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Rashes are common in children, and therefore, a frequent emergency department (ED) concern. The ED physician must be familiar with common rash patterns in children—the majority of which can be diagnosed by a complete history and physical examination.

The authors review the classic course of common pediatric diseases associated with rashes, including varicella-zoster virus, herpes simplex virus (HSV), roseola, and rubella. Understanding the classic patterns, disease progression, high-risk populations, and potential complications allow the ED physician to avoid unnecessary testing in low-risk patients with a classic presentation, and aggressively approach potentially significant rashes in high-risk populations (e.g., neonatal HSV).

This article also is designed to increase ED physicians' awareness of treatment strategies associated with common viral exanthems.

Note: This article contains information about an off-label use of oral antivirals on page 84.

— The Editor

Pediatric Viral Exanthems: Distinguishing the Benign from the Serious

Authors: **Catherine A. Marco, MD, FACEP**, Associate Professor, Surgery, Medical College of Ohio; Attending Physician, St. Vincent Mercy Medical Center, Toledo, OH; **Valerie R. Lint, DO, MS**, St. Vincent Mercy Medical Center, Toledo, OH; **Jennifer A. Schuster, MD,** St. Vincent Mercy Medical Center, Toledo, OH .

Reviewer: **Alfred Sacchetti, MD, FACEP,** Chairman, Emergency Medicine, Our Lady of Lourdes Medical Center, Camden, NJ.

Introduction

Dermatologic findings are common among pediatric emergency patients. More than 50 infectious agents have been identified that may cause exanthems in the pediatric population.[1] Results of one study demonstrated that 72% of cases of fever and rash in the pediatric population were caused by viruses; 20% by bacteria.[2] Many exanthems have specific patterns of lesions, distributions, and clinical history; some of the more common exanthems are detailed in this article. However, many viral exanthems are nonspecific, and establishing a definitive etiology may not be feasible.

Important historical elements include duration of symptoms; associated symptoms (e.g., fever, headache, gastrointestinal symptoms); time course of lesions; distribution; associated symptoms (e.g., itching, pain); and exacerbating and relieving factors. Medical history, medication usage, and allergies may be useful information. The physical examination should be conducted in a focused manner. General appearance and vital signs should be taken and stabilized. Details regarding the lesions should be documented, including size, color, secondary findings (e.g., scale, excoriations), and distribution. A differential diagnosis should be established, appropriate treatment

initiated, and timely follow-up ensured. In stable cases where the diagnosis is uncertain, supportive care and timely follow-up are appropriate.

Varicella-Zoster Virus

Varicella-zoster virus is a double-stranded DNA virus and a member of the *Herpes viridae* family. It is the unique etiologic agent that causes both varicella (chickenpox) and herpes zoster (shingles). The history of these two distinct, yet closely linked, viral exanthems date to 1888 when children developed primary varicella after exposure to individuals with acute zoster infections.[3] The origin of the word chickenpox is hypothesized to be from the word "chiche-pois," French for chickpea, used to describe the vesicle size. The term zoster is a Greek word that describes the binding of one's armor, similar to the distribution of the characteristic zoster rash.[4] Without immunization, the lifetime risk of varicella-zoster virus infection is about 95%, with most reported cases (90%) involving chickenpox in children 14 years and younger.[5] The lifetime risk of zoster is approximately 10-30%, with marked increases among the elderly (older than 85 years).[6] Varicella has a global distribution, but it is more prevalent in temperate climates, occurring most often during the months of March to May. However, zoster does not share in these same epidemiologic facts of season, geographic, or climate trends. Varicella is primarily a childhood illness. Although children rarely acquire zoster, children who are immunosuppressed (who have HIV infection or cancer or who take immunosuppressive drugs) are susceptible. It is considered one of the most infectious viral diseases in humans with an estimated 80-90% transmission rate among household contacts.[7] It is contagious by direct contact with infected vesicular fluid and inoculation by airborne transmission.

Acute varicella among young, healthy, nonpregnant individuals often follows a benign, self-limiting disease course. Prior to the appearance of the rash, a 1-2 day prodromal period may occur, which includes headache, malaise, and fever. However, children may present with the rash and fever simultaneously. The typical pruritic rash of varicella begins on the head, spreads centripetally, finally reaching the extremities. Proximal extremities are more affected than distal. The rash occurs in various stages of evolution from macules to papules, and then to vesicles on an erythematous base, commonly described as "dewdrop-on-a-rose-petal." After the vesicles have formed, they develop to pustules that eventually form crust and scab, while new crops of vesicles form during a 2-5 day period. Therefore, the chicken pox lesions classically will be found at various stages of this transformation. Adults, older children, and secondary cases of varicella acquired from household contacts typically will have a more severe course, with more lesions, more systemic complaints and a higher risk of serious complications, such as pneumonia, encephalitis, and death. Other severe sequelae that may occur include skin bacterial superinfection (the most common complication among healthy children), neurologic complications, (e.g., acute cerebellar ataxia and meningoencepahalitis), varicella pneumonia (an infrequent complication among children), asymptomatic transient hepatitis, immune-mediated thrombocytopenia, and rare ophthalmologic complications (e.g., keratitis and anterior uveitis). Varicella-zoster virus infection during pregnancy carries risks both to the mother and the infant.

After the resolution of primary varicella, the virus enters the latency phase, where it lies dormant in ganglia, most commonly the thoracic and trigeminal ganglia. With time, the cell-mediated immunity to varicella will decline. In conjunction with immunosuppressive factors, the latent virus may become active. The reactivation of varicella-zoster virus appears in one or more ganglia in the appearance of herpes zoster, also known as shingles. (*See Figure 1.*) Prior to the appearance of the lesions, a prodrome

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Editorial Group Head: Valerie Loner
Managing Editor: Martha Jo Dendinger
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Figure 1. Herpes Zoster

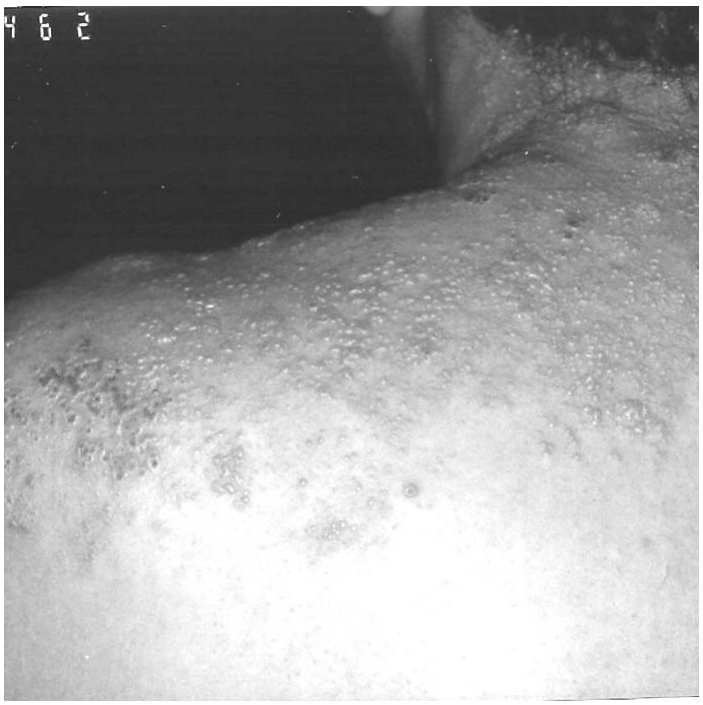
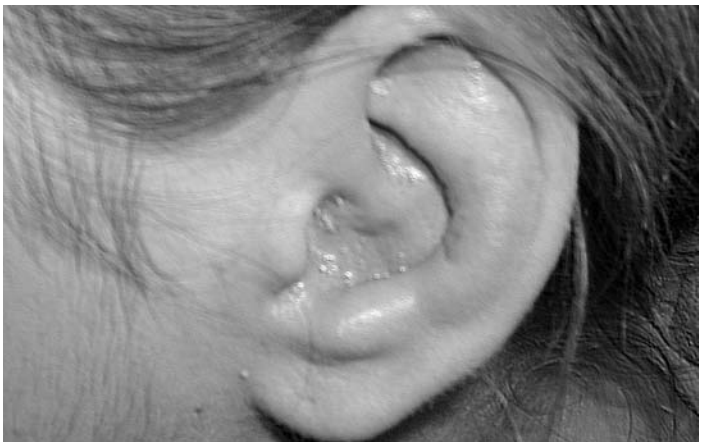


Figure 2. Ramsey-Hunt Syndrome (Herpes Zoster Oticus)



of burning, pain, itching, or tingling occurs and may last for several days. The vesicles are distributed along a dermatomal pattern, and may persist for up to four weeks if not treated with antiviral agents. Significant morbidity occurs among the elderly with the persistence of pain and dyesthesia, known as postherpetic neuralgia. Additionally, the disease course among immunocompromised patients tends to be more severe, involving multiple nerve roots, and in some cases leading to disseminated visceral disease. It is important to also examine for the presence of the ocular manifestations of varicella-zoster virus, since the virus can affect many ocular structures. Specifically, herpes zoster ophthalmicus affects the areas distributed by the ophthalmic division of the trigeminal nerve.[8] It can lead to chronic ocular inflammation, visual loss, tissue scarring, and debilitating pain. Ocular manifestations should be considered in any individual with involvement of the tip of the nose, known as Hutchinson's sign. In 1864, Hutchinson discovered that tissue involvement along the distribution of the nasociliary nerve is associated with ocular complications. Herpes zoster oticus may present with devastating otalgia coinciding with vesicular involvement of the external ear canal and pinna. (*See Figure 2*.) Ramsey-Hunt syndrome is the disease entity in which herpes zoster oticus produces a facial paralysis. The onset of pain in and around the ear, mouth, and the face may precede the rash for hours to days. Additional findings may include tongue lesions, alteration in taste, and the inability to fully close the ipsilateral eye.

The varicella vaccine was first developed in Japan in 1974. This live attenuated vaccine (Oka strain) was approved in 1995 for the use in the United States. The administration of this vaccine has affected the incidence of varicella in addition to the 80% decline in varicella-related hospitalizations.[9] The vaccine's effectiveness is estimated to be around 70-90%, with only mild, breakthrough cases. Children can receive the single-dose vaccine between 12-15 months of age; it is recommended that two doses be given 4-8 weeks apart for older children and adults. However, there remains controversy on the vaccine's lifetime immunity and the need for a booster.

