High-Risk Pediatric Orthopedic Pitfalls

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KEYWORDS

• Pediatric fracture • Limp • Septic arthritis • Abuse
• Compartment syndrome • Slipped capital femoral epiphysis

Key points

- For pediatric fractures, intra-articular injury, which can lead to osteoarthritis, is of even greater concern than physeal involvement, which can lead to growth disturbance but is correctable.
- For type I open fractures, in which the bone pokes through the skin creating a puncture from inside outward, treatment includes cleaning the wound, oral antibiotics, splinting and referral to an orthopedic surgeon within 24 hours.
- With supracondylar fractures, loss of pulse is not always an emergency as long as the hand is acceptably perfused, which is defined as warm and pink with less than 2 second capillary refill. In consultation with an orthopedist, such a fracture may be splinted in place and referred the next office day for evaluation and management.
- With supracondylar fractures, neural injury occurs in approximately 10% to 15% of cases. The outcome of such neurapraxia is recovery, which may take up to 6 months and therefore does not require immediate surgery.
- Compartmental syndrome in children differs from that in adults. Physical examination findings can be milder, children can be more difficult to examine, and they may not be able to describe their pain as clearly as adults. This difficulty can result in delayed diagnosis, so the level of suspicion should remain high.
- Axial joint septic arthritis (shoulder and hip) is treated with incision, drainage, irrigation, and debridement.
- Appendicular joint septic arthritis can often be diagnosed and treated by serial aspiration and antibiotics.
- Osteomyelitis with radiographic change will need operation and may wait until evaluation by an orthopedic surgeon within a week.

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- Osteomyelitis without radiographic change should not be treated with antibiotics (unless patients are in distress) until an osseous specimen is obtained.
- Articular hip disorders typically manifest as groin pain but may present as knee or thigh pain.
- In pediatric patients who have a limp, the prone position is optimal for examination, which allows uncoupling of the knee from the hip, in which the pathology of one may masquerade as the other.
- If the diagnosis of slipped capital femoral epiphysis (SCFE) is suspected, both hips should be imaged.
- Fifteen percent of children who have SCFE have mostly knee or distal thigh pain. Children who have SCFE who present with primarily knee pain are more likely to receive a misdiagnosis and have slips of greater severity.

Children are not small adults: they are peculiar and may confound even the experienced emergency provider who is more familiar with adults. The authors have selected some pediatric orthopedic pitfalls based upon frequency of occurrence, difficulty of diagnosis, or gravity of sequelae. The authors have sacrificed quantity at the alter of quality, to teach principles rather than to catalog. The authors' goals are brevity and clarity, and they hope that the included tables may serve for quick references in busy clinical settings.

FRACTURES

General

An understanding of pediatric bone anatomy is essential to the accurate description of a fracture (**Box 1** and **Table 1**). The location of a fracture relative to the growth plate has implications for growth and the eventual risk of osteoarthritis. The fracture pattern may influence the stability of a fracture and determine the type of immobilization required, the degree of weight bearing allowed, or the technique of operative treatment. Soft-tissue injury may impact risk of infection, healing, and functional outcome.

Displacement at a fracture has two components. Translation is measured in percentage of bone diameter; however, it is measured in millimeters when articular. Angulation is defined by the direction of the distal fragment (eg, dorsal in the sagittal plane, varus in the coronal plane) or by the direction of the apex of the fracture (eg, apex volar in the sagittal plane, apex lateral in the coronal plane). Rotational angulation (in the transverse plane) may be difficult to determine in standard anterior-posterior and lateral radiographs. Angulation is measured in degrees.

Fractures in children may be divided into non-physeal versus physeal. Physeal fractures have been classified by Salter and Harris (**Fig. 1**).[1] Although the Salter-Harris classification is venerable and widely established, physeal fractures may be divided according to treatment and prognosis into nonarticular versus articular (**Fig. 2**). Fractures are treated based upon acceptable criteria, as illustrated in **Table 2**. The most important factors that determine acceptable displacement are percent contribution of adjacent physis to growth and planes of motion of the adjacent joint. As a result, the fracture where the greatest amount of displacement is acceptable is the proximal humerus; the proximal physis accounts for 80% of total growth of the humerus (the highest contribution in the skeleton), and the shoulder joint allows motion in all planes.

Physeal/articular fractures require anatomic reduction, defined as within 2 mm of normal alignment, based upon the increased incidence of posttraumatic osteoarthritis seen in patients who have articular displacement greater than 2 mm.[2] Because the physis distinguishes bones in children from those in adults, it is often the focus of

Box 1
How to describe a fracture patient

1. Age
2. Mechanism
3. Date/time of injury
4. Bone involved
5. Location within bone
   - diaphyseal
   - metaphyseal
   - epiphyseal
6. Pattern of fracture
   - transverse
   - oblique
   - spiral
   - comminuted
7. Physeal involvement (±)
8. Articular involvement (±)
9. Soft tissue/skin:
   - open versus closed
   - swelling
   - neurovascular examination

discussions between parents and providers. However, in the balance of growth disturbance (with physeal injury) versus osteoarthritis (with articular displacement >2 mm), the latter is of greater concern to the surgeon. Growth disturbance is treatable (physeal bridge excision, corrective osteotomy, bone lengthening), whereas there is no effective reconstruction for osteoarthritis. An illustration of this principle is that, during open reduction and internal fixation of physeal/articular fractures, articular stability is never sacrificed for physeal preservation; trans-physeal (eg, with a plate or threaded implant), stable articular fixation is preferable to extra-physeal, less stable articular fixation.

