(v) Chronic ankle instability

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Abstract

Injuries to the ligaments of the ankle are common, especially in athletes. Symptomatic ankle instability develops in as many as 10–40% following an acute injury. The causes of symptoms are multifactorial, encompassing pre-existing patient factors predisposing to instability, functional instability and mechanical instability. Chronic ankle instability occurs when patients suffer recurrent episodes of ankle sprains and the majority can be successfully treated with a functional rehabilitation programme. Those that fail require consideration of surgical intervention. A full history, clinical examination, radiological investigation and an understanding of the pathomechanics involved are vital to ensure that the most appropriate surgical strategy is adopted. Pain and swelling are commonly associated symptoms and may be more disabling than the episodes of instability. Concurrent intra and extra-articular pathologies must be addressed to achieve a successful functional outcome. Surgical options include arthroscopy, ligament reconstruction techniques, hindfoot alignment procedures and gastrocnemius release. This article focuses on the anatomy, pathomechanics and treatment of chronic lateral ankle instability. Medial, syndesmotic and subtalar instability are also discussed.

Keywords ankle injuries; ankle joint; joint instability; lateral ligament ankle; subtalar joint

Introduction

Injuries to the lateral ankle ligament complex or "ankle sprains" are the commonest sports related injury, accounting for 16–21% of all musculoskeletal injuries. The incidence in the UK of 52.7/10 000/year equates to 300 000 injuries/year.[1] Following ankle injury the majority of patients undergo a functional rehabilitation programme, which is usually successful in returning patients to functional normality. There is little role for surgery in the acute phase.[2] However, some patients develop residual symptoms of pain and/or instability as a consequence, which may be underestimated in clinical practice. Symptomatic ankle instability can develop in as many as 10–40% of patients following an acute event, even after adequate conservative treatment.[3] Chronic ankle instability does not exist as a single pathologic entity and the symptoms of "*sprained ankle syndrome*" are frequently multifactorial. Treatment is not only based upon a proper history, physical examination and radiological investigations but consideration of the pathomechanics of the condition.

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Ankle joint anatomy and biomechanics

The ankle joint complex consists of three articulations: the talocrural, subtalar and distal tibiofibular joints. The three joints work together to allow coordinated movement of the hindfoot in three cardinal planes: the sagittal plane (plantarflexion–dorsiflexion), the frontal plane (inversion–eversion) and the transverse plane (internal and external rotation). Hindfoot motion does not occur in isolation but rather in a coordinated, coupled motion best described as pronation (dorsiflexion, eversion and external rotation) and supination (plantarflexion, inversion and internal rotation).[4]

The talocrural joint or '*mortice*' is formed by the articulation of the dome of the talus, the medial malleolus, the tibial plafond and the lateral malleolus. In isolation, the ankle joint is a modified hinge joint allowing dorsiflexion and plantarflexion. The sagittal plane motion of the ankle joint passes through the tips of the malleoli. Since the lateral malleolus is longer and is posterior to the medial malleolus, the plane is oblique to the plane of the floor and also to the transverse plane. As the ankle dorsiflexes, it also rotates externally and *vice versa*.[5]

The biomechanics of the ankle is complex, with three factors contributing to stability. In a loaded ankle the osseous anatomy is the most critical as the talus compresses into the bony mortice resulting in primary stability. In an unloaded ankle a combination of static ligamentous restraints and musculotendinous units play more vital roles, with each ankle ligament contributing a different function depending of the position of the foot and ankle in space.

Lateral ligaments

The lateral ankle ligament complex is composed of three main ligaments: the anterior talofibular, calcaneofibular and posterior talofibular ligaments (Figure 1). The anterior talofibular ligament (ATFL) is the weakest of the lateral ligaments. It has a load to failure 2–3.5 times lower than the calcaneofibular ligament (CFL) and two times lower than the posterior talofibular ligament

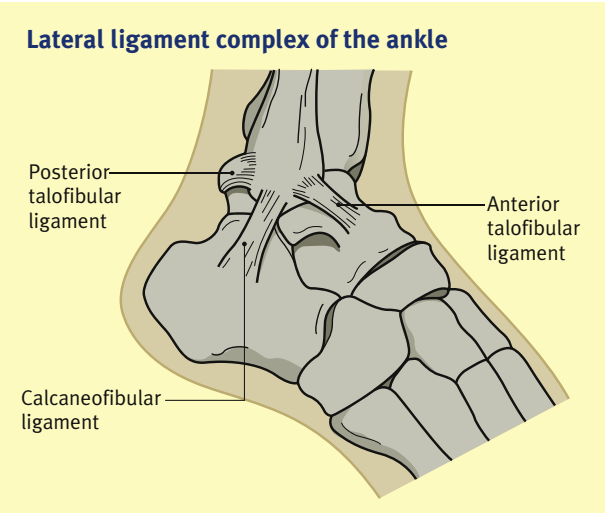

Figure 1 The lateral ligament complex of the ankle (to be redrawn).

(PTFL).[6] The ATFL is intra-capsular and originates 1 cm proximal from the tip of the lateral malleolus just anterior to the fibular facet. It extends distally and medially inserting onto the neck of the talus and functions as a check-rein when the foot is in an equinus or inverted position. It is therefore most vulnerable when the ankle is in plantarflexion and is the most frequently injured ligament. It is usually disrupted through its mid-substance. The ATFL is taut in plantarflexion and acts to prevent anterior displacement of the talus from the ankle and excessive inversion and internal rotation.

The CFL originates adjacent to the ATFL, approximately 8 mm from the tip of the fibula, and courses distally and posteriorly across both the ankle and subtalar joints to insert onto the lateral aspect of the calcaneus just behind and above the peroneal tubercle. It forms the floor of the peroneal tendon sheath. When the ankle is dorsiflexed, the ATFL is loose and the CFL is taut.

