may be used, particularly for obese children with respiratory insufficiency or sleep apnea. An outpatient referral to a dietitian specialist is often helpful.

C. Activity. The fact that individuals who increase their level of activity will maintain weight loss better than those who do not is well known. Exercise will increase lean body mass, which will increase the resting metabolic rate. Parents should be encouraged to be involved in the exercise plan formulated for their child and to reward their child's progress, even if only modest increases in activity occur. Such positive reinforcement can be a strong motivator for even more activity and will help the child's and the parents' morale.

D. Behavior modification. Behavior modification can help ensure long-term success in the management of obesity. Older children can learn to self-monitor their own food intake and activity and can learn how to obtain rewards other than food for their changed behavior. Exploring alternatives to overeating with the child is helpful. In most cases, psychotherapy is unnecessary.

E. Family. The success of any weight control program depends upon involvement of the family. Parents are responsible for most of the foods brought into the home, and they model for their children both eating and activity behaviors. Parents must be involved in the management plan from the beginning and may need as much support and encouragement

as their child. The physician should provide frequent follow-up in the office and use the opportunity to educate parents and their children about healthy, lifelong dietary habits.

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37 Diabetes Mellitus

Nancy J. Morwessel

I. Classification and Definition of Diabetes Mellitus (DM)

In 1997 the American Diabetes Association's Expert Committee on the Diagnosis and Classification of Diabetes Mellitus proposed extensive changes in the world classification and definition of diabetes and in the diagnostic criteria for diabetes. Modifications were approved in 1999.

A. The new classification defines diabetes by etiology, not treatment.

1. *Type 1 DM* (formerly insulin-dependent diabetes mellitus [IDDM] or juvenile-onset) is an autoimmune disease characterized by β-cell destruction, usually leading to absolute insulin deficiency.
2. *Type 2 DM* (formerly non–insulin-dependent diabetes mellitus [NIDDM] or adult-onset) extends from a state of insulin resistance with relative insulin deficiency to a state of defective insulin secretion with insulin resistance.
3. *Gestational DM* is any degree of glucose intolerance with first identification during pregnancy; if it persists 6 weeks after the pregnancy ends, it is reclassified.
4. *Impaired glucose tolerance (IGT) and impaired fasting glucose (IFG)* are metabolic stages between glucose homeostasis and diabetes and are risk factors for future diabetes and macrovascular disease.

B. The diagnosis of diabetes is based on abnormal fasting plasma glucose levels, casual plasma glucose levels, *and/or* oral glucose tolerance testing.

1. *Diabetes diagnosis by the measurement of fasting plasma glucose levels or casual plasma glucose levels is preferred.*
2. *Diabetes diagnosis by oral glucose tolerance testing is* ***not*** *preferred* because of cost, discomfort, and potential obstacles in obtaining the test and interpreting the results, resulting in a delay in the diagnosis of diabetes.
3. *The "normal" plasma glucose level is established as less than 110 mg/dL.* For epidemiologic study, diabetes prevalence and incidence are now based on a fasting plasma glucose of more than 126 mg/dL.

C. The 1997 American Diabetes Association diagnostic criteria are (a) with acute metabolic compensation, an individual must meet any of the three diagnostic criteria on one occasion or (b) without acute metabolic decompensation, an individual must meet any one of the three diagnostic criteria on two different days.

1. *Polyuria, polydipsia, and/or unexplained weight loss with a casual plasma glucose concentration of ≥ 200 mg/dL* (*casual* means any time of day, regardless of time since last caloric intake).

2. *Fasting plasma glucose of ≥ 126 mg/dL* (*fasting* means no caloric intake for at least 8 hours).

3. *Two-hour plasma glucose of ≥ 200 mg/dL during an oral glucose tolerance test,* performed as described by the World Health Organization.

II. New-Onset Diabetes Mellitus Presentation

A. History. Hyperglycemia, polyuria, nocturia, polydipsia, polyphagia, weight loss, abdominal pain, vomiting.

B. Physical examination

1. *Dehydration.*
2. *Signs of diabetic ketoacidosis,* including tachycardia, tachypnea, fruity breath, central nervous system depression, Kussmaul respirations.
3. *Type 2 DM,* including obesity, hypertension, hyperlipidemia, acanthosis nigricans, absent or mild ketonuria, mild or absent acidosis, initial insulin requirement of more than 1.5 U/kg per day, and/or family history of type 2 DM in parent or sibling.

C. Laboratory studies

1. *Electrolytes and renal group.*
2. *Blood gas.*
3. *Glucose.*
4. *Urinalysis for glucose and ketones.*
5. *Islet cell antibodies (ICA) if etiology is unclear.* In autoimmune diabetes (type 1 DM) specific islet cell antibodies that confirm the diagnosis may be present.
   a. ***Positive antibodies with symptoms of disease*** are consistent with type 1 DM.
   b. ***Negative antibodies with a stereotypical picture of type 1 DM*** (ketosis, acidosis, nonobese, expected response to treatment) are consistent with a diagnosis of type 1 DM. Currently, three specific ICA can be measured (ICA-512, GAD, IAA), along with a combination panel (ICA). The assumption is that many additional antibodies exist but are as yet unidentified. Therefore, negative antibodies raise the possibility of type 2 DM but do not negate the possibility of autoimmune islet cell destruction.
   c. ***Positive antibodies without symptoms of disease*** indicate risk for future development of type 1 DM but do not conclusively predict if or when the individual may progress to overt disease.
6. *Glycosylated hemoglobin ($HgbA_1c$) testing is not particularly helpful.* In the presence of overt hyperglycemia, the diagnosis of diabetes and initiation of treatment are not affected by knowledge of the duration of hyperglycemia.
7. *Once the patient is normoglycemic and metabolically stable* (generally more than 1 month after onset), *obtain baseline fasting lipid profile for those more than 5 years of age.*
8. *In type 1 DM, perform thyroid function tests if there is a history of thyroid disease in a first-degree family member.*

9. *In type 2 DM, once the patient is normoglycemic and metabolically stable* (generally more than 1 month after onset), *obtain baseline albumin excretion rate, echocardiogram, dilated funduscopic examination.* (At diagnosis, 40% adults with type 2 DM already have evidence of microvascular diabetes complications.)

III. Type 1 Diabetes Mellitus

A. General

1. *Type 1 DM represents approximately 10% of diabetes and affects 1 out of 500 Americans.*
2. *Incidence of type 1 DM is 5 to 43 per 100,000 live births among Caucasians of European descent.*
3. *Type 1 DM is the most common chronic illness of childhood.*
4. *Three-fourths of the cases of type 1 DM are diagnosed below the ages of 15 years* (peak between age 5 to 15 years) *with a median age of 12 years.* The rate of β-cell loss appears to correlate inversely with age, with a peak incidence at puberty. Boys are affected slightly more often than girls (1.1 : 1 to 1.2 : 1 ratio).
5. *Only 15% of individuals newly diagnosed with type 1 DM have any family history of type 1 DM.* Offspring of one parent with type 1 DM have a 2% risk (mother) to 6% risk (father) of developing type 1 DM. Depending on the combination of shared HLA haplotypes, siblings have a 5% to 40% risk of developing type 1 DM. Identical twins have the highest risk, with a concordance rate of 35% to 40%, demonstrating the mixed genetic and environmental basis of this disease.

B. Risk for developing other autoimmune disorders.

Individuals with type 1 DM and their relatives are at increased risk for autoimmune disease of the thyroid (Hashimoto thyroiditis and Graves disease), autoimmune gastritis (pernicious anemia), autoimmune adrenal disease (Addison disease), myasthenia gravis, vitiligo, and celiac disease.

C. New-onset type 1 DM

1. *Depending on metabolic status at time of diagnosis, treatment may be started on an outpatient or inpatient basis.*
2. *Blood glucose levels generally stabilize within 2 to 4 weeks.* The "honeymoon period" is characterized by very stable blood glucose levels on low-dose insulin and is a result of residual β-cell function. The honeymoon period begins in the first few weeks after initiating treatment with the resolution of the glucotoxic effects of acidosis and prolonged hyperglycemia. The honeymoon period generally lasts 2 to 24 months, with an average of 4 to 5 months. Blood glucose fluctuations and increasing insulin need are signs of deteriorating residual β-cell function.
3. *Initial self-management education consists of immediate "survival" skills needed by the child and family,*

including blood glucose monitoring, administration of insulin, identification and treatment of low blood glucose levels, carbohydrate counting, and urine ketone testing.

4. *Self-management education continues in the months following onset, gradually increasing in complexity.* Topics include interpretation of blood glucose trends, insulin adjustment, adjustments in insulin or carbohydrate intake for exercise, sick-day management, treatment of high blood glucose levels and ketosis, safety at school, and safe participation in sports.

5. *Anticipatory guidance for older children and teenagers includes information about smoking, alcohol, drugs, preconception care and pregnancy, and driving.*

D. Insulin requirements vary with age, weight, and blood glucose level

1. *See Table 37.1 for a description of available insulins.*

2. *Children before puberty generally require 0.6 to 1.2 U/kg per day.*

3. *Teenagers during puberty generally require 0.8 to 1.6 U/kg per day.*

4. *More insulin is needed to correct hyperglycemia than to prevent it;* therefore, children on "sliding scales" (increasing short-acting insulin doses to correct hyperglycemia at the time of the injection) are generally on higher daily doses of insulin.

Table 37.1. Description of available insulins

Short-Acting Insulins	Onset of Action	Peak Action	Duration of Action
Lispro insulin	10–20 minutes	1 hour	2–3 hours
Human regular insulin	45 minutes	2 hours	4–6 hours
Intermediate-Acting Insulins	**Onset of Action**	**Peak Action**	**Duration of Action**
Human NPH, Human Lente	1–2 hours	4–7 hours	10–14 hours
Human Ultralente	2–3 hours	7–9 hours (blunt peak, if present)	12–18 hours

Note: Human insulin has become the insulin of choice for new-onset diabetes patients. Eli Lilly manufactures the only available animal insulin—Iletin II, a purified pork insulin. Animal insulins as a rule have a longer duration of action. A change to human insulin may affect the required dose and distribution of insulin.

E. Conventional insulin therapy

1. *Two or three injections* a day of mixed intermediate-acting insulin and short-acting insulin.
2. *Blood glucose tests* before breakfast, lunch, dinner, and evening snack (minimum).
3. *Urine ketone tests* whenever blood glucose levels are more than 240 mg/dL.
4. *Carbohydrate counting* as the basis for a diabetic meal plan. Carbohydrates include the fruit, starch, and milk food groups, and may be used interchangeably, provided the total grams match the meal plan. High-sugar foods, including candy, cookies, and so on, can be substituted for other carbohydrates throughout the day in modest quantities. Fat and protein in growing children have an indirect effect on blood glucose levels; therefore, quantities are not strictly controlled.
5. *Meal plan* including three meals and three snacks per day, at specific times, consisting of defined carbohydrate quantities. The schedule and distribution of carbohydrates is designed to match insulin action. Once an insulin injection is given, the patient is committed to eating the required amounts of carbohydrate at the specified times to prevent hypoglycemia.
6. *Carbohydrate replacement for exercise.* In general, an extra 15 g of fast-acting carbohydrate is needed for every 30 to 45 minutes of vigorous activity to prevent exercise-induced hypoglycemia.
7. *Blood glucose goals that vary with age.* Goals need to be individualized. In general, blood glucose levels in the range of 70 to 150 mg/dL reflect acceptable glycemic control.
8. *The blood glucose goal at evening snack of more than 100 mg/dL.* If blood glucose is less than 100 mg/dL, an additional 15 g of carbohydrate is given.
9. *Treatment of hypoglycemia*
   a. ***Fifteen grams of fast-acting carbohydrate corrects blood glucose levels below 70 mg/dL.*** Examples of 15 g of fast-acting carbohydrate include 4 ounces of juice, 4 to 6 ounces of regular soda, 6 to 8 ounces of sports drink, and five lifesavers chewed up. Children should be discouraged from using "treats" when correcting hypoglycemia. High-fat, high-calorie foods digest too slowly to be effective in correcting hypoglycemia. In addition, many children will welcome hypoglycemia as a chance to get extra "treats," thus hindering efforts to stabilize blood glucose levels.
   b. ***Individual parameters are necessary;*** for example, younger children may receive treatment for glucose at levels less than 80 mg/dL.
   c. ***15 g of fast-acting carbohydrate generally should raise blood glucose levels approximately 50 mg/dL within 20 minutes.***

10. *Treatment of hyperglycemia*

a. ***Urine ketones should be tested whenever blood glucose levels are more than 240 mg/dL.***

b. ***If urine ketones are negative, extra insulin is rarely necessary for a one-time high blood glucose level.*** If hyperglycemia persists, extra short-acting insulin may be added to a usual injection time. Try to avoid extra injections if urine ketones are negative.

c. ***If urine ketones are positive, extra insulin is needed.*** Ketosis signifies near-absolute insulin deficiency. If it is time for a routine injection, additional short-acting insulin should be added. If it is not time for a routine injection, an additional injection of short-acting insulin should be considered, depending on the blood glucose level, amount of ketones, and time elapsed from the previous insulin injection. In the absence of illness or other conditions causing increased insulin need, insulin omission should be suspected if repeated episodes of ketonuria occur.

d. ***Carbohydrate-free and caffeine-free beverages should be encouraged (8 to 12 ounces every 30 minutes) if ketones are positive.***

e. ***Abdominal pain and vomiting can be warning symptoms of diabetic ketoacidosis (DKA);*** if they occur with hyperglycemia and ketosis, the patient must be assessed for dehydration and acidosis.

F. Intensified insulin therapy—basal/bolus insulin delivery system, whereby insulin is delivered separately to cover basal needs and bolus needs. Results of the Diabetes Control and Complications Trial (DCCT) 1993 comparing conventional insulin therapy with intensified insulin therapy showed that intensified therapy delayed the onset and slowed the progression of retinopathy, nephropathy, and neuropathy by 35% to 70% and that every 0.5% reduction in $HgbA_1c$ resulted in 20% to 40% reduction in microvascular and neurologic complications. However, intensified therapy was associated with more frequent, severe hypoglycemic events and weight gain.

1. *Basal insulin* keeps blood glucose levels steady, whether the individual is fasting or eating normally. *Bolus insulin* is given to cover food and to correct high blood glucose levels.

2. *Insulin is taken with every meal or snack; the dose varies, depending on the amount of carbohydrate to be eaten.* Therefore the patient does not have to eat a specific amount of carbohydrate at any specific time. Even though there are no specific rules about what to eat when, children should be encouraged to eat regularly throughout the day for optimal blood glucose control.

3. *Blood glucose tests are performed first thing in the morning, last thing before going to bed, and before each*

meal or snack. When trying to evaluate basal insulin doses, performing 6- to 8-hour fasting tests at different times of the day (test every 2 hours during the fast) is helpful. (Are blood glucose levels steady, increasing, or decreasing while fasting?) When evaluating bolus insulin doses, testing before the bolus and 2 to 3 hours after the bolus will help determine the effectiveness of the bolus dose. (Has the blood glucose level returned to baseline?)

4. *Two methods are used: continuous subcutaneous insulin infusion (CSII) via an external insulin pump, or multiple daily injections (MDI).*

a. ***CSII provides a continuous infusion of short-acting insulin delivered in units per hour 24 hours a day to cover basal insulin needs.*** The patient wears the pump 24 hours a day, only disconnecting for brief times (e.g., bathing, vigorous exercise). An infusion set is placed by the patient in the subcutaneous area of the abdomen, side, back, hip, or thigh. The infusion set is changed every 2 to 3 days, and the sites are rotated. At meal or snack times, the patient identifies the total carbohydrate grams to be eaten and calculates an appropriate insulin dose.

b. ***MDI meets basal insulin needs by two or more injections of intermediate-acting insulin per day*** (dose and timing unrelated to food intake). Short-acting insulin injections are taken with every meal or snack to cover food, and, when blood glucose levels are above goal, to correct hyperglycemia, as in CSII.

c. ***Glycemic control is improved in patients using lispro insulin instead of regular insulin to cover food and to correct hyperglycemia.*** The quicker onset of action and shorter duration of action of lispro insulin better matches postprandial glucose levels and allows for more rapid correction of hyperglycemia.

5. *Accurate carbohydrate counting* (grams, not exchanges) is essential to adequate glycemic control. Small errors can have big effects on glycemic control.

6. *Treatment of hypoglycemia and hyperglycemia* is the same as in conventional therapy, except that extra insulin for hyperglycemia correction can be given more precisely using the correction ratio.

G. Morning hyperglycemia: Somogyi phenomenon or dawn phenomenon?

1. *Somogyi phenomenon.*

a. ***Described as rebound hyperglycemia*** following undetected hypoglycemia at night.

b. ***Believed to be caused by an excess of counter-regulatory hormones*** in response to insulin-induced hypoglycemia.

c. ***Characterized by nighttime or early-morning sweating, nightmares, headaches, ketosis, hyperglycemia, glucosuria, and ketonuria.***

d. ***Suggested by blood glucose trends of less than 80 mg/dL at evening snack along with morning tests either below 80 mg/dL or above 280 mg/dL.*** If hypoglycemia is detected at night and treated, morning blood glucose levels are often in a normal range.

e. ***Reduce evening dose of insulin by at least 10% and test blood sugar every 2 to 3 hours if suspected;*** continue reducing evening insulin until hypoglycemia has been eliminated.

2. *Dawn phenomenon.*

a. ***Described as early-morning rise in blood glucose levels between 4:00 A.M. and 8:00 A.M. without prior hypoglycemia.***

b. ***Due to increased clearance of insulin.*** Hypoglycemia occurs when the clearance results in less available insulin, combined with the effect of the nocturnal growth hormone's interference with insulin action.

c. ***Not caused by the early-morning rise in cortisol.***

d. ***Testing of blood glucose at night to identify the dawn phenomenon as a cause of morning hyperglycemia.*** Test at midnight, 3:00 A.M., 5:00 A.M., and 7:00 A.M. to rule out hypoglycemia and the Somogyi phenomenon, and to determine when the blood glucose level is rising (e.g., high all night, high at midnight, high after 5:00 A.M.).

e. ***Moving the before-dinner intermediate-acting insulin to evening snack.*** This can be an effective way of improving insulin availability in the early morning without creating hypoglycemia at midnight. This results in three injections a day: combined short-acting and intermediate-acting insulin in the morning, short-acting insulin alone at dinner, and intermediate-acting insulin alone at evening snack. Changing the timing or quantity of carbohydrates at dinner and evening snack is not usually necessary. When the intermediate-acting insulin is moved to evening snack, reduce the dose by one-third initially until the blood glucose trends are well identified and nighttime hypoglycemia is ruled out.

H. Continuing care

1. *Quarterly visits with the diabetes health care provider* (primary care physician or endocrinologist/diabetologist), including history and physical examination, assessment of growth and nutrition, review of blood glucose test records and trends, insulin dose adjustment, surveillance for comorbidities and diabetes complications, anticipatory guidance, and continuing self-management education.

2. *At least annual assessment by a diabetes dietitian and a diabetes educator* are essential components of effective diabetes self-management.

3. *Quarterly measurement of glycosylated hemoglobin* serves as an indicator of overall glycemic control and a predictor of diabetes complication risk.

4. *Annual measurement of thyroid function tests* until growth is complete is imperative.
5. *Annual albumin excretion rate beginning 5 years after diagnosis (after onset of puberty)* should be tested.
6. *Annual ophthalmology dilated funduscopic examination beginning 3 to 5 years after diagnosis (and patient at least 10 years of age)* should be performed.
7. *Fasting lipid profile every 2 to 3 years* if no known cardiovascular risk exists (if cardiovascular risk is present, follow the National Cholesterol Education Program recommendations).
8. *Routine dental care* should take place every 6 months.

I. Sick day management

1. *If the child is able to eat and drink, the routine insulin doses and carbohydrate quantities should be given.*
2. *If the child cannot eat or drink, then do the following:*
   a. ***Give children on conventional therapy less intermediate-acting insulin*** (decrease usual dose by two-thirds to one-half).
   b. ***Give children on intensified therapy their usual basal insulin.***
   c. ***Give short-acting insulin as needed to maintain blood glucose levels 100 to 200 mg/dL.*** If blood glucose levels fall below 80 mg/dL, the child should start sipping on carbohydrate-containing liquids (juice, regular soda) to maintain blood glucose levels at more than 80 mg/dL.
3. *Blood glucose levels should be tested more frequently, at least every 4 hours.*
4. *Urine ketones should be checked if blood glucose levels are more than 240 mg/dL.*
5. *Abdominal pain and vomiting in the presence of hyperglycemia and ketosis are warning signs of DKA;* even if the history suggests an acute illness, DKA as an etiology for vomiting must be ruled out as soon as possible.

IV. Type 2 Diabetes Mellitus

A. General

1. *Type 2 DM is characterized by insulin resistance, β-cell dysfunction, and increased hepatic glucose production.* Individuals who are at risk for developing type 2 DM are believed to have increased insulin resistance with normal hepatic glucose metabolism and normal β-cell function for many years before developing glucose intolerance. As long as β-cell function can meet the increased demand for insulin caused by insulin resistance, normoglycemia is maintained in a hyperinsulinemic state. As β-cell function declines and impaired glucose tolerance develops, a subset of individuals will develop type 2 DM.
2. *Type 2 DM represents approximately 90% of diabetes.*
3. *Prevalence of type 2 DM in the United States varies among ethnic groups.*

a. Two percent to 8% are Caucasians.
b. Four percent to 12% are African-Americans.
c. Four percent to 19% are Mexican-Americans.
d. Thirty-five percent to 50% are Pima native Americans in Arizona (highest incidence in the world).
e. Fourteen percent to 21% are Asian-Americans.

4. *Offspring of one parent with type 2 DM have a 40% to 50% lifetime risk of type 2 DM; offspring of two parents with type 2 DM have an 80% to 100% lifetime risk.* Siblings have a 35% lifetime risk of developing type 2 DM, and identical twins have a concordance rate of 70% to 80%.

5. *Previously thought to be a disease of adults only, a rapidly increasing number of children and teenagers with type 2 DM exist, paralleling the increase in childhood obesity.*

B. Comorbidities

1. *Obesity occurs in more than 60% of people with type 2 DM.* Obesity may predispose individuals to type 2 DM, or it may be part of the genetic determinant of type 2 DM. Obesity is thought to contribute to type 2 DM risk by defects in insulin sensitivity and insulin secretion.

2. *Syndrome X, also called* ***metabolic syndrome*** *or* ***insulin resistance syndrome,*** *is characterized by glucose intolerance, type 2 DM, hypercholesterolemia, hypertriglyceridemia, elevated LDL cholesterol, elevated levels of small dense LDL, depressed HDL cholesterol levels, hypertension, microalbuminuria, high uric acid levels, atherosclerosis, and increased risk of coronary artery disease and stroke.* Insulin resistance–induced hyperinsulinemia is the central feature of the metabolic syndrome. The risk of myocardial infarction is up to 300% greater in individuals with syndrome X than it is in the general population.

C. New-onset type 2 DM

1. *Metabolic status.* Depending on metabolic status at time of diagnosis, treatment may be started on an outpatient or inpatient basis.

2. *Initial treatments.* See Section VII for information about correction of dehydration and acidosis and initiation of insulin therapy (if needed).

3. *Initial insulin therapy.* The benefits of initial therapy with insulin are debated. Individuals with type 2 DM appear in fact to be insulinopenic at time of presentation because of progressive β-cell dysfunction, following months and perhaps years of insulin resistance, hyperinsulinism, and hyperglycemia. In addition, chronic hyperglycemia induces a state of glucotoxicity, further increasing insulin resistance. Proponents of initial insulin therapy believe that early exogenous insulin administration provokes a more rapid correction of hyperglycemia and improvement in β-cell function and insulin sensitivity leading to a more successful transition to oral pharmacologic agents and/or diet and exercise management. Opponents believe that in-

sulin therapy at any time increases weight gain and the burden of diabetes care and decreases adherence to the diabetes treatment regimen.

4. *Blood Glucose testing.* For individuals taking insulin, blood glucose testing should be performed two to four times daily, depending on frequency of administration, type of insulin, and dose. For individuals taking oral pharmacologic agents, blood glucose testing should be performed one or two times daily (select the times for testing based on the need to assess fasting, preprandial, and/or postprandial glucose levels to interpret response to medication).

5. *Carbohydrates.* Carbohydrate counting is the basis for a diabetic meal plan; carbohydrates include the fruit, starch, and milk food groups and may be used interchangeably, provided the total grams match the meal plan. High-sugar foods, including candy, cookies, and so on, can be substituted for other carbohydrates throughout the day in modest quantities. If on insulin, a food plan with timed and controlled quantities of carbohydrate distributed throughout the day is necessary. Although fats and proteins in growing children have only an indirect effect on blood glucose levels, they do have calories and contribute to weight gain and thus should be taken into consideration when planning the diet. If the child is overweight, the dietitian may establish a food plan that limits fats and/or proteins to achieve weight goals.

6. *Initial self-management education.* Initial self-management education consists of immediate "survival" skills needed by the child and family, including blood glucose monitoring; administration of insulin or oral pharmacologic agents; identification and treatment of low blood glucose levels, including glucagon administration if on antihyperglycemic agents; carbohydrate counting; and urine ketone testing.

7. *Continuing self-management.* Education continues in the months following onset and gradually increases in complexity. Topics include interpretation of blood glucose trends, medication adjustment, sick-day management, treatment of high blood glucose levels and ketosis, safety at school, and safe participation in sports.

8. *Anticipatory guidance.* For older children and teenagers, include information about smoking, alcohol, drugs, preconception care and pregnancy, and driving.

D. Pharmacologic management

1. *Debate.* Some providers feel that pharmacologic management is a last resort if a trial of diet and exercise fails. Others feel that pharmacologic management should be initiated immediately, with a goal of decreasing or discontinuing medications as hyperglycemia resolves and patients experience success in diet and exercise lifestyle changes.

2. *Insulin dosage.* If insulin is used, initial doses may vary from as little as 0.1 U/kg per day to more than 2 U/kg per day, depending on need. Generally, a combination of intermediate-acting and short-acting insulins are given twice daily until blood glucose levels stabilize and acidosis resolves. Maintenance therapy is individualized for specific blood glucose patterns; for example, individuals with high morning blood glucose levels from presumed increased hepatic glucose production overnight often benefit from intermediate-acting insulin taken at bedtime.

3. *Oral pharmacologic agents.* Classified according to their mechanism of action in promoting normoglycemia, studies of the use of these drugs in children and teenagers, stimulated by the increasing incidence of type 2 DM in the young, are only beginning. See Table 37.2 for more information.

E. Diet and exercise therapy

Obesity is associated with insulin resistance; if an obese individual loses 10% to 20% of his body weight, insulin sensitivity improves (even though he may still be obese). Regular exercise improves insulin sensitivity; if a sedentary individual begins exercising 30 to 60 minutes per day, insulin sensitivity improves. The obvious assumption is that, given a choice, many people would prefer making these lifestyle changes to taking daily medications. Unfortunately, many barriers to success exist in this approach to DM management:

1. *Lasting lifestyle changes are notoriously hard to maintain,* particularly when the behaviors that need to change have been in place for many years.

2. *Many Americans live in a high-fat, high-calorie, fast-food, junk food, sedentary world, tied to a television or a computer.*

3. *Type 2 DM is overwhelmingly a family disease.* Most children or teens newly diagnosed with type 2 DM live with at least one family member with type 2 DM. If family members with DM are not following their own management plan, enlisting their support in the child's or teen's management plan will be very hard. Obesity is also often a family disease; asking children and teenagers to make diet and exercise lifestyle changes if they live in a family where overeating, being overweight, and being inactive constitute the norm may be nearly impossible. Therefore DM management involving diet and exercise lifestyle changes must involve the whole family to succeed.

4. *Many health care providers approach diet and exercise lifestyle changes as the "easy" solution and are impatient with failures and lack of progress.* The use of medication (especially insulin) may be presented as a threat or a punishment for failing to adhere to diet and exercise plans. Providers may find it difficult to link changes in therapy and increased intervention to outcomes, not the behavior (e.g., starting medication because blood glucose levels are persis-

Table 37.2. Oral pharmacologic agents for treatment of Type 2 DM

Sulfonylureas
- Stimulate increased insulin production
- Require functioning betacell for effect
- May cause hypoglycemia
- Cost and duration of action are the differences between drugs in this class
- Available preparations:
  - Amaryl (glimepiride)
  - Dymelor (acetohexamide)
  - Diabinese (chlorporpamide)
  - Glucotrol and Glucotrol XL (glipizide)
  - DiaBeta (glyburide)
  - Glynase (glyburide)
  - Micronase (glyburide)
  - Orinase (tolbutamide)
  - Tolinase (tolazamide)

Biguanides (1995)
- Increases insulin sensitivity (liver and muscle)
- Inhibits hepatic glucose release
- Increases risk of lactic acidosis; determine baseline liver and kidney function before initiating therapy
- Available preparation: Glucophage

Alpha-glucosidase inhibitors (1995)
- Delay the digestion of carbohydrates, causing slower elevation in postprandial glucose
- Thought to reduce the work of the betacell by lowering the demand for quick-release insulin in response to high-carbohydrate meals
- Side effects include flatulence and loose bowel movements
- Available preparations: Precose (acarbose), Glyset (miglitol)

Meglitinides (1998)
- Stimulates insulin secretion from the β-cells
- Quick onset and short duration of action suitable for food-related need for insulin
- Take immediately before eating
- Available preparation: Prandin (repaglinide)

Thiazolidinediones (TXDs) (1999)
- Increases insulin sensitivity
- Available preparations: Avandia (rosiglitazone maleate), Actos (piaglitazone hydrochloride)

Note: Rezulin (troglitazone), the first of the class of thiazolidinediones, was withdrawn from the U.S. market by the Food and Drug Administration in March 2000 due to reports of liver failure and related deaths. Two similar drugs, Avandia and Actos, offer the same benefits as Rezulin without the same risk, according the FDA. Visit the FDA's web site for details.

tently high, not because the patient did not lose weight or refuses to exercise). Time, patience, and the ability to listen without judging are needed to help patients identify rewards that they care about for changing diet and exercise behaviors. Finding small goals that are valued by the patient and that are achievable is a priority for building steady incremental successes for behavior change. Providers who function more as a coach and mentor, working with the patient as a partner and cheerleader, may be more successful than those who function as a directive decision maker telling the patient what he or she should and should not do.

