CHAPTER 31

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Sports Medicine in the Growing Child

SURGICAL PROCEDURE INDEX

INTRODUCTION

Risks of Injury during Sports Participation. It is estimated that approximately 30 million children and youth between the ages of 6 and 21 years engage in sports programs that are held outside of school, and 7.2 million participate in high-school sports programs (1).

Parents want to know if the benefits of the sports activities warrant the risks involved, so an understanding of sports-specific risks is crucial to provide a comprehensive approach to address this concern.

With appropriate surveillance studies of each sport, specific risks and patterns of injury associated with different sports can be determined and compared. From this data, it may be possible to develop specific interventions designed to reduce the frequency of injuries.

Epidemiology of Athletic Injuries in Children. What are the facts? What do we know about sports injuries in children and youth?

First, there are sport-specific data that identify the risk of injury to participants for most sports (2–5). The most comprehensive statistics on children's recreational injuries are available from the United States Consumer Product Safety Commission (CPSC). The CPSC operates the National Electronic Injury Surveillance System (NEISS) whereby data are gathered from the emergency departments of 100 hospitals throughout the United States. These data are then used in conjunction with other models involving the relation between emergency room visits and the number of injuries treated outside hospital emergency rooms to arrive at an estimate of the number of injuries treated for each specific age group in hospital emergency rooms, doctor's offices, clinics, and ambulatory centers. The figures for children and youth in 2008 reveal that injuries occurred most frequently in football, followed by basketball, soccer, and baseball and that over 2 million children under age 18 required medical attention for injuries (2) (Table 31-1).

An additional source of sport-specific data for high-school athletes is available through the Center for Injury Research and Policy at Nationwide Children's Hospital in Columbus, Ohio. Research examined data submitted by athletic trainers at 100 high schools in the United States. Information was collected for nine sports including boy's football, boys' and girls' soccer, girls' volleyball, boys' and girls' basketball, boys' wrestling, boys' baseball, and girls' softball (3).

The most severe injuries occurred in football and wrestling followed by girls' basketball and girls' soccer. Severe injuries occurred in approximately 15% of all sports-related injuries and were defined as loss of participation in sports of at least 21 days. Injuries to girls occurred more frequently than boys. Anatomic sites of injury in order of frequency were the knee (30%), ankle (12.3%), and shoulder (10.9%). One in four (28.3%) severe injuries required surgery with over half being knee surgeries.

Injuries to the upper extremity occur more frequently in younger children, due to falls, whereas lower extremity injuries occur more frequently in older children and adolescents (1, 4).

TABLE 31-1 National Electronic Injury Surveillance System (NEISS) Data Highlights—2008

	Number of Injuries		
Sport	**AGES 0–4**	**AGES 5–14**	**AGES 15–24**
Basketball	1653	156,115	229,487
Football	1351	214,492	192,778
Soccer	1088	82,707	80,732
Hockey	219	18,555	29,786
Lacrosse, rugby, ball games	2108	37,891	32,905
Baseball	4539	109,202	80,879
Volleyball	52	16,785	23,761
Racquet sports	52	4623	6767
Track and Field	17	8655	9688

Estimated Number of Injuries: Because NEISS is a probability sample, each injury case has a statistical weight. These are national estimates of the number of persons treated in U.S. hospital emergency departments with consumer product-related injuries and are derived by summing the statistical weights for the appropriate injury cases. The data system allows for reporting of up to two products for each person's injury; so a person's injury may be counted in two product or sports groups.

Injury surveillance identifies specific risks for specific sports and may lead to injury prevention by mandatory changes in equipment. Face masks and helmets used in hockey, shin guards used in soccer, and helmets used in baseball are examples of equipment modification put into place after injury surveillance rates indicated the need for change.

Despite the injuries seen in a sports medicine clinic, the documented health benefits of sports activity which include weight management, increased strength, flexibility and endurance, as well as improved self-esteem outweigh the risks of significant injury. Parents should be advised of both the recognized benefits and the sport-specific risks, in order to make an informed decision regarding their child's participation.

Injury Prevention. Prevention strategies for sports-related injuries in both children and adults generally lag behind injury management strategies. As participation in recreational and scholastic sports increases, there is a desire to examine strategies for injury prevention to lower the risk of injury (5, 6).

Injury prevention strategies include a thorough preparticipation physical evaluation to identify medical problems such as asthma or diabetes mellitus that affect training or participation and previous significant injuries such as fractures and sprains that should be assessed before clearing the athlete to participate (7, 8).

As part of the preparticipation physical evaluation, an assessment of general health, physical fitness, strength, flexibility, and joint stability and alignment should be performed (9).

A certain level of fitness should be attained before preseason practice begins and is the responsibility of the coach, parent, and athlete. As well as general aerobic fitness, sport-specific conditioning is recommended to prevent sport-specific injuries. Athletes involved in throwing sports should work on strengthening and stretching exercises for the shoulder girdle and upper extremity (10). Controversy exists as to the benefit of stretching programs in the prevention of muscle–tendon strains or apophysitis. There are no studies that have proven the efficacy of stretching in reducing the incidence of injury, but most coaches, trainers, and sports medicine personnel continue to advocate their use (8).

Probably, the most important individual in ensuring injury prevention is the coach. The coach should be qualified in sport-specific methods of training, injury prevention, injury recognition, and proper rehabilitation of the injured athlete before return to participation. An understanding and knowledgeable coach can make a lasting impression on the athlete, especially at the youth level.

Strength training for specific sports is permissible without any concern for overuse injury or effect on growth, as long as the program is supervised and submaximal weights are employed.

Principles of Rehabilitation. Rehabilitation is a process in which a series of structured activities enable an athlete to return to normal activity or function.

Although the physician will make the diagnosis and assess the functional limitations of the injury, physical therapy is actively involved in the rehabilitation process to enable the athletes to resume their previous level of activity.

The physician should supervise the rehabilitation process and determine when joint functions, muscle strength, and sport-specific functions are restored (7).

For minimal injuries such as minor contusions and sprains, the need for supervised rehabilitation is questionable. However, for major joint injuries such as significant ligament sprains, fractures, and significant resistant overuse syndromes, physical therapy will usually aid the athlete to a speedier return to activity and may also prevent further or repetitive injuries (8).

The phases of rehabilitation include the initial period of acute care when the limb is put at relative rest, and pain and inflammation are controlled by ice, elevation, and compression (7–9). The next phase, or intermediate phase, is aimed at the resolution of pain and restoration of joint motion, flexibility, and strength.

Later care involves progressive strengthening, functional and sport-specific drills, as well as proprioceptive training.

Finally, a maintenance program to prevent further injury is instituted.

Various modalities aid in this process.

Physical Modalities

Cold. For acute injuries, ice should be applied to decrease pain and swelling, blood flow, and muscle spasm. It is the agent of choice for nearly all acute injuries and even overuse injuries.

Heat. Heat is employed less commonly; it reduces pain and spasm and increases blood flow and soft-tissue relaxation. It has a limited role in acute injuries or overuse syndromes when swelling and inflammation are present.

Therapeutic Exercises. Once swelling and muscle spasm subside, therapeutic exercise is initiated to improve joint range of motion and to stretch and strengthen muscles.

Joint mobilization is best accomplished by active mobilization in which the athlete moves the injured joint. Passive mobilization utilizes another individual, usually a therapist, to move the patient's contracted joint. This technique is often complicated by exacerbation of the injury, tearing or stretching of soft tissues, and hemorrhage and should only be done by an experienced therapist when active mobilization has failed (7–9).

Active-assist mobilization is a combination of the two methods and has limited indications in the young athlete.

Stretching Techniques. Stretching techniques are designed to restore flexibility after an injury. Static stretching employs techniques in which the involved or target muscle is stretched or maintained for approximately 20 seconds. It is safer than ballistic stretching in which sudden bounces or joint motions are permitted. Ballistic stretching can cause activation of the stretch reflex and cause muscle–tendinous strain and is not recommended after acute injuries.

Strengthening. Strengthening is an important part of the rehabilitation program and includes isometric, isotonic, isokinetic, concentric and eccentric, closed kinetic chain, and functional exercises.

Isometric. The muscle contracts without changing length. Isometric exercises are most important in the early phase of rehabilitation after injury because the injured joint or muscle is not moved. The exercises are simple to perform and do not require specialized equipment. To the patient's relief, isometric exercises are relatively painless.

Isotonic. Isotonic exercise involves the contraction of muscle against fixed resistance while the joint moves through its arc of motion. Examples are free weights and weight machines. Isotonic exercises are initiated after pain and swelling subside and joint motion is restored. Motor performance is superior following isotonic exercise compared to isometric exercise.

Isokinetic. Isokinetic exercises replicate the speed of muscle contraction during specific activities and are usually provided by specific and expensive therapeutic machines. The exercises are performed at a constant velocity.

Concentric and Eccentric. Concentric exercises involve the contraction of a muscle during exercise (e.g., biceps curl). Eccentric exercises involve lengthening of the muscle while opposing gravity (e.g., elbow extension with free weights after biceps curl). Significant increase of muscle strength occurs with eccentric exercise (7, 8). Eccentric conditioning is introduced during the latter stages of rehabilitation.

Closed or Open Chain Kinetic Exercises. Closed chain kinetic exercise fixes a body part while performing work (e.g., foot on floor while performing squats), whereas open chain kinetic exercises do not fix the body part (e.g., leg lift). Closed chain exercise improves agonist and antagonist muscle contraction (9). These exercises are performed in the later stages of rehabilitation because they may cause pain.

Functional Exercise. Functional exercises reproduce patterns of movement involved in a specific sport and involve the integration of several muscle groups working together. This is the final step in the rehabilitation process before the athlete returns to the sporting activity.

Therapeutic Electrical Modalities

Electrogalvanic Stimulation. Electrogalvanic stimulation causes small muscle contractions that may reduce swelling and muscle atrophy (7–9).

Transcutaneous Nerve Stimulation. Transcutaneous nerve stimulation employs electrical stimulation to block pain impulses from the site of injury or site of surgery (7–9).

There is no objective evidence of the benefits of these two modalities in the treatment of athletic injuries in the child (7–9). Most sports injuries in the skeletally immature are amenable to nonoperative management. Not all injuries require supervised rehabilitation, but it has been shown to lessen recovery time and decrease reinjury rates.

Performance-enhancing Substances.

Widespread publicity about performance-enhancing drugs and the perception of societal reward for exceptional athletic success are the major reasons why young athletes consider the use of these substances (10).

Anabolic steroids in particular are under increasing scrutiny by international athletic organizations as well as the press in an attempt to publicize their role in the performance of elite athletes.

If used in conjunction with a strength training program and proper diet, anabolic steroids have been shown to increase muscle size and strength; but there is little, if any, evidence that their use resulted in improved performance or increased aerobic capacity (10–13).

Reports of steroid use for the last 10 to 20 years indicate patterns of use in up to 10% to 15% of boys and up to 2% to 4% of girls among high-school students (13). Anabolic steroid use is determined by a complex set of factors that include potential beneficial effects of anabolic steroids, dissatisfaction with current body size and strength, a peer group involved in their use, and a tendency toward risk-taking behavior (10–13).

Anabolic steroids are available in oral and injectable forms. The oral form is metabolized in the liver and converted to testosterone. The injectable form is directly absorbed into the circulation and is therefore less hepatotoxic than the oral form (12).

Adverse Effects. The adverse effects of anabolic steroids are well known, significant, and affect virtually every organ system (11, 12).

For the skeletally immature athlete, premature epiphyseal closure has been documented with the intake of a single cycle of anabolic steroids (10, 12). In addition, strains and ruptures of the tendons have been noted in young individuals without any predisposing tendonitis (11, 12).

Effects on the hepatobiliary system include transient elevation of liver enzymes, blood-filled cysts in the liver which may rupture and cause fatal hemorrhage, and benign and malignant neoplasms (11).

Anabolic steroids cause an elevation of blood pressure (reversible) and an increase in total cholesterol with a reduction in high-density lipoproteins (12, 13). Prolonged use may lead to arteriosclerotic heart disease and cardiomyopathy (12, 13).

Men taking anabolic steroids may experience acne, male pattern baldness, priapism, impotence, gynecomastia, and testicular atrophy (10–13). Women may develop masculinization including hirsutism, deepening of the voice, and baldness (10–12).

Aggression, emotional instability, and even psychosis have been reported (11, 12).

To effect a change in behavior, stiff penalties and peer pressure to avoid cheating are probably necessary. Imparting proper and current medical knowledge without the use of scare tactics may help. Encouragement and availability of proper programs in strength training, conditioning, proper nutrition, and acquisition of sporting skills is probably the best deterrent.

Strength Training in the Pediatric Population. Weight training or strength training by children or growing adolescents is a controversial topic. The controversy exists because of the belief that weight training causes damage to the physes or joints and the perceived association of weight training with performance-enhancing drugs. Both are enough to cause parents to question the potential benefits of weight training against its perceived risks to their child.

A number of studies have shown that children and adolescents can increase strength up to 40% with a low risk of injury (14–16). In girls and prepubescent boys, it is postulated that a gain in strength occurs due to enhanced recruitment of motor units rather than muscular hypertrophy (14–16).

Specific injuries have been reported in association with weight lifting in children, including distal radius and ulnar fractures, distal radial epiphyseal fractures, patellofemoral pain, clavicular osteolysis, pelvic apophyseal fractures, and meniscal tears (15, 17–19). In adolescents, lumbosacral injuries such as disc herniation, spondylolysis, and spondylolisthesis are common injuries (20). Power lifting and Olympic-style weight lifting are *not* recommended for the skeletally immature (14). However, there are no deleterious consequences to a well-supervised program of weight training in the growing youth, provided the movements are done in a slow, controlled fashion with submaximal weights (21, 22).

ACUTE INJURIES

Acute Patellar Dislocation. Patellar dislocations may be classified as acute, recurrent, or habitual. Acute dislocations tend to occur in adolescent, high-level athletes, whereas recurrent instability occurs in individuals with well-known anatomic variants such as ligamentous laxity, patella alta, and genu valgum (23). Even with acute patellar dislocations, there is commonly an underlying anatomical abnormality that predisposes to the dislocation. The common age for acute patellar dislocation is from 14 to 20 years (23–25).

Anatomy. Understanding the pathology of patellar dislocation requires an understanding of the anatomy of the extensor mechanism of the knee. There are three distinct layers around the patellofemoral joint as part of the extensor mechanism. The superficial layer involves the fascia overlying the sartorius muscle. The second layer comprises the patellar retinaculum and the medial patellofemoral ligament (MPFL). The final layer comprises the medial collateral ligament (MCL) and the joint capsule (26, 27).

The most important stabilizing structure of the patella is the MPFL. It arises from the adductor tubercle and inserts along the medial patellar border on its superior two-thirds. The MPFL varies widely in size, shape, and strength and provides from 50% to 80% of the restraining force to lateral displacement (28–34).

Mechanism of Injury. The most common mechanism is an indirect force applied to the knee. The foot is planted, knee flexed and in valgus, and an internal rotation moment applied to the femur (35). Patellar dislocation is commonly associated with this mechanism of injury in basketball, football, baseball, gymnastics, and also in falls (25).

Less frequently, direct forces applied to the medial side of the patella or to the lateral side of the knee with a valgus force may cause patellar dislocation (36).

History and Physical Examination. The patient usually describes a twisting injury with a mechanism not unlike that for an anterior cruciate ligament (ACL) tear. The patient may describe something moving out of position in the knee and then popping back into place. The knee quickly becomes swollen and the child is reluctant to move it.

On physical examination, the patella is almost always reduced or relocated. In the rare instance where it has not reduced, the child's knee is usually still flexed. The patella may be palpated on the lateral aspect of the lateral femoral condyle. If the knee is passively extended and a gentle medial force applied to the patella, the patella should reduce very easily. A large, tense hemarthrosis accompanies an acute patellar dislocation. Plain radiographs, specifically anteroposterior, lateral, and Merchant view (also known as *skyline views*), should be carefully evaluated for patellar reduction, lateral tilt, and osteochondral fracture. The Merchant view is an axial view of the patellofemoral joint with the knee flexed to a consistent 35 to 45 degrees (37) (Fig. 31-1).

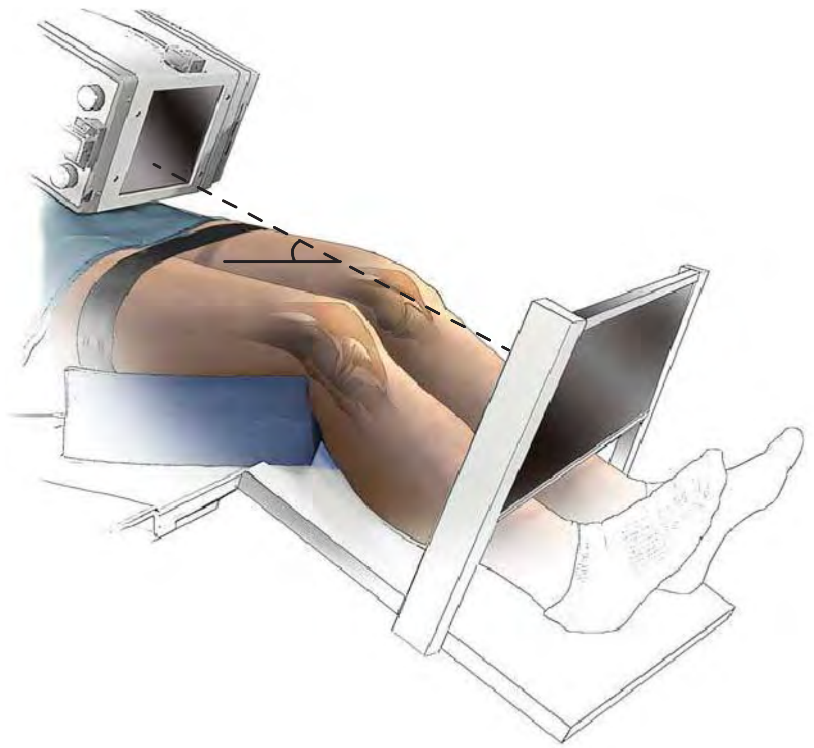

FIGURE 31-1. Tangential x-ray view for evaluating the patellofemoral joint. Merchant view allows the quadriceps mechanism to relax. The patella is not artificially held reduced in the distal femoral groove.

The mechanism of injury for an acute patellar dislocation is similar to an ACL tear, and careful examination to rule out the latter must always be performed.

Treatment. If the patella is still acutely dislocated, reduction should be accomplished promptly with the use of appropriate sedation if required. If the patella cannot be relocated with the patient supine, reduction can be facilitated by placing the patient prone. This allows the hamstrings to relax, and with gentle extension of the knee, the patella will reduce.

There is controversy regarding the most appropriate method of management following an acute patellar dislocation and the potential need for surgical repair in order to prevent recurrence. In two recent articles, there was no difference in redislocation rates between patients treated with surgical repair at the initial dislocation and those treated nonoperatively.

The evidence would strongly support conservative management after an acute patellar dislocation without an osteochondral fracture.

The incidence of osteochondral fracture following patellar dislocation ranges from 5% to 50% (36–39). The incidence is high enough to consider a magnetic resonance imaging (MRI) if the dislocation is associated with a large tense effusion. If an osteochondral fracture is detected, a knee arthroscopy is recommended to visualize the fragment and to determine if the fragment should be replaced or excised. If the fragment is >2 cm and has a significant bony component, fixation should be performed with any number of fixation techniques: low-profile headless cannulated screws countersunk to avoid abrasion, Herbert screws countersunk in the articular cartilage, or bioabsorbable pins or screws (35). In most cases, the fragment is smaller than 2 cm in diameter and should be excised. If significant anatomic abnormalities also exist, consideration of surgical correction at the time of treatment of the osteochondral fracture should be considered (40, 41).

Treatment of acute patellar dislocations without osteochondral fracture involves brief immobilization, then vigorous rehabilitation. The principles of rehabilitation have been elucidated previously and are aimed at resolving the hemarthrosis, reducing the pain, improving the range of motion, and increasing the strength of both the quadriceps and hamstrings (25, 34, 36).

Once the injured knee has been rehabilitated, return to sports is permitted. Prior to resumption of athletic activities, there should be no effusion, full range of motion, and restoration of at least 80% strength of the uninjured knee. The use of a patellar stabilization brace is recommended during sports.

Author's Preferred Recommendations. Following reduction of an acute patellar dislocation, it is imperative to obtain appropriate radiographs to rule out an osteochondral fracture. If symptoms persist, MRI may be helpful in visualizing a chondral or osteochondral defect (Fig. 31-2). If no fracture is detected, rehabilitation is begun. If an osteochondral fracture is detected, a knee arthroscopy determines whether it should be excised (if the fragment is <2 cm with very little subchondral bone) or replaced (if the fragment is >2 cm with significant subchondral bone). Replacement is accomplished by an arthrotomy with the use of small cannulated screws countersunk to the level of the subchondral component. Acute repair of medial structures including MPFL and patellar retinaculum is carried out, and usually a lateral retinacular release is done at the same time. The knee is immobilized for approximately 10 to 14 days in a soft dressing and knee immobilizer, followed by vigorous rehabilitation.

There are proponents of acute surgical repair in the absence of osteochondral fracture (40–42), but I prefer

FIGURE 31-2. Lateral image of knee (MRI) with large osteochondral defect of lateral femoral condyle after acute patellar dislocation. *Arrows* (*solid black*) point to defect in lateral femoral condyle.

nonsurgical treatment because 50% to 60% of patients older than 10 years will not experience a recurrence (43, 44).

Recurrent Patellar Dislocation

Clinical Features. Children with recurrent patellar instability have one or several features which predispose to the recurrence. Anatomic factors include an increased Q angle, increased femoral tibial valgus, excessive external tibial torsion, femoral condylar dysplasia, patella alta, and generalized ligamentous laxity (23–25, 35, 45–47). The Q angle is the angle formed by a long axis drawn along the quadriceps mechanism from the anterior superior iliac spine (ASIS) to the midaxis of the knee joint subtended by a long axis drawn along the patellar tendon. A normal Q angle is 10 degrees or less (Fig. 31-3A–C). Children with recurrent patellar instability exhibit a positive apprehension test. Apprehension is produced when an attempt is made to displace the patella laterally with the knee flexed approximately 30 degrees.

Surgical Management. Surgery is indicated when a patient has had three or four recurrences of patellar dislocation and the instability affects his or her lifestyle. Correction of the anatomic variants is crucial for the long-term outcome.

Surgery may entail soft-tissue surgery around the patella, including lateral retinacular release, reconstruction of the MPFL, vastus medialis advancement with medial reefing, Insall's proximal patellar realignment, or semitendinosus tenodesis.

Lateral retinacular release alone for patellar instability is rarely indicated. There are isolated reports of its success in the treatment of recurrent patellar dislocation, but its exact role in this condition remains to be determined (48, 49). Excessive lateral retinacular release combined with aggressive medial reefing may result in iatrogenic medial subluxation and must be avoided (29, 50, 51). Lateral retinacular release usually must be combined with reconstruction of the MPFL or by vastus medialis advancement.

Reconstruction of the MPFL has become popular in acute or early recurrent dislocation of the patella (29, 35). The MPFL may be reconstructed in several ways including free hamstring graft (semitendinosus tendon) (29) or by the use of an autologous quadriceps tendon (52) (Fig. 31-4A–E). It can be routed through drill holes in the patella and fixed to its normal origin at the adductor tubercle or fixed in a similar fashion using suture anchors.

In the case of the autologous quadriceps tendon, an 8 mm width × 60 mm length medial quadriceps tendon graft is harvested, pressed beneath the medial patellar retinaculum, and sutured to the intermuscular septum at the adductor tubercle. The graft is sutured in place with the knee in 30 degrees of flexion.

Thus drill holes are avoided, allowing the procedure to be performed in skeletally immature individuals. As a variation, the graft may be fixed to the femur at the same location using a suture anchor. In the situation of more chronic recurrent dislocations, a vastus medialis obliquus (VMO) advancement distally and laterally with medial reefing is performed in addition to the MPFL reconstruction (53–55).

Proximal Patellar Realignment (Insall Technique) (Figs. 31-5 to 31-8). With subluxation or dislocation of the patella in skeletally immature patients, the open growth plate of the tibial tubercle, which prohibits operations that transfer the origin of the patellar tendon, limits the surgeon's options. For the growing child with recurrent subluxation or dislocation of the patella—whether owing to malalignment, trauma, or mild ligamentous laxity (e.g., that seen in Down syndrome)—the proximal soft-tissue realignment described by Insall and colleagues (55, 56) provides a method of realigning the forces on the patella. For us, this method is preferable to detaching and then advancing the vastus medialis muscle. In cases in which advancement of the medialis muscle seems necessary, the muscle is usually so deficient that little is gained, and it is difficult to secure the muscle in place. The proximal realignment provides a secure repair with little tension on the suture lines and therefore earlier rehabilitation.

In cases of congenital dislocation associated with deficiency of the lateral femoral condyle or muscle structure, however, this operation is usually not sufficient. This is also true for children with Down syndrome or other collagen disorders who have severe ligamentous laxity and poor tissue for repair. In such cases, we prefer to combine elements of this procedure with the semitendinosus tenodesis of the patella.

Semitendinosus Tenodesis of Patella for Recurrent Dislocation (Figs. 31-9 to 31-15). The use of the semitendinosus tenodesis was first described by Galeazzi in 1922 (57, 58). The semitendinosus tenodesis procedure addresses several

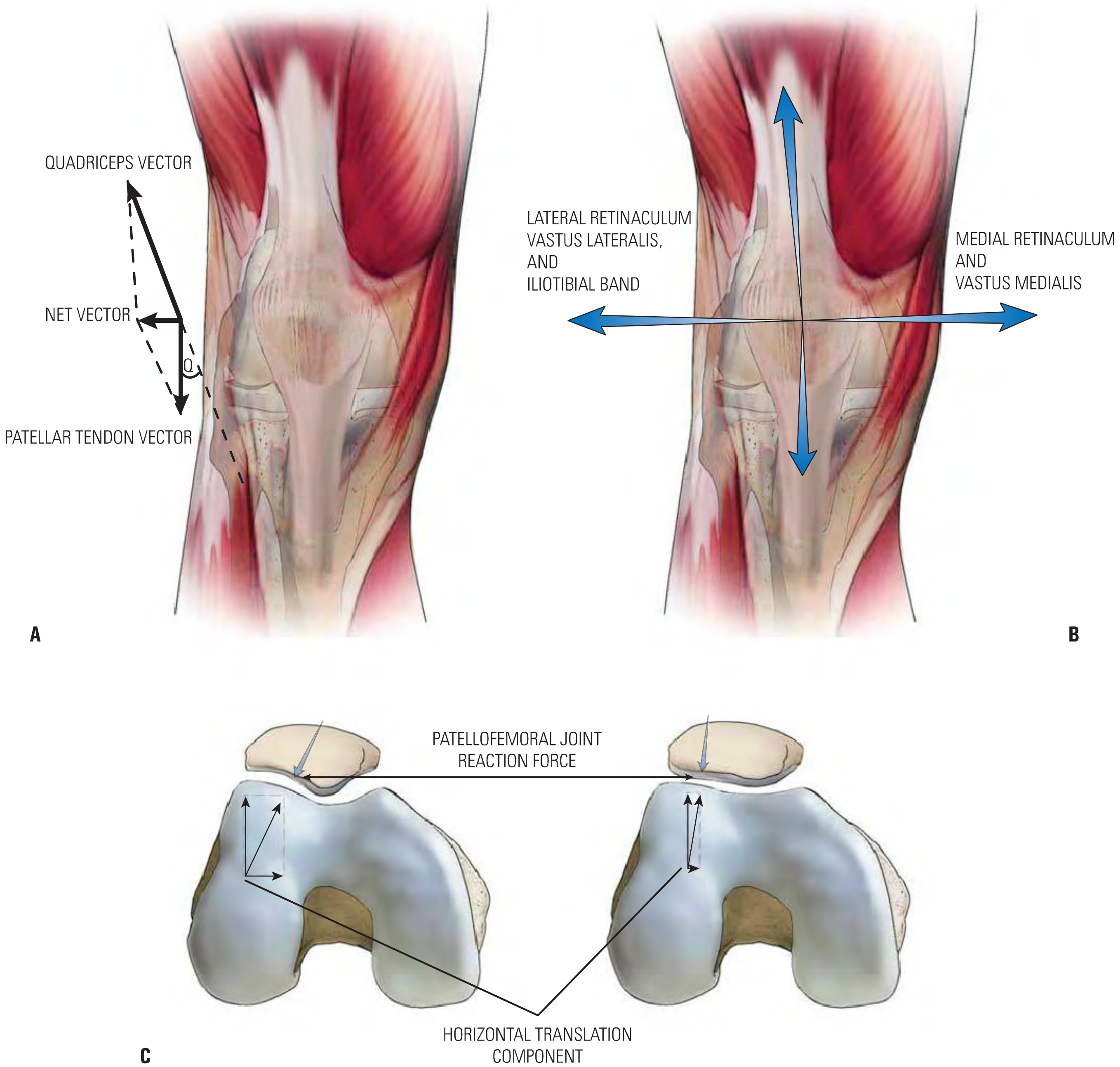

FIGURE 31-3. Patellofemoral biomechanics. **A:** The Q angle relates the direction of pull of the quadriceps mechanism to that of the patellar tendon. These are the two most powerful forces exerted on the patella. Their vector sum is directed laterally. **B:** There are additional soft-tissue forces applied to the patella. **C:** The laterally directed net vector is opposed by the patellofemoral articulation. If the groove is shallow, there is less potential resistance to horizontal translation than in knees with a deeper femoral groove. The dysplastic patellofemoral articulation results in less resistance to lateral translation and therefore greater sheer forces on the articular surface.

problems that the orthopaedic surgeon often encounters in the child with recurrent dislocation of the patella: ligamentous laxity, deficient lateral condyle, deficient medial musculature, and open growth plates. In all of the conditions in which recurrent dislocation of the patella is encountered (e.g., Down syndrome, congenital dislocating patella), the semitendinosus tendon is usually normal. We have found this procedure, often in combination with a proximal realignment, to be an excellent solution to the unusual problem of recurrent dislocating patella in skeletally immature children.

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Reconstruction of the MPFL (Fig. 31-4)

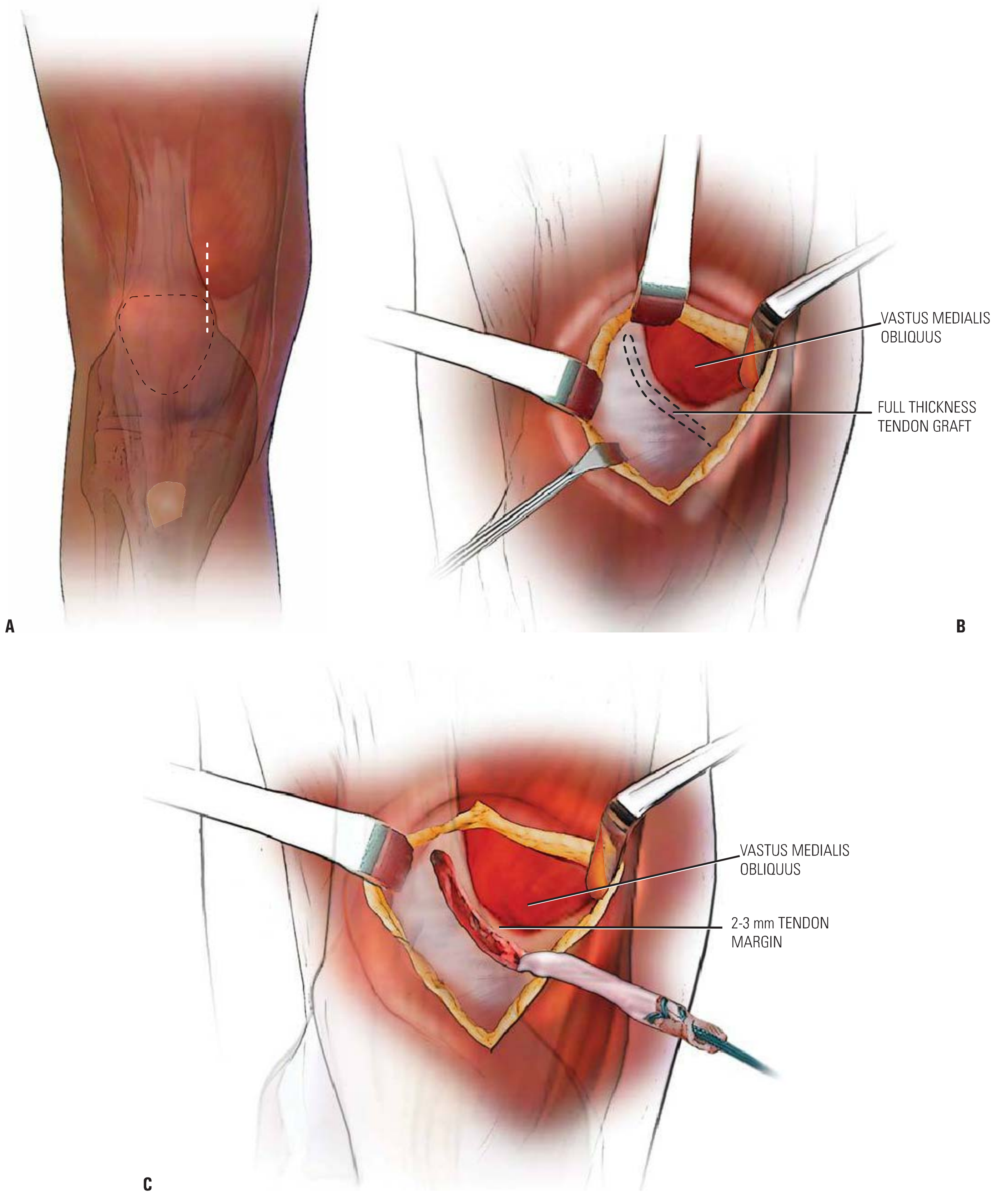
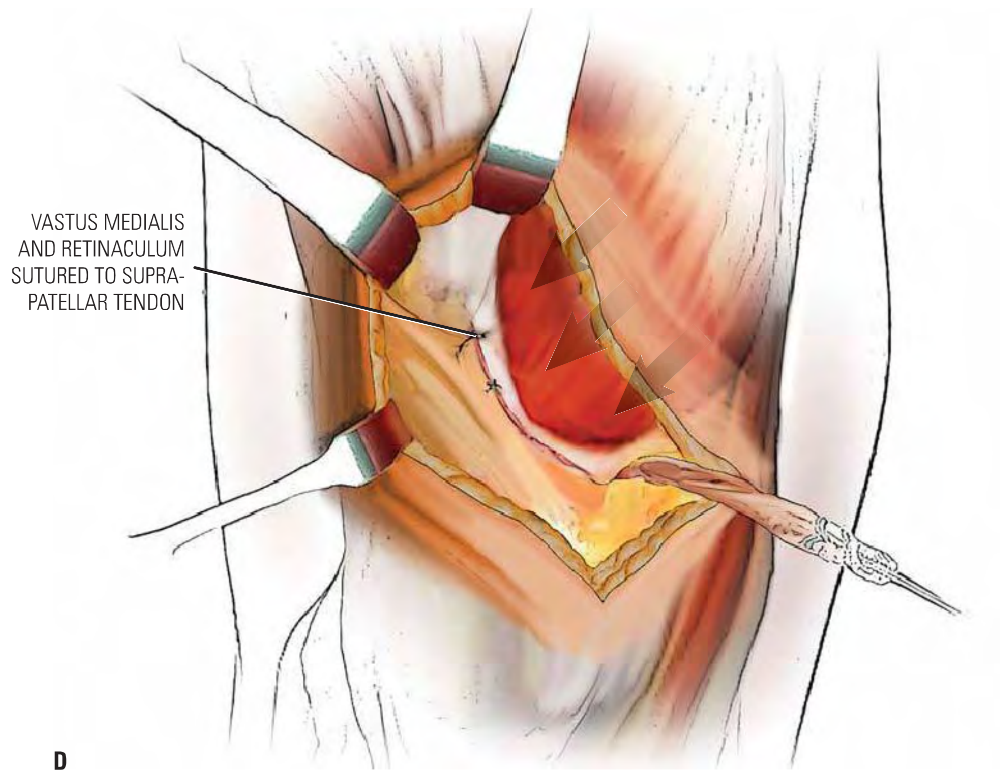


FIGURE 31-4. Reconstruction of the MPFL. A: Medial approach. **B:** Harvest full-thickness quadriceps tendon graft 60 mm long by 8 mm wide. Leave 2 to 3 mm of tendon with the VMO. **C:** Dissect the retinaculum free on its superficial and deep surfaces, posteriorly to the medial epicondyle. Puncture the medial retinaculum superficial to the medial epicondyle.



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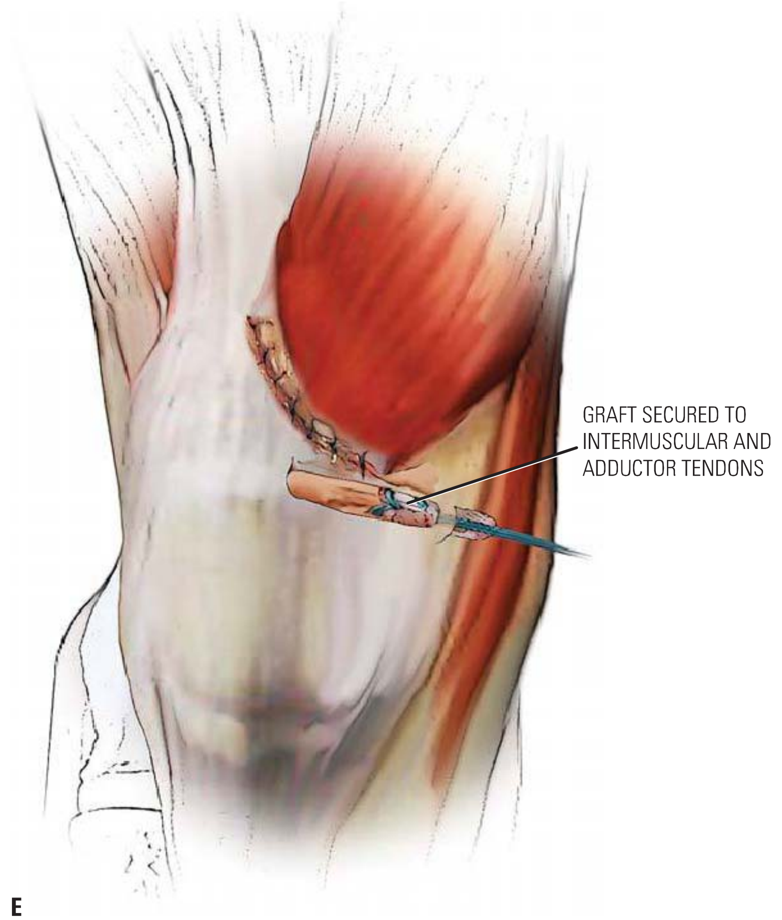


E

FIGURE 31-4. (*Continued*) **D:** The VMO and retinaculum are imbricated and reattached to the patella. **E:** With the knee in 30-degree flexion, the graft is secured to the intermuscular septum and adductor tendon insertion. Graft tension: assure that the patella can be laterally displaced 25% of its width.

