Approach to Knee Effusions

David J. Mathison, MD† and Stephen J. Teach, MD, MPH*†*

Abstract: The presence of an intra-articular knee effusion requires an extensive differential diagnosis and a systematic diagnostic approach. Pediatric knee effusions occur most commonly as acute hemarthroses after traumatic injury. However, the knee joint is susceptible to effusions secondary to a wide variety of atraumatic causes. Special attention is required in the atraumatic effusion to distinguish features of infectious, postinfectious, rheumatologic, hematologic, vasculitic, and malignant disease. This review discusses the various etiologies of both traumatic and atraumatic pediatric knee effusions highlighting the historical, physical examination, and laboratory characteristics to aid the emergency provider in diagnosis and initial management.

Key Words: knee effusion, knee injury, septic arthritis, joint effusion, fracture, anterior cruciate ligament, patellar dislocation, arthrocentesis, Lyme disease

(*Pediatr Emer Care* 2009;25: 773–790)

TARGET AUDIENCE

This CME activity is intended for physicians, nurse practitioners, and physician assistants who care for children in an emergency or primary care setting.

LEARNING OBJECTIVES

After participating in the activity, the paticipant should be better able to:

1. Outline a pragmatic approach to the child and adolescent with either an acute or a chronic knee effusion.
2. Use distinguishing historical and physical examination features to differentiate causes of traumatic knee effusions.
3. Describe various etiologies of atraumatic effusions and the important laboratory and radiographic features that can aid in diagnosis.

ILLUSTRATIVE CASES

A 14-year-old adolescent boy presents to the emergency department after injuring his knee playing soccer. He recalls pivoting and twisting his right knee, then hearing a "pop." He was unable to bear weight at the scene and was subsequently carried off the field. On examination, he has no obvious deformity but has a large effusion in his right knee with limited flexion. An anterior drawer test is limited by pain and swelling, but there is laxity on the Lachman maneuver. Results of both the McMurray test and the Bounce test are positive. His collateral ligaments appear intact, and his patella does not appear to be subluxable. A radiograph of his knee is negative for fracture or dislocation.

A 15-year-old adolescent girl presents to the emergency department with several weeks of worsening left knee pain. She denies fevers, foreign travel, sexual activity, or other joint symptoms. She spent the recent summer camping although has no known tick exposures. She has family members with lupus, but she is otherwise healthy and takes no medications. On examination, she has limited flexion of her knee. There is notable swelling with a fluid wave appreciated in the suprapatellar pouch. There is no warmth or erythema. A lateral radiograph shows signs of an effusion, but there are no bony abnormalities. The remainder the results of her physical examination is unremarkable.

Primary knee complaints are frequent in the pediatric emergency department, particularly among adolescent athletes who injure the knee more than any other body part, except the ankle.[1] Most knee pathologic diseases are related to minor trauma such as muscle strains, ligamentous sprains, and apophyseal overuse injuries. Patients with large knee effusions, however, are distinct. Although these effusions are most frequently hemarthroses from acute traumatic injuries, the clinician needs a broad differential to exclude the various nontraumatic causes of knee effusions specific to the pediatric patient (Table 1).

HISTORY

Patients with large effusions often have painful joints that are difficult to examine. It is therefore crucial to obtain a thorough history to elucidate risk factors for certain diseases. The clinician should ascertain information about acuity, duration, severity, signs of systemic disease, associated symptoms, and risk factors for various diseases. Examples of historical questions include the following:

- Is the swelling acute, subacute, or chronic?
- Has there been a traumatic event or repeated overuse?
- Are other joints involved?
- Are there signs of systemic illness (fevers, rash, etc)?
- Has there been recent travel to high-risk areas (ie, Lyme, brucellosis, tuberculosis)?
- Has there been any exposure to ticks?
- Have any new over-the-counter, herbal, or prescription medicines been used?
- Have there been any recent unprotected sexual encounters?
- Is there a coexisting chronic illness (ie, inflammatory bowel disease [IBD], lupus)?
- Is there a history of injury or surgery to the joint?
- Is there a family history of rheumatologic or autoimmune disease?

*Fellow (Mathison), Associate Chief (Teach), Division of Emergency Medicine, Children's National Medical Center; and †Instructor in Pediatrics (Mathison), Professor of Pediatrics and Emergency Medicine (Teach), George Washington School of Medicine and Health Sciences, Washington, DC.
Reprints: David J. Mathison, MD, Division of Emergency Medicine, Children's National Medical Center, 111 Michigan Ave NW, Washington, DC 20010 (e-mail: dmathiso@cnmc.org).

Unless otherwise noted below, each faculty's spouse/life partner (if any) has nothing to disclose.
The authors have disclosed that they have no significant relationship with or financial interests in any commercial companies that pertain to this educational activity.
All staff in a position to control the content of this CME activity have disclosed that they have no financial relationships with, or financial interests in, any commercial companies pertaining to this educational activity.
Lippincott CME Institute has identified and resolved all faculty conflicts of interest regarding this educational activity.

ISSN: 0749-5161

TABLE 1. Differential Diagnosis for Pediatric Knee Effusions

Traumatic
Ligamentous (ACL, PCL)
Fracture
Patellar dislocation
Meniscus injury
Nontraumatic
Infectious
Osteomyelitis with effusion
Classic bacterial (*Staphylococcus*, *Streptococcus*, gonorrhea)
Tick-borne disease (Lyme)
Zoonoses (brucellosis)
Mycobacterial (tuberculosis)
Fungal
Viral
Postinfectious
Reiter syndrome (seronegative reactive arthritis)
Acute rheumatic fever
Poststreptococcal reactive arthritis
Rheumatologic
Spondyloarthropathies (IBD, celiac, psoriatic arthritis)
Systemic lupus erythematosus
Juvenile idiopathic arthritis
Behçet disease
Vasculitis-associated
Henoch-Schönlein purpura
Wegener granulomatosis
Vitamin C deficiency
Hematologic
Hemophilia (factor deficiency)
Other coagulopathy (drug-induced or systemic disease)
Malignancy
Leukemia
Primary bone neoplasms (osteosarcoma, Ewing sarcoma)
Synovial sarcoma
Chondroblastoma
Metastatic disease
Other
Benign tumors
Infiltrative (gout, pseudogout)
Serum-like sickness reaction
Familial Mediterranean fever

Examples of descriptive questions specific to a traumatic event include the following:

- Was the injury a contact or a noncontact injury?
- What was the joint position during injury (ie, flexion, extension)?
- What was the mechanism of injury (ie, cutting, pivoting, twisting)?
- Have there been any mechanical symptoms (ie, locking, popping, or catching)?
- Was there any ability to bear weight after the injury?
- Have there been signs of instability (ie, "giving-way")?
- Where is the location of the pain?

ANATOMY

The knee is the largest joint in the body. It is formed by 3 articulations: the medial femoral and tibial condyles, the lateral femoral and tibial condyles, and the patella (Fig. 1). There are 4 major *ligaments* that provide stability: the anterior and posterior cruciate ligaments (ACL and PCL, respectively), which prevent dislocation of the tibia against the femur, and the medial and lateral collateral ligaments, which withstand medial and lateral stressors. The major *muscle groups* surrounding the knee are the quadriceps, the hamstring, the plantaris, the popliteus, and the gastrocnemius. The quadriceps femoris is the principal component for leg extension and one of the most powerful muscle groups in the body. It is composed of 4 parts: rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis. Leg flexion is principally accomplished by the hamstrings, composed of the semitendinosus, semimembranosus, and biceps femoris muscles. The popliteus, plantaris, and gastrocnemius all offer additional support in leg flexion.

The *patella*'s anterior position allows the quadriceps tendon to insert further from the joint's axis thus creating additional joint leverage. This mechanical advantage allows the joint to withstand friction from flexion and extension during running and to withstand compression of the quadriceps tendon during kneeling. The *menisci* (from the Greek word *meniskos*, meaning crescent) are wedge-shaped plates of fibrocartilage that act as shock absorbers for knee movements. The menisci are bridged by the transverse ligament of the knee, which allows them to move together during knee motion. The medial collateral ligament at its midpoint inserts into the medial meniscus, helping to explain why injuries to this ligament often have concomitant meniscal tearing. The *synovium* is a thin membrane that lines the knee capsule attaching to the lining of the articular

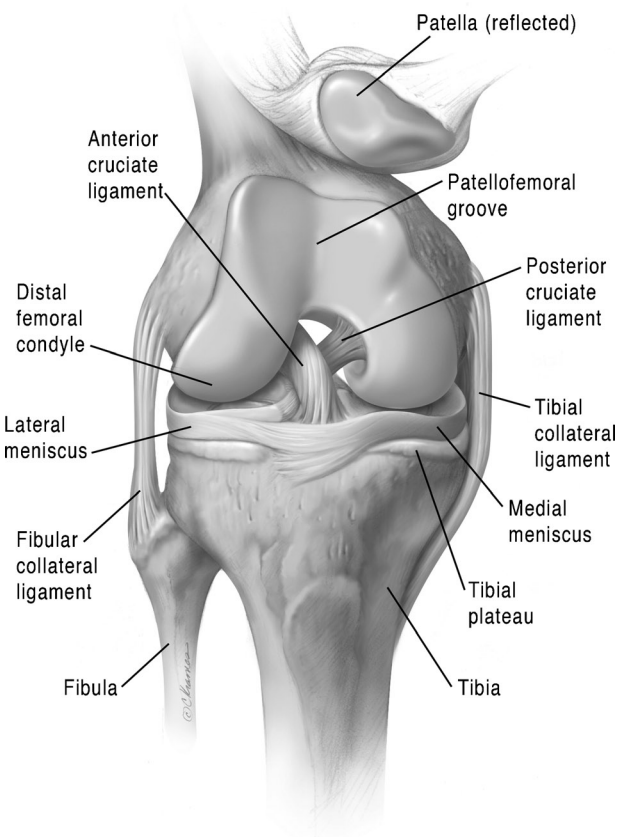

FIGURE 1. Knee anatomy. Illustration by Christy Krames, MA, CMI.

surfaces and fibrocartilaginous menisci. The synovium contains microvilli that secrete the clear mucoid synovial fluid. This fluid serves a critical role in joint lubrication, nutrition, and maintenance of joint integrity (by removing debris).

The apophysis is a secondary ossification center where a muscle-tendon unit inserts into bone. Apophyseal centers contribute to bone shape but not length and are therefore particularly vulnerable during growth spurts when long bones grow faster and often in a different direction than these sites of muscle-tendon insertion. The apophysis (also called traction epiphysis) is therefore prone to apophysitis and avulsion fractures peaking between ages 8 and 15 years. Apophysitis commonly involves the knee, particularly at the tibial tuberosity (Osgood-Schlatter disease) and the inferior pole of the patella (Sinding-Larsen-Johanssen [SLJ] syndrome).

PHYSICAL EXAMINATION

A thorough physical examination is often the key to diagnosis in the acutely traumatized knee. In the patient with a chronic or subacute knee effusion, the physical examination should focus on distinguishing traumatic, septic, and arthritic effusions.

Appearance

Comparison to the other knee joint is essential. Obvious deformities or dislocations should be immediately apparent. Erythema and warmth may be a sign of either infection or inflammation. The presence of a rash should be noted, particularly the classic erythema migrans of Lyme disease or the palpable purpura of Henoch-Schönlein purpura (HSP) vasculitis. In the chronically painful knee, it is helpful to evaluate variations in alignment such as genu varum or hip abnormalities that may be contributing risk factors for overuse injuries.

