2023

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Orthopaedic & Sports Medicine Service

LATEST ORTHOPAEDIC UPDATES

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Time	Event	Who
07:30 – 08:00	Arrival / Refreshments	
08:00	Welcome Message	Dr Doron Sher
	Hip Pain in Children and Adolescents	Dr Chris Spelman
	The Unhappy Total Knee	Dr Andreas Loefler
	Nutrition therapy optimising recovery after surgery	Dr Doron Sher
09:10	Panel Discussion	
	Ulnar sided wrist pain	Dr Kwan Yeoh
	Augmentation of the enthesis in rotator cuff repair	Dr Ivan Popoff
	Correction of deformity in the foot and ankle	Dr Todd Gothelf
	Common fractures of the foot and ankle	John Negrine
10.50	Panel Discussion	
11.10-11.40	**Morning Tea**	
	Physio question, education, Sponsor	
	Managing pars stress fractures	Dr Leigh Golding
	Taking a deeper history in Musculo-Skeletal (MSK) Consultations	Dr John Best
	Concussion	Dr Paul Mason
	PRPP Injection	Dr Paul Annett
13:00	Panel Discussion & Close	

Dr Christopher Spelman

M.B.B.S., MSpMd, F.R.A.C.S. (Ortho.)
Hip, Knee and Paediatric Orthopaedic Surgery

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Hip pain in Children and Adolescents

Age is key discriminating factor when assessing children for hip pain.

Certain conditions are much more common in different age groups, and your assessment can be tailored based on the likely differential diagnoses based on the child's age.

Hip Dysplasia

Hip dysplasia can present at any age. Although ideally would be picked up at birth or in infancy, there is no universal screening program in Australia and often children slip through.

Risk Factors

- First born
- Female
- **Breech**
- **Family History**
- Oligohydramnios
- Macrosomia

Key examination findings in babies

- Barlow's test
  - Can the hip be dislocated with flexion to 90, adduction and posterior directed force
- Ortolani's test
  - Can a dislocated hip be reduce with flexion o 90, abduction and anteriorly directed force
- Galeazzi's test
  - Hip flexed to 90, knee flexed to 90. Are the femurs and tibias the same length?

Associated packaging disorders – Does the baby have any of the following?

- Plagiocephaly
- Congenital Muscular Torticollis
- Metatarsus adductus
- Calcaneovalgus feet
- Infantile scoliosis or spinal dysraphism

Key examination findings in children and adolescents

- Limp (unilateral short leg gait if hip is dislocated, antalgic if dysplasia/labral pathology)
- Pain at extremes of motion
- Pain on FADIR or EABER
- Increased femoral neck anteversion (assessed on prone rotational profile)

Investigations

- Under 6 months – Bilateral hip DDH ultrasound
- 6 months to 12 years – Pelvis AP + frog lateral x-ray
- 12 years and over – Pelvis AP with coccyx centred and 1-2cm from symphysis (+ false profile x-ray if going for periacetabular osteotomy). MRI on patients over 16 or concern regarding labrum or cartilage.

Management

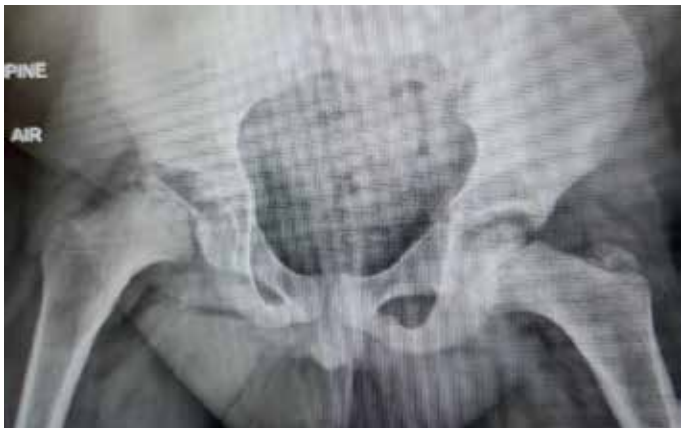
- Adductor stretching if mild and not requiring harness
- Bracing if before walking age (Pavlik harness or rhino brace).
- 6 months to 18 months (hip dislocated) Closed reduction + arthrogram +/- open reduction
- 18 months to 4 years – Open reduction + pelvic and femoral osteotomies as indicated

Perthes Disease

Idiopathic avascular necrosis of the femoral head

Important information:

- Bilateral in 20%
- Most common age at presentation is 4-8 years (can be anywhere from 2-16)
- Best outcomes in boys under 4 (most growth and remodelling potential)
- Multiple classification systems – no need to memorise



Clinical History

- Insidious onset groin pain and stiffness.
- Often presents after sport/exercise, especially trampolines

Examination

- Children often small for age and slim
- Limp due to antalgic, stiff hip gait
- Reduced ROM, especially abduction and FIR
- Pain on ROM (hip synovitis)