F. Continuing care

1. *Quarterly visit with diabetes health care provider* should include history and physical examination, assessment of growth and nutrition, review of blood glucose test records and trends, insulin or other medication adjustment, surveillance for comorbidities and diabetes complications, anticipatory guidance, and continuing self-management education.
2. *An annual assessment by a diabetes dietitian and a diabetes educator* are essential components of effective diabetes self-management.
3. *Measurement of glycosylated hemoglobin every 3 to 6 months* serves as an indicator of overall glycemic control and predictor of diabetes complication risk.
4. *Annual albumin excretion rate* should be tested.
5. *An annual ophthalmology dilated funduscopic examination* should be performed.
6. *Fasting lipid profile every 2 to 3 years* if no known cardiovascular risk exists. (If cardiovascular risk is present, follow the National Cholesterol Education Program recommendations.)
7. *Routine dental care* should occur every 6 months.

V. The Child's Developmental Participation in Care

A. The child's (or teen's) role

1. *Even young children can perform many of the tasks associated with diabetes care,* including blood glucose testing, insulin administration, and carbohydrate counting.
2. *Many parents strongly encourage children and teenagers to perform diabetes tasks independently,* thinking that it is good for their child "to be independent" as he will need to "take care of himself" as an adult.
3. *Teenagers are anxious to avoid parents' watchful eyes in all areas of their life,* as they practice decision making and learn how to react to successes and failures.
4. *Unfortunately, the ability to perform diabetes tasks is not the same as that required to perform these tasks reliably all day, day after day*—including troubleshooting problems and knowing when to ask for help.

B. The parents' role

1. *Ideally, parents should maintain daily knowledge of blood glucose tests, insulin doses, food decisions at home*

and away from home, responses to hyperglycemia, and treatment of hypoglycemia—in addition to occasionally giving injections, measuring insulin doses, doing blood glucose tests at night, and checking the blood glucose meter memory.

2. *Very few children and teenagers reliably keep written blood glucose records* (much less review them to identify important trends!). This is one task that parents can easily assume, thus preventing conflict over noncompliance or falsified records. The meter memory does not lie. Parents will know when tests are done, will know what the results are, and can discuss trends with the child or teen and jointly make decisions for change.

3. *The priorities of childhood and adolescence* (play, friends, school, sports) *do not readily include tedious and often uncomfortable diabetes care activities.* The supportive safety net provided by parents can allow the child to be a child, while gradually learning the self-care skills he will need as an adult.

C. Child/teen + parent partnership

1. *Children and teenagers who have sole responsibility for the tasks of daily diabetes care* (counting carbohydrates, measuring and administering insulin, testing and recording blood glucose levels) *have worse glycemic control and more adverse events* (e.g., severe hypoglycemia, DKA episodes).

2. *Children and teenagers who share responsibility for daily diabetes care with parents* (performing tasks, interpreting data, and troubleshooting decisions) *have better glycemic control and fewer adverse events.*

3. *Helping parents find ways to stay intimately involved with day-to-day diabetes care while encouraging the child or teen to learn more, do more, and make the transition from dependence to interdependence to independence* is the ultimate challenge—and reward—of the health care provider.

VI. "Top 10" List for Achieving and Maintaining Good Blood Glucose Control

(Children, teens, parents, and providers may rank these items in different order of importance; periodically negotiate priorities to identify mutual goals for DM care.)

A. Daily blood glucose testing

B. Accurate carbohydrate counting and following a meal plan

C. Taking of medications as prescribed

D. Daily exercise

E. Maintenance of records, review of records, interpretation of blood glucose results in the context of food and exercise

F. Quarterly glycosylated hemoglobin measurement

G. Frequent contact with diabetes care provider

H. Body image and weight perception acceptable to patient

I. Family support and involvement in diabetes care

J. Other "intangibles" acceptable to patient (perceived quality of life, amount and intensity of diabetes work, perception of control over diabetes)

VII. Treatment of Diabetic Ketoacidosis

A. Mild diabetic ketoacidosis: principles

1. *Correction of hyperglycemia and acidosis* by fluid resuscitation, electrolyte normalization, and administration of insulin and substrate.

2. *Individualized treatment for DKA.* The guidelines presented below are only a starting point and must be individualized to each patient, depending on clinical assessment and condition.

B. Mild diabetic ketoacidosis: management

1. *Assessment*
   a. Ketones negative or small
   b. Serum $CO_2 > 17$
   c. Dehydration < 7%
   d. pH >7.25
   e. Oral fluids intake

2. *Fluid resuscitation.* Depending on degree of dehydration, give ½ NS + 40 mEq/L KCl/KPO_4 at 3,000 mL/M^2 per 24 hours (for maintenance and replacement) for 24 to 36 hours (see Section VII.D.4.b & c).

3. *Insulin and substrate administration.* Start subcutaneous (SC) insulin administration and diabetic meal plan (three meals and three snacks with controlled carbohydrate content) using one of the following methods, depending on assessment:

   a. ***Bolus then split dose***:

      Day 1: Give 0.1 to 0.25 U of regular insulin per kilogram every 4 to 6 hours.

      (1) Begin with 0.1 U/kg and adjust dose based on response.

      (2) A second dose may have to be given at 2 hours if limited or no response, then go to every 4 to 6 hours.

      Day 2: Add up total insulin dose from previous day and give two-thirds of total before breakfast and one-third of total before dinner, dividing each dose into two-thirds intermediate-acting and one-third short-acting. Initially, proportionately more short-acting insulin may be needed until ketones clear and blood glucose levels stabilize.

   b. ***Immediate split dose.*** Start with 0.5 U/kg per day, divided two-thirds before breakfast and one-third before dinner, dividing each dose into two-thirds intermediate-acting insulin and one-third short-acting insulin.

   c. ***Known diabetic.*** Return to pre-DKA doses if appropriate for age and weight; additional short-acting insulin may be needed until the metabolic effects of DKA resolve.

C. Moderate to severe diabetic ketoacidosis: principles

1. *Criteria for intensive care unit admission include altered mental status, pH below 7.1, age less than 2 years with pH less than 7.25, blood glucose more than 1,000 mg/dL.*
2. *Cerebral edema is a life-threatening complication of DKA treatment.* Although the etiology is unknown, the risk of cerebral edema is reduced with careful administration of fluids and free water while avoiding overrapid correction of blood glucose levels.
3. *Risk of cerebral edema is highest in children less than 5 years of age, new-onset patients, patients with hypernatremic dehydration* (corrected serum sodium more than 150 mEq/L), *and in hyperosmolar coma.*

D. Moderate to severe diabetic ketoacidosis: management

1. *Assessment*
   a. ***Dehydration > 7%:*** (an asterisk [*] indicates ominous signs)
      (1) Heart rate > 15% above normal for age
      (2) Decreased BP*
      (3) Capillary refill > 4 seconds*
      (4) Acidosis (pH < 7.25)
      (5) Dry mucous membranes
      (6) Poor skin turgor
      (7) Sunken eyes or anterior fontanelle
      (8) Oliguria*
   b. ***Serum CO_2 < 17***
   c. ***Ketones moderate or large***
   d. ***Vomiting or not taking oral fluids***
   e. ***Altered mental status.***
      (1) Possible etiologies include cerebral edema, decreased oxygen to the brain, other vascular, central nervous system (CNS), or metabolic insults (including drugs).
      (2) Cerebral edema most often occurs 3 to 22 hours after initiation of fluid resuscitation. Onset can be sudden, without other CNS symptoms.
2. *Fluid resuscitation*
   a. ***Hour 1 to 2.*** Initial fluid bolus (10 to 20 mL/kg NS over 30 to 60 minutes; repeat if >10% dehydrated or required to prevent cardiovascular compromise).
   b. ***More than 1 to 2 hours, when cardiovascular status is stable.*** Start maintenance fluids ½ NS + 40 mEq/L KCl/KPO_4 at 3,000 mL/M^2 per 24 hours (see Section VII.D.4.b & c).
3. *Insulin and substrate administration*
   a. ***Insulin therapy***. Start intravenous (IV) insulin drip when patient is hemodynamically stable (0.1 U regular insulin/kg per hour). Monitor rate of hyperglycemia correction closely; it should not exceed a drop of 80 to 100 mg/dL per hour.
   b. ***Glucose infusion***. Add glucose to intravenous fluid (IVF) (5% or 10% dextrose solutions) if serum glucose is

decreasing >100 mg/dL per hour, and/or when the blood glucose falls below 300 mg/dL, regardless of rate of fall.

c. ***Stopping IV rehydration and initiating SC insulin therapy.*** When serum CO_2 is more than 18 and PO intake is tolerated, start SC insulin; discontinue insulin drip 30 minutes to 1 hour after first SC dose of Regular insulin (discontinue drip sooner if Humalog insulin). Maintain IVF therapy until the patient retains oral intake.

4. *Electrolytes*

a. ***Hypernatremic dehydration.*** Hyperglycemia causes artificial lowering of serum sodium because of the water shift into intravascular space.

(1) Infuse rapid bolus of NS; then continue NS infusion at 2,000 mL/M^2 per 24 hours along with insulin administration.

(2) *Proceed slowly with free water correction over a 72-hour period if corrected sodium (see item 6 immediately following) is more than 150 mEq/L.*

(3) Gradually add free water (3/4 NS or more hypotonic solutions at a higher IV rate as required). Correct sodium by 15 to 20 mEq/L per 24 hours.

(4) Correct serum glucose slowly; lower to 200 to 300 mg/dL over 24 hours with little or no decrease in the corrected serum sodium.

b. ***Potassium depletion.*** Acidosis causes the shift of potassium out of the intracellular space into the serum and eventually into the urine. Therefore a normal or low serum potassium in the presence of acidosis indicates severe total body potassium depletion. Insulin administration may rapidly lower serum potassium levels. Hypokalemia can result in cardiac arrhythmias. Potassium replacement should begin the second hour of therapy to avoid hypokalemia induced by insulin administration.

c. ***Phosphate depletion.*** Osmotic diuresis and insulin administration may decrease serum phosphate levels, causing depletion of 2,3-diphosphoglycerate (2,3-DPG)—shifting the oxyhemoglobin dissociation curve to the left if the acidosis is corrected too rapidly and thereby decreasing tissue oxygen delivery. IV phosphate administration may be necessary. Monitor carefully; excessive phosphate may induce hypocalcemic tetany.

d. ***Bicarbonate.*** Rarely used in routine DKA management, consider use only with extreme acidosis (pH < 7.00), after initial bolus of NS and if ventilatory support is immediately available. Administer 1 to 2 mEq/kg slowly over 2 to 4 hours to lower the risk of CNS acidosis.

5. *Persistence of acidosis.* This is a sign of insulin deficiency, whereas hyperglycemia generally reflects fluid status. Therefore persistent acidosis indicates continued need for IV insulin (and IV dextrose if blood glucose is below

300 mg/dL), and persistent hyperglycemia indicates continued need for replacement fluids.

6. *Calculation of corrected serum sodium in presence of hyperglycemia.* Use the following equation to calculate.

$$\text{Corrected serum sodium} = \text{patient's serum sodium} + 1.6 \times [(\text{patient's glucose} - 100)/100]$$

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Web Sites

http://www.aadenet.org (American Association of Diabetes Educators)

http://www.diabetes.org (American Diabetes Association)

http://www.cdc.gov/diabetes (Centers for Disease Control and Prevention)

http://www.hrsa.dhhs.gov (Health Resources and Services Administration)

http://www.cdc.gov/diabetes (CDC Diabetes Public Health Resource)

http://ndep.nih.gov (National Diabetes Education Program)

http://www.niddk.nih.gov (National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health)

http://www.jdfcure.org (Juvenile Diabetes Foundation)

http://www.childrenwithdiabetes.com (Children with diabetes)

http://www.disetronic.com (Insulin pump information)

http://www.minimed.com (Insulin pump information)

VIII

Hematologic Disorders

38 Iron Deficiency

Omer G. Berger

Iron deficiency is the state in which the amount of iron in the body is less than is required for normal formation of hemoglobin, iron-containing enzymes, and other functioning iron compounds. Iron deficiency remains a highly prevalent nutritional disease worldwide. In the United States, according to the third National Health and Nutrition Examination Survey (1988 to 1994), 9% of children aged 1 to 2 years were iron deficient, and 3% had iron deficiency anemia. Evidence from clinical trials and prospective studies underscores the importance of iron deficiency prevention by nutritional guidance and iron supplementation. Early diagnosis and treatment are necessary to prevent the chronic adverse effects of iron deficiency anemia on cognitive performance.

I. Pathogenesis

Iron deficiency occurs in infants because of rapid growth and a diet low in iron content. Neonatal iron stores are depleted by 4 to 6 months in full-term infants and by 2 to 3 months in premature infants. Perinatal blood loss and exchange transfusions increase the risk of iron deficiency. Typical iron-deficient children are 12 to 24 months old, still use a nursing bottle, and get most of their calories from milk and/or juice.

II. Signs and Symptoms

Severe iron deficiency may result in anemia with pallor, fatigue, and anorexia. As noted earlier, a greater proportion of children have iron deficiency without anemia, which may result in irritability, decreased attention span, and motor/mental delay. These effects are presumably due to depletion of critical iron-containing enzymes and dopamine receptors.

III. Diagnosis

The traditional diagnostic test for iron deficiency anemia has been measurement of hemoglobin/hematocrit. Electronic measurement of red blood cell (RBC) indices has improved the sensitivity of the complete blood count (CBC) in detecting iron deficiency without anemia (especially ↓ MCV and ↑ RDW). In the office or clinic, the diagnosis can be made using the CBC, including RBC indices and RDW, followed by a therapeutic trial of iron (ferrous sulfate, 3 mg/kg elemental iron divided BID PO) with a repeat hemoglobin determination after 3 weeks. An increase of 1.0 g/dL or more is diagnostic. Serum ferritin, a sensitive test of iron stores, may be helpful in confusing cases. Reticulocyte hemoglobin levels, which will be available soon with automated CBCs, are likely to improve the diagnostic accuracy of iron deficiency in the future.

IV. Prevention/Treatment

In full-term infants, prevention of iron deficiency is assured by the use of human milk or iron-supplemented formula during the first year of life with intake of iron-containing solid foods at appropriate times. After the age of 6 months, breast-fed infants should receive supplemental iron in the amount of 1 mg/kg per day. Iron-enriched cereal is the preferred first solid food.

In premature infants, iron stores may be depleted by the age of 2 months. Iron sulfate drops, 2 mg/kg per day of elemental iron divided BID, should be given until adequate dietary iron intake is established.

For infants and toddlers with confirmed iron deficiency, treatment with iron sulfate drops should be started at 3 to 4 mg/kg per day of elemental iron divided BID. Each dropperful (0.6 mL) contains 15 mg elemental iron. Therefore a 10-kg child should receive 0.6 mL twice daily. After 5 days, the hemoglobin will increase 0.25 g/day until a normal level is attained. Treatment should continue for 2 to 3 months to allow iron stores to reaccumulate.

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39 Lead Poisoning: Screening and Management

Omer G. Berger

Although childhood blood lead levels have decreased remarkably in the past 20 years, lead remains a common preventable environmental hazard. Lead is a potent neurotoxicant for which no threshold for toxic effects has been identified.

Cohort studies performed during the 1980s revealed that relatively low-level lead exposure was far more toxic than previously thought. Therefore the Centers for Disease Control (CDC) recommended in 1991 that all children receive blood lead testing. Subsequent screening data have revealed many areas where the prevalence of increased blood lead levels is low, prompting the CDC to recommend in its 1997 statement that children be screened according to risk factors. State health officials were advised to make screening recommendations for specific areas (e.g., zip codes).

I. Primary Prevention

Parents must be aware of possible sources of lead in the home environment or work place. Ideally, the risk of lead exposure is discussed during the 1- to 2-month well-child visit to allow the family to eliminate the lead hazard or to relocate before the child becomes mobile.

II. Secondary Prevention—Blood Lead Screening

A. Universal screening areas. High-risk zip codes (based on age of housing and elevated blood lead levels in children 12 to 36 months of age).

Age 6 to 36 months: Test two times, 12 months apart.
Age 36 to 72 months: If child was never tested, test at least one time.
Age 36 to 72 months: If child was tested at 6 to 36 months and blood lead levels were less than 10 μg/dL, perform lead risk questionnaire.

B. Targeted screening areas. All other zip codes.

Age 6 to 36 months: Perform lead risk questionnaire annually.
Age 36 to 72 months: Perform lead risk questionnaire.

C. Medicaid recipients screened as in part A of this section, regardless of zip code (Table 39.1)

III. Management of Elevated Lead Levels (Table 39.2)

Table 39.1. Basic lead risk questionnaire*

Does your child live in or regularly visit a house that was built before 1950? This question could apply to a home day care center or the home of a babysitter or relative.
Does your child live in or regularly visit a house built before 1978 with recent or ongoing renovations or remodeling (within the last 6 months)?
Does your child have a sibling or playmate who has or did have lead poisoning?

* Screen all children whose parent/guardian responds "yes" or "don't know" to any question.

Table 39.2. AAP recommendations regarding recommended follow-up and management

Blood Lead Level (BLL) (μg/dL)	Follow-up/Action
10–15	Provide education Repeat BLL within 3 months
15–19	Obtain environmental history Provide education regarding lead exposure and absorption Repeat BLL within 2 months
20–44	Perform history and physical, including environmental and nutritional assessment Provide education about lead exposure and absorption Refer child to local or state Health Department for detailed environmental investigation Consider consultation with clinicians experienced in treatment of lead poisoning
45–69	Complete history and physical examination Provide education as above Refer to local or state Health Department for detailed environmental investigation Provide chelation therapy in consultation with physician experienced in management of lead intoxication
≥70	Hospitalize and begin treatment immediately in consultation with physicians experienced in management of lead intoxication.

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40 Sickle Cell Disease Episodes and Infections

Paul S. Bellet

I. Sickle Episodes

Sickle episodes include vasoocclusive, sequestration, and aplastic episodes.

A. Vasoocclusive sickle episodes. Vasoocclusive episodes are due to intravascular sickling, ischemia, and tissue infarction. They most commonly involve the bones, lungs, liver, spleen, brain, and penis.

1. *Pain episode.* The most common vasoocclusive sickle episode is acute pain, which is due to bone marrow ischemia. Often there are no physical findings, but local tenderness, swelling, and warmth may occur. In children less than 5 years of age, painful swelling of the small bones of the hands and feet (dactylitis) is common. If the involved bone is near a joint, a joint effusion may occur. When fever occurs with bone tenderness, bone infarction is most likely, but osteomyelitis also should be considered.

   Pain management must be individualized. Many painful episodes can be managed at home with oral analgesics. The doses described in Table 40.1 are initial starting doses. Subsequent doses can be titrated as needed.

2. *Acute chest syndrome.* This acute illness is characterized by chest pain and a new pulmonary infiltrate on the chest radiograph. Fever is a variable finding. This syndrome has been attributed to thoracic bone (ribs, sternum, vertebrae) infarction, fat emboli from necrotic bone marrow, pneumonia, pulmonary infarction, and pulmonary thromboembolism. These children may become extremely ill. In general, a complete blood count, reticulocyte count, blood culture, chest radiograph, and measurement of oxygen saturation by pulse oximeter should be done. Management consists of intravenous fluids, effective analgesia, antibiotics if febrile, incentive spirometry to prevent pulmonary infiltrate/atelectasis, and close monitoring of vital signs, pulmonary status, and oxygenation. If the patient is hypoxic, oxygen and a packed red blood cell transfusion should be given. If the patient is very ill or has progression of the infiltrate, an exchange transfusion should be performed.

3. *Acute abdominal pain.* The etiology of this common problem in children with sickle cell anemia is unknown, although it has been attributed to mesenteric sickling. The findings may be indistinguishable from an acute surgical abdomen with fever, tenderness, guarding, and leukocytosis. Intrahepatic sickling may cause pain in the right upper quadrant with an enlarged tender liver, hyperbilirubinemia, and abnormal liver enzyme concentrations.

4. *Acute central nervous system event.* Acute vasoocclusion in the brain can result in a cerebrovascular accident

Table 40.1. Recommended starting dose and interval of analgesics for pain control in sickle cell disease

Medication	Dose/Rate	Comments
Mild pain		
Acetaminophen	15 mg/kg per dose Q4H PO (child) 325–650 mg Q4H PO (adult)	Often given with a narcotic to enhance analgesia
Ibuprofen	10 mg/kg per dose Q4–6H PO (child) 400 mg Q4–6H PO (adult)	
Naproxen	5 mg/kg per dose Q12H PO (child) 250–500 mg Q12H PO (adult)	
Moderate pain		
Codeine	1.0 mg/kg per dose Q4H PO	
Oxycodone	5 mg/dose Q4–6H PO	For older children
Severe pain		
Morphine sulfate	0.15 mg/kg per dose Q2–4H IV/IM 0.2–0.5 mg/kg per dose Q4–6H PO	Drug of choice
MS-Contin (sustained-release oral morphine)	0.3–0.6 mg/kg per dose Q12H PO	Recommended >12 years of age
Hydromorphone (Dilaudid)	0.02 mg/kg per dose Q3–4H IV/IM 0.04 mg/kg per dose Q3–4H PO	
Meperidine (Demerol)	1.0–1.5 mg/kg per dose Q3–4H IV/IM/PO	Not recommended because of increased seizures but can be used if patient cannot tolerate morphine or Dilaudid
Ketorolac (Toradol)	1.0 mg/kg IV first dose, then 0.5 mg/kg IV Q6H up to the maximum: 60 mg IV first dose, followed by 30 mg IV Q6H	Not to exceed 5 days

that may be fatal. It may occur spontaneously or in the setting of a painful or aplastic episode, viral infection, pneumonia, dehydration, or priapism. Neurologic signs include hemiparesis, focal seizures, speech defects, and gait disturbance. Immediate treatment includes a packed red blood cell transfusion as soon as possible to improve oxygen delivery and an exchange transfusion to reduce Hemoglobin S (HbS) to less than 25% of the total hemoglobin mass. Computed tomography should be performed to diagnose a ruptured cerebral aneurysm or other intracranial hemorrhage. When the patient is stable, magnetic resonance imaging and magnetic resonance angiography can be performed to further characterize the lesion. Conventional angiography may also be necessary. Chronic transfusion therapy given once a month to keep the HbS level less than 30% has been successful in reducing the risk of recurrent stroke.

5. *Priapism.* The penis becomes swollen and tender, and urination is often difficult. Management includes pain control, intravenous hydration, and placement of a urinary catheter for inability to void. A transfusion of packed red blood cells should be given to raise the hemoglobin to 9 to 10 g/dL. Some patients have been helped with adrenergic agonists such as oral pseudoephedrine or intrapenile epinephrine.

B. Acute sequestration. Acute splenic sequestration may be a fatal complication in children with homozygous HbS (HbSS), Hemoglobin SC (HbSC), or sickle-beta-thalassemia disease. Massive splenic enlargement, which traps a considerable portion of the red cell mass, can rapidly lead to severe anemia and hypovolemic shock. In many cases platelets are also trapped in the spleen, producing thrombocytopenia. The usual age of presentation in children with HbSS or sickle-beta-thalassemia is 1 to 3 years. With HbSC, acute sequestration usually develops when the child is older. The immediate goal of treatment is to expand the intravascular volume with crystalloid and then to transfuse with packed red cells. If available, whole blood may be given to expand intravascular volume and increase the oxygen-carrying capacity of the blood. Usually, the spleen becomes smaller in size in a few days, and the episode resolves. With recurrent sequestration, splenectomy is recommended. Partial splenectomy has been used successfully in a number of patients.

Sequestration may also occur in the liver. The usual clinical findings are liver enlargement, hyperbilirubinemia, severe anemia, and marked reticulocytosis. Because the liver is not as distensible as the spleen, pooling of red cells significant enough to cause cardiovascular collapse rarely occurs.

C. Aplastic episodes. Aplastic episodes are usually due to intercurrent viral or bacterial infections. Parvovirus infection in particular has recently been associated with aplastic episodes. Fatigue and dyspnea may occur when there is a progressive fall in the hematocrit without compensatory reticulo-

cytosis. When the reticulocyte count is zero, the hemoglobin can decrease 0.5 to 1.0 g/dL per day. Most of these crises are mild and self-limited, and require no therapy. Close follow-up is essential if the patient is not transfused. Transfusion may be required if the anemia becomes severe (Hb < 5 g/dL).

II. Infections

Infection is the most common cause of death in children with sickle cell anemia and is related to splenic dysfunction. Common infections in sickle cell disease include septicemia (*Streptococcus pneumoniae*, *Escherichia coli*), meningitis (*Streptococcus pneumoniae*), pneumonia (*Streptococcus pneumoniae*, *Mycoplasma pneumoniae*), urinary tract infection (*Escherichia coli, Klebsiella* species), osteomyelitis and septic arthritis (*Staphylococcus aureus*, *Streptococcus pneumoniae*, *Salmonella* species), and dysentery (*Salmonella* and *Shigella* species). Since the development of an effective vaccine, infections caused by *Haemophilus influenzae* type b are much less common. Sickle cell patients who present with a known infection or with fever should be seen promptly by a physician because the risk of septicemia is much higher than in the general population. The common occurrence of fever with no obvious source in young children with sickle cell disease makes the distinction between serious bacterial infections and benign self-limited viral infections a difficult problem. Upon presentation with significant fever (>101°F), the child with HbSS or sickle-beta-thalassemia should have a CBC, reticulocyte count, and blood culture. Other tests depend on the suspected diagnosis. In most cases, ceftriaxone 50 mg/kg intravascularly (IV) or intramuscularly (IM) should be given, even before any test results are available. The decision to hospitalize a child is based on the child's age, clinical appearance, height of fever, change in steady-state hematologic values, recent compliance with prophylaxis vaccines and penicillin therapy, and the parent's ability to return promptly if the child's condition deteriorates. Most patients without a known source of infection who are managed as outpatients are given oral amoxicillin 40 to 80 mg/kg per day divided TID (the dose of amoxicillin is dependent on the regional incidence of penicillin resistance in *Streptococcus pneumoniae*) for at least 3 days until the blood culture results are known. Every child should be reexamined the day following initiation of treatment.

Prophylactic penicillin VK (125 mg PO BID to age 3 years, then 250 mg PO BID) should be given to children less than 5 years of age with sickle cell disease. The author's center also recommends penicillin prophylaxis in patients with HbSC disease, but this is controversial. The polyvalent pneumococcal vaccine is recommended at 2 and 5 years of age in addition to routine childhood immunizations.

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IX

Neurologic Disorders

41 The Evaluation and Management of Headaches in Children

David N. Franz

Headache is the most common neurologic complaint to face the primary care physician. A straightforward diagnostic strategy and therapeutic intervention are successful in most cases. The primary diagnostic concerns are to (a) differentiate neurologic from nonneurologic causes and (b) differentiate neurologic causes associated with significant morbidity (e.g., malignancy, pseudotumor cerebri) from those that are less serious (e.g., migraine). Most pediatric patients with headache do not have a serious neurologic disorder. The responsibility of the primary care physician is to identify those children whose headaches result from a serious cause and to do so in a cost-effective manner.

I. History

History is the crucial element in the categorization of headache. This should be an active process, with questioning designed to include or exclude specific entities, rather than just rote interrogation. The headache episodes should first be characterized as accurately as possible with respect to frequency, duration, and quality of the pain. Are there any provoking factors or patterns to the attacks, such as ingestion of certain foods, particular activities, or times of day? Are the headaches of recent onset and increasing in severity or frequency, or are they chronic and nonprogressive? Is the patient experiencing an aura, visual or sensory changes, fever, or stiff neck?

Complex neurologic symptoms such as diplopia, hemiparesis, or aphasia can indicate structural central nervous system disease. Children who experience syncope or presyncope may have residual headache following a syncopal event, which can lead to an erroneous diagnosis of complicated migraine. Neurocardiogenic syncope or arrhythmia should be considered in patients with alteration in level of consciousness.

Patients with headache from nonneurologic causes, such as sinusitis or temporomandibular joint (TMJ) dysfunction, will often have pain involving the face, maxillae, or temporal regions, as opposed to hemicrania or holocephalgia. Associated fever and/or meningismus raise the possibility of subarachnoid hemorrhage or infection. These conditions need not be fulminant, as with aneurysmal rupture or bacterial meningitis. Venous angiomas and other types of vascular malformations may intermittently leak small amounts of blood (sentinel hemorrhage), producing episodic symptoms. Affected children may appear normal between events. The headache of subarachnoid hemorrhage is classically described as acute and severe (*"the worst headache I've ever had"*).

Subacute and chronic infectious processes (e.g., Lyme disease, fungal, mycobacterial, or viral meningoencephalitis) should

be considered, particularly in the immunosuppressed host or in one who has recently had a course of steroid therapy. Headaches that awaken the patient from sleep or that are relieved by upright posture can be related to increased intracranial pressure. Migraine sufferers usually prefer to lie down in a dark room during an attack. Sleep disturbances such as bruxism, snoring, enuresis, night terrors, or insomnia can be clues to underlying causes such as TMJ dysfunction, sleep apnea, epilepsy, or a psychologic disorder.

What has been tried for relief, and how have the headaches affected the child's daily activities? Some patients with "severe" headaches have nonetheless never missed school or favorite activities as a result. Conversely, changes in personality, school performance, or emotional state may indicate an ongoing encephalopathy. Issues of secondary gain, personal stressors, potential drug abuse, and pregnancy should be considered where appropriate. Cocaine and amphetamine abuse can produce headache, subarachnoid hemorrhage, toxic vasculitis, and stroke, even when not taken intravenously. Refractive error (eyestrain) is rarely a source of significant head pain. Likewise, allergies in the absence of rhinorrhea, nasal congestion, and tenderness over the affected sinus(es) are not commonly a source of headache pain. Finally, psychologic/emotional issues can trigger migraines and are an important factor as in any chronic disorder. These factors are widely underrecognized in the genesis and maintenance of persistent headaches. Intercurrent stressors and issues of secondary gain should always be explored in a sensitive, nonthreatening manner.

Table 41.1 divides headache symptoms into "Serious" (i.e., associated with serious neurologic pathology) and "Migraine."

Table 41.1. Characteristics of headache pain in children

Serious Etiology	Migraine
Always in the morning	Any time of day
Awakens from sleep	Better with recumbency
Worse with recumbency	Better with emesis
Early-morning vomiting	Obvious precipitants
Persistent neurologic deficit	Family history of migraine
Chronic progressive (increased severity or frequency)	Transient neurologic deficit
	Facial pain
Polydipsia, short stature	Stress
Personality change	"Tightness, aching, throbbing"
Immunosuppression	Bilateral "band"
Valvular heart disease	
"Worst headache I've ever had!"	
Excessively stereotyped	
Bruits	

II. Past Medical and Family History

The past medical history is important, particularly for identification of conditions that may indicate an increased risk of neurologic pathology or conditions that may help direct therapy, such as seizures, asthma, renal disease, or cardiac disease. A positive family history of migraine or severe episodic headache usually supports this diagnosis. However, such a history can be obtained in up to 60% of the general population. Head pain can be associated with certain types of inherited epilepsy, which may not always produce major motor seizures. Certain cerebrovascular malformations may be familial as well.

