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A Clear-Sighted Approach to Conjunctivitis: State of the Art Clinical Practice

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Emergency department (ED) physicians frequently are confronted with children with conjunctivitis. Although the majority of cases are simple bacterial or viral infections, the astute physician must maintain a high index of suspicion for diseases that may result in potential morbidity or mortality for the patient. The authors provide an in-depth analysis of the etiologic agents associated with conjunctivitis, clinical findings associated with each disease, and management options, including a thorough discussion of various topical and systemic therapies.

—The Editor

Epidemiology

Conjunctivitis is one of the most common ophthalmologic complaints managed by ED physicians.[1-3] The prevalence, etiologies, and management vary with the age of the child. Ophthalmia neonatorum is the most common infection seen in the neonatal period.[4-6] Estimated incidence ranges from less than 1% to 25% of newborns.[1,7] The prevalence of *Neisseria gonorrhea* and *Chlamydia trachomatis* in a given population will influence the rates of ophthalmia neonatorum, though many cases are caused by organisms that are not associated with sexually transmitted diseases.[7] Active maternal chlamydia infection is transmitted to newborns 50% of the time. One quarter to one-half of these infected newborns will develop conjunctivitis.[6,8] Caesarian section is not completely protective, nor is ocular prophylaxis with silver nitrate, erythromycin, or tetracycline. There is no change in the incidence of chlamydia conjunctivitis among neonates who receive ocular prophylaxis when compared to those who do not.[9]

Bacterial conjunctivitis is most common in older infants and children younger than 6 years of age. This especially is true during the winter months.[10] For children who develop otitis media in association with conjunctivitis (Conjunctivitis-Otitis Media Syndrome), exposure to siblings with similar symptoms and attendance at daycare have been identified as risk factors.[11] The advent of the Hib vaccine in the early 1990s has not changed the incidence of bacterial conjunctivitis, because most bacterial conjunctivitis is caused by nontypeable *Haemophilus influenzae*, which is not covered by the vaccine.[11] Though the majority of outbreaks of conjunctivitis are caused by viruses, non-typeable *Streptococcus pneumoniae* was

the etiologic agent in an outbreak in 2002 that affected nearly 700 Dartmouth college students.[10,12]

Viral conjunctivitis is most common in school-age children and adolescents, especially during the fall.[10,13] Most outbreaks of conjunctivitis are due to adenovirus; adenovirus types 8, 19, and 37 are responsible for most epidemic keratoconjunctivitis. In addition to the usual transmission by direct contact, poor hand washing by health care workers and contaminated ocular instruments and solutions have been implicated in the spread of disease.[2] Pharyngoconjunctival fever is caused by adenoviruses 3 and 7, with the majority of outbreaks generally involving children younger than 10. The highly contagious organisms generally are spread person-to-person, though outbreaks have been tied to contaminated ponds and pools.[2]

In late childhood and early adulthood, the incidence of allergic conjunctivitis increases, with 10-20% of Americans and Europeans affected.[14] Symptoms may be seasonal or perennial. While children with allergies are bothered by the nasal, ocular, and systemic symptoms, they do not tend to be upset with the symptoms or to limit their activities because of them.[15]

Etiology

Conjunctivitis is predominantly infectious, chemical, or allergic in origin.[16] Bacterial pathogens account for 65-80% of conjunctivitis, while viruses cause 13-20% of cases.[10,17] A red eye also may result from chemical irritation and inflammation from systemic disease. Risk factors and causative agents vary with the child's age.

Ophthalmia Neonatorum. The most frequent cause of neonatal conjunctivitis is chemical irritation. This presents during the first two days of life as a result of ocular prophylaxis.[1,19] Silver nitrate is 2-12 times more likely to cause irritation than erythromycin or tetracycline prophylaxis.[6] It first was used for ocular prophylaxis in newborns in 1884, and has been reported to cause conjunctivitis in 10-100% of infants treated.[6,19]

Bacterial pathogens include *C. trachomatis, N. gonorrhea, Staphylococcus aureus, Streptococcus viridans, H. influenzae,* and *S. pneumoniae*. Gram-negative organisms such as *Pseudomonas aeruginosa* need to be considered and may cause disease in neonatal intensive care patients.[3-6] There have been isolated reports of group B streptococcus as a cause of ophthalmia neonatorum.[18] *Streptococcus mitis* also has been cultured from up to 25% of symptomatic neonates.[7] Approximately half the time, however, an organism is not recovered on culture.[7] Viruses rarely cause conjunctivitis in newborns.[1]

More than half of infants younger than 2 months of age have aerobic bacterial agents as the etiology of their conjunctivitis, with a much smaller percentage caused by chlamydia.[7,20] Chlamydia infection in neonates is not prevented by ocular prophylaxis.[21-23] Between 2% and 20% of women have positive endocervical cultures for chlamydia during pregnancy,[24] and 18-50% of infants born to infected women will develop inclusion conjunctivitis during the first month of life. Transmission may be intrauterine or result from direct contact with the cervix at the time of delivery.[3,24] Delivery by caesarian section is not completely preventive.[3,6,8]

N. gonorrhea conjunctivitis is rare in the United States, as ocular prophylaxis has only a 1% failure rate for this organism.[1,4] Despite this, *N. gonorrhea* is one of the most important etiologic agents of ophthalmia neonatorum due to the aggressive course of the infection and high morbidity associated with untreated disease.[3] The emergence of beta-lactamase producing *N. gonorrhea* has made the selection of appropriate antibiotics imperative.[25] Conjunctivitis due to herpes simplex virus (HSV) likewise is rare, but is associated with high morbidity due to disseminated disease. The practitioner is aided in the diagnosis of HSV keratoconjunctivitis by the frequent presence of associated mucosal or skin findings.[1] *(See Table 1.)*

Bacterial Conjunctivitis. Beyond the neonatal period, bacterial conjunctivitis is twice as common as viral.[1,2,17,26] This especially is true in pre-school-age children.[1,17] *H. influenzae* accounts for 40-50% of bacterial conjunctivitis; *S. pneumoniae* accounts for 10%; followed by *Branhamella catarrhalis*.[1,17,26] The role of *S.*

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Table 1. Neonatal Conjunctivitis

ETIOLOGY	USUAL AGE AT ONSET	MANAGEMENT	COMPLICATIONS	PREVENTION
Chemical	< 24 hours	Observe	None, self-limited	Avoid use of silver nitrate
Neisseria gonorrhea	2-5 days	Admit, IV antibiotics, irrigation	Corneal ulceration/ perforation, blindness	STD screening and treatment during pregnancy
Chlamydia trachomatis	5-14 days	PO erythromycin	Blindness (rare)	STD screening and treatment during pregnancy
Herpes simplex virus type 2	Birth to 4 weeks	IV acyclovir and topical antivirals (e.g., trifluridine)	CNS involvement, disseminated disease	Screening during pregnancy; caesarian section for active maternal infection; avoid infected household contacts

Key: PO = oral; CNS = central nervous system;
STD = sexually transmitted disease; IV = intravenous

aureus has been debated in the literature. Some authors have reported a higher incidence of *S. aureus* growth in patients with conjunctivitis when compared to controls.[27] The majority report similar frequencies in cases and controls, supporting the idea of skin contamination of conjunctival cultures.[1,17,26] It has been proposed that Staphylococcal species may play a role when conjunctivitis follows trauma or surgery.[28] While the majority of conjunctivitis is caused by aerobes, the presence of mixed flora is common. Anaerobes are present 37-63% of the time, often mixed with aerobic organisms.[22,27] In one study, most anaerobes were not detected until seven days after culture, and thus could have been missed by conventional testing.[27] In sexually active teenagers, *N. gonorrhea* and *C. trachomatis* should be considered.[1,29] *N. gonorrhea* conjunctivitis in prepubertal children always should prompt an evaluation for sexual abuse, though cases not related to sexual contact have been reported.[30] *N. gonorrhea* conjunctivitis has developed in patients who are close household contacts of infected individuals, and in patients who have used the folk remedy of applying urine to the eyes to treat viral conjunctivitis.[31]

Conjunctivitis-Otitis Media Syndrome. As many as 25-40% of children with bacterial conjunctivitis may present with, or subsequently develop, otitis media.[3,20] Non-typeable *H. influenzae* is responsible for 60-85% of cases; and *S. pneumoniae* accounts for approximately 15% of cases.[20,26,32] Ninety percent concordance between middle ear and conjunctival isolates has been demonstrated in children with concurrent conjunctival infection and middle ear effusion.[32] The history of an affected sibling and daycare attendance are both risk factors for development of conjunctivitis-otitis media syndrome.[32] The frequency of this syndrome, and the beta-lactamase-producing organisms involved, both have implications for management.[7,33] Otitis media may be asymptomatic, or may develop after the conjunctivitis, complicating both diagnosis and therapy.[1,3]

Viral Conjunctivitis. Adenovirus causes 20% of cases of conjunctivitis and is responsible for most outbreaks, with prevalence highest in the fall and winter.[1,3,4,26] The common complaint of "pink eye" most often is associated with adenovirus type 3, while types 8, 19, and 37 are associated with epidemic keratoconjunctivitis.[34] When conjunctivitis is associated with pharyngitis (pharyngoconjunctival fever), adenovirus is the etiologic agent 65% of the time.[26] Other viral agents also may cause conjunctivitis, though less commonly. Enterovirus is responsible for outbreaks of acute hemorrhagic conjunctivitis.[31] Conjunctivitis due to primary infection with HSV peaks between ages 1 and 5. Most ocular infections are caused by HSV-1, though 75% of neonatal herpes infections are due to HSV-2.[2,35] Conjunctivitis is common in primary infections with varicella zoster virus (VZV), although corneal involvement is rare. Herpes zoster ophthalmicus complicates secondary HSV infections when the ophthalmic division of the trigeminal nerve is involved.[3,36] Conjunctivitis also can develop during mumps or rubella infections.[34]

Allergic Conjunctivitis. Allergic conjunctivitis is more common in late childhood and beyond.[36] Although prevalence has been estimated at 10-20% in adults, the incidence in children who present acutely with conjunctivitis is closer to 2%.[10,14] In addition to airborne allergens, conjunctivitis may result from an allergic reaction or irritation following contact with cosmetics, perfumes, detergents, ophthalmic solutions or preservatives, and contact lenses.[16,37] Irritation from smoke is another common cause of red eye.[10]

Miscellaneous. Prolonged use of vasoconstrictive eye drops has been associated with conjunctival hyperemia, follicular conjunctivitis, and eczematoid blepharoconjunctivitis. Symptoms resolve spontaneously when the medication is discontinued.[38] Other drugs, such as tetrahydrocannabinol, also have been linked to the condition. Autoimmune, inflammatory, and vasculitic conditions have been associated with conjunctivitis, as have irritants such as chlorine in swimming pools. Conjunctivitis is a prominent feature in Kawasaki disease.[23] Sjögren disease, though rare in children, may present with recurrent conjunctivitis and xerophthalmia.[39]

Pathophysiology

The conjunctivae possess both physical and immunologic defenses against infection. Tears not only wash away irritants and bacteria, but also contain IgA and lysosomes. The non-pathogenic bacteria that constitute normal eye flora defend against infection by virulent organisms. The lymphocytes, plasma cells, and neutrophils that comprise the conjunctival immune system provide additional protection against infection. Traumatic disruption of these normal defense mechanisms, or inoculation with virulent organisms, can lead to acute conjunctivitis.[1]

Infectious. Infectious conjunctivitis in neonates is the result of

direct inoculation of the conjunctival sac during the process of birth.[1] Organisms can be acquired within the uterus, or via passage through the cervix or lower genital tract.[19,24] The exact mechanism for transmission is unclear. While one study showed increased rates of conjunctival bacteria with vaginal delivery or prolonged rupture of membranes (PROM), another showed no difference with prolonged labor, PROM, or Caesarian section.[6,7] Neonatal conjunctivitis also may result from agents like *H. influenzae* or *S. mitis.* These common nasopharyngeal organisms are rarely found in vaginal isolates, suggesting transmission after delivery.[7]

Most of the organisms commonly implicated in conjunctivitis, such as *H. influenzae, S. pneumoniae,* and adenovirus, may colonize the nasopharynx.[1,13] Non-typeable *H. influenzae* has been recovered from 40-80% of asymptomatic children.[13,40] Several mechanisms have been postulated for conjunctival contamination. A hand that has been in the nose or mouth may inoculate the eye directly. HSV transmission results from direct contact with an active lesion in an infected person, or by autoinoculation.[3] Retrograde spread from the nasopharynx via the nasolacrimal duct may occur. The nasopharynx also offers a means of communication between the conjunctival sac and the middle ear, providing a possible mechanism for conjunctivitis associated with otitis media.[1]

Special mention should be made of transmission of *N. gonorrhea* beyond the neonatal period. In sexually active teenagers, *N. gonorrhea* conjunctivitis is largely the result of local contamination via autoinoculation from contaminated secretions on fingers.[29] In prepubertal children, the diagnosis of *N. gonorrhea* conjunctivitis should always prompt an evaluation for sexual abuse. There have been reports of *N. gonorrhea* conjunctivitis in toddlers that were attributed to non-sexual transmission resulting from close contact or co-sleeping with infected adults.[13,29,41,42] Although *N. gonorrhea* is very sensitive to drying, it may survive for short periods in a moist environment. Transmission from infected hands or fomites like bedding is possible.[42] Non-sexual transmission was most likely in a 1927 outbreak, during which 67 infants in the same hospital ward were infected.[43]

N. gonorrhea causes a hyperacute, purulent conjunctivitis. Stromal destruction results when enzymes and inflammatory cells, primarily polymorphonuclear leukocytes, migrate from the limbal vasculature into the corneal stroma, resulting in immune mediated tissue damage and corneal "melting." Microbial toxins are believed to play a role in the development of keratitis.[44]

Allergic. The clinical symptoms of allergic conjunctivitis result from an allergen-induced inflammatory response of the mucus membranes lining the eyelids and anterior ocular sclera.[14] The large supply of mast cells exposed in the conjunctiva is highly susceptible to allergen sensitization.[1] The majority of allergic conjunctivitis develops as a result of an acute, type I immediate hypersensitivity response, which may develop within minutes of exposure.[3,14,37] Airborne antigens dissolve in tears, and bind to IgE antibodies on the surface of mast cells in the conjunctival stroma. The mast cells then activate and degranulate, releasing histamine and other pro-inflammatory mediators.[14,45] Histamine binds to H_1 receptors on nerve cells, producing itching. Activation of H_1 receptors on endothelial cells produces vasodilatation and increased vascular permeability. Clinically, burning, itching, injection, edema, and limbal hyperemia result from the dilation of superficial conjunctival blood vessels.[45,46] The accumulation of fluid beneath the loosely attached bulbar conjunctiva produces chemosis.[46] Prostaglandins mediate ocular inflammation by contributing to edema, and by reducing the threshold to histamine-induced pain and itching.[37,45] For this reason, pharmaceutical approaches to controlling the symptoms of allergic conjunctivitis target histamine and prostaglandin synthesis.