Neurovascular Status

Evaluation of neurovascular integrity dictates how quickly to intervene in stabilizing the joint. Injury to the popliteal artery warrants emergent consultation with a vascular surgeon. Peroneal nerve and posterior tibial nerve injuries can also occur. Evaluation of neurovascular status should include palpation of the dorsalis pedis and posterior tibial pulses, plantar flexion of the ankle (posterior tibial nerve), eversion of the ankle (superficial peroneal nerve), extension of the great toe (deep peroneal nerve), and assessment of pedal sensation.

Presence of Effusion

The absence of "dimples" when comparing knees is often the first sign of an effusion (Fig. 2A). Large effusions (20–30 mL) will additionally occupy the suprapatellar space and displace the patella anteriorly. The balloon test uses the thumb and finger on each hand to compress the suprapatellar pouch against the femur rendering the effusion palpable with the thumbs. Similarly, by pushing the raised patella posteriorly, a palpable tap can be appreciated on the patella against the femoral condyles, known as the patellar ballottement test. In the absence of a large effusion, the clinician should attempt to elicit a fluid wave by milking the fluid from the lateral to medial side and then quickly pressing the opposite (lateral) side to see a recess created by the fluid wave, known as a bulge sign (Fig. 2B).

Evaluate for Patellar Dislocation

Acute dislocation is usually obvious on clinical examination. In a lateral patellar dislocation, the knee is held in 20 to 30 degrees of flexion, and there is tenderness along the medial edge of the patella and the medial femoral epicondyle. A patellar dimple sign is a palpable transverse defect at the medial joint line that can sometimes be appreciated as a sign of patellar tendon disruption associated with patellar dislocation or subluxation.

Palpation

This allows the clinician to localize the pain while feeling for signs of fracture (point tenderness or crepitus). The characteristic features of tendinitis and apophysitis are localized points of maximal tenderness to sites of ligamentous and tendon insertions. Common sites for apophysitis in the growing athlete include the tibial tuberosity and the inferior pole of the patella. Tenderness along the medial/lateral joint lines may indicate meniscal tears. In a patient with osteochondritis dissecans, tenderness can be elicited by direct palpation over the articular surface of the medial femoral condyle. A joint that is subjectively

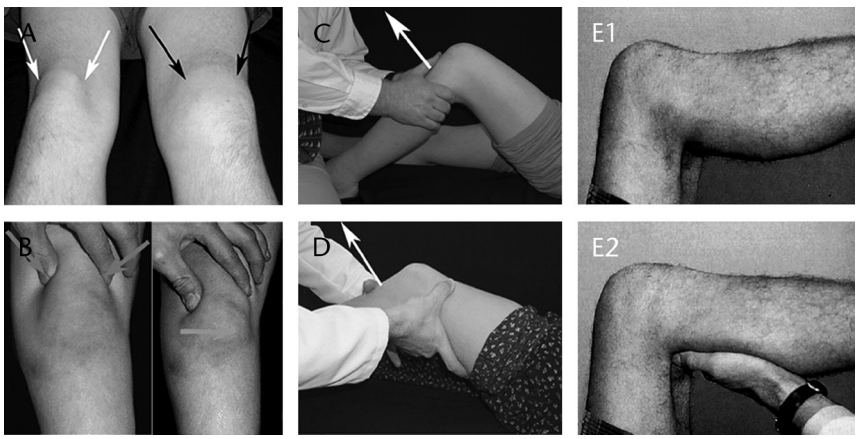

FIGURE 2. Physical examination technique. The presence of an effusion effaces the natural knee dimples (A).* An effusion can appear as fullness of the suprapatellar pouch (B)† and a fluid wave can be often be appreciated by sliding the patella. Both the anterior drawer test (C)* and the Lachman maneuver (D)* can demonstrate laxity of the ACL. The posterior sag test uses gravity to demonstrate laxity of the PCL (E1,2).‡ [*Reprinted with permission from Willis RB. *Lovell & Winter's Pediatric Orthopedics, 6th ed.* Morrissy RTW, Stuart L, eds. Lippincott Williams & Wilkins; 2006. †Reprinted with permission from *Staheli: Practice of Pediatric Orthopedics. 2nd ed.* Lippincott Williams & Wilkins, 2006. ‡Reprinted from *Clin Pediatr Emerg Med*, Vol 8, LaBella CR, Common acute sports-related lower extremity injuries in children and adolescents, 31–42, 2007, with permission from Elsevier Limited.

painful but does not have objective findings of swelling or pain on palpation differentiates an arthralgia from a true arthritis.

Passive Range of Motion (Flexion/Extension)

Limited flexion or extension can be caused by a large joint effusion or from other swelling to the knee area such as from a cellulitis, contusion, or a Baker cyst. Pain on flexion or extension may additionally be due to tendinitis in the hamstring or quadriceps muscle groups, respectively.

ACL Integrity

The anterior drawer test tests ACL integrity by stabilizing the foot and drawing the tibia anteriorly with the knee at 90 degrees and the hamstring relaxed (Fig. 2C). The Lachman test similarly evaluates ACL stability but with the knee at 20 degrees of flexion (Fig. 2D). If either test produces 5 mm or greater of translation/subluxation without a clear stop point, the joint has ACL laxity. The Lachman maneuver may be a more sensitive indicator of ACL injury than the anterior drawer test and is often easier to perform in the acute swollen knee.[2]

PCL Integrity

With the patient supine and knees at 90 degrees, the posterior sag test shows PCL laxity when the PCL-injured leg lags posteriorly relative to the contralateral side (Figs. 2E1 and E2). The quadriceps active test is performed by opposing the foot as the patient attempts to slide the tibia distally from a prone position. Posterior cruciate ligament laxity can cause an anterior tibial reduction with this maneuver. A posterior drawer test further demonstrates PCL laxity when the tibia can be posteriorly displaced using a posterior force on the tibia against the femur.

Meniscal Injury

A McMurray test is performed with the patient supine and the knee flexed, using internal and external tibial rotation while holding fingers against the medial and lateral joint lines. A positive test result is indicated by pain and a palpable "clunk," occurring as the torn meniscus is trapped during rotation. The specificity for meniscal injury of a positive McMurray test result is 97%.[3] The Apley (compression) test is a similar test for meniscal tears performed with the patient prone and with the knee flexed to 90 degrees. When applying pressure to the foot with medial and lateral rotation, a similar meniscal trapping can occur. A third maneuver is the bounce test, where the patient lies supine with the knee flexed to 15 degrees and the clinician drops it with gravity into extension. If a torn meniscus is present, the reflex contraction of the hamstring prevents the knee from hyperextending, which indicates a positive result on the bounce test. This "true locking" can also occur with a loose body or a torn ACL.

Collateral Stability

Both in full extension and at 30 degrees of flexion, the collateral ligaments are tested by applying medial and lateral stresses to assess the degree of laxity. Laxity greater than 30 degrees with either the varus stress test (lateral collateral ligament) or the valgus stress test (medial collateral ligament) is indicative of a collateral ligament sprain.

Patellofemoral Instability

This is a common cause of knee pain that can be elicited on physical examination after ruling out various ligamentous, meniscal, and bony abnormalities as described previously. In patellofemoral injury, the patellar inhibition test generates pain over the patella when the patient tightens the quadriceps against a force to the proximal patellar tendon. Another helpful test is the patellar apprehension test where the patella is pushed laterally with the knee at 30 degrees. A positive test result is indicated by immediate quadriceps contraction (leg extension) preventing the patella from subluxating, an indication of patellofemoral instability and often a sign of previous patellar dislocation.

DIAGNOSTIC EVALUATION

Most children with effusions of the knee, particularly in the setting of traumatic injury, require only plain radiographs of the joint for evaluation. Occasionally, laboratory testing and/or arthrocentesis may be necessary. Rarely will additional studies such as magnetic resonance imaging (MRI), ultrasound, or referral for arthroscopy play a role in the emergency department.

Imaging

A standard knee series should consist of anterior-posterior and lateral radiographs. Additional views are helpful, particularly a sunrise (infrapatellar/axial-patellar) view to look for patellar subluxation or fracture, oblique views to assess for tibial plateau fractures, and intracondylar views to visualize osteochondral lesions. The lateral view obtained at 15 to 30 degrees of flexion is the most helpful in visualizing a large knee effusion, which is seen with the widening of the suprapatellar bursa to greater than 5 mm in width. Lateral radiographs may be capable of detecting effusions as small as 1 to 2 mL.[4]

The Ottawa Knee Rules were created to decide when to order radiographs to evaluate for fractures in adults after knee trauma.[5] They were validated in adults with a sensitivity of 100% for clinically significant fractures (any bone fragment at least 5 mm displaced or any avulsion fracture with complete disruption of the ligament/tendon).[5–8] In the growing skeleton, however, small, nondisplaced fractures may be more clinically significant. The Ottawa criteria were validated in a prospective multicenter trial of 750 children aged 2 to 16 years with acute knee injuries[9] (Table 2). In this study, any bone fragments were considered "clinically significant." As in the adult study, the overall sensitivity and negative predictive value were 100%. Although the presence of an effusion is not part of the Ottawa criteria, hemarthrosis within 24 hours of injury has been correlated with fractures[10,11] and should be an automatic indication for knee radiographs.

In addition to evaluating for fracture and effusions, standard radiographs can demonstrate properties of inflammatory or infection such as bony erosion, epiphyseal enlargement, osteoporosis, and deformity of the joint surface. In children with chronic inflammatory joint disease, radiographs can be helpful to evaluate the degree of knee destruction.

Magnetic resonance imaging is the most sensitive and detailed modality for infectious and inflammatory disorders of the knee.[12,13] Magnetic resonance imaging detects early inflammatory changes in both the synovium and the bone, which can be useful in the acute nontraumatic effusion. It can also quantify synovial inflammation,[14–16] rendering it a useful tool in chronic inflammatory effusions to help guide response to therapies. Magnetic resonance imaging also offers a more detailed

TABLE 2. Ottawa Knee Rules in Children*

Inability to flex the knee to 90 degrees
Inability to bear weight both immediately and in the ED for 4 steps (limp is ok)
Isolated tenderness to the patella
Tenderness at the head of the fibula

*From Bulloch et al.[9]

view of soft tissue, which is advantageous in evaluating posttraumatic injuries, particularly with meniscus tears and ligamentous rupture.

The use of MRI in the posttraumatic hemarthrosis knee is debated. Although it can be a diagnostic aid, it does not seem that MRI is adequate in differentiating the need for surgical versus conservative therapy. Although it might be a sensitive tool to occasionally avoid unnecessary arthroscopy,[17,18] MRI may be insufficiently sensitive particularly in demonstrating meniscal and medial collateral ligament tears[19,20] while creating some false-positive meniscal findings. Other disadvantages of MRI include cost, availability, and need for sedation in children (often <6 years). Few pediatric centers have MRI accessibility from the ED for noncritical imaging.

Ultrasonography is fast, simple, inexpensive and free of radiation. It can assess for joint effusion, synovial thickening, and cartilage damage, although not as well as MRI. When the presence of a knee effusion is in question, ultrasound can be a helpful adjunct and guide for arthrocentesis of small effusions. Ultrasound is more accurate in detecting effusions than clinical examination.[21] Delaunoy et al[22] showed in cadaveric adults that at least 10 mL of blood or 7.4 mL of synovial fluid is required for ultrasound to detect a knee effusion.