Open fractures traditionally have been considered an emergency requiring operative treatment within 6 hours of injury. However, open fractures associated with a small skin laceration less than 1 cm, minimal surrounding soft-tissue injury, and minimal contamination are no longer considered to require emergent treatment.[3] Such fractures are classified as type I.[4] Types II and III describe increasing wound size and soft-tissue injury, including vascular and neural injury and need for reconstruction in addition to osseous fixation.[5] In type I open fractures, deformation of the limb allows the bone to poke through the skin creating a cutaneous puncture from inside outward. This type of puncture contrasts with an external object creating a large devitalized wound that brings contaminants from the outside inward, as in types II and III, necessitating irrigation, debridement, and fracture stabilization with internal fixation to optimize the environment for soft-tissue healing. Treatment for type I open fractures

Table 1 Osseous definitions	
Open fracture	Skin and soft-tissue wound that allows communication between the outside environment and the bone; also known as compound fracture
Comminuted fracture	Broken into multiple pieces
Greenstick fracture	A break in the convex cortex under tension caused by the bending of malleable bone
Torus fracture	A buckle in the concave cortex of malleable bone under compression; *Latin torus = buckle*
Epiphysis	End of long bone; secondary center of ossification; articular; separated from the rest of the bone by the physis
Metaphysis	Segment of bone between diaphysis and physis; most common site of infection
Diaphysis	Shaft of long bone; primary ossification center
Apophysis	Secondary center of ossification; site of insertion of muscle (eg, greater trochanter)
Condyle of bone	Paired articular swelling at the end of a long bone
Head of bone	Singular articular swelling at the end of a long bone

includes cleaning the wound, oral antibiotics, splinting, and referral to an orthopedic surgeon within 24 hours (**Table 3**).

Table 4 lists screening radiographic views for imaging of the skeleton. These radiographic views may be augmented by oblique views when a fracture is suspected (eg, based upon history, mechanism, severity of physical examination findings) or when there is a low tolerance for displacement (eg, in articular fractures [$\leq$2 mm]).

The most frequent long bone fracture in a child involves the distal metaphysis of the radius; most of these are treated with a cast. The fracture in a child that is most frequently fixed operatively involves the supracondylar region of the humerus. Supracondylar humerus fracture may be associated with vascular injury and neural injury.

Neurovascular Injury

Vascular injury in the setting of a supracondylar humerus fracture may occur when there is sufficient displacement to require operative fixation. Physical examination findings include loss of palpable pulse at the wrist, loss of pulse on ultrasound testing at the wrist, and loss of perfusion of the hand. The majority of vascular injuries in supracondylar humerus fractures result from stretch of the brachial artery by the proximal fragment. Although this may lead to loss of pulse, measured either by palpation or ultrasound, the vascular net around the elbow allows for collateral flow distal to the fracture. As a result, loss of pulse is not an emergency as long as the hand is acceptably perfused, which is defined as warm and pink with less than 2 second capillary refill. A fracture with an acceptably perfused hand may be splinted in place and referred the next office day for evaluation and management by an orthopedic surgeon. A fracture associated with a dysvascular hand, defined as cool, blue, greater than 2 mm capillary refill, is an emergency that must be addressed within 6 hours of injury. The treatment of all such fractures is reduction and internal fixation by the orthopedic surgeon. There is no role for preoperative diagnostic arteriography.[6]

Neural injury occurs in approximately 10% to 15% of supracondylar humerus fractures. Most frequently involved are median and radial nerves. The child is tested by

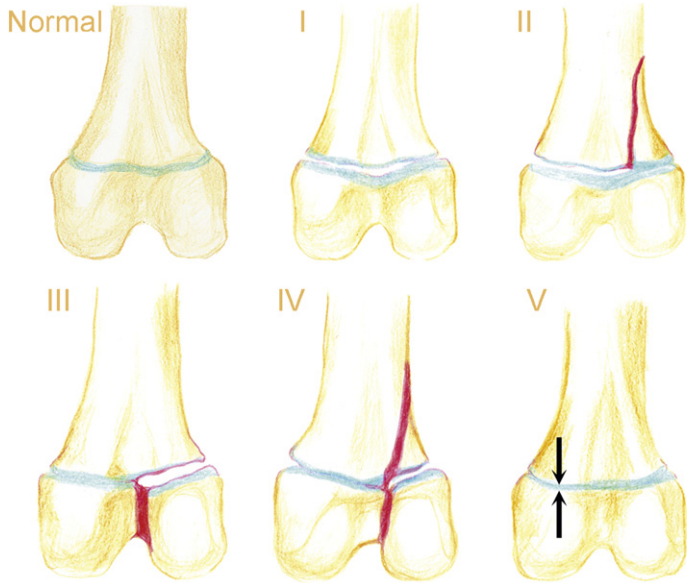
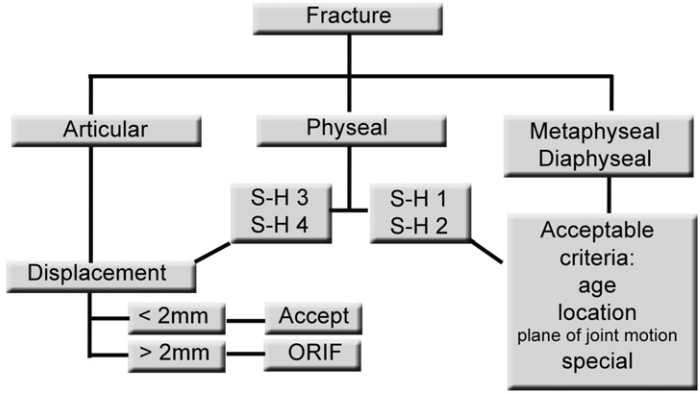


Fig. 1. Salter-Harris Classification. Illustration of the Salter-Harris classification of physeal fractures.* Normal: illustration of a normal pediatric distal femur. I: fracture through the physis. II: fracture through the physis that exits through the metaphysis. III: intra-articular fracture through the physis that exits through the epiphysis. IV: intra-articular fracture that involves the metaphysis and epiphysis, crossing the physis. V: crush injury to the physis. *Illustration by artist Joan R. Kaiser.

asking them to form the "OK sign" with the index and thumb (anterior interosseous branch of the median nerve) and the "thumbs up" sign (posterior interosseous branch of the radial nerve). The mechanism of this injury is stretched across the proximal fragment, and it represents neurapraxia and not nerve palsy. The usual outcome of such neurapraxias is full recovery that may take up to 6 months.[7–10] As a result,



*S-H: Salter Harris classification
**ORIF: Open reduction internal fixation

Fig. 2. Fracture Algorithm. ORIF; open reduction internal fixation; S-H, Salter Harris classification.