The PTFL is short, thick and is the strongest of the three ligaments and hence is rarely injured. It originates from the medial surface of the lateral malleolus and inserts into the posterior aspect of the talus. The talar and fibular insertions of the PTFL are broad. The PTFL is under tension only when the ankle is in extreme dorsiflexion, and provides restraint to both inversion and internal rotation when the ankle is loaded.[7]

Medial ligaments

The deltoid ligament has significantly higher load to failure than its lateral ligament counterparts and thus requires much greater force to injure. The anatomy of the deltoid ligament comprises of both superficial and deep components. The superficial deltoid originates from the anterior colliculus of the medial malleolus and inserts into both the navicular and the sustentaculum tali of the os calcis. The deep deltoid ligament is a key component of ankle stability. It originates from the posterior colliculus and inserts into the non-articular medial surface of the talus. Classically the superficial deltoid ruptures first followed by the deep deltoid at its talar insertion due to forced abduction or eversion. The biomechanical function of the deltoid ligament is to resist abduction and lateral translation of the talus. The deep deltoid ligament provides the greatest restraint against talar shift.[8]

Syndesmosis

The syndesmosis refers to the distal articulation between the tibia and fibula, and forms the stable roof of the talocrural joint. The joint is stabilized by a thick interosseous membrane that runs throughout the length of the two bones. There are three ligaments at the ankle: the anterior–inferior tibiofibular ligament (AITFL), the posterior–inferior tibiofibular ligament (PITFL) and the interosseous ligament (IOL). The AITFL is the most commonly injured and results in the so-called '*high ankle sprain*'. The IOL is both stronger and stiffer than the AITFL but is commonly injured in combination with the AITFL. The PITFL is smaller than the AITFL and is composed of both a deep portion, the transverse tibiofibular ligament, and a superficial portion. The PITFL contributes most towards the stability of the syndesmosis,[9] and acts to deepen the talocrural joint by projecting inferior to the tibia, preventing posterior translation of the talus.

Biomechanically, a limited degree of motion is necessary at the syndesmosis for normal ankle function. When the talus is wider anteriorly than posteriorly, as the ankle moves from plantarflexion to dorsiflexion the lateral malleolus externally rotates by 11° and the distance between the tibia and fibula increases by 1.5 mm.

Subtalar joint anatomy and biomechanics

The subtalar joint is formed by the articulation between the talus and os calcis. The joint is divided into anterior and posterior articulations separated by the sinus tarsi and canalis tarsi. The anterior joint consists of the talonavicular joint including the anterior and middle facets of the calcaneum. The posterior joint contains the posterior facet and its corresponding inferior talar surface. The anterior joint lies more medial than the posterior joint and has a higher centre of rotation. This results in a subtalar joint axis of rotation that is 42° upwards in the sagittal plane and 23° medial to the midline of the foot in the transverse plane[10] (Figure 2).

There is debate in the literature regarding the key ligamentous stabilizers of the subtalar joint, in both their terminology and reported functions. It is generally accepted that there are three ligamentous groups; the peripheral, deep and the retinacular ligaments. There are three peripheral ligaments, the calcaneofibular ligament (CFL), lateral talocalcaneal ligament (LCTL) and fibulotalocalcaneal ligament (FTCL). There are two deep ligaments, the cervical ligament (CL) and interosseous ligament (IOL).

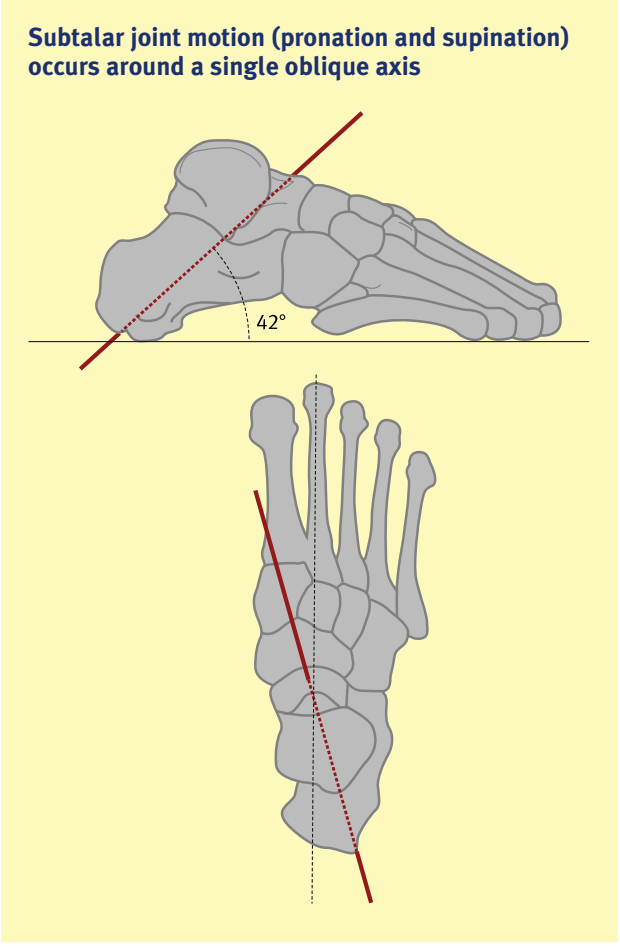

Figure 2 Subtalar joint motion (pronation and supination) occurs around a single oblique axis (to be redrawn).

The CFL is integral in preventing excessive supination at the subtalar joint, and its rupture may cause combined instability at the ankle and subtalar joints.

Pathomechanics of ankle sprains

Lateral ankle sprains most commonly occur as a result of excessive supination of the hindfoot about an externally rotated lower leg during the initial phase of heel strike. There is often sequential failure of the ligaments starting with the ATFL, followed by the CFL.[11] Isolated ruptures of the CFL are rare and may play a role in subtalar instability. Chapman classified acute injuries into three grades: Grade 1 — ligament stretch without macroscopic tearing, Grade 2 — macroscopic tearing of the ligament, Grade 3 — ligament rupture.[12] Chronic lateral ankle instability most often follows Grade 3 injuries.

There is little research describing predispositions to a first-time ankle sprain. A pathomechanical model proposed by Fuller[13] describes the cause of lateral ankle sprain as an increased supination moment around the subtalar joint axis. In the normal ankle, on heel strike the centre of pressure (COP) of the foot lies lateral to the subtalar joint axis and the ground reaction force (GRF) exerts a pronation moment. However, individuals with a rigid supinated hindfoot (calcaneal varus) would have a laterally placed subtalar axis relative to the GRF and this increased supination moment could cause excessive inversion and injury to the lateral ligaments (Figure 3). In addition, increased plantarflexion causes the subtalar joint axis to drift laterally and thus increases the risk of injury[14] (Figure 4). Inman described great variation amongst individuals in the alignment of the subtalar joint axis,[10] thus those individuals with a laterally placed subtalar axis would be predisposed to injury.