Laboratory Studies

In nontraumatic effusions, the following laboratory studies should be considered:

- Erythrocyte sedimentation rate (ESR) and C-reactive protein are nonspecific markers of inflammation that can be useful in differentiating infectious or inflammatory effusions from noninflammatory effusions (ie, hemophilia or benign tumor).
- A white blood cell count can additionally show signs of acute inflammation while also showing signs of systemic disease such as leukopenia from human immunodeficiency virus (HIV) or peripheral blasts from acute leukemia.
- Infectious studies should be targeted to specific diseases elicited in the history, such as zoonoses or arthropod-borne illnesses. Routine blood cultures should be performed on all patients with new-onset febrile effusions, although the yield is often low, even in the presence of a distal femoral osteomyelitis or a septic joint. Enzyme-linked immunosorbent assay for Lyme disease is a sensitive but not specific marker, and a positive result should be confirmed with Western blot immunoglobulin M/G titers. The febrile agglutinins test can be effective in identifying *Brucella* species in the patient who has had recent animal exposure or consumed unpasteurized products. Viral titers should be considered particularly in a patient with chronic blood transfusions at risk for hepatitis B or C. Urine polymerase chain reaction tests for *Chlamydia* and gonorrhea can be helpful in the sexually active patient.
- Streptococcal infection can be assessed with a throat swab for concurrent illness or by antistreptolysin O or anti–DNAse-B titers to assess for antecedent infection.
- Coagulation studies should be considered in the patient with a presumed bleeding diathesis, and an elevated partial thromboplastin time should be followed with specific factor levels.
- Rheumatologic markers such as an antinuclear antibody (ANA) and double-stranded DNA can help diagnose specific systemic illnesses such as systemic lupus erythematosus (SLE). In patients with chronic rheumatologic disease, measures of immune activation such as complement levels and neopterin may be helpful to gauge disease activity but rarely affect decision making in the emergency department.
- Creatine kinase elevation indicates rapid breakdown of muscle and can be helpful in distinguishing an acute myositis from an acute arthritis.
- Urinalysis can show additional markers of inflammation or signs of systemic disease. Pyuria can be present in serum sickness–like reaction or genitourinary tract infections such as urethritis. Microscopic hematuria can be a sign of intrinsic kidney disease such as from SLE nephritis or can occur from an acute vasculitis such as HSP.

Synovial Fluid Aspiration (Arthrocentesis)

Analysis of synovial fluid can distinguish an inflammatory from noninflammatory arthritis and further identify specific causative organisms, crystals, blood, or malignant cells (Table 3). Normal synovial fluid is acellular, clear, and viscous and contains a protein concentration similar to plasma and a glucose concentration about one-third of plasma. Diagnostic aspiration of the synovium should include a culture, Gram stain, lactate dehydrogenase, protein, glucose, cell count, and microscopic examination for crystals or malignant cells. Arthrocentesis has the additional therapeutic advantage of alleviating pressure and pain, particularly in the patient with an acute traumatic effusion. Contraindications to arthrocentesis include an overlying cellulitis or a neutropenic patient—both increasing the potential for bacteria seeding into the joint cavity.

Arthroscopy

Knee arthroscopy is the criterion standard for evaluating and repairing soft tissue injuries in the patient with traumatic hemarthrosis without significant bony abnormalities. Neither a clinical examination under anesthesia nor MRI is 100% sensitive for excluding intra-articular pathology, especially for non-ACL injuries. Although a Lachman ACL test under anesthesia may be as sensitive as 98%,[23] as few as 43% of patients with meniscal tears and 17% of patients with an osteochondral fracture were predictive with examinations under anesthesia before definitive arthroscopy.[24]

TABLE 3. Interpretation of Synovial Fluid Aspirate

	Noninflammatory	Inflammatory	Bacterial (Septic)	Hemorrhagic
Clarity	Transparent	Translucent-opaque	Opaque	Bloody
Color	Yellow	Yellow	Yellow-green	Red
Viscosity	High	Low	Variable	Variable
White blood cell counts, μL	200–2000	2000–10,000	>100,000	200–2000
Polymorphonuclear cells, %	<25	>50	>75	50–75
Protein, g/dL	1–3	3–5	3–5	4–6
Glucose, mg/dL	=blood	<blood	≪blood	=blood

ETIOLOGY

The etiology of knee effusions can be broadly categorized as either traumatic or nontraumatic. Nontraumatic effusions can be subdivided as infectious, postinfectious, rheumatologic, hematologic, vasculitis-associated, malignant, and others (Table 1).

Traumatic Effusions

Traumatic knee effusions are uncommon after knee injuries, particularly in younger children who have greater ligamentous laxity. Muscle strains, contusions, and overuse injuries are the most common pediatric knee injuries, all of which are extra-articular and are not associated with traumatic hemarthroses. Risk factors predisposing children and adolescents to knee injury include long cleats, alternative playing surfaces, lack of conditioning, female sex, previous injury, and anatomic abnormalities. The child with a traumatic knee effusion, however, is distinct and requires a thorough evaluation for diagnosis and management.

Anterior Cruciate Ligament

Either partial or complete ACL ruptures are the most common cause of acute hemarthrosis in adult athletes, occurring in as many as 70% to 86% of traumatic effusions.[18,24–26] In studies confined to the pediatric population (<17 years), however, ACL tears are less prevalent, occurring in 10% to 63% of traumatic effusions.[10,27–29] Anterior cruciate ligament tears are rare before the age of 11 and occur in females 6 to 8 times more frequently than in boys playing the same sports.[30,31] Anterior cruciate ligament injuries occur most commonly in competitive athletics but usually without any contact with another player.[32] The mechanics of these tears are often twisting injuries such as changing directions, decelerating abruptly, or landing from a jump. Athletes often feel a pop and have immediate swelling, instability, and the inability to bear weight. The sports most commonly implicated in the United States are basketball, soccer, football, and volleyball.

The knee examination is easier and more revealing soon after the injury before significant hemarthrosis, spasm, and pain develop. The Lachman test is generally the most sensitive sign on physical examination. The absence of a knee effusion virtually excludes an acute ACL tear.[33] Testing the contralateral joint is important to evaluate for constitutional laxity or congenital absence of the ACL. Radiographs will often show the presence of a joint effusion. A Segond fracture (lateral capsular sign) is an avulsion of the lateral tibia by the lateral capsular ligament and is pathognomonic for intra-articular injury, often an ACL tear. Radiographs should also evaluate for tibial eminence avulsion fractures (discussed below). Femoral avulsions are very rare.

Posterior Cruciate Ligament

Posterior cruciate ligament injuries often result from direct trauma to the anterior tibia against a flexed knee. Unlike ACL injuries, a PCL tear can occur without a joint effusion, and athletes often can continue playing sports after a substantial PCL tear. Seventy percent of PCL injuries occur at the tibial insertion, often occurring in conjunction with other ligamentous injury. A common athletic cause of PCL rupture is a home plate collision playing baseball. Posterior cruciate ligament rupture occurs more frequently in motor vehicle accidents particularly in the front passenger seat when the knee hits against the dashboard. Posterior cruciate ligament injury occurs in 38% to 44% of knee hemarthrosis from motor vehicle collisions; however, as few as 7% occur in isolation.[34,35] Rarely, an avulsion of the deep medial collateral ligament attachment on the tibia can be seen on a radiograph, conveniently known as a reverse Segond fracture.

Fractures

Isolated fractures often need to be displaced and associated with meniscal or ligamentous damage to cause a knee effusion. Fractures of the distal femur and proximal tibia or fibula often have swelling that extends well outside the joint space, easily distinguishing these injuries from an isolated hemarthrosis. Fractures of the tibial eminence and inferior patella, however, can be subtle and can clinically mimic soft tissue injuries—yet, are important to distinguish because they can require more aggressive treatment.

Tibial eminence (spine) fractures are a variant of ACL disruption in which the bony insertion of the ligament on the intercondylar tibial eminence is avulsed. Tibial eminence fractures occur most often between the ages of 8 and 14 and often in a fall from a bicycle or motorcycle.[36] Meniscal injury and collateral ligament injury can also occur with tibial spine fractures. A displaced tibial eminence fracture can limit full extension[37] and is often accompanied by long-term ACL laxity.[38] Tibial eminence fractures should not be confused with tibial plateau fractures. The proximal tibia head is composed of the medial and lateral tibial plateaus, as well as the intercondylar eminence. Tibial plateau fractures are most common in the elderly secondary to osteoporosis but can occur in children. They are sometimes referred to as "bumper" or "fender" fractures owing to their frequent occurrence from hitting the dashboard in motor vehicle collision.

Patellar fractures constitute up to 1% of all skeletal injuries.[39] They can occur either indirectly from abrupt deceleration causing rapid quadriceps contraction or directly from a force such as a fall or blunt trauma to the anterior patella. Patients with patellar fractures present with localized tenderness and hemarthrosis. Children and adolescents are more prone to avulsion injuries at the inferior patellar pole that include large sections of the chondral surface. These avulsions are known as "sleeve fractures" and can appear benign radiographically because the large amount of displaced cartilage is not visualized. On examination, sleeve fractures have a palpable gap at the inferior patella, and patients are unable to perform a straight leg raise. This fracture must be differentiated from SLJ syndrome, an overuse apophysitis syndrome in children 10 to 14 years characterized by fragmentation of the inferior patellar pole. Unlike sleeve fractures, SLJ syndrome is chronic, does not have hemarthrosis and is self-limited. Sleeve fractures must also be differentiated from a type 1 (lower pole) bipartite patella, a congenital fragmentation of the patella that occurs in 1% of the population. Contralateral films may be helpful to distinguish congenital bipartite patella.

Patellar Dislocations

Acute patellar dislocations are the second most common cause of traumatic hemarthrosis.[40] The mechanism of injury is a sudden internal rotation of the femur with the foot in a fixed position. Several anatomical factors can predispose children to primary and recurrent dislocations, including patella alta, lateral patellar displacement, increased Q angle, genu valgum, vastus medialis muscle hypoplasia, external tibial torsion, and increased femoral anteversion. As many as 90% of patellar dislocations reduce spontaneously with knee extension.[41] If the dislocation is still present in the ED, prompt reduction with analgesia and sedation is necessary. Patellar reduction can be accomplished by applying an anteromedial force to the lateral

side of the patella while another practitioner slowly extends the knee to relax the hamstrings.

First-time traumatic patellar dislocations have been traditionally treated conservatively with nonoperative management. However, without surgical correction, redislocation may occur in 44% of patients, and joint instability may occur in more than 50% of patients.[42] Open or arthroscopic procedures are recommended if there are large osteochondral fractures, major chondral injuries, substantial disruption of the patella-stabilizing structures, patellar subluxation, or redislocation.[43]

Osteochondral Fractures

Osteochondral fractures of the distal femur occur commonly with patellar subluxation and occur in as many as 31% to 72% of children with traumatic patellar dislocation.[44,45] However, only 32% may be seen on traditional radiographs, even with a sunrise view.[46] Because these lesions are often not visualized on traditional radiographs or even MRI, they can be neglected and found unexpectedly on arthroscopy or open procedures.[47] Osteochondral fractures in isolation are an uncommon cause of hemarthrosis but can occur in conjunction with other interarticular injuries such as ACL or PCL tears. The presence of fat globules in aspirated synovial fluid or the identification of lipohemarthrosis on knee ultrasound are additional signs that osteochondral fractures may be present. After 10 days of osteochondral displacement, the fragment may not fit back into place, and this fragmentation can lead to osteoarthritis. Surgical treatment depends on the delay after injury, the size, and the location of the fracture.