In healthy children, treatment of varicella is supportive and consists of antipruritics, skin hygiene, antipyretics, and adequate hydration. The use of antiviral agents such as acyclovir has been shown to be of clinical and economical benefit, however they have not been proven to decrease the complication rate among healthy children. Therefore, their use typically is reserved for newborns, preterm infants, children older than 13 years, and adults. Because replication occurs during the first few days, it is best to start the antiviral therapy within 24 to 48 hours from the onset of the rash. Intravenous acyclovir is recommended for the immunocompromised patient to help prevent dissemination and shorten the course. Moreover, antiviral agents should be administered for all patients who present with the zoster eruption within three days of the appearance of the skin lesions. Acyclovir, valacyclovir, and famciclovir are the primary antivirals available. Disseminated zoster should be treated with intravenous acyclovir. The addition of a steroid may reduce the incidence of postherpetic neuralgia, if administered within 72 hours of the appearance of lesions.

Herpes Simplex Virus

HSVs are DNA viruses that occur worldwide in pandemic proportions and commonly result in ED visits due to their frequency and intense pain-causing syndromes.

The viruses can exist as two separate types: herpes simplex virus type 1 (HSV-1) and herpes simplex virus type 2 (HSV-2); each has characteristic antigenic differences, and separate clinical and epidemiologic manifestations.[10] HSV infections can range from benign herpes labialis, to painful genital herpes and

even life-threatening neonatal herpes. The emergency clinician will face HSV infections commonly in the ED and should be aware of the diagnostic challenges and latest treatments—particularly for neonatal herpes and disseminated HSV—for which missed diagnosis and delayed treatment can be devastating.

Herpes viruses are common etiologies for acute skin infections that may be acquired at an early age and reach 80% incidence in the general adult population.[11] Transmission of the virus is by direct contact with mucous membranes or non-intact skin. The infection rate of HSV-1 is more frequent among children (younger than 5 years) of lower socioeconomic class, and increases to 70-80% by adolescent and early adulthood. Additionally, genital herpes is one of the most common sexually transmitted diseases worldwide.

Neonatal herpes simplex virus, a devastating disease of the newborn, is estimated at one per 3,000 to 20,000 live births.[12] HSV-2 is responsible for the majority of neonatal HSV cases, but 15-30% of the cases are due to HSV-1. Primary infection of genital HSV occurring during the third trimester of pregnancy, in a patient who has not had complete seroconversion by the onset of labor, leads to a 33% incidence of transmission to the infant. However, recurrent maternal infection has only a 3% incidence of viral transmission, due to protective maternal immunity against HSV-2. Occult, asymptomatic cervical shedding of the virus nevertheless leads to many, unpredicted neonatal infections.

Other HSV infections that warrant mention, but are beyond the scope of this article, include Kaposi varicelliform eruption (eczema herpeticum), herpes whitlow (vesicular outbreaks on the hands and fingers), herpes gladiatorum (eruption on the torso of athletes and wrestlers), herpetic sycosis (follicular infection of the bearded area) and disseminated HSV infection (usually among immunocompromised patients.)

The primary infection of both HSV-1 and HSV-2 are the most severe states, characterized by clusters of painful vesicles on an erythematous base. Systemic symptoms including fever, myalgias, and headache often are present. The characteristic lesions may persist for several weeks; however, resolution of the primary infection does not correlate with eradication of the virus.[13] The virus remains latent in the neural ganglion cells where reactivation may occur, resulting in less severe, episodic outbreaks in the same patient. During the sub-clinical phase, asymptomatic shedding may occur and sexual partners should be cautioned regarding possible transmission.

HSV-1 is more likely to recur orally leading to herpes labialis, also known as cold sores. Primary infection is characterized by painful vesicles on the lips, gingiva, palate, or the tongue, often preceded by a prodromal period of fever, sore throat, and tender lymphadenopathy. Also known as herpetic gingivostomatitis, children age 1-5 years are most commonly afflicted with symptoms lasting 10-14 days, although any age may suffer from the disease.[14] Recurrences usually present as lesions on the vermillion border preceded by pain, burning, itching, and paresthesias. Recurrences may be prompted by factors such as stress, fatigue, illness, dental trauma, and ultraviolet light.

HSV-2 infections most commonly affect the genital area. Outbreaks can occur within two days to two weeks post viral exposure and lead to a painfully debilitating state. Symptoms associated with this outbreak include fever, malaise, edema, inguinal lymphadenopathy, dysuria, and vaginal/penile discharge. Up to 25% of women with primary HSV-2 infections may develop the complication of aseptic meningitis.

The diagnosis of neonatal HSV can be challenging. The presentation includes nonspecific signs and symptoms such as irritability, lethargy, fever, and failure to thrive at one week of life. Most of the neonates do not develop skin lesions that suggest the diagnosis; thus, HSV should be considered in the differential of nonspecific illness in the neonatal period, especially among premature infants and those who have a low birth weight. Infants with congenital HSV infection—4% of all neonatal HSV infections—may be born with microcephaly, hydrocephalus, chorioretinitis, and vesicular skin lesions.

Oral and labial HSV-1 infections primarily are treated supportively with topical anesthetic agents for symptomatic relief. Oral antivirals (e.g., acyclovir) also may be used, but have not been approved for pediatric use.

Treatment regimens for HSV-2 differ for primary and recurrent infections. All primary HSV genital infections should be treated with antiviral medications, which include acyclovir, famciclovir, or valacyclovir.[15] All three regimens work to inhibit viral replication, thereby reducing the duration of the painful lesions and the viral shedding. All three agents may be used for suppressive therapy for patients with recurrent outbreaks during a period of six years or longer.

Neonatal HSV must be treated aggressively and the ED physician should treat all infants 8 weeks and younger with vesicular skin lesions with empiric acyclovir.[16] Prophylactic parenteral acyclovir should be started for infants born to mothers suspected of having HSV, particularly for infants born by vaginal delivery. All neonates suspected of having neonatal HSV should undergo a full septic workup and admission.

Erythema Infectiosum and Other Parvovirus B19 Infections

Erythema infectiosum was first recognized in 1889 as fifth disease because of its position among the list of other common childhood exanthems and the chronologic order in which they were first reported.[17] These included measles, scarlet fever, rubella, and a once-distinct disease called Dukes disease. In 1974, it was discovered to be caused by the virus parvovirus B19 and subsequently renamed erythema infectiosum for the corresponding blood bank code label.[18] The virus belongs to the large *Parvoviridae* family and is a commonly transmitted human pathogen. Erythema infectiosum occurs worldwide, but seems to be more prevalent in temperate, tropical climates. It is common in childhood, with peak rates between the ages of 5 and 14 years. Transmission of the virus occurs via respiratory droplets, making

Figure 3. Fifth Disease

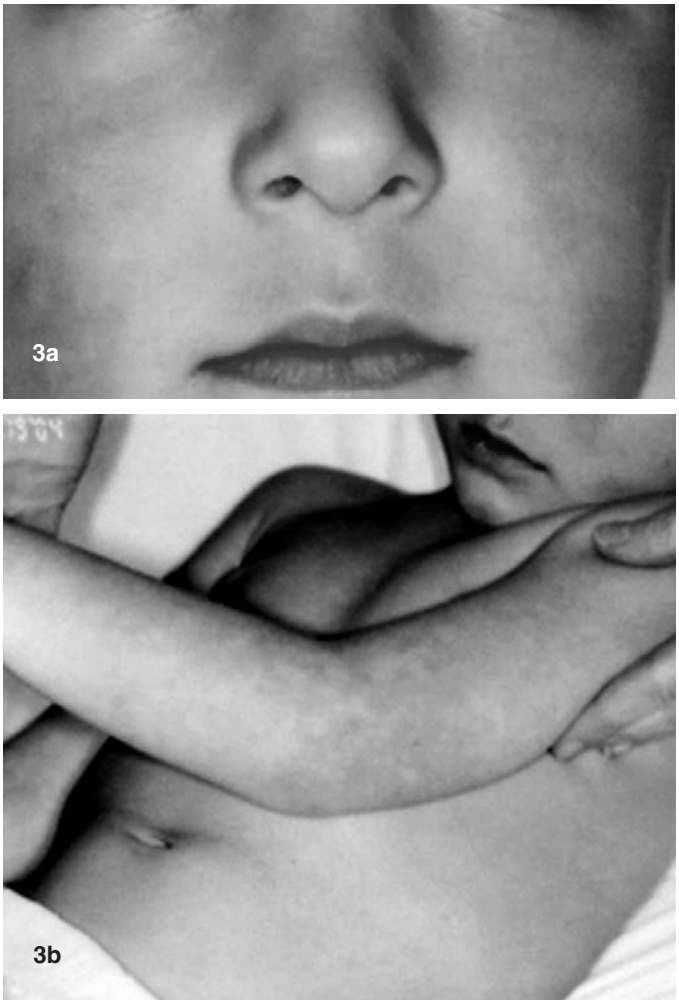


Figure 3. Fifth disease. **Figure 3a**: Note erythematous cheeks. **Figure 3b:** Reticular rash on limbs.

household transmission likely.

More than half of the cases are asymptomatic, with a rare prodromal phase that includes low-grade fever, headache, pharyngitis, myalgias, nausea, diarrhea, and joint pain.[19,20] The distinct facial erythema provides the basis for its other common name— "slapped cheek" disease, a result of red papules coalescing to form the red, edematous symmetrical cheek rash, which spares the periorbital areas and nasal bridge. The patient is no longer infectious when the cheek rash appears. Approximately two days after the onset of the facial erythema, the rash extends to the extremities in a unique, fishnet-like pattern that usually fades in 6-14 days. (*See Figure 3a and 3b.)* The slapped cheek appearance typically fades in four days. Certain triggers such as sunlight, stress, and temperature change may stimulate a recurrence during the two to three weeks following the extremity eruption.

Fifth disease is only one of several dermatologic, hematologic, and rheumatologic conditions caused by Parvovirus B19; other disease states include transient aplastic crisis, arthropathy, persistent anemia, and hydrops fetalis. It also has been suggested that Parvovirus B19 may play a role in Kawasaki's disease, Henoch-Schonlein purpura, juvenile rheumatoid arthritis (Still's disease), and giant cell arteritis.[21] Severe outcomes may occur in immunocompromised patients, the fetus, and patients with hemoglobinopathies. Patients with immunocompromised conditions, such as sickle cell, also may develop a serious, prolonged chronic anemia caused by an abrupt cessation of red-cell production resulting from a B19 infection. Other severe sequelae include the transplacental transmission to the fetus, which may lead to miscarriage or hydrops fetalis.

