LATERAL ANKLE/SUBTALAR JOINT INSTABILITY:
A Review of 42 Operative Cases

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Although the literature is very thorough on the pathophysiology and management of lateral ankle injuries, the author continues to search for answers to seemingly confusing clinical experiences. This paper reviews retrospectively 55 surgical cases for lateral ankle pathology of which 42 underwent stabilization by either a modified Brostom - Gould procedure, 34 cases or split peroneus longus lateral ankle stabilization, 8 cases. The other 13 cases were repairs of longitudinal tears in the peroneal tendons, predominantly peroneous brevis, lateral talar process factures, and subluxing peroneal tendon pathology.

Mechanical instability of the ankle is understood to be any movement of the ankle joint beyond the normal physiologic limits of the ankles range of motion Functional instability is the subjective feeling of ankle instability or recurrent, symptomatic ankle sprains due to proprioceptive and neuromuscular deficits.[1] Convention would then seem to dictate that any ankle in which both the calcaneofibular ligament and the anteriort talofib were found to be disrupted by visual inspection operatively would be termed mechanically unstable. It is the author's experience with 36 of the above cases in which the calcaneofibular ligament was found to be normal appearing intraoperatively that stimulated this paper.

All patients were treated by the author, all were referred after exhausting conservative care elsewhere, frequently with a history of anywhere from 8 months to 5 years of chronic lateral ankle pain and the vast majority were active duty US Marines. All patients complained of a subjective feeling of the ankle "giving way", recurrent ankle sprains, many had positive MRI findings and all felt the instability created a functional problem with their occupation and activities of daily living. In fact, the majority were on medical board by the military pending the anticipated resolution of their symptoms by surgical intervention.

It seemed counter intuitive to the surgeon to find that of the 42 cases, only 3 had complete ruptures of the calcaneofibular ligament and another 7 had what I subjectively termed partial tears or abnormal appearance. It is my belief that although we understand the entity of mechanically unstable ankles, we do much less so regarding functionally unstable ankles. This issue is further amplified by the general shift in clinical practice away from operative management of any acute injuries and a universal focus on functional recovery and conservative management. This places these patients all distinctly in the chronically unstable category and seeking surgical solutions.

The literature is "in" so to speak on the management of lateral ankle ligament injuries and the reader is referred to excellent articles and their references for those resources. They include the chapter Chronic Ankle Conditions by Schnirring-Judge and Perlman in the 3rd edition of McGlamry's Comprehensive textbook of Foot and Ankle Surgery, The Subtalar Joint Sprain by Gerard Yu in Update '97, Chronic Lateral Ankle Instability: Repair versus Reconstruction by Stapp, Smith and Challa in Update '96 and Tenodesis for Chronic Lateral Ankle Instability by Catanzariti and Mendicino in Update 2002.

In spite of all this research and reporting the author continues to mull over several questions regarding chronic lateral ankle instability such as:

Why is the calcaneofibular ligament relatively infrequently involved (23.8% in my series, Brostrom's original paper in 1966[2] states "about every fourth case there was also old rupture of the calcaneofibular ligament, which however, had healed without appreciable elongation in most cases), it seems counterintuitive? Brostrom found only 2 cases of his 60 to have notable elongation that failed to tense on supination, my series had 3 ruptures of 42 cases.

Why on occasion, 6 cases in this series, did the anterior talo fibular ligament appear normal? Are we actually treating subtalar instability in many of these patients and is this why I seem to be seeing an increased reading of sinus tarsi inflammation and ruptured interosseous ligaments on MRI in recent years? What accounts for those cases of seemingly unexplainable subtalar joint pain, not clearly fibrous coalitions or degenerative in nature? Is subtalar arthroscopy the answer?

Why do I seem to here more and more reports of

"high ankle sprains" yet don't see them in my practice? And lastly, if the Brostrom Gould procedure is the universally accepted technique for surgical stabilization, are we all doing it the same or just using the same terminology. It has been my observation at meetings and on viewing surgical tapes that many are calling a variety of surgical manipulations of the regional tissues a "Brostrom", when they may indeed be different techniques?

I believe the answer lies in a better understanding of a functionally unstable ankle and subtalar joint , one in which it is not necessarily mechanically unstable at all, but yet functions unstably.