Table 2
Pediatric fracture limits

Pediatric Fracture Limits[a]			
Bone	**Age**	**Angulation (°)**	**Translation**
Hand			
Phalanx	<10	25 apex dorsal/volar	Articular displacement <2 mm
Phalanx	>10	15 apex dorsal/volar	Articular displacement <2 mm
5th Metacarpal neck fracture (Boxer's or Brawler's)	All	30–40	
1st Metacarpal base (thumb)	All	30–40	
Wrist			
Distal radius physeal (Salter-Harris)	<10	30	2 mm articular; 50% displacment at physis in sagittal plane
Distal radius physeal (Salter-Harris)	>10	20	2 mm articular
Distal radius metaphyseal	<10	30 sagittal/20 coronal	Bayonette acceptable up to age 10 years
Distal radius metaphyseal	>10	20 sagittal	50% in coronal plane
Forearm			
Shaft	<10	30	Bayonette accepted
Shaft	>10	20	Bayonette accepted
Radial neck	All	30	30%
Humerus diaphysis	All	15–30 varus/valgus, 15–20 anterior/posterior	Bayonette accepted
Proximal humerus	<10	90	100%
Proximal humerus	>10	45	
Femur			
Femoral shaft	0–1[b]	30 varus/valgus, 30 anterior/posterior	2.5 cm shortening
Femoral shaft	1–5	15 varus/valgus, 20 anterior/posterior	2.5 cm shortening
Femoral shaft	>5[c]	minimal	minimal
Tibia			
Tibia diaphysis	<10	10 valgus, 10 varus, 10 anterior, 10 posterior.	10 mm shortening, evaluate subcutaneous border
Tibia diaphysis	>10	10 valgus, 10 varus, 10 anterior, 10 posterior.	5 mm shortening, evaluate subcutaneous border

[a] Authors' recommendations.
[b] Most deformity is accepted; these fractures are treated in a Pavlik harness with only the soft-tissue forces being corrective.
[c] All are treated surgically.

Table 3
Open fractures

Treatment of Open Fractures		
G/A Type	**I**	**II, III**
Wound Size	**Small**	**Large**
Mechanism	Inside out	Outside in
Agent	Bone	Foreign
Contamination	Clean	Dirty
Tetanus toxoid	YES	YES
Antibiotics	Oral	IV: cephalosporin, aminoglycoside
Irrigation	Local treatment	Operative I & D
Fixation	Immobilize	ORIF

Abbreviations: G/A, Gustillo Anderson classification; I & D, irrigation and debridement; ORIF, open reduction and internal fixation.

supracondylar humerus fracture with nerve out is not an emergency. The fracture may be referred the next office day and is treated by the orthopedic surgeon according to fracture principles uninfluenced by the neural status. Ulnar nerve injury is most often iatrogenic, from insertion of a medial distal humerus fixation wire at operation. Even in such cases, recovery is universal after removal of the wire.

FRACTURE: COMPARTMENTAL SYNDROME

In the steady state of limb perfusion, arterial inflow equals venous outflow (**Fig. 3**). In compartmental syndrome, this equilibrium is lost. Muscles, nerves, and vessels that

Table 4
Appropriate initial radiologic studies[a]

Proximal humerus	AP glenohumeral joint (Grashey) Axillary lateral
Humeral shaft fracture	AP/lateral humerus
Supracondylar humerus	AP/lateral distal humerus
Lateral condyle fracture	AP/lateral/two 45 degree obliques distal humerus
Proximal radius fracture	AP/lateral elbow Radiocapitellar view
Forearm fracture	AP/lateral forearm
Wrist fracture	AP/lateral wrist (clenched and unclenched fist)
Scaphoid fracture	AP/lateral/oblique hand or wrist Scaphoid view
Proximal femur fracture/SCFE	AP pelvis Frog leg or cross-table lateral hip
Femoral shaft	AP/lateral femur
Acute knee injury	AP/lateral/notch/Merchant knee
Tibial shaft	AP/lateral tibia
Ankle fracture	AP/lateral/mortise ankle
Foot fracture	Weight bearing AP/lateral/oblique foot

[a] Fracture-specific radiographs are listed only. In general, the joint immediately above and below the fracture should also be imaged (eg, the elbow and wrist should also be imaged in a forearm fracture). If injury or displacement is unclear, imaging the contralateral side may be of use.
Abbreviation: AP, anteroposterior.

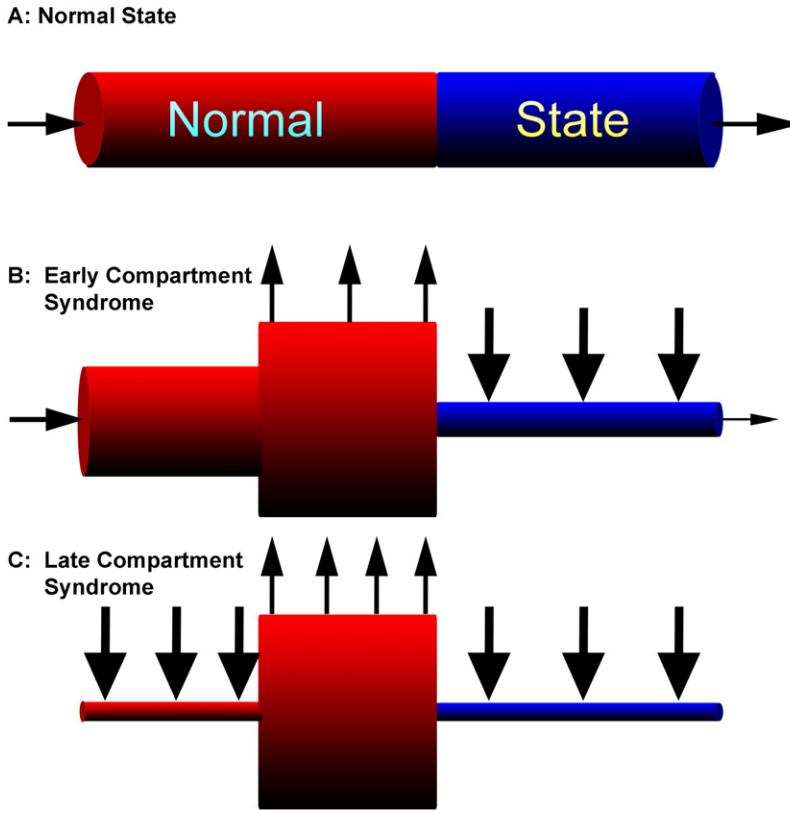


Fig. 3. Compartmental Syndrome. (*A*) Normal state: arterial inflow = venous outflow. (*B*) Early compartment syndrome: increase in compartment pressure decreases venous outflow. Arterial inflow preserved/augmented by inflammatory cascade and increased vascular permeability. Symptoms/signs: pressure (tense limb), pain (out of proportion). (*C*) Late compartment syndrome: decreased venous outflow leads to rapid increase in pressure, arterial flow compromised = ISCHEMIA. Symptoms/signs: paresthesias, lack of pulse, pallor.

course through the extremity do so within inelastic fascial membranes. Ischemia results when pressure in these compartments increases to become greater than the perfusion pressure. The forearm (especially after supracondylar humerus fracture) and leg (after tibia fracture) are most commonly affected.

