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Consensus of the 11th Round Table Cambridge, June 2024

Thomas Collins
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What, why and where? Cause versus effect of foot disorders

OrthoSolutions Group
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Consensus of the 11th Round Table Cambridge, June 2024

Course Facilitator:
Dishan Singh

Chairpersons:
Rick Brown
Mark Davies
Jitendra Mangwani
Lyndon Mason
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Hosted by:
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Distilled in this document are the thoughts and opinions, with consensus where possible, of 25 Orthopaedic Foot and Ankle Consultant Surgeons who gathered from across the United Kingdom, Germany and USA. A basis of invited lectures introduced open and frank discussion from which consensus was sought. The statements herein only represent those of individuals and no claim is made that they are irrefutable.
All the percentage figures quoted represent the proportion of the surgeons present who voted on the subject in discussion.

Preface

The 1st Round Table meeting was held in Padua, Italy in June 2011 and, thereafter, rapidly became a prominent highlight in the annual foot and ankle calendar. About 25 senior members of BOFAS and 2 to 3 invited international participants meet in a hotel setting for 3 days to discuss selected topics with ample time for an informal discussion. This Round Table approach is conducive to a more effective learning experience and generates considerable debate, but the group is able to reach a consensus on many issues. The proceedings of the meeting, the literature review, the personal experience, the discussions and the consensus views of all those who participate are collated in this booklet with the thorough, skilful and diligent assistance of 2 scribes (Thomas Collins and Elinor Flatt).

The theme for the 11th Round Table which was held in 2024 at Cambridge, England was *"What, Why and Where."* The focus was on the strategies to not only treat a disorder but also to identify and deal with its causes. We chose topics to discuss **what** is the problem, **why** did we get here and **where** are we going? Are long-term outcomes of surgery for e.g. the *progressive flat foot deformity* poor because we address the secondary effect rather than the cause?

Our distinguished local participants had the privilege of an international perspective from Professor Stefan Rammelt from Dresden, Germany and Dr. David Garras from Illinois, USA.

Ortho Solutions UK Ltd. have kindly provided financial and administrative support to the meeting since its inception. I would like to express my gratitude to Emma Keech and Sheena Easton for their hard work in ensuring the smooth running of the course.

This booklet collates the literature review and the views of all those who participated. This booklet does not represent Level I evidence derived from prospective randomized controlled trials but represents the compilation of the combined experience of 25 British and international orthopaedic surgeons.

We have selected a short list of references to keep the booklet concise and easily readable.

I hope that you will find something of use and relevant to your own practice.

Dishan Singh ***MBChB, FRCS (Orth)***
Retired Consultant Orthopaedic Surgeon
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Summary of Sessions

Session 1: Hallux valgus

Chaired by Jitendra Mangwani

1.1.What do we know about pathogenesis? *Carolyn Chadwick*

Throughout the literature the pathogenesis and aetiologies of hallux valgus (HV) are often discussed in combination despite being separate entities. As a reminder - pathogenesis is the process or mechanism by which a disease or disorder develops, whilst aetiology refers to the cause or set of causes of a disease or condition. These may be intrinsic, extrinsic or idiopathic in nature.

A review paper published in 2011 described the pathogenesis of HV including intrinsic and extrinsic factors contributing to this, anatomical variations, biomechanics and associated conditions.[1] Many further publications on the topic frequently refer to the work in this paper.

Gender

The true prevalence of HV with regards to gender is not clear as most of the data looks at patients undergoing surgery rather than diagnosis alone. It does however appear that the ratio for this is higher in favour of females (approximately 15:1). This may be accounted for by the increased incidence of ligamentous laxity, 1st ray hypermobility and choice of footwear in women. Osseous anatomy may also be a contributing factor with females tending to have a smaller and more rounded 1st metatarsal head, a more adducted metatarsal and different articular morphology both proximally and distally.[1]

Using a 3D CT modelling technique Yamatsu et al (2023) compared male and female control groups and a HV group. They concluded that there were several differences in the anatomy of the proximal phalanx and 1st metatarsal in the HV group, though it was difficult to conclude whether this was cause or effect. This study also looked at the size and orientation of the proximal and distal articulations of the metatarsal which are of relevance in terms of tarsometatarsal joint (TMTJ) obliquity and the distal metatarsal articular angle (DMAA). There is a well-known association of HV with TMTJ obliquity however this is based on radiographic appearances which do not consider the change in orientation when the foot is loaded. An increased DMAA is more commonly seen in juvenile HV and is often an unreliable measurement on radiographs as it can be altered depending on elevation or pronation of the metatarsal.[2]

When looking at the anatomy based on gender, the same study found that in male patients with HV, there tended to be an increased lateral inclination proximally of the proximal phalanx, a shorter & wider first metatarsal, a larger articular surface of the metatarsal head and a metatarsal head pronated relative to the proximal articulation. In female subjects with HV, the proximal phalanx tended to have a lateral and dorsal inclination proximally, with metatarsal changes similar to male HV patients but with added lateral inclination of the metatarsal head.[2]

Age

Although the most common age of presentation of HV is between the 4th and 6th decades, the initial pathological changes probably occur during adolescence. Joint kinematics and altered loading patterns, such as increased lateral pressure, increase the risk of HV, however age itself is a poor predictor of an increased HV angle.[1]

Genetics

There appears to be a genetic association with HV with 90% of patients presenting with HV having a 1st degree relative with the condition. It's thought to be an autosomal dominant pattern of inheritance with the strongest genetic link associated with male patients presenting with juvenile or adolescent HV.[1]

Extrinsic factors

Whilst there is an association between HV and increased first metatarsal loading, a valgus moment on the proximal phalanx, and repetitive trauma to the medial soft tissues, there has been no proven link with high BMI, excess walking or jobs involving manual work/heavy lifting i.e. risk factors for abnormal loading of the first ray.[1]

Footwear has also been implicated as a cause for HV. As the hallux is pushed forwards in a narrow toe box there tends to be increased first metatarsal loading, a valgus force on the hallux and a tendency for the metatarsal to pronate. However, this mechanism is more likely relevant in the progression rather than initiation of deformity as only a proportion of people who regularly wear shoes with a narrow toe box go on to develop HV. There is also a proportion of patients in populations that do not wear shoes who will go on to develop HV (2-33%).[3]

Initiation and progression of HV deformity

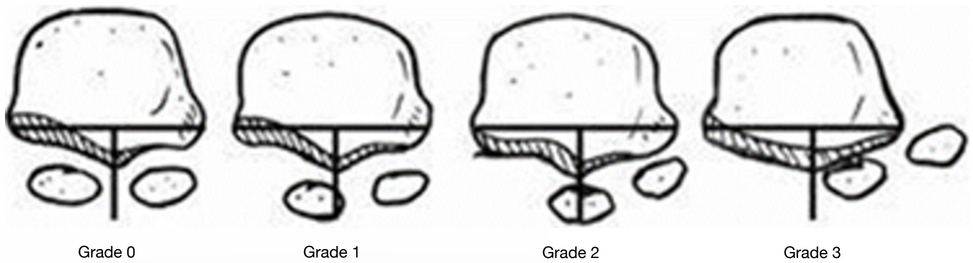
Stability of both the MTPJ and TMTJ are key to maintaining satisfactory alignment, however the first ray is inherently unstable. It relies on static structures in the form of the joint capsule, medial collateral ligament (MCL) and medial sesamoid ligaments. Dynamic stability is provided by the abductor and adductor hallucis working in synergy in a fashion similar to the rotator cuff, but also with contributions from extensor (EHL) and flexor hallucis longus (FHL).

It is still unclear what the initiating cause of HV is, however the primary failure appears to be in attenuation of the medial structures including the MCL and medial sesamoidal ligament. It has been hypothesized that enthesopathy of the metatarsosesamoid ligament may be the initiating factor in development of HV.[4]

Once the medial structures have failed, the metatarsal head is permitted to migrate medially and rotate in the frontal plane, whilst the proximal phalanx moves laterally and pronates in the axial plane. Varus deformity then develops at the TMTJ, the process of which may be accelerated by instability or obliquity at the joint.

The sesamoids lie within the tendon of flexor hallucis brevis (FHB). FHB inserts into the proximal phalanx of the hallux but is also tethered to the second metatarsal via the transverse metatarsal ligament. Therefore, as the first metatarsal moves medially due to loss of medial restraints, the sesamoids remain located relative to the 2nd metatarsal with apparent rather than true lateralisation. The lateral sesamoid comes to sit in the intermetatarsal space with the medial sesamoid sitting under the metatarsal head. This abnormal position of the medial sesamoid can lead to erosion and wear of cartilage and the normal intersesamoid crista on the plantar surface of the metatarsal (Figure 1). This flattening of the crista can lead to further instability. As the metatarsal head becomes more prominent medially, further pressure from footwear can cause bursal inflammation compounding any existing crowding within an enclosed shoe.

Figure 1



Deforming forces are important in the maintenance of HV. As the proximal phalanx deviates laterally, the FHL and EHL tendons bowstring laterally causing a valgus moment at the metatarasophalangeal joint (MTPJ). The metatarsal head is permitted to pronate as it falls off the medial sesamoid. Further pronation and plantarflexion of the hallux occur as the abductor hallucis is defunctioned and there is unopposed action of the adductor hallucis. As the metatarsal head elevates and medially displaces plantar pressure is transferred laterally, eventually causing transfer metatarsalgia to the lesser metatarsal heads (Figure 2).

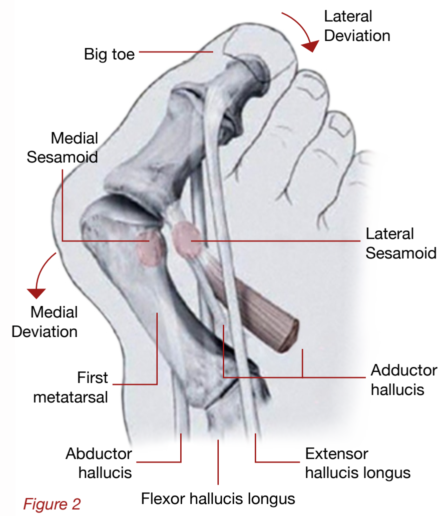


Figure 2

Deformity and rotation in HV

Traditionally HV was considered as a biplanar deformity with assessment made on radiographs in two planes, anteroposterior (AP) and lateral. HV was described as a valgus deformity in the transverse plane with elevation of the ray in the sagittal plane. Though first described by Mizuno in the 1950's there has been an increasing amount of focus in recent years on the rotational element of the metatarsal in the axial plane and the importance of pronation contributing to the deformity.[5]

The understanding of HV as a multiplanar deformity has been helped through the use of advanced imaging techniques. Whilst it is widely agreed that axial rotation is clinically relevant, no standardised methods for measurement currently exist. It has been shown in the literature that metatarsal pronation is associated with a decreased arch height[6], hypermobility, first ray torsional changes[7], and hallux rotation[8]. Okuda et al described the 'lateral head round sign' as a radiological marker of metatarsal rotation, whereby the lateral edge of the metatarsal head comes into view on the weight bearing AP view with pronation. It was postulated that this was a risk factor for development of HV but also a risk factor for recurrence after surgical correction. This, however, should be used with caution given the role that positional factors, such as dorsiflexion or plantarflexion of the first ray, may have on the projection of the metatarsal head on plain radiographs.[9]

A systematic review summarising the role of metatarsal rotation in HV and operative considerations for this concluded that metatarsal rotation is a deformity occurring independently of the HV angle and intermetatarsal angles on standard plain radiographs. Many additional rotational corrective procedures have been described with early results showing favourable outcomes, high patient satisfaction and decreased recurrence rates. However, the literature available is heterogenous, with most studies describing relatively new techniques, with short term follow up and results lacking statistical and clinical significance. More work is required to look at longer term outcomes and to determine at what level the rotation is occurring and should therefore be corrected i.e. at the TMTJ or within the first metatarsal itself.[10]

Contributing foot pathology

Metatarsus adductus is associated with an increased prevalence of HV. This deformity is seen more commonly in male patients and is associated with juvenile HV. Correction of HV in patients with metatarsus adductus is less predictable and recurrence rates are higher. This may be due to underestimation of the deformity and in severe cases consideration should be given to concomitant correction of second and third metatarsal deformities.[1]

Gastrocnemius tightness has been implicated as an intrinsic factor in the development of forefoot conditions such as HV. It has been observed that the gastrocnemius and hallux have a close anatomical and biomechanical relationship.[11,12] Biomechanical studies have shown that early heel raise during gait associated with gastrocnemius tightness increases load at the first MTPJ.[13] Gastrocnemius tightness also increases the windlass mechanism which, through tension of the middle and medial bands of the plantar aponeurosis inserting into the proximal phalanx base, increases the valgus force at the MTPJ.[1]

There is a consensus that ligamentous laxity increases the risk of developing HV through impairment of the load bearing capacity of the first metatarsal. This in turn induced higher deforming forces and moments at the first MTPJ contributing to malalignment here. Cho et al (2018) compared outcomes for patients with and without generalized ligamentous laxity after undergoing a proximal osteotomy for HV. There were no statistically significant differences between each group in terms of clinical and radiographic outcomes with generalized ligamentous laxity demonstrating no definitive effect on post-operative recurrence of HV.[14] First ray hypermobility may be part of generalised ligamentous laxity but should also be considered an entity and contributing factor by itself. A recent systematic review concluded that it is difficult to say whether it is the cause or consequence of HV, and although joint stabilising procedures such as the Lapidus are valid, there is evidence to show good results with other osteotomies that may both correct the HV and stabilise the first ray.[15]

Summary

Hallux valgus is a common condition with a multifactorial pathogenesis and aetiology. Whilst there is no doubt that the medial structures must fail for the deformity to occur, there remains ongoing debate as to the initiating cause and predictors of progression and symptoms. Intrinsic and extrinsic causes must be considered. Anatomical variation, altered biomechanics, and coexisting pathology all play an important role in understanding the deformity and how to treat it.

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1.2. Long-term results of metatarsal osteotomies +/- Akin

Adam Lomax

One of the main challenges in reviewing outcomes of metatarsal osteotomies is the variation in surgical techniques described in the literature. This includes variations in the osteotomy itself, intra-operative fixation, associated soft tissue releases and capsular repair, and any additional procedures. The second challenge is trying to define exactly what recurrence of hallux valgus (HV) is. Loss of radiographic measurements, in particular hallux valgus angle (HVA), are commonly quoted in the literature as defining recurrence, however whether this is any loss of the HVA, or reduction compared intraoperative imaging is often not reported. Unfortunately, there is also considerable heterogeneity in reporting of outcomes following osteotomies for HV, with many studies publishing retrospective data and no exact same technique repeated across the literature.

Chevron osteotomies

A meta-analysis of 17 studies looked at outcomes of distal metatarsal osteotomies with a minimum follow up of 5 years. The majority of osteotomies performed were Chevron osteotomies with no documented Akin osteotomies. Approximately half of the studies described having performed a lateral release of some description. There was a wide variation in the quoted recurrence rates of 0-73% across all the included papers.[1]

Two of the included papers followed up the same cohort of patients at separate 11- and 15-year time intervals. A distal Chevron osteotomy was performed on 90 feet with HV. Their technique described no fixation but stabilisation of the osteotomy with a capsular flap sutured through drill holes into the first metatarsal metaphysis (Figure 1). They described no lateral release or associated Akin osteotomies being performed in this cohort. They defined recurrence as a HVA of over 20 degrees on radiographs. A recurrence rate of 0% was documented at each follow up interval with 97.6% of patients being pain free and 95.8% being satisfied cosmetically (these figures remained the same across both papers).[2,3]

Torkki et al (2001) followed up 106 feet having undergone Chevron osteotomy plus lateral release without fixation. In this paper recurrence rates were documented at 62% based on a HVA of over 15 degrees at follow up. Despite this only 7 patients had a reoperation due to recurrence.[4]

Pentikainen et al (2015) found a recurrence rate of 73% when classified as a HVA over 15 degrees. They did however comment that no revision surgery was required as 'each case of recurrence was painless'. When they looked at pre-operative radiographic measurements, they found that increased HVA, IMA and DMAA all correlated with recurrence. Sesamoid position and lack of joint congruence pre-operatively were also significantly associated with recurrence.[5] This perhaps suggests that the Chevron osteotomy was being used in more significant deformities than it is powered for.

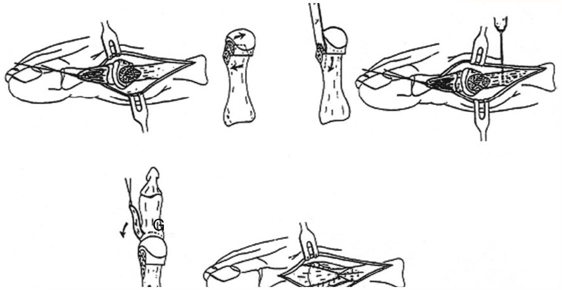

Figure 1

Scarf osteotomies

Early publications on Scarf osteotomies for HV in the 1990's showed relatively poor outcomes with loss of position, troughing on radiographs and nearly half of patients reporting dissatisfaction.[6] However, techniques and outcomes have progressed since then with improvements in patient reported outcomes.

A long-term follow up of a randomized controlled trial comparing Scarf and Chevron osteotomies found a similarly high recurrence rate in both groups (78% for Scarf and 73% for Chevron) when defined as a HVA over 15 degrees. However, pain and function scores did not correlate with recurrence and remained improved, with the majority of patients (81% Scarf and 76% Chevron) stating they would have the same treatment again. The authors suggested the lack of an associated Akin osteotomy and insufficient lateral release may have contributed to the high rate of radiological recurrence.[7]

Further work on long-term results following Scarf osteotomy with lateral release showed significant improvements in AOFAS scores at final follow up, with all radiographic measurements (HVA, IMA, DMAA, sesamoid position) having significantly improved from pre-op to final follow up. Despite this they still found a recurrence rate of 30% when defined as a HVA over 20 degrees.[8]

A systematic review of the Scarf osteotomy for HV found that the higher rates of documented recurrence were in those papers with longer term follow up, whilst recurrence rates in the short term (2 years or less) were much closer to 10%. This discrepancy between recurrence rates in the long versus short term are unclear, though the availability of short-term evidence is greater (9 studies with a mean follow up of 24 months, compared to 2 studies with long-term follow up). They concluded that there is 'grade B fair quality evidence that Scarf osteotomy is associated with a recurrence rate of up to 10%'.[9]

Addition of Akin osteotomy

A retrospective study of patients undergoing a Scarf osteotomy with and without an additional Akin osteotomy found superior radiological outcomes when concomitant Akin osteotomy is performed. Radiographic recurrence in this group was 1.6%, compared to 14.7% in the group that underwent Scarf osteotomy alone.[10] They then went on to look at factors that may influence recurrence and found statistically significant increases in HVA recurrence in the Scarf only group if the pre-operative proximal to distal phalangeal articular angle (PDPA) (Figure 2) on standing radiographs was over 8 degrees.[10,11]

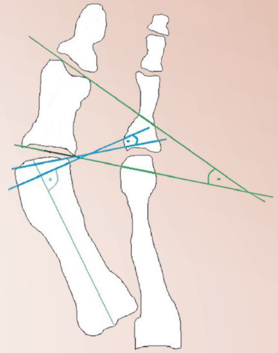

Figure 2

Summary

The long-term results available for Chevron and Scarf osteotomies are based on variable reporting of different techniques. They demonstrate poor rates of radiological recurrence but good patient satisfaction and low revision rates. This makes the results hard to apply to individual practice, however what we can say with more certainty is that a recurrence rate of 10% at 2 years appears reasonable with more consistent data for this across wider literature. Finally, research suggests that the addition of an Akin osteotomy should be considered when the PDPA is over 8 degrees on pre-operative radiographs.

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1.3. Long term results of Lapidus type operations

Rick Brown

The Lapidus procedure has been used in the treatment of hallux valgus (HV) for many years, though its use and how it is performed has progressed over the last few decades. Its popularity amongst foot and ankle surgeons has increased again recently due to its recognised benefit in the treatment of other foot and ankle pathology, in particular progressive collapsing flatfoot deformity (PCFD).[1,2] Variations of the procedure, such as the 'LapiCotton', have also been described for the treatment of more complex deformities (Figure 1).[3]

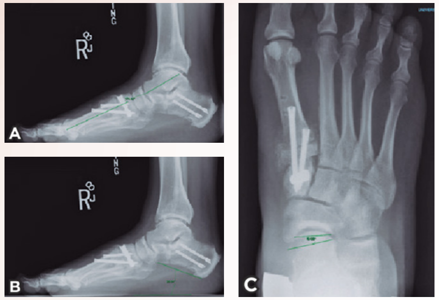

Figure 1

The Lapidus procedure is utilised for a number of underlying foot and ankle pathologies and when reviewing the literature for long term outcomes it is important to distinguish the pathology being treated. Indications for a Lapidus procedure include:

- First tarsometatarsal joint (TMTJ) instability (congenital or post-traumatic) with arch pain or HV
- Medial column collapse in PCFD
- Midfoot arthritis
- HV with an extreme intermetatarsal angle

Lapidus himself first published results for correction of metatarsus prima varus in HV in 1934. He believed that the primary cause of hallux valgus was a varus deformity in the first metatarsal. The original Lapidus procedure was described as an incision centred over the lateral surface of the cuneiform and base of first metatarsal, followed by repositioning of the first metatarsal parallel to the second, and distal capsulorrhaphy at the first metatarsophalyngeal joint (MTPJ) to realign the great toe onto the metatarsal. A heavy chromic suture was then used to fix the first metatarsal to the second.[4] Lapidus summarised the progression and outcomes of this original procedure in a further publication in 1956.[5]

Papers in the 1990's and 2000's sought to establish the longer-term outcomes of the Lapidus procedure with evidence to suggest good patient satisfaction. However, studies often did not standardise the underlying indications and there was an absence of objective PROMs.[6] Different iterations of the procedure were published throughout to 2000's with Trnka et al (2005) describing their modified Lapidus arthrodesis and recognition of non-union as a potential complication.[7]

Outcomes of the Lapidus procedure compared to other corrective procedures for HV have been shown to be comparable. Faber et al (2013) compared clinical and radiological outcomes for patients undergoing either a Hohmann (distal metatarsal closing wedge) osteotomy or Lapidus procedure for HV correction and found no significant difference between the 2 groups. There was also no difference in outcomes for the subgroup clinically assessed as hypermobile.[8]

When focussing on the potential long-term complications after the Lapidus procedure, the most documented in the literature include non-union, malunion, recurrent HV, persistent midfoot pain and hallux varus.

Non-union

In a review of the evolution of the Lapidus procedure and techniques used for this, it was documented that non-union rates may be as high as 20%. However non-union did not appear to influence function. It was also noted that with improved fixation devices the rate of non-union could be improved.[9] Early weight bearing following a Lapidus procedure does not appear to increase the rate of non-union.[10] Therefore, with the progression to more stable fixation techniques, foot and ankle surgeons can more confidently weight bear their patients earlier with a lower concern for ill effect on outcomes from non-union.

Malunion

Malunion may occur secondary to iatrogenic shortening, elevation of the first metatarsal, a congenitally short first metatarsal, and malrotation.