Many prescription and proprietary medications have headache as a side effect. Examples are psychostimulants, beta agonists, theophylline/caffeine, oral contraceptives, and sympathomimetic decongestants (ephedrine and pseudoephedrine). Migraine sufferers, particularly those who smoke cigarettes, are at increased risk for stroke. This risk is further increased by oral contraceptive use. Tetracyclines and chronic steroid use, especially when being tapered, have been associated with pseudotumor cerebri.

III. Physical Examination

The physical examination supplements the history in determining specific causes of headache pain. Blood pressure, heart rate, height, and weight should always be recorded. These can indicate the presence of systemic illnesses, such as hypertension, renal disease, or cardiac arrhythmia, which may be causing headache. Children with a postural component to their headache should have orthostatic blood pressure and heart rate determinations. Obese individuals are at increased risk for pseudotumor cerebri. Poor weight gain can indicate a malabsorptive or chronic inflammatory process. Failure to thrive with preservation of linear growth can be a sign of a diencephalic lesion or tumor (diencephalic syndrome).

Examination of the nasopharynx may reveal signs of sinusitis, dental caries/abscess, and tonsillar hypertrophy. Otoscopic assessment will rule out chronic or acute otitis media. Cervical and temporal muscle spasm or tenderness can indicate trauma or an overuse syndrome.

IV. Neurologic Examination

It is important for the physician to take the time to perform a careful ophthalmologic examination, including visualization of the optic disc and fundus. Besides papilledema, the fundus should be examined for spontaneous pulsations of the retinal vessels as they cross the disc margin. When present, spontaneous venous pulsations rule out increased intracranial pressure. Their absence does not, however, necessarily mean the reverse is true. Cranial nerves, deep tendon reflexes, and cerebellar function, including tandem walking, should be checked. Brisk but symmetric tendon reflexes in the absence of extensor plantar responses ("positive Babinski sign") or other abnormal signs generally do not indicate neurologic pathology. Muscle weakness is screened

for by checking upper-extremity drift. Despite a careful neurologic examination, most children with headache will have a normal neurologic examination when symptom-free. Examining the patient while he is experiencing a headache increases the likelihood of uncovering abnormal signs.

V. Laboratory Studies

"Routine" laboratory studies such as complete blood cell count (CBC), sedimentation rate, and thyroid function tests are of low yield and thus, not useful unless indicated by specific features on the history and physical examination. A possible exception may be serum riboflavin (B2) level. Persons with low levels often suffer from chronic headache and benefit from high-dose riboflavin supplementation (300 to 400 mg daily). Clinical improvement (except for placebo effects) is unlikely unless baseline levels are low. Neuroimaging studies are the primary consideration in the evaluation of the patient with headache, particularly to exclude structural central nervous system disease. Computed tomography (CT) and magnetic resonance imaging (MRI) are of low yield when performed indiscriminately in children with uncomplicated headache. Conversely, head pain caused by potentially serious lesions can, on rare occasions, be clinically indistinguishable from migraine or other more benign types of headache. There is no "cookbook" formula to define precisely which patients need neuroimaging. This judgment is made on clinical grounds and will continue to be for the foreseeable future.

Table 41.2 lists the factors that mandate neuroimaging in a headache patient. Additionally, the physician should consider imaging in patients (a) whose headaches seem atypical, (b) who have "Serious" symptoms listed in Table 41.1, or (c) who have concurrent systemic illnesses, such as congenital heart disease. CT scanning, with and without contrast, is sufficient in most patients to screen for structural lesions. CT is quicker, less expensive, and superior to MRI in the acute detection of subarachnoid blood. MRI is preferable for visualization of the brainstem, craniocervical junction, and cerebral white matter. Small vascular malformations may be better visualized on MRI and concur-

Table 41.2. Indications for neuroimaging in headache patients

Focal neurologic deficit
Alteration in mental status
Fever and/or stiff neck
Recent trauma
Prior history of neurologic disorder
"Complicated" migraine
Postural headache (i.e., worse with recumbency)
Acute progressive symptoms
Age less than 5 years

rent MR angiography. Very small malformations may be missed on MR angiography and require traditional angiographic techniques. MR venography is useful to exclude venous sinus thrombosis in cases of pseudotumor cerebri or hypercoagulable state.

Electroencephalography (EEG) is often requested as part of the evaluation of headache patients, but its usefulness is limited to identifying headaches that are part of an underlying epileptic syndrome. EEG has poor sensitivity for intracranial mass lesions. A normal EEG does not exclude a structural lesion. Conversely, focal findings on EEG (either epileptiform discharges or background slowing) are an absolute indication for neuroimaging. Nonetheless, EEG has no role in the routine screening of children with headache. Lumbar puncture (LP) should be performed in children suspected of having meningoencephalitis, pseudotumor cerebri, or subarachnoid hemorrhage (if clinically suspected, even if not apparent on neuroimaging). Before lumbar puncture, a mass lesion should be excluded with CT or MRI. Performance of the lumbar puncture should include opening and closing pressures. Normal cerebrospinal fluid (CSF) pressure in the lateral decubitus position is less than 200 mm of water (up to 250 mm of water in obese persons). Additional studies may be indicated if there is evidence of a coexistent systemic illness.

VI. Migraine

Most children with headache have migraine. Estimates of the incidence vary widely. Recurrent, severe headaches have been reported in 33% to 77% of children. Certainly, most migraine sufferers have their first attacks in childhood. This may be followed by a long period of quiescence, stretching well into adulthood. The relapsing and remitting character of migraine (plus the significant placebo effect reported in migraine) not only confounds estimates of its prevalence, but also hampers the evaluation of therapies. A variety of classifications have been employed for migraine. Functionally, it is usually adequate to consider three types: classical, common, and complicated. Tension-type headaches are now thought to be similar in pathogenesis to migraine, and therefore treatment strategies should be similar.

A. Classical migraine consists of a stereotypic, usually visual, aura followed by throbbing hemicranial pain, nausea, and vomiting.

B. Common migraine is generally less severe, has no aura, and may be described as a tight, bandlike holocephalgic pain.

C. Complicated migraine refers to headache associated with complex neurologic symptoms, such as hemiparesis, ophthalmoplegia, visual loss, or unconsciousness, provided that these symptoms are not attributable to another neurologic disorder.

Current thinking on the pathogenesis of migraine suggests a disturbance of central serotonergic neurotransmission, resulting in nausea and sensory disturbances (aura). Many, if not most, of the seemingly disparate medications for

migraine can be found to have direct or indirect effects upon central serotonin receptors. As an attack progresses, neurogenic inflammation develops, producing pain as well as vascular instability. This latter effect is felt to be responsible for focal deficits in complicated migraine.

D. Management. In addition to the "active" interventions of pharmacotherapy, there are several "passive" aspects that are essential to successful management. Patients should keep a "**headache diary**." A blank calendar is used to note the dates and times that headaches occur. An arbitrary scale is used to document intensity of symptoms. An example would be a scale from 1 to 10, where 1 is least severe and 10 is most severe. Patients should be asked to note any associated or precipitating factors. If such factors can be identified, avoidance of them in combination with the occasional use of simple analgesics (e.g., acetaminophen) may be sufficient. **Migraine precipitants** include exertion, emotional stress, bright lights, pungent odors, such as certain perfumes or cigarette smoke, and a variety of foods. Common food offenders are chocolate, cheese, caffeine, nitrates/nitrites (as in processed meats), and alcoholic beverages. In practice, patients are counseled on potential migraine precipitants and asked to note if any of these factors are associated with their headaches. These factors are then avoided, and any change in headache frequency is noted. In this way, a few culprits can often be implicated. A **psychologic component** to the headache should be presumed and inquired about. Reassurance and counseling on the part of the physician will often suffice to address such issues. Professional counseling, stress reduction, and biofeedback techniques can be extremely valuable and are typically underutilized in headache and pain management. Success with these interventions requires an ongoing commitment from the patient and family, as well as access to a qualified practitioner (usually a clinical psychologist).

VII. Pharmacotherapy

Pharmacotherapy of migraine is divided into abortive and prophylactic measures.

A. Abortive treatment (Table 41.3) should be administered as early in the attack as possible. Simple analgesics such as acetaminophen or ibuprofen should be tried first. The nonsteroidal antiinflammatory drug (NSAID) naproxen (Naprosyn, Anaprox, Aleve) is also effective. The effectiveness of acetaminophen or NSAIDs may be improved by coadministration of small amounts of caffeine (e.g., a cola beverage or soft drink). Excedrin Migraine, a proprietary headache remedy, is a combination of aspirin, caffeine, and acetaminophen and may be suitable for adolescents. Patients with migraine who suffer vomiting with their attacks may try promethazine (Phenergan) suppositories. Enabling the patient to sleep can be helpful in aborting more severe attacks: Diphenhydramine (Benadryl) is readily available and can be

Table 41.3. Drugs for migraine treatment: abortive therapy

Agent	Dosage*	Side Effects
Ibuprofen (e.g., Motrin)	10 mg/kg PO Q4–6H, (maximum: 800 mg/dose)	Gastritis, renal injury, inhibitation of platelet function
Naproxen (e.g., Naprosyn)	5 mg/kg PO BID (maximum: 375 mg PO BID)	Gastritis, renal injury, inhibitation of platelet function
Diphenhydramine (e.g., Benadryl)	1.5 mg/kg PO QID prn sleep (maximum: 50 mg/dose)	Anticholinergic toxicity, exacerbation of seizures, sedation
Promethazine (e.g., Phenergan)	0.5 mg/kg IV/PR Q4–6H prn (maximum: 50 mg/dose)	Extrapyramidal movements, anticholinergic toxicity, exacerbation of seizures, sedation
Prochlorperazine (e.g., Compazine)	0.1–0.15 mg/kg IM Q6H (can be given IV in children > 2 years of age) (maximum: 10 mg/dose)	Same as promethazine
Chlorpromazine (e.g., Thorazine)	1.0 mg/kg IM or IV Q6H	Same as promethazine

*Per dose unless otherwise indicated.

given at home. Excessive use of acetaminophen or NSAIDs can result in "analgesic-rebound," whereby headaches persist due to withdrawal from the analgesic agent, rather than from persistence of the migraine attack. In this situation, analgesics are withheld for 3 to 4 weeks, with emphasis on prophylactic treatment and psychologic interventions.

The selective serotonin agonists known as triptans are a new class of abortive agents for migraine. They have similar mechanisms of action and toxicity but differ in pharmacokinetics and bioavailability. Sumatriptan (Imitrex) was released first in the United States and has a rapid onset of action, a short half-life, and three dosage forms—tablet, injection, and nasal spray. The latter two formulations have superior bioavailability. Zolmitriptan (Zomig) has better oral bioavailability (and hence efficacy as an oral agent) than sumatriptan but is otherwise similar. Naritriptan (Amerge) also has good oral bioavailability and a significantly longer

half-life than the others. Its longer half-life can be helpful in patients subject to attacks of longer duration or in menstrual migraines where a sufferer is prone to recurrence for several days during her cycle. Other triptans are rizatriptan (Maxalt) and eletriptan (Relpax). A transient sensation of heaviness or dysesthesia about the chest, neck, and face often occurs in persons receiving a triptan. Since triptans can also cause coronary vasospasm and acute hypertension, this symptom may provoke understandable consternation. Triptans are contraindicated in patients with angina, coronary artery disease, or uncontrolled hypertension. They may also cause cerebral arterial vasospasm and are contraindicated in headache with significant ischemic symptomatology (e.g., hemiplegic and ophthalmoplegic migraine, cerebrovascular malformation). Patients receiving monoamine oxidase inhibitors should not receive triptans. Concomitant use with other serotonergic drugs, especially selective serotonin reuptake inhibitors (e.g., fluoxetine) may result in "serotonin syndrome." This consists of akathisia, encephalopathy, tachycardia, and other signs of excessive sympathetic nervous system activity. The triptans are not approved by the Food and Drug Administration for use in children but nonetheless have been used in pediatric patients with good effect. A prudent course of action is to administer the first dose of a triptan under medical supervision to judge efficacy and to manage any side effects that may occur.

Intravenous (>2 years of age) or intramuscular (any age) prochlorperazine (Compazine) can be given in the hospital setting. Intramuscular chlorpromazine (Thorazine) has also been advocated for abortive therapy. Patients should be monitored closely for side effects of hypotension and extrapyramidal reactions, such as oculogyric crisis, and other dystonic symptoms and signs. Drugs with sedative properties should not be given to patients in whom an intracranial mass, hemorrhage, or infection has not been excluded. Phenothiazine and antihistamine drugs should also be used cautiously in patients with epilepsy, as these agents can lower the seizure threshold.

B. Prophylactic therapy (Table 41.4) should be considered in children with one or more migraines per week. A daily dose of ibuprofen 10 mg/kg can be given as prophylaxis, and additional doses can be taken as needed for acute headache, up to 40 mg/kg per day or 800 mg/dose. However, because of possible analgesic rebound, daily ibuprofen or NSAIDs should not be given for more than 1 to 2 weeks. The goal is not to continue prophylaxis indefinitely, but to attain headache control for a period of 2 to 6 months (except NSAIDs), and then to discontinue medication. Amitriptyline, inexpensive, effective, and generally well tolerated, is given in a single daily dose. Cyproheptadine (Periactin) is preferred for very young children (<5 years of age) with migraine, those

Table 41.4. Drugs for migraine treatment: prophylactic therapy

Agent	Dosage*	Side Effects
Amitriptyline	10 mg PO QHS. Increase by 10 mg at weekly intervals until headache controlled, intolerable side effects, or maximum dose reaches 3 mg/kg per day.	Sedation, dry mouth, urinary retention, constipation
Cyproheptadine	0.12–0.25 mg/kg PO BID	Sedation, increased appetite
Propranolol	10 mg PO BID (or 0.5 mg/kg BID, whichever is less). Increase at weekly intervals until headache controlled, intolerable side effects, or maximum dose 4 mg/kg per day.	Lethargy, syncope, hypotension, exacerbation of bronchospasm
Valproate	250 mg or 5 mg/kg PO QHS (whichever is less). Increase at weekly intervals by same amount on QHS or BID schedule. Usual maximum dose 1,500 mg/day or 20–25 mg/kg per day	Lethargy, alopecia, increased appetite, thrombocytopenia, hepatic failure (rare). Perform complete blood count (CBC), diff. and liver function tests (LFT's) pretreatment and at maintenance dose

* Per dose unless otherwise indicated.

with concomitant allergic symptoms, and those intolerant to amitriptyline. Propranolol is effective but can cause lethargy and exacerbation of reactive airways disease. In older children and adolescents, valproate (Depakote) may also be used; a single dose of 250 to 500 mg PO HS is often effective. Potential problems include excessive weight gain, dysmenorrhea, and rarely, hepatic failure (approximate incidence one in 150,000). Complete blood count and liver function tests should be performed at baseline and 1 to 2 months after starting valproate. Prophylaxis should always be tapered, rather than abruptly discontinued, to avoid precipitating a

rebound headache. Beta-blocking drugs must always be tapered to avoid rebound hypertension or tachycardia.

VIII. Pseudotumor Cerebri

In pseudotumor cerebri, increased intracranial pressure presumably results from an excessive volume of CSF. These patients have postural headache, vomiting, and papilledema without obstructive hydrocephalus or an intracranial mass. Risk factors include obesity, tetracycline use, cachexia, excessive intake of fat-soluble vitamins, and chronic corticosteroid treatment, especially if tapered too rapidly. Affected individuals may have associated cranial neuropathies, most commonly of the sixth (abducens) or seventh (facial) nerves. Lumbar puncture, after appropriate neuroimaging, is both diagnostic and therapeutic. Sufficient CSF is drained to bring the CSF pressure to a normal level (i.e., <200 mm of water). Untreated, patients may suffer permanent visual loss or blindness. When symptoms recur after an initial lumbar puncture (LP), treatment options include agents to decrease CSF production, such as acetazolamide (Diamox) and furosemide (Lasix), or surgical intervention to enhance CSF drainage. Optic nerve fenestration is usually tried first; refractory cases may require lumboperitoneal shunting.

IX. Referral

Neurologic or neurosurgical consultation should be sought for any child who has headache associated with focal deficits, is under 5 years of age, does not respond to the abortive and/or prophylactic interventions described earlier, or has head pain felt to be related to a nonmigrainous etiology, such as pseudotumor, infection, or hemorrhage. Additionally, consultation should be considered in any patient with one or more of the "serious" symptoms noted in Table 41.1 or with a history of antecedent trauma or neurologic disorder such as seizures.

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42 Hearing Loss

Omer G. Berger

I. Introduction

Approximately 1 to 3 per 1,000 normal newborn infants and 2 to 4 per 100 infants in the intensive care population have significant bilateral hearing loss. In 1994, the AAP Committee on infant hearing endorsed the goal of universal detection of hearing loss before 3 months of age and of intervention by 6 months of age. In 1999 an AAP Task Force on Newborn Hearing recommended universal newborn hearing screening. All newborns should be tested using physiologic measures for both ears. Acceptable methodologies are evoked otoacoustic emissions (EOAE) and auditory brainstem response (ABR). These methods are rapid, noninvasive, and relatively cost-effective. However, one cannot be certain that a child "hears" until mature enough to indicate so behaviorally.

The current recommendation for congenital hearing loss is that it be fully evaluated by age 3 months so that effective intervention can take place by 6 months of age. At the present time, severe hearing loss is diagnosed at an average age of 14 months; and moderate hearing loss, at 24 to 36 months of age. The problem is compounded by the fact that approximately 20% to 30% of those affected acquire hearing loss in early childhood. Therefore, physicians should make responding to parental concerns about the child's hearing or language development a priority and should be aware of risk factors for congenital and acquired hearing loss as indicated in Table 42.1.

II. Diagnosis and Management

An audiologist with expertise in infants and young children can determine the type and severity of hearing loss and can participate in making recommendations for management. Hearing loss may be divided into sensorineural, conductive, and mixed sensorineural and conductive. Once identified, the child should undergo a multidisciplinary evaluation and assessment.

The team of professionals may include the following:

A. A physician expert in early childhood audiologic disorders.

B. An audiologist with experience in defining the nature of hearing loss and the use of amplification devices.

C. A speech pathologist or speech teacher with expertise in the assessment and intervention of communication skills in children.

The team will develop a program based on the child's needs that is consistent with the family's resources and concerns. Parents must be given sufficient information to enable them to give informed consent. Completing the full evaluation of the child within 45 days of referral is important so that intervention can begin promptly.

The child's primary care physician plays a key role in the support of the family and the coordination of consultations and

Table 42.1. Indicators associated with hearing loss (present in 50% of infants with hearing loss)

Age	Indicator
Birth–1 month Single screening required	Family history of hereditary sensorineural hearing loss Intrauterine infection Craniofacial anomalies Neonatal problems: birth weight (BW) < 1,500 g, increased bilirubin, ototoxic medications, Apgar scores 0–4 at 1 minute or 0–6 at 5 minutes Ventilator use < 5 days of age Bacterial meningitis Syndrome known to include hearing loss
1 month–2 years Single screening required	Parental concern regarding hearing and speech Bacterial meningitis Head trauma with loss of consciousness or fracture Syndrome that includes hearing loss Ototoxic medications Recurrent persistent otitis media with effusion < 3 months duration
1 month–3 years *Periodic* hearing testing required	*Indicators of delayed-onset sensorineural hearing loss* Family history of hereditary hearing loss Intraurine infections, such as cytomegalovirus Neurofibromatosis type II *Indicators of conductive hearing loss* Recurrent/persistent otitis media with effusion ≥ 3 months duration Disorders of eustachian tube function Neurodegenerative disorders

therapy. Periodic monitoring of the child's therapeutic program is essential to maximize the development of communication skills.

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43 Febrile Seizures

Julie A. Jaskiewicz

Febrile seizures are the most common convulsive events in childhood, occurring in 2% to 5% of children between 6 months and 5 years of age. Febrile seizures are most common during the second year of life and are slightly more frequent in males. Risk factors for developing a first febrile seizure are very high fever and a history of febrile seizures in a parent or close relative. As most febrile seizures are benign in childhood, the clinician's primary goal is to make an accurate diagnosis and to provide information and reassurance to the child and family.

I. Classification

Febrile seizures may be subdivided into two groups. **Simple febrile seizures** are defined as seizures occurring in a febrile child from 6 months to 5 years of age without evidence of intracranial infection, other neurodevelopmental abnormality, or history of afebrile seizures, and which are generalized, last less than 15 minutes, and occur only once in a 24-hour period. **Complex febrile seizures** are seizures occurring in febrile children that do not meet the criteria of simple febrile seizures (i.e., they may be prolonged [>15 minutes], may be focal, may occur more than once in a 24-hour period, may be associated with concurrent intracranial infection, may occur in a child with a preexisting neurologic abnormality, and/or may occur in a child with a history of afebrile seizures).

The most common type of febrile seizure in the pediatric population is the simple febrile seizure. These seizures typically occur soon after the onset of a febrile illness and may often be the first sign of illness. The child may be incontinent and hypopneic during the seizure and lethargic or sleepy afterwards. Soon after this postictal phase, the child returns to normal baseline activity with a normal neurologic examination. The evaluation and management strategies that follow are for simple febrile seizures.

II. Evaluation

The evaluation of a child with a first febrile seizure begins by obtaining a complete description of the event that occurred in order to classify the seizure as a simple febrile seizure. If the seizure fits the criteria of a simple febrile seizure, subsequent evaluation should focus on identifying the etiology of the child's fever.

A. Laboratory. Searching for the cause of the fever with appropriate laboratory tests may be warranted and will vary according to the child's age and clinical presentation. Identifying treatable causes of seizures associated with fever, including meningitis, encephalitis, and *Shigella* gastroenteritis, as well as other infectious conditions, such as roseola infantum that do not require specific treatment, is important. Toxins and drug exposures may also be considered.

The American Academy of Pediatrics recommends that a lumbar puncture be strongly considered for all children less

than 12 months of age with a first febrile seizure. A lumbar puncture should also be considered in febrile children with seizure between 12 and 18 months of age, as the clinical signs and symptoms of meningitis in this age group may be very subtle. Although older children (>18 months) in this situation do not routinely require a lumbar puncture, children with signs of meningeal irritation or children who are being treated with antibiotics should be considered for lumbar puncture. Other laboratory tests, such as complete blood count and blood and urine cultures, are obtained based on the child's symptoms and clinical appearance at the time of evaluation. Testing for serum electrolytes, calcium, phosphorus, and magnesium is not recommended in the evaluation of simple febrile seizures unless suggested by the history or physical examination.

B. EEG and imaging. Children who have febrile seizures may have abnormal findings on EEG, but these findings do not correlate with the risk of recurrence of febrile seizures or with the later development of epilepsy. Thus most neurologists do not recommend an EEG in the evaluation of an otherwise healthy child with a first simple febrile seizure. Similarly, neuroimaging is not routinely recommended for a child with a normal neurologic examination following a first simple febrile seizure. There are no data that suggest that children with simple febrile seizures have a higher incidence of intracranial abnormalities or that simple febrile seizures result in neurologic damage.

III. Natural History

It is generally believed that most children will "outgrow" their tendency to have simple febrile seizures, as the frequency of febrile infections and the height of fever with those infections diminishes with the increasing age of the child.

A. Risk of subsequent simple febrile seizures. The risk of having a second simple febrile seizure is most strongly influenced by young age (<12 months) at the time of the first febrile seizure and a positive family history of febrile seizures. The risk of recurrence for children less than 12 months of age is 50%, whereas children more than 12 months of age at the time of their first febrile seizure have a 30% chance of recurrence. Of those children who have a second febrile seizure, 50% will have a third. Most (75%) recurrent febrile seizures will occur within 1 year of the first seizure. No evidence exists showing that children who have more than one simple febrile seizure are at increased risk for cognitive delays or other neurologic complications.

B. Risk of subsequent afebrile seizures (epilepsy). The child who has a simple febrile seizure has only a slightly increased risk of subsequent afebrile seizure above that of the general population—1%. Infants who have had multiple simple febrile seizures and who were younger than 12 months of age at the time of the first febrile seizure are at slightly

increased risk of epilepsy—2.4%. The strongest risk factors for epilepsy following a febrile seizure are (a) a positive family history for afebrile seizures, (b) a preexisting neurologic abnormality, and (c) a complex first febrile seizure. The risk for epilepsy of children who have one such risk factor is 2% and that for two or more, 10%.

IV. Management

The benign nature of simple febrile seizures suggests that an appropriate management strategy should identify the type of febrile seizure, determine the cause of fever, treat any specific inciting event promptly, avoid unnecessary side effects from medication, and educate the family about the excellent prognosis.

A. Medication. The treatment of simple febrile seizures with anticonvulsants will not prevent future epilepsy. Although continuous anticonvulsant therapy with either phenobarbital or valproic acid can be 90% effective in preventing recurrent febrile seizures, both drugs have significant adverse side effects that outweigh any potential benefit from their use. Carbamazepine and phenytoin will not prevent recurrent febrile seizures. Acetominophen every 4 hours following a febrile seizure in the absence of anticonvulsants does not prevent recurrent febrile seizures. Oral diazepam, given at the time of fever, has been shown to effectively prevent recurrent febrile seizures, although side effects, including lethargy, sleepiness, and ataxia, can occur and should be weighed against the benefit of preventing another febrile seizure.

B. Education. Understandably, many parents are frightened when they witness their child's febrile seizure. Reassuring parents of the benign nature of febrile seizures and educating them about their child's risk of subsequent febrile seizures and epilepsy is vital. Primary care physicians are ideally suited to counsel families in the office, with the long-term goal of reducing parental anxiety. In most cases, children with simple febrile seizures should be followed without specific intervention. For situations of extreme parental anxiety, the physician may opt to prevent recurrent febrile seizures with oral diazepam, given in three divided doses for a total of 1 mg/kg per day, at the beginning of the child's febrile illness. If this management strategy is chosen, the physician must be careful to observe for side effects of the drug (e.g., lethargy) and differentiate these symptom from an underlying central nervous system infection.

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44 Vision and Strabismus

Omer G. Berger

I. Eye Examination in the Infant and Young Child

Examination of the eyes should be performed at birth and at all subsequent well-child visits. Formal vision screening should be done as soon as the child is able to cooperate with standard vision testing to permit early detection of visual deficits.

In newborns at risk for retinopathy of prematurity or those with a family history of retinoblastoma, congenital cataracts, or other genetic eye problems, an ophthalmologic examination is necessary in the nursery. Beginning at the 6-month well-child visit, infants should be evaluated regularly for fixation preference, alignment, and eye disease. The health care practitioner should obtain a history of vision and eye problems and listen carefully to parents' concerns about their children's vision or eye alignment, as they are rarely wrong.

The eye examination for children under 2 years of age includes motility, eye muscle balance, pupils, and red reflex. A penlight examination is sufficient to evaluate the conjunctiva, cornea, iris, and pupils. The corneal light reflex may be used to evaluate motility and muscle balance. Reflected light should appear symmetrically in relation to the pupil (Hirschberg's test). The red reflex is elicited with the ophthalmoscope at +1 diopter to screen for abnormalities of the retina or visual axis (e.g., a cataract). By 6 months of age, a symmetrical red reflex should be obtained during simultaneous examination of both eyes in dim light. Red reflexes that are equal in brightness and coloration indicate that vision and ocular alignment are very likely normal.

For children 2 to 4 years of age, additional measures may be included. Vision testing with picture cards is recommended at 3 years of age. If the child is unable to be tested, repeat testing is done 4 to 6 months later. If the child is unable to be tested after repeated valid attempts, referral to an ophthalmologist with experience in evaluating children is indicated. Ophthalmoscopy may be performed when the child is able to fixate while the practitioner evaluates the optic disc and the retinal vessels.

By 5 years of age, nearly all children can be tested for visual acuity and can undergo ophthalmoscopy. Any child who is untestable by this age should be referred to an ophthalmologist. After 5 years of age, periodic visual screening is conducted per recommendations for preventive health care.

II. Assessing Visual Acuity

For children who know the alphabet, Snellen acuity charts can be used in the office. For younger children, a picture chart, Allen picture cards, or the HOTV test (the eye chart read by the child) are effective in identifying children with unilateral refractive errors.

Refractive errors affect nearly 20% of the population before the late teenage years. Myopia is the most common significant refractive error requiring corrective lenses. Astigmatism and unilateral refractive errors may result in amblyopia. Referral guide-

Table 44.1. Screening guidelines

Age 3–5 Years	Tests	Referral Criteria
Distance visual acuity	Picture chart or Allen cards HOTV chart Snellen test	1. <20/40 *or* 2. Two-line difference between eyes
Ocular alignment	Hirschberg's test Unilateral Cover Test Random-Dot-E Stereo Test	Asymmetry of reflection Any eye movement <four of six correct
Over 5 Years	**Tests**	**Referral Criteria**
Distance visual acuity	Snellen test Tumbling E HOTV	1. <20/30 *or* 2. Two-line difference between eyes
Ocular alignment	Unilateral Cover Test @10 feet or Random-Dot-E Stereo Test	Any eye movement <four of six correct

lines for refractive errors or ocular malalignment are listed in Table 44.1. If vision screening results are questionable, the child should be retested to minimize overreferrals.

III. Assessing Ocular Alignment

The corneal light reflex test (Hirschberg's test) can be performed at each well-child visit, especially in very young and less cooperative subjects. The unilateral cover test and the random-dot-E depth perception test are more likely to detect subtle eye muscle imbalances. Detection is especially important in children with epicanthal folds, which cover a portion of the medial sclera, giving the false appearance of esotropia. For the cover test, the examiner instructs the child to look at a distant eye chart or bright toy. Then the examiner covers the left eye and looks for fixational movement in the right eye. If the right eye moves laterally, the eye is esotropic; if it moves medially, it is exotropic. The examiner then uncovers the left eye and swiftly covers the right eye, looking for fixational movement of the left eye similarly. If either eye moves during the test, suggesting ocular malalignment (strabismus), the child should be referred to an ophthalmologist.

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X

Musculoskeletal Disorders

45 Developmental Dysplasia of the Hip

Raymond C. Baker

Developmental dysplasia of the hip (DDH) occurs in approximately 0.1% to 0.2% of infants; the disorder has a predilection for females over males at a rate of 6 to 1. The female predilection is probably due to ligamentous laxity resulting from the influence of maternal hormones, estrogen, and relaxin. Sixty percent of affected hips appear on the left side, 20% on the right, and 20% are bilateral. In infants with a family history (first-degree relative affected) of DDH, the incidence is 10 times higher. The incidence of DDH is also higher in infants born in the breech position, in infants with certain other congenital abnormalities (torticollis, clubfoot, metatarsus adductus, and hyperextension of the knee), with primiparity, and with prenatal oligohydramnios. In about 5% of infants subsequently requiring treatment for hip dysplasia, hip examination is normal at birth.