We clearly understand several issues of lateral ankle pathology. We know the two primary ligaments are approximately 105 degrees apart, that the ATFL is stressed when the foot is planterflexed and CFL is relaxed. CFL is stressed when the foot is dorsiflexed. Fallet's study of 639 ankle injuries prospectively indicts an incidence of ATFL and CFL involvement 34.2% of the time.[3]

ATFL is universally altered in chronic ankle instability. We know 10 to 30% of all patients will develop chronic ankle instability. We believe from Freeman's work[4] that there is an afferent sensory pathway of proprioceptive fibers that directs appropriate position for joint function in addition to stimulating the muscular reflex arc for stabilization. Poor ligament healing may create a delay in sensory input and an attenuation and loss of proprioceptive function. We understand that early functional recovery provides the fastest recovery of ankle mobility and that secondary repair of the BrostromGould type are the standard, with caution given to patients over 225 lbs and instability of greater than 10 years or failed repairs.[5]

What we are not yet sure of is the role of subtalar joint instability and the role of stress to the ligaments of the tarsal canal. Most authors believe that the calcaneofibular ligament is the most important structure for subtalar stability followed by the ligaments of the sinus tarsi. Cadaveric studies seem to support this. Neither sectioning of the cervical nor interosseous ligaments causes much subtalar instability but sectioning of the lateral talocalcaneal ligament did.[6] Most mechanisms of subtalar joint sprain indicate inversion and dorsiflexion injuries. Of interest in this debate is the finding by Frey in 36 cases of subtalar arthroscopy with confirmed interosseous ligament injury that 7 had subtalar instability and that it manifest more as medial glide or shift of the calcaneous under the talus than an inversion.[7] Yu also speculates that perhaps the lateral talocalcaneal ligaments and joint capsule as well as the ligaments of the sinus tarsi complex play a larger role in subtalat instability then we think.[6]

Recent work by Tropp emphasizes the critical nature of subtalar joint mechanics to ankle instability. Effectively the subtalar joint allows the leg to rotate on the weight-bearing foot. Ground reactive forces act on the foot to create a moment acting on the subtalar joint. These forces normally act lateral to the subtalar joint axis and evert and dorsiflex the ankle. If the foot is inverted the axis moves laterally, producing an external load that further forces the foot into inversion. Interestingly a shoe makes the foot more vulnerable to hyperinversion because the added breadth of the shoe increases the length of the lever arm and friction between shoe and ground adds a shear force that creates more torque about the subtalar joint.[1]

It seems that functional instability lacks any clear relation to laxity or mechanical instability. The author's approach on this series was to perform a Brostrom Gould type repair with special emphasis on the retinacular reefing which will be pictured. No soft tissue anchors were used and the author performed all the surgeries himself. I do not have residents. The avoidance of soft tissue anchors was purposeful, it eliminated any bias or external pressure by product companies to book cases or "look" for lateral ankle cases to enhance my income in some fashion via either lecturing or endorsement of a certain product. In fact, the vast majority of these patients are active duty military, thereby covered by Tricare insurance and are my absolute worst payor, worse than Medicare. Soft tissue anchors have their place and can be very useful, they are not however absolutely essential

Based on these experiences I have modified some personal beliefs and conclusions on the exact pathophysiology of these delayed chronic ankle injuries and in so doing have theorized potential answers to my own previously posed questions above.

Brostrom himself concluded that rupture of the calcaneofibular ligament seems to be of minor importance in weakness and feeling of instability in the ankle and recurrent sprains. I concur. We may see less calcaneofibular ligament because we do less acute repairs today. There are highly likely factors specific to my subset of patients. Due to their military occupation and frequent mobilizations in Afganistan and Iraq post 9/11 they were probably all inadequately functionally rehabilitated. This is part of military life anyway where the emphasis is get back as quickly as possible. I propose that outside of a team physician role such as pro football or basketball, where injuries are more likely to be on stable level ground, my patients suffer more true plantarflexion injuries. This accounts for the absence of high ankle sprains and CFL ruptures. Those injuries likely occur more with the foot planted firmly on even ground and then forcibly dorsiflexed and inverted. A basketball or football player for

example cutting or planting on a hard unforgiving surface. Plantarflexion injuries may be more likely on uneven hilly terrain, running down hill or diving for cover from enemy fire. Less resistant surfaces such as desert sand and non athletic shoewear such as combat boots likely play a role. Combat boots may even increase the shear force component that I believe affect subtalar joint derangement.