There remains some debate in the literature as to how important iatrogenic shortening is on outcomes following Lapidus. One paper demonstrated a decrease in functional scores for 29 patients at 20 months follow-up if the metatarsal was shortened by more than 2.3mm.[11] A separate paper found an average shortening of 4.1mm following modified Lapidus procedures in 32 patients, but with no clinically significant metatarsalgia.[12] Good results, however, have been consistently demonstrated when first metatarsal shortening is minimised as much as possible.[13]

Dorsal metatarsal elevation may be addressed more successfully with a procedure such as the 'LapiCotton' whereby plantar flexion of the first metatarsal can be achieved using a block structural bone graft.[3]

Malrotation in HV has become increasingly topical amongst foot and ankle surgeons but remains a difficult deformity to quantify and treat. It can be a cause of recurrent HV or sesamoid pain following HV correction surgery. As previously discussed, the 'lateral round sign' (Figure 2) may be used as a measure of metatarsal rotation and is something that can be done in the operating room to quantify rotation corrected intra-operatively. With increased pronation the more rounded the lateral aspect of the metatarsal head appears.[14]
Pre-operatively, weight bearing CT imaging can be used as a reliable and reproducible way of assessing metatarsal rotation, though access to this imaging modality is often limited.[15]

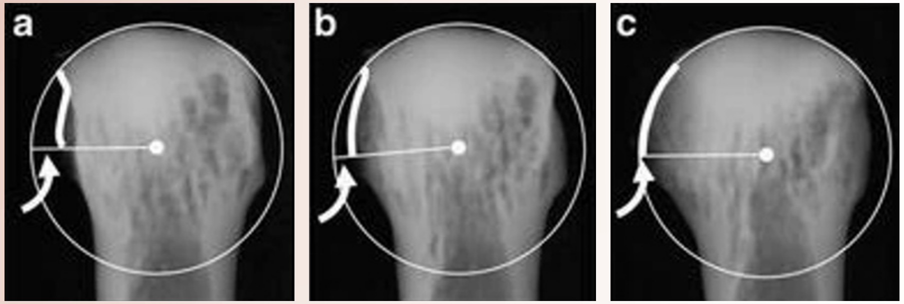

Figure 2

Recurrent HV

Recurrent HV is a recognised complication following a Lapidus procedure. The importance of good distal soft tissue realignment intra-operatively must be considered, as should associated deformities including hindfoot valgus and first metatarsal malrotation.

Hallux Varus

There is up to a 4% quoted rate of hallux varus following Lapidus for HV in the literature.[13] The cohort of patients assessed in this particular paper, however, had a relatively low pre-op HVA and IMA (32 and 16 degrees respectively), perhaps suggesting a Lapidus is too strong a corrective procedure for the less severe HV deformities.

Persistent midfoot pain

In patients with persistent midfoot pain following Lapidus correction two main possibilities should be considered; missed adjacent joint degeneration and excess inter-cuneiform laxity. The former can be mitigated with pre-operative 3D imaging in the form of MRI or CT scan to identify adjacent joint arthrosis. In cases of inter-cuneiform laxity there may be an argument for additional stabilization with the second ray.

Summary

Improved fixation techniques and better plating systems can reduce the non-union rate following a Lapidus procedure. In order to prevent post-operative malunion risk, shortening of the metatarsal should be minimised and consideration given to addressing dorsal elevation and malrotation of the first metatarsal. Proper distal re-alignment must not be overlooked and overcorrection into hallux varus can occur and should be avoided. It is worth considering potential adjacent midfoot joint degeneration and inter-cuneiform instability to reduce the risk of ongoing midfoot pain post-operatively.

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1.4. Role of metatarsal rotation

Shelain Patel

The role of metatarsal rotation in the development, progression and treatment of hallux valgus (HV) is becoming increasingly recognised.

Initial discussions about metatarsal rotation and its role in a number of foot and ankle pathologies are seen in the literature from the 1950's. However, the first main paper discussing first metatarsal rotation in the context of HV was published in 1972.[1] From the 1970's to the 1990's there was very little in the way of discussion on the topic within the literature, however between then and now publications on metatarsal rotation have increased exponentially.

A study group in Ireland discussed radiographic features that enable assessment of first metatarsal rotation in their 1993 paper. Using cadaveric samples plain radiographs were performed with varying amounts of rotation. They noted that with increasing pronation of the first metatarsal the inferior tuberosity at the base moved further lateral (Figure 1). Using these radiographic measurements for rotation, they also noted a relationship between increasing intermetatarsal angle (IMA) and the amount of pronation of the first metatarsal, though the causal effect of this association was unclear.[2]

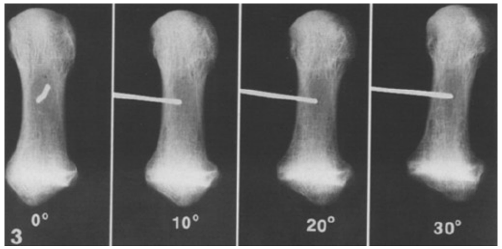

Figure 1

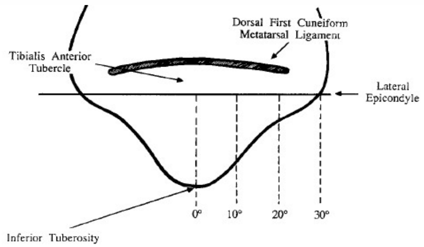

Figure 4 - Diagrammatic representation of the base of the metatarsal in the anteroposterior projection showing the inferior tuberosity position at 0˚, 10˚, 20˚, 30˚ of pronation (a model of pronation)

Further radiographic studies performed in patients on weight bearing radiographs have shown increased amounts of first metatarsal rotation in hallux valgus patients compared to normal control groups.[3]

More modern techniques in assessment of rotation with the use of weight bearing CT's have allowed further understanding of how best to evaluate metatarsal rotation in the context of HV. Initial research from the Stanmore group used weight bearing CT scans to define normal ranges for metatarsal pronation angles (MPAs) (-5 to 16 degrees) and alpha angles (-4 to 18 degrees) (Figure 2).[4] They then used these ranges to assess the prevalence of first metatarsal rotation in patients with HV. They found that there was abnormal rotation (based on MPA or alpha angle) in around a third of HV patients though there was only weak correlation between MPA, alpha angle and IMA. When looking at sesamoid rotation angle (SRA) however, there was a strong correlation with both IMA and hallux valgus angle (HVA).[5] It is hypothesized that the relationship between the sesamoids and metatarsal rotation is such that as the sesamoids migrate laterally, the force generated against the intersesamoid crista helps to drive metatarsal rotation. In more severe cases of HV we know that erosion of the crista occurs, meaning this rotational force is no longer being applied. Therefore, as the severity of HV increases (with increased HVA and IMA), the rotational deforming forces from the push of the sesamoids perhaps plateaus.

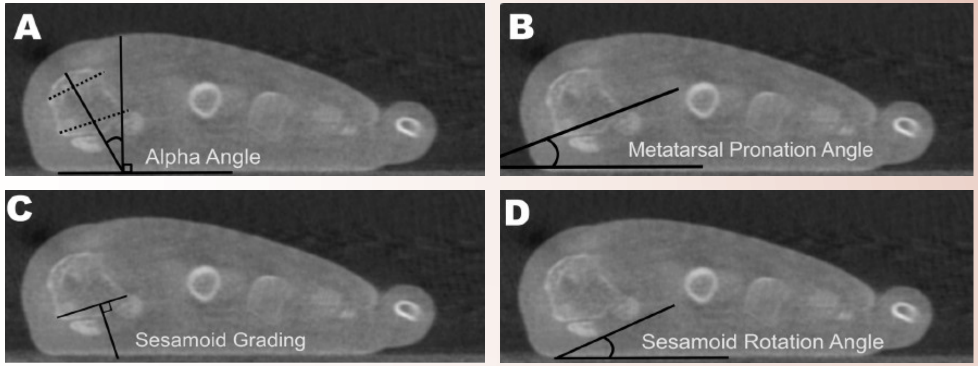

Figure 2

This is backed up by further research demonstrating that in cases of severe HV with a completely eroded crista, there was less pronation but a higher IMA when compared to those with normal anatomy. This again suggests the intersesamoid crista has a unique function in retaining the IMA.[6]

In papers assessing outcomes following HV correction, it has been shown that increased pre-operative pronation is associated with loss of correction and recurrence of deformity.[7] This may go some way as to explaining the relatively high quoted recurrence rates following corrective osteotomies where rotation has not been addressed.

In a series of 39 patients undergoing a Lapidus procedure for HV, those patients who had a degree of correction of rotation (as measured on pre- and post-operative weight bearing CT scans) had a significantly greater improvement in PROMS scores and a significantly lower rate of recurrence compared to those patients where there was no change to pronation.[8]

In the author's practice all patients with HV have a pre-operative weight bearing CT scan and metatarsal rotation is addressed intra-operatively if the MPA is higher than 16 degrees. It should be noted that this translates to a specific cohort of patients meaning only a small number of rotational osteotomies are performed. It is also noted that such osteotomies do confer a bigger operation with increased soft tissue dissection compared to a short Scarf osteotomy.

Overall, there is clear evidence to confirm the role of first metatarsal rotation in HV and outcomes following HV correction surgery. However, data remains limited on how best to correct metatarsal rotation and further research in this area would be of benefit.

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1.5. What are the causes of recurrence? *Jitendra Mangwani*

Recurrence of hallux valgus (HV) may be considered as clinical recurrence of symptoms and signs, radiological recurrence with changes to the hallux valgus angle (HVA), intermetatarsal angle (IMA), distal metatarsal articular angle (DMAA) and sesamoid position, or as biomechanical recurrence with transfer lesions or lesser toe deformities. Recurrence may or may not require further surgery.

When looking at radiological recurrence, studies have shown that an increased HVA or IMA following HV surgery may not be associated with recurrence of symptoms.[1,2] When assessing outcomes following procedures for HV, distinguishing between radiological recurrence with and without pain would be useful. It is also important to note the difference between cases of radiological recurrence whereby the deformity and radiological measurements progressively worsen following intervention, and those cases where there was incomplete deformity correction to accepted normal radiographic values at the time of surgery (often due to the severity of pre-operative deformity).

The cause of recurrent HV is usually multifactorial. Patient related factors such as preoperative anatomical predisposition, medical comorbidities and compliance with post-operative instructions all play a role. Surgical factors including the chosen procedure and technical competency of correction are also important.[3]

Skeletal immaturity

Historical figures for recurrence in juvenile or adolescent HV were as high as 50-60%. However, looking at more recent contemporary literature this figure appears closer to 8-10%, similar to the recurrence figures for adult HV.[4]

Skeletal immaturity

First ray hypermobility may occur in isolation or in the context or generalized hyperlaxity. In work by Faber et al (2004) patients with first tarsometatarsal joint (TMTJ) hypermobility were compared to patients without, following HV correction with either a Hohmann osteotomy or Lapidus procedure. There were no significant differences in clinical or radiological outcomes at 2 years noted for either group.[5] A prospective study documenting outcomes for treatment of HV with a proximal crescentic osteotomy and distal soft tissue repair noted that first ray mobility was routinely reduced to a normal level without the need for metatarsocuneiform joint fusion. They also noted that plantar gapping is not a reliable radiographic indication of sagittal plane hypermobility of the first ray.[6]

Further studies have shown no definitive effects on postoperative recurrence of HV due to generalized ligamentous laxity with comparable recurrence rates of 21.7% in those with laxity and 17.1% in those without (p=0.218).[7]

Anatomical predictors of recurrence

A large pre-operative HVA (over 37 degrees) and post-operative evidence of incomplete reduction of the sesamoids are strong predictors for recurrence of HV.[8,9]

A high DMAA is also a risk factor for HV recurrence with one paper suggesting a surgical correction goal of the DMAA to less than 11.3 degrees in order to reduce the recurrence risk following Scarf and Akin osteotomies.[10]

A systematic review and meta-analysis looking at data for approximately 3000 patients demonstrated that overall prevalence of HV recurrence was 24.86%. Pre-operative HVA and IMA showed a moderate positive relationship with recurrence, whilst post-operative HVA and sesamoid position showed a strong relationship with recurrence.[11]

Associated conditions

Thyroid dysfunction, in particular hypothyroidism, appears to be associated with forefoot pathology, especially HV and lesser toe deformities. The causal relationship and any effect on recurrence remains unclear but such correlations may offer an important opportunity in population health management, both in diagnosis and treatment.[12,13]

Rheumatoid arthritis (RA) is associated with HV and an increased risk of recurrence following treatment of HV, with a quoted range as broad as 4.5-60% for recurrence in the literature.[14,15] It has been shown that pre-operative HVA and first TMTJ angle are associated with an increased risk of post-operative radiological recurrence of HV in patients with RA.[16]

The role of pes planovalgus in HV is complex. It is unlikely that it is the initiating factor in development of HV but in the presence of pes planus the progression of HV is more rapid, especially in patients with RA, collagen deficiency disorders or neuromuscular disorders. The presence of pes planovalgus may predispose to the risk of recurrence following HV correction. Heyes et al (2020) showed an overall recurrence rate of 16% following a Scarf osteotomy for HV correction, with the pre-operative HVA and lateral talus-first metatarsal angles having a significant effect on the rate of recurrence.[17]

Data regarding complication and recurrence rates of HV deformity in patients with neuromuscular conditions is limited. When reported, isolated soft tissue procedures had extremely high rates of recurrence compared to other procedure types.[18]

Type of corrective procedure

A systematic review assessing outcomes following surgical treatment of HV demonstrated that the rate of recurrence was comparable across all surgery types, averaging 4.9%.[19] We need to be careful when reviewing the literature to confirm what measurement is being used to classify a recurrent HV. In a systematic review of longer term follow up it was noted that if using a HVA of over 15 degrees then recurrence rates following HV correction surgery would be quoted as high as 64%. If a HVA of over 25 degrees was used to classify recurrence, then rates drop to 5%. It is important to consider exactly which criteria is being used to define recurrence.[20]

When using rates of revision surgery as a marker for recurrence, a study comparing primary Chevron osteotomies, Lapidus procedures and closing base wedge osteotomies found no statistical difference in revision rates depending on method of correction used.[21]

A 15-year, single surgeon series showed that average time to recurrence after index surgery was 14 years. Whilst there was an association between an index surgery of a proximal osteotomy and quicker time to recurrence, there was no significant association between index surgery type and revision surgery type.[22]

Scarf osteotomy remains the most common procedure for surgical treatment of HV. A systematic review has shown that across 13 studies, 7 reported no recurrence following Scarf osteotomy, 6 studies reported recurrence rates of 3.6-11.3%, 1 study reported recurrence rates of 30% and 1 study reported recurrence rates at 78%.[23]

There is only one medium to long-term (over 60 months) outcome study assessing third generation minimally invasive (MIS) HV surgery utilising a validated clinical PROM score. This demonstrated a radiographic recurrence rate of 7.7%; comparable, but not superior to, open HV correction surgery.[24]

The use of intra-operative radiographs is supported with evidence to show that recurrence of HV can be reliably predicted from immediate post-operative non-weight-bearing radiographs, when assessing the HVA.[25]

Summary

Recurrence is a well-recognised complication of HV surgery however there is no consensus on a universal definition for this. High HVA, DMAA and negative lateral talus-first metatarsal angles correlate with HV recurrence. Both open and MIS techniques have a recurrence frequency with an average of approximately 5-10% between 5-10 years post-operative using a HVA of over 20 degrees as a threshold. To date, the published literature does not show any significant difference in the rate of recurrence depending on surgical procedure used at 5 years follow up.

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1.6. I prefer MIS techniques

Andrew Kelly

Until recently, the National Institute for Health and Care Excellence (NICE) guidance advised the use of minimally invasive surgery (MIS) in the foot ankle in the context of research alone. However, new guidance has been released in March 2024 on the use of MIS techniques in hallux valgus (HV) correction. NICE now recommend the use of MIS techniques as an option for HV correction with standard arrangements in place for clinical governance, consent, and audit. They also stipulate that such techniques should only be performed by a clinician with specific training and specialist experience, and that details of these procedures should be entered onto a registry.[1]

NICE based its recommendations on several papers, including meta-analyses, randomised controlled trials (RCT), cohort studies, and case series, with data on nearly 2000 patients. However, it should be noted that compared to open procedures, the number of described MIS techniques is limited (4 generations of MIS techniques compared to over 150 described open procedures). It should also be noted that 39 cohort studies and case series (with a total of 1245 patients) describing newer techniques were excluded by NICE in their review.

When reviewing MIS techniques, it is important to note the efficacy in terms of correction achieved, rates of recurrence, PROMS and patient satisfaction, but also the safety of procedures including complication rates, surgeon learning curve and radiation exposure intra-operatively.

Meta-analysis data shows that a number of papers have demonstrated statistically significant improvements in radiographic measurements from pre- to post-operative values following MIS correction of HV. When comparing MIS and open techniques there was similar radiographic correction.[1,2]

Recurrence rates are reported as being similar or lower to open operations (1-8%), though data is based on short term outcomes only with longer follow up required.1 Moving forward registry data will help ongoing assessment of outcomes following MIS surgery for HV.

Patient reported outcomes have been shown to significantly improve following MIS procedures for HV, with papers utilising AOFAS, MOxFQ, VAS pain, SF-36 and EuroQoL scores in their assessment of this. These improvements in post-operative outcome measures are comparable to those for open surgery but neither MIS or open techniques appear superior in this domain.[1] Unfortunately most RCT's comparing MIS and open techniques are underpowered.

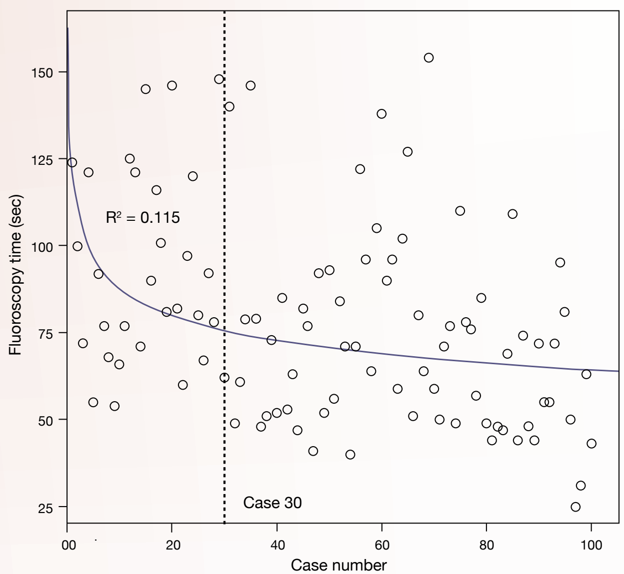
Interestingly one systematic review of 3 studies and 235 feet found a mean AOFAS score difference of 5 between pre- and post-operative assessments for both MIS and open techniques. Whilst there was no significant difference between the MIS and open groups, this mean difference of 5 points is under the 8 points required for a minimal clinically important difference (MCID) for AOFAS scoring.[2,3]

A meta-analysis reviewing a number of RCTs and cohort studies comparing MIS with Scarf or Chevron osteotomies for HV found early improvements in pain scores that were significantly better in the MIS surgery group. However, by 6 months there was no statistical significance in pain scores.[4]

It has been consistently demonstrated that radiation exposure is higher for MIS compared to open surgery. That being said, in a prospective observational study of intra-operative radiation exposure when performing MIS foot surgery (92% of cases being first ray surgery) the levels of radiation exposure recorded were lower than International Commission on Radiological Protection (ICRP) guidelines for occupational exposure and were comparable to those quoted in the literature for open surgical techniques.[5]

Several studies have looked at the learning curve for surgeons in performing MIS first ray surgery. When using operative time and radiation exposure as a measurement of this curve, it appears that this plateaus after around 30-40 cases (Figure 1).[6]

Figure 1



Complications commonly quoted as occurring in MIS first ray surgery include first metatarsal shortening, pseudo-bunion, dorsiflexion malunion and screw prominence. There is wide variation in the literature as to whether complication rates are significantly higher in either MIS or open surgery groups, and overall, it appears that rates are comparable.[1] As techniques develop, methods for dealing with MIS specific complications are also improving.

Summary

Overall, there is now good evidence to show that outcomes for MIS techniques are comparable to open techniques for first ray surgery. It appears that there is better early pain relief following MIS though this effect is comparable beyond 6 months. MIS techniques are rapidly advancing and ongoing research and collection of registry data will be useful to better understand longer term outcomes.

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1.7. I prefer open surgery *Simon Chambers*

As we've seen in the previous discussions there is a wealth of literature looking at open procedures for the treatment of hallux valgus (HV). Whilst some of the literature is varied in terms of descriptions of techniques, outcomes and recurrence rates, open procedures for HV remain widely used across the foot and ankle community and are therefore reliable procedures in the hands of most foot and ankle surgeons. They are safe procedures with a relatively small complication profile, and whilst variable recurrence rates are quoted in the literature, the number of these cases that are symptomatic remain low. Open procedures lend themselves better to teaching trainees on the basis of exposure, but also the learning curve, which is much steeper for minimally invasive surgery (MIS). Whilst studies have shown equivalent outcomes for MIS techniques, these certainly haven't been proven to be superior to open procedures. It is therefore the author's opinion that it is not necessary to move away from doing safe and reliable open surgery for HV, and towards minimally invasive surgery with a steep learning curve and no clear superior benefit to tried and tested techniques.

Consensus Questions

1. There is a postcode lottery in the provision of bunion surgery within the UK.
   a. Yes 22 **(100%)**
   b. No 0
2. The burden of bunions, including the impact on society and quality of life, is not fully appreciated by funding bodies within the UK.
   a. Yes 22 **(100%)**
   b. No 0
3. Clinical assessment of the tendo Achilles should be performed routinely for all patients presenting with hallux valgus.
   a. Yes 22 **(100%)**
   b. No 0
4. Hypermobility of the 1st TMTJ and generalised hyperlaxity should be routinely assessed for all patients presenting with hallux valgus.
   a. Yes 22 **(100%)**
   b. No 0
5. For hallux valgus all plain radiographs performed should be standing.
   a. Yes 22 **(100%)**
   b. No 0
6. Weight bearing CT imaging could provide clinically useful additional information that cannot be obtained through other imaging modalities.
   a. Yes 21 (95.5%)
   b. No 1 (4.5%)
7. PROMS show improvement after bunion surgery.
   a. Yes 22 **(100%)**
   b. No 0
8. Clinical recurrence of hallux valgus is defined as the return of preoperative symptoms i.e. pain or problems with footwear impacting daily function.
   a. Yes 19 (86.4%)
   b. No 3 (13.6%)
9. Intra-operative imaging should be available when performing bunion surgery
   a. Yes 20 (91%)
   b. No 2 (9%)
10. In the unit I work in, I have access to intra-operative imaging
    a. Yes 22 **(100%)**
    b. No 0
11. Rigid immobilisation in a plaster cast is not required following 1st metatarsal osteotomies for bunion surgery.
    a. Yes 22 **(100%)**
    b. No 0
12. Single dose intra-venous antibiotics should be used at the time of anaesthetic induction for bunion surgery.
    a. Yes 22 **(100%)**
    b. No 0
13. Following Lapidus surgery I routinely ensure a period of rigid immobilisation in a plaster cast for
    a. None 10 (45.5%)
    b. 2 weeks 1 (4.5%)
    c. 4 weeks 1 (4.5%)
    d. 6 weeks 10 (45.5%)

Session 2: Stress Fractures

Chaired by Adam Lomax

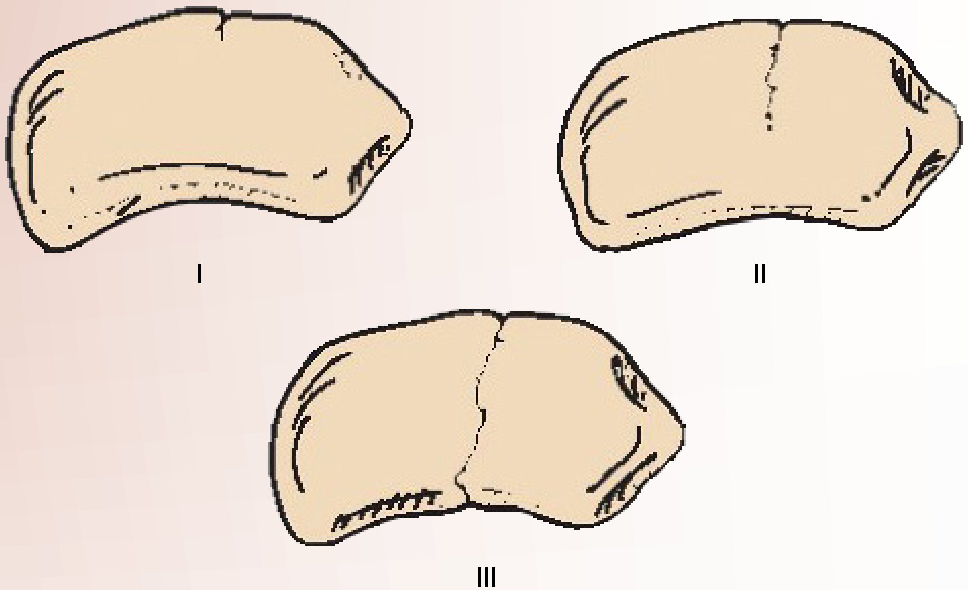
2.1. Navicular Stress Fractures

Stefan Rammelt

Navicular stress fractures are rare within the general population. However, they comprise 35% of bone stress injuries in athletes[1] such as distance runners, gymnasts and professional dancers. Navicular stress fractures are considered “high risk stress fractures” as they have the tendency to heal slower or go on to non-union[2].