I. Diagnosis

The diagnosis and prevention of DDH in the newborn depends on demonstrating hip instability, which may lead to frank dislocation or subluxation. Since early treatment is crucial, infants, especially those at increased risk, must be screened on a regular basis for DDH. Instability of the hip suggestive of DDH is demonstrated by means of the Ortolani and Barlow tests performed as part of the initial nursery postpartum examination and again at the first office postnatal visit.

A. Ortolani test. In this maneuver the infant is examined in the supine position. The pelvis is held on one side by one hand to stabilize it during the manipulation. The opposite hip is slowly and gently abducted with the other hand, at the same time pulling the femur forward using the greater trochanter as a fulcrum, as in Fig. 45.1. In the infant with an unstable hip, the examiner will feel a sudden shifting sensation and may hear or feel a "clunk" simultaneously as the hip reduces anteriorly.

B. Barlow test. In this maneuver, the infant is examined in the supine position. The pelvis is held on one side by one hand to stabilize it during the manipulation. The opposite hip is held by the other hand in the adducted, flexed position while gentle lateral pressure is exerted over the lesser trochanter, as in Fig. 45.2. In the infant with an unstable hip, a "clunk" may be felt as the hip subluxes posteriorly.

C. Diagnosis of hip dislocation in older infants. Beyond 6 to 8 weeks of age, the Ortolani and Barlow tests are likely to be negative in infants with dysplastic hips as the soft tissues surrounding the hip tighten. Hips should be examined at all subsequent routine well-child visits to age 2 years to rule out DDH undetected at earlier visits. In the older infant signs of dysplasia are the following:

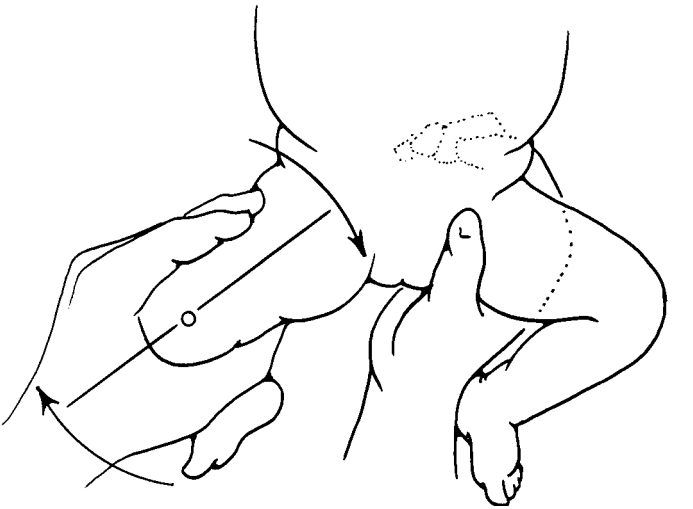

Fig. 45.1. The Ortolani test for hip instability.

1. *Incomplete or asymmetric abduction of the hips* (normal hip abduction is almost 90 degrees). This is the most reliable sign in bilateral hip dislocation.
2. *Leg length discrepancy with apparent shortening of the affected side* as measured either with the thighs and legs extended or with the infant supine with hips and legs flexed, legs adducted (the physician should look for asymmetry of the height of the knees [Galeazzi sign]).
3. *Posterior thigh and gluteal fold asymmetry.*
4. *Trendelenburg's sign caused by shortening and weakening of hip stabilizing muscles.* This is a late sign of DDH.

II. Confirmation and Treatment

The diagnosis of hip instability and dislocation is confirmed by real-time ultrasound of the hip in the newborn (up to 3 to 5 months of age) and by hip radiographs in the older infant (after the appearance of femoral nuclei of ossification when the bony architecture of the hip is developed enough for reliability of radiographs). Infants in whom instability, subluxation, or dislocation can be demonstrated should be referred to the orthopedist promptly. Treatment of hip instability or easily reduceable dislocation in the newborn period is usually uncomplicated and prevents subsequent dislocation and deformity. Treatment in the newborn period (and up to 5 to 6 months of age) consists of bracing or splinting the hips in the abducted, flexed position, com-

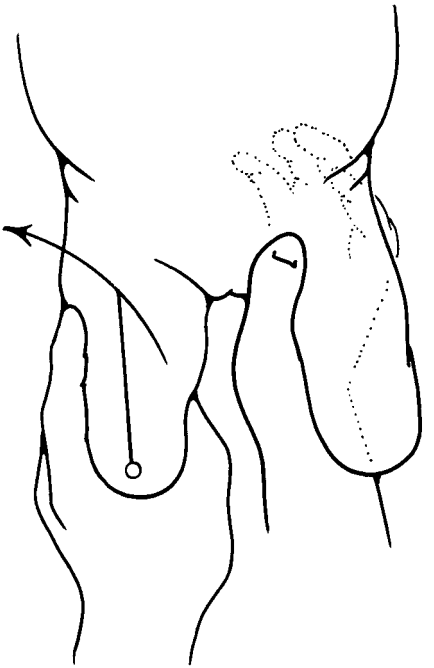

Fig. 45.2. The Barlow test for hip instability.

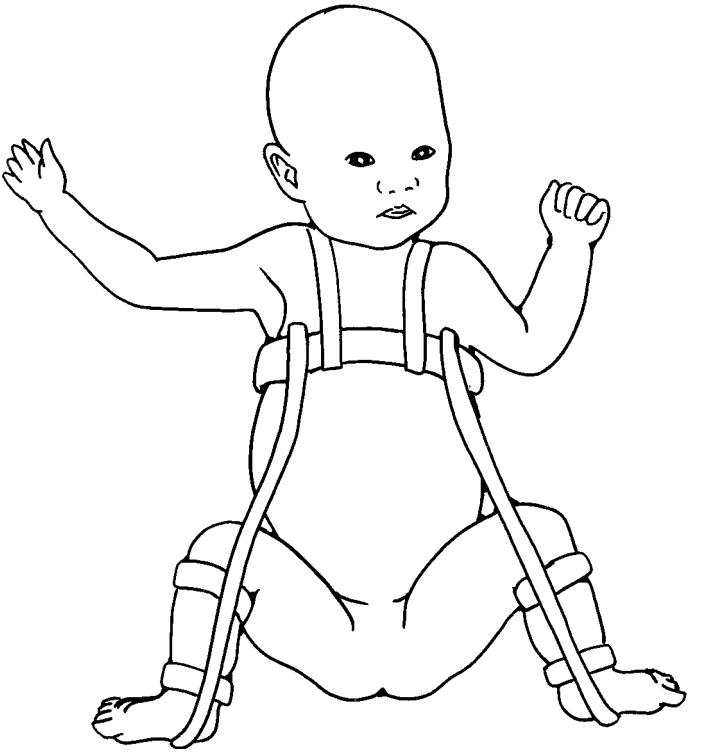

Fig. 45.3. The Pavlik harness. Treatment of hip instability consists of bracing or splinting the hips in the abducted, flexed position, commonly with the use of the harness.

monly with the use of the Pavlik harness (Fig. 45.3). The Pavlik harness has a success rate of 85%, with 2% incidence of avascular necrosis, a known complication of hip dislocation/subluxation. Beyond 6 months of age, other treatment modalities are used by the orthopedist, such as hip spica cast, open surgical reduction, and surgical release of the hip abductor and iliopsoas muscles.

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46 Genu Varum and Genu Valgum

Raymond C. Baker

I. Genu Varum (Bow Legs)

During growth and development of the lower extremities in children, physiologic bowing of the lower extremities as a variation of normal is common and usually resolves by 24 months of age. Coexisting internal tibial torsion exaggerates the appearance of bowing. The physician should anticipate questions about physiologic bowing and inform parents that this is usually normal and requires no treatment. However, quantitating the degree of deformity at each visit to document resolution, which usually begins by age 18 to 24 months, is important. A simple clinical method of quantitation is measuring the distance between the medial aspects of the knees with the child lying in the supine position, thighs and legs adducted to the midline with the medial malleoli touching (Fig. 46.1). If the distance is greater than 10 cm or if the condition does not improve by 24 months of age, the physician should consider orthopedic referral. Radiographs of the knees to rule out other conditions, such as rickets or tibia vara (Blount's disease), may be indicated, but this may be deferred to the orthopedist if orthopedic consultation is obtained.

II. Genu Valgum (Knock Knees)

Genu valgum is less common than genu varum and occurs in the older child between 3 and 5 years of age, more commonly in girls. Although several factors may affect the degree of genu valgum (lax medial collateral ligaments, pes varus, and obesity), it is a normal variation resulting from the process of remodeling of the lower extremities during growth and development. It is quantitated clinically by placing the child in the supine position with the thighs and legs adducted so that the medial aspects of the knees are touching and measuring the distance in centimeters between the medial malleoli of the ankles (Fig. 46.2). A distance greater than 10 cm at any age is an indication for orthopedic referral. Lesser degrees of genu valgum usually resolve by 6 to 8 years of age and rarely require orthopedic intervention. The physician should anticipate questions from parents about this condition and should discuss the transient nature of the condition.

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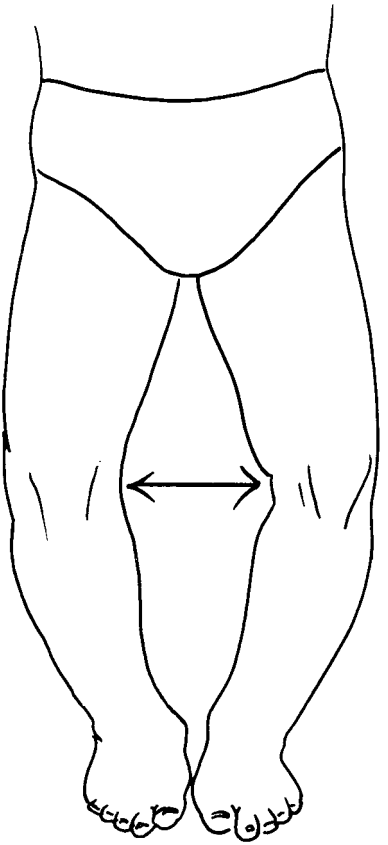

Fig. 46.1. Quantitation of genu varum (bow legs).

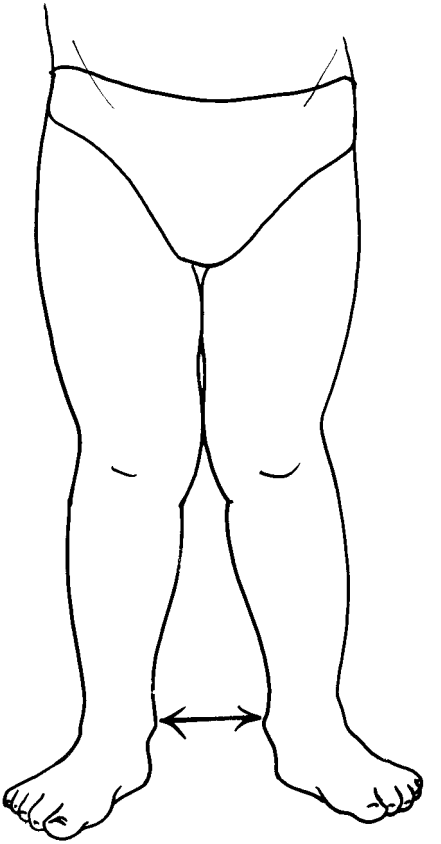

Fig. 46.2. Quantitation of genu valgum (knock knees).

47 Evaluation of the Patient with Intoeing

Raymond C. Baker

Intoeing (pigeon toes) in the infant and child is the most common orthopedic complaint parents have for the primary care physician during the course of well-child care. The most prevalent causes of intoeing are (a) metatarsus adductus (MA) in the young infant (birth to 6 months), (b) internal tibial torsion (ITT) in the toddler (12 to 24 months), and (c) femoral anteversion (FA) in the preschool- and school-aged child (2 to 5 years of age). Although all these are usually normal developmental variations and tend to resolve with growth of the child, they should be quantitated at each visit to document that resolution. A minority of patients with each of these deformities may require special exercises or orthopedic referral for excessive degrees of the deformity or for failure to resolve.

I. Physical Examination

The physical examination is performed with the child in the prone position, thighs extended and legs flexed to 90 degrees; the examiner is at the foot of the examining table (Fig. 47.1). This allows evaluation and quantitation of all three of the common causes of intoeing.

In this position, the three causes of intoeing can be differentiated. As the examiner looks down upon the bottoms of the feet, the "C"-shaped foot of metatarsus adductus is apparent (Fig. 47.2). Other findings in metatarsus adductus are a medial cleft, widened space between the first and second toes, and prominence of the base of the fifth metatarsal. Because MA is thought to result from molding secondary to intrauterine crowding, the physician should examine for other orthopedic deformities from molding, such as torticollis, hip dysplasia, and ITT. The degree of MA can be quantitated using an inexpensive plastic goniometer to measure the angle formed by a straight line drawn through the long axes of the hindfoot and forefoot, as indicated in Fig. 47.2.

If the foot is straight but there is internal rotation of the long axis of the foot relative to the femur, then internal tibial torsion is present (Fig. 47.3). Similar to MA, the physician should examine for the presence of other orthopedic deformities from molding, such as torticollis, hip dysplasia, and MA. The degree of ITT can be quantitated with a goniometer by measuring the angle formed by straight lines drawn through the long axes of the foot and the femur, as indicated in Fig. 47.3.

If both of these are normal and there is increased internal rotation at the hips tested with the child in the same position, then femoral anteversion is present (Fig. 47.4). Normal rotation at the hip is about 70 degrees external rotation and 50 degrees internal rotation. In FA, 80 to 90 degrees of internal rotation is usually present.

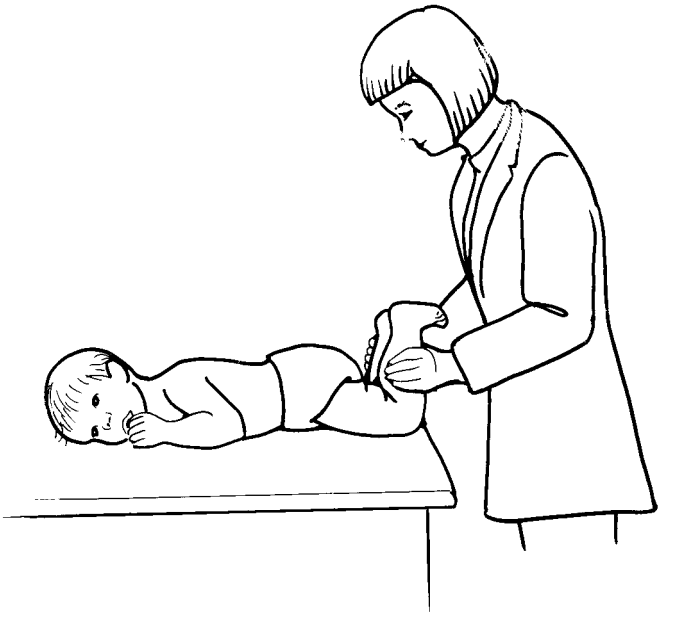

Fig. 47.1. Physical examination. Best position for performing the physical examination for intoeing.

II. Treatment of Metatarsus Adductus

The treatment of metatarsus adductus is dependent on the grade of the deformity present.

A. Grade I. The degree of angulation is less than 10 degrees; the forefoot can be passively stretched past neutral easily; and stroking the lateral aspect of the foot causes reflex straightening. No treatment is necessary, and the condition resolves spontaneously.

B. Grade II. The degree of angulation is more than 10 degrees, and the forefoot can be passively stretched past neutral easily. The physician should demonstrate stretching exercises for the caregiver and recommend them at each diaper change. (Stretching exercises consist of grasping the hindfoot in one hand and then grasping and stretching the forefoot as far past the neutral position as possible with the other hand to a count of 5. Perform five times, five times a day, or at each diaper change.) The infant should be checked for resolution of the MA at each visit. If it does not resolve by age 5 to 6 months, orthopedic referral is indicated.

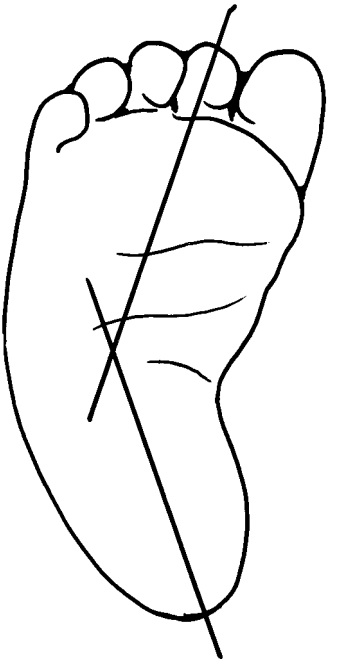

Fig. 47.2. Metatarsus adductus.

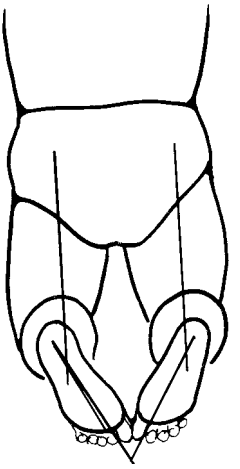

Fig. 47.3. Internal tibial torsion.

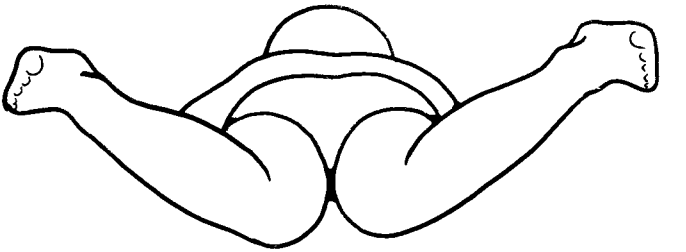

Fig. 47.4. Femoral anteversion. Increased internal rotation of the hip with femoral anteversion.

C. Grade III. Degree of angulation is more than 10 degrees, and the forefoot cannot be passively stretched past the neutral position. This requires immediate orthopedic referral for serial casting (preferably by no later than 2 months of age).

III. Treatment of Internal Tibial Torsion

Internal tibial torsion usually requires no specific treatment and resolves spontaneously. When diagnosed, the degree of torsion should be quantitated so that resolution can be measured at each well-child visit. If there is a significant decrease in the angle after the child has been walking for 1 year, the problem will usually resolve without further treatment. If the degree of angulation is more than 35 degrees at any age or if significant resolution has not occurred by the age of 2, then orthopedic referral is appropriate (for bracing in external rotation). The habit position of sitting or lying down on the feet with the hips and legs flexed and feet internally rotated underneath (Fig. 47.5) exacerbates this problem and should be discouraged.

IV. Treatment of Femoral Anteversion

Femoral anteversion is seen more commonly in females and is the most common cause of intoeing in preschool- and school-aged children. Family history is often positive. The hips can be internally rotated 80 to 90 degrees easily while external rotation is decreased or normal. The habit of sitting in the "W," or "TV," position (Fig. 47.6) tends to exacerbate this condition and should be discouraged. The child should be encouraged to sit Indian style with legs crossed. If the hips can be externally rotated greater than 25 degrees, then the deformity can be compensated, usually consciously by the child, by 10 years of age. Self-correction can be encouraged by activities that emphasize external rotation, such as ballet and ice skating. Orthopedic referral is rarely necessary for this condition. However, if the child's gait is affected enough to cause frequent falls, external rotation is limited to less than 25 degrees, the abnormality is unilateral, or no improvement occurs by 6 to 8 years of age, then referral to an orthopedist is appropriate.

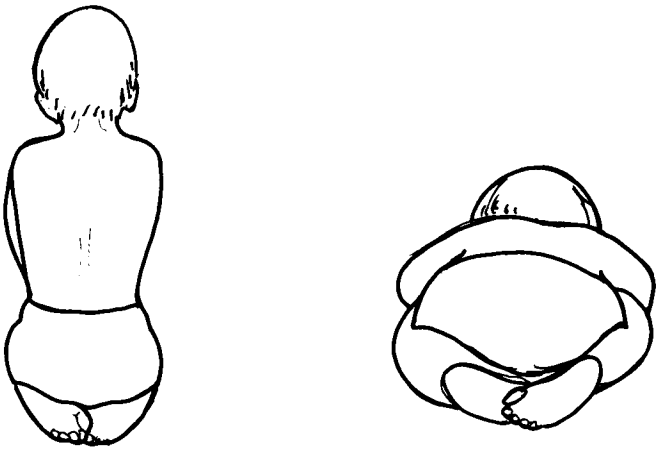

Fig. 47.5. Habit position exacerbating internal tibia torsion. These two positions exacerbate internal torsion and should be discouraged.

Fig. 47.6. Habit position exacerbating femoral anteversion. The "W," or "TV," position exacerbates femoral anteversion and should be discouraged.

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XI

Miscellaneous Topics

48 Antibiotic Selection and Compliance

Raymond C. Baker

Many different oral antibiotics are available that have a broad spectrum of activity against organisms causing the common infections encountered in the pediatric outpatient arena. The choice of antibiotic depends on many factors, the most important of which is the sensitivity of the probable etiologic organism. However, many subjective factors must also be considered in the individual setting. The final antibiotic choice by the primary care provider must take into account both the science of infectious diseases (organism sensitivity, mechanism of action, resistance factors, antibiotic stability, and antibiotic pharmacokinetics) and the psychobehavioral aspects of children (getting the child to take and retain the antibiotic) to ensure compliance in the patient and effectiveness against the organism. Antibiotic efficacy and organism sensitivities are addressed elsewhere in this book. The following information addresses the more practical and subjective aspects of antibiotic selection.

I. Taste

Many antibiotics have a bitter taste (especially penicillins) that is often poorly masked by flavorings. There are exceptions; for example, some of the cephalosporins have a more pleasing taste, but they tend to be more costly. At the same time, children are very different in their willingness to accept unpleasant-tasting medicine. Some children will take anything, and others need cajoling, bribing, and sometimes tears before "the medicine goes down." Parents usually know how well the child accepts medications; this information should be sought from them and used to help select the antibiotic (i.e., prescribing an antibiotic that tastes better even if it is more costly).

Several taste comparisons have been published in the pediatric literature in which antibiotics were ranked according to taste. In studies with adults sampling the different antibiotics, Lorabid, Suprax, Keflex, Cefzil, Duracef, Ceclor, Augmentin, and amoxicillin were considered palatable, whereas Prostaphlin, Dynapen, Biaxin, VeeTids, Vantin, Ceftin, Pediazole, and Sulfatrim were less tasty; erythromycin estolate (Ilosone) ranked significantly higher than the ethylsuccinate form (EES); and Zithromax outranked Biaxin significantly. In studies using children as tasters (who better to judge antibiotic tastes!), antibiotics ranked as good-tasting were Zithromax, Augmentin, Lorabid, and Suprax; lower-ranking antibiotics included Biaxin, Pediazole, Cefzil, and Vantin.

II. Frequency of Administration

Medications with a short half-life, which must be given often, may cause a problem with compliance. In general, minimizing the frequency with which medication is given increases the likelihood and ability of the parent to comply with the regimen.

Antibiotics that require only once-a-day dosing are Suprax, Cedax, and Zithromax; those requiring twice-daily dosing are trimethoprim/sulfamethoxazole, amoxicillin, Augmentin, Cefzil, Lorabid, Ceftin, and Biaxin. A strategy for improving the compliance in medications that must be given more frequently is to try to relate the medication administration to some other life event as a reminder (e.g., with meals or upon awakening and at bedtime).

III. Cost

Prescriptions are paid for in many ways. Some insurance companies and Medicaid generally pay for medications (especially if the medication requires a prescription). Some insurance companies require a copayment. Some parents must pay for the entire prescription out of pocket. Before prescribing an antibiotic, the physician should ask the parent how prescriptions are paid for; this may affect the choice. For example, the newer cephalosporins and macrolides tend to be costly, whereas the old stand-bys that are available as generics are less expensive (e.g., amoxicillin, penicillin, trimethoprim/sulfa, erythromycin).

IV. Absorption with Food

Some antibiotics are poorly absorbed in the presence of food in the stomach and therefore must be given between meals. Others are unaffected. Since young infants tend to be fed frequently, this may be an issue in selecting an antibiotic. In general, the macrolides, amoxicillin with clavulanate, the sulfas, and the cephalosporins are unaffected by food in the stomach. The tetracyclines, penicillin G, cloxacillin, dicloxacillin, ampicillin, and nafcillin should be given an hour before or 2 hours after meals.

V. Heat Stability (Refrigeration Requirement)

Most antibiotics that must be reconstituted with water at the time they are dispensed require refrigeration, and this will be indicated on the bottle. Although this does not commonly present a problem, it may be a consideration for families who do not have access to refrigeration or who are traveling. Refrigeration is not required for the sulfas, trimethoprim/sulfa, erythromycin, griseofulvin, clindamycin, loracarbef, and tetracycline. Most of the penicillins and cephalosporins require refrigeration.

VI. Drug Interactions

Relatively few drug interactions occur with the antibiotics, but some may require consideration when prescribed. Examples are the following:

A. Tetracycline *and* antacids and anticonvulsants.

B. Erythromycin *and* theophylline preparations and anticoagulants.

C. Amoxicillin *and* oral contraceptives.

D. Chloramphenicol *and* anticonvulsants (phenobarbital, phenytoin).

E. Clindamycin *and* diphenoxylate-atropine.

F. Furazolidone *and* alcohol.

G. Griseofulvin *and* phenobarbital.

H. Ketoconazole *and* terfenadine, astemizole, and cisapride.

I. Rifampin *and* anticoagulants and chloramphenicol.

J. Sulfonamides *and* anticoagulants and chloramphenicol.

K. Trimethoprim/sulfonamides *and* cyclosporine.

VII. Physiological Interactions

In children with chronic illnesses, attention should be paid to the mode of excretion of antibiotics when they are prescribed. Specifically, if the antibiotic is excreted in the urine (penicillins, aminoglycosides, cephalosporins), caution should be used in children with abnormalities of renal clearance. Likewise, in children with significant liver disease, drugs excreted in the bile should be used with caution (erythromycin, tetracyclines, chloramphenicol).

VIII. Adverse Reactions

Finally, the likelihood of an allergic reaction or other significant side effect should be considered, and a careful allergic history should be obtained. When taking a drug allergy history, the physician should seek details about the "allergic reaction," since parents may consider nonallergic side effects of antibiotics, such as nausea, vomiting, or diarrhea, allergic reactions. The following are examples of drug effects that may affect the antibiotic choice:

A. Serum-sickness-like reactions (3% to 5%) with cefaclor.

B. Erythema multiforme with sulfonamides.

C. Nausea with erythromycin (should be given with food).

D. Diarrhea with amoxicillin/clavulanate.

E. Urticarial/anaphylactic reaction with penicillins.

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49 The Evaluation of Fever in Infancy

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Children from birth to 36 months of age with fever but without localizing signs comprise a large fraction of ambulatory pediatric ill visits. Young children with fever frequently present a diagnostic and therapeutic problem for the health care provider. Although fever is frequently self-limited and the incidence of serious bacterial infections (SBI) in these children is relatively low, serious infections may be present. The most common SBI in this age group are bacterial meningitis, bacteremia, and urinary tract infection (UTI).

Proposed management strategies seek to identify infants (a) who are at low risk for SBI, which suggests outpatient management, or (b) who are at increased risk for SBI, which mandates further laboratory studies and expectant antibiotic therapy. In the very young infant, the physician should assume that the ability of the immune system to defend against pathogens is limited and that this ability matures during the early months of life. Such young infants are at higher risk for serious infections than are older children. Because of this, in many areas standard practice is to admit febrile neonates (birth to 28 days), to perform a complete diagnostic evaluation for SBI, and to administer parenteral antibiotics until results of blood, urine, and cerebrospinal fluid (CSF) cultures are reported negative. Investigations, however, have attempted to evaluate the efficacy of managing fever, even in young infants, in an ambulatory setting with or without empiric antibiotics. Infants and young children are usually divided into two groups based on age and ability to clinically assess severity of illness: birth to 2 months and 3 to 36 months.

I. Epidemiology

SBI include meningitis, bacteremia, urinary tract infection, pneumonia, enteritis, and bone or joint infections. Depending on clinical screening criteria and the definition of toxicity used, febrile infants less than 60 to 90 days of age with temperatures of more than 38°C rectally have a 1.4% to 17.3% probability of having an SBI, including 1.1% to 10.7% probability of bacteremia and 0.5% to 3.9% probability of meningitis. The risk of occult pneumococcal bacteremia in highly febrile children 3 to 36 months of age is reported to be between 2.5% and 11%. Ten percent to 25% of untreated patients with occult pneumococcal bacteremia experience complications, including 3% to 6% in whom meningitis develops.

II. Etiology

Etiologic agents of bacteremia in infants 2 months of age and younger include group B streptococci, *Streptococcus pneumoniae, Salmonella* sp, *E. coli*, *N. meningitidis*, and *Haemophilus influenzae* type b. The bacteria most commonly isolated from the blood of children aged 3 to 36 months with fever without a

source are *Streptococcus pneumoniae* (85%) and *Neisseria meningitidis* (3%). Infections caused by *Haemophilus influenzae* type b (Hib) have almost completely disappeared with routine administration of the Hib vaccine. When the recently developed conjugate pneumococcal vaccine becomes universal, the incidence of *Streptococcus pneumoniae* as an etiology of bacteremia (as well as other invasive diseases) is also likely to decrease significantly. Other pathogens less commonly associated with bacteremia include *Staphylococcus aureus, Streptococcus pyogenes,* and *Salmonella* sp.

Pathogens associated with bacterial meningitis in infants in the first 2 months of life include group B streptococci, *E. coli* and other enteric gram-negative bacilli, and *Listeria monocytogenes.* Pathogens typically causing community-acquired meningitis in older children, such as *Streptococcus pneumoniae* and *Neisseria meningitidis,* may also be found in younger infants. Once again, the proportion of children with meningitis caused by *Haemophilus influenzae* type b has decreased significantly because of widespread utilization of the vaccine against this organism.

III. Clinical Assessment and Management

No reliable means of identifying all children with SBI has been developed. However, screening criteria to identify infants and young children at low or high risk for SBI have been utilized by a number of investigators. These criteria most commonly involve history, physical examination, and specific laboratory parameters, which are then used to identify patients either who require no antibiotics and can be managed as outpatients or who require antibiotics as either an outpatient or an inpatient. Many practitioners are reluctant to base management decisions on clinical judgment alone, preferring such diagnostic tests with selected, and fairly frequent, use of antibiotics. The impact of the practice of empirical antibiotic use to the development of antibiotic resistance must be considered.

A. Young infants less than 3 months of age. Young infants with fever have been managed very conservatively, since they are notoriously difficult to assess clinically and are more prone to SBI because of their immature immune systems. Febrile neonates (0 to 28 days) in particular are considered high-risk patients. A complete evaluation should be performed, including complete blood count (CBC) and differential, blood culture, analysis and culture of CSF, and analysis and culture of urine obtained by catheterization or bladder tap. Stool culture and chest radiographs are obtained, if indicated. Antimicrobial therapy should be instituted in the hospital.