Meniscal Tears

Meniscal tears occur with twisting injuries, most commonly in football and basketball. With isolated meniscal tears, most children can ambulate after the injury, but pain and swelling follow during the next 24 to 48 hours. In an acute tear, patients often report a popping sensation. In a more subacute presentation, the meniscal tear can lead to instability when a fragment is trapped between the articular surfaces leading to a locking feeling or "giving out." Both acute and subacute presentations can have knee effusions, although the subacute are unlikely to develop hemarthroses. Meniscal tears may occur in isolation or in association with an ACL or PCL injury. Meniscal tears occur primarily in the skeletally mature athlete and are uncommon in children younger than 10 years, especially in those with morphologically normal menisci. In a study of children and adolescents correlating the physical examination with MRI and arthroscopy, the physical examination for meniscal injury had a sensitivity of 92.8%, a positive predictive value of 93.3%, and a specificity of 92.3%.[48]

Management Tips

The initial therapy for all traumatic effusions should focus on pain control. Sedation may be necessary for arthrocentesis or patellar reduction. In the absence of a significant fracture or neurovascular compromise, orthopedic consultation in the emergency department is usually not necessary. All patients with traumatic effusions require follow-up with an orthopedic surgeon within 7 days of injury where further diagnostic options such as MRI and arthroscopy would be discussed.

Nontraumatic Effusions

In the absence of acute or subacute injury, the causes of knee effusions are diverse and require a thorough history and physical examination to evaluate for key risk factors, family history, and signs of systemic disease.

Infectious Effusions

Osteomyelitis

Because osteomyelitis and septic arthritis are both primarily hematogenous in origin, it is not uncommon for a septic joint to present as a sequelae of acute hematogenous osteomyelitis (AHO). When bacteria seed the metaphysis of a long bone, there is potential for local spread either into the subperiosteum or into the joint capsule, the latter causing a septic joint. In a series of 66 patients with AHO, 22% had a septic arthritis at the joint adjacent to the infected bone.[49] The knee in particular is a common site for AHO joint seeding, which occurred in almost half of the tibia/fibula AHO cases in this series.

Septic Arthritis

Septic arthritis is an infection within a joint space, although the term is often used to indicate a bacterial infection. The septic joint is classically warm, swollen, and erythematous with limited range of motion. The patient is typically febrile, although fevers may be absent in as many as 40% of children.[50] According to the American College of Rheumatology, unexplained inflammatory fluid particularly in a febrile patient should be assumed to be infected until proven otherwise by culture.[51] A positive result in the synovial culture is diagnostic, and therefore, a synovial aspirate with Gram stain and culture should be performed on any patient with the clinical presentation of a septic joint.

A bacterial joint infection typically has a synovial cell count of greater than 50,000 white blood cells/μL with greater than 75% polymorphonuclear cells. However, a lower cell count may be a poor negative predictor for a septic joint. McGillicuddy et al[52] found that 39% of adult patients with culture-positive synovial aspirate had cell counts less than 50,000 cells/μL. Nelson et al[53] found that 34% of children with culture-proven bacterial arthritis had cell counts less than 25,000 cells/μL. Low cell counts in septic joints are more likely to occur in the immunocompromised host or in a partially treated infection. Conversely, a very elevated synovial cell count is not always indicative of a septic joint because a synovial leukocytosis can occur in other conditions such as leukemic infiltration, rheumatoid arthritis flares, atypical infections, gout, and reactions to intra-articular injections (hyaluronans).[54] However, the odds ratios for septic arthritis increases from 2.9 with counts greater than 25,000 cells/μL to 28 with counts greater than 100,000 cells/μL.[55]

In the first 2 months of life, *Streptococcus agalactiae* and *Staphylococcus aureus* are the most common organisms isolated from a septic joint. After the early infant period, *S. aureus* is the predominant organism until adolescence when *S. aureus* and *Neisseria gonorrhoeae* are the most prevalent.[50] Other organisms that less commonly invade the joint include *Streptococcus pyogenes* and *Streptococcus pneumoniae*. *Salmonella* species can occur in the child with sickle cell anemia, and gram-negative bacilli are more often isolated from the immunosuppressed host. *Staphylococcus epidermidis* is found most commonly in the infected prosthetic joint. The increasing prevalence of methicillin-resistant *S. aureus* should prompt the physician to consider empiric initial antibiotic coverage specific for this organism.

A history of recent exposure to zoonoses or other exposures is often helpful. In 2003, a case series of *Kingella kingae* septic arthritis transmitted from toddler-to-toddler was documented in a Minnesota daycare facility. Brucellosis is endemic in certain areas, especially in communities with human-animal contact. In

a recent Israeli series of septic arthritis (mean age, 14.7 years), Brucellosis was cultured in 11% of patients with the knee joint most commonly affected.[56] Eight of these patients had close contact with livestock or had consumed unpasteurized dairy products.

Gonococcal Arthritis

N. gonorrhoeae is a common cause of septic arthritis in adolescents.[50] Gonococcal joint disease is a manifestation of disseminated gonococcal infection but can occur in 2 distinct forms: a joint-localized suppurative arthritis and a more extensive disease. When occurring as monoarticular arthritis, it is infrequently associated with bacteremia. Gonococcemia, rather, has more systemic symptoms including polyarthralgias and skin lesions. Without genital symptoms, it is impossible to clinically distinguish a gonococcal arthritis from a septic joint, and the knee joint is the most common location for suppurative gonococcal arthritis. When aspirated, the synovial leukocyte count ranges between 40,000 and 60,000 cells/μL with greater than 80% polymorphonuclear cells.[57] Cultures of the synovial fluid are positive in fewer than 50% of cases of gonococcal arthritis,[58] even with special culture inoculum such as Thayer-Martin or Transgrow mediums. Gonococcal DNA amplification should be considered both from the synovial fluid and from the urine. The organism may also be cultured from the male urethra or female cervix.

Lyme Disease

Lyme borreliosis, caused by *Borrelia* species, is transmitted to humans by the *Ixodes ricinus* complex of ticks. In the United States, the causative organism is *Borrelia burgdorferi*, although other *Borrelia* species are found in Europe and Asia. Despite increased awareness and prevention of tick bites, the incidence of Lyme borreliosis continues to increase with a near doubling of reported cases in the United States for the past decade.[59]

The musculoskeletal manifestations of Lyme disease can be either arthralgias or arthritis and can be either polymigratory or monoarticular. Although most Lyme arthritis is preceded by the hallmark rash of erythema migrans,[60] the arthritis may be the initial manifestation in 2% to 6% of cases.[61,62] The knee is the most frequently involved joint. Lyme arthritis can persist long after the resolution of systemic symptoms although early antibiotic therapy helps shorten the duration of arthritis and prevent further arthritides.[63] Left untreated, 50% to 60% of patients infected with *B. burgdorferi* develop arthritis.[64] Although even without antibiotic therapy, arthritis flares become both less frequent and less severe over time and rarely occur 5 years after the initial Lyme presentation.[65]

Diagnostic testing in Lyme arthritis is best accomplished with a rapid Lyme enzyme immunoassay, although Western blot analysis is required for confirmation. Aspiration of joint fluid should be performed in all patients with suspected Lyme arthritis. The synovial aspirate usually shows counts between 1000 and 50,000 cells/μL with a negative Gram stain,[65] although more fulminant presentations can occur, featuring marked synovial leukocytosis with neutrophil predominance.[66] Serum studies are nonspecific—white blood cell counts are typically normal, and the ESR may be normal or elevated. When the results of serologic testing are not available at presentation, Lyme arthritis may need to be treated initially as a septic joint, even with a negative synovial fluid Gram stain.

Viral Arthritis

Viral arthritides tend to present with polyarthritis, often with a short duration as part of a prodrome or the onset of infection. The most common viruses causing arthritis in all ages are parvovirus, hepatitis B, hepatitis C, rubella, and the alphaviruses.[67] Arthritis has been described with the herpes virus family[68–70] (cytomegalovirus, herpes simplex, varicella, and Epstein-Barr), although this is very uncommon, especially given how frequently these infections can occur in the pediatric population. Viral illnesses in children may play a larger role in triggering an immune-mediated process rather than being the causative agent in an infectious arthritis. Diagnosis for viral arthritides is made by serologic testing, although viral culture or DNA in synovial fluid or from other tissues can be helpful.

Internationally, the mosquito-borne RNA alphaviruses have caused outbreaks of fevers, rash, and arthritis. Alphaviruses include Ross River, O'nyong-nyong, Mayaro, Barmah Forest, Semliki Forest, Chikungunya, and Sindbis viruses. Chikungunya caused widespread outbreaks affecting the French island of Réunion in 2005 and the Adhra Pradesh state of India in 2006.[71] While adult Chikungunya infection is often hallmarked by joint symptoms, children often have no associated arthritis.[72]

Patients with HIV infection have higher incidence of several arthropathies, the immunodeficiency, immune hyperactivity, and cytokine disregulation contribute to the higher incidence of several arthropathies that include septic arthritis, reactive arthritis, psoriatic arthritis, and the diffuse infiltrative lymphocytosis syndrome. In addition, some patients experience an HIV-associated arthritis, which is a self-limited syndrome that mimics other viral arthritides, often lasting less than 6 weeks.

Mycobacterial Arthritis (Tuberculosis)

Worldwide, 10% to 11% of extrapulmonary tuberculosis involves joints or bones, with the knee being the third most commonly affected joint, after the spine and hip.[73] In 2 recent international series each with greater than 100 septic arthritis patients, tuberculosis was the causative agent in 9% to 30% of cases,[56,74] although most patients were adults. Data on tuberculosis arthritis in the pediatric population are limited. Local factors such as surgical trauma or preexisting joint disease (SLE, juvenile idiopathic arthritis [JIA], etc) are known risk factors for developing a tuberculosis joint infection. Magnetic resonance imaging demonstrates several unique features of tuberculosis-associated joint destruction including synovitis with erosions, hypointense synovium (on T1), cartilage destruction, active and chronic pannus, and cystic lesions.[75]

Fungal Arthritis

In the immunocompetent host, fungal arthritis is very uncommon but should be considered in the patient with a persistent "septic-appearing" joint and negative synovial culture results. Alterations in human flora, disruption of mucocutaneous membranes, and impaired immune function may be predisposing factors. Fungal arthritis is typically seeded from hematogenous infection. Reported microorganisms responsible for fungal arthritis include *Candida*, *Aspergillus*, *Coccidioides*, *Histoplasma*, *Blastomycosis*, *Cryptococcus*, and *Sporothrix*. Candidal arthritis most commonly involves the knee and may occur more commonly after intra-articular administration of steroids, in the neonatal population, and in intravenous drug users regardless of HIV status. *Aspergillus* musculoskeletal disease should be considered particularly in children with chronic granulomatous disease. *Coccidioides* is endemic in the southwestern United States and can have an isolated monoarticular arthritis as part of primary pulmonary coccidioidomycosis or as part of a disseminated disease.[76] *Histoplasma* and cryptococcal arthritis most often occur from local invasion of infected bone into the synovial space but are both quite rare.[77] *Blastomycosis* spores are found in soil in the

Midwestern United States and, of all the fungal arthritides, is the only microorganism that can be identified by wet mount of synovial fluid.[78] *Sporothrix* arthritis can be very indolent with an interval of onset between 2 months and 8 years after the spore infection, most commonly through a minor skin wound.

Endocarditis

Musculoskeletal symptoms including a monoarthritis occur in as many as 25% to 41% of patients with infective endocarditis, sometimes as the initial symptom.[79,80] A monoarthritis is often inflammatory and sterile secondary to immune complex deposition in the synovium. But a septic arthritis can occur secondary to the hematogenous infection, particularly with *S. aureus*.

