OrthoSolutions Group

BONESUPPORT™

Consensus of the 9th Round Table

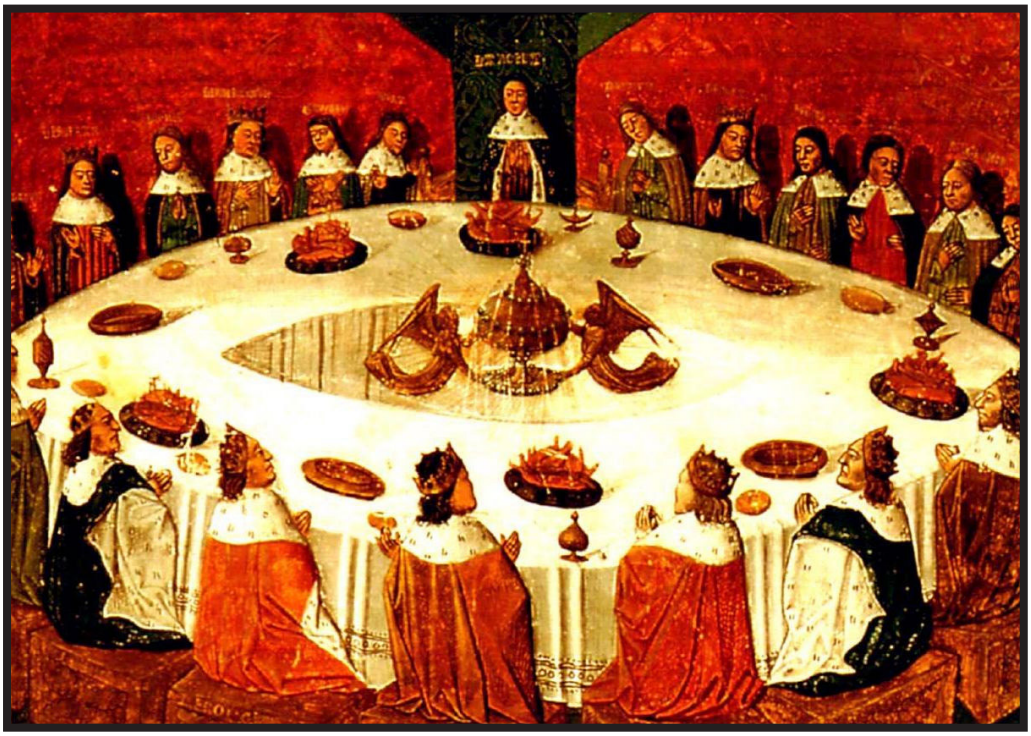
Bone Disorders of the Foot & Ankle

Kraków, September 2019

Karan Malhotra *FRCS (Tr & Orth)*

Shelain Patel *FRCS (Tr & Orth)*

Dishan Singh *FRCS (Orth)*



Aspects of Foot & Ankle Surgery

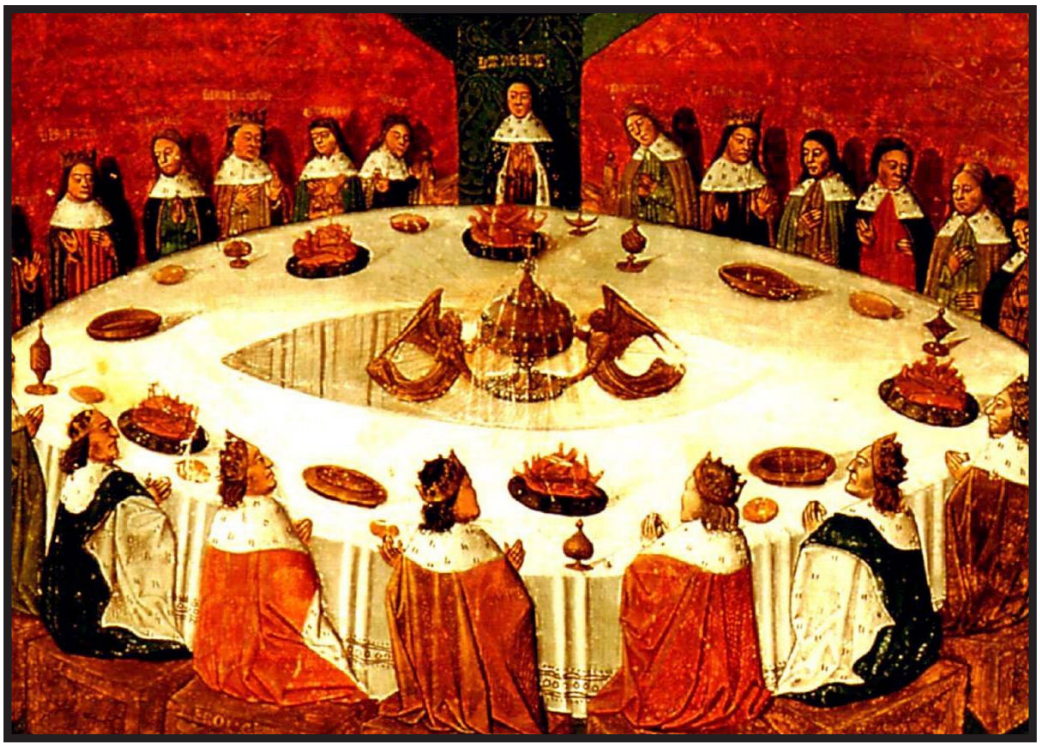
Bone Disorders of the Foot & Ankle

Consensus of the 9th Round Table

Kraków, 27th to 28th September 2019

Course Convener:

Dishan Singh



Hosted by:

OrthoSolutions Group & Bone Support

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 and Europe. Though eminence rather than true evidenced based medicine, this represents the concepts of over 200 years of combined experience. 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

When I designed the concept of the Round Table in foot and ankle surgery in 2011, it was based on an informal get together of senior foot and ankle surgeons to discuss aspects of foot and ankle surgery in an informal setting. The 9th Round Table in Kraków has once again not followed the format of a usual orthopaedic meeting, where faculty members lecture to delegates. As always, the meeting is unique in that all participants have an equal input to review the literature and present their individual experience on a topic - with ample time for an informal discussion of the subject in a relaxed setting. We then attempt, where possible, to reach a consensus to guide us and readers of this document on various aspects of management.

This year, I have selected topics dealing with bone disorders of the foot and ankle and the debate was indeed stimulating. Karan Malhotra and Shelain Patel were responsible for recording opinions and capturing the essence of the debates. Their valuable hard work is greatly appreciated, and 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 in order to keep the booklet small and easily readable.

I would like to thank our colleagues from Kraków (Artur Gądek, Henryk Liszka and Piotr Chomicki-Bindas) who contributed generously to both the organisation and the content of the meeting. We are also grateful to OrthoSolutions and Bone Support who provided an educational grant for the meeting. On a personal note, I thoroughly enjoyed organising the meeting and would like to express my sincere gratitude to Jo Millard for her invaluable input and support.

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

Dishan Singh ***MBChB, FRCS (Orth)***
Consultant Orthopaedic Surgeon
Royal National Orthopaedic Hospital
Stanmore, United Kingdom

October 2019

Summary of Sessions

Session 1 : Basic Sciences **Chair: Dishan Singh**

Topic	Speaker	Page
1.1 - Bone biology	Raman Dega	06
1.2 - Bone biomechanics	Tim Williams	08
1.3 - Osteoporosis, Calcium metabolism & Vitamin D	Rick Brown	10
1.4 - Bone healing	Rob Clayton	12
Discussion & Consensus	-	13

Session 2 : Factors Affecting Bone Healing **Chair: Nick Cullen**

Topic	Speaker	Page
2.1 - Evidence of role of smoking	Matt Welck	14
2.2 - Should fixation be completely rigid	Matt Welck	16
2.3 - Role of biologics to promote bone healing	Callum Clark	18
2.4 - Evidence for bone stimulation	Tim Clough	20
Discussion & Consensus	-	22

Session 3 : Managing Bone Loss / Bone Defects **Chair: Callum Clark**

Topic	Speaker	Page
3.1 - Which autograft is better?	Steve Hepple	24
3.2 - Allografts	Phil Vaughan	26
3.3 - Bone substitutes	Billy Jowett	28
3.4 - Metal cages & solid metal	Mike Karski	30
Discussion & Consensus	-	32

Session 4 : Miscellaneous **Chair: Patricia Allen**

Topic	Speaker	Page
4.1 - Stress fractures	Andy Goldberg	34
4.2 - Benign tumours	Ben Rudge	36
4.3 - Malignant tumours	Nick Cullen	38
4.4 - Avascular Necrosis	Henryk Liszka	40
4.5 - Bone dysplasias	Maneesh Bhatia	42
Discussion & Consensus	-	44

Session 5 : Non-Diabetic Foot Infection **Chair: Tim Clough**

Topic	Speaker	Page
5.1 - Osteomyelitis: when to debride / when to give antibiotics	Patricia Allen	46
5.2 - Unusual causes of bone infection	Piotr Chomicki-Bindas	48
5.3 - Implant related infection	Senthil Kumar	49
5.4 - Local *versus* systemic antibiotics	Anand Pillai	50
Discussion & Consensus	-	52

Session 6 : Diabetic Foot **Chair: Anand Pillai**

Topic	Speaker	Page
6.1 - When to operate on a Charcot foot	Ashok Acharya	54
6.2 - Role of debridement / preservation in foot ulcers	Krishna Vemulapalli	56
6.3 - Role of minimally invasive surgery in diabetic feet	Artur Gądek	58
6.4 - Foot attack	Dishan Singh	60
6.5 - Amputations	James Ritchie	62
Discussion & Consensus	-	64

Session 1: Basic Sciences

1.1 - Bone biology *(Raman Dega)*

Bones are living tissues with the ability for regeneration and self-repair. They interface well with inert materials and metal. Their basic functions include structural support, protection of vital organs, storage of minerals, and production of blood products in the marrow. Bone growth is through either intramembranous ossification (bone replaces connective tissue membranes), or endochondral ossification (bone replaces hyaline cartilage). Bone is composed of ***osteoblasts***, ***osteoclasts***, ***osteocytes***, ***bone matrix (osteoid)***, and ***hydroxyapatite***.

Osteoblasts comprise 4-6% of bone cells. They are derived from mesenchymal cells and their differentiation is controlled via various gene expression pathways including the Winless pathway, Runt related transcription factors and fibroblast growth factors. Once mature, osteoblasts synthesise and secrete organic bone matrix, osteocalcin, type I collagen and proteoglycans. Mineralisation occurs in 2 phases, the ***vesicular*** and ***fibrillar*** phases.

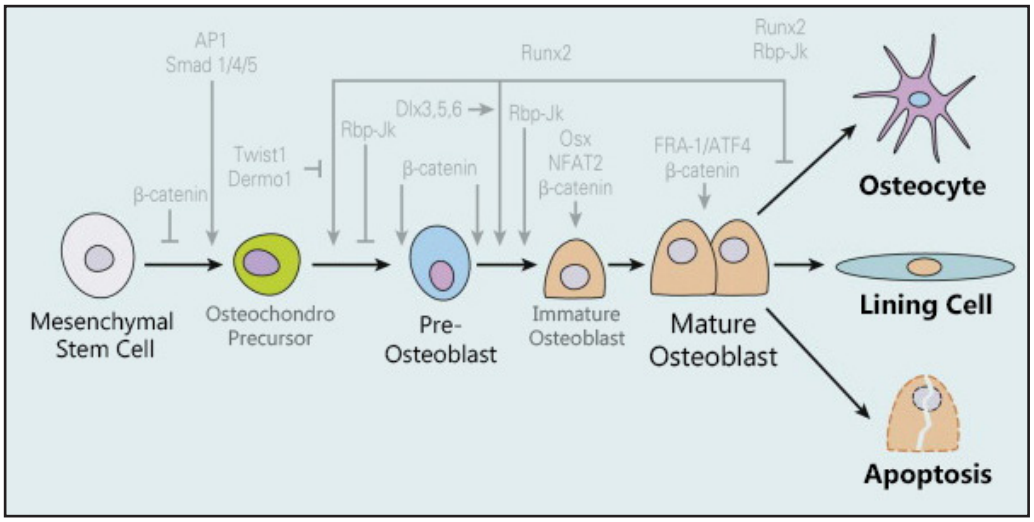
Vesicular phase:

- Matrix vesicles are release from osteoblasts
- Bind to proteoglycans in the matrix
- Negatively charged, bind / absorb Ca^{2+} ions
- PO_4^{3-} released via alkaline phosphatase and also absorbed

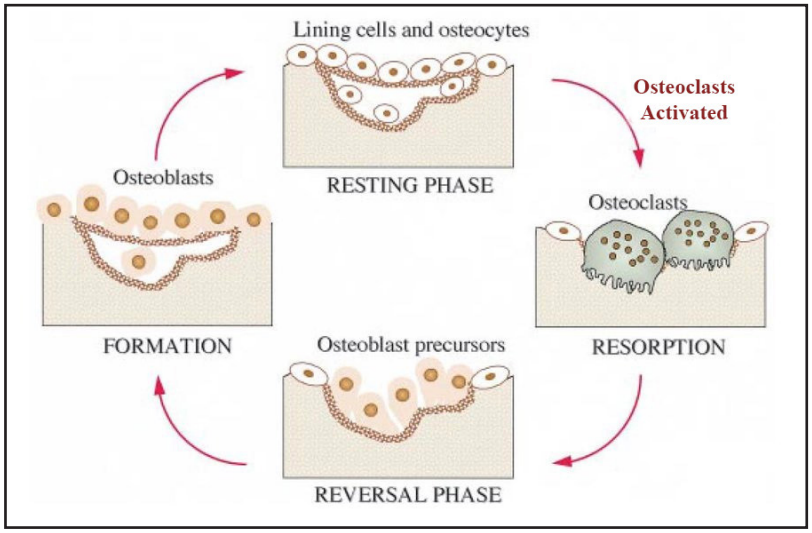
Fibrillar phase:

- Ca^{2+} and PO_4^{3-} ions inside the vesicles bind
- Create nuclei of hydroxyapatite crystals
- Grow and supersaturate
- Vesicles rupture and crystals deposit in collagen matrix

Osteoblasts either undergo apoptosis, form bone lining cells (quiescent, flat form), or form osteocytes. ***Osteocytes*** comprise 90-95% of bone cells and have a life span of up to 25 years. They play a vital role in orchestrating the activity of osteoblasts and osteoclasts. They sit within the lacunae of bone and communicate with adjacent osteocytes via cytoplasmic processes; this is the ***lacunar-canalicular system***. They serve as mechanoreceptors – loading the bone is surmised to deform cilia, protein complexes and the cytoskeleton of the osteocytes which in turn produce biochemical signals. They also respond to fluid flow during bone loading, opening ion channels and creating hyperpolarisation or depolarisation; this has traditionally been thought of as the ***piezoelectric effect***. Through these pathways, loading results in a net anabolic effect and lack of loading decreases anabolic activity and inhibits osteoblastic activity.



Osteoclasts are derived from a different cell line – the mononuclear line. Their differentiation and activation are controlled by RANK-Ligand, secreted by osteoblasts and osteocytes. During their activation there are 4 types of membrane domains – the ***sealing zone*** and the ***ruffled border*** contact the bone matrix, and the ***basolateral*** and ***functional secretory domains*** do not contact the matrix.



Bone formation and maintenance is through ***bone modelling*** and ***bone remodelling***. Modelling occurs from birth adulthood and results in new bone formation and bone growth. Bone remodelling is replacement of old bone tissue with new. Remodelling occurs via a ***basic multicellular unit (BMU)*** and undergoes several stages: *activation, resorption, reversal, formation and quiescence (resting phase)*. This is a highly complex cycle which is mediated by local and systemic factors including sex hormones and immunological mediators.

	Bone Modelling	**Bone Remodelling**
Arrangement of Osteoblasts / Osteoclasts	Different Surfaces	Same Surface
Activity	Continuous	Cyclical
Effect on bone mass	Fast	Slow

Summary:

- Bone is a living tissue with complex interactions between its various components
- A fine balance is needed for adequate repair and maintenence of bone health

References:

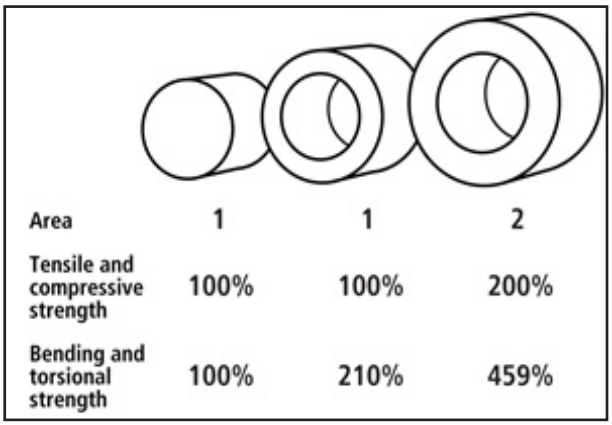
International Osteoporosis Foundation. **Introduction to Bone Biology: All About Our Bones.** *https://www.iof-bonehealth.org/introduction-bone-biology-all-about-our-bones (date last accessed 29/09/2019).*

Florencio-Silva R, Sasso GR, Sasso-Cerri E, *et al.* **Biology of Bone Tissue: Structure, Function, and Factors That Influence Bone Cells.** *Biomed Res Int 2015;2015:421746.*

Gusmao CV, Belangero WD. **How Do Bone Cells Sense Mechanical Loading?** *Rev Bras Ortop 2009;44-4:299-305*

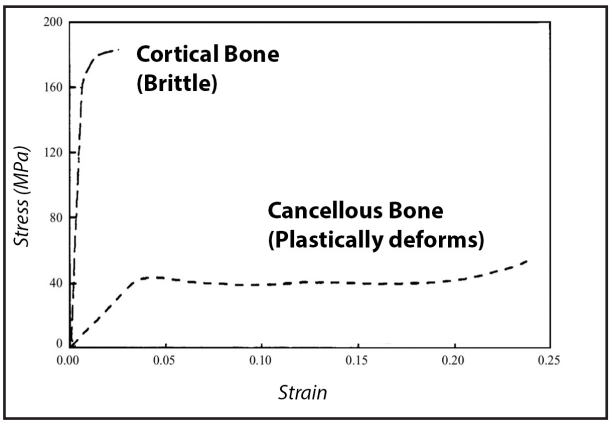
1.2 - Bone biomechanics *(Tim Williams)*

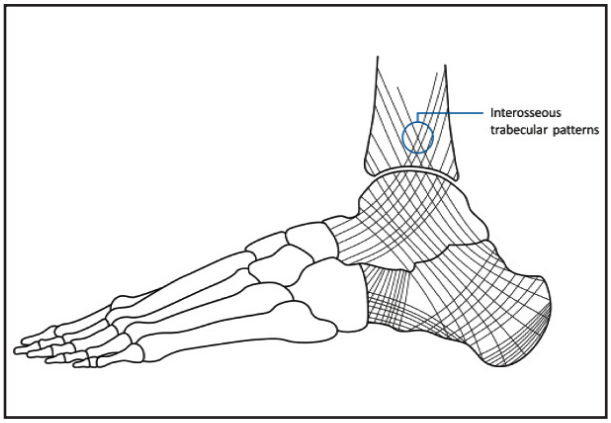
Bone is a bio-composite material which is strong under compression and tension, but weaker in torsion. ***Cortical bone*** is lightweight and tough but brittle. In long bones this is achieved by peripheralising the mass, the triangular cross section, and layers of collagen fibres orientated in different directions.



Area	1	1	2
Tensile and compressive strength	100%	100%	200%
Bending and torsional strength	100%	210%	459%

Trabecular bone has a lower yield to failure but can plastically deform prior to fracturing. It exhibits ***anisotropy*** (properties dependent on the direction of loading) and ***viscoelasticity*** (stress-strain characteristics are dependent on rate of applied strain). The mechanical role this plays is not fully understood, but trabecular bone becomes stiffer in compression and the faster it is loaded.





The ***poroelastic theory*** explains the rate dependent change in mechanical properties of bone by likening it to a sponge. On loading the fluid component of the matrix is forced through the various bony channels, but the rate at which this can occur is limited. At higher rates of load the rate of compression exceeds the rate of flow, which increases the internal pressure of the matrix and resists further load.

Viscoelastic properties of bone:

» ***Creep*** is the ability of a material to continually deform under the influence of a constant stress
» ***Stress Relaxation*** is decreased stress in response to constant strain

A***nisotropy*** is a result of trabecular tension and compression lines. These represent areas of increased bone density as remodelled by Wolff's law. Wolff stated that *'every change in the form and function of bones, or of their function alone is followed by certain definite changes in their internal architecture and equally definite secondary alteration in their external conformation, in accordance with mathematical law.'*

The ***internal architecture*** represents the trabecular pattern, and the ***external architecture*** represents cortical bone thickness. As discussed in *Section 1.1*, bone will remodel and increase or decrease its density in response to load, or lack thereof. Areas with reduced loading due to inactivity or, in the case of an implant, stress shielding, will therefore become susceptible to osteopenia, fatigue and fracture.

Summary:

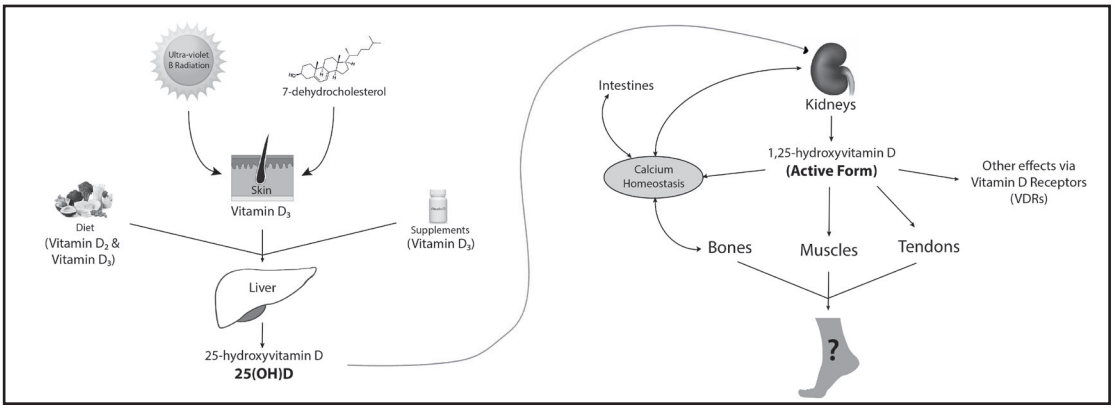
- Bone is lightweight, strong, and exhibits viscoelastic properties
- It remodels in accordance with loads placed on it (Wolff's law)

References:

Cowin SC. **Bone poroelasticity.** *J Biomech* *1999;32-3:217-38.*

1.3 - Osteoporosis, Calcium metabolism & Vitamin D *(Rick Brown)*

Vitamin D plays a vital role in calcium homeostasis, increasing intestinal, bony and renal absorption. Sunlight is an important source and lack of exposure to ultraviolet light at higher latitudes, particularly in the winter months, can result in deficiency. Other risk factors for low Vitamin D levels include clothing and occupations which minimise sunlight exposure, and poor diet.