Clinical Manifestations

The hallmark of conjunctivitis is eye redness. Hyperemia of superficial conjunctival vessels is more marked in the fornix and lessens toward the limbus of the cornea.[3,46-48] Edema of the conjunctiva and discharge of varying degrees are accompanying clinical features.[46,48] Purulent discharge and bilateral involvement may suggest a bacterial origin, whereas a watery or serous discharge may be suggestive of a viral infection.[3,13,26,48,49] *(See Table 2.)* Photophobia and eye pain are not typical of conjunctivitis, with the exception of epidemic keratoconjunctivitis and HSV keratitis.[3,34,47] Severe ocular pain, photophobia, visual disturbance, and involvement of the limbus are clinically significant and may indicate serious eye disease.[3,34,46,47] Of note, red eyes in contact lens wearers could be due to poor fit, corneal abrasion or ulcer, infectious conjunctivitis, or giant papillary conjunctivitis.[1,47,48] Ophthalmology consultation to guide further evaluation and management is recommended.[47]

Ophthalmia Neonatorum. Given the potential for significant morbidity, timely evaluation and treatment of all neonates with conjunctivitis is a priority. Conjunctivitis in the first several weeks of life can be classified according to age at presentation and clinical findings, although significant overlap exists.[1,3,50] *(See Table 1.)* Chemical conjunctivitis is characterized by vessel injection, watery discharge, and chemosis within the first 24 hours of life. No therapy is indicated, as this condition resolves spontaneously with 48 hours.[1,3,49-51]

N. gonorrhea conjunctivitis, while uncommon, is the most feared form of ophthalmia neonatorum. Symptoms begin in the first week of life, following a 2-5 day incubation period. Ocular prophylaxis may partially suppress active infection if already present at birth.[1,48,50] Rapid progression of inflammation leads to copious, thick, purulent secretions, eyelid swelling, and chemosis.[1,3,49-51] If timely treatment with parenteral antibiotics and eye irrigation is not instituted, the development of corneal ulceration and perforation, iridocyclitis, anterior synechiae and, in rare cases, panophthalmitis may result.[1,3,49-51]

The incubation period for *C. trachomatis* conjunctivitis generally ranges from five to 14 days, though it can extend up to several weeks.[3,8,51,52] The clinical presentation of these infants is variable. The degree of conjunctival erythema varies, and discharge may be minimal, or copious and purulent. There also may be severe eye edema, chemosis, and even pseudomembrane formation more reminiscent of *N. gonorrhea* conjunctivitis.[1,3,49,50]

Although rare, ocular infection with *Pseudomonas aeruginosa* potentially can lead to endophthalmitis, sepsis, and death. Infection typically is acquired in the nursery setting. The infection manifests initially on the fifth through 18th day of life, with ocular edema, lid erythema, purulent discharge, and pannus formation.[50]

Table 2. Distinguishing Features of Various Forms of Conjunctivitis

	BACTERIAL	VIRAL	ALLERGIC
Age	< 6 years	> 6 years	Late childhood to early adulthood
Discharge type	Purulent	Watery/serous	Mucoid
Conjunctival injection	Severe	Moderate	Mild
Unilateral/bilateral	Bilateral > unilateral	Unilateral > bilateral	Bilateral > unilateral
Associated signs/symptoms	Gritty sensation	Sick contact with red eyes	Itching, chemosis
Associated conditions	Otitis media	Upper respiratory infection/ pharyngitis (adenovirus)	Allergic rhinitis, asthma, eczema
Season	Winter	Fall	Spring/summer
Preauricular adenopathy	Uncommon	Common	Not present

Bacterial Conjunctivitis. The differentiation between bacterial and viral conjunctivitis can be difficult, though certain clinical findings can help guide diagnosis and management.[1-3,13] *(See Table 2.)* In bacterial conjunctivitis, tearing and ocular irritation begin in one eye followed by the abrupt development of mucopurulent or purulent drainage.[46] The development of conjunctival papillae, which have a vascular core, is a nonspecific response more common with bacterial disease.[1-3,13,48] The patient or caregiver may describe the presence of dried secretions and debris at the base of the eyelashes and matting of the eyelids upon awakening.[1,46] The infection spreads to the opposite eye within 48 hours.[46,50] Bilateral eye involvement, though seen in both viral and bacterial conjunctivitis, is more likely with bacterial disease.[3,13,26] In one series, bilateral disease was present in 50-75% of patients with *S. pneumoniae* or *H. influenzae* conjunctivitis, compared with 35% of patients with adenoviral infection.[26] Further subjective findings of mild pruritis and/or burning and a "gritty sensation" in the affected eye may correspond with the objective signs of redness, eyelid edema, and mucopurulent discharge.[2,3,50] Palpebral and bulbar conjunctivae are erythematous in typical cases (bulbar greater than palpebral), but are not hemorrhagic.[1,13] A concomitant acute otitis media (even in the absence of ear pain) points further toward a bacterial etiology.[1,3,13,32]

The practitioner confronted with a hyperacute conjunctivitis always should consider the possibility of infection with Neisseria species.[3,13,29,46,48] The development of a hyperacute purulent conjunctivitis in a sexually active teenager, supported by presence of a purulent genital discharge, should raise the suspicion of infection with *N. gonorrhea*.[29,46] The findings of bright red, chemotic eyes with copious purulent discharge, pronounced eyelid swelling, and tenderness on palpation are so severe that most patients seek medical care prior to infection of the contralateral eye.[46] As in neonates, sight-threatening complications, such as corneal perforation and loss of the eye, can result when correct diagnosis and treatment are delayed.[29,46,48] A similar presentation can be seen in conjunctivitis secondary to *Neisseria meningiditis*. Ocular involvement is unilateral in two-thirds of cases and can be complicated by invasive disease such as sepsis or meningitis.[2,3,48]

Viral Conjunctivitis. Conjunctival hyperemia, edema, and watery discharge characterize viral conjunctivitis.[3,46,48,49] A helpful finding may be conjunctival follicles, which represent lymphocyte aggregation and have an avascular center, in contrast to the papillae seen in bacterial disease.[2,3,47] Periorbital swelling can be dramatic and thus mistaken for preseptal cellulitis, especially with adenoviral infection.[2,13,49,53] However, with preseptal cellulitis, the lid swelling usually is painful, and the child is ill-appearing. Involvement is unilateral at the outset and often progresses to the opposite side after a few days, though this is less likely than with bacterial disease.[13,26] As might be expected, an upper respiratory tract infection may co-exist or precede the development of viral conjunctivitis. Preauricular lymphadenopathy, though not universally present, also is strongly suggestive of viral etiology.[2,46,48,51]

Adenovirus is the most common cause of viral conjunctivitis.[1-3,51] Ocular adenovirus infections can take several forms. Follicular conjunctivitis is characterized by ocular findings of hyperemia, watery discharge, palpebral edema, preauricular lymphadenopathy, and other signs of upper respiratory tract infection.[1,3,49,51]

In pharyngoconjunctival fever, pharyngitis and fever accompany eye involvement and bilateral preauricular nodes are common.[2,3,48] Eye findings may include small petechial conjunctival hemorrhages. This entity resolves over 4-14 days.[2,3,48] Although the signs and symptoms of fever and bilateral nonpurulent conjunctivitis may initially suggest Kawasaki disease, the absence of further diagnostic criteria can help to exclude this diagnosis.

Epidemic keratoconjunctivitis (EKC) occurs in older children and adults.[2,3,48] In contrast to pharyngoconjunctival fever, generalized upper respiratory tract symptoms and fever may be lacking. Corneal involvement is a dominant finding.[2,48] At the outset, diffuse epithelial keratitis leads to complaints of photophobia and foreign body sensation. Eyelid edema, chemosis, conjunctival hyperemia, and tender preauricular adenopathy also are present. Children may keep their eyes closed and rub them as a result of the foreign body sensation.[2,49] Follicle formation and subconjunctival hemorrhages may develop within 48 hours.[48,49] Punctate epithelial defects evolve in 7-10 days, causing corneal opacities. These opacities typically resolve without scarring, but may lead to minor reductions in visual acuity.[2] The eyelid edema and formation of inflammatory membranes that occurs may lead to a false diagnosis of preseptal cellulitis; again, with preseptal cellulitis, the lid swelling is painful and the child appears ill.[2,3,13,53]

Primary infection with HSV type 1 can present with an acute follicular conjunctivitis characterized by a red eye, chemosis, elevated follicles, and watery discharge. In the early stages, it may be clinically difficult to differentiate from adenoviral conjunctivitis.[54]

When vesicular lesions co-exist on the eyelid with the presence of corneal lesions, this infection can be easily differentiated from other forms of viral conjunctivitis.[1-3,51] Findings are unilateral in the majority of cases, and a tender preauricular node may be noted.[2,48,54] HSV conjunctivitis may persist for 2-3 weeks.[48] Corneal involvement is seen in 50% of cases and contributes to the key finding of severe ocular pain.[3,34,36,49,51] Dendritic lesions of the cornea on fluorescein staining are a pathognomonic finding of HSV keratitis.[2,3,49,51] In addition to pain, patients with acute keratitis may have tearing, reduced vision, and eye redness concentrated in the perilimbic region.[3,34] Diagnosis of recurrent HSV infection can be more difficult in the absence of the characteristic herpetic skin findings. A history of recurrent red eye or prior ocular skin findings may be helpful.[47] A high index of suspicion is required, particularly in children 1-5 years of age, to avoid missing potential corneal involvement.[2] Eye pain can be helpful in distinguishing this condition from more benign causes, although HSV keratitis occasionally can be painless because of induced corneal hypoesthesia.[47]

Both primary and recurrent infections with VZV may involve the conjunctivae. Vesicles and ulcers of the conjunctiva may be seen with primary infection.[3,36,47] Corneal involvement is much more common and can co-exist with iritis in recurrent VZV infection. Ophthalmologic consultation is warranted with both HSV and secondary VZV infections.[3,49]

Allergic Conjunctivitis. Allergic conjunctivitis is a response to allergen exposure and may have seasonal recurrence, given patterns of exposure to pollens, grasses, and animal dander.[1,3,34,46,48] Allergic conjunctivitis is associated with other atopic conditions such as eczema, asthma, and allergic rhinitis.[1,34,46,48] Family history, onset in late childhood or early adulthood, and seasonal or perennial recurrence are clues to diagnosis.[1,3,34,46] The hallmark of allergic conjunctivitis is ocular itching with varying degrees of bilateral conjunctival hyperemia, tearing, chemosis, eyelid edema, and stringy mucoid discharge.[1,3,13,34,46,48] Bilateral involvement and findings of papillary hypertrophy on palpebral conjunctiva are distinguishing features.[1,48]

Vernal conjunctivitis is a more serious form of allergic conjunctivitis.[1] It is more common in warmer climates, with onset in the prepubertal years.[1,48] In the palpebral form, giant papillae are pronounced in the upper lid and may produce a "cobblestone" appearance.[1,48] Gelatinous opacities straddle the limbis, the margin of the cornea overlapped by the sclera, especially in persons of African descent.[1,48] Eyelid swelling and stringy discharge are further manifestations. Possible complications include diffuse epithelial keratitis and ulceration of the corneal epithelium, which respond poorly to treatment.[1,48] Although vernal conjunctivitis is mostly a self-limited disease, recurrent or persistent symptoms mandate referral to an ophthalmologist, as topical steroids may be part of the short-term therapy.[48]

Atopic keratoconjunctivitis may develop in patients with atopic dermatitis. Clinical manifestations include itching or burning, eye redness, mucoid discharge, and photophobia.[1,48] A papillary response is more common on the lower eyelid, in contrast to the pronounced upper lid involvement in vernal conjunctivitis. Corneal involvement can be severe in recurrent bouts of conjunctivitis.[1,48] A family history of atopy, or skin findings consistent with atopic dermatitis, may exist.[48] Referral to an ophthalmologist to initiate and monitor possible topical steroid therapy is warranted.

Diagnostic Studies

The value of diagnostic studies in acute conjunctivitis varies with the age of the patient, the suspected etiology, and the technique of the examiner. In neonates, a presumptive diagnosis of chemical conjunctivitis can be confirmed when a gram stain of the conjunctival scrapings reveals no bacteria.[55] Patients with *N. gonorrhea* conjunctivitis will have gram-negative intracellular diplococci in conjunctival scrapings, and significant growth on chocolate agar or Thayer-Martin culture.[19,29,44] These colonies should be checked for penicillin resistance and beta-lactamase production.[44] Treatment should be initiated promptly. Do not wait for culture results if *N. gonorrhea* infection is suspected.[29] *P. aeruginosa*, a rare but serious cause of neonatal conjunctivitis, is identified by the presence of gram-negative rods and a positive culture.[3]

Gram stain is not helpful in detecting Chlamydia conjunctivitis. Identification is made when inclusions are seen in epithelial cells on a giemsa stain of conjunctival scrapings, or by culture or antigen testing.[19] To perform a culture, epithelial cells should be collected on a Dacron-tipped swab.[8] Specimens may be obtained from the conjunctiva or the nasopharynx.[56] Chlamydia also may be detected by DNA probe, direct fluorescent antibody, enzyme immunoassay, polymerase chain reaction, or ligase chain reaction. The sensitivity and specificity of all these methods are high.[8,56] Viral cultures should be obtained when HSV or VZV conjunctivitis is suspected. In HSV, the antigen also may be detected in vesicle aspirates. Immunofluorescence of vesicle scrapings can identify the virus in VZV conjunctivitis.[35,36]

Although adenovirus causes 20% of conjunctivitis, the poor availability and turnaround time of viral culture make culturing impractical.[28] When available, rapid direct fluorescent antibody testing may help guide management. Gram stains seldom are done in patients with acute conjunctivitis, due to poor sensitivity.[28,57] This partly is due to technique, as samples obtained by experienced practitioners have a higher yield. The swab should be swept over the inferior palpebral conjunctiva, taking care to avoid contamination from the skin on the eyelid or secretions in the inner canthus.[13] The presence of more than 15 white blood cells (WBCs) per high power field on a Gram stain of conjunctival exudate is predictive of a bacterial etiology.[11] Exudative specimens are not adequate for identifying specific organisms, since they contain more fibrin and cellular debris than intact cells.[2] Gram stains of conjunctival scrapings are more effective predictors.[13,17] Better specimens may be obtained when a topical anaesthetic is used prior to scraping with a spatula.[2] Testing may be worthwhile to determine the etiology for children who are being restricted from school or daycare.[2] When giemsa stains are performed on conjunctival scrapings, the presence of neutrophils suggest bacteria, while lymphocytes are associated with a viral etiology.[17] Eosinophils are present in the scrapings of 20-80% of allergic patients, but are not found in non-allergic patients.[1,17,37] Allergic conjunctivitis also may be diagnosed by the presence of a papillary conjunctival response on slit lamp exam of the everted

lid.[1] Of interest, conjunctival cultures are poor predictors of middle ear fluid cultures.[58]

Differential Diagnosis

The differential diagnosis for conjunctivitis, or the red eye, includes both ocular and systemic processes. Ocular causes fall into infectious, allergic, traumatic, anatomic, chemical, and inflammatory categories. *(See Table 3.)* Infectious causes are most common, and include bacteria, viruses, and Chlamydia. Amoebiasis is seen in other parts of the world, and should be considered if the travel history is relevant. Infections of other structures in the eye may be confused for conjunctivitis. These include uveitis, episcleritis, keratitis, blepharitis, and dacrocystitis. The first three cause circumcorneal injection or "ciliary flush," which is easily mistaken for conjunctival injection.[34] A history of unilateral involvement, pain, photophobia, or visual changes should prompt consideration of one of these entities. Allergens can cause both vernal and keratoconjunctivitis. Both airborne and contact allergens play a role. Trauma to the eye may cause corneal abrasion, subconjunctival hemorrhage, or foreign body deposition. All of these cause redness, pain, and irritation. The thick discharge and watery eye associated with nasolacrimal duct obstruction may be confused with conjunctivitis, though the conjunctiva themselves are not red.[10,16] Exposure to toxins or chemicals may cause eye redness, as can cosmetics, contact lenses, and the preservatives in ophthalmic solutions. Redness from "dry eye" results from decreased tear production, which may be due to heredity, irritants like smoke or pollutants, systemic diseases, or topical or systemic medications.[16,34] Glaucoma, though rare in children, is a serious cause of eye redness.[1,10]

Systemic processes also may be associated with conjunctivitis. The conjunctivitis associated with Kawasaki generally involves the bulbar conjunctiva, and is less likely to be purulent.[59] Patients with systemic lupus erythematosis (SLE) may present with keratoconjunctivitis sicca, or with follicular conjunctivitis. Their eyes tend to be painful and itchy, but without redness or discharge.[60] Other entities on the differential include Lyme disease, leptospirosis, juvenile rheumatoid arthritis, and Stevens-Johnson syndrome.[1]

Management

General Management. In addition to treating specific pathogens, therapy of conjunctivitis should provide relief of clinical symptoms, shorten the period of contagion, and decrease the likelihood of recurrence.[28] The ideal therapy should be easy to administer, have limited side effects, and be reasonably priced.[28] Regardless of etiology, all patients with conjunctivitis, and their caregivers, should practice good hand-washing and avoid sharing personal items that are likely to be contaminated, such as bedding and towels. Cool compresses, if tolerated, offer symptomatic relief of conjunctival irritation and swelling of periocular tissues.[3] Specific medications may be administered orally or topically, depending on suspected etiology and the constellation of symptoms. Management decisions are complicated by the fact that the etiology of individual cases often is difficult to determine clinically.[26] Many topical ophthalmic medications are not approved by the U.S. Food and Drug Administration for use in children, though they frequently are used. Adverse reactions are uncommon, though irritation frequently was associated with sulfacetamide solutions when their usage was more widespread. Preservatives in ophthalmic solutions may also cause irritation.[28] Among antibiotic ointments, neomycin is associated with higher rates of hypersensitivity reactions.[34] Topical medications have the additional disadvantage of being difficult to administer to young children.[13] Ointments are more difficult to administer, and cause brief blurring of vision, but are not washed out as easily by tears.[61] Eye drops can be administered by application to either the lower conjunctival sac or the inner canthus of the closed eye.[61,62] The second method is better tolerated by most children, and testing in adults suggests that this technique results in drug penetration of 66%, when compared to conventional administration.[62] Concerns remain that topical therapy, even when correctly administered, has the disadvantage of failing to treat or prevent the large number of cases of conjunctivitis-associated otitis media.

