



NOTE: To view the article with Web enhancements, go to:

<http://www.medscape.com/Medscape/OrthoSportsMed/journal/2000/v04.n02/mos5701.ryu/mos5701.ryu-01.html>.

Meniscal Lesions: Diagnosis and Treatment

Robert S. P. Fan, MD, Richard K. N. Ryu, MD

[Medscape Orthopaedics & Sports Medicine 4(2), 2000. © 2000 Medscape, Inc.]

Abstract

Lesions of the meniscus are commonly encountered in the practice of knee surgery. Our knowledge and understanding of the anatomy and function of the meniscus has evolved significantly over the past few decades. This, along with advances in arthroscopic surgery, have dramatically changed our surgical philosophy. Where once open total meniscectomy was the preferred treatment, efforts are now directed at meniscal preservation and even restoration. Commonly accepted treatment of meniscal disorders now includes arthroscopic partial meniscectomy, as well as meniscal repair. Currently, efforts are being studied to replace and/or regenerate the meniscus in an effort to restore function. This review intends to highlight the diagnosis and treatment of meniscal pathology.

Introduction

Meniscal lesions are among the most common knee disorders encountered by the practicing orthopaedic surgeon. Our knowledge and understanding of the meniscus has evolved significantly over the past several decades. The meniscus was once regarded as a vestigial structure that served no function, and appeared as little more than an embryologic remnant. This lack of appreciation for its function formed the basis for total meniscectomy. Advances in the knowledge of the anatomy and function of the meniscus, together with the development of arthroscopic surgery, have led to the foundation of contemporary meniscal treatment. Surgical philosophy has now matured from routine excision to preservation and even restoration. A fundamental and expanded knowledge of meniscal anatomy, biomechanics, and function is crucial to understanding meniscal pathology and treatment.

Anatomy

The menisci of the knee joint are fibrocartilaginous C-shaped disks that occupy the joint space between the femur and the tibia.

Embryologically, the menisci form from mesenchymal tissue and appear as distinct structures by the eighth to tenth week of gestational development. Initially highly cellular, the perinatal meniscus also has an abundance of blood vessels. Progressive and gradual changes occur from birth to mid-adolescence, consisting of decreasing cellularity, decreasing vascularity, and increasing collagen content. As the developing child becomes progressively more ambulatory, the collagen fibers become oriented in order to adapt to the weight-bearing stresses.^[1]

The meniscus represents fibrocartilaginous tissue composed of collagen and cells of either fibroblast or chondrocyte origin. The meniscus is approximately 75% water. The organic matrix is composed of approximately three quarters collagen, with type I collagen predominating.^[2] The collagen fibers are oriented in a characteristic fashion. The most superficial fibers are oriented radially. Most of the collagen fibers, however, are found in the deep layer and are arranged in a circumferential orientation, which follow the periphery. The radial fibers are woven between the circumferential fibers, which help to provide structural integrity. The arrangement of fibers enables them to resist the hoop stresses that are produced at the meniscus during weight bearing.^[3]

In cross section, the menisci are triangular, being thicker at the periphery and tapering to a thin free edge centrally. The superior surfaces are concave to accommodate the convexity of the femoral condyles. The medial meniscus is semilunar in shape and is thinner and narrower anteriorly. The posterior horn is thicker and wider, averaging approximately 10.6 mm.^[4] The anterior and posterior horns are attached to the intercondylar eminence with an additional slip from the posterior horn attaching to the posterior cruciate ligament. The peripheral circumference is firmly attached to the capsule by the coronary ligaments. The medial meniscus is also firmly attached to the posterior oblique ligament. The medial meniscus covers approximately 64% of the medial tibial plateau. The lateral meniscus covers approximately 84% of the lateral tibial plateau. It is more circular than the medial meniscus and is also more uniform in width (average, 12 to 13 mm).^[4]

The anterior and posterior horns of the lateral meniscus also attach to the intercondylar eminence, but in closer proximity to the anterior cruciate ligament than the medial meniscus. The peripheral attachment of the lateral meniscus to the capsule is thinner and looser than on the medial side. In addition, there is no attachment in the region of the popliteal hiatus, and there is no attachment of the lateral meniscus to the lateral collateral ligament (Figure 1).

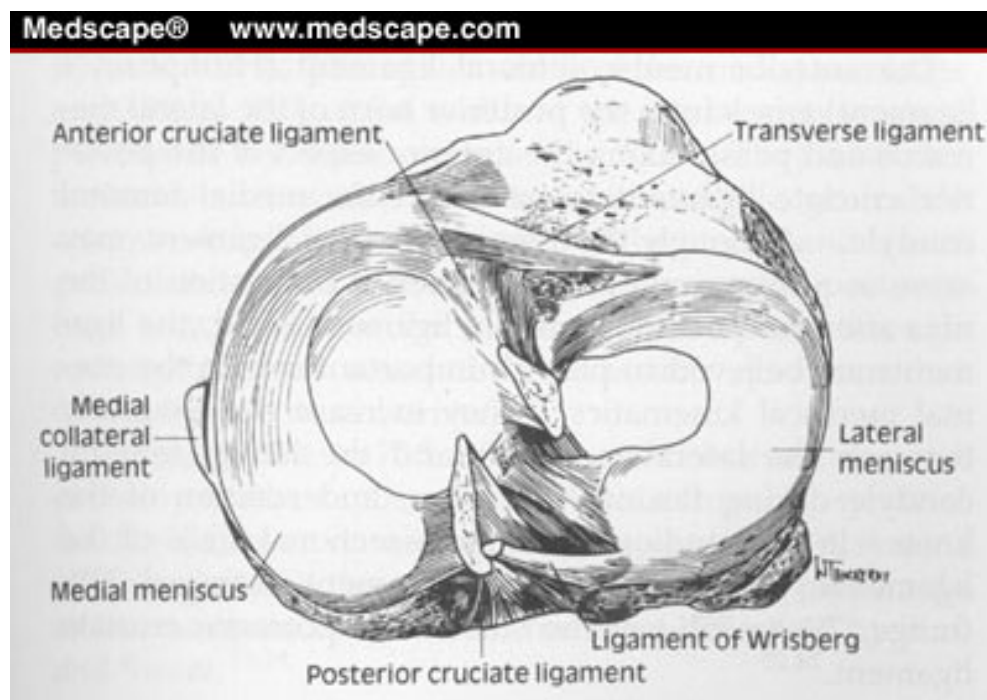


Figure 1. Schematic axial view of the tibial plateau, demonstrating the medial and lateral menisci. (Reprinted with permission.)

Warren R, Arnoczky SP, Wickiewicz TL. Anatomy of the Knee. In: Nicholas JA, Hershman EB, eds. *The Lower Extremity and Spine in Sports Medicine*. St. Louis, Mo: Mosby; 1986:657-694.

Menisiofemoral ligaments can be found in 70% of knees.^[5] These represent accessory knee ligaments that attach to the medial femoral condyle from the posterior horn of the lateral meniscus. The posterior menisiofemoral ligament of Wrisberg can be found coursing posterior to the posterior cruciate ligament. The anterior

meniscomfemoral ligament of Humphrey passes anterior to the posterior cruciate ligament. These vary considerably in size, but average 20% of the size of the posterior cruciate ligament.^[6]

The meniscus is typically an avascular structure with the primary blood supply limited to the periphery. Studies by Arnoczky and Warren have demonstrated that only the peripheral 10% to 30% of the meniscus is vascularized.^[7] These vessels are derived from the middle, medial, and lateral geniculate arteries. The inner free margin of the meniscus is avascular and is nourished by the synovial fluid through diffusion (Figure 2).



Figure 2. Frontal section of the medial compartment demonstrates the microvasculature of the medial meniscus. The perimeniscal capillary plexus (PCP) permeates through the peripheral border of the meniscus. F: Femur; T: Tibia. (Reprinted with permission.)

Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med.* 1982;10:90-95.

Biomechanics and Function

The menisci provide several integral elements to knee function. These include load transmission, shock absorption, joint lubrication, and joint nutrition and stability.