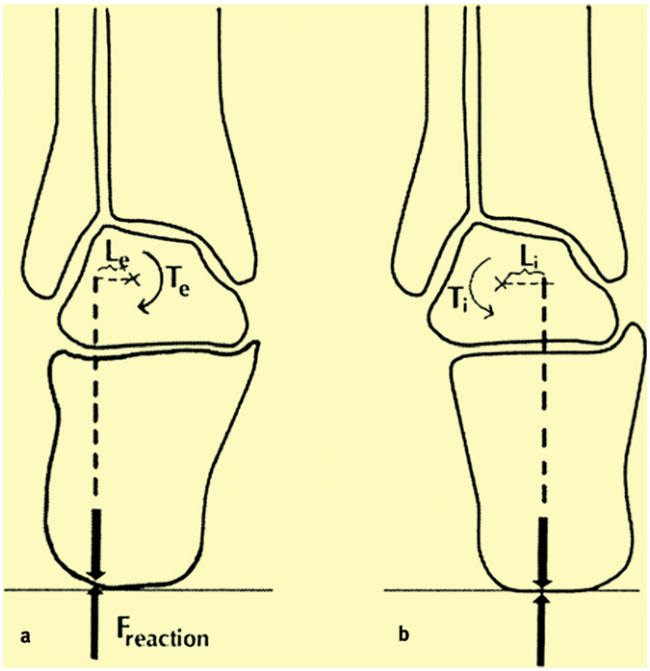

Figure 3 The subtalar joint axis of rotation passes through the talus. In the normal ankle (**a**), the ground reaction force acts lateral to the axis producing a pronating torque. In (**b**), where the heel is in varus, the force produces a supinating torque. (*Reproduced with kind permission from Tropp H 2002 Journal of Athletic Training*).

In addition to the stability provided by the osseous anatomy and ligamentous structures, the peroneal musculotendinous units provide a compensatory pronation moment during supination at the talocrural joint. Gauffin determined that the ankle everters are unable to withstand a supinating moment lever arm greater than 3–4 cm,[14] and if this is surpassed then ligamentous injury will occur.

Medial ankle sprains are rare in isolation and are more commonly combined with either a lateral ligament complex injury or fractures. A medial ankle sprain can occur after eversion and internal rotation injuries. The reported incidence of syndesmotic sprains range from 1% to 17% of all ankle injuries.[15] The primary causative force is external rotation. This causes failure of the AITFL and tearing of the interosseous ligament but the PITFL is usually preserved.

Although isolated subtalar joint injuries have been reported, these injuries typically occur in combination with chronic ankle instability. A significant proportion is due to an acute high-energy injury, and subtalar injuries may be present in 10–25% of individuals who have chronic ankle instability. A proposed mechanism of injury results from a sudden deceleration of the calcaneus with progression of the talus.

Pathomechanics of chronic instability

Chronic ankle instability (CAI) is defined as the occurrence of recurrent bouts of lateral ankle instability. The most common factor predisposing an individual to chronic instability is an initial acute event; however, the mechanisms of chronic instability are thought to be different from the acute injury. Classically, there are two factors thought to cause chronic instability although these are not mutually exclusive from one another. In 1965, Freeman[16] introduced the concept of functional instability, which occurs as a result of proprioceptive changes following ligament injury. Tropp et al[17] expanded on this model and defined mechanical instability as abnormal motion of the talus within the ankle joint due to pathologic laxity of the ligaments, and functional instability as motion beyond voluntary control but within the normal physiological range. In addition to instability, a significant proportion of patients develop chronic symptoms, which contribute to the overall morbidity and which may accentuate the sense of instability. Pain, swelling, locking and stiffness are commonplace and arise from pathologies that might not fall neatly into the two categories.

Mechanical instability

The precise definition of mechanical instability varies in the literature. It is traditionally thought of as the result of anatomical insufficiencies such as either ligamentous laxity, synovial changes or a fault in the kinematics of any of the three joints around the ankle. Mechanical instability can be measured *via* clinical examination, stress radiography or arthrometry.

Karlsson et al.[3] investigated the relationship between the degree of mechanical instability demonstrable with stress testing and symptoms. They determined that 10 mm or more of anterior draw or 9° of talar tilt was consistent with CAI. Alternatively, a difference of 3 mm of anterior draw or 3° of talar tilt with the functionally normal side was also significant. Since there is great variation in the normal physiologic ranges of motion, a comparison with the functionally stable ankle is the most accurate determinant.

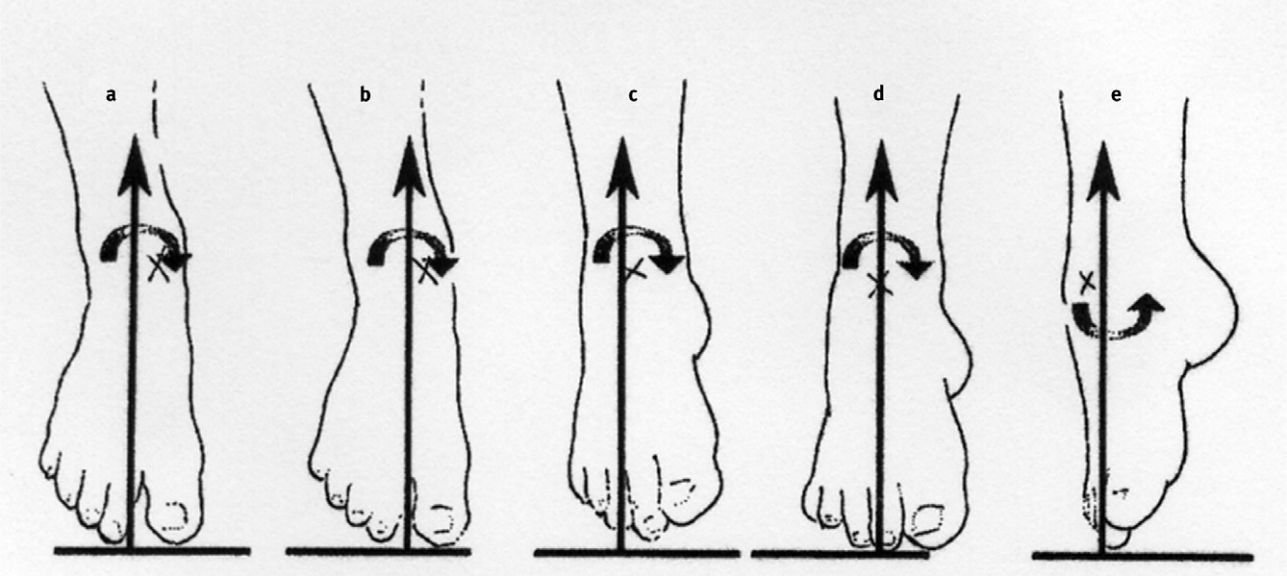

Figure 4 Diagram showing the lateral drift of the subtalar joint axis from (**a**) neutral to (**e**) plantarflexion and inversion, increasing the risk of injury. (*Reproduced with kind permission from Tropp H 2002 Journal of Athletic Training*).