Proximal Patellar Realignment (Insall Technique) Figs. 31-5 to 31-8

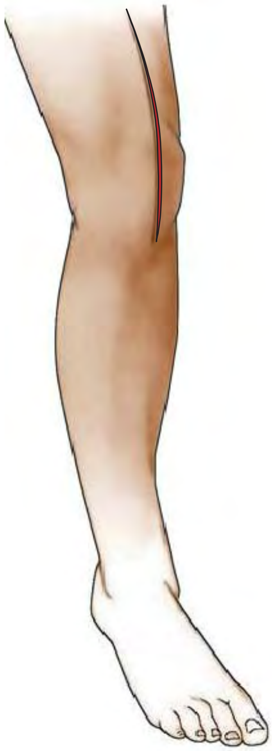
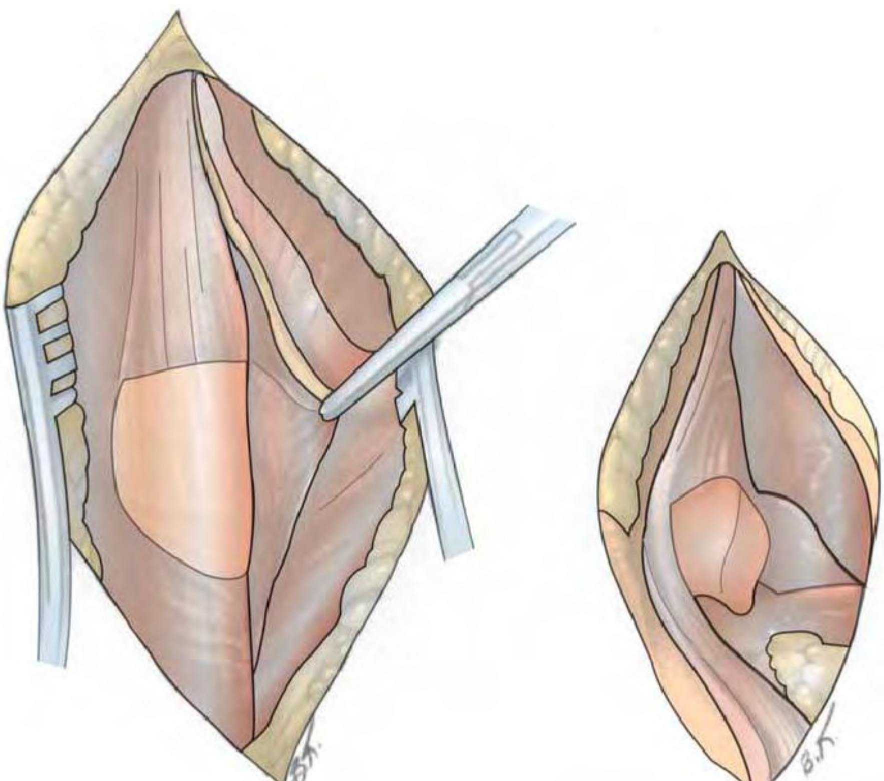


FIGURE 31-5. **Proximal Patellar Realignment (Insall Technique).** The operation is performed with the patient supine and with a bolster under the hip to avoid the need for an assistant to hold the leg in internal rotation. The incision begins in the midline, just below the junction of the middle and lower one-third of the thigh, and extends distally across the center of the patella to the tibial tubercle. The incision must be long enough to expose the entire quadriceps tendon.



A B

FIGURE 31-6. The flaps are reflected medially and laterally sufficiently to expose the medial and lateral border of the patella and the insertion of the vastus medialis and lateralis into the quadriceps tendon and detaches the vastus medialis from this tendon, leaving just enough tendon on the muscle on the muscle to hold sutures. As this incision is carried distally, it should be directed to cross the patella, dividing the medial one-third from the lateral two-thirds and then continuing down along the medial border of the patellar tendon. The quadriceps expansion overlying the medial one-third of the patella is then elevated subperiosteally from the patella **(A)**. This allows the patella to be turned up laterally, exposing the joint. By dividing the fat pad, the undersurface of the patella and the joint can be inspected **(B)**.

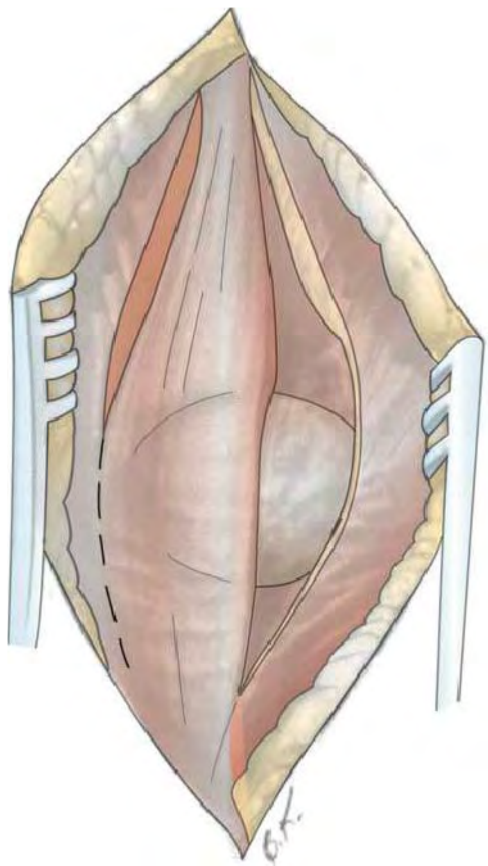


FIGURE 31-7. The next incision divides the lateral patellar retinaculum and separates the vastus lateralis from the quadriceps tendon. This incision begins at the quadriceps tendon proximally, opposite the medial incision. The vastus lateralis is detached, leaving a rim of tendon for suturing. As this incision approaches the patella, it skirts the lateral margin of the patella. The synovium should also be divided, with care taken to identify and coagulate the vessels that will be encountered. If the surgeon desires, the tourniquet can be released at this point to control any bleeding and then reinflated before beginning the repair.

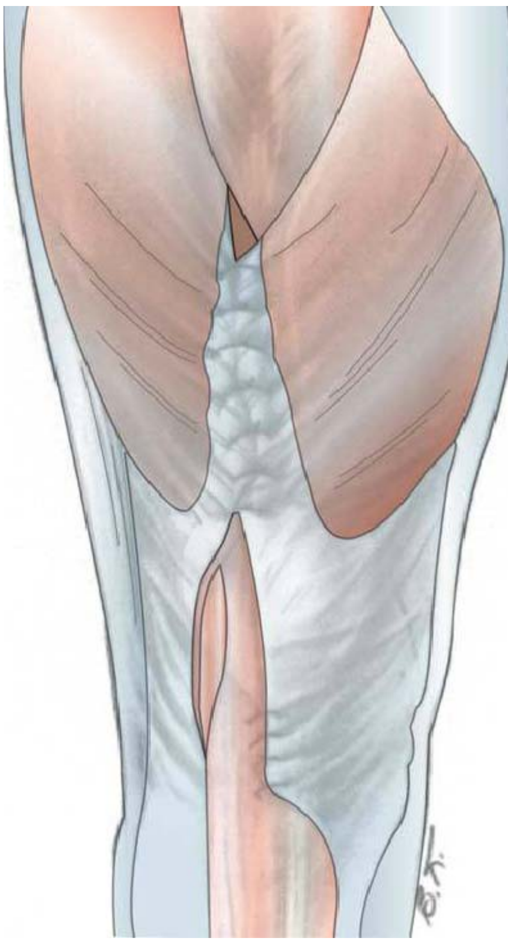


FIGURE 31-8. The repair is started proximally by bringing the cut edge of the vastus medialis and vastus lateralis together over the remaining portion of the quadriceps tendon, which is pushed deep to the repair. As the repair reaches the proximal pole of the patella, the patella begins to rotate medially, elevating the lateral portion of the patella. It is neither necessary, nor is it possible, to continue this repair across the entire patella because the medial periosteal flap does not reach the lateral retinaculum. Rather, when the patella is rotated and displaced medially to a sufficient degree, the medial flap is sutured to the periosteum on the lateral two-thirds of the patella without further effort to pull the patella medially. The knee can now be flexed to test the stability of the patella. The lateral incision is left open.

Semitendinosus Tenodesis of Patella for Recurrent Dislocation (Figs. 31-9 to 31-14)

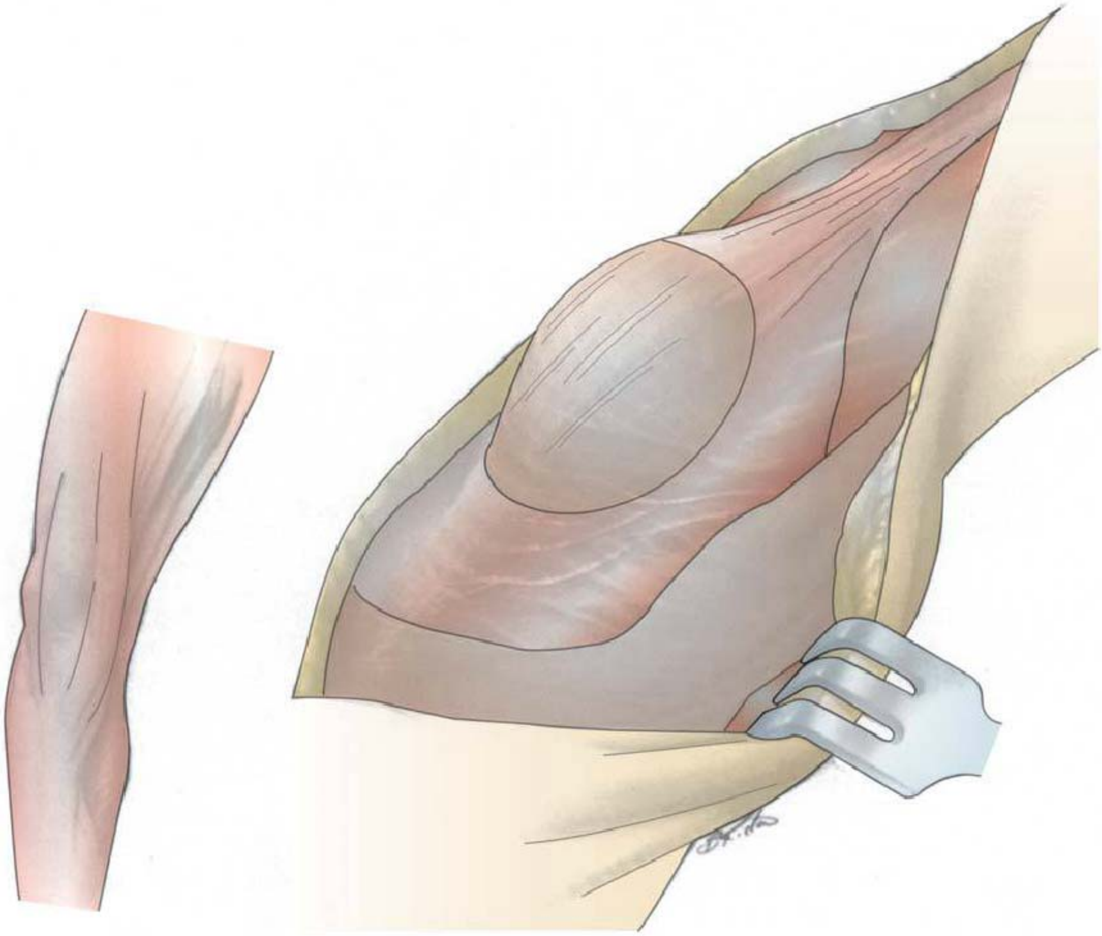


FIGURE 31-9. Semitendinosus Tenodesis of Patella for Recurrent Dislocation. The patient is placed supine on the operating table, and the entire leg is draped free. A tourniquet is used, and one incision is made. Although a medial parapatellar incision makes it slightly easier to reach the semitendinosus tendon, a long midline incision, as described for proximal realignment, is better cosmetically. The tendons on the medial side of the knee are illustrated **(A)**. Note the broad expanse of the sartorius, which is the most anterior. The gracilis tendon lies just behind the Sartorius. The semitendinosus is the most posterior, behind the knee, and is the deepest or most posterior tendon inserting into the tibia. It is easily distinguished from the gracilis, not only by the location of its insertion, but also by its size: it is a much larger tendon. The surgeon should not make the mistake of taking the gracilis tendon for repair.

The medial skin flap is elevated extensively around the medial side of the knees. The dissection must be carried both posteriorly and proximally. Flexing the kneed aids in this dissection. The infrapatellar branch of the saphenous nerve can usually be observed emerging from the Sartorius. Although a few of its sensory twigs may be divided, care should be taken with this nerve to avoid a large area of anesthesia **(B)**.

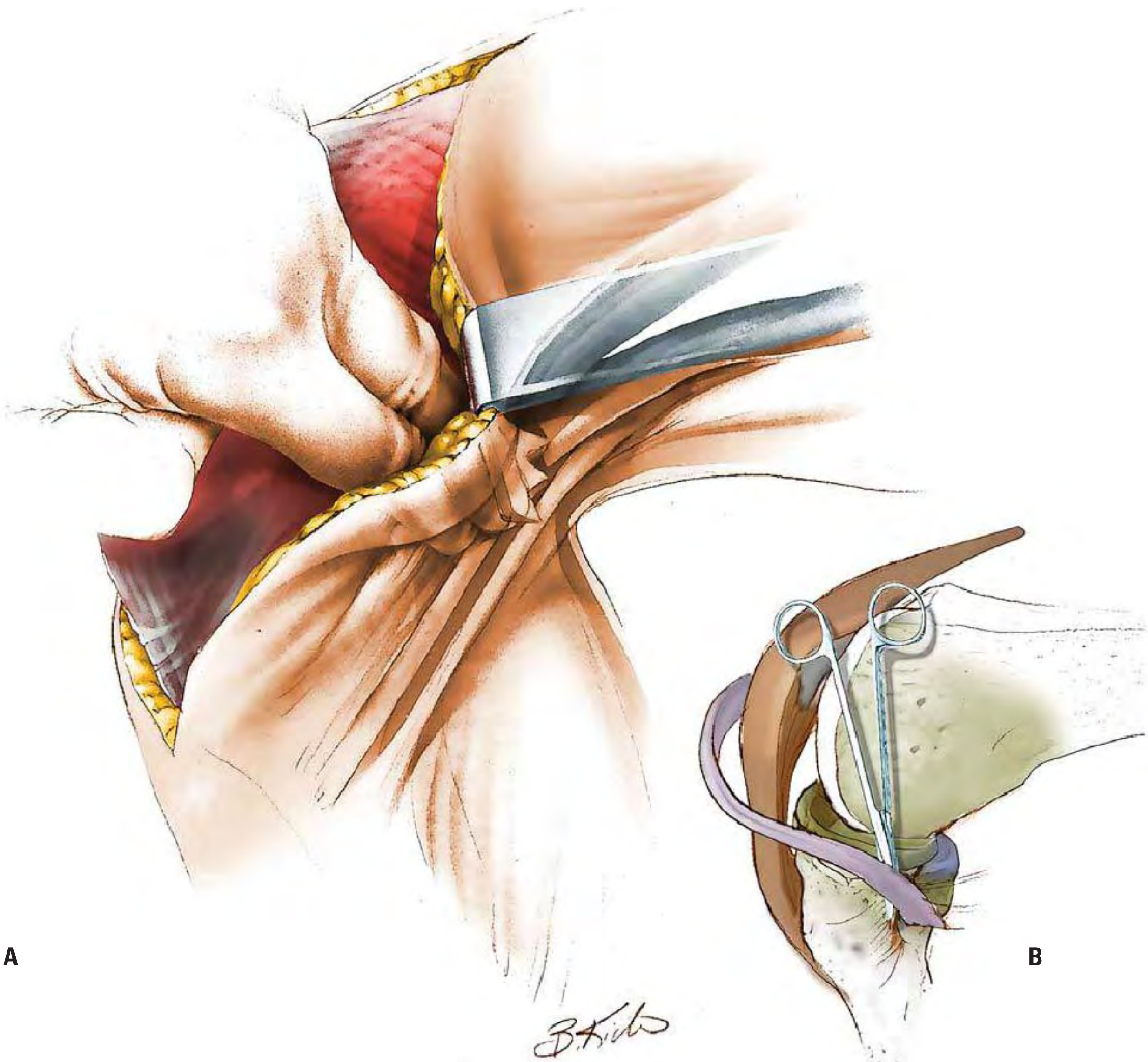


FIGURE 31-10. With the knee flexed, the skin flap is retracted with a long blade retractor and blunt dissection is continued posteriorly and proximally **(A)**. At this point care should be taken to avoid injury to both the infrapatellar branch of the saphenous nerve and the saphenous nerve itself (see Fig. 31-9). The tendon can be palpated. As mentioned previously, it is a larger structure than the gracilis tendon, which may be taken by mistake. It lies posterior to the Sartorius and gracilis tendons in this location. After the tendon is identified and exposed, it should be followed to its musculotendinous junction, where it is divided.

Next, the tendon should be followed to its insertion posterior to the Sartorius and gracilis tendons, freeing all extraneous attachments with care to avoid cutting the saphenous nerve **(B)**. If the tendon is not completely freed to its insertion, it will not have the proper direction and will soon become loose as the fascia that tethered it becomes stretched.

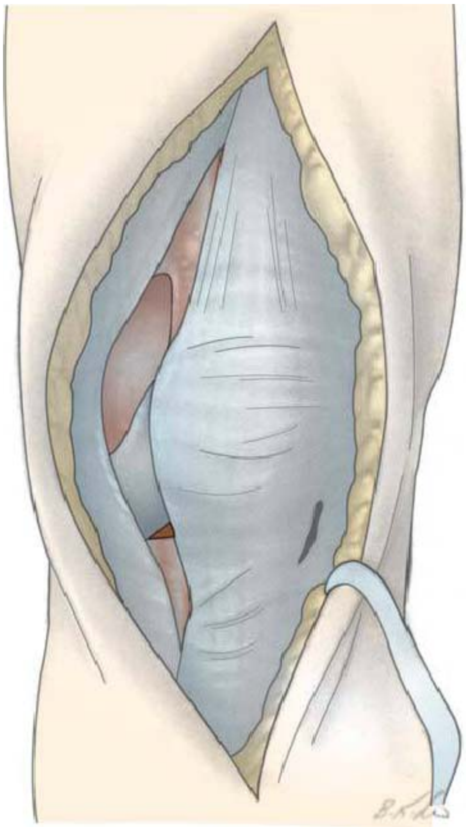


FIGURE 31-11. The lateral flap is now dissected to expose the lateral border of the patella. A complete lateral release should be performed, at the minimum, including both the capsule and the synovium. At this point, the surgeon can decide whether to perform a more extensive realignment of the patella with advancement of the vastus medialis muscle or a complete proximal realignment. If nothing more is to be done (as illustrated here for simplicity), a small incision should be made in the medial capsule at the distal end of the patella. This will allow palpation of the inferior surface of the patella for more accurate placement of the drill hole.

FIGURE 31-12. With the patella held in the desired position and the tendon pulled across the surface of the patella, the proper direction for the drill hole can be determined **(A)**. Starting at the inferior medial edge of the patella, a hole of sufficient size to allow passage of the tendon is drilled, emerging at the superior lateral corner of the patella **(B)**. In directing the drill, the surgeon must be careful to avoid penetrating the articular surface.

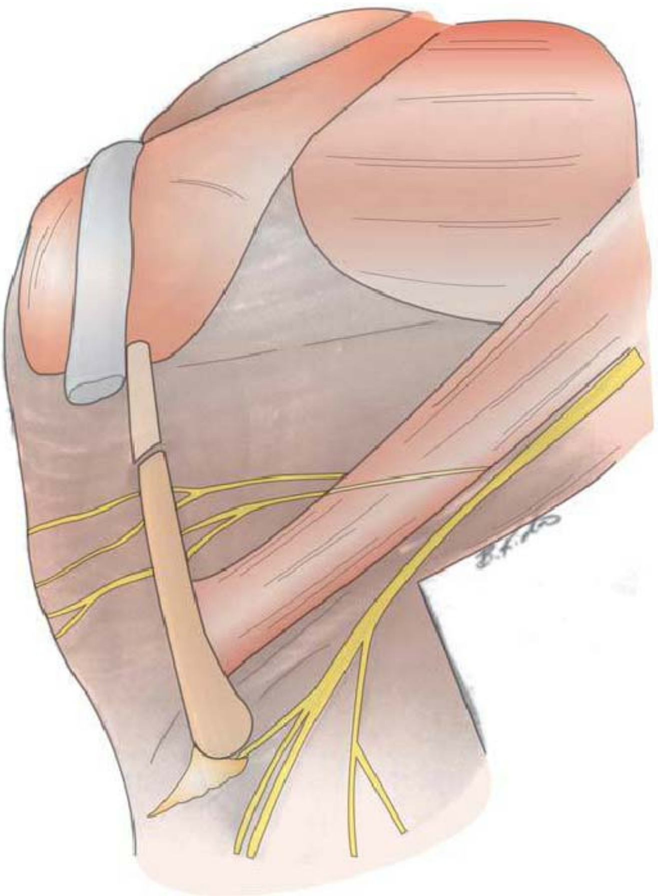


FIGURE 31-13. The tendon is drawn through the hole and pulled back on itself. Sufficient tension should be placed on the tendon to hold the patella in line with the intercondylar notch. This can be tested by flexing the knee while an assistant holds tension on the tendon. The tension should be sufficient to create laxity of the patellar tendon.

Note the infrapatellar branch of the saphenous nerve that penetrates the Sartorius muscle and branches over the medial capsule of the knee. The main branch of the saphenous nerve emerges from between the Sartorius and gracilis tendons to continue down the leg. Care must be taken during both the dissection and the routing of the tendon to be certain that those nerve are neither blocked nor kinked.

The operation is completed by suturing the semitendinosus tendon to the periosteum of the patella and, if sufficient length is available, to itself.

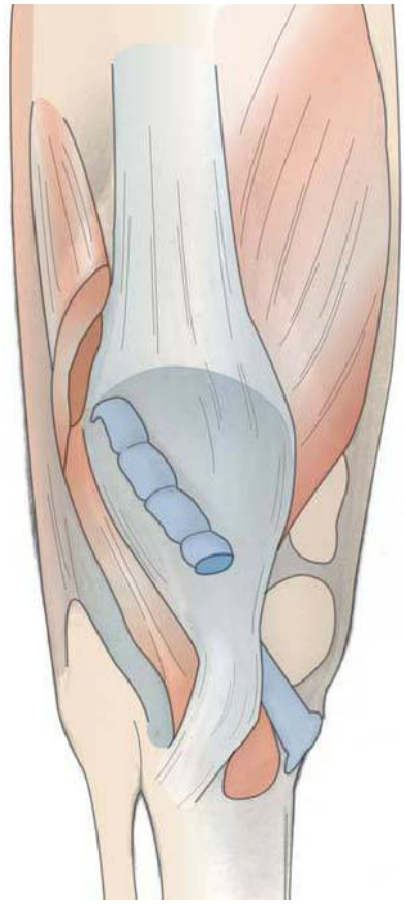


FIGURE 31-14. To restore tension to the patellar tendon and effect some redirection in its line of pull, a Goldthwait procedure can be added. This entails splitting the patellar tendon in half, detaching the lateral half, directing this half under the medial half of the tendon, and attaching it to the periosteum of the tibia under moderate tension. At the completion of this step, any muscle advancements or other steps to augment the realignment are completed, and the wound is closed over a suction drain.

This graft reproduces the vector of the patellotibial ligament. It can also be employed when there is persistent instability after already performing a lateral release and MPFL reconstruction or VMO advancement (Fig. 31-15).

If there is an excessive Q angle, distal realignment is advocated as well. If the individual is a skeletally mature youth, the tibial tubercle is osteotomized and shifted medially without distal transfer (Elmslie-Trillat procedure) (59) (Fig. 31-16A,B). A modification of the Elmslie-Trillat procedure is the Fulkerson procedure (60), in which a more generous osteotomy of the anterior tibial tubercle is performed and the tubercle transferred anteriorly and medially (Fig. 32-16C). This procedure is primarily reserved for patellofemoral pain in adults and is not recommended for instability in adolescents or young adults.

In the immature child with an excessive Q angle and open tibial tubercle apophysis, osteotomy is contraindicated because of the potential for growth arrest and genu

Text continued on page 1612

Semitendinosus Tenodesis of Patella for Recurrent Dislocation: The Galeazzi Procedure (Figs. 31-15 and 31-16)

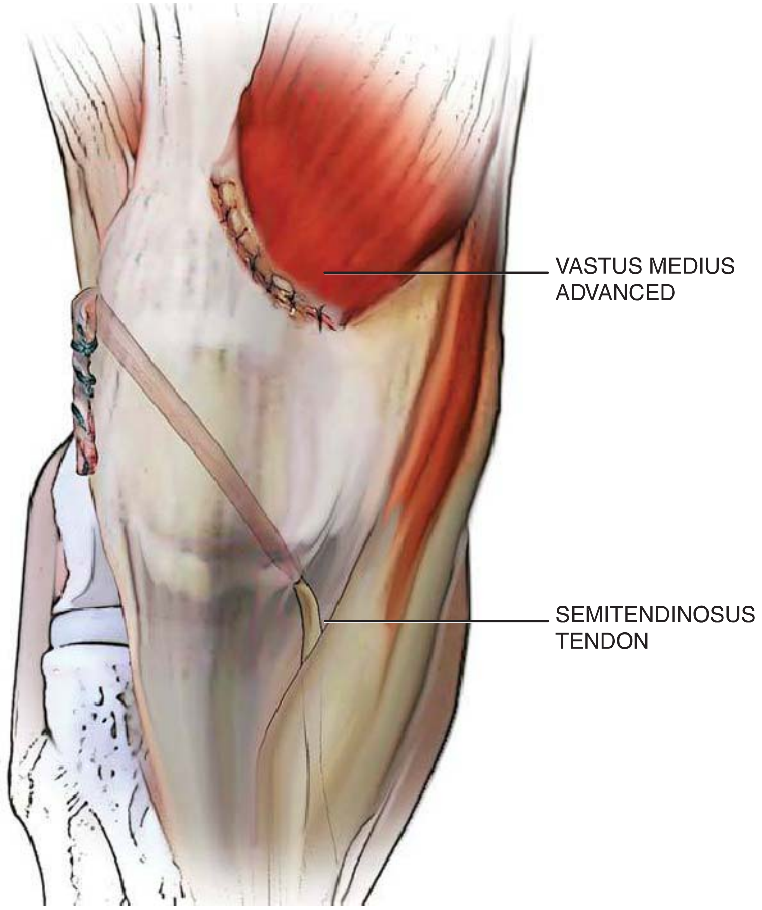


FIGURE 31-15. Semitendinosus Tenodesis of Patella for Recurrent Dislocation: The Galeazzi Procedure. The Galeazzi procedure transfers the semitendinosus to the inferior pole of the patella. From there, it courses through a drill hole placed obliquely through the patella, exiting the superior lateral aspect. The tendon is then sutured to the soft tissues. This provides a medial tether and effectively alters the net vector of the patellar tendon toward the medial side. Typically, the vastus medialis is advanced approximately one-third the width of the patella.

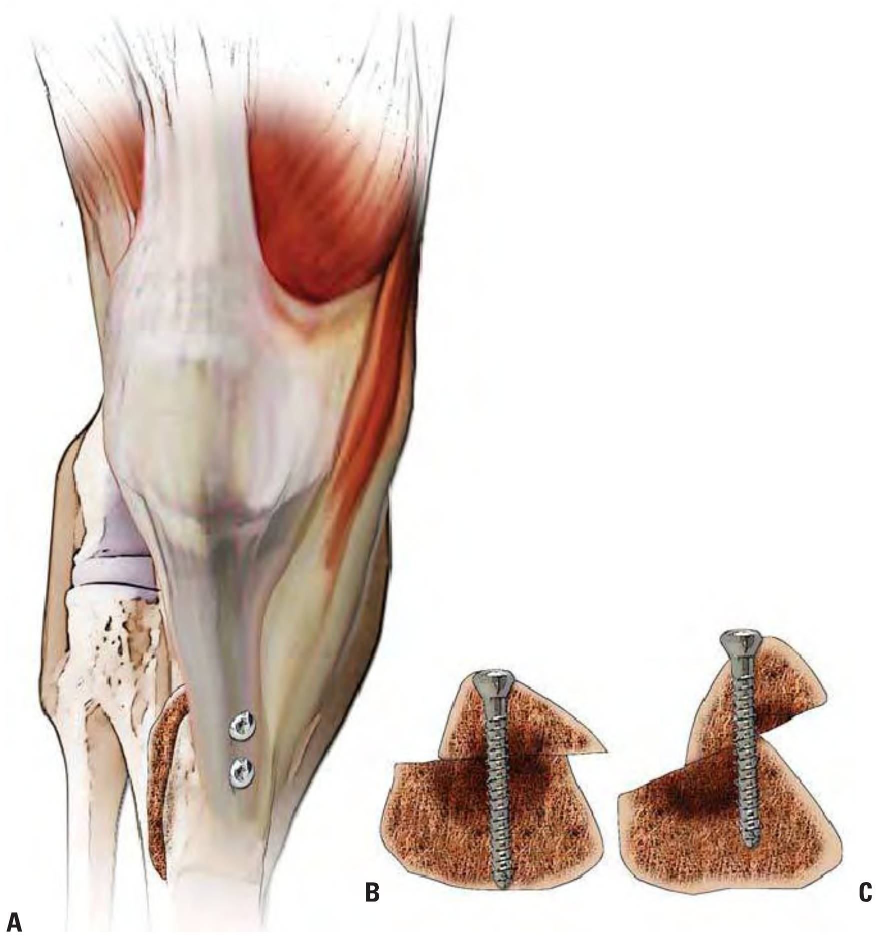


FIGURE 31-16. A, B: The Elmslie-Trillat technique shifts the tibial tubercle medially. The tubercle stays in the same plane. **C:** The Fulkerson modification involves an oblique cut that results in anterior translation as the tubercle is moved medially. This reduces the patellofemoral contact forces while shifting the pull of the patella medially.

FIGURE 31-17. The Roux-Goldthwait procedure splits the patellar tendon. The lateral half is transferred beneath the medial side and sutured to the periosteum along the metaphysis. This redirects the patellar tendon vector more medially.

recurvatum. In these cases, the patellar tendon may be split and the lateral half delivered beneath the medial portion of the patellar tendon and sutured to the periosteum of the proximal tibia medially by direct suture or by suture anchors (61) (Fig. 31-17). Acceptable results can be expected in up to 90% of cases employing the Roux-Goldthwait procedure.

An option is to sharply dissect the entire patellar tendon from its insertion using a scalpel and to resuture it more medially to restore a normal Q angle (62). Care must be taken not to move the insertion too distally, which may lead to patella baja and significant pain.

Rehabilitation following a patellar stabilization procedure is very important. It is crucial to move the knee early, and immobilization in a removable knee immobilizer for 3 to 4 weeks is sufficient for healing. Active range of motion and strengthening are essential parts of the rehabilitation program, and a resumption of sports activity can be anticipated in 4 to 6 months.

Author's Preferred Method. The critical steps in correcting recurrent patellar instability include a thorough analysis of anatomic factors preoperatively and intraoperative evaluation of the reconstruction. I employ a stepwise surgical protocol that almost always involves a lateral retinacular release and reconstruction of the MPFL. If the Q angle is within normal limits and the patella is stable, there is no need for further surgery. It is imperative that the knee is put through flexion and extension to ensure normal patellar tracking and to ensure the MPFL reconstruction is not too ambitious, in which case it will limit flexion. This is now my preferred method of surgical management of the adolescent with recurrent patellar dislocation.

If the Q angle is excessive, distal realignment is performed; the Roux-Goldthwait procedure is used for the skeletally immature patients up to 14 years of age and the Elmslie-Trillat procedure for patients older than 14 years.

Meniscal Injuries and Discoid Lateral Meniscus

Introduction. While the exact incidence of meniscal injuries in children and adolescents is unknown, these injuries are thought to be on the rise due to increased athletic participation, improved physician familiarity, and wider availability and use of MRI (63). The greater healing potential of the pediatric meniscus and the consequences of meniscectomy in a young active patient (increased contact forces and early osteoarthritic changes) underscore the importance of proper diagnosis and treatment of pediatric meniscal injuries. New surgical techniques have facilitated arthroscopic meniscal repair which has become the standard of care for repairable tears.

Anatomy and Function. The C-shaped medial meniscus covers 50% of the medial tibial plateau and has ligamentous attachments to the tibia (through the coronary ligament) and to the deep MCL through the meniscotibial ligament. These prevent the medial meniscus from translating more than 2 to 5 mm with knee motion. The circular lateral meniscus covers 70% of the lateral tibial plateau and lacks attachments to the fibular collateral ligament and at the popliteus hiatus. This results in increased mobility of the lateral meniscus, which normally translates 9 to 11 mm during knee motion. Meniscal blood supply arises from the geniculate arteries which form a peripheral perimeniscal synovial plexus. The developing meniscus is fully vascularized at birth, and its vascularity gradually diminishes to the peripheral 10% to 30% of the meniscus (red-red zone) by age 10 at which time it resembles the adult meniscus (64, 65). Synovial diffusion is responsible for nutrition of the central portion of the meniscus. The menisci are load sharing and reduce contact stresses across the knee joint, transmitting 50% to 70% of the load in extension and 85% of the load in 90 degrees of flexion (66).

Diagnosis. Nondiscoid meniscal tears usually occur in older children following a twisting knee injury (65, 67–69). Concurrent ligament injuries such as ACL tears are common (69). Meniscal injuries in children generally present with joint line pain and swelling. The physical exam by experienced examiners reliably diagnoses medial (62% sensitivity, 80% specificity) and lateral (50% sensitivity, 89% specificity) meniscal tears in children (70). The modified McMurray test—40 degrees of knee flexion with rotational varus/valgus stress—is appropriate for children who resist traditional McMurray test. Lachman test is reliable in children, although comparison to

FIGURE 31-18. Sagittal T2-weighted MRI demonstrating high intrameniscal signal (*white arrow*) in the posterior horn of the developing meniscus.

the other side is necessary (71). Knee radiographs including tunnel and sunrise views rule out alternative diagnoses including patellar subluxation, osteochondritis dissecans (OCD), and osteochondral loose bodies (72).

Sensitivity and specificity rates for MRI diagnosis of meniscal tears in skeletally immature children are 79% and 92% for medial meniscal tears and 67% and 83% for lateral meniscal tears (73). In this population, MRI does not improve diagnostic accuracy compared to physical exam by an experienced examiner (73). MRI diagnosis of meniscal tears in children younger than 12 years old has lower sensitivity (61.7% versus 78.2%) and specificity (90.2% versus 95.5%) compared with children 12 to 16 years old (73). The high vascularity of the developing meniscus may cause intrameniscal signal change that can be misinterpreted as a tear (Fig. 31-18) (74).

Management. Surgery is indicated for the majority of symptomatic meniscal tears in children. Small longitudinal tears (<10 mm) in the peripheral red-red zone that are manually displaceable by <3 mm will often heal on their own (75). For larger and unstable tears, meniscal repair is preferred over meniscectomy as high rates of osteoarthritis are noted at 10 to 20 years following meniscectomy (76).

Most meniscal tears (50% to 90%) in children are peripheral longitudinal tears that have high healing potential (77). Bucket handle tears are not uncommon and often involve a large portion of the meniscus. Given the consequences of partial meniscectomy and healing potential in this age group, meniscal repair should be attempted for most middle and peripheral third tears, especially in the setting of ACL reconstruction. Degenerative patterns such as horizontal cleavage tears, radial tears, and complex tears are less common and may require partial meniscectomy. In these cases, a judicious partial meniscectomy is recommended as contact forces increase with the volume of meniscal tissue removed (78).

Arthroscopic meniscal repair is accomplished using outside-in, inside-out, and all-inside techniques. Common surgical principles include repair site preparation with abrasion or trephination and anatomic reduction followed by stable fixation. Recent advances have expanded the role of all-inside repair. Second-generation suture-based systems limit chondral wear and provide suture compression across the tear. For smaller knees, all-inside repair places the posterior neurovascular structures at risk due to instrument size and overpenetration of the posterior capsule. A standard inside-out approach is safest in these cases. Anterior horn tears are best repaired with an outside-in approach.

Results. Few studies have specifically analyzed results of meniscal repair in children. The first report showed 100% clinical healing at 5-year follow-up on 26 patients of mean age 15.3 (range 11 to 17) who underwent 29 meniscal repairs using inside-out (25) and all-inside (4) technique (79). Another study analyzed arthroscopic meniscal repair on 71 children of mean age 16 (range 9 to 19, 88% were skeletally immature) using an inside-out technique on meniscal tears extending into the central avascular zone. At mean follow-up of 51 months, 53 of 71 patients (75%) were clinically healed including 39 of 45 (87%) who underwent simultaneous ACL reconstruction (80). More recently, retrospective results of meniscal repair in 12 children of mean age 13 (range 8 to 16) have been reported. At 3-year follow-up, seven patients were asymptomatic, two had occasional pain, and three (25%) required reoperation for partial meniscectomy (81). Long-term data are lacking and hence whether meniscal repair will lower the rate of future osteoarthritis remains unknown. Potential complications following meniscal repair in children include neurovascular injury, arthrofibrosis, and infection. Of the series above, only two cases of arthrofibrosis and one painful neuroma of the infrapatellar branch of the saphenous nerve were reported (79).

Discoid Meniscus Introduction. First described through cadaveric dissection by Young in 1887, discoid morphology implies a block-shaped, thickened, and enlarged meniscus that occupies a larger than normal percentage of the tibial plateau (Fig. 31-19). Occurring most commonly in the lateral meniscus (97%) and in the Asian population (15%), the true prevalence is unknown since many discoid menisci are asymptomatic (82, 83). Normal menisci are never discoid during development and hence discoid menisci likely represent a congenital variant (64, 84).

Anatomy and Classification. Watanabe's classic report on discoid menisci described three variants: type 1 (complete, stable) and type 2 (incomplete, stable) are uniformly thickened (8 to

FIGURE 31-19. Arthroscopic view of a block-shaped, thickened discoid lateral meniscus (*D*).

10 mm) and block shaped but differ in the amount of tibial plateau they cover (type 1—complete, type 2—<80%). Watanabe type 3 (unstable, Wrisberg-variant) discoids have varied appearance but lack peripheral posterior attachments except for the ligament of Wrisberg (85). Discoid menisci more likely represent a spectrum of abnormalities of size, shape, and peripheral rim stability. A recent series of 128 discoid lateral menisci (62% complete, 38% incomplete) found peripheral rim instability in 28%, with instability occurring at the anterior horn (47%), posterior third (39%), and middle third (11%) (86). Rim instability was associated with young age (mean age 8.2) and complete discoids (86).

Discoid menisci have less total number of collagen fibers and a more disorganized arrangement, which may be responsible for higher tear rates (87). Horizontal cleavage tears are most common (58% to 98%), likely from repetitive microtrauma causing delamination (72, 88–91). Tear pathology often shows mucoid fibrinous degeneration (91).

Diagnosis. Most discoid menisci are asymptomatic, although some unstable variants present early as the snapping-knee syndrome. Painless snapping occurs as the knee moves from flexion to extension and the unstable meniscus reduces back to its normal position. Physical exam may reveal a lateral joint line bulge with knee flexion and McMurray testing may elicit a clunk. The contralateral knee is involved in up to 20% of cases. Stable discoid menisci are asymptomatic but prone to tear and often present in older adolescents with signs of meniscal injury (joint line pain, effusion, positive McMurray test) (64).

Radiographs are usually normal, though classic findings include squaring of the lateral femoral condyle, cupping of the lateral tibial plateau, widening of the joint line, and meniscal calcification. MRI has low sensitivity (38.9%) compared to physical exam (88.9%) for diagnosis of discoid lateral meniscus in children (73). On MRI, sagittal images may show the bow-tie sign (three or more 5-mm slices with continuity between the anterior and posterior horns), while coronal images may show a meniscal diameter >15 mm or >20% of the total tibial width (Fig. 31-20A,B) (72, 92). Many discoid menisci are incomplete and appear normal (70). The Wrisberg variant may show subtle anterior subluxation of the posterior horn, with high T2 signal interposed between the posterior horn and capsule (93).

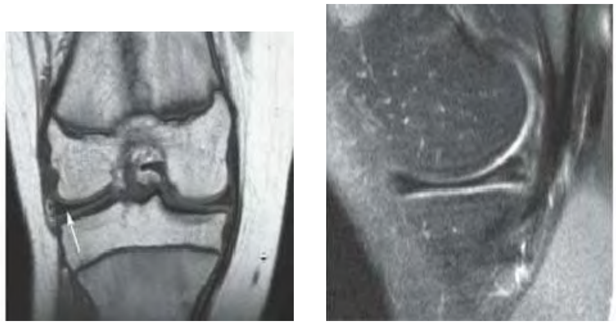


FIGURE 31-20. **A:** T1 weighted coronal image of a discoid lateral meniscus (*white arrow*) occupying the entire lateral tibial plateau. **B:** T2-weighted sagittal image showing continuity between the anterior and posterior horns (bow-tie sign).

Management. Asymptomatic discoid menisci are left alone. When noted incidentally during arthroscopy, most authors recommend no treatment as the knee may have adapted to discoid anatomy (70, 89). Symptomatic discoid menisci are best treated with preservation of meniscal tissue through arthroscopic saucerization as opposed to total meniscectomy.

The goal of arthroscopic saucerization of a discoid lateral meniscus is to create a stable and functioning meniscus that will provide adequate shock absorption without retearing (1, 409). Most authors recommend leaving a peripheral rim of tissue 6 to 8 mm intact as larger remnants are associated with higher retear rates (89, 91, 94). The indentation on the lateral femoral condyle and the size of the medial meniscus can also be used to guide the amount of resection (72). Saucerization is accomplished using a combination of low-profile arthroscopic baskets, shavers, and a meniscal knife (Fig. 31-21A–C). Cleavage tears within the zone of saucerization are excised. Following saucerization, peripheral rim stability is carefully assessed with a probe. Meniscal repair to capsule using standard technique is necessary for unstable areas of the saucerized meniscus.