The menisci act as a structural transition zone between the femoral condyles and tibial plateau. As such, they increase the congruence between the condyles and the plateau. The menisci appear to transmit approximately 50% of the compressive load through a range of motion of 0 to 90 degrees.^[8,9] The contact area is increased, protecting articular cartilage from high concentrations of stress. The circumferential collagen fiber orientation within the meniscus is uniquely suited to this capacity. As load is applied, the menisci will tend to extrude from between the articular surfaces of the femur and tibia. In order to resist this tendency, circumferential tension is developed along the collagen fibers of the meniscus as hoop stresses. The circumferential continuity of the peripheral rim of the meniscus is integral to meniscal function. Partial meniscectomy, or bucket-handle tearing, will still preserve meniscal function so long as the peripheral rim is intact. Conversely, if a radial tear extends to the periphery and interrupts the continuity of the meniscus, the load-transmitting properties of the meniscus are lost.^[9]

Fairbank^[10] was the first to significantly appreciate the load-bearing function of the meniscus with his observations in the postmeniscectomy knee. He documented an increase in degenerative changes of the articular surface after total meniscectomy, which he attributed to loss of meniscal function. Consequently, he recognized the potential for long-term alterations in joint function and biomechanics following total meniscectomy. The tibial femoral contact area decreased by up to 75% in postmeniscectomy knees as demonstrated by Baratz and Mengator.^[11] This decrease resulted in a 235% increase in contact stresses after total meniscectomy. Ahmed and Burke^[12] found a 40% increase in contact stresses. Other reports have been quite variable, with estimates of the increase in contact stress ranging from 450% to 700%.^[8] In contrast, partial meniscectomy results in only a 10% decrease in contact area and a 65% increase in contact stress.^[11]

Joint stability is also affected by the menisci. The medial meniscus is recognized as a secondary stabilizer to anterior translation.^[13] This becomes particularly important in the anterior cruciate ligament-deficient knee, in which an increase in anterior translation after total meniscectomy has been demonstrated.^[13] Consequently, the medial meniscus is vulnerable to injury in the anterior cruciate ligament-deficient knee as it attempts to limit anterior translation. The menisci have also been demonstrated to contribute to varus/valgus stability, as well as to internal and external rotational stability.^[14,15]

Meniscal motion has been studied with 3-dimensional MRI and cinematic MRI. Medial meniscal excursion was approximately 5.1 mm, and lateral meniscal excursion, 11.2 mm. The posterior horn excursion has been noted to be less than that of the anterior horn, both medially and laterally.^[16] DePalma^[17] has demonstrated that most lateral meniscal motion occurs after 5 to 10 degrees of flexion, whereas most medial meniscal displacement occurs after 17 to 20 degrees of flexion. The posterior oblique ligament is firmly attached to the posterior medial meniscus, thereby limiting its displacement and rotation. This likely accounts for the increased risk of injury to the medial meniscus. Conversely, the relatively increased mobility of the lateral meniscus is also responsible for the more frequent occurrence of injuries on the medial side.

Diagnosis

The clinical diagnosis of a meniscal lesion depends on the insight and experience of the physician. The patient with meniscal pathology typically presents with symptoms referable to the joint line, either medially or laterally. In traumatic cases, an injury is brought on with the knee in flexion, weight bearing, followed by rotation. A pop may or may not be felt. Symptoms are frequently worsened by flexing and loading the knee, and activities such as squatting and kneeling are poorly tolerated. Patients will frequently complain of a "pop" or "clunk" sensation as the knee is brought through the range of motion.

An effusion may be present to a varying extent. Patients most frequently will have specific joint line point tenderness. Often, the examiner may appreciate a small focus of swelling or boggiess in the area of the point tenderness, particularly if the knee is in flexion. A number of tests have been described in order to appreciate meniscal pathology. Apley's test is performed with the patient prone, and with the examiner hyperflexing the knee and rotating the tibial plateau on the condyles (Figure 3). Steinman's test is performed on a supine patient by bringing the knee into flexion and rotating (Figure 4). Reproduction of specific joint line pain with either of these 2 maneuvers is considered a positive test. McMurray's test is positive if a pop or a snap at the joint line occurs while flexing and rotating the patient's knee (Figure 5). Asking patients to squat and/or duck-walk will frequently reproduce symptoms. No test is specifically pathognomonic and, therefore, a combination of provocative maneuvers should be performed. In general, clinical diagnosis is more sensitive for pathology on the medial than the lateral side.^[18-20]

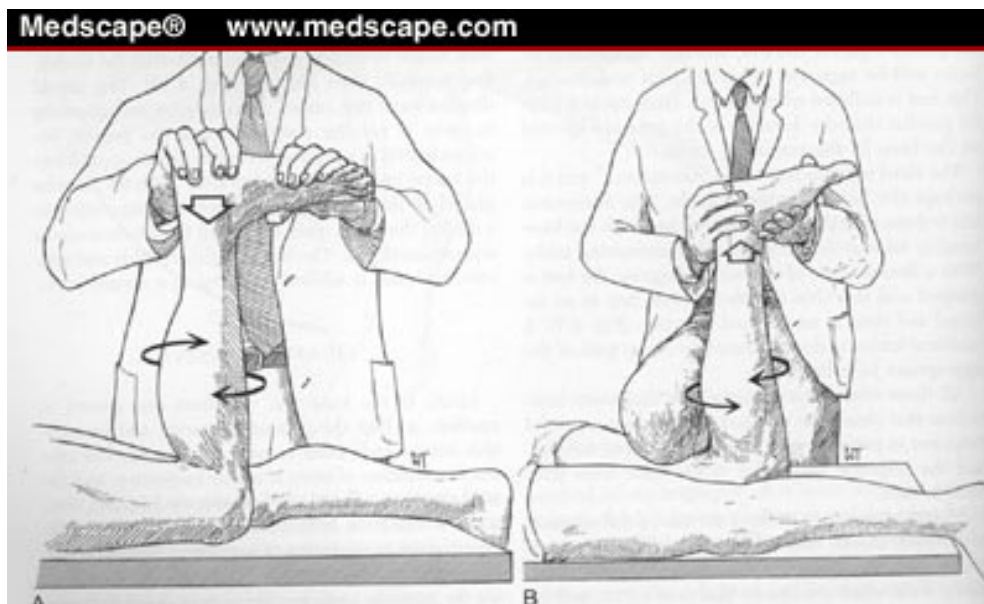


Figure 3. The Apley test is performed with patient in prone position by rotating the tibia on the femur and applying axial compression to reproduce joint line pain. (Reprinted with permission.)

Insall JN. Examination of the knee. In: Insall JN, ed. *Surgery of the Knee*. New York, NY: Churchill Livingstone; 1984:62, Figure 4.5.

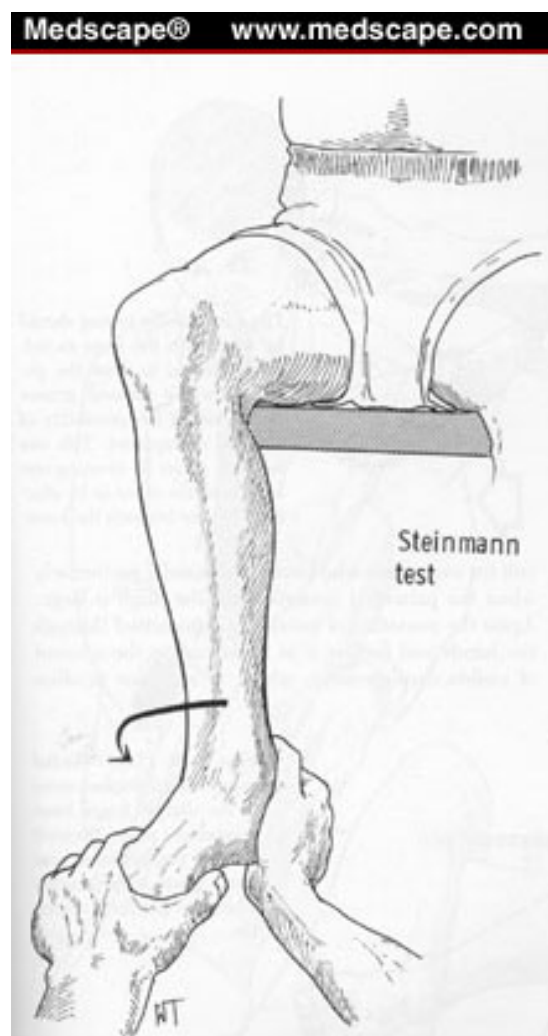


Figure 4. The Steinman test produces joint line pain when the tibia is rotated internally and externally while the knee is flexed over the examination table. (Reprinted with permission.)

Insall JN. Examination of the knee. In: Insall JN, ed. *Surgery of the Knee*. New York, NY: Churchill Livingstone; 1984:63, Figure 4.7.



Figure 5. The McMurray test is performed by flexing the patient's hip and knee and palpating for a pop or click along the joint line as the tibia is internally and externally rotated. (Reprinted with permission.)

