Physeal Fractures About the Knee

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Abstract

The knee is the most common site of injury in childhood sports, and with increased participation in organized sports, the potential for knee injuries has accordingly increased. The epiphyses and apophyses provide a site of injury unique to the immature patient. The distal femoral and proximal tibial physes and the tubercle apophysis respond differently to acute and repetitive load and often provide less resistance to traumatic forces than do surrounding ligament and bone. Treatment of displaced physeal fractures about the knee remains one of the more difficult problems in orthopaedics. Even with appropriate conservative or surgical treatment, a successful outcome is not ensured. The Salter-Harris classification system provides general guidelines regarding the risk of growth disturbance, but there are no clinical methods for quantifying the true extent of physeal damage in an acute injury. Ultimately, the value of a treatment method must be evaluated on the basis of not only the restoration of articular congruity and physeal anatomy but also the restoration of physeal function, as evidenced by the continuation of normal growth. The mechanism of injury, clinical evaluation, treatment, and outcomes for all epiphyseal injuries about the knee are discussed, as well as differences from adult injuries.

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The growth of club, recreational, and school sports has allowed more children to participate in sports than ever before.[1,2] With this increased participation comes increased potential for injury, and the knee is the most common site of injury in childhood sports.[2] Although the anatomy of the child's knee is similar to that of the adult's, the distal femoral and proximal tibial physes and tubercle apophysis present a unique potential for injury. These structures respond differently to acute and repetitive load and often provide less resistance to traumatic forces than do surrounding ligament and bone.

The purpose of this article is to review fractures of the physes, including the distal femoral physis, the proximal tibial physis, the tibial tubercle apophysis, and the intercondylar eminence. For each injury, the discussion of the evaluation and treatment will highlight the biomechanical and therapeutic differences from the adult knee.

General Considerations

Anatomy, Physiology, and Biomechanics

The distal femoral and proximal tibial physes are the largest and fastest-growing physes.[1,3,4] Physes are composed of four zones: the germinal zone, the zone of proliferation, the zone of hypertrophy, and the zone of provisional calcification. Although fractures usually occur through or near the zone of provisional calcification, the level of fracture varies according to the unique shape of the physeal plate, the amount of axial compression at the time of injury, and the obliquity of the forces causing the fracture.[4]

The frequency of growth deformities after physeal fractures is explained by three factors. First, the effect of growth arrest is exaggerated due to the rapid local growth in the injured bone. Second, injury tends to occur during adolescence, when growth is accelerated. Third, the physes are irregularly shaped, which enhances their stability but also predisposes them to fracture through more than one zone.[3]

Classification of Fractures

Fractures are typically classified according to the system proposed by Salter and Harris (Fig. 1).[5] In type I fractures, the fracture line traverses the physis, staying entirely within it. Type II injuries are the most common. The fracture line traverses the growth plate for a variable length and then exits obliquely through the metaphysis. Type III fractures also

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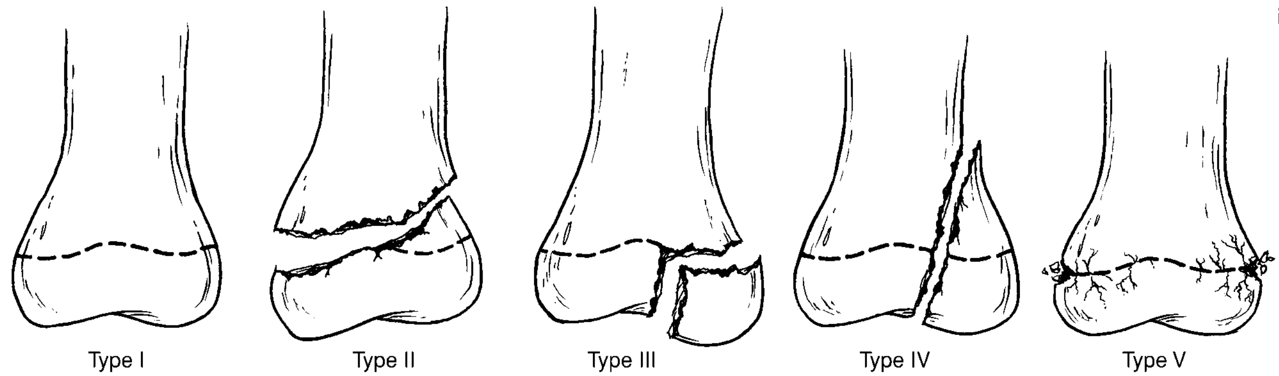