Postinfectious Effusions

Reiter Syndrome

Reiter syndrome, more recently termed "seronegative reactive arthritis," is defined as a sterile inflammatory arthritis that develops as a sequel to a remote infection. Classically, the arthritis is culture-negative; however, occasional demonstration of antigenic material (eg, *Salmonella* and *Yersinia* DNA or RNA) has created a blurred distinction between a reactive arthritis and a postinfectious one.[81] The arthritis typically appears between several days and several weeks after an infection. The characteristic infections cause either gastrointestinal or genitourinary symptoms. Causative organisms reported in children include *Salmonella*, *Clostridium difficile*, *Giardia*, *Shigella*, *Campylobacter*, *Yersinia*, and *Chlamydia*.[82–89]

The arthritis of Reiter syndrome is asymmetric, can be monoarticular or oligoarticular, and usually involves the lower extremity. Other clinical manifestations include enthesitis, hemorrhagic cystitis, uveitis, aortitis, heart block, balanitis, and keratoderma blenorrhagica. An HLA-B27 genotype is thought to be a predisposing factor, found in more than two-thirds of patients.[90] Approximately 25% to 50% of patients develop subsequent episodes of arthritis.[81] The initial treatment should be with potent nonsteroidal anti-inflammatory drugs (NSAIDs; naprosyn, indomethacin, or ibuprofen) and antibiotics for the underlying infection, if indicated (ie, *Chlamydia*).

Acute Rheumatic Fever

Acute rheumatic fever (ARF) is the delayed systemic reaction that occurs after a pharyngeal infection with group A *Streptococcus*. The diagnosis of ARF requires 2 major Jones criteria (migratory polyarthritis, carditis, chorea, erythema marginatum, and subcutaneous nodules) or 1 major and 2 minor criteria (arthralgias, prolonged PR interval, elevated ESR/C-reactive protein, and fevers) in the presence of an antecedent streptococcal pharyngitis. The arthritis of ARF is characteristically a polyarthritis, affecting large joints in quick succession. The polyarthritis usually starts in the lower extremities (often the knees) and the onset in different joints can overlap coining the "migratory" nature. Acute rheumatic fever has occasionally been diagnosed in unique populations with a monoarthritis, therefore strict adherence to the Jones criteria may result in underdiagnosis in these populations.[91,92] The arthritis of ARF is classically rapidly responsive to salicylates, although studies have shown a similar efficacy of naproxen[93] avoiding the risk of Reye syndrome from aspirin.

Poststreptococcal Reactive Arthritis

Poststreptococcal reactive arthritis (PSRA) is a poststreptococcal reaction that does not meet the Jones criteria for ARF. Whether PSRA falls under the umbrella of ARF or if it is a distinct entity is debated. Unlike ARF, PSRA features a nonmigratory arthritis that can involve multiple joints or be monoarticular. Poststreptococcal reactive arthritis has a shorter latency period (3–16 days),[94–96] and the arthritis is prolonged and poorly responsive to salicylates or NSAIDs.[97] Similar to ARF, the knee is the most commonly involved joint affecting 61% to 75% of PSRA patients.[95,98] Fever and a scarlatiniform rash are often present during the acute phase of pharyngitis but are absent when the arthritis appears.[96] Poststreptococcal reactive arthritis in children may be faster to resolve and less likely to recur relative to adults.[98] Poststreptococcal reactive arthritis is more common among patients with HLA-DRB1 genotyping and not HLA-B27, suggesting its pathogenesis to be more closely related to ARF than to other forms of reactive arthritis.[99] It is unclear if there is any increased incidence of carditis in PSRA and therefore if there is benefit from antibiotic prophylaxis for either recurrence of disease or the prevention of carditis.

Rheumatologic Effusions

Spondyloarthropathies

The spondyloarthropathies of childhood are a group of arthritides, typically beginning in adolescence that involve either the back (sacroiliitis) or the large joints of the lower extremities. The classic spondyloarthropathies are psoriatic arthritis, reactive arthritis, and that associated with gastrointestinal disease.

Juvenile psoriatic arthritis is defined as a psoriatic rash with at least 1 arthritic joint, although the specific criteria have been modified in recent years.[100] Patients without the characteristic psoriasis rash may have early disease and meet minor criteria that include nail pitting, dactylitis, and a family history of psoriasis. In a retrospective review of 63 patients with juvenile psoriatic arthritis, the median age of onset was 4.5 years, all patients were rheumatoid factor negative, and an ANA was positive in 50%.[101] The arthritis is usually pauciarticular, and other serum markers such as ANA, anticollagen antibodies, HLA-DR4, and HLA-DR8 occur in frequencies similar to patients with JIA.[102] The knee, however, is an infrequently involved joint. Juvenile psoriatic arthritis most commonly involves the small joints of the hands and feet.

Inflammatory bowel disease has several extraintestinal manifestations including a peripheral arthritis that affects 10% to 20% of patients.[103] The arthritis in IBD can be migratory, pauciarticular, or polyarticular, and recurrence is common. When the peripheral arthritis in IBD affects less than 6 joints, it is known as a type 1 arthropathy, which most frequently occurs with flares of the bowel disease. The knee is the most commonly affected joint, and the arthritis is self-limited. Several antigens are found in increased frequency in IBD patients with axial arthritis. Human leukocyte antigen B27 (HLA-B27), in particular, is found in 50% to 75% of IBD patients with axial arthritis.[104]

Patients with celiac disease can also have associated arthropathies. Adult patients with celiac disease have an associated arthritis in 46% of cases, reduced to 24% when adequately controlled on a gluten-free diet.[105] Data on pediatric celiac disease and arthritis are limited, but it seems that patients diagnosed with JIA have a higher prevalence of coexisting celiac disease,[106] perhaps as high as 7 times that of controls,[107] which may contribute to the arthritis.

Systemic Lupus Erythematosus

Systemic lupus erythematosus is an illness of immune disregulation that commonly presents during the first 2 decades

of life. Unlike the seronegative arthropathies, immune markers, specifically the double-stranded DNA and anti-Sm (Smith) antibodies, are highly specific for SLE. The arthritis for SLE is often a polyarthritis and can present with pain out of proportion to the clinical findings.[108] Other manifestations of SLE can include malar rash, nephropathy, pleuritis, encephalopathy, cytopenias, and psychiatric symptoms. It is not uncommon for patients to be diagnosed with other arthritides such as JIA before later developing more symptoms consistent with SLE.[109,110] Patients with SLE have increased susceptibility to invasive bacterial infections, likely secondary to hypocomplementemia, functional asplenia, steroid immunosuppression, leukopenia, or abnormal macrophage function. Pneumococcal infections are particularly more common in SLE; therefore, any patient with SLE and a septic-appearing joint should be covered with empiric antibiotic therapy for invasive pneumococcus.

Behçet Disease

Behçet disease (BD) is a chronic inflammatory disorder characterized by recurrent oral aphthae, ocular disease, skin lesions, neurologic disease, and arthritis. The international diagnostic criteria include recurrent aphthae, eye lesions (uveitis or retinal vasculitis), skin lesions (erythema nodosum, pseudovasculitis, papulopustular lesions, or acneiform nodules), and a positive pathergy test result. In an international series of 86 children with BD, the mean age of onset was 8.4 years with 3 cases presenting in the first year of life. Arthritis was present in 26% of cases with the knee involved most frequently.[111] Mortality from BD usually arises from a propensity for large vessel thrombosis, although vascular complications of BD are less frequent during childhood. Treatment options for BD include colchicine and thalidomide, although both can have dangerous adverse effects.[112]

Juvenile Idiopathic Arthritis

Formerly termed juvenile rheumatologic arthritis, JIA can be characterized either as pauciarticular (<5 joints), polyarticular (>5 joints), or systemic onset. For the patient presenting with a monoarticular knee effusion, the pauciarticular subtype is the most pertinent. Systemic-onset disease often presents with prolonged fevers and rash in an ill-appearing child that must be distinguished from malignancy, Kawasaki disease, and systemic infections. Pauciarticular JIA peaks in children aged 2 to 3 years, is less common in older than 5 years, and rarely occurs after age 10. For monoarticular arthritis, symptoms must be present for at least 3 months, excluding other causes of arthritis, to make a diagnosis of JIA. If two or more joints are involved, 6 weeks of arthritis symptoms must be present. Systemic symptoms such as rash and fevers are characteristically absent, and uveitis can be associated in up to 20% of cases.[113] The knee and other large joints are most commonly affected. The knee joint is the most common to manifest radiographic abnormalities, including synovitis, effusion, soft tissue swelling, and osteopenia.[114,115]

Vasculitis-Associated Effusions

Henoch-Schönlein Purpura

Henoch-Schönlein purpura is an acute leukocytoclastic vasculitis characterized by a palpable and purpuric rash usually involving the buttocks and legs. Arthritis occurs in 82% of children with HSP, and the knee joint is the second most commonly affected, causing arthritis in 61% of arthritis-affected children.[116] Although the rash is the means to diagnosis, the arthritis can precede the rash in as many as 25% of patients with HSP.[116–118] The arthritis is self-limited and responds to NSAIDs and joint rest. High-dose corticosteroids have been used with mixed results. Up to one-third of patients may develop a recurrence of symptoms.

Other vasculitides such as Wegener granulomatosis have associated joint symptoms including peripheral large joint monoarthritis. Although an uncommon pediatric vasculitis, as many as two-thirds of patients with Wegener granulomatosis may have musculoskeletal symptoms.[119] In addition to the vasculitides, vitamin C deficiency may cause vascular fragility, thus leading to hemarthrosis and subperiosteal bleeding.[120]

Hematologic Effusions

Bleeding disorders should be considered in acute hemarthrosis even with only minor trauma to the joint. Hemophilia A (factor VIII deficiency) is the most common bleeding disorder causing hemarthrosis, typically presenting before the age of 3 years. The knee is the most commonly affected joint followed by the elbow, ankle, hip, and shoulder. One joint is usually affected although multiple joints or bilateral involvement can occur. The acutely affected joint is painful and warm, often mimicking a septic joint. Therapy is aimed at correcting the factor deficiency (to a goal of >50% factor level) rather than aspiration for joint relief. Untreated hemorrhage or repeated hemorrhage can lead to intra-articular damage and osteoarthritis.

Other hematologic causes of hemarthrosis include coagulopathy secondary to systemic disease and other hemoglobinopathies. Coagulopathy-induced hemarthrosis can occur secondary to extensive liver disease, sepsis, or from exogenous heparin or warfarin administration. Hemoglobinopathies such as sickle cell disease can develop noninflammatory synovial effusions with crises, often with synovial leukocyte counts less than 1000/μL.[121] Recurrent hemarthroses, regardless of cause, may require synovectomy to prevent long-term intra-articular damage.

Malignant Effusions

Childhood neoplasms can present with a variety of symptoms, often before any radiographic or hematologic abnormalities. Neoplasms capable of presenting with knee effusions include leukemia, lymphoma, Ewing sarcoma, osteosarcoma, synovial sarcoma, and metastatic disease.

Leukemia is the most common malignancy of childhood. Monoarticular arthritis can be the only presenting feature in a subset of patients, which can classically be mistaken for JIA and other inflammatory diseases involving the joints.[122] Misdiagnosis, especially if therapy with corticosteroids is started, can lead to a delay in treatment and reduced efficacy of chemotherapy.[123] In a series of 414 children with a diagnosis of leukemia, bone pain was the most common musculoskeletal symptom (23.6%); however, arthralgia (22.4%) and arthritis (13.8%) were frequent initial complaints, with the knee joint being the most commonly affected.

