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The evaluation and diagnosis of the child with a limp can be challenging for the emergency physician. Causes can range from life-threatening infections to problems as simple as ill-fitting footwear. Most children who limp, however, do not require urgent interventions and can be followed clinically once life- or limb-threatening causes have been ruled out.[1,2] Often, a careful history and physical are all that are needed to narrow the differential; at other times, complex and invasive tests are needed to support the diagnosis.

The authors review common causes for the acutely limping child, with special attention to those etiologies that need emergent or urgent intervention and referral. Additionally, the authors offer clinical and historical clues to help decipher the cause.

—The Editor

Evaluation of the Child with a Limp

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Introduction

Evaluation of the limping child may be challenging to emergency physicians. Numerous barriers to establishing a definitive diagnosis may exist. History may be limited or unavailable, particularly in the younger age groups, and physical examination may be difficult due to age and apprehension. In addition to difficulties in obtaining an accurate history and localizing the source of limp in an uncooperative child, the lack of availability of definitive diagnostic studies can further complicate the evaluation.[1-4] The differential diagnosis of the limping child also is quite broad. *(See Table 1.)* Most children, fortunately, present with non-emergent causes of limp and can be referred and followed as outpatients.[1,2] By separating etiologies into traumatic and non-traumatic causes and focusing on common disease patterns, the physician can develop a simplified approach to establishing the correct diagnosis.

Physiology

A basic understanding of normal gait, gait patterns, and child development is clinically important for the emergency physician. Adult gait pattern, which usually is seen by 3 years of age, is divided into two major divisions—a swing portion and a stance portion.[1] The stance portion further can be subdivided into heelstrike phase, flat foot phase, heel-rise phase, and toe-off phase; these describe the process of shifting weight to various portions of the foot.[1] A limp is defined as any alteration of the normal gait cycle.[5] Abnormalities in the swing portion of gait often point to difficulty with range of motion, whereas problems with the stance portion more likely indicate difficulty bearing weight. Two common pathologic gait patterns include antalgic and Trendelenburg gaits. An antalgic gait, which is the most common abnormal gait pattern, is caused by unilateral limb pain.[1,2,4,6] An antalgic gait results in patients spending a greater portion of the gait cycle on the unaffected side.[7] Antalgic gaits commonly are seen in patients with infectious arthritis, fractures, and even foreign bodies. A Trendelenburg gait is painless and a result of muscle weakness or instability.[7] It is characterized by a pelvic tilt away from the affected side due to a relative weakness of the hip abductors.[1] This pattern commonly is seen with slipped capital femoral epiphysis (SCFE), Legg-Calvé-Perthes Disease (LCPD), or developmental dysplasia of the hips (DDH).[1,2]

History and Physical

Although essential to determining the cause of a limp, the history and physical exam can be misleading, especially in younger children. Caregivers and patients should be questioned carefully for diagnostic clues, such as developmental accomplishments, fever, time-course, type of gait disturbance, exacerbating and relieving factors, history of trauma, and the presence or absence of pain. A history of fever may indicate the presence of an inflammatory condition or infectious process and usually accompanies septic arthritis, transient synovitis, and osteomyelitis.[1,2] A recent growth spurt or history of obesity may increase the index of suspicion for SCFE. A high index of suspicion for trauma, a common cause of limp, always should be considered, regardless of history. During the physical exam, children should be completely undressed and ambulated through several gait cycles to help localize the cause of the limp. The bottom of the feet should be carefully inspected for foreign bodies. All joints should be observed in passive and active range of motion and assessed for the presence of warmth, tenderness, or effusions.

Atraumatic Limp

Introduction and Approach to the Patient. The pediatric patient who presents with a gait disturbance, but without a history of trauma, may represent a daunting challenge to even the most astute clinician. The extensive differential of non-traumatic pathologic processes that may present with pain in the lower extremities are numerous and can range from life-threatening problems, such as leukemia or osteogenic sarcoma, to limb-threatening causes, such as inflammatory or septic arthritis. Additionally, children with significant disease of the lower spine, such as diskitis, initially may present with a limp, as may patients with intra-abdominal or retroperitoneal pathology, such as appendicitis or iliopsoas abscess.

Fortunately, the most common causes of atraumatic lower extremity pathology often may be differentiated based on characteristic presentations and historical factors, such as age, gender, body habitus, polyarticular or monoarticular involvement, acuity of onset, and presence or absence of fever. In patients in whom the diagnosis is uncertain after examination, or when confirmation of clinical suspicion is needed, pertinent lab, radiographic, or ultrasonographic studies may be useful.

Inflammatory Disease. *Transient Synovitis.* Transient synovitis, also referred to as toxic synovitis, is the most common cause of hip pain and gait disturbance in children 3-10 years of age.[8] It can occur in any age group, is more common in males, and, in the majority of cases, there is a history of an antecedent viral illness in the 1-2 week period prior to presentation.[9] The exact etiology is unclear, although the male predisposition has

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Table 1. Differential Diagnosis for a Limping Child

COMMON	UNCOMMON
• Septic arthritis	• Appendicitis
• Osteomyelitis	• Diskitis
• Toxic synovitis	• Psoas abscess
• Legg-Calvé-Perthes disease	• Henoch-Schönlein Purpura
• Slipped capital femoral epiphysis	• Meningitis
• Juvenile rheumatoid arthritis/ inflammatory arthritis	• Recent immunization
• Osteochondritis dissecans	• Psychosomatic
• Chondromalacia patella	• Tumor/sarcoma
• Osgood-Schlatter disease	• Myositis ossificans
• Developmental dysplasia	• Rheumatic fever
• Trauma	• Leukemia
• Growth plate injuries (Salter-Harris class)	
• Foreign bodies	

Figure 1. Septic Arthritis in a Child

A child with septic arthritis of the right hip. The patient holds the hip in flexion, abduction, and externally rotated in the position of greatest comfort.

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led some authors to suggest that many cases are actually secondary to unreported trauma.[10]

Patients with transient synovitis usually present with relatively acute onset of pain in the hip or groin, or referred to the anterior thigh or knee. Not uncommonly, patients are more symptomatic in the morning, with some improvement as the day progresses.[11] They typically demonstrate an antalgic gait, but are capable of weight-bearing. The affected extremity usually is not held in an abnormal position,[9] although patients may demonstrate some modest limitation of range of motion, especially in abduction and internal rotation. Erythema, warmth, and joint swelling generally are not present. Large joints, especially the hip, commonly are affected.

Transient synovitis must be differentiated from more serious entities such as osteomyelitis, LCPD, and, especially, septic arthritis, which can pose the greatest challenge to the physician. As such, transient synovitis often is a diagnosis of exclusion. Transient synovitis, septic arthritis, and LCPD present in similar age groups, with complaints of hip pain and limp. Similarly, patients with transient synovitis may have an accompanying fever, but typically it is low-grade (less than 38° C), and these patients usually appear well.

Laboratory values in patients with transient synovitis usually are normal, although one occasionally may find a mild elevation of the erythrocyte sedimentation rate (ESR).[12] Radiographs, which are helpful to exclude other conditions such as LCPD and osteomyelitis, should include anteroposterior (AP) and frog-leg views of the hips, and usually are unremarkable. Ultrasound may demonstrate a small effusion. Arthrocentesis usually demonstrates a normal glucose and WBC count between 20,000 and 50,000. Radionuclide scanning or magnetic resonance imaging (MRI) evaluation also may be undertaken to rule out infection or LCPD, and again, these typically are normal.

The disease is self-limited, and its anticipated course is 1-2 weeks in duration.[8] Treatment is expectant and consists of bed rest and non-weight-bearing while significant pain persists, with gradual resumption of normal activities as symptoms subside. Non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen (10 mg/kg orally every eight hours), may be used, or acetaminophen (10 mg/kg) may be given for symptomatic relief. Full recovery should occur in 2-3 weeks, and persistent or worsening symptoms should prompt a search for another cause of pain.

Septic Arthritis. Septic arthritis, also known as acute suppurative arthritis, is the inflammation of a joint space secondary to a bacterial infection. The most common organism isolated is *Staphylcoccus aureus*. Septic arthritis occurs more frequently in younger children, with a mean age of 3-6 years.[12-16] Presenting symptoms in older children include an antalgic gait, a refusal to bear weight on the affected extremity, a tender and swollen joint and surrounding erythema. Neonates and infants may present with symptoms ranging from localized joint tenderness and muscle spasm to more general complains of irritability, decreased feedings, and dislike of being held.[14] *(See Figure 1.)*

More than 90% of cases of septic arthritis affect the joints of the lower extremity, with the knee the most commonly involved joint.[2] The high morbidity, including destruction of joint cartilage and bone with subsequent chronic arthritis in cases of missed or delayed treatment, make the emergency department (ED) physician's role critical in correctly identifying septic arthritis and immediately beginning antibiotic therapy.[14] The gold standard for diagnosis is identification of the organism on Gram stain and culture obtained by arthrocentesis. The diagnosis

Table 2. Comparison of Synovial Fluid

	INFLAMMATORY	SYNOVITIS	SEPTIC ARTHRITIS
Color	Yellow	Yellow	Serosanguinous /pus
Clarity	Cloudy	Opaque	Turbid
Viscosity	Low	Low	Very low
WBC	< 20,000	20,000-50,000	> 50,000-300,000
Crystals	±	Absent	Absent
Glucose	Normal	Normal/low	Very low

Key: WBC = white blood cell count

is highly suggested when aspirated synovial fluid shows pus, white blood cell (WBC) count greater than 50,000, elevated protein, and decreased glucose levels. *(See Table 2.)* Kocher identified four independent clinical predictors of septic arthritis, based on multivariate analysis, including: 1) history of an oral temperature greater than 38.5º C; 2) non-weight bearing; 3) ESR greater than 40 mm/hr; and 4) serum WBC greater than 12,000 cells/mm^3.[15] Early septic arthritis, however, may show a paucity of symptoms and signs without overtly positive Gram stains. Additionally, inflammatory arthritis can show elevated WBC counts and inflammatory markers, thus mimicking septic arthritis. Del Beccaro et al demonstrated that an ESR greater than 20 and a temperature greater than 37.5º C, in the correct clinical scenario, correctly will identify 97% of patients with septic arthritis. Unfortunately, using this criterion, 47% of patients with an eventual diagnosis of toxic-synovitis would have undergone an unnecessary arthrocentesis to rule out septic arthritis.[9] Regardless of presentation, the emergency physician must maintain a high index of suspicion for septic arthritis, as missing the diagnosis can lead to joint destruction and chronic morbidity.

Once the diagnosis of septic arthritis is entertained, timely administration of parenteral antibiotics is mandatory. Empiric intravenous broad-spectrum antibiotics, such as a third-generation cephalosporin and a penicillinase-resistant penicillin, is the usually recommended antibiotic regimen. In areas with a high prevalence of methicillin-resistant *S. aureus* (MRSA), vancomycin should be added to the treatment regimen. Once the pathogen is identified with susceptibilities, the spectrum can be narrowed. Ultimately, infected joints may need multiple aspirations and/or open surgical drainage with irrigations to reduce the pressure in the joint space. If left untreated, septic arthritis leads to joint destruction and adhesions, leaving the patient permanently disabled.

Juvenile Rheumatoid Arthritis. Juvenile rheumatoid arthritis (JRA) refers to a heterogeneous group of chronic inflammatory diseases involving the joints. By definition, the arthritis must present before the age of 16 years and involve at least one joint for more than six weeks.[17] The prevalence of JRA in the United States is estimated at approximately 86 per 100,000 children.[18]

The disease is classified into sub-classes, based on the disease onset, as follows: 1) systemic; 2) pauciarticular (1-4 joints involved); and 3) polyarticular (five or more joints). Systemic-onset JRA provides the greatest challenge to the ED physician, because the diagnosis requires the exclusion of other infectious causes of joint pain. Children with systemic JRA classically have two fever spikes during the day that resolve spontaneously within 1-2 hours.[17] This pattern of fevers, along with other systemic symptoms of generalized rash and serositis, can aid in the differentiation of system JRA from septic arthritis and osteomyelitis.[19]

Laboratory tests provide little assistance in diagnosing JRA. Rheumatoid factor (RF) and anti-nuclear antibody (ANA) routinely are ordered when JRA is suspected. RF has less than a 5% sensitivity for JRA,[20] and 40% of children tested were positive for ANA,[21] making both poor screening tests. X-rays should be obtained to look for joint destruction and to rule out other etiologies of joint pain.