Neonates. Chemical conjunctivitis in neonates resolves without treatment, and is best prevented by avoiding treatment with silver nitrate.[3] Bacterial ophthalmia neonatorum is best prevented by screening pregnant women for sexually transmitted diseases, and treating those who test positive. The majority of studies show no significant difference in the rates of Chlamydia conjunctivitis in neonates treated with silver nitrate, topical erythromycin, or tetracycline at the time of delivery.[9,63] Those who do show a small decrease in the incidence of Chlamydia conjunctivitis show no difference in the rates of nasopharyngeal colonization, which is associated with development of Chlamydia pneumonia.[24,64] Infants with Chlamydia conjunctivitis should be treated systemically with

Table 3. Differential Diagnosis of Red Eye*

OCULAR CAUSES
• Infectious
- bacterial, viral, chlamydial, amoebal
- keratitis conjunctivitis, episcleritis
- uveitis, iritis, endophthalmitis
- periorbital cellulitis, blepharitis, hordeolum, chalazion, trichiasis
- nasolacrimal duct obstruction, dacrocystitis
• Traumatic (foreign body, abrasion, iritis, globe injury)
• Allergic
• Chemical, irritative
• Other: Glaucoma, dry eye, contact lens
SYSTEMIC DISEASES
• Infectious disease (varicella, mumps, measles)
• Atopic dermatitis
• Kawasaki disease
• Collagen vascular disease
• Juvenile rheumatoid arthritis
• Inflammatory bowel disease
• Lyme disease
• Leptospirosis
• Stevens-Johnson syndrome

* List not inclusive

erythromycin ethyl succinate for 14 days.[8] This regimen has the additional advantage of treating Chlamydia pneumonia and eliminating nasopharyngeal colonization. Follow-up is necessary, since erythromycin therapy has a 20-30% failure rate.[19,65,66] The infant's mother and her sexual partner(s) also should be treated.[1] Azithromycin also has been tested in a very small series. Given once at a dose of 20 mg/kg, it eradicated symptoms and positive cultures in three of five infants studied. The same dose, given once a day for three days, effectively treated six out of seven infants.[65] *N. gonorrhea* conjunctivitis warrants hospitalization, frequent ocular irrigation, evaluation for associated sepsis or meningitis, and parenteral antibiotic therapy.[3,6,19] Given high rates of beta-lactamase producing organisms, and increasing resistance to penicillin and erythromycin, intravenous (IV) or intramuscular (IM) ceftriaxone is the treatment of choice.[3,6,19,25,44] *P. aeruginosa* and *N. meningitidis*, though rare causes of conjunctivitis, also have the potential to produce systemic disease, and also should be treated with systemic antibiotics. Gonococcal and *P. aeruginosa* conjunctivitis have greater potential for ocular damage, and should prompt an urgent ophthalmology consult.[3]

Bacterial Conjuntivitis after the Newborn Period. Bacterial conjunctivitis will resolve without treatment, but the use of topical or systemic antimicrobial agents shortens the time to clinical improvement and microbiologic cure.[1,3,57,67] Use of systemic agents has the advantage of decreasing the frequency of subsequent otitis media.[11] Therapy primarily should be directed at non-typeable *H. influenzae* and *S. pneumoniae*, which cause the majority of bacterial conjunctivitis in children. When children present with Conjunctivitis-Otitis Media Syndrome, the etiologic agent frequently is beta-lactamase-producing *H. influenzae*.[3,20] Oral therapy with a beta-lactamase-resistant antibiotic such as amoxicillin-clavulanate or a third-generation cephalosporin is recommended.[3,20] Topical therapy will lead to clearing of conjunctival cultures within days, but will not prevent the development of otitis media.[33] Conversely, oral treatment of otitis media will treat conjunctivitis without the addition of topical therapy.[13] Of note, follow-up of children with conjunctivitis-otitis syndrome at two weeks revealed high rates of persistent otitis.[11] This is consistent with the finding that oral therapy with amoxicillin does not eradicate *H. influenzae* from the nasopharynx.[11]

Topical Antimicrobials. The many topical preparations available have varying spectrums of activity, different side effects, and wide variations in cost. In addition to these considerations, the high rates of beta-lactamase production by *H. influenzae* and *S. pneumoniae* should be borne in mind when selecting appropriate therapy.[20] Trimethoprim-polymyxin B is inexpensive, offers broad coverage, and has few side effects.[61] Sodium sulfacetamide provides good gram-positive coverage, but stings when administered.[3,61] The aminoglycosides gentamicin and tobramycin provide excellent gram-negative coverage, but are expensive, and provide poor coverage of Streptococcus species and Chlamydia.[3,61] In addition, prolonged use may lead to corneal epithelial toxicity.[61] Erythromycin, while inexpensive and effective against gram positives and Chlamydia, provides poor coverage against *H. influenzae, B. catarrhalis,* Staphylococcal species, and gram-negative organisms. Local allergic reactions also have been reported.[2,3,61] For these reasons, use of topical fluoroquinolones often is recommended. Ciprofloxacin and ofloxacin are more expensive than the other agents, but offer excellent broad-spectrum coverage with few side effects other than irritation.[2,61,68] In rabbit models, topical ofloxacin had the additional advantage of remaining at therapeutic concentrations in the tears for hours longer than gentamicin or tobramicin.[69] Although the use of ciprofloxacin in children has been limited, following animal studies linking oral quinolone use to arthropathy in immature animals. Clinical studies in children do not support this.[70] A recent study of topical ciprofloxacin therapy for acute conjunctivitis in children found the drug to be safe and well tolerated; no non-ocular adverse events were reported.[70] Unfortunately, resistance to second- and third-generation fluoroquinolones has been increasing. Up to 30% of gram-positive ocular isolates no longer are sensitive to ciprofloxacin, ofloxacin, or levofloxacin.[10] The fourth-generation ophthalmic fluoroquinolones moxifloxacin and gatifloxacin are well tolerated and provide coverage against a wide range of organisms. Increased solubility of the agents allows them to penetrate tissues at higher levels, and the agents bind to two different bacterial enzymes, so that resistance is less likely to develop.[10] Regardless of which antimicrobial agent is chosen, preparations that combine antibiotics with steroids should be avoided. The steroid component may slow eradication of the infectious organisms, increase the risk of keratitis in patients with herpes conjunctivitis, and increase intraocular pressure.[61]

Viral. Specific therapy is not indicated for most viral conjunctivitis. Anti-viral and anti-inflammatory agents are no more effective at relieving symptoms than placebo.[71] Ketorolac ophthalmic solution, which provides relief of symptoms in allergic conjunctivitis, is no better than artificial tears at reducing discomfort, itching, tearing, foreign body sensation, or eyelid swelling in patients with viral conjunctivitis. Topical steroids also have been tested for relief of symptoms associated with viral conjunctivitis. Using a rabbit model, 1% prednisolone acetate solution significantly decreased both the symptoms of conjunctivitis and the presence of subepithelial infiltrates. Unfortunately, steroid therapy also increased viral replication and the duration of viral shedding.[72]

Certain viruses do warrant specific therapy. The topical antivirals trifluridine, iododeoxyuridine, and vidarabine have all proven useful in the treatment of HSV conjunctivitis.[35] Infants with ocular involvement from HSV should receive both topical therapy and parenteral acyclovir, due to the high morbidity and mortality associated with disseminated disease. Oral acyclovir also may be used for recurrent infections.[35] For recurrent VZV infections, oral or IV acyclovir are both effective when therapy is initiated within 72 hours of vesicle eruption. Foscarnet can be used for acyclovir resistant VZV.[21,36] Steroids may be indicated to prevent vision loss secondary to glaucoma from iritis, but should be prescribed only by an ophthalmologist. For immunocompetent patients with recurrent herpes zoster ophthalmicus, oral acyclovir and valacyclovir equally are effective as preventative agents and for reducing the duration and severity of pain.[21] Valacyclovir is not licensed for use in children.[35]

Allergic. The symptoms of allergic conjunctivitis can be relieved with cool compresses, oral antihistamines, and a variety

of topical agents. Different topical preparations contain non-steroidal anti-inflammatory agents, mast cell stabilizers, antihistamines, and vasoconstrictors, singly and in combination.[3,34,61] Ketorolac is superior to placebo in reducing many of the signs and symptoms of allergic conjunctivitis, though placebo alone is effective up to 70% of the time. Burning on installation of ketorolac drops is common, and the incidence of severe ocular irritation is higher than placebo.[45,73] Mast cell stabilizers like cromolyn sodium 4% and lodoxamine tromethamine 0.1% work by prevention, and may require days to weeks before an effect is seen.[3,61] Topical H_1 antagonists include levocabastine and emedastine difumarate, which clinically is superior due to greater potency and duration of action.[14,61] Topical antihistamines also may be combined with other agents. Olopatadine 0.1% solution is a combination antihistamine and mast cell stabilizer.[34,61] Combinations of antihistamines and vasoconstrictors include naphozoline-antazoline and naphozoline-pheniramine.[34,61] There is some concern that these agents may cause a reduction of body temperature in young children.[61]

Additional Aspects

Infections Associated with High Morbidity. While most conjunctivitis resolves spontaneously without treatment, several types are associated with higher morbidity if the diagnosis is missed. Untreated Chlamydia conjunctivitis can lead to conjunctival scarring and corneal infiltrates.[6] *P. aeruginosa* conjunctivitis, though rare, commonly leads to corneal perforation, endophthalmitis, and blindness, so early ophthalmologic consultation is warranted.[6,19] Death from sepsis and shock also has been reported, leading to recommendations for close monitoring and systemic therapy.[6] Ulcerative keratitis and corneal perforation can result in the loss of an eye when *N. gonorrhea* conjunctivitis goes unrecognized,[1,6,29,30,44] and the progression from purulent conjunctivitis to globe perforation may occur in as little as 24 hours.[2,44] Gram stain and culture of high-risk infants is recommended, even if prophylaxis has been given.[1] A high index of suspicion is required to prevent misdiagnosis in the older infant or child who presents with purulent conjunctivitis. In pre-pubertal children, 75% of cases initially are misdiagnosed.[30] A diagnosis of *N. gonorrhea* conjunctivitis beyond the neonatal period always should prompt an evaluation for other sites of infection, other sexually transmitted diseases, and consideration of the possibility of abuse.[3,30] Infection with *N. meningitidis* may be invasive or non-invasive. Ocular manifestations may include corneal ulcers, keratitis, subconjunctival hemorrhage, or iritis. Associated systemic disease can be deadly.[3,74] In addition to ophthalmologic consultation, a sepsis evaluation and systemic treatment for these patients is indicated. Chemoprophylaxis should be provided to close contacts of patients with invasive disease.[74] Ocular involvement from primary HSV infection is difficult to differentiate from adenoviral or chlamydial infections.[3] For this reason, infants with suspected viral conjunctivitis should be evaluated for disseminated or central nervous system herpes, which have mortality rates of 50-85%.[19] Affected infants should be hospitalized and treated with both IV acyclovir and topical trifluridine drops, since the cornea is avascular and not penetrated by the acyclovir.[19] Ophthalmologic consultation is indicated for herpes zoster ophthalmicus, which commonly involves the cornea and causes iritis.[3] Untreated recurrent herpes ophthalmicus leads to corneal opacification.[19]

Topical Drug Safety in Children. Young children require smaller doses of medication than adults to achieve effective ocular concentrations.[61] Despite this, toxicity from topical ophthalmic agents is uncommon.[75] Some preparations deserve special mention. Sulfacetamide drops have been associated with the development of both SLE and Stevens-Johnson syndrome.[3] Topical aminoglycosides can cause corneal ulceration and epithelial toxicity with prolonged use.[2,61] When corticosteroids are added to topical antibiotic preparations, they can aggravate herpes simplex and predispose to corneal involvement. In addition, these preparations may increase virus replication and duration of shedding, resulting in delayed healing. Superinfection also is more likely.[72,76] Topical corticosteroid therapy also can lead to cataracts, glaucoma, and increased intraocular pressure.[61,71,72,76] Young children are more susceptible to intraocular pressure elevation from steroids than teens and adults, and the visual loss from steroid induced glaucoma is irreversible.[77] Ophthalmic antihistamine decongestant preparations may cause rebound hyperemia with prolonged use.[36,44]

Systemic absorption of topical ophthalmic preparation occurs by two different routes. Medications that enter the nasolacrimal duct systemically may be absorbed through the nasal mucosa, or drain into the gastrointestinal tract.[61,75] Medications that are absorbed across the conjunctivae may achieve systemic concentrations comparable to those following intravenous administration.[78] Absorbed eye drops are less diluted in children's bloodstreams compared to an adult's, due to the smaller circulatory volume. Conversely, tear flow will dilute drops more quickly than ointment.[61] Suggestions to minimize systemic absorption of eye drops include punctal occlusion at the time of administration, followed by blotting to remove any excess medication. If the eyelids are closed at the time of administration, there will be less flow through the nasolacrimal duct.

Mode of Therapy. Uncomplicated bacterial conjunctivitis resolves with or without treatment within 7-10 days.[1] The duration of viral conjunctivitis is not changed by therapy. An argument could be made for not treating acute conjunctivitis for these reasons. Increasing drug resistance, especially to quinolones, offers another argument for not treating uncomplicated cases with antibiotics.[10] It has been shown, however, that antibiotic treatment of bacterial conjunctivitis shortens the duration of clinical symptoms, and brings on bacteriologic cure within 3-5 days. For this reason, most practitioners treat acute conjunctivitis with antibiotics. Debate persists about the optimal duration and mode of therapy. Many recommend topical therapy,[2,13,28] perhaps using eyedrops during the day and ointment at bedtime. Others recommend a full 10-day course of oral antibiotics in young children with conjunctivitis, especially if they are at high risk for otitis media.[13] Abbreviated oral therapy also has been proposed. One small study showed that patients treated with three days of oral antibiotics had clinical cure of their conjunctivitis, and none developed subsequent otitis. Patients treated with seven days of topical therapy were cured 90% of the time, but 40% had developed otitis media at the time of reevaluation.[79] Broad spectrum systemic therapy targeting *H.*

Table 4. Indications for Ophthalmology Consultation/Referral

- Neonatal gonococcal conjunctivitis
- Conjunctivitis in older infant, child, or adolescent with:
  - *N. gonorrhea* or *N. meningiditis*
  - *Pseudomonas aeruginosa*
- Persistent symptoms after 7-10 days
- Unexplained eye pain
- Herpetic keratitis
- Visual changes
- Conjunctivitis in contact lens wearer

influenzae and *S. pneumoniae* is a reasonable therapeutic option in younger children without obvious viral etiology. This age group has a higher risk for bacterial conjunctivitis and associated otitis media, and many parents find oral medicines easier to administer.[1] The cost of appropriate oral medications generally is equivalent to, or less than, the cost of newer, broad-coverage ophthalmic agents like fluoroquinolones.

Returning to School or Daycare. Most schools have policies preventing children with conjunctivitis from returning to school. Time away from school or daycare translates into time away from work for parents, or the need to make alternate arrangements for child care. Recommendations in the literature are conflicting. Studies of *H. influenzae* conjunctivitis outbreaks in daycare facilities have demonstrated different strains in different children—suggesting a low risk of contagion, and arguing against isolation. Conjunctival cultures match nasopharyngeal cultures in individual children, suggesting retrograde migration via a child's own lacrimal duct, rather than spread from child to child.[80,81] These findings are inconsistent with the reports that conjunctivitis-otitis syndrome, also caused by *H. influenzae*, has an increased incidence among siblings and playmates of affected individuals—suggesting spread between children.[7] Viral conjunctivitis is more contagious than bacterial. Twenty percent of all acute conjunctivitis is caused by adenovirus, which is highly contagious. It has been recommended that patients with adenoviral infections be kept home for a week.[2,36] Replicating virus is present in 95% of patients with adenovirus conjunctivitis on day 10 of the illness. This drops to 5% by day 16. Conservatively, these patients should avoid close contact and sharing of contaminated objects for two weeks following the onset of symptoms.[46] Further complicating things, fomites may remain infected with adenovirus for up to two months.[36] The group of viruses that cause epidemics of acute hemorrhagic conjunctivitis includes picornavirus, enterovirus, and coxsackie virus, in addition to adenovirus. These are highly infectious.[3] While the age of the child and clinical presentation can be helpful in distinguishing etiology and thus need for isolation, distinguishing the cause in the individual patient is not always easy. It has been pointed out that the same organisms that cause conjunctivitis also cause upper respiratory illnesses and otitis media, which are not reasons to exclude children from school.[81]

Disposition

The majority of patients with acute conjunctivitis safely can be started on topical or oral therapy and discharged with routine follow-up. There are several notable exceptions to this. Infants with Chlamydia conjunctivitis should be retested after treatment with erythromycin, which has a 20-30% failure rate.[8] Patients treated with topical agents who develop subsequent ear pain should be reassessed for the presence of otitis media. Those who present initially with conjunctivitis-otitis media syndrome should be re-evaluated to document resolution of the otitis, given the high rates of treatment failure. When allergic conjunctivitis is suspected, follow up should be arranged to monitor preventative therapy.

Several disease processes warrant immediate ophthalmologic consultation, due to severe or long-term consequences. These include *N. gonorrhea, P. aeruginosa, N. meningitidis*, HSV, and VZV. Conjunctivitis that persists for more than one week, and redness associated with pain or visual loss, also warrant rapid referral. *An ophthalmologist should be involved in all decisions to initiate therapy with preparations containing steroids. (See Table 4.)*

While outpatient therapy generally is adequate, some organisms which cause conjunctivitis also may cause systemic disease. Examples are *N. gonorrhea, N. meningitides, P. aeruginosa*, group B streptococcus in neonates, and HSV. When infection with these agents is suspected or confirmed, a sepsis evaluation should be performed, and parenteral therapy initiated. When conjunctivitis is associated with clinical signs of Kawasaki disease, inpatient admission is warranted.