Fig. 1 Classification of epiphyseal fractures according to the Salter-Harris system.[5] In type I fractures, the fracture line traverses the physis, staying entirely within it. In type II injuries the fracture line traverses the growth plate for a variable length and then exits obliquely through the metaphysis. Type III fractures also begin in the physis but exit through the epiphysis toward the joint. Type IV fractures involve a vertical split of the epiphysis, physis, and metaphysis. Type V fractures are crush injuries to the physeal plate.

begin in the physis but exit through the epiphysis toward the joint. Type IV fractures involve a vertical split of the epiphysis, physis, and metaphysis. Type V fractures are crush injuries to the physeal plate and usually are apparent only in retrospect.[5]

Treatment Principles

Treatment of physeal fractures follows several principles based on the unique physiology of the physis. Manipulation of a physeal fracture should be gentle to avoid further damage to the physis. In the tense and apprehensive child, general anesthesia provides the best relaxation and consequently allows the most gentle manipulation. Physeal fractures heal rapidly, with healing times generally half those for nonphyseal fractures in a patient of the same age. Weight-bearing on healing physeal fractures is discouraged because the physeal cartilage may be further damaged.

Fractures of the Distal Femoral Physis

Mechanism of Injury

Historically, the distal femoral physis has been recognized as a relatively common site of bone injury in children. In the 19th century, the wagon-wheel hyperextension injury of the distal femoral physis frequently produced neurovascular injury and severe deformity. Over time, the occurrence and complications associated with this fracture have decreased, and vehicular trauma and sports have become the most common causes of injury.

Excluding birth fractures, there are two types of distal femoral physeal fractures. Juvenile-type injuries (patient age, 2 to 10 years) more commonly follow a high-energy injury, such as occurs in a motor vehicle accident.[3,6] Common associated injuries include other extremity fractures, local vascular injuries, and intra-abdominal and intrathoracic injuries. Adolescent-type injuries (patient age, 11 years and older) frequently present after a low-energy injury, which is often sports-related.[3,6] The thinner perichondrial ring in the older age group provides less resistance to fracture.

The distal femoral physeal fracture is usually a Salter-Harris type II injury with displacement in the coronal plane.[1,4] A valgus force produces medial physeal separation, often with associated sprain of the medial collateral ligament. The force traverses the physis and exits laterally with an oblique fracture through the metaphysis. A hyperextension mechanism of injury, though rare, can be responsible, which is important because of the potential for neurovascular injury at the posterior edge of the displaced metaphysis.

Presentation and Evaluation

A patient with a distal femoral physeal fracture presents with a large hemarthrosis and local swelling of the extremity. There is circumferential tenderness over the physis and crepitus with motion. The prominence of the metaphyseal edge causes a deformity that is easily palpable. In the hyperextension injury, there is often anterior skin dimpling. Apparent laxity of the knee corresponds to that which would be seen in an adult ligamentous injury.

Evaluation should include a thorough examination for associated injuries as well as a careful appraisal of the knee. Anteroposterior, lateral, and oblique radiographs are required to evaluate the fracture. Stress radiographs may be needed to evaluate instability of the knee when plain radiographs appear normal, unless the instability is clearly of ligamentous origin. Care should be exercised with stress testing to prevent additional injury to the growth plate. In displaced hyperextension injuries, a careful neurovascular examination is necessary. An arteriogram is recommended if there is evidence of vascular compromise on clinical examination. If arteriography is not indicated, careful monitoring with Doppler or another noninvasive modality is recommended.

Treatment

The treatment of distal femoral physeal fractures is dictated by the fracture type and the amount of displacement. Nondisplaced fractures are immobilized in a long-leg cast or hip spica, depending on the patient's body type and the ability to maintain fracture immobilization.[3,6,7] Obese patients typically require more extensive immobilization. Knee position is dictated by the direction of fracture displacement. Posteriorly displaced fractures are immobilized in full extension, and anteriorly displaced fractures are immobilized in 30 degrees of flexion.

Displaced Salter-Harris type I or II injuries are gently manipulated with closed technique and immobilized in a cast.[3,6,7] Such injuries are usually stable when reduced. Riseborough et al[3] reported that 80% of Salter-Harris type II injuries can be managed conservatively. If the fracture is unstable, percutaneous smooth-pin fixation under fluoroscopic imaging is recommended. Preferably, the hardware should not cross the physis. However, displaced unstable type I injuries and unstable type II injuries with small metaphyseal fragments require transphyseal pins. Displaced type I and II fractures that cannot be acceptably reduced closed should be treated with open reduction.[1,3,4,6] The usual reason for irreducibility is trapping of a large medial periosteal flap in the fracture site.