Results. Total meniscectomy is still favored by a minority of authors who feel that residual discoid tissue is abnormal and will not function properly (95, 96). However, many studies report significantly high rates of osteoarthritis following total meniscectomy (68, 76, 90, 97, 98). One reported clinical and radiographic changes consistent with lateral compartment arthritis at 19.8 years follow-up in 10 of 17 children treated with total meniscectomy (90). Another analysis of 125 discoid menisci that underwent partial or total meniscectomy found that the partial meniscectomy group had better results at 5-year follow-up and long-term prognosis was related to the volume of meniscal tissue removed (97).

Recent studies show favorable short-term results for arthroscopic saucerization. Good to excellent clinical results and no degenerative changes on radiographs were reported in a series of 11 children treated with arthroscopic saucerization at 4.5 years follow-up (99). Another series of 27 consecutive children mean age 10.1 who underwent arthroscopic saucerization noted a 77%

FIGURE 31-21. **A:** Saucerization of a discoid lateral meniscus. **B:** Low-profile basket begins the saucerization. **C:** Final appearance after saucerization with a 6- to 8-mm rim of meniscal tissue preserved.

rate of peripheral instability requiring repair (88). At 3.1-year follow-up, excellent clinical results were noted in all patients. Long-term studies showing the efficacy of saucerization in preventing lateral compartment knee arthritis are lacking. Meniscal allograft transplantation may be an option for symptomatic patients who previously underwent total meniscectomy for discoid lateral meniscus. A recent report on meniscal allograft transplantation in 14 patients at mean follow-up of 4.8 years showed that Lysholm knee scores improved from 71.4 to 91.4, while six second-look arthroscopies revealed only one retear (100).

Tibial Spine Fracture

Introduction. The avulsion fracture of the tibial intercondylar eminence usually occurs in individuals between the ages of 8 to 14 with no predilection for gender. This is still a relatively rare injury accounting for about 2% of knee injuries or 3 per 100,000 children per year (101, 102). Classically, pediatric tibial spine fractures occurred from bicycling accidents, although they are also seen with pedestrian–motor vehicle accidents or sports injuries (103–105). Although far less common, tibial spine fracture can occur in adults and frequently involve lesions of the meniscus, capsule, or collateral ligaments because they are associated with higher energy mechanisms (106–111).

Fractures of the tibial spine are avulsion fractures of the ACL insertion and, in addition to disrupting ACL continuity, may, depending on the size of the fracture, involve the articular surface of the tibia (112, 113). Noyes has shown that as the subchondral bone fails, a elongation or stretch of the ACL occurs (112). This has led many authors to equate this injury to the midsubstance ACL rupture in adults (104, 105, 114–120).

Historically, treatment has evolved from closed treatment of all fractures to operative treatment of certain types. Garcia and Neer (121) reported 42 fractures of the tibial spine in patients ranging in age from 7 to 60 years with successful closed management in half their patients. Meyers and McKeever (103), recommended arthrotomy and open reduction for all displaced fractures, followed by cast immobilization with the knee in 20 degrees of flexion. Gronkvist et al. (117) reported late instability in 16 of 32 children with tibial spine fractures and recommended surgery for all displaced tibial spine fractures particularly in children over 10 years of age because of increased demand on the ACL–tibial spine complex. In a comparison of displaced tibial spine fractures, McLennan (122) reported on 10 patients treated with either closed reduction or arthroscopic reduction with or without internal fixation. After a second-look arthroscopy at 6 years, those treated with closed reduction had more knee laxity than those treated arthroscopically.

Modern treatment is based on fracture type. Fractures that are able to be reduced can be treated closed. Hinged and displaced fractures that do not reduce require open or arthroscopic reduction with internal fixation. A variety of treatment options have been reported, with the goal of obtaining a stable, pain-free knee. The prognosis for closed treatment of nondisplaced and reduced tibial spine fractures and for operative treatment of displaced fractures is good. Most series report healing with an excellent functional outcome despite some residual knee laxity (114, 115, 119, 120, 122–129). Potential complications include nonunion, malunion, arthrofibrosis, residual knee laxity, and growth disturbance (114, 115, 119, 120, 122–133).

Mechanism of Injury. The most common mechanism of tibial eminence fracture in children has been a fall from a bicycle, but with increased participation in youth sports at earlier ages and higher competitive levels, fractures resulting from sporting activities are being seen with increased frequency. The differential injury patterns of an ACL tear versus a tibial eminence fracture in the skeletally immature knee may be due to loading conditions, biomechanical properties, and anatomical differences (112, 113, 119, 134). The most common mechanism of tibial eminence fracture is forced valgus and external rotation of the tibia, although tibial spine avulsion fractures can also occur from hyperflexion, hyperextension, or tibial internal rotation. Slower loading rates, relative weakness of the incompletely ossified intercondylar eminence compared to the ligament midsubstance, greater elasticity of the ACL, and a wider intercondylar notch are believed to preferentially result in tibial spine avulsion fracture (112, 113, 119, 134).

In a biomechanical cadaver study, a fracture of the anterior tibial eminence was simulated by an oblique osteotomy beneath the eminence and traction on the ACL. In each specimen, the displaced fragment could be reduced into its bed by extension of the knee, likely affected by the lateral femoral condyle (135). In experimental models, midsubstance ACL injuries tend to occur under rapid loading rates, whereas tibial eminence avulsion fractures tend to occur under slower loading rates (112).

Additionally, intercondylar notch morphology may also influence injury patterns. In a retrospective study of 25 skeletally immature patients with tibial spine fractures compared to midsubstance ACL injuries, Kocher et al. (134) found narrower intercondylar notches in those patients sustaining midsubstance ACL injuries.

Physical Examination. As with patients with fractures around the knee joint, tibial spine fractures present with a painful swollen knee (hemarthrosis), limitation of knee motion, and difficulty with weight bearing. Evaluation should consist of a thorough history and physical examination. Sagittal plane laxity is often present, but the contralateral knee should be assessed for physiologic laxity. However, pain may make a thorough examination of the ligaments difficult. If possible, gentle stress testing should be performed to detect any tear of the MCL or LCL. Patients with a late malunion of a displaced tibial spine fracture may lack full extension because of a bony block. Patients with a late nonunion of a displaced tibial spine fracture may have increased knee laxity, a positive Lachman examination, and pivot-shift examination. A complete neurological and vascular examination should be performed.

Imaging. Standard roentgenograms and anteroposterior, lateral, and notch radiographic views are usually diagnostic. The fracture is best seen on the lateral and notch views (Fig. 31-22A,B). Radiographs should be carefully scrutinized as the avulsed fragment may be mostly nonossified cartilage with only a small, thin ossified portion visible on the lateral view. If necessary, computed tomographic (CT) scanning allows refined definition of the fracture anatomy.

MRI is not typically needed in the diagnosis and management of tibial eminence fractures in children. MRI may be helpful to confirm the diagnosis in cases with a very thin ossified portion of the avulsed fragment and to evaluate associated collateral ligament and chondral, meniscal, or physeal pathology; however these are uncommon. If distal pulses are abnormal or a dislocation is suspected, an arteriogram should be obtained.

Associated Injuries. Associated intra-articular injuries are relatively uncommon. Intercondylar eminence fractures may include or be associated with any combination of bone, chondral, meniscal, or ligamentous injuries (108). However, in a more recent series of 80 skeletally immature patients who underwent surgical fixation of tibial eminence fractures, Kocher et al. (136) found no associated chondral injuries

A

B

FIGURE 31-22. Anteroposterior **(A)** and lateral **(B)** radiographs of a displaced tibial spine fracture.

FIGURE 31-23. Meniscal tear in conjunction with a tibial spine fracture.

and associated meniscal tear in only 3.8% (3/80) of patients (Fig. 31-23). Associated collateral ligament injury or proximal ACL avulsion in conjunction with a tibial spine fracture has also been reported (137, 138).

Classification. The classification system of Meyers and McKeever (103, 139) is based on the degree of displacement and is widely used to classify fractures and to guide treatment (Fig. 31-24). Zaricznyj (140) later modified this classification to include a fourth type which are comminuted fractures of the tibial spine.

1. Type 1—minimal displacement of the tibial spine fragment from the rest of the proximal tibial epiphysis
2. Type 2—displacement of the anterior third to half of the avulsed fragment, which is lifted upward but remains hinged on its posterior border which is in contact with the proximal tibial epiphysis
3. Type 3—complete separation of the avulsed fragment from the proximal tibial epiphysis, usually associated with upward displacement and rotation

The interobserver reliability between type 1 and type 2/3 fractures is good; however differentiation between type 2 and 3 fractures may be difficult (134).

Surgical and Applied Anatomy. Between the condyles, the intercondylar eminence or spine is the insertion point for portions of the menisci and the anterior and posterior cruciate ligaments (PCLs). The tibial eminence is triangular and refers to the portion of the proximal tibia where there are two ridges of bone and cartilage. In the immature skeleton, the proximal surface of the eminence is covered entirely with cartilage. The ACL attaches distally to the anteromedial portion of the tibial intercondylar eminence (Fig. 31-25). The PCL inserts on the posterior aspect of the proximal tibia, distal to the joint line. Both menisci insert into the tibia in the region between the lateral and medial eminences, but there is no direct connection between the ACL and the menisci. In 12 patients with displaced tibial spine fractures that were unable to be reduced closed, Lowe et al. (141) reported that the anterior horn of the lateral meniscus and the ACL were attached simultaneously and pulling in different directions.

Meniscal or intermeniscal ligament entrapment under the displaced tibial eminence fragment can be common and may be a rationale for considering arthroscopic or open reduction in displaced tibial spine fractures (Fig. 31-26) (108, 136, 142, 143). Meniscal entrapment can prevent the anatomic reduction of the tibial spine fragment, which may result in increased anterior laxity or a block to extension and knee pain after the fracture has healed (104, 105, 117, 118, 122). Mah et al. (144) found medial meniscal entrapment preventing reduction in 8 of 10 children with type 3 fractures undergoing arthroscopic management. In a consecutive series of 80 patients who underwent surgical fixation of tibial eminence fractures which were not able to be reduced closed, Kocher et al. (136) found entrapment of the anterior horn medial meniscus (n = 36), intermeniscal ligament (n = 6), or anterior horn lateral meniscus (n = 1) in 26% of type 2 fractures and 65% of type 3

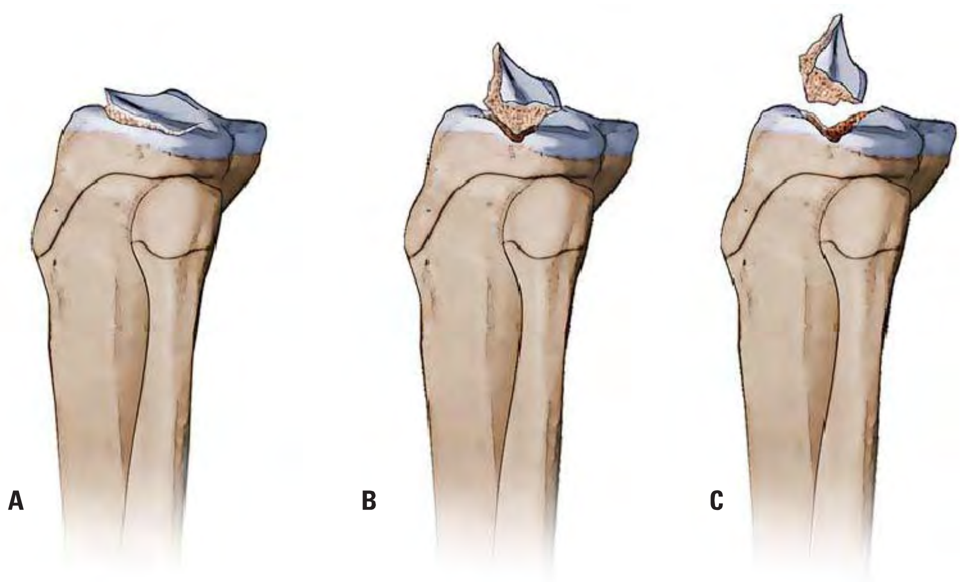


FIGURE 31-24. Meyers and McKeever classification system of tibial spine fractures in children. **A:** Type 1: Minimal displacement. **B:** Type 2: Displaced and hinged posteriorly. **C:** Type 3: complete displacement. (From Beaty JH, Kasser JR, eds. *Rockwood & Wilkins' fractures in children*. Philadelphia, PA: Lippincott Williams & Wilkins, 2010, with permission.)

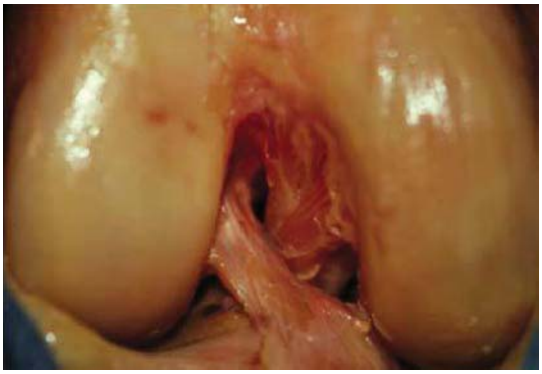


FIGURE 31-25. ACL insertion onto the anteromedial portion of the tibial eminence.

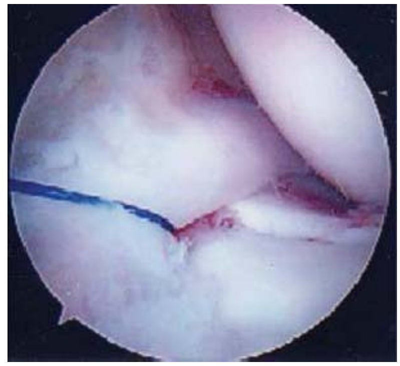


FIGURE 31-27. Use of a retention suture to retract the anterior horn of the medial meniscus.

fractures. The entrapped meniscus can typically be extracted with an arthroscopic probe and retracted with a retaining suture (Fig. 31-27).

Current Treatment Options. Current treatment options include cast immobilization (125, 127), closed reduction with immobilization (104, 120), open reduction with immobilization (127), open reduction with internal fixation (120, 128), arthroscopic reduction with immobilization (145), arthroscopic reduction with a variety of fixation methods, including: suture fixation (125, 130, 144, 146–148), wire (149), screw fixation (115, 125, 145, 150), anchor fixation (151), and bioabsorbable nail fixation (148). Many options still persist regarding the fixation of the fracture and have all been used with good success; most commonly, suture or screw fixation is used. Recent studies are equivocal in terms of strength of fixation, although suture fixation may be favored since it has the advantages of eliminating the risks of comminution of the fracture fragment, posterior neurovascular injury, and the need for hardware removal (147–149, 152).

The goal of treatment of a tibial spine avulsion is anatomic reduction; however, there is controversy regarding whether the tibial spine should be overreduced. Theoretically, overreduction may lead to excessive tightening of the ACL and limitation of knee motion (153). On the other hand, it is likely that permanent intersubstance stretching of the ACL occurs before the fracture (112), and therefore overreduction could be considered. Although further clinical or *in vitro* research is required, long-term evaluation of well-reduced tibial eminence fractures shows subtle increases in anteroposterior knee laxity without functional deficit (114, 117, 119, 124, 131, 141, 150).

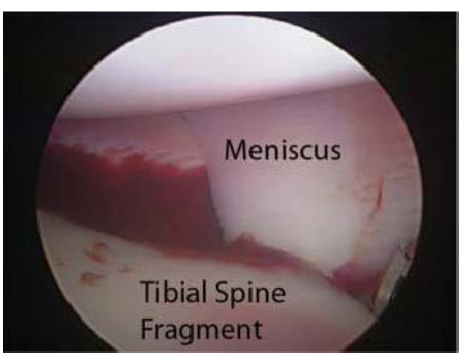


FIGURE 31-26. Anterior horn of the medial meniscus entrapped under tibial spine fragment.

Closed treatment is typically utilized for type 1 fractures and for type 2 or 3 fractures that are able to be successfully reduced closed. Aspiration of the hematoma is performed first and closed reduction is achieved by placement of the knee in full extension or 20 to 30 degrees of flexion. If the fracture fragment extends into the medial or lateral tibial plateaus, full extension may aid reduction through pressure applied by medial or lateral femoral condyle congruence, whereas fractures confined completely within the intercondylar notch may not reduce. Portions of the ACL are tight in all knee positions; therefore there may not be any one position that exists without traction being applied by the ACL which may prevent anatomic reduction. Radiographs are utilized to assess the adequacy of reduction.

Closed reduction can be successful for some type 2 fractures, but is frequently not successful for type 3 fractures. In their series, Kocher et al. (136) reported closed reduction in approximately 50% of type 2 fractures (26/49) with unsuccessful closed reduction in all 57 of the type 3 fractures. Arthroscopic or open reduction with internal fixation of type 2 and 3 tibial eminence fractures that do not reduce has been advocated due to the potential for clinical instability and loss of extension associated with closed reduction and immobilization, the ability to evaluate and treat injuries, and the opportunity for early mobilization (124, 142–144). For displaced type 2 and 3 fractures, Wiley and Baxter found a correlation between fracture displacement with measured knee laxity despite good patient function (5, 119).

Author's Preferred Treatment. The author's algorithm for the treatment of tibial spine fractures is shown in Figure 31-28.

Type 1 fractures are treated with cast immobilization after aspiration of the hematoma. A local anesthetic can be injected into the joint under sterile conditions if the patient is in severe

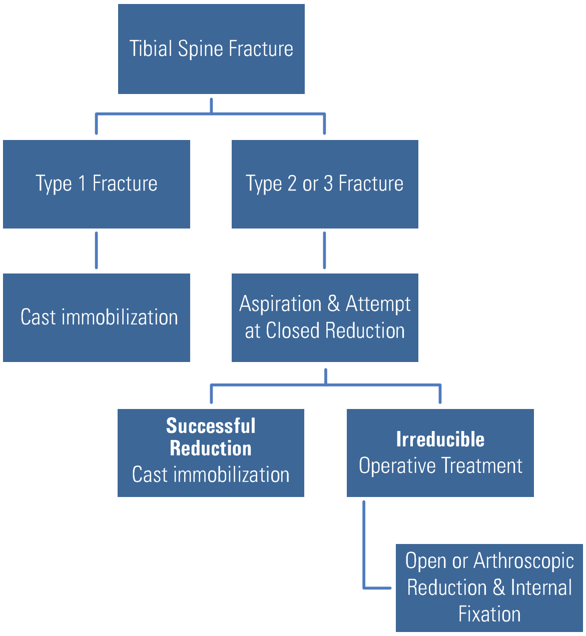


FIGURE 31-28. Author's preferred treatment algorithm for tibial spine fractures in children.

pain. A long-leg cast is applied in 0 to 20 degrees of flexion; we usually avoid a cylinder cast due to slippage and malleolar irritation. The patient and family are cautioned to elevate the leg to avoid swelling. Radiographs are repeated in 1 to 2 weeks to ensure that the fragment has not displaced and alignment is adequate. The cast is removed 6 weeks after injury. A hinged knee brace or a custom ACL brace is used, and physical therapy is initiated to regain motion and strength. Patients are typically allowed to return to sports at 3 months after injury if they demonstrate fracture healing and adequate motion and strength; the use of an ACL is encouraged for 6 months.

Type 2 fractures are initially treated with an attempt at closed reduction. The hematoma is aspirated and local anesthetic is injected into the knee under sterile conditions. Reduction is attempted at both full extension and 20 degrees of flexion. Radiographs are taken to assess reduction. If anatomic reduction is obtained, a long-leg cast is applied in the position of reduction and the protocol for type 1 fractures is followed. If the fracture does not reduce adequately or if the fracture displaces later, operative treatment is performed.

Type 3 fractures may be treated with attempted closed reduction; however this is usually unsuccessful and operative treatment is typically performed.

The author's preferred operative treatment is arthroscopic reduction and internal fixation. Open reduction through a medial parapatellar incision can also be performed per surgeon preference and/or experience or if arthroscopic visualization is difficult.

Arthroscopic Reduction and Internal Fixation with Epiphyseal Cannulated Screws. A standard arthroscopic operating room setup is utilized. The patient is placed supine and general anesthesia is typically used. A standard arthroscope can be used in most patients while a small (2.7 mm) arthroscope is used in younger children. An arthroscopic fluid pump is used at 35 torr in order to prevent excess bleeding and a tourniquet is routinely used. Standard anteromedial and anterolateral portals are established and accessory superomedial and superolateral portals are used for screw insertion. The hematoma is evacuated prior to the insertion of the arthroscope.

A thorough arthroscopic examination of the entire knee joint is conducted to evaluate for concomitant injuries. Frequently, we excise some portion of the anterior fat pad and ligamentum mucosum with an arthroscopic shaver for complete visualization of the intercondylar eminence fragment. An entrapped meniscus or intermeniscal ligament can be extracted with an arthroscopic probe and retracted with a retention suture inserted from outside in (Fig. 31-27). The base of the tibial eminence fragment is elevated (Fig. 31-29A) and the entire fracture bed debrided with an arthroscopic shaver and hand curette (Fig. 31-29B). Anatomic reduction is obtained using a probe, microfracture pick, or Kirschner wire with the knee in 30 to 90 degrees of flexion (Fig. 29-19C). Cannulated guide wires are placed through portals just off the superomedial and superolateral borders of the patella through the accessory portals at the base of the ACL. Fluoroscopic assistance is utilized to confirm anatomic reduction, guide correct wire orientation, and avoid the proximal tibial physis. A cannulated drill is used over the guide wires, and one or two screws are inserted based on the size of the tibial eminence fragment (Fig. 31-29D). Partially threaded 3.5-mm diameter screws (Fig. 31-29E) are used in children and 4.5-mm diameter screws are used in adolescents. The knee is evaluated through a full range of motion to ensure rigid fixation without fracture displacement and to ensure that there is no impingement of the screw heads in extension.

Postoperatively, patients are placed in a postoperative hinged knee brace and maintained touchdown weight bearing for 6 weeks postoperatively. Motion is restricted to 0 to 30 degrees for the first 2 weeks, 0 to 90 degrees for the next 2 weeks, and then full range of motion. The brace is kept locked in extension at night. Radiographs are obtained to evaluate the maintenance of reduction and fracture healing at 2 and 6 weeks (Fig. 31-30A–D). Cast immobilization in 20 to 30 degrees of flexion for 4 weeks postoperatively may be necessary in younger children unable to comply with protected weight-bearing and brace immobilization. Physical therapy is utilized to achieve motion, strength, and sport-specific training. Patients are typically allowed to return to sports at 12 to 16 weeks postoperatively depending on knee function and strength. Screws are not routinely removed. Functional ACL bracing is utilized if there is residual knee laxity.

Arthroscopic Reduction and Internal Fixation with Suture. Arthroscopic setup and examination is similar to the technique described for epiphyseal screw fixation. Accessory superomedial and superolateral portals typically are not used. A small incision is made just medial and distal to the tibial

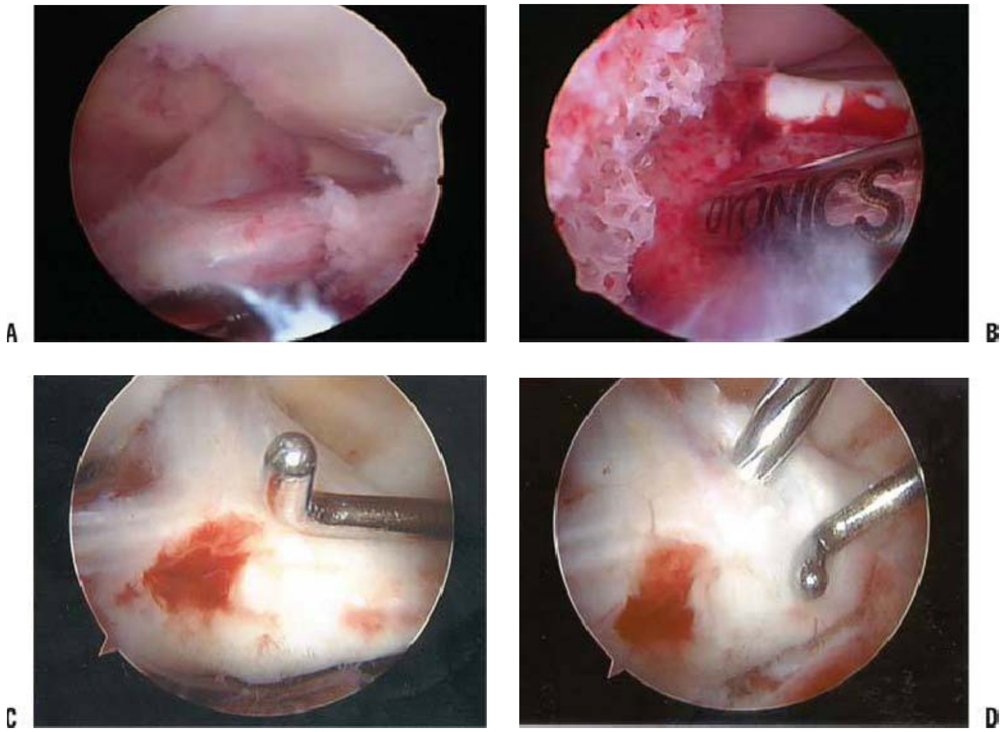


FIGURE 31-29. Arthroscopic reduction and insertion of cannulated screw internal fixation for a displaced tibial spine fracture. **A:** Tibial spine fragment. **B:** Elevation and debridement of the fracture bed. **C:** Reduction of tibial spine fragment. **D:** Drilling with a cannulated screw system. **E:** Insertion of a 3.5-mm screw.

FIGURE 31-30. Type 3 tibial spine fracture treated with arthroscopic reduction and screw fixation. **A:** Preoperative anteroposterior radiograph. **B:** Preoperative lateral radiograph. **C:** Postoperative anteroposterior radiograph. **D:** Postoperative lateral radiograph.

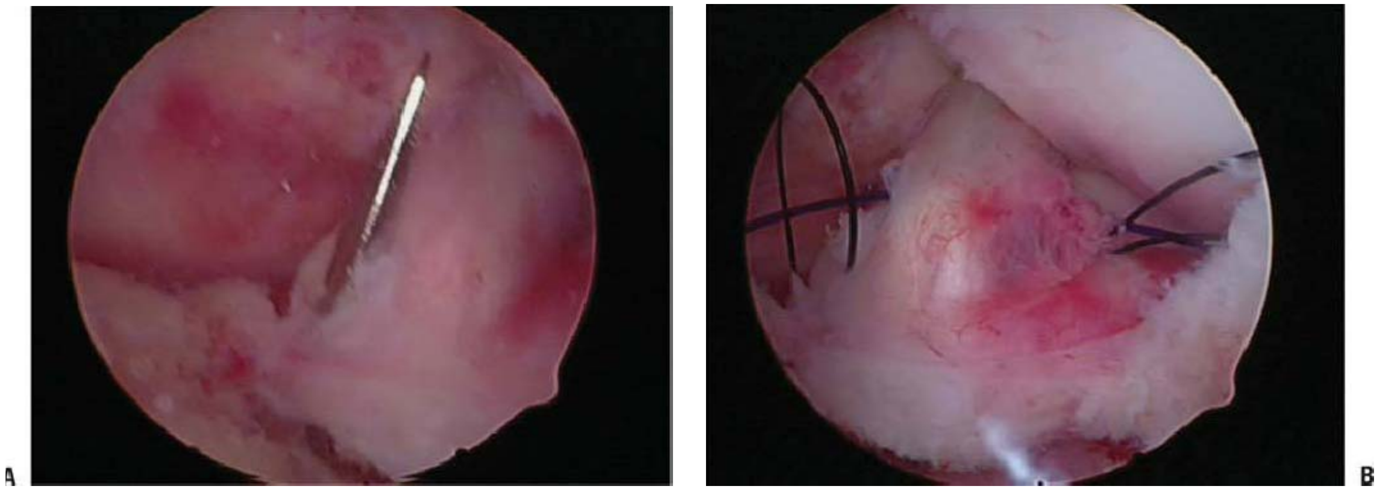


FIGURE 31-31. Treatment of a type 2 tibial spine fracture with arthroscopic reduction and suture fixation. **A:** Drilling of guide wire with ACL guide system. **B:** Hewson suture passers on either side of ACL and passage of absorbable sutures through the ACL. **C:** Final appearance after suture fixation.

tubercle as would be performed for an ACL reconstruction. After the fracture is debrided and slightly overreduced, a tibial ACL guide system with the tibial aimer set at 55 degrees is used to place two guide wires through the base of the ACL. The guide wires will traverse the tibial physis, but no cases of growth arrest after suture fixation have been reported. The guide wires are exchanged for Hewson suture passers and two heavy absorbable sutures are passed through the Hewson suture passers and the base of the ACL using a suture punch (Fig. 31-31A–C) or a suture lasso. The sutures are retrieved through the tibial tubercle incision, and the sutures are tied down onto the tibia. The procedure may be repeated for additional sutures. The postoperative protocol is the same (Fig. 31-32A–D).

Pearls and Pitfalls. When managing tibial eminence fractures with closed reduction, follow-up radiographs must be obtained at 1 and 2 weeks postinjury to verify maintenance of reduction. Late displacement and malunion can occur, par-

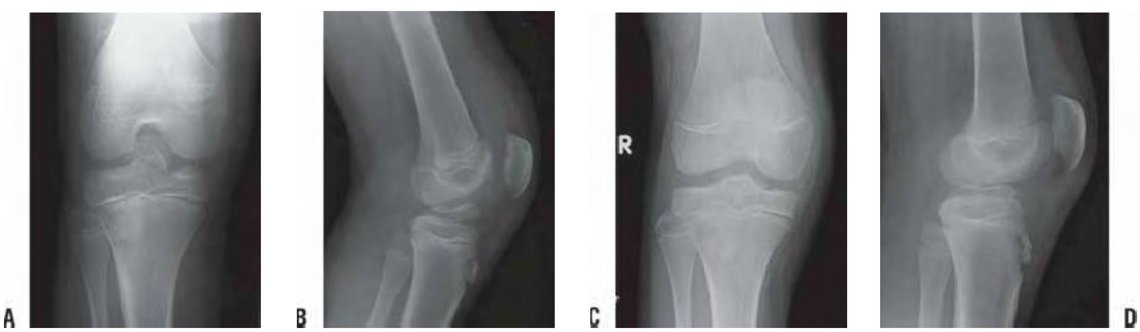


FIGURE 31-32. Type 2 tibial spine fracture treated with arthroscopic reduction and suture fixation. **A:** Preoperative anteroposterior radiograph. **B:** Preoperative lateral radiograph. **C:** Postoperative anteroposterior radiograph. **D:** Postoperative lateral radiograph.

ticularly for type 2 fractures. The injection of local anesthetic under sterile conditions can be helpful to minimize pain and allow for full knee extension in attempts at closed reduction.

During arthroscopic reduction and fixation of tibial spine fractures, visualization can be difficult unless the large hematoma is evacuated prior to the introduction of the arthroscope and bleeding from the fracture is controlled. Adequate inflow and outflow is essential for proper visualization, and we routinely use an arthroscopic pump and a tourniquet to achieve this. Careful attention should be paid to prepare the fracture bed to provide optimal conditions for bony healing. A slight overreduction of the fracture is attempted.

Epiphyseal cannulated screw fixation of small or comminuted tibial eminence fragments can fail due to inadequate bony purchase or further comminution; in these cases, suture fixation is preferred. If epiphyseal cannulated screw fixation is used, fluoroscopy is necessary to ensure that the screw does not traverse the proximal tibial physis, which may result in a proximal tibial physeal growth arrest (3, 129).

Early mobilization is useful to avoid arthrofibrosis which can occur with immobilization. However, in younger children, compliance with protected weight bearing and brace use can be problematic, and they must be casted.

Prognosis and Complications. The overall prognosis of tibial eminence fractures is good to excellent if satisfactory reduction is achieved as shown in most studies. However, some studies have found no difference in the outcome of displaced tibial spine fractures treated closed versus open or arthroscopic (112, 114, 117, 119, 120, 154). A majority of studies have found residual laxity of the knee after open or closed treatment for all tibial eminence fracture types up to 6 mm compared to the contralateral side (104, 105, 110, 114, 115, 120, 124–126, 154). Baxter and Wiley (114, 119) found excellent functional results without symptomatic instability in 17 pediatric knees with displaced tibial spine fractures, despite a positive Lachman examination in 51% of patients and increased measured mean knee laxity up to 3.5 mm. In a study of 12 pediatric knees undergoing open reduction and internal fixation, Smith found subluxation symptoms in two patients despite positive Lachman examinations in 87% of patients (131). Willis et al. (120) reported excellent clinical stability in all 50 children treated either closed or open despite a positive Lachman exam in 64% of patients and instrumented (KT-1000) knee laxity of 3.5 mm for type 2 fractures and 4.5 mm for type 3 fractures. Similarly, Janarv et al. (118) and Kocher et al. (124) also found excellent functional results despite persistent laxity in up to 80% of the patients even with an anatomic reduction.

This laxity is worse with type 3 injuries, pedestrian motor vehicle trauma, or with other associated ligament tears (104, 105, 110, 114, 115, 120, 124–126, 154). This increased laxity is likely due to intrasubstance stretching of the ACL during the injury. At the time of tibial spine fixation, the ACL often appears hemorrhagic within its sheath, but grossly intact and in continuity with the bony bed of tibia. However, ACL injury after previous tibial spine fracture is rare.

Poor results may occur after eminence fractures associated with unrecognized injuries to the collateral ligaments or physeal fracture (131, 155, 156). In addition, hardware across the proximal tibial physis may result in a growth disturbance with a recurvatum deformity (129). Malunion of type 2 and 3 fractures may cause bony impingement of the knee during full extension (126, 157). This can be corrected by excision of the malunited fragment and anatomic reinsertion of the ACL or, excision of the fragment and ACL reconstruction can be considered in adults and older adolescents.

Nonunion of type 2 and 3 tibial spine fractures treated closed can usually be managed by arthroscopic or open reduction with internal fixation with or without bone graft (133, 158, 159). Debridement of the fracture bed and the fracture fragment to bleeding bone is essential to optimize bony healing, and bone graft may be required in some cases. Excision of the fragment and ACL reconstruction can be considered in adults and older adolescents.

Arthrofibrosis, particularly, loss of extension, can occur after tibial spine fracture, even after anatomic reduction (132). This is thought to be due to the local increase in blood supply during healing, which leads to spine enlargement or arthrofibrosis which can cause a mechanical block to extension. Early range of motion and mobilization is essential to try to prevent loss of motion. Dynamic splinting and aggressive physical therapy can be employed during the first 3 months from fracture if stiffness is present. If stiffness persists after 3 months, a manipulation under anesthesia and a lysis of adhesions can be performed. Overly vigorous manipulation should be avoided in order to avert injury to the proximal tibial or distal femoral physis. A notchplasty can be performed if near skeletal maturity to regain extension.

Anterior Cruciate Ligament Injury. The knee is the most common site of injury in the skeletally immature athlete (160). The incidence of ACL tears appears to be on the rise. The treatment of these injuries is controversial. Nonoperative management can lead to functional instability and difficulty with cutting and pivoting sports. Additionally, the pathologic shear forces are associated with meniscal and chondral damage over time. ACL reconstruction in children and adolescents risks iatrogenic injury to the physis. This article reviews the historic perspective of ACL injuries in the young patient, clinical and diagnostic findings in children, treatment options, and results of treatment.

Historic Perspective. The ACL is the principal intra-articular stabilizer of the knee. As in adults, an ACL injury in a child or adolescent is usually a noncontact valgus injury. Prior to the 1980s, these injuries were thought to be rare in the pediatric athlete. Advances in diagnostic imaging and improved clinical acumen have allowed physicians to identify midsubstance ACL tears in patients with open physis (124, 136, 161).

The results of nonoperative management in children are consistently associated with poor outcomes (162, 163). Aichroth et al. reported on 23 children that were treated nonoperatively between 1980 and 1990. At final follow-up, meniscal tears were present in 15 knees, 3 osteochondral fractures occurred, and osteoarthritic changes developed in 10 knees. From 1980 to 1985, McCarroll followed 16 patients under the age of 14 with open physes and midsubstance tears of the ACL treated without reconstruction. Six patients underwent arthroscopy for meniscal tears. Only seven patients returned to sports, all experiencing recurrent episodes of giving way, effusions, and pain.

Attempts at primary repair of the ligament in children have resulted in poor outcomes (164). Engebretsen et al. presented eight adolescents who were followed 3 to 8 years after primary suture of a midsubstance rupture of the ACL. Only three patients had good function, and five were functionally unstable. Failure of primary repair has led to the development of a variety of procedures to stabilize the knee. Surgical options include transphyseal, partial transphyseal, and physeal-sparing reconstructions.

Tibial Spine Fractures and Partial Anterior Cruciate Ligament Tears. It is important to understand the different types of injuries that can occur in the skeletally immature patient. Partial ACL tears and avulsion fractures of the tibial spine are more common in the pediatric population (69). Excellent functional results have been reported following arthroscopic reduction and internal fixation of tibial spine fractures, although long-term follow-up does demonstrate some residual laxity, indicative of associated intrasubstance injury to the ACL (124). Many partial tears can be treated nonoperatively (165). Based on a prospective study of arthroscopically confirmed partial ACL tears, failure of nonreconstructive treatment has been associated with tears >50%, tears of the posterolateral bundle, older skeletal age, and presence of a pivot shift.

History and Physical Exam Findings. Important history questions include:

1. How did the injury occur?
   a. Was there contact with another athlete?
   b. Was there a fixed position of the foot and rotation or twisting movement?
2. Were you able to continue to compete?
3. Was there significant swelling directly after the injury?
4. Have there been previous injuries to the knee?

Our understanding of ACL tears in the setting of younger athletes has changed considerably. The tibial spine fracture was once thought to be the pediatric equivalent of an ACL tear. Midsubstance ACL ruptures are now diagnosed more frequently in pediatric athletes participating in cutting and contact sports. The typical presentation is a young athlete who has a decelerating, twisting injury. Approximately two-thirds of ACL injuries occur by noncontact mechanisms (166). The patient will often report a "pop" and the inability to return to the field. A large amount of swelling due to hemarthrosis is expected. The presentation is less dramatic in athletes who have had a prior partial tear of the ACL.

The findings on physical exam are dependent on the timing in relation to the injury. Directly after the injury, the stability of the knee can be tested on the sideline. The Lachman and pivot shift are positive before swelling and guarding occurs. When the patient presents for evaluation in the emergency department or clinic, the knee is typically swollen, compromising the ability to perform an accurate physical exam. Rates of ACL injury are reported between 10% and 65% in pediatric patients presenting with traumatic hemarthrosis of the knee; therefore, young athletes presenting with a hemarthrosis of the knee should raise suspicion for an ACL tear (167, 168). The differential diagnosis of hemarthrosis of the knee includes patellar dislocation, meniscal tear, osteochondral fracture, tibial spine fracture, and epiphyseal fracture of the femur or tibia.

A thorough examination of the knee must be performed to rule out concomitant injuries. Associated injuries include meniscal tears, posterior cruciate and/or collateral ligament tears, osteochondral fractures, and physeal fractures of the distal femur or proximal tibia. Given the higher prevalence of generalized ligamentous laxity in skeletally immature patients, a direct comparison to the contralateral knee should also be made. The Lachman and pivot-shift maneuvers are used to test for ACL insufficiency.

Imaging. Evaluation of the knee by MRI is an important part of the assessment, particularly in children. The MRI is useful to distinguish between partial tears, avulsions, and midsubstance tears of the ACL. Secondary findings in an acute injury include hemarthrosis and the presence of a bone contusion at the posterior lateral tibial plateau and anterior lateral femoral condyle. The MRI is useful for confirming the diagnosis of ACL tear, ruling out associated injuries, and assisting in preoperative planning (Fig. 31-33).

Indications and Timing of Surgery. Indications for ACL reconstruction in a skeletally immature patient include complete ACL tear with functional instability, partial ACL tear that has failed nonoperative treatment, and ACL injury with

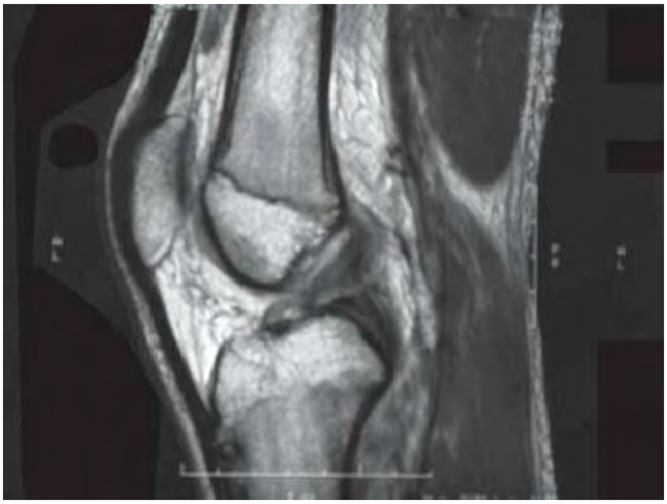


FIGURE 31-33. MRI demonstrating midsubstance ACL tear.

associated repairable meniscal or chondral injury. Due to higher rates of postoperative stiffness, acute ACL reconstruction is not recommended for isolated ACL tears (169). Surgery is typically delayed at least 3 weeks from the time of injury until adequate range of motion has been achieved and joint effusion minimized. Patients must be mature enough to participate in the extensive rehabilitation process following ACL reconstruction.