Insall JN. Examination of the knee. In: Insall JN, ed. *Surgery of the Knee*. New York, NY: Churchill Livingstone; 1984:62, Figure 4.6.

The differential diagnosis for meniscal pathology includes patellofemoral syndromes, osteoarthritis, inflammatory arthritides, osteochondritis dissecans, medial patella plica syndrome, and osteonecrosis. In addition, collateral ligament injury, stress fracture, and localized bursitis or tendinitis can mimic meniscal pathology. Finally, referred pain from a slipped capital femoral epiphysis, degenerative hip disease, lumbar radiculopathy, or other peripheral neuropathy should also be excluded.

Imaging

Plain radiographs are generally not helpful in evaluating meniscal lesions other than to rule out other bony or joint pathology. Arthrography has been used extensively in the past with reported accuracy rates of 60% to 97%.^[21] The primary disadvantage of arthrography is its invasive nature. Arthrography today has been largely supplanted by magnetic resonance imaging (MRI), which yields accuracy rates as high as 90% to 98%.^[21,22] MRI is noninvasive and highly accurate and has a very high negative predictive value.^[23]

There is considerable variation in the methodology of knee scanning among imaging centers. Experience of the centers and variations in such equipment as surface coils and magnetic field strength can play a role in determining the imaging protocol of the individual center.

A variety of imaging sequences may be selected. These include routine spin echo, inversion, recovery, fat suppression techniques, gradient recall acquisition in the steady state (GRASS), 3-dimensional Fourier transform imaging, and radial sequences. The repetition time (TR) and echo delay time (TE) can be manipulated to determine contrast during sequencing. This results in T1, T2, and proton density weighting images. To evaluate the menisci, T1 weighting, proton density weighting, or GRASS sequencing is essential to examine the menisci. The GRASS sequencing provides effective T2 weighting. The use of a dedicated circumferential knee coil provides optimal results. The menisci appear dark or low signal intensity on T1 and T2 weighting. Fluid appears as low- to intermediate-signal on T1 and proton density weighting and becomes bright or high signal intensity on T2 and GRASS-weighting images. MRI is ideal for studying the meniscus because of the low signal intensity of the fibrocartilage of the meniscus and the high signal intensity of fluid within a tear.^[24]

The normal-appearing meniscus is uniformly low in signal in both T1- and T2-weighted images. It should appear as triangular configurations on both the coronal and sagittal images.

A grading system has been developed to describe abnormal intrameniscal signal. Grade 1 is oval or globular in appearance and does not communicate with any meniscal surface. Grade 2 signal is more linear, but similarly does not communicate with the articular surfaces. Grade 3 signals within the meniscus are linear and should communicate with either superior or inferior articular surfaces. Grades 1 and 2 signals are consistent with intrasubstance myxoid degeneration, whereas grade 3 signal is consistent with a tear^[24] (Figures 6A,B).

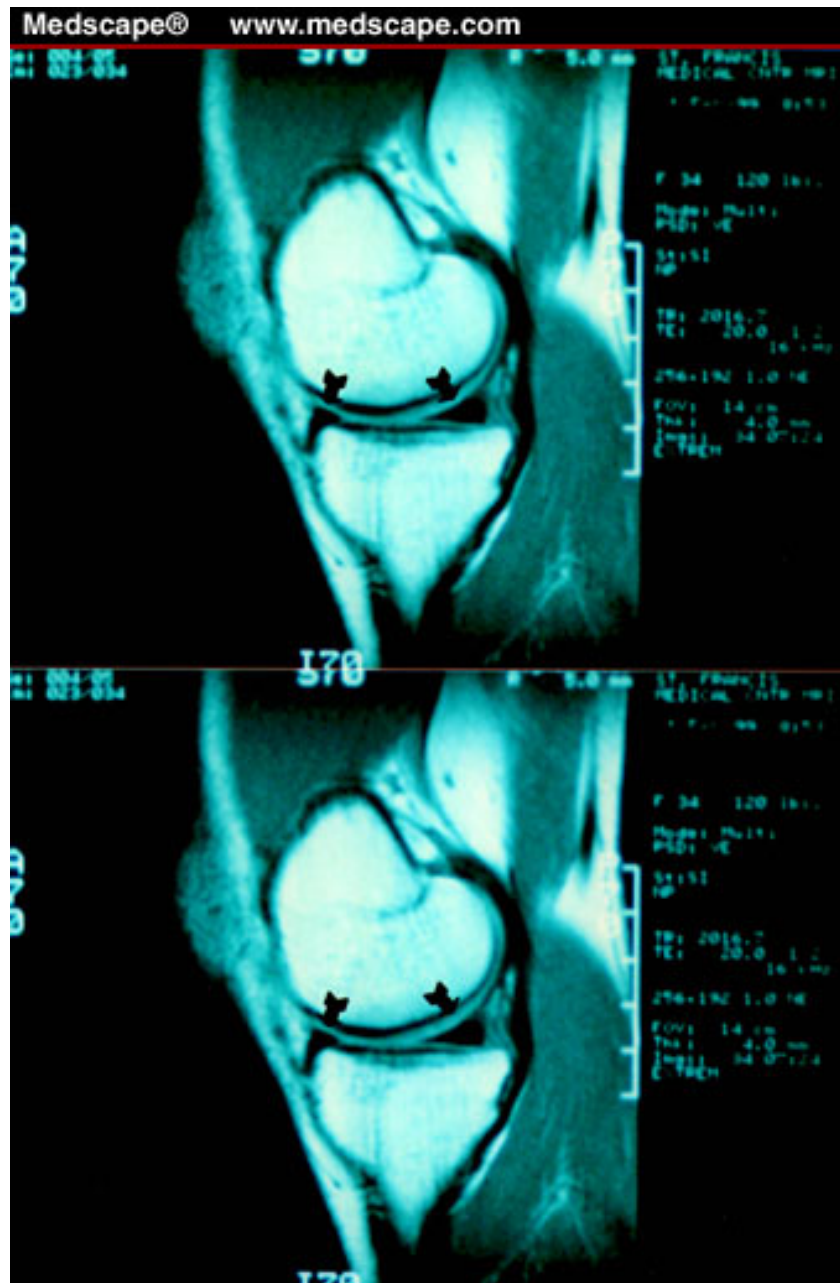


Figure 6A. Normal MR imaging of the knee demonstrating intact medial meniscus (arrows).