Management of patients with erythema infectiosum is symptomatic. Most patients do well with simple treatment measures including antipyretics, oral hydration, and antipruritic agents, if needed. Other treatments may be indicated for significant complications, including blood transfusions for patients with chronic hemolytic anemias who develop transient aplastic crisis and serial fetal ultrasounds for pregnant patients with positive IgM and IgG levels supporting parvovirus infection. There are no published recommendations for children returning to school once the diagnosis is made; however, children may return to school with the appearance of the facial rash. However, patients with aplastic crisis may be contagious for extended periods.

Pityriasis Rosea

Pityriasis rosea is a self-limited papulosquamous eruption frequently seen among adolescents and young adults during the fall and winter months. It is characterized by a classic rash that assumes a storiform or Christmas tree pattern and often is preceded by a small, circular lesion termed a "herald patch."

The exact etiology is still unclear, despite clinical and epidemiological evidence that suggest an infectious cause. Clinical support for a viral origin includes the prodromal upper respiratory symptoms, the characteristic rash pattern, and the spontaneous resolution of the lesions.[22] Epidemiological features that support a viral cause include community outbreaks and the peak incidence during the cold months.[23] Results from one study provided evidence of a viral etiology for pityriasis rosea by detecting human herpes virus-7 DNA in peripheral blood mononuclear cells from patients with acute pityriasis rosea.[24] However, these results have not been confirmed with subsequent studies.[25-27] The incidence of pityriasis rosea peaks at the ages of 20 to 29 years without a significant gender bias.[28-30]

The diagnosis of pityriasis rosea is made clinically, based upon history, physical examination, and the unique timing and appearance of the rash. Prior to the appearance of the generalized rash, a herald patch may be seen in 50 to 90% of cases. (*See Figure 4a*.) This single, circular lesion with defined borders precedes the rash by several days to a couple of weeks and often is confused with tinea corporis or nummular eczema. The lesion can be found most commonly on the neck or lower trunk area, but does occasionally appear on the extremities. The pathogno-

monic finding of the disease is the subsequent eruption of multiple, round to oval-shaped, salmon-colored plaques that follow the lines of skin cleavage creating a Christmas tree pattern. (*See Figure 4b*). These plaques are covered with a fine, white scale, which may be described as a "cigarette-paper" scale forming a collarette. The individual lesions may appear hyperpigmented in African-American patients, but are generally pink in nature. The rash appears rapidly over the trunk, neck, and proximal extremities. Unlike secondary syphilis, the palms and soles generally are spared. Pruritis is a common complaint and often persists 1 to 2 months before resolving spontaneously. A significant number of cases are preceded by symptoms of a mild, upper respiratory infection, but pityriasis itself is not considered contagious. Pityriasis rosea may have an atypical presentation including face and oral mucosal lesions in addition to involving the palms and soles.

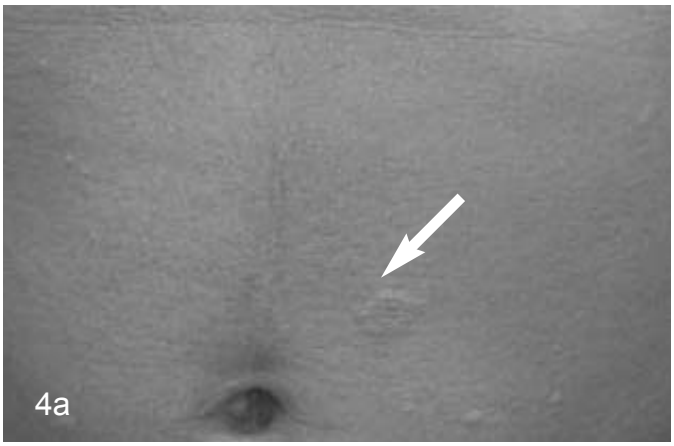
Other diseases to be considered in the differential diagnosis include tinea corporis, tinea versicolor, secondary syphilis, guttate psoriasis, nummular eczema, lichen planus, and drug eruptions.

Educating patients about the extended course of the illness is essential. Unlike other viral exanthems, pityriasis may last up to three months. While no workup for pityriasis is necessary, atypical presentations or symptoms that persist beyond the typical time course may indicate that further diagnostic tests are indicated.

Treatment is supportive and may include topical corticosteroids and antihistamines to provide relief of the prominent itching. Ultraviolet B light (UVB) may decrease the severity of the disease if used within the first week of eruption but should be done in conjunction with a dermatologist. Recurrence is rarely seen, and occurs twice as commonly in blacks (6%) as compared with whites (2% to 3%).[31]

Pityriasis is a self-limited disease frequently seen in the ED due to the prominent pruritis. For the emergency clinician, classic presentations require supportive treatment. For atypical or prolonged presentations, other more serious differential diagnosis, such as syphilis, should be ruled out.

Figure 4. Pityriasis Rosea in a 13-year-old Male



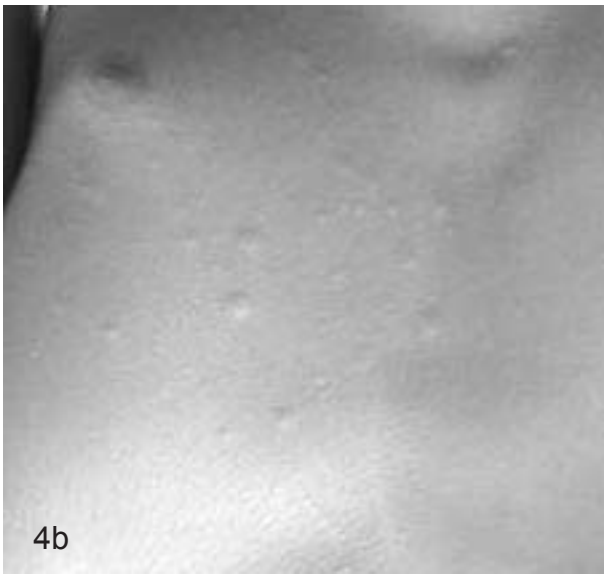


Figure 4a. Pityriasis Rosea in a 13-year-old male. Note herald patch on abdomen.
Figure 4b. Rash of pityriasis rosea.

Rubella

The rubella virus is a single-stranded RNA togavirus. It is spread by the respiratory route via nasopharyngeal droplets. The incubation period of rubella is estimated to be 2 to 3 weeks. Exposed persons are infectious prior to the outbreak of the exanthem; the infectious period continues for about five days after rash develops.[32,33]

Clinically, rubella typically begins as a mild respiratory illness.[34] Fever, headache, malaise, and anorexia are common symptoms. However, young children often have no prodromal symptoms prior to the appearance of the rash. Tender suboccipital, posterior cervical, and postauricular adenopathy are common. Typically, rubella is a mild, viral illness and results in minimal morbidity and mortality. Thrombocytopenia and encephalitis occur infrequently in children. Complications are more prevalent in adolescents and adults.

The rubella exanthem begins as macules and papules on the face, scalp, and neck.[35] The rosy pink lesions quickly spread to the rest of the body, including the hands and feet. Forchheimer's sign, seen in 20% of rubella cases, is a soft palate eruption of petechial spots. The exanthem appears and resolves over a three-day course, thus the term "three-day measles." Pruritus is common with the rash. Mild desquamation often occurs.

Rubella has become rare since the advent of the rubella vaccine in 1969. Incidence of rubella is less than one in 100,000. Although rare, congenital rubella remains a devastating condition. A fetus has the greatest chance of contracting rubella in the first 12 weeks of gestation. Risk declines as gestation progresses. Deafness, heart disease, and cataracts compose the classic triad of congenital rubella syndrome. Hearing loss results from sensorineural damage. Patent ductus arteriosus is the most common cardiac manifestation. Cataracts are usually bilateral, and

retinopathy also may be present. Neurological conditions, such as encephalitis and mental retardation, are also common. Chronic rubella infection results in growth retardation, hepatosplenomegaly, thrombocytopenia, jaundice, and purple skin lesions. An infant with congenital rubella is infectious for the first year of life.

The diagnosis of rubella can be made clinically. Suboccipital lymphadenopathy, rosy pink lesions, and an exanthem lasting three days are the clinical hallmarks of rubella. Serology may be performed for definitive diagnosis. Therapy is supportive only.

Rubeola

The measles virus is a single-stranded RNA paramyxovirus. Rubeola is endemic, highly contagious, and is a cause of significant morbidity and mortality. Prior to the advent of the measles vaccination, 95% of the population contracted measles by age 15 years. The virus is spread by both aerosolized respiratory droplets and direct contact. The virus remains virulent for at least two hours as droplets; the incubation period is typically 10-12 days. The carrier is infectious from the prodromal period until four days after the exanthem outbreak.[36]

The number of reported cases of measles has dropped dramatically in the United States since the advent of the measles vaccine. In 1998 and 1999, fewer than 70 cases were reported in the United States compared with the 4-5 million cases per year prior to immunization. Measles is most likely to infect preschoolers in low-income homes or in heavily populated areas. Most cases result from either noncompliance with recommended immunizations or primary vaccination failure. The endemic and virulent nature of measles obviates the need for its eradication. Worldwide, it is the No. 1 cause of vaccine-preventable death. The United States has decreased cases by 99% through routine series of two measles-mumps-rubella (MMR) vaccinations. The World Health Organization still is trying to decrease measles incidence in other parts of the world where it remains prevalent.[37] Rubeola is seen most often in the spring and winter.

Clinically, rubeola first appears as an influenza-like illness. The prodrome includes a high fever of 104-105ºF, malaise, and the classic C's of measles—cough, coryza, and conjunctivitis. The symptoms are present for 2-4 days prior to exanthem outbreak. The measles exanthem begins as erythematous macules and papules along the hairline and behind the ears. It spreads from the head in a downward and outward pattern. As time passes the initial lesions coalesce. The rash changes from a blanching, bright red rash to a brownish, non-blanching hue within 2-4 days of initial onset. Mild desquamation often occurs.

Koplik spots are pathognomonic for measles. The enanthem is on the buccal mucosa at the second molar. Lesions are bluish-white on an erythematous base. The Koplik spots are present during the prodromal phase and usually nonexistent at rash onset.