Pathogenesis

Due to repetitive, sub-traumatic overload where there is inadequate recovery time or inadequate regeneration. Radiological signs on MRI initially will include bone marrow oedema and microstructural trabecular lesions, which will lead on to macrostructural failure and fracture if either inadequate recovery time or regeneration takes place[2].



Saxena classification for navicular stress fractures

Risk Factors

- Female athlete triad (eating disorder, amenorrhea, osteoporosis)
- Low bone mineral density
- Vitamin D deficiency
- Hindfoot valgus
- High arched foot (navicular is at apex of the medial longitudinal arch)
- Gastrocnemius tightness (increases pressure on lever-arm of the forefoot)
- Limb length discrepancy
- Coalitions (increased loading of midfoot due to stiff hindfoot)
- Sudden increase in activity
- Hard training surfaces
- Change in footwear
- Potential anatomical predilection (force enters navicular through the talonavicular joint before being distributed across the cuneiforms. Force dissipation therefore occurs at the navicular)
- Potential poor vascular supply (16% have avascular zone reaching dorsal cortex)[3]
- Os supranaviculare[4,5]

Clinical Evaluation

Patients typically present with vague pain of insidious onset.
This typically worsens with activity and improves with rest. On examination, there is diffuse tenderness over the affected bone, sometimes with a visible swelling or palpable callus if a late presentation. It is important to check for contributing factors (gastrocnemius tightness, hindfoot valgus, low vitamin D/high gonadotrophin/low oestrogen)[2].

Imaging

- Plain radiographs are first line, but it takes 2-3 weeks to visualise fracture (sensitivity 18%)
- MRI shows early bone oedema
- Bone scan (sensitivity 100%, poor specificity)
- CT helps for deciding treatment and monitoring healing (sensitivity 100%)[2]

Differential Diagnoses

- Accessory bones (os naviculare/os tibiale externum, os supranaviculare)
- Bipartite navicular
- Müller Weiss disease (adults) / Köhler’s disease (paediatric)
- Post-traumatic deformity/non-union

Treatment

- **Conservative:** protected weight bearing, cessation of sports
- **Operative:** open reduction internal fixation +/- (vascularised) bone grafting
- **Adjuncts:** vitamin D + calcium substitution, extracorporeal shockwave therapy (ESWT), low intensity pulsed ultrasound (LIPUS)

Successful outcomes are achieved 72.0% of the time non-operatively, compared to 96.3% with operative management [Odds Ratio = 5.5][6]. In adolescent athletes, non-operative treatment was successful in 85% of cases[7]. Adolescents requiring operative treatment was more likely to be needed in older adolescents, a higher body mass index, and with high grade stress fractures.

Return to sport following bone stress injuries (BSI) at any site in the body, was longest in the navicular and talus (127 days; 95% CI [102 – 151 days])[8].
The shortest return to sport was in BSI of the posteromedial tibial shaft (44 days; 95% CI [27 – 61 days]). Over 90% of athletes successfully return to sport.

Summary

Navicular stress fractures often present with non-specific symptoms that results in a delayed diagnosis. They are a high risk stress fracture with regards to non-union. A trial of non-operative treatment is indicated in all adolescent and recreational athletes. Operative treatment has higher success rates in adult athletes. Treatment should be guided by risk factors. Treatment adjuncts should be used as they cause no harm.

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2.2. Metatarsal Stress Fractures

Phil Vaughan

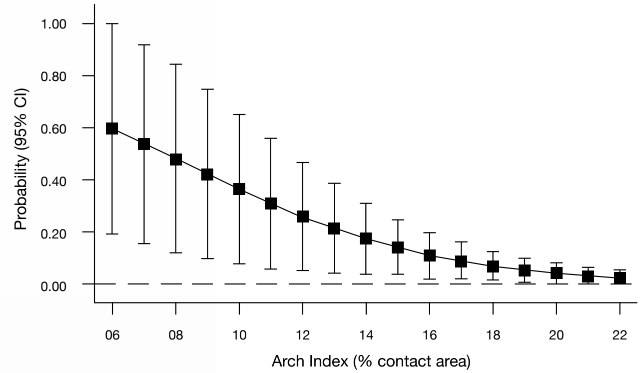
40% of stress fractures in the foot are in the metatarsals, but the distribution is not equal across all the metatarsals.

Risk Factors

- Extrinsic:
  - Sport/activity
  - Shoes
  - Training regimen (surface, distance, duration)
- Intrinsic:
  - Athlete (age, female, bone mineral density, BMI, genetics, female athlete triad, PMHx, steroids)
  - Foot (shape, metatarsal strength)

Risk Factors

More common in the cavus foot, due to a smaller contact area between the ground and foot, thereby causing increased pressure and likelihood of fracture (see figure). This is most apparent with a varus hindfoot that results in overload of the lateral border of the foot.



Metatarsal Variation

Structure

Lesser metatarsals are half the cross-sectional diameter of the 1st metatarsal. 2nd metatarsal is the longest and thinnest. The distribution of cortical thickness throughout individual metatarsals also varies along the length of the metatarsals[1], which influences location of the stress fractures.

Load

Load through the metatarsals is affected by the rigidity of the 1st - 3rd TMTJs. During push off the dorsoplantar (compressive) load is primarily at the 2nd metatarsal. When changing direction the mediolateral (tensile) load is primarily at the 5th metatarsal[2]. Plantar muscle fatigue and decreased 1st ray function (e.g. hallux valgus) can also increase the load going through the 2nd metatarsal.

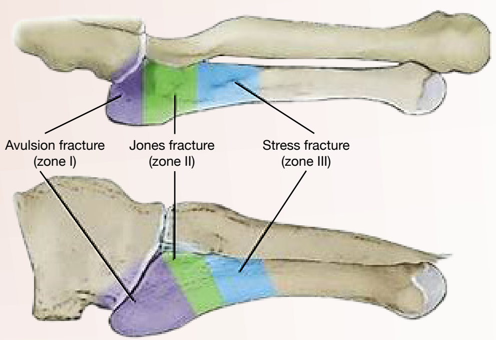
Relative bending strength/rigidity

When these factors are combined, it explains why the 2nd and 5th metatarsals are most susceptible to stress fractures, with the 2nd metatarsal having the lowest bending strength[3].

Blood supply

There are watershed areas of blood supply in the metatarsals, particularly at the 5th metatarsal base, which can be difficult to heal should a fracture occur at this area.

	2nd Metatarsal	5th Metatarsal
Direction of weakness	Dorsoplantar	Mediolateral
Area of overload	**Forefoot** • Metaphyseal fractures (tip toe sports, ballet) • Shaft fractures (runners military recruits)	**Lateral column** • Plantar flexion-inversion injury (basketball, football, tennis) • More common in varus hindfoot, metatarsus adductus, high 4-5 IMA
Non-union risk	***Low*** (shaft = compression; metaphyseal = watershed area therefore higher risk)	***High*** (stress fracture zone 3, tension/torsion force, watershed area)
Management	Predominantly ***conservative***	***Torg classification***



Torg Classification of Proximal Fifth Metatarsal Fractures	
Type 1	Narrow fracture line with sharp margins and no widening, minimal cortical hypertrophy, and no intramedullary sclerosis
Type 2	Wide fracture line with adjacent lucency that involves both cortices and demonstrates partial obliteration of the medullary canal by sclerosis at the site of fracture
Type 3	Wide fracture line with new periosteal bone formation and complete obliteration of medullary canal by sclerosis at the site of fracture

Investigations

- Vitamin D levels
- Bone profile
- Endocrine screen
- Bone mineral density

Management

Conservative

- Modify risk factors
  - Extrinsic: Shoes, terrain, training regimen, time off for recovery.
  - Intrinsic: Vitamin D3 800 – 1000 IU + Calcium 2000mg[4], Endocrine/Gynaecology review
- Boot/Cast/both
- Weight bearing status
- Length of restriction
  - Until callus formation

(Boot/Cast/both; Weight bearing status; Length of restriction; Until callus formation — No evidence)

Surgical

- Indicated for high risk of nonunion
  - 5th metatarsal (mostly)
  - Metaphyseal fracture
  - Regression/sclerosis on serial XRs
- Screw or plate weight bearing status
  - Screw has better bending and torsional rigidity[5]
  - Screw diameter >4.5mm[6]
- Bone graft
  - Particularly in re-fracture or large gap[7]

Adjuncts

- Bisphosphonates
  - Suppression of bone turnover
  - No risk reduction in future[8]
- Parathyroid hormone
  - Increase macrophages and therefore callus formation[9]
  - Non-FDA approved
- Low intensity pulsed ultrasound
  - No difference in outcome[10,11]
- Shockwave
  - Similar union rates to surgery[12]

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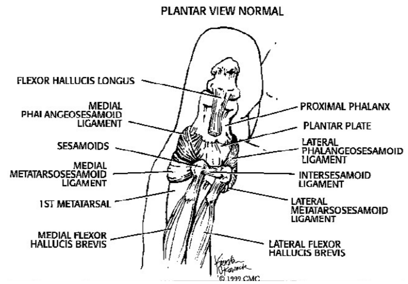
2.3. Sesamoid Fractures

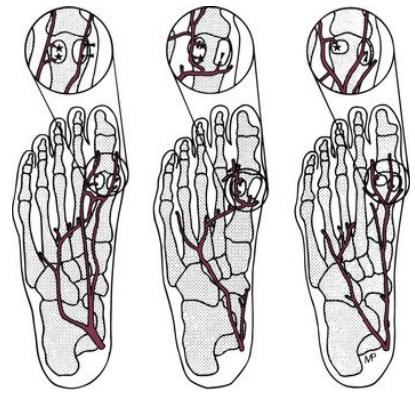
David Garras

Sesamoids act to augment muscle pull, diminish friction and modify pressure over a joint. At the 1st MTPJ they absorb weight across the 1st ray, protect FHL and increase the mechanical advantage of the intrinsic muscles.

Anatomy

Sesamoids have a complex suspensory mechanism that makes up part of the plantar stabilising structures. They are contained within the FHB and articulate with the 1st metatarsal head. The blood supply enters plantar and proximal, with minor supply distally. There are three normal variants as seen in the figure.





Sesamoid Injuries/Conditions

- Fracture
- Turf toe (disruption of FHB and plantar complex distal to sesamoids)
- AVN
- Bipartite sesamoid
  - Incidence 19 – 31% (80% tibial, 25 – 90% bilateral)
- Osteochondritis dissecans
- Degenerative joint disease
- Intractable plantar keratosis (IPK)

Fractures

The mechanism for sesamoid fractures is typically due to any of a fall onto forefoot, crush injury, hyperextension of the MTPJ or a chronic injury (ballet, runners).

Treatment

Conservative	Surgical
Cast/boot immobilisation • Toe spica extension • Plantar flexion Orthotics • Rigid plate • Pressure relief Taping regimens Bone stimulation	Bone grafting Fixation Fragment excision and restore mechanism Sesamoid excision and reconstruction

Approaches

A medial approach is standard practice for the tibial sesamoid. However, there are different approaches described for the fibular sesamoid including medial[1], dorsolateral[2] and plantar[3].

Surgical treatment

Fragment excision

Indicated for fractures involving <25%. Outcomes are poor, with residual pain and poor articulation of the remaining fragment within the trochlear[4].

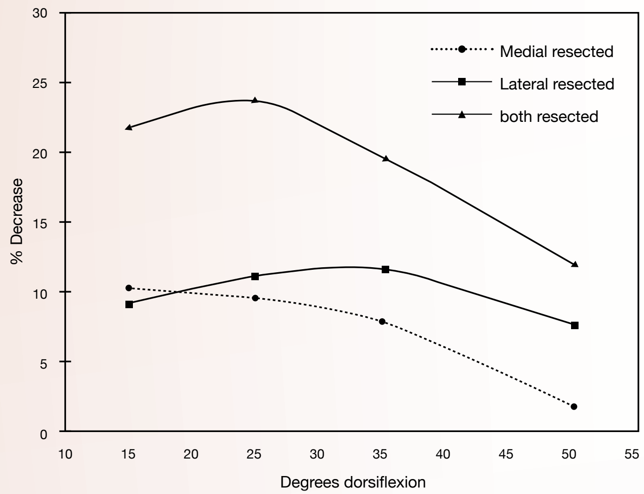
Sesamoidectomy

FHB push-off strength reduces following sesamoidectomy (tibial – 10%, fibula – 16%, both – 30%)[5].

The best outcomes for sesamoidectomy are where the FHB tendon complex is reconstructed[6].

- 20 out of 37 sesamoidectomies with excellent or good outcomes
- Direct repair of defect with overlying FHB and periosteum

Reduction in propulsive force due to sesamoid resection



Bone Grafting

Indicated for tibial hallux sesamoid stress fractures/non-unions, impact sports, chronic pain. Bone graft from metatarsal head can be used. Union is achieved in 90% after 12 weeks on average[7].

Complication Management

- Ongoing pain/adjacent sesamoid pain
  - Always excise sesamoids in stages
  - Separate stages by 1 year minimum if possible
  - Repair soft tissues well and protect post-operatively
- Progressive hallux valgus (tibial sesamoidectomy)
  - Consider realignment during primary procedure if needed
  - Release adductors if there is underlying hallux valgus
- Progressive hallux varus (fibula sesamoidectomy)
  - EHL transfer +/- IPJ fusion[8]
  - 1st MTPJ fusion
- Transfer IPK
  - Use orthotics
  - Plantar shaving if persistent pain
- Cock-up deformity
  - FHL tendon transfer +/- IPJ fusion[9]

Summary

Protect acute injuries from diastasis. Most sesamoid fractures do well with a period of immobilisation followed by orthotic management. For surgical treatment, aim to preserve sesamoids when possible. If sesamoidectomy is needed, robust soft tissue reconstruction is essential.

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2.4. Perimalleolar Stress Fractures

George Smith

The focussed history and investigation for perimalleolar stress fractures is consistent with the previous topics.

Posterior malleolus

There has only been one case report detailing a stress fracture of the posterior malleolus[1]. This occurred in a patient with rheumatoid arthritis on biologics and steroids. The patient healed after a brief period of immobilisation.

Lateral malleolus

Stress fractures of the lateral malleolus appear to be related to repetitive plantar flexor muscle contraction causing medial to lateral motion of the fibula or impingement of the talus on the distal fibula[2,3]. Bending forces are not well tolerated in cortical fibular bone[4]. Running on hard surfaces is also a possible etiological factor[4].

Treatment
Rest, immobilisation and cessation of activities for 4 - 7 weeks has been shown to be highly successful in resolving symptoms with no re-fractures, delayed union or non-union[5,6].

Limb malalignment
In neutral ankle alignment, the fibula undergoes approximately 6.4% of weight transmission[7]. Varus or valgus malalignment may play a role as a risk factor for lateral malleolar stress fractures through either a tensile force (varus) or direct bending moment on the distal fibula (pes planovalgus with sub-fibula impingement). There is one case report of a fibula stress fracture with a background of stage 4 pes planovalgus[8]. The patient failed conservative management in a weight bearing boot and subsequently received a tibiotalocalcaneal fusion.

Medial malleolus

Demographics and Incidence
0.6 – 4% of all lower limb stress fractures[9,10]

Almost exclusively in high demand athletic people[9,10]

Risk factors
Extrinsic: Training errors, poor/change in footwear, sudden increase in activity, weight gain.

Intrinsic: Varus malalignment, anteromedial spurs/impingement.

Any malalignment that shifts the mechanical axis towards the midline has been shown to be common in medial malleolar stress fractures (MMSF)[11–13]. Examples include increased external hip rotation, genu varum, forefoot varus and hindfoot varus. While the evidence is not conclusive, it is fair to consider varus malalignment a risk factor.

Anteromedial spurs are associated with medial malleolar stress fractures[14] and are thought to arise due to impingement at terminal ankle dorsiflexion, causing rotational shear forces through the medial malleolus. However, these spurs are also common in athletes[15,16] and many are asymptomatic[17].

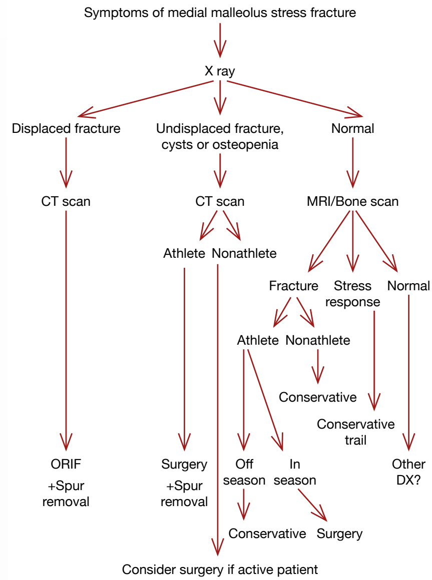
Diagnosis
Weight bearing X-rays will reveal a fracture line in 30% of MMSFs[18] and these are typically in patients with symptoms for 4 – 6 weeks. MRI is 100% sensitive[19].

Treatment
Treatment depends upon (20):
1. The presence of a fracture line, cyst or local osteopenia on the radiographs.
2. Displacement of the fracture.
3. Level of sports or athletic participation
4. Timing of the injury (in-season vs off-season)

However, there is a lack of good evidence to support either conservative or surgical management. Case reports and case series are therefore relied upon to shape practice. The aforementioned factors may also help inform management[20].

Outcomes
Operative management has been shown to shorten time to union, ranging from 2 weeks to 2.5 months compared to conservative management, but this comes with surgical risks[21,22]. There is no agreed protocol for conservative management with regards to weight bearing or duration of treatment. Faster return to sport by 2.5 weeks has been demonstrated for operative management[22]. Across multiple studies totalling 69 patients[11,21–24], only two went on to non-union, one surgically managed[21] and one conservatively managed[24].

Patients with anteromedial spurs that underwent fixation and arthroscopic debridement of the spur all went on to union and return to sport[13,14].



Unknowns of Medial Malleolar Stress Fractures

- Is surgery of benefit and for which cases?
- Are anteromedial spurs a cause of effect of MMSFs?
- If lower limb malalignment is present, should we be more aggressive with surgical treatment?
- Should we correct the malalignment?
- If treating conservatively, what should the protocol be?
- Is there a role for adjunctive therapy?

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Consensus Questions

1. Routine surgical management of 5th MT stress fractures requires bone graft
   a. Yes 0
   b. No 23 **(100%)**
2. In patients with stress fractures in the F&A, consideration should routinely be given to vitamin D supplementation
   a. Yes 23 **(100%)**
   b. No 0
3. In patients with stress fractures in the F&A, consideration should routinely be given to bone mineral density assessment
   a. Yes 1 (4.3%)
   b. No 22 (95.7%)
4. Vitamin D therapy should be recommended in all cases of stress fractures
   a. Yes 18 (78.3%)
   b. No 5 (21.7%)
5. For high-risk stress fractures, including navicular and 5th MT base stress fractures, consideration could be given to earlier surgical intervention
   a. Yes 23 **(100%)**
   b. No 0
6. Non-operative treatment for complete (type 3) navicular stress fractures is appropriate as 1st line treatment
   a. Always 0
   b. Sometimes 23 **(100%)**
   c. Never 0
7. Low intensity pulsed ultrasound could be useful in the treatment of stress fractures in the F&A
   a. Always 0
   b. Sometimes 15 (68.2%)
   c. Never 7 (31.8%)
8. For diagnosis of suspected navicular stress fractures … would be a standard investigation
   a. Xray 23 **(100%)**
   b. MRI 22 (95.7%)
   c. Bone scan 0
   d. CT 8 (34.8%)

9. When surgically treating sesamoid pathologies, proper and adequate soft tissue repair/reconstruction is vital
   a. Yes 23 **(100%)**
   b. No 2 (9%)

10. For routine assessment of sesamoid pathology I would include…
    a. XR (sesamoid views) 23 **(100%)**
    b. Bone scan 0
    c. MRI 2 (8.7%)
    d. CT 6 (26.1%)
    e. SPECT 3 (13.0%)
    f. USS 0
    g. Theatre fluoroscopy 0

11. Assessment of deformity should always form part of evaluation in perimalleolar stress fractures
    a. Yes 23 **(100%)**
    b. No 0

Session 3: Gastrocnemius tightness

Chaired by Lyndon Mason

3.1. Methods of assessment *Rod Hammett*

Gastrocnemius tightness can be defined as ankle dorsiflexion of less than 5 degrees during knee extension.[1] It is implicated in many pathologies, often with unclear causation, but for this review we will be discussing gastrocnemius tightness in the context of patients without underlying neurological conditions. Objective and consistent assessment can be difficult, therefore meaning it is also a difficult problem to study.

Methods of assessment for gastrocnemius tightness may include simple clinical tests, clinical tests with devices and imaging modalities.

Clinical assessment

Silfverskiold test is commonly used in clinical practice for assessment of gastrocnemius tightness. A positive test is equinus that resolves when the knee is flexed. A minimum difference between ankle dorsiflexion with knee flexion and knee extension has been quoted as 13 degrees though this figure differs within the literature.