Displaced type III and IV fractures can occasionally be successfully reduced by closed techniques and can be pinned percutaneously parallel to the physis. In the vast majority of cases, open reduction and internal fixation with smooth wires is required to restore articular congruity.[1,3,4,6]

Although damage to the physis at the time of injury is the most common cause of growth arrest, failure to obtain adequate alignment or loss of the initial reduction can also lead to growth disturbance or angular deformity.[4] Consequently, careful radiographic follow-up is recommended for all fractures. In the adolescent patient, fractures can be manipulated up to 10 days following injury, and repeat reduction is possible 1 week after a primary attempt.[1]

Outcome

Distal femoral physeal fractures generally heal predictably, but problems with angular deformity and shortening are more common than might be expected even after an anatomic reduction. Significant shortening is a consequence of 30% to 50% of adolescent fractures and of more than 50% of juvenile fractures.[3,4,7] Risk factors for these complications include metaphyseal displacement of greater than half the shaft diameter and relative skeletal immaturity.[4] Although growth arrest is noted in 6 to 12 months in most cases, final evaluation 2 years after the injury is recommended to identify atypical growth problems. Leg-length inequalities of less than 2 cm are usually managed conservatively. Larger discrepancies require contralateral epiphysiodesis or, rarely, an ipsilateral limb-lengthening procedure.

Angular deformity is caused by malunion or asymmetrical growth. An angular deformity greater than 5 degrees may develop in a third of patients with distal femoral physeal fractures.[2-4,7] Usually, local growth arrest occurs opposite the metaphyseal (Thurston-Holland) fragment.[5] In the common valgus injury, this causes medial growth arrest with a subsequent varus deformity. Treatment options include excision of the osseous bridge in a smaller lesion in a growing child or osteotomy in a larger lesion in a child approaching skeletal maturity.

In cases of neurovascular injury, the outcome is dependent on timely initial treatment. Peroneal nerve injury is managed by early closed manipulation of the fracture. Observation with late nerve exploration is indicated if there is no recovery. Popliteal artery injuries that are diagnosed and treated rapidly usually present no long-term problems. Reperfusion compartment syndrome must be considered, and fasciotomy may be necessary. Circumferential immobilization is to be avoided initially.

Fractures of the Proximal Tibial Physis

Mechanism of Injury

Although knee injuries in children are quite common, proximal tibial physeal injuries are not, representing fewer than 1% of all physeal injuries.[2,6] The physis is protected by ligamentous attachments on the proximal tibia distal to the epiphysis and by the presence of the proximal tibiofibular joint, which buttresses the physis. Proximal tibial physeal fracture usually occurs as a result of indirect force, with the most common mechanism of injury being a valgus force that produces a Salter-Harris type II fracture and an associated greenstick fracture of the fibula.[1,6,8,9] The physis opens medially, often with concomitant injury to the medial collateral ligament. The fracture line then traverses the physis and exits the lateral metaphysis, creating an oblique fracture.

As occurs with fractures of the distal femoral physis, hyperextension injuries occasionally involve the potential for serious injury. Posterior displacement of the metaphyseal fragment into the popliteal fossa also can cause neurovascular injury. The popliteal artery is tethered to the posterior tibial metaphysis by its articular branches, and the metaphyseal edge causes local compression or laceration of the artery in this position.

Presentation and Evaluation

Proximal tibial physeal fractures occur most commonly in boys aged 12 to 14 years.[8-10] The signs and symptoms include pain, swelling, hemarthrosis, and inability to bear weight. The displaced metaphyseal edge may be palpable subcutaneously, and in hyperextension injuries skin dimpling can be seen over the anterior metaphysis. Stress radiographs are useful when there is a question about the origin of the medial laxity (Fig. 2). Two thirds of these fractures are Salter-Harris type I or II injuries.

Fig. 2 Stress radiograph demonstrating physeal separation without joint-space widening.