Treatment Options. The choice of surgical technique is dependent on the physiologic age of the patient and the amount of growth remaining. For prepubescent children, violation of the tibial and femoral physis presents a risk of significant growth disturbance that would require limb lengthening or osteotomy. Animal studies have demonstrated a risk of physeal arrest with transphyseal ACL reconstruction (170, 171). A number of growth disturbances following ACL reconstruction in this age group have been documented (172). Radiographs and developmental findings are used to determine the physiologic age. Referencing radiographs of the left wrist to the atlas of Greulich and Pyle (173) provides an efficient means to determine the skeletal age. The physiologic age is based on the Tanner staging system (174) (Fig. 31-34) (Table 31-2).

The prepubescent child (Tanner stage 1 or 2) with a midsubstance ACL tear presents a difficult problem. Because of the large amount of growth remaining, the consequences of iatrogenic physeal arrest are severe. Unfortunately, activity modification such as refraining from cutting sports is difficult in this age group, and nonreconstructive treatment has been associated with meniscal and chondral injury (175–178).

Surgical techniques include physeal-sparing, transphyseal, and partial transphyseal reconstructions. In theory, the extra-articular reconstruction provides a method to restore stability and avoid risk of growth disturbance. At our institution, we use a modification of the MacIntosh ACL reconstruction to perform a physeal-sparing reconstruction with an extra-articular and intra-articular component that is described in detail later in the text.

The results of 44 patients with a mean follow-up of 5.3 years after this combined intra-articular and extra-articular ACL reconstruction were examined. There were two failures at 4.7 and 8.3 years and no episodes of growth disturbance. The mean International Knee Documentation Committee (IKDC) subjective knee score and the Lysholm score were 96.7 and 95.7, respectively (179).

Surgical treatment with conventional transphyseal tunnels has been described (180, 181). Liddle et al. described the results of 17 patients treated with transphyseal ACL reconstruction with four strand hamstring autograft. Eight Tanner stage 1 and nine Tanner stage 2 patients were followed for a mean of 44 months. The mean Lysholm score at follow-up was 97.5. There was one failure due to an additional injury. One child was noted to have a 5-degree valgus deformity.

An alternative physeal-sparing technique using epiphyseal tunnels has been described by Anderson (181) (Fig. 31-35). A transepiphyseal technique using fluoroscopy was performed in 12 patients. The mean IKDC subjective score was 96.4 at 4.1 years with no graft failures or growth arrests noted.

For adolescents with significant growth remaining (Tanner 3 and 4), we perform transphyseal ACL reconstruction with

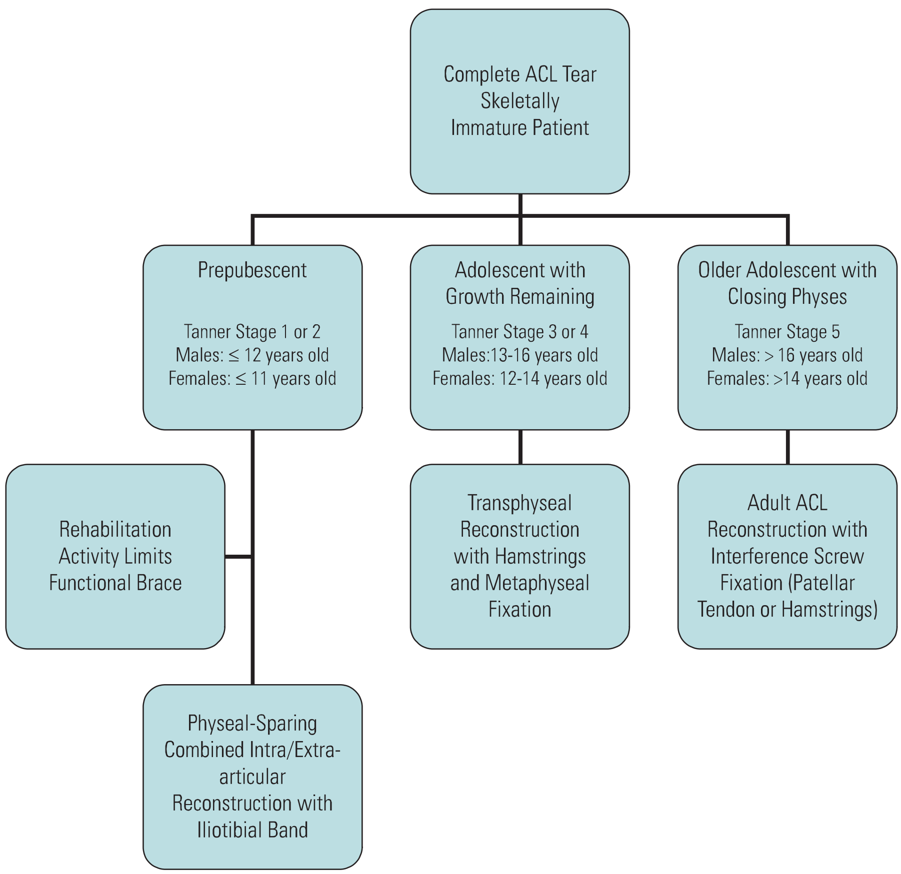


FIGURE 31-34. Algorithm for management of complete ACL injuries in skeletally immature patients.

TABLE 31-2 Tanner Staging Classification of Secondary Sexual Characteristics

Tanner Stage		Male	Female
Stage 1 (prepubertal)	Growth	5–6 cm/yr	5–6 cm/yr
	Development	Testes <4 mL or <2.5 cm No pubic hair	No breast development No pubic hair
Stage 2	Growth	5–6 cm/yr	7–8 cm/yr
	Development	Testes 4 mL or 2.5–3.2 cm Minimal pubic hair at base of penis	Breast buds Minimal pubic hair on labia
Stage 3	Growth	7–8 cm/yr	8 cm/yr
	Development	Testes 12 mL or 3.6 cm Pubic hair over pubis Voice changes Muscle mass increases	Elevation of breast; areolae enlarge Pubic hair of mons pubis Axillary hair Acne
Stage 4	Growth	10 cm/yr	7 cm/yr
	Development	Testes 4.1–4.5 cm Pubic hair as adult Axillary hair Acne	Areolae enlarge Pubic hair as adult
Stage 5	Growth	No growth	No growth
	Development	Testes as adult Pubic hair as adult Facial hair as adult Mature physique	Adult breast contour Pubic hair as adult
Other		Peak height velocity: 13.5 yr	Adrenarche: 6–8 yr Menarche: 12.7 yr Peak height velocity: 11.5 y

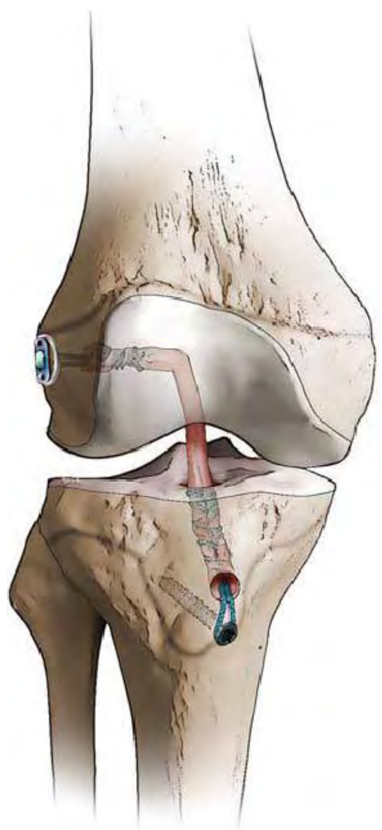


FIGURE 31-35. Physeal-sparing intra-articular ACL reconstruction with epiphyseal tunnels.

autogenous hamstrings. Sixty-one ACL reconstructions were reviewed in skeletally immature pubescent adolescents. Two patients underwent revision ACL reconstruction because of graft failure at 14 and 21 months postoperatively. For the remaining 59 knees, the mean IKDC subjective knee score was 89.5 and the mean Lysholm knee score was 91.2 (182).

Surgical Technique

Anterior Cruciate Ligament Reconstruction with the Iliotibial Band. For prepubescent children, Tanner 1 or 2, a physeal-sparing reconstruction is recommended (179). Skeletal age is usually <14 in males and <13 in females. The patient is placed supine on the operating room table. Examination under anesthesia is performed to confirm Tanner staging and verify ACL insufficiency. The operative extremity is prepped and draped from the level of the foot to the level of a tourniquet placed at the thigh. It is important to place the tourniquet as proximally as possible in case a counterincision is necessary to assist in harvesting the iliotibial (IT) band proximally. The insertion of the IT band on the tibia is palpated at Gerdy tubercle. The incision runs obliquely from the lateral joint line to the superior border of the IT band. The tourniquet is not routinely inflated in order to prevent tethering of the IT band. The incision is then made, and self-retaining retractors are placed. Dissection is carried down to the level of the IT band. In young, thin patients, the IT band may be quite superficial. The anterior and posterior borders of the IT band are defined (Fig. 31-36A). Posteriorly, the IT band blends with the lateral

Text continued on page 1627

Anterior Cruciate Ligament Reconstruction with the Iliotibial Band (Figs. 31-36 to 31-37)

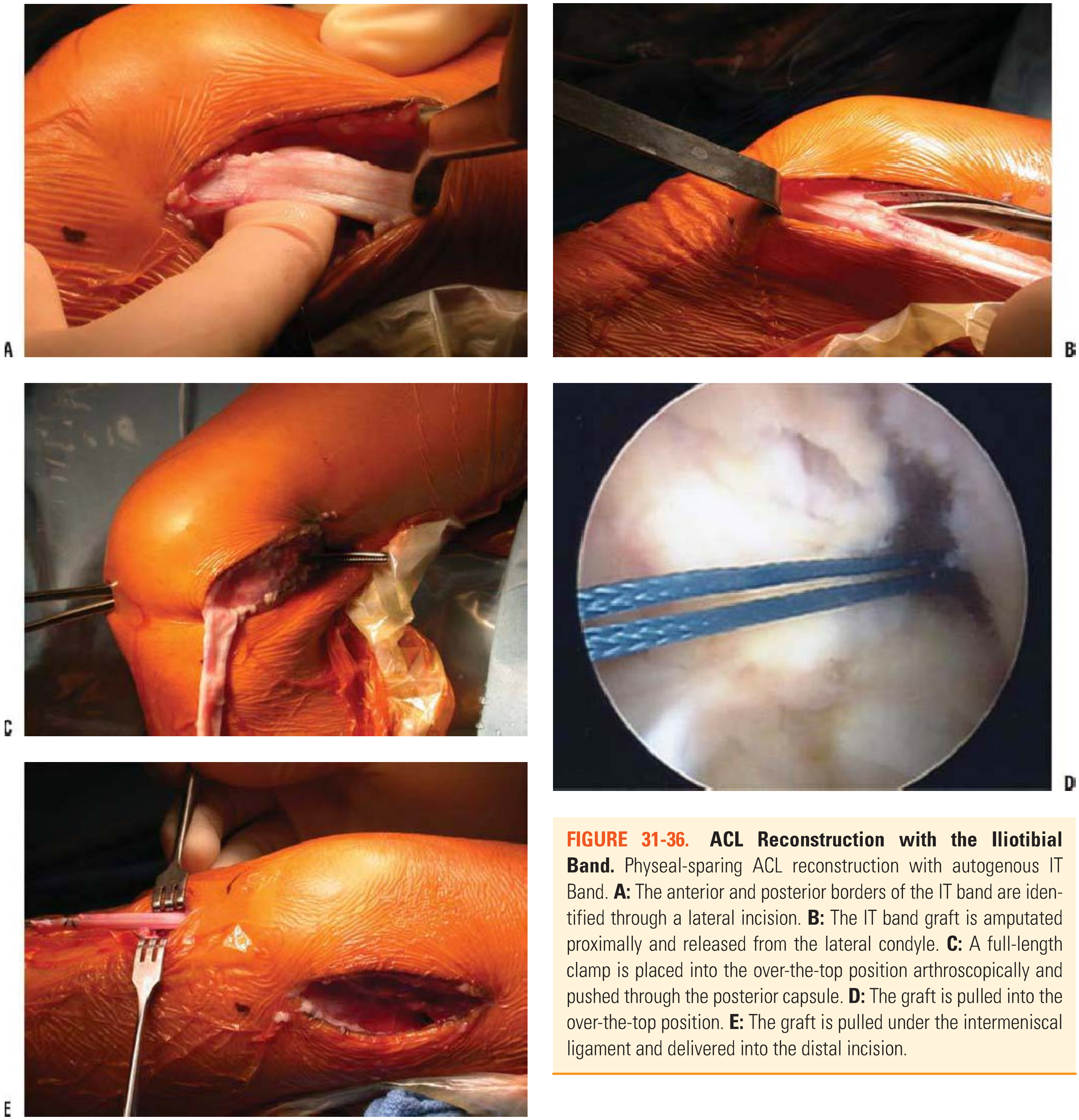


FIGURE 31-36. ACL Reconstruction with the Iliotibial Band. Physeal-sparing ACL reconstruction with autogenous IT Band. **A:** The anterior and posterior borders of the IT band are identified through a lateral incision. **B:** The IT band graft is amputated proximally and released from the lateral condyle. **C:** A full-length clamp is placed into the over-the-top position arthroscopically and pushed through the posterior capsule. **D:** The graft is pulled into the over-the-top position. **E:** The graft is pulled under the intermeniscal ligament and delivered into the distal incision.

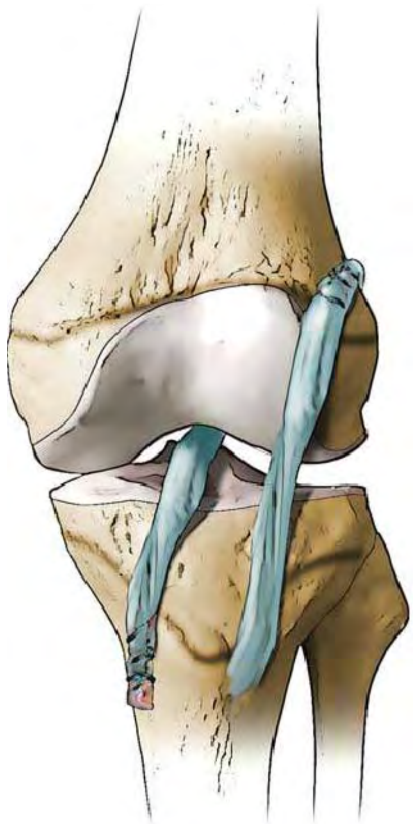


FIGURE 31-37. Combined intra-articular/extra-articular physeal-sparing ACL reconstruction.

hamstrings, and harvesting too far posteriorly risks injury to the common peroneal nerve. A Cobb elevator is used to dissect the subcutaneous tissue away from the IT band along its course.

A no. 15 blade is used to make an incision at the anterior border of the IT band, starting 2 cm above Gerdy tubercle. A Kelly clamp is placed in this incision and pushed posteriorly along the deep surface of the IT band until the intramuscular septum is palpated. The clamp is then passed through the posterior border of the IT band just above the intramuscular septum. The clamp is then spread in line with the fibers of the tendon to start the posterior split in the tendon. Adhesions to the underlying tissue are often present and should be released. A meniscotome is used to extend the two parallel incisions proximally in line with the fibers of the tendon. The incisions should be continued as proximally as possible to maximize graft length. The angled meniscotome is then used to amputate the graft proximally (Fig. 31-36B). If there is difficulty releasing the graft with the curved meniscotome, a counterincision is made near the tourniquet.

The graft is tubularized and a whip stitch is placed at its proximal end with no. 5 Ethibond. The tendon is separated from the underlying joint capsule and lateral femoral condyle. The capsule in this area is thin, but an effort should be made to maintain the integrity of the capsule to prevent fluid extravasation during later arthroscopy. The graft is left attached to Gerdy tubercle distally and tucked under the skin for the arthroscopic portion of the case.

The leg is elevated and the tourniquet is inflated. The anterolateral viewing portal is established, and the arthroscope is inserted. An anteromedial portal is established under arthroscopic visualization. Diagnostic arthroscopy is performed, and any associated injuries are treated. A limited notchplasty is performed to aid in visualization and identification of the over-the-top position on the distal femur. Excessive dissection should be avoided to prevent injury to the perichondral ring of the distal femoral physis during notchplasty. The distance from the femoral footprint of the ACL to the physis is typically 3 to 5 mm. Because there is no femoral tunnel, retaining a portion of the native ACL can help to maintain the position of the graft in the over-the-top position by acting as a sling.

Now it is necessary to pass the IT band into the over-the-top position. A full-length clamp is placed through the anteromedial portal and into the over-the-top position. The clamp is then passed through the joint capsule along the posterolateral femur and into the site of the IT band harvest (Fig. 31-36C). The clamp is then spread open to dilate a passage for the graft. The Ethibond sutures at the free end of the graft are placed into the clamp, and the graft is passed into the knee joint (Fig. 31-36D).

The distal insertion of the graft is then prepared. An additional 3-cm incision is made on the anteromedial aspect of the proximal tibial. The incision must be distal to the tibial physis and medial to the tibial tubercle apophysis. Fluoroscopy can be used to confirm the location of the metaphyseal incision. Dissection is carried down to the periosteum. Under arthroscopic visualization, a rasp is then passed along the periosteum and into the knee joint proximally. The rasp must enter the joint underneath the intermeniscal ligament. Using the rasp, a groove is then made in the tibial epiphysis to facilitate graft passage under the ligament and to translate the graft posteriorly in order to achieve a more anatomic position of the graft. The graft pulled is under the intermeniscal ligament with a clamp and delivered into the distal incision (Fig. 31-36E).

Graft fixation proceeds from proximal to distal. With the knee in 90 degrees of flexion, tension is applied to the graft, and the proximal aspect of the graft is sutured to the periosteum of lateral femoral condyle. This forms the extra-articular component of the reconstruction and helps to limit rotation of the tibia. Distally, an incision is made in the periosteum of the tibia. Periosteal flaps are raised medially and laterally in order to accommodate the diameter of the graft. Care is taken to avoid excessive dissection laterally as this risks injury to the tibial tubercle apophysis. Again, the location of this groove can be verified using fluoroscopy. A trough is then created in the tibia using a burr. With the knee in 20 to 30 degrees of flexion, distal tension is applied to the graft. No. 5 Ethibond sutures are then placed through the medial periosteum, the graft, and then the lateral periosteum. At least three sutures should be placed proximally in the femur and distally in the

FIGURE 31-38. MRI s/p physeal-sparing ACL reconstruction.

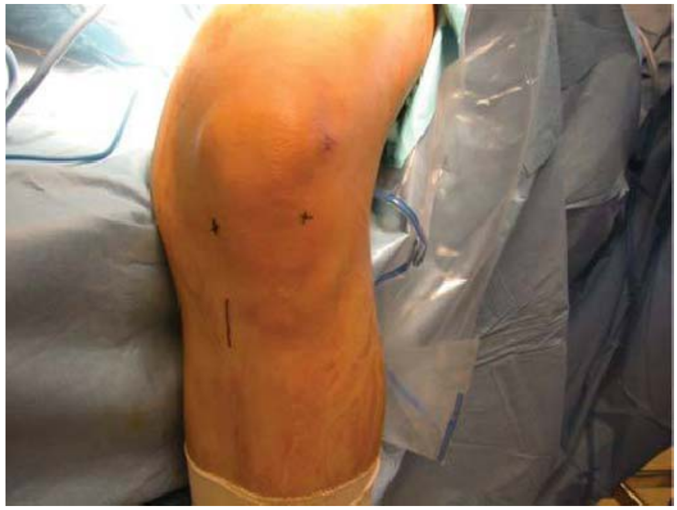


FIGURE 31-39. Incisions for transphyseal ACL reconstruction with hamstrings.

tibia (Fig. 31-37). Tibial fixation may be supplemented with a post if necessary. Wounds are closed in layered fashion with absorbable suture. A sterile dressing and a cryotherapy unit are applied to the knee. A hinged knee brace is placed over the cryotherapy unit.

Postoperative range of motion is limited from 0 to 30 degrees for 2 weeks. A continuous passive motion unit is used for 2 weeks. Flexion is gradually increased to 90 degrees from weeks 2 to 6, after which motion is unrestricted. Touchdown weight bearing in full extension is recommended for 6 weeks postoperatively. The patient may be placed into a simple hinge brace at 6 weeks. Jogging is instituted at 3 months with return to cutting sports at 6 months pending clearance. An ACL brace is worn for high-risk activities for the first 1 to 2 years after return to sport. Radiographs are obtained at 6 months to evaluate for growth arrest (Fig. 31-38). Clinical follow-up with assessment for leg-length discrepancy or angular deformity is done yearly for at least 2 years. Additional radiographs are obtained as indicated by clinical exam.

Modified Transphyseal Anterior Cruciate Ligament Reconstruction with Hamstrings Autograft. The modified ACL reconstruction is indicated in the adolescent with significant growth remaining. These patients are typically Tanner stage 3 with pigmented axilla and pubic hair for boys. The females at this stage are typically postmenarchal. For males, the bone age is from 14 years until skeletal maturity and for females 13 years until skeletal maturity. Adolescents nearing skeletal maturity (Tanner 5) can be treated as adults with conventional tunnels and bone plugs if desired.

Factors associated with growth arrest in transphyseal ACL reconstruction include placing hardware across the lateral distal femoral physis or tibial tubercle apophysis, bone plugs across the physis, large tunnels, and vigorous over-the-top dissection.

The patient is placed supine on the operating table. It can be useful to palpate the insertion of the hamstrings prior to prepping and draping the patient. A tourniquet is placed at the proximal thigh. The exam under anesthesia is performed. If the pivot shift is present, the hamstrings autograft is harvested initially. If the diagnosis is in doubt, diagnostic arthroscopy is performed first.

The leg is prepped and draped sterilely. The limb is exsanguinated and tourniquet inflated. The leg is placed in the figure of four position. Typically, the superior border of the medial hamstrings is 3 cm below the joint line. The superior and inferior borders of the hamstrings are marked 3 cm medial to the tibial tubercle (Fig. 31-39). A vertical incision is made, and dissection is carried down to the Sartorius fascia. Blunt dissection is used to separate the Sartorius fascia from the subcutaneous tissue. The gracilis and semitendinosus should be palpated just below the Sartorius fascia. The thin layer of Sartorius fascia is carefully incised. A right-angled clamp or Metzenbaum scissors are used to define the superior and inferior borders of the hamstrings tendons. The gracilis and semitendinosus are then isolated individually with vessel loops placed around each tendon (Fig. 31-40). A clamp may be passed deep to the tendons in order to apply distal traction on the tendons which will help free the tendons from the Sartorius fascia. The gracilis tendon is then dissected distally and released from its insertion on the tibia. Care should be taken to maintain a pick-up or clamp on the tendon to prevent proximal retraction after release. A whip-stitch is then placed in the free end of the tendon with no. 5 Ethibond suture. This is repeated for the semitendinosus (Fig. 31-41). Distal traction is again applied to the tendons individually and any adhesions are released. Special attention should be paid to adhesions from the semitendinosus to the medial head of the gastrocnemius. Such adhesions can be quite fibrous and risk diverting the tendon stripper which will

Text continued on page 1631

Modified Transphyseal Anterior Cruciate Ligament Reconstruction with Hamstrings Autograft (Figs. 31-40 to 31-50)

FIGURE 31-40. **Modified Transphyseal Anterior Cruciate Ligament Reconstruction with Hamstrings Autograft.** Gracilis and semitendinosus tendons identified.

FIGURE 31-41. Tendons isolated, released distally and whip stitched.

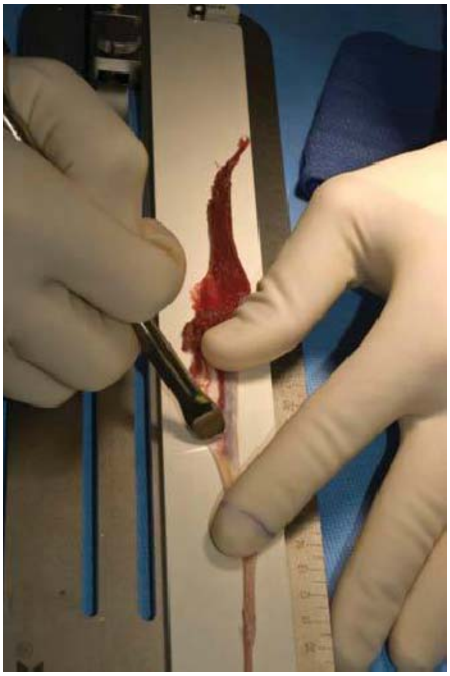


FIGURE 31-42. Graft preparation with removal of excess muscle.

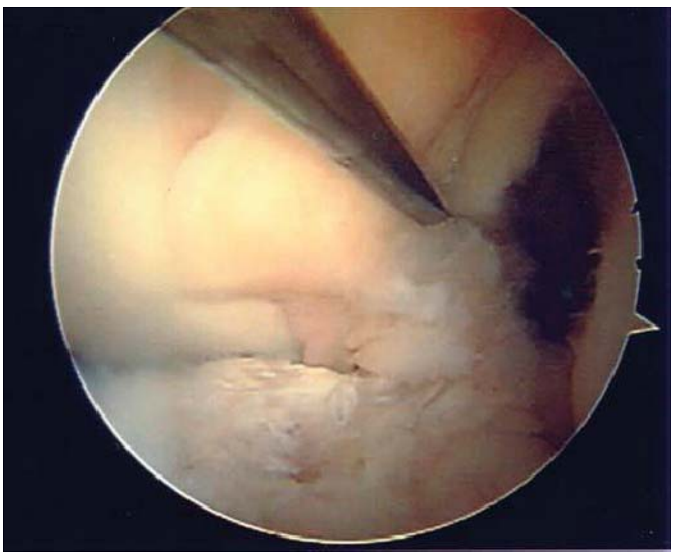


FIGURE 31-43. The ACL stump is debrided and the over-the-top position is visualized.

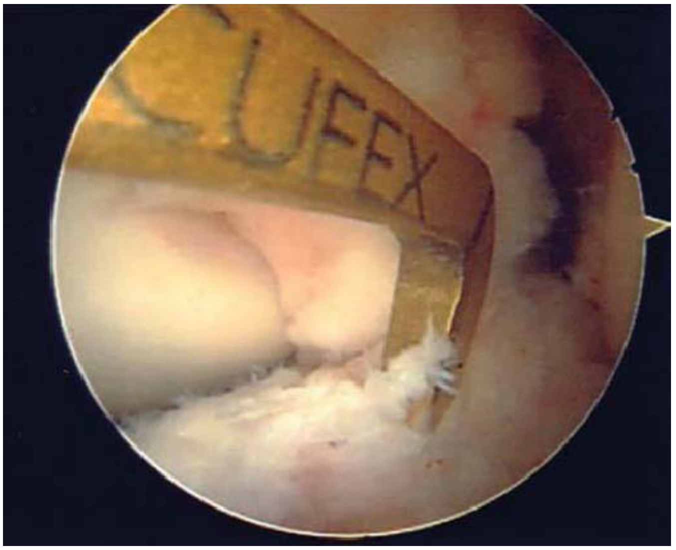


FIGURE 31-44. A tibial guide is used to make the tibial tunnel.

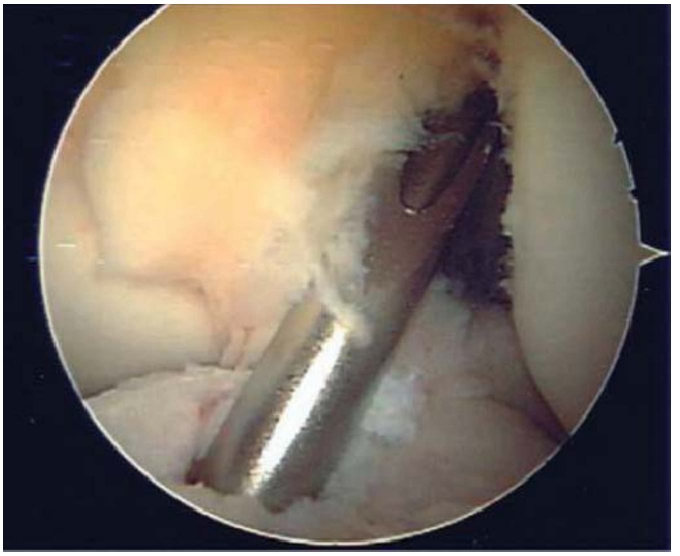


FIGURE 31-45. A transtibial femoral offset guide is used to make the femoral tunnel. Alternatively, the femoral tunnel can be drilled through a medial portal.

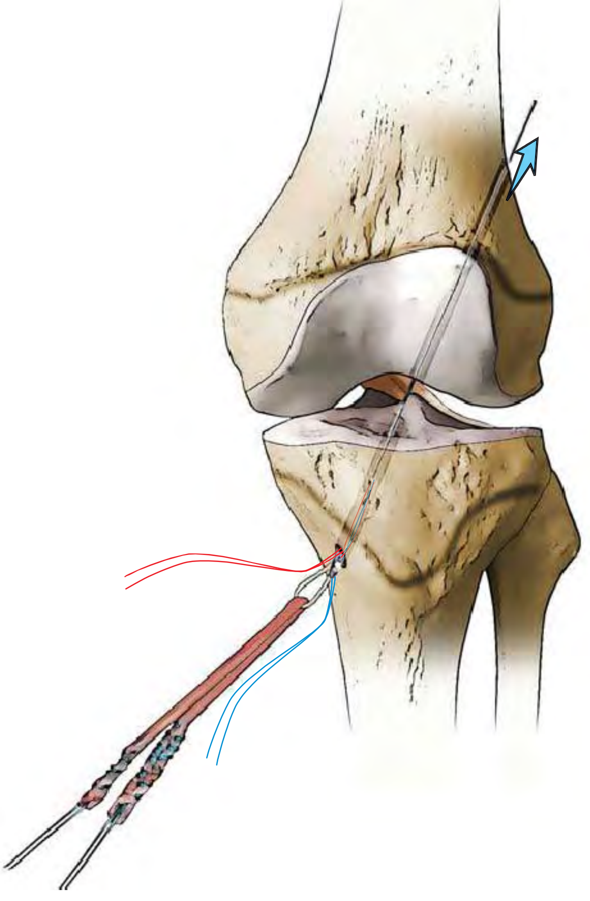


FIGURE 31-46. The leading sutures of the endobutton are passed through the tibial and femoral tunnels.

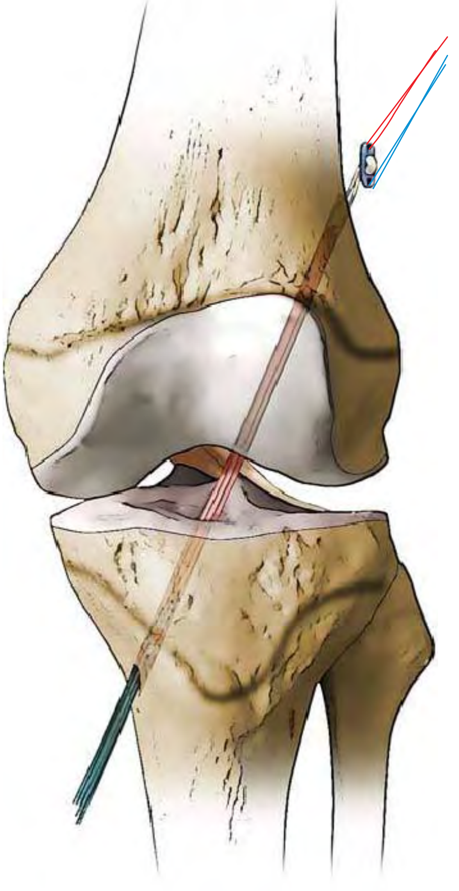


FIGURE 31-47. The graft is passed, and the endobutton is flipped on the femoral cortex.

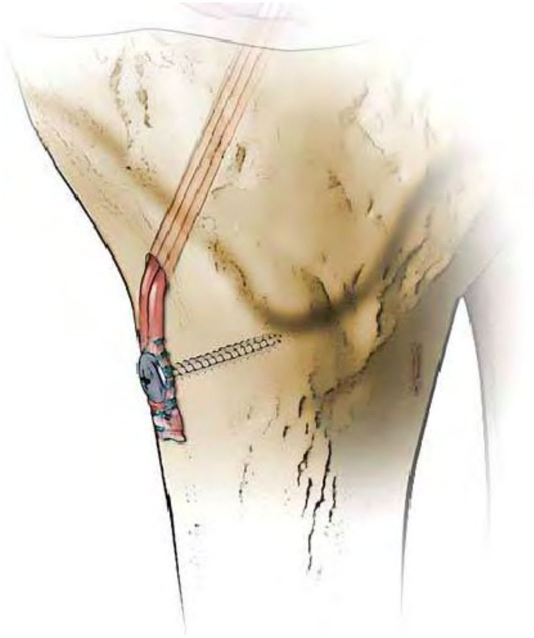


FIGURE 31-48. Alternative tibial fixation with a post.

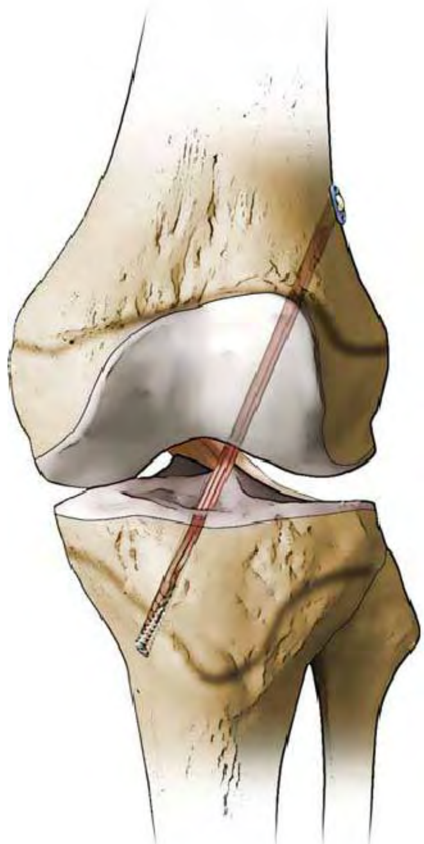


FIGURE 31-49. The graft is fixed distally using an absorbable interference screw.

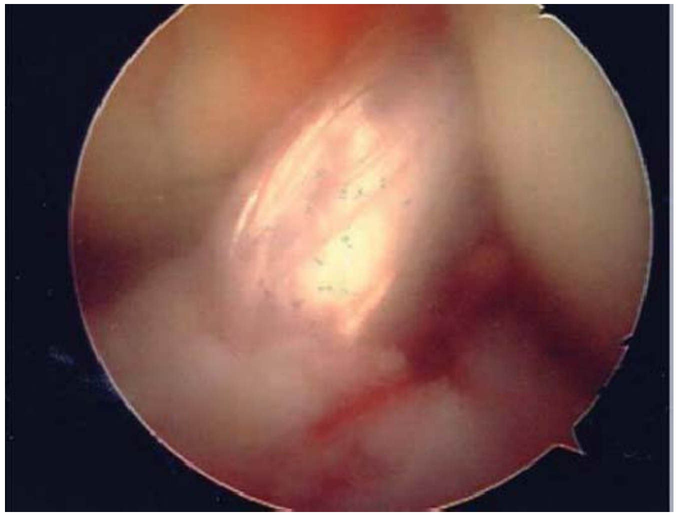


FIGURE 31-50. ACL reconstruction.

result in premature graft amputation. The tendons are then harvested with a closed-loop tendon stripper and taken to the back table for preparation (Fig. 31-42).

Excess muscle is removed from the proximal ends of the tendons and whipstitches are placed. The tendons are then folded over a closed-loop Endobutton to form a quadrupled graft, placed under tension, and covered with a moist sponge. Graft diameter is measured at this time.

A diagnostic arthroscopy is performed using standard anteromedial and anterolateral portals. The menisci are carefully evaluated as there should be a low threshold for meniscal repair in this patient population. The soft tissue is cleared from the notch with a shaver. A limited notchplasty is performed if necessary for visualization or to avoid impingement (Fig. 31-43). The tibial guide is set at 55 degrees. With the leg hanging over the side of the table, the tibial guide is placed through the anterior medial portal. In order to avoid the tibial tubercle apophysis, the guide wire entry point on the tibia should be medial through the same incision used to harvest the hamstrings. The guide is typically at 20 degrees to the knee in the sagittal plane. The entrance for the tip of the guide wire is 5 mm in front of the PCL. It should be in line with the posterior portion of the anterior horn of the lateral meniscus and slightly more medial than lateral in the joint. The tibial tunnel is then drilled based on the width of the harvested graft (Figs. 31-44 and 31-45).

A bump is placed under the thigh to keep the knee at 90 degrees. A long guide pin is placed through the tibial tunnel to the over-the-top position on the femur. The pin is drilled through the proximal femur. The 4.5-mm endobutton drill is then advanced through the lateral cortex. The pin and drill are then removed from the knee. A depth gauge is used to determine the total tunnel length. The depth of the femoral tunnel is determined by subtracting the length of the endobutton loop from the total length. An additional 8 mm is required to flip the Endobutton. For example, if the total tunnel length is 60 mm and a standard Endobutton length of 15 mm is used, the necessary femoral tunnel depth would be 45 mm.

An additional 8 mm is added in order to flip the Endobutton, making the total drill depth 53 mm.

The pin is then placed back into the femoral tunnel transtibially. The acorn drill corresponding to the width of the graft is then used to ream to the calculated femoral depth. Excess bone should be removed with the shaver. If there is any question regarding the adequacy of the tunnels, the arthroscope can be placed through the tibial tunnel to visualize the femoral tunnel.

A looped no. 5 Ethibond suture is then placed at the end of the guide pin and pulled through the knee for use as a passing suture. The loop should remain visible through the distal incision with the two free ends exiting proximally through the femoral tunnel and skin. The two passing Endobutton sutures of the endobutton are then passed through the looped no. 5 Ethibond, and the loop is pulled through the tibial and femoral tunnels (Fig. 31-46). The two Endobutton sutures are then used to pass the graft through the tibial and femoral tunnels. The Endobutton is flipped on the femoral cortex and fixation confirmed by pulling distally on the graft (Fig. 31-47). The arthroscope is placed back into the knee to evaluate the ACL graft. Additional notchplasty may be performed if there is evidence of graft impingement in extension. The length of the tibial tunnel should also be evaluated. The tibial physis can be visualized by placing the scope into the tibial tunnel. A gross measurement of the metaphyseal portion of the tibial tunnel can be made by bringing the scope up to the level of the physis and measuring the length of the scope within the tunnel. If this distance is <25 mm, an interference screw should not be used for fixation. A post, spike washer, or staple may be used alternatively (Fig. 31-48).

If the tibial tunnel is of sufficient length, the graft is then fixed on the tibial side with a bioabsorbable interference crew (Fig. 31-49). Tension is applied to the graft at all times, and the knee is held in 20 to 30 degrees of flexion. Generally, the screw size matches the tibial tunnel diameter. The Endobutton sutures are cut and removed from the femur. The wounds are irrigated and closed in a layered fashion with absorbable suture. Sterile dressings, a cryotherapy unit, and a hinged knee brace are applied. Range of motion is limited from 0 to 90 degrees for the first 6 weeks postoperatively. Touchdown weight bearing with the knee in extension is maintained for 2 weeks. Home CPM is used for 2 weeks starting at 0 to 30 degrees immediately after surgery and advancing up to 90 degrees. After 6 weeks, rehabilitation and clinical follow-up are identical to physeal-sparing reconstruction (Figs. 31-50 to 31-52).

FIGURE 31-51. One month postoperative x-ray.

OSTEOCHONDRITIS DISSECANS

Introduction. OCD is an acquired, yet potentially reversible lesion of the subchondral bone as well as of the overlying cartilage. Originally described by Paget (183) (Paget) as "quiet necrosis," the condition remains an enigma as to etiology, pathogenesis, and in some cases the prognosis, and treatment. Juvenile osteochondritis dissecans (JOCD) has a much better prognosis than its adult counterpart and will be the focus of this discussion. It is a lesion of the articular cartilage and subchondral bone before closure of the growth plate and was originally described by Roberts (184). OCD affect males more commonly than females (between 2:1 and 3:1). An increase in sport participation by females and younger children has increased the prevalence of OCD in these populations.