Signs of leukemia such as fever, elevated ESR, anemia, arthritis, and leukocytosis can be nonspecific because they are frequently seen in systemic inflammatory diseases such as SLE, IBD, and systemic JIA. Other signs of malignancy may include thrombocytopenia, lymphadenopathy, hepatosplenomegaly, neutropenia, and blast cells on the peripheral smear. Peripheral white blood cell counts can be normal, and peripheral blast cells may be absent at the onset of leukemic disease.[122–124] If a patient has abnormal blood counts or if the diagnosis is in question, the patient should have a bone marrow aspiration to definitively evaluate for malignancy before starting empiric steroid therapy.

Osteosarcoma and Ewing sarcoma are the primary bone tumors of the lower extremity and can present with knee pain but

infrequently have concomitant joint disease from local invasion.[125] Each of these bone tumors have characteristic radiographic appearances, easily differentiating a primary bone problem from a disease localizing to the joint. Primary malignancies of the joint include synoviosarcoma and chondrosarcoma. Metastatic disease to the joint can occur most frequently from neuroblastoma, soft tissue sarcomas, and primary bone tumors.

Other Causes of Joint Effusions

Benign Tumors

Rarely, synovial hemangiomas and pigmented villonodular synovitis can cause a hemarthrosis. Pigmented villonodular synovitis is characterized by the proliferation of the synovial membrane of joints, tendon sheaths, and bursae. Pigmented villonodular synovitis is more locally aggressive and classically features darkly pigmented synovial fluid from hemosiderin deposits. Synovial hemangiomas are mostly intra-articular cavernous vascular tumors, enveloped in a synovial membrane. Patients with synovial hemangiomas usually develop symptoms in childhood, often before age 10 and before age 16 in approximately 75% of cases.[126] Both of these tumors can be locally invasive but do not metastasize, and both most commonly involve the knee joint. Synovial hemangiomas and pigmented villonodular synovitis often present with recurrent nontraumatic hemarthrosis of the suprapatellar pouch, occasionally with a palpable mass.[127–129] Results of knee radiographs are usually normal. Magnetic resonance imaging can be diagnostic, but a synovial biopsy is often required.

Infiltrative Disease

Crystal-induced arthritis caused by gout and pseudogout are a frequent cause of monoarthritis in adults but are quite rare in pediatrics. A history of diuretic use and renal stones often accompanies gouty arthritis. Microscopic examination of joint aspirate reveals negative birefringent rods in gout and positive birefringent rectangles or rhomboids in the calcium pyrophosphate crystals of pseudogout.

Drug Reaction

Serum sickness–like reaction is a systemic hypersensitivity reaction usually secondary to drug reaction that can present with fevers, lymphadenopathy, rash, proteinuria, and joint symptoms. In a study of 283 patients with acute arthritis, 15 cases were attributed to a serum sickness–like reaction, 12 of which had circulating immune complexes and 3 of which with immunoglobulin E antibodies specific to penicillin.[130] Generally, arthralgias are more common than arthritis with effusions, although both are possible, and large joints such as the knees are most commonly affected. Serum sickness is a self-limited illness with symptoms often resolving 1 to 3 weeks after the exposure. Penicillins and sulfa-containing drugs are most frequently implicated.

Familial Mediterranean Fever

Also called recurrent polyserositis, familial Mediterranean fever is a disease of recurrent episodes of peritonitis, pleuritis, and arthritis associated with fevers. It occurs mostly in Sephardic Jews along with people of Armenian, Turkish, and Arabic descent caused by a mutation in the *MEFV* gene. Protracted monoarthritis can persist for many weeks to many months.[131] Cases of chronic massive knee effusions in children with familial Mediterranean fever have been reported.[132]

SUMMARY

The approach to the pediatric knee effusion should be a thorough inquiry incorporating a broad differential diagnosis encompassing both acute and chronic illnesses as well as isolated and systemic diseases. Although the athlete with acute trauma is the most common cause of knee effusion in pediatrics, a comprehensive approach should discern between the many other nontraumatic hemarthroses and inflammatory arthritides.

REFERENCES

1. Willis RB. Sports medicine in the growing child. In: Morrissy RTW, Stuart L, eds. *Lovell & Winter's Pediatric Orthopedics*. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
2. Torg JS, Conrad W, Kalen V. Clinical diagnosis of anterior cruciate ligament instability in the athlete. *Am J Sports Med*. 1976;4:84–93.
3. Jackson JL, O'Malley PG, Kroenke K. Evaluation of acute knee pain in primary care. *Ann Intern Med*. 2003;139:575.
4. Hall FM. Radiographic diagnosis and accuracy in knee joint effusions. *Radiology.* 1975;115:49–54.
5. Stiell IG, Greenberg GH, Wells GA, et al. Derivation of a decision rule for the use of radiography in acute knee injuries. *Ann Emerg Med*. 1995;16:405.
6. Stiell IG, Greenberg GH, Wells GA, et al. Prospective validation of a decision rule for the use of radiography in acute knee injuries. *JAMA*. 1996;275:611.
7. Bachman LM, Haberzeth S, Steurer J, et al. The accuracy of the Ottawa Knee Rule to rule-out knee fractures: a systematic review. *Ann Intern Med*. 2004;140:121.
8. Emparanza JI, Aginaga JR. Validation of the Ottawa Knee Rules. *Ann Emerg Med*. 2001;38(4):364–368.
9. Bulloch B, Neto G, Plint A, et al. Validation of the Ottawa Knee Rules in children: a multicenter study. *Ann Emerg Med*. 2003;42:48.
10. Matelic TM, Aronsson DD, Boyd DW Jr, et al. Acute hemarthrosis of the knee in children. *Am J Sports Med*. 1995;23(6):668–671.
11. Fagan DJ, Davies S. The clinical indications for plain radiography in acute knee trauma. *Injury.* 2000;31:723.
12. Gylys-Morin VM. MR imaging of pediatric musculoskeletal inflammatory and infectious disorders. *Magn Reson Imaging Clin N Am*. 1998;6(3):537–559.
13. Johnson K, Gardner-Medwin J. Childhood arthritis: classification and radiology. *Clin Radiol*. 2002;57(1):47–58.
14. Buchmann RF, Jaramillo D. Imaging of articular disorders in children. *Radiol Clin North Am*. 2004;42:151–168.
15. Ostergaard M, Stoltenberg M, Lovgreen-Nielsen P, et al. Magnetic resonance imaging-determined synovial membrane and joint effusion volumes in rheumatoid arthritis and osteoarthritis: comparison with the macroscopic and microscopic appearance of the synovium. *Arthritis Rheum*. 1997;40(10):1856–1867.
16. El-Miedany YM, Housny IH, Mansour HM, et al. Ultrasound versus MRI in the evaluation of juvenile idiopathic arthritis of the knee. *Joint Bone Spine*. 2001;68(3):222–230.
17. Munshi M, Davidson M, MacDonald PB, et al. The efficacy of magnetic resonance imaging in acute knee injuries. *Clin J Sports Med*. 2000;10(1):34–39.
18. Bachmann G, Jürgensen I, Leiers S, et al. MRI in hemarthrosis of the knee joint: assessment of cause of bleeding, injury pattern and therapy. *Rofo*. 1996;165(3):270–275.
19. Oei EH, Nikken JJ, Verstijnen AC. MR imaging of the menisci and cruciate ligaments:. *Radiology.* 2003;226(3):837–848.
20. Lundberg M, Odensten M, Thuomas KA, et al. The diagnostic

validity of magnetic resonance imaging in acute knee injuries with hemarthrosis. A single-blinded evaluation in 69 patients using high-field MRI before arthroscopy. *Int J Sports Med*. 1996;17(3): 218–222.

21. Hauzeur J, Mathy L, DeMaertekaer V. Comparison between clinical evaluation and ultrasonography in detecting hydrathrosis of the knee. *J Rheumatol*. 1999;26:2681–2683.
22. Delaunoy I, Feipel V, Appelboom T, et al. Sonography detection threshold for knee effusion. *Clin Rheumatol*. 2003;22:391–392.
23. Hardaker WT, Garrett WE, Bassett FH. Evaluation of acute traumatic hemarthrosis of the knee joint. *South Med J*. 1990;83(6):640–644.
24. Bomberg BC, McGinty JB. Acute hemarthrosis of the knee: indications for diagnostic arthroscopy. *Arthroscopy*. 1990;6(3):221–225.
25. Maffulli N, Binfield PM, King JB, et al. Acute haemarthrosis of the knee in athletes. *J Bone Joint Surg [Br]*. 1993;75-B(6):945–949.
26. DeHaven KE. Diagnosis of acute knee injuries with hemarthrosis. *Am J Sports Med*. 1980;8(1):9–14.
27. Bergstrom R, Gillquist J, Lysholm J, et al. Arthroscopy of the knee in children. *J Pediatr Orthop*. 1984;4:542–545.
28. Eiskjaer S, Larsen ST, Schmidt MB. The significance of hemarthrosis of the knee in children. *Arch Orthop Trauma Surg*. 1988;107:96–98.
29. Stanitski CL, Harvell JC, Fu F. Observations on acute knee hemarthrosis in children and adolescents. *J Pediatr Orthop*. 1993;13: 506–510.
30. Hughes G, Watkins J. A risk-factor model for anterior cruciate ligament injury. *Sports Med*. 2006;36(5):411–428.
31. Medvecky MJ, Bosco J, Sherman OH. Gender disparity of anterior cruciate ligament injury. Etiological theories in the female athlete. *Bull Hosp Jt Dis*. 2006;59(4):217–226.
32. McNair PJ, Marshall RN, Matheson JA. Important features associated with acute anterior cruciate ligament injury. *N Z Med J*. 1990;103: 537–539.
33. Hall FM. Radiographic features of anterior cruciate ligament tear. *Radiology*. 2002;222(2):576.
34. Fanelli GC. Posterior cruciate ligament injuries in trauma patients. *Arthroscopy*. 1993;9(3):291–294.
35. Lu KH, Hsiao YM, Lin ZI. Arthroscopy for acute knee haemarthrosis in road traffic accident victims. *Injury*. 1996;27(5):341–343.
36. Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am*. 1979;52(8):1677–1684.
37. Roberts JM, Lovell WW. Fractures of the intercondylar eminence of tibia. *J Bone Joint Surg*. 1970;52A:827.
38. Willis RB, Blokker C, Stoll TM, et al. Long-term follow-up of anterior tibial eminence fractures. *J Pediatr Orthop*. 1993;13:361.
39. Broströaum A. Fracture of the patella: a study of 422 patellar fractures. *Acta Orthop Scand Suppl*. 1972;143:1–80.
40. Harilainen A, Myllynen P, Antilla H, et al. The significance of arthroscopy and examination under anesthesia in the diagnosis of fresh injury haemarthrosis of the knee joint. *Injury*. 1988;19:21–24.
41. LaBella CR. Common acute sports-related lower extremity injuries in children and adolescents. *Clin Pediatr Emerg Med*. 2007;8:31–42.
42. Cofield RH, Bryan RS. Acute dislocation of the patella: results of conservative treatment. *J Trauma*. 1977;17:526–531.
43. Stefancin JJ, Parker RD. First-time traumatic patellar dislocation. *Clin Orthop*. 2007;455:93–101.
44. Toupin JM, Lechevallier J. Osteochondral fractures of the external femoral condyle after traumatic patellar dislocation during physical exercise in children [Article in French]. *Rev Chir Orthop Reparatrice Appar Mot*. 1997;83(6):540–550.
45. Nomura E, Inoue M, Kurimura M. Chondral and osteochondral injuries associated with acute patellar dislocation. *Arthroscopy*. 2003;19: 717–721.
46. Stanitski CL, Paletta GA. Articular cartilage injury with acute patellar dislocation in adolescents: arthroscopic and radiographic correlation. *Am J Sports Med*. 1998;26:52–55.
47. Nietosvaara Y, Aalto K, Kallio PE. Acute patellar dislocation in children: incidence and associated osteochondral fractures. *J Pediatr Orthop*. 1994;14:513–515.
48. Stanitski CL. Correlation of arthroscopic and clinical examinations with magnetic resonance imaging findings of injured knees in children and adolescents. *Am J Sports Med*. 1988;26:2–6.
49. Christiansen P, Frederiksen B, Glazowski J, et al. Epidemiologic, bacteriologic, and long-term follow-up data of children with acute hematogenous osteomyelitis and septic arthritis: a ten-year review. *J Pediatr Orthop*. 1999;8:302–305.
50. Fleisher GR. Infectious disease emergencies. In: Fleisher GR, Ludwig S, eds. *Textbook of Pediatric Emergency Medicine*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2000:768–769.
51. Guidelines for the initial evaluation of the adult patient with acute musculoskeletal symptoms: American College of Rheumatology Ad Hoc Committee on Clinical Guidelines; 1996.
52. McGillicuddy DC, Shah KH, Friedberg RP, et al. How sensitive is the synovial fluid white blood cell count in diagnosing septic arthritis? *Am J Emerg Med*. 2007;25(7):749–752.
53. Nelson JD. The bacterial etiology and antibiotic management of septic arthritis in infants and children. *Clin Pediatr*. 1972;50(3):437–440.
54. Krey PR, Bailen DA. Synovial fluid leukocytosis. A study of extremes. *Am J Med*. 1979;67(3):436–442.
55. Margaretten ME, Kohlwes J, Moore D, et al. Does this adult patient have septic arthritis? *JAMA*. 2007;297:1478.
56. Eder L, Zisman D, Rozenbaum M, et al. Clinical features and aetiology of septic arthritis in northern Israel. *Rheumatology*. 2005;44:1559–1563.
57. Rice PA. Gonococcal arthritis (disseminated gonococcal infection). *Infect Dis Clin North Am*. 2005;19:853–861.
58. Wise CM, Morris CR, Wasilauskas BL, et al. Gonococcal arthritis in an era of increasing penicillin resistance. Presentations and outcomes in 41 recent cases (1985–1991). *Arch Intern Med*. 1994;154(23): 2690–2695.
59. Centers for Disease Control and Prevention. Lyme disease—United States. *JAMA*. 2000;1259–1260.
60. Steere AC, Bartenhagen NH, Craft JE, et al. The early clinical manifestations of Lyme disease. *Ann Intern Med*. 1983;99(1):76–82.
61. Steere AC, Sikand VK. The presenting manifestations of Lyme disease and the outcomes of treatment. *N Engl J Med*. 2003;348(24):2472–2473.
62. Gerber MA, Shapiro ED, Burke GS, et al. Lyme disease in children in southeastern Connecticut. *N Engl J Med*. 1996;335(17):1270–1274.
63. Dattwyler RJ, Luft BJ, Kunkel MJ, et al. Ceftriaxone compared with doxycycline for the treatment of acute disseminated Lyme disease. *N Engl J Med*. 1997;337(5):289–294.
64. Steere AC, Schoen RT, Taylor E. The clinical evolution of Lyme arthritis. *Ann Intern Med*. 1987;107:725–731.
65. Hu L. Lyme arthritis. *Infect Dis Clin North Am*. 2005;19:947–961.
66. Willis AA, Widmann RF, Flynn JM, et al. Lyme arthritis presenting as acute septic arthritis in children. *J Pediatr Orthop*. 2003;23: 114–118.
67. Naides SJ. Viral arthritis. In: Kelley WN, Harris ED, Budd RC, Genovese MC, eds. *Textbook of Rheumatology*. Philadelphia, PA: Elsevier Saunders; 2005.
68. Friedman HM, Pincus T, Gibilisco P. Acute monoarticular arthritis caused by herpes simplex virus and cytomegalovirus. *Am J Med*. 1980;69:241.