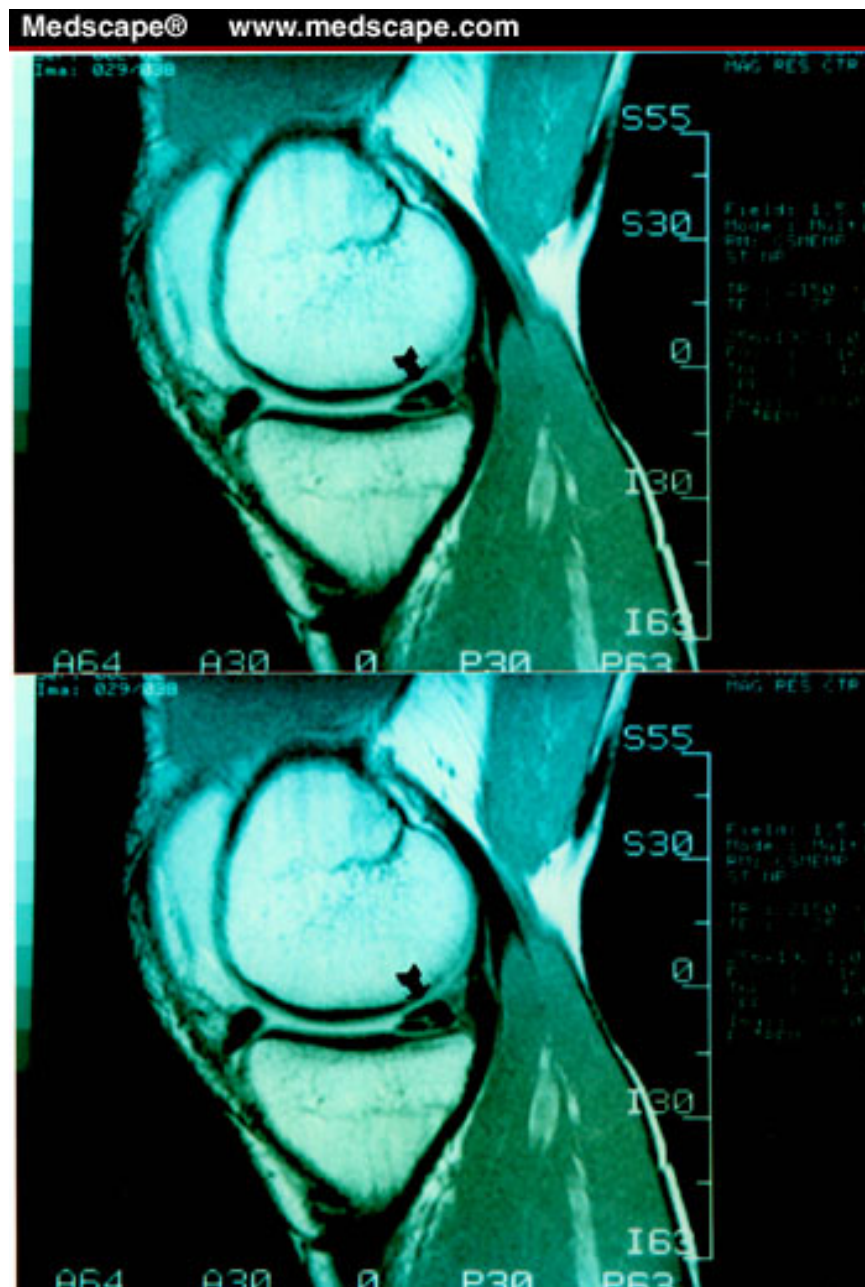


Figure 6B. High signal intensity within the posterior horn of the medial meniscus (arrows) extending through the surface, diagnostic of meniscal tear.

In addition, focal alteration of meniscal size or an irregular configuration should raise suspicions of a torn meniscus.

False positives for meniscal pathology are well known. These occur commonly at the junction of the transverse meniscal ligament with the anterior horn of the lateral meniscus, or at the lateral meniscus in the region of the popliteal hiatus. In addition, there is a normal superior recess in the posterior horn of the medial meniscus. Furthermore, the menisiofemoral ligaments of Humphrey and Wrisberg can mimic a tear in the posterior horn on the lateral meniscus.

Meniscal Tears

Meniscal tears can be either traumatic or degenerative in nature. Meniscal tears are uncommon in persons under 10 years of age, but become increasingly common during and after adolescence.^[1] Degenerative tears can be found in as much as 60% of the population over age 65.^[25] The majority of these tears, however, are

asymptomatic and occur in association with degenerative joint disease. The changing patterns of meniscal injury with chronological age most likely correlate with normal alterations in collagen fiber orientation with aging, as well as increasing intrasubstance degeneration.

The majority of meniscal tears affect the medial meniscus and tend to involve the posterior horn. Meniscal tears are either partial or full thickness and stable or unstable. An unstable tear is one where the entire tear or a portion thereof can be displaced into the joint space. There it may become trapped, causing pain by traction at the meniscocapsular junction. It may be responsible for symptoms of catching, locking, and effusion.

Meniscal injuries can be further classified based on their tear patterns^[26] (Figure 7). A vertical or longitudinal tear occurs in line with the circumferential fibers of the meniscus (Figure 8). If long enough, this tear is known as a bucket-handle tear. At arthroscopy, the bucket-handle tear may be seen as being attached anteriorly and posteriorly. Alternatively, it may be detached at either end or transected in the middle with unstable anterior and posterior flaps. A bucket-handle tear may displace into the intercondylar notch, where it may cause true locking of the knee joint.

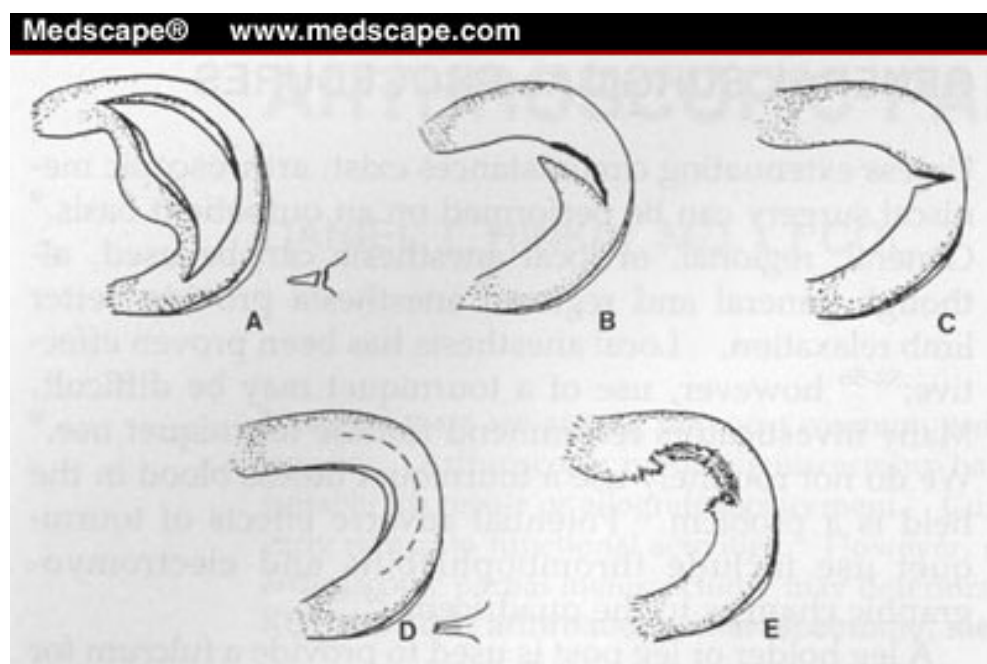


Figure 7. Diagram of meniscal tear patterns: (A) Vertical or longitudinal (Bucket-handle), (B) Flap or Oblique, (C) Radial or Transverse, (D) Horizontal, (E) Complex degenerative. (Reprinted with permission.)

Hinkin DT. Arthroscopic partial meniscectomy. In: Balderston RA, Miller MD, eds. *Operative Techniques in Orthopaedics*. Philadelphia, Pa: WB Saunders; 1995:30, Figure 1.

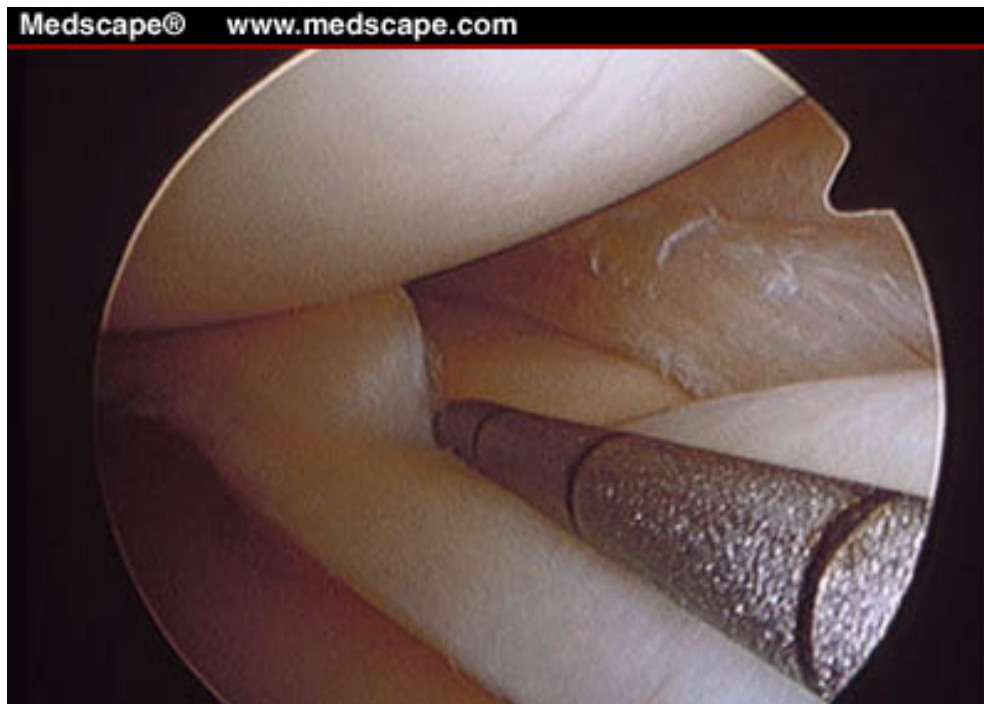


Figure 8. Vertical longitudinal (bucket-handle) tear.

Oblique tears are also known as flap or parrot beak tears and are perhaps the most common (Figure 9). These occur generally at the junction of the posterior and middle thirds.