In the ED, the diagnosis of JRA rarely can be made definitively. The diagnosis should be considered only after exclusion of infectious causes of joint pain. First-line therapy for JRA consists of NSAIDs, and can be initiated in the ED.[20,22,23] The use of oral steroids and other second-line therapies, such as intra-articular steroid injections or methotrexate, typically are administered only in consultation with a specialist because of potentially serious side effects. A patient diagnosed with JRA requires referral to a rheumatologist for long-term management.

Osteomyelitis. Osteomyelitis is an infection of the bone or bone marrow. The disease is classified by the length of time the infection has been present. Acute osteomyelitis is an infection of fewer than two weeks' duration, and is the most common form in children. Sub-acute (2-6 weeks' duration) and chronic (more than 6 weeks) more frequently affect the adult population.

Bones become infected by one of three mechanisms: 1) hematogenous spread; 2) infection from a contiguous source; or 3) direct inoculation from wounds, trauma, or surgery.[24] Hematogenous seeding is the leading cause of acute osteomyelitis, and affects children more frequently due to the blood flow in growing bones. After approximately 1 year of age, diaphyseal blood vessels no longer cross the growth plate, and instead end in slow-moving pools of blood in the metaphysis.[25] This area of relative stasis provides an ideal location for the implantation of hematogenous infections in long bones.

The incidence of childhood osteomyelitis is on the decline in developed countries, in large part because of the *Haemophilus influenzae* vaccine. Despite the decline, 1 in 1000 neonates and 1 in 5000 children younger than 13 years in the United States are afflicted with this disease each year. *S. aureus* is the most commonly identified pathogen, responsible for nearly half the single organism infections.[25] Group B Streptococcus, *S. pneumoniae*, and gram-negative bacilli are other common pathogens. Several unique clinical scenarios are classically associated with otherwise uncommon organisms such as Salmonella infections in sickle cell patients, *Pseudomonas aeruginosa* infections in penetrating trauma, and *Neisseria gonorrhoeae* in newborns and sexually active teenagers.[26]

Diagnosing acute osteomyelitis in the ED can be challenging, especially differentiating between joint (septic arthritis) and bone involvement. Though often thought of as separate disease entities, recent studies show concurrent infections in 34-47.5% of cases.[25,27] Patients with osteomyelitis often present with localized tenderness, overlying soft-tissue erythema, and warmth, as well as systemic symptoms such as fevers, irritability, and poor feeding (infants). Gait disturbances nearly always are present when the lower extremity (i.e., femur, tibia, and fibula) is affected.[28]

Laboratory studies have been shown to be of little assistance in making the diagnosis of osteomyelitis. The WBC count has been proven to not be a sensitive indicator of osteomyelitis, while ESR and C-reactive protein (CRP) are more sensitive, but not specific.[24,29] Blood cultures and direct needle aspirates identify the bacterial pathogen in 50-75% of cases, respectively.[29]

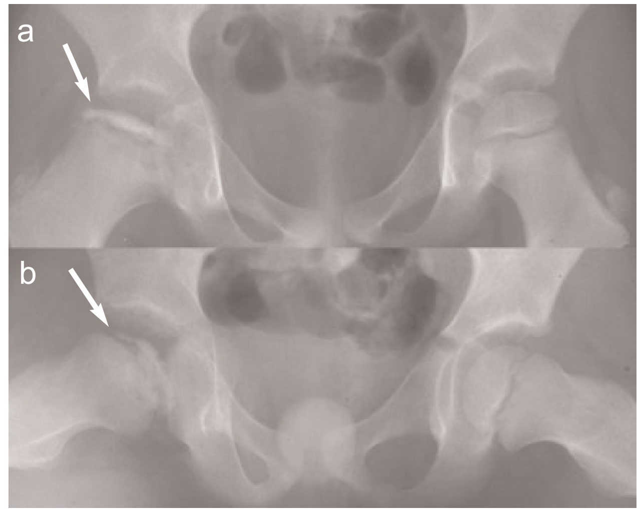
The first step in imaging the affected area typically should be a plain radiograph. The classic signs of bone destruction and pereosteitis often are not evident until 10-14 days after disease onset, though early signs of soft-tissue swelling may be present. Radiographs also are useful to help exclude other diagnoses, including fractures and bone masses or tumors.[30,31] Ultrasound examination may be useful if thickening and elevation of the periosteum are identified. Fluid collections may be identified and localized, and this may aid diagnostic and therapeutic aspirations.[30]

Bone scintigraphy (bone scan) identifies areas of inflammation, and is 82% sensitive in diagnosing osteomyelitis.[26] Bone scans lack the specificity and ability to precisely delineate the extent of bone involvement. Although computed tomography (CT) and MRI scans are more specific, both are considered secondary tests.[30,31]

Once osteomyelitis is suspected, intravenous empiric antibiotics are required. Antibiotics should not be delayed for culture recovery, and should be administered parenterally. Empiric antibiotics should cover *S. aureus*, as well as Group A and B Streptococcus, gram-negative bacilli, and *H. influenzae*. This can be accomplished using a penicillinase-resistant penicillin and a third-generation cephalosporin.[24] In regions of the country with high rates of MRSA, vancomycin should replace the penicillin. In sickle cell patients and patients with puncture wounds, where Salmonella and Pseudomonas are suspected, respectively, a quinolone must be added to the above regimen.[22] All patients with osteomyelitis require admission to the hospital.

Disorders of the Hip. *Legg-Calvé-Perthes Disease.* LCPD is an idiopathic avascular necrosis of the capital femoral epiphysis (CFE), the ossification center of the femoral head. LCPD was first described in 1909 by Arthur Legg, Jacques Calvé, and George Perthes, who were working in pediatric tuberculosis hospitals in Boston, France, and Germany, respectively.[32] The budding technology of roentgenography allowed them to identify patients who had been misdiagnosed with tuberculous arthritis, but who, in fact, suffered what Calvé termed "a pseudo-coxalgia."[33]

Figure 2 a-b. Legg-Calvé-Perthes Disease (LCPD) Radiographs



2a. Anteroposterior view of pelvis and hips show increased density and compression deformity of the epiphysis of the right hip when compared to the left.
2b. Similar findings in frog-leg view.

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LCPD is seen in children ages 2-12 years, with the vast majority being 5-9 years of age. There is a 4:1 to 5:1 male predominance, and many of the patients are of small stature, with relative skeletal immaturity. There also is a reported association of LCPD with patients who demonstrate deficiencies in protein S and protein C and those with thrombophilia and hypofibrinolysis.[9] The vascular insult may be secondary to arterial or venous thrombosis, with the latter being more common. Approximately 10-20% of patients will develop bilateral disease.

LCPD patients generally present with a history of several days of a painless limp, usually of the Trendelenburg type. Physical exam may demonstrate mild restriction of range of motion, mostly in abduction and internal rotation. Atrophy of the proximal thigh muscles may be noted in long-standing cases. Patients are non-toxic, and there usually are no symptoms of systemic disease, such as fever or rash.

The diagnosis can be made or suggested radiographically. In patients with established disease, AP and frog-leg films of the hips and pelvis will demonstrate characteristic findings of compression deformity and increased density of the capital femoral epiphysis. *(See Figure 2 a-b.)* Early cases may require MRI or radionuclide scanning for detection, although there is no demonstrable treatment benefit to early diagnosis. There typically is no effusion, and arthrocentesis is not indicated, except in cases where septic arthritis is suspected.

The age of onset carries significant prognostic implications. Younger patients (usually younger than 6 years of age) are much less likely to sustain long-term sequelae. It is postulated that the higher proportion of cartilage in the less mature epiphysis retains a greater capacity for remodeling than the more ossified epiphysis of the older patient.[32] Between the ages of 6 and 9 years, patients have a 35-40% risk of significant complications. Patients older than 10 years at onset, with a residual deformity of the femoral head, have nearly 100% incidence of degenerative osteoarthritis in later life.[9] Radiologic findings that are prognostic are based on the degree of involvement of the lateral pillar (lateral one-third of the CFE). Patients with little or no involvement of the lateral pillar typically do well without treatment. Patients with lateral pillar height loss up to 50% have a less benign prognosis, and patients who demonstrate a loss of height greater than 50% generally do poorly, with higher incidences of degenerative arthritis.[32]

Accordingly, orthopedic treatment decisions are based on factors including patient age at the time of onset, radiologic measures of the severity of disease, and the symptomatic expression of the deformity.[34] Patients younger than 5-6 years, or those with minimal lateral pillar collapse, generally are treated conservatively, with only expectant observation. In this group, patients with mild to moderate symptoms may benefit from periods of bed rest and/or abduction stretching therapy in an attempt to ameliorate discomfort and maintain joint mobility. In older patients or those with more advanced disease, the use of casts or orthotics may be indicated, and surgical containment procedures may be of benefit in cases of severe disease. Expeditious orthopedic referral and symptomatic treatment are indicated.

Slipped Capital Femoral Epiphysis. SCFE is the most common non-traumatic cause of pathology and pain in the adolescent hip. In the United States, the incidence of SCFE varies between two and 10 cases per 100,000 population.[35] It more commonly is seen in males, with gender predominance between 2:1 and 3:1. The age of onset usually is 9-16 years. Most patients with SCFE are overweight, with greater than half of the patients measuring above the 95th percentile for weight. Many of these patients also demonstrate delayed skeletal maturation. Conversely, a second group of patients at risk are tall, thin adolescents who have recently undergone a growth spurt. Estimates of the incidence of bilateral disease range from 21-80%.[36] Most patients develop contralateral symptoms within 1.5 years of their initial diagnosis.[36] Close monitoring of patients with unilateral disease, therefore, is warranted to ensure early detection of bilateral disease.

The exact etiology of SCFE is unclear, but it is postulated to be the result of excessive loading of the femoral head, either acutely or chronically, resulting in shear forces beyond the load range of the physeal cartilage. The subsequent abnormality is a displacement of the CFE posteriorly and inferiorly on the femoral neck metaphysis. SCFE has also been observed as a complication of endocrinopathies, such as hypothyroidism, pituitary disease, pseudohypoparathyroidism, and others.[37]

Patients with SCFE may present with complaints of either acute onset or chronic symptoms of pain in the hip, groin, knee, or anterior thigh. Some patients may give a history of an inciting event, such as a jump or a fall, while others note an insidious and more gradual onset, with or without an abrupt worsening of their symptoms. The onset history and radiographic findings determine the classification of patients into four groups.[9] The first group are patients with preslip. These patients have symptoms and historical and physical findings consistent with SCFE, and a widening of the physis without evidence of displacement. These often are found in the contralateral hips of known SCFE patients who are being monitored for bilateral involvement, but these also can be seen in new patients with early disease. The second group has an acute SCFE and is diagnosed in cases with fewer than three weeks of symptoms and no antecedent complaints. Pain and limp usually begin abruptly, without a history of trauma. Symptoms usually are severe enough to render the patient incapable of weight bearing. The third group of patients have an acute-on-chronic slip and experience mild symptoms, usually of several months' duration, with a sudden exacerbation or progression of discomfort as a result of a new slip superimposed on a chronic one. These patients similarly are incapacitated and often are unable to bear weight on the affected extremity. The fourth and most common group of patients with SCFE presents with a chronic SCFE. These patients usually are symptomatic for months, and displacement of the CFE has occurred gradually enough to maintain continuity with the femoral neck. The resulting symptoms are mild, and the patient usually adapts with a slightly antalgic gait and some degree of external rotation.