The evidence for the role of ***Vitamin D deficiency*** in foot and ankle pathology is far from concrete. Smith *et al.* found that 40% of patients with foot and ankle injuries had a low Vitamin D level and that Vitamin D levels tended to be lower in those with a fracture than an ankle sprain. Givon *et al.* found that Vitamin D levels were lower in soldiers with stress fractures than in those without, although majority of the soldiers had deficiency and there was no population control to compare with. Other military studies have reported similar findings. Boszczyk *et al.* found that Vitamin D deficiency was high in patients with delayed union of foot and ankle fractures, but that similar levels of deficiency were also seen in patients without non-union.

Vitamin D supplementation has been shown to improve bone healing in animal models, but in humans most studies have included calcium co-supplementation which makes interpretation of its effect difficult. A randomised control trial demonstrated increased fracture callus formation in proximal humeral fractures when patients were given both Vitamin D and calcium. Other studies have shown that Vitamin D and Calcium supplementation reduced the risk of stress fractures by 20% in naval recruits. A meta-analysis of 68,000 patients suggested that Vitamin D supplementation alone was not effective at preventing fractures but did become effective when calcium was co-supplemented. However, most people in the UK have a calcium within the normal range and may not need the calcium supplementation.

Vitamin D supplementation recommendations from Endocrine Society & NICE:

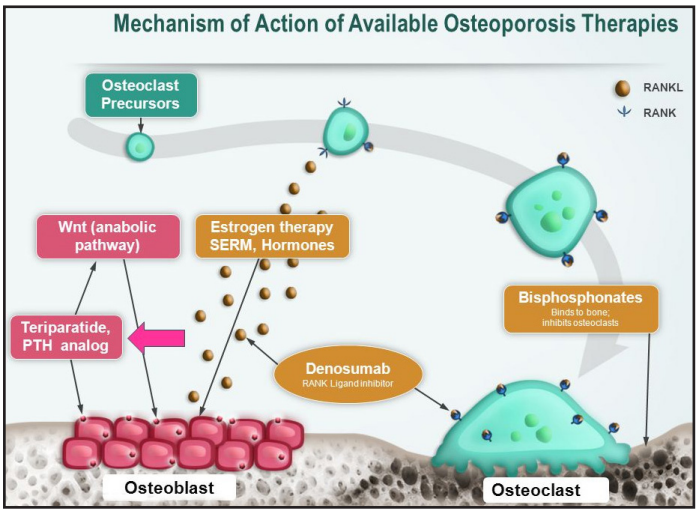
» ***Supplementation:*** adults with deficiency of Vitamin D (< 20 ng/ml) receive 50,000 IU / week for 8 weeks and then 1,500-2,000 IU / day thereafter

» ***Maintenence:*** to maintain Vitamin D levels, 600 IU (< 70 years) to 800 IU (> 70 years) total daily intake (from all sources) is sufficient. High risk groups may require oral supplementation of 400 IU / day to achieve this

Osteoporosis is defined by the WHO as a bone density < 2.5 standard deviations below the mean peak bone density for individuals of the same race and gender. ***Primary osteoporosis*** is the more common type, is hormone related, and consists of Type 1 and Type 2. ***Type 1*** is post-menopausal and seen in women between the ages of 50 and 70. ***Type 2*** is senile osteoporosis and affects both men and women over the age of 70. ***Secondary osteoporosis*** is seen in both men and women and is related to other

causes including drugs, endocrine disorders, marrow disorder, malignancy, inflammatory conditions and poor nutrition.

Treatment involves lifestyle modifications, Vitamin D and calcium supplements, treatment of any underlying pathology and exercise to maintain controlled loading of the musculoskeletal system. Pharmacological treatment includes bisphosphonates, oestrogens, and other newer agents which either inhibit osteoclast activity (e.g. Denosumab), or increase osteoblastic activity (e.g. Teriparatide). These monoclonal antibodies may prove in the future to have a role in the management of patients with delayed bone union.



Summary:

- Vitamin D level are often low in patients with foot and ankle fractures and stress fractures
- Prophylactic Vitamin D and calcium *may* reduce stress fracture risk, but there is insufficient evidence regarding its routine use in other aspects of foot and ankle surgery
- Daily supplementation of 400 IU / day may be sufficient to prevent deficiency, but higher doses are required to treat deficiency, with or without calcium
- There is unclear benefit or testing at-risk patients *versus* supplementation

References:

Boonen S, Lips P, Bouillon R, *et al.* **Need for additional calcium to reduce the risk of hip fracture with vitamin d supplementation: evidence from a comparative metaanalysis of randomized controlled trials.** *J Clin Endocrinol Metab* *2007;92-4:1415-23.*

Boszczyk AM, Zakrzewski P, Pomianowski S. **Vitamin D concentration in patients with normal and impaired bone union.** *Pol Orthop Traumatol* *2013;78:1-3.*

DIPART Group. **Patient level pooled analysis of 68 500 patients from seven major vitamin D fracture trials in US and Europe.** *BMJ* *2010;340:b5463.*

Givon U, Friedman E, Reiner A, *et al.* **Stress fractures in the Israeli defense forces from 1995 to 1996.** *Clin Orthop Relat Res* *2000-373:227-32.*

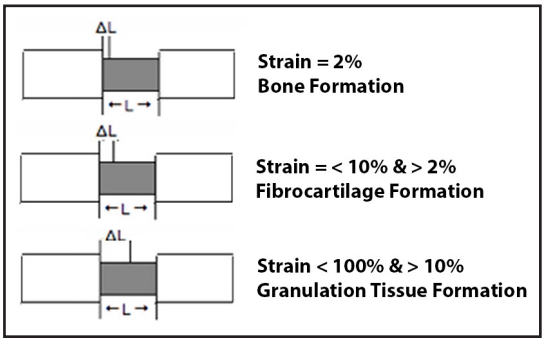
Ruohola JP, Laaksi I, Ylikomi T, *et al.* **Association between serum 25(OH)D concentrations and bone stress fractures in Finnish young men.** *J Bone Miner Res* *2006;21-9:1483-8.*

Smith JT, Halim K, Palms DA, *et al.* **Prevalence of vitamin D deficiency in patients with foot and ankle injuries.** *Foot Ankle Int* *2014;35-1:8-13.*

1.4 - Bone healing *(Rob Clayton)*

Lamellar bone comprises most normal adult bone and consists of collagen fibres arranged in parallel layers. It may be cortical or cancellous. In ***cancellous bone*** the trabecular patterns of bony struts are orientated in the direction of greatest stress as described in *Section 1.2*. ***Woven bone***, in adults, is seen main at fracture sites during healing, or in areas of pathological conditions. It consists of randomly oriented collagen fibres with reduced mechanical strength. Surrounding the bone is the vascular periosteum, from whence osteoblasts migrate during bony repair. The prerequisites for bone healing are adequate blood supply and adequate mechanical stability. Healing can be either direct (primary) or indirect (secondary).

Direct bone healing requires *compression* and *absolute stability*. It does not result in callus formation. ***Contact healing*** occurs when there is direct contact between the cortical bone on either side of a fracture. The gap must be < 10 μm and the interfragmentary strain < 2%. Under these conditions cutting cones forms and cross the fracture gap. The Haversian system is restored at the same time as bony healing. ***Gap healing*** occurs when there is a slightly larger gap between fragments ~500 μm, although rigid fixation is still required. Here, the gaps are first filled with woven bone which undergoes remodelling to lamellar bone by cutting cones, *i.e.* union and remodelling of the Haversian system do not occur simultaneously.



Other factors impacting bone healing:

» ***Local:*** soft tissue injury, devascularisation, soft tissue interposition, bony necrosis

» ***Systemic:*** malnutrition, smoking, medications which modify the immune system or inflammatory cascade, systemic illness

Indirect bone healing requires relative stability and healing occurs by callus. Initial healing is relatively rapid, but significant remodelling must later occur. The ***inflammation phase*** lasts for a week and is mediated by a variety of inflammatory and growth factors. During this time a haematoma forms, vascular ingrowth occurs, and necrotic bone ends are resorbed by osteoclasts. The ***repair phase*** occurs as first periosteal callus, then bridging soft callus and finally intramedullary callus forms. This is replaced by woven bone which forms a hard callus, and ultimately this is remodelled. During the ***remodelling phase*** the normal lamellar system is formed, and the medullary cavity reconstitutes.

Summary:

- Bone healing proceeds according to the strain in the local environment and the vascularity
- This can be influenced by fixation and soft tissue management

References:

Albright JA, Brand RA. *ed.* **The Scientific Basis of Orthopaedics. (2nd ed.)** *Appleton and Lange, 1987.*

Ramachandran M, *ed.* **Basic Orthopaedic Sciences: The Stanmore Guide.** *Edward Arnold (Publishers) Ltd, 2007.*

Session 1 - Discussion & Consensus

Vitamin D:

» **How many surgeons routinely *measure* Vitamin D levels in patients prior to *primary* fusion surgery?**

Yes	0	(0%)
No	25	(100%)

» **How many surgeons routinely *measure* Vitamin D levels in patients prior to *revision* fusion surgery?**

Yes	9	(36%)
No	16	(64%)

» **How many surgeons routinely *give* Vitamin D levels in patients prior to *revision* fusion surgery?**

Yes	6	(24%)
No	19	(76%)

» **Of those surgeons advising supplementation of Vitamin D, how many also advise co-supplementation of Calcium?**

Yes	1	(17%)
No	5	(83%)

It was acknowledged that the benefit of routine supplementation of Vitamin D in foot and ankle patients is unclear. However, most surgeons felt it was appropriate to test for, or presumptively treat, Vitamin D deficiency prior to revision fusion surgery. It was noted that the conclusions of the study by the DIPART group published in the BMJ (see references of *Section 1.3*) was considered controversial. It was generally agreed that it is not currently clear whether calcium co-administration is required.

Fracture Union:

» **When assessing bony union using plain radiographs, is it acceptable to diagnose union on the basis of 3 out of 4 cortices (on the AP and lateral) appearing to have united?**

Yes	16	(76%)
No	9	(9%)

Although plain radiographs are often used for assessing fusion, it was generally agreed that the clinical picture was of greater importance and that if there was concern regarding non-union, a CT scan is preferred. It was also generally agreed that in the case of, for example, an ankle fusion, between 30% and 50% fusion on a CT scan may be sufficient evidence of union if the patient was asymptomatic, although progression across serial scans is a better predictor of union than a single scan.

Session 2: Factors Affecting Bone Healing

2.1 - Evidence of role of smoking *(Matt Welck)*

Smoking amongst patients is common; in the UK, 14.7% of adults smoke cigarettes and 7.1% vape electronic cigarettes. Although the number of cigarette smokers is reducing, the number of people who smoke e-cigarettes is rising; 31% of vapers are former smokers who used the products to quit cigarettes. A survey of BOFAS members in 2006 found 99% of surgeons were aware of the negative impact of smoking on foot and ankle surgery but only 9% specifically documented this on the consent form. Bettin *et al.* found less than 44% of smokers are aware of the effect it has on wound / bone healing.

Cigarette smoke contains at least 4,000 compounds, including gaseous, volatile, and non-volatile particle compounds. Inhaling smoke can be split into a ***volatile phase*** of approximately 500 gases including *Carbon monoxide*, *Ammonia*, *Hydrogen cyanide* and *Benzene*, and a ***particulate phase*** containing approximately 3,500 chemicals including *nicotine*; 70 of these are ***carcinogenic***.

Acute effects of smoking:

- ***Nicotine*** decreases prostacyclin production and increases platelet adhesion, leading to vasoconstriction and formation of microvascular clots
- ***Carbon Monoxide*** preferentially binds to haemoglobin, preventing Oxygen binding
- ***Pro-inflammatory mediators*** are released which promote inflammation and potentiate pain pathways

There is strong evidence that ***smoking negatively impacts orthopaedic surgery***. A recent systematic review (Al-Bashaireh *et al.*) found smoking was associated with *low bone mineral density*, *delayed union* of fractures, *periprosthetic osteolysis* and *implant failure*. This is also true of foot and ankle surgery: Krannitz *et al.* found bony union after hallux valgus correction took 69 days, 120 days, and 78 days in non-smokers, smokers, and second-hand smokers respectively. In a cohort of 160 subtalar, talonavicular, calcaneocuboid, and double / triple arthrodeses, smokers were 2.7 times less likely to unite (Ishikawa *et al.*).

Chronic effects of smoking:

- ***Reduced bone mineral density*** via lower expression on BMPs and bone marrow progenitor cells
- ***Small airway collapse and ciliary inhibition*** which reduces gas exchange
- ***Atherosclerosis and peripheral artery disease*** which reduces Oxygen availability
- ***Reactive Oxygen species*** inhibit immune cells and fibroblasts, leading to wound dehiscence
- ***Reduced thickness and strain ratio of tendons*** which become thicker and harder

Smokers are 3.3 times more likely to develop ***wound complications*** after ankle ORIF than non-smokers (Saleh *et al.*), with higher rates of wound complications after calcaneal fracture ORIF (Soni *et al.*) and total ankle arthroplasty (Lampley *et al.*). Furthermore, patients who smoke may also ***experience more pain*** and report ***lower patient satisfaction scores*** after foot and ankle surgery (Beahrs *et al.*).

Smoking cessation reduces acute inflammation which improves peripheral blood flow, and within 8 hours Carbon monoxide levels in the blood stream normalise. However, it takes several weeks for respiratory cilia to reactivate and regrow, and up to 10 years for bone mineral density to improve. The evidence ***supports encouraging smoking cessation prior to surgery***. In a series of 602 patients undergoing forefoot surgery, complication rates for currently, previously and never-smoking patients were 36.4%, 16.5% and 8.5% respectively (Bettin *et al.*). Each additional week of cessation reduces the

relative risk of general surgical complications by 19% (Mills *et al.*) and smoking cessation 4 weeks before and 4 weeks after surgery reduces perioperative complications by 20% (Lindström *et al.*).

E-cigarettes provide a vaporised form of nicotine delivery and contain less particulate and volatile chemicals than cigarettes. Theoretically, it is better than smoking in terms of risk of cancer and heart disease, but the ***effect on surgical complications is less clear*** as it does still *alter DNA sequences*, *lead to endothelial toxicity*, *reduce cutaneous blood flow* and it is associated with *higher wound complication rates* than non-smokers/non-vapers. The effect on bone healing is not known.

Cotinine is a metabolite of nicotine which can be measured on urine dip-stick testing. It normalises two weeks after smoking cessation, so is of value when establishing whether patients are still smoking. However, 10% of patients questioned in a recent survey tested positive despite claiming to have stopped and it is unknown if the problem lay with the test or patient (Salandy *et al.*).

Summary:

- Cigarettes and vapes should be stopped for as long as possible before and after surgery
- A minimum of 4 weeks is recommended for low risk surgeries
- Adjuncts which may improve outcomes in smokers include increasing plaster immobilisation time, nutritional supplementation, avoiding tourniquets, and consideration of post-operative anticoagulation to reduce micro-angiopathic events

References:

Al-Bashaireh AM, Haddad LG, Weaver M, *et al.* **The Effect of Tobacco Smoking on Musculoskeletal Health: A Systematic Review.** *J Environ Public Health* *2018;2018:4184190.*

Beahrs TR, Reagan J, Bettin CC, *et al.* **Smoking Effects in Foot and Ankle Surgery: An Evidence-Based Review.** *Foot Ankle Int* *2019;40-10:1226-32.*

Bettin CC, Gower K, McCormick K, *et al.* **Cigarette smoking increases complication rate in forefoot surgery.** *Foot Ankle Int* *2015;36-5:488-93.*

Ishikawa SN, Murphy GA, Richardson EG. **The effect of cigarette smoking on hindfoot fusions.** *Foot Ankle Int* *2002;23-11:996-8.*

Krannitz KW, Fong HW, Fallat LM, *et al.* **The effect of cigarette smoking on radiographic bone healing after elective foot surgery.** *J Foot Ankle Surg* *2009;48-5:525-7.*

Lampley A, Gross CE, Green CL, *et al.* **Association of Cigarette Use and Complication Rates and Outcomes Following Total Ankle Arthroplasty** *Foot Ankle Int* *2016;37-10:1052-9.*

Lindström D, Sadr Azodi O, Wladis A, *et al.* **Effects of a perioperative smoking cessation intervention on postoperative complications: a randomized trial.** *Ann Surg* *2008;248-5:739-45.*

Mills E, Eyawo O, Lockhart I, *et al.* **Smoking cessation reduces postoperative complications: a systematic review and meta-analysis.** *Am J Med* *2011;124-2:144-54 e8.*

Saleh H, Konda S, Driesman A, *et al.* **Wound-Healing Issues Following Rotational Ankle Fracture Surgery: Predictors and Local Management Options.** *Foot Ankle Spec* *2018:1938640018810422.*

Salandy A, Malhotra K, Goldberg A, *et al.* **Can a urine dipstick test be used to assess smoking status in patients undergoing planned orthopaedic surgery? a prospective cohort study.** *Bone Joint J* *2016;98-10:1418-24.*

Soni A, Vollans S, Malhotra K, *et al.* **Association Between Smoking and Wound Infection Rates Following Calcaneal Fracture Fixation.** *Foot Ankle Spec* *2014;7-4:266-70.*

2.2 - Should fixation be completely rigid? *(Matt Welck)*

Bone healing requires a favourable mechanical and biological environment. The mechanical environment is determined by the stability conferred upon a construct which in turn dictates strain. ***Strain*** is the change in gap (fracture or fusion) divided by the size of gap. According to ***Perren's strain theory***, the ***strain percentage*** dictates which tissue is formed between the ends of bones, and this is influenced by stability. This is discussed in *Session 1.4*.

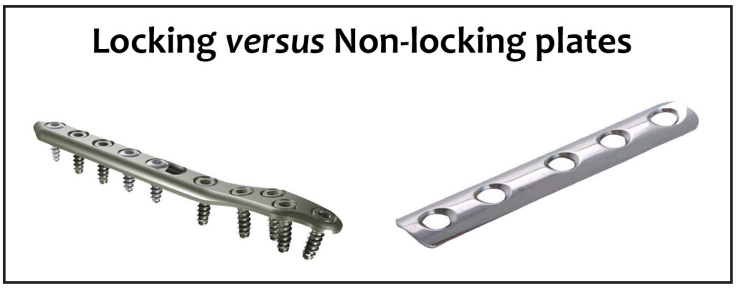
Types of Stability:

» ***Absolute stability:*** no motion occurs at fracture site under physiological load. This requires anatomical reduction and compression for bone to heal which can be achieved with *lag screws*, *compression plates*, *tension band wiring*, etc. If this type of stability is used and the bone ends are not apposed, then atrophic non-union will result

» ***Relative stability:*** micromotion occurs at the fracture or arthrodesis site under physiological load which results in callus formation. This can be achieved using *casts*, *external fixators*, *bridge plating*, *IM nailing*, *wires* etc. If this type of stability is used but a high strain environment persists, then hypertrophic union will occur.

» ***'Absolutive' stability:*** This novel term was coined by Kojima and Pires and implies rigid fixation without compression as may be employed with locking plates of the distal tibia, distal radius, midfoot etc. Bones would still be expected to heal without callus due to rigidity of fixation, so long as there is good bony contact.

The past 15 years has seen a dramatic increase in the use of ***locking plates*** in foot and ankle surgery. This is driven by a need for more rigidity in smaller bones where bone quality may be impaired e.g. osteoporosis, rheumatoid arthritis. However, the *number and types of screws used*, and *their position relative to the fracture* or union site will determine whether absolute or relative stability is conferred. Unless compression is achieved, locking plates should be used to confer relative stability which can be achieved by using non-locking screws where possible, increasing the working length by skipping holes over areas of instability and aiming for a ***screw to hole ratio of 0.4 - 0.5 per fragment***.



In ***fibular fractures***, no significant difference has been found in biomechanical strength of fixation between ***locking and nonlocking one-third tubular plates***, and ***locking and nonlocking pre-contoured periarticular plates*** using osteoporotic Weber B fracture models. A retrospective review of 145 ankle fractures treated with either semi-tubular plate, low contact dynamic compression plates and pre-countered distal fibula locking plates found no significant difference between these plates for complications or reoperation (Lyle *et al.*).

Similarly, several biomechanical papers have compared ***locking and nonlocking constructs*** in an osteoporotic Sanders type IIB ***calcaneal fracture*** model and found locking plates conferred no mechanical advantage in resisting torque or in the number of cycles to failure.

However, in ligamentous and fracture associated ***Lisfranc injuries***, better short to medium term clinical outcomes with lower reoperation rates are reported with bridging locking plates than with traditional trans-articular screw constructs.

In the setting of ***first MTP joint arthrodesis***, locking plates confer greater stiffness in load-to-failure testing than either compression screws alone or non-locked plated. Using locking plates in isolation confers a trend toward increased non- union rates whilst dorsal locking plate in combination with a lag screw has union rates of up to 98%, although this figure does not differ significantly from that reported with 2 cancellous cross screws.

Conflicting biomechanical reports exist when comparing ***locking plate constructs against intramedullary nails*** in the setting of ***tibio-talar-calcaneal arthrodesis***. O'Neill *et al.* reported stiffness was significantly lower in the locking plate group whilst Ohlson *et al.* reported no difference in *initial or final stiffness*, *load to failure*, or *construct deformation*. Variability between plate designs and nail designs is likely to be important in these results.