Outcomes of this assessment are technique dependent. The original technique described the initial correction of valgus to lock the hindfoot, locking of the Chopart joint, application of 2kg of force specifically under the second metatarsal head, and passive movement at the knee to prevent recruitment of anterior compartment extensors and a false positive result.[2-4] It is therefore not surprising that it has been shown to be an unreliable test with low inter and intra-observer repeatability. Despite this, it is still widely used.[5]

Standing assessments, such as the lunge test, have been shown to be more reliable. Assessment of ankle dorsiflexion in a weight bearing position with the knee extended can be performed. Measurements may be taken with an inclinometer or goniometer. The potential issues with this examination are that it may not be possible to perform in patients with significant pain or who cannot tolerate weight bearing. The force applied is also not controlled.[6,7]

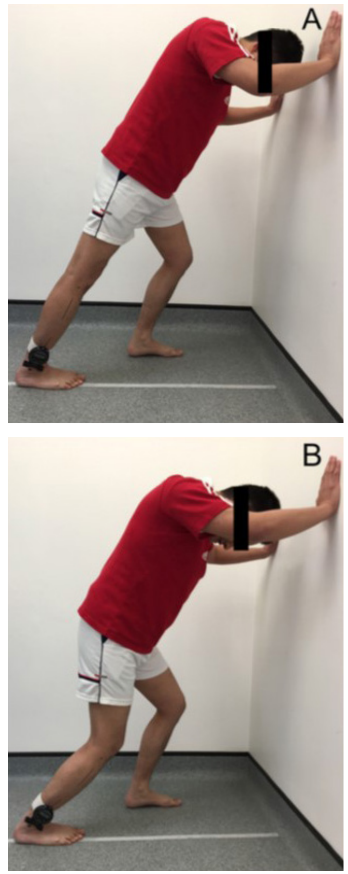


Figure 1

The weight bearing lunge test has been shown to be reproducible with good inter and intra-observer reliability. In population based studies of healthy ankles it was shown that an ankle dorsiflexion index of over 13 degrees with the knee extended lies outside of 2 standard deviations and can therefore be considered abnormal.[8,9]

Maestro also described the Taloche test in which, when the foot of a patient with gastrocnemius tightness is placed on an inclined plane, they are unable to maintain their balance (Figure 2).[4]

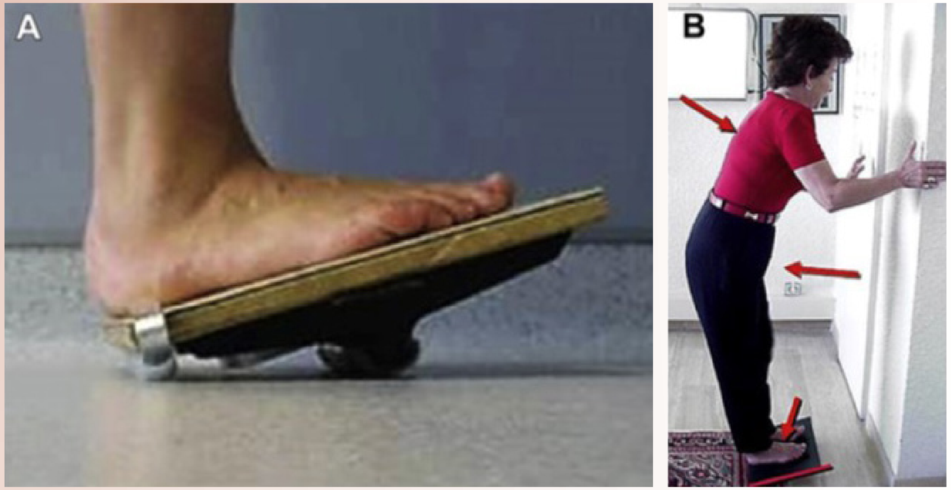


Figure 2

Clinical tests with devices

Goniometers and electro-goniometers have typically been the clinical standard used to measure range of movement, or contractures, around a joint. However, research has shown poor intra-observer reliability and an accuracy within only a 5-6 degree range, even in the most experienced hands. There is also no ability to account for the force applied. This, in particular, makes their use in research limited.[10]

Numerous other measurement devices have been used in an attempt to assess and quantify gastrocnemius tightness, including a torque range of motion device and three-dimensional tracking. The suggested benefits of measurement devices are that they can fix the foot position, control the applied force, may be easier to use and are more reproducible than clinical examination assessments. However, the evidence for these is comprised of a highly heterogenous group of studies, the outcomes of which may not be applicable across the usual patient populations. There is also a cost implication to consider.[5,11-14]

Imaging tests

The use of imaging tests for gastrocnemius tightness tends to be limited to the research laboratory setting, in particular when looking at results of interventions for contractures. Examples include ultrasound tape techniques to measure the length of the muscle belly, 3D ultrasound and ultrasound combined with magnetic or optical tracking. MRI studies with measurements may also be used.

There are detectable and distinct effects of gastrocnemius tightness on gait kinematics and therefore kinematic assessment may be useful. However, it has been shown that the majority of this effect is occurring at the hip and knee joint with increased compensatory flexion at both of these. The main effects of gastrocnemius tightness on kinematics have been shown to occur in the late stance phase of the gait cycle (i.e. when the knee is extended).[15,16]

The increased plantar pressures with gastrocnemius tightness have been well documented with a peak in forefoot pressure on measurements shown to occur in late stance. These increases in plantar pressure have been shown to be similar in cases of both triceps surae and isolated gastrocnemius tightness.[17]

Mechanography can be used to assess ground reaction forces and has been used in the context of research to assess plantar flexion strength pre and post gastrocnemius release.[18]

Summary

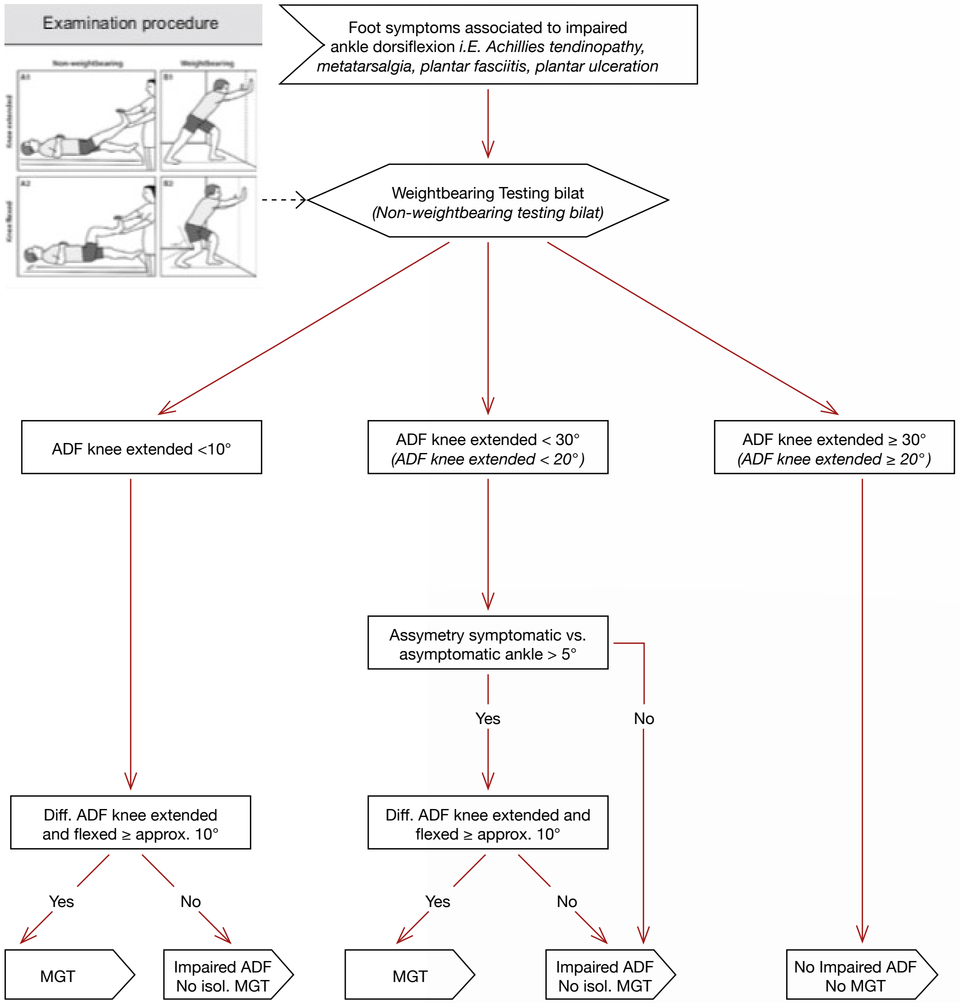
The best method of assessment of gastrocnemius tightness in the research setting is probably a device such as an equinometer with a controlled force plate and ability to digitally measure dorsiflexion. This will differ to the best method of assessment in the outpatient clinic.

One of the main problems is that multiple studies have documented on gastrocnemius tightness, however there is a wide variation in what is classified as tight. This depends on the magnitude of loss of movement, force applied, anatomical landmarks and measurement devices used.

Whilst Silfverskiöld test is still commonly used, and relied upon in clinic studies, it has been shown to be unreliable and unreproducible, particularly in subtle or isolated gastrocnemius contractures. This most likely limits its use in research.[19] In clinical practice a consistently performed Silfverskiöld test by the same observer is probably acceptable, though other clinical tests, such as the weight bearing lunge test, are more reliable and reproducible.

Baumbach et al (2016) published a decision pathway to aid in assessment and diagnosis of gastrocnemius tightness that may be useful in both clinic and research settings. Their algorithm provides a consistent definition of impaired ankle dorsiflexion and gastrocnemius tightness to guide treatment strategies and study their effectiveness (Figure 3).[20]

Figure 3



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3.2. Conservative treatment

Lyndon Mason

The difficulty with attributing cause and effect in gastrocnemius tightness given its association with a wide variety of foot and ankle conditions is well recognised.

The anatomy of the gastrocnemius muscle has been well documented. Cadaveric studies have shown that the tendoachilles rotates as it approaches its insertion on the calcaneus, leaving the medial head as the most posterior/superficial structure (Figure 1). This in part explains why medial head of gastrocnemius release works well as a procedure.[1] Whilst the medial head forms the most superficial/posterior portion of the Achilles tendon, it has also been shown to be the most active portion of the gastrosoleus complex.[2] The Achilles tendon becomes continuous with the plantar fascia via the periosteum of the calcaneum, meaning that their pathologies are closely related.[3]

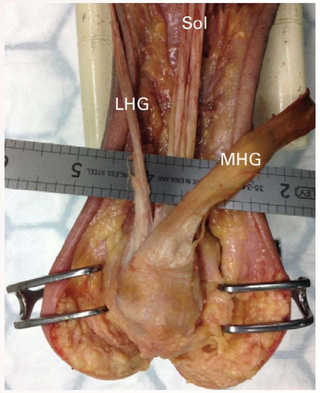

Figure 1

Aetiology of heel pain

A 'terrible triad' for increased risk of developing heel pain has been well documented.

1. There is a linear correlation between calf tightness and VAS scores for heel pain, with ankle dorsiflexion of less than 0 degrees increasing the risk of developing heel pain by 23-fold.[4]
2. With a BMI of over 30 there is a 2.9 times increased risk in developing heel pain. Large amounts of force go through the Achilles tendon and plantar fascia during gait; on walking, a force of 3.9 x body weight goes through the Achilles tendon and 1.8 x body weight through the plantar fascia. On running this increase to 7.7 x body weight and 3.7 x body weight respectively. There is an association between hyperlipidaemia and Achilles tendon disorders, interestingly with a more pronounced increased risk in underweight individuals with higher LDL cholesterol levels than overweight individuals overall.[5]
3. Standing jobs where patients spend a prolonged portion of their day weight bearing also increases the risk of developing heel pain by 3.6.

Sleep position has also been implicated in calf tightness. 20% of people sleep in the prone position which typically rests the ankle in equinus, increasing the risk of developing heel pain. Sleep studies have also shown that with increased age the period of time of postural immobility also increases. This perhaps, in part, explains the higher rates of Achilles tendinopathy in older age groups, along with the pathophysiological changes occurring in the ageing tendon.

The pathological stages of tendinopathy are well documented. It was originally thought that interventions with conservative treatment could only be successful if implemented at the earliest reactive stage of tendinopathy (occurring within minutes to days), however ongoing evidence suggests that there is the potential for good outcomes in terms of pain improvement if treatment is targeted during the dysrepair stage of tendinopathy.[6]

There is also a demonstrated association between depression, anxiety and stress, and heel pain. Cotchett et al (2016) showed that for every 1 unit increase in a DASS (depression, anxiety and stress) scale, the odds ratio for plantar heel pain increased by 1.3.[7]

Treatment in plantar fasciopathy

Without treatment patients with plantar fasciopathy can go on to develop long term pain and symptoms. Evidence has shown that after 15 years, 44% of these patients have ongoing heel pain if left untreated.[8]

First line treatment should consist of weight loss, reduction of calf tightness and activity modification. Plantar fascia specific stretches as well as tendoachilles stretching exercises should be implemented, with 90% of patients demonstrating improvements in pain at 2 years following treatment with this alone.[9]
Whilst plantar fascia stretching exercises have been shown to result in the most significant improvement in patient reported outcome scores, the addition of other conservative treatment modalities such as analgesics and heat & silicon heel pads, can improve this further.[10]

Orthotics also have a role in conservative treatment of plantar fasciopathy. Customized insoles have been shown to improve pain, foot function and ultrasound findings in plantar fasciitis.[11]

Figure 2

There is evidence to show the use of night splints (Figure 2) can offer a relatively good and quick effect on heel pain symptoms,[12] however rigid night splints tend to be poorly tolerated. The author proposes the use of a specially designed pillow (Figure 3) that can be placed under the ankle joint to reduce the amount of equinus occurring when sleeping in the prone position.

Figure 3

In summary non-surgical recommendations for plantar fasciopathy and calf tightness include weight loss, physiotherapy with targeted stretching exercises, and combined orthotics and night splinting. The evidence for most other non-operative treatments, such as PRP injections, Botox and shock wave therapy is limited but the placebo effect should not be underestimated and may be used to satisfy patients.

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3.3. Surgical treatment

Lyndon Mason

The anatomical site of gastrocnemius release can be divided into proximal tendon (Silfverskiöld procedure), gastrocnemius muscle belly (Baumann procedure), at the insertion of gastrocnemius aponeurosis into soleus fascia (procedures include Strayer, Vulpius and Baker) and at the Achilles tendon (procedures include Hoke, White and Paley) (Figure 1).

There is a fairly widespread practice when it comes to operative gastrocnemius release and choice of procedure tends to be based on surgeon experience and preference.

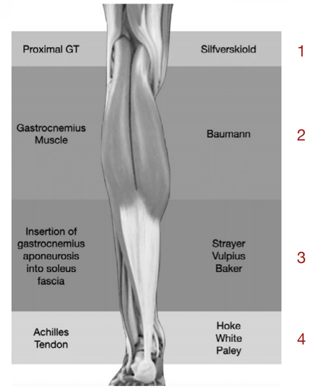

Figure 1

Surgery in Plantar Fasciopathy

There are a number of studies looking at outcomes following gastrocnemius release for patients with plantar fasciopathy. There is a variety within the literature in which anatomical zone the release is performed. There are consistently significant improvements in patient recorded outcome measures in the short to midterm follow up across these studies.[1-3]

In particular a recent large retrospective case series (Slullitel et al. 2024) demonstrated a significant improvement in both VAS and FAAM scores at 3 years post-operative, with a 1.3% complication rate.[4]

An RCT looking at outcomes for patients with chronic plantar fasciitis following proximal medial gastrocnemius recession also showed significant improvement in AOFAS, VAS and MOxFQ scores at 6 year follow up.[5]

A meta-analysis study has shown that surgical interventions are effective in providing short to midterm symptomatic improvement for plantar fasciitis refractory to non-operative treatment, though current evidence is equivocal regarding choice of operative treatment.[6]

Factors associated with poor outcomes following isolated gastrocnemius recession for heel pain include tobacco use, high BMI and prior foot and ankle surgeries, highlighting the importance of also addressing modifiable risk factors.[7]

Overall gastrocnemius release is a recommended procedure to patients with plantar fasciopathy and heel pain refractory to non-operative treatment. Plantar fasciotomy should only be performed with caution given the demonstrated risk of developing lateral column pain following this procedure.[3]

Surgery for non-insertional Achilles tendinopathy

Evidence for outcomes following gastrocnemius release in non-insertional tendinopathy mostly consists of level II-IV evidence. Systematic reviews looking at this evidence show that there is a significant improvement in patient reported outcomes (including VAS, FFI, VISA-A and AOFAS scores) across studies for those patients undergoing gastrocnemius release, though there is a 9.45% rate of documented complications. The complications seen tended to depend on the zone of surgical release, with reduced rates of nerve related complications seen in Zone 1 & 2 releases.[8,9]

The lack of high-level evidence for gastrocnemius release in non-insertional Achilles tendinopathy may be due to the fact that the majority of these cases settle with conservative treatment. However, the available evidence suggests that there are improvements in outcomes with surgery and that gastrocnemius lengthening is reported as the safest and as offering the highest improvement.

Surgery for forefoot overload

For those patients with symptoms relating to forefoot overload, gastrocnemius recession has been shown to increase heel contact time, increase ankle dorsiflexion and shift the gait line medially on gait analysis. There is also documented improvements in patient reported outcome measures following this.[10]

There is concern regarding long term problems with calf weakness following operative gastrocnemius release, with reduction in power of the triceps surae documented in a number of small studies. One paper showed that despite an overall statistically significant reduction in leg strength following surgery for gastrocnemius release (Strayer procedure), there was no significant difference in force reduction between the operated and non-operated leg. This perhaps suggests that any weakness is secondary to the patient having undergone foot & ankle surgery, rather than secondary to the muscle release itself.[11]

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3.4. Gastrocnemius lengthening as an adjunct procedure

Nijil Vasukutty

Gastrocnemius tightness is implicated in a number of foot and ankle pathologies. It is well known that deformity of the hindfoot will change the line of pull of the Achilles tendon; for example, in hindfoot valgus the line of pull moves laterally, whilst in varus it moves medially (Figure 1). A tight gastrocnemius can change the overall biomechanics of the foot and ankle. In the sagittal plane limited dorsiflexion of the ankle causes the talus to become more plantarflexed. In the transverse plane the metatarsals become more abducted and in the coronal plane the calcaneus will tend to evert. It is well documented in kinematic studies that with loss of ankle dorsiflexion there is increased pressure on the metatarsal head during gait.[1]

Figure 1

Studies have also shown that there is an increased incidence of gastrocnemius tightness in patients with foot pain[2] and Barouk has described the use of isolated proximal medial gastrocnemius release for treatment of metatarsalgia.[3] Therefore release of a tight gastrocnemius can be a useful adjunct to other boney and soft tissue procedures in a number of foot and ankle conditions and deformities.

Gastrocnemius release in Total Ankle Replacement (TAR)

There are a number of studies assessing the effects of gastrocnemius or tendoachilles lengthening (TAL) following TAR. Jeng et al demonstrated increased tibiotalar dorsiflexion on radiographs in patients who had a TAL or gastrocnemius recession following TAR. However, in the TAL group this was also associated with loss of plantarflexion, suggesting that gastrocnemius recession may be the better choice of procedure for TAR patients.[4]

DeOrio et al utilised digital photographs to assess maximal passive dorsiflexion following gastrocnemius recession in 29 patients undergoing TAR.
They demonstrated that gastrocnemius recession resulted in a significant increase in ankle dorsiflexion following TAR which was seen irrespective of whether patients had a positive Silfverskiöld test on intra-operative assessment.[5]

In a study looking at gait mechanics following TAL or gastrocnemius recession as an adjunct procedure in TAR, peak dorsiflexion angles and ankle range of motion showed a significantly greater improvement at 1 year post-op in those that underwent a lengthening procedure, however overall outcomes between both groups were equivalent.[6]

Gastrocnemius release in flatfoot correction

The close relationship between gastrocnemius function and flatfoot deformities is well known, as with hindfoot valgus the line of pull of gastrocnemius shifts laterally and ultimately leads to shortening of the gastro-soleus complex.

A case series of 24 patients undergoing combined FDL transfer, medial displacement calcaneal osteotomy, lateral column lengthening and gastrocnemius recession for stage II posterior tibial tendon insufficiency, had a significant improvement in functional outcomes without any concerns of plantarflexion weakness.[7]

A systematic review evaluating available clinical and radiographic evidence for incorporation of gastrocnemius recession or TAL into surgical correction of adult acquired flatfoot deformity found that there was no high-level evidence to support clinical outcomes. Support for lengthening procedures in flatfoot reconstruction remains largely based on expert opinions and case series and its effect is difficult to distinguish from the clinical contribution of associated corrective procedures.[8]

Gastrocnemius release in diabetic foot disease

Evidence from gait analysis and pedography studies clearly demonstrates increased forefoot pressures with a tight gastrocnemius, with the potential to precipitate forefoot ulcers in diabetic patients. Tiruveedhula et al suggested a 2-stage treatment protocol for forefoot ulcers in diabetic patients. This consisted of an outpatient TAL and subsequent proximal metatarsal osteotomy if ulcers persisted or recurred. In a cohort of 96 feet they found that TAL followed by a walking cast for 6 weeks was effective at healing forefoot ulcers in 96% of cases.[9] The International Working Group for Diabetic Foot (IWGDF) advise TAL procedures as part of surgical treatment for forefoot ulcers.

Small studies have also demonstrated good results for isolated gastrosoleus recession for diabetic midfoot ulcers.[10]

TAL is also commonly performed for midfoot Charcot with the aim of restoring calcaneal pitch, reducing plantar midfoot pressures and preventing further midfoot collapse. This has been shown to slow, and in some cases recede, early stages of midfoot Charcot neuropathy.[11]

Gastrocnemius release in trauma

There are a number of review articles and expert opinions discussing TAL in trauma. It is hypothesized that a tight gastrocnemius may predispose to certain injuries due to increased plantarflexion and altered loading through the midfoot. It is also hypothesized that a tight gastrocnemius may increase the risk of failure of fixation or joint arthrosis following trauma, however there is no high-level evidence assessing this.[12]

Gastrocnemius release in amputations

There is some evidence to show that performing a TAL at the time of a transmetatarsal amputation reduces the risk of requiring repeat procedures.[13]

Summary

There is limited high level evidence for the use of gastrocnemius lengthening procedures as an adjunct in the surgical treatment of other foot and ankle problems, however it remains a commonly performed procedure. There is case series evidence for its use in ankle arthroplasty and diabetic foot conditions and some level IV biomechanical evidence for its use in the treatment of forefoot conditions and transmetatarsal amputations. Good evidence for its use in flatfoot correction is lacking, thought potentially due to the difficulties in distinguishing improvements in outcomes from associated clinical procedures.
Evidence for gastrocnemius lengthening in trauma remains very limited.

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3.5. Is gastrocnemius release just modern day bloodletting?

Dishan Singh

Historically medical conditions were considered in terms of the 'four humours': blood (air), yellow bile (fire), black bile (earth) and phlegm (water). Bloodletting was a common medical treatment during these times and decision to use this treatment was based on very little in the way of objective measurements. Doctors would 'feel it in their waters', arguably in the same way modern foot and ankle surgeons evaluate for gastrocnemius tightness given the paucity of standardised assessment tools for this. Similar to bloodletting gastrocnemius release may be considered a safe and simple procedure, though we know there are complications associated with this. There are numerous case series published on the outcomes of gastrocnemius release but the majority are in the context of self-limiting conditions. The scientific reasoning behind gastrocnemius release remains unclear.

The original assessment tool for gastrocnemius contracture was described by Nils Silfverskiöld in the early 1900's. Silfverskiöld was an orthopaedic surgeon, Swedish aristocrat, bon vivant, and Olympic gymnast who spent a lot of his practice treating patients with Cerebral Palsy.[1] He published a case series of his clinical test and an operation for treating spastic conditions by 'reduction of two-joints muscles of the leg to one-joint muscles' through release of both gastrocnemius heads at their insertion on the distal femur and reinsertion on the tibia.[2] This however ignores the contribution of gastrocnemius to the subtalar joint, and the fact that it should really be considered a 'three-joint muscle'.