Investigation

- Pelvis AP + frog lateral x-rays. Functional x-rays with hips in maximal abduction to assess reducibility
- MRI if diagnosis unclear

Treatment goals

- Containment
  - Keep the femoral head within the acetabulum to allow remodelling
- Pain relief
  - Limit activity, analgesia with NSAIDS
- Range of motion
  - Preserve hip ROM, particularly abduction which is key to maintain containment

Non-surgical Management

- Hip range of motion exercises
- Reduce impact exercise during fragmentation phase (crutches, wheelchair etc)
- Corticosteroid injection into hip joint under CT or ultrasound guidance
- Broomstick casts/Abduction splinting
- Slings and springs

Surgical Management

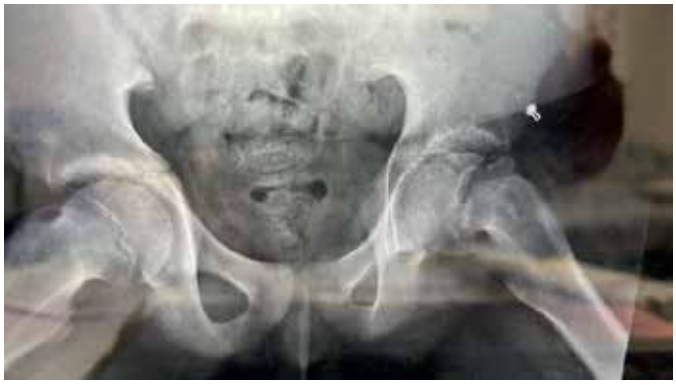
- Varising proximal femoral osteotomy – to reduce femoral head into acetabulum and allow remodelling
- Periacetabular osteotomy – Correct secondary hip dysplasia caused by remodelling
- Relative femoral neck lengthening – correct abductor moment arm by restoring position of greater trochanter relative to femoral head

<u>Slipped Capital Femoral Epiphysis (SCFE)</u>

Displaced of the femoral epiphysis relative to the femoral neck

Important information

- Onset 10-16 (must have open femoral physis)
- Bilateral 20-30%
- Strongly associated with obesity
- Associated with endocrinopathies (thyroid etc)
- Key predictor of outcome stable vs unstable (can the patient weight bear)
  - 50% AVN rate in unstable slips, 0% in stable



Clinical history

- Often presents with knee pain rather than hip pain
- Limp, particularly after sport
- Need to ask duration of symptoms (acute vs chronic)

Examination

- Limp/antalgia
- Obligate external rotation with hip flexion

Investigation

- Pelvis AP + frog lateral x-ray
- Looking at Klein's line, Southwick angle
- Bone scan pre-operatively if planning open reduction to assess head perfusion

Management

- Always surgical
    - Pin insitu vs capital realignment
- Proximal femoral flexion/valgising osteotomy to correct residual deformity

Septic arthritis vs Transient Synovitis

Septic arthritis is a bacterial infection of the joint. It is a surgical emergency and requires urgent treatment. Transient synovitis is a diagnosis of exclusion. Any unwell child with fever and limp should be immediately sent to the emergency department.

Transient synovitis

- Rare under 2
- Must be afebrile
- Must have normal bloods (especially CRP and WCC)
- Should resolve with NSAIDS
- Normally have a preceding viral illness in the 2 weeks prior

Septic Arthritis

- Febrile >38.5
- Acute onset
- Raised CRP and WCC

Investigation

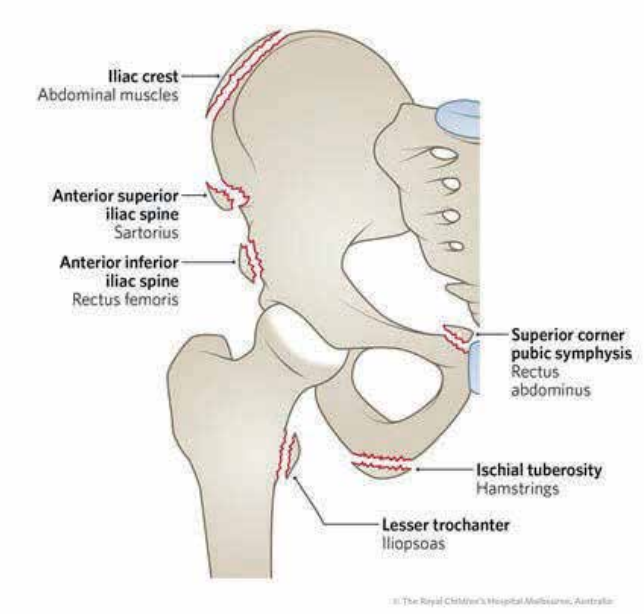
- Bloods
- Ultrasound and x-ray of affected joint

Trauma

Most diagnoses can be made from history

Avulsion injuries in teenagers are very common, especially AIIS in soccer players, sprinters and jumpers.