69. Fierman AH. Varicella-associated arthritis occurring before the exanthem. Case report and literature review. *Clin Pediatr*. 1990;29(3):188–190.
70. Sigal LH, Steere AC, Niederman JC. Symmetric polyarthritis associated with heterophile-negative infectious mononucleosis. *Arthritis Rheum*. 1983;26:553.
71. Ligon BL. Reemergence of an unusual disease: the Chikungunya epidemic. *Semin Pediatr Infect Dis*. 2006;17:99–104.
72. Halsted SB. Chikungunya. In: Feigin RD, Cherry JD, Demmler GJ, Kaplan S, eds. *Textbook of Pediatric Infectious Diseases*. 5th ed. Philadelphia, PA: Saunders; 2004:2178–2184.
73. Malaviya AN, Kotwal PP. Arthritis associated with tuberculosis. *Best Pract Res Clin Rheumatol*. 2003;17(2):319–343.
74. Ntsiba H, Bazebissa R, Lamini N, et al. Knee septic arthritis: 100 cases report in intertropical zone [Article in French]. *Bull Soc Pathol Exot*. 2004;97(4):244–246.
75. Sawlani V, Chandra T, Mishra RN, et al. MRI features of tuberculosis of peripheral joints. *Clin Radiol*. 2003;58:755–762.
76. Bayer AS, Guze LB. Funagl arthritis II coccidioidal synovitis: clinical, diagnostic, therapeutic, and prognostic considerations. *Semin Arthritis Rheum*. 1979;8(3):200–211.
77. Bayer AS, Choi C, Tillman DB. Fungal arthritis V. Cryptococcal and histoplasmal arthritis. *Arthritis Rheum*. 1980;9(3):218–227.
78. Saiz P, Gitelis S, Virkus W, et al. Blastomycosis of long bones. *Clin Orthop Relat Res*. 2004;421:255–259.
79. Roberts-Thomson PJ, Rischmueller M, Kwiatek RA, et al. Rheumatic manifestations of infective endocarditis. *Rheumatol Int*. 1992;12(2): 61–63.
80. Gonzalez-Juanatey C, Gonzalez-Gay MA, Llorca J, et al. Rheumatic manifestations of infective endocarditis in non-addicts. A 12-year study. *Medicine (Baltimore)*. 2001;80(1):9–19.
81. Hannu T, Inman R, Granfors K, et al. Reactive arthritis or post-infectious arthritis? *Best Pract Res Clin Rheumatol*. 2006;20(3): 419–433.
82. Kanakoudi-Tsakalidou F, Pardalos G, Pratsidou-Gertsi P, et al. Persistent or severe course of reactive arthritis following *Salmonella enteritidis* infection: a prospective study of nine cases. *Scand J Rheumatol*. 1998;27:431–434.
83. Cron RQ, Gordon PV. Reactive arthritis to *Clostridium difficile* in a child. *West J Med*. 1997;166:419–421.
84. LeBlanc CM, Birdi N. *Giardia lamblia* associated arthritis in a nineteen-month-old child. *J Rheumatol*. 1999;26:2066–2067.
85. Hannu T, Mattila L, Siitonen A, et al. Reactive arthritis attributable to *Shigella* infection: a clinical and epidemiological nationwide study. *Ann Rheum Dis*. 2005;64(4):594–598.
86. Hannu T, Mattila L, Rautelin H, et al. *Campylobacter*-triggered reactive arthritis: a population-based study. *Rheumatology (Oxford)*. 2002;41(3):312–318.
87. Kousa M. Evidence of chlamydial involvement in the development of arthritis. *Scand J Infect Dis Suppl*. 1983;32:116–121.
88. Hill GJ, Lillicrap MS. Arthritis associated with enteric infection. *Best Pract Res Clin Rheumatol*. 2003;17(2):219–239.
89. Simonet ML. Enterobacteria in reactive arthritis: *Yersinia*, *Shigella*, and *Salmonella*. *Rev Rhum Engl Ed*. 1999;66(suppl 1):14S–18S.
90. Barth WF, Segal K. Reactive arthritis (Reiter's syndrome). *Am Fam Physician*. 1999;60:499–507.
91. Ferrieri P. Proceedings of the Jones Criteria Workshop. *Circulation*. 2002;106(19):2521–2523.
92. Carapetis JR, Currie BJ. Rheumatic fever in a high incidence population: the importance of monoarthritis and low grade fever. *Arch Dis Child*. 2001;85(3):223–227.
93. Hashkes PJ, Tauber T, Somekh E, et al. Naproxen as an alternative to aspirin for the treatment of arthritis of rheumatic fever: a randomized trial. *J Pediatr*. 2003;143(3):399–401.
94. Deighton C. beta Haemolytic streptococci and reactive arthritis in adults. *Ann Rheum Dis*. 1993;52:475–482.
95. Jansen TL, Janssen M, deLong AJ, et al. Post-streptococcal reactive arthritis: a clinical and serological description, revealing its distinction from acute rheumatic fever. *J Int Med*. 1999;245: 261–267.
96. Ahmed S, Ayoub EM. Poststreptococcal reactive arthritis. *Pediatr Infect Dis J*. 2001;20:1081–1082.
97. Shulman ST, Ayoub EM. Poststreptococcal reactive arthritis. *Curr Opin Rheumatol*. 2002;14:562–565.
98. Mackie SL, Keat A. Poststreptococcal reactive arthritis: what is it and how do we know? *Rheumatology*. 2004;43:949–954.
99. Ahmed S, Ayoub EM, Scornik JC, et al. Poststreptococcal reactive arthritis: clinical characteristics and association with HLA-DR alleles. *Arthritis Rheum*. 1998;41(6):1096–1102.
100. Toll ML, Lio P, Sundel RP, et al. Comparison of Vancouver and International League of Associations for rheumatology classification criteria for juvenile psoriatric arthritis. *Arthritis Rheum*. 2007;59(1): 51–58.
101. Roberton DM, Cabral DA, Malleson PN, et al. Juvenile psoriatric arthritis: follow up and evaluation of diagnostic criteria. *J Rheumatol*. 1996;23(1):166–170.
102. Southwood TR, Petty RE, Malleson PN, et al. Psoriatic arthritis in children. *Arthritis Rheum*. 1989;32(8):1007–1013.
103. DeVos M. Joint involvement in inflammatory bowel disease [review]. *Aliment Pharmacol Ther*. 2004;(suppl 4):36–42.
104. Weiner SR, Clarke J, Taggart N, et al. Rheumatic manifestations of inflammatory bowel disease. *Semin Arthritis Rheum*. 1991;20:353.
105. Lubrano E, Ciacci C, Ames PRJ, et al. The arthritis of coeliac disease: prevalence and pattern in 200 adult patients. *Br J Rheum*. 1996;35: 1314–1318.
106. Lepore L, Martelossi S, Pennesi M, et al. Prevalence of celiac disease in patients with juvenile chronic arthritis. *J Pediatr*. 1996;129: 311–313.
107. Stagi S, Giani T, Simonini G, et al. Thyroid function, autoimmune thyroiditis and coeliac disease in juvenile idiopathic arthritis. *Rheumatology*. 2005;44(4):517–520.
108. Klein-Gitelman M, Reiff A, Silverman ED. Systemic lupus erythematosus in childhood. *Rheum Dis Clin North Am*. 2002;28: 561–577.
109. Ragsdale CG, Petty RE, Cassidy JT, et al. The clinical progression of apparent juvenile rheumatoid arthritis to systemic lupus erythematosus. *J Rheumatol*. 1980;7:50–55.
110. Saulsbury FT, Kesler RW, Kennaugh JM, et al. Overlap syndrome of juvenile rheumatoid arthritis and systemic lupus erythematosus. *J Rheumatol*. 1982;9:610–612.
111. Kone-Paut I, Yurdakul S, Bahabri S, et al. Clinical features of Behcet's disease in children: an international collaborative study of 86 cases. *J Pediatr*. 1998;132(4):721–725.
112. Kari JA, Shah V, Dillon MJ. Behcet's disease in UK children: clinical features and treatment including thalidomide. *Rheumatology*. 2001; 40:933–938.
113. Saurenmann RK, Levin AV, Feldman BM, et al. Prevalence, risk factors, and outcome of uveitis in juvenile idiopathic arthritis: a long-term follow up study. *Arthritis Rheum*. 2007;56(2):647–657.
114. Martel W, Holt J, Cassidy J. Roentgenologic manifestations of juvenile rheumatoid arthritis. *AJR Am J Roentgenol*. 1962;88: 400–423.
115. Cohen PA, Job-Deslandre CH, Lalande G, et al. Overview of the

radiology of juvenile idiopathic arthritis (JIA). *Eur J Radiol*. 2000;33(2):94–101.