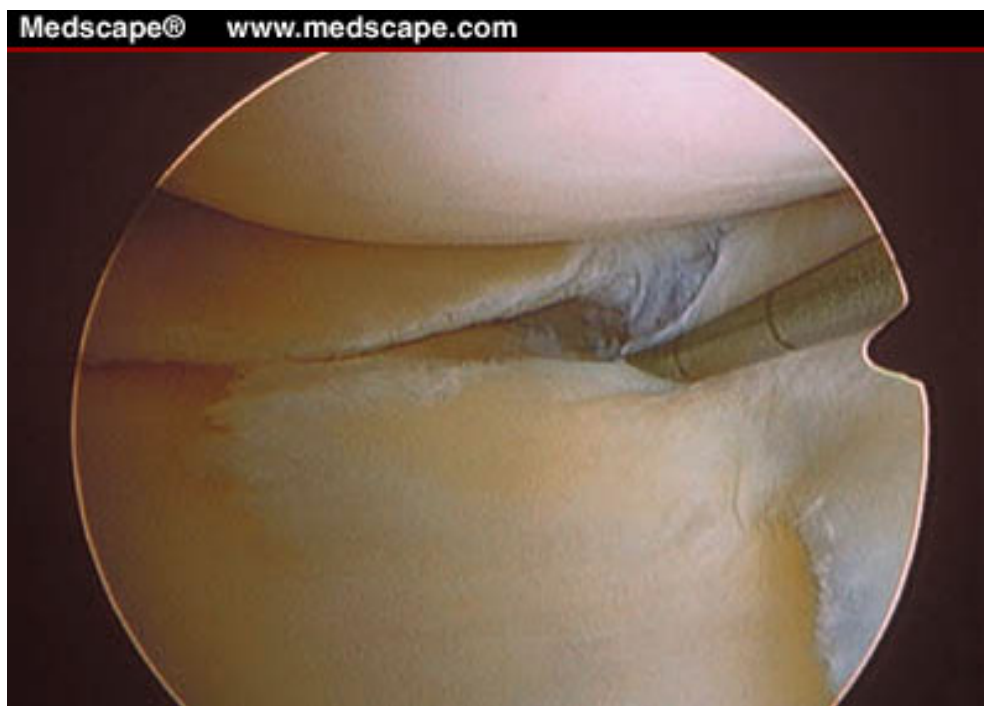


Figure 9. Flap tear.

Radial tears occur in a similar location. They extend from the inner free margin toward the periphery (Figure 10). If such a tear reaches the periphery, it transects the meniscus and renders the hoop stress-distributing capacities of the meniscus useless. Such a tear is the functional equivalent of a total meniscectomy.



Figure 10. Radial tear.

Horizontal cleavage tears usually occur in older individuals. They extend from the inner free margin peripherally to the intrameniscal substance where myxoid degeneration may be present. These tears divide the meniscus into superior and inferior flaps, either of which may be unstable (Figure 11).



Figure 11. Horizontal cleavage tear.

Complex degenerative tears occur in older patients. Osteoarthritic changes may be visible on plain radiographs, and chondromalacia of the articular surfaces is commonly encountered. The tears occur in multiple planes (Figure 12).



Figure 12. Complex degenerative tear.

Treatment of meniscal tears includes simple observation, meniscectomy, and meniscal repair. Tears that are stable, < 1 cm in length, and that do not cause significant mechanical symptoms may be treated with simple observation.^[27] Those tears that are unstable and contribute to mechanical symptoms are treated with operative intervention. As early as 1936, King^[28] drew several important conclusions based on studies of dogs. He showed that a tear within the substance of the meniscus in all likelihood would never heal, but that a tear through the periphery of the meniscus may heal.

Total meniscectomy was advocated as recently as 1971 for meniscal pathology.^[29] With the advent of the arthroscope, as well as the recognition of the importance of the menisci to knee function and load transmission, the role of partial meniscectomy has become much more viable.

Patients with tears that are unstable, occur in the inner two thirds of the meniscal substance, and cause mechanical symptoms are candidates for partial meniscectomy. Metcalf^[26,30] has outlined principles for partial meniscectomy, which include removing all unstable fragments, contouring the meniscus to a relatively smooth, stable rim, and avoiding obtaining a perfectly smooth rim. He advocated switching portals in order to adequately assess the meniscal contour and favored frequent use of a probe to provide tactile feedback. He also noted that the meniscocapsular junction should be protected. Both motorized and hand instruments should be used.

The indications for meniscal repair continue to evolve. Factors affecting success include tear age, location and pattern, age of the patient, as well as any associated injuries. Tears amenable to repair include unstable tears > 1 cm in length and occurring in the outer 20% to 30% toward the periphery, or in the so-called red-red zone.^[31,32] Those tears occurring more toward the junction of the red-white zone may also heal, and the decision to repair should be made based on the clinician's judgment. Ideal candidates for repair are vertical, longitudinal tears occurring within 3 mm of the peripheral rim.

The knee should also be ACL-stable or stabilized. The prognosis for a meniscal repair decreases in the ACL-deficient knee, as the meniscus is required to play an increased role in restricting anterior posterior translation, thus placing the repaired tissue at risk. Success rates for meniscal repair average approximately 70% to 80% in most series. Repairs performed in conjunction with an ACL reconstruction, however, offer a greater success rate, on the order of 90%.^[31-39]

Both open and arthroscopically assisted meniscal repair techniques have been described. Open meniscal repair offers the advantage of better preparation of the tear site. However, only the most peripheral of tears in the red-red zone are amenable to this technique because of exposure and accessibility. Long-term follow-up of open meniscal repairs has revealed success rates ranging from 84% to 100%.^[33]

Arthroscopically assisted meniscal repairs have been described as inside-out, outside-in, and all-inside techniques. Henning^[36,37] first described the inside-out technique of arthroscopic meniscal repair. Inside-out techniques utilize zone-specific cannulas to pass sutures through the joint and across the tear. The sutures are swaged onto flexible needles. A small posterior joint line incision is used to retrieve the sutures and tie directly on the capsule. The use of a posterior retractor, such as a gynecologic speculum, is vital in order to protect the posterior neurovascular structures.

The outside-in techniques have been described by Warren^[32] and Morgan and Casscells.^[34] Outside-in techniques involve passing sutures percutaneously through spinal needles at the joint line across the tear, and then retrieving the sutures intra-articularly. Mulberry knots can then be tied on the intra-articular free ends of the suture. A small incision is then made at the joint line, where the protruding suture ends are retrieved and tied directly on the capsule. An alternative technique is to retrieve the intra-articular portion of the suture with another pass across the tear using a wire snare and tying the suture back on itself on the capsule. This technique eliminates the need for Mulberry knots. A potential disadvantage of the outside-in technique is difficulty in reducing the tear and opposing the edges while passing the sutures.

The all-inside technique was traditionally used to perform repairs of the far posterior horns, where a posterior accessory portal is used, along with passing a suture with a suture hook device.^[40,41] The suture would then be tied intra-articularly. More recently, technologic advances have brought about a number of implantable anchors, arrows, screws, and staples that facilitate meniscal repair without the need for accessory incisions or portals. These devices can be found of permanent, as well as absorbable materials. Although the pullout strength of some of these devices has been shown to approximate those of mattress sutures in cadaveric studies,^[42,43] there have been no long-term clinical studies that compare them to more traditional repair techniques (Figure 13).



Figure 13. Meniscal repair completed with meniscal arrows in place.

Essential principles of meniscal repair include preparing the tear with a rasp or abradar, establishment of a hemarthrosis or use of a fibrin clot, and the presence or establishment of a stable knee.^[31,32,36,38,40]

Postoperative rehabilitation of the knee is controversial. Factors to consider include the nature of the tear, the stability of the repair, and the presence of a stable knee. If the repair is performed in conjunction with an ACL reconstruction, many surgeons do not deviate from their postoperative ACL rehabilitation protocol. If the meniscal repair is performed as an isolated procedure, it is reasonable to limit either range of motion, weight bearing or both. It seems reasonable to permit a range of motion from 0 to 90 degrees, as well as full weight bearing in a brace locked at 0 degrees extension for 6 weeks. Return to sports can be anticipated within 4 to 6 months.

The complication rate of meniscal repair approaches 2% and is most commonly neurovascularly related.^[44] An awareness of the saphenous nerve and its infrapatellar branch on the medial side, as well as the peroneal nerve on the lateral side, is paramount.

Discoid Meniscus

The discoid meniscus is an anatomic variant that primarily affects the lateral meniscus. Rarely, it has also been shown to affect the medial side.