Apart from complete, displaced hamstrings avulsions most can be treated non surgically.



Management is symptomatic and avoidance of stretching/displacing avulsed fragment until bony union has occurred.

Labral tears in teenagers should always be investigated for hip dysplasia.

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NOTES:

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Joint Replacement & Spine Surgery

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The Unhappy Total Knee

The human knee is a complex joint, which connects long lever arms, the femur and the tibia. It glides, twists, and bends, but is also constrained by the cruciate and collateral ligaments. Injury, overuse and disease can damage the articular surfaces resulting in pain, stiffness and deformity.

The ultimate treatment for the arthritic knee is a replacement, which has been done for more than 50 years. Both the prostheses and the surgical technique have improved in that time. We have better materials, more precise implantation, as well as better perioperative care. Whist 80% of patients are satisfied and are likely to have long term benefits from a knee replacement, some 20% of patients remain unhappy.

Apart from major complications, like infection and fracture, there are numerous other factors for unsatisfactory outcomes. Surgical factors include implant sizing, especially oversizing of the femoral component, implant positioning, especially malrotation of the femoral component, and balancing the knee in flexion and extension. Patella mal-tracking and adjustment of ligaments are additional challenges, which need to be addressed at the time of surgery. Failure to correctly address any of these steps can result in mechanical disfunction of the knee replacement.

Adequate postoperative care of the wound and early physiotherapy is essential in regaining good function of the knee replacement. Pain and stiffness are the most common complaints, as well as weakness, instability, swelling and clicks. Some knees look good, but feel bad, whilst other look bad, but feel good.

There are a number of patient related factors, which may contribute to unsatisfactory outcomes. These include comorbidities, anxiety and depression, social circumstances, poor pain tolerance and unrealistic expectations. Lack of appropriate exercise and rarely overdoing it can also contribute to unsatisfactory outcomes.

Patient selection, patient education, preoperative planning and good postoperative care will help to improve outcomes. Patients need to have realistic expectations and feel supported in the perioperative period. In addition to physical support many patients need emotional and psychological support. Hospitals, surgery and rehab programs are often unfamiliar to patients and they feel loss of control. This can lead to fear and misunderstandings. Good and regular communication is essential.

Some knee replacements need to be revised. Infection or periprosthetic fractures are the most common causes for revision. Less common causes for revision are stiffness or instability. These cases require careful evaluation as revision does not always lead to better function. Prosthetic failure is relatively rare. Hypersensitivity is very rare but does occur.

Unhappy patients need additional support. They need careful evaluation. They need more physical support. Some also need psychological attention.

NOTES:

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Using Nutrition to Optimise Recovery After Orthopaedic Surgery

Your nutritional status is a strong predictor of postoperative outcomes. Adequate nutritional consumption is essential after surgery to mitigate the loss of muscle mass, strength, and functionality. The goal of post-operative nutrition is to promote nitrogen balance, reduce the loss of lean mass, and facilitate rapid tissue healing and recovery.

Protein intake is probably the most important factor for modulating surgical stress and supporting recovery but most surgical patients significantly under-consume protein, taking in about 1/5 to 1/3 of estimated requirements.

Surgery stimulates a cascade of inflammatory, immune, and metabolic responses that result in a hypermetabolic-catabolic state. There is upregulation of glucagon, cortisol, and proinflammatory cytokines which leads to significant catabolism of hepatic and muscle glycogen. Gluconeogenesis in the liver is also significantly upregulated, relying on lactate, amino acids, and glycerol as primary precursors.

These all lead to an interference with insulin secretion, preventing blood glucose clearance (Insulin resistance) which lasts for hours to weeks depending on the individual. This is far worse in patients with diabetes or pre-existing hyperglycemia.

This is a problem because a raised blood sugar impairs immune function and significantly impacts on surgical outcomes. It causes higher rates of infection, heart attacks, kidney failure, re-operation and ultimately, death. This applies to every type of surgery, not just Orthopaedics. There have quite literally been hundreds of studies on this topic. On the other hand earlier post-operative feeding with the right types of foods is associated with reduced infection complications, improved healing, and decreased length of stay.

Skeletal Muscle

If we go right back to basics: Skeletal muscles are the muscles used to move your bones and joints. As a tissue it has a unique ability to alter its metabolism and the size of myofibers in response to changes in mechanical loading. Chronic mechanical loading leads to an increase in skeletal muscle mass and an enlargement of muscle fibers, while prolonged mechanical unloading results in a significant decrease in muscle mass and the cross-sectional area of muscle fibers (muscle atrophy).

If you look at a muscle cell under a microscope you will see striations. This pattern is formed by a series of basic units called Sarcomeres that are arranged in a stacked pattern throughout the muscle tissue. These are repeated throughout the muscle and there are thousands of sarcomeres in each muscle cell. Muscle is a specialized tissue which can change its length, that is contract/shorten or lengthen and it is the change in length of the proteins in the sarcomere that causes the muscle to change it's length.