Compartmental syndrome is characterized by the following sequence.[11] In the setting of the fracture, increased arterial inflow and hemorrhage from the ends of the fracture fragments cause increased pressure within the compartment. Increased pressure leads to compression followed by occlusion of venous outflow, which further increases compartmental and intra-arterial pressure. Increased intra-arterial pressure increases hemorrhage. When the intra-compartmental pressure approaches the arterial pressure, arterial inflow decreases and ischemia results. Transition to anaerobic metabolism in the setting of ischemia leads to the buildup of lactic acid, loss of the osmolar gradient, and leaky capillary membranes, which leads to a cascade of neutrophil activation, free-radical generation, and intravascular coagulation.[12] The result is a feedback loop that progresses to muscle necrosis.

The physical examination findings in compartmental syndrome are a tense limb and pain, and when advanced, paresthesias, lack of pulse, polar (coolness), and pallor. Pain is the most sensitive sign and is characteristically severe, out of proportion to the injury, and refractory to routine analgesia. Pain with passive stretch of a tendon that runs through the compartment is most suggestive of compartment syndrome.

A tense limb with firm compartments on palpation is worrisome for compartmental syndrome. Invasive pressure measurement can be a useful adjunct to clinical examination, with an intra-compartmental pressure greater than 30 mmHg and a calculated difference between the diastolic blood pressure and intra-compartmental pressure less than 30 mmHg both suggestive of compartmental syndrome with sensitivity of greater than 80%.[13–15]

Pressure and pain are associated with venous obstruction, although the latter three (paresthesias, lack of pulse, and pallor) are characteristic of arterial insufficiency. Intervention initiated after onset of paresthesias, lack of pulse, or pallor may be too late to avoid permanent muscular necrosis, with its attendant loss of function and risk of infection. Plaster cast univalving can reduce compartment pressure by 40% to 60% and release of the underlying padding may reduce pressure by an additional 10%.[16] When external restrictive pressure generators are removed and compartment pressures remain unacceptable, surgical fasciotomy to allow dissipation of pressure becomes necessary. For full recovery from muscular injury, fasciotomy must be instituted within 6 hours of onset. In adults, intervention after 36 hours is not recommended; muscle recovery is sufficiently unlikely to be outweighed by a high risk for infection caused by exposure of dead tissue.

Compartmental syndrome in children differs from that in adults. Physical examination findings can be milder and children can be more difficult to examine and may not be able to describe their pain as clearly as adults. This difficulty can result in delayed diagnosis, so the level of suspicion should remain high.

BONE AND JOINT INFECTION

Joints may be divided into axial, including hip and shoulder, or appendicular, including elbow, wrist, knee, and ankle. Appendicular joints are distinguished by being visible and palpable, making them easier to access for aspiration and easier to follow for response to treatment. The knee is the most frequently infected pediatric joint. Pyarthritis of the hip is known to have the most severe consequences.

Appendicular infections often present with swelling, erythema, tenderness, and decreased joint range of motion. This decreased range of motion is often caused by swelling and splinting by patients. Axial infections are more difficult to diagnose clinically, and therefore supplemental tests are often necessary. One study that reviewed children diagnosed with either transient synovitis or septic arthritis of the hip found four predictive criteria for septic arthritis. These criteria were fever greater than 38.5°C, inability to bear weight on the affected limb, leukocyte count greater than 12,000/mm^3, and erythrocyte sedimentation rate greater than 40 mm/h. The predicted probability of septic hip arthritis in these subjects was approximately 10% for one predictor, approximately 35% for two, approximately 73% for three, and approximately 93% for four.[17] In addition, C-reactive protein less than 1 mg/dL has an 87% negative predictive value,[18] and a repeat visit to the doctor may have positive predictive value.[19] The neonate may not demonstrate the same immune response to infection, so these criteria should be applied cautiously to this population.

Axial joint infection is treated with incision, drainage, irrigation and débridement. This treatment may be performed emergently if the child is systemically in distress

or to avoid destruction of articular cartilage. Multiple studies have shown the destructive action of bacterial infection on articular cartilage. Poor prognosis has been associated with a delay in diagnosis/treatment greater than 4 days.[20] Children who have an unclear diagnosis of joint infection may be brought back within 4 days for a repeat assessment and surgical treatment, if necessary, without significant long-term consequence.

For appendicular joints, aspiration is central to diagnosis and treatment. Aspiration provides fluid for analysis, including cell count, Gram stain, culture and sensitivity, and crystals, and may be sufficient treatment when supplemented with antibiotics.[21] The following is an ideal sequence:

1. Joint aspiration in the emergency department, which is often the best setting for such a procedure in children
2. Admission to hospital for intravenous antibiotics
3. Immobilization of the joint
4. Modification of antibiotics according to culture and sensitivity analysis
5. Ensuring clinical response, including defervescence greater than 24 hours and reduced pain with improved range of motion
6. Ensuring laboratory response, including C-reactive protein less than 2 mg/dL
7. Discharge to home on oral antibiotics
8. Cessation of antibiotics when erythrocyte sedimentation rate is less than 20 mm/hr

In this sequence, the typical duration of intravenous antibiotics is less than 1 week, and the overall antibiotic course is less than the traditional 6 weeks.

Bone infections may be divided into those that are with and without change seen on radiograph. In the absence of radiographic change, the bone infection may be treated in the same manner as an appendicular joint infection, beginning with aspiration when the infected bone is accessible. Accessibility is associated with the following: metaphyseal location (the most common site) where the cortex is thin; hyperemia that softens bone; or the presence of periosteal abscess that obviates the need to penetrate the bone. Correct location for needle insertion may be determined by point of maximal tenderness, overlying erythema, and a mini fluoroscope to locate the nearest metaphysis.