Summary

Conjunctivitis is a common ocular complaint in children who present to the ED. Etiologies include infection, allergy, trauma, and systemic inflammatory conditions. The prevalence of different entities varies with age, season, and exposure to infectious agents, allergens, and irritants. While most conjunctivitis is self-limited and resolves spontaneously, specific therapy can reduce the duration of symptoms in bacterial or allergic cases. Recognizing patterns of presentation for these common entities can help to direct specific management. A wide variety of topical and systemic agents are available to treat both conditions, and vary in both cost and spectrum of activity.

While complications from conjunctivitis are rare, several disease entities are associated with significant morbidity and mortality. The practitioner should learn to recognize conditions associated with long-term visual complications, institute prompt therapy, and referral to ophthalmology.

References

1. Gigliotti F. Acute conjunctivitis. *Pediatr Rev* 1995;16:203-208.
2. Weiss A. Acute conjunctivitis in childhood. *Curr Probl Pediatr* 1994;14:4-11.
3. Teoh DL, Reynolds S. Diagnosis and management of pediatric conjunctivitis. *Pediatr Emerg Care* 2003;19:48-55.
4. Fransen L, Van der Berghe P, Mertens A, et al. Incidence and bacterial aetiology of neonatal conjunctivitis. *Eur J Pediatr* 1987;146:152-155.
5. Sandstrom KI, Bell TA, Chandler JW, et al. Microbial causes of neonatal conjunctivitis. *J Pediatr* 1984;105:706-711.
6. O'Hara M. Ophthalmia neonatorum. *Pediatr Clin North Am* 1993;40:715-725.
7. Krohn MA, Hillier SL, Bell TA, et al. The bacterial etiology of conjunctivitis in early infancy. *Am J Epidemiol* 1993;138:326-332.
8. American Academy of Pediatrics. Chlamydial infections. In: Pickering LK, ed. *Red Book: 2003 Report of the Committee on Infectious Diseases.* 26th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003:235-243.
9. Chen JY. Prophylaxis of ophthalmia neonatorum: Comparison of silver nitrate,

tetracycline, erythromycin and no prophylaxis. *Pediatr Infect Dis J* 1992;11: 1026-1030.
10. Chou TM, Dorfman MS, Wagner RS. Bacterial conjunctivitis: Staying a step ahead of the bugs. *Contemp Pediatr* 2003;20:2-7.
11. Harrison CJ, Hedrick JA, Block SL, et al. Relation of the outcome of conjunctivitis and the conjunctivitis-otitis syndrome to identifiable risk factors and oral antimicrobial therapy. *Pediatr Infect Dis J* 1987;6:536-540.
12. Martin M, Turco JH, Zegans ME, et al. An outbreak of conjunctivitis due to atypical *Streptococcus pneumoniae*. *N Engl J Med* 2003;348:1112-1121.
13. Wald ER. Conjunctivitis in infants and children. *Pediatr Infect Dis J* 1997; 16:S17-S20.
14. Verin P, Easty D, Secchi A, et al. Clinical evaluation of twice-daily emedastine 0.05% eye drops (emadine eye drops) versus levocabastine 0.05% eye drops in patients with allergic conjunctivitis. *Am J Ophthalmol* 2001;131:691-698.
15. Juniper EF, Howland WC, Roberts NB, et al. Measuring quality of life in children with rhinoconjunctivitis. *J Allergy Clin Immunol* 1998;101:163-170.
16. Jackson WB. Differentiating conjunctivitis of diverse origins. *Surv Ophthalmol* 1993;38:S91-S104.
17. Weiss A, Brinser JH, Nazar-Stewart V. Acute conjunctivitis in childhood. *J Pediatr* 1993;122:10-14.
18. Pöschl JM, Hellstern G, Ruef P, et al. Ophthalmia neonatorum caused by group B streptococcus. *Scand J Infect Dis* 2002;34:921-922.
19. Datner EM, Jolly BT. Pediatric ophthalmology. *Emerg Med Clin North Am* 1995;13:669-679.
20. Block SL, Hedrick J, Tyler R, et al. Increasing bacterial resistance in pediatric acute conjunctivitis (1997-1998). *Antimicrob Agents Chemother* 2000;4: 1650-1654.
21. Colin J, Prisant O, Cochener B, et al. Comparison of the efficacy and safety of valaciclovir and acyclovir for the treatment of herpes zoster ophthalmicus. *Am Acad Ophthalmol* 2000;107:1507-1511.
22. Brook I. Anaerobic and aerobic bacterial flora of acute conjunctivitis in children. *Arch Ophthalmol* 1980;98:833-835.
23. Smith LB, Newburger JW, Burns JC. Kawasaki syndrome and the eye. *Pediatr Infect Dis J* 1989;8:116-118.
24. Numazaki K, Wainberg MA, McDonald J. Chlamydia trachomatis infections in infants. *CMAJ* 1989;140:615-622.
25. Doraiswamy B, Hammerschlag M, Pringle G, et al. Ophthalmia neonatorum caused by ß-lactamase-producing *N. gonorrhoeae*. *JAMA* 1983;250:790-791.
26. Gigliotti F, Williams W, Hayden F, et al. Etiology of acute conjunctivitis in children. *Pediatrics* 1981;98:531-536.
27. Perkins RE, Kundsin RB, Pratt MV, et al. Bacteriology of normal and infected conjunctiva. *J Clin Microbiol* 1975;1:147-149.
28. Lohr JA. Treatment of conjunctivitis in infants and children. *Pediatr Ann* 1993; 22:359-364.
29. Pellerano RA, Bishop V, Silber TJ. Gonococcal conjunctivitis in adolescents, recognition and management. *Clin Pediatr* 1994;33:114-116.
30. Lewis LS, Glauser TA, Joffe MD. Gonococcal conjunctivitis in prepubertal children. *AJDC* 1990;144:546-548.
31. Alfonso E, Friedland B, Hupp S, et al. *Neisseria gonorrhoeae* conjunctivitis: An outbreak during an epidemic of acute hemorrhagic conjunctivitis. *JAMA* 1983;250:794-795.
32. Bodor FF, Marchant CD, Shurin P, et al. Bacterial etiology of conjunctivitis-otitis media syndrome. *Pediatrics* 1985;76:26-28.
33. Bodor FF. System antibiotics for treatment of conjunctivitis-otitis media syndrome. *Pediatr Infect Dis J* 1989;8:287-290.
34. Hara JH. The red eye. *Am Fam Phys* 1996;54:2423-2430.
35. American Academy of Pediatrics. Herpes simplex virus infections. In: Pickering L, ed. *Red Book: 2003 Report of the Committee on Infectious Diseases*. 26th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003:344-353.
36. Steinkuller PG, Edmond JC, Chen RM. Ocular infections. In: Feigin RD, Cherry JD, eds. *Textbook of Pediatric Infectious Diseases*, 4th ed. Philadelphia: WB Saunders; 1998:786-806.
37. Friedlaender M. Conjunctivitis of allergic origin: Clinical presentation and different diagnosis. *Surv Ophthalmol* 1993;38:S105-S114.
38. Soparkar CN, Wilhelmus KR, Koch DD, et al. Acute and chronic conjunctivitis due to over-the-counter ophthalmic decongestants. *Arch Ophthalmol* 1997;115: 34-38.
39. Bartunková J, Šeivá A, Vencovský J, et al. Primary Sjögren's syndrome in children and adolescents: Proposal for diagnostic criteria. *Clin Exp Rheumatol* 1999;17:381-386.
40. American Academy of Pediatrics. *Haemophilus influenzae* infections. In: Pickering L, ed. *Red Book: 2003 Report of the Committee on Infectious Diseases.* 26th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003:293-301.
41. Folland DS, Burke RE, Hinman AR, et al. Gonorrhea in preadolescent children: An inquiry into source of infection and mode of transmission. *Pediatrics* 1997;60:153-156.
42. Shore WB, Winkelstein JA. Nonvenereal transmission of gonococcal infections to children. *Pediatrics* 1971;79:661-663.
43. Cooperman MB. Gonococcus arthritis in infancy. *Am J Dis Child* 1927;33:932.
44. Ullman S, Roussel TJ, Culbertson WW, et al. *Neisseria gonorrhoeae* keratoconjunctivits. *Ophthalmol* 1987;94:525-531.
45. Ballas Z, Blumenthal M, Tinkelman DG, et al. Clinical evaluation of ketorolac tromethamine 0.5% ophthalmic solution for the treatment of seasonal allergic conjunctivitis. *Surv Ophthalmol* 1993;38:S141-S148.
46. Leibowitz HM. The red eye. *N Engl J Med* 2000;343:345-351.
47. Levin AV. Eye-Red. In: Fleisher GR, Ludwig S, eds. *The Textbook of Pediatric Emergency Medicine*, 4th ed. Philadelphia: Lippincott Williams and Wilkins, 2000:231-235.
48. Schwab IR, Crawford JB. Conjunctiva. In: Vaughan D, Asbury T, Riordan-Eva P, eds. *General Ophthalmology*, 15th ed. New York: McGraw-Hill 1999:1.
49. Levin, AV. Ophthalmic emergencies. In: Fleisher GR, Ludwig S, eds. *The Textbook of Pediatric Emergency Medicine,* 4th ed. Philadelphia: Lippincott Williams and Wilkins; 2000:1561-1568.
50. Olitsky SE, Nelson LB. Disorders of the conjunctiva. In: Behrman RE, Kliegman RM, Jenson HB, eds. *Nelson Textbook of Pediatrics,* 16th ed. Philadelphia: WB Saunders, 1998;786-806.
51. Baker R. Conjunctivitis and hordeolum. In: Baker R, ed. *Pediatric Primary Care: Ill-Child Care.* Philadelphia: Lippincott Williams and Wilkins, 2001;53-56.
52. 1998 Guidelines for Treatment of Sexually Transmitted Diseases. Centers for Disease Control and Prevention. *MMWR Recomm Rep* 1998;47(RR-1):1-11.
53. Ruttum MS, Ogawa G. Adenovirus conjunctivitis mimics preseptal and orbital cellulitis in young children. *Pediatr Infect Dis J* 1996;15:266-267.
54. Uchio E, Takeuchi S, Itoh N, et al. Clinical and epidemiological features of acute follicular conjunctivitis with special reference to that caused by herpes simplex virus type 1. *Br J Ophthalmol* 2000;84:968-972.
55. O'Hara M. Ophthalmia neonatorum. *Pediatr Clin North Am* 1993;22:353-356.
56. Hammerschlag M, Roblin P, Gelling M, et al. Use of polymerase chain reaction for the detection of chlamydia trachomatis in ocular and nasopharyngeal specimens from infants with conjunctivitis. *Pediatr Infect Dis J* 1997;16:293-297.
57. Gigliotti F, Hendley O, Morgan J, et al. Efficacy of topical antibiotic therapy in acute conjunctivitis in children. *Pediatrics* 1984;104:623-626.
58. Groothuis JR, Thompson J, Wright PF. Correlation of nasopharyngeal and conjunctival cultures and middle ear fluid cultures in otitis media: A prospective study. *Clin Pediatr* 1986;25:85-88.
59. Burns JC, Mason WH, Glade MP, et al. Clinical and epidemiologic characteristics of patients referred for evaluation of possible Kawasaki disease. *Pediatrics* 1991;118:680-686.
60. Chan DQ. Neurologic, ophthalmic, and neuropsychiatric manifestations of pediatric systemic lupus erythematosus. *Optometry and Vision Science* 2000;77:388-394.
61. Wallace DK, Steinkuller PG. Ocular medications in children. *Clin Pediatr* 1998;37:645-652.
62. Smith SE. Eyedrop instillation for reluctant children. *Br J Opthalmol* 1991; 75:480-481.
63. Hammerschlag MR, Cummings C, Roblin PM, et al. Efficacy of neonatal ocular prophylaxis for the prevention of chlamydial and gonococcal conjunctivitis. *N Engl J Med* 1989;320:769-772.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

64. Hammerschlag M, Chandler J, Alexander R, et al. Longitudinal studies on chlamydial infections in the first year. *Pediatr Infect Dis J* 1982;1:395-400.
65. Hammerschlag MR, Gelling M, Roblin PM, et al. Treatment of neonatal chlamydial conjunctivitis with azithromycin. *Pediatr Infect Dis J* 1998;17: 1049-1050.
66. Stenberg K, Mardh PA. Treatment of chlamydial conjunctivitis in newborns and adults with erythromycin and roxithromycin. *J Antimicrob Chemother* 1991;28:301-307.
67. Schiebel NE. Use of antibiotics in patients with acute bacterial conjunctivitis. *Ann Emerg Med* 2003;41:407-409.
68. Osato M, Jensen HG, Trousdale MD, et al. The comparative in vitro activity of ofloxacin and selected ophthalmic antimicrobial agents against ocular bacterial isolates. *Am J Ophthalmol* 1989;108:380-386.
69. Richman J, Zolezio H, Tang-Liu D. Comparison of ofloxacin, gentamicin, and tobramycin concentrations in tears and in vitro MICs for 90% of test organisms. *Antimicrob Agents Chemother* 1990;34:1602-1604.
70. Gross R, Hoffman R, Lindsay R. A comparison of ciprofloxacin and tobramycin in bacterial conjunctivitis in children. *Clin Pediatr* 1997;36:435-444.
71. Shiuey Y, Ambati BK, Adamis AP, et al. A randomized, double-masked trial of topical ketorolac versus artificial tears for treatment of vial conjunctivitis. *Amer Acad Ophthalmol* 2000;107;1512-1516.
72. Romanowski EG, Roba LA, Wiley L, et al. The effect of corticosteroids on adenoviral replication. *Arch Ophthalmol* 1996;114:581-585.
73. Tinkelman DG, Rupp G, Kaufman H, et al. Double-masked, paired-comparison clinical study of ketorolac tromethamine 0.5% ophthalmic solution compared with placebo eyedrops in the treatment of seasonal allergic conjunctivitis. *Surv Ophthalmol* 1993;38:S133-S140.
74. American Academy of Pediatrics. Meningococcal infections (*Neisseria meningitides*). In: Pickering LK, ed. *Red Book: 2003 Report of the Committee on Infectious Diseases*. 26th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003:430-436.
75. Palmer EA. How safe are ocular drugs in pediatrics? *Ophthalmol* 1986;93: 1038-1040.
76. American Academy of Ophthalmology. Conjunctivitis. Preferred practice patterns. San Francisco, CA: American Academy of Ophthalmology, 1998.
77. Ohji M, Kinoshita S, Ohmi E, et al. Marked intraocular pressure response to instillation of corticosteroids in children. *Am J Ophthalmol* 1991;112:450-454.
78. Shell JW. Pharmacokinetics of topically applied ophthalmic drugs. *Surv Ophthalmol* 1982; 26:207-218.
79. Wald ER, Serdy C, Guerra N, et al. Short course: Oral antibiotic treatment of bacterial conjunctivitis. [Abstract 727] *Pediatr Res* 1993;33:124A.
80. Trottier S, Sternberg K, Anderson von Rosen I, et al. *Haemophilus influenzae* causing conjunctivitis in day-care children. *Pediatr Infect Dis J* 1991;10:578-584.
81. Gigliotti F. Management of the child with conjunctivitis. *Pediatr Infect Dis J* 1994;12:1161-1162.

Physician CME Questions

81. Which of the following is most consistent with bacterial conjunctivitis?
    A. A 10-year-old asthmatic presents with one week of rhinorrhea and red, itchy eyes during the spring.
    B. A 6-year-old presents in October with three days of right-sided eye redness, nasal congestion, and rhinorrhea.
    C. A 4-year-old presents with one day of left ear pain during the winter. His left eye is red, and was stuck shut upon awakening.
    D. A 7-year-old presents with severe unilateral eye pain, redness, and mild photophobia in August.

82. In children younger than 6 years of age, bacterial conjunctivitis is more common than viral conjunctivitis.
    A. True
    B. False

83. The advent of the Hib vaccine has *not* changed the incidence of bacterial conjunctivitis.
    A. True
    B. False

84. The most frequent cause of neonatal conjunctivitis is:
    A. *C. trachomatis.*
    B. *N. gonorrhea.*
    C. chemical irritation.
    D. *S. viridans.*

85. Ocular prophylaxis has a very high failure rate for elimination of *N. gonorrhea.*
    A. True
    B. False

86. Which of the following is *not* true regarding allergic conjunctivitis?
    A. The incidence in children is about 65%.
    B. It is most common in late childhood and adults.
    C. It may result from exposure to airborne allergens.
    D. It may result from smoke exposure.

87. Prolonged use of vasoconstrictive eye drops has been associated with:
    A. conjunctival hyperemia.
    B. follicular conjunctivitis
    C. eczematoid blepharoconjunctivitis.
    D. All of the above

88. Which of the following is true regarding *N. gonorrhea* conjunctivitis in the newborn?
    A. Symptoms typically begin in the first week of life.
    B. Neonates typically have copious, thick, purulent secretions.
    C. Complications include the development of corneal ulcerations and perforations.
    D. All of the above

89. Which of the following is *not* typical for bacterial conjunctivitis?
    A. The child is usually younger than 6 years of age.
    B. The child usually has a purulent discharge and severe conjunctival injection.
    C. Unilateral eye involvement is more common than bilateral eye involvement.
    D. Patients may have a "gritty" sensation in their eye.