Functional instability

Injuries to the lateral ligaments results in neuromuscular changes to the stabilizing muscles around the ankle, leading to a proprioceptive deficit. Weakness of the peronei have been reported amongst individuals with CAI as well as impaired reflexive response times. Most patients presenting with CAI will invariably have an element of functional instability and it is often the predominant problem.

Other factors

DiGiovanni et al.[18] demonstrated the presence of multiple other pathologies in patients presenting with CAI, which may cause pain, disordered kinematics or mechanical impairments, and this can manifest themselves as a sense of instability. Synovial hypertrophy with anterolateral impingement, osteochondral injuries, intra-articular loose bodies, degenerative changes and peroneal tendon injuries are common. Pre-existing factors will also contribute biomechanically towards CAI such as a varus hindfoot, tight tendo-Achilles or gastrocnemius, and generalized joint laxity.

Clinical evaluation

History

Patients presenting with chronic ankle instability usually report a preceding history of a significant ankle sprain. Typically, patients complain of repeated episodes of giving way, particularly on walking on uneven ground. Aside from instability, intermittent swelling and pain may accompany these episodes. The presence of pain, especially if consistent on weightbearing, should raise the suspicion of intra-articular pathology such as an osteochondral lesion or soft tissue impingement. The mechanism of injury, in particular the position of the foot at the time of injury, is important but often the patient's account does not correlate well with the injured structures.

Clinical examination

A thorough examination is essential to determine intra-articular and extra-articular causes of symptoms. Tenderness is usually maximal over the lateral gutter (ATFL) and Molloy's impingement test may reveal impingement syndrome due to post-traumatic synovitis.[19] Tenderness over the deltoid ligament usually indicates a more complex injury. Peroneal tendon subluxation may manifest itself as a sense of instability and can be unmasked with the foot in maximal dorsiflexion and eversion. Hindfoot examination must also exclude a gastrocnemius contracture (Silfverskiold test) and a varus heel, both of which would accentuate the instability.

Neurological status should be assessed and documented, as sural and peroneal nerve palsies are a rare complication of lateral ligament injuries. The two most important tests for evaluation of ankle instability are the anterior draw test and inversion stress test (talar tilt test).

Anterior draw test: with the patient relaxed, anterior subluxation of the talus can easily be demonstrated. With the foot in 20° of plantarflexion, the tibia is pushed backwards against the fixed foot or the foot drawn forwards against the tibia (Figure 5). The characteristic sign is the '*suction sign*' as the skin is sucked inwards over the lateral gutter.

Inversion stress test: excessive hindfoot inversion with the foot in a plantigrade position may indicate tibiotalar laxity, and is usually positive where there is complete CFL disruption. Both ankles should be tested simultaneously to determine asymmetry (Figure 6). It is sometimes difficult to differentiate between ankle and subtalar motion, and palpation of the talar neck may help.

Medial and syndesmotic instability test: the Kleiger test can demonstrate medial and syndesmotic instability. With the knee flexed to 90°, the foot is externally rotated. A positive test reveals pain in the area of injury. The '*squeeze test*' for syndesmotic injury involves squeezing the fibula at the mid-calf. The pain should be felt distally at the level of the syndesmosis.

Molloy impingement test: the Molloy impingement[19] test is the cardinal physical sign for ankle synovial impingement. The ankle

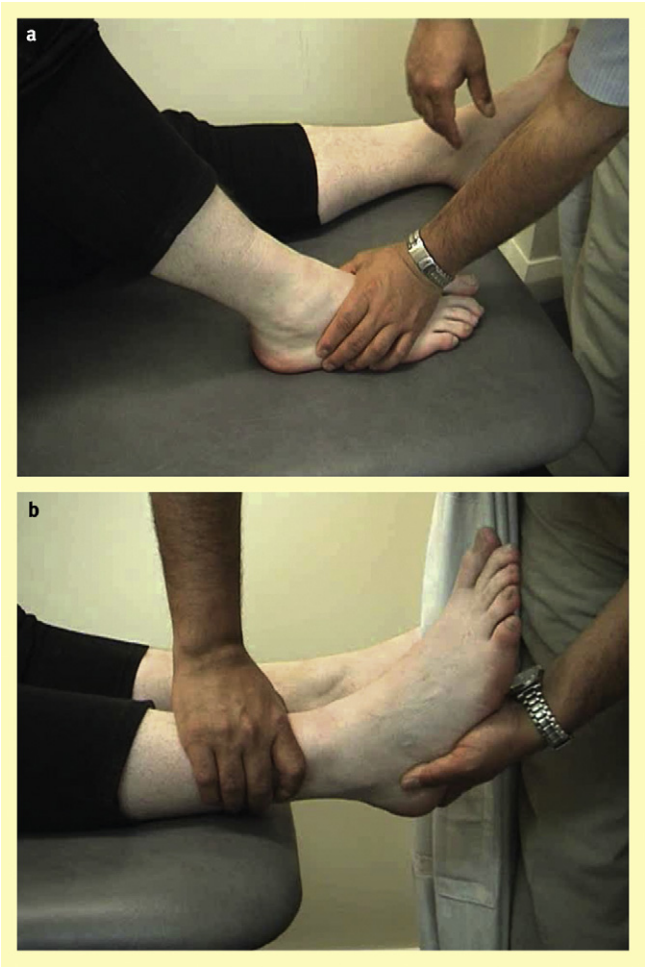

Figure 5 The anterior draw test. It can be performed either with the foot fixed or free.

is dorsiflexed with finger pressure in the joint line. The appearance of or increase in pain under the finger is a positive test. The test may have to be repeated at different points on the joint line. This is both highly sensitive and specific for predicting synovitis and hypertrophy.

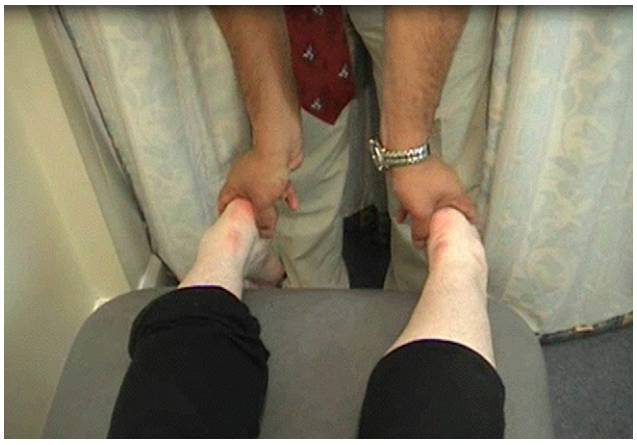

Figure 6 The talar tilt test, which is best performed bilaterally for comparison.