Radiographic change requires greater than or equal to 50% bone loss. In addition, a ''hole in bone'' appearance represents an intraosseous abscess that is walled off by a sclerotic margin (known by the Latin involucrum, ''wrapper''), and that may contain a central fragment of necrotic bone (known by the Latin sequestrum, ''isolated thing''). The involucrum and sequestrum are impenetrable by antibiotics. This fact, together with extent of bone loss, make surgical intervention for débridement essential when there is radiographic change. After adequate surgical treatment, which may require multiple débridements, treatment resembles that for bone infection without radiographic change.

A bone or joint infection is only an emergency if children manifest signs of systemic infection (eg, cutaneous flushing, hypotension). Pyarthritis is regarded as urgent and is treated ideally within 6 hours with evacuation, either by way of a needle or incision. However, gray-zone presentations may be observed and reassessed within 4 days of onset without significant clinical sequela. Osteomyelitis with radiographic change will need operation and may wait until evaluation by an orthopedic surgeon within a week. Osteomyelitis without radiographic change should not be treated with antibiotics (unless patients are in distress) until an osseous specimen is obtained.

LIMPING CHILD

Disturbance of gait producing a limp may result from pain, weakness, neuromuscular imbalance, or deformity. An understanding of the gait cycle, a careful history, thorough physical examination, appropriate radiographic imaging, and laboratory studies can help the emergency physician greatly narrow the possible diagnoses causing the limp.

Gait may be divided into a swing phase, during which the referenced limb is off the ground, and a stance phase, which may be further divided into heel strike, flat foot, and push off. An antalgic gait is defined by a shortened stance phase, which in the extreme is manifested by a refusal to walk. A common theme in hip deformities that produce a limp is a Trendelenburg gait. This gait results from a shift of the body over the affected hip to reduce the momentum exerted on weak abductor muscles, which aids them in maintaining a horizontal pelvis during stance phase. In milder deformities, the gait may be apparent only after several cycles and may give way to pain as the hip abductors increasingly fatigue. This type of pain may be distinguished by its more lateral location, where the hip abductors are attached to the ilium and greater trochanter. By contrast, pain in articular hip disorders is typically referred anteriorly in the region of the groin and less frequently in the anteromedial thigh and knee. This obeys the law of Hilton, which states that a nerve supplying a muscle that acts upon a joint will supply the given joint and the skin over the distal attachment of the given muscle.[22] In the hip, the obturator and femoral nerves supply motor innervation to the adductors and rectus femoris respectively, and sensory innervation of the hip joint and the anteromedial thigh.

During physical examination the child should be undressed fully and the resting position of the limbs should be noted. The examination should be performed in supine and prone positions. The supine position may show hip obligate lateral rotation with flexion as seen in hip pyarthritis or SCFE. The prone position has the distinct advantage of allowing uncoupling of the knee from the hip, in which the pathology of one may masquerade as the other. In the prone position, the knee may be ranged from extension to flexion without moving the hip. In the supine position, moving the knee requires flexion of the hip, making it difficult at times to tell which joint is the offender. In addition, as in examination for torsion, the prone position allows simultaneous comparison of hip rotation (especially medial), which is the most sensitive to disease. Finally, the prone position can reveal a hip flexion contracture that may be concealed by lumbar hyperlordosis.

Studies of the limping child show that the cause is not always identified; no definitive diagnosis is made in up to 30% of cases.[23]

Most Common Cause: Transient Synovitis

Although the cause remains uncertain, transient synovitis may be thought of as a reactive arthritis that affects the hip. Often there is a history of antecedent infection and the condition occurs most frequently in the infant or young child. Transient synovitis is characterized by an antalgic gait, normal temperature to low fever, reduced range of motion of the hip, and normal to mildly elevated serologic markers of inflammation. Ultrasound shows a non-echogenic effusion. Treatment consists of symptomatic care, including activity modification and nonsteroidal antiinflammatory agents. In the majority of patients, symptoms and signs resolve over 1 week without sequelae.

Transient synovitis is a diagnosis of exclusion and must be distinguished from hip pyarthritis (*vide supra*). Typically in transient synovitis, patients do not appear to be systemically ill, are able to walk, motion of the hip is supple in the mid range while restricted at the extremes, and improvement begins within 72 hour. Temperature less

than 37.5°C and a erythrocyte sedimentation rate less than 40 mm/hr may be used to help exclude pyarthritis.[24] It is essential to recognize that these distinctions are guidelines; the two disorders present on a continuum and may overlap. Where doubt exists, ultrasound-guided or fluoroscopically-guided diagnostic aspiration of the hip with fluid analysis for white blood cell count and bacterial staining and culture should be performed.

Discitis

Discitis represents infection of the metaphysis of the body of a vertebra that manifests in the intervertebral disk. It presents as a triad of pain, fever, and reduced intervertebral disk height on spinal radiograph. Pain in the infant typically manifests as refusal to walk; the child may complain of vague abdominal pain and often it is not until adolescence that the process can be localized to the back. Access to the intervertebral disk occurs through vascular channels that traverse the ring epiphyses in the immature skeleton.

The radiographic signs of discitis, intervertebral disk space narrowing and osseous erosion of the endplates, may be absent up to 2 to 6 weeks after the onset of symptoms.[25] In cases of clinical suspicion with negative radiographs, technetium bone scan can confirm the diagnosis and increased uptake can be seen as early as 7 days after symptom onset.[25] MRI can also assist in the diagnosis, but should usually be reserved for patients who have negative radiographs and bone scans, and for patients whose clinical course has not responded to antibiotics. Discitis may be diagnosed and treated before and without radiographic change.

The most common offending organism is *Staphylococcus aureus*. Biopsy is usually not performed because the cultures are often negative. Patients should be started on antistaphylococcal intravenous antibiotic therapy empirically and are typically transitioned to oral therapy with clinical resolution. Bracing is controversial.

Slipped Capital Femoral Epiphysis (SCFE)

SCFE is the second most commonly missed time-sensitive pediatric orthopedic problem, secondary to fracture, with frequent delays in diagnosis and treatment.[26] The disorder is characterized by disruption of the proximal femoral physis. The metaphysis displaces anteriorly and superiorly relative to the epiphysis, which remains tethered in the acetabulum by the ligamentum teres. SCFE is often likened to an ice cream scoop (epiphysis) falling off its cone.