Skeletal muscle cells are long and cylindrical. They are protein dense and have many nuclei which allows for greater production of proteins and enzymes. There are also mitochondria and endoplasmic reticulum. Within the muscle fiber the proteins are organized into structures called myofibrils which then contains thousands of sarcomeres

connected in series. Within each sarcomere you will find thin and thick filaments called Actin and Myosin. The details here are a bit complicated but essentially a chemical reaction cause these 2 to 'slide' over each other which changes the length of the muscle.

Protein

The basic building block of a protein is called an amino acid. Just like joining many glucose together forms a complex carbohydrate, joining many amino acids together eventually forms a protein. If you join 2 amino acids together it forms a peptide and this releases an H and an HO which essentially means H2O is released, otherwise called a dehydration reaction or polymerization. A dipeptide is simply 2 amino acids joined together and an oligopeptide has 3-10 amino acids. A polypeptide is more than 10, but usually 300-1000 amino acids and is otherwise known as protein. These are folded in a special way to have biological activity.

Essential Amino Acids

Ruminant animals can make all their own amino acids but humans need to ingest some, otherwise known as the essential amino acids. All amino acids are essential for the normal functioning of the body but some amino acids can be manufactured in the body while the only way to get others is to eat them because the body is unable to make them. Remember that a major source of amino acids is re-using the stuff already in the body so not all of it needs to be ingested. The maintenance of skeletal muscle mass is dependent on the balance between the rates of muscle protein synthesis and protein degradation.

Protein is made in little factories called ribosomes. Their main function is to convert genetic code into an amino acid sequence and to build protein polymers from amino acid monomers. The number of these units and the efficiency of the units determines how much protein is made.

Skeletal muscle protein synthesis and protein breakdown are regulated by an intricate network of signalling pathways that get activated or inactivated in response to various stimuli such as mechanical tension, nutrients, hormones/growth factors, etc.

How much protein do we need to eat to get the amino acids we need to survive? Building muscle is not a straightforward process. It requires the interaction of various enzymes and hormones. As an adult there also seems to be an on/off switch with the switch only turning on when a critical amount of amino acids are present in our system.

As a child, hormones drive growth much more strongly and even quite malnourished children will continue to grow reasonably well. IGF1, Growth hormone and insulin are the main drivers of the process in kids.

In adults the best combination for muscle building is: Exercise and having amino acids available at about the same time: Exercising makes you keep more and lose less.

I'm not going to spend much time on Mtor other than to say that mTORC1 plays a central role in regulating all of these processes, and therefore controls the balance between anabolism and catabolism in response to environmental conditions

The main driver for mTOR is the amino acid Leucine. Leucine is essential and the main sources of it are animal proteins, meat, fish, chicken and diary but it is also found in things like tofu and navy beans.

SO why is leucine so important?

It seems that Leucine is a bit of an on/off switch when it comes to protein synthesis. If your meal has less that 2g of leucine then as an adult you will not get a metabolic response to build muscle. Having 3g is better than 2g but having 5g or more does not seem be any better than having 3, that is there is not a dose response curve, it is more of an on/off switch.

We know that for most animal proteins you will need to consume about 30 grams of protein to get 3 grams of leucine because Leucine is about 8% of most animal proteins. Remember that 20 grams of protein is not a 20gram steak but about a 100g steak, fish or piece of chicken. In a nutshell, if you eat protein at a meal which does not reach this leucine threshold then what you eat can't be used for muscle building..... which is what we want to happen when the patient is recovering from their operation. We would ideally like to reach this trigger at least twice each day.

The protein quality provided makes a very big difference to this and unfortunately plant proteins are nowhere near as effective in promoting muscle building as animal proteins are.
We also need to understand that there is a big difference between daily caloric requirements and daily protein requirements. The amount of protein required to build muscle is an absolute number. If you calculate protein the other way, that is as a percentage of daily caloric requirements you almost certainly will not get enough protein at a single meal to build muscle.

Key Points:

- If you want to recover from surgery and build up your skeletal muscle then stay away from carbohydrates and fats and stick to protein combined with strength training.

- Just being in hospital does not mean you should lose control of your food choices.

- My job as a surgeon doesn't stop once the wound is closed. I need my patients to heal, recover and restore optimal function in the shortest time possible.

- Providing more and better protein to patients provides satiety, keeps blood sugars in the normal range and hopefully gets the patient to the point of reaching their leucine threshold to start building muscle to aid recovery.