Radiographic evaluation should include AP pelvis, lateral hip, and frog-leg views. Findings on x-ray will reflect the category of disease. Preslip patients will demonstrate some physeal widening without CFE displacement. Acutely slipped hips will demonstrate displacement without significant remodeling of the CFE. Acute-on-chronic slips will reveal displacement as well as evidence of remodeling that, presumably, predated the slip. Chronic slips will demonstrate displacement, and callus formation may be visible, usually at the inferomedial aspect of the physis.

SCFE also may be classified on the basis of the degree of displacement. Mild cases slip less than one-third the diameter of the femoral neck, moderate cases are one-third to one-half the neck diameter, and severe cases exceed one-half the diameter. *(See Figure 3.)* Ultrasound, radionuclide bone scans, CT, and MRI may be helpful, but in general are not superior to plain films in establishing the diagnosis. Lab tests typically are normal and not necessary.

Treatment of SCFE is intended to prevent further displacement and to reduce the risk of complications and chronic sequelae. Currently, the most common treatment is closed reduction followed by epiphysiodesis, or surgical closure of the CFE,[38] typically through fluoroscopically guided percutaneous placement of fixation screws through the femoral neck into, but not through, the CFE. Cases of more severe chronic slips often require an osteotomy at the physis, the base of the femoral neck, or at the trochanter to compensate for the irreversibly remodeled CFE.

The most common serious complication of SCFE is osteonecrosis, which can affect all or part of the epiphysis. This is more common in patients who are pinned without reduction of the CFE. The second most common complication is chondrolysis, or degenerative change in the acetabular and femoral articular cartilages,[39] which occurs in 3-7% of cases. The surgical treatment of osteonecrosis may be undertaken early or may be delayed, sometimes for years, until the patient has reached adulthood.

Emergency physicians must maintain a high index of suspicion, a low threshold for obtaining appropriate radiographs, and a vigilant eye when interpreting those films. Patients in whom the diagnosis is made or strongly suspected should be referred emergently to an orthopedist, with appropriate symptomatic treatment initiated in the ED. Delayed or missed diagnosis carries the potential for an increased incidence of osteonecrosis and subsequent long-term sequelae.[1]

Developmental Dysplasia of the Hip. DDH is an uncommon cause of gait disturbance in the pediatric population, as it usually is detected on neonatal screening examinations. However, it may present in a young child who did not receive a complete evaluation as a newborn or infant.

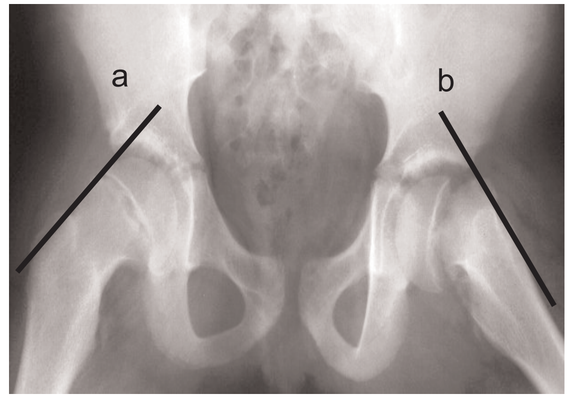
Epidemiologically, more than half of these patients are first-born infants, and one-third to one-half were breech presentations. There is a 9:1 female predominance, and a 20% incidence of positive family history. The majority of these children will demonstrate generalized ligamentous laxity.[40]

Patients with previously undiagnosed DDH will present with an abnormal and non-painful (Trendelenburg) gait, usually described as "waddling." Other findings may include exaggerated lumbar lordosis, toe walking, or leg-length discrepancy.[9]

Physical screening examination may yield a positive Ortolani or Barlow test in newborns or neonates with DDH. The Ortolani test consists of holding the thigh flexed and in abduction with anterior traction applied to the thigh, leading to a palpable reduction of the dislocated hip. The Barlow test consists of axial loading of the thigh in the flexed and adducted position. If the hip dislocates, which readily is appreciated by the examiner, the test is considered positive.[41] After 6 months of age, the Ortolani and Barlow tests are not applicable, and one may find only a positive Galleazzi sign. This test is performed with the patient in the supine position and requires the examiner to place the feet at the same level and compare the height of the knees. If the knees are not at the same level, one must infer a leg-length discrepancy, typically a shortening of the lower of the two sides. Note that this test will yield a false-negative result in the patient with bilateral disease.

Radiographic findings include abnormal alignment of the femoral head with respect to the acetabulum and pelvis. Treatment is variable, based on age at presentation and on whether the hip is dislocated, subluxed, or merely unstable (dislocatable). In cases of delayed diagnosis and progressive deformity, surgery often is necessary. Appropriate management in the ED includes timely orthopedic evaluation.

Figure 3. Slipped Capital Femoral Epiphysis (SCFE) Radiograph



a. Chronic slipped capital femoral epipysis (SCFE) in a patient with bilateral disease is evidence by a line drawn through the superior margin of the cortex of the femoral neck, which intersects only the lateral margin of the epiphysis or femoral head (Klein's lines).
b. Acute SCFE with gross displacement. Klein's line does not transect even a portion of the femoral head. In a normal view, the line drawn along the femoral neck margin would transect the lateral one-third of the epiphysis. *(Photo courtesy of Valerie Lint, DO.)*

Disorders of the Knee. Patients who present with complaints of knee pain in the absence of trauma are common. The knee is a complex, weight-bearing joint given to a variety of developmental and structural problems that may disturb the gait. Fortunately, the most common of these problems usually occur in adolescents or teenagers who are able to localize their symptoms to the knee.

Osgood-Schlatter Disease. This is one of the most frequent causes of non-traumatic knee pain in children 10-15 years of age. It is slightly more common in males than females, particularly those who are athletic or very physically active, and often is bilateral.[42] This condition is the result of repetitive traction of the quadriceps muscle on the patellar tendon at its insertion on the immature apophysis of the anterior tibial tubercle. Onset typically is gradual, although there may be a history of minor trauma to the area.

On physical examination, there is point tenderness and often swelling over the anterior tibial tubercle. Patients usually will have increased pain with extension of the knee against resistance or with passive flexion of the knee. Effusion, erythema, or warmth are uncommon, and their presence should prompt consideration of an alternate diagnosis.

Radiographs frequently demonstrate local soft-tissue swelling and a "ratty" appearance of the ossifying apophysis. The condition is self-limiting, and the patient and parents should be reassured that with rest and symptomatic treatment such as ice, NSAIDs, and acetaminophen, normal activities may be resumed when tolerated. This may be quickly, if improvement with NSAID therapy is sufficient, or may take up to 24 months following complete closure of the apophysis.[9]

Osteochondritis Dissecans. Osteochondritis dissecans may occur in other joints, but most commonly is found in the knee, usually involving the lateral surface of the medial femoral condyle. It results from an avascular insult to a small portion of the subchondral bone in this region, possibly due to repeated trauma from the tibial spine. This condition usually is seen in adolescents, particularly among athletic or very physically active patients. The history may be one of gradual and insidious onset, or, in 40-60% of cases, there may be a history of recent or antecedent trauma to the knee.[43]

Findings on physical examination may vary. With the knee in flexion, one may be able to directly palpate the involved area of the femoral condyle, eliciting point tenderness.[9] Wilson's sign may be positive and is specific for osteochondritic lesions of the medial femoral condyle. The sign is elicited when the knee is flexed to 90° and then slowly extended while the tibia is held in internal rotation. The sign is positive if the patient develops pain at approximately 30° of flexion, the point where the tibial spine contacts the medial femoral condyle.[44] A small effusion may be present; however, one should not find signs of an acute inflammatory process such as erythema or warmth. The ligamentous structures of the knee are stable on examination. If actual avulsion of the avascular fragment has occurred, the clinician may elicit a history of locking of the knee or find evidence of it on examination.

Radiographs may be positive only on the tunnel view of the knee. A loose intra-articular foreign body may be present if actual avulsion of a fragment has occurred. MRI examination may further orthopedic assessment of the stability of the lesion and allow staging of the disease, but is not necessary for initial diagnosis.[43]

Treatment usually consists of NSAIDs for symptomatic relief, as well as rest and avoidance of activities that worsen symptoms. Arthroscopic intervention may be necessary in cases of intra-articular loose bodies.

Chondromalacia Patella (Patellofemoral Syndrome). The exact cause of chondromalacia patella is unknown, although abnormal tracking of the patella over the femoral condyles is a contributory factor.[45] Patients may present with this condition at any age, but it is more common among adolescents and teenagers. Symptoms usually consist of dull, aching pain localized to the knee, and patients often will complain of discomfort behind the patella. The onset of symptoms may be gradual, or the complaints may arise after some direct trauma. Pain often is worse during activities such as climbing steps or riding a bicycle, or after prolonged periods of sitting with the knee in a flexed position, the so-called "movie sign."[46]

On examination, one may find tenderness of the medial posterior patella, and with application of posteriorly directed pressure applied to the patella with the knee in slight flexion. There also may be some crepitus palpable with flexion and extension of the knee.[47] Radiographs may demonstrate some blurring of the periosteum of the posterior patellar surface. This best may be visualized on the sunrise view of the knee.

Treatment generally is expectant, with rest, NSAIDs, and quadriceps-strengthening physical therapy.

Other Significant, Non-traumatic Causes of Gait Disturbance. As listed in Table 1, a wide variety of conditions not directly involving the lower extremity may present with a limp as the initial symptom. These include such significant problems as intervertebral diskitis,[2] appendicitis, pelvic inflammatory disease, leukemia, and both malignant and benign bony tumors.

A discussion of these diagnoses is beyond the scope of this article; however, one always must be cognizant of the possibility that a child with a limp and no history of trauma warrants a careful and complete investigation before symptoms are ascribed to a benign process.

Traumatic Limp

Introduction and Approach to the Patient. A second broad category of acute limp results from traumatic injuries. Regardless of history, trauma always should be considered, particularly with younger children. The inability of the child to describe symptoms and localize pain makes pediatric trauma care a special challenge that requires experience and great patience on the part of the examining physician.[48] Furthermore, injury patterns that are unique to pediatrics pose diagnostic dilemmas. The areas of immature pediatric skeletons composed of growth plates or physis represent the "weak link" in pediatric bone.[37] Injuries in adults that often cause sprains with injuries to soft tissue and tendon more often result in injuries to the bone or growth plate in children. While a detailed discussion of pediatric trauma is beyond the scope of this article, a few classic patterns are described.