Some investigators have advocated selecting subgroups of febrile infants from 28 to 90 days of age and stratifying them as either low- or high-risk for serious bacterial infections. Infants who are at low risk for SBI based on certain criteria may be managed either with or without antibiotics as

outpatients. Low-risk criteria have been studied by the Rochester group and include the following:

1. *Infant appears generally well.*
2. *Infant has been previously healthy:*
   a. Born at term (≥37 weeks gestation).
   b. Did not receive parenteral antibiotics.
   c. Was not treated for unexplained hyperbilirubinemia.
   d. Has not received and was not receiving antimicrobial agents.
   e. Has not been previously hospitalized.
   f. Has no chronic or underlying illness.
   g. Was not hospitalized longer than mother.
3. *Physical examination reveals no evidence of skin, soft-tissue, bone, joint, or ear infection.*
4. *Laboratory values:*
   a. Peripheral blood white blood cells (WBC) 5,000 to 15,000/μL
   b. Absolute band form count ≤ 1,500/μL.
   c. ≤10 WBC per high-power field (×40) on microscopic examination of a spun urine sediment.
   d. ≤5 WBC per high-power field (×40) on microscopic examination of a stool smear (for infants with diarrhea).

A physician thoroughly familiar with caring for young children must evaluate patients. Infants who meet such clinical criteria appear to be at low risk for SBI and may be candidates for outpatient management. Close follow-up visits are mandatory.

Alternatively, some recommend that low-risk infants be managed as outpatients and receive therapy with an antibiotic that will empirically treat the presumed agents of bacteremia. A third-generation cephalosporin, such as ceftriaxone at a dose of 50 mg/kg intramuscularly is a suitable antibiotic because of its long half-life. The child should return for reevaluation within 24 hours. Any child who cannot return for repeat evaluation should be admitted to the hospital. At the return evaluation, results of cultures should be reviewed. Ill-appearing infants or those with positive blood or CSF cultures must be admitted for therapy. A second intramuscular injection of ceftriaxone may be an option. Continued close observation and immediate return to the hospital if needed must be always assured.

B. Three to 36 Months. Well-appearing older children, 3 to 36 months of age, with fever below 39°C without a source need neither laboratory testing nor antibiotics. Parents, however, must be instructed to return if fever persists for more than 48 to 72 hours or if the child's condition deteriorates.

The appropriate and cost-effective evaluation and management of children with fever of ≥39°C are varied both in the pediatric literature and in practice. Conservative management consists of attempting to identify a subgroup of febrile children who are at increased risk for SBI, specifically occult

bacteremia (OB), by obtaining a CBC, blood culture, and urinalysis and culture (the latter on all females and males under 6 months of age). Chest radiographs and cultures of stool are performed if indicated by the history. A child with a WBC count ≥15,000/μL may be at increased risk for OB.

A conservative approach in the child so identified as being at increased risk for OB would be expectant antibiotic therapy pending culture results. Before penicillinase-producing *H. influenzae* became significant (and before *Haemophilus* immunization), oral amoxicillin alone or procaine penicillin and amoxicillin were commonly used. Currently, intramuscular ceftriaxone 50 mg/kg is popular due to its long half-life and its extended coverage. Patients should be reevaluated within 24 hours. Ill-appearing children or those with positive blood cultures should be admitted for further treatment.

The overwhelming majority of cases of OB in 3- to 36-month-old children in areas with widespread Hib vaccination are now due to *S. pneumoniae.* Most pneumococcal bacteremias resolve spontaneously without sequelae. Furthermore, no evidence suggests that antibiotic therapy in these children prevents meningitis, the most severe but rare complication of OB. Overuse of antibiotics, on the other hand, is clearly the major reason for development of drug-resistant bacteria. Concerns about cost containment, the dramatically decreased incidence of OB due to *H. Influenzae* since the vaccine, and increased knowledge and experience with OB have prompted some practitioners to resist the urge to use antibiotics empirically in children without localizing signs who look well. The site of care and the relationship between the physician and the patient affect the decision. In a primary care setting with patients who are followed on a regular basis, the practitioner may elect to follow such patients clinically with no laboratory evaluation (or with blood culture only). If the fever persists beyond 24 to 48 hours or if the patient does not respond to antipyretics with improved clinical appearance, further work-up may be appropriate. On the other hand, a physician evaluating a febrile infant unknown to him/her in an emergency room or urgent-care setting is more likely to obtain a CBC and blood culture and treat expectantly using the preceding criteria.

Any ill- or toxic-appearing child in this age range must be admitted to the hospital following appropriate laboratory evaluation, usually including a CBC, blood culture, and CSF evaluation. Toxicity is generally defined as a clinical appearance consistent with sepsis (e.g., unconsolable irritability, lethargy, signs of poor perfusion, hypoventilation, cyanosis). Unfortunately, clinical assessment of toxicity is the least objective screening criterion available to the primary care physician and requires the most clinical experience. McCarthy attempted to quantitate clinical toxicity by developing the Infant Observation Scale, but the appearance of toxicity is

affected by the presence of fever. Some children may appear toxic while the temperature is elevated but become less ill appearing, even playful and well appearing, after defervescence. This has led to the common practice of giving antipyretics to febrile children upon arrival in a care facility even before evaluation. Improved clinical appearance with defervescence may obviate the need for lumbar puncture and admission in some children.

No management strategy will identify all patients at risk for SBI. Meticulous clinical assessment, judicious use of laboratory testing, and close follow-up are warranted when caring for the febrile infant and young child.

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50 Management of Infants with Perinatal HIV Exposure

Raymond C. Baker

I. Introduction

The second decade of the AIDS epidemic has seen a shift in the mode of transmission of the human immunodeficiency virus (HIV) toward heterosexual activity and intravenous drug use. This trend has resulted in an increasing number of infants who are exposed perinatally to HIV. Currently, almost 2,000 children under age 13 in the United States are HIV infected (predominantly perinatal acquisition), and 3,700 have AIDS. Of HIV-infected infants and children, about 60% are African-American; 24%, Hispanic; and 14%, Caucasian. The risk of perinatal vertical transmission of HIV from mother to infant (mother not on antiretroviral therapy) is 25% to 30%. If the mother receives antiretrovirals during pregnancy (and the infant receives antiretrovirals at birth and in the few weeks of life), the rate of vertical transmission drops to 7% to 8%; if the infant is delivered by C-section and the mother is treated with antiretroviral agents, the risk of vertical transmission is only 2%.

Because of the effectiveness of these interventions in preventing vertical transmission, the primary strategies in the prevention of pediatric HIV infection are (a) identifying HIV infection in pregnant women, (b) treating them (and their infants) with antivirals during the pregnancy, and (c) performing C-section on HIV-infected mothers in an attempt to prevent postnatal infection resulting from contamination with infected maternal blood during a vaginal birth. An additional strategy to maximize the effectiveness of antiretroviral therapy in infants who do prove HIV infected is confirmation and treatment as early as possible. Although long-term studies of the beneficial effects of early treatment of HIV-infected infants have not yet been performed, general principles of therapy of infectious diseases suggest that clinical outcome is improved in infants diagnosed and treated as early as possible with antiretroviral drugs.

The management of infants born to HIV-positive mothers therefore requires a cooperative effort between the obstetrician and the pediatrician. The obstetrician must (a) assess risk factors and test pregnant women at increased risk for HIV infection (an alternative strategy would be to test all pregnant women for HIV infection), (b) treat HIV-positive pregnant women with antiretroviral agents during the pregnancy, and (c) consider C-section to reduce the possibility of vertical transmission even further. The pediatrician must (a) be aware of the mother's HIV status and treat HIV-exposed infants following delivery with antiretrovirals, (b) test infants of HIV-positive women for HIV infection in order to initiate early therapy if positive, (c) provide appropriate primary care to infants regardless of HIV status, and (d) provide appropriate HIV-specific care to infected infants.

II. History

The evaluation and management of the infant born to an HIV-infected woman begins with the maternal and perinatal history, which should include specific HIV-related information as follows:

A. Maternal history

1. *Mode of transmission* to mother (sexual, drug use, blood products).
2. *Symptoms of HIV infection.* (The rate of HIV transmission is increased in mothers with more advanced disease.)
3. *Sexually transmitted disease (STD) history* and prenatal STD screening. (Maternal STDs are associated with an increased rate of HIV transmission to both sexual partners and infant.)
4. *Hepatitis B and C testing results.*
5. *HIV therapy during pregnancy.* (Rate of transmission decreased in women treated with zidovudine during the last two trimesters of pregnancy.)
6. *CD4 count and plasma HIV RNA levels.* (Rate of HIV transmission is increased with lower maternal CD4 counts and higher HIV RNA levels.)
7. *HIV status of other children.* (Rate of HIV transmission is increased if previous children were HIV infected.)
8. *Length of gestation.* (Rate of premature delivery is increased in HIV-infected infants.)

B. Perinatal History

1. *Length of labor and interval between rupture of membranes and delivery.*
2. *Type of delivery.*
3. *Complications of delivery.*
4. *Apgar Scores.*
5. *Nursery course.*

III. Physical Examination

The newborn physical examination should be thorough with special attention to

A. Birth weight. (HIV-infected infants tend to have lower birth weights.)

B. Evidence of congenital cytomegalovirus (CMV), herpes, syphilis, or toxoplasmosis. (These may be associated with HIV infection.)

C. Head circumference. (HIV-infected infants may have smaller head circumferences.)

IV. Laboratory Evaluation

HIV infection can now be diagnosed definitively in most infants by 1 month of age and in virtually all by 6 months of age. A positive test (culture, DNA, or RNA PCR) should be confirmed by a repeat test as soon as possible to initiate appropriate therapy. Once HIV infection is confirmed, the infant's viral burden should be determined by quantitative HIV RNA assay, which will suggest prognosis for disease progression. The preferred

diagnostic test is HIV DNA PCR or HIV RNA PCR performed within 48 hours of birth, at 1 month, and at 4 to 6 months. HIV culture is also reliable, but is much more difficult to perform and is not available everywhere. PCR testing can be sent out to diagnostic laboratories more easily, and results are available much sooner.

Other laboratory tests that may suggest HIV infection and that can be performed at the same time as the specific HIV tests are CD4+ T-lymphocyte counts and percentages (HIV infection results in a decreased absolute CD4+ count and decreased percentage of T4 lymphocytes), and complete blood count (CBC) (anemia, neutropenia, and thrombocytopenia may be associated with HIV infection).

V. Medical Management

In the period before HIV status is established, the infant should be presumed HIV positive from a management perspective and should undergo regular evaluation combining routine well-child procedures with expectant laboratory testing.

A. Routine well-child care examinations. Examinations should be performed at regular intervals with special attention to possible early signs of HIV infection, such as failure to thrive, chronic diarrhea, lymphadenopathy, and recurrent monilial disease of the mouth and diaper area.

B. Intercurrent illnesses. HIV-infected infants tend to have a higher incidence of common infections, such as otitis media, skin infections, and pneumonia, as well as opportunistic infections.

C. Immunizations. The immunization schedule for infants born to HIV-infected mothers is the same as the routine immunization schedule.

D. *Pneumocystic carinii* pneumonia (PCP) prophylaxis. Most experts in pediatric HIV infection recommend PCP prophylaxis beginning at age 4 to 6 weeks in the infant born to an HIV-positive mother until HIV status is known. Current recommendations for PCP prophylaxis are (a) first choice—oral trimethoprim (tmp)/sulfamethoxazole (smz) suspension (80 mg tmp and 400 mg smz/5 mL), dosed at 150 mg tmp/750 mg smz/m^2 PO divided BID on 3 consecutive days per week (e.g., Monday, Tuesday, and Wednesday) or (b) second choice—dapsone, 2 mg/kg PO QD in children unable to tolerate tmp/smz.

E. Cytomegalovirus. Urine culture for CMV at birth or first postnatal visit to allow monitoring and treatment for symptomatic CMV infection.

VI. Medical Management of HIV-Infected Infants and Children

Although the medical management of HIV-infected infants and children is beyond the scope of this handbook, several principles should be followed in providing their medical care. The primary care physician for HIV-infected infants and children may be a generalist or specialist, depending on individual ability,

availability, interest, and support (from colleagues, medical institution). Because of the many needs of families affected by HIV, both medical and psychosocial, the primary care physician must serve as both primary care provider and coordinator of care. The core HIV team should include the primary care physician, nurse, and social worker working closely with nutrition, psychology, and pharmacy. Other disciplines that are often needed for consultation include infectious disease, immunology, pulmonology, hematology/oncology, and dentistry. Essential ancillary services needed include pharmacy, laboratory, and radiology.

A. Routine well-child care

1. *Immunizations.* In addition to routine immunizations, HIV-infected infants require yearly influenza immunizations beginning at 6 months of age and tuberculin skin testing at least every 12 months, depending on the local prevalence of HIV disease and tuberculosis. Routine immunizations should include DTaP, Hib, IPV, HBV, MMR, and conjugated pneumococcal vaccine. In asymptomatic infants who are not immunosuppressed, varicella vaccine is also recommended at 12 to 15 months of age.
2. *Nutrition and growth.* Monitoring growth and providing nutritional information is critical to the successful management of the infant with HIV infection. Increasing the caloric density of routine infant formulas by using special high-density formulas (e.g., Pediasure, Ensure) and caloric additives (e.g., Polycose) may promote growth that has been slowed by the disease process. Each visit should include measurements of height, weight, head circumference, triceps skinfold thickness, and upper arm circumference.
3. *Development.* Careful monitoring of development by routine developmental examination and periodic neuropsychiatric testing are the most sensitive indicators of disease progression, which dictates therapeutic choices.

B. Intercurrent illnesses and opportunistic infections. HIV-infected infants and children have increased susceptibility to all of the common illnesses of childhood, especially recurrent otitis media, pneumonia, urinary tract infection, skin infections, and viral infections. Careful attention should be paid to prophylactic dental care to avoid complications of dental infections. Additionally, opportunistic infections, most commonly *Pneumocystis carinii* pneumonia, CMV disease, toxoplasmosis, *Mycobacterium avium* complex (MAC), and pulmonary tuberculosis, should be considered at each intercurrent illness and aggressively pursued.

C. PCP prophylaxis. PCP prophylaxis with oral trimethoprim/sulfamethoxazole suspension in the doses indicated earlier is recommended in HIV-infected children less than 6 years of age in the following circumstances:

1. History of previous episode(s) of *Pneumocystis carinii* pneumonia.
2. CD4+ percentage less than 15%.

3. CD4+ count lower than 500 cells/μL.
4. Age under 12 months.

D. Other prophylaxis. Prophylaxis against toxoplasmosis, MAC, and candidiasis is indicated in some situations.

E. Monitoring. HIV-infected patients require routine monitoring of certain laboratory parameters to allow decisions regarding primary therapy, prophylactic therapy, complications of the disease, disease progression, and prognosis. Currently, the combination of CD4+ counts/percentages and plasma HIV RNA levels show the greatest correlation with these parameters of illness. Other testing that should be performed periodically might include CBC with differential, liver function tests, renal function tests, neuropsychiatric testing, CT/MRI scans, chest radiography, and ophthalmologic examination.

F. Antiretroviral chemotherapy. The decision regarding when to begin antiretroviral chemotherapy is a complex one that does not have universal consensus among experts. Some or all of the following criteria are used to determine when to initiate therapy with currently available chemotherapeutic drugs:

1. Evidence of immune suppression based on CD4+ count/percentage.
2. High or increasing plasma HIV RNA levels.
3. Clinical symptoms related to HIV infection (CDC HIV Pediatric Classification System: Clinical Categories).
4. Blanket treatment. Some experts recommend treatment of all HIV-infected children regardless of age, symptoms, immune status, or virologic status.

G. Antiretroviral agents. Antiretroviral agents currently available fall into three categories that are commonly used in combination:

1. *Nucleoside analog reverse transcriptase inhibitors (NRTI)*
   a. Didanosine
   b. Lamivudine
   c. Stavudine
   d. Zalcitibine
   e. Zidovudine
   f. Abacavir
2. *Nonnucleoside analog reverse transcriptase inhibitors (NNRTI)*
   a. Nevirapine
   b. Delavirdine
   c. Sustiva
3. *Protease inhibitors (PI)*
   a. Saquinavir
   b. Indinivir
   c. Ritonavir
   d. Nelfinavir
   e. Amprenavir

VII. Psychosocial Care

Social service plays an integral role in the care of HIV-affected families. Counseling, interacting with community social agencies to obtain services for families, and monitoring the psychologic and emotional needs of the family are necessary for the success of the HIV team.

VIII. Terminal/Hospice Care

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Web Sites

National Pediatric and Family HIV Resource Enter Web Site: www.pedshivaids.org/

51 Physical Abuse of Children

Robert A. Shapiro and Amy L. Baxter

Child abuse is a problem that virtually all primary care physicians will encounter in their practice. Physicians are required by state laws to report suspected physical abuse or neglect to the legally mandated agency in their community. Although the exact legal definition of *child abuse* varies from state to state, an accepted medical meaning of physical abuse includes any injury to or death of a child less than 18 years of age resulting from the intentional commission of an act(s) by the child's caregiver. A commonly accepted medical meaning of *neglect* includes any injury to, suffering of, or death of a child under 18 years of age resulting from the omission of an act(s) by the child's caregiver.

I. Statistics

In 1998 there were more than 2.8 million referrals for alleged child maltreatment in the United States. Of the 66% investigated, 903,000 of those reports were verified as abuse by children's protective agencies—12.9 victims for every 1,000 children. Twenty-three percent of the reports were for physical abuse, and 54% were for neglect. In 1998, 1,100 deaths in the United States were due to child abuse; 77% were children 3 years of age or younger.

II. Risk Factors

Child abuse has been reported in households of all nationalities and ethnicities. The likelihood of physical abuse increases in families on welfare or with maternal drug use. Other factors that may increase a child's risk for physical abuse include pregnancy or birth complications. The risk of neglect increases in children with mental or physical disabilities and temperamental or needy behavior. More than 85% of perpetrators of child abuse are parents or other relatives of the victim. Although abuse is often diagnosed in families in whom specific risk factors are absent, known risk factors for caretakers abusing their children include the following:

- **A. Caregivers were abused or neglected as children.**
- **B. Caregivers are isolated.**
- **C. Caregivers lack resources to handle stress.**
- **D. Caregivers are young or impulsive.**
- **E. Caregivers lack knowledge of child development.**
- **F. Caregivers suffer from mental illness.**

III. Prevention

Awareness of risk factors in families can enable the physician to maintain increased vigilance for abuse. At early well-baby visits, inquiring about spousal abuse, neighborhood violence, and child care plans can help guide prevention discussions. The physician should discuss parents' attitudes about disciplining infants and should emphasize the dangers of shaking a baby. The provider should let parents know that it is common to get tired and frustrated with a new baby and that it is acceptable to leave the child crying in a safe crib until the parent feels calmer. Many cities have help lines that caregivers can call if they feel overwhelmed.

IV. History

When evaluating a child for child abuse, the history should document the how, where, when, and with whom for all suspicious injuries. Abuse should be suspected whenever (a) the injury is inconsistent with the degree or mechanism of trauma, (b) the injury is incompatible with the child's developmental abilities, or (c) the history is changed by the historian without an adequate explanation. Fictitious histories are likely to be incomplete and may contain conflicting details. Abuse should be seriously considered if there is an unreasonable delay in seeking medical care and if injuries are diagnosed in nonambulating children.

V. Specific Injuries

A. Bruises. Bruises are the most common type of injury from abuse. Certain features help in distinguishing between accidental and inflicted bruises. Accidental bruises most commonly occur over body areas that lack "padding," such as the forehead, elbows, knees, and shins. Bruises over padded parts of the body, such as the cheeks, abdomen, flanks, buttocks, and thighs, are injured less often by accidental activities and need to be examined more thoroughly for possible abuse. The physician should suspect abuse if the child has a large number of bruises over different areas of the body, especially if the bruises appear to be of different ages. Bruises on nonambulatory children are unusual and are often signs of abuse. Pattern injuries, such as loop marks, handprints, fingerpad bruises, and trauma from other recognizable objects, are strongly suggestive of abuse. Parallel linear petechial injuries may signify a high-velocity impact from a stick, fingers, cord, and so on. Choking must be considered when petechiae around the eyes are present along with bruising around the neck.

Color photographs of all injuries should be obtained if abuse is suspected. In most states, parental consent is not required. A measuring tape should be placed next to the injury before taking the picture so that size is recorded. Sketches should be made in the medical record documenting the location, size, shape, and color of the bruises. The age of the bruise cannot be determined by its color alone. Recent bruises may be tender and/or swollen, and the overlying skin may be abraded. Resolution of the bruise may occur within a few days or many weeks.

B. Fractures. In the nonambulatory infant, a fracture should alert the physician to the possibility of abuse. Rib, metaphyseal, and scapular fractures are cause for high suspicion of child abuse. Fractures resulting from allegedly minor trauma and multiple fractures of various ages are typical findings in the abused child. The presence and degree of callous about the fracture site will roughly date a fracture. Typically, callous appears at the fracture site within 10 to 14 days.

C. Burns. The appearance of the burn usually provides clues as to the cause. Water immersion burns cause a sharp-bordered "glove-and-stocking" distribution on an extremity. Scalds from "dipping" a child into hot water are clearly demar-

Table 51.1. Length of time to cause 3° burns

Water T° (F)	Time to cause 3° burn
120	10 minutes
122	5 minutes
127	1 minute
130	30 seconds
140	5 seconds
150	2 seconds

Adapted from Katcher M. Scald burns from hot tap water. *JAMA* 1981;246:1219–1222.

cated, sparing areas that the child keeps protected (e.g., spared skin folds between inner thighs with burns on buttocks). Children who are held firmly in a tub of hot water may have sparing to the buttocks if they are pressed down onto the bottom of the tub. Grease burns show a pattern of "dripping or running" down the skin or of splattering. Cigarettes burns are round and the diameter of the cigarette. Table 51.1 lists the time required to cause third-degree burn in adults exposed to water at different temperatures.

D. Abdominal trauma. Abdominal trauma results from direct blows to the abdomen. Signs of injury may be minimal. Specific injuries include traumatic pancreatitis and pseudocyst, duodenal hematoma, intestinal rupture, and mesenteric injury. Liver laceration or splenic rupture may have abrupt presentations due to blood loss, or they may be minimally symptomatic.

E. Bites. Human bites must be distinguished from animal bites. Animal bites are usually smaller, deeper, and narrower than human bites and often cause a ripping type of injury. Differentiating a child's bite from the bite of an adult can be difficult. Expert evaluation is advised. Bites should be photographed using a high-quality 35-mm or digital camera and should include a tape measure in the camera field so that accurate measurements can be made. If the bite is fresh and the skin has not yet been washed, saliva from the perpetrator may be present on the victim's skin. The saliva may be forensic evidence that should be collected. A slightly moistened piece of sterile gauze can be used to swab the skin over the bite. The gauze should then be placed into a paper (not plastic) envelope that is labeled with the patient's name, a description of the source of the specimen, the name of the collector, and the date/time of collection. The envelope must be sealed and locked in a secure location until given to police.

F. Head injury. Inflicted head injury, compared with other injuries from child abuse, causes the greatest morbidity and mortality. Inflicted head injury may be caused by blunt trauma

or severe shaking, which is commonly known as shaken baby syndrome (SBS). Presenting signs include altered mental status, irritability, vomiting, seizures, or respiratory arrest. External evidence of injury is frequently absent in SBS; retinal hemorrhages are found in approximately 80% of victims. Brain imaging by CT or MRI may demonstrate a subdural hematoma and/or subarachnoid bleed, cerebral contusions, diffuse axonal injury, or other intracranial injuries. Other injuries, such as rib fractures, metaphyseal fractures, and abdominal injury, can also be seen in SBS victims.

VI. Evaluation

A platelet count, prothrombin time (PT), and partial thromboplastin time (PTT) to rule out a bleeding diathesis should be obtained in children with suspicious, nonpatterned bruising. A skeletal survey to rule out occult fractures should be performed in children under the age of 2 years who are suspected child abuse victims. The skeletal survey should include an anteroposterior (AP) and lateral of the skull and thorax, lateral cervical and lumbar spine, AP pelvis, oblique hands, and AP views of the arms, legs, and feet. A bone scan can augment information obtained by the skeletal survey and is particularly useful for identifying acute rib fractures. A follow-up skeletal survey obtained 14 days after the initial survey may clarify questionable findings and reveal additional fractures. In addition to an ophthalmologic consultation, a head CT or MRI scan should be obtained in infants with suspected SBS. Head ultrasound has little value in screening for SBS because of the low sensitivity of this procedure. Hepatic transaminase and amylase levels, urinalysis for occult blood, and appropriate abdominal imaging should be performed if there is any concern about occult abdominal trauma. Although abdominal trauma in children will usually cause tenderness or abdominal bruising, liver lacerations may be unsuspected and asymptomatic in up to 6% of physical abuse cases. The physician should always consider other diagnoses and perform an appropriate work-up to exclude other conditions that can mimic child abuse.

VII. Reporting

Physicians are required to report all cases of suspected child abuse to the local child protective services (CPS) or to the police. The physician should not accuse the family of abusing the child but should explain his/her concerns about possible abuse and his/her legal requirement to report. The following statement is one example of how to begin talking about possible abuse with the child's parent/guardian: *"Your child's injuries seem too severe to have been caused by the incident that you are describing. I am concerned that someone may be hurting your child. Do you have any of these same concerns?"*

Although it is usually best to explain your concerns to the family before making a report and ordering further studies, if you fear that the child's safety may be jeopardized by notifying the family, the CPS social worker should be informed first. The initial report should be phoned to the CPS social worker and should followed by a complete written report. The report should describe the

injuries observed, the history given, and the reason for the report. Nonmedical terminology should be used throughout the report so that social workers, police officers, and lawyers can understand the findings and the physician's concerns. Physicians are immune from civil or criminal lawsuits brought by the family for making reports of alleged child abuse if the report is made in good faith.

VIII. Follow-up

The physician must maintain objectivity throughout the investigation, answer the questions of social workers and police, and be willing to testify in legal proceedings.

IX. Disposition

Once abuse is suspected, the child must be protected from further harm. The CPS worker, not the physician, is responsible for the safety of the child. If the child's home is unsafe and the CPS social worker is unable to secure safe placement, the physician may choose to hospitalize the child. Some states allow the physician to place a temporary "medical hold" on the discharge of a child; other states require an order by a juvenile court judge.

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Web Sites

http://www.calib.com/nccanch (National Clearinghouse on Child Abuse and Neglect)

http://w3.ouhsc.edu/ccan/page8.html (Directory of child abuse links)

http://www.aap.org/policy/05126.html (American Academy of Pediatrics policy on Shaken Baby Syndrome)

http://www.ndacan.cornell.edu (National Data Archive on Child Abuse and Neglect)

52 Sexual Abuse of Children

Robert A. Shapiro and Amy L. Baxter

Sexual abuse is the exploitation or involvement of a child in sexual activities. Sexual activities include, but are not limited to, exposure, fondling, genital contact, and pornography. Statutory rape criteria vary somewhat from state to state and include age of the child (often less than 14 years), difference in age between participants (usually 3 to 4 years), and capacity of the child to consent in situations of same-age sexual activity.

I. Statistics

The prevalence of sexual abuse is difficult to determine because it often remains undiagnosed. In addition, studies investigating prevalence and incidence have varying definitions of what constitutes abuse. In 1998, more than 100,000 substantiated reports of sexual abuse in the United States occurred, which represented 12% of all child maltreatment victims. Sexual abuse has been diagnosed in children of all ages, races, and socioeconomic groups. The perpetrator is often a male family member, family friend, or neighbor who has access to the child or authority over the child.

II. Presentation

Children may present to their primary care providers because of behavioral or physical symptoms or recent disclosure of abuse by the child or a sibling of abuse. If a report has been made to the children's protective services (CPS), a CPS social worker may ask to have the child examined as part of an investigation. Although many sexually abused children have no symptoms, others may act out sexual behaviors, experience sleep disturbances, develop phobias, perform poorly in school, or regress in their behavior. Abused children may complain of genital-rectal symptoms, such as pain, itching, redness, discharge, or bleeding.

Sexual abuse should be considered in all children presenting with secondary enuresis, encopresis, or other genitourinary complaints. Lice in eyelashes (pediculosis palpebrarum) should likewise prompt an investigation of sexual abuse, as pubic lice are virtually always the etiologic organism. Pubic lice can be distinguished from body and head lice when examined microscopically under low-power magnification (See Chapter 6).

III. History

The purpose of the interview is to collect information that may validate the history of abuse, to determine what testing needs to be done, and to begin the healing process for the child. Children who have been previously interviewed about the alleged abuse should not be requestioned about the same issues by the physician.

The history should be obtained in a calm, relaxed, and accepting manner. Before referring to body parts, the physician should ask the child or caregiver what words she or he uses when referring to the genitalia and rectum. Anatomic dolls or drawings should not be used without specific training, as they may provide

a source of leading information and render the interview invalid. The physician should encourage the child to tell the story himself using open-ended, nonleading questions (e.g., *"Tell me what happened,"* not *"Who hurt you?"*). During the interview telling the child that disclosing the abuse was the right thing to do and that you will try to prevent the abuse from happening again can be helpful. If the alleged perpetrator is a family member or a close friend, the child's parents may have difficulty believing that the abuse occurred. When parental support is absent, children often recant because of family pressures.

The history should be thoroughly documented in the child's chart. All historic statements should be attributed to the person who made them (e.g., *"per the mother, the child was. . ."*), and statements made by the child should be delineated with quotation marks.

IV. Forensic Evidence

A. In most states, if the last sexual assault occurred less than 72 hours ago and the nature of the assault suggests that seminal fluid, pubic hair, saliva, or blood belonging to the perpetrator might be recovered from the patient's body or clothing, then forensic specimen collection is required. The physician should refer the patient immediately to an emergency facility or similar site capable of forensic evidence collection. Instruct the patient not to bathe or to change clothes before the examination. Advise the family to bring a change of clothes, as the garments the child is wearing may be kept as evidence. If the child has changed clothes, advise caregivers to bring those garments in a paper bag.

B. If there is no possibility of forensic material being present and no acute injury is suspected, the physical examination can be scheduled for a convenient time and place. Children with acute injuries or symptoms should be examined urgently to evaluate and to document injury and need for treatment.

V. Examination

The purpose of the examination is to look for supporting evidence of sexual abuse, to screen for sexually transmitted diseases (STDs), and to reassure the child that his or her body is, or will be, fine. Physical findings of acute trauma should be documented. A colposcope is the best tool to capture photodocumentation of genital and rectal injury. When no colposcope is available, a 35-mm or digital camera may be used. Examinations are best done by professionals trained in sexual abuse evaluations. When this is impossible, positive findings should be confirmed and followed up by an experienced examiner.