90. Which of the following is characteristic of a patient with HSV keratitis?
    A. Patients usually have the acute onset of symptoms and severe eye pain.
    B. Patients typically have a clear discharge.
    C. On fluorescein staining, a finding of dendritic lesions of the cornea is pathognomonic.
    D. All of the above

Answer key:

81. C	86. A
82. A	87. D
83. A	88. D
84. C	89. C
85. B	90. D

In Future Issues: **Status Epilepticus**

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Conjunctivitis

Neonatal Conjunctivitis

ETIOLOGY	USUAL AGE AT ONSET	MANAGEMENT	COMPLICATIONS	PREVENTION
Chemical	< 24 hours	Observe	None, self-limited	Avoid use of silver nitrate
Neisseria gonorrhea	2-5 days	Admit, IV antibiotics, irrigation	Corneal ulceration/ perforation, blindness	STD screening and treatment during pregnancy
Chlamydia trachomatis	5-14 days	PO erythromycin	Blindness (rare)	STD screening and treatment during pregnancy
Herpes simplex virus type 2	Birth to 4 weeks	IV acyclovir and topical antivirals (e.g., trifluridine)	CNS involvement, disseminated disease	Screening during pregnancy; caesarian section for active maternal infection; avoid infected household contacts

Key: PO = oral; CNS = central nervous system;
STD = sexually transmitted disease; IV = intravenous

Distinguishing Features of Various Forms of Conjunctivitis

	BACTERIAL	VIRAL	ALLERGIC
Age	< 6 years	> 6 years	Late childhood to early adulthood
Discharge type	Purulent	Watery/serous	Mucoid
Conjunctival injection	Severe	Moderate	Mild
Unilateral/bilateral	Bilateral > unilateral	Unilateral > bilateral	Bilateral > unilateral
Associated signs/symptoms	Gritty sensation	Sick contact with red eyes	Itching, chemosis
Associated conditions	Otitis media	Upper respiratory infection/ pharyngitis (adenovirus)	Allergic rhinitis, asthma, eczema
Season	Winter	Fall	Spring/summer
Preauricular adenopathy	Uncommon	Common	Not present

Differential Diagnosis of Red Eye*

OCULAR CAUSES

- Infectious
  - bacterial, viral, chlamydial, amoebal
  - keratitis conjunctivitis, episcleritis
  - uveitis, iritis, endophthalmitis
  - periorbital cellulitis, blepharitis, hordeolum, chalazion, trichiasis
  - nasolacrimal duct obstruction, dacrocystitis
- Traumatic (foreign body, abrasion, iritis, globe injury)
- Allergic
- Chemical, irritative
- Other: Glaucoma, dry eye, contact lens

SYSTEMIC DISEASES

- Infectious disease (varicella, mumps, measles)
- Atopic dermatitis
- Kawasaki disease
- Collagen vascular disease
- Juvenile rheumatoid arthritis
- Inflammatory bowel disease
- Lyme disease
- Leptospirosis
- Stevens-Johnson syndrome

* List not inclusive

Indications for Ophthalmology Consultation/Referral

- Neonatal gonococcal conjunctivitis
- Conjunctivitis in older infant, child, or adolescent with:
  - *N. gonorrhea* or *N. meningiditis*
  - *Pseudomonas aeruginosa*
- Persistent symptoms after 7-10 days
- Unexplained eye pain
- Herpetic keratitis
- Visual changes
- Conjunctivitis in contact lens wearer

HSV Keratitis: Summary of Findings

- Acute onset
- Severe eye pain
- Clear discharge/tearing
- Unilateral
- Erythema worse in perilimbal region
- Lid swelling
- Tender preauricular node
- Dendritic pattern on fluorescein staining
- Prior or active herpetic skin lesions

Selected Ophthalmologic Agents

GENERIC NAME	BRAND NAME	TYPE OF AGENT	DOSAGE AND LOWER AGE LIMIT*	COST OF TREATMENT**
Ciprofloxacin 0.3%	Ciloxan	Antibiotic	≥1yr 1-2 gtt q2h while awake for 2 days, then 1-2 gtt q4hr while awake for next 5 days	$$$$$
Ofloxacin 0.3%	Ocuflox	Antibiotic	≥1yr 1-2 gtt q2-4hr for 2 days then qid for 5 more days	$$$$$
Gentamicin 0.3%	Gentacidin	Antibiotic	Mild-mod infection: 1-2 gtt q4hr Severe infection: up to 2 gtt q1hr initially	$$ (generic)
Tobramycin 0.3%	Tobrex	Antibiotic	Mild-mod infection: 1-2 gtt q4hr Severe infection: 2 gtt q 1hr initially	$-$$ (generic)
Trimethoprim + polymyxin B sulfate	Polytrim	Antibiotic	≥ 2 mos 1 gtt q3hr for 7-10 days	$$$$
Levofloxacin 0.5%	Quixin	Antibiotic	≥ 1 yr: 1-2 gtt q2hr while awake (max 8 times/day) on days 1 and 2, then 1-2 gtt q4hr while awake on days 3-7 (max 4 times/day)	$$$$$
Ketoralac 0.5%	Acular	NSAID	≥ 12 yrs: 1 gtt qid	$$$$$
Pemirolast 0.1%	Alamast	Mast cell stabilizer	≥ 3 yrs: 1-2 gtt qid	$$$$$
Nedocromil 2%	Alocril	Mast cell stabilizer	≥ 3 yrs: 1-2 gtt bid at regular intervals	$$$$$
Lodoxamide 0.1%	Alomide	Mast cell stabilizer	≥ 2 yrs: 1-2 gtt qid for up to 3 months	$$$$$$$
Cromolyn 4%	Crolom	Mast cell stabilizer	≥ 4 yrs: 1-2 gtt 4-6 times/day at regular intervals	$$$$$
Emedastine 0.05%	Emadine	Antihistamine	≥ 3 yrs: 1 gtt up to 4 times/day	$$$$$
Olopatadine 0.1%	Patanol	Antihistamine, mast cell stabilizer	≥ 3 yrs: 1 gtt twice daily	$$$$$$$
Azelastine 0.05%	Optivar	Antihistamine, mast cell stabilizer	≥ 3 yrs: 1 gtt in affected eye bid	$$$$$$
Ketotifen 0.025%	Zaditor	Antihistamine, mast cell stabilizer	≥ 3 yrs: 1 gtt in affected eye q 8-12 hours	$$$$$
Naphazoline 0.025%, pheniramine 0.3%	Naphcon A	Vasoconstrictor, antihistamine	≥ 6 yrs: 1-2 gtt up to 4 times/day	$-$$
Antazoline 0.5%, naphazoline 0.05%	Vasocon-A	Vasoconstrictor	≥ 6 yrs: 1-2 gtt up to 4 times/day	$$-$$$$$

KEY: gtt = drop(s); NSAID = nonsteroidal anti-inflammatory drug; $$ = $10-20; $$$ = $20-30; $$$$ = $30-40; $$$$$ = $40-50; $$$$$$ = $50-60; $$$$$$$ = $60-70

* Not recommended for patients younger than age listed.

** Price ranges provided solely for relative comparison. Prices obtained by authors from retail pharmacies July 2003.

Adapted from Prescribing Reference Inc. *Prescribing Reference for Pediatricians*, Spring/Summer 2003.

Supplement to *Pediatric Emergency Medicine Reports*, September 2003: "A Clear-Sighted Approach to Conjunctivitis: State of the Art Clinical Practice." *Authors:* **Linda Arnold, MD,** Assistant Professor of Pediatrics; Section of Pediatric Emergency Medicine, Yale-New Haven Children's Hospital, New Haven, CT; **Walter J. Eppich, MD,** Clinical Fellow, Section of Pediatric Emergency Medicine, Yale-New Haven Children's Hospital, New Haven, CT.

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Non-accidental Injury: Recognizing Child Abuse in the Pediatric Trauma Patient

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The recognition of non-accidental injury is critical for a pediatric trauma patient. In the year 2000, almost 3 million reports of child abuse were made to social service agencies.[1] More importantly, 1200 children died from neglect or abuse in that same year.[1] Forty-four percent of the fatalities were children younger than 1 year of age.[1] Not only are these statistics alarming, but they point out the need for emergency department (ED) and trauma physicians and nurses to recognize non-accidental injury and aggressively protect the children who seek our medical expertise and protection.

—The Editor

Child abuse and neglect are not uncommon, and clinicians who provide trauma care to children will encounter from several to many cases during a career. Child abuse is defined by federal legislation that provides minimum guidelines for states to incorporate into their criminal and civil statues. Subsequently, each state in the United States has a working legal definition of child abuse and neglect.[2-5]

There are four basic types of child maltreatment—physical abuse, sexual abuse, emotional abuse, and child neglect.[5] Child neglect is the most common form of child maltreatment investigated and is the failure to provide for a child's basic needs. This can include physical, emotional, medical, supervisional, and educational neglect. Each state has different definitions that help determine if a given situation meets the standard for neglect. Child Protective Services (CPS) is the branch of public social services that investigates reports of child abuse and neglect in which caretakers are involved as possible perpetrators. Law enforcement is involved in many of these cases and also has primary responsibility for investigating cases in which strangers and noncaretakers are the alleged perpetrators. CPS has to investigate any reported child maltreatment and then provide support services to the family if indicated.

Physical abuse and sexual abuse probably are the most common types of child maltreatment seen in ED settings. The physicians and nurses in these situations have the responsibility of recognizing clues to abuse or neglect and reporting them to appropriate agencies.

A medical provider must be very familiar with his or her state's reporting laws covering child maltreatment. All states list health care workers as mandated reporters of child maltreatment. To report a case to CPS, the provider need only have a suspicion that maltreatment or neglect has occurred, not proof.[2] CPS is

given the job of investigating any report when the agency believes the report meets a minimal standard of likelihood. All states mandate reporting without risk of liability if the report is made without malicious intent.[2] A medical provider can incur liability for failing to report a case of suspected child maltreatment. It is important to emphasize to parents (or other caretakers) that reporting is not an accusation, but is a legal requirement based on the assessment to that point.

Important factors to assess in possible abuse or neglect situations are the history, the child's developmental abilities, physical examination findings, and the presence of other risk factors for abuse and neglect. These risk factors include, but are not limited to, young maternal age (< 15 years), prior social service investigations, prior law enforcement involvement, substance abuse in the family, domestic violence, low socioeconomic status, and disability or prematurity of the child.[4,6,7] It also is important to remember that the absence of risk factors does not rule out abuse, and is not a reason to fail to report if suspect findings are present.

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The intended audience for this publication is emergency, family, osteopathic, and general practice physicians and nurses who have contact with trauma patients.

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The abused child presents a unique set of epidemiologic, physiologic, and social circumstances.[9,10] The clinical history often is difficult to obtain, and deliberately may be obfuscatory, false, or contradicted by physical findings or known patterns of injury. The presentation frequently is delayed, allowing many mechanisms of secondary injury to become well established. Abused children, especially infants, often present with nonspecific complaints and findings.[11-13] These children frequently present with respiratory distress or apnea, but vomiting, mental status changes (lethargy or irritability), poor feeding, or nonspecific behavioral changes also are common.[11,14] Seizures are reported in up to 70 % of patients.[11] Many abused children are repetitively traumatized; thus, the presenting incident may simply be the worst (or most recent) of several episodes.[15,16]

An unsuspecting physician may accept a false history of trauma and not consider the possibility of abuse. To identify abused children, child abuse needs to be included on the differential list of all pediatric injuries.[4] In most instances, child maltreatment quickly will be eliminated from the differential, but a small dose of suspicion is needed to identify abused children.

The diagnosis of child abuse is made like all others—a careful history, complete physical examination, and supporting laboratory and radiographic data lead to a working diagnosis. In some cases, the physical examination alone indicates abuse, but in most cases, the comparison of the history of trauma and the resulting injuries suggests the diagnosis. The following are general indicators of possible child abuse:[4]

- Infants and children with unexplained injuries;
- The history provided does not adequately explain the injuries;
- The history provided changes with time;
- The history provided does not correlate with the child's developmental abilities; or
- There is an unexplained delay in seeking medical care.

Clinically, child abuse may be obvious or subtle. A child with external signs of trauma, known as a "battered" child, should be treated the same as any trauma victim.[12,13,17] Abused children with less obvious presentations often have injuries falling into certain patterns. Retinal hemorrhages are common after head injury from child abuse, and need to be documented and taken into consideration with other clinical and radiographic findings.[11,18,19] Retinal hemorrhages are not all the same and should be described thoroughly, including amount, type, distribution, and side affected (bilateral, unilateral, or asymmetric).

Suspicion of nonaccidental trauma should prompt immediate investigation. Social work, CPS, and any appropriate social or legal authorities should be involved as soon as possible.

History and Physical Examination

The initial history often is the first clue to the detection of child abuse. Obtain a detailed history, including location, time, and mechanism of any injury described. The identification of significant injury unaccompanied by a history of trauma is cause for concern. If the history does not seem to explain the injury identi-

fied, the possibility of abuse should be considered. Parents who provide a false history of trauma to explain a child's abusive injuries most commonly offer simple household trauma as an explanation.[12] For example, infants or toddlers with abusive head injury often are said to have fallen off a bed or couch.

It is important to document the events from the time of the injury leading to the medical visit. Some parents, in an effort to explain inflicted injuries, provide various histories to explain an injury, or change the story once additional injures are identified. A comparison of histories obtained from various health care providers, pre-hospital personnel, emergency physicians, hospitalists, or intensivists may reveal inconsistencies. Also, compare histories obtained from adult caregivers present at the time of injury.

An unexplained delay in seeking medical care should be recorded and explored. In an effort to prevent detection of abuse by professionals, parents occasionally will keep their injured child at home, despite the obvious need for medical intervention.

The physical examination of the child must be complete. Examine all surfaces of the child's skin carefully, and document any injuries. In infants, subtle external injuries are often a clue to more serious internal injury, and should not be dismissed. Bruises, burns, and scars should be measured, and their size, shape, location, and color recorded. Photographs are an important adjunct to the recorded physical examination, but are not an appropriate substitute for accurate medical documentation.

Craniofacial, head, face, and neck injuries occur in more than half of the cases of child abuse.[3] Careful intraoral and perioral examination is necessary in all cases of suspected abuse. Some authorities believe that the oral cavity may be a central focus for physical abuse because of its significance in communication and nutrition.[3] The injuries most commonly are inflicted by blunt trauma with an instrument, eating utensils, hands, or fingers or by scalding liquids or caustic substances.[3,20] The abuse may result in contusions; lacerations of the tongue, buccal mucosa, palate, gingiva alveolar mucosa, or frenum; fractured, displaced, or avulsed teeth; facial bone and jaw fractures; burns; or other injuries. These injuries, including a lacerated frenum, also can result from unintentional trauma. Gags applied to the mouth may leave bruises, lichenification, or scarring at the corners of the mouth.

Laboratory and Radiographic Data

Abused children often have occult injuries, and some medical diseases may mimic abuse. Therefore, the laboratory and radiographic evaluation of the abused child is guided not only by the history and physical examination findings, but also by the above considerations. Necessary laboratory and/or radiographic testing varies by age, injury pattern, and severity. The following studies may be appropriate in the evaluation of an abused child.

Laboratory Studies. *Hematologic Evaluation.* A complete blood count (CBC) with platelet count, prothrombin time (PT), and partial prothrombin time (PTT) are indicated for children who present with bleeding or bruising. Elevations of PT and PTT may be the result of severe inflicted head injury.[21]

Liver Function Tests/Pancreatic Enzymes. Alanine aminotransferase (ALT), aspirate aminotransferase (AST), amylase, and/or lipase may be elevated with acute liver or pancreatic injury. Such injuries can be asymptomatic, and screening is recommended for injured infants and children in whom the abdominal examination may not be a sensitive indicator of injury.

Urinalysis. The urinalysis is used as a screen for renal or bladder trauma, and can detect myoglobinuria secondary to rhabdomyolysis from severe beatings.

Toxicology screens are indicated for infants and children with unexplained neurological symptoms.