It should be noted that the original description of Silfverskiöld's test was not based on the range of movement achieved at the ankle but states that the gastrocnemius contracture should be 'measured by the strength that is needed to produce passive dorsal flexion of the foot with the knee bent or stretched respectively', though this was never quantified and is described based on testing in patients with increased tone.[1,2]

In the 1970's procedures for gastrocnemius tightness became further popularised following the publication of work from Seattle quoting equinus as 'the greatest symptom producer in the foot'.[3] This thinking was taken on into the 1990's by surgeons in Seattle, including the renowned Ted Hansen, who advocated the need for gastrocnemius release in patients with forefoot symptoms, in addition to surgical correction of forefoot pathology including hallux valgus and pes planovalgus. By this time the iteration of Silfverskiölds test being utilised had moved on from the strength required to produce passive dorsiflexion to the range of movement noted with the knee extended versus the knee flexed.
Further publications from this Seattle group assessed these differences in

range of passive dorsiflexion comparing patients with foot pathology and a control group without. DiGiovanni's paper showed an association between foot pathology and loss of ankle dorsiflexion range of movement but with no clear cause and effect and did not give a clear-cut answer as how to define gastrocnemius tightness.[4] Following these publications the gastrocnemius recession became a popular orthopaedic procedure, not only in the USA but also on the European continent. Based on a symposium held in France in 2006, P and LS Barouk's work on gastrocnemius tightness was published as a textbook in 2012, in which they have labelled it as a cause for multiple pathologies, including forefoot and midfoot overload, inferior heel pain, Achilles tendinopathy and back pain. However, the cause and effect remained unclear.[5]

In 2015, DiGiovanni published a literature review titled 'Gastrocnemius recession for foot and ankle conditions in adults: Evidence-based recommendations', in which it was concluded that the procedure was being performed by foot and ankle surgeons with increasingly enthusiastic support for a number of foot and ankle conditions, however with little scientific support behind its application with the majority of evidence level III and IV studies.[6]

The first population based observational study assessing gastrocnemius was published by the Royal National Orthopaedic Hospital group in 2019. Assessing 800 normal limbs in 400 patients, it was found that assessment using goniometer or equinometer measurements were unreliable, though the lunge test had better inter and intra-observer reliability. The main concerns with this test, however, are the fact that the amount of force and subtalar joint movement cannot be controlled. That being said it is a weight bearing test and is therefore perhaps most true to the physiological role of gastrocnemius.[7] In this same study the difference in ankle dorsiflexion with knee extension and knee flexion was plotted for the same participants with no foot and ankle symptoms. The mean difference was 6 degrees though with a normal difference being anywhere from 0 to 13 degrees.[7]

In a further study, comparisons were made between a control group and patients with foot and ankle pathology. In those patients with foot and ankle symptoms there was a significant increase in the difference in ankle dorsiflexion with the knee extended and the knee flexed (utilising the normal range of 0-13 degrees +/- 2 standard deviations as per the first study). When these patients were broken down into those with forefoot versus mid or hindfoot pathology the difference was only significant for those in the forefoot pathology group. Again, this shows a clear association between gastrocnemius tightness and foot and ankle pathology, with 1/3 of patients with forefoot pathology having gastrocnemius tightness. However, there remained 2/3 of patients with forefoot pathology who did not have gastrocnemius tightness and it remains unclear as to the cause and effect relationship.[8]

Kowalski is often quoted as a way of explaining the link between gastrocnemius tightness and forefoot overload in gait. A book published by the Belgian surgeon describes walking pedobarograms through the use of pictorial graphs but unfortunately without data to quantify this. Graphs demonstrating forefoot overload are also confusing with the suggestion that forefoot overload continues to occur during the swing phase of gait.[9]

In fact, further dynamic studies demonstrate that compensatory knee flexion in patients with gastrocnemius tightness with no clear evidence to suggest forefoot overloading.[10]

In a gait analysis study of patients with gastrocnemius tightness it was noted that there is little difference in the ankle dorsiflexion throughout the gait cycle compared to a control group. There is, however, increased knee flexion throughout the stance phase in those with gastrocnemius tightness (Figure 1).[11]

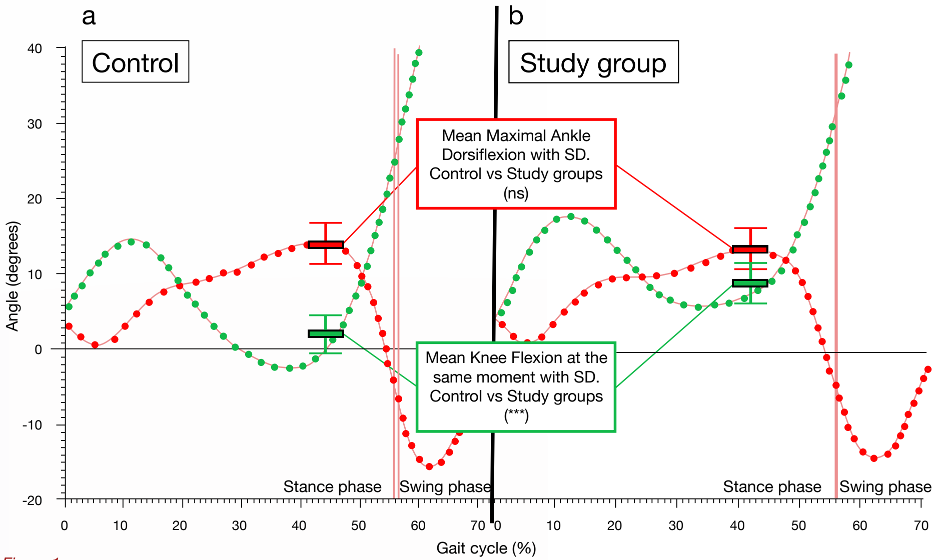

Figure 1

In conclusion, there is clear evidence that there is an association between gastrocnemius tightness and foot and ankle pathology, however the direction of the cause-and-effect relationship remains unclear. Whilst it is often assumed that foot and ankle pathology is a result of gastrocnemius tightness, it may be considered that this tightness occurs secondary to foot pain and symptoms. We no longer treat patients with bloodletting and need to consider that the gastrocnemius release may really act as a placebo. Is it time for a trial of sham surgery?

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Consensus Questions

1. Do you perform a gastrocnemius release in your regular practice?
   a. Yes 24 **(100%)**
   b. No 0
2. In your routine practice in patients with a clinically tight gastrocnemius would you consider a lengthening procedure for **heel pain**?
   a. Yes 23 (96%)
   b. No 1 (4%)
3. In your routine practice in patients with a clinically tight gastrocnemius would you consider a lengthening procedure in **forefoot procedures**?
   a. Yes 16 (67%)
   b. No 8 (33%)
4. In your routine practice in patients with a clinically tight gastrocnemius would you consider a lengthening procedure in midfoot procedures?
   a. Yes 23 (96%)
   b. No 1 (1%)
5. In your routine practice in patients with a clinically tight gastrocnemius would you consider a lengthening procedure in ankle procedures?
   a. Yes 24 **(100%)**
   b. No 0

6. What technique should be used for clinical assessment of gastrocnemius tightness in normal practice?
   a. Silfverskiold test 24 **(100%)**
   b. Lunge test 0
   c. Taloche test 0
   d. Goniometer 0
7. Which of this do you include in your routine conservative treatment protocol for gastrocnemius tightness?
   a. Physiotherapy 24 **(100%)**
   b. Orthotics 22 (92%)
   c. Night positioning device 12 (50%)
8. At which sites do you routinely perform gastrocnemius release/lengthening?
   a. Zone 1 8 (33%)
   b. Zone 2 5 (21%)
   c. Zone 3 16 (67%)
   d. Zone 4 5 (21%)
9. When performing gastrocnemius release procedures in theatre what is your routine patient positioning?
   a. Supine 14 (58%)
   b. Prone 2 (9%)
   c. Other 8 (33%)
10. If doing a solitary gastrocnemius recession do you routinely give VTE prophylaxis post-operatively?
    a. None 8 (42%)
    b. 2 weeks 5 (26%)
    c. 4-6 weeks 6 (32%)
11. What weight bearing protocols do you follow after a solitary gastrocnemius release?
    a. Immediate weight bearing 24 **(100%)**
    b. Non-weight bearing 0
12. What immobilisation do you use following a solitary gastrocnemius release?
    a. Plaster 0
    b. Boot 12 (50%)
    c. Night splint 2 (8%)
    d. None 10 (42%)

Session 4: Talar Osteochondral Lesions

Chaired by Anand Pillai

4.1. What do we know about pathogenesis and natural history?

James Ritchie

Aetiology

Trauma appears to be the primary cause for osteochondral lesions of the talus (OLTs), with 93% of lateral OLTs and 62% of medial OLTs associated with trauma[1]. Atraumatic causes include:

- Malalignment of lower limb/morphology of the ankle
- Instability
- Avascular necrosis
- Vitamin D deficiency
- Endocrine disorders
- Iatrogenic (steroids, radiation, chemotherapy)
- Genetic predisposition

Ankles are at risk of OLTs due to being a highly congruent joint with subsequently thin cartilage over the talar dome (approx. 1.11mm in women and 1.35mm in men). Any decrease in joint congruity will leave the ankle vulnerable to OLTs. Talar blood supply may also increase the risk of OLTs.

Pathogenesis

<u>Trauma</u>

OLTs can occur as a result of either a single event or repetitive microtrauma, where the talar dome impacts on the fibula (lateral) or tibia (medial)[2] causing cartilage bruising, softening or delamination with or without bone bruising, fissuring or fracturing.

Once the trauma has occurred, the damage can either heal or progress. During progression cartilage loses its glycosaminoglycan (GAG) side chains and proteoglycans, thereby becoming less hydrophilic. This causes the water within cartilage to escape, with pressure loading forcing it into fissures, resulting in cysts.

Pain from these lesions is thought to occur due to increased pressure in subchondral bone or cysts as pain fibres are present in osteons.

<u>Malalignment</u>

With malalignment there will be extra-articular changes in the loading of the ankle, with potentially a change in fibula length which would affect the congruency at the ankle and leave it susceptible to OLTs as previously mentioned.

Ankle morphology has been shown to play a part in the development of OLTs[3]. Medial OLTs are significantly more common in patients with:

- a medially deviated talus
- a talus that is flatter in the sagittal plane
- a talus that is wider at the front

The above parameters have also been identified with increased risk of lateral OLTs following trauma, albeit with a laterally deviated talus rather than medially[4].

The medial malleolus appears to be smaller in those with medial OLTs[5] and the deep deltoid ligament attachment tends to be broader and more proximal[6]. In fact, the fibres of the deltoid ligament were attached to the OLT in 76.7% of cases. An association with loss of the medial longitudinal arch was identified, which led to the hypothesis that increased traction on the medial side of the foot/ankle contributes to medial OLTs[6].

The increase in focal stress from malalignment has been hypothesised to cause an adaptive response by cartilage and/or subchondral bone as per Wolff's law[7], which explains how cartilage can sometimes be intact over an OLT. Another theory explains this finding through osseous hypertension, leading to decreased vascularisation and poor cell metabolism[8].

These theories beg the question of whether alignment should be corrected to prevent OLTs. Correcting malalignment in patients with shoulder OLTs via osteotomy, has been shown to significantly improve pain and reduce lesion size, albeit not always to complete resolution of the lesion[9].

<u>Ankle Instability</u>

Ankle instability may also play a role in pathogenesis. Patients with medial OLTs and lateral ligament instability have been found to present at an earlier stage of the disease with smaller lesions[10], suggesting that instability causes OLTs to become symptomatic earlier. In addition, a meta-analysis of 402 isolated syndesmotic injuries found OLTs in 22-24% of patients[11], suggesting an association.

<u>Other Pathogenesis Mechanisms</u>

In animal models, a failure of enchondral ossification causes necrotic islands in subchondral bone (osteochondrosis latens), which can also be caused by vascular insult. This can either heal or progress if there is biomechanical trauma to the lesion[12]. Overall, pathogenesis may be vascular, mechanical, genetic or a combination[13].

<u>Is there a spectrum of disorder?</u>

Considering the evidence, it may be that there is a spectrum regarding the causation of OLTs. At one end there are normal joints overwhelmed by abnormal activity/trauma, and at the other end normal activity is sufficient to cause failure due to either, abnormal joint mechanics creating excessive stress on the joint or a preexisting biological failure that weakens the joint. Anecdotally, it would seem that medial OLTs occur at the biomechanical end of the spectrum, and lateral OLTs at the traumatic end.

Natural History

Paediatrics

So called "stable" lesions usually do well with conservative management, with 58 – 100% achieving good results[14,15]. However, there is no consensus on what conservative management is; treatments include activity reduction, immobilisation, and/or simple analgesia. Younger age seems to be the most important factor in achieving a good outcome. The evidence suggests that while many patients OLTs heal up fully, others have persistent radiological changes that are asymptomatic[14,15].

It is rare for OLTs in children to remain symptomatic into adulthood[16] and even rarer to progress to arthritis (6.7%)[17]. This has been shown in studies with long term follow up (>21 years).

Adults

Systematic review of OLTs in adults has shown good outcomes in 59% of patients treated with activity restriction, and 41% treated with immobilisation[18]. However, this review comprised 14 small studies of low-level evidence.

Progression of OLTs in adults has been shown to be fairly slow, with the majority of lesions remaining stable over the medium term and not progressing to OA[19]. Even advanced lesions progress slowly, with evidence showing that 54% of patients have good or excellent clinical function[20]. Approximately 65% will show mild degenerative changes at 7 years follow up, with none showing severe degeneration. There is therefore no correlation with clinical outcome.

Summary

- Pathogenesis:
  - Trauma, malalignment and ankle morphology are important factors
  - Physeal and/or vascular failure may be involved but the mechanisms are not fully understood
  - Genetics may play a role
- Natural history:
  - Many OLTs will heal without treatment
  - Others will become asymptomatic
  - Relatively few progress to OA
  - Many OLTs are found incidentally and it is estimated that up to 66% are asymptomatic[21]

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4.2. What to look for in assessment and investigations

Anand Pillai

Assessment

The majority of patients presenting with osteochondral lesions of the talus (OLTs) are between 20 – 40 years old. They typically have vague and nonspecific symptoms with no clear physical examination signs.

History

- Residual pain after ankle sprain or other ankle trauma
- Sprain that doesn't respond to first line treatments (RICE, simple analgesia, physiotherapy)
- Exercise-related pain
- Local tenderness
- Minor limitations in range of motion of ankle and/or subtalar joint
- Deep, intermittent pain in the ankle with weight bearing
- Mechanical symptoms (clicking, locking, catching)
- Occasional swelling and joint instability
- Non-specific ankle pain (which may not correspond to the location of the lesion)

The most common complaint is exercise-related pain followed by mechanical symptoms[1]. OLTs are often undetected, therefore a high index of suspicion is required.

Examination

- Areas of tenderness
- Limitations in range of motion
- Hindfoot alignment
- Ankle stability tests
  - Anterior drawer
  - Talar tilt
- Compare to other side

Investigations

- X-rays
  - Often lack diagnostic accuracy
  - Include a mortice view with 4cm heel rise as a significant number of OLTs will be found more posteriorly[1]
- MRI
- CT/Weight bearing CT/SPECT
- Arthroscopic evaluation

Accuracy

Routine radiological examination misses up to 50% of OLTs[2]. When compared with history taking, examination and standard x-rays, adding a mortice view with a 4cm heel raise will double to diagnostic odds ratio[3]. MRI has the highest sensitivity (0.96) of any investigation, while CT is more specific (0.99)[3].
When used in combination, history + examination + XRs + CT or MRI are both very effective in correctly diagnosing OLTs[3].

While MRI is very good at identifying OLTs and assessing abnormalities of the cartilage, the true size of the lesion can often be obscured by concomitant bone marrow oedema, leading one to think the lesion is larger than it is[4].
CT, however, is accurate in sizing lesions and assessing the bony profile.
The overlying cartilage cannot be assessed unless an intra-articular contrast CT is performed. CTs also have the added benefit of usually being more widely available with a shorter acquisition time compared to MRI[5]. Weight bearing CT is starting to become available to clinicians. It has the added benefits of allowing one to assess the alignment and loading of the ankle, which helps with planning approaches if osteotomies are required. A proportion of OLTs have talar and tibial lesions, WBCT demonstrates relationship between these.

Classifications

There are many classifications available for OLTs, dependant on which imaging modality you are using[6–10]. Below is an overview of the different classifications taken from Lan et al, 2021[11].

Observations

T. Lan, H.S. McCarthy, C.H. Hulme et al. — *Journal of Arthroscopy and Joint Surgery 8 (2021) 231–237*

Table 1
Overview of the classification systems used across the different assessment modalities used in the diagnosis of OLTs. OC = osteochondral fragment; SCB = subchondral bone.

X-Ray	MRI	CT	Arthroscopy	
Berndt and Harty[13]	Hepple[34]	Ferkel[36]	ICRS[37]	Cheng-Ferkel[38]
I – subchondral compression	1 - articular damage only	I - cystic lesion, intact overlying articular cartilage	1 – superficial zone softening or fissure	A – smooth and intact cartilage, but soft
II – partially detached OC fragments	2a – Articular cartilage damage with subchondral fracture (- odema)	IIA – cystic lesion with articular surface communication	2 – lesions extend <50% depth	B – rough articular surface
III – fully detached OC fragments	2b – Articular cartilage damage with subchondral fracture (+odema)	IIB – overlying non-displaced OC fragment	3 – lesions extend >50% depth but not into SCB	C – fissures and fibrillations present
IV – displaced OC fragments	3 – detached non-displaced OC fragment	III – non-displaced OC fragment with lucency	4 – lesion extends into the SCB	D – cartilage flap or exposed SCB
	4 – detached and displaced OC fragment	IV – displaced OC fragment		E – loose, non-displaced fragments
	5 – subchondral cysts			F – displaced fragment

- Medial OLTs are generally larger and deeper, while lateral OLTs are smaller and shallower[11].
- Lateral OLTs tend to be more symptomatic, possibly due to higher baseline contact pressures in the lateral compared to medial ankle[11].
- 98% of lateral OLTs are associated with trauma[12].
- The rate of coexisting talar and tibial lesions is up to 35%[13].
- 37% of tibial shaft fractures have a concomitant occult OLT at 12 months[14].
- There is a strong correlation between symptomatic knee and ankle osteochondral defects and vitamin D deficiency (up to 90%)[15].
- Bone bruises seen in post-traumatic ligament tears are potential precursors of OLTs[16].

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4.3. Non-operative Management and Outcomes

Simon Clint

Management

The literature on non-operative management for OLTs is sparse and primarily retrospective case series’ covering a range of treatments.

Published non-operative management options include:

- Benign neglect
- Activity modification
- Weight bearing restrictions
- Guided hyaluronate injection
- PRP injection
- Prolotherapy
- Shockwave therapy

Outcomes

There is a single systematic review looking at non-operative management for OLTs[1]. This paper pooled 868 patients from 30 papers, had a median follow up of 37 months (3 – 288) and covered a wide range of treatments. Indications were recorded in 40%. A combined success rate of 45% from 372 patients was found, with a conversion to surgery rate of 46% from 400 patients. Progression of OA on x-rays in 9%, on CT in 11%, and on MRI in 12%.

Benign Neglect

A case series followed up 35 chronic Berndt and Hardy stage 5 OLTs over an average 3 years post-diagnosis and 7 years post-onset[2]. Patients were simply observed at follow up, 54% had “good” or “excellent” outcomes, with 17% increasing and 5.7% decreasing in size. In patients with complete clinical recovery, CT showed 75% had an unchanged lesion.

A further paper looked at 50 OLTs originally referred for pain and/or stability[3]. They found that 70% of patients reported their pain had decreased or fully resolved at an average 52 month follow up (27 – 124). Repeat MRIs showed no substantial progression of OLTs. Further follow up of the same cohort at 11 – 20 years post-presentation, found only 2 had undergone further treatment. 92@ had AOFAS over 80, with a median of 94. The medial VAS was 0. 73% showed no deterioration on x-rays.

Activity Modification

Two papers looked at activity modification in isolation for OLTs. Neither mention the indications for treatment. The earlier paper[4] did not use VAS or any objective measures of pain, but it did record an average post-treatment AOFAS of 68.2. The later paper[5] did note an improvement in VAS from 3.8 to 0.9, but did not use any outcome measures. The conversion rates to surgery between the papers were very contrasting at 61% and 11% respectively. Across the papers, the lesion remained unchanged radiologically in 83% deteriorated in 11% but with no arthritic changes.

Weight Bearing Restrictions

4 papers with a total of 51 patients receiving anything from 3 – 8 weeks in cast, found varying results. One paper had a 55% success rate, while another had a conversion to surgery rate of 63%. Only one paper included VAS as a pain score, with an improvement of 9 to 5.4.

Hyaluronate Injection

A case series looked at 30 patients receiving 3 hyaluronate injections over 1 month[6]. At 12 week follow up, significant improvement in AOFAS was seen from 52 to 98, indicating a "trend" towards improved range of motion and function. However, most patients still reported pain at final follow up.

PRP vs Hyaluronate

An RCT looking at PRP vs hyaluronate in patient with previously failed non-surgical management (of unknown duration), found that the ankle hindfoot scale (AHFS) significantly improved in both groups at 28 weeks follow up, with a significant difference in favour of PRP for the AHFS, stiffness and function as measured by VAS[7].

PRP vs Prolotherapy

A retrospective cohort study with 12 month follow up compared PRP with prolotherapy. Patients received 3 injections over 9 weeks. The VAS and AOFAS significantly improved in both groups but not between groups, therefore no benefit of PRP over prolotherapy was found[8].

Shockwave Therapy

A single case series combined 11 ankles and 29 knees with osteochondritis dissecans (OCD). Patients received one treatment under GA and were followed up for 12 months. No indications or post-treatment regime was detailed. Pain during activities of daily living (ADLs) improved from 72% to 27% at 12 months, but this was not differentiated by joint.

Summary

- Approximately 50% improve without surgery.
- If conservative treatment works, the long-term outcomes seem good.
- There is a lack of evidence to support one treatment over another.

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4.4. Debridement and Grafting for OLTs *David Garras*

Operative Considerations

When deciding upon surgical management there are several aspects that need to be considered to maximise outcomes:

Mechanical Factors	Patient Factors
Is the OLT isolated? Stage of the OLT Size of the OLT Uncorrected malalignment Ankle instability "Kissing lesions" of the tibia and talus	Chronic steroid use Smoking status BMI Previous infection at operative site Presence of inflammatory disease

Once non-operative management has been exhausted, surgical management can be guided by the severity of osteochondral lesion of the talus (OLT) as described by Berndt and Harty[1] and later modified to include MRI[2] and CT findings[3].

Surgical Options

- **Stage 1 and 2:**
  - Debridement + microfracture
- **Stage 3, 4 and 5:**
  - Debridement + grafting (OATS, ACI, bulk allograft)
  - Retrograde drilling
  - Fixation
  - Replacement

OLTs with subchondral cysts (Stage 5) have poor outcomes with microfracture[4], therefore, alternative treatments have developed fill the defect.

Debridement + Microfracture

For simple lesions, debridement and microfracture yields good results with low complication rates and quick recovery[5]. The importance of microfracture compared to simple excision of the OLT, has been demonstrated via success rates of each (88% vs 38% respectively)[5].

Fixation of OLT

Fixation is typically reserved for large-enough OLTs, to ensure correct reduction. The size has been debated, with early papers quoting "one third of the talar dome"[6] or ">7.5mm"[7]. Current publications do not put a limit on the size of lesion for fixation[8]. The most important factor in achieving a good outcome is union of the bony fragment, as this was linked to cartilage quality on MRI and arthroscopic evaluation[9]. Different methods of fixation are available, typically screws[10] and/or bioabsorbable implants are used[9].

Grafting Options

<u>Osteochondral Autograft Transplantation System (OATS):</u>

- Taken from non-weight bearing portion of the knee
- Transported to recipient site
- Inserted into prepared holes at recipient site
- Usually reserved for revision surgery following failure of microfracture
- Approximately 12% reoperation rate[11]

<u>Autologous Chondrocyte Implantation (ACI):</u>

- Graft harvesting performed
- Send for culture in lab (4 – 6 weeks). Newer techniques are available that allow a single stage matrix-augmented ACI, e.g. Arthrex AutoCart™.
- Implantation into defect

<u>Cartilage Allograft:</u>

- Human tissue allograft – live chondrocytes from donated tissue
- Single procedure

<u>Bulk Allograft</u>

- For large defects of the talar body[12]
- Cadaveric allograft used to replace half of talus
- Fixed in place with headless compression screws
- Systematic review has shown a significant improvement in AOFAS ankle/hindfoot and VAS pain scores post-operatively, with an average graft survival rate of 86.6%[13].

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4.5. Arthroplasty in the Management of OLTs *Howard Davies*

There are multiple implants available for both discrete lesions and resurfacing of the talus. However, there are no clear indications for these implants in terms of location or size of the OLT, or if they should be used in primary or revision surgery. Furthermore, there are no large studies, RCTs or long-term outcome data. The available implants are detailed below.

HemiCAP®

The HemiCAP® is the most well-known of the partial joint replacements for the talus. it is a two-part implant, with a titanium peg and cobalt chrome surface. It is designed for the top to sit flush to the talar dome at the site of the OLT. It was developed in 2007 by Niek van Dijk, primarily as a treatment for failed medial talar defects. There are 15 different implant sizes to match the geometry of the medial talar dome. The offset sizes range for 0.5 – 2mm in the sagittal plane and 3.5 – 5.5mm in the coronal plane.