Laboratory results often show leukopenia with lymphocytopenia. Generalized lymphadenopathy and splenomegaly may be seen as extracutaneous manifestations of measles.[38] Complications may include pneumonia, otitis media, and diarrhea. Children younger than 5 years most often incur complications. Pneumonia is the most common cause of death in children with measles. Hepatitis, thrombocytopenia, and encephalitis also may occur.

Three types of encephalitis may occur as complications from rubeola. Acute post-infectious measles encephalitis (APME), occurs about two weeks after rash onset. Clinical features include fever, altered mental status, seizures, and headache. Periventricular demyelination has been found in these patients. APME mortality is 10-20%. Measles inclusion body encephalitis (MIBE), manifests 1-6 months after primary measles infection. It affects immunocompromised individuals as a result of continued viral replications in neurons. Typically, no fever occurs, but altered mental status and seizures are present. One in 1 million cases of measles in the United States leads to the development of subacute sclerosing panencephalitis (SSPE), which lies dormant for 7-10 years after the initial measles infection. Inflammation and demyelination of cortical and subcortical gray and white matter occurs, and clinical symptoms arise, including slowly progressive mental retardation, motor dysfunction, seizures, coma, and death.[39]

Atypical measles has declined since the advent of the live attenuated vaccine. Exposure to wild type measles following killed measles vaccination may result in atypical measles. Although rare, this also has occurred following live attenuated vaccination. Koplik spots, conjunctivitis, and runny nose are absent. The illness comprises fever, myalgias, headache, cough, and rash. However, symptoms are more severe, and the exanthem differs in location and appearance. Rash begins at the wrists and ankles as well as the palms and soles. The rash spreads inward and upward. This presentation may be confused with Rocky Mountain spotted fever. The exanthem varies from maculopapular to vesicular and may be erythematous to purpuric. Pneumonia almost always is found in these patients, and pleural effusions are common.

Vaccination is available in a combination with mumps and rubella vaccine—MMR—or as a single agent vaccine. Both are live attenuated vaccines. Any patient with anaphylaxis to eggs or neomycin may have a severe, allergic reaction to the measles vaccine. Vaccination is contraindicated in pregnancy, immunocompromised patients, and cancer patients. Previously raised questions of the measles vaccination as the cause of inflammatory bowel disease and autism have been studied. No evidence has been found to support such claims. In approximately 10% of patients, mild fever and rash may develop after immunization. Transient thrombocytopenia is a recognized side effect of vaccination.

If a measles case is suspected, the individual should be isolated. Anyone exposed to measles who has not been vaccinated, who is pregnant, or younger than 1 year should receive prophylaxis with immunoglobulin within six days of contact. The measles vaccine should be given five months later.

Diagnosis of measles can be made clinically, but it is difficult prior to onset of the exanthem. For definitive diagnosis, acute

and convalescent titers can be measured or enzyme-linked immunosorbent assay (ELISA) can be performed. During epidemics, collection of specimens should be done for genotyping.

Treatment of measles is primarily supportive, and should include oral hydration, antipyretics, and analgesics. Pneumonia, otitis media, persistent fever, or other significant illness may necessitate hospital admission and/or antibiotic treatment. Ribavirin may be helpful in complicated measles complications. If the patient is in an area deficient in vitamin A, 400,000 units should be given as it has been shown to decrease morbidity and mortality.

Roseola Infantum

Roseola is thought to be of viral origin, yet no specific entity has been isolated. The rotavirus and human herpes virus-6 have been reported as sources of roseola. Several other viruses also have been reported to be associated with roseola. The incubation period is 1-2 weeks. Roseola infects males and females equally. Most cases occur in spring and early fall. Children between 6 months and 3 years of age account for nearly all cases of roseola.[40]

Clinically, fever is the first symptom, and may be 104-105ºF. Fever may be persistent or intermittent. The febrile child is playful and nontoxic in appearance. Occasionally, febrile seizures occur. On physical exam, cervical lymphadenopathy, pharyngeal erythema with or without exudates, and otitis media may be found.

The exanthem appears suddenly after fever resolution, which is where the name "roseola subitum" originated. The rash begins on the trunk and spreads upward to the neck and proximal extremities. It is pink in color and can be macular, papular, or maculopapular. The exanthem may be as short in duration as a few hours, or it may last up to 2 days. No pruritus or flaking occurs.[41]

Diagnosis is made clinically. High fever in a well-appearing child, rapid decline of fever, and sudden onset of rash are the keys to clinical diagnosis. Leukopenia with lymphocytosis may be seen on laboratory examination. Therapy is supportive and may include antipyretics and oral hydration.

Infectious Mononucleosis

Infectious mononucleosis may affect any pediatric age group and is caused by the Epstein-Barr virus (EBV), a DNA herpes virus.[42] Because infection is often mild and undiagnosed in early childhood, most clinically recognized cases occur in adolescents and young adults.[43] Most adults have been exposed and have titers against the Epstein-Barr virus. There is no typical seasonal variation. The virus is transmitted via salivary secretions. The incubation period is typically 30-50 days. Clinical symptoms typically include fever, headache, malaise, fever, pharyngitis, and lymphadenopathy. Splenomegaly and hepatomegaly also may occur. Symptoms may last from a few days to several months. Significant complications of hemolytic anemia, aplastic anemia, pneumonitis, and neurologic problems occur rarely.

The exanthem of infectious mononucleosis has been estimated to occur in approximately 5-50% of cases. The typical appearance is a nonspecific maculopapular eruption, although petechial, scarletiniform, erythema multiforme, and urticarial lesions also may be seen.[44] The lesions typically appear about day four of the illness. The trunk and arms often are involved early, and lesions may spread to the face and remainder of the body. Patients who receive ampicillin or amoxicillin are at increased risk of developing the rash, and up to 80% of patients with mononucleosis who are treated with ampicillin may develop the rash. The drug-related rash is thought to be due to production of drug-specific antibodies with immune complex deposition in the skin. The drug-related eruption typically occurs 7-10 days after initiation of antibiotic therapy. The rash is not associated with subsequent penicillin allergies.

Infectious mononucleosis also may be associated with other dermatologic findings. Erythema nodosum, exacerbation of preexisting acne vulgaris, urticaria, and anaphylaxis have been reported.

The potential for splenic rupture should be considered in cases of infectious mononucleosis. Splenomegaly occurs in approximately 8-50% of patients[45-47] and often is undetected clinically. Splenic rupture occurs in 0.1-0.5% of cases,[48] and usually occurs in the first three weeks of illness, with most (80%) cases occurring in patients age 25 years and younger.[49]

Laboratory studies are considered optional, as the diagnosis is primarily clinical. The heterophile antibody test is available, and the sensitivity varies from 63-84%; specificity is between 84-100%. The Monospot heterophile antibody test commonly is used, and becomes positive 5-7 days after onset of symptoms, and may be falsely negative if performed early in the disease course. EBV serology may be performed to confirm acute infection. Liver transaminases may be elevated modestly, and peak at 2-3 weeks.

Treatment consists of supportive care, including adequate rest, hydration, nutrition, antipyretics, and analgesics. Steroids may be prescribed for patients with significant pharyngitis and dysphagia. Patient education is essential, particularly regarding prognosis and supportive care. Contact sports should be avoided, although the precise time until return to full activity is debatable. Published recommendations include abstinence from contact sports for 3 weeks to 6 months.

Hand, Foot, and Mouth Disease

Hand, Foot and Mouth Disease (HFMD) refers to a viral exanthem first described in 1958.[20] It usually is caused by enteroviruses, including coxsackievirus A16 and enterovirus 71. Cases have occurred less commonly from infection with coxsackie A4-7, A9, A10, B1-3, and B5. HFMD is highly contagious and is spread by fecal-oral contamination. Pediatric patients younger than 10 years—typically younger than 4 years—are affected most commonly. Epidemics often occur in the late summer and early fall, although sporadic cases often are

seen. In the United States, epidemics may be seen approximately every three years.

Clinical manifestations are quite predictable. Following the incubation period of 3-6 days, a prodrome may occur with fever, malaise, cough, anorexia, and abdominal pain. Within the next few days, clinical manifestations typically include oral lesions, followed by vesicular lesions on the hands and/or feet. Lesions may begin as erythematous papules and macules, and/or vesicles, which typically progress to erosions or ulcers, and disappear during the ensuing 5-10 days. The lateral and dorsal aspects of fingers commonly are involved. Lesions often occur in a pattern along skin lines. Oral lesions occur most commonly on the hard palate, tongue, and buccal mucosa. Oral intake may be compromised by pain.

Complications are rare.[50] Scarring is rare. Reported complications include myocarditis, meningoencephalitis, pulmonary edema, and death. Neurologic complications including myoclonus, paralysis, meningitis, and encephalitis have been reported.[51-53]

Treatment consists of supportive measures to decrease discomfort and improve oral intake. Malaise and pain may be treated with antipyretic agents, such as acetaminophen or ibuprofen. Oral lesions may be treated symptomatically with diphenhydramine elixir, or magnesium hydroxide. Although acyclovir has shown improvement of symptoms in one small study,[54] the lack of large-scale studies demonstrating efficacy currently precludes routine use.

Other Enteroviral Exanthems

A variety of enteroviruses may cause symptoms including fever, malaise, gastrointestinal complaints, meningitis, and rash. Infections tend to peak in summer and autumn months. The enterovirus exanthem typically exhibits a nonspecific maculopapular eruption; although petechiae, mimicking a meningococcal infection, may be seen.[55] Petechiae have been reported with coxsackievirusA9, A4, B2-5, echovirus 9, and echovirus 3,4, and 7 infections.[56]

Conclusions

Pediatric viral exanthems occur commonly among the emergency pediatric population. Accurate historical information and physical examination can be crucial in establishing the correct diagnosis. Many viral exanthems are benign and resolve spontaneously with supportive care. Certain exanthems require specific interventions and have been addressed in this manuscript.