Summary:

- Fixation need not always be rigid since sometimes anatomical reduction is not required
- In these cases constructs that provide relative stability are sufficient
- Surgeons should identify the type of stability required and use appropriate reduction techniques with the correct hardware (used in an appropriate manner to achieve the desired goal)
- There is good evidence to support locking plate technology in Lisfranc injuries where joint sparing surgery is performed but universal adoption of locking plates in other foot and ankle conditions is not supported by the current evidence base

References:

Kojima KE, Pires RES. **Absolute and relative stabilities for fracture fixation: the concept revisited.** *Injury 2017;48 Suppl 4:S1.*

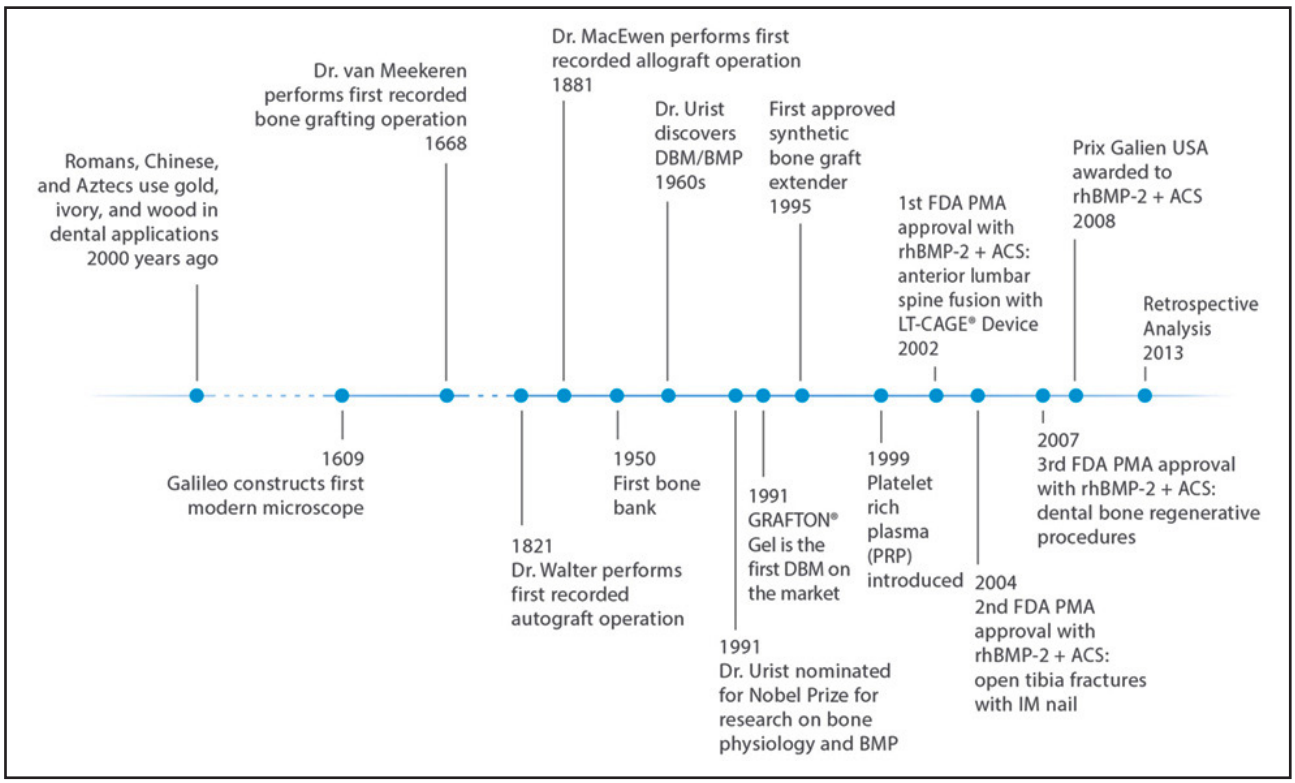
Lyle SA, Malik C, Oddy MJ. **Comparison of Locking Versus Nonlocking Plates for Distal Fibula Fractures.** *J Foot Ankle Surg 2018;57-4:664-7.*

O'Neill PJ, Logel KJ, Parks BG, *et al.* **Rigidity comparison of locking plate and intramedullary fixation for tibiotalocalcaneal arthrodesis** *Foot Ankle Int 2008;29-6:581-6.*

Ohlson BL, Shatby MW, Parks BG, *et al.* **Periarticular locking plate vs intramedullary nail for tibiotalocalcaneal arthrodesis: a biomechanical investigation.** *Am J Orthop (Belle Mead NJ) 2011;40-2:78-83.*

2.3 - Role of biologics to promote bone healing *(Callum Clark)*

Bone healing can be improved by using products that are ***osteogenic***, ***osteoconductive*** and ***osteoinductive***. This is discussed further in *Session 3*. Bone grafts or substitutes can also provide structural support or act as a delivery system for antibiotics. Urist and Dowell described the use of ***demineralised bone*** in 1968 which has since been used extensively to fill cavitary defects. Whilst it is known to be osteoinductive and osteoconductive in animals, this has yet to be proven in human trials.



The first ***bone graft extender*** was used in 1995. They are ceramic based scaffolds that are osteoconductive. They mimic the mineral phase of bone and have pores which allow for ***mesenchymal cell adhesion***, ***proliferation***, and ***differentiation*** into mature osteoblasts. However, as porosity increases, mechanical strength decreases. They are formed from either *hydroxyapatite*, *tricalcium phosphate*, *calcium sulphate*, or *biphasic calcium phosphate*. These materials are discussed further in *Session 3.3*.

In 2002, ***Recombinant Human Bone Morphogenic Protein - Type 2 (rhBMP-2)*** was approved for anterior lumbar interbody fusions after it was found to promote mesenchymal stem cell activity and reverse bone loss in osteopenic mice *(osteoinductive)*. However, a Cochrane review from 2010 found there was paucity of data on its use in fracture healing and considerable industry involvement was present. A similar product, ***rhBMP-7*** (OP-1), has been withdrawn from the market following lawsuits where the product was mixed with synthetic bone void filler and autograft in an unapproved manner resulting in complications including excessive bone growth and osteolysis.

Platelet-derived Growth Factor:

» Angiogenic
» Mitogenic and chemotactic effects on mesenchymal cells
» Co-factor required for fibroblast mitosis

Daniels *et al.* (in a company sponsored study) published one of the few series that investigated the effects of orthobiologics in foot and ankle surgery. They randomised patients undergoing hindfoot fusions to received either purified ***recombinant human platelet-derived growth factor BB homodimer (rhPDGF-BB)*** with a ***betatricalcium phosphate (β-TCP)–collagen matrix*** or autograft. Clinical success at one year (defined by a 20 point improvement in VAS score and lack of revision) was 91% in the orthobiologic-bone substitute combination group *versus* 78% in the autograft group. Furthermore fusion was quicker in rhPDGF-BB / β-TCP although fusion rates remained comparable at 84% and 80% respectively. It was unclear why *'clinical success'* would be different with the exception perhaps of avoiding donor site morbidity.

Stem-cell therapy:

» ***Embryonic stem cells*** are pluripotent *i.e.* they can differentiate to any cell type in the human body
» ***Tissue specific stem cells*** can differentiate into cell types for the specific tissue in which they reside
» ***Mesenchymal stem cells (MSCs)*** are derived from connective tissue, fat, and blood; they are capable of differentiating in bone, cartilage, muscle and fat

Hernigou *et al.* compared injection of ***bone marrow mesenchymal stem cells (BM-MSCs)*** delivered in an autologous bone marrow concentrate (BMC) to ankle non-union sites in diabetic patients and compared them to patients undergoing traditional revision techniques with iliac crest bone grafting. Union was observed in 82.1% of patients in the former group compared to 62.3% in the latter group whilst maintaining a lower rate of complications.

Summary:

- Bone allografts, substitutes and orthobiologics have been shown in laboratory studies to help bone healing
- Clinical trials have also shown these agents aid bone healing of fractures and arthrodesis although they are not a substitute for adequate joint preparation and suitable fixation
- The exact role of each specific agent has not been well defined within the literature and thus choice is dependent upon availability and the philosophy of the operating surgeon

References:

Daniels TR, Younger AS, Penner MJ, *et al.* **Prospective Randomized Controlled Trial of Hindfoot and Ankle Fusions Treated With rhPDGF-BB in Combination With a beta-TCP-Collagen Matrix.** *Foot Ankle Int 2015;36-7:739-48.*

Hernigou P, Guissou I, Homma Y, *et al.* **Percutaneous injection of bone marrow mesenchymal stem cells for ankle non-unions decreases complications in patients with diabetes.** *Int Orthop 2015;39-8:1639-43.*

Urist MR, Dowell TA. **Inductive substratum for osteogenesis in pellets of particulate bone matrix.** *Clin Orthop Relat Res 1968;61:61-78.*

2.4 - Evidence for bone stimulation *(Tim Clough)*

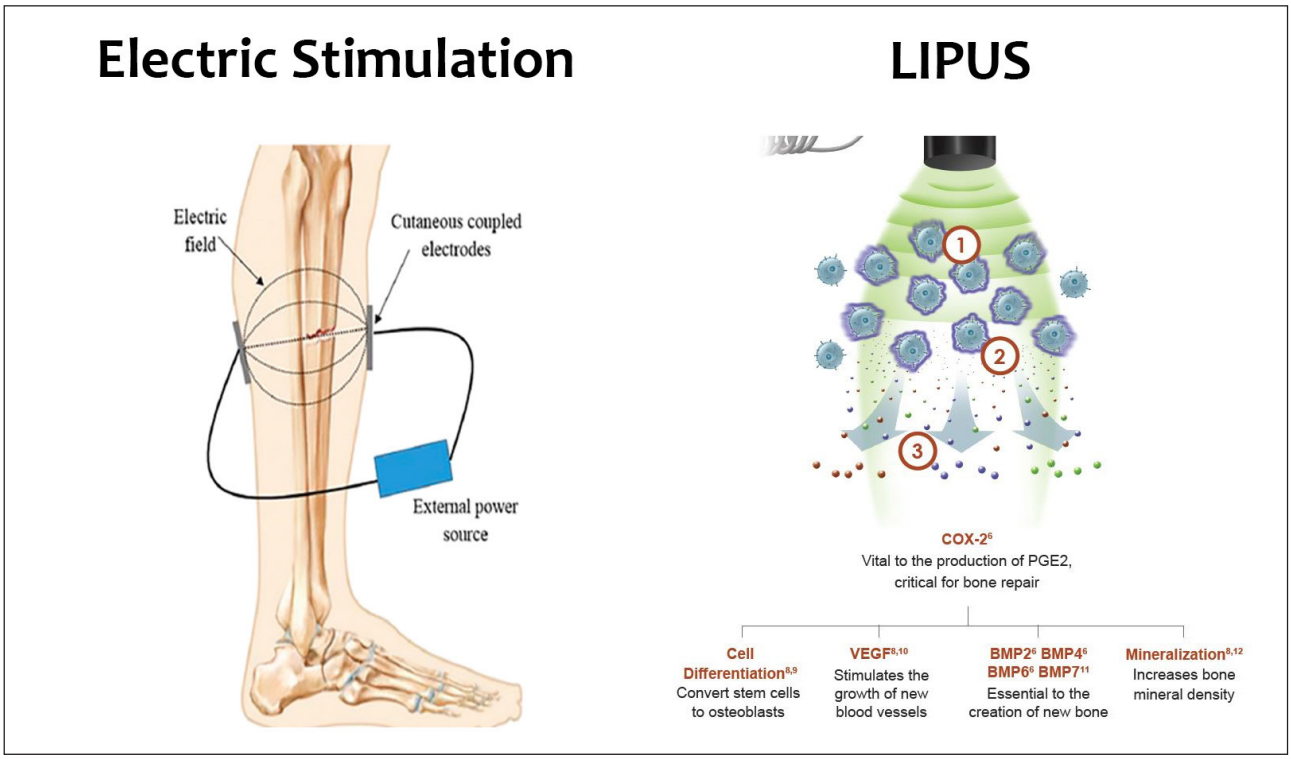
Bone stimulation can be internal or external. External devices work in three ways: ***low-intensity pulsed ultrasound***, ***electric stimulation***, and ***electromagnetic stimulation***. The TRUST trial investigated 501 patients with tibial shaft fractures treated with intramedullary nailing, randomised to use either an EXOGEN® or sham device post-operatively. They found compliance was moderate since only 73% of patients administered ≥50% recommended treatments and it did not accelerate union / improve rate of union.

Methods of external bone stimulation:

» ***Low intensity pulsed ultrasound (LIPUS)*** is provided by devices such as EXOGEN® (Bioventus). It delivers ultrasound waves through soft tissue to bone which activates cell receptors creating cascade reaction and increased COX-2. This activates osteoclasts and macrophages to remove bone debris and stimulates immature bone marrow to produce bone and cartilage. It also accelerates maturation and ossification of bone. The EXOGEN® device is used for 20 minutes per day for up to 6 months.

» ***Electric stimulation*** is provided by devices such as the Biomet OrthoPak®. It transmits potentials of between 1-10V, frequencies between 20-200Hz and electric fields between 1-100mV/cm. Cutaneous coupled electrode pads are placed either side of a fracture or arthrodesis site and this allows an electric field to be formed between them. The electric currents theoretically stimulate production of *BMP 2, 3, 4, 5, 6, 7 and 8* which in turn stimulates *Osterix* production. This is a transcription factor for osteoblast differentiation which act on proteoblasts that ultimately leads to increased bone mass. The Biomet OrthoPak® is advised to be used for Use 270 days for 24 hours per day which has implications for patient compliance.

» ***Electromagnetic stimulation*** is provided by devices such as the DonJoy CMF® (Combined Magnetic Field) Bone Growth stimulator. It creates a stress gradient that drives fluid through canaliculi from high to low pressure. This exposes osteoblast membranes to flow-related shear stress and electrical potential activation which in turn increases osteoblast secretion IGF-II and osteoblastic DNA synthesis. The CMF® device is advised to be used for 30 minutes per day for an average of 6 months.



Recently published retrospective work by Majeed *et al.* evaluated the effect of six months of EXOGEN® usage in patients with non-union following foot and ankle surgery finding it led to good outcomes:

Region treated with EXOGEN® (Majeed *et al.*)	**Union Rate**
Forefoot / Midfoot	78%
Hindfoot	67%
Fractures	93%

NICE has recently updated its guidance in 2018 regarding LIPUS stating that the poor-quality evidence regarding its use suggests it should only be used with special arrangements for clinical governance, consent, and audit or research. A meta-analysis of randomised sham-controlled trials of electric stimulation found moderate quality evidence from 15 trials that stimulation reduced radiographic non-union rates by 35% (Aleem *et al.*). A Cochrane review of electromagnetic field stimulation for treating delayed union or non-union of long bone fractures in adult found that whilst the intervention gave a risk ratio of 1.96 for union being realised, the difference was not statistically significant.

Summary:

- Routine use of bone stimulators is not recommended
- There may be a role for bone stimulation if union is not progressing but the optimal type of stimulator has not been established
- Using a bone stimulator earlier than six months after surgery may be better than if used after more than six months

References:

Aleem IS, Aleem I, Evaniew N, *et al.* **Efficacy of Electrical Stimulators for Bone Healing: A Meta-Analysis of Randomized Sham-Controlled Trials.** *Sci Rep 2016;6:31724.*

Ebrahim S, Mollon B, Bance S, *et al.* **Low-intensity pulsed ultrasonography versus electrical stimulation for fracture healing: a systematic review and network meta-analysis.** *Can J Surg 2014;57-3:E105-18.*

Majeed H, Karim T, Davenport J, *et al.* **Clinical and patient-reported outcomes following Low Intensity Pulsed Ultrasound (LIPUS, Exogen) for established post-traumatic and post-surgical nonunion in the foot and ankle.** *Foot Ankle Surg 2019; epub, ahead of print.*

Phillips M, Baumhauer J, Sprague S, *et al.* **Use of Combined Magnetic Field Treatment for Fracture Nonunion.** *J Long Term Eff Med Implants 2016;26-3:277-84.*

Trust Investigators Writing Group, Busse JW, Bhandari M, *et al.* **Re-evaluation of low intensity pulsed ultrasound in treatment of tibial fractures (TRUST): randomized clinical trial.** *BMJ 2016;355:i5351.*

Session 2 - Discussion & Consensus

Smoking in Foot & Ankle:

» **How many surgeons present routinely discuss the risks of smoking during the consent process?**

Yes	25	(100%)
No	0	(0%)

» **How many surgeons would not routinely perform a *primary* ankle fusion on an active smoker?**

Would not routinely operate whilst still smoking	7	(28%)
Would still offer surgery / operate	18	(72%)

» **How many surgeons would not routinely perform a *revision* ankle fusion on an active smoker?**

Would not routinely operate whilst still smoking	17	(68%)
Would still offer surgery / operate	5	(20%)
Undecided / would depend on situation	3	(12%)

Session 3: Managing Bone Loss / Bone Defects

3.1 - Which autograft is better? *(Steve Hepple)*

An ***autograft*** is bone or tissue taken from the host from one site and transferred to another to replace a deficiency. In foot and ankle surgery bone autograft may be used to *fill a void*, to *provide structural support*, and for *biological stimulation*. Autograft is *osteoconductive*, *osteoinductive*, and *osteogenic*. It does not provoke an immune response, is readily available and cheap, and may be used with local antibiotics. Cons include donor site morbidity, increased operating time and limited supply for larger defects. The efficacy of autograft may be limited by several host factors including vascularity of host site, abundance of progenitor cells, size of defect, presence of infection, immunosuppression, previous radiotherapy and drugs such as nicotine and NSAIDs.

Types of Autograft:

» ***Cortical graft*** is useful as structural graft but has a lower cell count and has limited osteogenic and osteoinductive potential. It provides immediate strength, but this gradually reduces over 6 months due to resorption. This strength is regained by 12 to 18 months.

» ***Cancellous graft*** is the most commonly used in foot and ankle surgery. It consists of a high quantity of mesenchymal stem cells and endothelial cells which can proliferate and promote healing. It has a high surface area and is immediately active, producing vascularisation within 2 days, new bone within 2 weeks, and begins to remodel within 8 weeks.

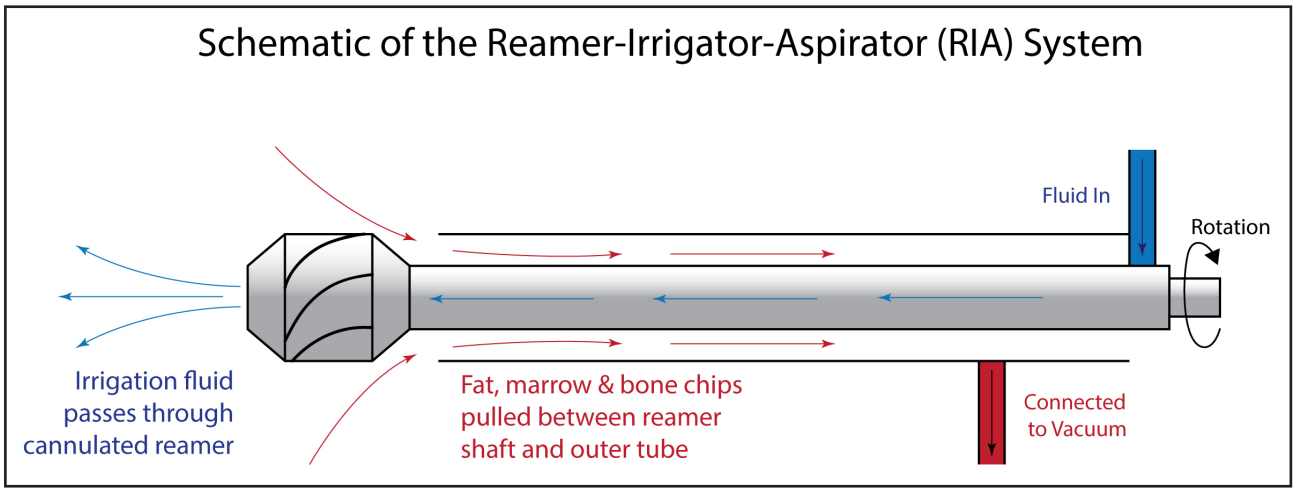
» ***Vascularised graft*** consists of cortico-cancellous graft on a vascular pedicle. This improves incorporation and healing although it requires a much longer procedure and has more donor site morbidity. It may be used to treat larger bone defects.

Iliac crest graft is often considered the gold-standard as it can be used to harvest cortical, cancellous, cortico-cancellous and vascularised grafts, and large volumes are available. However, complications range from 10-20% minor complications to 5% major complications which include: infection, deep hematoma, vascular injury, hernias, nerve / ureter injury, and persistent pain.

Proximal tibial graft can also generate high volume of graft and is easy to access during foot and ankle surgery. It has a lower complication rate and main risks involve persistent pain, fracture, knee penetration, and subsequent radiological appearance that may be confused with tumour. Other harvest sites less frequently used include the ***distal tibia***, ***greater trochanter***, ***calcaneum*** and ***distal radius***.

The ***Reamer Irrigator-Aspirator (RIA) system*** may also be used to harvest large quantities of cancellous bone from the femur. This is rich in stem cells and growth factors, is less painful than iliac crest graft, and has a low risk of fracture. However, it requires specialised equipment, fluoroscopy, appropriate planning, and the patient may require a post-operative blood transfusion.

Other options which have been investigated include ***Bone Marrow Aspirate,*** which likely has its effect through introduction of progenitor endothelial cells which improve blood supply, and ***Platelet Rich Plasma***, which introduces growth factors. Neither of these has conclusive evidence to support routine use.



There is little clinical evidence to recommend one donor site over another. Harvest sites closer to the axial skeleton tend to have a greater count of pleuri-potent stem cells. However, more distal graft sites are also suitable despite reduced cell count; it is therefore unclear whether the osteoblastic component of the graft is as important as the endothelial proliferation potential.

Summary:

- There is little clinical evidence to recommend one site of autograft harvest over another
- Where there is adequate host vascularity and a good graft bed, harvest may be from the calcaneum or distal tibia (low volumes), or ipsilateral proximal tibia (larger volumes)
- Where host biology is poor or there is reduced vascularity, iliac crest graft may be preferred

References:

Bauer TW, Muschler GF. **Bone graft materials. An overview of the basic science.** *Clin Orthop Relat Res* 2000-371:10-27.

Lim ZXH, Rai B, Tan TC, *et al.* **Autologous bone marrow clot as an alternative to autograft for bone defect healing.** *Bone Joint Res* 2019;8-3:107-17.