Evidence

Van Dijk himself reviewed his results through a prospective case series looking at 38 patients with a mean follow up of 5.1 years[1]. There was a 5.3% (2 patients) failure rate during this time, both of which received ankle fusion. AOFAS and VAS scores significantly improved post-operatively, except rest pain. In addition, the reoperation rate was high at 55.3% (21 procedures). These included 7 cheilectomies, 12 medial malleolar screw removals and 2 calcaneal osteotomies.

Another case series of 31 patients with a mean 50 month follow up found similar results, with AOFAS improvement from 47.6 to 79.1, a 3.2% revision rate (1 failure), and a 42% reoperation rate (13 procedures)[2].

A further retrospective study of 12 patients who received the HemiCAP® for previous failed OLT surgery, found the AOFAS, VAS and FFI disability and pain sub-scale scores all significantly improved[3]. 5 of the 12 patients were not satisfied with their outcomes which caused the authors to not recommend the HemiCAP®. However, the OLTs treated were not isolated to the medial talus, thereby contradicting the implant indications.

A cadaveric study assessing the biomechanics of the HemiCAP® found that if the implant sat just 0.25mm proud, the peak contact stresses reached 220%. While the HemiCAP® has the potential to restore normal joint mechanics, contact stresses are highly sensitive to implant positioning.

Talus resurfacing

There are two studies looking at talus resurfacing, aka ankle spacer, totalling 12 patients[4,5]. There was a 30% failure rate at 18 months, where patients were revised to total ankle replacement. Two of the patients had modest pain reduction and improvement in function.

3D Printing

Episealer®

The same principles are used as with the HemiCAP®, however, it is designed and manufactured for specific OLTs using CT scans. A single case report with a 5 year follow up has yielded good results, with the patient reporting reduced pain, improvements in clinical outcomes and return to their manual job and recreational football[6]. Further studies have been planned, with a prospective study based in Bologna having recently completed recruitment, and a retrospective cohort study based in Sweden and Germany.

Partial Talus Replacement

Partial talus replacements have been published in the literature as a case report[7], using a CT scan to produce patient specific instrumentation and a 3D printed implant that completely replaces the talar dome. At 2 years follow up, the patient reported return to sporting hobbies (golf, walking, swimming) with minimal pain and maintenance of pre-operative range of motion.

Ankle Hemiarthroplasty

3D printed ankle hemiarthroplasty has also been published in the literature as a single case report[8], for a large medial OLT that had failed microfracture and allografting. The patient reported complete relief of pain and return to activity at 14 months follow up, with AOFAS score improving from 43 to 80.

Summary

- There is evidence in favour of HemiCAP® for:
  - Revision surgery
  - Larger, medial lesions
- It is technically difficult to achieve good results
- There are high reoperation rates
- Continued rest pain is a problem
- Early results are not good for talus resurfacing
- 3D printed implants may work well → studies are in process

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Consensus Questions

1. Would you routinely scan all soft tissue injuries of the ankle?
   a. Yes 0
   b. No 24 **(100%)**
2. Would you routinely bring back a patient for a repeat scan if significant oedema was noted on an MRI?
   a. Yes 0
   b. No 24 **(100%)**
3. I would routinely follow up patients with an asymptomatic lesion noted on imaging.
   a. Yes 0
   b. No 24 **(100%)**
4. My first line investigations for OCLs are…
   a. XR 22 (91.7%)
   b. MRI 24 **(100%)**
   c. CT 1 (4.2%)
5. I would perform CT and MRI in combination.
   a. Always 0
   b. Sometimes 23 **(100%)**
   c. Never 0
6. If a patient presents with a soft tissue ankle injury and significant bone bruising evident on MRI what follow up would you offer?
   a. PIFU 23 **(100%)**
   b. Discharge 1
   c. Routine 0
7. My first line treatment for symptomatic lesions would be non-operative management.
   a. Yes 24 **(100%)**
   b. No 0
8. My first line non-operative treatment for a symptomatic lesion would include offer of injection.
   a. Always 1 (4.2%)
   b. Sometimes 18 (75%)
   c. Never 4 (16.7%)
9. I would consider vitamin D replacement for patients presenting with OLTs.
   a. Always 2 (8.4%)
   b. Sometimes 16 (66.7%)
   c. Never 5 (20.8%)
10. For OLTs >1.5cm my first line surgical management would be microfracture/bone marrow stimulation.
    a. Yes 23 (95.8%)
    b. No 1 (4.2%)
11. Weight bearing lower limb alignment views should be performed before surgical management.
    a. Always 0
    b. Sometimes 23 (95.8%)
    c. Never 1 (4.2%)
12. Following initial surgical treatment with microfracture/bone marrow stimulation, for a recurrent OLT representing with symptoms after 2 years I would consider further microfracture/bone marrow stimulation.
    a. Always 5 (20.9%)
    b. Sometimes 17 (70.8%)
    c. Never 1 (8.3%)
13. My preferred primary surgical procedure for OLT <1.5cm is…
    a. Microfracture 24 **(100%)**
    b. Other 0
14. Prior to revision surgery in OLTs, cases should be discussed as part of a specialist MDT/network.
    a. Yes 21 (87.5%)
    b. No 3 (12.5%)

Session 5: Midtarsal/Lisfranc injuries

Chaired by Mark Davies

5.1. Column theory of the midfoot

Mark Davies

The column theory has been commonly used to aid in description of midfoot stability and to help understand and guide management in midfoot fractures. However, the question remains as to whether midfoot structure is more complex than the column theory explanation.

Recognising the significance of some midfoot injuries does not appear to be consistent across the UK. Understanding how foot anatomy relates to function and being able to apply basic principles is of paramount importance in the diagnosis and management of these injuries. The importance of CT imaging in being able to understand the significance of a midfoot injury and to help guide management is recognised but this may not always be available in certain units.

In 1954 JH Hicks effectively first described the role of the windlass mechanism in midfoot function. This mechanism acts without the need for muscular action, but rather by creating tension across the plantar fascia through dorsiflexion of the digits leading to functional restoration of the anatomical arch.[1] The next year he went on to describe the foot as consisting of five independent weight bearing units that function in the absence of any muscular action, similar to the windlass mechanism he already described. What was important about this description of foot anatomy was that each unit can act biomechanically as a composite beam structure. In this theory again, the importance of tensioning of the plantar fascia in restoring anatomical function is recognised.[2]

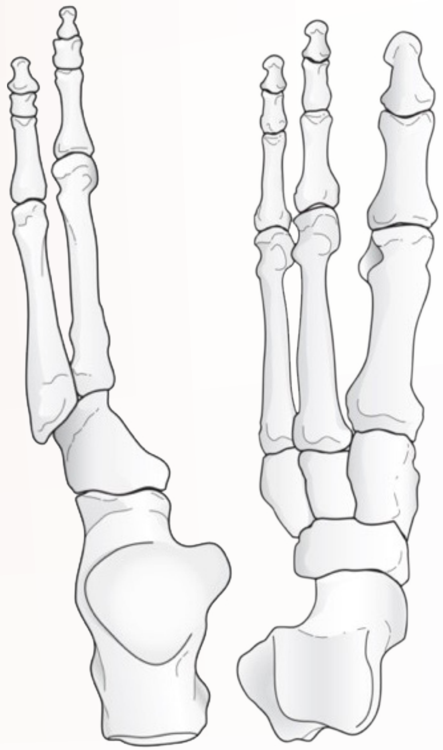
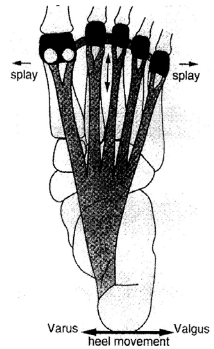


Figure 1

The columns of the foot were described in Dillwyn Evans paper ‘Relapsed club foot’ (JBJS 1961). In this paper 2 columns are described: the lateral column consisting of the calcaneus, cuboid and 4th & 5th metatarsals, and the medial column consisting of the talus, navicular, cuneiforms and 1st-3rd metatarsal bones (Figure 1).[3]

In published work throughout the 1970’s and 1980’s there is an appreciation and commonality of describing midfoot injuries in terms of the columns of the foot, sometimes described as the ‘pes talus’ and ‘pes calcaneus’.[4,5] This two-column theory of the foot is further described throughout published work on anatomy and mechanics of the foot throughout the 1990’s and beyond.[6]

Transverse and longitudinal tie-bar system



Both systems tightens when weight bearing on forefoot

Figure 2

In the late 1990’s David Stainsby presented work highlighting the role of the plantar plate and ‘deep transverse metatarsal ligament tie-bar’ in the pathological anatomy and mechanics of the foot. In particular he recognised the role of the plantar plate and transverse metatarsal ligaments in reconstituting the transverse arch of the foot and thus maintaining the anatomical structure of the mid and forefoot (Figure 2).[7]

Therefore, it has been suggested that foot injuries can be considered in terms of 2 longitudinal columns; the lateral column centred around the cuboid, and the medial column centred around the navicular. However, the transverse arch (centred around the cuneiforms as the pivotal cornerstone) of the foot, and its role in midfoot injuries, should not be overlooked.[8]

Further descriptions of managing midfoot injuries highlight the importance of restoring the lateral and medial columns, but also the re-tensioning of the plantar aponeurosis and therefore the transverse arch.[9]

Traditionally midfoot injuries may have been considered as either Chopart or Lisfranc injuries, however studies have shown that there are a significant proportion of injuries that are combined.[10] Such injuries may be under-recognised suggesting a potential problem with diagnosis and treatment that can influence the outcome of entire foot function in the mid to long term.

Work to try and classify both cuboid and navicular fractures has shown that whilst there are often clear patterns of injury, there is also considerable overlap between both classification systems for cuboid and navicular fractures, highlighting the prevalence of those combined injuries and the importance of considering associated injuries when faced with midfoot trauma.[11, 12]

Isolated longitudinal injuries can occur and should be considered as they may be devastating despite potentially appearing benign on initial radiographs. When managing longitudinal injuries of the 1st ray the direction, and more importantly, exit of force should be considered as this can significantly affect the stability of the medial column.[13] Oftentimes these injuries do not have a high energy mechanism and run the risk of not prompting the appropriate index of suspicion.

Using all of these principles, a guide to managing midfoot trauma has been suggested:

- **Maintain** – the appropriate lengths of the lateral and medial columns, and the relationship between the forefoot and hindfoot.
- **Preserve** – talonavicular joint function and the fourth and fifth metatarsal joints.
- **Use** – stable fixation to maintain anatomic reductions or primary arthrodesis.
- **Allow** – adequate time for bone and soft tissue healing before considering removal of any bridging metalwork.

Summary

In conclusion, when managing midtarsal injuries the foot can be considered in terms of two columns, however the importance of reconstituting the transverse arch through re-tensioning of the plantar aponeurosis should not be overlooked. Unfortunately, studies show that there may still be an underappreciation, and therefore inadequate management, of those combined injuries and clinicians should maintain an index of suspicion for more significant injuries despite an unconvincing history or initial plain radiographs. Use of CT imaging in midtarsal or midfoot trauma can aid in recognising more significant injuries. Furthermore, research has demonstrated poor outcomes associated in delayed treatment of these injuries with closed reduction and percutaneous pinning, suggesting early definitive management with open reduction and fixation techniques by specialist Foot & Ankle Surgeons only should be strongly considered.[14]

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5.2. Traumatic navicular and cuboid fractures *Stefan Rammelt*

The so called Chopart joint was named following the publication of a case report of amputation at the mid-tarsal joints by Francois Chopart in the mid 1700's. Likewise the term Lisfranc joint originates from descriptions of amputation at the tarsometatarsal joints in a textbook by Jacques Lisfranc in the late 1700's. Subsequently these terms have been adopted to refer to injuries around these joints.

At the levels of the Chopart joint we must consider the 4 bony components (calcaneus, cuboid, talus & navicular) and their articulations, but also the strong ligamentous complexes that lead it to behave as one functional unit.

Main and Jowett (1975) studied the mechanism of midtarsal joint injuries. They described how the injury sustained depends upon the foot position at the time of injury and direction of the deforming force. Forced adduction of the forefoot on the hindfoot leads to medial stress at the midtarsal joint, most commonly leading to fractures of the talar head and navicular. Forced abduction of the forefoot on the hindfoot leads to lateral stress at the midtarsal joint and more commonly fractures involving the anterior process of calcaneum and cuboid. 40% of injuries were also described as longitudinal compression injuries with a smaller proportion being crush or plantar injuries.[1]

When considering injuries around the Chopart joint it should be noted that with a compression injury on one side there is likely to be a failure in tension on the other side, which may or may not manifest as a fracture. Therefore, there should be a low threshold for considering associated ligamentous injury and instability.[1,2]

Research from the last 4 decades has repeatedly demonstrated that these injuries may be overlooked or underestimated, with a paper in 2023 quoting that these injuries are still missed in up to 30-40% of cases. It is thought that this is due to the overall low incidence of these injuries, the fact that patients can present with variable symptoms and signs, and inadequate initial radiographic imaging. Though perhaps most importantly midtarsal injuries may be frequently considered as isolated navicular or cuboid fractures with a lack of recognition of the joint characteristics and functional unit, leading to underappreciation of their significance.[1,3,4]

Assessment

Early recognition of more severe injuries around the Chopart joint is vital to prevent long-term complications with thorough clinical assessment and sufficient imaging paramount in preventing an incorrect 'midfoot sprain' diagnosis.

When assessing patients clinically it is important to have a high index of suspicion, in particular when the patient presents with signs such as plantar ecchymosis. This indicates that there must be disruption of the strong plantar ligaments of the foot and points towards a more severe or unstable injury.[5,6]

Proper initial radiographs in the correct projection can also guide the clinician towards an appropriate diagnosis. True lateral, dorsoplantar with 20 degree tilted tube, and 45 degree oblique views of the foot should be performed. Disruption of the Cyma line on a true lateral radiograph of the foot is an important clue towards Chopart instability and weight bearing views (sometimes including those of the contralateral foot) can also guide clinicians towards unstable injuries.[7,8]

Clinicians should have a low threshold for CT imaging if any suspicion is raised in initial clinical assessment or imaging. CT imaging not only aids in diagnosis of injuries of uncertain significance but also helps to guide operative planning. In particular CT scans are useful for detecting pathology such as central comminution, locked dislocations and ligament avulsions.[6]

Classification

Zwipp classified injuries around the Chopart joint based on the bone and ligament injuries involved. Purely ligamentous injuries were shown to be the most infrequent but most severe in terms of mechanism of injury and outcomes. Involvement of the soft tissues in these injuries should not be overlooked. Transnavicular and transcuboidal injuries were the most common injuries noted with transtalar and transcalcaneal being less so (Figure 1). In over 50% of cases there was a combined injury involving anywhere from 2 to all 4 bones of the Chopart joint. It would therefore be amiss to refer to isolated navicular or cuboid fractures unless proven otherwise.[4]

Transtalar fractures, whilst less common than transnavicular or transcuboidal injuries, do occur and a fracture of the talar head on imaging should point the clinician towards a Chopart injury. A double contour of the talar head on plain radiographs should raise suspicion of this.[4]

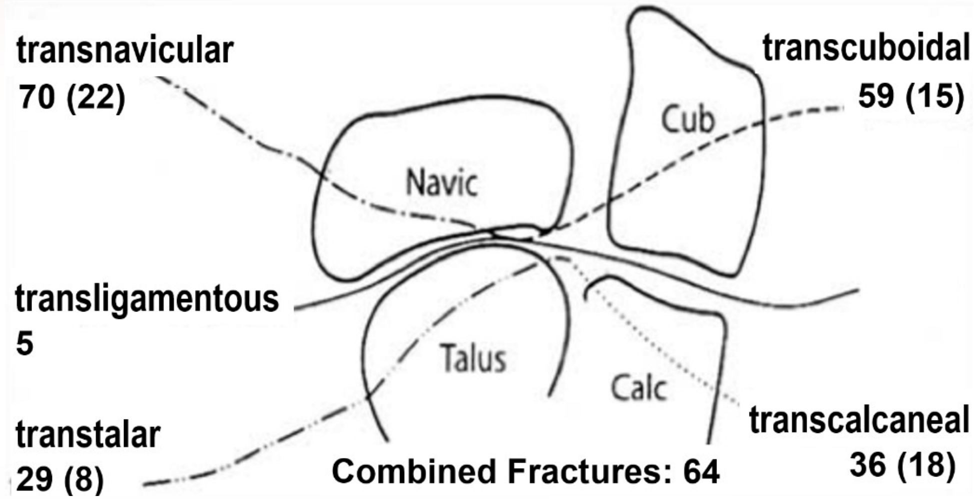

Figure 1

Transnavicular fractures are the most commonly seen and whilst there is clearly a medial compression injury, associated lateral distraction injuries must be considered. Likewise in cases of transcuboidal fractures, medial sided tension injuries must also be considered.[4]

Management

In managing lateral compression injuries (i.e. transcuboidal and transcalcaneal) injuries operatively a lateral utility approach has been described (analogous to the medial utility approach) with an incision extending distally from the tip of the fibula in line with the 4th metatarsal (Figure 2). This provides good access to the calcaneocuboid joint above the peroneal tendons and the lateral tarsometatarsal joints if required.[9]

Distraction and proper restoration of length of the lateral column, before fixation on the compression side, should be performed. Persistent instability may occur on the medial side if this has failed in tension, in which case transfixation of the talonavicular joint with K-wires can be a useful adage.

Figure 2

For those combined injuries (most commonly involving the navicular and cuboid but may be any combination of bones & joints), again proper open reduction and internal fixation, starting with either navicular or cuboid, must be performed. K-wires may be useful in fixation of small bony fragments not amenable to a screw, however closed reduction and percutaneous k-wire fixation of these fractures is not sufficient for definitive treatment.[10]

Prognosis and outcomes

Factors relating to prognosis in these injuries include polytrauma, associated ipsilateral foot fractures (seen in up to 30% of cases), combined injuries, purely ligamentous injuries and injuries requiring primary fusion. Importantly, as something we can influence, the quality of reduction and fixation (i.e. open reduction, internal fixation) in these injuries has also been shown to be a significant prognostic indicator. Factors not relating to prognosis in these injuries include age, gender and site of the fracture.[4,11,12]

In terms of long-term outcomes, a recent study has shown that injuries of the Chopart joint can have a significant effect on gait kinematics of the limb.[13] Long-term results are shown to decline from one through to four part fractures, with the poorest outcomes seen in transligamentous injuries. Outcomes have also been shown to correlate directly with the quality of reduction at the time of surgery.[4]

Potential complications seen following operative management of these injuries include wound edge necrosis, infection, complex regional pain syndrome (seen more commonly with a delayed diagnosis), avascular necrosis of the navicular, non-union and malunion (with shortening of either medial or lateral column increasing the risk of deformity and arthritis).

Summary

- Clinical signs may be subtle and there should be a high index of suspicion for significant and unstable injuries around the midtarsal joints.
- Proper radiographic projections and CT imaging is vital in ensuring a proper diagnosis is made and for appropriate operative planning.
- Concomitant injuries occur in up to 30% of cases and should not be missed.
- Over 50% are combined injuries and associate instability at the opposite side of the foot must be considered.
- Anatomical joint reduction is of vital importance in managing these injuries and affects long term outcomes. The medial and lateral columns must be restored and rebalanced.
- Closed reduction and k-wire fixation as definitive treatment has the risk of malreduction and failure.
- In cases of malunion early correction or fusion should be considered.

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5.3. Lisfranc injuries

Jane Madeley

Lisfranc injuries refer to those affecting the tarsometatarsal joints and their supporting ligaments, including the Lisfranc ligament. Injuries may involve the bone, ligaments or a combination of both. Variants of this injury may also involve the intercuneiform and naviculocuneiform joints and this must not be disregarded.

Anatomy

The cuneiforms and base of metatarsal bones are trapezoidal in shape and together the joints form a so called 'Roman arch'. The recessed base of second metatarsal acts as the cornerstone for this arch. The strong ligamentous complex (dorsal, interosseous and plantar portions) between the base of second metatarsal and medial cuneiform help to maintain stability here. The interosseous portion (named the Lisfranc ligament) is the largest and strongest ligament within this complex, followed by the plantar, then dorsal ligaments.[1] The plantar ligament has often been seen to have 2 slips from the medial cuneiform – one to the base of second and one to the base of third metatarsals.