Diagnostic Imaging. Imaging studies often are critical in the assessment of the infant and young child with evidence of physical injury, and they also may be the first indication of abuse in a child who is seen with an apparent natural illness.[4,22,23] When viewed in conjunction with clinical and laboratory studies, imaging findings commonly provide support for allegations of abuse. For severely abused infants, the imaging findings alone may form the basis for a diagnosis of inflicted injury.[22] The role of imaging in cases of suspected abuse is not only to identify the extent of physical injury when abuse has occurred, but to elucidate all imaging findings that may point to alternative diagnoses. Because most conventional imaging studies performed in these settings are noninvasive and entail minimal radiation risks, recommendations about imaging should focus on examinations that provide the highest diagnostic yield at acceptable cost.[22]

Roentgenographic Skeletal Survey. The skeletal survey is an important adjunct to the evaluation of abused infants and toddlers, and is indicated for all children younger than 2 years with any suspicious injury.[4] Guidelines for the appropriate imaging methods have recently been updated by the American Academy of Pediatrics.[22]

Although skeletal injuries rarely pose a threat to the life of the abused child, they often are the strongest radiologic indicators of abuse. In fact, in an infant, certain patterns of injury are sufficiently characteristic to permit a firm diagnosis of inflicted injury in the absence of clinical information.[22] This fact mandates that imaging surveys performed to identify skeletal injury be performed with at least the same level of technical excellence routinely used to evaluate accidental injuries. The "body gram" (a study that encompasses the entire infant or young child on one or two radiographic exposures) or abbreviated skeletal surveys have no role in the imaging of these subtle but highly specific bony abnormalities.[22]

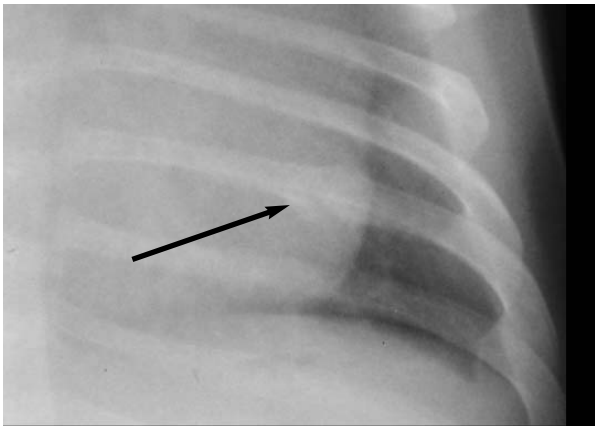
In general, the radiographic skeletal survey is the method of choice for global skeletal imaging in cases of suspected abuse. The standard skeletal survey imaging protocol that has been developed by the American College of Radiology is given in Table 1.[22,24] Anteroposterior and lateral views of the skull are mandatory even when cranial computed tomography (CT) has been performed because skull fractures coursing in the axial plane may be missed with axial CT. Skeletal injuries, especially those requiring orthopedic management, necessitate at least two radiographic projections. Oblique views of the thorax increase the yield for the detection of rib fractures. *(See Figure 1.)* Recent

Table 1. Standard Skeletal Survey

APPENDICULAR SKELETON	AXIAL SKELETON
Humeri (AP)	Thorax (AP and lateral)
Forearms (AP)	Pelvis (AP; including middle and lower lumbar spine)
Hands (Oblique PA)	Lumbar spine (lateral)
Femurs (AP)	Cervical spine (lateral)
Lower legs (AP)	Skull (frontal and lateral)
Feet (AP)	

AP= anteroposterior; PA=posteroanterior

Figure 1. Oblique View to Detect Rib Fracture



Shallow oblique radiograph demonstrating a subtle rib fracture in a victim of child abuse.

evidence suggests that a follow-up skeletal survey approximately two weeks after the initial study increases the diagnostic yield, and this procedure should be considered when abuse is strongly suspected.[24,25] The repeated study may permit more precise determination of the age of individual injuries. Lack of interval change may indicate that the initial radiographic finding is a normal anatomic variant or is related to a bone dysplasia.

Radionuclide Bone Scan. A bone scan identifies areas of increased bone turnover, and is a sensitive method for detecting rib fractures fewer than 7-10 days old, subtle diaphyseal fractures, and early periosteal elevation.[24] A bone scan is most commonly used as an adjunct to the skeletal survey when abuse is strongly suspected and the skeletal survey is normal. Conversely, the skeletal survey can be repeated 3-4 weeks after the initial survey to increase the detection of healing injuries.

Computed Tomography Scan. A CT scan is the method of choice for diagnosing acute intracranial, pulmonary, and solid abdominal organ abnormalities in children with serious injuries.[22-24]

Magnetic Resonance Imaging (MRI). MRI scans of the brain are more sensitive than CT scans in detecting certain traumatic injuries, including axonal shearing, cortical contusions, and brainstem injuries.[22-24]

Manifestations of Physical Abuse

Nonaccidental injuries may affect any organ system in the body, alone or in combination. The following are common manifestations of child abuse.

Head Trauma. Trauma is the most common cause of death in childhood, and inflicted head injury is the most common cause of traumatic death in infancy.[6,11,26,27] On average, among children hospitalized for blunt trauma, those injured by abuse sustain more severe injuries, use more medical services, and have worse survival and functional outcome than children with unintentional injuries.[16,26,28-30] This is despite a plethora of interventions developed over the last 30 years, including legislatively mandated reporting and the establishment in 1974 of the National Center on Child Abuse and Neglect as a mechanism to increase knowledge of the problem and identify steps to prevent it.[6]

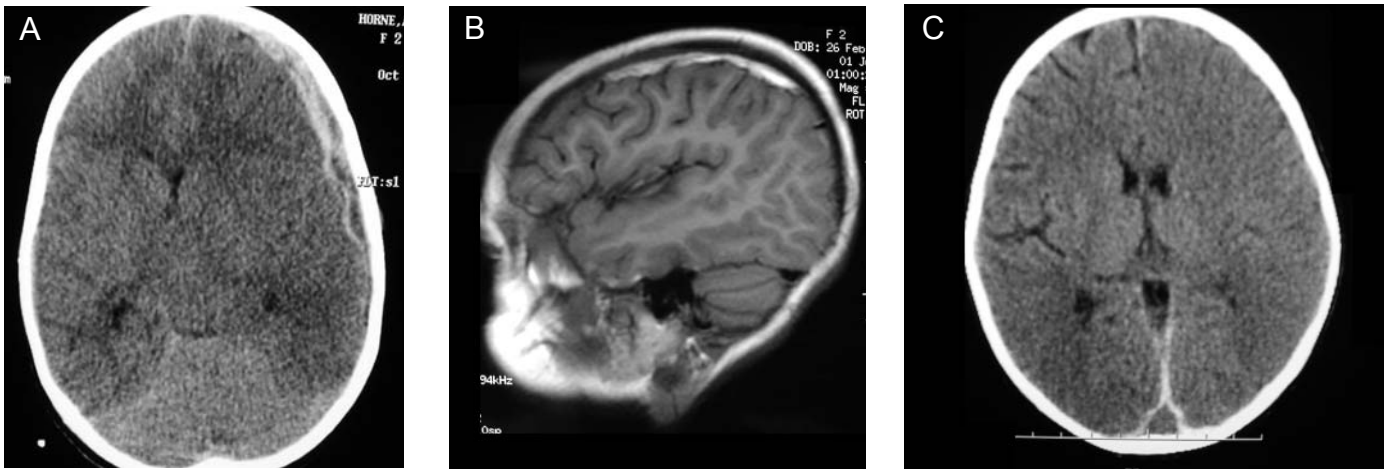
The major issue plaguing the description of abuse-related injuries to young children has been and continues to be accurate diagnosis.[26,31,32] The dire consequences of either false-positive or false-negative diagnosis intensifies the need to establish accurate diagnostic criteria.

Because of some unique features of the infant brain, the risk for severe intracranial injury is great.[12] With a large head-to-body ratio and relatively weak neck muscles, infants are more susceptible to the acceleration/deceleration forces of abusive head trauma. In addition, the immature brain, not fully myelinized, has only 25% of its adult weight at birth, and 75% by age 2 years.[33] There is more subarachnoid space, and therefore a greater vulnerability to shearing of blood vessels causing significant hemorrhage. There is a common misconception that infants will tolerate an acute insult better than their adult counterparts because the fontanel is open; however, it must be remembered that the brain is encased by the inelastic dura.[33] The infant has a shorter craniospinal axis than the adult and thus has less of a "potential space" to displace blood volume and cerebrospinal fluid; this can make the infant brain more susceptible to increased intracranial pressure and secondary brain injury.[33]

Head injuries in infants and toddlers can be difficult to diagnose because symptoms are often nonspecific.[12] Vomiting, fever, irritability, and lethargy are common symptoms of a variety of conditions seen in children, including head trauma. When caretakers do not give a history of injury and the victim is preverbal, an abusive head injury mistakenly can be diagnosed as a less-serious condition. When medical personnel fail to recognize that the child's symptoms are secondary to nonaccidental head injury, the child is frequently re-injured or has serious complications of the unrecognized, untreated head injury.[34]

The primary brain injury attributed to child abuse was originally thought to result from repetitive accelerative-decelerative forces of shaking, hence the term "shaken-baby syndrome."[11] However, biomechanical studies subsequently suggested that the inertial forces developed from shaking were insufficient to cause diffuse axonal injury (DAI), suspected to be the primary mechanism of neuronal injury after abuse.[11] Because impact with even a soft surface, such as a bed or crib, could theoretically cause DAI, the concept of the "shaken-impact syndrome" developed.[11] Recent reports have shown, however, that diffuse changes consis-

Figure 2. CT and MRI in Children with Subdural Hemorrhage and Ischemic Changes



A—Computed tomography (CT) from a 2-year old showing subdural hemorrhage, edema, and shift. **B**—T1-weighted magnetic resonance imaging (MRI) scan showing subdural hemorrhage in a nonaccidental trauma victim. **C**—Interhemispheric hematoma demonstrated on CT scan.

tent with hypoxic-ischemic damage are more common than DAI, which is found mostly in the brainstem, and that cervical spine injury is frequent.[9,35-37] This suggests that the inertial forces of shaking cause brainstem and spinal injuries, which lead to respiratory impairment and subsequent hypoxic injury.

Apnea induced by shaking or by shaking combined with impact plays a major role in the pathophysiology of nonaccidental head trauma and accounts for the poor outcome in this group of patients.[14,38] Trauma induced apnea causes cerebral hypoxia and possibly ischemia, which may be more fundamental to outcome than the mechanism of injury (shaken vs shaken with impact), subdural hemorrhage, subarachnoid hemorrhage, diffuse axonal injury, parenchymal shear, or brain contusion. In addition, the timing of the primary injury is closely linked to the onset of apnea.[37,39]

CT scanning is a mainstay of the diagnosis of nonaccidental head injury.[22,40] Subdural or subarachnoid hemorrhage almost always can be detected on CT scans, although the more subtle findings may be missed by less experienced observers.[40-44] The most frequent CT findings in nonaccidental traumatic brain injury are a combination of subdural convexity and interhemispheric hematomas.[16,26,45] The interhemispheric subdural hematoma is particularly characteristic of the shaking or shaking-impact mechanisms in which angular acceleration and deceleration forces are involved. *(See Figure 2.)*[46] The majority of subdural hemorrhages in children younger than 2 years are due to child abuse.[40,43] MRI is useful in detecting and characterizing small extraaxial hemorrhages in infants with equivocal CT findings.[46,47]

The neuroradiologic findings of abuse often are present within a few hours of the assault, particularly when the event results in hypoxia-ischemia.[14,37,46] In these cases, the CT may show findings of edema as early as 2 hours following the documented event.[46] The earliest CT sign is often a loss of cerebral gray-white matter differentiation in the cortical and subcortical regions, in deep basal ganglia and capsular regions, or in both. Hypoxic-ischemic brain injury may result from associated brainstem injury with apnea, from progressive edema and increased intracranial pressure, or from additional suffocation or strangulation.[46]

All infants and children with suspected intracranial injury must undergo either cranial CT or MRI. *(See Figure 2.)* Strategies should be directed toward the detection of all intracranial sequelae of abuse and neglect with a thorough characterization of the extent and age of the abnormalities. In the acute care setting, efforts are directed toward rapid detection of treatable conditions. Subsequent studies are designed to delineate more fully all abnormalities, determine the timing of the injuries, and monitor their evolution.[44,46]

Radiologists evaluating imaging studies of young children, even those in whom there is no suspicion of physical abuse, should routinely assess subtle signs of chronic brain damage in addition to acute findings.[16,41,44] Investigators have identified elevated rates of cortical atrophy, ventriculomegaly, and subdural hygroma only in children with inflicted traumatic brain injury.[16]

MRI is the best modality to assess fully intracranial injury, including extraaxial collections, intraparenchymal hemorrhages, contusions, shear injuries, and brain swelling, or edema.[43] Imaging should be performed with T_1 and T_2 weighting with proton-density or inversion-recovery sequences to differentiate cerebrospinal fluid collections from other water-containing lesions. Diffusion imaging is a new and valuable technique for the evaluation of cerebral ischemia and likely will have a role in the assessment of inflicted cerebral injury.[36,48] Abused infants may not demonstrate neurologic signs and symptoms, despite significant CNS injury. The MRI offers the highest sensitivity and specificity for diagnosing subacute and chronic injury and should be considered whenever typical skeletal injuries associated with shaking or impact are identified.

Table 2. Nonaccidental Trauma: Hints to Facilitate an Early Diagnosis

FRACTURES

- No single fracture is pathognomonic of child abuse.
- Certain patterns of fractures should increase the concern for abuse, including multiple fractures and fractures of different ages.
- Multiple, bilateral posterior rib fractures in infants are almost always the result of abuse.

BABIES WITH BRUISES

- With advancing age and motor skills, bruises occur more commonly.
- Bruises in mobile infants normally are located on the front of the body, typically on the shins and forehead.
- Bruises in infants younger than 9 months who are not yet beginning to ambulate should lead to consideration of abuse or illness.
- Bruises in toddlers that are located in atypical areas, such as the trunk, genitalia, neck, hands or buttocks, should be considered abuse.

SKULL FRACTURES: ACCIDENTAL VS. NON-ACCIDENTAL

- Inflicted
  - History does not correlate with mechanism of injury, OR no history
  - Associated with intracranial injury, especially subdural hematomas
  - Bilateral
  - Non-parietal
  - Comminuted/Stellate
  - Depressed
  - Wider than 1 mm
  - Associated with other injuries
  - Crossing suture lines
- Accidental
  - History consistent with injuries
  - Not associated with intracranial injury
  - Unilateral
  - Parietal
  - Linear
  - Nondepressed
  - Narrower than 1 mm
  - Involves only one body area
  - Does not cross suture lines

Many studies have demonstrated the usefulness of MRI in suspected acute nonaccidental head injury. It is particularly useful for the diagnosis of subdural hematomas in the subtemporal area, illustrating tearing of the bridging veins, delineating subdural hematomas of different ages, and demonstrating hemorrhages at the gray-white matter junction.[47] All these features are not well defined by CT.

Several selected points culled from an extensive literature review may prove useful in recognizing nonaccidental trauma in infants and children. *(See Table 2.)*[2,27]

There is an abundance of literature attesting to the fact that short falls (< 4 feet), such as from beds, couches or down stairs, do not cause serious injury in children, except the case of epidural hematoma, which can occur after short falls.[2,26,49-58] However, some of the most severe infant head injuries occur when an adult falls on the stairs while carrying an infant.[55,59,60] The relative severity of these injuries is probably the result of two different injury mechanisms: fall from a height and a fall down stairs.[60] These two mechanisms can increase the impact force.

Simple skull fractures are common in accidental falls, with complex fractures seen less frequently.[26] Subdural hematomas and subarachnoid hemorrhages seldom are seen and retinal hemorrhages virtually are never seen in short falls.[2]

Inflicted head injury in infants commonly presents as shaken baby syndrome. Altered level of consciousness, coma, seizure, listlessness/lethargy, irritability, apnea or respiratory difficulty, and poor feeding can be associated with shaken baby syndrome. Although not pathognomonic for child abuse, retinal hemorrhages occur in up to 75-85% of these cases and frequently correspond with subdural or subarachnoid bleeding.[13,18] Skeletal trauma, such as sternal fractures and posterior rib fractures, frequently are seen in shaken baby syndrome due to the placement of the perpetrator's hands. Acceleration/deceleration forces are created as the head whips forward, stopping as the chin strikes the chest, then backward until the occiput strikes the back, as well as when the baby is thrown on the sofa or bed. Shearing injuries to the intracranial bridging veins and incompletely myelinated cortical nerves are sustained. CT scanning is an excellent tool for detecting subdural and subarachnoid hemorrhages, especially when associated with retinal hemorrhages. Epidural hemorrhage in infants is less likely to be caused by shaking and more likely to be due to a blow or fall.[13,61,62] Epidural hemorrhages are rarely a result of abuse.

Finally, the evaluating physician also must be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.[2,12,63] These include accidental injury, certain coagulopathies, vascular diseases, infections, metabolic disorders, neoplastic diseases, certain therapies, and some congenital and dysplastic disorders. Infants who present with unexplained subdural and retinal hemorrhages most often are victims of child abuse. However, an autosomal recessive metabolic disorder, glutaric acidemia type 1, is a known cause of these findings in children.[63,64] Urine organic acid testing will show a characteristic large peak of glutaric acid and 3-hydroxy glutaric acid. The diagnosis can be confirmed through testing of enzyme activity in cultured fibroblasts and leukocytes or through genetic mutation analysis.