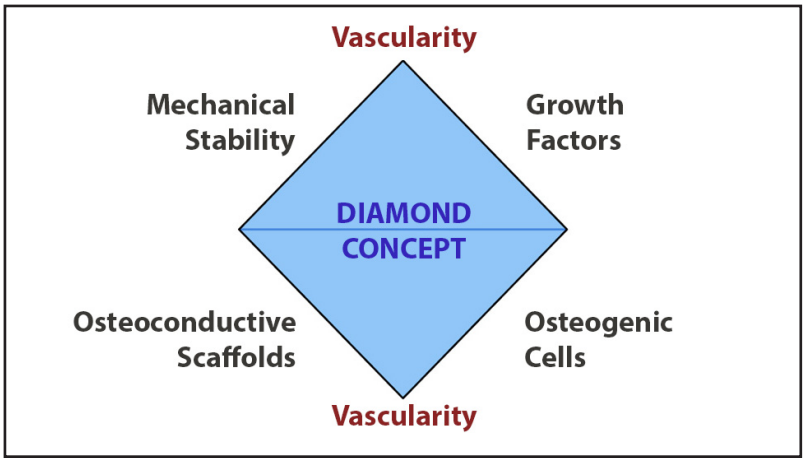
Miller CP, Chiodo CP. **Autologous Bone Graft in Foot and Ankle Surgery.** *Foot Ankle Clin* 2016;21-4:825-37.

Roberts TT, Rosenbaum AJ. **Bone grafts, bone substitutes and orthobiologics: the bridge between basic science and clinical advancements in fracture healing.** *Organogenesis* 2012;8-4:114-24.

Whitehouse MR, Lankester BJ, Winson IG, *et al.* **Bone graft harvest from the proximal tibia in foot and ankle arthrodesis surgery.** *Foot Ankle Int* 2006;27-11:913-6.

3.2 - Allografts *(Phil Vaughan)*

Giannoudis et al. published their ***Diamond Concept*** which reinforces that for healing to occur an appropriate scaffold is required. The ideal bone graft should be *osteoconductive* (appropriate permeable scaffold), *osteoinductive* (growth factors and BMPs), *osteogenic* (cellular), biocompatible, and enable good vascularity in a mechanically stable environment.



Allograft may be used in foot and ankle surgery to fill defects and aid fusion in the setting of trauma, tumour, non-union and deformity correction. Compared to autograft, it has the advantage of shorter operative times, lack of donor site morbidity and availability of large volumes if required. However, it is expensive, and there are the risks of disease transmission and host response to the allograft. Availability is dependent on tissue banks.

Stages of incorporation of allograft:

- ***Inflammatory:*** Initial necrosis and rejection
- ***Revascularisation:*** Ingrowth of new vessels to and through graft
- ***Osteoinduction:*** Permeating of BMP / growth factors
- ***Osteoconduction:*** Ingrowth and laying down of new host bone
- ***Remodelling:*** Laying down of new Haversian system

In the UK, bone allograft is available via the NHS Tissue bank. This provides a selection of safe bone which has been adequately quarantined and processed. It is available in several formats: *shaped bone graft*, *osteochondral graft*, *structural / cortical graft*, *morselised / cancellous graft*, *cortico-cancellous graft*, *massive allograft*, *femoral heads*, *demineralised bone matrix* and *cellular allograft*.

Cellular allograft incorporates mesenchymal stem cells (MSC) into a ***demineralised bone matrix (DBM)***. This is possible as MSCs are immunologically privileged and do not contain cellular markers necessary for activation of the host immune cascade. The resulting scaffold is termed ***cellular bone matrix (CBM)*** and although it provides no structural benefit it is highly osteoconductive and osteoinductive.

There is Level II evidence and a Grade B recommendation for use of allograft versus autograft in the foot and ankle. Both techniques have equivalent union rates, time to graft incorporation and complication rates. Cellular allograft has been shown as equivalent to autograft for the treatment of contained bone defects in ankle fusion. There have been similar findings for the use of DBM in hindfoot fusion and osteochondral defects of the talus. Other uses include the use of osteochondral graft for the ankle, femoral head allograft for hindfoot fusion with large defects, and bone block arthrodesis of the subtalar joint.

Ways in which allografts are processed (multiple techniques may be used):

» ***Fresh-Frozen:*** preserved cellular component, with greatest osteogenic and osteoinductive potential, but greatest immune response and disease risk. 5-year shelf life in the freezer. Maintains >90% of compressive and torsional strength

» ***Freeze-Dried:*** prolongs shelf life (at room temperature), however, there is disruption of collagen creating a graft that is brittle, but which can still function well under compression

» ***Irradiated:*** effective at decontamination, including blood borne viruses, but reduces mechanical properties of graft significantly. Osteoconductive only. Loses > 50% strength in axial compression

» ***Washed:*** reduces host response by depleting cell and marrow content, reducing osteogenic potential

» ***DBM:*** cortical graft demineralised in hydrochloric acid. Removes cellular component and structural strength but maintains BMPs (osteoinductive and osteoconductive)

Summary:

- Allograft may be used with good results in the foot and ankle for primary fusion and the treatment of large defects in the setting of a well vascularised bed
- It is important to choose the right graft type for the defect being managed, bearing in mind the mechanical properties of the graft and the level of graft processing

References:

Chou LB, Halligan BW. **Treatment of severe, painful pes planovalgus deformity with hindfoot arthrodesis and wedge-shaped tricortical allograft.** *Foot Ankle Int 2007;28-5:569-74.*

Dekker TJ, White P, Adams SB. **Efficacy of a Cellular Bone Allograft for Foot and Ankle Arthrodesis and Revision Nonunion Procedures.** *Foot Ankle Int 2017;38-3:277-82.*

Galli MM, Protzman NM, Bleazey ST, *et al.* **Role of Demineralized Allograft Subchondral Bone in the Treatment of Shoulder Lesions of the Talus: Clinical Results With Two-Year Follow-Up.** *J Foot Ankle Surg 2015;54-4:717-22.*

Giannoudis PV, Einhorn TA, Marsh D. **Fracture healing: the diamond concept.** *Injury 2007;38 Suppl 4:S3-6.*

Pelker RR, Friedlaender GE, Markham TC. **Biomechanical properties of bone allografts.** *Clin Orthop Relat Res 1983-174:54-7.*

Yanez Arauz JM, del Vecchio JJ, Amor RT, *et al.* **Non-irradiated frozen structural allograft in reconstructive surgeries of the hindfoot and midfoot.** *Foot Ankle Surg 2014;20-2:120-4.*

3.3 - Bone substitutes *(Billy Jowett)*

The ideal bone substitute should be biocompatible, structurally similar to bone, easy to use, safe, cost effective, osteoconductive and osteogenic. Bone substitutes avoid the risks associated with autograft and allograft. They may be derived from biological products or wholly synthetic.

Biological substitutes include *demineralised bone matrix (DBM)*, *platelet rich plasma (PRP)*, *bone morphogenic proteins (BMPs)*, *hydroxyapatite* and *coral*. **Synthetic substitutes** include *calcium sulphates*, *calcium phosphates*, *biphasic calcium phosphates*, *tricalcium phosphate*, *bioactive glass* and *polymer based substrates*.

Biological substitutes:

» ***DBM*** is processed to remove the mineral component of bone but maintain BMPs and growth factors. It consists of 95% collagen and is osteoinductive and osteoconductive. It has poor structural properties and therefore is often combined with a carrier. It does not suffer from immunological rejection.

» ***PRP*** is a concentrate of platelets and growth factors. It has no mechanical properties, and there is no evidence to validate its use as a standalone bone substitute.

» ***BMPs*** are part of the TGF-β family and are osteoinductive. They are produced as recombinant human BMPs and have demonstrated promising results in the treatment of non-unions.

» ***Hydroxyapatite*** is biocompatible, osteoconductive and has good mechanical properties, particularly in resisting compression. It has a very slow rate of resorption, persisting for 3 years after implantation.

» ***Corals*** have a skeleton similar to spongy bone. They are formed from calcium carbonate and are resorbed by bone and transformed into hydroxyapatite. They can be used as carriers for BMPs. They do not produce an inflammatory response.

Synthetic substitutes:

» ***Calcium sulphates*** have a similar structure to bone and are osteoconductive. They are absorbed rapidly (1 to 3 months) at a rate that exceeds new bone deposition. They have no mechanical strength, are available as pellets, powder or pastes and serve as a vector for delivery of antibiotics or growth factors.

» ***Calcium phosphates*** can be delivered as a cement. Their nanocrystalline structure resembles hydroxyapatite and is osteoconductive. They resorb over 2 years. They are available as pastes.

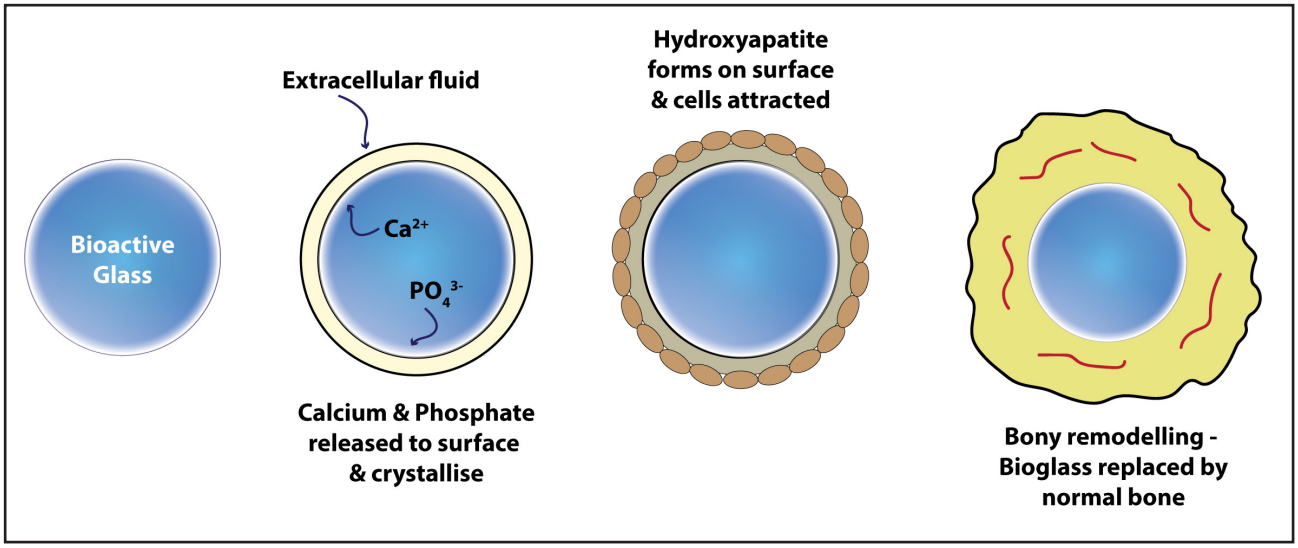
» ***Tricalcium phosphates*** are considered the gold standard for synthetic bone. They resorb slower than calcium sulphates and provide mechanical resistance for up to 13 to 20 weeks. They are osteoconductive.

» ***Biphasic calcium phosphates*** are synthetic hydroxyapatites which are osteoconductive and resorb faster than hydroxyapatite. They bond well with host bone but have less resistance to compression than cortical bone and organic hydroxyapatite.

» ***Bioactive glasses*** are silicates which are combined with calcium and phosphate oxides. When exposed to extracellular fluid the surface releases calcium and phosphate which mineralise and form hydroxyapatite. This is then remodelled to normal bone and all the glass is absorbed. They are osteoinductive, immunologically inert and porous. They are, however, brittle and have low mechanical strength.

» ***Polymer based substitutes*** are based on collagen and can be biodegradable (PLA) or non-degradable (PMMA). They can also mimic collagen with porosity and osteoconductive properties (polycapolactone). They are often used as carriers for growth factors or antibiotics.

Due to the large number of products available and lack of large trials it is difficult to make evidence-based recommendations. A Grade B recommendation can be made for the use of calcium phosphates and sulphates in the management of displaced, intraarticular calcaneal fractures. A Grade B recommendation can be made for the use in DBM in hindfoot and ankle fusions.



Summary:

- Bone substitutes may be biological or synthetic and have varied properties
- There is a wide variety of choice but few studies to guide practice
- It is important to understand the properties of any substitute used and choose the correct type for the particular clinical problem

References:

Campana V, Milano G, Pagano E, *et al.* **Bone substitutes in orthopaedic surgery: from basic science to clinical practice.** *J Mater Sci Mater Med* 2014;25-10:2445-61.

Fernandez de Grado G, Keller L, Idoux-Gillet Y, *et al.* **Bone substitutes: a review of their characteristics, clinical use, and perspectives for large bone defects management.** *J Tissue Eng* 2018;9:2041731418776819.

Habraken W, Habibovic P, Epple M, *et al.* **Calcium phosphates in biomedical applications: materials for the future?** *Materials Today* 2016;19-2:69-87.

Hannink G, Arts JJ. **Bioresorbability, porosity and mechanical strength of bone substitutes: what is optimal for bone regeneration?** *Injury* 2011;42 Suppl 2:S22-5.

Pryor LS, Gage E, Langevin CJ, *et al.* **Review of bone substitutes.** *Craniomaxillofac Trauma Reconstr* 2009;2-3:151-60.

Wang W, Yeung KWK. **Bone grafts and biomaterials substitutes for bone defect repair: A review.** *Bioact Mater* 2017;2-4:224-47.

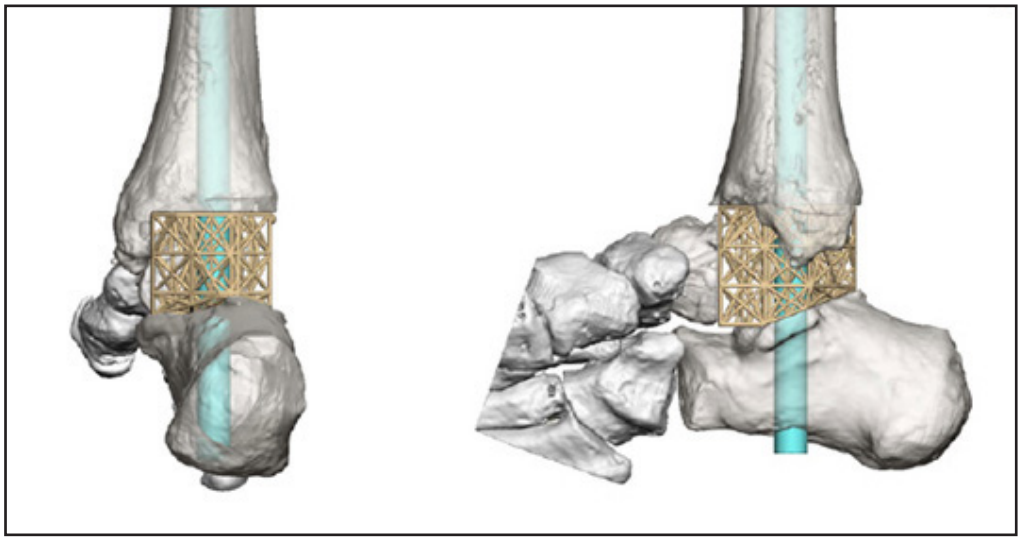
Wee J, Thevendran G. **The role of orthobiologics in foot and ankle surgery: Allogenic bone grafts and bone graft substitutes.** *EFORT Open Rev* 2017;2-6:272-80.

3.4 - Metal cages & solid metal *(Mike Karski)*

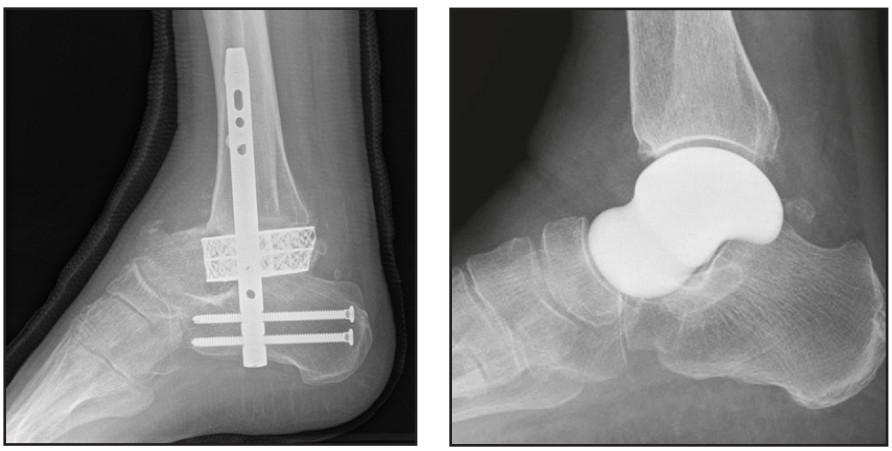
Bone graft alone may be insufficient for larger, more complex foot and ankle defects, such as revision ankle arthroplasty where there are large cysts and / or bone loss. Autograft may not be suitable due to limitations in the quantity available for harvest, whereas high non-union rates (up to 50%) have been reported when allograft has been used in this setting. Metal cages or implants may have a role to play in such cases.

Tantalum cages are solid trabecular metal cages with porous structures, similar to cancellous bone, which encourages bony ingrowth. They are strong and have a modulus of elasticity similar to that of cortical bone. Sagherian *et al.* reported on three cases of failed ankle arthroplasty revised to tibio-talar-calcaneal (TTC) fusion using these cages and report no complications, with fusion achieved at 3 months. However, Aubret *et al.* reported less favourable results with 5 out of 11 patients having ongoing pain and three patients requiring further surgery.

Palmanovich *et al.* reported on seven patients undergoing either TTC or subtalar fusion using a ***spinal fusion cage*** and autograft. They achieved union in all cases by 6 to 12 months and had one case of infection. ***Custom, 3D printed titanium cages*** may also be used; these can be designed to allow a nail and / or screws to pass through the cage. The shape can be planned to preserve bone whilst filling the defect, and they can be packed with autograft, allowing bone to incorporate through the cage.



For severe talar fractures / AVN, a **3D printed talus** can also be created to replace the diseased talus. This is a novel technique with a few published case reports, and no long-term results as yet.



Summary:

- Metal implants can be used to manage large bony defects
- Solid metal / tantalum cages appear to have a high non-union / complication rate
- Metal cages allow filling with autograft and may be custom designed. They are thus more versatile and may be appropriate for complex cases
- The use of 3D printed implants is an emerging technique, but there are limited series and no long term follow up thus far

References:

Aubret S, Merlini L, Fessy M, *et al.* **Poor outcomes of fusion with Trabecular Metal implants after failed total ankle replacement: Early results in 11 patients.** *Orthop Traumatol Surg Res 2018;104-2:231-7.*

Palmanovich E, Brin YS, Ben David D, *et al.* **Use of a spinal cage for creating stable constructs in ankle and subtalar fusion.** *J Foot Ankle Surg 2015;54-2:254-7.*

Ruatti S, Corbet C, Boudissa M, *et al.* **Total Talar Prosthesis Replacement after Talar Extrusion.** *J Foot Ankle Surg 2017;56-4:905-9.*

Sagherian BH, Claridge RJ. **Salvage of failed total ankle replacement using tantalum trabecular metal: case series.** *Foot Ankle Int 2015;36-3:318-24.*

Session 3 - Discussion & Consensus

Autograft:

» **How many surgeons routinely use bone graft in primary hindfoot fusions?**

Yes	8	(32%)
No	17	(68%)

» **What is your preferred harvest site for cancellous autograft?**

Iliac crest	9	(36%)
Proximal tibia	9	(36%)
Other	7	(28%)

» **For those surgeons who harvest graft from the proximal tibia, which is your preferred side of harvest?**

Medial side	9	(36%)
Lateral side	3	(12%)
Do not routinely harvest from proximal tibia	13	(42%)

» **What is your preferred harvest site for distal harvests?**

Calcaneal	17	(68%)
Distal medial tibia	7	(28%)
Do not routinely perform distal harvests	1	(4%)

» **How many surgeons would use bone marrow aspirate for revision arthrodesis surgery?**

Yes	5	(20%)
No / Unsure	20	(80%)

Allograft / Bone substitutes / Metal cages:

» **How many surgeons use structural allograft for complex reconstructions?**

Yes	14	(56%)
No	11	(44%)

» **What is your preferred method for filling of large bone defects when revising an ankle arthroplasty to a fusion?**

Autograft	3	(12%)
Structural allograft	8	(32%)
Metallic Cages	7	(28%)
Do not routinely do this procedure	7	(28%)

» **How many surgeons routinely use bone substitute to augment a primary subtalar fusion?**

Yes	0	(0%)
No	25	(100%)

» **If you had a small, contained bony defect, for e.g. around the tibia or talus, what would be your preferred choice of material to fill it?**

Autograft	12	(48%)
Allograft	0	(0%)
Synthetic substitute	8	(32%)
Unsure	5	(20%)

» **What is your preferred choice of material for filling a cyst around an ankle replacement?**

Autograft	11	(44%)
Allograft	4	(16%)
Calcium sulphates / calcium phosphates	4	(16%)
Unsure / not part of routine practice	6	(24%)

Session 4: Miscellaneous

4.1 - Stress fractures *(Andy Goldberg)*

Stress fractures occur when there is cyclical overload at an intensity lower than the maximum bone strength in non-pathological bone. Classically they occur 6-8 weeks after the onset of insult, such as increased intensity training, and would present with symptoms of pain and swelling without an acute injury. History and identification of risk factors is thus important in diagnosis.

Imaging is required to confirm the diagnosis. Plain radiographs are often ordered as a first line imaging modality but have a high false negative rate since signs may only be seen 2-4 weeks after onset of pain. MRI is sensitive and specific since signal changes can be seen within the bone before macroscopic cortical discontinuity is present. CT is usually reserved when MRI is contraindicated. SPECT-CT can be used but has a high dose of radiation and is mostly used to assess the pars interarticularis rather than the foot or ankle. Arendt and Griffiths used MRI to classify severity and guide immobilisation time:

Grade of injury	MRI findings	Recommended rest from sport
1	STIR Positive	3 weeks
2	STIR and T2-weighted positive	3 to 6 weeks
3	T1 and T2 positive, without cortical rupture	12 to 16 weeks
4	T1 and T2 positive, with cortical rupture and visible fracture line	16 weeks

Forces acting on the bone lead to deformation which progresses to *plastic deformity* and *microfractures*. *Overloading*, *repetitive movements*, and *inadequate recovery* lead to chronicity as osteoclastic activity supersedes osteoblastic activity. Eventually, macro-fractures occur, and normal bone healing is initiated.