More recently Mason et al (2020) have described the lateral Lisfranc ligament in a cadaveric study (Figure 1). This plantar ligament spans from the second to fifth metatarsal to blend with the long plantar ligament and appears separate from the intermetatarsal ligaments connecting each metatarsal. This ligament perhaps explains why lateral instability often resolves following stabilisation of the medial three tarsometatarsal joints.[2]

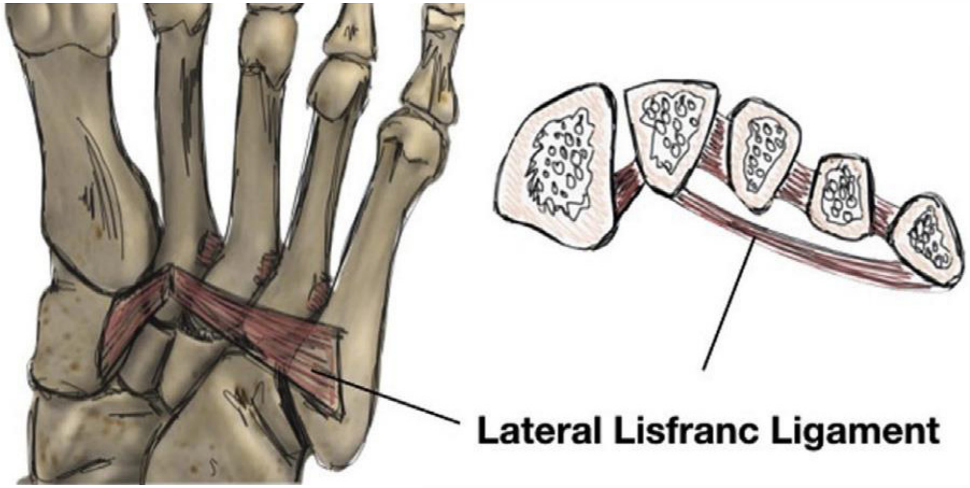

Figure 1

Classification

The original Quenu and Kuss classification was based on the concept of 3 columns. This was later modified by Hardcastle and then Myerson to include classifications based on direction or displacement, congruency and complexity of the injury. Myerson also commented on the fact that whilst commonly considered as solely transverse injuries, there is often an associated longitudinal component.[3]

Nunley and Vertullo have more recently developed a classification system to describe the lower-grade Lisfranc injuries. This system describes injuries based on clinical symptoms, bone scan findings, first to second metatarsal distance on weight bearing dorsoplantar radiographs and height of the medial longitudinal arch on weight bearing lateral radiographs.[4]

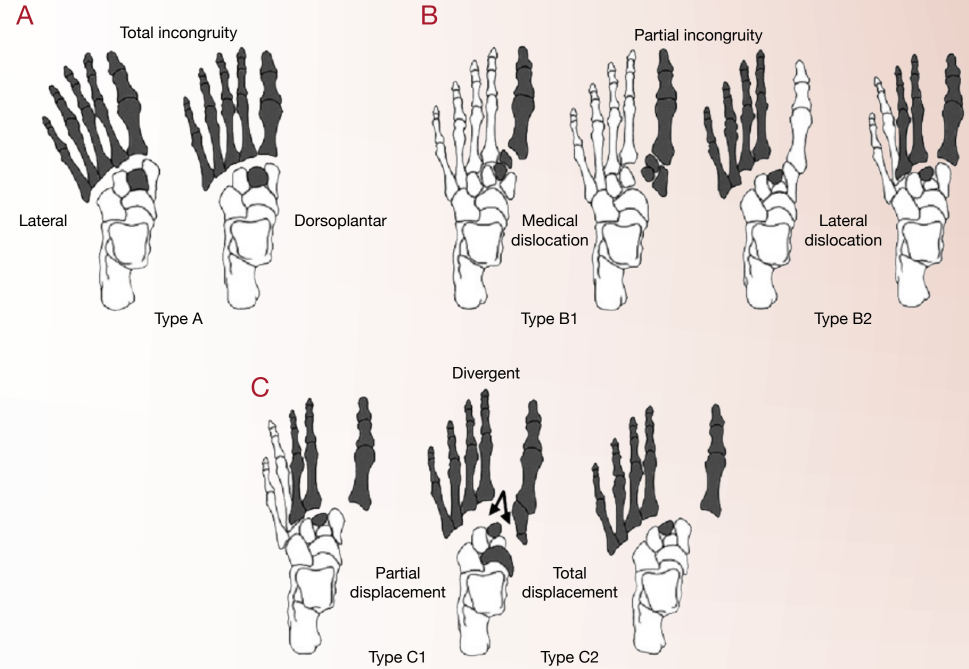

Figure 2

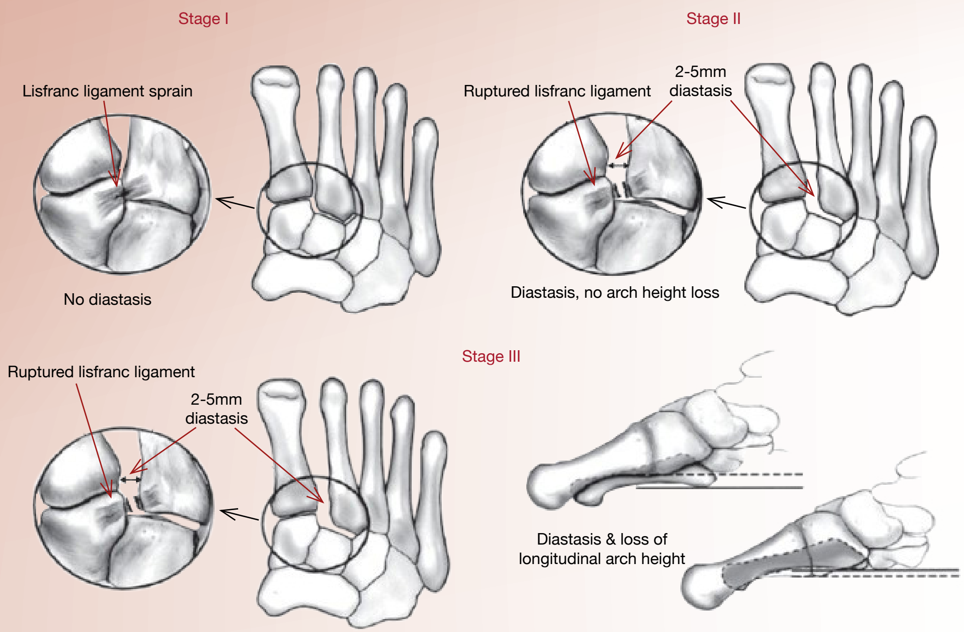

Figure 3

Mechanism

Traditionally approximately 70% of Lisfranc injuries were diagnosed following high energy mechanisms. An increasing proportion of these injuries are being seen following lower energy mechanisms, particularly in the more elderly population or following sports injuries. A recent paper (Stodle et al 2020) suggested the ratios of high to low energy injuries being seen is now reverse, hypothesising that this may be due to improvements in imaging and clinical index of suspicion.[5]
Indirect injuries tend occur with a longitudinal force being applied to the forefoot resulting most commonly in plantarflexion and abduction, though the exact mechanism and pattern will depend on the position of the ankle and forefoot at time of injury.

Direct injuries tend to result from a crush type mechanism with these injuries being associated more with soft tissue damage and risk of neurovascular compromise.

Often these injuries may be missed or underappreciated at the time of initial presentation. Trevino et al reported that this was the case in 20% of cases though this is improving with increased awareness and the availability of cross-sectional imaging. Unrecognised or untreated Lisfranc injuries can result deformity, degenerative change, or a combination of both.[6]

Diagnosis

This requires a high index of suspicion for these injuries through thorough history regarding the mechanism of injury & whether the patient is able to weight bear, and through clinical examination (in particular looking for plantar ecchymosis or pain on stressing the tarsometatarsal joints).
Weight bearing radiographs, with consideration of imaging of the contralateral foot for comparison, should be performed where the patient is able to do so. Correct radiographic views must be obtained with a 20 degree tilt for dorsoplantar radiographs to allow proper visualisation of the tarsometatarsal joints (which are typically angled by 28.9 degrees relative to the floor).[7]

When performing weight bearing radiographs of both feet for comparison it has been shown that this should be performed with feet on separate cassettes to ensure the beam is centred over the second metatarsal in both feet (Figure 4).[8]
Studies have shown that a gap of over 2mm between the medial cuneiform and base of second metatarsal on radiographs has a 96% sensitivity for diagnosing unstable Lisfranc injuries.[9]

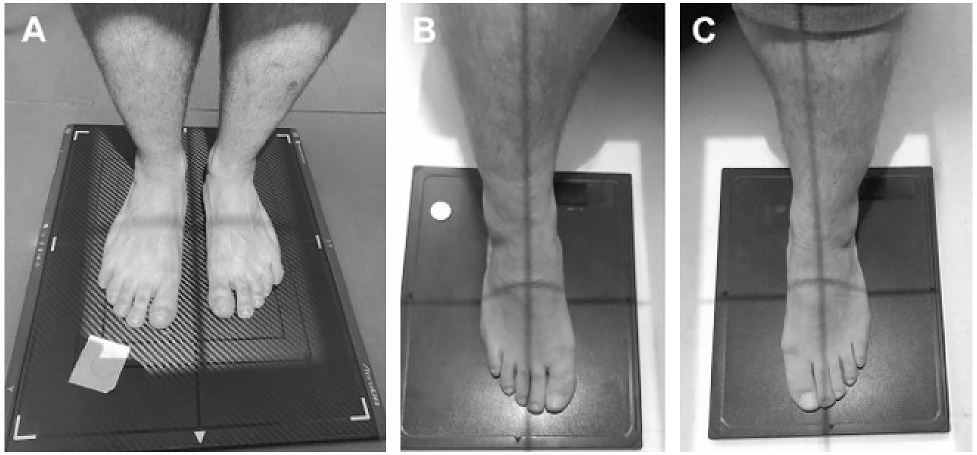

Figure 4

Cross-sectional imaging with a CT scan allows more detailed assessment, in particular looking at whether the rays and columns are aligned. Other indications of instability on CT imaging include diastasis between the first and second rays or the cuneiforms, diastasis between the base of second metatarsal and medial cuneiform, a fleck sign, loss of arch height and other associated fractures.
Weight bearing CT scans are an emerging diagnostic tool with cadaveric studies demonstrating sensitivity of this imaging to partial or complete Lisfranc rupture.[10]

Kennelly et al compared the utility of weight bearing radiographs and CT imaging for subtle Lisfranc injuries. In those patients with weight bearing radiographs positive for a Lisfranc injury, only 54% had a Lisfranc injury reported on CT imaging. Conversely for those that had a negative weight bearing radiograph only 12% went on to have a diagnosis of a Lisfranc injury on CT. They concluded that bilateral weight-bearing radiographs were preferable for diagnosis, though CT imaging is often necessary for operative planning. They also suggested that for those patients that were unable to tolerate weight bearing initially then a further review with a second attempt at weight bearing radiographs was a reasonable course of action.[11]

MRI is rarely used in the acute diagnosis of Lisfranc injuries though may be used for those patients presenting with persistent pain or symptoms of instability despite good alignment on radiographs and CT. MRI imaging can provide information on ligament involvement or may demonstrate bone bruising.

Examination under anaesthetic may also be considered if diagnosis is not clear from clinical examination or imaging.

Treatment

A number of treatment algorithms for managing Lisfranc injuries have been suggested in the literature.

Non-operative treatment appears to have increased with a systematic review in 2023 demonstrating an estimated incidence of 1.6/100,000 of non-operative treatment in the 1980's compared to 9.2-14/100,000 in the 2020's. This increase in rates of non-operative management is largely due to improved imaging and diagnosis of more subtle injuries. Whilst the diagnostic criteria used to decide on non-operative treatment was relatively consistent between those studies included, the treatment protocols were not. Irrespective of the non-operative treatment given there was a range of 0-54% of patients across these studies that went on to develop secondary diastasis and require operative treatment, perhaps suggesting the need for clinicians to consider the role of dynamic instability over time rather than relying solely on initial imaging. Despite this, the outcomes of those undergoing delayed surgery were similar to those undergoing primary surgery and the best outcomes in those managed non-operatively were seen in patients where a CT had been performed and demonstrated less than 2mm displacement.[12]

In those patients where operative treatment is most appropriate the importance of anatomical reduction of the joints must not be underestimated. Therefore, open reduction should be the gold standard in these injuries. This allows direct visualisation of the joint, removal of any interposed soft tissue, but also conversion to primary arthrodesis if significant chondral damage is noted.

Traditionally the most common mode of fixation was with trans-articular screws, though it is now recognised that there is some chondral damage that occurs with this technique.[13]

The use of bridging plates as an alternative can help to overcome this issue. Studies have shown that the results of bridging plating are equivalent to trans-articular screws and are therefore a valid alternative. Long term follow up is still required to assess whether this also translates to lower rates of post-traumatic arthritis. The poorest outcomes have been noted in patients who underwent combined fixation with both plates and trans-articular screws. Overall, the importance of anatomical reduction has been recognised in influencing patient outcomes.[14-16]

For more subtle ligamentous injuries suture button techniques may be employed with equivalent results demonstrated when compared to screw fixation.[17]

If there is ongoing lateral ray instability following stabilisation medially, k-wires may be utilised at the fourth and fifth tarsometatarsal joints.

Primary arthrodesis in Lisfranc injuries has been discussed extensively in the literature. Ly and Coetzee (2006) published work demonstrating improved outcomes for primary fusion in ligamentous Lisfranc injuries.[18] Barnds et al 2018 reviewed the cost implications of arthrodesis vs ORIF for Lisfranc injuries and showed that primary arthrodesis was more expensive with a higher rate of complications. They also noted that only 2.5% of those patients undergoing ORIF as the primary operation went on to have arthrodesis.[19]

A systematic review and meta-analysis of outcomes following fixation or fusion for Lisfranc injuries showed an overall greater increase in AOFAS and VAS scores in those undergoing primary arthrodesis. They did however note that their results were skewed somewhat by the results of Ly and Coetzee and when that paper was excluded from their analysis the difference was much less profound. There is an apparent higher rate in the literature of metalwork removal in those undergoing ORIF but it is not clear what proportion is due to planned metalwork removal versus removal due to complications.[20]

In terms of salvage procedures for those that have presented late or have a failed primary procedure, arthrodesis has been shown to be the most reliable option. It has again been highlighted the importance of CT imaging for operative planning and the need to achieve anatomical alignment of the joints.[21]

Summary

- Consistent diagnostic criteria and stability assessment are required to prevent both under and over treatment of these injuries.
- Whilst weight bearing radiographs utilising appropriate projections are useful in the diagnosis of these injuries, CT imaging remains an important adjunct for operative planning.
- The best non-operative management of Lisfranc injuries is not clear. There is potentially a role for immediate weight bearing to provoke and allow early identification of missed instability.
- When managing Lisfranc injuries operatively, anatomical reduction of the joints is the most important influencer on outcomes and therefore open reduction is the gold standard.
- Bridge plates are becoming a commonly utilised method for fixation with literature showing equivalent results to trans articular screws without the potential risk of chondral damage. Whether this translates to long-term reduction in post-traumatic arthritis is yet to be demonstrated.
- There is evidence to suggest that primary arthrodesis in purely ligamentous Lisfranc injuries can improve outcomes, though the significance of this is unclear.

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Consensus Questions

1. In a patient with an equivocal diagnosis and who is able to tolerate weight bearing, is it useful to do a standing AP and lateral plain radiograph?
   a. Yes 20 **(100%)**
   b. No 0
2. In a patient with an equivocal diagnosis and who is able to tolerate weight bearing, is it acceptable to request a contralateral standing plain radiograph to guide diagnosis?
   a. Yes 20 **(100%)**
   b. No 0
3. In the management of all midfoot trauma CT should be considered.
   a. Yes 19 (95%)
   b. No 1 (5%)
4. Weight bearing CT could improve imaging assessment of patients with midfoot trauma.
   a. Yes 18 (90%)
   b. No 2 (10%)
5. Does the panel feel that the ratio of incidence of high to low energy injuries has been reversed so that we are now more commonly detecting low energy injuries?
   a. Always 20 **(100%)**
   b. Sometimes 0
6. Do k-wires provide sufficient stability for the sole definitive fixation of these injuries?
   a. Always 0
   b. Sometimes 10 (50%)
   c. Never 10 (50%)
7. Given that the best predictor of outcome for these injuries is anatomical reduction does the panel agree that percutaneous reduction is not acceptable?
   a. Yes 20 **(100%)**
   b. No 0

8. Should Lisfranc and Chopart injuries be definitively managed by a foot and ankle surgeon?
   a. Yes 20 **(100%)**
   b. No 0
9. In a purely ligamentous Lisfranc injury, is there a need for primary fusion techniques as the definitive surgical treatment?
   a. Always 0
   b. Sometimes 16 (80%)
   c. Never 4 (20%)
10. Is there a role for postoperative CT assessment following fixation of these injuries?
   a. Always 0
   b. Sometimes 20 **(100%)**
   c. Never 0

Session 6: Progressive Flatfoot Deformity

Chaired by Rick Brown

6.1. Ligaments vs Tendons in the Progressive Collapsing Foot Deformity

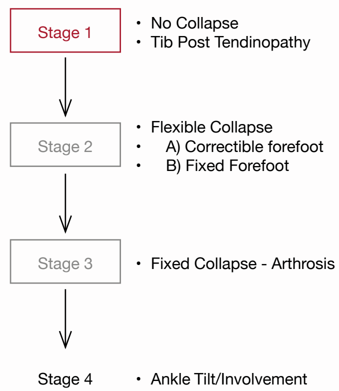
Tim Williams

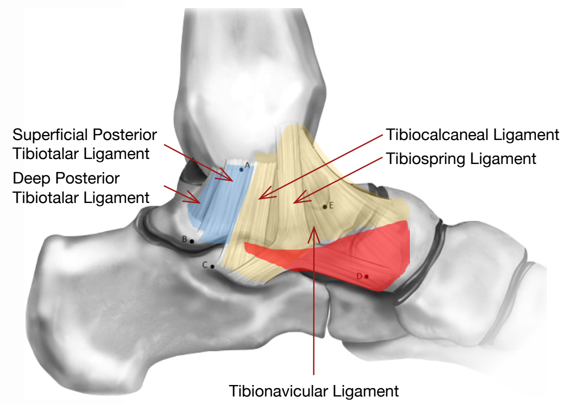
Historically it had been thought that tibialis posterior tendon insufficiency/lengthening is what leads to a progressive flatfoot. This theory highlights the tibialis posterior as responsible for converting the foot from a shock absorber into a rigid lever. It was thought to accomplishes this by being a shock absorber, an arch supporter, and an inverter and plantar flexor of the foot. The tibialis posterior tendon travels behind the medial malleolus and has a broad attachment into the foot at the navicular, cuneiforms, MT 2-4 bases and the cuboid[1]. The medial malleolus acts like a pulley, which enables tibialis posterior to invert the foot. This mechanism gave rise to the classical description by Johnson and Strom[2], that linked the lengthening and failure of tibialis posterior to the different stages of the collapsing flatfoot. This was later modified to include ankle pathology[3].

Calcaneonavicular (Spring) Ligament

We are now beginning to think differently about a multi-factorial cause of a flatfoot deformity and whether this may be failure of ligaments rather than solely tibialis posterior[4]. For example, when the tibialis posterior tendon is transferred for a foot drop, you do not get a progressive flat foot deformity[4]. In addition, it has been shown that injury to the spring ligament causes progressive collapse[5]. Similarly, this has been demonstrated with the deltoid ligament: sectioning of the superficial deltoid increases eversion and pronation of the foot, while sectioning the deep deltoid leads to talar tilt[6,7]. The spring and deltoid ligaments are intimately related in their structure and function. They are inelastic restraints that support the medial longitudinal arch. It is failure of the deltoid-spring ligament that we now believe to initiate the progressive flat foot deformity.

Sequence of Collapse





This figure shows the commonly accepted sequence of a collapse, and indicates the ligaments which may be responsible for each stage in colour coding.

Deltoid Ligament

The deltoid ligament not only helps to stabilise the arch of the foot but is well known as a stabiliser of the ankle. Damage will lead to talar tilt and an anterior-external rotational draw to the ankle. Just like lateral stability loss leads to peroneal tendinopathy, antero-posterior instability of the deltoid ligament can lead to tibialis posterior tendinopathy even without a flat foot in stance or with only a subtle terminal stance over-pronation. Superficial deltoid injury, therefore, has ramifications in that it can lead to stage 1 tibialis posterior tendinopathy and possibly be the start of a progressive flat foot. This needs to be understood and considered when deltoid ligament injury is identified.

<u>Situations to consider a Deltoid Reconstruction</u>

Signs of deltoid laxity

- Visible medial laxity to anterior draw
- Copious Saline infiltration volume when accessing joint in arthroscopy
- Torn deep deltoid fibres
- EUA after lateral reconstruction

Signs of secondary damage from deltoid laxity

- Tender medial gutter or anteromedially
- Anteromedial tibia (osteophyte presence +/- synovitis)
- Tender tibialis posterior at tip of medial malleolus

The Role of Tibialis Posterior

If ligamentous structures are the cause of the progressive flat foot, then what is the role of the tibialis posterior tendon? Tibialis posterior has some interesting features:

- It is the second strongest muscle in the lower leg after the gastro-soleus complex, yet it is a weak plantar flexor.
- Initiates heel raise
- Large excursion of 2cm
- Relatively long tendon containing elastin, with the potential to store large amounts of energy when eccentrically contracted
- Broad insertion across entire plantar foot

<u>Tibialis Posterior during Stance Phase of Gait</u>

The adjacent figures demonstrate the tibialis posterior tendon in:

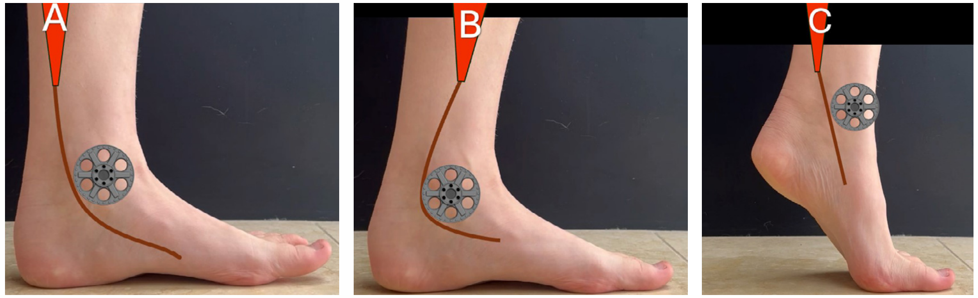
- Mid stance phase with the tendon in maximum excursion (A).

The tibialis posterior tendon is anchored to the plantar foot, which is in turn anchored to the floor by the weight of the body.

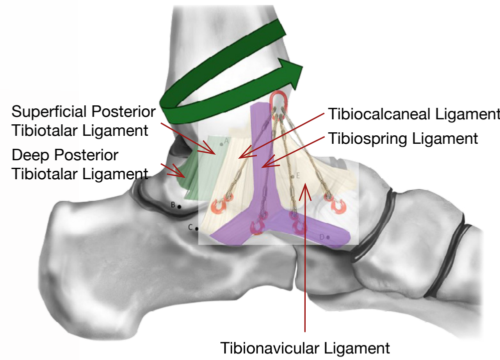
- Late stance phase when the tendon is eccentrically loading and generating potential energy storage (B).

As the ankle goes into dorsiflexion, the body weight goes directly over the midfoot, anchoring it even more into the floor.

- Terminal stance phase with the tibialis posterior muscle concentrically contracting (C).



As tibialis posterior contracts, the tendon straightens and exerts force across the mobile pulley, that is the medial malleolus (represented by the wheel in the figures), thereby externally rotating the tibia. The tibialis posterior acts on the medial malleolus and not the foot, because the foot is anchored into the floor.



The consequence of this is that the deltoid ligament, which is anchored to the medial malleolus and acting as an inelastic tie-bar link (see figure below), pulls the foot into inversion and supination.

Summary

1. Progressive flat foot is caused by rapid or attritional failure of the deltoid-spring ligament.
2. Progressive flat foot can start with a superficial deltoid ligament injury.
3. Progressive flat foot affects the function of the tibialis posterior tendon and is not caused by it.
4. We still do not fully understand the workings of gait in its normality to therefore comment entirely as to why we get pathology. We need to learn more about this going forward.

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6.2. What is the effect of Rotation in Progressive Collapsing Foot Deformity?

Chandra Pasapula

When considering the progressive collapsing foot deformity (PCFD), rotation can occur in multiple ways on the bones, joints and mechanical axes. This is a complex topic with interplay of ground reaction forces (GRF), ligament/tendon forces and mechanical axis deviations. The introduction of weight bearing CT has superseded plain films regarding evaluation of flat feet, in particular talus rotation.

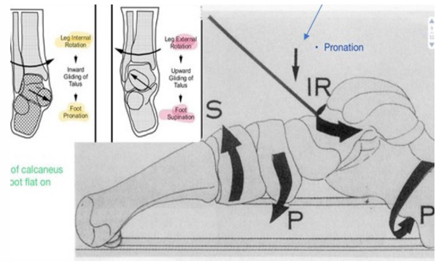
What rotates?

There is a lot of natural rotation that occurs in gait:

- Tibia internally rotates approx. 10°
- Talus internally rotates in terminal stance
- The medial column pronates due to peroneus longus action

As we are interested in PCFD, this topic will focus on subtalar axis rotation and its interplay with tendons and ligaments.

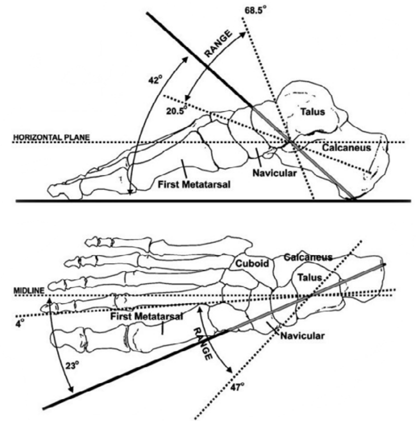
Subtalar axis internally rotates with pronation



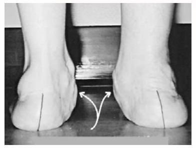
Subtalar Axis

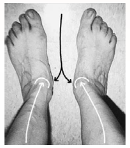
The subtalar axis is dependent upon the morphology of the subtalar joint articulating surfaces and is closely linked to talus position. It penetrates the anterior talar neck, following the line of the talar neck and body centrally. The subtalar axis varies throughout the gait cycle and with the position of the foot (see figure):

- Pronation ⟶ IR + plantarflexion of the talus **(midstance phase)**
- Supination ⟶ ER + dorsiflexion of the talus **(heel off/terminal stance phase)**



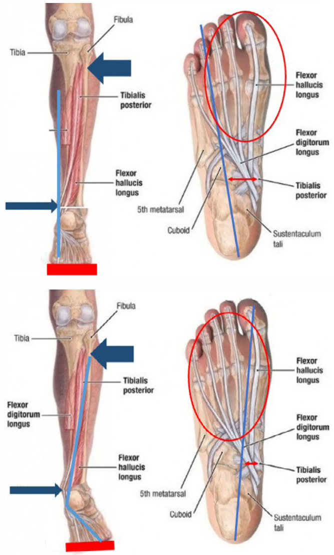
Clinical Assessment





Subtalar axis deviation can be clinically assessed by inspection from the top and back, looking for abnormal convexity of the medial midfoot (black arrows in photo) and medially positioned and internally rotated soft tissue contour of the talar neck and head (white lines and arrows). This is important because **confirming talus internal rotation, effectively confirms internal rotation of the subtalar axis.**

Association with PCFD



In normal gait, a GRF-inversion moment is generated through a stable first ray in terminal stance. This is at maximal distance from the subtalar axis, which follows the talus which is forced to externally rotate with tibial external rotation peaking at mid/terminal stance. Tibial external rotation and hindfoot inversion is augmented by tibialis posterior. The tibialis posterior insertion has a maximal distance from the subtalar axis (see longer red arrow in figure), which is beneficial in augmenting the GRF-inversion moment.