This may lead to more subtalar sprains as well. As the CFL remains relaxed in plantareflexion, the next logical structures stressed are the contents of the sinus tarsi. Improved MRI sequencing and reading may also account for higher clinical suspicions of interosseous ligament tears. The ATFL is the roof of the sinus tarsi in a sense as any resident whom has wandered too far dorsally during the lateral dissection of a triple and cut into it can tell you and once ruptured in the plantarflexed foot it seems logical that the force propagates to the sinus tarsi. The talus is destabilized laterally after an ATFL rupture and has no muscular attachments. It is tethered primarliy by the interosseous ligaments. The CFL does not even tether or attach to the talus. I believe the assessment of subtalar instability must come AFTER ATFL rupture, the primary restraint of ankle stability or the anterior cruciate ligament of the ankle as Inman called it. Perhaps chronic functional lateral ankle stability is really weakening/rupture/attenuation of the ATFL and the ligamentous constraints of the sinus tarsi and not CFL at all or rarely.

Those patients that have pain to inversion or rotatory stress on clinical exam yet turned out to have normal CFL probably had subtalar joint pain. During this same time frame of these cases I performed 35 isolated subtalar joint fusions of which 5 did not have a firm diagnosis yet had chronic subtalar pain and often peroneal spasm. They may best be termed fibrous coalitions but all imaging was normal and no evidence of fibrous coalition was encountered intraoperatively. Subtalar pathology is likely under-appreciated and probably at least in part responsible for the functional instability clinically in those patients in which I encounterd a normal appearing ATFL during surgery.

Subtalar joint instability may very well be more of a horizontal or transverse plane phenomenon than an inversion or frontal plane instability. Frey's study indicates this and the cadaveric studies' conclusions about the role of the calcaneofibular ligament may be inaccurate in a functional sense, especially with plantarflexion injuries and with the variables added by shoegear. They also don't necessarily account for considerations of stability AFTER an ATFL rupture.The subtalar motion is screw like and lends itself more to translatory instability than inversion in my view. This also correlates more with forces at play in a subtalar joint dislocation. The instability is likely

more anterior than along the wall of the posterior facet where CFL resides, again in the plantarflexed foot

These observations may differ from accepted conclusions historically due to better MR scans and reading and the advent of subtalar arthroscopy that confirms more subtalar pathology and tarsal canal ligamentous involvement.

The surgical repair of the Brostrom Gould type is an anatomic repair. I extend the incision higher than normal, coursing from the distal inferior tib fib ligament region along the anterior course of the fibula to the level of the CFL. This allows for exposure of the anterior ankle joint and distal tibial as well as inspection of the talar dome. Associated pathology in this series was typically fibular and tibial fracture fragments and 5 Basset's lesions were encountered and debrided. Other pathology was two hypertrophic peroneal tubercles and old calcaneal beak fractures Ankle arthroscopy is reserved in my hands for symptomatic medial ankle pain in conjunction with lateral instability. Effective repair is critical and I feel the role of the extensor retinacular repair is under emphasized. Many people use the term Brostom yet the surgical manipulations of the local tissue is often varied due to surgeon experience, beliefs or talent. Because the retinaculum arises in three separate bands from the floor of the sinus tarsi it has the ability to stabilize the subtalar joint as well. I use a three location pants over vest repair along the anterolateral ankle. The "pants" is the the retinaculum, the "vest" is the periosteum on the fibula. The first throw reinforces the ligament free region between the ant inf tib fib lig and the ATFL. This is the region of universal anterolateral ankle joint capsule disruption. The second throw is directly over the ATFL which may or may not need to be imbricated.

The third throw is between the ATFL and the CFL and essentially over the lateral process of the subtalar joint. All three throws are placed with #2 fiberwire by Arthrex, it is simply the strongest suture available and then hand tied with the foot in neutral to slight eversion. This approach covers all anatomic variants of the ATFL which can arise in 2 bands , beginning just inferior to the anterior inferior tib fib ligament and an inferior band near the CFL.

Stress testing was not routinely done as it was not going to change procedural selection and ligaments were inspected intraoperatively. The split tendon transfer procedures were done in one case of a failed Brostom in a a noncompliant patient 8 months postop, one in a case of complete rupture of ATFL, CFL and a peroneoud brevis tear, and one case of subtalar instability without lateral ankle pathology and 5 cases where clinically I felt

subjectively a Brostrom would not be strong enough.