Radiographic investigation

Although various imaging modalities have been utilized in the evaluation of ankle instability, the two key investigations are stress radiography and MRI.

Stress radiography

The purpose of stress radiography is to demonstrate mechanical instability of the tibiotalar joint. The inversion stress view is a comparative antero-posterior radiograph taken with the foot in neutral and the hindfoot in maximal inversion. Both anterior draw and talar tilt are tested (Figure 7). Muscle guarding due to pain may limit its sensitivity and intra-articular local anaesthetic has been shown to improve accuracy. There is no consensus as

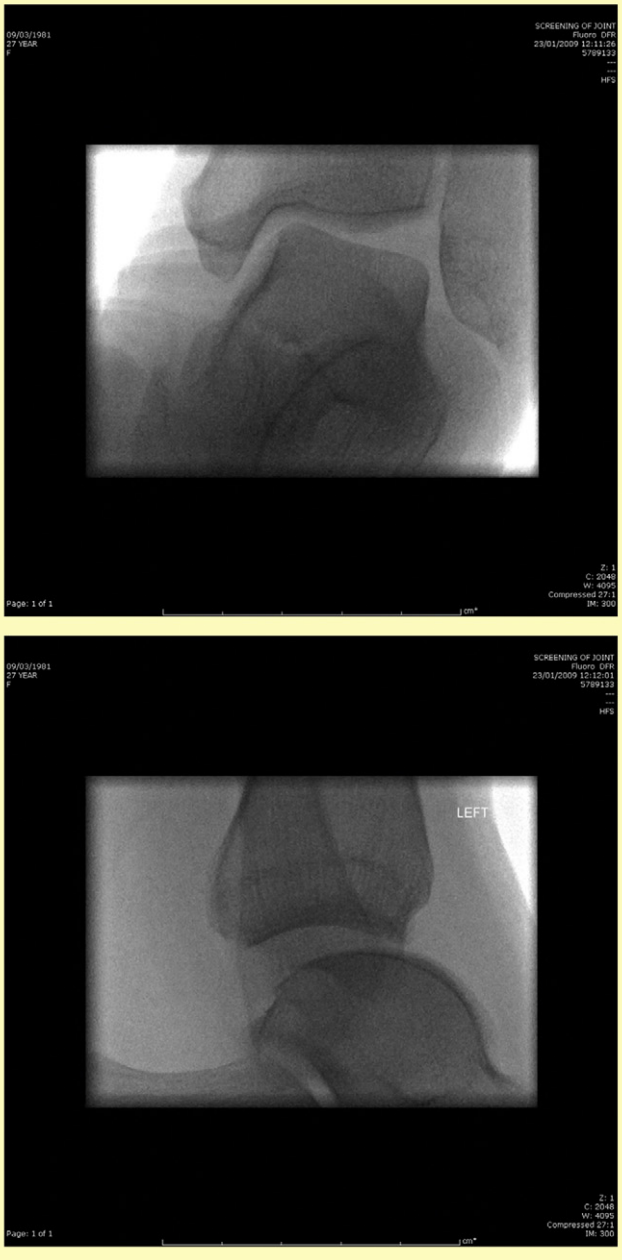

Figure 7 Stress radiography showing abnormal 18° of talar tilt and 6 mm of anterior draw.

to what is the normal range of talar tilt (0–23°). Chrisman and Snook[20] determined that a 10° difference between the two sides was 97% sensitive for ATFL and CFL injury. According to Safran,[21] a 5° difference may be clinically significant.

Syndesmotic instability can be demonstrated radiographically with the Kleiger test (Figure 8). Comparative views are recommended, but instability is likely where: (1) the tibiofibular clear space on the mortice view is >5 mm, (2) the tibiofibular overlap on the antero-posterior view is <6 mm, and (3) there is tibiofibular overlap on a mortice view of <1 mm.

Magnetic resonance imaging

The primary purpose of MRI is to identify conditions that may give rise to symptoms of instability such as osteochondral lesions, peroneal tendon tears, impingement syndrome and loose bodies. Although MRI can identify the degree of injury to the ligaments, it does not confirm whether or not the ankle is mechanically unstable. MRI is useful for patients presenting primarily with chronic pain, to determine whether arthroscopic treatment should be offered prior to consideration of ligament reconstruction (Figure 9).

Other investigations

Arthrography or MRI-arthrography is of limited value in the evaluation of chronic lateral instability. A rupture of the CFL allows contrast medium to enter the peroneal tendon sheath but penetration into the subtalar joint is not indicative of injury. Peroneal tendography can be utilized in a converse manner and may be useful when a tendon tear is suspected.

Ultrasound is a comparatively new technique and is largely operator-dependent. Ligament ruptures can be identified and stress testing can be performed. It may prove to be a useful technique in the assessment of subtalar instability.

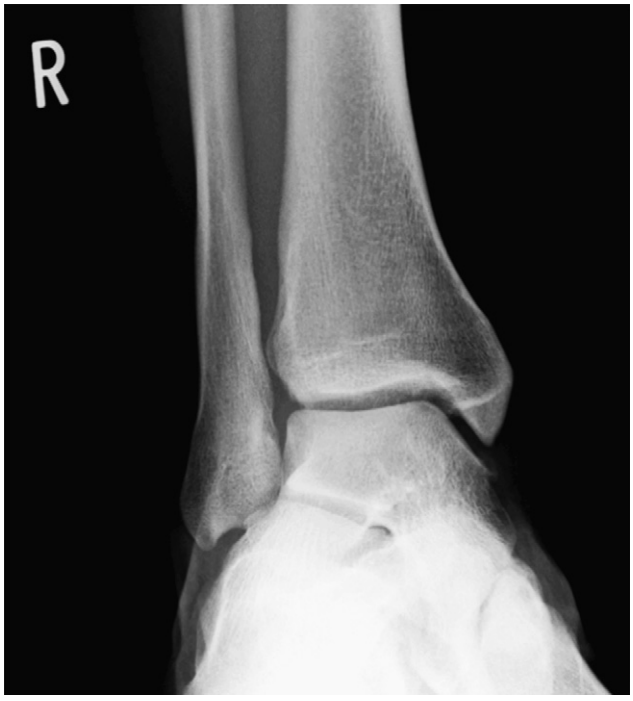

Figure 8 The Kleiger test of syndesmotic instability. Significant widening is noted with no tibiofibular overlap on the mortice view.