Salter-Harris Fracture Classification. Common in children, injuries of the growth plates or physis pose an increased morbidity and diagnostic challenge for the clinician. The Salter-Harris classification is used in the description and prognosis of injuries involving the growth plates of skeletally immature children. *(See Figure 4.)* A Salter-Harris Type I fracture is a horizontal fracture through the physis or growth plate. Since no cortical or radio-opaque bone is involved, these injuries often are not visualized on radiographs and most are diagnosed clinically. They account for approximately 5% of physeal injuries.[49] Radiographically, a non-displaced Salter-Harris Type I fracture will appear normal or near normal. Comparison views may help to identify subtle displacement, but do not alter treatment. Any child with tenderness at the physis should be splinted, with close follow-up regardless of radiographic findings. Salter-Harris Type I injuries are treated with closed reduction and casting for approximately four weeks.[22] Overall, these injuries have an excellent prognosis.[37] Salter-Harris Type II fractures involve the physis and metaphysis and do not involve the joint space. These are the most common Salter-Harris injuries seen in children and account for approximately 75% of growth plate injuries.[49] Salter-Harris Type II fractures do not involve the joint space and, therefore, overall also have excellent prognosis.[22] Salter-Harris Type II fractures are treated with closed reduction and short casting for four weeks.[22]

Type III injuries are intra-articular fractures involving the physis and epiphysis. These injuries account for approximately 10% of physeal injuries and usually occur in children with a partially closed physis.[49] These injuries have a higher incidence of posttraumatic arthritis, but carry a good prognosis with proper anatomic correction.[37] Therefore, they often require open reduction and internal fixation to avoid growth disturbances.[22]

Type IV injuries involve the physis, epiphysis, and metaphysis with an oblique fracture line. The fracture pattern is intra-articular and, therefore, can result in generally a worse prognosis than the previously mentioned three patterns. The relative incidence is approximately 10%.[49] These intra-articular injuries often require open reduction and precise anatomic alignment to assure good outcomes.[37,50]

Salter-Harris Type V injuries are perhaps the most difficult to diagnose. These injuries are the result of crushing force to the physis. Radiographs of the involved growth plate often appear normal, though comparison views can be helpful in determining subtle differences. Although displacement of the epiphysis is rare, these injuries carry the poorest prognosis, with growth disturbances and premature cessation of bone growth.[37] Fortunately, they are the rarest of the Salter-Harris injuries and represent approximately 1% of growth plate injuries.[49]

Knee Injuries. Cartilage and ligaments are far stronger than bone, and injuries that normally would result in knee sprains in adults are more likely to result in fractures or growth plate injuries in children. The results are several injury patterns uncommon in adults but that commonly are seen in children. Sprains of the knee are quite common in adolescents and adults, but are rare in children. Hyperextension injuries of the knee that usually result in anterior cruciate ligament tears in adults often result in tibial spine avulsions in children.[51-53] Additionally, violent flexion against a contracted quadriceps muscle that would lead to patellar tendon tears in adults will lead to tibial tubercle avulsions in children.[51] Tibial spine and tibial tubercle avulsions can be difficult to diagnose, and negative radiographs do not rule out these injuries. Children with significant pain and clinical effusions should be placed in a long leg splint or knee immobilizer with orthopedic follow up in 1-2 days.

Lower Leg. *Toddler's Fracture.* Injuries of the lower leg are common in children, with the distal fibula and tibia being the most commonly injured sites.[51,54,55] One fracture pattern, unique to pediatrics, requires mention because of the common confusion with child abuse.

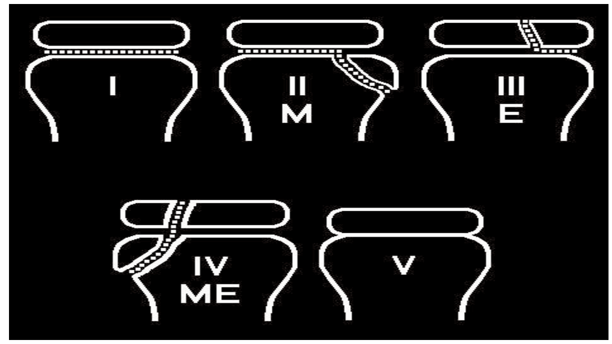
Children ages 1-5 are at risk for a childhood accidental spiral tibial fracture otherwise known as "toddler's fracture."[26,51] Children with these injuries may present without a history of trauma, or may be too young to relay a history of minor trauma. The mechanism of injury is a twisting force, often the result of a young child learning to walk. Symptoms include tenderness, warmth, antalgic gait, or refusal to bear weight. These occult injuries are difficult to diagnose and may be seen only on follow-up radiographs or bone scan. The astute clinician must, therefore, be alert to the diagnosis. Fortunately, the toddler's fracture is a stable fracture that often is treated conservatively with a long leg cast.[26] While long-bone injuries in children of this age are uncommon and should raise the suspicion for abuse, the emergency clinician must be aware of this diagnosis so as not to unnecessarily pursue a child abuse workup.

Ankle Injuries. While ankle sprains are common in the adult and adolescent population, children seldom suffer from sprained ankles. The ankle commonly is injured in children; however, and accounts for approximately 5% of fractures and 15% of all growth plate injuries.[51] The physis usually will separate or fracture before disruption or spraining of an adjacent strong or flexible ligament.[52] The three lateral ankle ligaments—anterior talofibular, calcaneofibular, and posterior talofibular—are prone to rupture and tear in the adult, but give way to fractures in children. A Salter-Harris Type I fracture of the distal fibula is the most common ankle fracture seen in children. Ankle injuries with swelling and tenderness around the growth plate should be treated conservatively as occult fractures with splinting and close follow-up in 1-2 days. Distal fibula injuries heal well with few complications.[56]

Foreign Bodies. Often overlooked, the diagnosis of plantar foreign body in the nonverbal child should be considered.

Radiographs may be helpful in the diagnosis of foreign body, but cannot exclude the presence of all foreign bodies. Many types of small glass particles, wood, plant matter, and plastics can be missed on x-ray. Ultrasound may play an increasingly important role in the identification and removal, but is not always readily available.[57,58] The astute clinician always should keep a high index of suspicion for foreign body and patients suspected of having retained foreign bodies should be given anticipatory guidance and follow-up in 1-2 days with a plastic, podiatric, or orthopedic surgeon. Tetanus should be updated and pro-

Figure 4. Salter-Harris Classification of Fractures



Key: M=metaphysis; E=epiphysis

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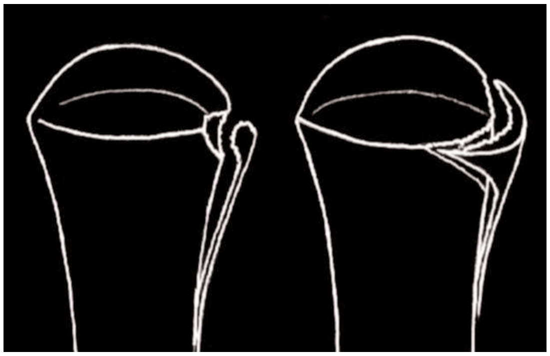
Figure 5. Fractures Suggestive of Abuse



The injuries shown above, a corner fracture (left) and bucket handle fracture (right), are highly suggestive of child abuse.

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phylactic antibiotics with gram-positive coverage may be prescribed in cases of deep, heavily contaminated wounds or for patients with underlying immunocompromised states. The use of antibiotics in otherwise healthy children with simple superficial foreign bodies remains controversial.

Child Abuse. With any pediatric traumatic injury, child abuse must be considered in the differential. Two-thirds of victims of physical abuse are younger than 3 years, and one-third are younger than 6 months.[22] Certain subtle historical clues and injury patterns to the lower extremity should raise suspicion of abuse. For example, inconsistencies between historians or frequently changing histories from a single historian should alert the clinician to a possible abuse case. Likewise, inconsistencies between mechanism and injury, such as a spiral femur fracture in a non-ambulatory child, also should raise suspicion.

When evaluating the limping child, two particular fracture patterns may strongly suggest abuse. *(See Figure 5.)* The bucket-handle fracture, which is an avulsion fracture of the distal metaphysis of a long bone, is nearly pathognomonic for child abuse. This pattern is produced by extrinsic traumatic forces with a rotational component. Additionally, corner fractures also are highly suggestive of abuse and warrant further workup, including an ophthalmologic exam and skeletal survey. Femur fractures, which require high energy forces, always should raise suspicion of abuse. As many as 70% of children younger than 1 year of age with femur fractures are victims of abuse.[51,59] All cases of suspected abuse require consultation and discussion with the primary physician as well as child protective services. Physicians are mandated by law to report all cases of suspected child abuse and neglect; however, reporting methods may vary by state.[60]

Conclusion

The orthopedic complaint of limp is common in the pediatric ED population and may pose many diagnostic dilemmas. Knowledge of normal gait problems and physiology are helpful in determining the cause. While the differential for the acutely limping child remains lengthy, an understanding of common injury and disease patterns and a simplified approach often will assist in determining the diagnosis. In those cases for which a diagnosis is uncertain, life- or limb-threatening causes should be ruled out, and patients should be referred for timely outpatient follow up. Whenever possible, the emergency physician should communicate with the primary or consulting physician to assure close follow up. Patients and families should be given clear discharge instructions, with anticipated course of disease and reasons to return to the ED.

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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.

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Physician CME Questions

21. Which of the following is true regarding the limping child?
    A. Most cases of limp are life-threatening and require urgent referral.
    B. Ligaments are underdeveloped and often rupture before bone.
    C. Growth plates represent a weak point in the pediatric skeleton.
    D. Tendon injuries are common with decreasing age.

22. Which of the following is characteristic of an antalgic gait?
    A. It is the result of pain on the affected side.
    B. It is a result of muscle weakness on the affected side.
    C. It can be described as a waddling gait.
    D. It results in an increased stance portion of the gait cycle.

23. Which of the following is true of septic arthritis?
    A. It is most common in neonates.
    B. It usually is preceeded by a viral syndrome.
    C. It typically results in a Trendelenburg gait disturbance.
    D. Patients may show a paucity of signs and symptoms in early stages.
    E. It most commonly is caused by Salmonella.

24. Which of the following is characteristic of Legg-Calvé-Perthes Disease?
    A. Peak incidence at 5-9 years of age
    B. More common in women
    C. Can be treated with steroids
    D. Most cases require admissionn
    E. Most cases result in an antalgic gaitn

25. Which of the following statements regarding slipped capital femoral epiphysis (SCFE) is true?
    A. The majority of patients with SCFE are male.
    B. The majority of patients with SCFE are tall and thin
    C. The majority of patients with SCFE present with an acutely slipped CFE.
    D. The majority of patients with SCFE will require emergent surgical intervention.

26. Pediatric patients with SCFE commonly present with complaints of pain in which of the following areas?
    A. Knee
    B. Thigh
    C. Groin
    D. All of the above

27. Which of the statements regarding developmental dysplasia of the hip (DDH) is true?
    A. The minority of the patients with DDH are first-born children.
    B. There is a low incidence of breech presentation among patients with DDH.
    C. Most patients with DDH are male.
    D. Most patients with DDH are diagnosed in the newborn period.
    E. All of the above are true.

28. Which of the following best describes a Salter-Harris Type IV fracture?
    A. It involves the epiphysis only.
    B. It commonly results from a crushing force.
    C. It is the most common Salter-Harris pattern.
    D. It has an excellent prognosis when treated with closed reduction.
    E. It involves both the metaphysis and epiphysis.

29. Which of the following common injury patterns seen in children is suggestive of child abuse?
    A. A 3-year-old with a spiral tibial fracture
    B. An 11-year-old male with tibial spine avulsion
    C. A 9-year-old with Salter-Harris Type IV injury
    D. A 9-year-old tibial tubercle avulsion
    E. A 1-year-old with a corner fracture of the femur

30. Which of the following is true regarding osteochondritis dissecans?
    A. It most commonly is found in the knee.
    B. It usually is seen in toddlers.
    C. There usually is no history of trauma.
    D. Wilson's sign is negative.
    E. Tunnel view radiographs of the knee are always negative.

Answer Key:

21. C	26. D
22. A	27. D
23. D	28. E
24. A	29. E
25. A	30. A

In Future Issues: **Moderate Head Injuries**

PEDIATRIC Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Child with a Limp

Differential Diagnosis for a Limping Child

COMMON	UNCOMMON
• Septic arthritis	• Appendicitis
• Osteomyelitis	• Diskitis
• Toxic synovitis	• Psoas abscess
• Legg-Calvé-Perthes disease	• Henoch-Schönlein Purpura
• Slipped capital femoral epiphysis	• Meningitis
• Juvenile rheumatoid arthritis/ inflammatory arthritis	• Recent immunization
• Osteochondritis dissecans	• Psychosomatic
• Chondromalacia patella	• Tumor/sarcoma
• Osgood-Schlatter disease	• Myositis ossificans
• Developmental dysplasia	• Rheumatic fever
• Trauma	• Leukemia
• Growth plate injuries (Salter-Harris class)	
• Foreign bodies	

Septic Arthritis in a Child

A child with septic arthritis of the right hip. The patient holds the hip in flexion, abduction, and externally rotated in the position of greatest comfort.