A. Preparation. Before the examination, comfort the child by discussing examination positions. Some examiners demonstrate positions using a doll. When time allows, have the child practice correct positioning while still clothed. Permit the child to have control about who stays with him or her

during the examination. Assure the child that the examination will not be painful.

B. Technique: examination positions

1. *Frog leg.* Position the child in a supine position with his or her knees out and soles together, knees resting on the examination table. The child can lie on an examination table or sit on his or her parent's lap, whichever seems most comfortable.

2. *Knee/chest.* Have the child get on all fours, with knees spread wider than shoulders. Then have the child touch his or her chest to the examination table, maintaining the knee placement with a swayed backbone. Use a drape while adjusting the child's position. The knee/chest position is particularly useful to visualize vaginal foreign bodies and the posterior hymenal rim.

C. Technique: examination. Examine the external genitalia for signs of injury and infection. Examine the perineum for injuries, condylomata, herpetic lesions, bruises, tears, or discharge. A vaginal speculum is contraindicated in almost all prepubertal examinations. Visualize the hymen by holding onto the labia majora with the thumb and forefinger of each hand and retracting them outward (toward the examiner), laterally, and downward toward the rectum. When done properly, the introitus will open and the inner hymenal ring will be visible. Examine the hymen for signs of trauma.

The delicate nonestrogenized hymen is very sensitive and may fold onto itself, making the hymeneal rim difficult to visualize. "Floating" the rim (drip 1 to 2 mL of warmed saline onto the hymen, causing the hymenal ring to float into view) is a painless aid to visualization, particularly in the knee/chest position.

D. Findings: genitalia

1. *Prepubertal female.* When documenting, refer to locations of findings using a "clock face" orientation, with the urethra at 12 o'clock and midline posterior fourchette at 6 o'clock (Fig. 52.1). Girls less than 2 years of age, as well as older girls after puberty, may have redundant, estrogenized hymenal tissue with bumps or tags. A nonestrogenized hymen is often crescent shaped with little or no tissue between 10 and 2 o'clock, possibly with bumps or tags. Other normal findings include labial adhesions, ridges inside the vagina, and bands between the labia minora and urethra. Clefts or defects of the hymen may indicate injury, particularly when found in the posterior rim between 5 and 7 o'clock. Absent hymen between 3 and 9 o'clock probably indicates changes from previous injury. An enlarged hymenal opening without signs of trauma should not be considered indicative of vaginal penetration.

Approximately 5% to 15% of abused prepubertal girls will have clear evidence of sexual abuse on genital examination; 15% will have findings that are suspicious or sug-

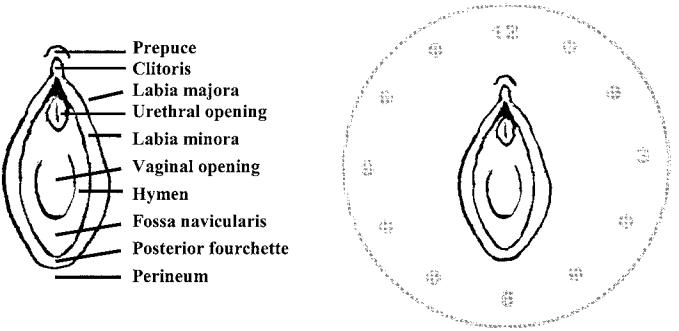

Fig. 52.1. Prepubertal female anatomy and orientation. (A) Anatomy. (B) Orientation.

gestive of abuse, but not diagnostic; 50% will have nonspecific findings; and 30% will have normal genital examinations. The likelihood of significant physical findings increases if the child provides a history of bleeding and when the examination is conducted soon after the alleged abuse.

2. *Pubertal female.* Inspect the external genitalia for acute injury, condylomata, herpes, and lice. Perform, if possible, an internal speculum and bimanual examination, and obtain routine cultures for STDs. Findings suggestive of hymenal injury can be easily confused with the normal adolescent hymenal anatomy of redundant folds and notching.

3. *Male.* Inspect the genitalia for infection or injury.

B. Findings: rectum. Examine the rectum for trauma, including scars, bruising, or tears. Normal or nonspecific findings include anal tags, thick or smooth skin in the midline, anal gaping with stool in the rectal vault, flattened or thickened anal folds, fissures, and delayed venous congestion of the perianal tissues. Immediate anal dilatation ≥15 mm without stool in the rectal vault is suspicious. A deep perianal laceration extending beyond the external anal sphincter is an indication of penetrating trauma.

VI. STD Testing

The algorithm below (Fig. 52.2) will assist in deciding when to culture for STDs. In prepubertal males, cultures are indicated if symptoms are present or if the perpetrator is documented to have an STD. In both males and females, condylomata, genital lesions consistent with the herpes simplex virus, or pediculosis palpebrarum should prompt the health care provider to consider cultures for gonorrhea and *Chlamydia* and blood testing for syphilis and HIV. When obtaining *N. gonorrhea* (GC) and

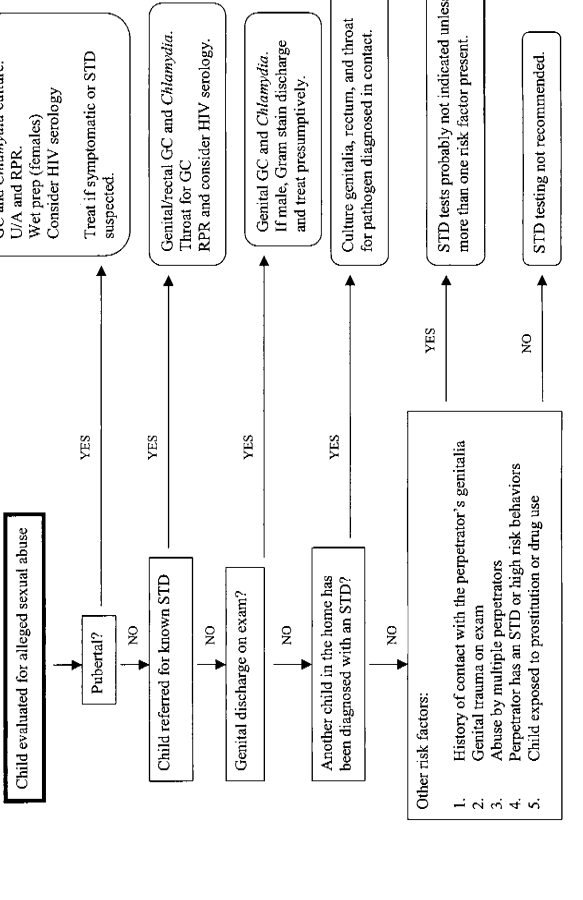

Fig. 52.2. Algorithm for testing for sexually transmitted disease.

Chlamydia cultures in prepubertal girls, sample the mucosa just proximal or distal to the hymen with a moistened type I Calgiswab. In pubertal females, sample the cervix. Presumptive positive gonococcal cultures on Thayer-Martin media must be confirmed by at least two different confirmatory tests (biochemical, enzyme substrate, or serologic). Only GC and *Chlamydia* cultures should be accepted as evidence of infection in prepubertal children. Rapid tests, including DNA amplification, are currently inadequate for legal documentation.

VII. Treatment

Prepubertal children do not require prophylactic antimicrobial treatment for GC or *Chlamydia* unless infection is thought to be likely. Gonococcal vaginitis is usually symptomatic in prepubertal girls. Adolescents should be offered STD prophylaxis, and, if the assault was within 72 hours, pregnancy prophylaxis.

VIII. STD Prophylaxis

Trichomoniasis, bacterial vaginosis, *Chlamydia*, and gonorrhea are the most frequently diagnosed infections among adolescents who have been sexually assaulted. Hepatitis B infection can be prevented by postexposure administration of hepatitis B vaccine and should be considered for adolescents who have not received the vaccine. Prophylaxis guidelines are published periodically by the Centers for Disease Control (CDC) in the *Morbidity and Mortality Weekly Report.* Current updates appear on its website. The CDC recommends a follow-up examination for adolescents 2 weeks after the assault.

Treatment consists of

Cefixime 400 mg PO × 1 *or* ceftriaxone 125 mg IM × 1
or
Spectinomycin 40 mg/kg IM × 1 (max. 2 g) for PCN-allergic patients
and
Azithromycin 1 g PO × 1, *or* doxycycline 100 mg PO BID × 7 days (if ≥ 9 y/o)
and
Metronidazole 2 g PO × 1

IX. Pregnancy Prophylaxis

Prophylaxis against pregnancy should be offered in all cases of sexual assault that may result in pregnancy. Prophylaxis consists of two Ovral tablets within 72 hours of the assault, followed by 2 additional tablets 12 hours after the first dosage. Consider Benadryl 25 to 50 mg one-half hour before administering Ovral to prevent nausea.

X. Reporting

All states require physicians to report suspected sexual abuse.

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Web Sites

http://www.calib.com/NCCANCH/ (National clearinghouse on child abuse and neglect)

http://wonder.cdc.gov/wonder/prevguid/p0000480/p0000480.htm (Specific updated recommendations for evaluation and treatment of sexual assault victims)

53 Symptomatic Therapy of Children

Raymond C. Baker

I. Introduction

Symptomatic therapy refers to medications or treatments suggested or prescribed by a physician to alleviate patients' symptoms, unrelated to the disease process. This term is used in contrast to medications or treatments prescribed by physicians that effectively reverse a disease process, such as antibiotics for bacterial infection or steroids for asthma. Since so many illnesses of childhood are self-limiting and without specific treatment, symptomatic therapy is often the only therapy a physician can offer a parent. However, symptomatic therapy, like any other intervention, has both therapeutic advantages and toxic potential. The decision to use medications to treat symptoms should therefore be mitigated by several factors.

A. Advantages of symptomatic therapy

1. *Subjective improvement with improved function.*
2. *Placebo value to child (and parent).*
3. *Something for the parent to do to lessen the feeling of helplessness.*
4. *Positive course of action.*
5. *A compromise with a parent's demand for medication.* Deters the parent from seeking less optimal medical care elsewhere.

B. Disadvantages of symptomatic therapy

1. *Cost.* Over-the-counter (OTC) symptomatic medications are usually less expensive than prescription medications and should be recommended whenever possible. The physician should be aware of the parents' financial status and method of payment for prescriptions when making decisions, such as whether to treat and whether to use OTC or prescription medications. Most medications for symptomatic therapy are available OTC.
2. *Ineffectiveness.* Symptomatic medications are often relatively ineffective.
3. *Impact.* Symptomatic medications have little impact on the course of the illness.
4. *Child's resistance.* Some children resist taking medications of any kind.
5. *Masking of symptoms.* Symptomatic medications might mask symptoms of advancing disease.
6. *Vigilance.* False sense of security from the medication might make a parent (or physician) less vigilant.
7. *Side effects.* Undesirable side effects may occur.
8. *Parental mindset.* Symptomatic therapy may create a mindset in the parent that every symptom requires a medication.

II. Ideal Symptomatic Medication

The ideal symptomatic medication should be inexpensive (especially OTC), tasty, nontoxic, readily available (especially OTC), easy to administer (e.g., infrequent dose schedule, oral), and effective for the symptom being treated. Few symptomatic medications fulfill all these criteria, making the decision to treat a particular child one that requires knowledge of the medication, the illness, the patient, and the family. For the symptoms that follow, symptomatic medications available will be described in terms of effectiveness, availability, and potential side effects/contraindications. Whenever possible, home remedies will also be suggested that might be helpful (albeit usually for their placebo effect).

III. Key

A. Trade name. Trade name medications are indicated by capitalization of the first letter of the medication (e.g., Benadryl).

B. Generic name. Generic medications are indicated by all-lower-case letters (e.g., diphenhydramine).

C. Prescription drugs. Prescription medications will be indicated with italicized letters (e.g., *Viscous Xylocaine*).

IV. Cough

Although widely available in products formulated for children, cough suppressants have not been shown to be effective when subjected to double-blind, placebo-controlled studies using pediatric patients. They have been shown somewhat effective in adolescents and adults, suggesting that their use should be limited to older children and adolescents. Nonetheless, because use of the nonnarcotic cough suppressant dextromethorphan is so widespread in younger children, some guidelines for its use in preadolescents follow. The use of cough suppressants should be limited to the treatment of cough resulting from irritation of the respiratory tract due to viral inflammation and minor secretions **only if the cough is interfering with the child's normal activities, such as sleep.** Cough suppressants should not be used in bronchospasm or conditions in which large amounts of secretions are being produced (e.g., cystic fibrosis, bronchiectasis).

A. The primary nonnarcotic cough suppressant is dextromethorphan, which is widely available OTC, either alone (e.g., Robitussin DM) or in combination with decongestants and antihistamines and marketed as multisymptom cold medications (e.g., Triaminic DM, Dimetapp DM). Dextromethorphan is contraindicated in infants under 12 months of age because of possible respiratory depression.

B. Narcotic cough suppressants include *codeine* and *hydrocodone,* usually in combination with other cold medications, such as decongestants and antihistamines (e.g., *Naldecon CX, Hycomine Pediatric Syrup*), and are available only by prescription. These medications are not indicated in children under 6 years of age because of possible respiratory depression. Their use in children is usually not warranted because

they have greater toxicity and abuse potential than dextromethorphan and have only marginal superiority over dextromethorphan as a cough suppressant in adult studies.

C. Expectorants (e.g., guaifenesin, Robitussin) theoretically should be helpful in cough by thinning secretions, making cough more effective. However, these medications are not effective and should be considered only for their placebo value.

D. Home remedy. Hard candy (e.g., lemon drops, cough drops) in older children (over 4 years of age).

V. Congestion

Congestion refers to blockage of upper respiratory passages secondary to inflammation of viral or allergic origin. Treatment may be indicated if the symptoms interfere with sleep and feeding.

A. Topical decongestants are effective and relatively inexpensive. Short-acting phenylephrine (e.g., Neo-Synephrine) is available in both adult and pediatric strengths. Long-acting oxymetazoline (e.g., Afrin) is available in adult strength only. Because topical decongestants can cause rebound symptoms (rhinitis medicamentosa) if used too long, their use in children should be limited to the first 3 to 5 days of the illness. For young infants, they should be used even more cautiously—**nighttime only** for no more than **3 to 5 days** (using the **weakest strength—0.125%**). Topical decongestants are especially helpful during the first few days of a viral respiratory tract infection in infants who cannot sleep because of nasal obstruction.

B. Oral decongestants (e.g., pseudoephedrine, Sudafed) likewise are effective decongestants when used on a short-term basis, as is true with topical decongestants. They are generally safe for use in infants and children more than 6 months of age and are relatively inexpensive. They are often combined with antihistamines, cough suppressants, or both and are sold as multisymptom cold medications (e.g., Novahistine, Triaminic DM, Dimetapp DM).

C. Home remedy. A cool mist humidifier may be helpful in the congested infant to thin mucus and to hydrate mucous membranes. A cool mist humidifier is preferred over a steam vaporizer, which has the potential to scald the fingers of a curious child. In addition, the cool mist humidifiers usually hold more liquid than the steam vaporizer and can last overnight. Saline or plain tapwater nose drops may also be instilled in the infant's congested nares to loosen secretions for easier removal by a nasal aspirator.

VI. Sore Throat/Sore Mouth (Stomatitis)

A. Sore throat. Topical analgesic liquids (benzocaine, diphenhydramine, *lidocaine,* phenol) are effective but are difficult to apply to the posterior pharynx, where the pain of pharyngitis is usually greatest. Some are available as a spray

(Vick's Chloraseptic spray), which may reach the posterior pharynx in the cooperative older child but are not easily applied to the younger child. Topical benzocaine (e.g., Cepacol, Chloraseptic) is also available in lozenge form and is marketed for pharyngitis pain. However, the analgesic effect is mostly on the tongue and anterior structures of the mouth with little effect in the posterior pharynx. Oral analgesics, especially ibuprofen, are somewhat effective in relieving sore throat pain.

B. Sore mouth/stomatitis. Topical analgesics are effective for the pain of stomatitis but may be difficult to apply in the uncooperative younger child who is at the age most likely to get stomatitis. Topical benzocaine (Oragel), lidocaine (*Viscous Xylocaine*), diphenhydramine (Benadryl), or a 1:1:1 mix of diphenhydramine, Kaopectate, and 2% *Viscous Xylocaine* are all effective if they can be applied. Sometimes a cotton- or sponge-tipped applicator can be used to apply these liquids in younger children; older children can swish and spit. These medications should not be swallowed because of systemic effects. Oral analgesics, especially ibuprofen, are somewhat effective if topical therapy is not feasible.

C. Home remedies for sore mouth and throat. Cold applied topically is also effective, although of shorter duration. Foods, such as ice cream, popsicles, cold pudding, and cold liquids, may be better tolerated by young children than medications. These have the advantage of providing some fluids that may be needed in the child whose intake has decreased because of the illness. Older children and teenagers may find relief of sore throat pain by gargling with warm saline solution, taking frequent sips of cold liquids, or using hard candy drops, such as lemon drops, as a salivary secretagogue.

VII. Earache

A. Medications. The pain of otitis media can be severe enough to interfere significantly with sleep (both infant and parent), feeding, and play activities in the infant. Both topical and systemic effective pain relief are available for the pain of otitis media. Topical benzocaine (e.g., *Auralgan*) has been shown to be effective, but because of the viscous nature of the vehicle, parents have difficulty instilling the product into the ear canal sufficiently to contact the tympanic membrane, which is necessary for the medication to be effective. A single application by the physician or nurse at the time of the diagnosis, followed by a recommendation for oral analgesics to be administered at home, may provide acute relief. Satisfactory oral analgesia can usually be obtained with acetaminophen for mild pain and with ibuprofen for more severe pain. (See the following discussion.)

B. Home remedies. Some infants may find relief from warmed mineral oil or olive oil (sweet oil) applied topically as a home remedy, but this should not be recommended if a per-

foration might be present (ear drainage) or tympanostomy tubes are in place. This remedy may be particularly helpful for those middle-of-the-night calls from anxious parents who have no oral analgesics available because it may provide relief until the infant sees the physician the following morning. The wise physician (who also appreciates a good night's sleep) should recommend that parents keep an oral analgesic in the home and know when and how to use it.

VIII. Nausea and Vomiting (Infectious Origin)

A. Oral medications of the phenothiazine and antihistamine categories (e.g., *prochlorperazine, Compazine, promethazine, Phenergan,* diphenhydramine, Benadryl) have been used for many years for nausea and vomiting in children and adolescents. They are marginally effective for children with these symptoms (of infectious origin), although they are more effective for nausea and vomiting resulting from chemotherapy. Phenothiazines are contraindicated in children under 6 years of age because of their neurologic side effects (drowsiness, dystonic reactions) and toxic potential. More important, antiemetics are usually unnecessary since vomiting is well tolerated by young children and is usually self-limiting because of its infectious etiology. In older children (more than 6 years), these medications may be considered in some situations. However, the physician should be aware of young children in the home and caution the parents about appropriate security of these medications. New antiemetics have been developed that have fewer toxicities but are indicated specifically for nausea and vomiting related to chemotherapy (e.g., *Zofran*).

B. Home remedies. A more common practice in infants and young children, however, is to discontinue solid intake and replace with easily absorbed liquids in order to replace and maintain body fluids and to decrease gastrointestinal activity. For this purpose home remedies can be suggested, including clear liquids such as sodas, Kool-Aid, or fruit juices. Electrolyte contents of the replacement fluid are not of great importance for the first 24 to 48 hours, and most vomiting has ceased by this time. Instructing parents to give small amounts frequently is important so that the vomiting reflex will not be triggered.

IX. Diarrhea (Infectious Origin)

A. Oral medications for diarrhea are available, usually of the opiate and anticholinergic categories (e.g., loperamide, Immodium), and are effective in decreasing the numbers and volume of stool output. However, they are contraindicated for young children (<2 years) for several reasons. Most diarrhea is self-limiting and well tolerated; the course of diarrhea in certain infectious etiologies may be prolonged by these medications; and "third spacing" may occur. In older children and adolescents, loperamide may be used to maintain normal activities, such as school attendance. Bismuth subsalicylate

(e.g., Pepto-Bismol) is effective in large doses in enterotoxigenic diarrhea. In certain bacterial diarrheas, antibiotics are indicated. Because of significantly greater toxic potential with *Lomotil,* this drug should not be prescribed in the pediatric population.

B. Home remedies. In infants with diarrhea of infectious origin, instituting a clear liquid diet to replace and maintain body fluids and to decrease gastrointestinal activity may be appropriate. Again, the electrolyte content of the liquids is probably not important unless the diarrhea exceeds 2 to 3 days' duration. If diarrhea has been present longer than 2 to 3 days, glucose-electrolyte oral rehydration solutions (e.g., Pedialyte, Rehydralyte, Infalyte) in small, frequent amounts (daily dose calculated on basis of losses and body weight) for 12 to 24 hours is appropriate. These are usually available in plain and flavored varieties. Oral rehydration with glucose/electrolyte solution is followed by return to a regular diet, including cow's milk–based formulas.

X. Constipation

Constipation is a common complaint in infants and children and may require intervention although most constipation is self-limiting. Before considering intervention, differentiating constipation (hard consistency) from infrequent passage or apparent difficulty in the passage of stool is important. Considerable variability in frequency of bowel movements exists in infants and children. Furthermore, infants, before successful toilet training, lack the mechanical advantage offered by the sitting position to have bowel movements. This often results in a greater effort to pass a bowel movement, which may be interpreted by parents as constipation.

A. Medications. Many effective laxatives, mostly OTC, are available. **Stimulants** are usually reserved for older children rather than infants, as they are usually unnecessary and may cause cramping. Examples of stimulant laxatives are bisacodyl (Dulcolax), phenolphthalein (Ex-Lax, Feen-A-Mint), and senna (Senokot). **Osmotic** laxatives, which act by drawing water into the bowel, are less cramping and are effective at most ages beyond infancy (e.g., magnesium hydroxide). **Stool softeners** (e.g., mineral oil, docusate, Maltsupex, lactulose) and fiber medications (e.g., Metamucil) may be indicated in some children, depending on the etiology of their constipation.

B. Home remedies are commonly suggested for constipation, especially in infants, and are somewhat effective, although their placebo value probably exceeds their actual efficacy. The addition of Karo syrup or molasses, 2 to 3 teaspoons per 4 ounces, to infant formula is a time-honored suggestion that may help. Other dietary manipulations include prune juice (stimulant), which is helpful in infants, and increased dietary fiber, such as bran cereals, popcorn, raisins, prunes, figs, spinach, carrots, and beets in older infants and children.

XI. Pain

There is a tendency among physicians to underestimate pain in infants and young children. Crying may be misinterpreted as fear and anxiety, and physicians may fear masking symptoms of advancing disease with effective pain management. In fact, pain management is clearly indicated in infants and children for the same reasons as in adults—to feel better and to allay anxieties that are commonly expressed as crying and irritability in young children. The quantitation of pain in children requires innovative techniques, such as using a scale of 1 to 10 or a spectrum of cartoon faces, to determine the degree of pain management necessary. The parent is usually helpful in determining the degree of pain in children by interpreting visual cues, for example, that may elude the physician.

A. Medications. Many effective pain medications are available for children and should be selected based on the degree of pain that is present. For mild pain, acetaminophen, 15 mg/kg per dose Q4 to 6H is effective in infants and children. For moderate pain, ibuprofen, 10 mg/kg per dose Q6H (or other nonsteroidal antiinflammatory agents) or *codeine,* 0.5 to 1.0 mg/kg per dose Q4–6H can be used. For severe pain, narcotic analgesics, orally or parenterally, including *morphine, meperidine* (*Demerol*), *hydromorphone* (*Dilaudid*), and *Fentanyl* (available as Fentanyl suckers), are used with appropriate precautions and attention to underlying conditions that might contraindicate their use.

B. Home remedies for mild pain may include cold or heat applied topically (e.g., musculoskeletal pain, cellulitis).

XII. Fever (Infectious Origin)

The treatment of fever as a symptom is controversial. Evidence exists that fever of infectious origin may be beneficial by providing an environment unfavorable for the infecting organism. Fever may also produce some subjective benefits, such as encouraging decreased activity during the illness and serving as an indicator of the course of the illness. Furthermore, antipyretics are not without toxicities.

On the other hand, treatment of fever may significantly improve the child subjectively, encouraging better oral intake; it may prevent recurrent febrile seizures; and antipyresis decreases insensible water losses.

A. Medications. Fever of infectious origin virtually always responds to acetaminophen in a dose of 15 mg/kg per dose PO or PR Q4 to 6H or ibuprofen, 10 mg/kg per dose PO Q6 to 8H. Failure to respond usually is secondary to inadequate dosing, either because the dose was too small, the child did not retain the dose, or heat loss was prevented by inappropriate wrapping.

B. Sponging with tepid water is probably not any more effective than simply undressing the infant after an appropriate antipyretic has been given. If sponging is performed, it must not be started until **20 to 30 minutes after an**

appropriate oral or rectal antipyretic has been given to avoid shivering and an increase in the body core temperature. The exception is in the treatment of fever that is not of infectious origin, such as heat stroke, in which rapid lowering of body temperature is achieved solely with external cooling without antipyretics.

MEDICATION ADMINISTRATION IN INFANTS AND CHILDREN

I. Oral Medications

Oral medications commonly come in *liquid forms*—which include syrups (sucrose solutions), elixirs (alcoholic preparations sweetened with sucrose), and suspensions (superior form for medications with bad taste in solution)—and *solid forms*—which include tablets, chewable tablets, coated tablets (easier to swallow), and capsules. (Most capsules can be pulled apart, allowing contents to be mixed with other vehicles.) Liquids have the advantage of being able to be administered in almost unlimited dose sizes, but they also have the disadvantage of taste. Tablets and capsules (except for chewable tablets) have no taste, but most children cannot learn to swallow them until they reach early school age.

To dispense liquid oral medications accurately, the measuring device has to be standardized and graduated in milliliters. Household teaspoons and tablespoons vary too much in size to use for the accurate measurement of liquid medications. Three inexpensive dispensing devices greatly improve the accuracy of the dose and are commonly dispensed along with the medication by the pharmacist. These are the oral dosing syringe (e.g., Exacta-Med Dispenser), which is usually graduated in both fractions of teaspoons and milliliters; the dispensing spoon, a tubular container with a spoon bowl built into the open end (e.g., Ezy-Dose, Handy Hook Spoon), which is similarly graduated; and the graduated medicine dropper.

It is usually easier to administer medication to young infants than to older infants and children, since infants tend to accept bad-tasting medications better than older infants and children, who have learned the intricacies of parental manipulation. The graduated medicine dropper and the dispensing syringe are good choices for infants since the medication can be gently squirted directly into the mouth. If the infant objects to the taste and closes his mouth, the medication can be squirted into the buccal space, where it is usually swallowed, perhaps with the help of a pacifier quickly slipped into place after giving the medication. Sometimes the medication can be put into a nipple (with an enlarged hole) for easy administration to bottle-dependent infants.

A common mistake parents make is to mix the medication with milk or other liquid the infant enjoys and give it in a bottle. The problem with this method is that most infants will detect the different taste and not want to take the whole amount (which must be given if the whole dose is to be given). Usually giving the

liquid medication as is works better (the pharmaceutical companies have done their best to disguise the taste) because the quantity of liquid is as small as possible. This can be followed, if necessary, by a better-tasting "chaser" to take away the bad taste. If the infant spits the medication out, then all or part of the dose will need to be repeated (the quantity must be estimated based on the amount rejected). A small minority of infants will repeatedly vomit oral medications, making an alternative route (usually parenteral) necessary. Although the vomiting may result from a hysterical crying scene, more commonly it is due to the child's illness itself and resolves after 24 hours, making parenteral treatment necessary only for the first few doses.

In older infants and children who still require liquid medications but are fussy about taste, oral administration of medications can be a real challenge. Most parents are well aware of their child's track record on taking oral medications and can help the physician decide what is the best oral medication to use. If the parent says that the child is likely to resist taking bad-tasting medications, several things the physician suggests might make the process a little easier:

A. Select a medication that you know tastes good if you have a choice. With the many choices of oral antibiotics available now, usually one or more have a better taste. Unfortunately, the better-tasting antibiotics usually cost more.

B. Select a medication that requires infrequent dosing—once or twice a day. If the child does make a major production out of taking the medication, limit the number of major productions to once or twice a day!

C. Improve the taste, if you must use a bad-tasting medication (and some cannot be disguised even by the yummiest of flavorings), by using the tablet or capsule form of the medication, crushed if necessary, and mixed with a vehicle that is more acceptable to the child. Hershey' chocolate syrup, mushy ice cream, and grape juice are good choices.

D. Administer the medication very cold.

E. Give the medication through a narrow straw with the tip of the straw near the back of the tongue, bypassing most of the taste buds.

F. Follow the medicine with a strong-tasting chaser, such as root beer or juice.

G. Use positive reinforcement by offering a small reward after taking the medication (e.g., sticker, piece of candy, etc.). Remember, it is a bribe if you give the reward first; it is positive reinforcement if you give it after!

H. Spend the time to teach the child how to swallow tablets or capsules. Children as young as 5 or 6 years of age may be able to learn if the incentive is strong enough. Several tips on teaching children how to swallow tablets include the following:

1. *Start with small coated tablets that are easier to swallow.*
2. *Practice with small candies and work up to larger ones* (e.g., Jimmies, Smarties, TacTacs, M&M's, Good & Plenty's—in order of increasing size).
3. *Swallow with mushy ice cream, apple sauce, or pudding.*
4. *Swallow with soda.*

II. Rectal Medications

The nurse or physician should demonstrate the insertion of a rectal suppository if the parent has never given one before. Instruct the parent to (a) place a finger cot onto the index finger of their dominant hand; (b) apply Vaseline liberally to the suppository itself, the perianal area, and the index finger; (c) hold on to the suppository with the thumb and third finger and position the end of the suppository at the anal orifice, (d) push on the bottom of the suppository with the index finger; and (e) follow the suppository with the index finger tip up to the first joint of the finger to ensure it enters the rectal vault and remains there.

III. Topical Eye Medications

Applying topical eye medications is inherently frightening to a child and must be done gently and with a clear explanation. The usual concerns of the child are that it will *"hurt when it touches my eye."* If the parent has never used eyedrops or ointments, the nurse or physician should demonstrate for the parent. To administer an ophthalmic ointment, the medication should be warmed to body temperature before administration. (Warming also makes the ointment come out of the tube more easily.) Then ask the child to tilt the head back a little, gently pull down the lower eyelid with the thumb, and deposit a strip about 0.5 cm long on the lower palpebral conjunctiva. Then close the eyelids and massage gently to distribute the medication. Warn older children that the vision will be a little blurry for awhile afterward. Topical eyedrops can be applied in a similar manner to cooperative children.