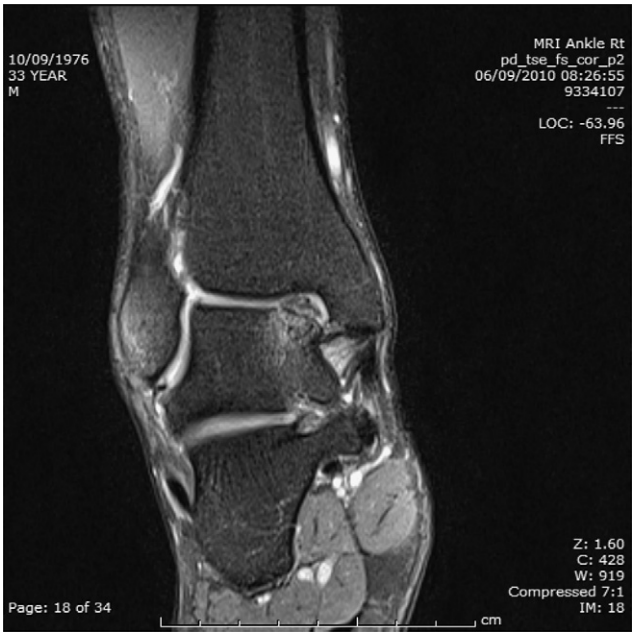

Figure 9 T2 weighted MRI showing an unstable osteochondral lesion of the medial talar dome.

Non-surgical treatment

Early rehabilitation of acute injuries is the main aim. A combination of isokinetic strength training with proprioception training shortens rehabilitation and can be prophylactic for recurrent injury. In the case of CAI, rehabilitation follows a similar course; the aim being to improve the functional component of any instability. Ankle supports such as an ankle stirrup brace may be utilized, as needed. Patients with pure mechanical instability are less likely to respond to conservative measures.

Surgical treatment

Surgery should be considered following complete workup and failure of conservative management. Surgical planning should initially be based on the presence or suspicion of any intra-articular pathology and whether or not ankle arthroscopy is indicated.

Ankle arthroscopy

DiGiovanni,[18] Kibler[22] and Ogilvie-Harris[23] all found a high incidence of intra-articular pathology in CAI. Synovitis, impingement lesions, osteochondral lesions, spurs and loose bodies are all amenable to arthroscopic treatment, which can lessen symptoms of mechanical instability (Figure 10). Malviya et al.[24] performed an initial ankle arthroscopy with screening and found a similarly high incidence of intra-articular pathology. Treatment of these lesions resulted in 53% of their patients not requiring lateral ligament reconstruction.

Kibler believed that fluid extravasation from ankle arthroscopy did not compromise ligament reconstruction and practice varies as to whether the arthroscopy is performed as a combined or staged procedure. With a high reported rate of resolution of symptoms with an arthroscopy alone, it would seem logical to perform an initial arthroscopy and subsequently proceed to ligament reconstruction only if required. Further research is

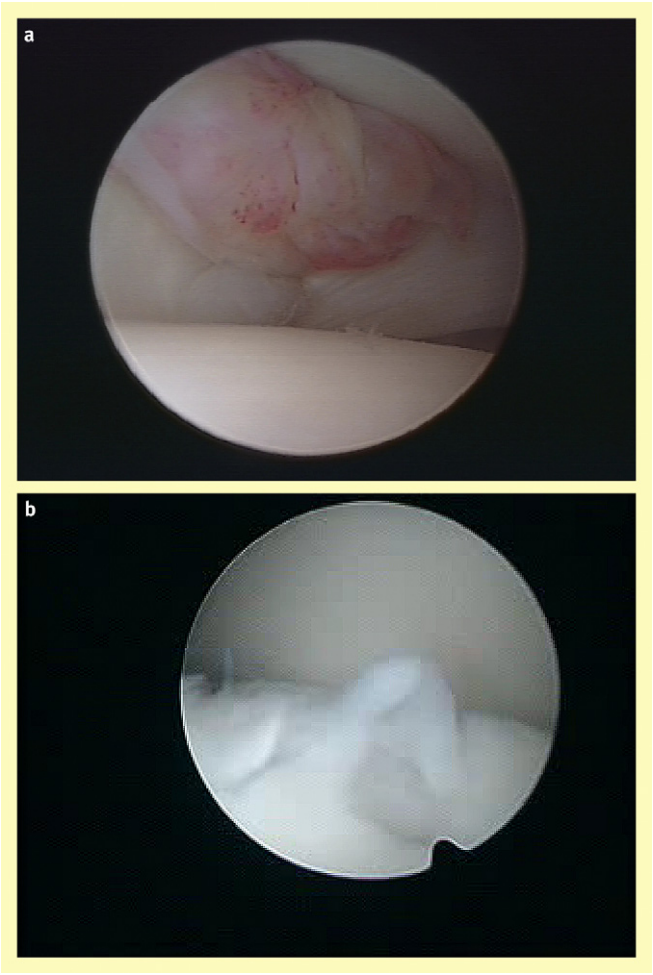

Figure 10 (**a**) A syndesmotic impingement lesion with synovitis. (**b**) An unstable osteochondral lesion of the medial talar dome.

required in this area. The authors' practice is to perform an initial ankle arthroscopy in all suspected cases of intra-articular pathology, and subsequently proceed to ligament reconstruction 10–12 weeks post-operatively if instability has not resolved. A combined procedure is only performed where no intra-articular pathology is identified.

Surgical stabilization of chronic lateral ankle instability

There are over 20 different described surgical reconstruction procedures available for chronic lateral ankle instability. All have reported success rates of over 80%. There are two main approaches to surgical stabilization: anatomical and non-anatomical. In contemporary practice, the anatomic repair remains the procedure of choice although the techniques have to be individualized according to the patient and their anatomy.

Biomechanical studies have shown more stability with anatomic repair *versus* non-anatomic but the clinical outcomes may largely be the same. There are only a few randomized controlled trials comparing the two techniques.

Anatomic stabilization: Brostrom[25] described a repair whereby the ruptured lateral ligaments are reapproximated to the bone, resulting in restoration of normal anatomy. Gould et al.[26] described a modification in which the inferior extensor retinaculum is advanced to reinforce the repair (Figure 11).