Etiology. The exact etiology of this unusual condition is unknown; it is probably multifactorial (184–190). Proposed

FIGURE 31-52. Four years postoperative x-ray.

causative factors include mechanics (trauma), ischemia, and genetic factors (185, 190–194).

Langenskiöld (195) demonstrated that removal and replacement of a portion of articular cartilage of the distal femur in rabbits produced a lesion similar to OCD in humans. Biomechanical studies using finite-element analysis have demonstrated that stresses are greatest in the subchondral bone of the medial femoral condyle and are maximal at 60 degrees of flexion (196).

Mechanisms that have been shown to cause osteochondral fractures are similar to forces responsible for causing JOCD and include impaction of a tibial spine, direct impaction forces resulting in a "bone bruise," joint compression forces, and rotational injury patterns (190, 197–200). In addition, Smillie (200) has suggested that the juvenile form may be caused by a disturbance in the ossification of the epiphysis itself, resulting in the separation of small islands of bone from the main bony epiphysis.

Green and Banks (186), in their classic article, noted that the basic process in OCD was aseptic necrosis involving the subchondral bone. However, most other histologic studies of OCD do not reveal ischemic changes, but a reparative process at the interface between the fibrocartilage and the bone (201–204). With the advent of MRI to evaluate OCD lesions of the knee, it is apparent that ischemic changes of bone are rare.

A few articles, including those of Mubarak and Carroll (193) and another by Ribbing (204), discuss the familial nature of the lesion, indicating an autosomal dominant inheritance pattern. However, most other large series refute the hereditary nature of the lesion (205).

The condition is most likely caused by multiple factors, including repetitive mechanical trauma or stress, in highly active children and adolescents (206).

Symptoms and Signs. Knee pain is the chief complaint in most children. The pain is activity related, and its location is nonspecific. With progression of the disease, patients may have complaints of giving way, locking, catching, or swelling. In a large multicenter study carried out by Hefti et al. (207), 32% of patients had no or minor pain at presentation.

Tenderness may be elicited with deep palpation over the involved femoral condyle. A positive Wilson test occurs with the knee flexed 90 degrees and internally rotated. As the knee is extended, the patient complains of pain that is relieved by external rotation. For patients with classic lesions, the tibial eminence impinges on the OCD lesion in internal rotation and extension, whereas, external rotation moves the eminence away from the lesion. The knee examination of patients with JOCD however is notoriously poor for detecting the presence of this disorder (208).

Imaging. Skeletally immature patients suspected of having JOCD should undergo radiographic studies. A lateral view helps identify whether lesions are on the external flexion surface and can show features consistent with normal, benign accessory ossification centers. The axial view is helpful if a lesion of the patella or trochlea is suspected. A "notch view" in 30 to 50 degrees of knee flexion may help identify the lesions of the posterior femoral condyle and the "tunnel view" may identify the lesion.

MRI is a useful adjunct to determine the extent of articular cartilage involvement and the stability of the lesion (209–214) (Fig. 31-53A,B). A high-intensity signal on T2-weighted images between the lesion and the surrounding subchondral bone means that synovial fluid from the joint is present between the lesion and the underlying subchondral bone,

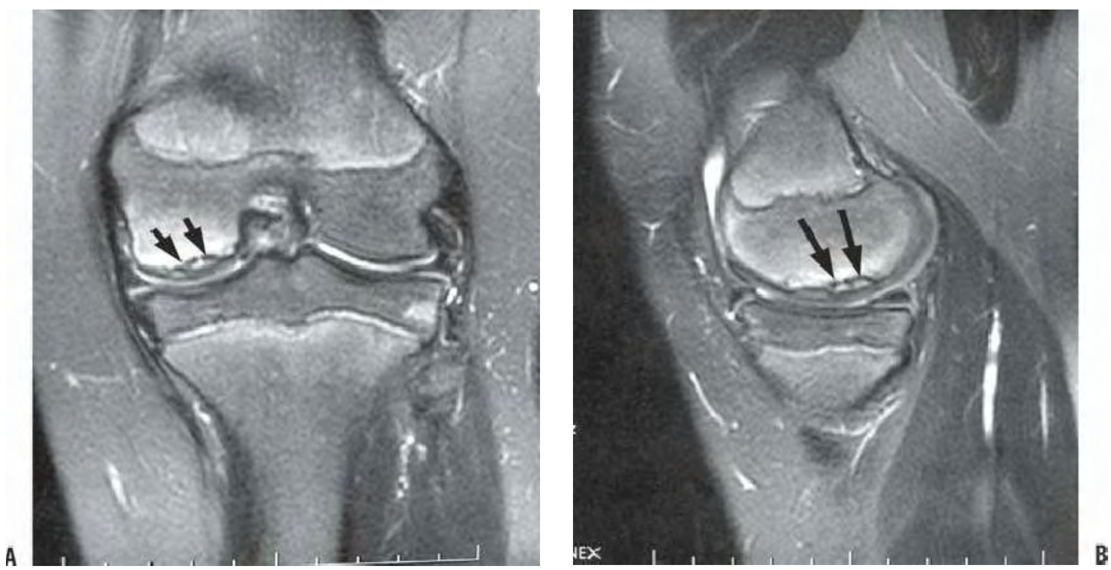


FIGURE 31-53. **A:** MRI of a knee demonstrating OCD lesion (*black arrows*) in the classical location (lateral aspect of medial femoral condyle). The lesion appears to be stable, with an intact articular surface. **B:** Lateral image of same knee (*black arrows* outline lesion).

implying instability of the lesion (209). In children, the signal may represent a line of healing vascular granulation tissue, but it is pathognomonic of instability in the adult variety of OCD (215). MRI with gadolinium enhancement improves the ability to assess lesion stability, and MRI arthrography with gadolinium allows for even greater improvement (216) (Fig. 31-53).

Natural History. The true natural history of OCD is not known, but a large multicenter European study has given clues (207). In this study, patients with JOCD (patients with open physes) did better than adults; 22% of these patients with JOCD had abnormal knees at follow-up, whereas 42% of adults with OCD had abnormal knees.

The outcome of JOCD is significantly better in lesions that are stable at the time of presentation and in the classic location as opposed to any other location. Patients who were less active had a better result at follow-up than did active athletes. Patients with stable lesions at diagnosis did better with conservative (nonoperative) treatment than did those with surgery, regardless of the type of nonoperative treatment. Conversely, patients with unstable lesions did better with surgery than did those with nonoperative treatment. There was no superior method of fixation or resurfacing, as the numbers in these groups were too small for statistical analysis.

Treatment Recommendations. The prognosis for JOCD is considerably better than the prognosis in its adult counterpart. The goals of treatment in JOCD include preservation of articular cartilage and stability of the lesion.

Nonsurgical Treatment. For small stable lesions in skeletally immature patients, nonsurgical treatment if often recommended. Nonsurgical management limits high-impact activities by instituting short-term immobilization and protected weight bearing. It is of interest that in the European multicenter study, the results of all conservative treatment methods, including cast immobilization, bracing, physiotherapy, and non–weight bearing, were the same (207). Refraining from sports for 3 to 6 months is probably efficacious in children and adolescents with OCD lesions (183, 210, 216). Wall et al. studied the healing potential of stable JOCD. The study found that after 6 months of nonoperative treatment, 16 (34%) of 47 stable lesions did not progress to healing. The size of the lesion determined by MRI was the strongest prognostic variable (217).

Plain radiographs are employed to evaluate bridging of bone across the lesion; JOCD lesions heal in an average of 4 to 5 months (188, 216, 218).

In summary, patients with intact JOCD lesions should undergo a period of nonoperative treatment, which commonly includes activity restriction and bracing or casting for 6 to 10 weeks. Uncooperative patients should be managed in a cylinder cast. Refraining from sports for up to 6 months is advisable to allow lesions to heal.

Surgical Treatment. If the lesion fails to heal after nonoperative treatment of 6 months, or if the lesion is unstable, arthroscopic evaluation and treatment are indicated. Guhl classified lesions arthroscopically as (a) intact lesion, (b) early separated lesion, (c) partially detached lesion, (d) salvageable craters and loose bodies, and (f) unsalvageable craters and loose bodies (218).

Intact lesions are usually drilled in a transarticular or retrograde manner to promote healing. The theory is that vascular ingrowth occurs in the small channels created by the Kirschner-wires or drill. Excellent results have been reported by several authors using the transarticular drilling technique (188, 216, 218). Some authors prefer not to violate the articular surface and use an extra-articular drilling method with or without bone graft to stimulate healing (214, 219, 220). Hefti's (207) multicenter study was more discouraging than these studies. In a subgroup of 58 patients demonstrating marked sclerosis, little benefit or healing was noted.

In the situation of early separation or partial detachment, internal fixation of the lesion is indicated (207, 218). This can be accomplished by various methods. Bioabsorbable pin fixation was performed on 11 patients age 12 to 16 with 32 months follow-up. One case had early synovitis treated with nonsteroid antiinflammatory drugs (NSAIDs). Union was noted in all patients by MRI (221). Bone strips were used in 11 patients aged 11 to 20 with 48 months follow-up with 90.9% satisfactory, and all patients returned to competitive sports (222). Arthroscopic titanium Herbert screw fixation was performed in 14 patients aged 12 to 35 with 50-month follow-up. At second-look arthroscopy, 14 out of 15 knees showed stable fragments (223). Postoperatively, the patients remain non–weight bearing for 6 to 8 weeks after fixation. A second-look arthroscopy is scheduled to see if the fixation is raised before the resumption of weight bearing. If the screw heads are too prominent, they are turned in further to prevent articular cartilage erosion.

If the lesion is partially detached, the bed should be freshened down to bleeding bone. The bed may require a small amount of bone grafting before the OCD lesion is replaced and fixed. It is important to achieve articular congruity at the completion of the fixation procedure (215). For unsalvageable craters and loose bodies, the loose body or bodies are removed, and the edges of the articular cartilage trimmed. Fragment excision alone appears to have poor long-term results, although in the short-term knee function may be excellent. In the European Pediatric Orthopaedic Society study, Hefti et al. (207) reported 48% poor results after fragment excision alone. Because of the poor results, they recommended some technique to restore the articular surface. Anderson and Pagnani (224) also reported a preponderance of poor results in young patients at follow-up an average of 5 years after fragment excision.

Osteochondral plugs have recently been presented as a biologic alternative to the use of hardware to provide bone graft as well as overlying articular cartilage. The advantages of osteochondral plugs are that they create vascular access channels, provide a biological bridge between cartilage and bone, and also secure the lesion. A study was conducted to evaluate the outcomes of patients who failed nonoperative treatment for unstable OCD lesions of the knee who were treated with osteochondral plugs to secure the lesions. MRI scans showed a continuous cartilage bridge in all cases and the visual analog pain scale improved from a preoperative level of 3.1 to a 2-year post operative level of 10 indicating a return to full activities by 2 years.

For full-thickness defects, the restoration of the articular surface may also be accomplished by recruiting mesenchymal stem cells via drilling, picking, or abrasion arthroplasty, and replacement via osteochondral allografts (225–232), mosaicplasty, or autologous chondrocyte regeneration (233, 234). If cartilage regeneration via mesenchymal cell stimulation is selected, then the authors prefer drilling and pick treatment over abrasion because the former do not flatten the convex surface of the femur and therefore do not increase the force per unit area at the edges of the lesion. Osteochondral autografts provide advantages of filling the defect with local autologous tissue and disadvantages include donor site morbidity and cartilage surface incongruity. A comparison of microfracture and osteochondral plug transplantation was performed in a randomized prospective study in the knee joint in children, and both groups showed encouraging results. The osteochondral autograft group, however, had superior functional and objective results at an average follow-up of 4.2 years. Osteochondral allografts can provide complete fill of the defect, and there is no donor site morbidity from these procedures. The challenges of using allograft tissue include the increased risk of disease transmission and difficulty in locating a size-matched donor. To minimize the risk of disease transmission, a screening processes is performed which can leave a window of 3 or 4 weeks for graft implantation. For young patients with large defects, autologous chondrocyte implantation is an option because at the current time this tissue most closely approximates native hyaline cartilage. The disadvantages are that the process entails two procedures, the process has increased expense relative to other procedures, and the long-term results are unknown in pediatric patients.

In summary, knee OCD is seen and treated with increasing frequency. Stable intact OCD lesions in young patients have a high rate of healing with nonoperative measures. If patients have persistence of stable lesions beyond 6 months of nonoperative treatment, then arthroscopic drilling should be considered to promote healing. Unstable lesions require surgical treatment with fixation and possible bone grafting. While long-established sclerotic lesions can be difficult to fix, the results of excision of large lesions from weight-bearing areas are poor. All of the cartilage resurfacing techniques need further study, and refinement before definitive statements regarding long-term prognosis in children and adolescents can be widely recommended.

Treatment recommendations are summarized in Figure 31-54.

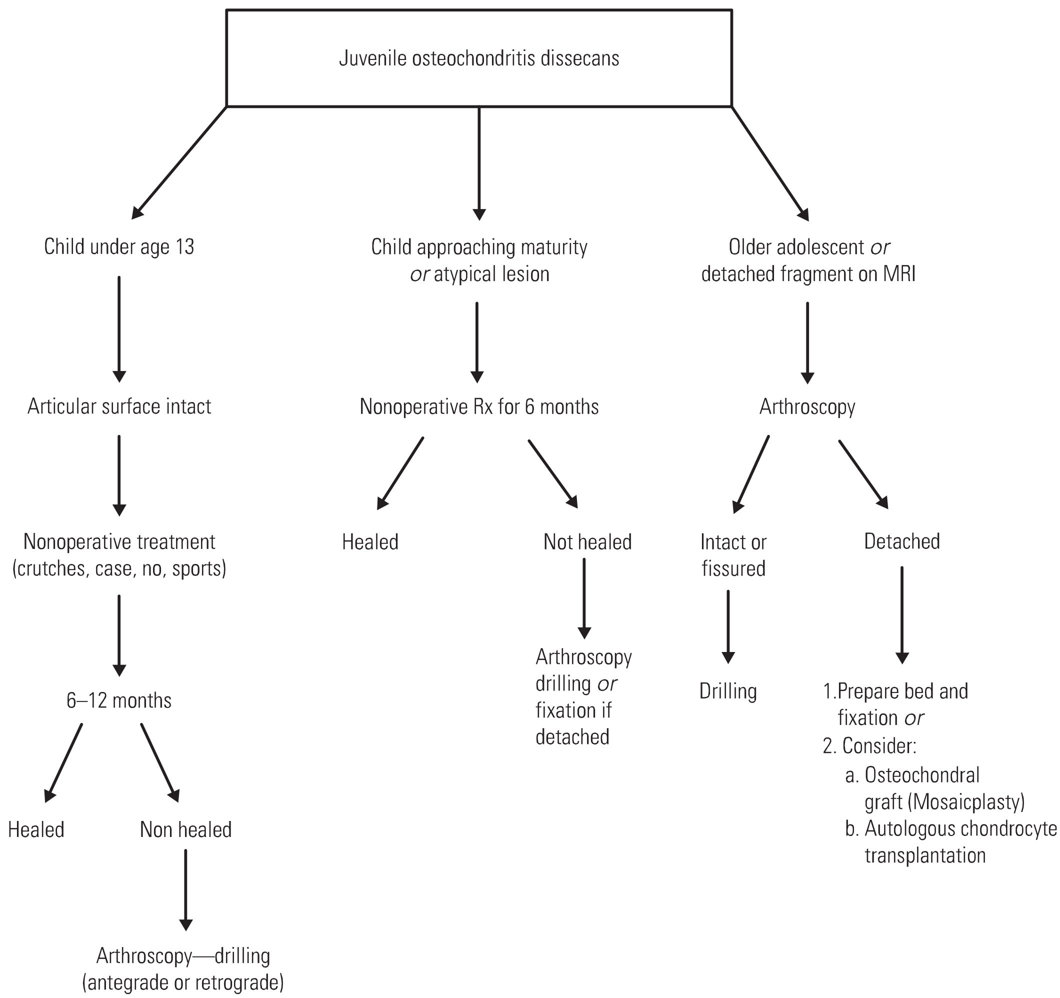


FIGURE 31-54. Algorithm for the treatment of JOCD of the knee.

Quadriceps Contusion. Blunt trauma to the anterior thigh is not an uncommon injury that occurs when playing football, hockey, and even noncontact sports such as soccer or basketball. If the force is severe enough, it will result in muscle hemorrhage, followed by the formation of granulation tissue over the course of the next few weeks, which can mature into a dense collagenous scar and lead to significant disability (235). It is therefore important to recognize this injury early to prevent long-term problems.

In the early stages of hemorrhage, significant bleeding may occur. It is accompanied by thigh swelling, pain, and loss of knee flexion.

Radiographs of the femur, including hip and knee, should be taken to rule out fracture and epiphyseal separation. The differential diagnosis should also include osteomyelitis and tumor (osteosarcoma or Ewing sarcoma), which can be ruled out with a careful history and normal laboratory workup.

Initial treatment consists of rest, ice, compression, and elevation (RICE). The knee and thigh may be further protected by employing a knee immobilizer and crutches. When the pain and muscle spasm subside, gentle active range of motion is begun. Passive stretching to increase knee flexion is not permitted and will exacerbate bleeding and formation of scar tissue. Progressive strengthening and exercise are permitted after 90 degrees of knee flexion is obtained. Moderate-to-severe contusions take from 4 to 6 weeks, on an average, to heal before return to sports participation (236–238). Minor contusions take considerably less time.

A careful evaluation of the athlete is performed before allowing full participation in sports. Knee motion of at least 120 degrees, at least 80% strength of the opposite leg, and functional agility are required (236–238). Special thigh guards can be employed to protect from further injury.

Complications of quadriceps contusion include the very rare situation of compartment syndrome of the thigh and myositis ossificans.

Anterior compartment syndrome of the thigh is usually manifested by severe thigh swelling and pain after a significant contusion and has also been described after relatively minor trauma in a patient with a bleeding disorder. Like its counterpart in the arm or leg, it demands fasciotomy to prevent muscle necrosis (239).

Myositis ossificans traumatica is a complication after severe quadriceps muscle contusion or after reinjury and occurs in up to 20% of quadriceps contusions (236).

Radiographically, flocculated densities appear at 2 to 4 weeks postinjury within the muscle mass, and periosteal new bone may also be seen. By 3 to 6 months, the bony changes stabilize (239, 240).

Despite these radiographic changes, the athlete often exhibits no functional deficit. No treatment is required if the patient is functioning well, and full participation in sports is permitted.

Loss of knee flexion and pain may rarely occur, in which case surgical excision should be undertaken, but only after the myositis has matured, which usually takes 6 months. Plain radiographs on a sequential basis will provide evidence that the lesion is mature and not continuing to ossify. A bone scan may be helpful in showing the lesion to be relatively quiescent in its uptake of radionucleotide, which is suggestive of maturity of the lesion. NSAIDs may be used before and after excision of the myositis ossificans to prevent recurrence, but there is no evidence that NSAIDs are beneficial in this clinical situation.

Ankle Sprains. In the adult population, ankle sprains comprise 25% of athletic injuries (241–243). Younger children are more likely to suffer an injury to the distal fibular physis, whereas ankle sprains are more common in adolescents.

Mechanism of Injury. Almost all ankle sprains are caused by a plantar flexion and inversion injury. The lateral ligaments, namely, the anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament, are injured in that sequence.

With the ankle in plantar flexion and inversion, the effect of bony stability is minimized and the lateral ligaments become the primary lateral stabilizers, with the anterior talofibular becoming the most important (241).

The differentiation between physeal injury (fracture) and ligamentous injury is made primarily on the basis of the anatomical location of the pain and tenderness. If the maximal tenderness is directly over the distal fibula, a fracture or physeal injury is suspected and x-rays are taken. If the maximal tenderness is directly over the anterior talofibular ligament or calcaneofibular ligament, there is little need for x-rays (242). If there is excessive swelling and it is difficult to determine the exact area of maximal tenderness, one should err on the side of caution and obtain three views of the ankle—anteroposterior, lateral, and mortise.

Classification and Management. Ligament injuries are classified according to the severity and disruption of the anatomic structure of the ligaments (244). Grade I sprains (mild) have no appreciable disruption of tissue, and there is minimal loss of function. Grade I sprains involve the anterior talofibular ligament only. Grade II sprains (moderate) have some disruption of tissue and partial loss of function with involvement of the anterior talofibular and calcaneofibular ligaments. Grade III sprains (severe) have significant or complete disruption of tissue with involvement of all the lateral ligaments and even the deltoid medially. There is marked loss of function. The type of sprain is best determined by the anatomic location of the pain and swelling and the degree of disability of the patient.

Grade III sprains and interosseous ligament injuries are more prone to develop osteochondral fractures and chronic instability. The diagnosis of interosseous ligament injury is based largely on the mechanism of the injury, the physical findings, and in rare instances radiographic findings.

Interosseous ligament injuries are universally seen in conjunction with a deltoid ligament injury. They are seen when the mechanism of injury is pronation–abduction, pronation–external rotation, and supination–external rotation of the foot. If the syndesmosis is significantly disrupted, squeezing the fibula and tibia together proximally will cause pain distally at the site of the syndesmosis in the ankle. In addition, increased side-to-side mobility of the talus in the ankle mortise when the distal leg is grasped is indicative of a syndesmosis injury. Plain radiographs that show widening of the syndesmosis width >5 mm are indicative of a syndesmosis rupture.

Rehabilitation of Patients with Ankle Sprains. Rehabilitation after an acute ankle sprain is divided into three phases. Phase I consists of rest and protection (brace, cast, splint, crutches, and ice wrap), control of swelling (ice, compression, and elevation), and early weight bearing. Phase II is aimed at reducing residual swelling and restoring range of motion of the ankle as well as strength, followed by low-impact aerobic training. The final phase (Phase III) includes proprioceptive exercises and sports-specific skills such as running, cutting, and jumping, with a gradual return to sports. With the return to sports, the athlete may benefit from an ankle stabilization brace or taping, although there is no evidence that they prevent further injury (245).

Chronic ankle instability in skeletally immature athletes is distinctly uncommon. A careful clinical and radiographic examination of the ankle and hindfoot is mandatory in patients with continuing symptoms. It is important to differentiate between functional instability and mechanical instability in the patient who complains of giving way after an ankle sprain. Functional instability is a subjective feeling of giving way during physical activity, occurring in up to 50% of patients following an ankle sprain. Its exact mechanism is unclear, but it is thought to be due to a disorder of proprioception, muscle control, and ligamentous stability. Functional instability is best managed with proprioceptive training (ankle tilt board), muscular strengthening, and the use of ankle taping or bracing for athletic activities.

Mechanical instability indicates incompetence of the stabilizing ligaments of the ankle and is demonstrated clinically by the ankle drawer test and talar tilt stress radiographs. A side-to-side difference of 10 mm or more of anterior talar translation and a talar tilt of 9 degrees or more on stress radiographs is highly suggestive of mechanical instability (246).

In addition to chronic ankle instability, the differential diagnosis includes tarsal coalition and osteochondral fracture or OCD of the talar dome.

In the rare case of chronic ankle instability in the young athlete, ligamentous reconstruction may be necessary. A variety of options exist to reconstruct the anterior talofibular ligament and calcaneofibular ligament, among them the Evans procedure (247), Watson-Jones technique (248), and the Chrisman-Snook modification of the Elmslie procedure (249). The most widely used reconstruction method is the Bröstrom repair, a direct repair and imbrication of the anterior talofibular and calcaneofibular ligaments (250). Biomechanical and clinical data support this anatomic reconstruction method (251, 252).

Avulsion Fractures of the Pelvis.

Avulsion fractures of the pelvis are not uncommon injuries seen in adolescents and young adults. Avulsion fractures occur primarily between the ages of 14 and 25 years and account for approximately 15% of pelvic fractures in children (253–255).

Etiology. The usual mechanism is a sudden and forceful concentric or eccentric muscle contraction, which occurs with rapid acceleration or deceleration.

This mechanism is commonly seen in particular sporting activities such as sprinting and jumping sports, as well as soccer and football (253). The same mechanism that would cause a muscle or tendon strain in an adult may cause an apophyseal avulsion in an adolescent.

Clinical Features. The common avulsions are from the ASIS due to violent contraction of the sartorius as seen in jumping or running, from the anterior inferior iliac spine (AIIS) due to overpull of the straight head of the rectus femoris, and from the ischial tuberosity due to forceful contraction of the hamstrings (Fig. 31-55). Avulsions of the AIIS are often seen in participants of sports involving kicking action, and avulsions of the ischial tuberosity are seen in gymnasts and hurdlers.

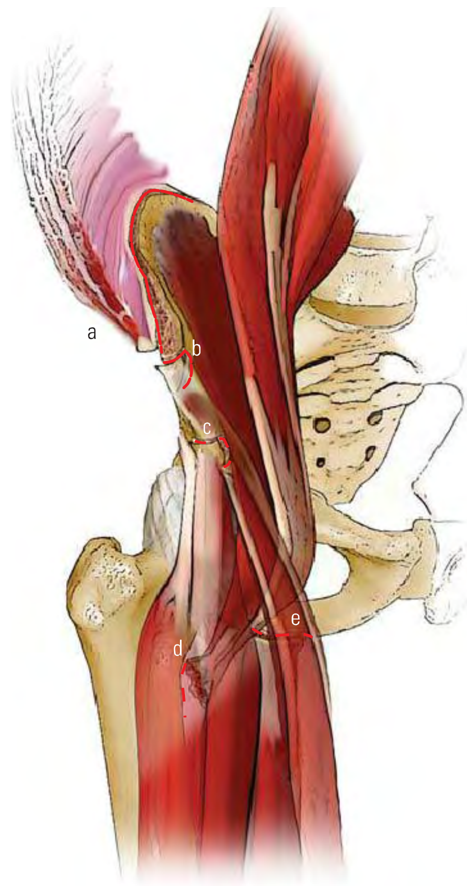


FIGURE 31-55. Avulsion fractures of the growing pelvis result from traction injuries where major muscle groups insert into or originate from apophyses about the pelvis. The abdominal and trunk muscles insert into the iliac apophysis **(A)**. The sartorius originates from the anterior superior iliac apophysis **(B)**. The direct head of the rectus femoris originates from the anterior inferior iliac apophysis **(C)**. The iliopsoas inserts into the lesser trochanteric apophysis **(D)**. The hamstrings originate from the ischial apophysis **(E)**.

The ischial tuberosity appears at 15 years of age and may not unite until 25 years of age, making ischial avulsions possible even in early adulthood (255).

With all apophyseal avulsions, there may be a history of antecedent prodromal pain signifying apophysitis before the avulsion. Athletes with avulsions present with local pain, swelling, and tenderness confined to the avulsed area. Pain is reproduced by active or passive stretch of the involved muscle.

Radiographic Features. Plain radiographs are usually sufficient to identify the area of avulsion. Special views to place the ASIS or AIIS in profile may help in delineating the avulsion. If the lesion is acute, the diagnosis is usually straightforward.

However, if the patient is seen several weeks after the inciting event, the radiographs may be misinterpreted as showing a neoplasm or infection. A careful history and normal laboratory values aid in making the correct diagnosis (Fig. 31-56).

Treatment. The recommended treatment of patients with pelvic avulsion fractures has generally been rest, followed by a specific rehabilitation program. Metzmaker and Pappas (256) outlined a five-stage rehabilitation program that consists of rest to relax the involved muscle groups as well as ice wrap and analgesics, initiation of gentle active and passive motion, resistance exercises after 75% of motion is regained, stretching and strengthening exercises with an emphasis on sports-specific exercises, and finally return to competitive sports.

Surgical intervention with attempts at open reduction and internal fixation has been recommended for isolated incidents, but there appears to be no superiority of operative intervention over conservative management (256).

Patients should be advised that the wait for return to competitive athletics may be prolonged. The earliest that such a return can be expected is 6 weeks, but it is not uncommon for complaints to persist for up to 4 to 6 months (253, 257).

FIGURE 31-56. Anteroposterior radiograph of right hip demonstrating how avulsion fracture of ischium may be mistaken for neoplasm or infection.

Dislocations of the Shoulder (Glenohumeral Joint)

Incidence. There is a wide disparity in shoulder dislocation rates among different groups of pediatric patients. Dislocations in teenagers are quite common especially those in high school and college. It has been estimated that 40% of shoulder dislocations occur in patients <22 years of age (258). Dislocations of the glenohumeral joint in preadolescent athletes however are quite rare. Overall, the incidence in children younger than 12 represents <5% of all glenohumeral dislocations (259–264).

Anatomic Considerations. The shoulder has little intrinsic stability due to the fact that the large humeral head articulates with the small shallow glenoid fossa. This is described as a low glenoid to humerus ratio. The average transverse diameter of the glenoid is 25 mm and the average transverse diameter of the humeral head is 45 mm (265). This allows for range of motion in the shoulder joint in multiple planes which is accomplished at the expense of joint stability. The shoulder is considered a ball and socket joint; however the glenoid humeral shape and size discrepancy described has drawn analogies to a golf ball on a tee (265).

Since the boney configuration of the humerus and glenoid is not conducive to stability, there are a number of static and dynamic stabilizers that contribute to this. Static stabilizers include negative intra-articular pressure, the glenohumeral ligaments, as well as the labrum. The superior, middle, and inferior glenohumeral ligaments provide anterior stability. The superior glenohumeral ligament plays a role in providing inferior stability and the anterior band of the inferior glenohumeral ligament is a major stabilizer with the shoulder in an abducted and externally rotated position. Dynamic stabilizers include the rotator cuff and the long head of the biceps tendon which contribute to joint compression. In addition to the rotator cuff and biceps, the deltoid and scapulothoracic muscles position the scapula to provide maximum stability at the glenohumeral joint.

Mechanism of Injury. Traumatic dislocations in children occur with the same mechanism as those seen in adults, including forced abduction and external rotation injuries during contact sports as well as significant falls onto an outstretched hand.

Classification. There are two broad categories of dislocation which include traumatic or atraumatic. This widely used classification system is that of Rockwood (266) who noted that of 44 cases of dislocation, 8 were traumatic and 36 were atraumatic (Table 31-3).

Clinician Features

Acute Dislocation. A child with an acute traumatic anterior shoulder dislocation may present with the arm held in slight abduction and external rotation. The humeral head may be

TABLE 31-3 Classification of Dislocation of the Glenohumeral Joint in Children

Traumatic Dislocation
As a result of true traumatic force, proximal humerus may displace anteriorly, posteriorly, or inferiorly
May occur at birth or later as a result of injury to brachial plexus or central nervous system
Atraumatic Dislocation—Voluntary or Involuntary
Occurs in a number of nontraumatic causes
Congenital abnormalities or deficiencies
Hereditary joint laxity problems such as Ehlers-Danlos syndrome
Developmental joint laxity problems
Emotional and psychiatric disturbances
Other

From Dameron TB, Rockwood CA. Part 2. Subluxations and dislocations of the glenohumeral joint. In: Rockwood CA, Green DP, eds. *Fractures*, 2nd ed., Vol. 3. Philadelphia, PA: JB Lippincott, 1984, with permission.

palpated anteriorly in the subcoracoid region with anterior dislocation. With traumatic posterior dislocation, the arm is held adducted and in marked internal rotation and the humeral head may be palpated posteriorly. With either dislocation, the normal rounded contour of the shoulder is lost, and any attempt to move the shoulder either actively or passively is typically very painful.

A careful history and physical exam are vital to the diagnosis in the diagnosis of isolated and recurrent episodes, especially in the young athlete. Patients frequently recall a specific traumatic event as well as a reduction maneuver occurring spontaneously or with assistance. The clinician should record whether the shoulder became relocated at the scene of the injury or in the emergency room. Patients with atraumatic dislocations classically do not recall a specific traumatic event. These patients usually have not had a formal reduction maneuver and do not have marked pain or dysfunction following their instability episodes. These patients may also describe multiple directions of translation with anterior and posterior subluxation or dislocation being more common than inferior.

The physical examination includes an evaluation of active and passive range of motion, as well as shoulder and upper arm strength. Most important for the assessment of instability are the evaluation of translation of the humeral head on the glenoid and apprehension and relocation testing. The stability examination should include an evaluation of both shoulders in order to distinguish pathologic laxity from physiologic laxity. The shoulder examination should also include a complete examination of the cervical spine.

Recurrent Dislocation. Glenohumeral stability may be assessed with the patient in the sitting or supine position. The sitting position requires a relaxed cooperative patient, but the supine position is usually preferred, especially with provocative tests for dislocation.

TABLE 31-4 Grading of Glenohumeral Stability Based on Translation of the Humeral Head

Grade	Description
1	No increase in translation
2	Translation to the glenoid rim
3	Translation on to the glenoid rim with spontaneous reduction (subluxation)
4	Translation beyond the glenoid rim (dislocation)

From Pagnani MJ, Galinat BJ, Warren RF. Glenohumeral instability. In: David Drez Jr, ed. *Orthopaedic sports medicine. Principles and practice*. Philadelphia, PA: WB Saunders, 1994:580–623, with permission.

Translation of the humeral head is first evaluated with the shoulder in the neutral position, in external rotation for anterior inferior testing, and in flexion and internal rotation for posterior inferior translation. The amount of translation in each direction is quantified and compared to the healthy shoulder. A grading system has been employed as shown in Table 31-4 (266).

Provocative Tests. These are often referred to as *apprehension tests* and reproduce the mechanism of instability (dislocation) that the patient recognizes.

The anterior apprehension test is performed by abducting and externally rotating the shoulder 90 degrees in each direction. As more force is gently applied, the athlete will become apprehensive of an impending dislocation and either adduct and internally rotate the shoulder or demonstrate their concern by changing facial expression or by making a sound.

For the posterior apprehension test, the shoulder is flexed to 90 degrees and internally rotated with a posterior force applied to the shoulder joint through the upper extremity.

Relocation tests are performed to validate the examiner's suspicions of shoulder instability and are best performed with the patient in the supine position. After the anterior apprehension test has been performed, a hand is placed anteriorly over the upper humerus and a posteriorly directed force is applied while again performing the apprehension test. If positive, the relocation test should relieve the patient's apprehension and can be verified by removing the posterior force to see if the apprehension returns.

The posterior relocation test is accomplished in the opposite manner, with a hand held over the posterior aspect of the upper humerus (applying an anteriorly directed force) while the posterior apprehension test is performed.

Inferior or multidirectional instability is evaluated by the sulcus test. With the patient sitting, the humerus is grasped distally just above the elbow, and an inferiorly directed force is applied while stabilizing the scapula. A dimple or gap will appear over the lateral shoulder as the humeral head is translated inferiorly. The sulcus sign is pathognomonic of multidirectional instability (267).

To assess for generalize ligamentous laxity, which is often seen in multidirectional and voluntary dislocations, a number of clinical tests should be quickly assessed, including thumb-to-forearm abduction, hyperextension of metacarpal phalangeal joints (fifth finger >90 degrees), elbow hyperextension >10 degrees, and palms to floor with knees extended (268). If the athlete has two or more of these signs, the diagnoses and implications of generalized ligamentous laxity should be considered. Surgery is generally contraindicated in this group of patients.

Treatment Recommendations

Nonsurgical. Prereduction films should be taken in most patients to confirm the direction of the dislocation and to rule out fracture. However, the diagnosis of anterior dislocation is readily apparent with the harm held in slight abduction and external rotation with the humeral head palpable anteriorly. If the treating physician is experienced in diagnosis and management, reduction of the dislocation without prior x-rays is permitted. On the playing field, this is accomplished by gentle traction on the arm in slight abduction, forward flexion, and internal rotation prior to the onset of muscle spasm.

In the emergency room, reduction is best accomplished by appropriate sedation and placing the patient prone, with the arm hanging free and 5 to 10 lb (2 to 5 kg) of weight attached to the upper extremity.

Therapy should be aimed at restoration of motion and then a specific strengthening program. The athlete should work vigorously on the anterior rotator cuff (supraspinatus and subscapularis) as well as on the periscapular muscles following an anterior dislocation. In the rare case of a posterior dislocation, the posterior rotator cuff muscles or external rotators (infraspinatus and teres minor) should be isolated and strengthened. Four specific exercises have been shown to strengthen glenohumeral muscles (269).

1. Press-up exercise. In seated position, the patient lifts his or her boy from a chair by placing the hands on the chair and extending the upper extremities.
2. Elevation of the arm in the sagittal plane.
3. Elevation of the arm in the scapular plan with the arm internally rotated and thumbs pointed down.
4. Horizontal adduction from the prone position with the arm externally rotated.

In addition to restorative motion and strengthening exercises, nonoperative treatment for first-time dislocators has classically been preceded by several weeks of immobilization in internal rotation. It has been noted by some that when the arm is immobilized in external rotation, the Bankart lesion is more accurately positioned along the glenoid rim to ensure proper anatomic healing, thereby decreasing the likelihood of redislocation (270–272). Compliance for external rotation bracing in adolescents remains challenging, and improved treatment outcomes with external rotation bracing verses internal rotation have not been universally duplicated (273) (Fig. 31-57).

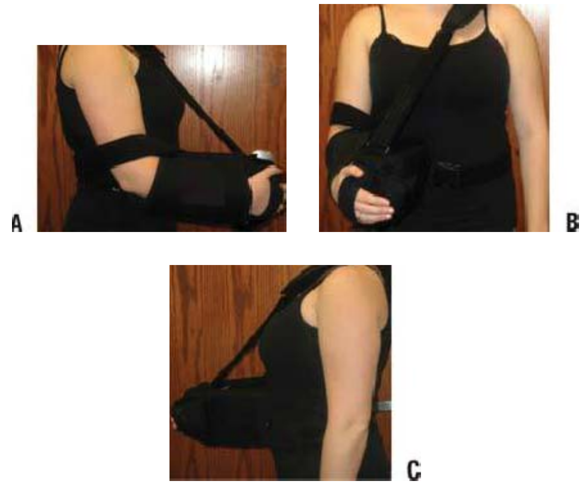


FIGURE 31-57. Patient immobilized in external rotation brace after acute anterior glenohumeral dislocation **(A–C)**.

Surgical. The redislocation rate after the initial event varies among adolescents. Rates have been described as low as 25% and as high as 100% in adolescents with open physes; however most series in these patients report rates over 50% (258–264, 274–276). The high rate of redislocation has prompted some surgeons to recommend surgical repair of the Bankart lesion by arthroscopic technique immediately after the initial dislocation (264–269, 275, 277, 278) (Fig. 31-58A–D). A shared decision-making process is helpful for patients and families for all athletes after the physician provides athletes with the risks and benefits of nonoperative and operative intervention. Surgical treatment should be considered in collision sport adolescent athletes following initial dislocation.

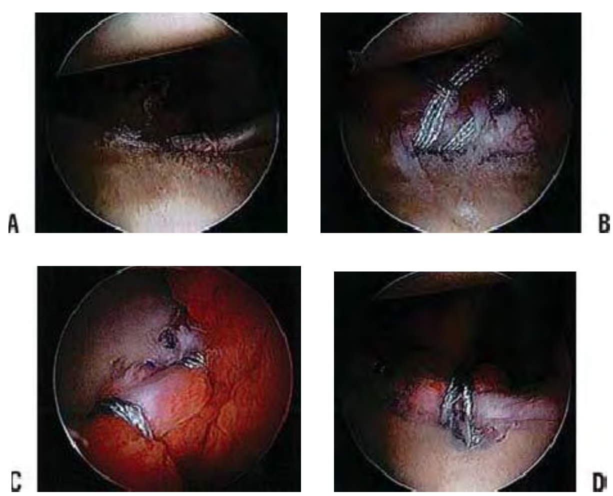


FIGURE 31-58. **A:** Soft-tissue Bankart lesion repair in a patient with repetitive shoulder dislocation. **B:** Sutures around the elevated soft-tissue Bankart in labrum and capsule. **C:** Anterior view of the shoulder showing labral repair. **D:** Status postlabral repair.

Complications

Recurrence. In almost all cases of recurrent shoulder dislocation, the instability is due to a combination of injuries to the labrum including bone or soft-tissue Bankart lesions as well as avulsion and attenuation of the capsule from the anterior glenoid. Surgery, be it arthroscopic or open technique, is aimed at restoration or repair of this anterior capsulolabral complex to eliminate the Bankart lesion (278–280) (Fig. 31-58A–D).

For the young athlete with recurrent traumatic multidirectional instability, an arthroscopic or open capsular shift is recommended. Careful preoperative evaluation of these patients is necessary to differentiate them from the atraumatic group. In the latter group, surgery has a much greater incidence of failure (266, 267).

Atraumatic dislocation is divided into voluntary dislocation, in which the patient learns to voluntarily subluxate or dislocate the glenohumeral joint, and involuntary dislocation, in which the dislocation occurs with a specific event such as carrying heavy weight. Involuntary atraumatic dislocation is often seen in association with generalized ligamentous laxity in connective tissue disorders such as Ehlers-Danlos or Marfan syndromes (267).