Abdominal Trauma. Severe abdominal injury is an uncommon, but well recognized, manifestation of abuse.[65] The history almost always is misleading, and rarely includes a history of abdominal trauma. Less severe injury is under-recognized and underreported, because symptoms are non-specific and external indications of abdominal trauma are often lacking.[23]

Most abusive abdominal injury is caused by blunt trauma, resulting in solid organ injury, perforation of hollow viscous, or shearing of mesenteric vessels.[4,65-68] Isolated solid organ injuries are most common with both accidental and inflicted injuries,

although hollow visceral injuries more commonly are associated with abuse.[4] Recent reports of abdominal trauma secondary to abuse reveal that liver and spleen injuries are most common, followed by duodenojejunal rupture, duodenal rupture, and pancreatic, vena cava, and renal trauma.[66,67] These injuries are thought to be due to compression of abdominal viscera against the vertebral column following a punch or a kick.[66] The small size of the child's abdomen predisposes them to multiple organ injury. Children with severe liver or mesenteric injury usually present with signs and symptoms of acute bleeding, including hemorrhagic shock.[66] Children with intestinal perforation typically present with signs of peritonitis. Their presentation for medical care can be delayed by hours or days, but an accurate history (often lacking) should demonstrate progressive abdominal symptoms.[66] Mortality is extremely high owing to delays in presentation and the magnitude of injuries.[68]

Abused children occasionally have asymptomatic abdominal injuries, which can be detected with evaluations of serum liver function tests (LFTs), amylase, and lipase.[66] Abdominal trauma is diagnosed by physical examination, screening LFTs, amylase, lipase, urinalysis, sonography and abdominal CT.[65-68]

Injuries involving the duodenum are a common finding in blunt trauma to the abdomen, and include duodenal hematomas or transection. Such injuries to the duodenum occur because the ligament of Treitz is a relatively fixed structure and allows compression of the duodenum against the vertebrae. *(See Figure 3.)*[65] It is not infrequently associated with injuries to the adjacent organs. Unfortunately, there are often delays in making the diagnosis because the retroperitoneum, in which part of the duodenum lies, offers some protection and this contributes to the morbidity and mortality of these injuries.

Life-threatening intraabdominal injuries can exist with few signs or symptoms. One research team describes a 2½ -year-old child who presented with relatively few symptoms, who at post mortem examination was found to have peritonitis secondary to a duodenal rupture which was considered to have occurred 2-3 days before death.[69]

Liver injuries also can be occult with no evidence of external injuries. One study found, in children suspected of nonaccidental injury who had no history or physical signs of abdominal trauma, evidence of occult liver lacerations on CT in 6% of cases.[70] Researchers also found that raised transaminases were associated with these cases and consider that this is a useful indicator of occult liver injury.[70]

Nonaccidental trauma as a cause of pancreatic injury is not uncommon. One group reported one-third of their series of 49 cases of pancreatitis in children as being associated with nonaccidental injury.[71] As in adults, pancreatitis in children is associated with considerable morbidity and chronic pancreatitis may develop.

Fractures. Fractures often are seen in physical abuse cases.[72] They also are a common accidental childhood injury. As in the preceding cases, the clinician must ask, "Is the injury consistent with the history and developmental stage?"

Abusive fractures are more common in younger children (< 5 years of age), often present without a history of trauma, and often are characterized by a delay in seeking medical care.[72] Any fracture can be the result of abuse.[3] It is the history, physical examination, and additional evaluation that are crucial in differentiating accidental from nonaccidental trauma. In children younger than 2 and selectively in children younger than 5 years of age, a skeletal survey is in order to look for other injuries, some of which may be occult.

A common type of history given in both abusive and unintentional injuries is a fall. Although a history of a fracture in a minor fall should be investigated, single unintentional fractures can occur from falls of fewer than two feet, and falls from fewer than four feet can result in injury to more than one bone.[73]

Radiographically, some fractures are more suspect for abusive injury than others.[24,72,74] Fractures that are highly specific for abusive injury in infants include the following: posterior rib fractures, scapular fractures, spinous process fractures, sternal fractures, and classic metaphyseal lesions. The classic metaphyseal lesions often are called bucket handle or corner fractures, and occur at the end of the long bones at the growth plate. Fractures with moderate specificity for abuse are complex skull fractures, digital fractures, vertebral body fractures, epiphyseal separations, fractures of different ages, and multiple fractures. Fractures common in childhood with low specificity for abuse are linear skull fractures, long bone fractures, and clavicular fractures. The appearance of subperiosteal new bone formation in infants also is common, but can be a normal variant. A pediatric radiologist may help with this evaluation when appropriate.

Linear parietal skull fractures are common whether unintentional or secondary to abuse.[72] However, skull fractures that are multiple, depressed, diastatic more than 3 mm, bilateral, or cross suture lines are more suggestive of intentional injury, especially coupled with a suspicious history.[24,72,73]

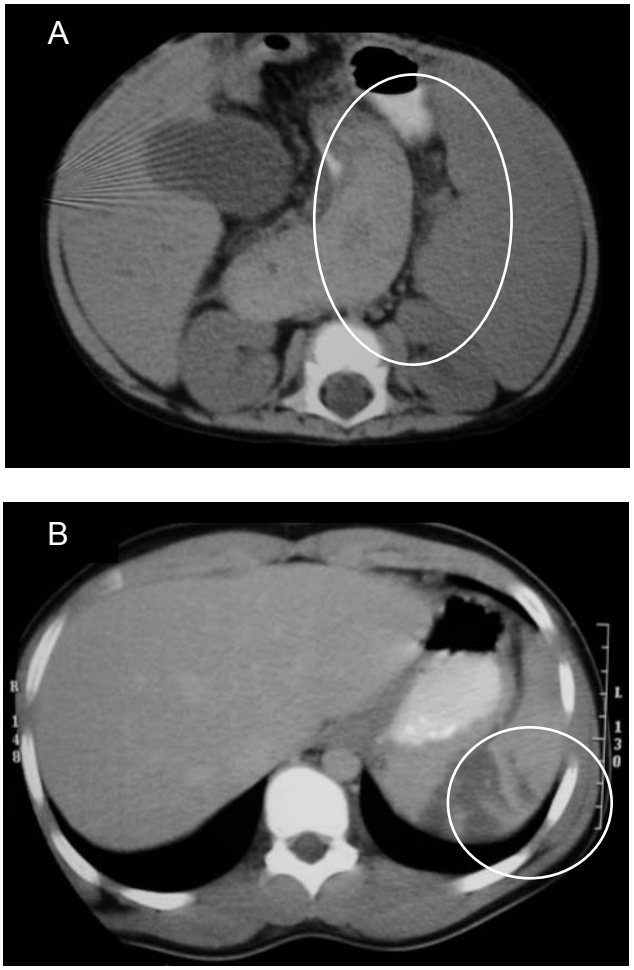
The humerus is the most commonly fractured bone in battered children.[72,75] Several authors have reported that the majority of humerus fractures in infants younger than 15 months are intentional.[72]

Rib fractures commonly are seen in abused children, with 90% of abuse-related fractures occurring in children younger than 2 years.[72,76,77] In infants, rib fractures are the most frequent fracture of abuse.[72,78] In general, rib fractures in children are much less common than in adults owing to their more compliant chest walls. Therefore, rib fractures, especially multiple fractures, are very suggestive for abuse in children younger than 2 years in the absence of major blunt trauma or prior bone pathology.[72]

Abusive fractures typically occur when the infant is manually grabbed around the thoracic cage and violently squeezed and shaken. This anteroposterior compressive force results most frequently in multiple, symmetrical, posterior rib fractures where mechanical stress is at its greatest. With increasing force, lateral then anterior fractures occur.[79] Of importance, rib fractures from cardiopulmonary resuscitation are rare in infants and young children.[72]

Diseases such as osteogenesis imperfecta, rickets, and osteomyelitis can mimic abusive injuries.[72] Most have other clini-

Figure 3. Abdominal Trauma



A—Duodenal hematoma. **B**—Splenic lacerations.

cal manifestations that distinguish them from abuse, and may be diagnosed by simple blood tests.[4]

Bruises. A bruise is an area of skin discoloration caused by the escape of blood from ruptured underlying blood vessels after an injury. It is the process of hemoglobin degradation and its expression through the "window" of the skin that determines the color of a bruise.[80] They are a common injury in children and often are accidental. However, bruises may be a sign of physical abuse.[80-85] To evaluate whether a bruise is abusive or accidental, it is important again to look at the history, the child's developmental level, the pattern of the bruising, and other findings on physical examination.

In general, bruises are very difficult to age by appearance alone, especially in children. A yellow color to the bruise indicates that it is probably more than 18 hours old. However, red, blue, purple, and brown can be seen at any time. The healing of bruises is affected by the area of skin involved, the depth of the bruise, the amount of blood in the bruise, and other factors.[80] Therefore, the dating and aging of bruises is inexact.[4]

The history of the bruise is important—both from the caretaker and the child, if obtainable. When was the first time the bruise was noted? What is the reported mechanism—fall, play, sports? Does the mechanism of injury match the bruise seen? Could the child do what is reported?

In the physical examination, it is important to look at all areas of skin and document any injuries. The area of the body affected is important because some areas are more suspect for abuse than others.[83-85] Some areas of the body such as the back, buttocks, neck, cheek, ear, thighs, genitalia, and hands are atypical areas of accidental bruises. Grab marks sometimes are seen on the upper arms. Some bruises may appear patterned. Patterns of concern for abusive injury are looped cord marks, hand prints, and other patterns that could be matched to an object such as a shoe, belt, or other implement.[2] Patterns of abusive bruising also may be determined by the anatomy of the injured body part rather than the shape of the injuring object. Two examples include patterned abusive bruises of the buttocks and the pinnae.[86,87]

Bite marks are lesions that may indicate abuse.[3] Bite marks should be suspected when ecchymoses, abrasions, or lacerations are found in elliptical or ovoid patterns. Bite marks may have a central area of ecchymoses. The normal distance between the maxillary canine teeth in adult humans is 2.5-4.0 cm, and the canine marks in a bite will be the most prominent on deep parts of the bite.[3] Bites produced by dogs and other carnivorous animals tend to tear flesh, whereas human bites compress flesh and can cause abrasions, contusions, and lacerations but rarely avulsions of tissue. If the intercanine distance is less than 2.5 cm, the bite may have been caused by a child. If the intercanine distance is 2.5-3.0 cm, the bite was probably produced by a child or small adult; if the distance is greater than 3.0 cm, the bite was probably by an adult.[3] The pattern, size, contour and colors of the bite mark should be evaluated by a forensic odontologist or forensic pediatrician or pathologist if an odontologist is not available. A photograph, taken at a right angle to the bite should be taken with an identification tag and scale marker in the photograph. Because each person has a characteristic bite pattern, a forensic odontologist may be able to match dental models (casts) of a suspected abuser's teeth with photographs of the site.

The differential diagnosis for bruises is long.[2,82] Mongolian spots often are confused with bruises. Mongolian spots do not fade or change over days to weeks. A repeat examination can make the diagnosis if there are questions. Minimal accidental trauma may result in dramatic bruises suggesting nonaccidental trauma in some medical problems such as idiopathic thrombocytopenic purpura, hemophilia, and vitamin K deficiency.[82,88] In settings of inappropriate bruising, it sometimes is appropriate to obtain a complete blood count and coagulation studies.

In some cultures, there are practices that can be confused with abusive bruising. These are cupping and coining.[82] These cultural practices are done with the intent of helping the child, and some report that it feels good.

The key question remains, "Is this injury consistent with the history provided by the caretaker, and is it consistent with the child's developmental abilities?" Several studies have looked at bruises in relation to development.[83,84] Based on their research,

Sugar, et al., reported, "Those who cannot cruise don't bruise."[83] They found that those children who are not yet cruising (walking while holding onto furniture) do not have significant bruises on their bodies. There may be one or two isolated bruises over bony prominences (e.g., forehead, knee), but most of these children do not have bruises when examined. Bruises on the face and neck necessitate a good history owing to the concern for physical abuse.

Burns. Burns occur when tissue is damaged by heat, chemicals, sunlight, electricity, or nuclear radiation.[89] The history related to how burns occurred and how that correlates with the burn pattern is more important than the depth of burns. But in investigating burns, records must include specific details about pattern, location, and degree. Burns can be classified into first-, second-, third-, and fourth-degree, based on the depth and severity of the burn.

First-degree burns are the most common and rarely require hospitalization. There is superficial tissue damage often characterized by painful erythema without blisters. These usually heal without scarring. An example would be the common sunburn. Other than raising issues of possible neglect, this severity of burn usually is not related to abuse.

Second-degree burns are considered partial-thickness burns. They are characterized by clear fluid-filled blisters that are very painful and sensitive to temperature and air. The lesions often blanch with pressure. They usually heal within 1-2 weeks and sometimes scar.

Third-degree burns involve the full thickness of the skin. The injury is characterized by the finding of charring or translucent white tissue with mottling. Over time, the overlying tissues may develop a leathery, dry appearance. There is minimal pain in the charred tissue because the nerve endings have been damaged. There often is marked edema, and the color can vary from white to gray to red to black or charred. These burns always scar and often require excision and grafting.

Fourth-degree burns involve not only all layers of the skin, but also subcutaneous fat and deeper structures. They usually have a charred appearance. A unique situation seen in child abuse with fourth-degree burns is when a body (a child) is placed in an operating microwave oven.[90] There are only a handful of case reports in the literature, all involving children. In such cases, the tissue injury often is worse than it appears because of the way that microwave ovens cook. This type of burn will show relative layered tissue sparing where the skin and underlying muscle are burned with relative sparing of the subcutaneous fat.[91] A child with this injury must be closely monitored for complications, preferably in a specialized burn unit.

Burns also can be classified as thermal, electrical, chemical, or radiation. The most commonly seen in abuse are thermal burns. Electrical burns can be seen in children when electrical cords are chewed (corners of the mouth) or outlets explored (fingertips). Chemical burns and radiation burns rarely are seen. Depending on severity and location of burns, some children do need to be transferred to the nearest burn facility.

In evaluating burns in children, there are several factors to consider. These include the history, physical examination, the child's developmental level, and the presentation of the injury to medical care.

When obtaining a history, the clinician first investigates the reported mechanism for the burn. It is important to note in the medical record the reported history and who reported it. In abuse cases, the history of the injury sometimes changes over time or with different witnesses. This can sometimes occur with accidental injuries, but such inconsistencies should raise a red flag for possible abuse.

The examiner also should look at the child's developmental level, both reported and observed. The clinician should remember to observe whether details provided by the caretaker are similar to those observed in interactions with the child. In evaluating burns, the examiner must ask himself or herself such questions as could that child have reached up and grabbed that cup of hot tea? Is the history compatible with the child's developmental level? CPS or law enforcement agencies usually can provide scene evaluations when necessary.

With possible child abuse, the physical examination is more than gathering information to assess the patient's injuries and provide care; it is a source of very valuable information about mechanisms of injury and possible abuse. What is the pattern of the burn injury—immersion, flow, or contact? Does this pattern match with the reported history? As an example, the stocking-and-glove distribution seen in immersion burns is fairly specific for nonaccidental injury.[4] These have a clear line of demarcation between the burned and unburned skin that looks like a sock line. There often are no splash marks. This is indicative of the limb being held forcefully in hot water. A donut distribution on the buttock area also can be seen with immersion burns. This is seen when the child is held in hot water in a tub (or sink). The donut appearance of central sparing and peripheral burns is related to the surface (usually of the tub) protecting the central skin, and the fluid in contact with the periphery resulting in second-degree burns. Skin in contact with other skin (e.g., between buttocks, behind knees) also is spared. By noting the burn pattern, the position the child was in at the time of the burn often can be recreated. As a protected area of the body, genital burns are uncommon accidental injuries. They often are seen in abusive situations, especially those involving toileting accidents.[4]

Burns may be inflicted by contact with hot solids, such as irons, radiators, stoves, or cigarettes.[4] Inflicted burns are characteristically symmetrical, deep, and leave a clear imprint of the hot instrument. Dermatologic and infectious disease can mimic abusive burns, including toxin-mediated staphylococcal and streptococcal infections, impetigo (which can be mistaken for cigarette burns), and phytophotodermatitis.[4,82]

How the child presented to medical care also is important. Did the child receive what appears to be a second-degree burn four days ago and is just now coming for treatment? Who is bringing the child for treatment? In abuse situations, there often is a delay in seeking appropriate medical care.

In completing the physical examination, the provider should be alert to other signs of trauma or neglect such as failure to

thrive or other injuries and scars. The physical examination should be fully documented with diagrams and pictures, if possible. This can help with longitudinally following healing of the burn and can assist greatly with recall in court if needed.

Conclusion

Neglect and abuse remain a difficult and emotionally charged topic. Occurring behind closed doors, it is unobserved and confessions are rare.[92] There are myriad presentations, and abuse and neglect may mimic other disease processes. While there is significant morbidity and mortality, the diagnosis and treatment are intertwined with legal issues of parental rights and family preservation.

Identifying children who are victims of abuse is a difficult and unpleasant part of pediatric health care. However, it also can be rewarding in that it can serve as the first step in saving or improving a child's life. Health care professionals must keep in mind that children are the victims of these horrible acts and that they are often silent victims. They must count on us to identify and rescue them. Don't forget to keep child abuse on the differential diagnosis of all childhood injuries.