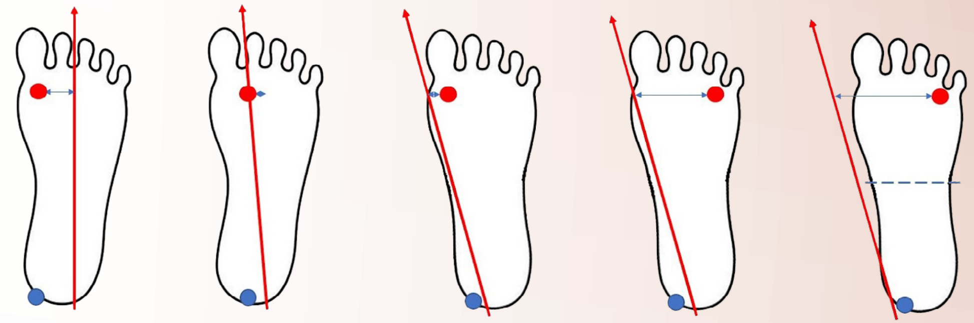
In PCFD, spring ligament laxity allows medial deviation of the talar head, internalising the subtalar axis whose motion is amplified at the forefoot, decreasing the GRF-inversion moment.
Tibialis posterior reacts by trying to increase its force output to correct these changes. Eventually, with persistent spring ligament laxity the subtalar axis internally rotates and medially deviates further, leading to a lateral GRF (see red oval in lower figure) that tibialis posterior tries to compensate for.

It does not take much to generate an internally rotated subtalar axis. It has been shown that sectioning of the spring ligament in isolation induces peri-talar rotational changes, particularly internal rotation of the talus and subtalar axis deviation[1]. The **spring ligament**, therefore, serves as the "lynchpin" to controlling peri-talar rotations and supporting the medial arch.

Tibialis Posterior Involvement

Incremental tensioning of tibialis posterior was found to be ineffective in counteracting the peri-talar rotational changes[1]. A further study looking into tensioning of tibialis posterior to correct a planovalgus foot, found that tibialis posterior partially restores hindfoot and forefoot alignment but not the arch[2]. A non-physiological 700% increase in tibialis posterior force was noted to be required to restore foot alignment. Thus, foot alignment around the subtalar axis is, therefore, more dependent on ligaments. Moreover it is not just tibialis posterior that tries to increase its pull, abductor hallucis and gastro-soleus do the same[3,4].

Achilles Involvement



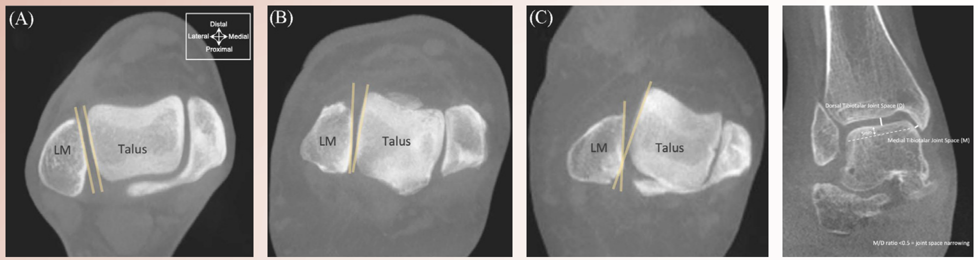
This figure demonstrates the relationship between the subtalar axis, GRF at the forefoot, and pull of the tendoachilles (TA).

The TA also becomes overactive in PCFD to accommodate the reduced lever arm as the subtalar axis deviates medially. The arc of motion of the subtalar axis is centred around the centre of the talar head. In addition there is a greater arc of motion of the subtalar axis as the site of the forefoot GRF moves laterally which challenges TA further, requiring yet more compensation from TA[5].

Eventually, the centre of pressure starts to deviate laterally, causing the zone of instability to spread from the medial to the lateral column, thereby destabilising the lateral column[6,7].

Talus Internal Rotation

The talus internally rotates as the tibia internally rotates in PCFD, but what we are just starting to understand from standing CTs is that the talus also internally rotates within the ankle mortice[8,9] (see CT images). This represents a wider failure of the ligaments.

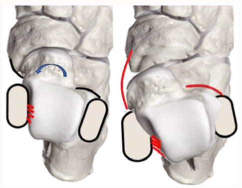


Control — Moderate Abduction Group — Severe Abduction Group

Coronal and sagittal plane stability of the first ray is required to ensure the high forces generated in terminal stance (especially with accelerated gaits) are transmitted appropriately through the medial column of the foot (see figure).
Over 60% of the GRF is transmitted through the medial column.

Any incongruency of the talonavicular joint, results in a lateral force directed medially (small blue arrow) with decreased force longitudinally through the medial column (see figure).
This lateral force (the smaller blue arrow) is creating a second moment that is acting on the talus, causing internal rotation and talar torque with cyclical loading.

What keeps the Talus in Place?



There are 4 main constraints to the talus:
1. Bony architecture (contributes 30% of rotational stability[10])
2. Spring ligament
3. ATFL
4. Deep deltoid ligament

In PCFD, the talus goes into plantarflexion, causing a failure to engage the widest part of the talar body. The talus is now reliant on the ligaments to keep itself in place. However, the spring ligament has already failed, leaving only the ATFL and deep deltoid ligament (see figure). The ATFL acts in tension, which it is experiences in PCFD, therefore, it continues to function. In contrast the deep deltoid ligament undergoes shear, which eventually causes deep deltoid to fail. In addition, the talus has no muscles that act upon it to exert dynamic control.

Summary

The effects of rotation on PCFD include:
1. Talus malrotation causes widespread foot dysfunction
2. Alteration of biomechanics of tendons and forces going through them
3. Leads to deep deltoid failure (before ankle joint degeneration (Johnson & Strom Stage 4)
4. Talar rotation causes more medial sided tibiotalar joint degeneration (than is appreciated on x-rays)
5. Overload of tendon and muscle groups

Take Home Points

- Subtalar axis rotation is critical to foot function.
- Internal rotation of the talus, with respect to the ankle, is a characteristic of PCFD and is accentuated in more severe abduction deformities.
- Internal rotation of the talus within the ankle mortice, indicates there is more ankle pathology in PCFD patients than recognized.

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6.3. What is the role of Lateral Column Lengthening?

Jane Madeley

Distraction of the lateral column at the anterior process of the calcaneum or calcaneocuboid joint (CCJ) is used to compensate for the functional shortening caused by a flat foot deformity.

How does it work?

The procedure works by creating a 3D effect of rotating the forefoot around the talonavicular joint (TNJ). This not only corrects forefoot abduction, but also serves to support the medial arch (see figure).

Cadaveric study has shown the correction of the axial plane (forefoot abduction) and sagittal plane (talonavicular dorsiflexion) deformities to be true[1]. Just a 6mm lengthening was found to be enough to correct the deformity in both planes, indicating the power of a lateral column lengthening (LCL).

No osteotomy   10 degrees

Importantly, a shift in the peak plantar pressure from medial to lateral was demonstrated. Therefore, it is vital not to over correct when performing a LCL, to prevent lateral sided pathology from occurring.

What are the Indications?

- Flexible flatfoot with forefoot abduction
  - i.e. Indicated when 40% talonavicular uncoverage[2] as measured on a weight bearing AP x-ray of the foot
- Should be used in conjunction with bony and soft tissue corrections
- Ca be an adjunct to medial displacement calcaneal osteotomy (MDCO) or utilised on its own

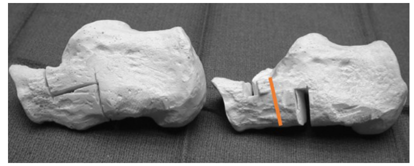
Techniques

1. Osteotomy of the anterior process/mid-section of the calcaneum

<u>Evans osteotomy</u>

- Start 1.3mm posterior to CCJ
- Direct osteotomy from posterolateral to anteromedial to avoid posterior facet and sinus tarsi
- Open the gap with laminar spreader/ stacked osteotomes/Hintermann distractor
- Use fluoroscopy to check correction
- Graft the osteotomy site
  - No statistical difference between allograft or autograft. There is a tendency to increased non-union with larger graft size[3].
  - Rectangular grafts more powerful, but wedges are best to avoid subluxation of CCJ and TNJ[4].

<u>Z-osteotomy</u>



- Aims to reduce collapse and loss of position that can occur with an Evans osteotomy[5]
- Large surface area, therefore potential reduction in non-union risk (no evidence).
- Can be combined with MDCO
- Technically difficult

2. Distraction fusion of CCJ

- Prepare joint surfaces
- Distraction under fluoroscopic guidance until desired correction achieved
- Bone graft and fixation
- Creating a stiff lateral column through fusion could, in theory, lead to reduced functional outcomes. However, this is not found in the literature. issues with LCL arise due to over correction[6].

Whichever technique is used it is vital to use intra-operative fluoroscopy to guide correction. Overcorrection will restrict subtalar motion and increase lateral pressures. Checking passive eversion and forefoot adduction intra-operatively can help to reduce risk of overcorrection.

Which procedure is best?

When comparing LCL (via distraction CCJ arthrodesis) with MDCO, LCL showed greater correction of forefoot abduction and longitudinal arch, with results lasting longer. MDCO had higher reoperation rates (due to screw removal). However, LCL had higher rates of OA in adjacent hindfoot joints[7].

Evans osteotomy has been shown to have a higher satisfaction compared to distraction arthrodesis, with no statistical difference in correction or non-union rates between techniques[8]. A systematic review comparing the same techniques found no significant differences between the overall outcomes of each technique. However, they did recommend that distraction arthrodesis may be a more reliable technique in patients with a high BMI[9].

Risks and Complications

- The predominant risk is development of lateral foot pain from increased plantar-lateral pressure or increased CCJ pressure due to overcorrection.
  - The use of trial wedges to determine graft size may help reduce the incidence of lateral foot pain[10].
- Failure to recognise compensatory forefoot supination, in which case
  - Consider a Cotton osteotomy or plantarflexion 1st TMTJ fusion
- Graft collapse or loss of correction
  - Use locking plate to maintain position
- Non-union
  - Approximately 8 – 9%[9]
- Under or over correction
- Peroneal tendon symptoms
  - Due to lateral distraction or prominent metalwork
- Sural nerve symptoms
  - Due to lateral distraction

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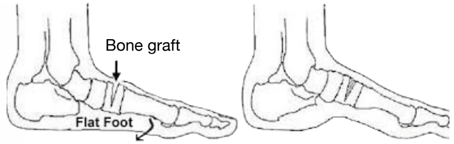
6.4. Why add a Cotton-type Osteotomy?

Robert Clayton

What is a Cotton Osteotomy?

The Cotton osteotomy is a dorsal opening wedge osteotomy of the medial cuneiform. The plantar hinge needs to be kept intact and the wedge filled with bone graft, thereby restoring the arch of the foot.



Indications

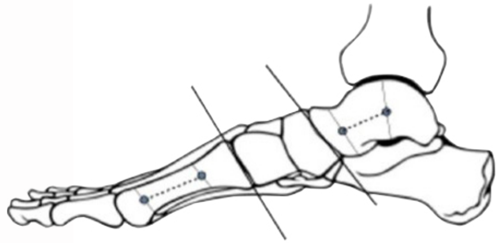
- Progressive collapsing flatfoot correction
- Flexible forefoot varus with stable 1st ray
- Adjunct to triple fusion and hallux valgus correction

Technical Tips

- Dorsal approach
- Minimal release of joints to maintain stability
- Carefully identify NCJ and TMTJ on inspection and fluoroscopy
- Osteotomy under fluoroscopic guidance and by feel
- Use corticocancellous graft
  - From calcaneum if in conjunction with MDCO
- Use thumb on plantar MT head to gauge size of wedge required

Supportive Evidence

Cotton osteotomy has been shown not to improve Meary's angle. However, it did improve the medial arch sag angle (see figure) and was shown to be more effective with an Evans osteotomy, rather than a MDCO[1].



Meary's Talo - 1st Metatarsal Angle

MASA - Medial Arch Sag Angle

Using a Cotton osteotomy was found to be the most powerful predictor for achieving a good radiographic correction in a flatfoot deformity[2].

As an adjunct to triple arthrodesis, Cotton osteotomy was the primary factor in achieving correction in patients with forefoot supination[3]. However, this is also correctable through the arthrodesis.

A recent consensus meeting in the United States decided upon the indications for Cotton osteotomy in PCFD[4]. They agreed:

1. A stable longitudinal arch is required (9/9)
2. Adequate correction has been obtained when the 1^{st} metatarsal head can be felt level with the 5^{th} on pushing up the forefoot (9/9)
3. Wedge size required is 5 – 11m (9/9)
4. Presence of "some instability" does not preclude a Cotton osteotomy (8/9)
5. 1^{st} TMTJ fusion is required if the TMTJ is "grossly unstable" or gapping on lateral x-ray (9/9)

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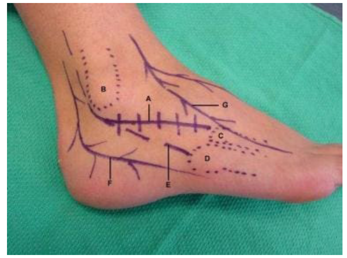
6.5. When to do a Triple Fusion?

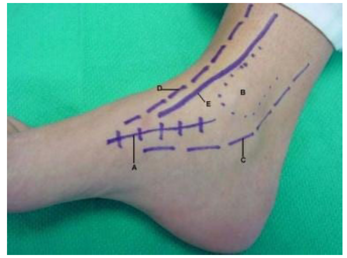
Roland Walker

The triple arthrodesis was first described by Ryerson in 1923[1] and was originally described for stabilising the hindfoot in polio patients. This was an evolution of Hoke's technique described to fuse the STJ and TNJ[2].

Operative Technique

The traditional technique uses a lateral approach from the tip of the fibula to the base of the 4^{th} metatarsal and a medial approach between tibialis anterior and posterior. The foot is rotated and realigned around the TNJ and held in place with internal fixation devices.





Indications for Triple Fusion in PCFD

- A stiff, flat foot with an intact ankle joint (Johnson & Strom stage 3)
- To provide a pain free plantigrade platform before ankle replacement in Johnson & Strom stage 4
- Johnson & Strom stage 2 in high BMI patients
- Johnson & Strom stage 2 in hypermobile patients
- Failed surgery for Johnson and Strom stage 2
  - e.g. cut out of FDL transfer/internal brace
- Missed traumatic rupture of tibialis posterior/delto-spring ligament

As mentioned above, the triple arthrodesis was originally designed to treat polio, and it is certainly still indicated for neuromuscular flat feet (polio, multiple sclerosis, cerebral palsy), as well as tarsal coalition and inflammatory arthropathy. However, the most common reasons for performing a triple arthrodesis have changed over time[3]:

1. Tibialis posterior insufficiency (30%)
2. Inflammatory arthropathy (19%)
3. Neuromuscular disorders (18%)
4. Post-traumatic arthritis (13%)
5. Primary osteoarthritis (9)
6. Congenital talipes equinovarus (7%)
7. Others (3%)

The subjective outcomes of triple arthrodesis (for all indications) are good – 75%, fair – 18% and poor 7.9%[3].

Technical Considerations

Approaches

While a double approach (medial and lateral) is traditionally used the pure medial approach has been described for triple fusion[4,5]. The reasoning behind this was concern regarding tension on the lateral would closure following correction.

Good outcomes with this technique have been described in 15/17 patients, with comparable or better corrections and no wound complications[4].
Using this approach in high-risk patients (e.g. Charcot feet), again showed no wound complication[5].

The anatomical considerations of the approach have also been studied[6], with the medial approach in the bed of tibialis posterior being found to be relatively safe. The neurovascular bundle is located 2cm below the middle facet of the STJ, and care is needed not to strip deep deltoid of the medial talus.

Which joints need Fusion?

The medial approach has also been used for double arthrodesis[7], where CCJ fusion is perhaps not necessary as it will be offloaded mechanically by distraction following correction of the collapsed foot shape. Indeed it is argued that correction could more powerful as the CCJ does not have the be closed. 78% had satisfied outcomes, 89% union rate and no wound complications.

What sort of Fixation?

All studies use large (5mm or more) cannulated screws for the STJ and TNJ, with one screw per joint being sufficient[8]. Staples or small plates are used for the CCJ.

Do we need Bone Graft?

Bone graft has been shown to not be required for triple fusion[8], with a large study showing 4% non-union rate, good outcomes in 75% of patients, fair in 20% and poor in 5%. Bone graft is therefore not routinely needed for double or triple arthrodesis.

Additional Procedures

There is a lot of heterogeneity included in the literature concerning triple fusion. There are many additional procedures that are commonly included as a part of the overall foot correction, as they are often necessary to achieve a good outcome. Some of these include:

- Achilles lengthening
- Gastrocnemius release
- Peroneal tendon release
- Deltoid reconstruction for valgus ankle
- NCJ or 1st TMTJ arthrodesis

Novel Techniques

- Arthroscopic triple fusion
- Lateral column lengthening arthrodesis with triple fusion
- Cuboid osteotomy with triple fusion - If unable to fully correct forefoot supination through the Chopart joints

Challenges and Controversies

- How to manage progressive valgus drift of the ankle?
  - Routine transfer of FDL?
  - Should we intervene in patients starting to drift but asymptomatic?
- What to do with tibialis posterior ⟶ Excise? Advance?
  - Does it have a role in an advanced flatfoot or is it simply a pain generator?

There is no evidence to suggest an answer to these questions.

Johnson & Strom Stage 4

Correctable stage 4 flat foot:

If there is reasonably well-preserved cartilage, doing a triple arthrodesis and a deltoid +/- spring ligament reconstruction is not unreasonable when trying to preserve the ankle joint.

Stiff stage 4 flat foot:

Total ankle replacement (TAR) following STJ, double or triple arthrodesis shows comparable pain and AOFAS at mid-term follow up when compared to TAR alone. However, the TAR above a fusion had more lysis around the implants[9].

Johnson & Strom Stage 2

The role of fusion in a flexible flat foot is not yet defined. However, this may be indicated in:

- High BMI patients
- Hypermobile patients
- Severe deformity
- As a revision procedure for failed MDCO and FDL transfer

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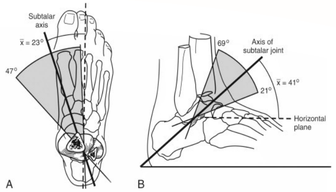
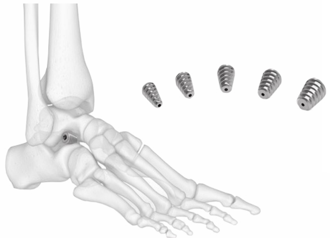
6.6. Why risk using an Arthroereisis Screw?

Shelain Patel



It is still unclear as to why some flat feet cause pain and others do not. It is also assumed that form follows function and vice versa. So, for painful flat feet, if we correct the shape, then the patients' pain should resolve. However, this is not always the case.

The STJ has a rotational moment moving in both axial and sagittal planes. This allows the calcaneum to sink into valgus and the talus to plantar flex, causing the talar head to become uncovered from its sling.



One of the aspects of flat foot correction should, therefore, be to correct subtalar rotation. To this end, an arthroereisis screw within the sinus tarsi aims to correct this subtalar rotation.

How is it commonly used?

Most literature on arthroereisis screws concerns use within paediatric orthopaedics. Arthroereisis screw +/- tendoachilles lengthening normalised every radiological parameter in paediatric flexible flat feet[1], except talonavicular coverage which was better but not normal.
Paediatric flat feet will not typically be grossly abducted at the TNJ, because the delto-spring ligaments will be intact. Therefore, an isolated arthroereisis screw theoretically could have a role in most paediatric flat feet. The paediatric and adult flat feet populations are very different.

Does it work in adults?

Within the adult population, arthroereisis screw is not usually used in isolation as it is in children. When used in combination with other procedures, arthroereisis screw corrected all but the talonavicular coverage angle in adults[2]. This suggests that if you want to use an arthroereisis screw, you should not use it to correct the abduction across the TNJ, but it will correct other parameters.

What are the Risks?

1. Sinus tarsi pain[3]
   a. 11.5% in isolated arthroereisis
   b. 30.3% in associated arthroereisis
2. Over-correction
3. Extrusion

Why use it?

As previously mentioned, the arthroereisis screw will correct all the flat foot parameters except forefoot abduction. Therefore, if your goal is to correct hindfoot alignment and you are concerned about doing a calcaneal osteotomy, then you can use an arthroereisis screw.

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Consensus Questions

1. Spring ligament damage is a more important aetiological factor than tibialis posterior disease
   a. Yes 18 (94.7%)
   b. No 1 (5.3%)
2. Deltoid ligament damage/disease has a role in PCFD
   a. Yes 19 **(100%)**
   b. No 0
3. Internal rotation of the talus has a role in PCFD
   a. Yes 19 **(100%)**
   b. No 0
4. A classification system describing the wide range of damaged structures in PCFD is more useful than the tibialis posterior dysfunction classification of Johnson and Strom
   a. Yes 9 (47.4%)
   b. No 10 (52.6%)
5. PCFD is mainly a clinical diagnosis, therefore, in the management of early PCFD, MRI is not mandatory.
   a. Yes 16 (84.2%)
   b. No 3 (15.8%)
6. In PCFD, an important role for MRI is…
   a. To define early degenerative change (i.e. stage 2 vs 3)
      i. Yes 10 (52.6%)
      ii. No 9 (47.4%)
   b. To diagnose a spring ligament tear
      i. Yes 4 (21.1%)
      ii. No 15 (78.9%)

c. To assess quality of tibialis posterior
   i. Yes 11 (57.9%)
   ii. No 8 (42.1%)
d. To assess quality of FDL
   i. Yes 7 (36.8%)
   ii. No 12 (63.2%)
e. To exclude other pathologies
   i. Yes 18 (94.7%)
   ii. No 1 (5.3%)

7. For a patient, who has tibialis posterior tenosynovitis (Johnson & Strom stage 1) and inflammatory arthropathy has been excluded, a steroid injection (ultrasound/clinically guided) is useful.
   a. Always 0
   b. Sometimes 8 (44.4%)
   c. Never 10 (55.6%)

8. In a patient with advanced PCFD, I perform lateral column lengthening.
   a. Frequently 0
   b. Sometimes 12 (63.2%)
   c. Never 7 (36.8%)

9. Lateral column lengthening is a difficult surgical procedure which has a role in PCFD when there is TNJ abduction
   a. Yes 12 (60%)
   b. No 8 (40%)

10. For a morbidly obese patient (BMI >35) with PCFD without joint changes (Johnson & Strom stage 2), fusion surgery should be considered earlier than in a similar patient with a normal BMI.
   a. Always 19 **(100%)**
   b. Sometimes 0

11. In a patient with advanced PCFD and severe hindfoot valgus, a double fusion is a better procedure than a triple fusion.
   a. Always 5 (27.8%)
   b. Sometimes 13 (72.2%)
   c. Never 0

12. When deciding whether to transition from non-operative to surgical management, the most important factor is the patient's opinion.
   a. Yes 17 (89.5%)
   b. No 2 (10.5%)

Notes

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