116. Saulsbury F. Henoch-Schönlein purpura in children. Report of 100 patients and review of the literature. *Medicine (Baltimore)*. 1999;78(6): 395–409.
117. Allen DM, Diamond LK, Howell DA. Anaphylactoid purpura in children (Schonlein-Henoch syndrome). *Am J Dis Child*. 1960;99:833–854.
118. Robson WL, Leung AK. Henoch-Schonlein purpura. *Adv Pediatr*. 1994;41:163–194.
119. Noritake DT, Weiner SR, Bassett LW, et al. Rheumatic manifestations of Wegener's granulomatosis. *J Rheumatol*. 1987;14(5):949–951.
120. Mowad CM, Howe KL, Guzzo CA. Unexplained hemothorax, hemarthrosis, and palpable purpura. *Hosp Pract*. 1995;30:55.
121. Schumacher HR. Arthritis associated with sickle cell disease and other hemoglobinopathies. In: Kelley WN, Harris ED, Ruddy S, Sledge CB, eds. *Textbook of Rheumatology*. Philadelphia, PA: WB Saunders; 1993.
122. Ostrov BE, Goldsmith DP, Athreya BH. Differentiation of systemic juvenile rheumatoid arthritis from acute leukemia near the onset of disease. *J Pediatr*. 1993;122:595–598.
123. Revesa T, Kardos G, Kajtar P, et al. The adverse effect of prolonged prednisolone pretreatment in children with acute lymphoblastic leukaemia. *Cancer*. 1985;55:637–640.
124. Trapani S, Grisolia F, Simonini G, et al. Incidence of occult cancer in children presenting with musculoskeletal symptoms: a 10-year survey in a pediatric rheumatology unit. *Semin Arthritis Rheum*. 2000;29(6):348–359.
125. Halla JT, Hirsch V. Monoarthritis as the presenting manifestation of localized Ewing's sarcoma in an older patient. *J Rheumatol*. 1987;14(3):628–631.
126. Yercan HS, Okeu G, Erkan S. Synovial hemangiohamartomas of the knee joint. *Arch Orthop Trauma Surg*. 2007;127:281–285.
127. Carroll MB, Higgs JB. Synovial haemangioma presenting as a recurrent monoarticular haemarthrosis. *Arch Dis Child*. 2007;92:623–624.
128. Wirth T, Rauch G, Ruschoff J, et al. Synovial heamangioma of the knee joint. *Int Orthop*. 1992;16:130.
129. Frassica FJ, Bhimani MA, McCarthy EF, et al. Pigmented villonodular synovitis of the hip and knee. *Am Fam Physician*. 1999;60:1404.
130. Kunnamo I, Kallio P, Pelkonen P, et al. Serum-sickness–like disease is a common cause of acute arthritis in children. *Acta Paediatrica*. 1986;75(6):964–969.
131. Sohar E, Gafni J, Prass M, et al. Familial Mediterranean fever: a survey of 470 cases and review of the literature. *Am J Med*. 1967;43: 227–253.
132. Salai M, Zemmer D, Segal E, et al. Chronic massive knee effusion in familial Mediterranean fever. *Semin Arthritis Rheum*. 1997; 27(3):169–172.

CME EXAM
INSTRUCTIONS FOR OBTAINING *AMA PRA CATEGORY 1 CREDITS*™

Pediatric Emergency Care includes CME-certified content that is designed to meet the educational needs of its readers. An annual total of 12 *AMA PRA Category 1 Credit*™ is available through the twelve 2009 issues of the Pediatric Emergency Care. This activity is available for credit through December 15, 2009.

Credit Designation Statement

Lippincott Continuing Medical Education Institute designates this educational activity for a maximum of one (1) *AMA PRA Category 1 Credit*™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Accreditation Statement

Lippincott Continuing Medical Education Institute is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education or physicians.

To earn CME credit, you must read the article in *Pediatric Emergency Care* and complete the quiz, answering at least 80 percent of the questions correctly. Mail the completed quiz to the Lippincott Continuing Medical Education Institute, 530 Walnut Street, 8th Floor East, Philadelphia, PA 19106; fax: 267-757-0651. Only the first entry will be considered for credit, and must be postmarked by the expiration date. There is no cost for CME credit or quiz processing. Answer sheets will be graded and certificates will be mailed to each participant within 6 to 8 weeks of participation.

CME EXAMINATION
NOVEMBER 2009

Please mark your answers on the ANSWER SHEET.

Approach to Knee Effusions, *Mathison and Teach.*

1. Which of the following is *not* a physical examination maneuver used to assess for meniscal injury?
   a. The Bounce test
   b. The McMurray test
   c. The Balloon test
   d. Apley's (compression) test

2. Which of the following is *not* an Ottawa criterion for obtaining a knee radiograph following a traumatic injury?
   a. Isolated patellar bone tenderness
   b. Inability to weight bear for four steps in the ED
   c. Presence of a knee effusion
   d. Pain at the proximal fibular head
   e. Inability to weight bear for four steps immediately after the injury

3. Which of the following characteristics is more consistent with the arthritis of acute rheumatic fever (ARF) as opposed to a post-streptococcal reactive arthritis (PSRA)?
   a. Latency period of 3–16 days
   b. Monoarticular arthritis
   c. Poorly responsive to salicylates
   d. Migratory polyarthritis

4. A 10-year-old boy with no significant medical history presents to the ED with fever and knee pain. Physical examination shows a warm and slightly erythematous left knee with a palpable effusion and flexion limited by pain. You suspect an acute bacterial septic joint. Arthrocentesis is performed. Which of the following synovial results would most argue against your suspected diagnosis?
   a. Synovial leukocyte count >80,000
   b. Negative synovial gram stain
   c. Synovial protein ≥3 g/dL
   d. Synovial glucose ≥ serum glucose

5. Which of the following statements is *not* a characteristic of a Lyme monoarthritis of the knee?
   a. It can occur before or without erythema migrans
   b. Early antibiotic therapy shortens the duration of the arthritis
   c. The preferred screening test is a serologic enzyme immunoassay (EIA) lyme antibody panel
   d. The synovial leukocyte count is always less than 40,000 cells/mm^3

Novel Influenza A(H1N1): Clinical Presentations, Diagnosis, and Management, *Jain and Goldman.*

6. Which of the following children should *not* receive antiviral treatment of suspected novel influenza A(H1N1) infection?
   a. A 12-year-old boy with severe cerebral palsy
   b. An 18-month-old girl with mild uncomplicated illness
   c. A 10-year-old boy with asthma
   d. A 4-year-old girl with mild uncomplicated illness
   e. A 7-year-old girl with an influenza-like illness (ILI) complicated by radiologic signs of pneumonia

7. Which of the following statements is TRUE regarding testing for novel influenza A(H1N1)?
   a. Rapid influenza diagnostic tests are highly sensitive for detecting the H1N1 virus
   b. Immunocompromised children should only be tested for H1N1 if they have an illness that meets the case definition for ILI
   c. Either reverse transcription-polymerase chain reaction or viral culture may be used to definitively diagnose H1N1
   d. Any child who presents with an ILI should undergo testing for H1N1
   e. Confirmatory diagnostic testing is mandatory before initiating antiviral treatment of H1N1

8. Which of the following statements is FALSE regarding the treatment of novel influenza A(H1N1)?
   a. Oseltamivir is currently authorized for use in children younger than 1 year
   b. Most circulating H1N1 strains are susceptible to M2 inhibitors
   c. Antiviral medications are most effective if given 48 hours of illness onset
   d. H1N1 resistance to oseltamivir has been detected primarily in individuals who were immunocompromised or who were receiving antiviral prophylaxis
   e. Most children affected by H1N1 require only supportive treatment
9. A daycare worker is on vacation when she develops an ILI 3 days into her trip. She returns to work after she has been afebrile for 48 hours. Which of the following statements is *true*?
   a. All of the children in the daycare should receive antiviral prophylaxis because of exposure to H1N1 through the daycare worker
   b. Outbreaks of H1N1 have been frequently reported in schools, daycares, and camps
   c. The daycare worker is no longer in the infectious period of her illness when she returns to work
   d. The daycare worker's 16-month-old niece (whom she was visiting when she became ill) should receive antiviral prophylaxis for 5 days
   e. Zanamivir may not be used for prophylaxis in children under the age of 7 years
10. Which of the following measures is *not* recommended to limit the spread of illness caused by novel influenza A(H1N1)?
   a. Frequent hand washing
   b. Vaccination of high-risk individuals
   c. Isolation of sick children in the emergency department
   d. Avoidance of individuals who are sick with an ILI
   e. Use of N95 masks by healthy individuals in the community

ANSWER SHEET FOR THE *PEDIATRIC EMERGENCY CARE* CME PROGRAM EXAM November 2009

Please answer the questions on page 787 by filling in the appropriate circles on the answer sheet below. Please mark the one best answer and fill in the circle until the letter is no longer visible. To process your exam, you must also provide the following information:

Name (please print): _____

Street Address _____

City/State/Zip _____

Daytime Phone _____

Specialty _____

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

Your evaluation will help us assess whether this CME activity is congruent with LCMEI's CME mission statement and will assist us in future planning of CME activities. Please respond to the following questions:

1. Did the content of this CME activity meet the stated learning objectives?
[ ] Yes [ ] No

2. On a scale of 1 to 5, with 5 being the highest, how do you rank the overall quality of this educational activity?
[ ] 5 [ ] 4 [ ] 3 [ ] 2 [ ] 1

3. Was the activity's format (ie, print, live, electronic, Internet, etc.) an appropriate educational method for conveying the activity's content?
[ ] Yes [ ] No

4. Did this CME activity increase your knowledge/competence in the activitys topic area? **If No, please explain why not.**
[ ] Yes [ ] No

_____

_____

5. As a result of participating in this CME activity, will you be changing your practice behavior in a manner that improves your patient care? **Please explain your answer.**

_____

_____

6. Did you perceive any evidence of bias for or against any commercial products? **If yes, please explain.**
[ ] Yes [ ] No

_____

_____

7. How long did it take you to complete this CME activity?
_____hour(s) _____minutes

8. Please state one or two topics that you would like to see addressed in future issues.

_____

_____

[ ] YES! I am interested in receiving future CME programs from Lippincott CME Institute! (Please place a check mark in the box)

Mail by January 15, 2010 to
Lippincott CME Institute
770 Township Line Road, Suite 300
Yardly, PA 19067

CME EXAM ANSWERS

Answers for the Pediatric Emergency Care CME Program Exam

Below you will find the answers to the examination covering the review article in the August 2009 issue. All participants whose examinations were received by September 15, 2009 and who achieved a score of 80% or greater will receive a certificate from Lippincott CME Institute.

EXAM ANSWERS

August 2009

1. b
2. d
3. a
4. c
5. d