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Physician CME Questions

61. Which of the following medications increases the likelihood of developing a rash associated with infectious mononucleosis?
    A. Acetaminophen
    B. Ampicillin
    C. Aspirin
    D. Ibuprofen
    E. Trimethoprim-sulfamethoxazole

62. Which of the following treatment modalities *is not* appropriate for infectious mononucleosis?
    A. Antipyretics
    B. Analgesics
    C. Antibiotics
    D. Hydration
    E. Steroids

63. Which of the following agents commonly causes Hand, Foot and Mouth Disease?
    A. Coxsackievirus A16
    B. Parvovirus B19
    C. Herpes zoster virus
    D. Epstein-Barr virus
    E. Human herpes virus-6

64. In which of the following months would primary varicella-zoster infection most likely occur?
    A. November
    B. March
    C. August
    D. October
    E. December

65. Which of the following conditions is a potential sequelae of acute varicella?
    A. Acute cerebellar ataxia
    B. Pneumonia
    C. Transient hepatitis
    D. Immune-mediated thrombocytopenia
    E. All of the above

66. What of the following treatments is most appropriate for erythema infectiosum (fifth disease)?
    A. Preventive immunization
    B. Intravenous immunoglobulin
    C. Topical antiviral agents, such as acyclovir
    D. Oral antiviral agents, such as acyclovir
    E. Supportive care and patient education

67. Which of the following clinical conditions is synonymous with measles?
    A. Roseola
    B. Erythema infectiosum
    C. Rubeola
    D. Roseola subitum
    E. Mumps

68. Which of the following statements is *not* true regarding varicella?
    A. Adults and secondary cases of varicella acquired from household contacts typically have a more severe disease course.
    B. Acute varicella among young, healthy, nonpregnant individuals typically follows a benign, self-limiting disease course.
    C. Varicella is primarily a childhood illness.
    D. It is not very infectious with transmission rates estimated at 20-30%.

69. A 3-year-old female presents with facial redness, followed by a reticular rash on the extremities. The child is happy, playful, and lacks systemic symptoms. The most likely diagnosis is:
    A. erythema infectiosum.
    B. roseola.
    C. rubella.
    D. infectious mononucleosis.
    E. varicella-zoster.

70. An 18-month-old child presents with a rash. His mother states that the child ran a high fever for two days, but was happy and playful. Yesterday, the fever resolved, and his mother noted a pink maculopapular rash that started on the trunk, and then spread to the face. The child is happy, playful, and has some mild, pharyngeal erythema. The most likely diagnosis is:
    A. erythema infectiosum.
    B. roseola.
    C. rubella.
    D. infectious mononucleosis.
    E. varicella-zoster.

Answer Key:

61. B
62. C
63. A
64. B
65. E
66. E
67. C
68. D
69. A
70. B.

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.

CME Instructions

Physicians participate in this continuing medical 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 that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

In Future Issues:

Toxicology

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Pediatric Viral Exanthems

Fifth Disease

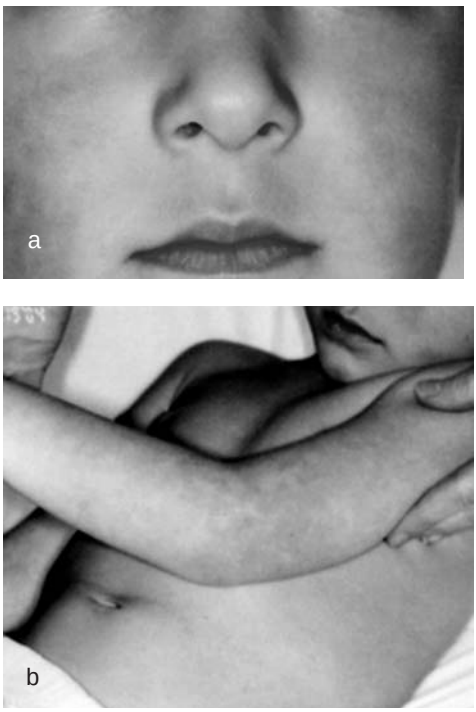
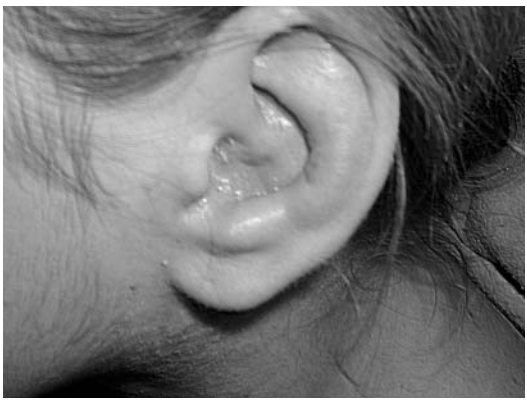


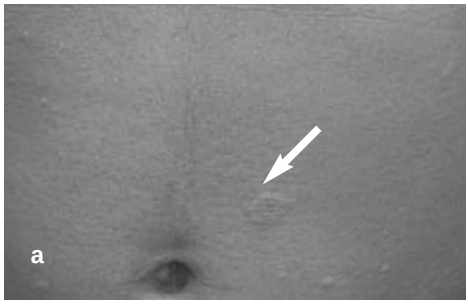
Figure. Fifth disease. **Figure a**: Note erythematous cheeks. **Figure b**: Reticular rash on limbs.

Ramsey-Hunt Syndrome (Herpes Zoster Oticus)



Herpes Zoster

Pityriasis Rosea in a 13-year-old Male



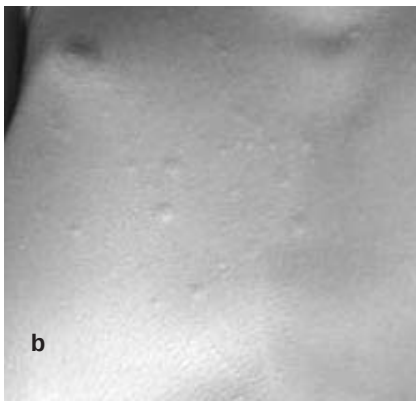


Figure a. Pityriasis Rosea in a 13-year-old male. Note herald patch on abdomen.
Figure b. Rash of pityriasis rosea.

Supplement to *Pediatric Emergency Medicine Reports*, July 2004 "Pediatric Viral Exanthems: Distinguishing the Benign from the Serious" *Authors:* **Catherine A. Marco, MD, FACEP**, Associate Professor, Surgery, Medical College of Ohio; Attending Physician, St. Vincent Mercy Medical Center, Toledo, OH. **Valerie R. Lint, DO, MS**, St. Vincent Mercy Medical Center, Toledo, OH; **Jennifer A. Schuster, MD**, St. Vincent Mercy Medical Center, Toledo, OH. *Peer reviewer:* **Alfred Sacchetti, MD, FACEP,** Chairman, Emergency Medicine, Our Lady of Lourdes Medical Center, Camden, NJ. *Pediatric Emergency Medicine Reports' "Rapid Access Guidelines."* **Vice President and Group Publisher:** Brenda Mooney. **Editor-in-Chief:** Ann Dietrich, MD, FAAP, FACEP. **Editorial Group Head:** Valerie Loner. **Managing Editor:** Martha Jo Dendinger. For customer service, call: **1-800-688-2421.**

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Supplement to *Emergency Medicine Reports*, *Pediatric Emergency Medicine Reports*, *ED Management*, and *Emergency Medicine Alert*

July/August 2004

Dealing with potential cervical spine injuries (CSIs) is a frequent occurrence for any physician who manages trauma patients. The importance of the routine practice of clearing the cervical spine should not be underestimated. Missed spine fractures may have devastating consequences for both the patient and the physician. Although evaluating a patient with a potential CSI is common, many controversies exist regarding immobilization, imaging selection, and management strategies. Since, fortunately, CSIs are relatively uncommon, large, controlled series with definitive answers do not yet exist. The author has created an outstanding, two-part article that presents the evidence currently available for evaluation and clearance of the cervical spine in adult trauma patients. The first part of this series reviews cervical spine immobilization, patient selection for imaging and clinical decision rules for cervical spine radiography. This two-part article is critical for any physician who manages patients with potential cervical spine trauma.

—The Editor

The Evaluation and Clearance of the Cervical Spine in Adult Trauma Patients: Clinical Concepts, Controversies, and Advances, Part 1

Authors: **S.V. Mahadevan, MD, FACEP**, Assistant Professor of Surgery/Emergency Medicine, Stanford University School of Medicine; Associate Chief, Division of Emergency Medicine, and Medical Director, Stanford University Emergency Department, Stanford, CA; and **Misty Navarro, MD**, Senior Emergency Medicine Resident, Stanford-Kaiser Emergency Medicine Residency, Stanford, CA.

Reviewer: **Andrew D. Perron, MD, FACEP, FACSM**, Residency Program Director, Maine Medical Center, Portland, ME.

Introduction

The evaluation of the cervical spine for potential injury is viewed widely as one of the most challenging aspects of trauma care. Though neck pain and tenderness are common findings in trauma patients presenting to the emergency department (ED), CSI is found in only 0.9-6% of all blunt trauma patients.[1-5] The emergency physician's goal is to identify patients with injuries that could result in permanent dysfunction or catastrophic disability. The implications of a missed injury are profound and could have life-altering consequences for the both the patient and physician. Lifetime medical care costs exceed $1 million per injury victim, while the annual nationwide care costs for these patients exceed $2 billion.[6] In one series, missed spinal fractures were responsible for 3% of malpractice claims, and ensuing spinal cord injuries accounted for more than 9% of total dollars paid in claims.[7]

Therefore, it is not surprising that this area has been the subject of extensive research and controversy in an attempt to define the most accurate and cost-effective approach to clearing the cervical spine. This two-part review article will describe the evalua-

tion of traumatic CSIs, including when to suspect CSIs, how to protect the cervical spine from further injury, the practice of clinical cervical spine clearance, radiographic clearance of the cervical spine, the diagnosis of isolated ligamentous injury, and the syndrome of spinal cord injury without radiographic abnormality (SCIWORA). This review will focus primarily on patients 14 years and older as anatomic and biomechanical features of the pediatric cervical spine differ significantly from those of the adult. For further information regarding pediatric cervical spine injuries please refer to the July 2003 edition of *Trauma Reports*, "Pediatric Cervical Spine Injuries: Avoiding Potential Disaster."

Suspicion of Cervical Spine Injuries

More than 1 million blunt trauma patients with potential CSIs are evaluated in U.S. EDs each year; approximately 30,000 of these patients have cervical spine fractures or dislocations, and 10,000 of these patients suffer spinal cord injury.[3,8,9] Fractures of the cervical spine are not always readily apparent. CSIs are the most commonly missed severe injury with serious implications for the patient and physician.[10] Failure to identify the neurologically intact patient with an unstable fracture or ligamentous injury may lead to the development or progression of severe neurologic compromise. Patients with a delayed or missed initial diagnosis of CSI have been found to have an associated 7.5-fold increase in the incidence of secondary neurologic injury as compared with patients in whom the diagnosis is made initially.[9] One group of authors emphatically states, "A single missed unstable CSI is an unacceptable disaster."[11] Considering the large number of trauma patients evaluated each year and the potential consequences of a missed CSI, the question arises: In whom should we suspect CSI? While it is true that all blunt trauma victims are potentially at risk, certain historical and clinical features may raise the clinician's level of suspicion.