All patients have done well to my knowledge with the exception of 2 patients whom had continued pain around the lateral ankle and subtalar joint. I could not explain their pain. One was lost to followup and the other declined any further treatment. A long term followup will be attempted on these patients but is unlikely to be fruitful. The nature of the military is that if they are doing fine they don't come back for followup after 3 months, there are more pressing concerns in their lives and the lives of their families. Many were seen at 6 months and a few at 1 year but the average was approximately 3 months. Deployment issues particularly at Cherry Point and Camp Lejuene NC for the Afganistan and Iraq conflicts affected followup tremendously. Neverthless, it is felt that these results clinically matched the literature as universally well accepted and tolerated. The reality for these active military personnel is that after they are out of the cast and back to full activities or doing rehab on base they do not return to the civilian provider, myself, unless there is a problem.

Standard postoperative management was 6 weeks non weightbearing in a cast and then weeks in a fracture boot followed by ankle brace support and functional rehab with physical therapy.

It is my hope that the reader will find this article to be clinically honest, useful in their own hands in sense of shared clinical experiences and perhaps stimulating as we all continue to query why we do what we do and what works for our patients.

Figure 1A. Incision used for brostrom-gould procedure. Incision is extended more superiorly to better visualize across the anterior ankle joint and the region of the anterior inferior tibfib ligament.

Figure 1B. Cadaveric specimen.
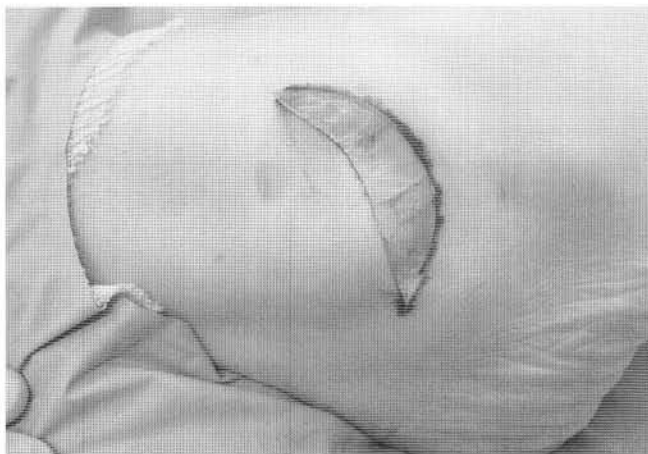
Figure 2. Dissection through the skin in to subcutaneous tissue. Multiple veins are frequently encountered and handtied or bovied. Inspection for branches of the superficial peroneal nerve is performed.

Figure 3. Blunt dissection of subcutaneous tissue off the extensor retinaculum is performed.

Figure 4. Blunt dissection is continued, this is critical to expose the retinaculum for later incorporation in the closure.
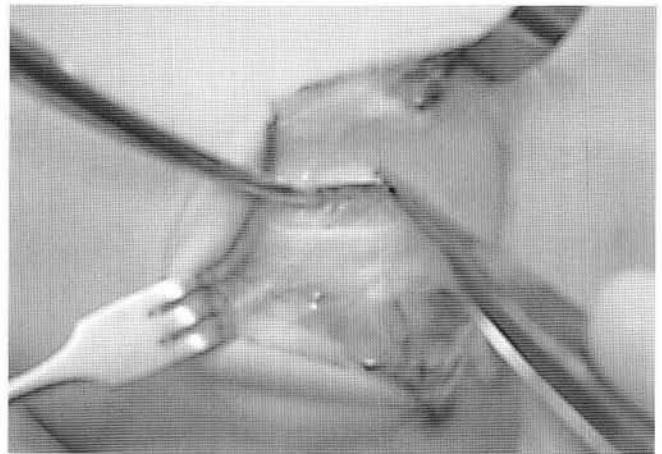
Figure 5A. The retinaculum when exposed correctly is a readily identifiable distinct layer.
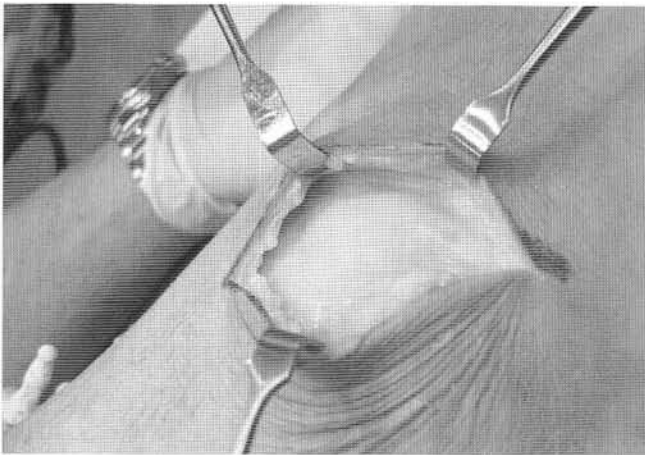