The incision is performed anterior to the lateral malleolus, parallel to its border in a curvilinear approach. Dissection is carried out down to the joint capsule. The capsule and ATFL are divided at the lateral malleolus, leaving a cuff of tissue, and imbricated *via* transosseous sutures or anchors. The peroneal tendon sheath is then opened to determine the quality of the CFL. Avulsions proximally or distally can be reattached in the same manner but a midsubstance tear may prove impossible to

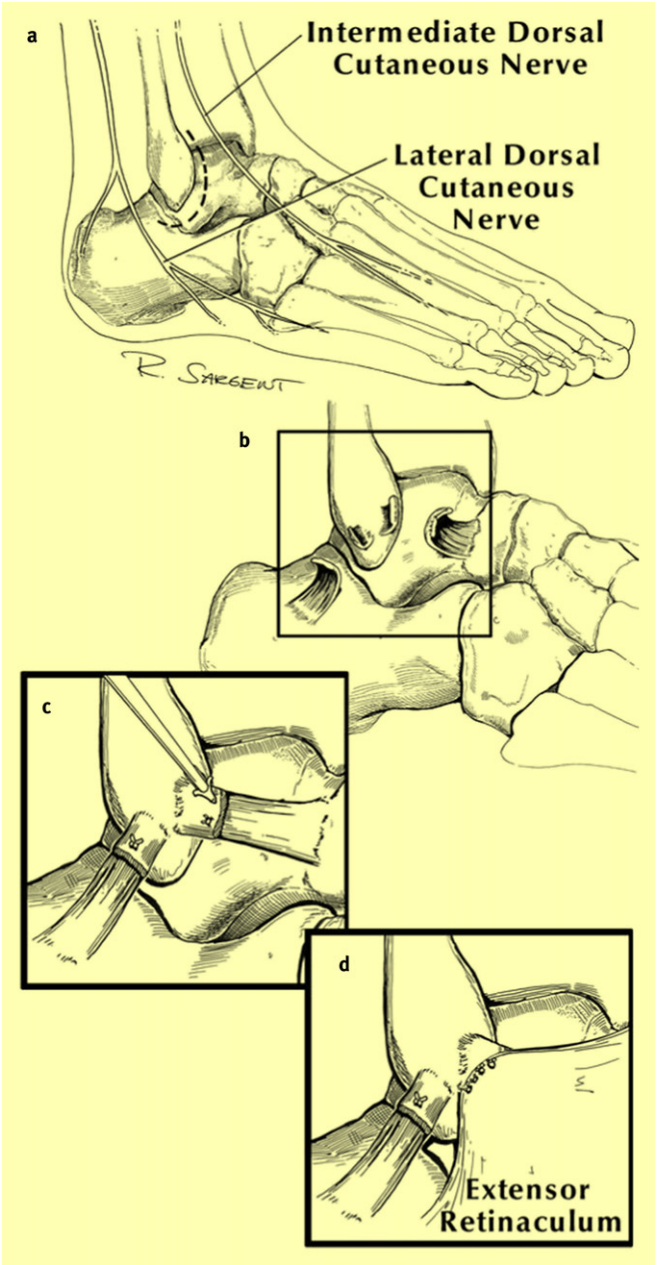

Figure 11 The Brostrom-Gould lateral ankle ligament reconstruction: (**a**) The surgical approach between the branches of the superficial peroneal and sural nerves. (**b**) Tears of the ATFL and CFL. (**c**) Direct repair and imbrication of ligaments. (**d**) The Gould modification with mobilization and advancement of the inferior extensor retinaculum. (*Reproduced with kind permission from J Baumhauer 2002 Journal of Athletic Training*).

imbricate. Additional stability to the subtalar joint may be obtained by imbrication of the inferior extensor retinaculum over the repair to the periosteum of the fibula, as described by Gould.[26] Post-operatively, a walking plaster or boot is applied for 4–6 weeks. Thereafter, the patient may commence proprioceptive training with a U-stirrup support. Athletes are instructed to use protective taping/bracing for 6 months after the repair.

Reported functional outcomes are excellent, with 87–95% success rates.[25–27] The advantages of anatomical repair include minimal surgical exposure, repair of host anatomy, preservation of ankle and subtalar joint motion and low morbidity. Unsatisfactory outcomes are reported in patients with generalized hypermobility, long standing ligament insufficiency and previous surgery. Other relative contraindications include obesity, hindfoot malalignment and very high demand athletes. The most severe common complication is iatrogenic injury to the superficial peroneal or sural nerve.

Non-anatomic stabilization: non-anatomic reconstruction utilizes a tendon or other graft as a weave tenodesis. The most common graft involves the use of peroneus brevis. The commonly used techniques are the Evans[28] (Figure 12) and Chrisman–Snook (Figure 13) procedures.[20]

The Evans procedure involves harvesting half of the peroneus brevis tendon, leaving the free arm attached distally to the base of the fifth metatarsal. The free arm is then passed though a drill hole in the lateral malleolus, from anterior to posterior, and is then sutured to itself. The degree of tension can be adjusted according to the position of the foot.

The Evans tenodesis does not recreate the normal biomechanical functions of the ATFL and CFL. Whilst ankle motion is not affected, anterior translation is not well controlled and subtalar motion is decreased.

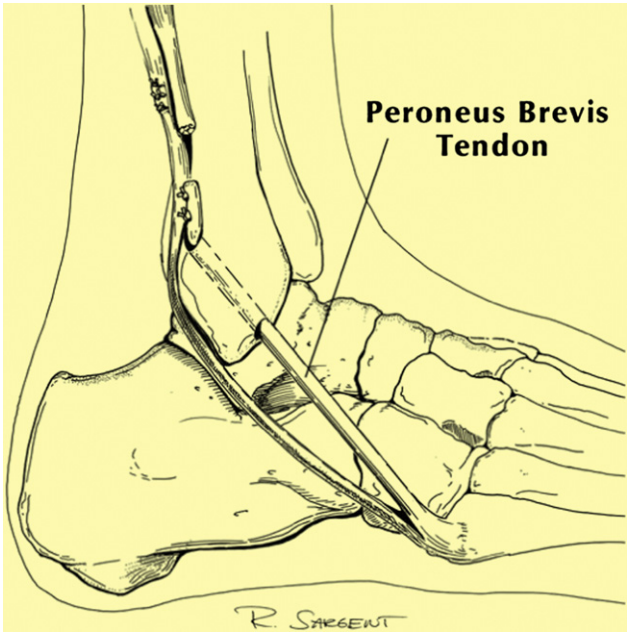

Figure 12 The Evans ankle tenodesis. One-half of peroneus brevis is harvested, leaving it attached at the fifth metatarsal base. It is passed from anterior to posterior through a drill hole in the fibula and sutured to itself. (*Reproduced with kind permission from J Baumhauer 2002 Journal of Athletic Training*).