Risk factors for stress fractures:

» ***Intrinsic risk factors:*** genetics, ethnicity, gender, anatomical variance
» ***Extrinsic risk factors:*** activity levels, level of fitness, nutritional status, equipment (e.g. footwear)

Levels of ***Vitamin D*** may affect formation and healing of stress fractures. This is discussed and referenced in *Session 1.3* but is briefly recounted. Givon *et al.* studied 2,591 Israeli soldiers and showed Vitamin D deficiency was a risk factor for stress fractures. Ruohola *et al.* studied 756 male, Finnish military recruits and found recruits with lower levels of Vitamin D had an increased incidence of stress fractures. A blinded RCT of 3,700 female navy recruits showed Vitamin D supplementation lowered risk of stress fracture; the same study also showed that amenorrhea conveyed a 91% higher risk. (Lappe *et al.*)

However, defining the normal range of serum blood 25(OH)D level is controversial. Whilst a value of more than 50 nmol/l has previously been deemed sufficient, in 2011 the Institute of Medicine revised this to 90-100 nmol/l. In practice, this may lead to more diagnoses of Vitamin D deficiency, particularly in the winter months when levels are inherently lower. Genetic abnormality in the Vitamin D receptor allele frequency has been suggested as a risk factor for stress fractures. However, a recent meta-analysis (Gao *et al.*) did not find a statistically significant association; an increased risk was found in population-

based studies but not in hospital-based studies. Therefore, there may be a selection bias and further work is required on this.

Race may play a role: stress fractures in one military population have been shown to be twice as likely in Caucasian than Afro-Caribbean soldiers independent of sex. However, this was attributed to difference in bone density rather than race as an independent risk factor. Age does not appear to play a role; stress fractures are as likely in the elderly as the younger population and incidence differences are likely to be multifactorial. However, gender does play a role in the aetiology. Multiple studies have found that females are at increased risk of stress fracture compared to males although the association is complex and is attributable to multiple factors including hormones, nutrition, body fat, and activity levels.

Immobilisation has traditionally been the accepted treatment but there is a recent trend to avoid complete immobilisation and the associated deleterious effects on muscle strength and conditioning. Immobilisation may be needed when there is a high risk of non-union such as in stress fractures of the *navicular*, *sesamoids*, *patella & posteromedial tibia* but in these fractures early fixation and stabilisation is also justified.

Other therapies for management of stress fractures:

» ***Hyperbaric Oxygen:***	controversial evidence for use
» ***Bisphosphonates:***	usually used in refractory cases only
» ***Platelet rich plasma (PRP):***	no evidence to support use
» ***Recombinant parathyroid hormone:***	rarely used
» ***Low intensity pulsed ultrasound:***	conflicting evidence for use
» ***Electromagnetic stimulation:***	no evidence to support use

Summary:

- Stress fractures result from repetitive load
- Risk factors should be identified, and extrinsic factors should be corrected to give the best outcomes and avoid repeat injuries

References:

Arendt EA, Griffiths HJ. **The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes.** *Clin Sports Med 1997;16-2:291-306.*

Gao J, Wang L, Zhu J. **Influence of BsmI polymorphism in vitamin D receptor gene on the risk of fracture in Caucasian populations: a meta analysis.** *Int J Clin Exp Med 2015;8-1:589-97.*

Gao XR, Yu YG. **Meta-Analysis of the Association between Vitamin D Receptor Polymorphisms and the Risk of Autoimmune Thyroid Disease.** *Int J Endocrinol 2018;2018:2846943.*

Lappe J, Cullen D, Haynatzki G, *et al.* **Calcium and vitamin d supplementation decreases incidence of stress fractures in female navy recruits.** *J Bone Miner Res 2008;23-5:741-9.*

Milgrom C, Finestone A, Levi Y, *et al.* **Do high impact exercises produce higher tibial strains than running?** *Br J Sports Med 2000;34-3:195-9.*

Ruohola JP, Kiuru MJ, Pihlajamaki HK. **Fatigue bone injuries causing anterior lower leg pain.** *Clin Orthop Relat Res 2006;444:216-23.*

4.2 - Benign tumours *(Ben Rudge)*

Benign tumours can arise from soft-tissues or bones. They can be common, and history, examination and imaging will help distinguish them from malignant lesions. The most common tumours are listed below. It is beyond the scope of this chapter to discuss all benign tumours so the focus shall be on two of the most common: ganglion cysts and plantar fibromata.

Common types of benign foot and ankle tumours:

- ***Arising from soft-tissue:*** ganglion, plantar fibroma, lipoma, PVNS, haemangioma, neurinoma, angiomyoma, glomangioma, lymphangioma, fibrolipoma, desmoplastic fibroma, glomus tumour
- ***Arising from bone:*** osteochondroma, osteoid osteoma, enchondroma, aneurysmal bone cyst, intraosseous fibroma, giant cell tumour, non-ossifying fibroma, chondroma chondroblastoma, fibrous dysplasia, osteoblastoma, unicameral bone cyst

Ganglia are single or multiloculated cysts with mucinous contents arising from either a joint capsule or tendon sheath. The wall is lined by synovial cells without a true epithelial lining. 11% occur in the foot and only if they are symptomatic do they require treatment. There is controversy amongst surgeons regarding the best treatment. Kliman and Freiberg reviewed 33 patients with ganglia around the foot or ankle. Nine of the 21 patients (42%) who had surgery had a recurrence whilst only four recurred in the 12 patients (33%) who had aspiration and steroid injection. In the wrist, Ahmad Shah *et al.* recently identified a similar finding of 13% recurrence with aspiration followed by intralesional triamcinolone acetate injection *versus* 17% recurrence with excision. In light of these recurrence rates and inconvenience of ganglionectomy in the foot, aspiration and steroid injection may be considered safe, simple and as if not more effective than surgery.

Plantar fibromata were first described by Georg Ledderhose in 1897 and are caused by an overproduction of fibroblasts. They are bilateral in 25% of cases and associated with Dupuytren's disease in 9-25% of cases, and with Peyronie's disease in up to 4% of cases. The risk factors are similar to Dupuytren's and include: *middle-age*, *Caucasian race*, *male gender*, *family history*, *excessive alcohol consumption*, *liver disease*, *diabetes* and *epilepsy*. They present as slow growing nodules which can be painful. When it reaches a critical size the pressure effect can negatively impact gait. Unlike Dupuytren's disease, it does not effect the skin and rarely causes contractures. The differential diagnoses are serious enough that imaging should be considered to confirm the diagnosis in first time presentations. These include: *PVNS*, *leiomyoma*, *rhabdomyosarcoma*, *neurofibroma* and *liposarcoma*.

Stages in plantar fibroma development:

- ***Proliferative stage:*** increased fibroblastic activity
- ***Active/involution stage:*** fibroblasts mature and differentiate into myofibroblasts with increased collagen formation (nodule formation)
- ***Residual stage:*** reducing maturation and collagen formation (scar contracture)

Treatment is only considered in symptomatic patients and if possible, activity modification and orthotics should be commenced before other ***non-surgical*** options. Surgery is considered the last option due to the reportedly high rate of recurrence but may have a role in certain situations, *e.g.* to improve gait. The table below is adapted from Carroll *et al.* and summarises various non-surgical treatment methods.

Treatment	Mechanisms of Action	Results
Offloading orthotics	Offloads fibroma	Does not affect size or progression of fibroma. Provides symptomatic relief
Radiation	Ionizing radiation disrupts TGF-β produced by myofibroblasts during proliferation phase	Requires multiple sessions. 50% of patients report decrease in size of lesion
Extracorporeal shock wave	Mechanism of action unknown. Theorised to damages lesion resulting in autophagy	Pain reduction and softening of lesions reported as early as 2 weeks after treatment
Steroids	Decreases expression of VCAM-1 and alters production of TGF-β and bFGF	Reduces size and pain of lesion, however, lesion can reoccur after several years
Anti-oestrogen	Decreases contraction rates of myofibroblasts	No in vivo studies evaluating efficacy
Verapamil cream	Inhibits collagen production and increases collagenase activity	No reports in plantar fibromata, but decreases plaque size in Peyronie's disease
Collagenase injections	Contains 2 types of collagenase AUX-1 and AUX-2, which degrade collagen	Decreases contractions in Peyronie's and Duyuptren's but not in plantar fibromata
Colchicine	Inhibits microtubule polymerization by binding to tubulin	Not proven effective, more studies are needed to evaluate efficacy
Hyaluronidase	Catalyse degradation of hyaluronic acid	Multiple anecdotal reports but no published studies

Van der Veer *et al.* reported 60% of plantar fibromata reoccurred following surgical excision (100% with limited excision, 25% after total plantar fasciectomy). Post-operative radiotherapy may reduce this risk but is associated with impaired foot function, lymphoedema, fibrosis and fracture of irradiated bone.

Summary:

- Ganglia and plantar fibromata comprise the majority of foot and ankle soft-tissue tumours
- Ganglia may be effectively treated with aspiration and steroid injection
- Plantar fibromata have a high recurrence rate following excision so non-operative management should be exhausted prior to offering surgical treatment
- Whilst surgical excision is indeed a last resort, it is not necessarily something surgeons should wholly discount as it may have a role in certain situations, *e.g.* debulking to improve gait

References:

Ahmad Shah A, Raina AH, Ganie MA, *et al.* **Comparison of Aspiration Followed by Intra-Lesional Steroid Injection and Surgical Excision in Management of Dorsal Wrist Ganglion.** *World J Plast Surg* *2019;8-2:181-4.*

Carroll P, Henshaw RM, Garwood C, *et al.* **Plantar Fibromatosis: Pathophysiology, Surgical and Nonsurgical Therapies: An Evidence-Based Review.** *Foot Ankle Spec* *2018;11-2:168-76.*

de Bree E, Zoetmulder FA, Keus RB, *et al.* **Incidence and treatment of recurrent plantar fibromatosis by surgery and postoperative radiotherapy.** *Am J Surg* *2004;187-1:33-8.*

Kliman ME, Freiberg A. **Ganglia of the foot and ankle.** *Foot Ankle* *1982;3-1:45-6.*

van der Veer WM, Hamburg SM, de Gast A, *et al.* **Recurrence of plantar fibromatosis after plantar fasciectomy: single-center long-term results.** *Plast Reconstr Surg* *2008;122-2:486-91.*

4.3 - Malignant tumours *(Nick Cullen)*

Primary musculoskeletal malignancies are generally uncommon forming only 1% of cancer burden in adults and 10% in children. Since < 5% of all malignancies of bone and soft tissues affect the foot, the true burden of disease is difficult to establish and can result in a delay in diagnosis. Malignant neoplasms of the foot are usually primary, and rarely metastatic. The foot provides a unique challenge in the tissue preservation needed for a functional foot *versus* oncological principles of tumour resection.

Problems faced with malignant tumours in the foot:

- Multiple, small muscle compartments
- Terminal neuro-vascular branches
- Delicate soft tissue cover
- Highly specialised fat pad on the sole, that if excised cannot be adequately replaced by free flaps
- Tumour can pass between fascia along tendon sheaths

Chondrosarcoma and ***Ewing's sarcoma*** are the commonest malignant bone tumours arising in the foot and ankle followed by ***osteosarcoma***. ***Synovial sarcoma*** is the commonest soft tissue malignancy in the foot and ankle; it is known as the *'great mimicker'* for being clinically confused with more benign conditions such as gouty arthritis, ganglia, plantar fasciitis, synovitis, and plantar fibromata. Staging is by the TNM or Enneking *(table below)* classification systems.

Enneking Stage	Grade	Tumour	Metastasis
IA	G1 - Low grade	Intra-compartmental (T1)	M0
IB	G1 - Low grade	Extra-compartmental (T2)	M0
IIA	G2 - High grade	Intra-compartmental (T1)	M0
IIB	G2 - High grade	Extra-compartmental (T2)	M0
IIIA	G1 or G2	Intra-compartmental (T1)	M1
IIIB	G1 or G2	Extra-compartmental (T2)	M1

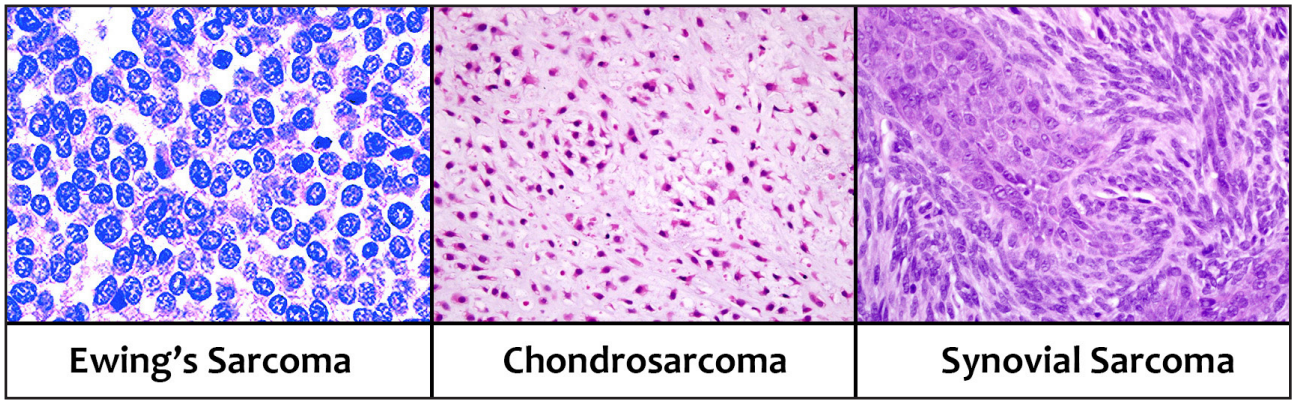
'Red flag' **criteria that should prompt urgent referral to local tumour service:**

- Deep, sub-fascial location
- > 5cm diameter ('golf-ball' size)
- Increase in size
- Painful mass
- Recurrent tumour

Chondrosarcomas present in the middle-aged male, with possible history of local trauma. The tarsal bones and short tubular bones of the feet may be involved. Radiographs show endosteal erosion, cortical destruction, indistinct margins, matrix calcification, and associated soft tissue masses. The main differential diagnosis is enchondroma.

Ewing's Sarcoma presents in childhood or adolescence with majority of cases arising < 25 years of age. The diaphysis of the long bones is affected. Radiographs show long, permeative, lytic lesions with soft tissue masses. Presentations around the foot have a 5-year survival of only 53% dropping to 14% if metastases are present.

Synovial Sarcoma does not arise from synovium but from dual epithelial/mesenchymal differentiation. The peak incidence is 20-30 years of age, although 30% occur in under 20s. They typically arise in a peri-articular position but can rarely occur within the joint. They are high grade tumours with invasive features. The tendency is for late local recurrence and to metastasise to the lungs. Surgery and radiation therapy achieve excellent local control, but the presence or absence of distant metastasis remains the most accurate prognostic factor of survival.



Ewing's Sarcoma	Chondrosarcoma	Synovial Sarcoma

Tumours should be staged and graded under the direction of the local tumour service. Biopsies must be performed at the tertiary referral tumour unit and performed by unit / team performing definitive surgery.

Summary:

- Clinical features of malignant tumours affecting the foot and ankle may be indistinguishable from benign conditions at time of presentation
- Therefore there is commonly a delay from presentation to diagnosis in excess of 1 year, warranting a high degree of vigilance by the surgical team
- Patients with suspected malignant lesions of the foot and ankle should be managed in a specialist centre with a multi-disciplinary team
- Surgical excision remains the mainstay of treatment for sarcomas and many patients will require a partial or complete amputation in order to achieve clear surgical margins

References:

Adkins CD, Kitaoka HB, Seidl RK, *et al.* **Ewing's sarcoma of the foot.** *Clin Orthop Relat Res* 1997-343:173-82.

Cadman NL, Soule EH, Kelly PJ. **Synovial Sarcoma; an Analysis of 34 Tumors.** *Cancer* 1965;18:613-27.

Chou LB, Ho YY, Malawer MM. **Tumors of the foot and ankle: experience with 153 cases.** *Foot Ankle Int* 2009;30-9:836-41.

Ozdemir HM, Yildiz Y, Yilmaz C, *et al.* **Tumors of the foot and ankle: analysis of 196 cases.** *J Foot Ankle Surg* 1997;36-6:403-8.

Papagelopoulos PJ, Mavrogenis AF, Badekas A, *et al.* **Foot malignancies: a multidisciplinary approach.** *Foot Ankle Clin* 2003;8-4:751-63.

4.4 - Avascular necrosis *(Henryk Liszka)*

Osteonecrosis (AVN) is a process in which bone becomes devitalised through disruption of the intramedullary blood supply and can progress to structural collapse and degradation. Trauma is the leading cause in the foot and ankle. Other causes are listed in the table below. Pathogenesis varies by aetiology and different processes may overlap: *vascular interruption*, *impaired angiogenesis*, *endothelial damage*, *vascular occlusion*, *coagulopathy*, *lipid accumulation / dysfunction*. Radiographs are usually sufficient for diagnosis, but MRI is the most sensitive imaging modality for early detection and reveals bone marrow oedema.

Trauma	Drug / Environment	Disease
Fracture; Dislocation; Soft-tissue injury	Systemic corticosteroid use; Excessive alcohol consumption; Smoking	Thrombotic disease (e.g. sickle cell); Inflammatory conditions (e.g. SLE); HIV

AVN of the talus is post-traumatic in 75% of cases, and idiopathic in the rest. It may be induced by chemotherapy (e.g. following renal or bone marrow transplantation) or corticosteroid use. Treatment depends on whether it is in the early or late stages.

Treatment of talar AVN:

» ***Early stage (pre-collapse):*** protected weightbearing & bracing; core decompression & bone grafting; vascularised grafts; subtalar fusion

» ***Late stage (post-collapse):*** tibiotalar fusion; tibio-talar-calcaneal fusion; ceramic talar replacement

Navicular AVN may occur as ***Köhler's disease*** occurs in children. This presents with medial midfoot pain and swelling. Microtrauma to the cartilaginous navicular by the surrounding tarsal bones leads to disruption of the vascular supply of the bone. Long-term outcomes are favourable regardless of treatment method, although a short period of immobilisation with a walking cast may reduce the duration of symptoms. ***Müller-Weiss disease*** occurs in adults. The talar head shifts laterally over the os calcis which drives the subtalar joint into varus creating a *paradoxical flatfoot* (hindfoot varus rather than valgus). Orthotic management involves rigid insoles with medial arch support and a lateral heel wedge to reduce supination of the heel. The Dwyer calcaneal osteotomy combined with lateral displacement seems to be a good alternative to the different types of perinavicular fusions that do not address pathomechanics.

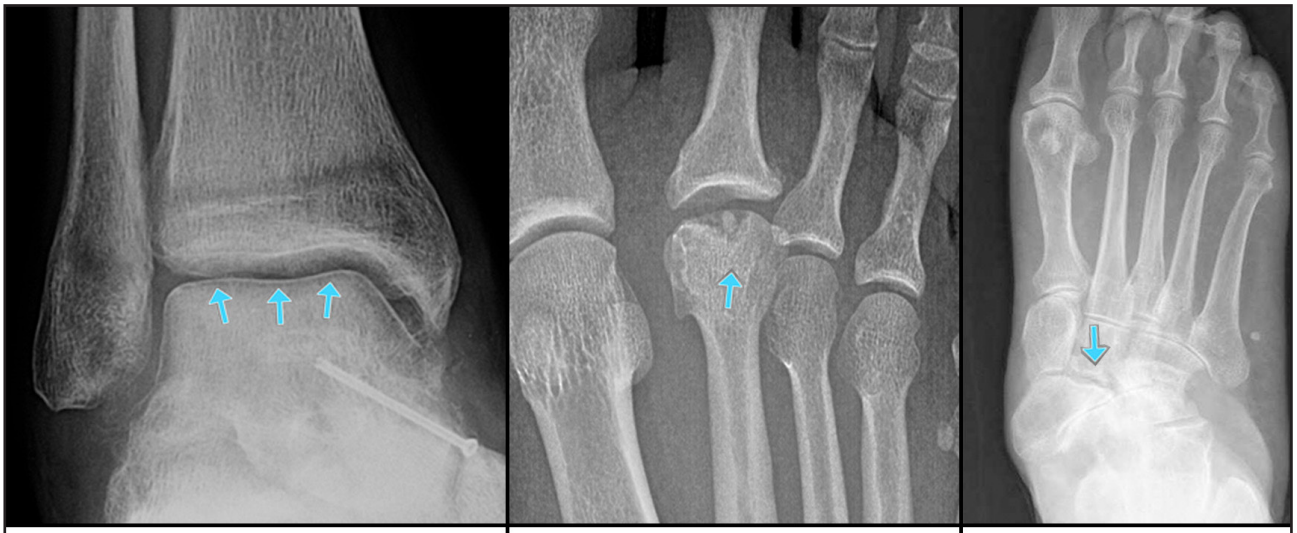
AVN of the first metatarsal most commonly results iatrogenically after hallux valgus surgery. This can be reduced by limiting soft tissue stripping around the plantar metatarsal head to ensure that saw cuts do not violate the vascular supply.

Management options for Friedberg's disease:

» ***Non-surgical:*** footwear modification, orthotics, corticosteroid injection (improves associated capsulitis), PRP / stem-cell injections (no evidence)

» ***Surgical:*** debridement, osteotomies (dorsal closing wedges / shortening), interposition arthroplasty (dorsal capsule / EDL / EBD), osteochondral grafting, arthroplasty (Cartiva / silicone / ceramic)

Lesser metatarsal AVN is commonly known as Freiberg's disease and can affect all metatarsal heads, although the second is most commonly afflicted (68%). Bilateral symptoms occur < 10% of patients. Despite significant radiographic findings, patients are often asymptomatic and require no or only modest nonsurgical management. No consensus exists on which treatment is best, but the various options are listed above.



AVN of the sesamoids more commonly affects the medial sesamoid. It is thought to occur due to repetitive trauma, resulting in microfractures. Fragmentation is a late finding. Non-operative treatment consists of offloading, but the rate of success is unclear. Operative treatment involves excision of the involved sesamoid and usually results in resolution of pain, however, complications include hallux varus or valgus and loss of push-off strength.