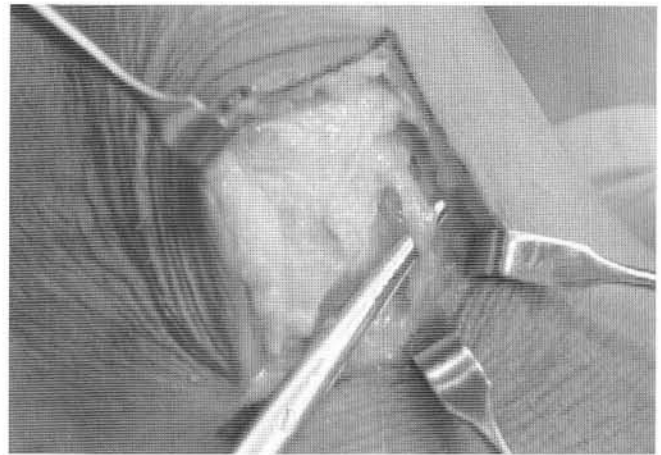Figure 5B. Cadaveric specimen showing the deep fascia and exposure of the extensor retinaculum.

Figure 5C. Cadaveric specimen showing the superficial peroneal nerve. A venous plexus is often identified here as well.
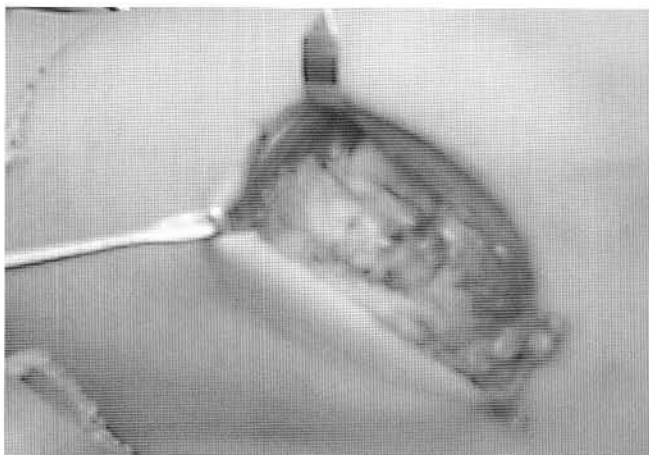