For infants and younger children who are more likely to resist and squeeze the eyes shut, eyedrops may be easier to apply and can be applied by a single adult without help. The parent sits on the floor with legs slightly abducted and extended, forming a "V." The infant is positioned supine on the floor with the upper extremities trapped beneath the parent's legs and the child's head held by the parent's upper legs at the point of the "V." The drops, which have been warmed to body temperature, are then placed in the inner corner of the eye (which is usually tightly closed!). The parent then sets the eyedrop bottle down and with the thumb and index finger pulls the lids apart gently, allowing the drops to enter the eye. After the first time or two, the child usually realizes the process does not hurt and resists less.

IV. Topical Ear Medications

Children are usually not resistant to ear drops, but applying them so they come into contact with all ear surfaces, including the ear drum, can be tricky. The parent should be instructed to

pull up and back (children) or just back (infants) on the pinna of the ear to straighten out the ear canal. The drops are then allowed to run down the posterior wall of the ear canal so that they reach the bottom of the canal (the ear drum) without an air pocket getting between the medication and the ear drum. Depending on the medication, a cotton plug may be placed in the outer canal to retain the medication.

V. Topical Nasal Medications

For older children, nasal spray is easier to use; for infants and young children who may resist, drops are preferred. To apply nasal drops to the uncooperative infant, tell the parent to place the infant on his back on the parent's lap with the head slightly hanging off the end of the parent's knees. Alternatively, the infant can be laid on the edge of a bed with the head slightly hanging down. The parent may need some assistance holding the child's head still. The drops are placed in both nostrils; then the infant should be kept in the same position for about a minute to allow the medication to distribute. The infant can be encouraged to sniff the medications back further into the nostrils by having the parent hold a hand momentarily over the baby's mouth during inspiration, forcing him to breathe in through his nose. Nasal spray is applied with the child standing up with instruction to "sniff" the medication back immediately after spraying.

ADDITIONAL READING

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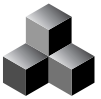

Appendices

Appendix A Guidelines for Antimicrobial Drug Dosages for Pediatric Patients

Table A.1. Guidelines for antimicrobial drug dosages for pediatric patients*

Medication†	Available		Dosing‡
Amoxicillin (Amoxil, Trimox)	Liq: Drops: Ctabs: Tabs:	125, 200, 250, 400 mg/5 mL 50 mg/mL 125, 200, 250, 400 mg 500, 800 mg	15–30 mg/kg BID (Susceptible strains, including sensitive *S. pneumoniae*) 40–45 mg/kg BID (maximum dose 1,000 mg) (*S. pneumoniae* with intermediate resistance)
Amoxicillin/clavulanate (Augmentin)	Liq: Ctabs: Tabs:	125, 200, 250, 400 mg/5 mL 125, 200, 250, 400 mg 500, 875 mg	20–22 mg/kg BID
Azithromycin (Zithromax)	Liq: Tabs:	100, 200 mg/5 mL 250, 600 mg	10 mg/kg QD day 1, followed by 5 mg/kg QD days 2–5 (maximum 500 mg/day)
Cefaclor (Ceclor)	Liq: Caps:	125, 187, 250, 375 mg/5 mL 250, 500 mg	15–20 mg/kg BID (maximum 1,000 mg/day)
Cefadroxil (Duricef)	Liq: Tabs: Caps:	125, 250, 500 mg/5 mL 1,000 mg 500 mg	15 mg/kg BID
Cefixime (Suprax)	Liq: Tabs:	100 mg/5 mL 200, 400 mg	8 mg/kg QD
Cefpodoxime (Vantin)	Liq: Tabs:	50, 100 mg/5 mL 100, 200 mg	5 mg/kg BID (maximum dose 200 mg)
Cefprozil (Cefzil)	Liq: Tabs:	125, 250 mg/5 mL 250, 500 mg	15 mg/kg BID

Ceftibuten (Cedax)	Liq: Caps:	90 mg/5 mL 400 mg	9 mg/kg QD (maximum 400 mg/day)
Cefuroxime (Ceftin)	Liq: Tabs:	125, 250 mg/5 mL 125, 250, 500 mg	15 mg/kg BID (maximum 1,000 mg/day)
Cephalexin (Keflex)	Liq: Tabs: Caps:	250 mg/5 mL 250, 500 mg 250, 500 mg	10–12 mg/kg QID
Clarithromycin (Biaxin)	Liq: Tabs:	125, 250 mg/5 mL 250, 500 mg	7.5 mg/kg BID (maximum 1,000 mg/day)
Clindamycin (Cleocin)	Liq: Caps:	75 mg/5 mL 75, 150, 300 mg	3–6 mg/kg TID
Doxycycline (Vibramycin)	Liq: Tabs: Caps:	25 mg/5 mL 100 mg 50, 100 mg	2–4 mg/kg QD
Erythromycin/sulfisoxazole (Pediazole)	Liq:	200/600 mg/5 mL	0.25 mL/kg QID
Erythromycin (EES, Ilosone, Eryped)	Liq:	200, 400 mg/5 mL (EES, Eryped) 125, 250 mg/5 mL (Ilosone)	10–12 mg/kg TID (maximum 100 mg/kg per day)
	Drops: Ctabs: Tabs:	100 mg/2.5 mL (Eryped) 200 mg (Eryped) 400 mg (EES) 500 mg (Ilosone)	
	Caps:	250 mg (Ilosone)	
Fluconazole (Diflucan)	Liq: Tabs:	50, 200 mg/5 mL 50, 100, 200 mg	3–6 mg/kg QD (maximum 600 mg/day)

(continued)

Table A.1. ***Continued.***

Medication†	Available		Dosing‡
Griseofulvin (Grifulvin V)	Liq: Tabs:	125 mg/5 mL 250, 500 mg	20–25 mg/kg QD (maximum 1000 mg/day) (with fatty food)
Ketoconazole (Nizoral)	Tabs:	200 mg	3.3–6.6 mg/kg QD
Loracarbef (Lorabid)	Liq: Caps:	100, 200 mg/5 mL 200, 400 mg	15 mg/kg BID
Minocycline (Minocin)	Liq: Caps:	50 mg/5 mL 50, 100 mg	4 mg/kg QD first dose followed by 2 mg/kg BID
Metronidazole (Flagyl)	Tabs: Caps:	250, 500 mg 375 mg	5 mg/kg TID (maximum 750 mg/day) (Giardiasis)
Nystatin (Mycostatin)	Liq: Tabs: Pastilles:	100,000 U/mL 500,000 U 200,000 U	2 mL QID
Penicillin V (V-Cillin K)	Liq: Tabs:	125, 250 mg/5 mL 250, 500 mg	10–20 mg/kg TID 250 mg BID (streptococcal pharyngitis)
Rifampin (Rifadin)	Caps:	150, 300 mg	10–20 mg/kg QD (maximum 600 mg/day)
Sulfisoxazole (Gantrisin)	Liq: Tabs:	500 mg/5 mL 500 mg	30–37 mg/kg QID (maximum 6,000 mg/day)

Terbinafine (Lamisil)	Tabs:	250 mg	Weight 15–20 kg: Weight 20–40 kg: Weight >40 kg: (Tinea capitis)	62.5 mg QD 125 mg QD 250 mg QD
Tetracycline (Sumycin, Achromycin V)	Liq: Tabs: Caps:	125 mg/5 mL (Sumycin) 250, 500 mg 250, 500 mg	5–12 mg BID	
Trimethoprim/ sulfamethoxazole (Bactrim, Septra)	Liq: Tabs:	40/200 mg/5 mL 400/80, 80/160 mg	0.5 mL BID	

Sources: Pickering LK, ed. *2000 red book: report of the Committee on Infectious Diseases*, 25th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2000.
Physicians' Desk Reference, 21st ed. Montvale, NJ: Medical Economics Company, Inc.; 2000.
Prescribing Reference for Pediatricians. New York: Prescribing Reference, Inc.; Fall-Winter 1999–2000.
Abbreviations: Liq, Suspension/Liquid/Solution; Caps, Capsules; Tabs, Tablets; Ctabs, Chewable tablets.
* Oral drugs only. Dosages are for pediatric patients out of the newborn period. This list is not comprehensive and does not represent preferences.
† Generic (brand name).
‡ Medication amounts are per dose unless otherwise indicated.

Appendix B Composition of Commercially Available Oral Rehydration Solutions and Selected Clear Liquids

Table B.1. Composition of commercially available oral rehydration solutions and selected clear liquids

Name	CHO	Kcal (per L)	Na (mEq/L)	K (mEq/L)	Base (mEq/L)	Osmolality (mOsm/L)
Pedialyte	Glucose, fructose	100	45	20	30	250
Rehydralyte	Glucose	100	75	20	30	300
Infalyte (formerly Ricelyte)	Rice syrup solids	120	50	25	30	200
WHO/UNICEF Formula	Glucose	80	90	20	30	310
Gatorade*	Fructose, glucose	1,000	20	3	3	330
Cola*	Sucrose/corn syrup	2,800	2	N	13	750
Kool-Aid* (sweetened)	Sucrose	480	N	N	N	334

Abbreviation: N, negligible (<2.0 mEq/L).
* Not recommended for rehydration.

Appendix C Blood Pressure Norms

Table C.1. Blood pressure levels for the 90th and 95th percentiles of blood pressure for girls 1–17 years of age by percentiles of height

		Systolic BP (mm Hg) by percentile of height							Diastolic BP (DBP5) (mm Hg) by percentile of height						
Age (yr)	% ile	5%	10%	25%	50%	75%	90%	95%	5%	10%	25%	50%	75%	90%	95%
1	90th	98	98	99	101	102	103	104	52	52	53	53	54	55	55
	95th	101	102	103	104	106	107	108	56	56	57	58	58	59	60
2	90th	99	99	101	102	103	104	105	57	57	58	58	59	60	60
	95th	103	103	104	106	107	108	109	61	61	62	62	63	64	64
3	90th	100	101	102	103	104	105	106	61	61	61	62	63	64	64
	95th	104	104	106	107	108	109	110	65	65	66	66	67	68	68
4	90th	101	102	103	104	106	107	108	64	64	65	65	66	67	67
	95th	105	106	107	108	109	111	111	68	68	69	69	70	71	71
5	90th	103	103	105	106	107	108	109	66	67	67	68	69	69	70
	95th	107	107	108	110	111	112	113	71	71	71	72	73	74	74
6	90th	104	105	106	107	109	110	111	69	69	69	70	71	72	72
	95th	108	109	110	111	113	114	114	73	73	74	74	75	76	76
7	90th	106	107	108	109	110	112	112	71	71	71	72	73	74	74
	95th	110	111	112	113	114	115	116	75	75	75	76	77	78	78
8	90th	108	109	110	111	112	114	114	72	72	73	74	74	75	76
	95th	112	113	114	115	116	117	118	76	77	77	78	79	79	80
9	90th	110	111	112	113	114	116	116	74	74	74	75	76	77	77
	95th	114	115	116	117	118	119	120	78	78	79	79	80	81	81

(continued)

Table C.1. ***Continued.***

Age (yr)	% ile	Systolic BP (mm Hg) by percentile of height							Diastolic BP (DBP5) (mm Hg) by percentile of height						
		5%	10%	25%	50%	75%	90%	95%	5%	10%	25%	50%	75%	90%	95%
10	90th	112	113	114	115	116	118	118	75	75	76	77	77	78	78
	95th	116	117	118	119	120	122	122	79	79	80	81	81	82	83
11	90th	114	115	116	117	119	120	120	76	77	77	78	79	79	80
	95th	118	119	120	121	122	124	124	81	81	81	82	83	83	84
12	90th	116	117	118	119	121	122	123	78	78	78	79	80	81	81
	95th	120	121	122	123	125	126	126	82	82	82	83	84	85	85
13	90th	118	119	120	121	123	124	124	79	79	79	80	81	82	82
	95th	122	123	124	125	126	128	128	83	83	84	84	85	86	86
14	90th	120	121	122	123	124	125	126	80	80	80	81	82	83	83
	95th	124	125	126	127	128	129	130	84	84	85	85	86	87	87
15	90th	121	122	123	124	126	127	128	80	81	81	82	83	83	84
	95th	125	126	127	128	130	131	131	85	85	85	86	87	88	88
16	90th	122	123	124	125	127	128	129	81	81	82	82	83	84	84
	95th	126	127	128	129	130	132	132	85	85	86	87	87	88	88
17	90th	123	123	124	126	127	128	129	81	81	82	83	83	84	85
	95th	127	127	128	130	131	132	133	85	86	86	87	88	88	89

Source: Rosner B, Prineas RJ, Loggie JMH, et al. Blood pressure nomograms for children and adolescents, by height, sex, and age, in the United States. *J Pediatr* 1993;123:871. Reproduced with permission.

Table C.2. Blood pressure levels for the 90th and 95th percentiles of blood pressure for boys 1–17 years of age by percentiles of height

		Systolic BP (mm Hg) by percentile of height							Diastolic BP (DBP5) (mm Hg) by percentile of height						
Age (yr)	% ile	5%	10%	25%	50%	75%	90%	95%	5%	10%	25%	50%	75%	90%	95%
1	90th	94	95	97	99	101	102	103	49	49	50	51	52	53	54
	95th	98	99	101	103	105	106	107	54	54	55	56	57	58	58
2	90th	98	99	101	103	104	106	107	54	54	55	56	57	58	58
	95th	102	103	105	107	108	110	110	58	59	60	61	62	63	63
3	90th	101	102	103	105	107	109	109	59	59	60	61	62	63	63
	95th	105	106	107	109	111	112	113	63	63	64	65	66	67	68
4	90th	103	104	105	107	109	110	111	63	63	64	65	66	67	67
	95th	107	108	109	111	113	114	115	67	68	68	69	70	71	72
5	90th	104	105	107	109	111	112	113	66	67	68	69	69	70	71
	95th	108	109	111	113	114	116	117	71	71	72	73	74	75	76
6	90th	105	106	108	110	112	113	114	70	70	71	72	73	74	74
	95th	109	110	112	114	116	117	118	74	75	75	76	77	78	79
7	90th	106	107	109	111	113	114	115	72	73	73	74	75	76	77
	95th	110	111	113	115	117	118	119	77	77	78	79	80	81	81
8	90th	108	109	110	112	114	116	116	74	75	75	76	77	78	79
	95th	112	113	114	116	118	119	120	79	79	80	81	82	83	83
9	90th	109	110	112	114	116	117	118	76	76	77	78	79	80	80
	95th	113	114	116	118	119	121	122	80	81	81	82	83	84	85

(continued)

Table C.2. ***Continued.***

Age (yr)	% ile	Systolic BP (mm Hg) by percentile of height							Diastolic BP (DBP5) (mm Hg) by percentile of height						
		5%	10%	25%	50%	75%	90%	95%	5%	10%	25%	50%	75%	90%	95%
10	90th	111	112	113	115	117	119	119	77	77	78	79	80	81	81
	95th	115	116	117	119	121	123	123	81	82	83	83	84	85	86
11	90th	113	114	115	117	119	121	121	77	78	79	80	81	81	82
	95th	117	118	119	121	123	125	125	82	82	83	84	85	86	87
12	90th	115	116	118	120	121	123	124	78	78	79	80	81	82	83
	95th	119	120	122	124	125	127	128	83	83	84	85	86	87	87
13	90th	118	119	120	122	124	125	126	78	79	80	81	81	82	83
	95th	121	122	124	126	128	129	130	83	83	84	85	86	87	88
14	90th	120	121	123	125	127	128	129	79	79	80	81	82	83	83
	95th	124	125	127	129	131	132	133	83	84	85	86	87	87	88
15	90th	123	124	126	128	130	131	132	80	80	81	82	83	84	84
	95th	127	128	130	132	133	135	136	84	85	86	86	87	88	89
16	90th	126	127	129	131	132	134	134	81	82	82	83	84	85	86
	95th	130	131	133	134	136	138	138	86	86	87	88	89	90	90
17	90th	128	129	131	133	135	136	137	83	84	85	86	87	87	88
	95th	132	133	135	137	139	140	141	88	88	89	90	91	92	93

Source: Rosner B, Prineas RJ, Loggie JMH, et al. Blood pressure nomograms for children and adolescents, by height, sex, and age, in the United States. *J Pediatr* 1993;123:871. Reproduced with permission.

Appendix D Surface Area Nomogram

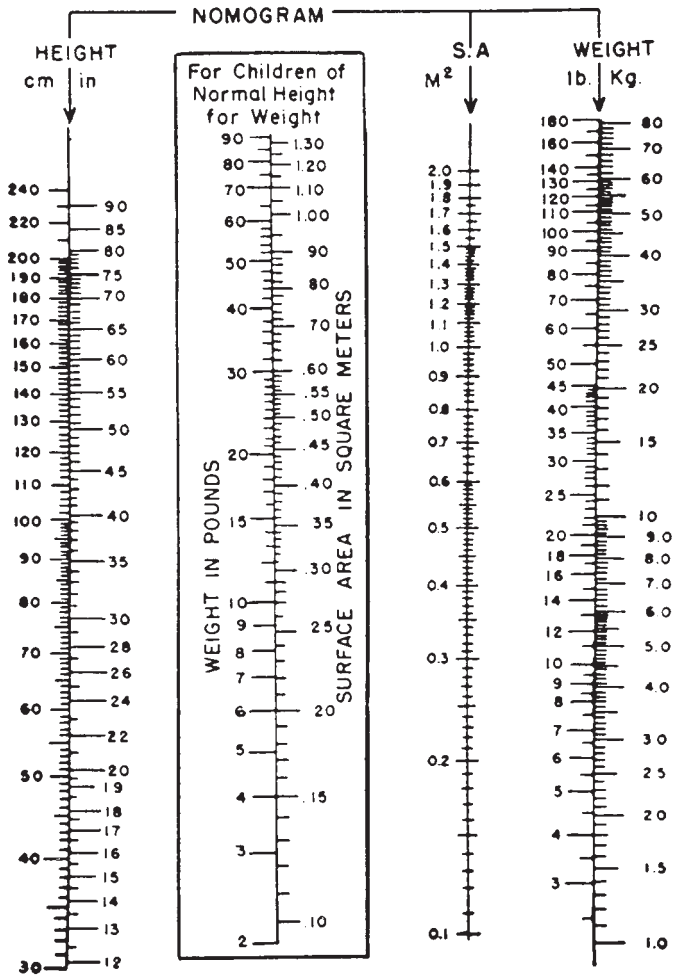

Figure D.1. Nomogram for estimation of body surface area.
Source: Behrman RE, Kliegman RM, Nelson WE, et al. *Nelson textbook of pediatrics*, 14th ed. Philadelphia: WB Saunders; 1992. Reproduced by permission.

Appendix E Body Mass Indices

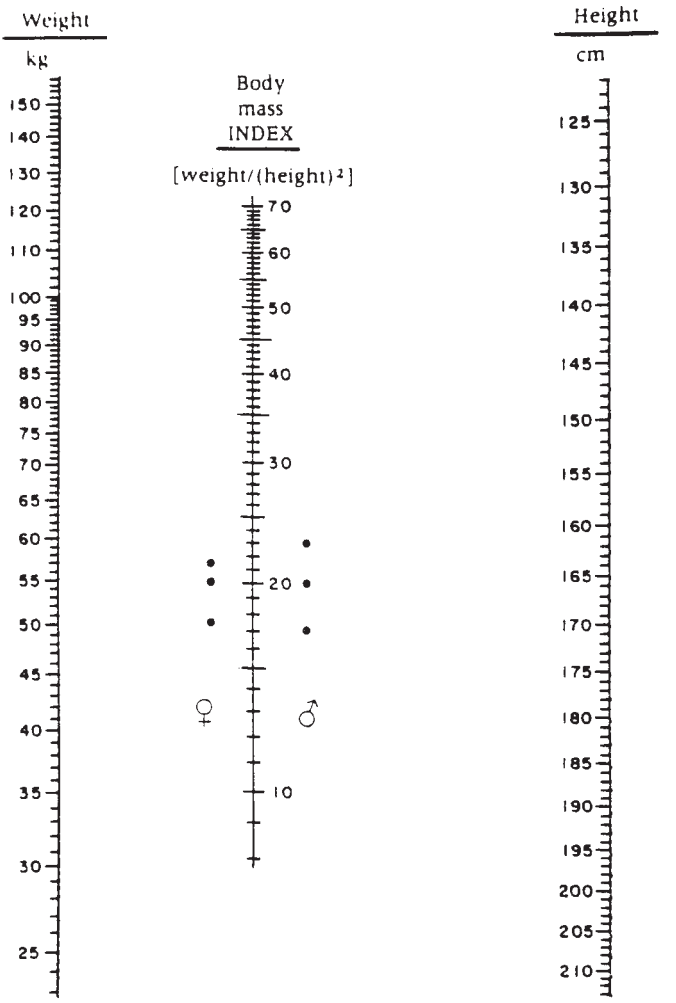

Figure E.1. Body mass index nomogram.
Source: McAnarney ER, Kreipe RE, Orr DP, Comerci GD. *Textbook of adolescent medicine.* Philadelphia: WB Saunders; 1992. Reproduced with permission.

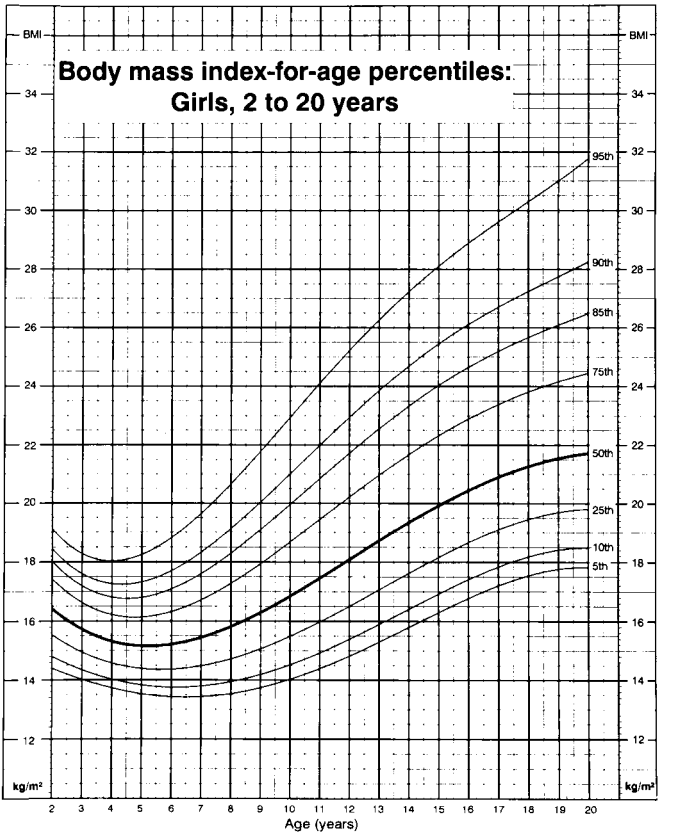

Figure E.2. Girls, body mass index-for-age percentiles.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

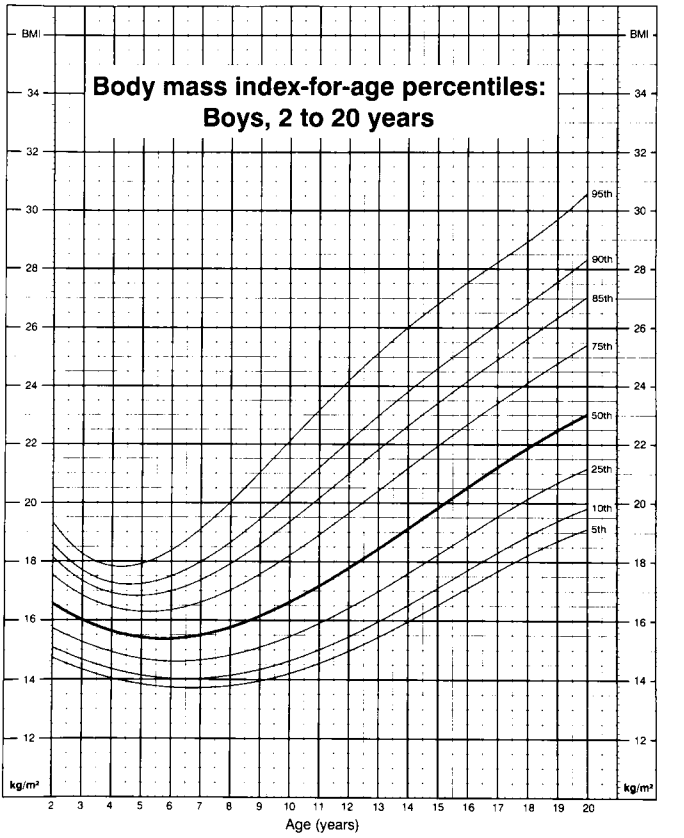

Figure E.3. Boys, body mass index-for-age percentiles.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts.* Atlanta, GA: Centers for Disease Control and Prevention, 2000.

APPENDIX F Clinical Stages of Pubertal Development

Table F.1. Clinical stages of pubertal development: Tanner stages

Tanner Stage	Girls (Breast)	Boys (Genitalia)	Both Sexes (Pubic Hair)
I	None	Prepubertal penis (< 7 cm) Testes < 2.5 cm long	None
II	Budding less than diameter of areola	Prepubertal penis (< 7 cm) Scrotal thinning Testes > 2.5 cm long (4–6 mL in volume)	Few dark, thick hairs over mons and labia or base of penis and scrotum
III	Breast greater than diameter of areola	Enlarging penis > 7 cm Testes 3–4 cm long (6–10 mL in volume)	Visible dark hairs over mons and labia or base of penis and scrotum
IV	Puffy areola	Larger penis with developed glans Testes 4–5 cm long (10–15 mL in volume)	Thick hair distribution over wider area
V	Adult	Adult	Adult

Adapted from Marshall WA, Tanner JM. Variations in the patterns of pubertal changes in boys. *Arch Dis Child* 1970;45:13 and Sizonenko PC. Normal sexual maturation. *Pediatrician* 1987;14:191.

Appendix G Sequence of Sexual Maturity

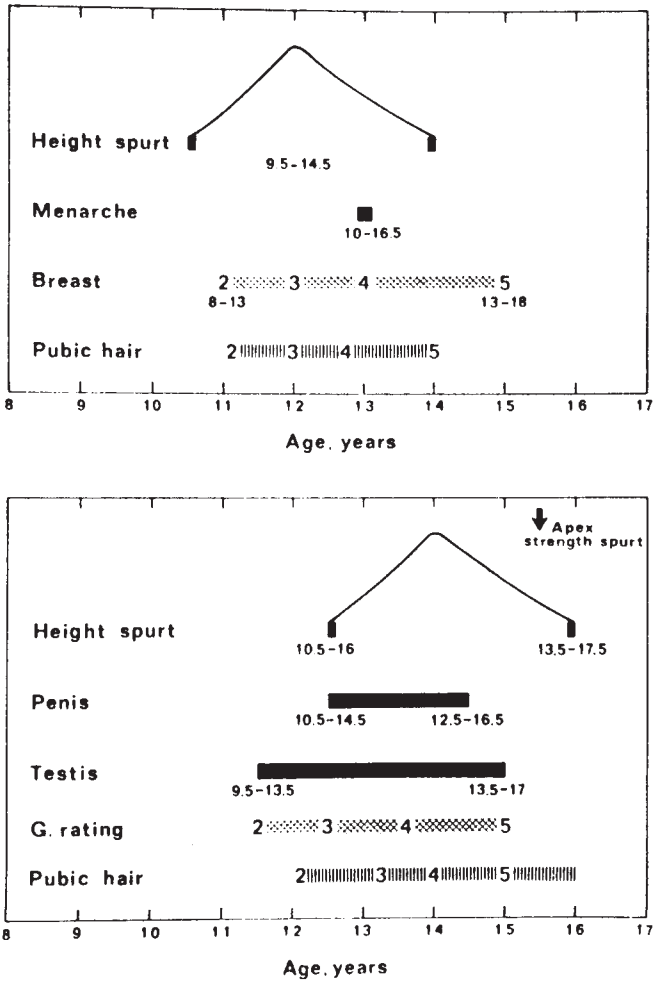

Figure G.1. Sequence of sexual maturity.
Source: Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. *Arch Dis Child* 1970;45:22. Reproduced with permission.