Summary:

- Osteonecrosis afflicts many bones in the foot but the incidence is probably underreported
- Treatment broadly follows the principles adhered to in other parts of the body
- No consensus on treatment exists for each stage of disease for each bone although patients should be fully informed of the options available

References:

Assouline-Dayan Y, Chang C, Greenspan A, *et al.* **Pathogenesis and natural history of osteonecrosis.** *Semin Arthritis Rheum* *2002;32-2:94-124.*

Carmont MR, Rees RJ, Blundell CM. **Current concepts review: Freiberg's disease.** *Foot Ankle Int* *2009;30-2:167-76.*

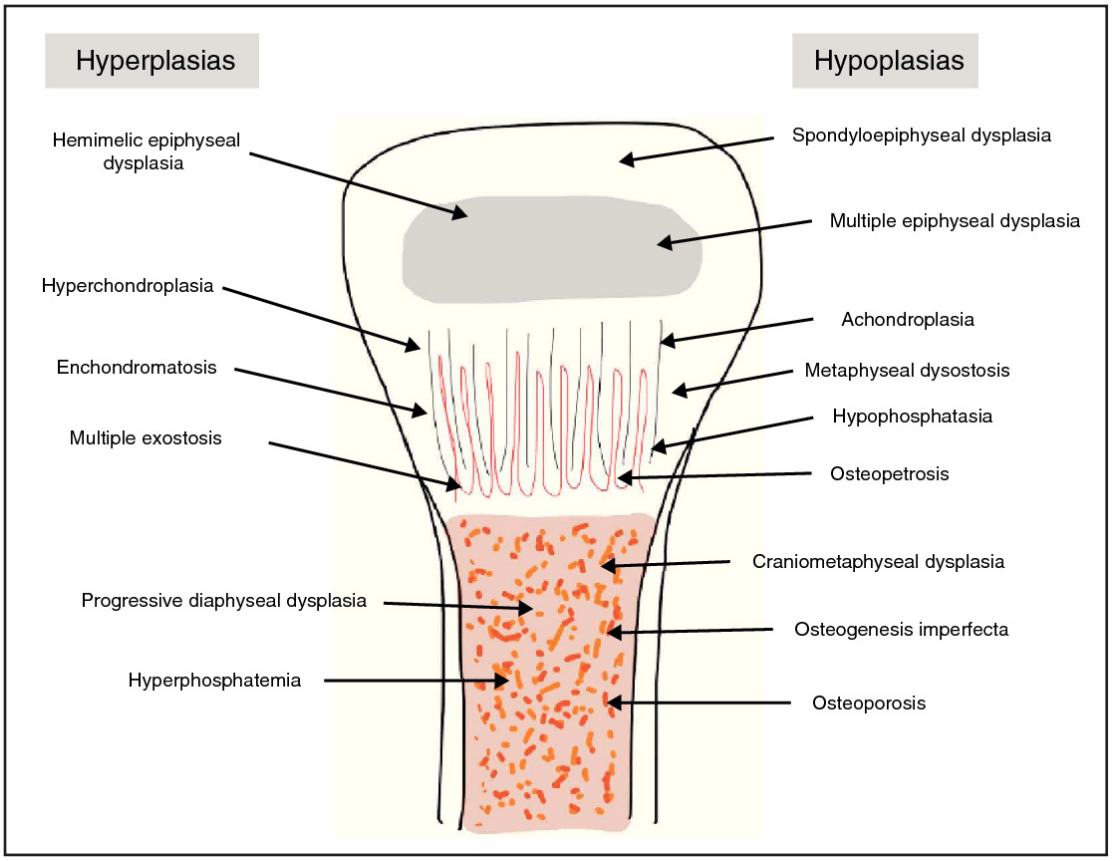
Cooper C, Steinbuch M, Stevenson R, *et al.* **The epidemiology of osteonecrosis: findings from the GPRD and THIN databases in the UK.** *Osteoporos Int* *2010;21-4:569-77.*

Gross CE, Haughom B, Chahal J, *et al.* **Treatments for avascular necrosis of the talus: a systematic review.** *Foot Ankle Spec* *2014;7-5:387-97.*

Multiple Authors from: **Avascular necrosis of the foot and ankle.** *Foot Ankle Clinics* *2019;24-1;1-172*

4.5 - Bone dysplasias *(Maneesh Bhatia)*

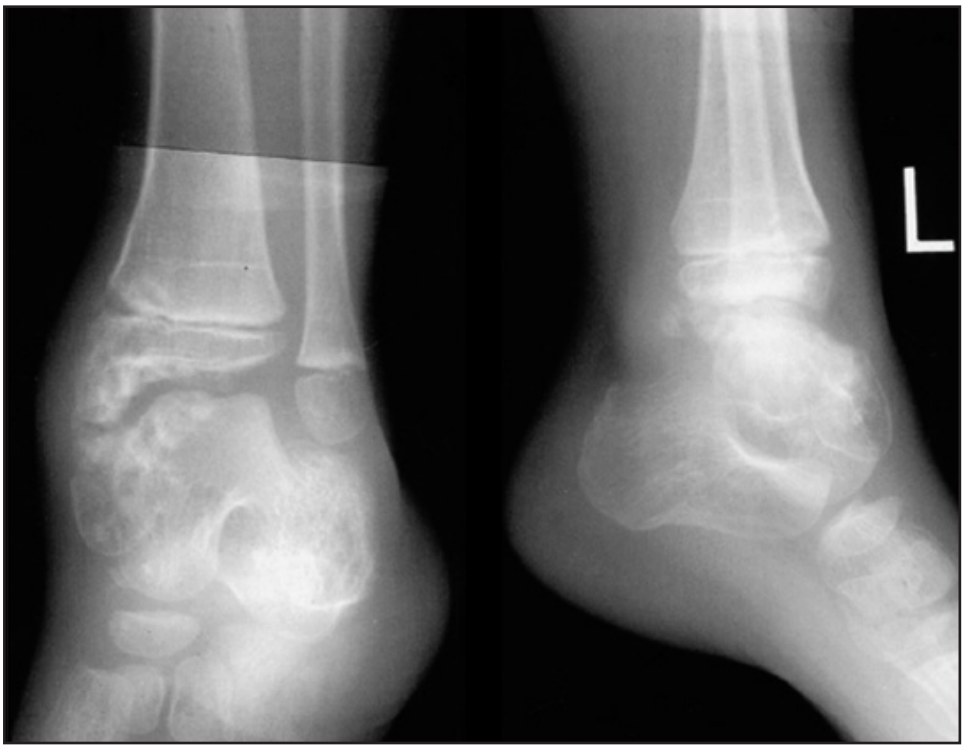
Bone dysplasias are disorders of growth and development that affect bone and cartilage. Most result in short stature (≥3 SD below the mean height for age). They can result from spontaneous genetic mutations or be inherited and many of the genes mutated in skeletal dysplasias encode proteins that play critical roles in the growth plate.



Rubin *et al.* proposed a classification dependent upon anatomical origin: whether the abnormality was in the *epiphysis*, *physis*, *metaphysis* or *diaphysis,* and furthermore, whether there was over or undergrowth. Bonafe *et al.* recently published a master list of dysplasias in which they identified 436 genetic skeletal diseases, stemming from mutations in 364 genes, divided into 42 groups. Dysplasias are uncommon with 1 case per 4,000-5,000 live births whilst lethal dysplasias form 0.95 per 10,000 deliveries. ***Achondroplasia*** is the most common nonlethal skeletal dysplasia while ***thanatophoric dysplasia*** is the commonest lethal skeletal dysplasia.

Detailed history including family history is crucial. An examination should identify the size ratios between upper and lower limbs and then identify which segments are affected. A full set of radiographs (spine, pelvis, extremities, hands, feet & skull) are needed and ideally should be taken prior to growth plate fusion.

Dysplasia epiphysealis hemimelica (Trevor's disease) is a rare developmental disorder affecting 1 in 1,000,000 persons. The epiphyses of knee and ankle joints can be affected in a manner seen in the radiographs below. In the ankle, the pattern of disease involves distal medial tibial epiphysis and talus. The natural history is continual growth until skeletal maturity. It therefore causes considerable disability in ankle function due to joint incongruity caused by the lesion's relentless growth during childhood. Early surgical removal to improve joint incongruity is recommended, with subsequent surgery to ensure continued congruity as required.



Multiple Epiphyseal Dysplasia (MED) also affects the foot and ankle. Ingram *et al.* identified 50 patients with MED and noted 50% had marked radiographic abnormality of the ankle despite relatively minor ankle pain. 50% also had small, broad feet with stubby toes and despite widespread radiographic changes, arthritis was not seen in ankle or foot.

Summary:

- Bone dysplasias are uncommon, can have complex deformity and should be managed in a multi-disciplinary setting
- Arthritis of hips and knees are common in epiphyseal dysplasias but the foot and ankle tend to be spared, except in Trevor's disease
- Metaphyseal dysplasia can lead to primary or secondary deformity at the ankle

References:

Bonafe L, Cormier-Daire V, Hall C, *et al.* **Nosology and classification of genetic skeletal disorders: 2015 revision.** *Am J Med Genet A* *2015;167A-12:2869-92.*

Ingram RR. **The foot and ankle in multiple epiphyseal dysplasia.** *Foot (Edinb)* *1993;3-3:139-43.*

Rubin P. **On Organizing a Dynamic Classification of Bone Dysplasias.** *Arthritis Rheum* *1964;7:693-708.*

Session 4 - Discussion & Consensus

Stress fractures:

» **What is your primary investigation for a suspected stress fracture?**

Plain radiograph	21	(84%)
Plain radiograph + ultrasound	2	(8%)
MRI	2	(8%)

» **In the case of an athlete with a metatarsal stress fracture of the proximal third of the 2nd metatarsal, with 2 weeks of pain and unable to participate in sport: should the patient be immobilised in an Aircast (or similar) boot?**

Yes	17	(68%)
No	8	(32%)

» **How many surgeons routinely measure Vitamin D levels in patients with stress fractures?**

Yes	17	(68%)
Give Vitamin D without testing	5	(20%)
No	3	(12%)

With regard to stress fractures it was suggested that in addition to checking Vitamin D levels, bone mineral density should also be checked, however, a consensus was not reached with regard to this.

Soft tissue & malignant tumours:

» **With regard to the management of a soft tissue lump in the foot, how many surgeons would routinely get confirmatory imaging when the suspected diagnosis was a plantar fibroma?**

Yes	20	(80%)
No	5	(20%)

» **In the above setting would any surgeon make the diagnosis based solely on clinical findings and then discharge the patient?**

Yes	0	(0%)
No	25	(100%)

» **In the above setting how many surgeons would be satisfied on the basis of an ultrasound scan and then discharge the patient?**

Yes	19	(76%)
No, would not be satisfied with ultrasound	6	(24%)

» **How many surgeons would routinely biopsy a lesion with a suspected diagnosis of plantar fibroma?**

Would biopsy to confirm diagnosis	0	(0%)
Would not routinely biopsy if diagnosis confirmed on imaging	25	(100%)

» **How many surgeons would routinely follow-up a patient with plantar fibroma, which was confirmed on their imaging modality of choice?**

Yes	1	(4%)
No	24	(96%)

» **What are the currently assembled surgeons' preferred treatment options for symptomatic plantar fibroma?**

Corticosteroid injection	5	(20%)
Hyalase injection	2	(8%)
Shockwave	1	(4%)

» **How many surgeons would routinely scan a soft tissue lump > 3 cm in diameter which otherwise appears benign (e.g. ganglion), and which you are not considering surgery on?**

Yes, would scan	17	(68%)
No, would not scan	4	(16%)
Would not scan, but would aspirate and send for histology	4	(16%)

» **What are the currently assembled surgeons' preferred treatment options for the non-operative management of a ganglion around the foot and ankle?**

Corticosteroid injection + aspiration	13	(52%)
Aspiration only	3	(12%)
Neither	9	(36%)

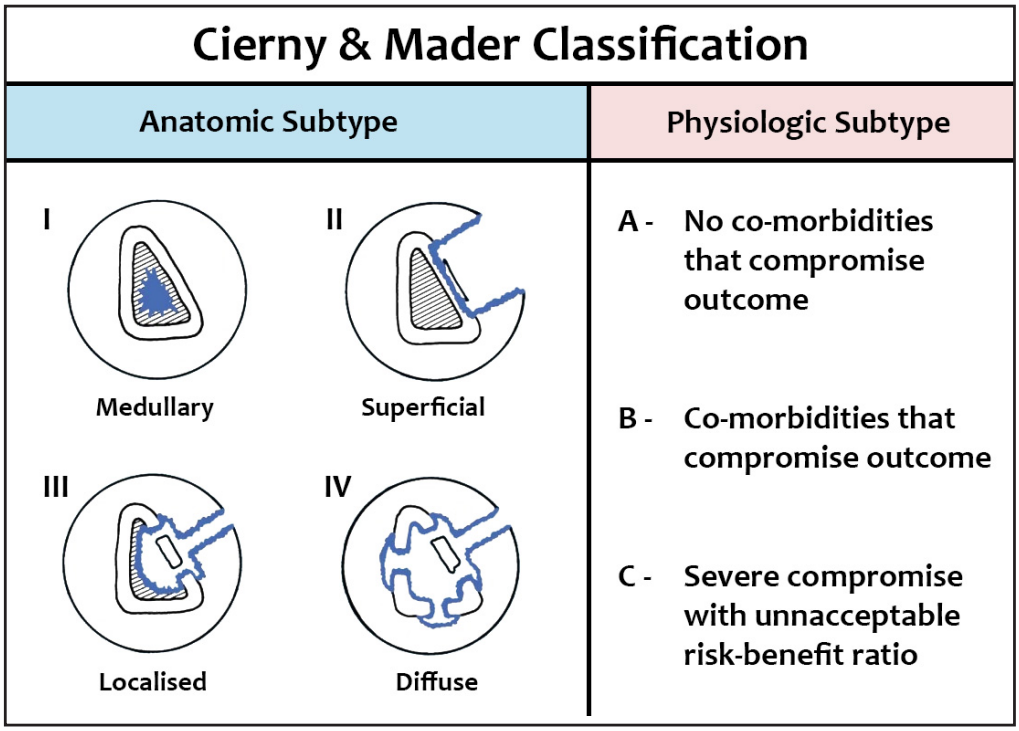
With regard to plantar fibroma, apart from the consensus questions above, it was determined that 10 consultants (40%) had performed surgery for symptomatic cases. Additionally, 6 consultants (24%) had performed surgery on more than one occasion.

With regard to malignant foot and ankle tumours, it was acknowledged that sarcomas of the foot and ankle are rare. The vast majority of consultants present had seen at least one soft tissue and one bony sarcoma during the span of their career, but very few had seen more than two. It was also commented upon that even in malignant tumours, night pain is rarely a feature of foot and ankle tumours, and so the index of clinical suspicion should be high even in cases without this clinical symptom.

Session 5: Non-Diabetic Foot Infection

5.1 - Osteomyelitis: when to debride / when to give antibiotics *(Patricia Allen)*

Osteomyelitis is an inflammatory bone condition caused by an infecting organism. Usually a single bone is affected and if it becomes chronic the result is compromised soft tissue surrounded by dead, infected bone, as well as reactive new bone. Osteomyelitis arises from either *haematogenous spread*, *direct inoculation of bone*, or *contiguous spread*. Most cases are caused by ***Staphylococcus aureus***; other common pathogens are ***Streptococci***, ***Enterobacteriaceae*** and ***anaerobic bacteria***.



A ***biofilm*** may be produced around implants and is discussed in *Session 5.3*. This is a highly structured community of bacteria that adopt a distinct phenotype and communicate through cell to cell signals and adhere to inert or living structures. They express surface components called *adhesins* which bind to proteins in host material. Once attached, a *polysaccharide matrix* is produced which forms a substrate in which colonies may develop, forming the biofilm. Once sufficient numbers are present, maturation and further development of the mature biofilm occurs, and some bacteria enter a dormant state. Bacteria are protected from the host immune system within the glycocalyx. In addition, some bacteria, such as ***Staphylococcus aureus,*** can invade living cells and can survive inside osteoblasts.

Diagnosis of osteomyelitis (features seen on imaging):

- ***Radiographs:*** osteopenia, cortical breaches, periosteal reaction, involucrum, sequestra
- ***Ultrasound:*** collections
- ***MRI:*** high signal on T2 / fat suppression, not good for cortical sequestra, overestimates extent in acute phase
- ***CT:*** bony destruction, sequestra

In the foot and ankle, ***acute osteomyelitis*** is usually treated with intravenous antibiotics for 2 weeks followed by oral antibiotics for a further 2-4 weeks. Antibiotics alone can be given if: the diagnosis is made within a few days of onset symptoms, there is no dead bone / abscess on imaging, there is no associated septic arthritis and there is rapid response to treatment. ***Surgery should be considered*** when there is a collection present, if there is progressive bony destruction, or if there is no response to treatment.

Chronic osteomyelitis cannot be cured by antibiotics alone since they cannot overcome biofilm or dead bone. ***Long term antibiotics*** are acceptable if suppressive treatment alone is desired (*e.g. physiologic subtype C*) otherwise surgery can be curative if all affected bone is resected and six weeks of antibiotics are administered post-operatively.

For ***debridement***, general surgical principles are followed. All infected or necrotic tissue is debrided and at least *five deep samples* should be sent for microbiology at start of surgery using separate instruments for each sample. Avoid touching instrument tips, and prevent skin contact with instruments / samples. Any abscesses should be drained, and copious lavage performed with saline or 0.05% aqueous chlorhexidine (*see discussion section*). Gloves, drapes and instruments should then be changed, and the tourniquet released to confirm adequacy of bone debridement by visualisation of bleeding bone. Finally, any dead space should be filled.

After debridement ***either intravenous of oral antibiotics may be used***. A Cochrane review in 2013 looked at 8 trials involving 282 patients with post-traumatic osteomyelitis. There was no difference between oral and intravenous antibiotics. These findings have been corroborated by the non-inferiority OVIVA trial published this year in the NEJM (Li *et al.*). Huang *et al.* undertook a meta-analysis comparing ***short and long durations of treatment***. They found short-course antibiotics were safe and effective in children with acute osteomyelitis, but longer courses of antibiotics were preferred in vertebral osteomyelitis, particularly if Staphylococcus aureus was the pathogen.

Summary:

- Acute osteomyelitis should be treated with antibiotics unless there is a local collection, progressive bony destruction, or treatment is failing
- Chronic osteomyelitis can be suppressed with long-term antibiotics but is otherwise treated with surgery followed by antibiotics

References:

Cierny G, 3rd, Mader JT, Penninck JJ. **A clinical staging system for adult osteomyelitis.** *Clin Orthop Relat Res 2003-414:7-24.*

Conterno LO, Turchi MD. **Antibiotics for treating chronic osteomyelitis in adults.** *Cochrane Database Syst Rev 2013-9:CD004439.*

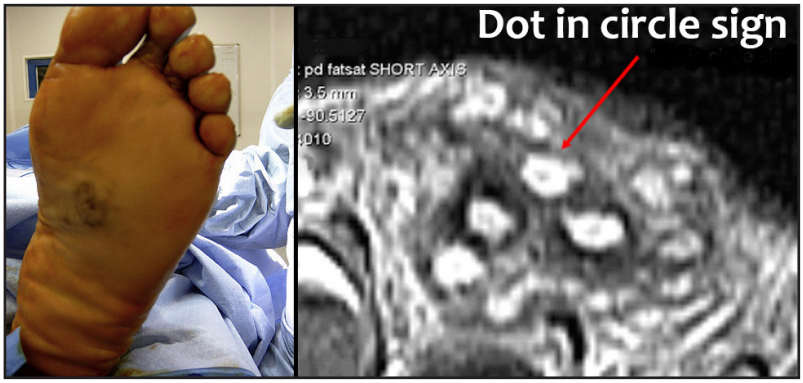
Huang CY, Hsieh RW, Yen HT, *et al.* **Short- versus long-course antibiotics in osteomyelitis: A systematic review and meta-analysis.** *Int J Antimicrob Agents 2019;53-3:246-60.*

Li HK, Rombach I, Zambellas R, *et al.* **Oral versus Intravenous Antibiotics for Bone and Joint Infection.** *N Engl J Med 2019;380-5:425-36.*

5.2 - Unusual causes of bone infection *(Piotr Chomicki-Bindas)*

Unusual infections in the foot are a diagnostic and therapeutic challenge. Some may be endemic to an area or susceptible population, whilst others may be rare in general. They often develop slowly, without pathognomonic signs. Increased global travel and migration may increase spread. This session cannot discuss all unusual infections, and so the focus shall be on two: ***tuberculosis (TB)*** and ***mycetoma***.

Musculoskeletal ***TB*** is uncommon; it accounts for ~10% of extrapulmonary TB cases and TB of the foot and ankle constitutes 2-8% of all musculoskeletal cases. A high index of suspicion is necessary for diagnosis as clinical sings can be misleading, and imaging can mimic other conditions. Diagnosis is established via tissue biopsy. Biopsy requests should specifically query TB since prolonged, TB-specific cultures are required. Treatment is ***pharmacological***, and NICE recommends initial *quadruple therapy* of ***rifampicin***, ***ethambutol***, ***pyrazinamide*** and ***isoniazid*** (with pyridoxine for prophylaxis against isoniazid-induced neuropathy) for two months. ***Streptomycin*** is used outside the UK, but rarely used within it unless there is intolerance to standard therapy or resistance to isoniazid. Subsequently, daily treatment with ***rifampicin*** and ***isoniazid*** is continued for at least a further four months. ***Surgery*** is rarely required unless an open biopsy is performed to establish diagnosis. Surgery may also be performed for the treatment of late sequelae or prophylaxis in cases where there is high risk of joint destruction.



Mycetoma is also known as ***Madura foot***. It can be caused by fungi (*eumycetoma*) or bacteria (*actinomycetoma*). Clinical presentation forms a typical triad: initially a *painless subcutaneous mass* is seen, with later *sinus formation* and *purulent discharge containing granules*. It is most common in Sudan, Mexico, Venezuela, and India but cases are also seen in the UK and Europe. *Actinomycetomas* are more commonly found in South and Central America, whereas *eumycetomas* are more common in Africa. All ages can be affected although men between 20-40 years are 3-5 times more likely to be affected. USS or MRI can show typical granulomata featuring a ***'dot in circle' sign*** *(above)*. *Actinomycetomas* are treated with ***antibiotics*** whereas *eumycetomas* are treated with ***systemic antifungals*** and ***surgical excision***. Systemic antifungals are necessary since surgery alone has up to a 90% recurrence rate for *eumycetoma*.

Summary:

- TB and Myceoma are unusual causes for foot and ankle bone infection and require a high index of suspicion to diagnose

References:

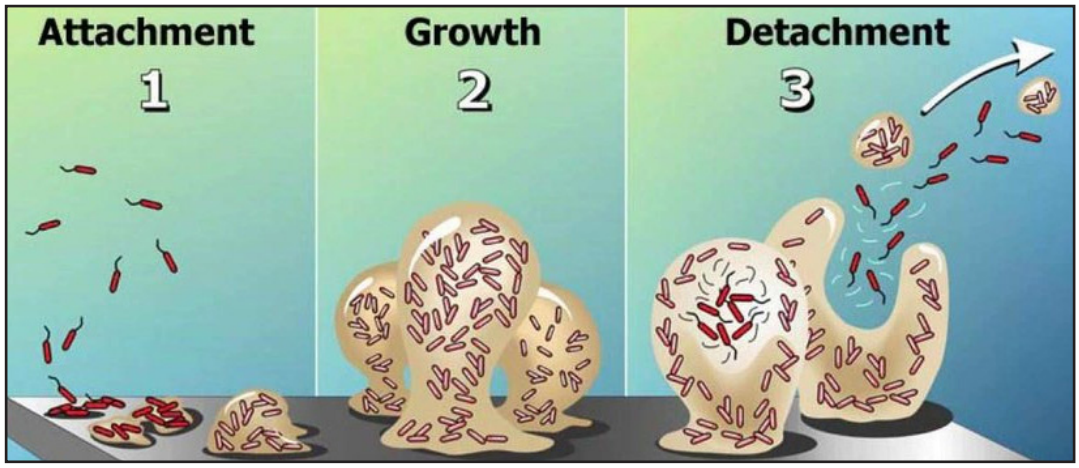
Leonard MK, Blumberg HM. **Musculoskeletal Tuberculosis.** *Microbiol Spectr* 2017;5-2.