Boys are affected more often than girls, and black and Hispanic children are affected more frequently than white children.[27] The diagnosis usually correlates with the rapid growth spurt of puberty. The average age of diagnosis is 11.2 years in girls and 12.7 years in boys, and the age at onset is declining.[27] SCFE is often associated with obesity; over half of these patients are above the 95th percentile for weight.[28]

Bilateral SCFE is common, and approximately 50% of the cases of bilateral SCFE present with simultaneous bilateral involvement.[29] If the diagnosis is suspected, both hips should be imaged. The risk of contralateral involvement is 2335 times higher in patients with the diagnosis of a unilateral slip than those who have not had a slip.[30]

There is an increased incidence of SCFE in patients who have endocrinopathies, such as hypothyroidism, hypogonadism, and growth hormone therapy.[28] Most children who have SCFE do not have a diagnosed endocrinopathy, but because of the high prevalence of a common somatotype (eg, obese, male, hypogonadal, pubescent), many think that these children have a subtle underlying endocrine disorder.[28]

Adolescents typically present with pain and a limp. Approximately 85% complain primarily of hip, groin, or proximal thigh pain, but 15% have mostly knee or distal thigh

pain.[31] Patients who present with primarily knee pain are more likely to receive a misdiagnosis and to have slips of greater severity.[31] The vast majority of patients will report symptoms lasting weeks to months, or even years.

On examination, patients who are ambulatory will have an antalgic or Trendelenburg gait, and the affected extremity may appear externally rotated and short. There is a loss of hip range of motion, especially internal rotation, but also flexion and abduction. On hip flexion, there is an obligate external rotation of the hip because the proximal metaphysis abuts and slides along the anterior acetabular rim.

The diagnosis can usually be made with anteroposterior (AP) and frog-leg lateral pelvis radiographs. The frog-leg lateral film demonstrates subtle displacement more clearly than the standard AP pelvis. The physis will appear widened and blunted, and in cases of chronic SCFE, may show signs of remodeling. Klein's line, drawn along the anterosuperior aspect of the femoral neck on the AP radiograph, should intersect the epiphysis in the normal hip.[32] This line is tangential to or distant from the slipped epiphysis (**Fig. 4**). In cases of high clinical suspicion and negative radiographs, the diagnosis of a pre-slip can be made with bone scintigraphy.

There are three characteristics of SCFE that are especially useful to the orthopedic consultant:

Chronicity: symptoms for less than 3 weeks are described as acute and greater than 3 weeks as chronic.
Stability: a patient who is able to walk on a SCFE has a stable condition, whereas if they are unable, it is described as an unstable condition.
Severity: this refers to the angle subtended by the capital epiphysis and shaft of the femur, and is described as mild when this is less than 30°, moderate between 30°to 60°, and severe when greater than 60°.

Complications of SCFE, notably osteonecrosis of the head of the femur and chondrolysis (loss of articular cartilage) of the hip-joint (defined as joint width $\leq$2 mm), are associated with acute, unstable, and severe presentations.[33]

Treatment in the emergency setting is removal of weight bearing from the affected limb. Although crutches may achieve this goal, a wheel chair in an over-weight and noncompliant teenager may be safer, with consultation with an orthopedic surgeon in less than 8 hours.

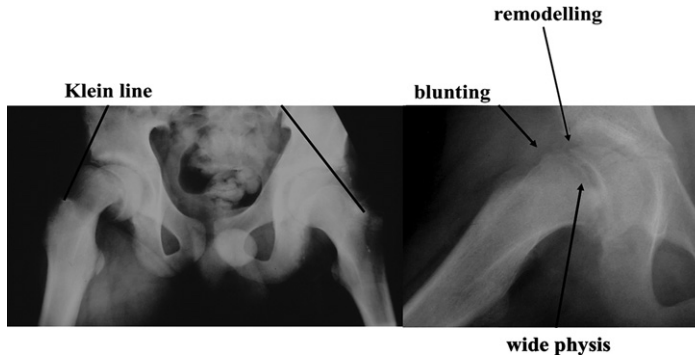


Fig. 4. Radiographic signs of SCFE.

NON-ACCIDENTAL TRAUMA

Child abuse has an unacceptably high rate of morbidity and mortality; 35% of children who are returned to their environment without intervention will be reinjured and 5% will die of abuse.[34] Thirty percent of physically abused children will require orthopedic treatment.[35]

The US Department of Health and Human Services reported that approximately 794,000 children were victims of abuse in 2007.[36] The following are risk factors for non-accidental trauma: **Box 2**

- Young age: children younger than 4 years make up 30% of total child abuse victims and 75% of the fatalities.[36]
- Single parent: this increases risk of child abuse by 77% compared with two-parent homes.[37]
- Low economic status: annual family income less than $15,000 increases risk for abuse by 22 times compared with income greater than $30,000.[37]
- Comorbidity: nearly 50% of non-accidental trauma victims older than 3 years have psychiatric or neurologic conditions.[38]

Whether race correlates with a difference in maltreatment is debatable.[36,37] There is no significant gender difference.[36]

The most frequent physical manifestation of child abuse is soft-tissue injury (>90%).[39] It is therefore imperative that the child be undressed fully for an inspection of the skin in its entirety. Although a characteristic lesion may indicate the method or instrument used (eg, iron mark, cigarette burn, scalded/dipped baby), most soft-tissue injuries are in an unremarkable pattern. The incidence of musculoskeletal injury in non-accidental trauma correlates with age. Eighty percent of fractures in abused children occur before 18 months of age; by contrast, 85% of accidental fractures occur after 5 years of age.[40]

High-risk fracture patterns are listed in **Table 5**. The diaphysis of a long bone is the most common site of fracture in physical abuse.[41] Approximately 80% of femur fractures in children younger than 2 years are associated with abuse.[42] A humeral diaphysis fracture before 3 years of age represents non-accidental trauma until proven otherwise.[40,43] In contrast to diaphyseal humerus fracture, supracondylar humerus fracture is the most frequent indication for operative fixation of a fracture in a child. Associated features that increase the significance of diaphyseal fractures are exuberant callus caused by inadequate immobilization, multiple fractures and fractures in various stages of healing, inappropriate clinical history, and delay in seeking medical attention.[35,43]