In patients with voluntary dislocation of the glenohumeral joint, there are a significant portion who have associated psychological or even psychiatric disorders (281, 282). Surgery should be cautioned against in this specific subset of patients, but psychological support and rehabilitation will often help (267, 281, 282).

Surgery in the involuntary atraumatic group should only be contemplated after failure of a vigorous muscle strengthening program involving all the muscle groups of the shoulder for at least 6 to 12 months. The rare patient who fails this program may be a candidate for an inferior capsule shift procedure, but the failure rate is significantly higher due to the association with ligamentous laxity (267, 268).

A small group of athletes taking part in sports with a high demand for throwing will complain of pain and decreased ability to throw. On examination, the athlete demonstrates signs of rotator cuff impingement and inflammation, with pain and weakness on resisted supination testing. There are subtle signs of glenohumeral instability upon translation testing and provocative maneuvers. These patients respond well to rest, cessation of throwing, and NSAIDs for 2 to 4 weeks, followed by a vigorous rehabilitation program once the pain has resolved. Specific strengthening of the rotator cuff and scapular stabilizers is employed and also an examination of the throwing mechanics of these athletes. Results with these nonoperative regimes are encouraging (264, 274, 283–286).

Neurologic Injury. Injury to the axillary nerve is not uncommon following traumatic or anterior dislocation, and musculocutaneous nerve injury has also been reported (265, 267). In both cases, these injuries are all almost traction neuropraxias and will resolve spontaneously. A sulcus sign is elicited with directed pressure on the arm, with the arm in neutral rotation with the patient seated. In the case of open surgical approaches, knowledge of the anatomic location, course, and direction of these nerves is essential to prevent iatrogenic injury (286–289).

OVERUSE INJURIES

Anterior Knee Pain in Adolescents.

Anterior knee pain is a common entity seen in the adolescent, both the competitive athlete and nonathlete. Anterior knee pain can occur as the result of a number of musculoskeletal conditions and can prove challenging for the orthopaedist to sort out. These conditions may include “chondromalacia patellae” or idiopathic anterior knee pain, Osgood-Schlatter syndrome, Sinding-Larsen-Johansson (SLJ) syndrome, synovial plica, or patellar or quadriceps tendinitis.

Anterior knee pain in adolescents is usually caused by repetitive overload conditions rather than a specific traumatic event.

Differential Diagnosis.

Any child or adolescent with anterior knee pain must have a careful hip examination to rule out referred pain from hip pathology such as slipped capital femoral epiphysis or Legg-Calvé-Perthes disease. In particular, any loss of hip motion or an abnormal gait demands careful clinical and radiographic evaluation of the hip.

Osgood-Schlatter Syndrome.

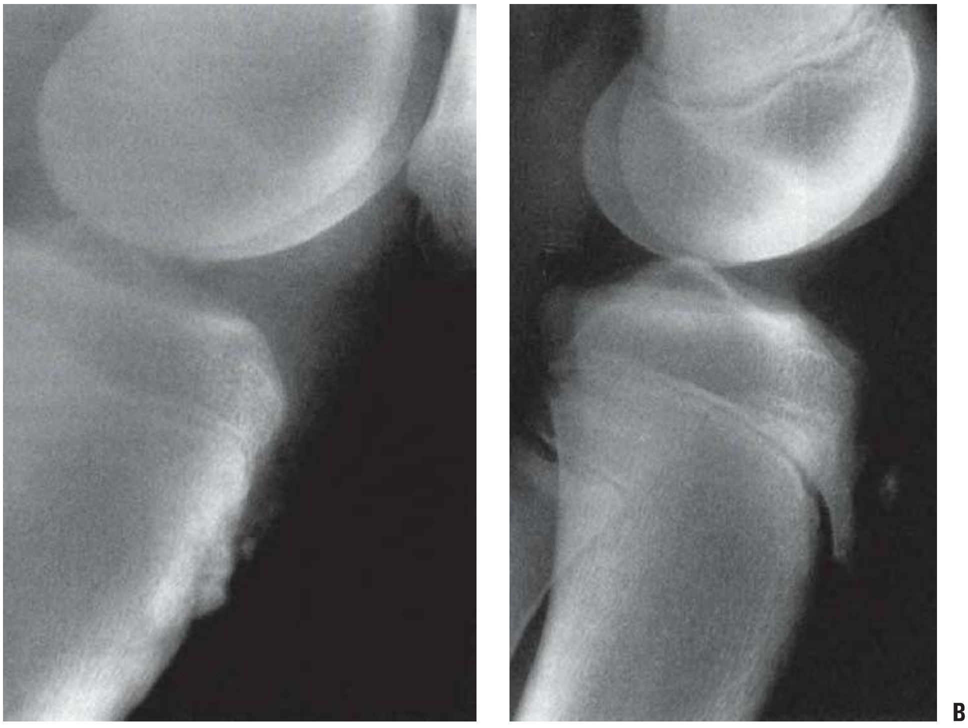
Osgood-Schlatter syndrome or disease (OSD) is a traction apophysitis of the tibial tubercle. This is an entity that is common in athletes between 10 and 15 years of age, particularly in those involved in jumping sports. Osgood-Schlatter syndrome is historically more prevalent in boys, but occurs with some frequency in girls as well.

The cause of OSD is repetitive traction on the secondary ossification center of the tibial tuberosity. It probably represents a true avulsion or stress fracture of the tibial tuberosity ossification center. Some authors have postulated that OSD occurs more frequently in patients with patella alta (290), while others have postulated the exact opposite as a contributing factor, namely, patella infera or baja (291).

Clinical Features. Symptoms vary widely in adolescents with this condition. In the acute phase, pain and tenderness directly over the tibial tubercle are noted. After the acute phase heals, the pain and tenderness subside, and the only positive physical finding may be an anterior mass. Pain in this phase occurs usually after physical activity.

Radiographic Features. The diagnosis is almost always a clinical one, and x-rays merely confirm what is already known from history and physical examination. If the condition is bilateral, plain radiographs are not necessary. However, in unilateral cases, x-rays should be ordered to rule out other pathology such as tumor or infection.

Plain radiographs (true lateral view of knee with leg internally rotated 10 to 20 degrees to place the tibial tubercle in profile) usually show fragmentation of the tubercle or a loose ossicle separate from the underlying tuberosity. Further



A B

FIGURE 31-59. Osgood-Schlatter disease. **A:** Typical radiographic findings include a prominence of the tibial tubercle with irregularity of the bone at the insertion of the patellar tendon. **B:** In some cases, a separate ossicle may form and not unite. If persistently symptomatic, this ossicle may require excision.

investigations such as MRI, CAT scans, or ultrasound are not necessary (Fig. 31-59A, B).

Natural History. In a study of 50 adults who had OSD in childhood, 76% had no symptoms or any limitation of function, 60% had pain when kneeling, and most noted the presence of a prominent tibial tubercle (288). OSD has been noted to be a predisposing factor in cases of tibial tubercle fracture, but no direct cause-and-effect relation has been demonstrated (292–294).

Treatment

Nonsurgical. The treatment of OSD in the growing child is always nonoperative. Time, rest, and occasional immobilization usually result in marked improvement of symptoms (295–299). Activity should be limited until the pain resolves and the athlete demonstrates a full painless range of knee motion. Use of ice wraps can help the acute situation and NSAIDs may relieve some of the pain. After the acute phase, a maintenance program of stretching, especially of the quadriceps, and strengthening of the quadriceps and hamstrings may help the athlete.

Surgical. If the patient is skeletally mature and still symptomatic, excision of the loose ossicle resolves the symptoms in most cases (300, 301).

Sinding-Larsen-Johansson Syndrome. SLJ syndrome is a condition similar to OSD, but affects only the proximal attachment of the patellar tendon to the inferior pole of the patella. It typically affects children at a slightly younger age than is the case with OSD, namely, 10 to 12 years.

Patients present with point tenderness at the inferior pole of the patella. Radiographs may show calcification at the inferior pole (Fig. 31-60). The condition is thought to be due to repetitive tensile stress at the junction of the tendon and bone (302, 303).

Treatment is similar to that for OSD. Little is known of the natural history of SLJ syndrome. Most patients respond to rest and NSAIDs (304).

There is little, if any, evidence of the benefits of NSAIDs for the osteochondroses. In almost all cases where they are

FIGURE 31-60. SLJ disease. The lateral radiograph best demonstrates the irregularity at the inferior pole of the patella.

administered, cessation of physical activity may be just as important in relieving the symptoms. However, their use seems justified, given the inflammatory nature of these conditions.

Synovial Plica Syndrome. Synovial plica are normal synovial folds within the knee joint that can cause knee pain. With trauma or repetitive motion, the plicae may hypertrophy, causing pain and signs of intra-articular pathology (305–309).

Clinical Features. The most common plica to cause symptoms is the medial patellar plica, which anatomically runs from the superior medial pole of the patella or midpatella to the medial patellar fat pad (305). Other plicae which have been described are the suprapatellar, lateral, and infrapatellar plicae (309) (Fig. 31-61).

Most plicae are asymptomatic, but occasionally this condition is a true symptomatic entity. The patient complains of anterior or anteromedial knee pain, often after repetitive activities such as running, jumping, or squatting. The athlete may complain of a popping or snapping sensation with their knee in midflexion. Giving way may also be a symptom.

Physical examination usually reveals tenderness directly over the plica as it comes over the medial femoral condyle to the infrapatellar fat pad, and possibly a palpable snapping sensation as the knee is flexed from 30 to 60 degrees of flexion while the patient is weight bearing or standing. In some cases, synovial plicae are associated with signs of lateral patellar instability.

Radiographic Features. Plain radiographs are normal, and other imaging studies have not proven to be of value. This syndrome is a clinical diagnosis of exclusion after other causes of knee pain and popping have been ruled out.

Pathoanatomy. Symptomatic plicae demonstrate hypertrophy and inflammation which lead to thickening and eventual fibrosis. With significant fibrosis, changes in the articular surface and even subchondral bone may occur (310).

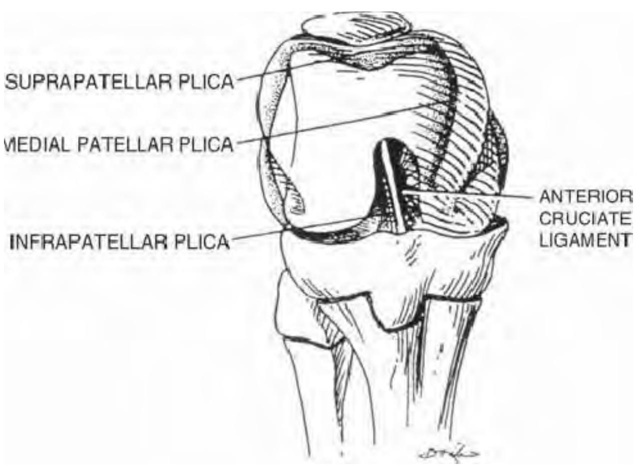


FIGURE 31-61. Plicae of the knee. The suprapatellar (superior) plica, the midial plica, which is one of the most commonly symptomatic, and the inferior plica (ligamentum mucosum), which overlies the anterior cruciate ligament.

Treatment Recommendations

Nonsurgical. Treatment should consist of nonoperative therapy including rest, ice wrap, NSAIDs, and a gradual strengthening program, especially of the quadriceps and hamstrings, avoiding terminal extension if pain is reproduced (311).

Surgical. Rarely, symptoms will fail to resolve after months of nonoperative treatment. Arthroscopic resection of the plica is indicated in those rare cases, with excellent results expected in 90% of cases (307, 312–320). Simple division of the plica has been associated with recurrence and is not recommended. At the time of surgical resection, the joint is thoroughly examined for other causes of internal derangement such as meniscal tear or patellar maltracking.

Hoffa Syndrome. Originally described by Hoffa (321) in 1904, very little is known about this condition in children or adolescents. Anatomic studies of the fat pad have revealed a densely innervated tissue, and because of its anatomic location, its role as a possible cause of anterior knee pain is debated (322–326). It has been mentioned as a source of pain or pathology in conjunction with other entities such as patellar tendinitis, meniscal tear, ACL disruption and reconstruction, impingement after intramedullary nail placement, and tibial osteotomy (310, 326).

Symptoms are similar to those in patients with patellar tendinitis or SLJ syndrome. In the latter condition, maximal tenderness is at the inferior pole of the patella. In Hoffa syndrome, the maximal area of tenderness is at the anterior joint line on either side and deep to the patellar tendon.

Management is nonsurgical and consists of rest, ice, and NSAIDs. A diagnostic intra-articular local anesthetic injection directly into the fat pad may be of help in those patients whose symptoms fail to resolve. There is little information as to the efficacy of surgical management for this condition (327). Because such doubt exists as to the validity of the diagnosis, surgical recommendations as a treatment option cannot be made (328).

Idiopathic Anterior Knee Pain. Physicians and surgeons are generally unwilling to admit that a patient's complaints defy a plausible explanation. However, anterior knee pain in children and adolescents can exist in the absence of positive physical findings for any of the pathologic conditions causing knee pain. In that case, the term "idiopathic anterior knee pain" is used (328, 329).

These patients typically complain of anterior knee pain with insidious onset. The pain is often bilateral, more commonly worse in one knee. The pain is made worse with physical activity such as running, jumping, squatting, going up and down stairs, or after prolonged sitting with the knee in flexed position.

Physical examination may reveal so-called miserable malalignment including excessive internal femoral torsion, external tibial torsion, mild genu valgum with medial deviation of the patella, and a tendency to pes planus or foot pronation. Patients typically point to the entire anterior aspect of the knee as the location of the pain. The patients usually do not have an effusion and have a full range of motion. The patella may be hypermobile and have some evidence of maltracking, but without signs of patellar subluxation or dislocation. There may be atrophy of the quadriceps and patellar crepitus with flexion and extension of the knee or a patellar compression test.

Examination of patellar tracking should include assessment of the Q angle, lateral tilt, and lateral tracking.

Radiographic Assessment. Plain radiographs including an anteroposterior, lateral, notch view, and Merchant view should be taken (Fig. 31-1). In most cases, the plain radiographs are normal. Unless the clinical diagnosis is suggestive of another pathology, there is no indication to proceed to other imaging studies such as MRI.

Treatment and Prognosis. Historically, the term "chondromalacia patellae" has been used to describe this entity (330). "Chondromalacia patellae" implies changes in the articulation between the patella and femoral sulcus, including softening, fibrillation, or erosion of the articular surface. In children and adolescents with idiopathic anterior knee pain, however, the articular surface is often normal. Articular cartilage has no sensory nerve endings, and with the lack of articular cartilage changes in these patients, the source of the pain is not definite. Therefore, one should avoid the use of the term "chondromalacia patellae" in the case of patients with idiopathic anterior knee pain.

In what should be a classic article, Sandow and Goodfellow reported on the natural history of untreated anterior knee pain in adolescent girls followed up for 2 to 8 years (331). The symptoms of most of these patients resolved over time or were significantly improved.

Treatment should be almost exclusively nonsurgical and consist of activity modification, flexibility exercises of the quadriceps and hamstrings, strengthening exercises of the same muscle groups, and the use of other modalities such as ice, heat, ultrasound, and transcutaneous electrical muscle stimulation (331–338). Some patients will benefit from foot orthotics, especially if pes planus is a component of the problem. Knee orthotics such as a patellar stabilization brace or patellar sleeve or strap may also be beneficial.

In summary, idiopathic anterior knee pain in adolescents is commonly referred to as a "headache of the knee" (331). The orthopaedist must assume the role of the "knee psychiatrist" when treating these patients, with a careful and complete clinical history and physical examination, and almost evangelical enthusiasm for nonoperative treatment. It is essential that the patient and his or her family understand that the course of symptoms may be prolonged, but with growth and maturation, activity modification, and an organized rehabilitation program, there is excellent prognosis for return to physical activities and improvement in symptoms (328).

Popliteal Cysts. Popliteal or Baker cysts are a common entity seen in young children. The common presentation is the discovery of an asymptomatic mass by the mother of the affected child on the posteromedial aspect of the knee at the popliteal crease. The age at presentation is usually 4 to 8 years. Pain is an uncommon feature. There is usually no associated intra-articular pathology in children with this symptom (339).

Plain radiographs demonstrate no bony abnormality. Transillumination of the cyst or ultrasound can document the cystic nature of the lesion and rule out solid soft-tissue lesions such as rhabdomyosarcoma. MRI is only indicated when a cystic lesion is not identified on ultrasound (340).

Anatomically, the cyst arises from the posterior aspect of the knee joint itself, between the medial head of the gastrocnemius and the semimembranosus. Although it may be firmly attached to the fascia of the medial gastrocnemius, it almost always communicates with the knee joint. Spontaneous resolution of popliteal cysts tends to occur, but this often takes up to 12 to 24 months (339).

Surgical excision is rarely indicated and should probably be considered only in cases where rapid enlargement has occurred or pain is a major feature. Recurrence rates are significant after surgery, and the treatment of choice should be watchful waiting and parental reassurance.

Shin Splints. "Shin splints" is an outdated term which is applied to virtually every type of lower leg pain as a result of overuse. A more appropriate term is shin pain.

Definition. Shin pain refers to a condition that produces pain and discomfort in the leg due to repetitive running or hiking (341). The condition is limited to musculotendinous inflammations and diagnosis should exclude stress fractures and ischemic disorders. Nevertheless, stress fracture and chronic exertional compartment syndrome are part of the differential diagnosis of leg pain in the running athlete.

Etiology. The etiology of shin splints has been variously reported in the literature. The pain is usually appreciated on the posteromedial border of the tibia from an area approximately 4 cm above the ankle to a more proximal level approximately 10 to 12 cm proximal. It was felt that symptoms were due to an inflammation and overload of the posterior tibial tendon (339, 340). Drez (342) has used the term "medial tibial stress syndrome" to describe this pain. Postmortem studies have demonstrated that the site of pain along the posteromedial border of the tibia corresponds to the medial origin of the soleus muscle (343).

Clinical Features. The physical examination consistently demonstrates tenderness along the posteromedial border of the tibia, centered at the junction of the proximal two-thirds and the distal one-third. There is no pain along the subcutaneous border of the tibia, and active and passive motion of the foot and ankle are usually negative. Active resistant plantar flexion and toe raises may elicit the pain.

Radiographic Features. Routine x-rays are usually interpreted as normal, but there may be hypertrophy of the posterior tibial cortex with subperiosteal lucency or scalloping. Faint periosteal new bone may be present.

Other Imaging Modalities. Evaluation of troublesome shin pain may include a bone scan. In medial tibial stress syndrome, there is increased uptake in a longitudinal pattern along the posteromedial tibial cortex at the exact site of pain and tenderness (344, 345). Tibial stress fractures demonstrate a transverse pattern of increased uptake (344, 345).

Differential Diagnosis. Shin pain in the adolescent athlete can be due to a multitude of causes and should be evaluated for a specific diagnosis. Possible causes of shin pain include medial tibial stress syndrome, stress fracture, exertional compartment syndrome, benign or malignant tumor, infection, and other rare causes.

Natural History of Medial Tibial Stress Syndrome. Medial tibial stress syndrome is associated with a sudden increase in athletic activity, especially running (346). A study at the U.S. Naval Academy demonstrated that inactive recruits were twice as likely to develop symptoms as were recruits who had been actively training (347). In the same study, rest was shown to be the most important element of treatment.

Treatment Recommendations

Nonsurgical. With rare exceptions, most patients respond to nonsurgical treatment. Rest or restriction of causative factors is the most important modality and is usually combined with ice, NSAIDs, stretching of lower leg muscle groups, and the use of foot orthotics.

It has been demonstrated that patients with medial tibial stress syndrome have a higher incidence of forefoot pronation (343–345). Foot orthotics designed to control or elevate the medial ray of the foot may prove to be beneficial (346–348).

Stretching of the gastrocnemius soleus complex has merit because tight heel cords have been shown to be more prevalent in patients with shin splints (342, 347).

Most patients show some improvement in symptoms 7 to 10 days after cessation of activity, but recurrence is a common problem, especially if the athlete returns to the preinjury activity level too quickly. A gradual return to full exercise over 6 weeks is recommended.

Surgical. In cases of resistant medial tibial stress syndrome after 6 to 12 months of nonsurgical management, surgery in the form of release of the investing fascia overlying the medial soleus (the soleus bridge) and division of the medial soleus origin and periosteum may be indicated (339, 347). Surgery in adults has been performed with success but is rarely indicated in the growing child.

Chronic Exertional Compartment Syndrome

Definition. Chronic exertional compartment syndrome is also called *exercise-induced compartment syndrome*; it is characterized by increased intracompartmental pressure that is sufficient to cause pain in the leg and is usually precipitated by running (348).

Etiology. With exercise and muscle contraction, significant elevations of intracompartmental pressure, up to 80 mm Hg, occur (349, 350). Muscle weight and size increase up to 20% during exercise and, because of the unyielding compartment space, lead to increased pressure that eventually exceeds the capillary filling pressure, causing ischemia and pain (350). The ischemia is never severe enough to cause muscle necrosis in exertional compartment syndrome. Indeed, other studies have cast doubt on the ischemic theory of causation (351).

Clinical Features. The typical presentation is an individual who develops leg pain in one of the muscle groups of the lower leg after training, usually running. The pain is initially dull and may persist after training has ended. Paraesthesia on the plantar aspect of the foot or the dorsum of the foot indicates involvement of the deep posterior compartment or anterior compartment respectively. These are the two most frequent compartments involved (348, 349, 352, 353).

The physical examination is often normal in the patient at rest. Examination of the foot and entire limb for mechanical axis deviations and rotational abnormalities should be performed.

Diagnostic Studies. If there is a high index of suspicion of exertional compartment syndrome, pressure measurements using either a slit catheter or a wick catheter should be performed to evaluate compartment pressure both at rest and after exercise (349, 350, 354–357).

Pedowitz et al. (357) developed the following criteria:

1. Pre-exercise pressure >15 mm Hg.
2. A 1-minute postexercise pressure of >30 mm Hg.
3. A 5-minute postexercise pressure in excess of 20 mm Hg.

Other studies employed as alternatives to compartment pressure testing, including MRI, near infrared spectroscopy, and thallium-201 single-photon emission CT scans, have been inconclusive (358, 359).

Natural History. Unfortunately, conservative treatment methods for exercise-induced compartment syndrome, including the use of orthotics, NSAIDs, activity modification, and physical therapy modalities, have been shown to be unsuccessful in cases where pressure measurements have been obtained (347, 349, 352, 353). These studies were performed only in adults.

Treatment Recommendations

Surgical. Fasciotomy of the affected compartment is the treatment of choice (347, 349, 353, 360). Pressure measurements of all four compartments should be conducted prior to surgery, as more than one compartment may be involved. The tibialis posterior may reside in a separate, deep posterior compartment, and careful evaluation should be undertaken after decompression (351, 360).

Options for fasciotomy include (a) fibulectomy (356); (b) perifibular fasciotomy (356); and (c) double-incision fasciotomy (360).

Author's Preferred Recommendations. I prefer to use multiple, limited, skin incisions for obvious cosmetic reasons. The fascia between the skin bridges must be divided to ensure that the compartment is adequately released. In release of the anterior and lateral compartments, care must be taken to protect the superficial peroneal nerve. Visualization and protection of the saphenous vein and nerve must be done with release of the superficial and deep posterior compartments. The skin is closed primarily, unlike an acute compartment syndrome.

Postoperatively, the patient is kept on crutches for 3 to 4 days, but allowed ambulation and early range of motion of adjacent joints. Strengthening is commenced when the wounds are healed, and the patient can be expected to return gradually to running after 3 to 4 weeks.

Approximately 90% of patients with chronic compartment syndrome are significantly improved with fasciotomy (347, 353, 361). Recurrence of the symptoms has been reported to be 3.4% in a large series (347). Preoperative evaluation including compartment pressure monitoring is essential prior to surgical intervention.

Trochanteric Bursitis

Iliotibial Band Syndrome. In the skeletally immature athlete, trochanteric bursitis is invariably associated with IT band tendonitis or "snapping hip syndrome." Clinically, there is tenderness directly over and distal to the greater trochanter. Pain is accentuated with adduction and external rotation of the hip. The patient can often voluntarily reproduce the snapping with a trick maneuver. Occasionally, the pain may also be experienced just above and at the knee on the lateral epicondyle. Associated factors that may contribute to this condition include a broad pelvis, leg-length discrepancy, and excessive pronation of the foot (362).

Treatment. This is a condition which generally resolves with nonsurgical treatment (362). Treatment is aimed at stretching the IT band and decreasing the inflammation with NSAIDs. In extreme cases or cases that fail to resolve, local corticosteroid injection into the IT band and greater trochanteric bursa may be indicated.

Surgery. Rarely, if nonsurgical methods fail to resolve the symptoms, operative release or lengthening of the IT band may be required (362).

Stress Fractures. Stress fractures are becoming more common in children because of an increased level of participation in organized athletics, earlier sports specialization, year-round sports, and participation in multiple teams during the same season. Stress fractures arise from repeated submaximal stresses applied to normal bone or normal stresses applied to abnormal bone and can present as a spectrum spanning from a mild microfracture to a complete fracture. Running is the sport most commonly linked with stress fractures and the tibia is the most commonly affected part of the body (363).

Stress fractures most commonly occur as a result of excessive repetitive or unaccustomed stress. The repeated stress is below the threshold required to cause acute fractures; however it is sufficient to disrupt the body's normal bone remodeling mechanism. While an increase in osteoblast activity can occur as a consequence of physical training, an abrupt onset of physical training especially after prolonged inactivity tilts the balance toward the stimulation of osteoclast activity. The abrupt change can be an increase in duration, intensity, or frequency of activity. Stress fractures are believed to occur in this early, predominantly osteoclastic remodeling period (364).

The most commonly affected areas that are affected by stress fractures are the proximal tibia, femoral neck, femoral diaphysis or the distal femoral metaphysis, medial malleolus, and the metatarsals.

Classification. This more commonly found abnormal stress applied to normal bone is described as a fatigue fracture. It is less common for athletes to have abnormal bone; however these conditions whereby normal stress causes fractures in abnormal bones are described as insufficiency fractures. Stress fractures predominantly occur in children and adolescents in strenuous activities such as running and jumping sports and have been noted infrequently in children involved in free play (365, 366).

Incidence. Military recruits have provided a wealth of information regarding a cohort of relatively young healthy active participants. A prospective study was performed on Israeli male recruits aged 17 to 26, and of these 783 recruits, the risk for stress fractures was inversely proportional to age. Each year, above the age of 17, the risk for stress fractures was noted to have been reduced by 28% (367). Stress fractures most commonly affect the tibia and fibula in younger patients. A retrospective review was performed on 154 military patients aged 17 to 29, and of the 143 stress fractures identified 99% were located at the tibia (368).

Risk Factors. Many factors have been correlated with stress injuries in pediatric athletes including an excessive rate of exercise progression, anatomic malalignment, a history of stress injuries, changes in strength and flexibility associated with growth, and increased body mass index (366). A well-understood risk for stress fractures is a rapid increase in training intensity which can be commonly found in young athletes implementing new training protocols or beginning team preseason training regimens. The female athlete triad of menstrual irregularity, osteopenia, and disordered eating should alert the treating physician to the potential for an increased risk for stress injuries. A difference was noted in cumulative stress fractures with an incidence of 4% in girls with a regular menstrual history versus 15% in girls with irregular or absent menses (369). A prospective, multicenter cohort study was performed to investigate risk factors, and among 146 collegiate athletes those more likely to develop medial tibial stress syndrome had been participating in athletic activity for fewer than 5 years, had a history of medial tibial stress syndrome or stress fracture, or had used orthotics (370). In a study of adolescent athletes with open physes, patients were primarily treated with reduction in weight bearing; however, 7 of 21 patients reported unsatisfactory outcomes and four patients with tibial stress fractures had persistent symptoms and were changed from reduced weight bearing to plaster cast immobilization. Risk factors for tibial stress fractures include hip external rotation, knee malalignment, smaller tibial width, a poor level of conditioning, hard terrain, as well as nutritional factors.

Differential Diagnosis. Chronic leg pain can occur due to a variety of reasons in young athletes. The main concern in diagnosis is differentiating a stress fracture from a malignant bone tumor, especially with some periosteal new bone formation. The physician must also rule out other conditions such as benign tumors including osteoid osteoma, infection, inflammatory arthritis, or soft-tissue injury. Shin splints are discomfort in the leg from musculotendinous inflammation as a result of repetitive impact exercises or the use of foot flexors in sports such as running. In patients with proximal discomfort, the examiner should check for referred pain from the patellofemoral region as patients with the terrible triad of femoral anteversion, genu valgum, and pes planus. This terrible triad of malalignment issues is a predisposing factor in the development of overuse injuries of the patellofemoral region and the lower legs.

Clinical Features. When assessing a young athlete in whom an overuse injury is suspected a careful and detailed history is important in differentiating stress fractures from other conditions. Patients with overuse injuries frequently complain of an insidious onset of pain that is most intense when they are involved in repetitive physical activity such as running jumping or dancing (365, 366). Because young athletes occasionally deny having symptoms to avoid resting, the information provided by parents and coaches can be valuable in terms of describing earlier injuries as well as a change in the athlete's technique, performance, and disposition. Night pain is uncommon in stress fractures but is common is osteoid osteoma or malignant bone tumors such as osteogenic and Ewing sarcoma.

The physical examination should include an evaluation of the entire kinetic chain including alignment, gait, limb length, muscle strength, and flexibility which may reveal tightness, appropriate flexibility, or pathologic laxity. Stress fractures are noted to occur more commonly in athletes with foot abnormalities such as pes cavus and pes planus (366, 371–373). The exam also includes palpation of tender areas at the site of the stress fracture; however, swelling is not a reliable or consistent clinical finding (364).

Imaging Studies. Plain radiograms are inconspicuous in the early phase of a stress fracture; however, they can be useful in the late phase because they can show a periosteal reaction or a fracture line such as the dreaded tibial black line. Several weeks to several months can elapse from the start of a patient's symptoms until the time that plain radiographic changes begin to appear (Fig. 31-62). Technetium scans are helpful to confirm the diagnosis of a stress fracture, and changes can be noted using this technique as early as 12 to 15 days following the onset of injury and symptoms. It can at times be challenging using bone scintigraphy to distinguish the difference between a stress fracture of the bone and shin splints from musculotendinous inflammation. MRI has been described as a method of differentiating stress reactions of the bone from other conditions in the legs. A study was performed on 22 athletes whose average age was 16 years. It was noted that fat-suppressed MRI showing a localized wide high signal at a localized area of bone marrow for stress fractures was useful for discrimination between stress fractures and shin splints before the plain radiographs showed a detectable tibial periosteal reaction (371). Radiographs, technetium bone scans, and MRI were also obtained in symptomatic legs in runners, and the MRI findings correlated with established bone scan grading systems and more precisely defined the site of injury (374). In stress fractures, MRI shows bandlike areas of very low signal intensity in the intramedullary area, continuous with the cortical bone. On T1 images, there is decreased signal intensity in the marrow (375) (Fig. 31-63).

FIGURE 31-62. Plicae of the knee. The suprapatellar (superior) plica, the medial plica, which is one of the most commonly symptomatic, and the inferior plica (ligamentum mucosum), which overlies the ACL.

FIGURE 31-63. MRI of the same patient as in Figure 31-53. *Arrow* points to fracture line.

Treatment. Treatment of stress fractures in the child or adolescent depends on the site of injury, the severity of symptoms, and the patient's age (365, 376, 377).

The principles of treatment in young patients revolve around the pediatric sports abc's of activity modification, bracing and continued rehabilitation, and prevention. The activity modification includes education and instruction for the young athlete to partake in exercise that does impart forces on the injured area. This at times includes eliminating the sport that the athlete participates in altogether. Bracing for lower extremity stress fractures may include boot or cast immobilization or off-the-counter orthotics for 4 to 6 weeks to allow sufficient bone deposition to occur. Continued rehabilitation and prevention includes advancement to strengthening, flexibility exercises and training, as well as instruction regarding already addressed factors in the workup including nutritional influences, graduated training regimen increases, and an awareness of training surfaces.

Surgical Treatment. There are some anatomical areas that are prone to prolonged symptoms or in some cases nonunion including the femoral neck, tibial diaphysis, medial malleolus, and tarsal navicular (366).

Femoral shaft stress fractures are at low risk of nonunion or displacement and can be managed with activity modification. Femoral neck stress fractures are at high risk of nonunion or displacement, potentially leading to avascular necrosis (AVN). Surgical treatment is therefore recommended and is most commonly performed with cannulated compression screws.

For tibial stress fractures, the posterior medial compression side stress fracture is the most common. This is a low-risk stress fracture and most heal with 4 to 6 weeks of pneumatic boot immobilization. The tibial anterior tension side fracture is less common; however in this hypovascular region, callus is generally not formed and patients are at risk for delayed union or nonunion (378). In the case of delayed union, a patellar tendon-bearing cast or commercial orthosis should be employed for 4 to 6 months before surgery is considered. If nonunion exists in the skeletally mature adolescent, options to treat the nonunion include the use of an intramedullary nail with or without bone grafting and fibular osteotomy (379). In the skeletally immature individual with a diaphyseal tibial stress fracture that fails to heal after 6 months of nonoperative treatment, operative treatment is indicated. Excision of the fibrous nonunion and autogenous bone grafting from the iliac crest with possible fibular osteotomy has been employed successfully in this rare situation (379). For medial malleolus stress fractures, the fracture line tends to traverse in an oblique manner from the intersection of the medial malleolus and the tibial plafond. Nonoperative management including restricted weight bearing as well as casting or bracing has a higher failure rate than other low-risk fractures. Operative intervention therefore can be performed to facilitate early healing and recovery.

In the case of nonunion of tarsal navicular stress fractures, recommended treatment includes excision of the nonunion site and autogenous bone grafting. In the case of 5th metatarsal fracture nonunion, recommended treatment includes intramedullary screw fixation with or without bone grafting.

Return to Sports. While the greatest concern for a young athlete is the timeliness of return to sports, the treating physician must assess multiple variables. Return to play should be guided by factors indicative of healing and injury prevention including the cessation of pain, radiographic evidence of a healed fracture, elimination of inciting causes and instruction regarding training methods, and a graduated exercise program.

Sever Disease

Definition. Sever calcaneal apophysitis is a self-limited inflammatory condition of the os calcis in growing children.

Clinical Features. The typical child with Sever disease is from 9 to 14 years of age and involved in running sports (380). The condition is bilateral in most cases and presents as diffuse pain and tenderness over the prominence of the heel rather than exclusively on the plantar aspect as is seen in plantar fasciitis (381).

Natural History. Originally described by Sever (382) in 1912, the condition is presumed to be an inflammatory condition of the calcaneal apophysis. Radiographs are normal, and the condition in all cases improves over time.

Treatment. With rest, activity modification, and passage of time, the condition always resolves (380, 382, 383). Ice, oral NSAIDs, stretching exercise of the Achilles tendon and plantar fascia, and heel pads or inserts may offer some relief from the discomfort (380, 382, 383). Cast immobilization may rarely be necessary for the patient with severe, incapacitating symptoms (383). Reassurance and time are the basis of treatment of this common condition.

Stress Physeal Reactions. Repetitive stress in the upper extremity may lead to a specific stress reaction in the physis. This reaction is not uncommonly seen in the distal radial physis of skeletally immature gymnasts who load their wrists with significant compressive forces while participating in vault and floor exercises (Fig. 31-64). Radiographically, the stress reaction is noted as a radiolucency and irregularity on the metaphyseal side of the physis, similar to the radiolucency seen in the medial aspect of the proximal tibia in adolescent tibia vara and in the femoral neck in patients with slipped capital femoral epiphysis.

Caine et al. (384) found radiographic abnormalities in the distal radius in up to 85% of competitive gymnasts who were skeletally immature. The same study found an increased incidence of positive ulnar variance in the wrists of nonelite gymnasts (384). The hypothesis of the study was that repetitive compressive forces to the distal radius led to premature growth arrest resulting in a positive ulnar variance. Positive ulnar variance has been associated with secondary wrist problems including tears of the triangular fibrocartilage complex and ulnar impingement syndrome.

Athletes involved in throwing sports can develop the same symptoms and radiographic abnormalities in the proximal humerus. This condition is commonly seen in Little League pitchers (385) (Fig. 31-65A, B).

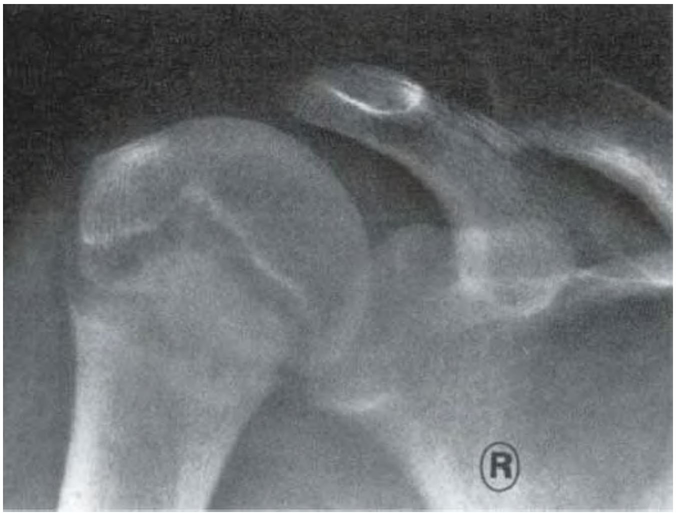
FIGURE 31-64. Anteroposterior radiograph of wrist of gymnast with 3-month history of wrist pain. Note radiolucency on the metaphyseal side of growth plate (physis) indicative of stress physeal reaction.

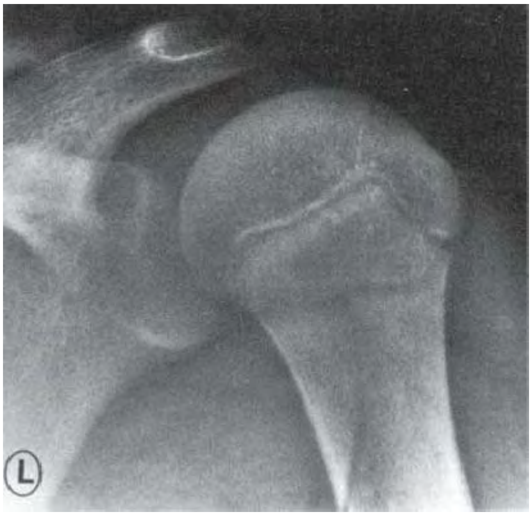
Treatment of these overuse stress reactions is centered on the discontinuation of the offending force or modification of activities until the symptoms of pain subside. Baseball pitchers may need to refrain from pitching for 2 to 3 months until the pain that accompanies the throwing motion totally subsides.

Gymnasts with symptomatic radial physeal stress reaction should refrain from loading their wrists for up to 2 to 3 months to allow the reactive changes to heal.

Strengthening exercises to specifically strengthen the wrist flexors should be undertaken in gymnasts, and taping or bracing to limit wrist dorsiflexion may help prevent recurrence.

A





B

FIGURE 31-65. Radiographs of the proximal humerus in a 14-year-old, right-handed baseball pitcher with shoulder pain that progressed over the final few weeks of the season. **A:** Widening and irregularity of the physeal plate are present in the right shoulder. **B:** Radiograph of the left shoulder is provided for comparison.

With rest and refraining from the inciting or offending exercise, the radiographic changes seen at the physis and metaphysis will resolve.

Elbow Problems in the Throwing Athlete. The elbow of the immature athlete is susceptible to injury when the athlete throws too frequently or uses a style that puts too much stress on the elbow joint. While young athletes have excellent healing potential and regenerative capacities, the apophyses and physes are areas of weakness in young throwers. In a youth baseball study of 476 pitchers, 50% of 9- to 14-year-olds reported elbow or shoulder pain (386).

During the throwing motion, a significant valgus moment is applied to the elbow joint (385). This valgus moment results in excessive tensile or distraction forces on the medial aspect of the joint (medial epicondyle and ulnar collateral ligaments) and excessive compressive forces on the lateral aspect of the joint (radiocapitellar joint) (381, 387) (Fig. 31-66). Recent literature has emphasized the need for proper pitching mechanics in order to reduce injury risk (388, 389). Better pitching mechanics have been shown to increase efficiency as well as create lower valgus load on the elbow and rotational torque on the humerus (388).

Safety guidelines published in *USA Baseball News* recommended limits of 52 ± 15 pitches per game for 8- to 10-year-olds, 68 ± 18 for 11- to 12-year-olds, and 76 ± 16 for 13- to 14-year-olds (390–392). Similarly, a USA Baseball Medical Advisory Council created guidelines in 2006 and recommended limits for youth baseball pitchers. They recommended for 9- to 10-year-old pitchers limits of 50 pitches per game and 75 pitches per week. For 11- to 12-year-old pitchers, the recommendation is 75 pitches per game and 100 pitches per week; and for 13- to 14-year-old pitchers, it is 75 pitches per game and 125 pitches per week. Pitchers are also discouraged from pitching for more than one team in a given season and from returning to the mound in a game once he or she has been removed (393). Youth pitchers are also at increased risk of elbow and shoulder injury when they throw curveballs and sliders, and youth pitchers should therefore avoid throwing breaking pitches (386). Other controlled laboratory studies on youth baseball pitchers show that the curveball when compared with other pitches such as the fastball does not put the shoulder or elbow at an increased risk for injury (391, 392). The authors suggest that increased risk of injury is due to the amount of pitching rather than the type of the pitch (394).