Figure 6A. The ligament free region between the anterior inferior tib fib lig and the anterior talofib lig is incised. It is usually thicked , scarred capsular, synovial type tissue but not always.
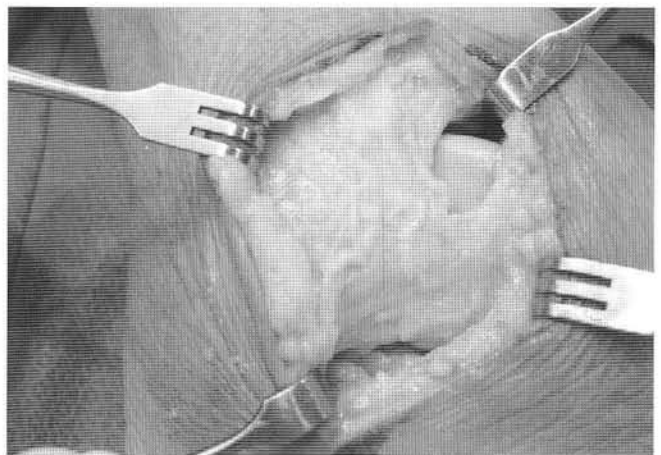
Figure 6B. Cadaveric specimen, note the excellent visibility to the ankle joint. The ATF ligament is intact.
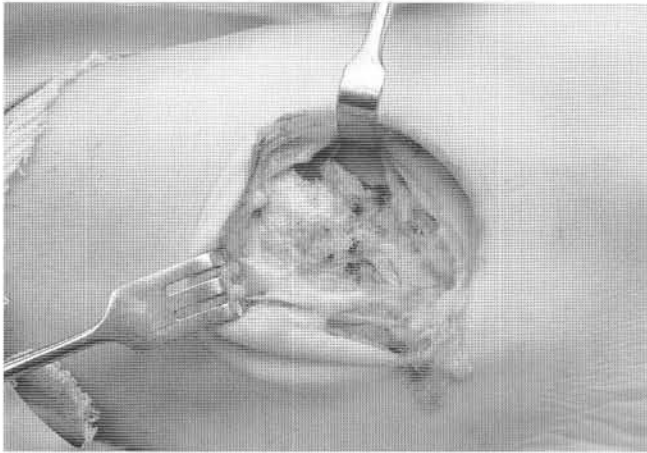
Figure 7. Clinical view into ankle joint anteriorly.
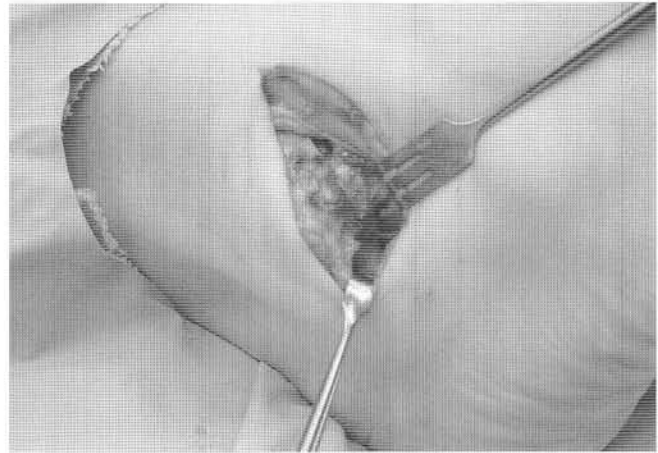
Figure 8. Inspection of the calcaneofibular ligament via retraction of the peroneal tendons.
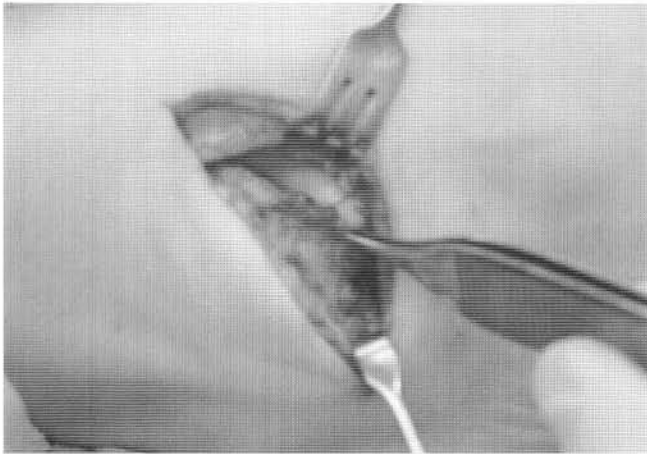
Figure 9A. ATF ligament is inspected and incised for imbrication.
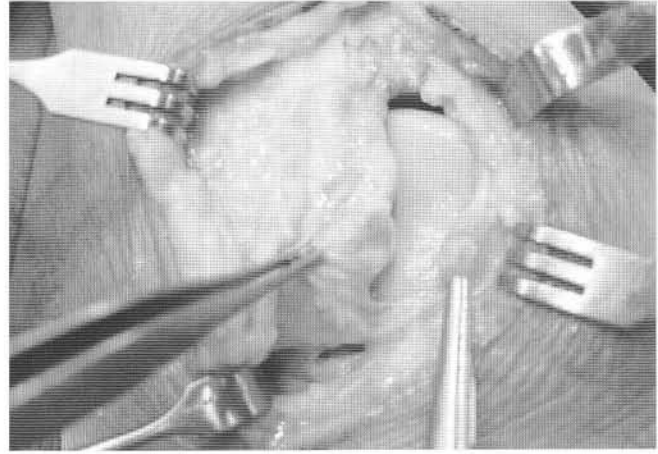
Figure 9B. Cadaveric specimen, note the distinct difference in crosssectional appearance of the incised ligament versus surrounding soft tissue and capsule.
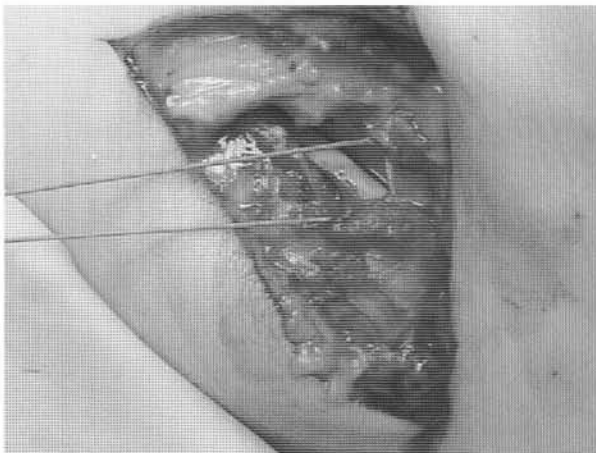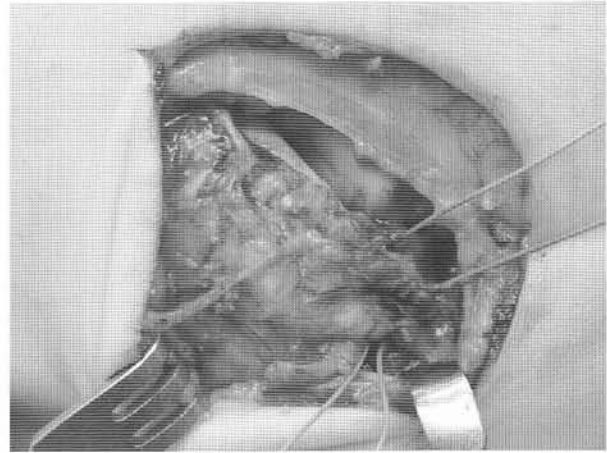