On a global level, traffic accidents involving motor vehicles, bicycles, or pedestrians account for the majority of CSIs and almost 50% of all spinal cord injuries.[12] Falls, sporting and recreational accidents, work related injuries, and violence are responsible for the remainder of adult spinal cord injuries.[12] Falls account for the most frequent cause of cervical spine injuries in adults older than 65 years.[13-15] Some authors have attempted to identify patients with characteristics placing them at increased risk for CSI. (*See Table 1*.) Generally accepted, high-risk mechanisms of injury include high-velocity blunt trauma (e.g., motor vehicle accidents at speeds more than 35 mph), motor vehicle accidents with a death at the scene, a drowning or diving accident, and falls from higher than 10 feet.[16-19] While the mechanism of injury alone has not been shown to be a predictor of clinically significant CSI, providers should maintain a high index of suspicion when evaluating patients from such accidents.[17,20] Concomitant use of alcohol or other intoxicants in these settings also should raise the possibility of injury, as their use is associated with increased risk-taking behavior and may produce an unreliable clinical examination. High-risk physical exam findings include evidence of direct cervical injury, the presence of pelvic or extremity fractures, altered mental status, significant head or facial injury, evidence of a thoracic or lumbar fracture, and paresthesias or burning in the extremities.[16-19]

Patients with a history of a prior cervical spine disease or surgery, or disease states characterized by a rigid spine (e.g., ankylosing spondylitis, diffuse idiopathic hyperostosis) are also at increased risk of CSI. In this subset of patients, less force generally is required to produce an injury; therefore, what might be considered relatively minor trauma actually may result in a significant CSI.[21] These patients generally suffer from chronic pain; therefore, differentiating an acute vertebral injury from a chronic process may be more difficult.

Patients older than 65 years have an increased relative risk (RR 2.09; 95% CI 1.77-2.59) of CSI.[22] The prevalence of cervical spine fractures among elderly patients is greater than all other age groups.[23] This fact has been attributed to the tendency of elderly patients to fall, their increased risk for motor vehicle accidents, and the significant incidence of senile osteopenia.[13,14] Unlike young adults, elderly patients are more likely to sustain injuries from low-energy mechanisms. In one study, falls from standing or seated heights—a mechanism unlikely to produce

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Editorial Group Head: Valerie Loner
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Table 1. High-risk Criteria for CSI

- High-velocity blunt trauma
- Significant motor vehicle accident (rollover, ejection)
- Direct cervical region injury
- Falls/diving injuries
- Rigid spine (i.e., ankylosing spondylitis)
- Significant head/facial injury
- Altered mental status (including alcohol, drugs, intoxicants, loss of consciousness, and mental illness)
- Abnormal neurologic exam
- Prominent neck pain/tenderness
- Thoracic/lumbar spine fracture

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injury in young adults—accounted for 30% of CSIs in those 65 years or older and 40% of injuries in patients older than 75 years.[13]

Protecting the Cervical Spine

During the initial evaluation of any trauma patient, assume that the cervical spine may be injured. An important consideration in such patients is the prevention of pathological motion of injured cervical vertebrae, which may create or exacerbate a spinal cord injury. (*See Figures 1A and 1B.*) It is estimated that 3-25% of spinal cord injuries occur after the initial traumatic insult, either during transport or early in the course of management.[24] Rogers et al found that 10% of spinal cord injuries occurred or were exacerbated following the initiation of medical care.[25] The mishandling of such patients has been associated with poor clinical outcomes.[24]

As the unstable cervical spine is prone to additional injury from manipulation, complete spinal immobilization has been utilized to limit motion until a spinal injury has been excluded. Proper and secure spinal immobilization is accomplished with a long backboard, a rigid cervical collar, lateral support devices (e.g., tape blocks or towel rolls), and tape or straps to secure the patient to the backboard.[26] Padding may improve positioning and comfort.

Early management of the patient with a potential CSI begins with proper immobilization at the scene of an injury. Emergency medical service personnel are trained to immobilize any trauma victim with potential for CSI—symptomatic or not. As a result, cervical spine immobilization is one of the most frequently preformed prehospital procedures.[26] An estimated 5 million patients are immobilized at a cost of $75 million a year in the United States.[27] This conservative practice arose from reports citing lack of immobilization as the cause of neurological deterioration among acutely injured trauma patients transported to medical facilities for definitive care. In a retrospective case series report, 26% of the 123 trauma patients reviewed suffered major neurologic deterioration prior to admission to the hospital as a result of patient mishandling. The report cited failure to immobilize the patients as the primary cause.[28] Though immobilization makes sense and provides theoretical protection, no case control studies

Figure 1. Trauma Patient with a Cervical Spine Fracture

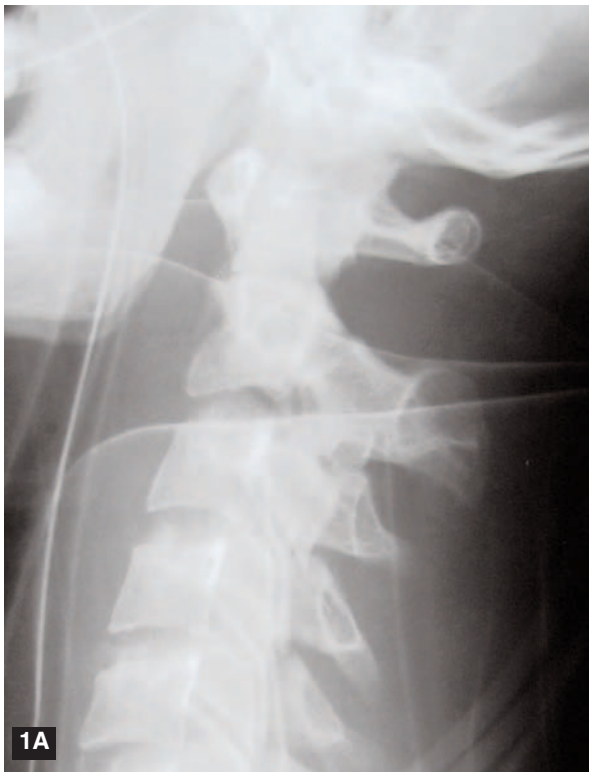


Figure 1A: A trauma patient with an unrecognized unstable dens (C_2) fracture.

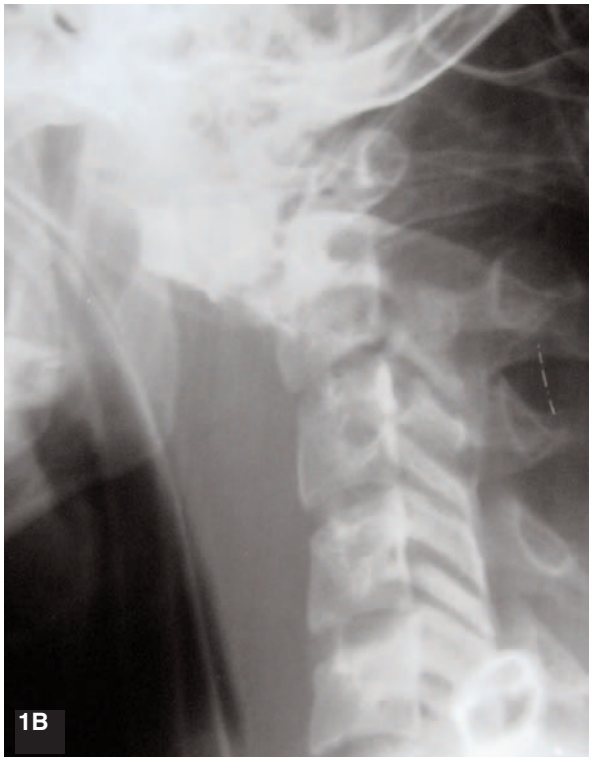


Figure 1B: Inadvertent neck flexion leads to subluxation of the dens fracture and narrowing of the spinal canal.

Images courtesy of S.V. Mahadevan, MD, and Michael Zucker, MD.

or randomized trials have addressed the effect of spinal immobilization on clinical outcomes after CSI.[24]

Furthermore, spinal immobilization carries inherent risks and has a modest morbidity. Complaints of occiput and back pain are common following immobilization, and immobilization also may result in unnecessary radiographic evaluation.[29] Immobilization increases the risk for aspiration, impairs normal respiratory function, and places patients at risk for pressure sores.[30,31] Patients at greatest risk for pressure sores are those who remain on the spine board for more than two hours without being turned.[32] The application of rigid cervical collars is associated with elevation of intracranial pressure (ICP) and potentially could exacerbate a concomitant head injury.[33] In certain cases, the application of the hard collar itself actually may do more harm than good. Papadopoulos et al describe a case of an 82-year-old male with ankylosing spondylitis and a fixed flexion deformity of the cervical spine who walked into an ED complaining of neck pain after a fall. After plain films revealed a fracture at C_6, the patient was placed in a rigid cervical collar (*See Figure 2A.*), subsequently leading to an iatrogenic spinal cord injury and death.[34] Rather than forcing a patient with fixed flexion deformity into a neutral position, it may be better to simply immobilize the patient in flexion to prevent further injury. (*See Figure 2B.*)

While evaluating a patient with a potential CSI, it is important to maintain cervical spine precautions until an injury has been excluded. Techniques for moving patients with spine fractures are designed to protect and preserve neurologic function. The logroll maneuver commonly is employed to remove a patient from the backboard or examine the thoracic and lumbar spine. In this technique, one person stabilizes the head and neck while assistants turn the body, turning the patient as one unit. (*See Figure 3.*) The logroll maneuver should be performed with care as it presents an opportunity for movement of the spine at an unstable segment.[35] Alternatives to the logroll maneuver include the HAINES method (High Arm IN Endangered Spine) and the multihand/fireman lift.