Appendix H Growth Charts

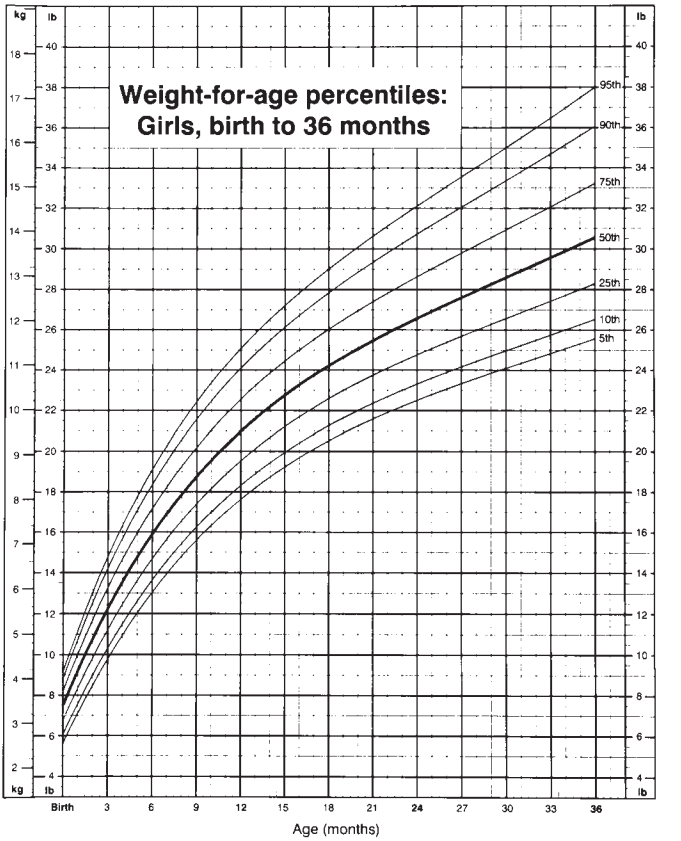

Figure H.1. Weight for age, girls, birth to 36 months.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

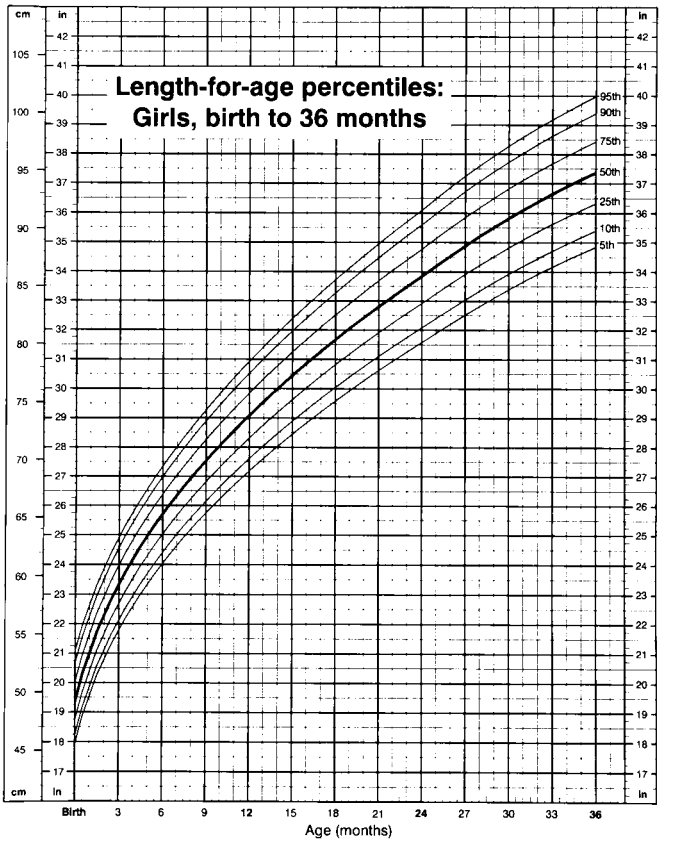

Figure H.2. Length for age, girls, birth to 36 months.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

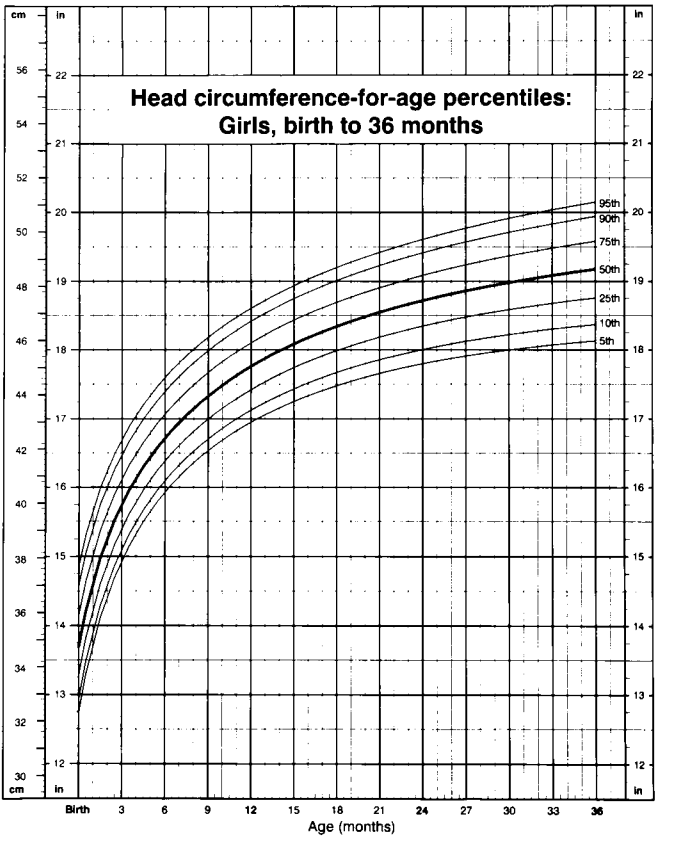

Figure H.3. Head circumference for age, girls, birth to 36 months. Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

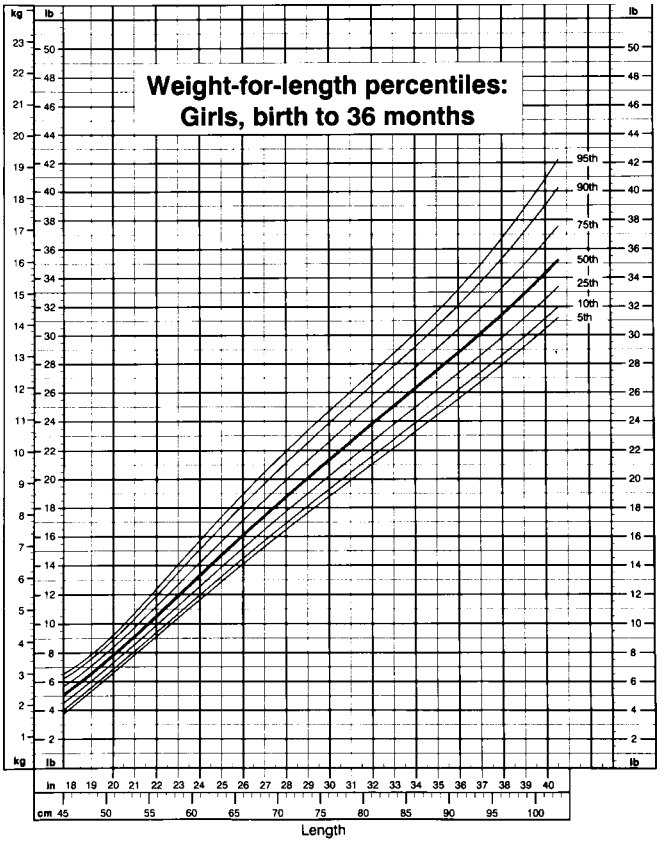

Figure H.4. Weight for length, girls, birth to 36 months.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics, *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

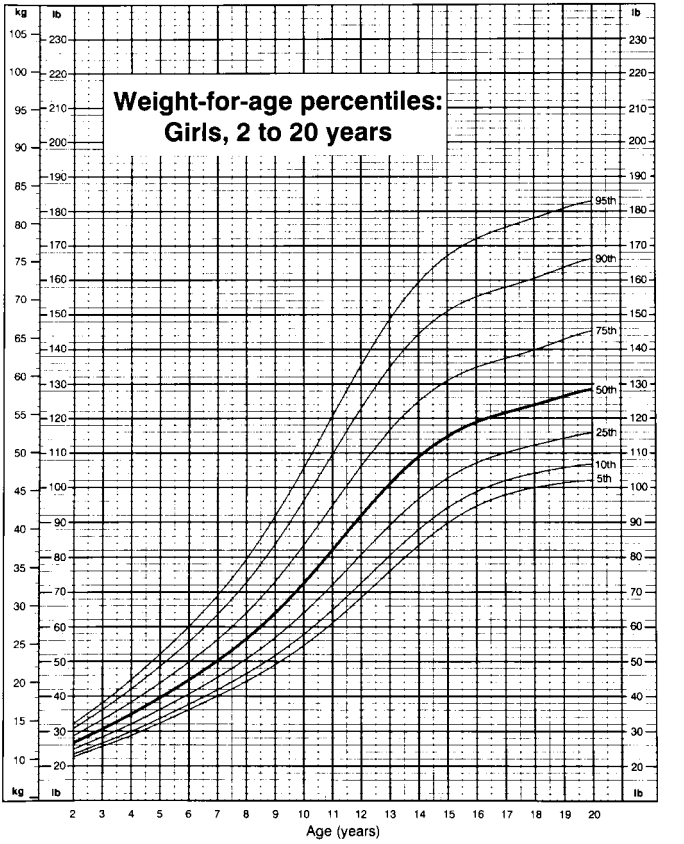

Figure H.5. Weight for age, girls, 2 to 20 years.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

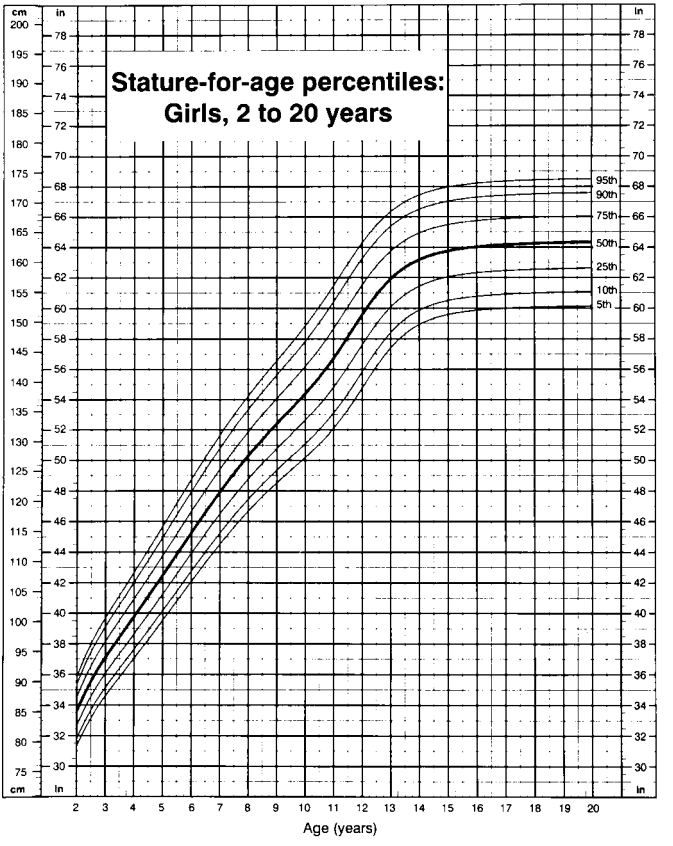

Figure H.6. Stature for age, girls, 2 to 20 years.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

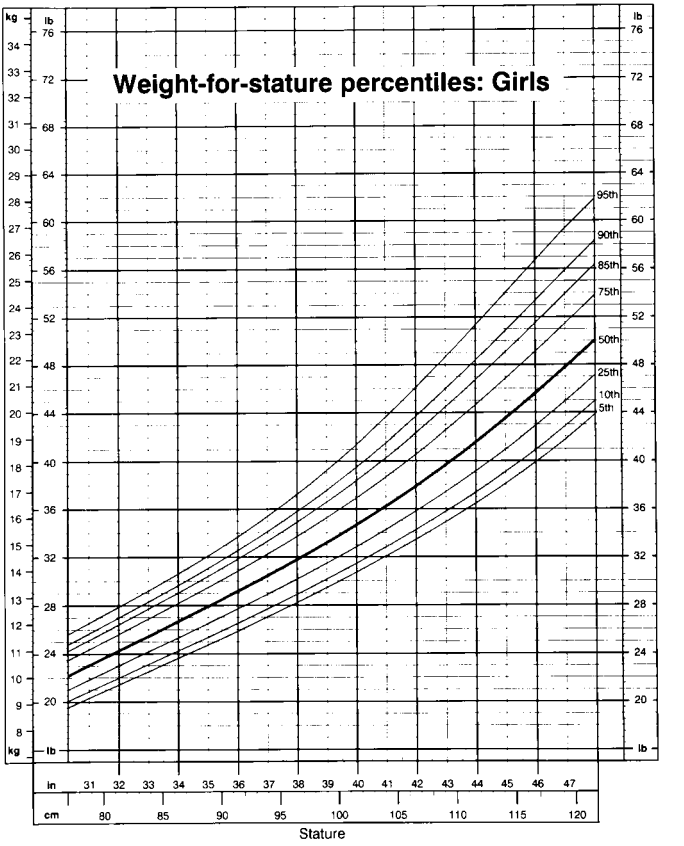

Figure H.7. Weight for stature, girls.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

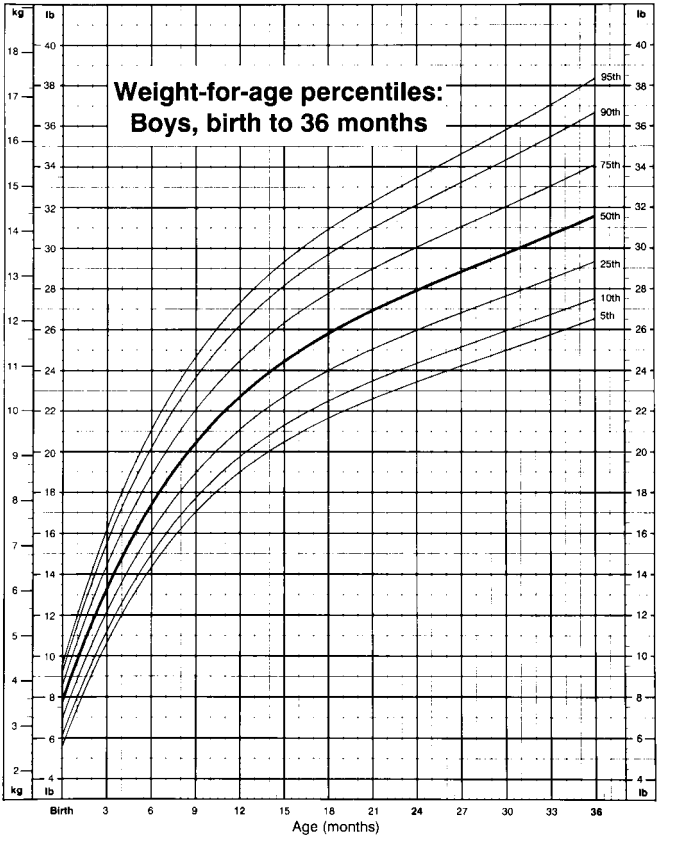

Figure H.8. Weight for age, boys, birth to 36 months.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

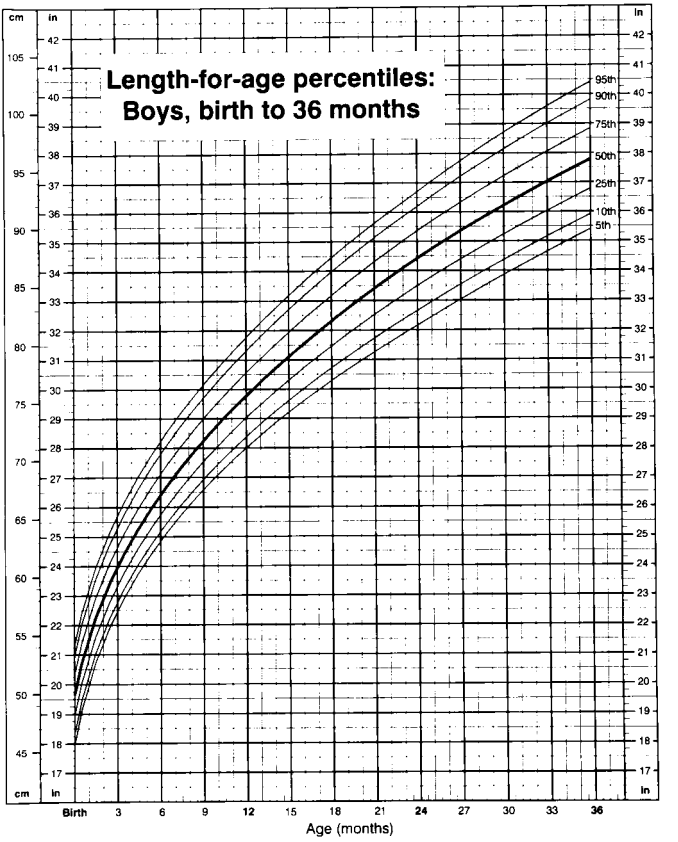

Figure H.9. Length for age, boys, birth to 36 months.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

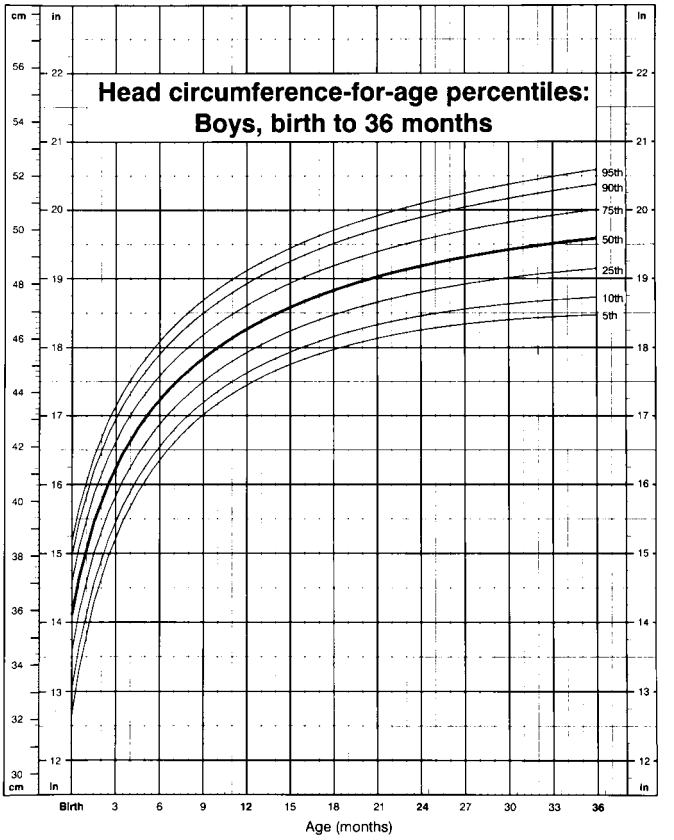

Figure H.10. Head circumference for age, boys, birth to 36 months. Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

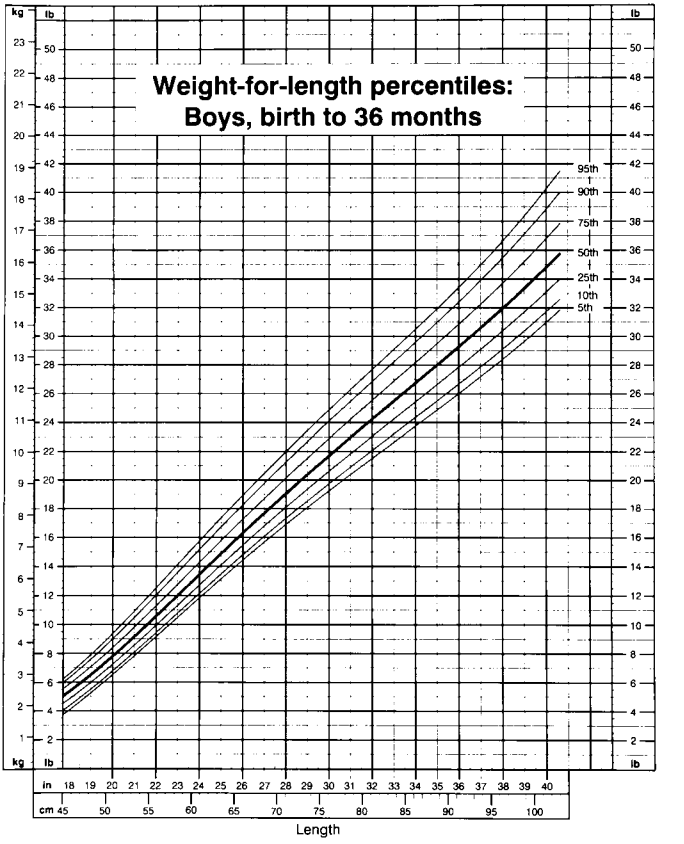

Figure H.11. Weight for length, boys, birth to 36 months.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

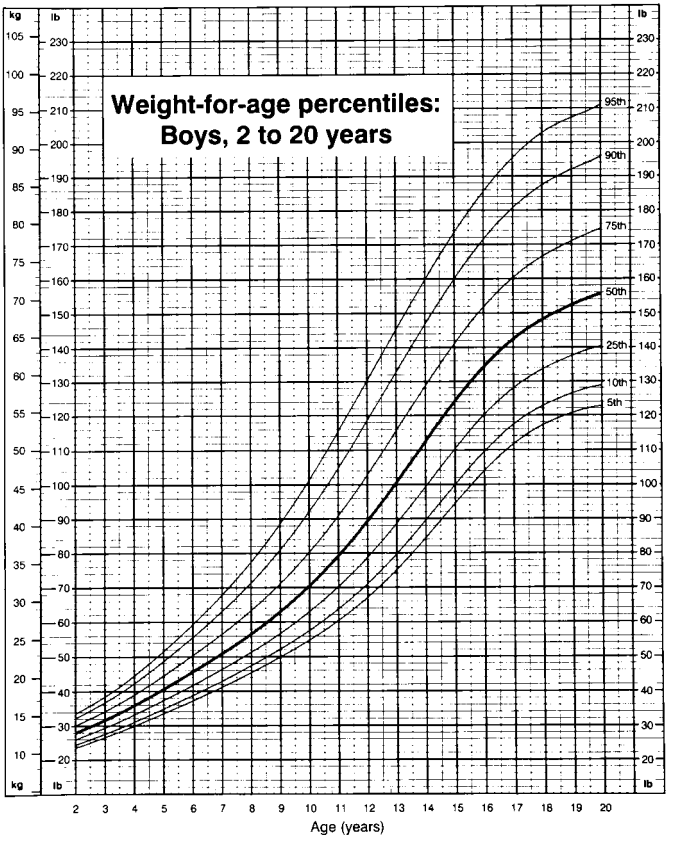

Figure H.12. Weight for age, boys, 2 to 20 years.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

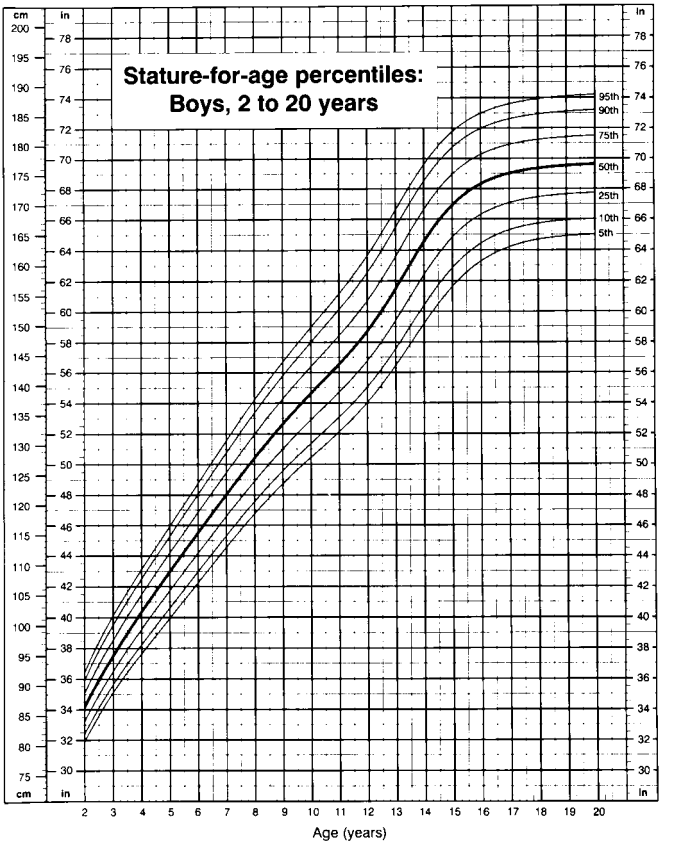

Figure H.13. Stature for age, boys, 2 to 20 years.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

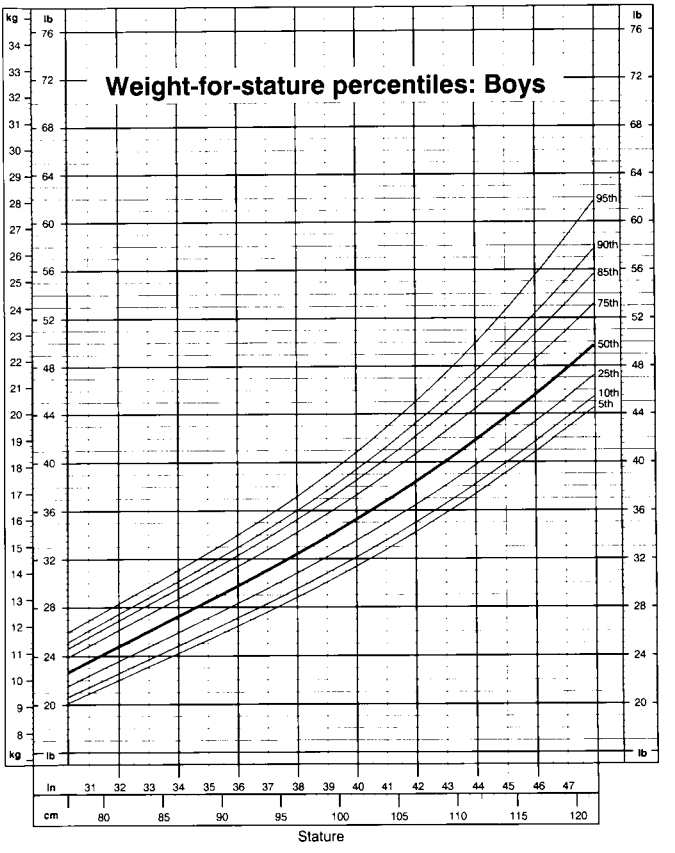

Figure H.14. Weight for stature, boys.
Source: Centers for Disease Control and Prevention, National Center for Health Statistics. *CDC Growth Charts*. Atlanta, GA: Centers for Disease Control and Prevention, 2000.

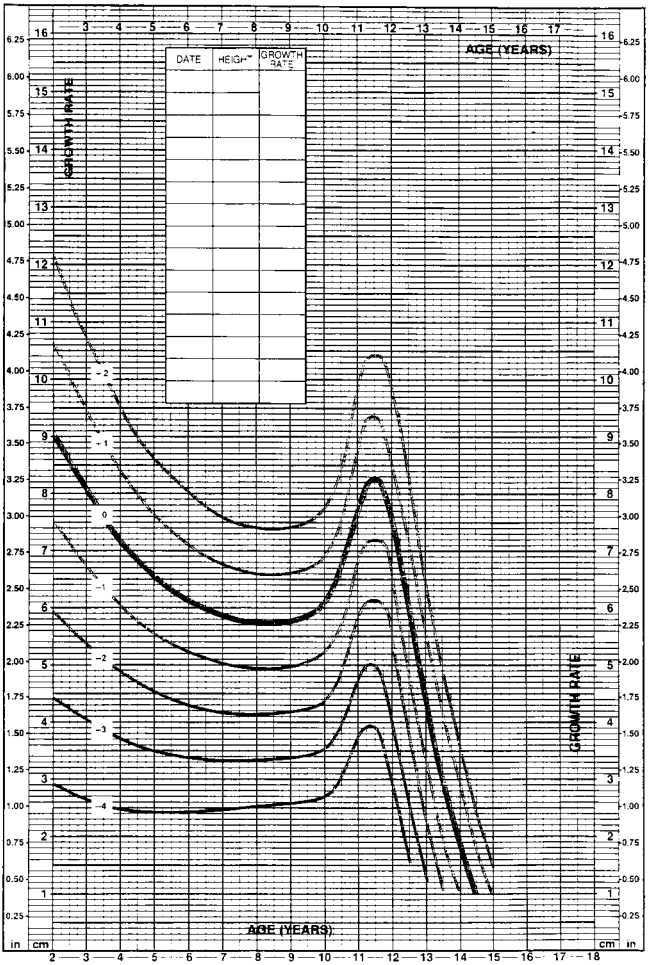

Figure H.15. Growth velocity, girls, 2 to 15 years of age.
Source: Genetech, Inc., 1988. Reproduced with permission.

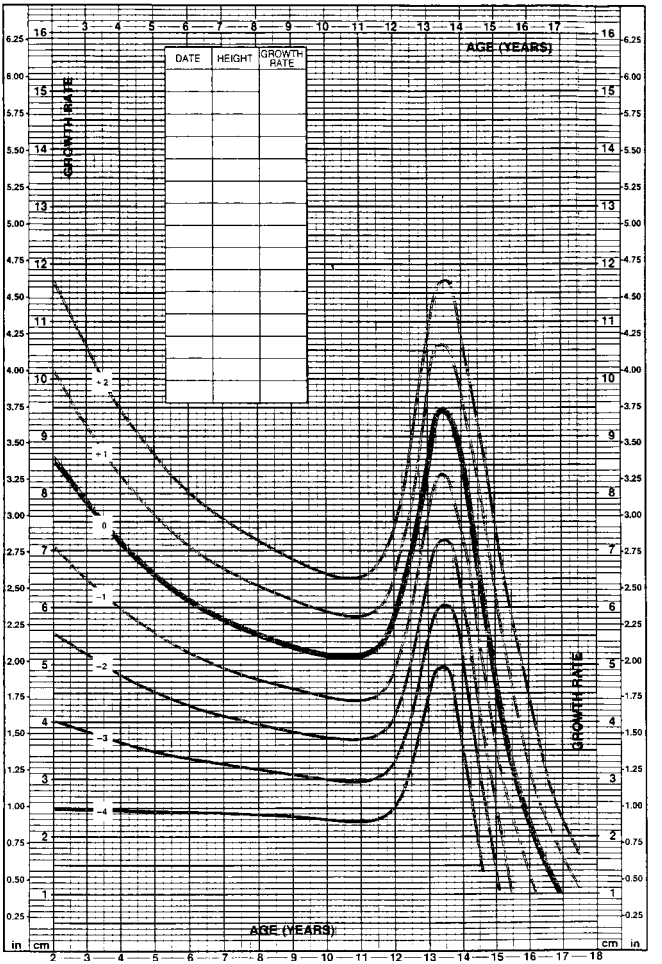

Figure H.16. Growth velocity, boys, 2 to 15 years of age.
Source: Genetech, Inc., 1988. Reproduced with permission.

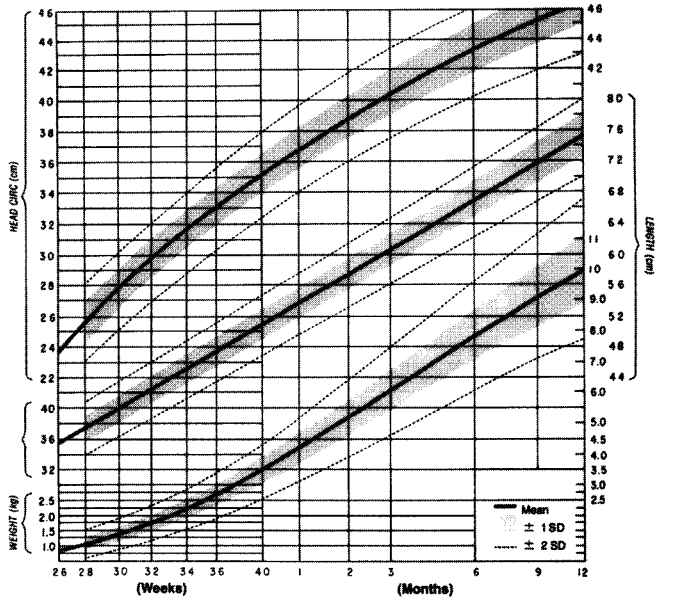

Figure H.17. Growth record for premature infants, birth to 1 year, male and female.
Adapted from: Babson SG, Benda GI. Growth graphs for the clinical assessment of infants of varying gestational age. *J Pediatr* 1976;89:814. Reproduced with permission.

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