Watanabe^[45] has classified the discoid meniscus as complete, incomplete, and Wrisberg ligament types. Complete and incomplete discoid menisci vary in their degree of tibial plateau coverage. The Wrisberg ligament type is fairly normal in shape, but there is no posterior coronary ligament attachment. Instead, the lateral meniscus attaches to the menisiofemoral ligament of Wrisberg (Figure 14).

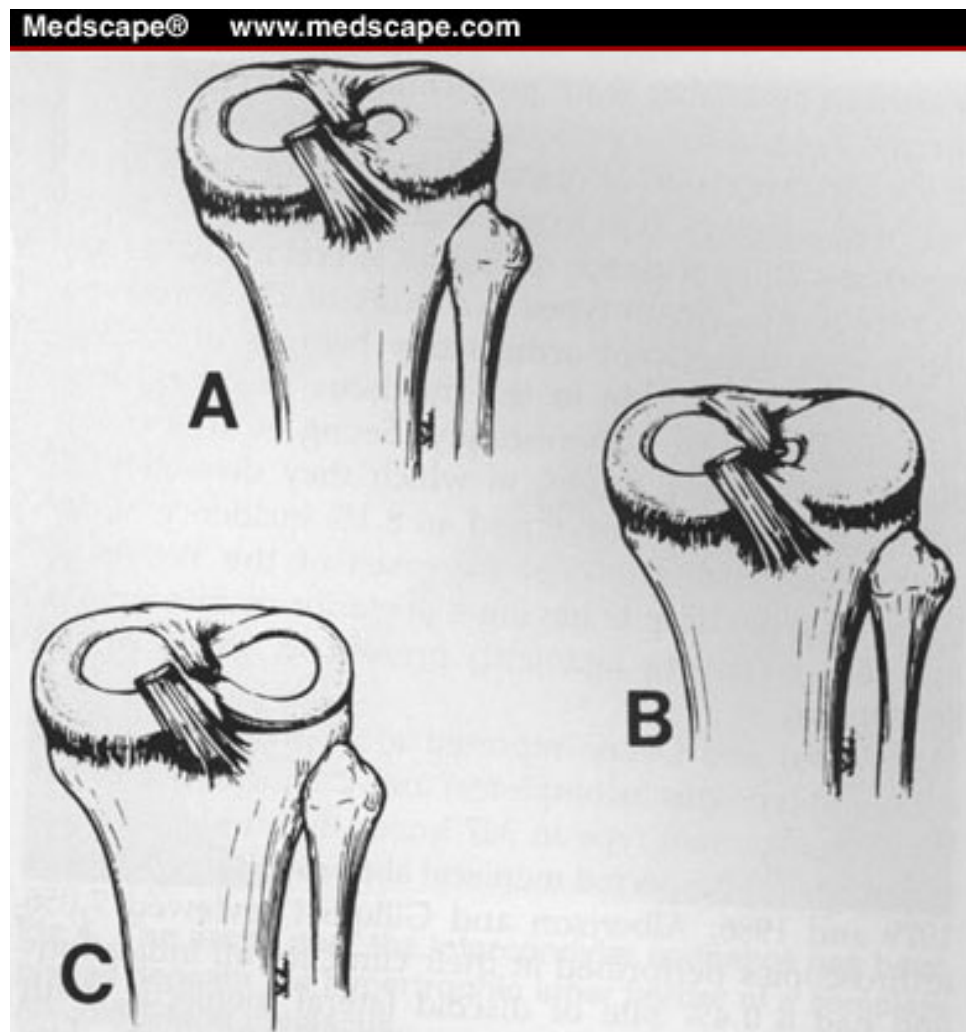


Figure 14. Watanabe classification of discoid lateral meniscus: (A) Incomplete, (B) Complete, (C) Wrisberg-ligament variant. (Reprinted with permission.)

Neuschwander DC, Dres D, Finney TP. Lateral meniscal variant with absence of the posterior coronary ligament. *J Bone Joint Surg Am.* 1992;74: 1186-1190.

The discoid meniscus is an uncommon finding. The incidence has varied from 0.4% to 5% in arthroscopic studies. Interestingly, the incidence from studies in the Japanese and Korean populations has ranged from 8% to 15%.^[46]

The discoid lateral meniscus was first described in 1887.^[47] Smillie^[48] wrote that the discoid meniscus represented a relative failure of absorption during different stages of development. Alternatively, Kaplan,^[49] in 1957, described how abnormal motion of the discoid meniscus might lead to hypertrophy and result in a discoid shape. The exact etiology of discoid meniscus remains unclear.

The discoid lateral meniscus is usually asymptomatic (Figure 15). With the complete and incomplete types, the menisci usually become symptomatic when a meniscal tear occurs. Consequently, the signs and symptoms of the pathology are more reflective of a meniscal tear. The discoid meniscus is then identified upon arthroscopy.



Figure 15. Discoid meniscus.

The snapping knee syndrome is usually associated with the Wrisberg ligament variant. Abnormal motion of the meniscus results from the lack of posterior capsular attachment. Subluxation of the meniscus through flexion and extension then results in a snapping sensation at the joint line.

The treatment of a discoid meniscus depends on its type and association with a tear. If a discoid meniscus is discovered without evidence of a tear, then its presence should be considered incidental, and it should be left intact. If a tear is associated with a complete or incomplete discoid meniscus, then partial meniscectomy should be performed as a saucerization technique. The goal should be to resect enough tissue to result in a well-contoured, 6-mm stable rim.^[50,51] For the Wrisberg ligament variant, the traditional treatment has been total meniscectomy. More recently, techniques have been developed to reduce the meniscus and repair it by providing a posterior attachment.^[52]

Meniscal Cysts

Parameniscal cysts occur relatively infrequently. They are usually associated with horizontal cleavage tears. However, isolated cysts without meniscal pathology have also been reported. Although the incidence of cysts is usually higher on the lateral side, some studies report an equal incidence. [53,54]

Meniscal cysts were first described by Ebner^[55] in 1904. Their incidence ranges from 1% to 22%.^[56-58] Several theories have been proposed regarding cyst etiology, including traumatic origin, as well as purely degenerative origin. Barrie^[59] performed histopathologic studies that provided great insight into cyst etiology. He postulated that meniscal cyst formation originated by influx of synovial fluid through microscopic and gross tears in the substance of the meniscus. In 112 cysts, he demonstrated a meniscal tear with a horizontal component, as well as a tract that provided an exchange of fluid between the joint and the cyst. Meniscal cysts typically are multilocular and are lined with synovial endothelial tissue. Meniscal cysts have been reported, however, in the absence of meniscal pathology, a factor that may alter the surgical treatment of the meniscal cyst.

In the absence of a meniscal tear, it has been proposed that a parameniscal cyst may develop from a compression injury to the periphery of a meniscus that has central degeneration.^[60] A meniscal cyst may then develop more peripherally, leaving the body of the meniscus abnormal, but not torn. In addition, cyst-like structures may develop that are histologically different from those associated with meniscal tears.^[61]

A meniscal cyst may present with signs and symptoms consistent with typical meniscal pathology. Intermittent swelling at the joint line is variable, while pain over the area is quite common. Pisani^[62] described that a lesion that decreases in size with knee flexion and increases with extension is consistent with a meniscal cyst.

The MRI is valuable for confirming the presence of a suspected meniscal cyst and identifying any concurrent meniscal tear (Figure 16).