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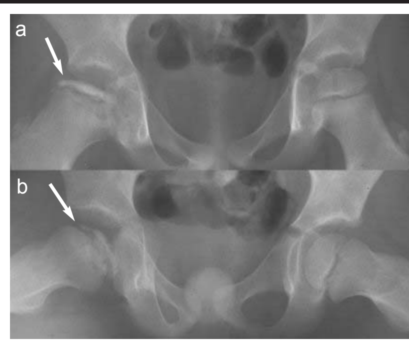
Comparison of Synovial Fluid

	INFLAMMATORY	SYNOVITIS	SEPTIC ARTHRITIS
Color	Yellow	Yellow	Serosanguinous /pus
Clarity	Cloudy	Opaque	Turbid
Viscosity	Low	Low	Very low
WBC	< 20,000	20,000-50,000	> 50,000-300,000
Crystals	±	Absent	Absent
Glucose	Normal	Normal/low	Very low

Key: WBC = white blood cell count

Legg-Calvé-Perthes Disease (LCPD) Radiographs



a. Anteroposterior view of pelvis and hips show increased density and compression deformity of the epiphysis of the right hip when compared to the left.
b. Similar findings in frog-leg view.

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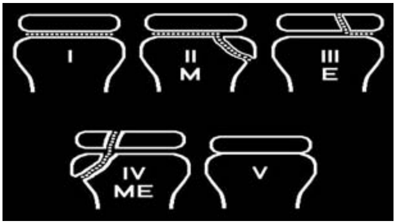
Salter-Harris Classification of Fractures



Key: M=metaphysis; E=epiphysis

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Fractures Suggestive of Abuse

The injuries shown above, a corner fracture (left) and bucket handle fracture (right), are highly suggestive of child abuse.

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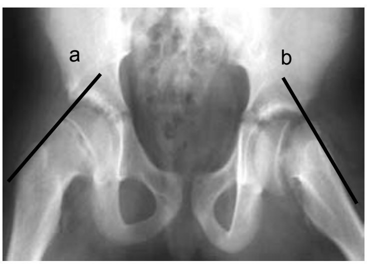
Slipped Capital Femoral Epiphysis (SCFE) Radiograph



a. Chronic slipped capital femoral epipysis (SCFE) in a patient with bilateral disease is evidence by a line drawn through the superior margin of the cortex of the femoral neck, which intersects only the lateral margin of the epiphysis or femoral head (Klein's lines).
b. Acute SCFE with gross displacement. Klein's line does not transect even a portion of the femoral head. In a normal view, the line drawn along the femoral neck margin would transect the lateral one-third of the epiphysis. *(Photo courtesy of Valerie Lint, DO)*

Supplement to *Pediatric Emergency Medicine Reports*, March 2004: "Evaluation of a Child with a Limp." *Authors:* **David S. Brancati, DO,** Department of Emergency Medicine, St. Vincent Mercy Medical Center and Mercy Children's Hospital, Toledo, OH; **John Jewell, MD,** Department of Emergency Medicine, St. Vincent Mercy Medical Center and Mercy Children's Hospital, Toledo, OH; and **Michael Omori, MD,** Mercy Children's Hospital Department of Pediatric Emergency Medicine; Faculty, St. Vincent Mercy Medical Center Emergency Medicine Residency Program. *Peer reviewer:* **Andrew D. Perron, MD, FACEP,** Residency Program Director, Department of Emergency Medicine, Maine Medical Center, Portland.

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Evaluation and Management of Blunt and Penetrating Thoracic Trauma

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Trauma to the thoracic cavity is responsible for approximately 10-25% of all trauma-related deaths,[1,2] with the majority of these deaths occurring after arrival at the emergency department (ED). The mortality for isolated chest injury is relatively low (less than 5%); however, with multiple organ system involvement, the mortality approaches 30%.[1]

This article dissects the critical aspects of thoracic trauma and highlights acute care management strategies.

— The Editor

Introduction

Thoracic trauma may be either blunt or penetrating. Compression, deceleration forces, and direct impact are mechanisms that result in the injury patterns seen in thoracic trauma. Unlike blunt trauma, penetrating trauma results from extrinsic violation of the integrity of the thoracic cavity. These injury mechanisms may result in pneumothorax, hemothorax, pulmonary contusion, or injuries to the mediastinal structures. With the proliferation of firearms, penetrating chest injuries are becoming more prevalent, and nearly all result in development of a pneumothorax, with hemothorax occurring in almost 75% of the cases.[1] The use of scoring scales such as injury severity scale, abbreviated injury severity scale, or the thoracic trauma severity score may assist the ED physician and trauma team in algorithmic decision-making. Although controversies abound regarding management of traumatic arrest patients, the decision to perform thoracotomy should be individualized. Moreover, fewer than 10% of blunt chest injuries and 15-30% of penetrating injuries require thoracotomy.[2]

Morbidity and mortality from chest injuries results from the injury pattern itself and physiologic derangements such as hypoxia, hypercarbia, and acidosis. The pathophysiology of these clinical entities arises from inadequate tissue perfusion from hypovolemia (secondary to blood loss), ventilation and perfusion mismatch from pulmonary contusion, or change in intrathoracic pressure from either tension or open pneumothorax.[2]

The presence of severe respiratory distress is associated with a high mortality, with 10% of these patients requiring intubation at the scene or immediately on arrival in the ED.[1] The most common associated risk factors for respiratory distress include multiple rib fractures, shock, pneumothorax, hemopneumothorax, and coma.[1]

Thoracic Injuries

Thoracic cage injuries result from the direct effect of trauma to the chest wall or the thoracic cage. They include flail chest and rib, sternal, thoracic spine, and scapular fractures.

Rib Fractures and Flail Chest. The ribs are the most commonly injured structures in the thorax.[2] This injury should be suspected in patients with localized chest wall pain or contusion over one or more rib segments after trauma. The upper ribs, first through third, are well protected by the bony framework of the upper limb; protective structures include the scapula, humerus, and clavicle, along with their muscular attachments.[2] Fracture of the first, second, or third rib requires a significant amount of force and should lead to high index of suspicion for injuries involving the head, neck, spinal cord, tracheobronchial tree, lungs, or the great vessels.[1,2] The mortality associated with these fractures is approximately 15-30%.[1,2]

The middle ribs, four through nine, are most commonly affected by blunt trauma.[2] A direct force striking anterior-to-posterior on the thoracic cage fractures these ribs along their shafts and tends to drive the ends of the bones into the thoracic cavity. This subsequently leads to intrathoracic injuries such as pneumothorax or lung contusion.[2] Furthermore, fracture of the lower ribs (ribs 10-12) should heighten suspicion for intra-abdominal injuries involving the liver and spleen.

A flail chest results when fractures occur in two or more ribs, or a fracture involves multiple segments of a rib. This results in a lack of continuity between the fractured segments and the rest of the thoracic cage, resulting in the disruption of normal chest wall movement. The morbidity from flail chest is not from the paradoxical chest wall movement produced, but rather from the underlying injury to the lung parenchyma (pulmonary contusion), and persistent splinting that results from severe pain. The pulmonary contusion leads to ventilation perfusion mismatch, leading to hypoxia. *(See Figure 1; see more on pulmonary contusion in the section dedicated to this entity.)*

Elderly and Pediatric Patients. The presence of rib fractures in older individuals carries significant morbidity and mortality. An elderly patient with a rib fracture resulting from blunt chest trauma has twice the morbidity and mortality of younger patients.[4,5] For each additional rib fracture, mortality increases by 19%.[4] Similarly, pediatric patients who present with rib fractures should alert the practitioner to possible child abuse. Although a rib fracture could occur in a pediatric patient who sustains blunt chest trauma, it is very rare. Its presence portends the possibility of concomitant severe injuries. Thus, geriatric and pediatric patients with multiple rib fractures warrant extreme vigilance.

The initial evaluation of a patient with a rib fracture involves palpation for localized pain, crepitus, subcutaneous emphysema, and deformities. In simple rib fractures, chest radiography alone will suffice. The purpose of the study is not to visualize the rib per se, but to evaluate for possible coexisting complications such as pneumothorax. Patients who sustain rib fractures from significant trauma to the thoracic cage, in addition to chest radiography, require computed tomography (CT) of the chest to qualify the extent of intrathoracic organ involvement (e.g., hemopneumothorax or aortic, bronchial, esophageal, cardiac, or diaphragmatic injuries).

Initial therapeutic intervention for an isolated rib fracture focuses on adequate ventilation and analgesia for pain control. Many therapeutic regimens are available for pain management. These modalities include opioid analgesics, transcutaneous electric nerve stimulation, non-steroidal anti-inflammatory drugs, and regional nerve blocks (intercostals, epidural, interpleural, and thoracic paravertebral).[6,7]

In patients with simple flail chest and minimal or no lung contusion, analgesia or intercostal nerve blocks with chest physiotherapy may be adequate. In patients with flail chest and moderate to severe pulmonary contusion, evidence of hypoxia, and signs of shock, mechanical ventilation should be initiated. Mechanical ventilation should be considered in individuals with flail chest and shock, three or more associated injuries, severe head injury, underlying pulmonary disease, fracture of eight or more ribs, or age older than 65 years.[1] Bergeron et al showed that after adjusting for severity, co-morbidity, multiple rib fractures, and age, patients older than 65 years had five times the odds of dying when compared to those younger than 65 years.[8] In patients with flail chest and two or more injuries, early mechani-

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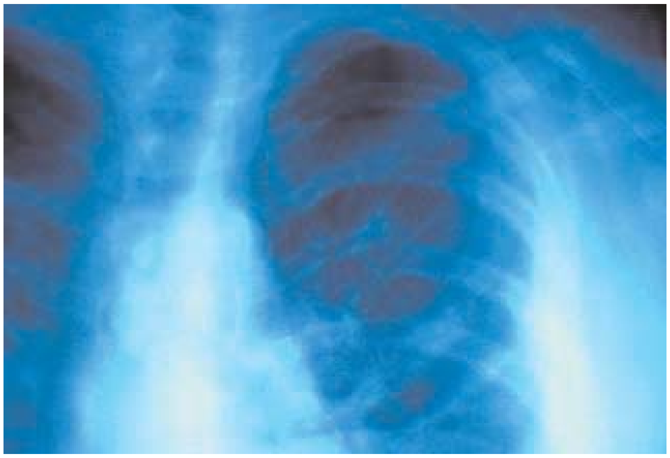
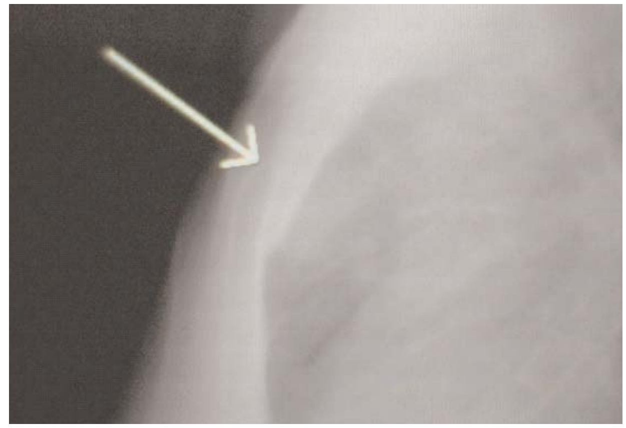
Figure 1. Rib Fractures with Pulmonary Contusion



The above image demonstrates multiple rib fractures and a pulmonary contusion.

Figure 2. Sternal Fracture with Posterior Displacement



A 78-year-old man who sustained a seatbelt injury in a motor vehicle accident. The arrow shows a sternal frature with posterior displacement. (Image courtesy of A. Adewale, MD.)

cal ventilatory support has been shown to reduce the mortality from 69% to 7%.[1]

The disposition of a patient with a rib fracture resulting from blunt chest trauma should be individualized. Patients with isolated rib fractures may be discharged home safely with good analgesia and an incentive spirometer. In patients with multiple rib fractures, admission often is recommended for pain control and to minimize the potential associated morbidities. All geriatric patients with multiple rib fractures require admission for management.[4,5,8]

Sternal Fracture. Sternal fractures commonly result from a direct blow to the chest wall during motor vehicular accidents or from falls, and account for approximately 8% of thoracic injuries.[9] The majority of these injuries result from direct contact with the steering wheel or from seatbelt compression. The presence of a sternal injury should alert practitioners to the possibility of an underlying cardiac or pulmonary injury. However, in the absence of concomitant injuries and other comorbidities, the incidence of morbidity is only about 5.4%,[9] and mortality is approximately 0.7-0.8%.[1,9] Previously, fractures of the sternum were believed to be harbingers of underlying cardiac injury; however, multiple recent studies[9-12] have proven otherwise. It is prudent to remember anecdotal cases of ventricular wall rupture associated with a sternal fracture.[13] *(See Figure 2.)*

The initial diagnostic modalities for sternal fracture are posterior-anterior and lateral chest radiographs. Although some studies have reported better diagnostic yield with sternal ultrasound and bone scintigraphy,[14,15] chest radiography still remains the initial diagnostic aid.