References

1. U.S. Department of Health and Human Services, Administration on Children, Youth and Families, Child Maltreatment 2000 (Washington, DC: U.S. Government Printing Office; 2002).
2. Block RW. Child abuse: Controversies and imposters. *Curr Prob Pediatr* 1999;29:253-272.
3. American Academy of Pediatrics, Committee on Child Abuse and Neglect, American Academy of Pediatrics Dentistry, Ad Hoc Work Group on Child Abuse and Neglect. Oral and dental aspects of child abuse and neglect. *Pediatrics* 1999;104:348-350.
4. Christian CW. Recognition of child abuse in the pediatric trauma patient. *Trauma Reports* 2000;1:1-12.
5. Jaim AM. Emergency department evaluation of child abuse. *Emerg Med Clin North Am* 1999;17:575-593.
6. DiScala C, Sege R, Li G, et al. Child abuse and unintentional injuries. *Arch Pediatr Adolesc Med* 2000;154:16-22.
7. Overpeck MD, Brenner RA, Trumble AL, et al. Risk factors for infant homicide in the United States. *N Engl J Med* 1998;339:1211-1216.
8. Starling SP, Holden JR, Jenny C. Abusive head trauma: The relationship of perpetrators to their victims. *Pediatrics* 1995;95:259-262.
9. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children I: Patterns of brain damage. *Brain* 2001;124:1290-1298.
10. Shannon P, Becker L. Mechanisms of brain injury in infantile child abuse. *Lancet* 2001;358:686-687.
11. Duhaime A, Christian CW, Rorke LB, et al. Nonaccidental head injury in infants—the "shaken-baby syndrome." *N Engl J Med* 1998; 338:1822-1828.
12. Scribano PV. Abusive head trauma. *Pediatric Case Reviews* 2002; 2:87-94.
13. Keenan H, Runyan DK. Shaken baby syndrome: Lethal inflicted traumatic brain injury in young children. *NCMJ* 2001;62:340-343.
14. Johnson DL, Boal D, Baule R. Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* 1995;23:305-310.
15. Alexander RL, Crabbe L, Sato Y, et al. Serial abuse in children who are shaken. *Am J Dis Child* 1990;144:58-60.
16. Ewing-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Child's Nerv Syst* 2000;16:25-34.
17. Kempe CH, Silverman FN, Steele BF, et al. The battered-child syndrome. *JAMA* 1962;18:17-24.
18. Morris MW, Smith S, Cressman J, et al. Evaluation of infants with subdural hematoma who lack external evidence of abuse. *Pediatrics* 2000;105:549-553.
19. Mei-Zahav M, Uziel Y, Raz J, et al. Convulsions and retinal hemorrhage: Should we look further? *Arch Dis Child* 2002;86:334-335.
20. Stricker T, Lips U, Sennhauser FH. Oral bleeding: Child abuse alert. *J Paediatr Child Health* 2002;38:528-529.
21. Hymel KP, Abshire TC, Luckey DW, et al. Coagulopathy in pediatric abusive head trauma. *Pediatrics* 1997;99:371-375.
22. American Academy of Pediatrics, Section on Radiology. Diagnostic imaging of child abuse. *Pediatrics* 2000;105:1345-1348.
23. Nimkin K, Kleinman PK. Imaging of child abuse. *Pediatr Clin North Am* 1997;44:615-635.
24. Care M. Imaging in suspected child abuse: What to expect and what to order. *Pediatr Ann* 2002;31:651-659.
25. Kleinman PK, Nimkin K, Spevak MR, et al. Follow-up skeletal surveys in suspected child abuse. *Am J Roentgenol* 1996;165:893-896.
26. Reece RM, Sege R. Childhood head injuries: Accidental or inflicted? *Arch Pediatr Adolesc Med* 2000;154:11-15.
27. Perkin RM, Moynihan JA, McLeary M. Current concepts in the emergency management of severe traumatic brain injury in children. *Trauma Reports* 2000;1:1-16.
28. Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics* 1998;102:300-307.
29. Haviland J, Russell RIR. Outcomes after severe non-accidental head injury. *Arch Dis Child* 1997;77:504-507.
30. Ewing-Cobbs L, Prasad M, Kramer L, et al. Inflicted traumatic brain injury: Relationship of developmental outcome to severity of injury. *Pediatr Neurosurg* 1999;31:251-258.
31. Herman-Giddens ME, Brown G, Verbiest S, et al. Under ascertainment of child abuse mortality in the United States. *JAMA* 1999;282:463-467.
32. Leventhal JM. The challenges of recognizing child abuse: Seeing is believing. *JAMA* 1999;281:657-659.
33. Conway EE. Nonaccidental head injury in infants: "The shaken baby syndrome revisited." *Pediatr Ann* 1998;27:677-690.
34. Jenny C, Hymel KP, Ritzen A, et al. Analysis of missed cases of abusive head trauma. *JAMA* 1999;282:621-626.
35. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children II: Microscopic brain injury in infants. *Brain* 2001; 124:1299-1306.
36. Stoodley N. Non-accidental head injury in children: Gathering the evidence. *Lancet* 2002;360:271-272.
37. Nashelsky MB, Dix JD. The time between lethal infant shaking and onset of symptoms. *Am J Forensic Med Path* 1995;16:154-157.
38. Atkinson JLD. The neglected phase of head injury: Apnea and catecholamine surge. *Mayo Clin Proc* 2000;75:37-47.
39. Willman KY, Bank DE, Senae M, et al. Restricting the time of injury in fatal inflicted head injuries. *Child Abuse Negl* 1997;21:929-940.
40. Jayawant S, Rawlinson A, Gibbon F, et al. Subdural hemorrhages in infants: Population based study. *BMJ* 1998;317:1558-1561.
41. Wells RG, Vetler C, Land P. Intracranial hemorrhage in children younger than 3 years. *Arch Pediatr Adolesc Med* 2002;156:252-257.
42. Feldman KW, Bethel R, Shugerman RP, et al. The cause of infant and toddler subdural hemorrhage: A prospective study. *Pediatrics* 2001;108:636-646.
43. Kemp AM. Investigating subdural hemorrhage in infants. *Arch Dis Child* 2002;86:98-102.
44. Dias MS, Backstrom J, Falk M, et al. Serial radiography in the infant shaken impact syndrome. *Pediatr Neurosurg* 1998;25:77-85.
45. Wells RG, Vetler C, Land P. Intracranial hemorrhage in children younger than 3 years: Prediction of intent. *Arch Pediatr Adolesc Med* 2002;156:252-257.
46. Barnes PO, Robson CD. CT findings in hyperacute nonaccidental brain injury. *Pediatr Radiol* 2000;30:74-81.
47. Barlow KM, Gibson RJ, McPhillips M, et al. Magnetic resonance imaging in acute nonaccidental head injury. *Acta Paediatr* 1999;88:734-740.
48. Biousse V, Suh DY, Newman NJ, et al. Diffusion weighted magnetic reso-

nance imaging in shaken baby syndrome. *Am J Ophthalmol* 2002;133: 249-255.
49. Baxter AL, Bechtel K, Pierce MC. Abuse or not abuse: That is the question. *Pediatr Emerg Care* 2002;18:203-208.
50. Chadwick DL, Chin S, Salerno C, et al. Deaths from falls in childhood: How far is fatal? *J Trauma* 1991;31:1353-1355.
51. Tarantino CA, Dowd MD, Murdock TC. Short vertical falls in infants. *Pediatr Emerg Care* 1999;115:5-8.
52. Lyons TJ, Oates RK. Falling out of bed: A relatively benign occurrence. *Pediatrics* 1993;92:125-127.
53. Nimityongskul P, Anderson LD. The likelihood of injuries when children fall out of bed. *J Pediatr Orthop* 1987;7:184-186.
54. Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics* 1977;60:533-535.
55. Joffe M, Ludwig S. Stairway injuries in children. *Pediatrics* 1988;82: 451-461.
56. Hall JR, Reyes HM, Hurvat M, et al. The mortality of childhood falls. *J Trauma* 1989;29:1273-1275.
57. Warrington SA, Wright CM. Accidents and resulting injuries in premobile infants: Data from the ALSPAC study. *Arch Dis Child* 2001;85:104-107.
58. Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma* 1991;31:350-352.
59. Greenes DS, Schutzman SA. Clinical indicators of intracranial injury in head-injured infants. *Pediatrics* 1999;104:861-867.
60. Chiaviello CT, Christoph RA, Bond GR. Stairway-related injuries in children. *Pediatrics* 1994;54:679-681.
61. Shugerman RP, Paez A, Grossman DC, et al. Epidural hemorrhage: Is it abuse? *Pediatrics* 1996;97:664-668.
62. Beni-Adani L, Flores I, Spektor S, et al. Epidural hematoma in infants: A different entity? *J Trauma* 1999;46:306-311.
63. Soden SE, Dasouki MJ, Walsh IR. A 9-month-old baby with subdural hematomas, retinal hemorrhages, and developmental delay. *Pediatr Emerg Care* 2002;18:44-47.
64. Hartley LM, Khawaja OS, Verity CM. Glutaric aciduria type 1 and nonaccidental head injury. *Pediatrics* 2002;107:174-175.
65. Ng CS, Hall CM, Shaw DG. The range of visceral manifestations of nonaccidental injury. *Arch Dis Child* 1997;77:167-174.
66. Rothrock SG, Green SM, Morgan R. Abdominal trauma in infants and children: Prompt identification and early management of serious and life-threatening injuries. Part I: Injury patterns and initial assessment. *Pediatr Emerg Care* 2000;16:106-115.
67. Gaines BA, Ford HR. Abdominal and pelvic trauma in children. *Crit Care Med* 2002;30(suppl):416-423.
68. Holland AJA, Cass DT, Glasson MJ, et al. Small bowel injuries in children. *J Paediatr Child Health* 2000;36:265-269.
69. Fossum RM, Descheneaux KA. Blunt trauma of the abdomen in children. *J Forensic Sci* 1991;36:47-50.
70. Coant PN, Kornberg AE, Brody AS, et al. Markers for occult liver injury in cases of physical abuse in children. *Pediatrics* 1992;89:274-278.
71. Ziegler DW, Long JA, Philippert AI, et al. Pancreatitis in childhood: Experiences with 49 patients. *Ann Surg* 1988;207:257-261.
72. Sills RM, Pena ME, Parson SK. Bones, breaks, and the battered child: Is it unintentional or is it abuse? *Pediatric Emergency Medicine Reports* 1998; 3:1-10.
73. Leventhal JM, Thomas SA, Rosenfield NS, et al. Fractures in young children: Distinguishing child abuse from unintentional injuries. *Am J Dis Child* 1993;147:87-92.
74. Kleinmann PK. Diagnostic imaging in infant abuse. *Am J Radiol* 1990;155: 703-712.
75. Strait RT, Siegel RM, Shapiro RA. Humeral fractures without obvious etiologies in children less than 3 years of age: When is it abuse? *Pediatrics* 1995;96:667-671.
76. Swischuk LE. Apnea and cyanosis in an infant. *Pediatr Emerg Care* 1993;9:241-243.
77. Cadizow SP, Armstrong KL. Rib fractures in infants: Red alert! *J Paediatr Child Health* 2000;36:322-236.
78. Kleinmann PK, Marks SL, Nimkin K, et al. Rib fractures in 31 abused infants: Postmortem radiologic-histopathologic study. *Radiology* 1996;200: 807-810.
79. Merten DF, Carpenter BC. Radiologic imaging of inflicted injury in the child abuse syndrome. *Pediatr Clin North Am* 1990;37:815-837.
80. Schwartz AJ, Ricci LR. How accurately can bruises be aged in abused children? *Pediatrics* 1996;97:254-257.
81. Labbe J, Caouettee G. Recent skin injuries in normal children. *Pediatrics* 2001;108:271-276.
82. Scales JW, Fleischer AB, Sinai SH, et al. Skin lesions that mimic abuse. *Contemporary Pediatrics* 1999;16:137-145.
83. Sugar NF, Taylor JA, Feldman KW. Bruises in infants and toddlers: Those who don't cruise rarely bruise. *Arch Ped Adolesc Med* 1999;153:399-403.
84. Carpenter RF. The prevalence and distribution of bruising in babies. *Arch Dis Child* 1999;80:159-163.
85. Dunstan FD, Guildea ZE, Kontos K, et al. A scoring system for bruise patterns: A tool for identifying abuse. *Arch Dis Child* 2002;86:330-333.
86. Tsaroulas N. Buttock lesions: A sensitive issue at day care. *Pediatr Ann* 2001;30:586-590.
87. Feldman KW. Patterned abusive bruises of the buttocks and the pinnae. *Pediatrics* 1992;90:633-636.
88. Wetzel RC, Slater AJ, Dover GJ. Fatal intramuscular bleeding misdiagnosed as suspected nonaccidental injury. *Pediatrics* 1995;95:771-773.
89. Cabinum-Foeller E. In-Hospital Evaluation. In, Perkin RM, Swift JD, Newton DA, eds. *Pediatric Hospital Medicine—Textbook of Inpatient Management.* Philadelphia: Lippincott, Williams and Wilkins; 2003:775-778.
90. Alexander RA, Surrell JA, Cohle SD. Microwave oven burns to children: An unusual manifestation of child abuse. *Pediatrics* 1987;79:255-260.
91. Surrell JA, Alexander RC, Cohle SD, et al. Effects of microwave radiation on living tissue. *J Trauma* 1987;24:935-939.
92. Boal DKB, Feldman AH, Krugman RD. Controversial aspects of child abuse: A roundtable discussion. *Pediatr Radiol* 2001;31:760-774.

CME/CE Questions

1. Which of the following sites of injury is the least specific for child abuse?
   A. Scapular fractures
   B. Rib fractures
   C. Metaphyseal fractures
   D. Clavicular fracture

2. All of the following skull fractures more commonly are encountered in abusive rather than in accidental injury *except:*

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

A. bilateral skull fractures.
B. fractures crossing suture lines.
C. isolated linear parietal skull fractures.
D. multiple skull fractures.

3. In a child with suspected physical abuse, the American College of Radiology recommends a full skeletal survey. This survey most appropriately is described by which of the following?
   A. A single frontal view of the entire child
   B. Anteroposterior and lateral views of the upper and lower extremities
   C. Multiple dedicated images of the axial and appendicular skeleton, including additional views as needed to document sites of injury
   D. Anteroposterior and lateral views of the skull plus a single anteroposterior view of the chest and abdomen

4. A 1-year-old girl has a history of not moving her arm for one day. The family denies any history of trauma. The child is acting normally and has no other evidence of acute injury. An x-ray reveals a midshaft humeral fracture. The next step in the work up of this child would be:
   A. to order a CT scan of the head to rule out a chronic subdural hematoma.
   B. to order liver enzymes to help rule out intra-abdominal injury.
   C. to obtain a skeletal survey to rule out other fractures.
   D. to apply a sling and swath and have the patient follow up with orthopedics.

5. Which one of the following statements concerning nonaccidental trauma is *not* true?
   A. Child abuse needs to be considered in all pediatric injuries.
   B. Subdural hematomas frequently are seen in children who fall out of bed.
   C. If the history does not seem to explain the identified injuries, the possibility of abuse should be considered.
   D. An unexplained delay in seeking medical care in an injured child is an indicator of nonaccidental trauma.

6. Which one of the following statements concerning nonaccidental head injury is *not* true?
   A. Inflicted head injury is the most common cause of traumatic death in infancy.
   B. Children with intentional injuries have worse functional outcome than children with unintentional injury.
   C. Infants will tolerate an acute brain injury better than adults because the fontanel is open.
   D. Head injuries in infants can be difficult to diagnose because the symptoms often are nonspecific.

7. When physicians fail to recognize that the child's symptoms are secondary to nonaccidental head injury, the child is frequently re-injured or has serious complications of the unrecognized, untreated head injury.
   A. True
   B. False

8. All of the following statements concerning imaging in nonaccidental pediatric head injury are true *except*:
   A. The most frequent presentation in nonaccidental trauma is a combination of subdural convexity and interhemispheric hematomas.
   B. MRI is useful in detecting small hemorrhages in infants with equivocal CT findings.
   C. The majority of subdural hematomas in children younger than 2 years are due to child abuse.
   D. Epidural hemorrhage is a common finding in shaken-infant syndrome.

9. Which one of the following statements regarding bruising in infants and children is *not* true?
   A. The dating and aging of bruises is precise.
   B. Some areas of the body such as the back, neck, and ears are atypical areas of accidental bruises.
   C. Mongolian spots often are confused with bruises.
   D. Children who are not yet walking do not have significant bruises on their bodies.

10. Severe abdominal injury is not a recognized manifestation of child abuse.
    A. True
    B. False

CME/CE Objectives

Upon completing this program, the participants will be able to:

a.) Quickly recognize or increase suspicion for non-accidental trauma in a pediatric patient;

b.) Be educated about rapid stabilization and management of a child who has sustained non-accidental injury;

c.) Understand the various diagnostic modalities available to recognize non-accidental trauma, and know the appropriate use of each modality;

d.) Integrate the ability to recognize suspicious injury patterns for non-accidental trauma into their clinical practice.

Answer Key:

1. D	6.C
2. C	7.A
3. C	8.D
4. C	9.A
5. B	10.B

In Future Issues: **ED Thoracotomy**