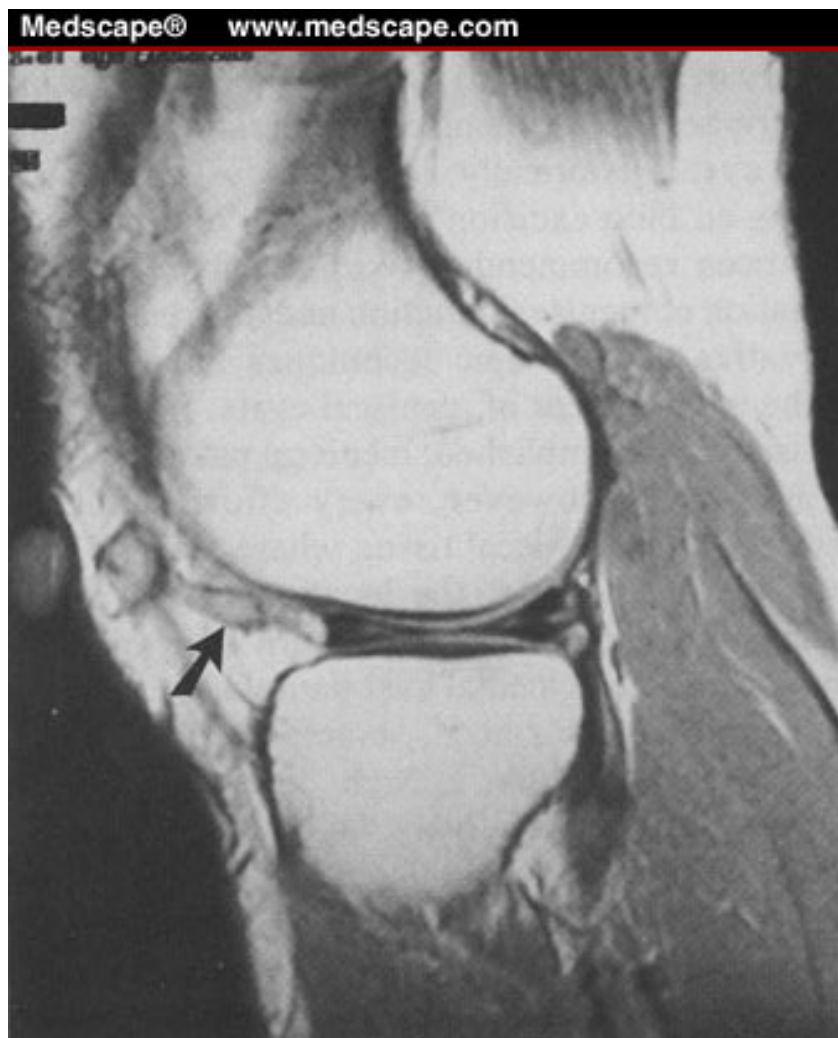


Figure 16. MRI finding of complex serpiginous tract (arrow) associated with lateral meniscal tear, with cyst presenting adjacent to the patellar tendon. (Reprinted with permission.)

Ryu RKN, Ting AJ. Arthroscopic treatment of meniscal cysts. *Arthroscopy*. 1993;591-595, Figure 3.

The management of a meniscal cyst consists of diagnostic arthroscopy to determine the presence of a meniscal tear. In the presence of a meniscal tear, partial meniscectomy followed by arthroscopic cyst decompression is the treatment of choice. If a tear is not confirmed at the time of arthroscopy, then open-cyst decompression with peripheral meniscal repair becomes the logical treatment option, thereby leaving the body of the meniscus unviolated. In the presence of a small meniscal tear, an arthroscopic limited partial meniscectomy may be performed, and if no tract is identified, then conversion to an open cystectomy may similarly preserve the peripheral meniscal body.^[52,53,63]

Current Concepts

Meniscal Allograft Reconstruction

Meniscal transplantation has evolved over the past 10 years as a promising technique. The recognition that meniscal sacrifice leads to late onset of degenerative arthritis has led investigators to search for techniques to alter the long-term consequences of complete or subtotal meniscectomy. Meniscal allograft transplantation intends to restore meniscal function through increase of contact area, decrease in contact stress, joint stabilization, shock absorption, and lubrication.

The indications for meniscal reconstruction continue to evolve. Generally speaking, patients who have undergone

a subtotal or total meniscectomy with a stable knee resulting and no evidence of mal-alignment are candidates for meniscal replacement. Among patients with articular cartilage chondromalacia, the procedure should ideally be limited to those with grades 1 or 2. The meniscal allograft is harvested and procured according to standards established by the American Association of Tissue Banks^[64] and is typically fresh frozen. Precise sizing of the meniscal allograft is correlated by true lateral x-ray measurement of the anterior posterior width of the tibial plateau.^[65] The procedure is performed arthroscopically. Techniques with and without use of bone plugs or a bone bridge have been described. The presence of bone plugs or bridge provides the advantage of improved stability and bone to bone healing. This theoretically results in improved hoop stress transfer and meniscal stability. The remainder of the graft fixation is then performed with the meniscal repair technique of choice.

Follow-up studies have shown that meniscal allografts healed to the periphery in a similar manner as typical meniscal repairs^[66-68] (Figure 17). To date, the function of the transplanted tissue has not been established. Long-term studies that examine the ability of the transplanted tissue to alter the progression of degenerative changes in the postmeniscectomized knee in a prospective fashion are needed in order to determine the long-term benefit of this specific procedure.



Figure 17. Completed meniscal replacement.

Meniscal Regeneration

Currently, the search is under way for a synthetic meniscal replacement. The biomechanical properties of the naturally occurring meniscus provide an enormous challenge for any synthetic material to match. One meniscal replacement strategy focuses on regeneration of meniscal tissue. The theory of meniscal regeneration is based on similar scenarios for skin regeneration in burn patients, as well as nerve regeneration. In the case of meniscal regeneration, a collagen scaffold acts as a resorbable regeneration template, where the scaffold resorbs at a controlled rate to allow for meniscal regeneration. Clinical studies investigating this technique are ongoing.^[69,70]

Currently, meniscal allograft transplantation, as well as meniscal regeneration, remain areas of clinical research. As such, these topics remain controversial and there is no consensus opinion with regard to their widespread clinical application.

References

1. Clark CR, Ogden JA. Development of the Menisci of the Human Knee Joint, *J Bone Joint Surg Am.* 1983;65:538-547.
2. Arnoczky S, Adams M, DeHaven K, et al. Meniscus. In: Woo SY, Buckwalter JA, eds. *Injury and Repair of the Musculoskeletal Soft Tissues.* Chicago, Illinois: American Academy of Orthopaedic Surgeons; 1987:487.
3. Bullough PG, Munuera L, Murphy J, et al. The strength of the menisci of the knee as it relates to their fine structure. *J Bone Joint Surg Am.* 1970;52:564-570.
4. Ferrer-Roca O, Vilalta C. Lesions of the meniscus. Part I: Macroscopic and histologic findings. *Clin Orthop.* 1980;146:289-300.
5. Heller L, Langman J. The Menisconfemoral ligaments of the human knee. *J Bone Joint Surg Br.* 1964;46:307-313.
6. Harner CD, Livesgay GA, Choi NY, et al. Evaluation of the sizes and shapes of the human anterior and posterior cruciate ligaments: A comparison study. *Transactions of the Orthopaedic Research Society.* 1992;17:123.
7. Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med.* 1982;10:90-95.
8. Radin EL, Delamotte F, Maquet P. Role of the menisci in the distribution of stress in the knee. *Clin Orthop.* 1984;185:290-294.
9. Shrive NG, O'Connor JJ, Goodfellow JW. Load bearing in the knee joint. *Clin Orthop.* 1978;131:271-287.
10. Fairbank TJ. Knee joint changes after meniscectomy. *J Bone Joint Surg Br.* 1948;30:664-670.
11. Baratz ME, Mengator R. Meniscal tears: The effect of meniscectomy and repair on intra-articular contact areas and stress in the human knee. *Am J Sports Med.* 1986;14:270-275.
12. Ahmed AM, Burke DL. In vitro measurement of static pressure distribution in synovial joints-Part I: Tibial surface of the knee. *Journal of Biomechanical Engineering.* 1983;105:216-225.
13. Levy IM, Torzilli PA, Warren RF. The effect of medial meniscectomy on anterior-posterior motion of the knee. *J Bone Joint Surg Am.* 1982;64:883-888.
14. Markolf KL, Bargar WL, Shoemaker SC, Amstutz HC. The role of joint loading and knee stability. *J Bone Joint Surg Am.* 1981;63:570-585.
15. Seales KS, Haynes DW, Nelson CL, McLeod PC, Gardes MH. The effect of meniscectomy on knee stability. *Transactions of the Orthopaedic Research Society,* 1981, p 236.
16. Thompson WO, Thaete FL, Fu FH, Dye FF. Tibial meniscal dynamics using three dimensional reconstruction of magnetic resonance images. *Am J Sports Med.* 1991;19:210-216.
17. DePalma AF. *Diseases of Knee: Management in Medicine and Surgery.* Philadelphia: Lippincott; 1954.
18. Gillies H, Seligson D. Precision in diagnosis of meniscal lesions: A comparison of clinical arthrography and arthroscopy. *J Bone Joint Surg Am.* 1979;61:343-346.
19. DeHaven KE, Collins RC. Diagnosis of internal derangements of the knee. *J Bone Joint Surg Am.* 1975;57:802-810.
20. Fowler PJ, Lubliner JA. The predictive value of five clinical signs in the evaluation of meniscal pathology. *Arthroscopy.* 1989;5(3):184-186.
21. Polly DW, Callaghan JJ, Sikes RA, McCabe JM, McMahan K, Savory CG. The accuracy of selective magnetic resonance imaging compared with the findings of arthroscopy of the knee. *J Bone Joint Surg Am.* 1988;70:192-198.
22. Crues JV, Mink JH, Levy P, et al. Meniscal tears of the knee, accuracy of MRI imaging. *Radiology.*