NOTES:

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Ulnar sided wrist pain

A DIFFICULT DIAGNOSIS

- Source:
  - Bone; joint; ligament; tendon; other
- Mechanism:
  - Acute; chronic
- Precise history & examination
- Investigations
- Targeted injections

ACUTE INJURIES

FRACTURES

- Base 4th & 5th metacarpals
  - CT scan!
- Hamate, triquetrum, pisiform
  - CT scan!
- Distal ulna
- Treatments:
  - Immobilisation
  - Surgery

DISLOCATIONS

- 4th & 5th CMC joints
  - Be suspicious → CT scan!
- Distal radioulnar joint (DRUJ)
- Treatment:
  - Surgery
  - Immobilisation

LIGAMENTS

- Triangular fibrocartilage complex (TFCC)
- Radioulnar ligament
- Lunotriquetral ligament

- Treatments:
    - Immobilisation
    - Early rehabilitation
    - Surgery:
        - Debridement
        - Repair

CHRONIC CONDITIONS

JOINT/LIGAMENT

- TFCC sprain or tear
    - Rehabilitate
    - Surgery:
        - Arthroscopic or open
- Ulnocarpal abutment syndrome
    - Surgery:
        - Osteotomy: Ulna or radius
        - Arthroscopy
- Arthritis
    - DRUJ
    - Pisotriquetral (PT) joint
    - Treatments:
        - Non-surgical
        - Surgery: Many different types, eg. DRUJ replacement, excision pisiform.

TENDON

- Extensor carpi ulnaris (ECU)
    - Tendinopathy, tear, instability
    - Treatment:
        - Non-surgical
        - Surgery

UNUSUAL

- Vascular
    - Ulnar artery
    - Embolus
- Neuroma
- Ulnar nerve compression
- Peripheral vascular disease

NOTES:

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ORTHOSPORTS

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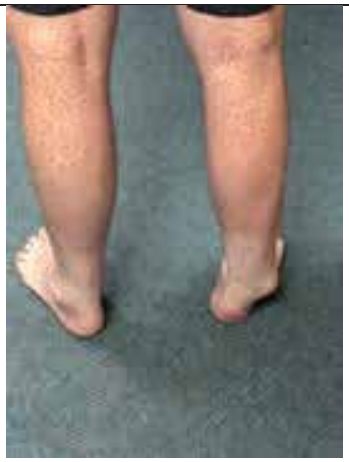
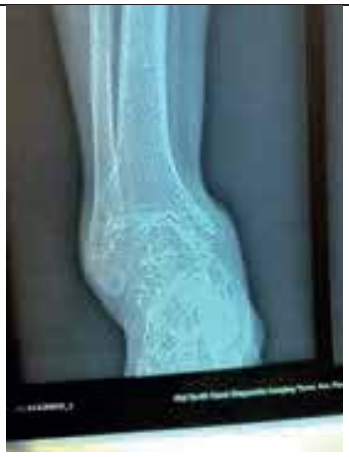
Correction of Deformity in the Foot and Ankle

Correction of deformities is the cornerstone of Foot and Ankle Surgery. When evaluating a foot or ankle deformity, a number of general principles are adhered to in order to obtain optimal outcomes for patients. I will attempt to outline these general principles in order to have a better understanding of how we approach cases.

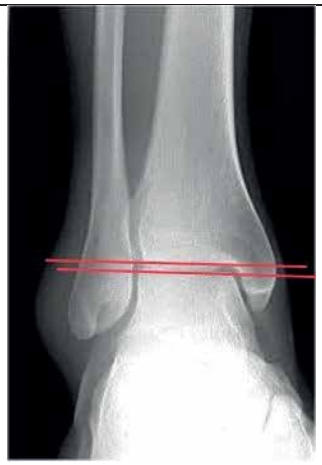
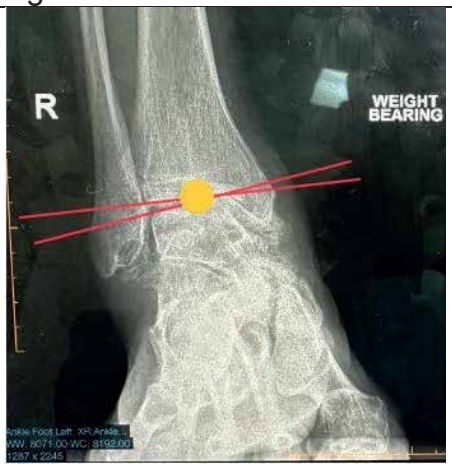
1. Where is the deformity?

a. It is important to determine where the deformity comes from, whether it is the forefoot, midfoot hindfoot, ankle. etc. Around the midfoot, hindfoot, and ankle joints, this can be most challenging. Generally, an x-ray will always help, but WEIGHTBEARING x-rays are essential.