Figure 10. Suturing of the ATF ligament.

Figure 11. Suturing of the CF ligament. If the ligament is attenuated, it is often easier to place the suture lines first , then cut the ligament and then reef or imbricate it.
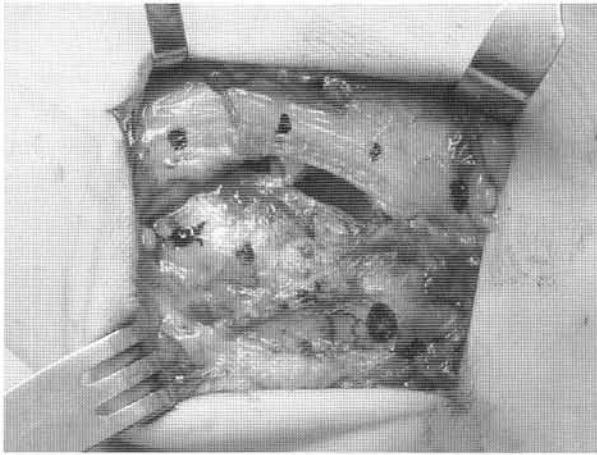
Figure 12A. Proposed placement of the 3 separate suture lines of #2 fiberwire or similar in the extensor retinaculum.
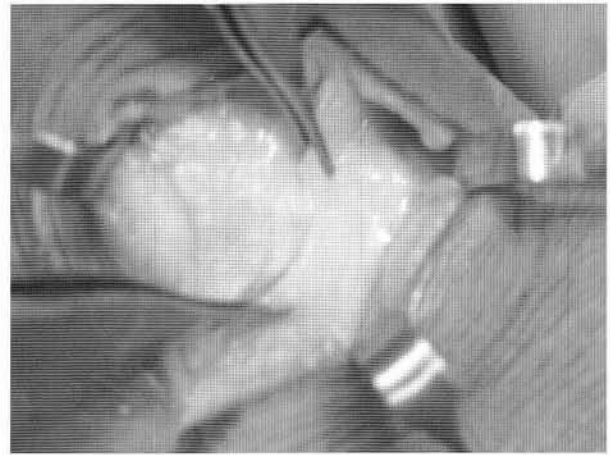

Figure 12B. Cadaveric specimen showing the proposed mobilization of the extensor retinaculum.
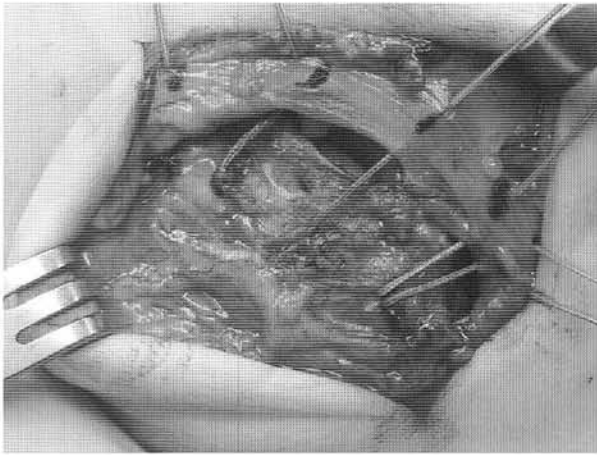