The extent of the evaluation and management should be guided by the severity of the injury and co-morbid factors. Most studies recommend a baseline electrocardiogram (ECG) and creatinine kinase (CK-MB),[1,11,12] with a 2-D echocardiogram reserved for those patients whose clinical presentation warrants the diagnostic test. Althought there are significant variations in institutional management of sternal fractures, the disposition of these patients should be individualized. Most patients with an isolated sternal fracture and no significant underlying medical problems may be discharged safely with appropriate analgesia.[10-12]

Thoracic Spine Injury. The thoracic spine supports the posterior segment of the thoracic cage. The first 10 vertebrae are fixed owing to their articulation with the thoracic cage.[1] Thoracic spine fractures often result from a fall from a height, a motor vehicle collision (especially with ejection), or a motorcycle accident.[16] Motorcycle accidents are the most common mechanisms because of the forced hyperflexion of the thoracic spine.[17,18] Fractures of the thoracic spine usually result from an excessive force, and an anatomically narrow thoracic spinal canal leads to a high incidence of associated neurologic complications.[1,2] Thoracic spinal fractures include anterior wedge compression, burst, chance, and fracture dislocation injuries. *(See Figure 3.)*

The initial evaluation of a patient with a possible spinal injury involves palpation of the spine for step off or subluxation, deformity, or midline tenderness. In most minor blunt chest trauma patients, an ordinary thoracic spine radiograph is adequate for evaluation. However, in severe multi-trauma patients with decreased levels of consciousness, severe alcohol intoxication, or respiratory distress requiring mechanical ventilation, it may be difficult to adequately assess the thoracic spine with routine radiographs. In these patients, CT of the spine has been shown to have better sensitivity, specificity, and negative and positive predictive values when compared to routine thoracic spine radiography.[19-21] Hence, in severe multi-trauma patients, CT scan of the thoracic spine should be the diagnostic study of choice.

The initial therapeutic management of a potential thoracic spinal injury associated with chest trauma involves spinal immobilization until radiographic clearance. However, because of the high incidence of spinal cord involvement associated with thoracic spine injury, the early utility of steroids is very controver-

Figure 3. Thoracic Spinal Fracture

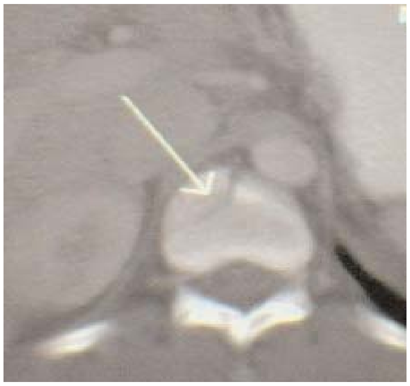
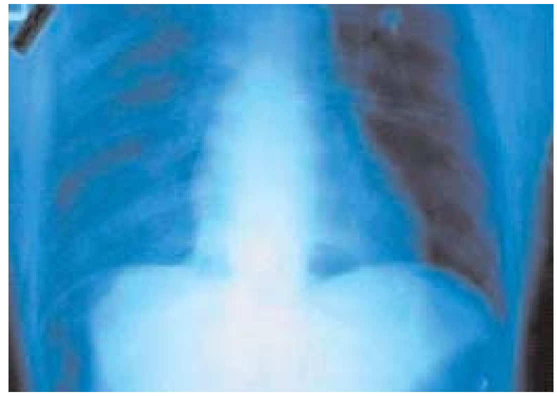


Image courtesy of A. Adewale, MD.

Figure 4. Hemopneumothorax



The chest radiograph above demonstrates a hemopneumothorax in a 49-year-old male involved in a motor vehicle collision.

sial. While some studies support the utilization of corticosteroids,[22,23] others do not consider it as a mandated standard of care, but rather as a treatment option.[24,25] Currently, the National Acute Spinal Cord Injury Studies (NASCIS) advocates the early use of steroids.

Intrathoracic Injuries

Pneumothorax. Pneumothorax results when air enters the potential space between the parietal and visceral pleura. This injury can be caused by either penetrating or blunt trauma.[2] The most common cause of pneumothorax in blunt trauma is a lung laceration with air leak.[2] It is a common complication of chest trauma, and a recognizable cause of preventable death.[26,27] About 6.7% occur without rib fractures, while the incidence increases with number of rib fractures.[28]

For the normal functioning of the pulmonary apparatus, the thorax is completely filled by the lungs, which are held to the chest wall by surface tension between the pleural surfaces. The presence of air in the pleural space leads to eventual collapse of the lungs. This collapse results in a ventilation/perfusion (V/Q) mismatch, because the blood perfusing the non-ventilated area is not oxygenated.[2] This ultimately results in respiratory distress and hypoxia. Early in the course of a pneumothorax or a small pneumothorax, the patient may be asymptomatic. However, as the intrathoracic pressure increases because of air in the pleural space, a simple asymptomatic pneumothorax may become a life-threatening tension pneumothorax.

The diagnosis of pneumothorax often is accomplished by clinical examination and chest radiography. However, in a severely traumatized patient, supine chest radiography may miss a pneumothorax.[29] Adjunctive diagnostic aids, such as ultrasonography and CT scan, have been shown to have both high sensitivity and specificity in diagnosing pneumothorax.[29,30]

The management of simple pneumothorax is expectant. However, when mechanical ventilation is indicated, prophylactic chest tube thoracostomy should be performed to prevent tension pneumothorax. In acutely dyspneic patients with hemodynamic instability after chest trauma, rapid needle decompression followed by tube thoracostomy is the standard of care.

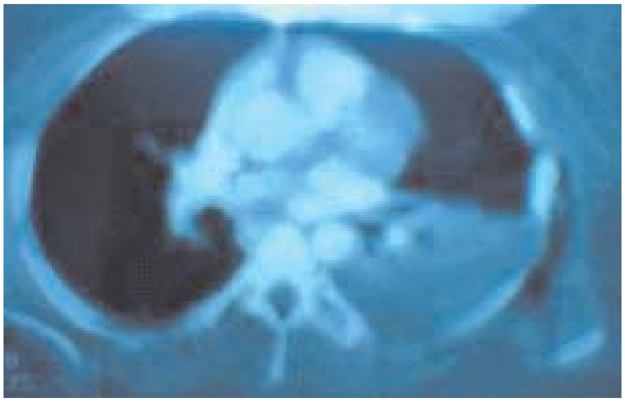
Hemothorax. Hemothorax commonly results from lacerations to the lungs, intercostal vessels, or the internal mammary artery due to either blunt or penetrating trauma.[1,2] Patients with large hemothoraces often are dyspneic, with some degree of respiratory distress because of restricted ventilation. *(See Figure 4.)*

The diagnosis of hemothorax should be made promptly. Although the majority of these injuries are diagnosed during the initial phase of assessment and management of the patient, there have been reported cases of delayed manifestation up to eight hours after initial presentation.[31] The initial diagnostic method is the chest radiograph, and a hemothorax that is large enough to be apparent on chest radiograph should be evacuated. However, in a supine trauma patient, up to 1000 mL of blood could be missed on a radiograph.[1] Studies showing the utility of ultrasound have produced mixed results.[32,33] The use of CT scans has been shown to be the most sensitive and specific for diagnosing and accurately assessing hemothoraces in chest trauma patients.[34-36] CT adequately quantifies the extent of the injury complex and the underlying complications.

The initial management of a hemothorax involves the insertion of a large-caliber chest tube for drainage, or open thoracotomy. In the majority of patients with hemothoraces, tube thoracostomy alone is adequate and is effective in more than 80% of the cases.[37] In the presence of persistent bleeding, videothoracoscopic evaluation and treatment have been shown to be as effective as open throracotomy, and minimize the complications that accompany thoracotomy.[37] The presence of more than 1500 mL of blood in the initial chest tube drainage, drainage of more than 200 mL an hour for 2-4 hours, or ongoing transfusion requirements mandate surgical exploration with open thoracotomy.[2] *(See Figure 5.)*

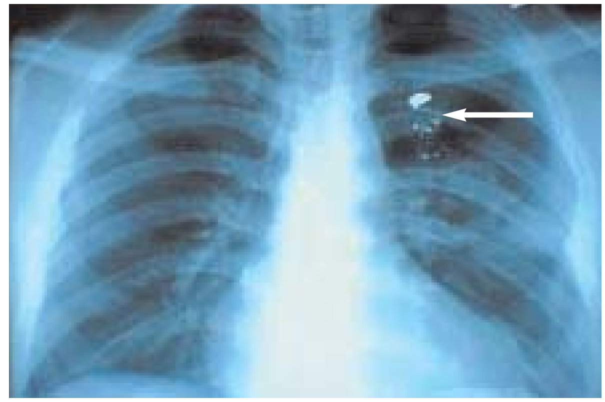
Pulmonary Contusion. Contusion to the lung parenchyma is a significant cause of morbidity and mortality resulting from thoracic trauma. It is the most common cause of lethal chest injury.[2] Usually a result of direct force to the chest wall, the injury results

Figure 5. Hemothorax and Pulmonary Contusion



A CT scan of the chest that demonstrates a hemothorax and pulmonary contusion. (Images courtesy of A. Adewale, MD.)

Figure 6. Rib Fracture from Gunshot Wound, with Pulmonary Contusion



An isolated rib fracture from a gunshot wound to the chest with a pulmonary contusion. The arrow indicates a bullet fragment.

from a coup and countrecoup effect. The transmitted force produces direct damage to the lung parenchyma and associated hemorrhage and edema. In the majority of cases, respiratory failure develops over days rather than instantaneously.[2]

The pathophysiology of pulmonary contusion is poorly understood, and only minimal advances have been made in the past 20 years.[38] The primary injury to the lung results in increased blood flow to the uninjured lung and parenchyma due to reflex decrease in pulmonary vascular resistance. This results in the extravasation of fluid into the alveoli and the interstitial spaces. With aggressive resuscitation, the level of the edema and blood in the lung parenchyma and interstitial spaces increases, thus producing a V/Q mismatch that is manifested as worsening hypoxia, hypercarbia, and acidosis.[1]

The diagnosis of pulmonary contusion is dependent on the extent of the lung parenchyma involved. Initial radiographic manifestations may be minimal or significant, depending on the individual patient. Even so, the chest radiograph should be the initial diagnostic aid, and for the majority of patients will show diffuse opacification of the involved lung parenchyma. *(See Figure 6.)* CT scan of the chest may be used in patients to adequately identify the exact lung segment involved, quantify the contusion volume, and act as a prognosticator of morbidity and mortality.[39,40]

The management of pulmonary contusion depends on the severity, associated injuries, and co-morbid conditions of the patient. The physiologic parameters that determine the severity of a pulmonary contusion in a chest trauma patient include: oxygen saturation less than 90% that is not responding to routine oxygen supplementation (nasal canula, bag-valve-mask, or face-mask); PaO_2 less than 65 mmHg; persistent tachypnea or FiO_2/PaO_2 less than 300.[1,2,41]

The primary management goal is to maintain adequate ventilation. The severity of the injury determines the modality of mechanical ventilation utilized. The need for intubation should be individualized, since the majority may be managed non-invasively.[42] In a patient with a contused lung, the plateau airway pressure is increased, while static compliance and $ETCO_2$ is decreased.[43] Independent lung ventilation (ILV) now is a commonly used modality for managing pulmonary contusion, because it is effective in reducing V/Q mismatch, improving oxygenation with a setting of tidal volume (TV) and positive end-expiratory pressures (PEEP) to keep pulmonary plateau pressure below safe thresholds for barotrauma.[42,43] Presently, the use of lung protective ventilatory strategy with low TV and high PEEP has become a standard practice.[43,44] In most severe cases, placing the patient in a decubitus position (involved lung dependent position), use of nitrous oxide (because of its less dense characteristics), utility of pressure-control ventilation with paralysis, and the use of high-frequency oscillator have been shown to improve survival.