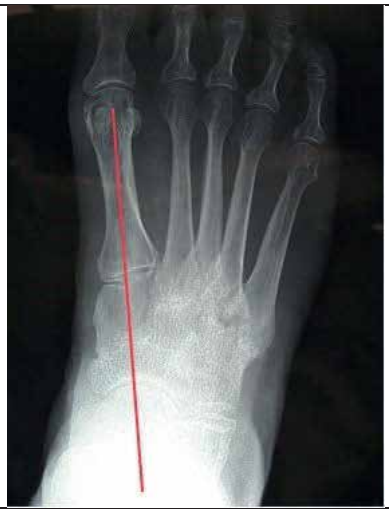




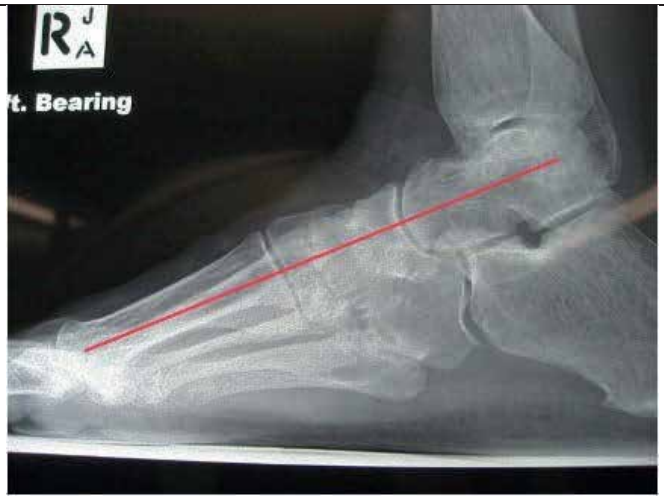
Caption: The patient above has a varus deformity of the foot. The Weight bearing radiograph reveals that the deformity lies in the ankle joint. Notice the varus alignment of the ankle joint with arthritis.

b. Normal alignment of the foot and ankle must be recognised to allow the identification of abnormal alignment. Here are some important ways to determine Normal alignment.

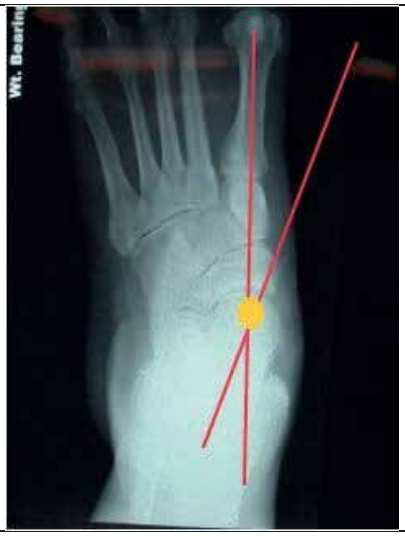



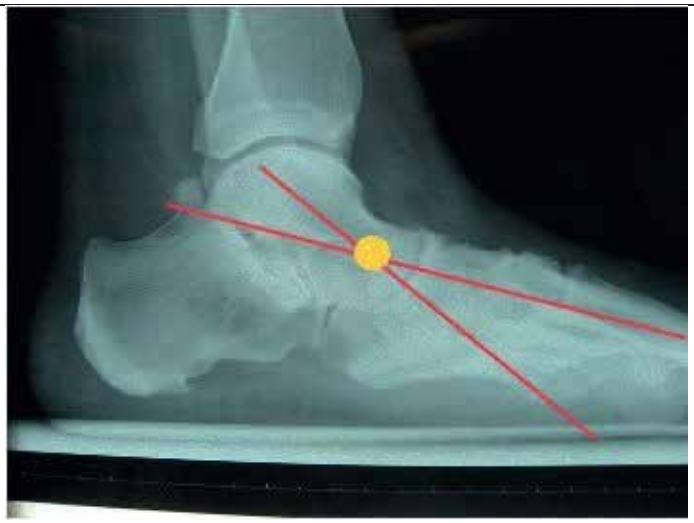
Caption: The image on the far left reveals a normal weight bearing x-ray of the ankle. Notice the joint surfaces are parallel. In the image on the right, the ankle joint surfaces are not parallel due to uneven arthritis.





Caption: The weight bearing radiographs of the feet demonstrate normal alignment. Notice a line driven through the talus on both views is in line with the first metatarsal.





Caption: The weight bearing radiographs to the left demonstrate abnormal alignment at the talonavicular joint. Notice how the Meary's line is not in line with the first metatarsal in both views. Note the CORA at the yellow circle.

c. When dealing with a deformity, it is best to identify the Center of Rotational Angulation (CORA). Corrections are generally made as close to the CORA as possible, as corrections further away may cause a zig-zag deformity. In addition, the CORA is usually where the pathology lies, and is best addressed in order to help symptoms.

d. In the case of the flat foot deformity above, the correction is best made around the talonavicular joint where the CORA is located (Yellow circle).