Treatment

Salter-Harris type I and II injuries are treated with closed manipulation and application of a long-leg cast.[8-10] A type II fracture is usually stable after manipulation; when unstable, percutaneous metaphyseal pinning is indicated. Ideally, smooth pins are placed through the metaphyseal fragment parallel to the physis. If the fragment is too small, however, transphyseal pins may be required. When closed manipulation fails, open reduction is indicated.[8-10] A periosteal flap interposed in the fracture site prevents reduction of the fracture. Open reduction with pin fixation and noncircumferential immobilization are then necessary. Radiographs are required to detect early or late displacement of a satisfactorily reduced fracture.

Salter-Harris type III and IV injuries are usually treated with open reduction and internal fixation.[8-10] As with any intra-articular fracture, correction of articular incongruency is mandatory. Minimally displaced fractures are often quite unstable, and internal fixation should be considered even in these fractures.[8] As in treating a distal femoral fracture, smooth pins parallel to the physis are used whenever possible. If nonoperative treatment is elected, the reduction must be carefully followed up for displacement.

Outcome

Proximal tibial physeal fractures heal predictably, and nonunions are rare. Problems with shortening and angulation are less common than with distal femoral injuries because of the smaller contribution of the proximal tibial physis to overall limb growth. Limb-length inequality of more than 1 cm can be expected in 20% of patients.[8] As with the distal femur, growth arrest is more common than elsewhere, even in Salter-Harris type I and II fractures.[8,9] Treatment is by contralateral epiphysiodesis or ipsilateral lengthening, depending on the amount of shortening that must be overcome to achieve leg-length equality.

Angular deformity occurs due to asymmetrical growth arrest or malunion. This is treated with physeal bar excision if less than 50% of the physis is involved. Osteotomy is required when a larger lesion is present.[6,11]

Contaminated open fractures of the proximal tibial physis are a particular problem.[10] These open fractures are frequently the result of lawn-mower injuries. Poor results in terms of growth arrest and angulation are more common in younger patients and in those with a greater magnitude of injury.[9]

Fractures of the Tibial Tubercle Apophysis

Mechanism of Injury

Acute isolated fracture of the tibial tubercle is rare, despite the frequency of chronic injury in this anatomic region. As in the juvenile Tillaux fracture, the differential rate of physeal closure of the tubercle apophysis plays an important role in the tubercle fracture. In the period of preclosure of the physis (i.e., between the ages of 12 and 16 years), the apophysis matures, and columnar hypertrophic cells become dominant.[12,13] The columnar cells do not resist traction as well as their fibrocartilaginous precursors. A severe concentric or eccentric contraction of the extensor mechanism produces a fracture through this level of the apophysis.[12-15]

Acute apophyseal fracture must be distinguished from Osgood-Schlatter disease. Although both conditions involve traction force through the immature physis, the rate of loading and the magnitude of the force are different. Osgood-Schlatter disease is a chronic avulsion of the anterior surface of the tubercle, whereas tubercle fracture is an acute avulsion through the apophysis.[13] Several reports suggest that Osgood-Schlatter disease may predate apophyseal fracture in up to 20% of patients.[13-15] Ogden et al[13] have suggested a biomechanical link between these conditions, but no study has documented this hypothesis definitively.

Ogden et al[13] developed a classification system for tubercle fracture. In that system, fractures are divided into three types on the basis of the severity of the displacement (Fig. 3). Type I is a distal fracture through the physis with breakout through the secondary ossification center. Type II is a more proximal fracture through the cartilage bridge between the ossification center of the tubercle and the proximal tibial physis. A type III fracture propagates upward through the proximal tibial physis into the knee. Type I fractures are the most common, but the exact incidence of this injury is not clear because of cross-classification with proximal tibial physeal fractures.[3,13]