Blunt Myocardial Injury. Blunt myocardial injury (BMI) commonly is caused by high-speed motor vehicle collisions, although injuries resulting from low-velocity collisions have been reported.[1] Due to different criteria used in diagnosing BMI, the incidence is difficult to determine. However, the prevalence ranges from 3-56%, depending on the study. The mechanisms of this injury include direct blows, athletic trauma, industrial crush, blasts, rapid deceleration, or falls from a height.[1]

A direct blow to the precordium (such as the chest wall striking the steering wheel in a motor vehicle collision, or the handlebar or a guardrail in a motorcycle collision, or being struck by a high-velocity object such as a baseball) creates a force that compresses the myocardium against the spine. *(See Figure 7.)* Because of the relatively free anterior-posterior movement of the heart, the momentum generated from a rapid deceleration accident maintains the heart in a uniform, straight-line motion, resulting in a direct strike against the internal sternum.[1,2] BMI includes injuries such as cardiac contusion, cardiac concussion (commotio cordis), cardiac chamber rupture, or valvular disruption.

The manifestations of BMI originate from a direct injury to the myocardium that results in sub-endocardial hemorrhage, in turn leading to the formation of localized edema and the mobilization of the inflammatory response system. The resulting inflammatory changes cause a redistribution of coronary flow that may manifest as ischemic type chest pain.[1] The severity of the presentation is contingent upon the underlying coronary artery disease, since transient coronary redistribution can produce a total or near-total occlusion of coronary vessels that already are stenosed. The worsening stenosis may lead to signs of acute coronary syndrome or acute myocardial infarction (AMI).

The clinical manifestations of this injury, in addition to ischemic or ischemic-like chest pain, include rhythm and conduction disturbances (e.g., multiple premature ventricular contractions and premature atrial contractions, atrial fibrillation, ventricular tachycardia, etc.), tachycardia out of proportion to volume loss or pain, right bundle-branch block (RBBB), and heart failure.[1,2] Also, the clinician should be suspicious of BMI in the presence of increased central venous pressure (CVP) and the absence of obvious cause.

Presently, there is no gold standard for diagnosing cardiac contusion. The true diagnosis only can be ascertained through direct visualization of the injured myocardium. Currently utilized diagnostic aids include:

- Chest radiography—The presence of pulmonary contusion or sternal fracture on chest x-ray should lead to a high index of suspicion for BMI;
- ECG—Its utility is controversial. However, recent studies have shown its advantages as a stratification and medical decision-making tool. Velmahos et al show that "the combination of normal ECG and cardiac TnI at admission and 8 hours later rules out the diagnosis of significant blunt cardiac injury."[45]
- Cardiac enzymes—Its utility also is controversial. However, multiple recent studies[46,49-52] are beginning to show its usefulness in determining if a patient has sustained significant BMI. Mori et al showed that "abnormal titers of cardiac TnI suggesting myocardial contusion may be found in more than half of the patients with blunt chest trauma."[46]
- Echocardiography—This is a very good screening and adjunctive test for evaluating BMI. Because of the low yield and lack of specificity of transthoracic echocardiography (TTE), transesophageal echocardiography (TEE) is the modality of choice when BMI is suspected. Chirillo et al showed that TTE "has a low diagnostic yield in severe blunt chest trauma, while transesophageal echocardiography provides accurate diagnosis in a short time at the bedside."[47]
- Magnetic resonance imaging (MRI)—The ability of cardiac MRI to adequately visualize the entire myocardium and the adjoining great vessels might increase its utility in chest trauma patients. Descat et al reported two cases of myocardial contusion diagnosed using MRI.[48]

The management of BMI depends on the clinical manifestations. Although the majority of the cases are benign, some may present with AMI,[53,54] persistent hypotension, or cardiogenic shock that warrants acute intervention. Recent literature proposes the use of ECG and TnI as the initial screening tools. If the initial ECG and TnI (four hours after the injury) are normal in the absence of concurrent injuries, the patient safely may be discharged from the ED.[52] However, if the ECG or the TnI is abnormal, further workup is indicated. The patient should be admitted to a monitored unit with serial cardiac enzymes and a TEE to adequately evaluate the cardiac apparatus. In the absence of abnormalities on TEE and the normalization of cTnI, the patient should be reassessed, and the clinical parameters should determine the disposition.

Figure 7. Blunt Myocardial Injury

A 7-year-old boy who presented with dizziness and chest pain after being struck on the chest by a baseball. (Image courtesy of A. Adewale, MD.)

Commotio Cordis. Commotio cordis, or cardiac concussion, is the most common cause of traumatic death in an athlete.[55-57] It causes sudden or near cardiac death in the absence of structural abnormalities, and results from an object directly striking the chest wall at a phase of ventricular depolarization.[56] Ventricular fibrillation is the most commonly induced arrhythmia. The survival rate is very low when the condition is not recognized. With the popularity of automatic electric defibrillators in public places, the survival from this phenomenon could be improved, since the ventricular fibrillation may respond to rapid defibrillation.

Esophageal Injury. Traumatic esophageal injury is very uncommon.[2,57] When it occurs, injury to the esophagus most commonly results from penetrating trauma; however, it also can result from blunt injury to the lower thoracic cavity. Upper esophageal injury may accompany lower cervical or upper thoracic spine injuries, while distal injuries are rarely caused by blunt chest trauma.[57,58] The mechanism of this injury is the forceful expulsion of gastric contents into the esophagus caused by a severe blow to the lower chest wall or upper abdomen. The resulting increase in intragastric pressure being transmitted to the esophagus results in a linear tear of the lower esophagus. This tear leads to the leakage of gastric contents into the mediastinum.[2]

The clinical presentation of the disease is similar to that of an esophageal tear secondary to persistent, profuse retching. In a severely traumatized patient, the diagnosis initially may be missed, and delayed diagnosis may lead to increased morbidity and mortality.

Esophageal rupture should be suspected in a patient with blunt trauma and one of the following features:

- Left hemothorax or pneumothorax without rib fracture;
- A significant blow to the lower sternum or epigastria with pain out of proportion to the apparent injury;
- Presence of particulate matter in chest tube drainage; or
- Presence of pneumomediastinum.

The diagnosis of esophageal perforation is very challenging. If suspected, a non-ionic contrast esophagogram should be performed.[1] However, this study carries a high false-negative rate, and in patients with a high degree of suspicion for this injury, flexible esophagoscopy in conjunction with esophagogram may increase diagnostic yield.

The management of esophageal perforation is very controversial and is guided by the location of the perforation (hypopharynx, cervical, thoracic, or distal). While some authors propose surgical management for all perforations,[59-63] some propose conservative management based on Cameron's criteria (i.e., minimal signs of sepsis, disruption contained within the mediastinum, drainage of cavity back into the esophagus, and minimal symptoms).[64] According to Rios et al, "Conservative treatment should be considered for patients meeting Cameron's criteria, since their evolution is favorable with low morbidity and mortality."[64]

Despite technical and nutritional advances, the mortality rate for esophageal injuries ranges from 5-25% for those treated within 12 hours, to 25-66% for those treated after 24 hours.[1] Regardless of the management approach, esophageal perforation is a life-threatening condition that requires early diagnosis and management to minimize the morbidity and mortality.

Diaphragmatic Injury. Blunt injury to the diaphragm is uncommon, occurring in about 0.8-8% of hospitalized chest trauma patients.[65] The incidence of the laterality of this injury varies depending on the study. However, most studies report the incidence of left-side injury to be between 60-90%.[1,66-68] This probably is because of the protection provided by the liver on the right and the possible left posterior lateral weakness of the diaphragm.[1,2] Because of the significant contribution of the diaphragm to normal ventilation, injury to this structure may lead to significant respiratory compromise.

This injury often initially is missed unless the defect is large enough to cause acute herniation of the abdominal viscera into the thoracic cavity. In this instance, the chest radiograph will show the gastrium in the thoracic cavity or a coiled nasogastric tube in the thoracic cavity. Smaller defects may cause a delay in diagnosis until the abdominal viscera slowly migrates into the thoracic cavity, causing compression of the adjacent lung leading to either bowel strangulation or tension pneumothorax.[1]

Diaphragmatic injuries create a diagnostic dilemma. The chest radiograph is diagnostic in most cases of large ruptures; however, in the small defects, the injury often is missed or the chest radiograph is misinterpreted as showing an elevated diaphragm, acute gastric dilatation, a loculated pneumohemothorax, or a sub-pulmonary hematoma.[2] The general consensus for the modality of choice for diagnosis is the helical CT; multiple studies have shown helical CT scan with sagittal and coronal reconstruction to be very sensitive in diagnosing diaphragmatic injury.[65,69,70] In one study, sensitivity approaches 84%, with specificity of 77%.[70] The most accurate modality for diagnosis is MRI. MRI is capable of directly acquiring both coronal and sagittal images, and allows the evaluation of the entire diaphragm, and shows the exact site and size of rupture in all cases as reported in one study.[69] However, its usefulness is limited in the acutely traumatized patient. Its utility is beneficial in otherwise stable patients with blunt chest trauma with a high index of suspicion for isolated diaphragmatic injury. With technological advances in the field of thoracic surgery, videothoracoscopic evaluation of the diaphragm in the hand of an experienced surgeon is emerging as a diagnostic modality.[71,72]

The management of diaphragmatic injury mostly is surgical. Most trauma patients with suspected diaphragmatic injury undergoing exploratory laparotomy for any intra-abdominal injury should have the diaphragm evaluated for possible defect. However, with the advances in laparoscopic techniques, thoracolaparoscopic repair is becoming the modality of choice for repair of an isolated diaphragmatic injury.[71] Isolated diaphragmatic injury in the absence of other surgical injuries carries low mortality, and Bergeron et al showed that operative repair can be deferred without appreciable increase in mortality if no other indications mandate surgery.[72]

Blunt Aortic Injury. Blunt traumatic thoracic aortic injury (BAI) is a rare, but very lethal, condition. It often occurs as a result of a decelerating injury from high-speed motor vehicle collision (low-speed in older population) or a fall from a height.[1] The incidence of BAI is about 6.8 per 100,000 motor vehicle occupants, with a steady increase with increasing age.[73] Approximately 80-90% of the patients die at the scene,[1,73] and up to 50% of the remaining patients die within 24 hours if not promptly diagnosed and treated.[1] Of the survivors who make it to the hospital, the ultimate survival rate is lower for patients who are older than 60 years.[73]

Three mechanical factors contribute to aortic rupture. These factors include shearing stress, bending stress, and torsion stress.[1] As deceleration occurs, a gradient is created between the mobile aortic arch and the immobile descending aorta. This gradient places the aortic isthmus under tension, and the resultant shearing stress can lead to rupture or tear opposite the fixation site.[1] Bending stress results from the hyperflexion of the aortic arch produced by the downward traction exerted by the heart, and torsion stress results from anterior posterior compression of the chest, with the heart displaced to the left combined with an intravascular pressure wave transmitted to the aorta.[1] When combined, these three forces produce maximum stress to the inner surface of the aorta at the ligamentum arteriosum, which is the point of greatest fixation.[1]

The clinical presentation of BAI is vague, since specific signs and symptoms often are absent.[2] Most patients with free rupture, as elucidated earlier, die at the scene. However, patients with contained rupture who make it to the hospital may exhibit transient hypotension, dysphagia, hoarseness, or acute dyspnea.[1,74] Although clinical factors (e.g., multiple rib fractures, flail chest,