2. Is a deformity FLEXIBLE or FIXED?

a. A flexible deformity means that the joints are moveable, and with a correction is planned, one can consider a soft tissue correction.
    i. Soft tissue correction involves joint releases, shifting of tendons, or transferring tendons. The concept of tendon transfers is to shift a muscle to help to correct a deformity.
    ii. One example of this is a peroneus longus to peroneus brevis transfer in a cavovarus foot correction, which takes a muscle that causes first ray plantarflexion and moves the muscle to become and ankle everter.
    iii. Flexible deformities may also be fixed with osteotomies. The bone is redirected outside of the joints to re-position the joint in better alignment.
    iv. One example of an osteotomy is a bunion correction. A metatarsal osteotomy re-directs forces around the forefoot and preserves the joint.
b. A FIXED deformity means that the joints cannot be correctable. This may be due to a longstanding malposition of the joints, or can be from arthritis.

    i. Fixed deformities are generally corrected by fusing the affected joint. An example may be a midfoot fusion.

3. Is surgery necessary or are there alternatives?

a. Each case is general considered for its own merits. Generally, symptoms have to be present in relation to the deformity present and considering of benefits and risks of surgery. Benefits have to outweigh the risks to consider surgery.
b. Alternatives to surgery may include orthotics, bracing, and shoe modification. These alternatives can be attempted with a podiatrist or orthotist. Bracing or orthotics help to re-distribute forces around the foot and ankle to support the deformity and help with ambulation.
c. One example of a bracing is with an adult acquired flat foot deformity. An above ankle brace may help to stabilise the ankle joint and support ambulation when the posterior tibialis tendon loses strength.

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ORTHOSPORTS

Common Fractures of the Foot and Ankle

"If all else fails talk to the patient!"

When seeing a patient with a fracture it is as important to talk to the patient about the mechanism of injury as it is to look at the x-ray that the patient is thrusting into your hand.

Did the patient twist their ankle on a 3 cm uneven piece of pavement? Did they slip on a wet surface in a supermarket? Did they fall 10 m out of a building? Was their foot run over by a 10 ton truck? Did they fall off a horse?

Clearly the former represent low energy injuries and the latter high energy injuries.

Importantly, when a patient is injured on the sporting field were they able to keep playing or were they stretchered off in spectacular fashion and taken to hospital by helicopter?

When examining a patient with a fracture the clinical signs are: swelling, deformity and point tenderness over bone.

Most patients with foot and ankle fractures do not need an MRI scan!

Ankle fractures:

Ankle fractures are extremely common and usually result from a twisting injury. They are often associated with sprains. As indicated above a fractured ankle sustained twisting it on a footpath is different to an ankle fracture sustained by falling 10 m out of a building.

Ankle fractures are broadly classified as being below (Weber A), at (Weber B) or above the syndesmosis (Weber C). Fractures below the syndesmosis will generally heal nonsurgically. Fractures at the syndesmosis sometimes require surgery and fractures above the syndesmosis nearly always require surgery.

Ankle sprains often result in avulsion fractures from the distal fibula, lateral wall of the talus, lateral wall of the calcaneus or the anterior process of the calcaneus. These avulsion fractures are generally small and are generally managed as ankle sprains would be with the customary rest, ice, compression, elevation.

Larger fragments sometimes need to be reattached. The best way to image ankle fractures in this situation is a fine cut CT scan.

Fractures of the fifth metatarsal:

These fractures frequently accompany ankle sprains. Again, when examining a patient with an ankle sprain it is very important to palpate the fifth metatarsal for tenderness over bone.

Broadly speaking fifth metatarsal fractures occur in the tuberosity proximally, the junction of the metastasis and the diaphysis and in the distal shaft.

Tuberosity fractures will generally become pain free whether or not the fracture is displaced and whether or not the fracture unites. Indeed, a painless pseudarthrosis with fibrous tissue bridging the gap will usually be painless and not require any further treatment.

The long spiral fracture of the fifth metatarsal sometimes known as the dancer's fracture has a somewhat sinister radiological appearance but generally unites uneventfully.

The fracture which generally causes the most trouble is the fracture at the diaphysis/metaphysis junction. This is known as the Jones' fracture. This fracture can either occur as an acute fracture or as a stress fracture. The optimal management is six weeks in plaster nonweight bearing. Unfortunately, even with this treatment there is a 25% incidence of non-union.

I usually consider open reduction internal fixation in the high demand patient or the athlete which increases the chance of union to approximately 90% and substantially decreases the risk of re-fracture.

Toe Fractures

Toe fractures particularly fifth toe fractures are common and often result from getting the toe caught on a piece of furniture.

If the toe is reasonably straight and the fracture does not involve the joint the patient will usually make an uneventful recovery but needs to be warned that the toe will swell for up to 6 months. This is particularly an issue in ladies who want to wear fashionable shoes!

If the toe is not straight they can generally be straightened in the office under local anaesthesia and then buddy taped to the adjacent toe for a period of 3 to 6 weeks.