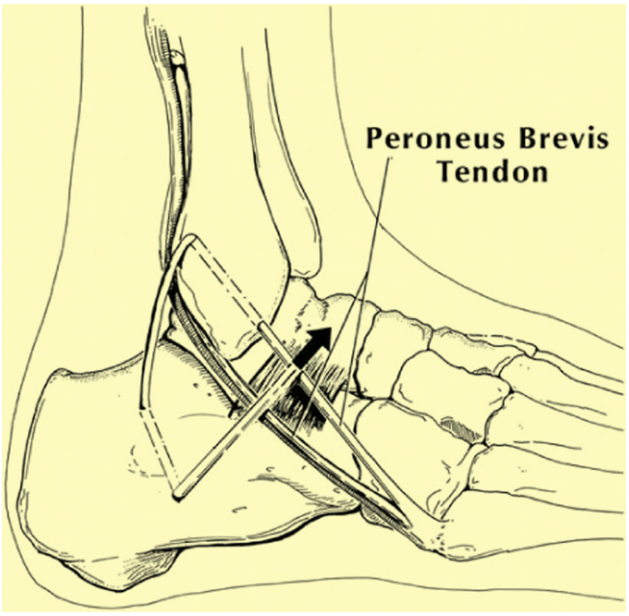

Figure 13 The Chrisman–Snook ankle tenodesis. One-half of peroneus brevis is harvested, leaving it attached at the fifth metatarsal base. It is passed anterior to posterior through the distal fibula and through a calcaneal bone tunnel and sutured back on itself. (*Reproduced with kind permission from J Baumhauer 2002 Journal of Athletic Training*).

The Chrisman–Snook procedure uses a split peroneus brevis tendon detached proximally, similar to the Evans procedure. It aims to reconstruct the function of the ATFL and CFL more accurately and involves weaving the tendon from anterior to posterior through the fibula and subsequently though a drill hole in the calcaneum before the tendon is sutured back upon itself. As a result, stability is better than the Evans procedure but it has the highest incidence of loss of inversion. Post-operatively, patients are placed in a walking plaster or boot for 6 weeks and a U-stirrup for a further 6 weeks. Active proprioceptive training is commenced at 6 weeks.

Reported early results are similar to anatomic repair, with 80–90% success rates. The greatest limitation of these procedures is the decrease in subtalar joint motion and the risk of sural nerve injury. Long-term follow-up studies have shown gradual deterioration of stability, restriction of range of motion, increased risk of degenerative changes and less satisfactory overall results as compared to anatomic reconstructions.

The advantage of non-anatomic reconstruction is the increased strength of the repair, which is particularly important in athletes where stability is more important than motion. In most cases, reconstruction is reserved for when the tissues are so severely attenuated that an anatomic repair cannot be carried out.

Surgical stabilization of syndesmotic instability

Surgical treatment of syndesmotic instability should be considered in symptomatic patients where mechanical instability is demonstrated on radiography. Syndesmotic stabilization can be performed in the subacute phase (6 weeks–6 months) and

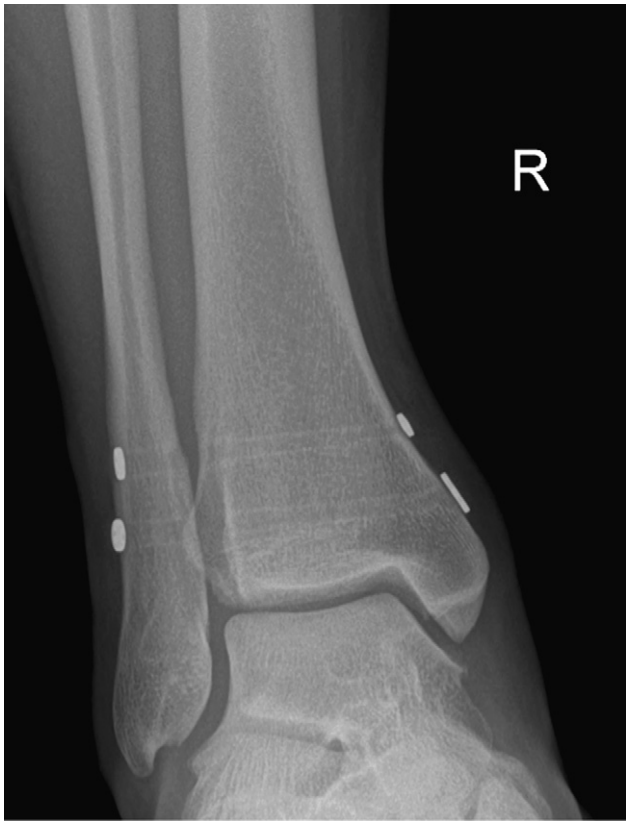

Figure 14 Syndesmotic reconstruction with suture button fixation. The syndesmosis has been reduced following arthroscopic debridement.

multiple methods have been described. The commonest techniques are insertion of a syndesmotic screw, bioabsorbable screws and suture button fixation.[29] Reduction of the syndesmosis is facilitated by reduction clamps under radiographic control. Arthroscopic debridement should be performed concurrently, to allow accurate reduction of the syndesmosis and to treat any intra-articular pathologies. If the joint surface is already degenerate on both sides then arthrodesis or arthroplasty may be needed.

The aim of surgery is to allow a degree of fibrotic healing of the syndesmotic ligaments using an implant strong enough to resist diastasis, to ensure early mobilization and to allow a degree of physiologic micromotion (Figure 14). There are a number of controversies with regards to implant positioning, post-operative management and implant removal but no technique has been shown to be superior.

Conclusion

Chronic ankle instability is not a single pathologic entity, and comprises a spectrum between functional and mechanical instability. Treatment should be based upon careful evaluation of the pathomechanics of the injury, clinical assessment and stress radiography. Arthroscopy should be considered where patients present with chronic pain suggestive of intra-articular pathology. Factors predisposing the patient to chronic instability such as a gastrocnemius contracture or hindfoot varus should be noted and treated as part of the surgical plan. A focused course of conservative management will often resolve patient symptoms, particularly if functional instability is the predominant problem. The results of surgical treatment of mechanical instability are excellent and the technique employed should be chosen based upon functional demand, patient factors and the pathological anatomy of the injury. ◆

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