Parker L, Singh D, Biz C. **The dot-in-circle sign in Madura foot.** *J Foot Ankle Surg* 2009;48-6:690 e1-5.

5.3 - Implant related infection *(Senthil Kumar)*

Periprosthetic infections may be thought of as ***early*** *(less than 4 weeks after surgery)* or ***late*** *(more than 4 weeks after surgery)*. The importance of classifying them thus is that prior to four weeks, it is less likely that a ***biofilm*** will have developed. The composition of a biofilm is discussed in greater detail in *Session 5.1.* Formation of a biofilm increases antibiotic resistance by up to a factor of 1,000. Furthermore, the more central bacterial cells are metabolically less active, and therefore harder to eradicate with bacteriostatic antibiotics. In order to remove the biofilm, the implant must be removed also.

In order to prevent biofilm formation steps should be taken to ***reduce bacterial seeding*** at time of primary surgery. This involves *minimising duration of surgery, minimising wound exposure* and *administering prophylactic antibiotics* prior to surgery. Post-operatively adhesion / bonding of the implant to the bone needs to occur before a biofilm can form. This is sometimes termed ***'the race for the surface'*** and is aided by implants with hydrophilic surfaces (titanium is better in this regard than stainless steel).



It is widely accepted that bone infection compromises bone quality and can lead to ***instability***, but it is less clear whether instability predisposes to infection. There may be instances where eradication of infection is particularly challenging and, in these scenarios, the surgeon must evaluate whether the construct has sufficient rigidity to facilitate an environment in which infection can be overcome. Although it seems counterintuitive to place metal-ware around an infection in the foot or ankle, it must be remembered that spinal infections are frequently, successfully treated with antibiotics and stabilisation when instability is a risk. ***This was discussed further in the discussion / consensus session.***

Summary:

- The understanding of implant related infections is continuously improving
- Distinguishing early from late infections is important as they are treated differently
- Biofilm plays an important role in established infections since the success of disease eradication is reduced unless it is managed aggressively
- After debridement, a stable environment for healing may help prevent further biofilm formation

References:

Izakovicova P, Borens O, Trampuz A. **Periprosthetic joint infection: current concepts and outlook.** *EFORT Open Rev* 2019;4-7:482-94.

5.4 - Local *versus* systemic antibiotics *(Anand Pillai)*

Despite advances in aseptic techniques and the use of antibiotic prophylaxis prior to surgery, rates of post-operative infection linger between 1% and 2%. This can increase to 33% in 2-stage revision surgeries for infection. ***Staphylococcus aureus*** is the causative pathogen in 75% of cases and recurrence of infection after decades of quiescence remains a problem.

Bacterial reservoirs:

» ***Abscess*** communities

» On the implant, within the ***biofilm***
Resistance and tolerance can occur within the biofilm and high local concentrations of antibiotics are required to penetrate the biofilm, which can be difficult to achieve with oral or intravenous antibiotics alone

» In cortical bone within the ***osteocyte-lacunar-canalicular network (OLCN)***
The OLCN has only recently been discovered and it is theorised bacteria can reside within this network for decades, with an inexhaustible supply of nutrients, whilst evading immune attack

The surgical strategy for eradication of infection includes *disruption of the biofilm* (requires implant removal), *debridement*, and *bony resection* to remove bacteria in the OLCN, However, it is unclear how much bony debridement is required to achieve this and greater resection inevitably results in instability, as discussed in *Session 5.3*. Thus, tissue and implant removal alone are often insufficient and providing concentrations of antibiotics well above the minimum inhibitory concentration is vital to penetrate biofilm and reduce bacterial load. It is unclear whether ***oral***, ***intravenous*** or ***local antibiotics*** are superior. Each has advantages and disadvantages as detailed in the table below (from Gomes *et al.*):

Therapy type	Advantages	Disadvantages
Intravenous	- Delivery of antibiotic to areas that cannot be reached with oral therapy - Choice of a large set of agents - Arrest or eradication of infection in most cases (in conjunction with surgical debridement)	- May require hospitalisation or OPAT - Lack of patient compliance - Systemic drug toxicity - Even with prolonged therapy relapse is not uncommon - Expensive
Oral	- Ease of administration - Reduced duration of hospitalisation - Reduced health care costs	- Therapeutically unpredictable - Capacity to replace IV therapy is controversial - Limited choice of agents
Local	- Avoids high serum concentrations of the antibiotic - Delivers antibiotic directly to the infection site - Reduced duration of hospitalization and health care costs	- Lack of proven efficacy in randomised clinical trials

Local antibiotics are thus advantageous for many reasons and various delivery systems have been employed. ***Bone cement (PMMA)*** is the earliest local carrier system used effectively in orthopaedics. Whilst there is good data to support its use, the non-biodegradable PMMA can attract *glycocalyx producing*

bacteria, as it provides a potential surface for bacterial colonisation, and thus requires removal. For this reason, ***biodegradable carriers*** were manufactured.

An ideal biodegradable antibiotic carrier should:

» Deliver high concentrations of drug reliably, at the point of delivery
» Degrade and breach or prevent biofilm
» Be non-toxic, systemically and locally
» Be completely biodegradable, i.e. no removal needed
» Have composite properties such as ability to stimulate / form bone
» Be easy to use with consistent elution, and be cost-effective

Gentamicin loaded collagen fleece had reportedly high rates of success although the initial product had a rapid rate of elution (95 % of gentamicin released in first 1.5 hours). Other products developed include ***polyester***, ***amylose***, ***alginate***, ***chitosan*** and ***borate glass***. However, the current vogue is for ***Calcium based carriers*** of which *Osteoset-T*, *Stimulan* and *Cerament* are some of the most widely used.

In a ***Calcium based carrier***, the carrier vehicle is either *Calcium sulphate* alone or in combination with *hydroxyapatite* ($Ca_{10}(PO_4)_6(OH)_2$). There is a paucity of publicly available literature that compares the pharmacokinetics of each individual carrier for each individual antibiotic. They universally tend to elute very high doses of antibiotics for the first 3-5 days with antibiotics detectable in the blood stream and urine for up to 30 days after. Levels above the minimum inhibitory concentration are present for up to 4 weeks with Gentamicin, and above biofilm inhibitory concentration for 14 days.

Summary:

- Local antibiotics carriers provide higher local concentration of cytotoxic agents initially than intravenous or oral methods
- There is increasing evidence to support the use of these agents particularly in high-risk patients such as diabetics
- They help reduce the dead space incurred by aggressive debridement
- The SOLARIO trial is currently recruiting patients to identify whether using local antibiotic therapy allows shorter courses of oral or intravenous antibiotics, in order to limit antibiotic resistance, side effects and cost. With recruitment expected to complete in 2022, results would not be expected before 2023

References:

Gomes D, Pereira M, Bettencourt AF. **Osteomyelitis: an overview of antimicrobial therapy.** *Braz J Pharm 2013;49-1.*

Masters EA, Trombetta RP, de Mesy Bentley KL, *et al.* **Evolving concepts in bone infection: redefining "biofilm", "acute vs. chronic osteomyelitis", "the immune proteome" and "local antibiotic therapy".** *Bone Research 2019;7.*

Panagopoulos P, Tsaganos T, Plachouras D, *et al.* **In vitro elution of moxifloxacin and fusidic acid by a synthetic crystallic semihydrate form of calcium sulphate (Stimulan).** *Int J Antimicrob Agents 2008;32-6:485-7.*

Stravinskas M, Horstmann P, Ferguson J, *et al.* **Pharmacokinetics of gentamicin eluted from a regenerating bone graft substitute: In vitro and clinical release studies.** *Bone Joint Res 2016;5-9:427-35.*

Session 5 - Discussion & Consensus

Implant related infection:

» **When debriding infected bone / implant, how many surgeons routintely take 5 samples for microbiology?**

Yes	16	(64%)
No	9	(36%)

» **In the setting of a late infection of a total ankle replacement, planned for a 2-stage revision, what are surgeons' preferences for treatment during Stage 1?**

Antibiotic loaded cement	17	(68%)
Antibiotic loaded void filler	4	(16%)
Both cement and void filler	3	(12%)
Neither / Other	1	(4%)

It was discussed that when taking samples for microbiology, 5 separate sets of instruments should be used (i.e. forceps and pots), one for each sample. One additional sample should be sent for histology. A number of consultants present worked at trusts where there were packs available with the additional sets of instruments required for this. Discussion also ensued as to whether histology was useful in a non-specialist centre. It was agreed that for these cases it was helpful to have a microbiologist with a special interest in bone infection, but in either case there should be a conversation with the microbiology team regarding the individual case.

Regarding debridement of osteomyelitis, it was acknowledged that it is sometimes difficult to know how much debridement is needed to eradicate all infection. It was suggested that taking a marginal bone specimen is helpful to determine whether the area left behind is disease free. When performing lavage of osteomyelitis, Hydrogen peroxide (H_2O_2) is contraindicated due to the risk of tissue damage and the risk of air embolism: the MHRA have issued guidance that Hydrogen peroxide is not to be used in closed body cavities or on deep or large wounds. Iodine based solutions, such as Betadine, are also inactivated upon contact with blood and so are less useful. Aqueous Chlorhexidine can be used.

Finally, there was a discussion regarding the role of stability in preventing recurrence of infection. This is an area that is still poorly understood, however any residual bacteria that remain dormant, may be reactivated by movement / mechanical factors and thus stability is preferred if achievable.

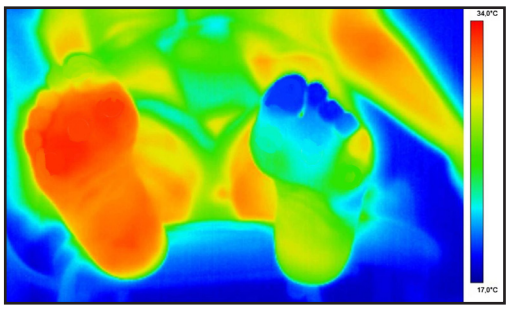
Session 6: Diabetic Foot

6.1 - When to operate on a Charcot foot *(Ashok Acharya)*

The main causes of diabetic foot morbidity are *Charcot neuropathic foot*, *infection*, *ischaemia* and *ulceration*. Charcot foot deformity results from a lack of protective sensation and is often accompanied by ulceration and / or infection. The most widely used clinical staging system for Charcot neuroarthropathy was described by ***Eichenholtz*** in 1966. He described three stages in the clinical and radiographic evolution of diabetic neuropathic feet. Stage 0 was later added by Shibata *et al.* to classify patients with clinical symptoms, but no radiographic changes (although changes may be seen on MRI).

Eichenholtz Stage	Radiographic Findings	Clinical Findings
0 (Prodromal)	Normal radiographs	Swelling, erythema, warmth
1 (Fragmentation)	Osteopenia, fragmentation, joint subluxation or dislocation	Swelling, erythema, warmth, ligamentous laxity
2 (Coalescence)	Absorption of debris, sclerosis, fusion of larger fragments	Decreased warmth, decreased swelling, decreased erythema
3 (Consolidation)	Consolidation, joint arthrosis, fibrous ankyloses, rounding of bone fragments	Absence of warmth, swelling and erythema, stable joint ± fixed deformity

Surgery for Charcot foot is aimed at ***bony stabilisation*** and ***correction of deformity*** to allow bracing, to reduce the risk of neuropathic ulceration, and to allow infected / ulcerated tissue to heal. In cases of acute infection or progressive ulceration the limb is at risk and therefore surgery is required irrespective of stage. However, in the absence of these criteria, the classical teaching is to wait until the active inflammation in the foot has settled *(Eichenholtz Stage 2/3)* before considering operative intervention. Clinically, resolution of inflammation is measured by return of the limb to normal temperature which can be assessed with ***thermography. Doppler spectrum analysis*** is another technique that has been described to monitor inflammation by assessing blood flow.



Thermography (skin temperature measurement) is performed using infrared thermometers and is the more commonly used and cost-effective technique. Skin temperature is measured in both feet at pre-determined sites and non-operative treatment is continued until the temperature of the affected foot is within 2.2°C of the normal foot. This is considered an indirect measure of the end of *Stage 1* and the beginning of *Stage 2*. Historically, foot temperature measurement was used to identify Charcot feet at risk of ulceration, but it is unclear how it came to be the investigation of choice to time corrective surgery. The cut-off of 2.2°C appears to have been first described by Lavery et al in 2007. They reported on the role of home-monitoring of foot temperatures in preventing ulceration. Patients were instructed to decrease their weightbearing activities and return for a medical review if a temperature difference of

more than 2.2°C developed between the affected and control feet. This threshold was chosen based on their previous work which indicated patients with a temperature gradient of > 2.2 developed ulceration.

Doppler Spectrum Analysis was described by Wu et al. They performed doppler ultrasonography of the first dorsal metatarsal artery in 15 patients with unilateral acute Charcot foot to monitor the resolution of the Charcot process. Doppler analysis was done every 2 weeks using a 10 MHz linear probe, and patients remained non-weightbearing in a bivalved cast for the duration of the study. With successful treatment the *monophasic* Charcot foot doppler spectrum was found to revert to the normal *triphasic* spectrum. This occurred after a mean duration of 13.6 weeks.

There is insufficient evidence to support one particular method of Charcot foot monitoring over others to guide timing of surgery. NICE looked at 9,817 studies in 2011 but could not make any recommendation on the optimal time for surgical management. Other authors have reached similar conclusions.

Earlier operative intervention during Stage 1 disease has been reported, with some success. Simon *et al.* and Mittlmeier *et al.* have both published case-series in which fusion was undertaken in Charcot feet during *Eichenholtz Stage 1*; all fusions were considered successful in both series, even when undertaken in the presence of ulceration. Earlier surgery has the advantage of avoiding significant deformity and minimising the impact of prolonged non-operative treatment on the patients' lives.

Summary:

- There is insufficient evidence to support the generally held recommendations for optimal timing of surgery in Charcot feet
- Although surgery is classically delayed until the disease has progressed to *Eichenholtz Stage 2*, earlier intervention may be required to prevent significant deformity
- Well-designed prospective trials comparing early versus delayed surgical intervention are required

References:

Lavery LA, Armstrong DG. **Temperature monitoring to assess, predict, and prevent diabetic foot complications.** *Curr Diab Rep 2007;7-6:416-9.*

Mittlmeier T, Klaue K, Haar P, *et al.* **Should one consider primary surgical reconstruction in charcot arthropathy of the feet?** *Clin Orthop Relat Res 2010;468-4:1002-11.*

National Institute for Health and Care Excellence. **Diabetic foot problems: inpatient management of diabetic foot problems.** *https://www.nice.org.uk/guidance/ng19/evidence/appendix-k-deleted-text-from-nice-guideline-cg119-pdf-15672915542 (date last accessed 29/09/2019).*

Pinzur MS, Schiff AP. **Deformity and Clinical Outcomes Following Operative Correction of Charcot Foot: A New Classification With Implications for Treatment.** *Foot Ankle Int 2018;39-3:265-70.*

Schneekloth BJ, Lowery NJ, Wukich DK. **Charcot Neuroarthropathy in Patients With Diabetes: An Updated Systematic Review of Surgical Management.** *J Foot Ankle Surg 2016;55-3:586-90.*

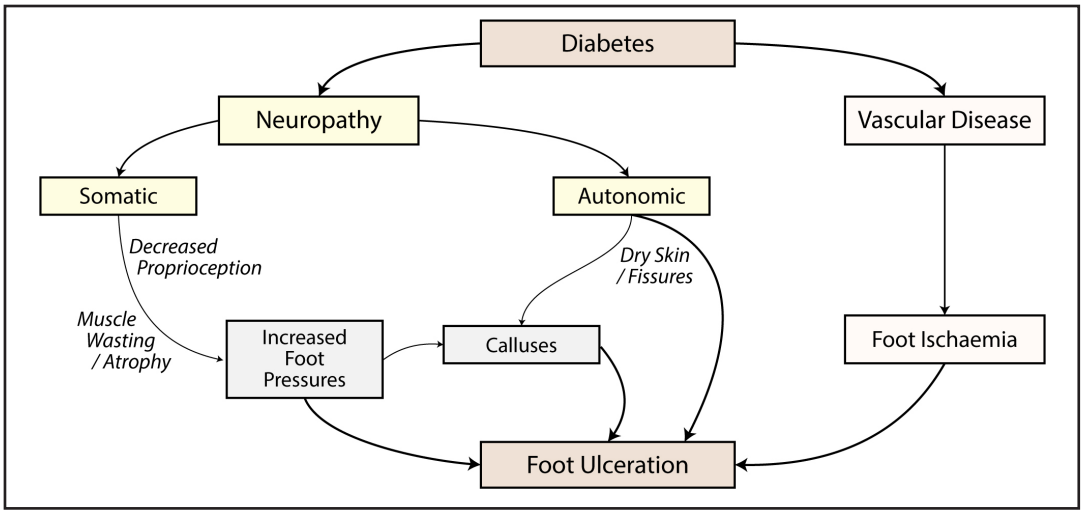
Simon SR, Tejwani SG, Wilson DL, *et al.* **Arthrodesis as an early alternative to nonoperative management of charcot arthropathy of the diabetic foot.** *J Bone Joint Surg Am 2000;82-A-7:939-50.*

Ugwu E, Adeleye O, Gezawa I, *et al.* **Predictors of lower extremity amputation in patients with diabetic foot ulcer: findings from MEDFUN, a multi-center observational study.** *J Foot Ankle Res 2019;12:34.*

Wu T, Chen PY, Chen CH, *et al.* **Doppler spectrum analysis: a potentially useful diagnostic tool for planning the treatment of patients with Charcot arthropathy of the foot?** *J Bone Joint Surg Br 2012;94-3:344-7.*

6.2 - Role of debridement / preservation in foot ulcers *(Krishna Vemulapalli)*

Diabetes creates a significant health burden for the patient and the NHS. Management of diabetic foot ulceration costs the NHS ~£1 billion annually and is associated with a 40% mortality at 5 years. Ulceration in diabetes may be *neuropathic* (54%), *ischaemic* (10%), or *neuro-ischaemic* (34%). The pathogenesis is complex and often a combination of microtrauma / increased pressure, neuropathy and peripheral vascular disease. Once established, ulcers demonstrate diminished capacity to respond to growth factors and slower migration of fibroblasts to injured areas. Cells at the periphery of ulcers, in particular, become pathological and inhibit healing.



Treatment is aimed at pressure relief, infection control and re-vascularisation. Debridement is the process whereby all tissue incompatible with healing are removed from a wound. It removes necrotic tissue and biofilm, reduces bacterial burden, and encourages formation of granulation tissue. Debridement may be ***autolytic***, ***biological***, ***hydrosurgical***, ***sharp*** or ***ultrasonic***. Debridement to healthier margins, capable of healing, is required and this needs to be coupled with offloading.

Types of debridement:

» ***Autolytic debridement*** uses the body's own enzymes and moisture to rehydrate and liquefy eschar. It uses occlusive / semi-occlusive dressings and / or antimicrobial products to create a balanced wound environment. It is commonly used and although slow, is appropriate for smaller wounds or for maintenance after surgical debridement

» ***Biological debridement*** uses larvae from the green bottle fly to remove necrotic / devitalised tissue. Larvae secrete proteolytic enzymes to liquefy dead tissue which they ingest. They then neutralise bacteria in their gut. They also change the local pH which increases oxygenation and activity of growth factors. This is a highly selective and rapid process, although it is expensive

» ***Hydrosurgical debridement*** uses a high energy saline stream to remove devitalised tissue. It is quick and selective, but cannot remove all the required tissue and requires specialised equipment

» ***Sharp debridement*** uses a scalpel or other surgical instruments to remove dead / devitalised tissue. This is carried out to just before the viable tissue level. The debridement is therefore subtotal and must be accompanied by adjunctive therapy but is quick, selective and does not require anaesthesia

» ***Ultrasonic debridement*** uses USS to dissect devitalised tissue off the wound bed. It is quick and selective and can be used for maintenance of debridement over a number of sessions

The goal of debridement is to *prevent / reduce infection* and *prevent amputation*. However, the best method is not clear. Piagessi *et al.* found that aggressive surgical debridement followed by antibiotics and appropriate orthoses improved outcomes as compared to dressings and orthotics alone. In another study the same unit also found that offloading with a total contact cast post-debridement improved outcomes. Saap *et al.* found that adequate debridement of bone, ulcers and the wound-bed increased the likelihood of achieving wound closure after treatment.

A meta-analysis by Elraiyah *et al.* found that ***autolytic therapy*** increased the healing rate of diabetic foot ulcers, and that ***larval therapy*** reduced the risk of amputation. It also found that ***surgical debridement*** reduced wound healing times. However, they could not recommend one form of treatment over the others due to lack of comparative studies. They recommended the choice of treatment should depend on local expertise and logistics, and patient preference.

NICE have recommended that care of the diabetic foot should be by a ***multidisciplinary team*** according to local expertise. Other therapies include *electrical therapy*, *PRP gels*, *wound matrices*, *hyperbaric Oxygen* and *growth factors*. These do not have sufficient evidence to make recommendations and should not be offered unless as part of a clinical trial.