If the patient requires emergent airway management, precious time should not be wasted obtaining a single lateral radiograph of the cervical spine to exclude a CSI. This approach delays definitive airway management and provides a false sense of security; a single view is inadequate to exclude a CSI. Numerous studies have shown that the proper, safe, and effective approach to managing these patients is rapid sequence intubation with in-line immobilization.[36-40] This approach does not significantly increase the risk of neurologic injury in unstable cervical spine fractures. Paralyzing the patient reduces the risk of patient movement during intubation while immobilization of the head and neck in the neutral position throughout the procedure prevents hyperextension of the neck by the laryngoscopist. (*See Figures 4A and 4B.*)

Clinical Decision Rules for Cervical Spine Radiography

In addition to maintaining a high level of suspicion of possible CSI, trauma care providers should have an organized, thoughtful approach to the evaluation of these patients. The high-risk nature of these injuries raises the question: Do all blunt trauma victims need routine cervical spine imaging?

During the 1970s and 1980s, several papers cautioned physicians about the occurrence of occult or painless CSIs in blunt trauma victims.[41-46] Researchers in one study went as far as to recommend that all blunt trauma patients should have a lateral radiograph to exclude CSI, a recommendation that persists in Advanced Trauma Life Support training to date.[47] However, careful review of the literature finds these occult or painless injuries were not truly asymptomatic;[48] that in each of these cases, either the patient was evaluated inadequately or had a clinical finding that increased the risk for a CSI.

Fear of missing an occult CSI in the multi-system trauma patient has prompted many physicians to order cervical spine radiography on virtually all blunt trauma patients, regardless of their clinical presentations. The result is that many cervical spine x-rays are ordered to detect a few injuries; 98% of these radiographic studies are normal or negative.[49] Each year in the United States, an estimated 800,000 cervical spine radiographs are ordered by 25,000 emergency physicians at a cost of $180 million per year.[49] It is predicted that the radiation from these studies

Figure 2. Immobilization of Patient with Fixed Flexion Deformity

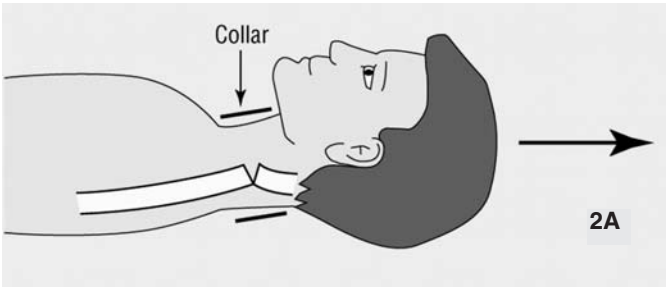


Figure 2A: Applying a hard collar to a patient with major cervical kyphosis and a fracture angulates the fracture and causes cord injury.

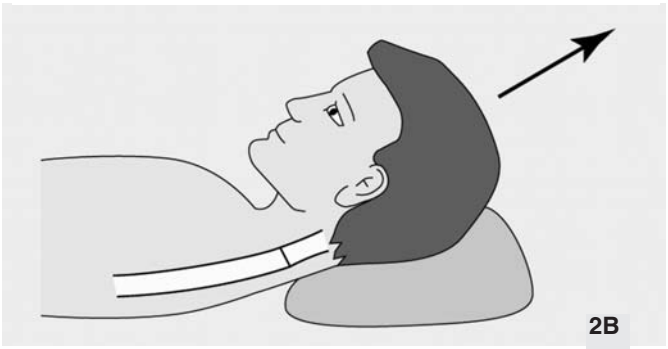


Figure 2B: Immobilizaton in flexion by placing sandbags under the occiput reduces the fracture and prevents cord damage.

Reprinted with permission from: Papadopoulos MC, Chakraborty A, Waldron G, et al. Lesson of the week: Exacerbating cervical spine injury by applying a hard collar. BMJ *1999:319:171-172.*

Figure 3. Logroll Maneuver

These photographs demonstrate the correct technique for performing a logroll maneuver.

Photos courtesy of S.V. Mahadevan, MD.

Figure 4. Immobilization of the Cervical Spine during Endotrachial Intubation

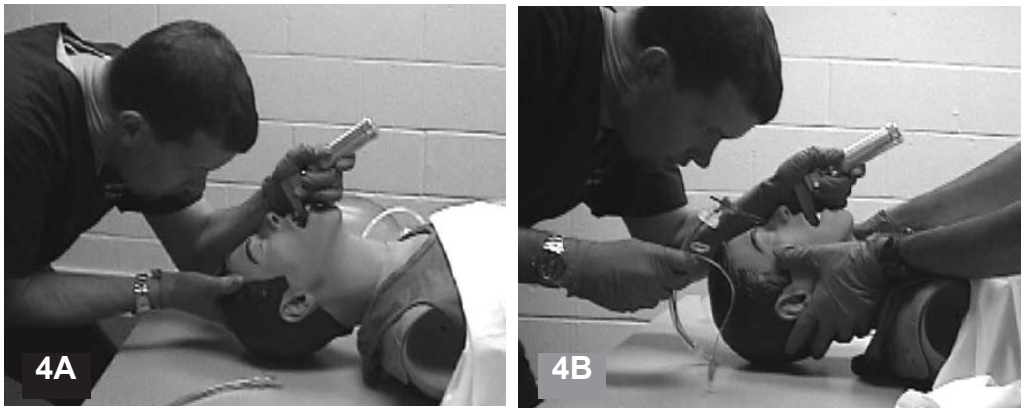


Figure 4A: Absence of in-line immobilization allows hyperextension of the neck.
Figure 4B: Use of in-line immobilization to prevent inadvertent neck extension.

Photos courtesy of S.V. Mahadevan, MD.

will lead to 3,760 thyroid cancers.[2]

Imaging all blunt trauma victims also leads to prolonged ED work-ups, lengthy immobilization, and may delay other therapeutic interventions and the care of other ED patients. To conserve resources, prevent morbidity, and act in a cost- and time efficient manner, a risk stratification approach is necessary to determine which patients require imaging.

During the late 1980s and early 1990s results from several small studies suggested that patients with certain low-risk clinical criteria have a low probability of CSI.[17,20,50-59] The largest of these studies had a sensitivity of 100% for excluding injury, but did not include enough patients (n=974) or cervical spine fractures (n=27) to justify widespread use.[49]

National Emergency X-Radiography Utilization Study (NEXUS). Results from the NEXUS trial presented clinicians with strong evidence to support the selective exclusion of cervical spine imaging in low-risk, blunt trauma patients. NEXUS consisted of a large, federally supported, multi-center, observational study designed to validate a clinical decision rule comprising five clinical criteria, commonly known as the NEXUS low-risk criteria.

The five clinical criteria (*See Table 2.*) include: 1) tenderness at the posterior midline of the cervical spine; 2) focal neurologic deficit; 3) abnormal level of alertness; 4) any evidence of intoxication; and 5) clinically apparent painful injury that might distract the patient from the pain of a CSI. The presence of any one criterion mandates the need for cervical spine radiography. The absence of all five clinical criteria strongly suggests that a patient has a low probability of CSI and, therefore, does not require cervical spine radiographs.

This NEXUS trial enrolled 34,069 patients at 21 different EDs across the United States—more than 20 times as many patients with CSI than any previous study. Of the 818 patients in this study with CSIs, all but eight injuries were identified by the presence of one of the five aforementioned criteria. Of the eight missed injuries, only two were considered clinically significant and only one required surgery. The patient requiring surgery was a 57-year-old male who was a belted driver involved in a head-on collision. He briefly lost consciousness; had scapular and clavicular tenderness and right arm paresthesias; and was diagnosed with a clavicle fracture and a C_6 lamina fracture. In retrospect, this patient actually may represent a misapplication—rather than a failure—of the clinical decision rule as the patient had a loss of consciousness, a clavicle fracture, and paresthesias.[60]

Overall, the five clinical criteria had a sensitivity of 99.0% and a negative predictive value of 99.8%. Using this clinical decision rule, the authors found that cervical spine x-rays could have been avoided in 4,309 patients (12.6%)—translating into a substantial reduction (approximately 100,000) in the number of cervical spine radiographs ordered in the United States each year, a cost savings of approximately $100 million annually and 474

Table 2. NEXUS Criteria

- Posterior midline cervical spine tenderness
- Focal neurologic deficit
- Abnormal level of alertness
- Any evidence of intoxication
- Clinically apparent painful distracting injury

Table 3. Distracting Painful Injuries

- Long bone fracture
- Visceral injury necessitating surgical consultation
- Large laceration
- Degloving or crush Injury
- Large burns
- Any other injury producing acute functional impairment

fewer predicted thyroid cancers. The NEXUS trial's findings subsequently were validated in both the pediatric and geriatric populations.[61,62]

Though the five clinical criteria were not defined explicitly, the individual criteria and decision rule as a whole showed good to excellent interobserver reliability.[63] A distracting painful injury (DPI) was not defined explicity, but described as any painful injury that potentially could distract the patient's attention from a CSI. (*See Table 3.*) On data analysis of a subset of patients' from the NEXUS database, researchers found that fractures (58%) and soft-tissue injuries (16%) accounted for the majority of DPIs.[64]