Metaphyseal-epiphyseal fractures of the distal femur, tibia, and proximal humerus usually heal with no consequence but are highly clinically relevant for their association

Box 2
Risk factors for abuse

Risk Factors:
Young age
Single-parent home
Income <$15,000
Neurologic/Psychiatric condition

Table 5
Characteristic injuries of non-accidental trauma

Most Common	Suspicious
Soft-tissue injury	Metaphyseal-epiphyseal fractures: corner fracture bucket handle fracture
—	Distal humerus transphyseal fracture
—	Femur fracture in child before walking
—	Humerus diaphyseal fracture in child <3y
—	Rib fractures, especially posterior
—	Fractures in various states of healing

with abuse. Children evaluated with these fractures should be evaluated for shaking injuries, such as head trauma. Because of indirect force (eg, shaking), the periosteum avulses the immature bone near the physis in a planar fashion, resulting in the radiologic finding of a corner fracture or bucket handle fracture (**Fig. 5**).[44] In contrast, accidental trauma usually produces metaphyseal fractures that are closer to the diaphysis, such as in a Salter-Harris type II fracture or torus fracture.

Other fractures that should cause a high level of suspicion are posterior rib fractures and scapula fractures. The mobility of the ribs makes them difficult to break accidentally. Their posterior aspect is struck directly as the child flees. The scapula is a dense, flat bone embedded in muscle on all sides, and the energy required to fracture it is high and rarely occurs with everyday childhood trauma.

The principal morbid entity in the differential diagnosis of non-accidental fracture is osteogenesis imperfecta. This may be distinguished by

- Osseous quality (eg, gracility, osteopenia, global deformation)
- Presence of wormian bones, which represent multiple non-coalesced centers of ossification in the skull
- Family history
- Blue sclera, caused by thinning and translucency exposing the subjacent choroid
- Short stature
- Possible associated broken and yellow-brown teeth, caused by fragility of the dentin
- Possible associated hearing loss

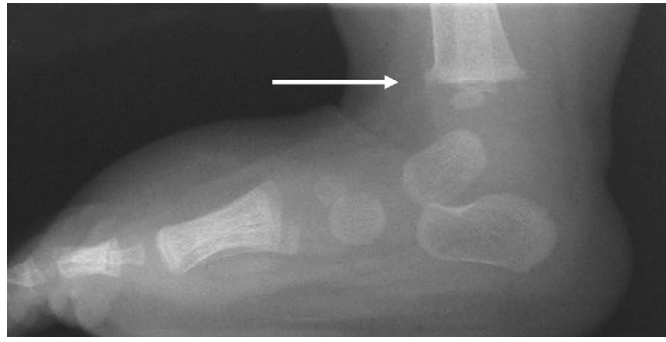


Fig. 5. Corner fracture. Radiograph of a distal tibia corner fracture. The classic metaphyseal-epiphyseal injury can be seen clearly in this radiograph.

Abused children continue to be inadequately treated. Physeal-metaphyseal fractures can have a mild clinical presentation, characterized by less pain and less displacement than other fracture patterns.[43] Up to 90% of children who have clinical evidence of abuse do not receive radiographs.[39] A radiographic skeletal survey should be performed in all children younger than 3 years in whom abuse is suspected:

- AP imaging of the appendicular skeleton: femur, tibia-fibula, foot, humerus, radius-ulna, and hand
- AP and lateral imaging of the axial skeleton: spine, chest, and skull
- A survey 10 to14 days after the initial evaluation may reveal occult fracture based upon periosteal new bone formation.[45]

Every family must be evaluated equally, regardless of socioeconomic status, appearance, or other biasing trait. Evaluation is multidisciplinary, including physician, social worker, nurse, mental health professional, and a representative from Child Protective Services.[46] Document every aspect of the presentation fully; approximately 40% of charts do not contain adequate information to determine the cause of fracture in children.[47] If there is uncertainty, admit patients.

SUMMARY

Pediatric fractures, compartment syndrome, limp, infection, SCFE, and nonaccidental trauma can all be a challenge to diagnose and treat for the emergency room physician and orthopedic surgeon. Missed diagnoses and mismanagement of these conditions can have disastrous consequences for patients and family members. A high level of awareness and the proper diagnosis are the first steps in management. With the described treatment algorithms in this review article, physicians can better avoid the associated pitfalls of these urgent orthopedic conditions.

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REFERENCES

1. Salter RB, Harris WR. Injuries involving the epiphyseal plate. J Bone Joint Surg Am 1963;45:587–622.
2. Knirk JL, Jupiter JB. Intra-articular fractures of the distal end of the radius in young adults. J Bone Joint Surg Am 1986;68(5):647–59.
3. Skaggs DL, Friend L, Alman B, et al. The effect of surgical delay on acute infection following 554 open fractures in children. J Bone Joint Surg Am 2005;87(1): 8–12.
4. Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am 1976;58:453–8.
5. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type III (severe) open fractures: a new classification of type III open fractures. J Trauma 1984;24:742–6.
6. Shaw BA, Kasser JR, Emans JB, et al. Management of vascular injuries in displaced supracondylar humerus fractures without arteriography. J Orthop Trauma 1990;4(1):25–9.
7. Cramer KE, Green NE, Devito DP. Incidence of anterior interosseous nerve palsy in supracondylar humerus fractures in children. J Pediatr Orthop 1993;13(4):502–5.