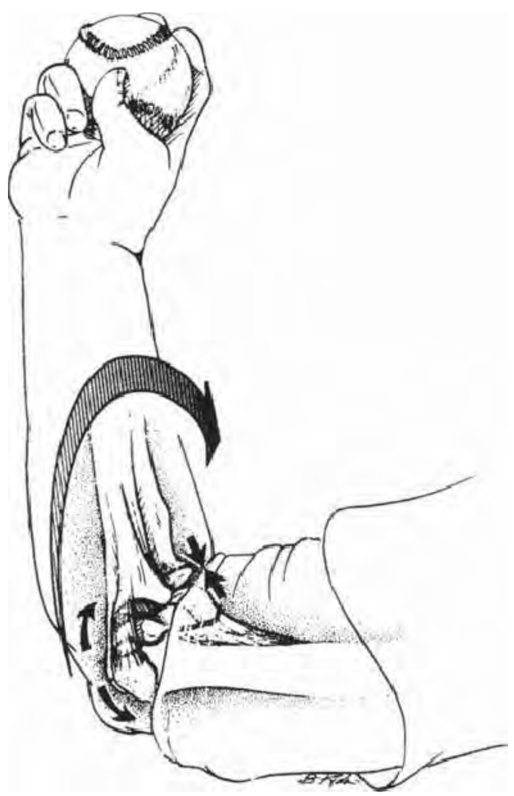


FIGURE 31-66. Pitching produces a valgus moment (*large curved arrow*) at the elbow. There are compressive forces (*straight arrows*) across the radiocapitellar joint and tension (*curved arrows*) across the medial epicondyle and MCL.

"Thrower's elbow" is the term used to describe all the facets of overuse injury in the young throwing athlete. The term was originally proposed in 1960 and encompasses a spectrum of conditions including medial epicondyle apophysitis, capitellar OCD, radial head deformation, olecranon apophysitis, and flexion contracture (390).

Pathophysiology. Risk factors that cause elbow problems in the young athlete involved in throwing sports have been studied extensively in recent years. Factors responsible include age of the pitcher, skeletal maturity, tissue strength, muscle strength, level of conditioning, throwing speed and style, types of pitches thrown, number of pitches per outing, and number of outings per season (386, 390, 391, 411).

In a study that analyzed elbow and shoulder problems in the young throwing athlete, 47% of pitchers complained of shoulder or elbow pain, and 68% of the elbow pains were medial (382, 389). Twenty-eight percent of the pitchers complained of elbow pain specifically, and throwing split finger pitches or sliders increased the incidence of elbow complaints (386).

Two groups of adolescent pitchers were asked to complete a survey in an effort to isolate risk factors for upper extremity injury: 95 pitchers who had shoulder or elbow surgery and 45 pitchers with no significant pitching-related injury. Results showed that injury was most strongly associated with overuse and fatigue (395). High-pitch velocity and showcase participation also increased the risk for injury.

Medial Epicondyle Apophysitis. The muscle, tendon, and bone of the medial epicondyle resist traction forces better than the apophyseal cartilage. These repetitive stresses cause microfractures in the apophyseal cartilage not unlike those seen in Osgood-Schlatter syndrome. The apophysis is seen to widen on x-ray films. Clinically, these patients tend to be in their preadolescent years around the ages of 10 to 13. A gradual onset of pain is most common including pain with activity such as hard throwing.

On examination, point tenderness over the medial epicondyle is elicited. The pain may also be increased if a valgus stress is applied to the elbow or if resisted wrist flexion or pronation is tested. Patients may also present with swelling and a flexion contracture in more severe chronic cases. Radiographs may be normal or show medial epicondyle fragmentation or physeal widening. Comparison radiographs can help delineate subtle abnormalities.

Treatment. Treatment for most of the overuse injuries in the skeletally immature athlete is rest and avoidance of the offending activity, and the elbow is no exception. Rest, ice, and the use of acetaminophen or NSAIDs should result in improvement of symptoms. A gradual return to sports may resume when the athlete is symptom free, requires no medication, and has a normal examination. An interval progressive throwing program can typically be instituted at 6 to 12 weeks following the onset of treatment. If the athlete develops a true avulsion of the medial epicondyle, treatment should consist of cast immobilization if the apophysis is displaced 5 mm or less. Surgical fixation of the medial epicondyle should be considered if it is displaced more than 5 mm in a throwing athlete or gymnast. Fixation can be accomplished with a small cannulated screw, which allows early range of motion to minimize the risk of elbow stiffness. The screw does not need to pass through the opposite cortex. A fully threaded screw with washer is employed to provide enough stability to allow early range of motion. The epicondyle is positioned without removal of physeal cartilage. The screw is not routinely removed unless it becomes prominent and symptomatic to the patient.

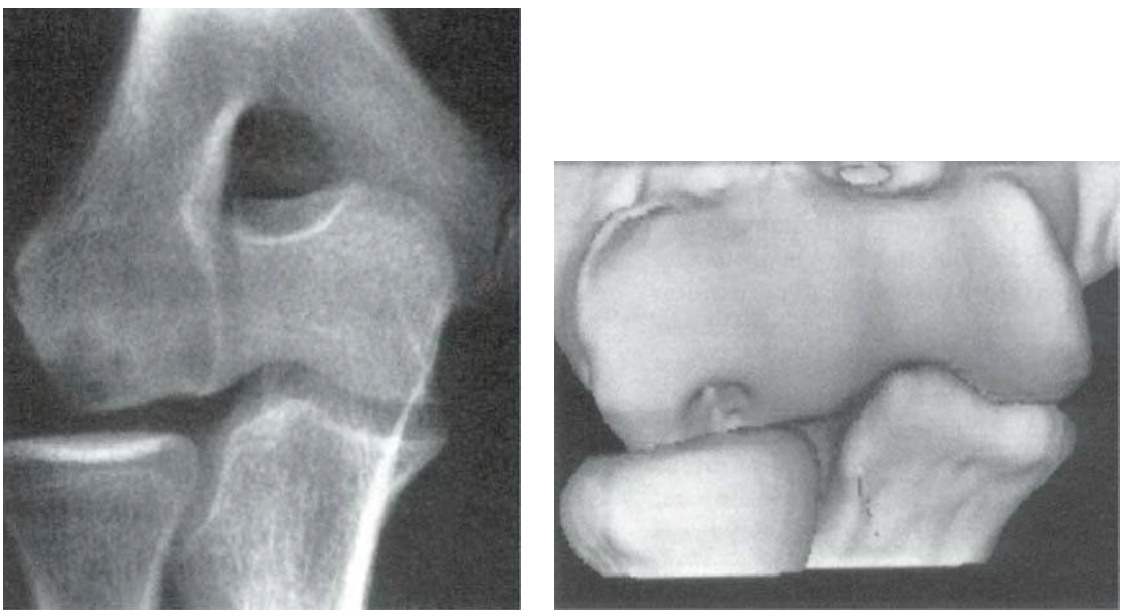
Capitellar Osteochondritis Dissecans. JOCD of the elbow should be differentiated from juvenile osteochondroses of the capitellum or "Panner disease" (396, 397). Originally described by Panner (397) as a posttraumatic AVN, the juvenile form occurs in children who are younger than 10 years; it is atraumatic and probably due to variation in ossification rather than typical OCD. It is self-limited and resolves spontaneously (396). OCD is differentiated from Panner disease by its older age of presentation (i.e., age 10 years or older), incomplete involvement of the capitellum, loose body formation, and prolonged clinical course.

The vast majority of patients are males in baseball and females in gymnastics. The true incidence in baseball players and gymnasts remains unknown.

Pathoanatomy. The pathologic entity of OCD involves infarction in the subchondral bone followed by resorption of the necrotic bone and changes suggestive of repair. The exact mechanism is not yet determined, but likely involves an interplay between ischemia, trauma, and genetic factors. Other minor factors that may have potential roles in the etiology are a biomechanical mismatch between the radial head and the capitellum, blood dyscrasia, and endocrine abnormalities.

The late cocking and early acceleration phases of throwing causes valgus stress and increased compressive loads at the radiocapitellar joint (398). Similarly, gymnasts experience lateral compression of the elbow when performing routines that convert the arms to weight-bearing extremities. The elbows of female gymnasts are in valgus in full extension and, when combined with an increased carrying angle compared to males, result in large axial loading to the lateral side of the elbow.

Evaluation. OCD of the capitellum presents with an insidious onset of lateral elbow pain in the dominant extremity. A family history of OCD may be helpful since a genetic predisposition is likely (Fig. 31-67). If loose bodies are present,



A B

FIGURE 31-67. Osteochondral lesion of the capitellum in a 15-year-old baseball player with a painful elbow. **A:** The subchondral plate of the capitellum appears intact. Subchondral cysts are present. **B:** The CT scan with three-dimensional reconstruction demonstrates the bony defect of the capitellum, including loss of the subchondral plate in that area.

additional complaints of locking and catching may be reported. The patient will usually have tenderness directly over the capitellum and a small effusion. There is often a loss of terminal extension, and occasionally, mild losses of flexion and forearm rotation. Forearm rotation may elicit crepitus in the radiocapitellar joint.

Standard radiographic views of the involved elbow should be obtained for patients with lateral elbow pain. Radiographs of the contralateral elbow may help when subtle findings are likely. OCD lesions are seen as focal areas of lucency in the subchondral bone with surrounding subchondral sclerosis, most commonly in the anterior–distal surface of the capitellum. A characteristic semilunar rarefied zone called the crescent sign may be seen. Irregularity and enlargement of the radial head, as also seen in Panner disease, may be noted. MRI may be helpful for detecting early lesions by revealing marrow edema and unstable lesions by showing intervening fluid on T2-weighted images.

Nonoperative Treatment. Treatment is governed by stage and clinical findings. Symptomatic, skeletally mature patients and skeletally immature patients with intact, stable lesions shown on MRI are best managed nonsurgically with rest and cessation of throwing. The activity modification reduces excessive axial load to the radiocapitellar joint. A gradual return to activities is allowed once full range of motion, strength, and endurance have returned. A progression of activities is allowed as long as the patient remains asymptomatic, and full participation is generally achieved after 3 to 6 months. The lesions, however, may take up to 12 months to heal. A recent study of 39 baseball players (mean age, 12.8 years) with capitellar OCD showed that nonoperative treatment is appropriate for early lesions as the healing potential is still high. However, the potential to spontaneously heal is low in advanced cases of OCD, thereby indicating surgical treatment (399). Early diagnosis and treatment therefore provide a more favorable outcome.

Operative Treatment. Surgical management may be necessary if symptoms continue with nonsurgical care. Persistent symptoms may indicate loose bodies, articular cartilage fractures, or displacement of an osteochondral fragment. If the lesion is unstable, the base should be freshened and fixed with pins, screws, or bioabsorbable nails. Surgical intervention often involves excision of partially attached lesions and loose bodies, as well as subchondral drilling or abrasion chondroplasty. Recent literature points to autologous osteochondral mosaicplasty as a relatively effective treatment for advanced lesions. Nineteen adolescent patients were evaluated to determine the efficacy of autologous osteochondral mosaicplasty for advanced elbow OCD lesions in teenage athletes. All but two patients returned to a competitive level of the sport that they had previously played, and clinically significant improvements were noted in elbow range of motion and clinical elbow scores (400). Recipient and donor sites have been evaluated in separate studies, and MRI findings indicate that the graft incorporation to the surrounding tissues occurs around or after 6 months postoperatively (401). For the recipient site, no adverse effects on donor knee function were found based on Lysholm and IKDC scores; however MRI indicates that the donor site is resurfaced with fibrous tissue (402).

The use of multiple arthroscopic lateral portals can raise concern for trauma to the lateral ligamentous complex of the elbow; however studies show that correct placement of arthroscopic portals or lateral mini arthrotomy can avoid these structures and allow for full visualization of the defect (403, 404).

Rehabilitation is begun early to promote restoration of motion and strength. The decision to resume throwing sports is one that should not be made until the lesion is healed and the elbow fully rehabilitated.

The skeletally immature elbow is susceptible to overuse injury due to the forces applied to the elbow during throwing (381, 387, 391, 392, 405–408). When the elbow becomes painful, coaches, trainers, and medical personnel should advise cessation of throwing. Fortunately, nonoperative treatment is successful in alleviating most of the conditions associated with "Thrower elbow," but the athlete, parents, and coaches must be educated as to the reasons for the problem and ways to prevent it from recurring.

This chapter outlines the many potentially acute and overuse injuries that can occur to the skeletally immature athlete. Despite these risks, the beneficial aspects of recreational and organized sports far outweigh the hazards. Most important of all the benefits is the development of an active and healthy lifestyle, which offers countless rewards not only during childhood and adolescence but also in adult life.

REFERENCES

1. National Federation of State High School Associations (NFHS). *2005–2006 High School Athletics Participation Survey.* Indianapolis, IN. NHFS, 2006.
2. U.S. Product Safety Commission NEISS coding manual 2008. National electronic injury surveillance system. Sports-related injuries treated in U.S. Hospital Emergency Departments. *Ages* 0–18, 2008.
3. Darrow CJ, Collins CL, Yard EE, et al. Epidemiology of severe injuries among United States high school athletes 2005–2007. *Am J Sports Med* 2009;37(9):1798–1805.
4. Barfield WR, Gross RH. Injury prevention in care of the young athlete. In: Sullivan JA, Anderson SJ, eds. *Care of the young athlete*. Rosemont, IL: AAOS and American Academy of Pediatrics, 2000:121–130.
5. Bijur PE, Trumble A, Harel Y, et al. Sports and recreation injuries in U.S. children and athletes. *Arch Pediatr Adolesc Med* 1995;149:1009–1116.
6. Hergenroeder AC. Prevention of sports injuries. *Pediatrics* 1998;101: 1057–1063.
7. Anderson S. Principles of rehabilitation in care of the young athlete. In: Sullivan JA, Anderson SJ, eds. *Care of the young athlete*. Rosemont, IL: American Academy of Pediatrics and AAOS, 2000:267–280.
8. Kibler WB, Lee PA, Herring SA, eds. *Functional rehabilitation of sports and musculoskeletal injuries*. Gaithersburg, MD Aspen Publishers, 1998.
9. Young JL, Press JM. The physiologic basis of sports rehabilitation. *Phys Med Rehabil Clin N Am* 1994;5:9–36.
10. American Academy of Pediatrics Committee on Sports Medicine and Fitness. Adolescents and anabolic steroids: a subject review. *Pediatrics* 1997:99:904–908.
11. Brower K. Anabolic steroids: addictive, psychiatric and medical consequences. *Am J Addict* 1992;1:100–114.
12. Griesmer BA. Performance enhancing substances in care of the young athlete. In: Sullivan JA, Anderson SJ, eds. *Care of the young athlete*. Rosemont, IL AAOS and American Academy of Pediatrics, 2000:95–104.

13. Johnson MD. Anabolic steroid use in adolescent athletes. *Pediatr Clin North Am* 1990;37:1111–1123.
14. Sewall L, Micheli LJ. Strength training for children. *J Pediatr Orthop* 1986;6:143–146.
15. Brady TA, Cahill BR, Bodnar LM. Weight training related injuries in the high school athlete. *Am J Sports Med* 1982;10:1–5.
16. Weltman A, Janney C, Rians C. The effect of hydraulic resistant strength training in prepubertal males. *Med Sci Sports Exerc* 1986;18:629–638.
17. Scavenius M, Iversen BF. Non-traumatic clavicular osteolysis in weight lifters. *Am J Sports Med* 1992;20:463–446.
18. Gumbs VL, Segal D, Halligan JB, et al. Bilateral distal radius and ulnar fractures in adolescent weight lifters. *Am J Sports Med* 1982;10:375–375.
19. Ryan JR, Salciccioli GG. Fractures of the distal radial epiphysis in adolescent weight lifters. *Am J Sports Med* 1976;4:26–26.
20. Brown EW, Kimball RG. Medical history associated with adolescent powerlifting. *Pediatrics* 1983;72:636–644.
21. Regwold G. Does lifting weights harm a prepubescent athlete? *Phys Sportsmed* 1982;10:141–144.
22. American Academy of Pediatrics. Committee on sports medicine. Weight training and weight lifting: information for the pediatrician. *Phys Sportsmed* 1983;11:157–161.
23. Atkin DM, Fithian DC, Marangi KS, et al. Characteristics of patients with primary acute lateral patellar dislocation and their recovery within the first 6 months of injury. *Am J Sports Med* 2000;28(4):472–479.
24. Bensahel H, Souchet P, Pennecott GF, et al. The unstable patella in children. *J Pract Orthod (B)* 2000;9(4):265–270.
25. Busch MT. Care of the young athlete. In: Morrissy R, Weinstein S, eds. *Pediatric orthopaedics by Lovell and Winter*. Philadelphia, PA: Lippincott Williams & Wilkins, 2000.
26. Dye SF. The knee as a biologic transmission with an envelope of function: a theory. *Clin Orthop* 1996;325:10–18.
27. Warren LF, Marshall JL. The supporting structures and layers on the medial side of the knee: an anatomical analysis. *J Bone Joint Surg* 1979;61A(1):56–62.
28. Conlan T, Garth WP Jr, Lemons JF. Evaluations of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg* 1993;75-A(5):682–693.
29. Drez D, Edwards TB, Williams CS. Results of medial patellofemoral ligament reconstruction in the treatment of patellar dislocation. *Arthroscopy* 2001;17(3):298–306.
30. Rieder B, Marshall JL, Koslin B, et al. The anterior aspect of the knee joint. *J Bone Joint Surg* 1981;63A(3):351–356.
31. Desio SM, Burks RT, Bachus KN. Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med* 1998;26(1):59–65.
32. Hautanaa PV, Fithian DC, Kaufman KR, et al. Medial soft tissue restraints in lateral patellar instability. *Clin Orthop* 1998;349:174–182.
33. Burks RT, Desio SM, Bachus KN, et al. Biomechanical evaluation of lateral patellar dislocations. *Am J Knee Surg* 1998;11(1):24–31.
34. Nomura E. Classification of lesions of the medial patellofemoral ligament in patellar dislocation. *Int Orthop* 1999;23(5):260–263.
35. Hinton RY, Sharma KM. Acute and recurrent patellar instability in the young athlete. *Orthop Clin North Am* 2003;34:385–396.
36. Larsen E, Lauridsen F. Conservative treatment of patellar dislocations. *Clin Orthop Relat Res* 1982;171:131–136.
37. Merchant AC, Mercer RL, Jacobsen RH, et al. Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg* 1974;56A:1391–1396.
38. Buchner M, Baudendiskel B, Sabo D, et al. Acute traumatic primary patellar dislocation: long-term results comparing conservative and surgical treatment. *Clin J Sports Med* 2005;15(2):62–66.
39. Palmer S, Kallio P, Donnell S, et al. Acute patellar dislocation in children and adolescents: a randomized clinical trial. *J Bone Joint Surg Am* 2008;90:463–470.
40. Ahmad CS, Stein BE, Matuz D, et al. Immediate surgical repair of the medial patellar stabilizers for acute patellar dislocation. A review of eight cases. *Am J Sports Med* 2000;28(6):804–810.
41. Nikku R, Nietosvaara Y, Kallio PE, et al. Operative versus closed treatment of primary dislocation of the patella. Similar 2 year results in 125 randomized patients. *Acta Orthop Scand* 1997;68(5):419–423.
42. Cash JD, Hughston JC. Treatment of acute patellar dislocation. *Am J Sports Med* 1988;16(3):244–249.
43. McManus F, Rang M, Heslin DJ. Acute dislocation of the patella in children: the natural history. *Clin Orthop* 1979;139:88.
44. Hawkins RJ, Bell RH, Annisette G. Acute patellar dislocations. The natural history. *Am J Sports Med* 1986;14(2):117–120.
45. Caylor D, Fites R, Worrell TW. The relationship between quadriceps angle and anterior knee syndrome. *J Orthop Sports Phys Ther* 1993;17(1):11–16.
46. Fairbank JC, Pynsent PB, van Poortvliet JA, et al. Mechanical factors in the incidence of knee pain in adolescents and young adults. *J Bone Joint Surg* 1984;66B(5):685–693.
47. Livingston LA, Mandigo JL. Bilateral Q angle asymmetry and anterior knee pain syndrome. *Clin Biomech* 1999;14(1):7–13.
48. Metcalf RW. An arthroscopic method for lateral release of the subluxating or dislocating patella. *Clin Ortho Relat Res* 1982;167:9.
49. Schonholtz GJ, Zahn MG, Magee CM. Lateral retinacular release of the patella. *Arthroscopy* 1987;3:269.
50. Hughston JC, Deese M. Medial subluxation of the patella as a complication of lateral retinacular release. *Am J Sports Med* 1988;16(4):383–388.
51. Nonweiler DE, Delee JC. The diagnosis and treatment of medial subluxation of the patella after lateral retinacular release. *Am J Sports Med* 1994;22(5):680–686.
52. Noyes FR, Albright JC. Reconstruction of the medial patellofemoral ligament with autologous quadriceps tendon. *J Arthrosc Rel Surgl* 2006;22(8):904. e1–7.
53. Madigan R, Wissinger HA, Donaldson WF. Preliminary experience with a method of quadricepsplasty in recurrent subluxation of the patella. *J Bone Joint Surg Am* 1975;57:600.
54. Baksi DP. Restoration of dynamic stability of the patella by pes anserinus transposition. *J Bone Joint Surg Br* 1981;63:399.
55. Insall J, Falvo KA, Wise DW. Chondromalacia patellae. *J Bone Joint Surg Am* 1976;58:1.
56. Insall J, Bullongh PG, Burstein AH. Proximal "tube" realignment of the patella for chondromalacia patellae. *Clin Orthop* 1979;144:63.
57. Baker RH, Carroll N, Dewar FP, et al. The semitendinosus tenodesis for recurrent dislocation of the patella. *J Bone Joint Surg Br* 1972;54(1):54–103.
58. Letts RM, Davidson D, Beaule P. Semitendinosus tenodesis for repair of recurrent dislocation of the patella in children. *J Pediatr Orthop* 1999;19(6):742–747.
59. Brown DE, Alexander AH, Lichtman DM. The Elmslie-Trillat procedure: evaluation in patellar dislocation and subluxation. *Am J Sports Med* 1984;12:104.
60. Fulkerson JP. Anteromedialization of the tibial tubercle for patellofemoral malalignment. *Clin Orthop* 1983;177:176.
61. Chrisma OD, Snook GA, Wilson TC. A long-term prospective study of the Hauser and Roux Goldthwait procedures for recurrent patellar dislocation. *Clin Orthop* 1979;144:27.
62. Benoit B, Laflamme GY, Laflamme GH, et al. Long-term outcome of surgically-treated habitual patellar dislocation in children with coexistent patella alta. *J Bone Joint Surg Br* 2007;89-B(9):1172–1177.
63. Kramer DE, Micheli LJ. Meniscal tears and discoid meniscus in children: diagnosis and treatment. *J Am Acad Orthop Surg* 2009;17:698–707.
64. Clark CR, Ogden JA. Development of the menisci of the human knee joint. Morphological changes and their potential role in childhood meniscal injury. *J Bone Joint Surg Am* 1983;65:538–547.
65. Greis PE, Bardana DD, Holmstrom MC, et al. Meniscal injury: I. Basic science and evaluation. *J Am Acad Orthop Surg* 2002;10:168–176.
66. Ahmed AM, Burke DL. In-vitro measurement of static pressure distribution in synovial joints—Part I: Tibial surface of the knee. *J Biomech Eng* 1983;105:216–225.
67. Busch MT. Meniscal injuries in children and adolescents. *Clin Sports Med* 1990;9:661–680.
68. Manzione M, Pizzutillo PD, Peoples AB, et al. Meniscectomy in children: a long-term follow-up study. *Am J Sports Med* 1983;11:111–115.
69. Stanitski CL, Harvell JC, Fu F, Observations on acute knee hemarthrosis in children and adolescents. *J Pediatr Orthop* 1993;13:506–510.

70. Kocher MS, Micheli LJ. The pediatric knee—evaluation and treatment. In: Insall JN, ed. *Surgery of the knee*, 3rd ed. Philadelphia, PA: Churchill Livingstone, 2001:1374–1376.
71. Flynn JM, Mackenzie W, Kolstad K, et al. Objective evaluation of knee laxity in children. *J Pediatr Orthop* 2000;20:259–263.
72. Kocher MS, Klingele K, Rassman SO, Meniscal disorders: normal, discoid, and cysts. *Orthop Clin North Am* 2003;34:329–340.
73. Kocher MS, DiCanzio J, Zurakowski D, et al. Diagnostic performance of clinical examination and selective magnetic resonance imaging in the evaluation of intraarticular knee disorders in children and adolescents. *Am J Sports Med* 2001;29:292–296.
74. Takeda Y, Ikata T, Yoshida S, et al. MRI high-signal intensity in the menisci of asymptomatic children. *J Bone Joint Surg Br* 1998;80: 463–467.
75. Weiss CB, Lundberg M, Hamberg P, et al. Non-operative treatment of meniscal tears. *J Bone Joint Surg Am* 1989;71:811–822.
76. Lohmander LS, Englund PM, Dahl LL, et al. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. *Am J Sports Med* 2007;35:1756–1769.
77. Fu F, Baratz ME. Meniscal injuries. In: DeLee J, Drez D, eds. *Orthopaedic sports medicine: principles and practice*, vol. 2. Philadelphia, PA: WB Saunders, 1994:1146–1162.
78. Baratz ME, Fu FH, Mengato R, Meniscal tears: the effect of meniscectomy and of repair on intraarticular contact areas and stress in the human knee. A preliminary report. *Am J Sports Med* 1986;14:270–275.
79. Mintzer CM, Richmond JC, Taylor J, Meniscal repair in the young athlete. *Am J Sports Med* 1998;26:630–633.
80. Noyes FR, Barber-Westin SD. Arthroscopic repair of meniscal tears extending into the avascular zone in patients younger than twenty years of age. *Am J Sports Med* 2002;30:589–600.
81. Accadbled F, Cassard X, Sales de Gauzy J, et al. Meniscal tears in children and adolescents: results of operative treatment. *J Pediatr Orthop B* 2007;16:56–60.
82. Jordan MR. Lateral meniscal variants: evaluation and treatment. *J Am Acad Orthop Surg* 1996;4:191–200.
83. Young R. The external semilunar cartilage as a complete disc. In: Cleland J, Mackey JY, Young RB, eds. *Memoirs and memoranda in anatomy*. London, UK Williams and Norgate, 1887:179–187.
84. Kaplan EB. Discoid lateral meniscus of the knee joint; nature, mechanism, and operative treatment. *J Bone Joint Surg Am* 1957:39-A: 77–87.
85. Watanabe M, Takeda S, Ikeuchi H. *Atlas of arthroscopy*, 3rd ed. Tokyo, Japan: Igaku-Shoin, 1979:75–130.
86. Klingele KE, Kocher MS, Hresko MT, et al. Discoid lateral meniscus: prevalence of peripheral rim instability. *J Pediatr Orthop* 2004;24:79–82.
87. Atay OA, Pekmezci M, Doral MN, et al. Discoid meniscus: an ultrastructural study with transmission electron microscopy. *Am J Sports Med* 2007;35:475–478.
88. Good CR, Green DW, Griffith MH, et al. Arthroscopic treatment of symptomatic discoid meniscus in children: classification, technique, and results. *Arthroscopy* 2007;23:157–163.
89. Hayashi LK, Yamaga H, Ida K, et al. Arthroscopic meniscectomy for discoid lateral meniscus in children. *J Bone Joint Surg Am* 1988;70: 1495–1500.
90. Raber DA, Friederich NF, Hefti F. Discoid lateral meniscus in children. Long-term follow-up after total meniscectomy. *J Bone Joint Surg Am* 1998;80:1579–1586.
91. Smith CF, Van Dyk GE, Jurgutis J, et al. Cautious surgery for discoid menisci. *Am J Knee Surg* 1999;12:25–28.
92. Silverman JM, Mink JH, Deutsch AL. Discoid menisci of the knee: MR imaging appearance. *Radiology* 1989;173:351–354.
93. Singh K, Helms CA, Jacobs MT, et al. MRI appearance of Wrisberg variant of discoid lateral meniscus. *AJR Am J Roentgenol* 2006;187: 384–387.
94. Atay OA, Doral MN, Leblebicioglu G, et al. Management of discoid lateral meniscus tears: observations in 34 knees. *Arthroscopy* 2003;19: 346–352.
95. Habata T, Uematsu K, Kasanami R, et al. Long-term clinical and radiographic follow-up of total resection for discoid lateral meniscus. *Arthroscopy* 2006;22:1339–1343.
96. Okazaki K, Miura H, Matsuda S, et al. Arthroscopic resection of the discoid lateral meniscus: long-term follow-up for 16 years. *Arthroscopy* 2006;22:967–971.
97. Kim SJ, Chun YM, Jeong JH, et al. Effects of arthroscopic meniscectomy on the long-term prognosis for the discoid lateral meniscus. *Knee Surg Sports Traumatol Arthrosc* 2007;15:1315–1320.
98. Wroble RR, Henderson RC, Campion ER, et al. Meniscectomy in children and adolescents. A long-term follow-up study. *Clin Orthop Relat Res* 1992;180–189.
99. Ogut T, Kesmezacar H, Akgun I, et al. Arthroscopic meniscectomy for discoid lateral meniscus in children and adolescents: 4.5 year follow-up. *J Pediatr Orthop B* 2003;12:390–397.
100. Kim JM, Bin SI. Meniscal allograft transplantation after total meniscectomy of torn discoid lateral meniscus. *Arthroscopy* 2006:22:1344–1350. e1341.
101. Luhmann SJ. Acute traumatic knee effusions in children and adolescents. *J Pediatr Orthop* 2003;23(2):199–202.
102. Skak SV, Jensen TT, Poulsen TD, et al. Epidemiology of knee injuries in children. *Acta Orthop Scand* 1987;58(1):78–81.
103. Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1970;52(8):1677–1684.
104. Oostvogel HJ, Klasen HJ, Reddingius RE. Fractures of the intercondylar eminence in children and adolescents. *Arch Orthop Trauma Surg* 1988;107(4):242–247.
105. Pellacci F, Mignani G, Valdiserri L. Fractures of the intercondylar eminence of the tibia in children. *Ital J Orthop Traumatol* 1986;12(4):441–446.
106. Ahmad CS, Stein BE, Jeshuran W, et al. Anterior cruciate ligament function after tibial eminence fracture in skeletally mature patients. *Am J Sports Med* 2001;29(3):339–345.
107. Delcogliano A, Chiossi S, Caporaso A, et al. Tibial intercondylar eminence fractures in adults: arthroscopic treatment. *Knee Surg Sports Traumatol Arthrosc* 2003;11(4):255–259.
108. Falstie-Jensen S, Sondergard Petersen PE. Incarceration of the meniscus in fractures of the intercondylar eminence of the tibia in children. *Injury* 1984;15(4):236–238.
109. Kendall NS, Hsu SY, Chan KM. Fracture of the tibial spine in adults and children. A review of 31 cases. *J Bone Joint Surg Br* 1992;74(6):848–852.
110. Rademakers MV, Kerkhoffs GM, Kager J, et al. Tibial spine fractures: a long-term follow-up study of open reduction and internal fixation. *J Orthop Trauma* 2009;23(3):203–207.
111. Toye LR, Cummings DP, Armendariz G. Adult tibial intercondylar eminence fracture: evaluation with MR imaging. *Skeletal Radiol* 2002;31(1):46–48.
112. Noyes FR, DeLucas JL, Torvik PJ. Biomechanics of anterior cruciate ligament failure: an analysis of strain-rate sensitivity and mechanisms of failure in primates. *J Bone Joint Surg Am* 1974;56(2):236–253.
113. Woo SL, Hollis JM, Adams DJ, et al. Tensile properties of the human femur-anterior cruciate ligament-tibia complex. The effects of specimen age and orientation. *Am J Sports Med* 1991;19(3):217–225.
114. Baxter MP, Wiley JJ. Fractures of the tibial spine in children. An evaluation of knee stability. *J Bone Joint Surg Br* 1988;70(2):228–230.
115. Berg EE. Pediatric tibial eminence fractures: arthroscopic cannulated screw fixation. *Arthroscopy* 1995;11(3):328–331.
116. Clanton TO, DeLee JC, Sanders B, et al. Knee ligament injuries in children. *J Bone Joint Surg Am* 1979;61(8):1195–1201.
117. Gronkvist H, Hirsch G, Johansson L. Fracture of the anterior tibial spine in children. *J Pediatr Orthop* 1984;4(4):465–468.
118. Janarv PM, Westblad P, Johansson C, et al. Long-term follow-up of anterior tibial spine fractures in children. *J Pediatr Orthop* 1995;15(1):63–68.
119. Wiley JJ, Baxter MP. Tibial spine fractures in children. *Clin Orthop Relat Res* 1990;255:54–60.
120. Willis RB, Blokker C, Stoll TM, et al. Long-term follow-up of anterior tibial eminence fractures. *J Pediatr Orthop* 1993;13(3):361–364.

121. Garcia A, Neer CS II. Isolated fractures of the intercondylar eminence of the tibia. *Am J Surg* 1958;95(4):593–598.
122. McLennan JG. Lessons learned after second-look arthroscopy in type III fractures of the tibial spine. *J Pediatr Orthop* 1995;15(1):59–62.
123. Bale RS, Banks AJ. Arthroscopically guided Kirschner wire fixation for fractures of the intercondylar eminence of the tibia. *J R Coll Surg Edinb* 1995;40(4):260–262.
124. Kocher MS, Foreman ES, Micheli LJ. Laxity and functional outcome after arthroscopic reduction and internal fixation of displaced tibial spine fractures in children. *Arthroscopy* 2003;19(10):1085–1090.
125. Lee YH, Chin LS, Wang NH, et al. Anterior tibia spine fracture in children: follow-up evaluation by biomechanical studies. *Zhonghua Yi Xue Za Zhi (Taipei)* 1996;58(3):183–189.
126. Mah JY, Adili A, Otsuka NY, et al. Follow-up study of arthroscopic reduction and fixation of type III tibial-eminence fractures. *J Pediatr Orthop* 1998;18(4):475–477.
127. Molander ML, Wallin G, Wikstad I. Fracture of the intercondylar eminence of the tibia: a review of 35 patients. *J Bone Joint Surg Br* 1981; 63-B(1):89–91.
128. Mulhall KJ, Dowdall J, Grannell M, et al. Tibial spine fractures: an analysis of outcome in surgically treated type III injuries. *Injury* 1999;30(4):289–292.
129. Mylle J, Reynders P, Broos P. Transepiphyseal fixation of anterior cruciate avulsion in a child. Report of a complication and review of the literature. *Arch Orthop Trauma Surg* 1993;112(2):101–103.
130. Jung YB, Yum JK, Koo BH. A new method for arthroscopic treatment of tibial eminence fractures with eyed Steinmann pins. *Arthroscopy* 1999;15(6):672–675.
131. Smith JB. Knee instability after fractures of the intercondylar eminence of the tibia. *J Pediatr Orthop* 1984;4(4):462–464.
132. Gholve PA, Voellmicke KV, Guven M, et al. Arthrofibrosis of the knee after tibial spine fracture in children: a report of two complicated cases. *HSS J* 2008;4(1):14–19.
133. Lombardo SJ. Avulsion of a fibrous union of the intercondylar eminence of the tibia. A case report. *J Bone Joint Surg Am* 1994;76(10): 1565–1568.
134. Kocher MS, Mandiga R, Klingele K, et al. Anterior cruciate ligament injury versus tibial spine fracture in the skeletally immature knee: a comparison of skeletal maturation and notch width index. *J Pediatr Orthop* 2004;24(2):185–188.
135. Smillie IS. *Injuries of the knee joint*, 5th ed. Edinburgh, UK: Churchill-Livingstone, 1978.
136. Kocher MS, Micheli LJ, Gerbino P, et al. Tibial eminence fractures in children: prevalence of meniscal entrapment. *Am J Sports Med* 2003;31(3):404–407.
137. Hayes JM, Masear VR. Avulsion fracture of the tibial eminence associated with severe medial ligamentous injury in an adolescent. A case report and literature review. *Am J Sports Med* 1984;12(4):330–333.
138. Robinson SC, Driscoll SE. Simultaneous osteochondral avulsion of the femoral and tibial insertions of the anterior cruciate ligament. Report of a case in a thirteen-year-old boy. *J Bone Joint Surg Am* 1981;63(8): 1342–1343.
139. Meyers MH, McKeever KF. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1959;41-A(2):209–220; discussion 220–202.
140. Zaricznyj B. Avulsion fracture of the tibial eminence: treatment by open reduction and pinning. *J Bone Joint Surg Am* 1977;59(8):1111–1114.
141. Lowe J, Chaimsky G, Freedman A, et al. The anatomy of tibial eminence fractures: arthroscopic observations following failed closed reduction. *J Bone Joint Surg Am* 2002;84-A(11):1933–1938.
142. Burstein DB, Viola A, Fulkerson JP. Entrapment of the medial meniscus in a fracture of the tibial eminence. *Arthroscopy* 1988;4(1):47–50.
143. Chandler JT, Miller TK. Tibial eminence fracture with meniscal entrapment. *Arthroscopy* 1995;11(4):499–502.
144. Mah JY, Otsuka NY, McLean J. An arthroscopic technique for the reduction and fixation of tibial-eminence fractures. *J Pediatr Orthop* 1996;16(1):119–121.
145. McLennan JG. The role of arthroscopic surgery in the treatment of fractures of the intercondylar eminence of the tibia. *J Bone Joint Surg Br* 1982;64(4):477–480.
146. Huang TW, Hsu KY, Cheng CY, et al. Arthroscopic suture fixation of tibial eminence avulsion fractures. *Arthroscopy* 2008;24(11):1232–1238.
147. Liljeros K, Werner S, Janarv PM. Arthroscopic fixation of anterior tibial spine fractures with bioabsorbable nails in skeletally immature patients. *Am J Sports Med* 2009;37(5):923–928.
148. Shepley RW. Arthroscopic treatment of type III tibial spine fractures using absorbable fixation. *Orthopedics* 2004;27(7):767–769.
149. Sharma A, Lakshmanan P, Peehal J, et al. An analysis of different types of surgical fixation for avulsion fractures of the anterior tibial spine. *Acta Orthop Belg* 2008;74(1):90–97.
150. Reynders P, Reynders K, Broos P. Pediatric and adolescent tibial eminence fractures: arthroscopic cannulated screw fixation. *J Trauma* 2002;53(1):49–54.
151. Vega JR, Irribarra LA, Baar AK, et al. Arthroscopic fixation of displaced tibial eminence fractures: a new growth plate-sparing method. *Arthroscopy* 2008;24(11):1239–1243.
152. Ahn JH, Yoo JC. Clinical outcome of arthroscopic reduction and suture for displaced acute and chronic tibial spine fractures. *Knee Surg Sports Traumatol Arthrosc* 2005;13(2):116–121.
153. Lubowitz JH, Grauer JD. Arthroscopic treatment of anterior cruciate ligament avulsion. *Clin Orthop Relat Res* 1993(294):242–246.
154. Wilfinger C, Castellani C, Raith J, et al. Nonoperative treatment of tibial spine fractures in children-38 patients with a minimum follow-up of 1 year. *J Orthop Trauma* 2009;23(7):519–524.
155. Meyers MH. Isolated avulsion of the tibial attachment of the posterior cruciate ligament of the knee. *J Bone Joint Surg Am* 1975;57(5):669–672.
156. Sullivan DJ, Dines DM, Hershon SJ, et al. Natural history of a type III fracture of the intercondylar eminence of the tibia in an adult. A case report. *Am J Sports Med* 1989;17(1):132–133.
157. Fyfe IS, Jackson JP. Tibial intercondylar fractures in children: a review of the classification and the treatment of mal-union. *Injury* 1981;13(2):165–169.
158. Keys GW, Walters J. Nonunion of intercondylar eminence fracture of the tibia. *J Trauma* 1988;28(6):870–871.
159. Vargas B, Lutz N, Dutoit M, et al. Nonunion after fracture of the anterior tibial spine: case report and review of the literature. *J Pediatr Orthop B* 2009;18(2):90–92.
160. Smith AD, Tao SS. Knee injuries in young athletes. *Clin Sports Med* 1995;14:629–650.
161. Rang M, Pring ME, Wenger DR. *Rang's children's fractures*. Philadelphia, PA: Lippincott Williams & Wilkins, 2006.
162. Aichroth PM, Patel DV, Zorrilla P. The natural history and treatment of rupture of the anterior cruciate ligament in children and adolescents. A prospective review. *J Bone Joint Surg Br* 2002;84:38–41.
163. McCarroll JR, Rettig AC, Shelbourne KD. Anterior cruciate ligament injuries in the young athlete with open physes. *Am J Sports Med* 1988;16:44–47.
164. Engebretsen L, Svenningsen S, Benum P. Poor results of anterior cruciate ligament repair in adolescence. *Acta Orthop Scand* 1988;59:684–686.
165. Kocher MS, Micheli LJ, Zurakowski D, et al. Partial tears of the anterior cruciate ligament in children and adolescents. *Am J Sports Med* 2002;30:697–703.
166. Noyes FR, Bassett RW, Grood ES, et al. Arthroscopy in acute traumatic hemarthrosis of the knee. Incidence of anterior cruciate tears and other injuries. *J Bone Joint Surg Am* 62:1980;687–695:757.
167. Eiskjaer S, Larsen ST, Schmidt MB. The significance of hemarthrosis of the knee in children. *Arch Orthop Trauma Surg* 1988;107:96–98.
168. Vahasarja V, Kinnuen P, Serlo W. Arthroscopy of the acute traumatic knee in children. Prospective study of 138 cases. *Acta Orthop Scand* 1993;64:580–582.
169. Shelbourne KD, Wilckens JH, Mollabashy A, et al. Arthrofibrosis in acute anterior cruciate ligament reconstruction. The effect of timing of reconstruction and rehabilitation. *Am J Sports Med* 1991;19: 332–336.