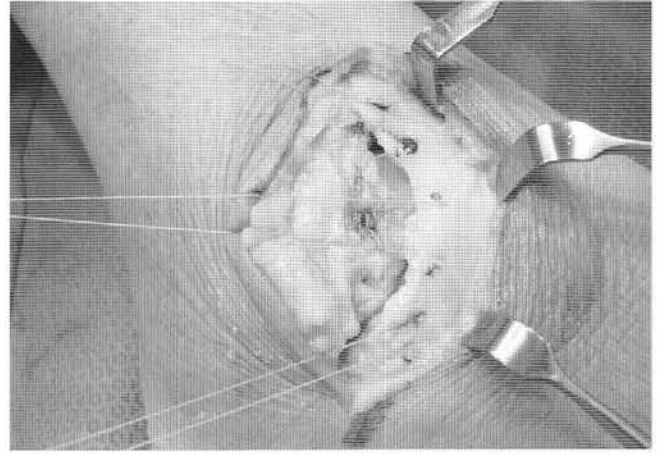Figure 13A. Suture lines placed in the retinaculum

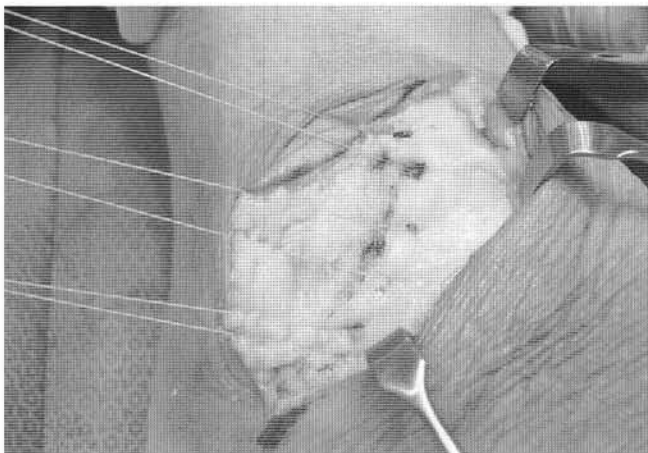Figure 13B. Cadaveric specimen, suture lines in place

Figure 13C. Suture lines advanced, in a pants over vest fashion, pants is the retinaculum, vest is periosteum on the fibula.
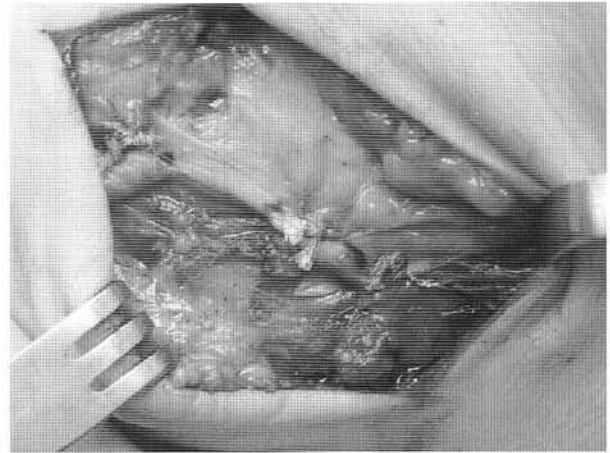

Figure 14A. Closure of the extensor retinaculum repair to the periosteum on the fibula.
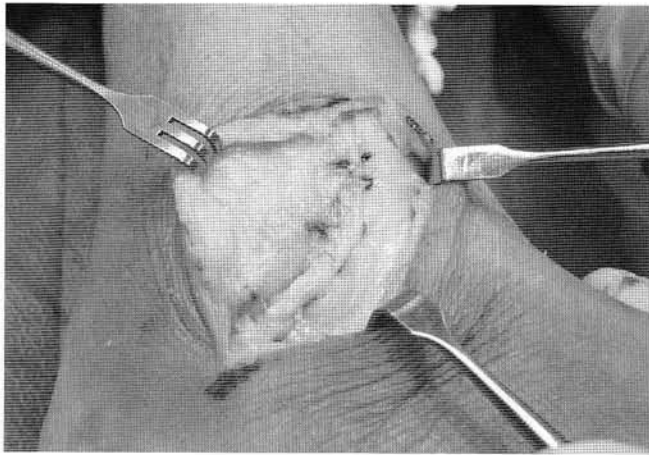
Figure 14B. Cadaveric specimen of the same.

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ADDITIONAL REFERENCE

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