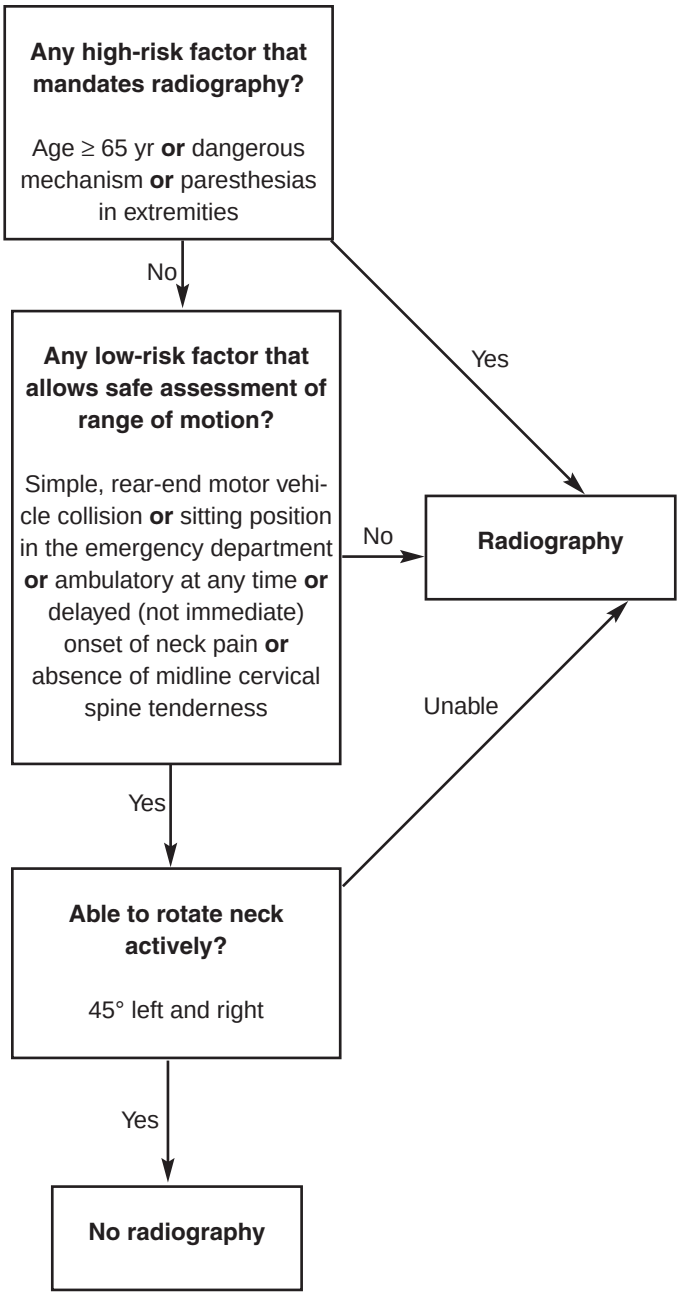
Canadian C-spine Rule (CCR). In hopes of narrowing the scope of patients requiring imaging, the Canadian C-spine group conducted a large, prospective, cohort study to generate data for a new decision rule. The group's paper contended that although the NEXUS criteria were highly sensitive (99.6%), the rule's reduced specificity (12.9%) could lead to patients being imaged unnecessarily.[65]

Results from the CCR derivation study generated a decision rule comprising three sequential questions to determine if imaging is necessary.[65] (*See Figure 5.*) The first question screens the patient for any high-risk factors that mandate radiography. These high-risk factors include age older than 65 years, a dangerous mechanism, and paresthesias in the extremities. The authors clarify what constitutes a dangerous mechanism to include such events as a fall from higher than 3 feet, axial load to the head, a high-speed motor vehicle collision (MVC), a bicycle collision, and a motorized recreational vehicle accident. If a high-risk factor is deemed present, the patient should undergo an imaging study; if not, one proceeds to the next question.

The second question seeks to determine if there is a single, low-risk criterion that might allow for safe assessment of range of motion. The low-risk criteria are defined as a simple, rear-end MVC, patient in a sitting position in the ED, patient ambulatory at any time, delayed onset of neck pain, or absence of midline cervical spine tenderness.

If any one of these low-risk factors is present and all high-risk

Figure 5. The Canadian C-Spine Rule



For patients with trauma who are alert (as indicated by a score of 15 on the Glasgow Coma Scale) and in stable condition and in whom cervical spine injury is a concern, the determination of risk factors guides the use of cervical spine radiography. A dangerous mechanism is considered to be a fall from an elevation higher than/equal to 3 ft or 5 stairs; an axial load to the head (e.g., diving); a motor vehicle collision at high speed (>100 km/hr) or with rollover or ejection; a collision involving a motorized recreational vehicle; or a bicycle collision. A simple, rear-end motor vehicle collision excludes being pushed into oncoming traffic, being hit by a bus or a large truck, a rollover, and being hit by a high-speed vehicle.

factors are absent, the third question is asked: Can the patient actively rotate the neck 45° both to the left and right? If the patient is able to complete this task, the CCR states cervical spine imaging is not required.

This derivation study consisted of 8,924 patients with 151 clinically important fractures. The authors determined that the CCR had a sensitivity of 100%, identifying all 151 clinically important fractures, and had a specificity of 42%. They estimated that use of the CCR would result in a 15.5% reduction in cervical spine radiography, compared with a 12.6% reduction using the NEXUS clinical decision rule.

However, the CCR has explicit exclusion criteria and cannot be used with any of the following patient populations: 1) age younger than 16 years; 2) abnormal Glascow Coma Scale score; 3) abnormal vital signs; 4) injury older than 48 hours; 5) penetrating trauma; 6) paralysis; 7) history of vertebral disease; 8) patient seen previously for the same injury; or 9) pregnancy.

NEXUS VS CCR. The CCR validation study consisted of 8,283 patients with 169 clinically important injuries and dealt with the confirmed results of the derivation study. The authors found the Canadian C-spine Rule had a sensitivity of 99.4% and a specificity of 45.1%. One of the 169 clinically important injuries was missed using the CCR.[66] However, in 845 cases — almost 10% of the study's patients—the study physicians were uncomfortable testing the patient's range of motion for unclear reasons. The inclusion of these patients could have affected the CCR's sensitivity or specificity adversely. Additionally, using the CCR, the study physicians misinterpreted the need for radiography in 5.2% of cases.

In addition to validating their rule, the CCR authors decided to retest the NEXUS low-risk criteria against their derivation and validation study populations.[66,67] They found NEXUS low-risk criteria had only a 90.7-92.7% sensitivity, and that use of the NEXUS rule would have missed 7-10% of patients with clinically important cervical spine injuries. These findings were in stark contrast to the NEXUS validation study, which found the rule to be 99.6% sensitive and was inconsistent with the large body of literature collected prior to the NEXUS trial.[68] Considering there are approximately 11,000 CSIs in the United States each year and based upon CCR studies' findings, the NEXUS decision rule could miss as many as 1,000 injuries each year. Yet, since the publication of the NEXUS validation study, there have been no case reports of injuries missed by the NEXUS criteria.

What could account for such a discrepancy? A number of methodological issues make interpretation of the NEXUS rule using CCR data suspect. The study physicians' failure to detect certain physical findings and the use of surrogate criteria introduces the potential for misclassification errors. For example, a fractured mandible and distal radius fracture were not classified as distracting injuries, and patients with extremity paresthesias were documented as not having a focal neurologic deficit. Such misclassification errors could result in inappropriate use of any clinical decision rule and serious medical errors.

Rather than being forced to choose one rule or the other, the astute clinician should be familiar with both, and employ them based upon the clinical scenario. In some instances, the combination of the rules may improve the positive predictive value while maintaining a high sensitivity.[68] As Mower and Hoffman positively note, "the development and validation of the NEXUS criteria and Canadian C-spine rule represent an embarrassment of riches for emergency physicians, who will have the luxury of choosing to use either or both of these instruments, depending on their clinical setting, ease and applicability, and individual practice styles."[68]

Summary

The first part of this two-part series has focused on the identification of patients at risk for CSI, immobilization, and controversies regarding the indications for radiographs. Early identification of patients who are high-risk for CSI, either based on the mechanism of injury, age, previous cervical spine disease process, or use of mind-altering substances will facilitate an early diagnosis and protection of the spine. Complete immobilization of the cervical spine will prevent additional injury to an unstable cervical spine and minimize further neurologic deterioration. In addition, physician awareness of the currently available clinical decision rules for cervical spine radiography may assist in a timely diagnosis for injuries and eliminate unnecessary radiographs in the appropriate patient categories. Part two of this series will include a discussion of imaging modalities for cervical spine trauma including plain radiographs, flexion/extension films, CT, and MRI. A thorough discussion on ligamentous CSIs will be presented as well as a discussion of SCIWORA.

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CE/CME Objectives

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

a.) Quickly recognize or increase suspicion for cervical spine injuries;

b.) Identify indications and techniques for cervical spine immobilization;

c.) State clinical decision rules for cervical spine radiography in a trauma victim; and

d.) Discuss controversies associated with imaging of the cervical spine.

CE/CME Questions

1. Which of the following mechanisms is *not* considered high-risk for cervical spine injury?
   A. Rollover motor vehicle collision
   B. Simple, rear-end motor vehicle collision
   C. Diving or drowning accidents
   D. Falls from higher than 10 feet
   E. Bicyclist struck by automobile

2. If a patient with suspected cervical spine injury requires emergent airway management, the clinician should:
   A. obtain a single, lateral radiograph of the cervical spine prior to intubation attempt to exclude a cervical spine fracture or subluxation.
   B. perform oropharyngeal intubation with rapid sequence intubation while maintaining in-line immobilization.
   C. avoid oropharyngeal intubation and perform nasopharyngeal intubation to secure airway.
   D. avoid oropharyngeal intubation and perform cricothyrotomy to secure airway.

3. Obtaining imaging of the cervical spine in all blunt trauma patients:
   A. is the standard of care.
   B. leads to prolonged ED work-ups and lengthy immobilization.
   C. is not associated with significant morbidity or increased cost.
   D. is essential because a significant number of cervical spine injuries may be occult.

4. Which of the following conditions is *not* a component of the NEXUS clinical criteria?
   A. Focal neurologic deficit
   B. Clinically apparent painful distracting injury
   C. Posterior midline cervical spine tenderness
   D. Ability to actively rotate the neck 45° to left and right
   E. Abnormal level of alertness

5. Which of the following conditions is considered a low-risk factor that allows for safe assessment of range of motion according to the Canadian C-spine Rule?
   A. Age younger than 65 years

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.

B. Ambulatory at any time
C. Midline cervical spine tenderness
D. Bicycle collision

6. Which of the following conditions may be considered a distracting injury?
   A. Long bone fracture
   B. Large laceration
   C. Large burns
   D. Crush injury
   E. All of the above

7. Which of the following patients is *not* at increased risk for a cervical spine injury?
   A. A 78-year-old male
   B. A 65-year-old male with ankylosing spondylitis
   C. A 54-year-old female with a history of neck strain
   D. A 63-year-old female with diffuse idiopathic hyperostosis
   E. A 50-year-old with previous cervical spine surgery

8. Which of the following criteria is *not* high risk for cervical spine injury in a 50-year-old male?
   A. Lumbar spine fracture
   B. Abnormal neurologic examination
   C. A fall from 3 feet
   D. Significant facial injury

9. Which of the following patients requires cervical spine radiography?
   A. A 65-year-old male who fell 10 feet and has neck pain
   B. A 37-year-old female with posterior midline cervical spine tenderness
   C. An intoxicated 27-year-old male who has been involved in a motor vehicle collision rollover
   D. All of the above

10. Which of the following criteria does *not* mandate radiography according to the Canadian C-spine Rule?
   A. Age older than 65 years
   B. Dangerous mechanism
   C. Fall from 2 feet
   D. Paresthesias in an extremity

Answer Key:

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

In Future Issues:

Evaluation and Clearance of the Cervical Spine, Part 2