170. Guzzanti V, Falciglia F, Gigante A, et al. The effect of intra-articular ACL reconstruction on the growth plates of rabbits. *J Bone Joint Surg Br* 1994;76:960–963.
171. Houle JB, Letts M, Yang J. Effects of a tensioned tendon graft in a bone tunnel across the rabbit physis. *Clin Orthop Relat Res* 2001;275–281.
172. Kocher MS, Saxon HS, Hovis WD, et al. Management and complications of anterior cruciate ligament injuries in skeletally immature patients: survey of the Herodicus Society and The ACL Study Group. *J Pediatr Orthop* 2002;22:452–457.
173. Greulich WW, Pyle SI. *Radiographic atlas of skeletal development of the hand and wrist*. Stanford, CA: Stanford University Press, 1959.
174. Tanner JM, Whitehouse RH. Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Arch Dis Child* 1976;51:170–179.
175. Andersson C, Odensten M, Good L, et al. Surgical or non-surgical treatment of acute rupture of the anterior cruciate ligament. A randomized study with long-term follow-up. *J Bone Joint Surg Am* 1989;71:965–974.
176. Giove TP, Miller SJ III, Kent BE, et al. Non-operative treatment of the torn anterior cruciate ligament. *J Bone Joint Surg Am* 1983;65:184–192.
177. McDaniel WJ Jr, Dameron TB Jr. The untreated anterior cruciate ligament rupture. *Clin Orthop Relat Res* 1983;172:158–163.
178. McDaniel WJ Jr, Dameron TB Jr. Untreated ruptures of the anterior cruciate ligament. A follow-up study. *J Bone Joint Surg Am* 1980;62:696–705.
179. Kocher MS, Garg S, Micheli LJ. Physeal sparing reconstruction of the anterior cruciate ligament in skeletally immature prepubescent children and adolescents. *J Bone Joint Surg Am* 2005;87:2371–2379.
180. Liddle AD, Imbuldeniya AM, Hunt DM, et al. Transphyseal reconstruction of the anterior cruciate ligament in prepubescent children. Transepiphyseal replacement of the anterior cruciate ligament using quadruple hamstring grafts in skeletally immature patients. *J Bone Joint Surg Br* 2008;90:1317–1322.
181. Anderson AF. Transepiphyseal replacement of the anterior cruciate ligament in skeletally immature patients. A preliminary report. *J Bone Joint Surg Am* 2003;85-A:1255–1263.
182. Kocher MS, Smith JT, Zoric BJ, et al. Transphyseal anterior cruciate ligament reconstruction in skeletally immature pubescent adolescents. *J Bone Joint Surg Am* 2007;89:2632–2639.
183. Paget J. On production of some of the loose bodies in joints. *St. Barth Hosp Rep* 1870;6:1.
184. Roberts J. Osteochondritis dissecans. In: Kennedy J, ed. *The injured adolescent knee.* Baltimore, MD: Williams & Wilkins, 1979.
185. Cahill B. Treatment of juvenile osteochondritis dissecans and osteochondritis dissecans of the knee. *Clin Sports Med* 1985;4(2):367–384.
186. Green WT, Banks HH. Osteochondritis dissecans in children. *J Bone Joint Surg Am* 1953;35-A(1):26–47; passim.
187. Hughston JC, Hergenroeder PT, Courtenay BG. Osteochondritis dissecans of the femoral condyles. *J Bone Joint Surg Am* 1984;66(9):1340–1348.
188. Kocher MS, et al. Functional and radiographic outcome of juvenile osteochondritis dissecans of the knee treated with transarticular arthroscopic drilling. *Am J Sports Med* 2001;29(5):562–566.
189. Lindholm TS, Osterman K. Treatment of juvenile osteochondritis dissecans in the knee. *Acta Orthop Belg* 1979;45(6):633–40.
190. Fairbanks HT. Osteo-chondritis dissecans. *Br J Surg* 1933;21:67–82.
191. Clanton TO, DeLee JC. Osteochondritis dissecans. History, pathophysiology and current treatment concepts. *Clin Orthop Relat Res* 1982(167):50–64.
192. Milgram JW. Radiological and pathological manifestations of osteochondritis dissecans of the distal femur. A study of 50 cases. *Radiology* 1978;126(2):305–311.
193. Mubarak SJ, Carroll NC. Familial osteochondritis dissecans of the knee. *Clin Orthop Relat Res* 1979(140):131–136.
194. Mubarak SJ, Carroll NC. Juvenile osteochondritis dissecans of the knee: etiology. *Clin Orthop Relat Res* 1981;157:200–211.
195. Langenskiöld A. Can osteochondritis dissecans arise as a sequel of cartilage fracture in early childhood? An experimental study. *Acta Chir Scand* 1955;109(3–4):206–209.
196. Twyman RS, Desai K, Aichroth, PM. Osteochondritis dissecans of the knee. A long-term study. *J Bone Joint Surg Br* 1991;73(3):461–464.
197. Kennedy JC, Grainger RW, McGraw RW. Osteochondral fractures of the femoral condyles. *J Bone Joint Surg Br* 1966;48(3):436–440.
198. Rosenberg NJ. Osteochondral fractures of the lateral femoral condyle. *J Bone Joint Surg Am* 1964;46:1013–1026.
199. Smillie IS. *Injuries of the knee joint*. Edinburgh, UK: Churchill Livingstone, 1970.
200. Smillie IS. *Osteochondritis dissecans*. Baltimore, MD: Williams & Wilkins, 1960.
201. Chiroff RT, Cooke CP. Osteochondritis dissecans: a histologic and microradiographic analysis of surgically excised lesions. *J Trauma* 1975;15(8):689–696.
202. Rogers W, Gladstone H. Vascular foramina and arterial supply of the distal end of the femur. *J Bone Joint Surg Am* 1950;32:867–874.
203. Reddy AS. Evaluation of the intraosseous and extraosseus blood supply to the distal femoral condyles. *Am J Sports Med* 1998;26:415–419.
204. Ribbing S. Multiple hereditary epiphyseal disorders and osteochondrosis dissecans. *Acta Radiol* 1951;36(5):397–404.
205. Petrie PW. Aetiology of osteochondritis dissecans. Failure to establish a familial background. *J Bone Joint Surg Br* 1977;59(3):366–367.
206. Wall E, Von Stein D. Juvenile osteochondritis dissecans. *Orthop Clin North Am* 2003;34(3):341–353.
207. Hefti F, et al. Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. *J Pediatr Orthop B* 1999;8(4):231–245.
208. Wilson JN. A diagnostic sign in osteochondritis dissecans of the knee. *J Bone Joint Surg Am* 1967;49(3):477–480.
209. De Smet AA, Ilahi OA, Graf BK. Reassessment of the MR criteria for stability of osteochondritis dissecans in the knee and ankle. *Skeletal Radiol* 1996;25(2):159–163.
210. De Smet AA, Ilahi OA, Graf BK. Untreated osteochondritis dissecans of the femoral condyles: prediction of patient outcome using radiographic and MR findings. *Skeletal Radiol* 1997;26(8):463–467.
211. Dipaola JD, Nelson DW, Colville MR. Characterizing osteochondral lesions by magnetic resonance imaging. *Arthroscopy* 1991;7(1):101–104.
212. Hinshaw MH, Tuite MJ, De Smet AA. "Dem bones": osteochondral injuries of the knee. *Magn Reson Imaging Clin N Am* 2000;8(2):335–348.
213. Nawata K, et al. Anomalies of ossification in the posterolateral femoral condyle: assessment by MRI. *Pediatr Radiol* 1999;29(10):781–784.
214. Wall E, Nosir H, Smothers C. Juvenile osteochondritis dissecans of the femoral condyle: healing prognosis based on x-ray and gadolinium enhanced MRI. In: American Academy of Orthopaedic Surgeons Annual Meeting—Specialty Day, San Francisco, CA, 2001.
215. O'Connor MA, et al. Osteochondritis dissecans of the knee in children. A comparison of MRI and arthroscopic findings. *J Bone Joint Surg Br* 2002;84(2):258–262.
216. Kramer J, et al. MR contrast arthrography (MRA) in osteochondrosis dissecans. *J Comput Assist Tomogr* 1992;16(2):254–260.
217. Wall EJ, et al. The healing potential of stable juvenile osteochondritis dissecans knee lesions. *J Bone Joint Surg Am* 2008;90(12):2655–2664.
218. Aglietti P, et al. Arthroscopic drilling in juvenile osteochondritis dissecans of the medial femoral condyle. *Arthroscopy* 1994;10(3):286–291.
219. Guhl JF. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res* 1982;167:65–74.
220. Anderson AF. et al. Antegrade drilling for osteochondritis dissecans of the knee. *Arthroscopy* 1997;13(3):319–324.
221. Din R, Annear P, Scaddan J. Internal fixation of undisplaced lesions of osteochondritis dissecans in the knee. *J Bone Joint Surg Br* 2006;88(7):900–904.
222. Navarro R, et al. The arthroscopic treatment of osteochondritis dissecans of the knee with autologous bone sticks. *Arthroscopy* 2002;18(8):840–844.
223. Makino A, et al. Arthroscopic fixation of osteochondritis dissecans of the knee: clinical, magnetic resonance imaging, and arthroscopic follow-up. *Am J Sports Med* 2005;33(10):1499–1504.

224. Anderson AF, Pagnani MJ. Osteochondritis dissecans of the femoral condyles. Long-term results of excision of the fragment. *Am J Sports Med* 1997;25(6):830–834.
225. Bugbee WD. Fresh osteochondral allografts. *J Knee Surg* 2002;15(3): 191–195.
226. Convery FR, Meyers MH, Akeson WH. Fresh osteochondral allografting of the femoral condyle. *Clin Orthop Relat Res* 1991;273:139–145.
227. Gill TJ. The treatment of articular cartilage defects using microfracture and debridement. *Am J Knee Surg* 2000;13(1):33–40.
228. Levy A. Osteochondral autograft for the treatment of focal cartilage lesions. *Oper Tech Orthop Surg* 2001;11(2):108–114.
229. Mahomed MN, Beaver RJ, Gross AE. The long-term success of fresh, small fragment osteochondral allografts used for intraarticular post-traumatic defects in the knee joint. *Orthopedics* 1992;15(10):1191–1199.
230. Solheim E. Mosaicplasty in articular cartilage injuries of the knee. *Tidsskr Nor Laegeforen* 1999;119(27):4022–4025.
231. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res* 2001;391(Suppl):S362–S369.
232. Tomford WW, Springfield DS, Mankin HJ. Fresh and frozen articular cartilage allografts. *Orthopedics* 1992;15(10):1183–1188.
233. Brittberg M, Faxen E, Peterson L. Carbon fiber scaffolds in the treatment of early knee osteoarthritis. A prospective 4-year follow-up of 37 patients. *Clin Orthop Relat Res* 1994;307:155–164.
234. Peterson L, et al. Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation: results at two to ten years. *J Bone Joint Surg Am* 2003;85-A(Suppl 2):17–24.
235. Walton M, Rothwell AG. Reactions of thigh tissues of sheep to blunt trauma. *Clin Orthop* 1983;176:273–281.
236. Jackson DW, Feagin JA. Quadriceps contusions in young athletes: relation of severity of injury to treatment and prognosis. *J Bone Joint Surg Am* 1973;55:95–105.
237. Lipscomb AB, Thomas ED, Johnston RK. Treatment of myositis ossificans traumatica in athletes. *Am J Sports Med* 1976;4:111–120.
238. Rothwell AG. Quadriceps hematoma: a prospective clinical study. *Clin Orthop* 1982;171:97–103.
239. An HS, Simpson M, Gale S, et al. Acute anterior compartment syndrome in the thigh: a case report and review of the literature. *Orthop Trauma* 1987;1:180–182.
240. Norman A, Dorfman HD. Juxtacortical circumscribed myositis ossificans: evolution and radiographic features. *Radiology* 1970;96:301–306.
241. Barker HB, Beynnon BD, Renstrom P. Ankle injury risk factors in sports. *Sports Med* 1997;23:69–74.
242. Anderson SJ, Sullivan JA. *Ankle in care of the young athlete*. Rosemont, IL: AAOS and American Academy of Pediatrics, 2000:413–424.
243. Stiell IG, Greenberg GH, McKnight RD, et al. Decision rules for the use of radiography in acute ankle injuries. Refinement and prospective validation. *JAMA* 1993;269:1127–1132.
244. Dyment PG, ed. *Sports medicine: health care for young athletes*. Elk Grove Village, IL: American Academy of Pediatrics, 1991:216.
245. Anderson SJ. Evaluation and treatment of ankle sprains. *Compr Ther* 1996;22:30–38.
246. Karlsson J, Bergsten T, Lasinger O, et al. Surgical treatment of chronic lateral instability of the ankle. *Am J Sports Med* 1989;17:268–274.
247. Evans GA, Hardcastle A, Frenyo D. Acute rupture of the lateral ligaments of the ankle. *J Bone Joint Surg Br* 1984;66:209.
248. Younes C, Fowles JV, Fallaha M, et al. Long-term results of surgical reconstruction for chronic lateral instability of the ankle: comparison of Watson-Jones and Evans techniques. *J Trauma* 1988;28:1330–1334.
249. Chrisman OD, Snook GA. Reconstruction of lateral ligament tears of the ankle: an experimental study and clinical evaluation of seven patients treated by a new modification of the Elmslie procedure. *J Bone Joint Surg Am* 1969;51:904–912.
250. Brostrom L. Sprained ankles VI. Surgical treatment of "chronic" ligament ruptures. *Acta Chir Scand* 1966;132:551–565.
251. Gould N, Seligson D, Gassman J. Early and late repair of lateral ligaments of the ankle. *Foot Ankle* 1980;1:84–89.
252. Wilkerson GB, Pinerola JJ, Caturano RW. Invertor versus evertor peak torque and power deficiencies associated with lateral ligament injury. *J Orthop Sports Phys Ther* 1997;26:78–86.
253. Waters PM, Millis M. Hip and pelvic injuries in the young athlete. *Clin Sports Med* 1988;7:513.
254. Watts HG. Fractures of the pelvis in children. *Orthop Clin North Am* 1976;7:615.
255. Canale ST, King RE, Wilkins KE. Pelvic and hip fractures. In: Rockwood CA Jr, King RE, eds. *Fractures in children*. Philadelphia, PA: JB Lippincott, 1984.
256. Metzmaker JN, Pappas AM. Avulsion fractures of the pelvis. *Am J Sports Med* 1985;13:349.
257. Schlonsky J, Olix ML. Functional disability following avulsion fracture of the ischial epiphysis. *J Bone Joint Surg Am* 1972;54:641.
258. Deitch J, et al. Traumatic anterior shoulder dislocation in adolescents. *Am J Sports Med* 2003;31(5):758–763.
259. Heck CC. Anterior dislocation of the glenohumeral joint in a child. *J Trauma* 1981;21(2):174–175.
260. Hoelen MA, Burgers AM, Rozing PM. Prognosis of primary anterior shoulder dislocation in young adults. *Arch Orthop Trauma Surg* 1990;110(1):51–54.
261. Hovelius L. Anterior dislocation of the shoulder in teen-agers and young adults. Five-year prognosis. *J Bone Joint Surg Am* 1987;69(3): 393–399.
262. Rowe CR. Prognosis in dislocations of the shoulder. *J Bone Joint Surg Am* 1956;38-A(5):957–977.
263. Wagner KT Jr, Lyne ED. Adolescent traumatic dislocations of the shoulder with open epiphyses. *J Pediatr Orthop* 1983;3(1):61–62.
264. Wasserlauf BL, Paletta GA Jr. Shoulder disorders in the skeletally immature throwing athlete. *Orthop Clin North Am* 2003;34(3):427–437.
265. Matsen FA, Thomas SC, Rockwood CA. Anterior glenohumeral instability. In: Rockwood CA, Matsen FA, eds. *The shoulder*. Philadelphia, PA: WB Saunders, 1990.
266. Pagnani MJ, Galinat BJ, Warren RF. Glenohumeral instability. In: David Drez Jr, ed. *Orthopaedic sports medicine. Principles and practice*. Philadelphia, PA: WB Saunders, 1994:580–623.
267. Dameron TB, Rockwood CA. Subluxations and dislocations of the glenohumeral joint. In: Rockwood CA, Green DP, eds. *Fractures*. Philadelphia, PA: JB Lippincott, 1984.
268. Pasque CB, Maginnis DW, Yurko-Griffin L. Shoulder in care of the young athlete. In: Sullivan JA, Anderson SJ, eds. *Care of the young athlete*. Rosemont, IL: American Academy of Orthopaedic Surgeons and American Academy of Pediatrics, 2000:323–347.
269. Townsend H, et al. Electromyographic analysis of the glenohumeral muscles during a baseball rehabilitation program. *Am J Sports Med* 1991;19(3):264–272.
270. Itoi E, et al. A new method of immobilization after traumatic anterior dislocation of the shoulder: a preliminary study. *J Shoulder Elbow Surg* 2003;12(5):413–415.
271. Itoi E, et al. Position of immobilization after dislocation of the glenohumeral joint. A study with use of magnetic resonance imaging. *J Bone Joint Surg Am* 2001;83-A(5):661–667.
272. Kido T, et al. Dynamic stabilizing function of the deltoid muscle in shoulders with anterior instability. *Am J Sports Med* 2003;31(3):399–403.
273. Finestone A, et al. Bracing in external rotation for traumatic anterior dislocation of the shoulder. *J Bone Joint Surg Br* 2009;91(7):918–921.
274. Kocher MS, Waters PM, Micheli LJ. Upper extremity injuries in the paediatric athlete. *Sports Med* 2000;30(2):117–135.
275. Marans HJ, et al. The fate of traumatic anterior dislocation of the shoulder in children. *J Bone Joint Surg Am* 1992;74(8):1242–1244.
276. Schlonsky J, Olix ML. Functional disability following avulsion fracture of the ischial epiphysis. Report of two cases. *J Bone Joint Surg Am* 1972;54(3):641–644.
277. Jones KJ, et al. Functional outcomes of early arthroscopic Bankart repair in adolescents aged 11 to 18 years. *J Pediatr Orthop* 2007;27(2):209–213.
278. Bankart AS. The pathology and treatment of recurrent dislocation of the shoulder joint. *Br J Surg* 1939;26:24–38.

279. Gill TJ, et al. Bankart repair for anterior instability of the shoulder. Long-term outcome. *J Bone Joint Surg Am* 1997;79(6):850–857.
280. Koss S, Richmond JC, Woodward JS Jr. Two- to five-year followup of arthroscopic Bankart reconstruction using a suture anchor technique. *Am J Sports Med* 1997;25(6):809–812.
281. Huber H, Gerber C. Voluntary subluxation of the shoulder in children. A long-term follow-up study of 36 shoulders. *J Bone Joint Surg Br* 1994;76(1):118–122.
282. Rowe CR, Pierce DS, Clark JG. Voluntary dislocation of the shoulder. A preliminary report on a clinical, electromyographic, and psychiatric study of twenty-six patients. *J Bone Joint Surg Am* 1973;55(3):445–460.
283. Ireland ML, Andrews JR. Shoulder and elbow injuries in the young athlete. *Clin Sports Med* 1988;7(3):473–494.
284. Ireland ML, Satterwhite YE. Shoulder injuries. In: Zarins B, Andrews JR, Wilk KE, eds. *Injuries in baseball*. Philadelphia, PA: Lippincott Williams & Wilkins, 1998:271–281.
285. Oberlander MA, Chisar MA, Campbell B. Epidemiology of shoulder injuries in throwing and overhead athletes. *Sports Med Arthrosc* 2000;8:115–123.
286. Patel PR, Warner JJ. Shoulder injuries in the skeletally immature athlete. *Sports Med Arthrosc* 1996;4:99–113.
287. Bach BR Jr, et al. An unusual neurological complication of the Bristow procedure. A case report. *J Bone Joint Surg Am* 1988;70(3):458–460.
288. Richards RR, et al. Injury to the brachial plexus during Putti-Platt and Bristow procedures. A report of eight cases. *Am J Sports Med* 1987;15(4):374–380.
289. Willis RB, Galpin RD. Dislocations of the shoulder. In: Letts RM, ed. *Management of pediatric fractures.* New York , NY: Churchill Livingstone, 1994:159.
290. Aparicio G, Abril JC, Calvo E, et al. Radiologic study of patellar height in Osgood-Schlatter disease. *J Pediatr Orthop* 1997;17(1):63–66.
291. Lancourt JE, Cristini JA. Patella alta and patella infera. Their etiological role in patellar dislocation, chondromalacia and apophysitis of the tibial tubercle. *J Bone Joint Surg Am* 1975;57(8):1112–1115.
292. Krause BL, Williams JP, Catterall A. Natural history of Osgood-Schlatter disease. *J Pediatr Orthop* 1990;10(1):65–68.
293. Bolista MJ, Fitch RD. Tibial tubercle avulsions. *J Pediatr Orthop* 1986;6(2):186–192.
294. Chow SP, Lam JJ, Leong JC. Fractures of the tibial tuberosity in adolescents. *J Bone Joint Surg Am* 1990;72(2):231–234.
295. Ogden JA, Southwick WO. Osgood-Schlatter's disease and tibial tuberosity development. *Clin Orthop* 1976;116:180.
296. Crigler NW, Riddervole HO. Soft tissue changes in x-ray diagnosis of the Osgood-Schlatter lesion. *Va Med* 1982;109:176.
297. Kujala UM, Kvist M, Heinonen O. Osgood-Schlatter's disease in adolescent athletes. *Am J Sports Med* 1985;13:236.
298. Willner P. Osgood-Schlatter's disease: etiology and treatment. *Clin Orthop* 1969;62:178.
299. Bowers KD Jr. Patellar tendon avulsion as a complication of Osgood-Schlatter's disease. *Am J Sports Med* 1981;59:56.
300. Mital MA, Matza RA, Cohen J. The so-called unresolved Osgood-Schlatter lesion. *J Bone Joint Surg Am* 1980;62:732.
301. Binazzi R, Felli L, Vaccari V, et al. Surgical treatment of unresolved Osgood-Schlatter lesion. *Clin Orthop* 1993;289:202–204.
302. Ogden JA, McCarthy SM, Jokl P. The painful bipartite patella. *J Pediatr Orthop* 1982;2(3):263–269.
303. Kay JJ, Freiberger RH. Fragmentation of the lower pole of the patella in spastic lower extremities. *Radiology* 1991;101:97.
304. Medlar RC, Lyne ED. Sinding-Larsen-Johansson disease. Its etiology and natural history. *J Bone Joint Surg Am* 1978;60(8):113.
305. Dupont JY. Synovial plica of the knee. Controversies and review. *Clin Sports Med* 1997;16(1):87.
306. Apple JS, Martinez S, Hardaker WT, et al. Synovial plicae of the knee. *Skeletal Radiol* 1982;7(4):251–254.
307. Barber FA. Fenestrated medial patella plica. *Arthroscopy* 1987;3(4):253.
308. Dandy DJ. Anatomy of the medial suprapatellar plica and medial synovial shelf. *Arthroscopy* 1990;6(2):79.
309. Dupont JY. Role of synovial plicae in pathology of the knee. *Rev Chir Orthop Reparatrice Appar Mot* 1985;71(6):401.
310. Duri ZA, Patel DV, Aichroth PM. The immature athlete. *Clin Sports Med* 2002;21(3):461.
311. Amatuzzi MM, Fazzi A, Varella MH. Pathologic synovial plica of the knee. Results of conservative treatment. *Am J Sports Med* 1990;18(5):466.
312. Ewing JW. Plica: pathologic or not? *J Am Acad Orthop Surg* 1993;1(2):117.
313. Andersen E, Paulsen TD. Plica mediopatellaris-arthroscopic resection under local anaesthesia. *Arch Orthop Trauma Surg* 1986;106(1):18.
314. Flanagan JP, Trakru S, Meyer M, et al. Arthroscopic excision of symptomatic medial plica. A study of 118 knees with 1–4 years follow-up. *Acta Orthop Scand* 1994;65(4):408.
315. Hardaker WT, Whipple TL, Basset FH III. Diagnosis and treatment of the plica syndrome of the knee. *J Bone Joint Surg Am* 1980;62(2):221.
316. Johnson DP, Eastwood DM, Witherow PJ. Symptomatic synovial plicae of the knee. *J Bone Joint Surg Am* 1993;75(10):1485.
317. Maffuli N, Testa V, Vapasso G. Mediopatellar synovial plica of the knee in athletes: results of arthroscopic treatment. *Med Sci Sports Exerc* 1993;25(9):985.
318. Munziger U, Ruckstuhl J, Scherrer H, et al. Internal derangement of the knee joint due to pathologic synovial folds: the mediopatellar plica syndrome. *Clin Orthop* 1981;155:59.
319. Stanitski CL. Knee overuse disorders in the pediatric and adolescent athlete. *Instr Course Lect* 1993;42:483.
320. Patel D. Plica as a cause of anterior knee pain. *Orthop Clin North Am* 1986;17(2):273.
321. Hoffa A. The influence of the adipose tissue with regard to the pathology of the knee joint. *JAMA* 1904;43:795–796.
322. Biedert RM, Sanchis-Alfonso V. Sources of anterior knee pain. *Clin Sports Med* 2002;21(3):335–347.
323. Biedert RM, Lobenhoffer P, Latterman C, et al. Free nerve endings in the medial and posteromedial capsuloligamentous complexes: occurrence and distribution. *Knee Surg Sports Traumatol Arthrosc* 2000;8(2):68–72.
324. Biedert RM, Stauffer E, Friederich NF. Occurrence of free nerve endings in the soft tissue of the knee joint. A histologic investigation. *Am J Sports Med* 1992;20(4):430–433.
325. Kennedy JC, Alexander IJ, Hayes KC. Nerve supply of the human knee and its functional importance. *Am J Sports Med* 1982;10(6):329–335.
326. Duri ZA, Aichroth PM, Dowd G. The fat pad. Clinical observations. *Am J Knee Surg* 1996;9(2):55–66.
327. Ogilvie-Harris DJ, Giddens J. Hoffa's disease: arthroscopic resection of the infrapatellar fat pad. *Arthroscopy* 1994;10(2):184–187.
328. Shea KG, Pfeiffer R, Curtin M. Idiopathic anterior knee pain in adolescents. *Orthop Clin North Am* 2003;34:377–383.
329. Stanitski CL. Patellofemoral mechanism-anterior knee pain. In: Stanitski CL, et al., eds. *Pediatric and adolescent sports medicine*. Philadelphia, PA: WB Saunders, 1994:335–370.
330. Bentley G. Chondromalacia patellae. *J Bone Joint Surg Am* 1970;52(2):221–231.
331. Sandow MJ, Goodfellow JW. The natural history of anterior knee pain in adolescents. *J Bone Joint Surg Br* 1985;67(1):36–38.
332. Dehaven KE, Dolan WA, Mayer PJ. Chondromalacia patellae in athletes. Clinical presentation and conservative management. *Am J Sports Med* 1979;7(1):5–11.
333. Dehaven KE, Dolan WA, Mayer PJ. Chondromalacia patellae and the painful knee. *Am Fam Physician* 1980;21(1):117–124.
334. Insall JN. Patella pain syndromes and chondromalacia patellae. *Instr Course Lect* 1981;30:342–356.
335. Yates C, Grana WA. Patellofemoral pain-a prospective study. *Orthopedics* 1986;9(5):663–667.
336. Yates CK, Grana WA. Patellofemoral pain in children. *Clin Orthop* 1990;255:36–43.
337. Grelsamer RP. The nonsurgical treatment of patellofemoral disorders. *Oper Tech Sports Med* 1999;7(2):65–68.
338. Bennett JG, Stauber WT. Evaluation and treatment of anterior knee pain using eccentric exercise. *Med Sci Sports Exerc* 1986;18(5):526–530.

339. Andrish JT, Bergfeld JA, Walheim JA. A prospective study on the management of shin splints. *J Bone Joint Surg Am* 1974;56:1697–1700.
340. Bates P. Shin splints—a literature review. *Br J Sports Med* 1985;13: 365–366.
341. American Medical Association. *Subcommittee on classification of sports injuries. Standard nomenclature of athletic injuries*. Chicago, IL: American Medical Association, 1966:122–126.
342. Drez D. *Therapeutic modalities for sports injuries*. Chicago, IL: Year Book Medical Publishers, 1989.
343. Holder LE, Michael RH. The specific scintigraphic pattern of "shin splints in the lower leg": concise communication. *J Nucl Med* 1984;25:865–869.
344. Brill DR. Sports nuclear medicine. Bone imaging for lower extremity pain in athletes. *Clin Nucl Med* 1983;8:101–106.
345. Lieberman CM, Hemingway DL. Scintigraphy of shin splints. *Clin Nucl Med* 1980;5:31.
346. Lehman WL Jr. Overuse syndromes in runners. *Am Fam Physician* 1984;29:157–161.
347. Detmer DE. Chronic shin splints. Classification and management of medial tibial stress syndrome. *Sports Med* 1986;3:436–446.
348. Detmer D, Sharpe K, Sufit R, et al. Chronic compartment syndrome: diagnosis, management and outcomes. *Am J Sports Med* 1985;13(3): 162–170.
349. Logan J, Rorabeck C, Castle G. The measurement of dynamic compartment pressure during exercise. *Am J Sports Med* 1983;11(4):220–223.
350. McDermott A, Marble A, Yabsley R, et al. Monitoring dynamic anterior compartment pressures during exercise. *Am J Sports Med* 1982;10(2): 83–89.
351. Shah SN, Miller BS, Kuln JE. Chronic exertional compartment syndrome. *Am J Orthop* 2004;32(7):335–341.
352. Fronek J, Mubarek S, Hargens A, et al. Management of chronic exertional anterior compartment syndrome of the lower extremity. *Clin Orthop* 1987;220:217–227.
353. Martins M, Backaert M, Vermant G, et al. Chronic leg pain in athletes due to a recurrent compartment syndrome. *Am J Sports Med* 1984;12(2):148–151.
354. Aubrey B, Sienkiewicz P, Mankin H. Chronic exercise-induced compartment pressure elevation measured with a miniaturized fluid pressure monitor. *Am J Sports Med* 1988;16(6):610–615.
355. Matsen F, Mayo K, Sheridan G, et al. Monitoring of intramuscular pressure. *Surgery* 1976;79(6):702–709.
356. Matsen F, Winquist R, Krugmire R. Diagnosis and management of compartment syndromes. *J Bone Joint Surg* 1980;62A(2):286–291.
357. Pedowitz D, Hargens A, Mubarek S, et al. Modified criteria for the objective diagnosis of chronic compartment syndrome of the leg. *Am J Sports Med* 1990;18(1):35–40.
358. Amendola A, Rorabeck CH, Vellet D, et al. The use of magnetic resonance imaging in exertional compartment syndromes. *Am J Sports Med* 1990;18:29–34.
359. Trease L, van Every B, Bennell K, et al. A prospective blinded evaluation of exercise thallium-201 SPECT in patients with suspected chronic exertional compartment syndrome of the leg. *Eur J Nucl Med* 2001;28:688–695.
360. Mubarek S, Owen C. Double incision fasciotomy of the leg for decompression in compartment syndromes. *J Bone Joint Surg* 1977;59A:184.
361. Rorabeck C, Fowler P, Nott L. The results of fasciotomy in the management of chronic exertional compartment syndrome. *Am J Sports Med* 1988;16(3):224–227.
362. Zoltan DJ, Clancy WG, Keene JS. A new approach to snapping hip and refractory trochanteric bursitis in athletes. *Am J Sports Med* 1986;14:201.
363. Matheson GO, et al. Stress fractures in athletes. A study of 320 cases. *Am J Sports Med* 1987;15(1):46–58.
364. Awbrey BJ, Sienkiewicz PS, Mankin HJ. Chronic exercise-induced compartment pressure elevation measured with a miniaturized fluid pressure monitor. A laboratory and clinical study. *Am J Sports Med* 1988;16(6):610–615.
365. Micheli LJ. Overuse injuries in children's sports: the growth factor. *Orthop Clin North Am* 1983;14(2):337–360.
366. Micheli LJ. Stress fractures. In: Letts RM, ed. *Management of pediatric fractures*. New York, Churchill Livingstone, 1994:973.
367. Milgrom C, et al. Youth is a risk factor for stress fracture. A study of 783 infantry recruits. *J Bone Joint Surg Br* 1994;76(1):20–22.
368. Ruohola JP, Kiuru MJ, Pihlajamaki HK. Fatigue bone injuries causing anterior lower leg pain. *Clin Orthop Relat Res* 2006;444:216–223.
369. Lloyd T, et al. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med Sci Sports Exerc* 1986;18(4):374–379.
370. Hubbard TJ, Carpenter EM, Cordova ML. Contributing factors to medial tibial stress syndrome: a prospective investigation. *Med Sci Sports Exerc* 2009;41(3):490–496.
371. Giladi M, et al. Stress fractures. Identifiable risk factors. *Am J Sports Med* 1991;19(6):647–652.
372. Lysens RJ, et al. The accident-prone and overuse-prone profiles of the young athlete. *Am J Sports Med* 1989;17(5):612–619.
373. Paty JG Jr. Diagnosis and treatment of musculoskeletal running injuries. *Semin Arthritis Rheum* 1988;18(1):48–60.
374. Fredericson M, et al. Tibial stress reaction in runners. Correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. *Am J Sports Med* 1995;23(4):472–481.
375. Lee JK, Yao L. Stress fractures: MR imaging. *Radiology* 1988;169(1): 217–220.
376. Micheli LJ. Overuse injuries in children. In: Lovell WW, Winter RB, eds. *Pediatric orthopedics*. Philadelphia, PA: Lippincott, 1986:1103.
377. Walter NE, Wolf MD. Stress fractures in young athletes. *Am J Sports Med* 1977;5(4):165–170.
378. Rettig AC, et al. The natural history and treatment of delayed union stress fractures of the anterior cortex of the tibia. *Am J Sports Med* 1988;16(3):250–255.
379. Green NE, Rogers RA, Lipscomb AB. Nonunions of stress fractures of the tibia. *Am J Sports Med* 1985;13(3):171–176.
380. McKenzie DC, Taunton JE, Clement DB, et al. Calcaneal epiphysis in adolescent athletes. *Can J Appl Sport Sci* 1981;6:123.
381. Pappas AM. Elbow problems associated with baseball during childhood and adolescence. *Clin Orthop Relat Res* 1982(164):30–41.
382. Sever JW. Apophysitis of the os calcis. *NY State Med J* 1912;95: 1025–1029.
383. Micheli LJ, Ireland ML. Prevention and management of calcaneal apophysitis in children: an overuse syndrome. *J Pediatr Orthop* 1987;7:34.
384. Caine D, Howe W, Ross W, et al. Does repetitive physical loading inhibit radial growth in female gymnasts? *Clin J Sports Med* 1997;7(4):302–308.
385. Hansen NM Jr. Epiphyseal changes in the proximal humerus of an adolescent baseball pitcher. A case report. *Am J Sports Med* 1982;10(6): 380–384.
386. Lyman S, et al. Effect of pitch type, pitch count, and pitching mechanics on risk of elbow and shoulder pain in youth baseball pitchers. *Am J Sports Med* 2002;30(4):463–468.
387. Fleisig GS, et al. Kinetics of baseball pitching with implications about injury mechanisms. *Am J Sports Med* 1995;23(2):233–239.
388. Davis JT, et al. The effect of pitching biomechanics on the upper extremity in youth and adolescent baseball pitchers. *Am J Sports Med* 2009;37(8):1484–1491.
389. Fortenbraugh D, Fleisig GS, Andrews JR. Baseball pitching biomechanics in relation to injury risk and performance. *Sports Health* 2009;1:314–320.
390. Andrews JR, Fleisig GS. How many pitches should I allow my child to throw? *USA Baseball News* 1996;4:5.
391. Gerbino PG. Elbow disorders in throwing athletes. *Orthop Clin North Am* 2003;34(3):417–426.
392. Lyman S, et al. Longitudinal study of elbow and shoulder pain in youth baseball pitchers. *Med Sci Sports Exerc* 2001;33(11):1803–1810.
393. Andrews JR, Fleisig GS. How many pitches should I allow my child to throw? *USA Baseball News* April, 1996.
394. Dun S, et al. A biomechanical comparison of youth baseball pitches: is the curveball potentially harmful? *Am J Sports Med* 2008;36(4): 686–692.
395. Olsen SJ II, et al. Risk factors for shoulder and elbow injuries in adolescent baseball pitchers. *Am J Sports Med* 2006;34(6):905–912.

396. Laurent LE, Lindstrom BL. Osteochondrosis of the capitulum humeri: Panner's disease. *Acta Orthop Scand* 1956;26(2):111–119.
397. Panner HA. A peculiar affection of the capitulum humeri resembling Calvé-Perthes disease of the hip. *Acta Radiol* 1929;10:234–242.
398. Ganley TJ, et al. Overuse injuries to the physes in young athletes: a clinical and basic science review. *Univ Penn Ortho J* 1998;11(Spring):27–35.
399. Mihara K, et al. Nonoperative treatment for osteochondritis dissecans of the capitellum. *Am J Sports Med* 2009;37(2):298–304.
400. Iwasaki N, et al. Autologous osteochondral mosaicplasty for osteochondritis dissecans of the elbow in teenage athletes. *J Bone Joint Surg Am* 2009;91(10):2359–2366.
401. Iwasaki N, et al. Sequential alterations in magnetic resonance imaging findings after autologous osteochondral mosaicplasty for young athletes with osteochondritis dissecans of the humeral capitellum. *Am J Sports Med* 2009;37(12):2349–2354.
402. Iwasaki N, et al. Donor site evaluation after autologous osteochondral mosaicplasty for cartilaginous lesions of the elbow joint. *Am J Sports Med* 2007;35(12):2096–2100.
403. Davis JT, et al. Dual direct lateral portals for treatment of osteochondritis dissecans of the capitellum: an anatomic study. *Arthroscopy* 2007;23(7):723–728.
404. Pill SG, et al. Osteochondritis dissecans of the capitellum: arthroscopic-assisted treatment of large, full-thickness defects in young patients. *Arthroscopy* 2003;19(2):222–225.
405. Andrews JR. Bony injuries about the elbow in the throwing athlete. *Instr Course Lect* 1985;34:323–331.
406. Lindholm TS, Osterman K, Vankka E. Osteochondritis dissecans of elbow, ankle and hip: a comparison survey. *Clin Orthop Relat Res* 1980(148):245–253.
407. Morrey BF, An KN, Stormont TJ. Force transmission through the radial head. *J Bone Joint Surg Am* 1988;70(2):250–256.
408. Werner SL, et al. Biomechanics of the elbow during baseball pitching. *J Orthop Sports Phys Ther* 1993;17(6):274–278.
409. Kocher MS. Discoid lateral meniscus saucerization and repair. In: Micheli LJ, Kocher MS, eds. *The pediatric and adolescent knee*, 1st ed. Philadelphia, PA: Elsevier, 2006:264–271.
410. Aoki Y, et al. Magnetic resonance imaging in stress fractures and shin splints. *Clin Orthop Relat Res* 2004;421:260–267.
411. Nissen CW, et al. A biomechanical comparison of the fastball and curveball in adolescent baseball pitchers. *Am J Sports Med* 2009;37(8): 1492–1498.