Summary:

- Diabetic foot ulcers are associated with significant morbidity and mortality
- Aggressive treatment is required with debridement, off-loading and restoration of vascularity
- The method of debridement will be dictated by local preference and expertise

References:

Brem H, Stojadinovic O, Diegelmann RF, *et al.* **Molecular markers in patients with chronic wounds to guide surgical debridement.** *Mol Med 2007;13-1-2:30-9.*

Elraiyah T, Domecq JP, Prutsky G, *et al.* **A systematic review and meta-analysis of debridement methods for chronic diabetic foot ulcers.** *J Vasc Surg 2016;63-2 Suppl:37S-45S e1-2.*

Piaggesi A, Schipani E, Campi F, *et al.* **Conservative surgical approach versus non-surgical management for diabetic neuropathic foot ulcers: a randomized trial.** *Diabet Med 1998;15-5:412-7.*

Piaggesi A, Viacava P, Rizzo L, *et al.* **Semiquantitative analysis of the histopathological features of the neuropathic foot ulcer: effects of pressure relief.** *Diabetes Care 2003;26-11:3123-8.*

Ragnarson Tennvall G, Apelqvist J. **Health-related quality of life in patients with diabetes mellitus and foot ulcers.** *J Diabetes Complications 2000;14-5:235-41.*

Saap LJ, Falanga V. **Debridement performance index and its correlation with complete closure of diabetic foot ulcers.** *Wound Repair Regen 2002;10-6:354-9.*

Stojadinovic O, Brem H, Vouthounis C, *et al.* **Molecular pathogenesis of chronic wounds: the role of beta-catenin and c-myc in the inhibition of epithelialization and wound healing.** *Am J Pathol 2005;167-1:59-69.*

6.3 - Role of minimally invasive surgery in diabetic feet *(Artur Gądek)*

As discussed in the previous sections, management of the diabetic foot represents a challenging problem where the clinician must balance the risks of infection, poor vascularity and ulceration against the risk of progressive joint destruction and subluxation / dislocation. Early recognition and prevention are key, but if conservative treatment fails, surgery may be required to prevent further deformity / collapse of the foot.

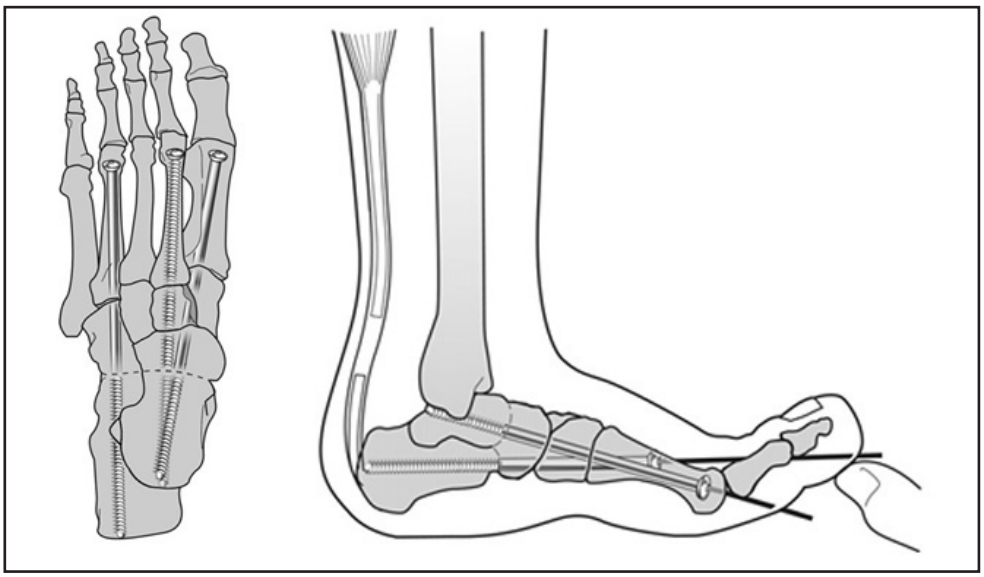
Apart from Charcot deformity, other pathologies that may be encountered include ***lesser toe deformities***, ***equinus deformity***, ***limited joint mobility***, ***intrinsic muscle atrophy*** and ***subluxation of the MTP joints***. Addressing all these deformities surgically runs the risk of significant wound complications and / or infection on account of poor vascularity and wound healing.

The recent advent of ***minimally invasive techniques*** provides the surgeon with options to reduce the complications of open procedures. However, the principles and goals of treatment remain the same and are aimed at *correcting deformity*, *preventing further deformity*, and *relieving areas of increased pressure* to prevent ulceration. In the case of a rocker-bottom deformity with midfoot collapse, this involves realigning the joints, restoring the medial and lateral columns, and creating a plantigrade foot.

Minimally invasive procedures available:

»	***Claw / hammer toes:***	PIPJ plantar release + FDB tenotomy ± phalangeal osteotomy
»	***MTPJ subluxation:***	DMMO + percutaneous extensor tenotomy + MTPJ arthrolysis
»	***Equinus deformity:***	Gastrocnemius release or percutaneous Achilles tendon lengthening
»	***Rocker bottom foot:***	Percutaneous burring of bony prominences (from non-weightbearing area)
»	***Charcot foot:***	Single or two stage correction with intramedullary, percutaneous beaming

A number of strategies have been described for dealing with the more complex Charcot deformities. Miller *et al.* described percutaneous correction in one or two stages, depending on the degree of deformity. For severe deformities they performed an initial stage which consisted of percutaneous tenotomies, wedge osteotomies, and application of a ring external fixator to distract and realign the joints over a period of 2 months. The definitive procedure consisted of minimally invasive arthrodesis with percutaneous cartilage removal and percutaneously inserted, cannulated, intramedullary bolts.



Open Procedures	Minimally Invasive Procedures
Long skin incisions	Percutaneous / short incisions
Close to / over operated site	Can be remote from operated site
Prolonged wound healing	Faster healing
Higher risk of infection	Lower risk of infection
May compromise future operative fields	Can be converted to traditional surgery

Summary:

- Minimally invasive techniques may be utilised in the treatment of deformities in diabetic feet and a number of treatments have been described
- Potential advantages include reduced wound complications and faster healing, which may allow intervention earlier in the disease process than traditional techniques
- Studies comparing minimally invasive and open procedures in the diabetic foot are required

References:

Batista F, Magalhaes AA, Nery C, *et al.* **Minimally invasive surgery for diabetic plantar foot ulcerations.** *Diabet Foot Ankle 2011;2.*

Biz C, Gastaldo S, Dalmau-Pastor M, *et al.* **Minimally Invasive Distal Metatarsal Diaphyseal Osteotomy (DMDO) for Chronic Plantar Diabetic Foot Ulcers.** *Foot Ankle Int 2018;39-1:83-92.*

Botezatu I, Laptoiu D. **Minimally invasive surgery of diabetic foot - review of current techniques.** *J Med Life 2016;9-3:249-54.*

Lamm BM, Gottlieb HD, Paley D. **A two-stage percutaneous approach to charcot diabetic foot reconstruction.** *J Foot Ankle Surg 2010;49-6:517-22.*

Lamm BM, Siddiqui NA, Nair AK, *et al.* **Intramedullary foot fixation for midfoot Charcot neuroarthropathy.** *J Foot Ankle Surg 2012;51-4:531-6.*

Miller RJ. **Neuropathic Minimally Invasive Surgeries (NEMESIS):: Percutaneous Diabetic Foot Surgery and Reconstruction.** *Foot Ankle Clin 2016;21-3:595-627.*

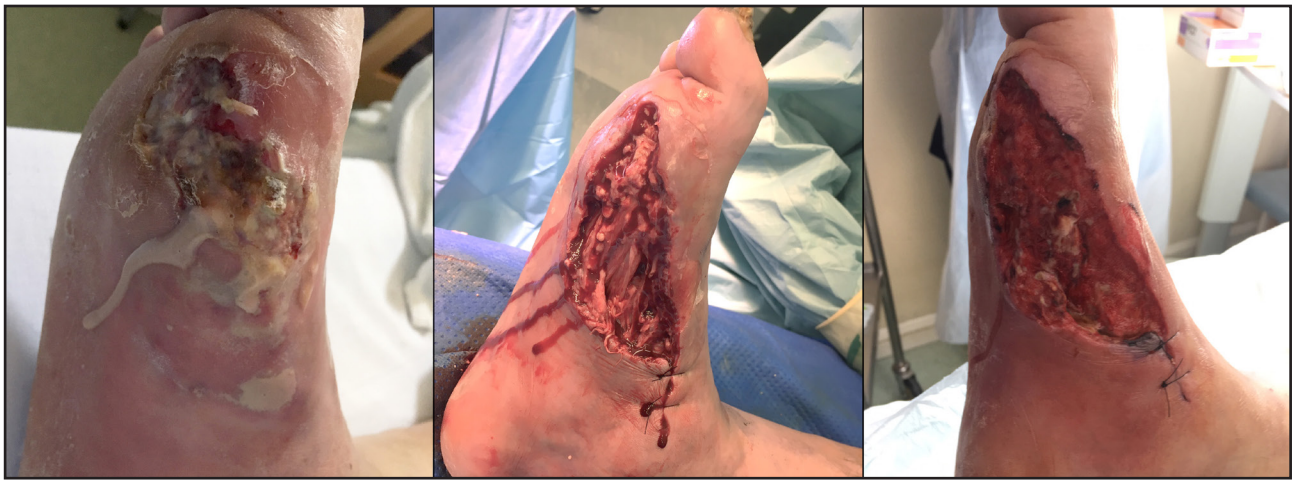
6.4 - Foot attack *(Dishan Singh)*

The term ***'foot attack'*** is a novel one designed to convey a sense of urgency in the management of a diabetic patient with severe, rapidly progressing foot infection. However, it is not well defined in the orthopaedic literature. The term was first introduced as ***'diabetic heart attack of the foot'*** in a blog post to illustrate the severity and seriousness of diabetic foot infections. This was modified to *'foot attack'* by Diabetes UK a few years later and has since been described by NICE in 2015 and BOFAS in 2016. NICE, however, did not use the term 'foot attack', and BOFAS used a more narrow definition *(see box below)*.

Defining and treating *'Foot Attack'*:

» **Definition (by Vas *et al.*):** '*an acutely inflamed foot with rapidly progressive skin and soft tissue necrosis*'

» **BOFAS recommendation:** 'a foot abscess in a diabetic can quickly become limb or life threatening. Prompt drainage is essential. Uncertainty and procrastination, such as for imaging, can de disastrous.'



Vas *et al.* from King's College Hospital diabetic foot unit, defined a ***typical foot attack*** as above, but also used the term ***atypical foot attack*** in the absence of infection, for ischaemia / Charcot feet.

Types of diabetic foot attack (Vas *et al.*):

» ***Typical* - infected foot attack** is a severe infection with spreading necrosis. Urgent treatment with debridement and antibiotics is required initially, followed by correction of ischaemia if present

» ***Atypical* - ischaemic foot attack** is a severe, critical ischaemia with or without tissue loss. There is a narrow window to prevent limb threatening ischaemia and it requires urgent revascularisation

» ***Atypical* - acute Charcot neuroarthropathy** usually presents as a hot, swollen foot without ulceration. This needs urgent diagnosis, exclusion of infection and off-loading to prevent joint destruction

Currently there are no international guidelines for managing typical, infective diabetic foot attacks. It is however important that all suspected cases have urgent hospital admission and work-up with surgical planning. Clinical signs include: *a red, hot swollen foot*; *subcutaneous bullae*; *collection of pus*; *crepitus; or gas in the tissues on plain radiograph*. If required, debridement should be undertaken as an emergency by the general orthopaedic or vascular team, with revascularisation as appropriate. Intensive wound management follow-up is required.

Phases of management of infected foot attack (from Vas *et al.*):

» ***Phase 1:*** Admission, antibiotics, resuscitation, imaging
MRI and arterial duplex are desirable but should not delay surgical treatment

» ***Phase 2:*** Radical debridement of all infected tissue, down to health bleeding tissue and copious lavage
Plan relook at 48 hours and arrange for other investigations if not already done

» ***Phase 3:*** Revascularisation (if required) and daily wound management with the multidisciplinary team
Target antibiotics, perform medical optimisation and plan for skin graft if required

The surgical principles include using long, deep, longitudinal incisions. Explore the tendon sheaths if required as infection often tracks along tendons. Devitalised tissue and any tissue suspected of being compromised must be excised. The wound should be left open for a second look.

Discussion ensued on this topic; it was accepted that acute Charcot foot and ischaemia are urgent conditions, but they do not have the same degree of urgency as a diabetic foot abscess. Methods of distinguishing acute Charcot foot from infection was also discussed. It was acknowledged that this is often difficult in the absence of soft tissue necrosis as they can appear similar and the CRP in diabetic foot infections is often normal. Signs to look for include the improvement of symptoms on elevating and immobilising the leg, which would suggest Charcot, and subcutaneous gas on the radiographs, which would suggest infection. Majority of gathered surgeons felt the term 'foot attack' should only be used to refer to purely infected cases.

Summary:

- Diabetic foot attack is a term used to convey an appropriate sense of urgency regarding a limb threatening situation
- It is as yet not clearly defined
- It requires urgent investigation and treatment which may be instituted by a non-specialist, but which ultimately will require a multidisciplinary approach

References:

Diabetes UK. **Fast Track for a Foot Attack: reducing amputations.** *https://www.diabetes.org.uk/professionals/position-statements-reports/specialist-care-for-children-and-adults-and-complications/fast-track-for-a-foot-attack-reducing-amputations (date last accessed 29/09/2019).*

National Institute for Health and Care Excellence. **Diabetic foot problems: prevention and management.** *https://www.nice.org.uk/guidance/ng19/chapter/1-Recommendations (date last accessed 29/09/2019).*

British Orthopaedic Foot & Ankle Society. **Managing the Acute Diabetic Foot Abcess - "Foot Attack".** *https://www.bofas.org.uk/Portals/0/BOFAS%20DM%20Feet.pdf?ver=2016-10-13-230932-247 (date last accessed 29/09/2019).*

HuffPost. **Beware of the Diabetic Heart Attack of the Foot.** *https://www.huffpost.com/entry/diabetes-foot-attack_b_1030267? (date last accessed 29/09/2019).*

Vas PRJ, Edmonds M, Kavarthapu V, *et al.* **The Diabetic Foot Attack: "'Tis Too Late to Retreat!".** *Int J Low Extrem Wounds 2018;17-1:7-13.*

Vig S, Alchikhal T, Turner B. **The foot attack: Where are the defence mechanisms?** *Br J Diabetes Vasc Dis 2014;14-2:72-4.*

6.5 - Amputation *(James Ritchie)*

Every year there are over 7,000 amputations involving diabetic feet in the UK. These are often due to *spreading infection*, *intractable ulceration* or infection, and *deformity*. In order to prevent amputations, management by a multidisciplinary team is required, which includes *diabetic control*, *perfusion optimisation* (micro- and macro-vascular perfusion problems can coexist), *offloading footwear* and *surveillance*.

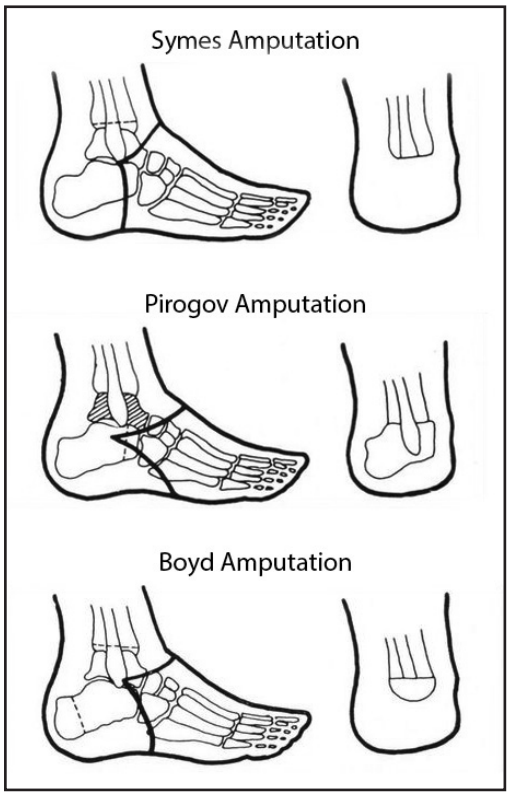
However, despite best efforts some patients will still require amputation. The goal of surgery in these cases is to produce a stump that will: *heal well*, *be pain free*, and *accept a prosthesis* optimal for the individual patient's needs. Prior to amputation patients should be optimised medically and receive psychological assessment and counselling.

When performing amputations basic principles must be followed: all suspect tissue, including ulcer tracts, must be debrided; multiple samples should be sent for culture and thick soft tissue flaps should be maintained. Care should be taken not to undermine the soft tissues, with a preference for shortening to achieve soft tissue coverage. A tourniquet may be applied but should not be inflated if possible.

Levels at which amputations may be performed:

- ***Lesser toes:*** This is commonly done, the level is determined by extent of the disease. Plantar flaps are made longer than dorsal flaps where possible and tendons are cut cleanly and allowed to retract. They create little disability and defects may be treated with spacers
- ***Hallux:*** This results in a reduced push off during stance, which can impair walking speed. If possible, some proximal phalanx should be preserved (insertion of FHB)
- ***Ray amputation:*** Performed if the metatarsal head is compromised. It avoids destabilising the adjacent toes, but results in transfer metatarsalgia. 20% of first ray amputations need revision but 5th ray amputations are well tolerated. When amputating the 5th, preserve peroneus brevis insertion where possible
- ***Partial foot amputations:*** May be performed at a number of levels. In general, the length of bone left does not impact function to a significant extent and therefore it is acceptable to resect more proximally if it allows better soft tissue coverage. Specific examples of partial foot amputations include trans-metatarsal amputation, amputation through the TMTJ joints (Lisfranc amputation), and amputation through the talonavicular and calcaneocuboid joint (Chopart amputation). In all of these cases the Achilles tendon may need to be lengthened and tendons may need to be transferred to prevent equinus deformity.
- ***Ankle amputations:*** Consist of the Syme, Pirogov and Boyd amputations. In the Syme, there is disarticulation through the ankle with trimming of the malleoli. The heel pad is used to cover the stump and it allows limited ambulation without a prosthesis. In the Pirogov and Boyd amputations, part of the calcaneus is left behind and fused to the tibia. This gives greater length but the complications of both are much higher.
- ***Trans-tibial:*** This is the highest level at which most patients can achieve normal ambulation. The level of resection depends on the soft tissues. In most cases the posterior flap is created longer. It allows for a multitude of prosthetic options.

Negative pressure dressings may be used as they have been shown to improve the rate of healing and the proportion of wounds which heal, but they do not necessarily result in an improvement in quality of life. (Liu *et al.*). Shaikh *et al.* demonstrated that primary closure is acceptable in forefoot amputations provided there has been meticulous debridement and tension free closure. In most cases the amputation and debridement can be performed in one procedure.



Some studies have compared partial foot amputation with trans-tibial amputations and found no significant difference in energy expenditure and quality of life. Brown *et al.* found that patients with trans-metatarsal amputations had better ambulation than those with trans-tibial amputations, but that 25% needed revision of the stump to a more proximal level.

Summary:

- When performing amputation, the level of resection is dictated by the soft tissues
- Soft tissue coverage is more important than preserving length
- There is some weak evidence to support use of negative pressure dressings
- Partial foot amputations may have better function than trans tibial amputations, but it depends on the level of resection and the overall benefit is unclear

References:

Armstrong DG, Lavery LA, Diabetic Foot Study C. **Negative pressure wound therapy after partial diabetic foot amputation: a multicentre, randomised controlled trial.** *Lancet* 2005;366-9498:1704-10.

Brown ML, Tang W, Patel A, *et al.* **Partial foot amputation in patients with diabetic foot ulcers.** *Foot Ankle Int* 2012;33-9:707-16.

Dillon MP, Barker TM. **Preservation of residual foot length in partial foot amputation: a biomechanical analysis.** *Foot Ankle Int* 2006;27-2:110-6.

Liu Z, Dumville JC, Hinchliffe RJ, *et al.* **Negative pressure wound therapy for treating foot wounds in people with diabetes mellitus.** *Cochrane Database Syst Rev* 2018;10:CD010318.

Shaikh N, Vaughan P, Varty K, *et al.* **Outcome of limited forefoot amputation with primary closure in patients with diabetes.** *Bone Joint J* 2013;95-B-8:1083-7.

Session 6 - Discussion & Consensus

Timing of surgery in Charcot foot:

» **How many surgeons would operate on an unstable, unbraceable Charcot foot, irrespective of stage?**

Yes	23	(92%)
No - always wait until fragmentation phase is over	2	(8%)

» **How many surgeons would consider a tendoachilles lengthening to prevent worsening deformity in midfoot Charcot during the fragmentation phase (Stage 1)**

Yes	20	(80%)
No	5	(20%)

It was noted that temperature monitoring may need to continue for an extended period in patients with acute Charcot. It was the general consensus that monitoring was required weekly initially, and subsequently every time the plaster was changed - as the disease stabilises, the interval between monitoring could be increased. It was also noted that patients treated in a total contact cast should have thromboprophylaxis for the first 6 weeks of treatment, in accordance with NICE guidance.

With regard to ***minimally invasive surgery (MIS)***, there was a discussion as to whether percutaneous techniques provided sufficient rigidity. The general consensus was that intramedullary rods should be supplemented with plates, although this would no longer be minimally invasive. It was acknowledged that preservation of the soft-tissue envelope with percutaneous techniques may mean that healing can occur with less rigid fixation, but there is as yet insufficient evidence in this area. Although majority of the assembled body of consultants had little experience with MIS of the diabetic foot, it was felt that there may be a role in using percutaneous, techniques should surgery be required for stabilisation of a Grade 1 Charcot foot.

Foot attack:

» **How many surgeons agree that the definition of 'foot attack' should refer to purely infected cases?**

Yes	24	(96%)
No - it should be subclassified into 3 types	1	(4%)

Discussion ensued regarding foot attack: this discussion is detailed at the end of *Section 6.4*. It was also noted that only five of the consultants present worked at trusts which had a '***foot attack protocol***'.

Convened participants of the 2019 Round Table Meeting

Ashok Acharya
Patricia Allen
Maneesh Bhatia
Rick Brown
Piotr Chomicki-Bindas
Callum Clark
Rob Clayton
Tim Clough
Nick Cullen
Raman Dega
Artur Gądek
Andy Goldberg
Steve Hepple
Billy Jowett
Mike Karski
Senthil Kumar
Henryk Liszka
Anand Pillai
James Ritchie
Ben Rudge
Dishan Singh
Phil Vaughan
Krishna Vemulapalli
Matt Welck
Tim Williams

OrthoSolutions Limited
West Station Business Park
Spital Road, Maldon
Essex, CM9 6FF
United Kingdom

T: +44 1621 843 599
F: +44 1621 858 953
E: sales@orthosol.com

www.orthosolutions.com

BONESUPPORT Holding AB
Scheelevägen 19
SE-223 70 Lund
Sweden

T: +46 46 286 53 70
F: +46 46 286 53 71
E: info@bonesupport.com

www.bonesupport.com