Table 1. Radiographic Findings Suggestive of Aortic Injury[1,2]

- Widened mediastinum
- Aortic knob obliteration
- Rightward deviation of the trachea
- Obliteration of the aorto-pulmonary window
- Left mainstem bronchus depression
- Rightward deviation of the esophagus
- Paratracheal stripe widening
- Presence of apical cap
- Left hemothorax

Figure 8. Thoracic Aortic Dissection

CT-scan of a 78-year-old female involved in a motor vehicle accident. The arrow shows a thoracic aortic dissection. (Image courtesy of A. Adewale, MD.)

pulse deficits, or hoarseness without laryngeal injury) may be suggestive of BAI, about one-third of patients with this injury have no external signs of thoracic injury on initial physical examination.[1]

The diagnosis of BAI requires a high index of suspicion. A chest radiograph remains the most appropriate initial screening modality, with a negative predictive value approaching 95%.[75] The supine chest x-ray may not show the classic findings; however, the presence of a widened mediastinum mandates further investigation. On chest x-ray, the presence of a widened mediastinum and a hemothorax in a patient with transient hypotension should increase the suspicion of aortic injury.[74] *(See Table 1.)*

The radiographic algorithm utilized after the initial chest x-ray should be dictated by the patient's clinical condition. Although aortography still is the recognized gold standard, fast spiral helical CT scan has emerged as a diagnostic study that potentially will supplant aortography.[76] Multiple studies[77-80] have shown spiral CT angiography to have a sensitivity of 96-100%, specificity of 98-100%, and accuracy of 99-100%. In the hemodynamically stable patient, contrast-enhanced helical CT has a critical role in the exclusion of BAI and prevents unnecessary thoracic aortography.[80]

The role of TEE in the evaluation of BAI has been well documented.[75,77] Since the specificity and sensitivity of TEE are similar to those of helical CT, the indication for TEE is for the hemodynamically unstable patient with suspected BAI.[75,77] Although TEE and CT have similar diagnostic accuracy, TEE allows for the diagnosis of associated cardiac injuries, and is more sensitive than CT for the identification of intimal or medial lesions of the thoracic aorta.[77] *(See Figure 8.)*

While aortography still is the recognized gold standard for diagnosing BAI, in the case of equivocal aortographic findings, intravascular ultrasound (IVUS), with sensitivity approaching 92%, and specificity of 100%, could be used as an adjunctive diagnostic aid.[81] The role of MRI in the evaluation of trauma patients remains indeterminate. Despite sensitivity and specificity approaching 100% for aortic injury, MRI's utility in trauma patients is not feasible logistically because of the requirements of a metal-free environment and the long period of time that the patient must lie in isolation in a quiet room.[1]

The management of BAI typically is surgical following the initial resuscitation using the American College of Surgeons Advanced Trauma Life Support protocol. Emergent surgical intervention is the accepted standard of care. However, in some selected cases, such as patients deemed to be at high operative risk because of associated injuries or pre-existing medical conditions, or in stable patients for whom conditions for surgery are not ideal, delayed surgical intervention may be warranted.[1] The surgical approach utilized is institutionally dependent.

Tracheobronchial Injury. Injury to the tracheobronchial area occurs very rarely and often is associated with other injuries.[84] Although this injury potentially is fatal, it often is overlooked on initial assessment.[2] The reported mortality for tracheobronchial injury (TBI) has fallen from 36% in the 1950s to less than 9% in the 1970s.[85] The mechanism of this injury results from the effect of rapid deceleration on a relatively mobile bronchial structure and its fixed proximal segments.[1] The majority of these injuries occur within 2 cm of the carina, or at the origin of the lobar bronchi.[1,86]

The common clinical presentations of TBI are signs of respiratory distress (dyspnea, stridor, or hemoptysis), subcutaneous emphysema, and sternal tenderness.[1,87,88] The presence of pneumomediastinum, pneumothorax, widened mediastinum, or deep cervical emphysema on chest radiograph may suggest TBI.[1,2,84,88]

The diagnosis of TBI requires a high index of suspicion. The morbidity and mortality increases if not diagnosed early.[87] The initial screening modality is the chest radiograph, which often demonstrates the signs suggestive of TBI (as described in the paragraph above). According to one study, the CT scan of the chest demonstrated similar findings to the chest radiograph, but failed to confirm the diagnosis.[89] The presence of the "fallen lung" sign (a collapsed lung in a dependent position hanging on the hilum by its vascular attachment) on CT scan is highly suggestive of TBI.[84] Tracheobronchoscopy is the definitive diagnostic modality of choice.[1,2,84,88] On bronchoscopy, the injury pattern visualized is a transverse tear in the main bronchus involved or a disruption at the origin of an upper lung bronchus, while the trachea shows a vertical tear in the membranous portion near its attachment to the tracheal cartilages.[1]

Since most patients with TBI present with pneumothorax or tension pneumothorax, initial needle decompression with subsequent thoracostomy tube placement is required. The presence of persistent air leak with proper chest tube placement and drainage is highly suggestive of TBI until proven otherwise.[1,2,89]

The presence of persistent hypoxia despite intubation and chest tube placement mandates the use of temporizing opposite main stem bronchus intubation to provide adequate oxygenation,[2] and also minimizes the effect of the ventilation and perfusion mismatch. In some instances, blind endotracheal intubation may be difficult, owing to anatomic distortion from pharyngeal injuries, paratracheal hematoma, or the TBI itself. For these patients, immediate operative intervention is required.[2] However, in the more stable patients, acute surgical intervention could be delayed until inflammation and edema resolve.[2,90] The definitive surgical treatment involve the reestablishment of the anatomic continuity of the tracheobronchial tree if the lesion affects more than one-third of the circumference.[86]

Finally, independent of mechanism and anatomic location of injury, delay in diagnosis is the single most important factor influencing outcome.[91]

Penetrating Chest Injury. Penetrating chest injuries (PCIs) are more common in urban medical centers. Most of these injuries involve firearms and knives. Injuries involving the cardiac, vascular, and transmediastinal structures are the most lethal, with prehospital mortality approaching 86% for cardiac injuries, and 92% for extrapericardial vasculatures.[92] Of patients who make it to the hospital alive, only about 5-15% require thoracotomy.[1] Of those who survive to the hospital, the mortality for those with cardiac injury is 21.9%, and 1.5% for those without cardiac injury.[93] Survival rates from stab wounds generally are much higher than those from gunshot wounds.[1,93]

The injury pattern in PCI may include extensive lung laceration *(See Figure 9),* a sucking chest wound, or mediastinal traversing injuries. A sucking chest wound acts as a one-way valve that allows air to enter the pleural cavity during inspiration and none to leave during expiration. This eventually leads to an expanding or tension pneumothorax.[1] In the prehospital setting, it is imperative that the wound be covered with occlusive dressings on only three sides. This allows air to escape the pleural cavity during expiration and, thus, prevents development of a tension pneumothorax. On arrival to the ED, the wound should be examined and covered completely with occlusive dressing, with simultaneous insertion of a chest tube at a site other than the initial injury location.[1,2]

Wounds that traverse the mediastinum may involve the great vessels, heart, tracheobronchial tree, or the esophagus.[2] The overall mortality for these injuries approaches 20%.[2] The evaluation of these injuries in hemodynamically stable patients can be performed non-operatively.[94] Trauma ultrasound may be used for assessing pericardial tamponade, spiral CT for evaluating transmediastinal injuries, and organ-specific studies (e.g., esophagogram, aortography, bronchoscopy, thoracolaparoscopic evacuation of retained hemothorax, or repair of diaphragmatic injury) are minimally invasive management techniques for stable PCI patients.[94]

Figure 9. Penetrating Chest Injury

This penetrating chest injury was sustained by a motorcyclist who struck an embankment in a high-speed collision (Image courtesy of A. Adewale, MD.).

In hemodynamically unstable patients, there should be a high index of suspicion for exsanguinating thoracic hemorrhage, tension pneumothorax, or pericardial tamponade.[1,2] In this situation, a bilateral chest tube thoracostomy is warranted to decompress possible hemopneumothorax and document volume of blood in chest tube drainage.[2] The performance of ED thoracotomy is mainly for evacuation of pericardial blood, direct control of exsanguinating hemorrhage, open cardiac massage, or cross-clamping the descending aorta to slow blood loss below the diaphragm and increase perfusion to the brain and heart.[1,2] With a bleak survival report for emergent ED thoracotomy, each facility should develop a uniform guideline for performance of this procedure. *(See* Trauma Reports *2003;4:1-12 for a thorough discussion of ED thoracotomy.)* Recent studies[95,96] show that the presence of signs of life on arrival to the hospital, in addition to the mechanism of injury and location of major injury, should be the determinants of the indications for emergent thoracotomy.

Conclusion

Thoracic cavity trauma carries significant morbidity and mortality because of the vital structures it involves. With technologic advances, most of these injuries now can be evaluated with minimally invasive diagnostic aids. The evaluation and management of injuries involving this cavity should be individualized, with special consideration for the pediatric and geriatric population.

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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.

CE/CME Questions

1. The most commonly injured structure(s) in the thorax is/are:
   A. the sternum.
   B. the clavicle.
   C. the ribs.
   D. the lungs.

2. The indication for surgical exploration or videothoroscopic evaluation of hemothorax in a chest trauma patient is:
   A. initial chest tube drainage of 500 mL.
   B. the drainage of 100 mL/hr of blood for 2-4 hrs.
   C. spontaneous resolution of bleeding.
   D. initial chest tube drainage of more than 1500 mL.

3. The "fallen lung sign" is suggestive of which injury?
   A. Tension pneumothorax
   B. Esophageal tear
   C. Tracheobronchial injury
   D. Hemopneumothorax

4. One of the physiologic parameters that determine the severity of pulmonary contusion is:
   A. initial oxygen saturation less than 95%.
   B. initial PaO_2 less than 65 mmHg.
   C. initial PaO_2/FiO_2 less than 400.
   D. initial respiratory rate of 42.

5. The gold standard for diagnosing cardiac contusion is:
   A. electrocardiogram.
   B. cardiac enzymes.
   C. echocardiogram (TEE or TTE).
   D. There is no gold standard for diagnosing cardiac contusion.

6. The most commonly induced arrythmia in commotio cordis (cardiac concussion) is:
   A. ventricular tachycardia.
   B. asystole.
   C. ventricular fibrillation.
   D. supraventricular tachycardia.

7. Esophaphageal rupture should be suspected in blunt trauma patients with which of the following features?
   A. Multiple rib fractures
   B. Left hemothorax or pneumothorax without rib fractures
   C. Lack of particulate matter in chest tube drainage
   D. Persistent retching

8. A coiled nasogastric tube in the thoracic cavity is indicative of which of the following injuries?
   A. Blunt aortic injury
   B. Tracheobronchial injury
   C. Diaphragmatic injury
   D. Pulmonary contusion

9. Regarding the diagnosis of blunt aortic injury, which of the following statements is *not* true?
   A. Aortography is still the gold standard.
   B. CT scan angiography is as specific and sensitive as aortography.
   C. Magnetic resonance imaging is the most specific and has replaced aortography as the gold standard.
   D. Transesophageal echocardiography is quick, sensitive, specific, and enables evaluation of the cardiac apparatus.

10. Which of the following statements regarding transmediastinal injuries (stab or gunshot) is true?
    A. A gunshot wound to the heart has a better survival rate.
    B. A stab wound to the heart has a better survival rate.
    C. Survival rates for stab wounds and gunshot wounds to the chest are the same.

CME Objectives

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

a.) Recognize or increase index of suspicion for thoracic trauma;

b.) Identify how to correctly and quickly stabilize and manage thoracic trauma;

c.) Employ various diagnostic modalities for thoracic trauma; and

d.) Recognize both likely and rare complications that may occur.

Answer Key

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

In Future Issues: **Trauma Ultrasound**