- 1987;164:445-448.
23. Mink JH, Deutsch AL. Magnetic resonance imaging of the knee. *Clin Orthop*. 1989;244:29-47.
 24. Mink JH, Reicher MA, Crues JV. *Magnetic Resonance Imaging of the Knee*. New York, NY: Raven Press; 1987.
 25. Noble J, Hamblen DL. The pathology of the degenerative meniscus lesion. *J Bone Joint Surg Br*. 1975;57:180-186.
 26. Metcalf RW. Arthroscopic meniscal surgery. In: McGinty JB, ed. *Operative Arthroscopy*. New York, N.Y.: Raven Press; 1991:203-236.
 27. Weiss CV, Lundberg M, Hamberg P, DeHaven KE, Gillquist J. Non-operative treatment of meniscal tears. *J Bone Joint Surg Am*. 1989;71:811-822.
 28. King D. The healing of the semilunar cartilage. *J Bone Joint Surg Am*. 1936;18:333-342.
 29. Smillie JS. *Injury to the Knee Joint*. 4th ed. Edinburgh: Churchill Livingstone; 1971.
 30. Metcalf RW. The torn medial meniscus. In: Parisien JS, ed. *Arthroscopic Surgery*. New York, NY: McGraw Hill; 1988:96-98.
 31. DeHaven KE. Peripheral meniscal repair. alternative to meniscectomy. *J Bone Joint Surg Br*. 1981;63:463.
 32. Warren RF. Arthroscopic meniscal repair. *Arthroscopy*. 1985;1:170-172.
 33. DeHaven KE, Black KP, Criffiths HJ. Open meniscal repair: technique and two to nine-year results. *Am J Sports Med*. 1989; 17(6):788-795.
 34. Morgan CD, Casscells SW. Arthroscopic meniscus repair: a safe approach to the posterior horn. *Arthroscopy*. 1986;2:3-12.
 35. Ryu RK, Dunbar WH. Arthroscopic meniscal repair with two year follow-up. A cinical review. *Arthroscopy*. 1988;4:168-173.
 36. Henning CE. Arthroscopic repair of meniscus tears. *Orthopaedics*. 1983;6:1130-1132.
 37. Henning CE, Lynch MA, Yearout KM, et al. Arthroscopic meniscal repair using an exogenous fibrin clot. *Clin Orthop*. 1990;252:64-72.
 38. Morgan CD, Wojtys EM, Casscells SW, et al. Arthroscopic meniscal repair evaluated by second look arthroscopy. *Am J Sports Med*. 1991;19:632-637.
 39. Scott GA, Jolly BL, Henning CE. Combined posterior incision in arthroscopic intra-articular repair of the meniscus. *J Bone Joint Surg Am*. 1986;68:847-861.
 40. Morgan CD. The "all inside" meniscus repair. *Arthroscopy*. 1991;7:120-125.
 41. Cooper DE, Arnoczky SP, Warren RF. Meniscal repair. *Clin Sports Med*. 1991;10:335-351.
 42. Dervin GF, Downing KJ, Keene GC, McBride DG: Failure strengths of suture versus biodegradable arrow for meniscal repair: an in vitro study. *Arthroscopy*. 1997;13(3):296-300.
 43. Albrect-Olsen PM, Lind T, Kristensen G, Falkenberg B. Failure strength of a new meniscus arrow repair technique: biomechanical comparison with horizontal suture. *Arthroscopy*. 1997;13:183-187.
 44. Small NC. Complications in meniscal repair. *Complications in Orthopaedics*. 1987;July/August:109-112.
 45. Watanabe M. Arthroscopy of the knee joint. In: Helfet AJ, ed. *Disorders of the Knee*. Philadelphia, Pa.: Lippincott; 1974:45.
 46. Dickhaut SC, DeLee JC. The discoid lateral meniscus syndrome. *J Bone Joint Surg Am*. 1982;64:1068-1073.
 47. Young RB. The external semilunar cartilage as a complete disc. In: Cleland J, MacKay JY, Young RB, eds. *Memoirs and Memoranda in Anatomy*. London, England: Williams and Norgate; 1889:179.
 48. Smillie IS. Congenital discoid meniscus. *J Bone Joint Surg Br*. 1948;30:671-682.

49. Kaplan EB. Discoid lateral meniscus of the knee joint: nature, mechanism, and operative treatment. *J Bone Joint Surg Am.* 1957;39:77-87.
50. Hayashi LK, Yamaga H, Ida K, et al. Arthroscopic meniscectomy for discoid lateral meniscus in children. *J Bone Joint Surg Am.* 1988;17:1495-1500.
51. Ikeuchi H. Arthroscopic treatment of the discoid lateral meniscus. Technique and long-term results. *Clin Orthop.* 1982;167:19-28.
52. Rosenberg TD, Paulos LE, Parker RD, Harner CD, Gurley WD. Discoid lateral meniscus. Case report of arthroscopic attachment of a symptomatic Wrisberg ligament type. *Arthroscopy.* 1987;3(4):277-282.
53. Ryu RKN, Ting AJ. Arthroscopic treatment of meniscal cysts. *Arthroscopy.* 1993;9:591-595.
54. Parisien JS. Arthroscopic treatment of cysts of the menisci -- a preliminary report. *Clin Orthop.* 1990;257:154-158.
55. Ebner A. Ein fall von ganglion am kniegelenks-meniskus, *Muencherer Med Wochenschr.* 1904;51:1737-1739.
56. Lantz B, Singer KM. Meniscal cysts. *Clin Sports Med.* 1990;9:707-745.
57. Hertz J. Cysts of the semilunar cartilage of the knee joint. *J Int Coll Surg.* 1955;24:257-264.
58. Smillie IS. *Injuries of the Knee Joint.* 4th ed. Edinburgh: Churchill Livingstone; 1970.
59. Barrie HJ. The pathogenesis and significance of meniscal cysts. *J Bone Joint Surg Br.* 1979;61:184-189.
60. Pedowitz RA, Feagin JA, Rajagopalan S. A surgical algorithm for treatment of cystic degeneration of the meniscus. *Arthroscopy.* 1996;12(2):209-216.
61. Barber FA, Ryu RKN, Regan WD. Review of "A surgical algorithm for treatment of cystic degeneration of the meniscus." *Arthroscopy.* 1996;12(2):213-214.
62. Pisani AJ. Pathognomonic signs for cysts of the knee cartilage. *Arch Surg.* 1947;54:188-190.
63. Regan WD, McConkey JP, Loomer RL, Davidson RG. Cysts of the lateral meniscus, arthroscopy versus arthroscopy plus open cystectomy. *Am J Sports Med.* 1989;5:273-281.
64. American Association of Tissue Banks. *Standards of Tissue Banking.* Arlington, Va.: American Association of Tissue Banks; 1997.
65. Garrett JC, Stevenson RN. Meniscal transplantation of the human knee. A preliminary report. *Arthroscopy.* 1991;7:57-62.
66. Garrett JC. Meniscal transplantation: a review of forty-three cases with two- to seven-year follow-ups. *Sports Medicine Arthroscopy Review.* 1993;1:164-167.
67. Shelton WR. Meniscal allotransplantation: an arthroscopically assisted technique. *Arthroscopy.* 1993;9:361.
68. Milachowski KA, Weismeier K, Wirth CJ. Homologous meniscus transplantation. Experimental and clinical results. *Int Orthop.* 1989;13:1-11.
69. Stone KR, Rodkey WG, Webber RJ, McKinney L, Steadman JR. Future directions. Collagen based prostheses from meniscal regeneration. *Clin Orthop.* 1990;252:129-135.
70. Stone KR, Steadman JR, Rodkey WG, Li ST. Regeneration of meniscal cartilage with use of a collagen scaffold. Analysis of preliminary data. *J Bone Joint Surg Am.* 1997;79(A):1770-1777.



Medscape Search Options

Select a database to search, enter a search term, then click "go." [Advanced Search Forms](#)

All material on this website is protected by copyright. [Copyright](#) © 1994-2001 by Medscape Inc. All rights reserved. This website also contains material copyrighted by 3rd parties. Medscape requires 3.x browsers or better from [Netscape](#) or [Microsoft](#).