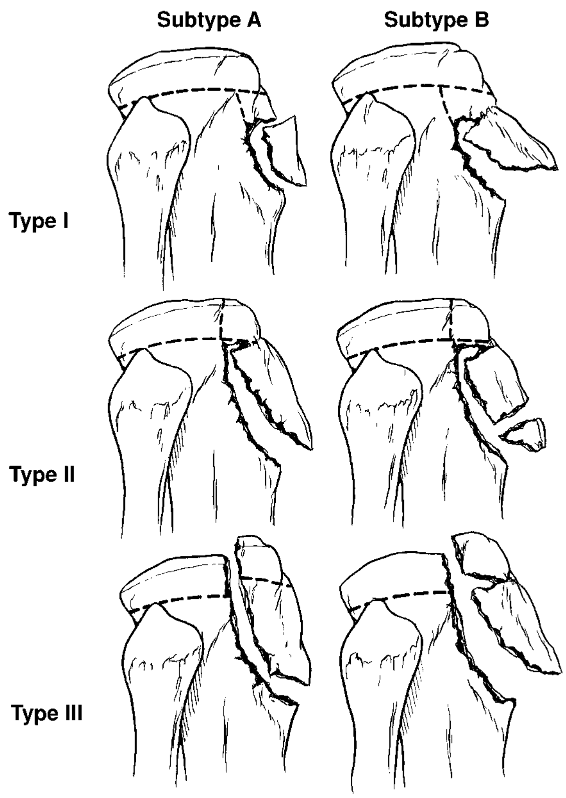

Fig. 3 Classification of tibial tubercle fractures according to the system developed by Ogden et al.[13] Type I is a distal fracture through the physis with breakout through the secondary ossification center. Type II is a more proximal fracture through the cartilage bridge between the ossification center of the tubercle and the proximal tibial physis. A type III fracture propagates upward through the proximal tibial physis into the knee. Subtype A fractures are noncomminuted; subtype B fractures are comminuted.

Presentation and Evaluation

The patient presents with a history of acute injury during participation in a jumping sport, such as basketball. The patient is usually a boy aged 13 to 16 years.[2,13,15] The knee is held flexed due to hemarthrosis, hamstring spasm, and, in type III fractures, discontinuity in the extensor mechanism. There is a palpable osseous deformity. Patella alta is observed in fractures with significant displacement. Active knee extension is possible in type I fractures but not in type II or III injuries.[13,15]

Evaluation should include anteroposterior, lateral, and oblique radiographs of the knee. The lateral view is important for classification. Oblique views may demonstrate unexpected propagation of the fracture into the knee.

Treatment

Nondisplaced type I injuries are treated with a cylinder cast with the knee in full extension for 4 to 6 weeks.[13,15] Displaced type I, II, and III fractures are usually treated with open reduction, internal fixation, and cast immobilization in extension for 6 weeks.[1,2,13,15] Open reduction often reveals a periosteal flap interposed in the fracture site. This flap is removed and repaired with direct suturing after fixation of the osseous injury. Fixation options include the use of a screw or tension-band wire and direct suture.[1,13-15] Anatomic reduction decreases the possibility of patella baja or patella alta resulting from inadvertent shortening or lengthening of the extensor mechanism.

Outcome

Complications following a tibial apophyseal fracture are uncommon. The fracture heals well when treated by epiphysiodesis. Usually, no leg-length disparity or recurvatum deformity occurs, since the injury happens in the age group of patients who are undergoing apophyseal closure.[13,15] Prominence of the tubercle with local sensitivity and patellofemoral pain due to patella alta or patella baja may occur.

Fractures of the Intercondylar Eminence

Mechanism of Injury and Classification

Intercondylar eminence fractures occur in patients under 15 years of age.[1,15] The usual mechanism is a hyperextension force. The anterior cruciate ligament (ACL) attaches to the tibia on and between the medial and lateral tibial spines in the skeletally immature person. The tibial attachment of the ACL is broad and offers greater resistance to tensile load than does the intercondylar bone itself.[1] Consequently, hyperextension and rotational forces that would cause ACL disruption in the adult often cause an intercondylar eminence avulsion fracture in the child. Although eminence fractures remain more common, isolated ACL disruptions are now recognized in immature athletes. In eminence fractures, ultimate failure occurs through bone, but interstitial ACL injury and stretching are recognized as significant components of this injury.[16-18]

Meyers and McKeever[19] proposed the classification of this injury (Fig. 4). Type I fractures are nondisplaced. Type II fractures are partially displaced and are often hinged posteriorly. Type III fractures are displaced completely. Type IV fractures are displaced and rotated. Interposition of the transverse meniscal ligament can be noted in type II and type III fractures.[1]