8. Campbell CC, Walters PM, Millis MB. Neurovascular injury and displacement in type III supracondylar humerus fractures. J Pediatr Orthop 1995;15:47–52.
9. Culp RW, Osterman AL, Davidson RS, et al. Neural injuries associated with supracondylar fractures of the humerus in children. J Bone Joint Surg Am 1990;72(8): 1211–5.
10. Dormans JP, Squillante R, Sharf H. Acute neurovascular complications with supracondylar humerus fractures in children. J Hand Surg Am 1995;20(1):1–4.
11. Matsen FA III, Hargens AR. Compartment syndromes and Volkmann's contracture. Philadelphia: WB Saunders; 1981.
12. Matsson-Hultén L, Holmström M, Soussi B. Harmful singlet oxygen can be helpful. Free Radic Biol Med 1999;27:1203–7.
13. Janzing HM, Broos PL. Routine monitoring of compartment pressure in patients with tibial fractures: beware of overtreatment!. Injury 2001;32(5):415–21.
14. Heppenstall RB, Sapega AA, Scott R, et al. The compartment syndrome. An experimental and clinical study of muscular energy metabolism using phosphorus nuclear magnetic resonance spectroscopy. Clin Orthop Relat Res 1998; 226:138–55.
15. Matsen FA 3rd, Winquist RA, Krugmire RB Jr. Diagnosis and management of compartmental syndromes. J Bone Joint Surg Am 1980;62(2):286–91.
16. Halanski M, Noonan KJ. Cast and splint immobilization: complications. J Am Acad Orthop Surg 2008;16(1):30–40.
17. Kocher MS, Mandiga R, Zurakowski D, et al. Validation of a clinical prediction rule for the differentiation between septic arthritis and transient synovitis of the hip in children. J Bone Joint Surg Am 2004;86(8):1629–35.
18. Levine MJ, McGuire KJ, McGowan KL, et al. Assessment of the test characteristics of C-reactive protein for septic arthritis in children. J Pediatr Orthop 2003;23: 373–7.
19. Luhmann SJ, Jones A, Schootman M, et al. Differentiation between septic arthritis and transient synovitis of the hip in children with clinical prediction algorithms. J Bone Joint Surg Am 2004;86:956–62.
20. Choi IH, Pizzutillo PD, Bowen JR, et al. Sequelae and reconstruction after septic arthritis of the hip in infants. J Bone Joint Surg Am 1990;72:1150–65.
21. Herndon WA, Knauer S, Sullivan JA, et al. Management of septic arthritis in children. J Pediatr Orthop 1986;6:576–8.
22. Hilton J. On rest and pain: a course of lectures on the influence of mechanical and physiological rest in the treatment of accidents and surgical diseases, and the diagnostic value of pain. In: Jacobson WHA, editor. Lectures delivered at the Royal College of Surgeons of England 1860–1862. New York: William Wood and Company; 1879. p. 191–210.
23. Fischer SU, Beattie TF. The limping child: epidemiology, assessment and outcome. J Bone Joint Surg Br 1999;81:1029–34.
24. Del Beccaro MA, Champoux AN, Bockers T, et al. Septic arthritis versus transient synovitis of the hip: the value of screening laboratory tests. Ann Emerg Med 1992; 21:1418–22.
25. Szalay EA, Green NE, Heller RM, et al. Magnetic resonance imaging in the diagnosis of childhood discitis. J Pediatr Orthop 1987;7(2):164–7.
26. Skaggs DL, Roy AK, Vitale MG, et al. Quality of evaluation and management of children requiring timely orthopaedic surgery before admission to a tertiary pediatric facility. J Pediatr Orthop 2002;22:265–7.
27. Lehmann CL, Arons RR, Loder RT, et al. The epidemiology of slipped capital femoral epiphysis: an update. J Pediatr Orthop 2006;26(3):286–90.

28. Aronsson DD, Loder RT, Breur GJ, et al. Slipped capital femoral epiphysis: current concepts. J Am Acad Orthop Surg 2006;14:666–79.
29. Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis. A study of children in Michigan. J Bone Joint Surg Am 1993;75:1141–7.
30. Schultz WR, Weinstein JN, Weinstein SL, et al. Prophylactic pinning of the contralateral hip in slipped capital femoral epiphysis: evaluation of long-term outcome for the contralateral hip with use of decision analysis. J Bone Joint Surg Am 2002; 84:1305–14.
31. Matava MJ, Patton CM, Luhmann S, et al. Knee pain as the initial symptom of slipped capital femoral epiphysis: an analysis of initial presentation and treatment. J Pediatr Orthop 1999;19(4):455–60.
32. Klein A, Joplin RJ, Reidy JA, et al. Slipped capital femoral epiphysis: early diagnosis and treatment facilitated by "normal" roentgenograms. J Bone Joint Surg Am 1952;34:233–9.
33. Loder RT, Richards BS, Shapiro PS, et al. Acute slipped capital femoral epiphysis: the importance of physeal stability. J Bone Joint Surg Am 1993;75:1134–40.
34. Schmitt B, Clemmens M. Battered child syndrome. In: Touloukin R, editor. Pediatric trauma. St Louis (MO): Mosby-Year Book; 1990. p. 161–87.
35. Akbarnia B, Torg JS, Kirkpatrick J, et al. Manifestations of the battered-child syndrome. J Bone Joint Surg Am 1974;56:1159–66.
36. US Department of Health and Human Services. Child maltreatment 2007. Available at: http://www.acf.hhs.gov/programs/cb/pubs/cm07/cm07.pdf. Accessed May 10, 2009
37. Sedlak AJ, Broadhurst DD. Executive summary of the third national incidence study of child abuse and neglect. US Department of Health and Human Services. Available at: http://www.childwelfare.gov/pubs/statsinfo/nis3.cfm. Accessed May 10, 2009.
38. Loder RT, Feinberg JR. Orthopaedic injuries in children with nonaccidental trauma. J Pediatr Orthop 2007;27(4):421–6.
39. McMahon P, Grossman W, Gaffney M, et al. Soft-tissue injury as an indication of child abuse. J Bone Joint Surg Am 1995;77:1179–83.
40. Worlock P, Stower M, Barbor P. Patterns of fractures in accidental and non-accidental injury in children: a comparative study. Br Med J (Clin Res Ed) 1986;293: 100–2.
41. Merten DF, Radowski MA, Leonidas JC. The abused child: a radiological reappraisal. Radiology 1983;146:377–81.
42. Anderson WA. The significance of femoral fractures in children. Ann Emerg Med 1982;11(4):174–7.
43. Carty HM. Fractures caused by child abuse. J Bone Joint Surg Br 1993;75: 849–57.
44. Kleinman PK. Diagnostic imaging in infant abuse. AJR Am J Roentgenol 1990; 155(4):703–12.
45. Kleinman PK, Nimkin K, Spevak MR, et al. Follow-up skeletal surveys in suspected child abuse. AJR Am J Roentgenol 1996;167(4):893–6.
46. McDonald KC. Child abuse: approach and management. Am Fam Physician 2007;75(2):221–8.
47. Oral R, Blum KL, Johnson C. Fractures in young children: are physicians in the emergency department and orthopedic clinics adequately screening for possible abuse? Pediatr Emerg Care 2003;19(3):148–53.