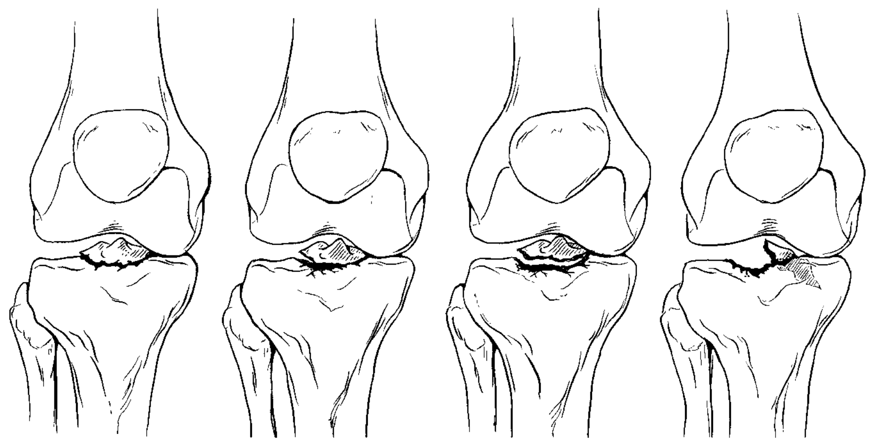

Fig. 4 Classification of intercondylar eminence fractures according to the system of Meyers and McKeever.[19] Type I fractures are nondisplaced. Type II fractures are partially displaced and are often hinged posteriorly. Type III fractures are displaced completely. Type IV fractures are displaced and rotated.

Presentation and Evaluation

Tibial eminence fractures occur in children and adolescents aged 8 to 15 years and are often associated with a fall. A young child with a hemarthrosis after a fall from a bicycle should be considered to have an eminence fracture until proved otherwise.[19] The knee is flexed due to hemarthrosis and hamstring spasm. The Lachman test is positive if hamstring spasm can be overcome. Associated injuries often include medial collateral ligament injury and ipsilateral extremity fracture.

Routine radiographs will demonstrate an eminence fracture. Visualization of fracture displacement is best on lateral views. Oblique radiographs aid in determining extension of the fracture into the plateau. Radiographs are often deceptive in the young patient, as the eminence may be largely cartilaginous. The visualization of small flecks of bone in the notch of a young patient should raise suspicion of an eminence fracture. Magnetic resonance imaging or stress radiography, possibly with the patient under general anesthesia, is recommended for evaluation.

Treatment

Treatment of intercondylar eminence fractures varies according to the fracture type. Type I injuries are treated by immobilization in a cylinder cast in 0 to 20 degrees of flexion.[1,17,19] Slight flexion relaxes ACL tension on the fragment, thus theoretically decreasing the risk of displacement of a nondisplaced fracture. However, this position may contribute to development of an extensor lag.

Treatment of type II injuries is controversial. Open reduction is recommended if closed manipulation with the knee extended does not reduce the fragment.[1] Open reduction allows anatomic restoration and prevents displacement of the fracture during immobilization. Fixation options include direct suturing with a heavy absorbable suture. Suture fixation may cross the physis. Arthroscopically assisted open reduction and internal fixation[16] is now an alternative to traditional arthrotomy.

Type III and type IV eminence fractures are always treated by open reduction and internal fixation.[1,17-19] Postoperative immobilization in a cylinder cast in extension for 6 to 8 weeks is recommended in all cases.

Outcome

Outcomes after intercondylar eminence fractures are variable. Extensor lag, though usually unrecognized by the patient, has been noted in 10% to 100% of cases.[1,16,18] Immobilization in flexion may contribute to this problem. Nonunion of displaced fractures treated nonoperatively has been reported.[6] Untreated eminence fracture results in instability secondary to malunion or nonunion of the fracture or ACL elongation at the time of injury.[1,2,16,17] A third of patients with malunion or nonunion present with instability complaints, while two thirds decrease activity to minimize symptoms.[2] Anatomic reduction and internal fixation produces predictable healing, but instability is not eliminated.

Several reports have documented late anterior laxity as well as clinical instability despite anatomic fracture healing.[16-18] Plastic deformation of the ACL prior to the eminence avulsion has been implicated in this situation. Late laxity varies according to the severity of the initial injury. Type I injury produces no late laxity, while type II and type III injuries commonly produce more than 4 mm of anterior laxity.[16] Residual collateral laxity can also be seen secondary to associated collateral injury.

Summary

Treatment of displaced physeal fractures about the knee remains one of the more difficult problems in orthopaedics. Even with appropriate conservative or surgical treatment, a successful outcome is not ensured. Unfortunately, the damage to the physis at the time of injury is a variable over which the surgeon has no control. The Salter-Harris classification of a fracture provides general guidelines regarding the risk of growth disturbance, but there are no clinical methods for quantifying the true extent of physeal damage in an acute injury. Thus, outcomes remain somewhat unpredictable. Ultimately, the value of a treatment method must be evaluated on the basis of not only the restoration of articular congruity and physeal anatomy but also the restoration of physeal function, as evidenced by the continuation of normal growth.

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