In the great toe intra-articular fractures which are displaced sometimes need open reduction internal fixation to prevent the development of arthritis.

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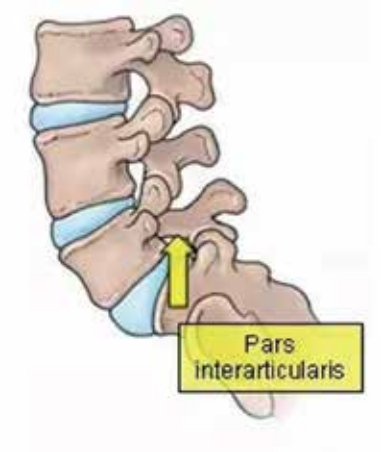
Lumbar Pars Stress in Adolescent Athletes

Lumbar Pars Stress:

- Most common cause of back pain in adolescents
- Can either be from pars stress (bone oedema) or true fracture
- If undiagnosed can lead to chronic pars defect
- Chronic pars defect increases the risk of:
    - Increased episodes of LBP later in life
    - Spondylolisthesis
    - Limitations to athletic performance
- Modern MRI sequences are sensitive & specific for detecting this now



Patient Selection for Imaging:

Adolescents > young adults

- Repetitive extension / lumbar rotation
- Unilateral sports
- Contralateral to dominant side
- High sporting load (or quick change in load)
- Pain on extension
- Painful lumbar quadrant test (extension-rotation)
- Athletes that can commit to a period of offloading (fracture resolution may not be possible if offloading is delayed)

Imaging:

- MRI with STIR / VIBE sequence? Pars stress reaction
- Presence of bone oedema (with or without fracture line) is more likely to be amenable to full resolution with offloading
- Fracture line in the absence of bone odema may be chronic and thus may not heal with offloading

Management – 1st Stage:

- Aim for fracture resolution, minimal bone odema, No symptoms in recent history (4 wks) and normal clinical exam
- Will need strict offloading (No running. Bike and some Rx Tx allowed).
- Repeat MRI at 3m – assess if this is improving
- Continued offloading & further repeat imaging likely required
- Fine slice single level CT (0.5mm) can be used to assess bony healing if cost of MRI is major concern

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Management – 2nd Stage:

- Aim for a safe return to pre-injury level of sports & exercise
- Reload to sports should be slow (3m) and progressive
- Consider frequency, time, type & intensity of activity
- Prioritise things they most enjoy first
- If any symptoms recur – immediate offload and re-image

Key Take Away:

1. Back pain in adolescents is most likely to be pars stress
2. If they are active & have ability to offload – consider MRI
3. Pars stress or fracture often requires 6-9 months of offloading
4. #s with no oedema may be chronic and not respond to offloading
5. Offload = Complete offload from sport.
6. Suggest Clinical follow up at 12 weeks.
7. Repeat MRI when pain free for 4 weeks.
8. Progressive reload once bone oedema minimal
9. Progressive reload to sport 3-6m +/-

Reference:
Orchard, J., Saw, R., Kountouris, A., Redrup, D., Farhart, P., & Sims, K. (2023). Management of lumbar bone stress injury in cricket fast bowlers and other athletes. South African Journal of Sports Medicine, 35(1). DOI: 10.17159/2078- 516X/2023/v35i1a15172

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Taking a deeper history in Musculo-Skeletal (MSK) Consultations *genetics, metabolism and body mass*

1. Why this topic?

Knowing our patients more deeply can help change their lives. Most non-acute presentations of MSK dysfunction are due to pain or reduced functional ability. Ongoing inflammation and poor healing capacity are major factors in some patients more than others. The MSK consultation with a healthcare provider, such as a physiotherapist, is often an entry point into highly relevant and more complex contributing factors which affect the patient's recovery.

The influences of media, poor health information and the search for a 'quick-fix-guru' is ubiquitous which does not help our patients. It should motivate us and strengthen our role as health care professionals as we deepen our knowledge of our patients and add to level of care and professionalism. We will look at three areas of relevance – one's genetics, metabolism and body morphology. Clinical cases will be presented.

2. Genetics

Genetic factors are a major influence in joint and bone disorders and to a lesser degree tendon and muscle disorders. I explain to patients that our genetics is "the study of our genes and our heredity—of how certain qualities or traits are passed in families". (Modified from nigms.nih.gov).

Depending on the patient and the presentation, I would frame some common questions like this… 'tell me about your family':

- Has anyone had joint replacements or arthritis like rheumatoid?
- Has anyone dislocated joints such as their shoulder?
- Have family members snapped their tendons like the achilles?
- Do you have family members with conditions like psoriasis of the skin, autoimmune problems (like Lupus or Hashimoto's') or inflammation in the bowel (like Crohn's disease or ulcerative colitis) that can cause malabsorption?
- Does anyone have osteoporosis/weak bones? Did your parents or grandparents lose much height as they got older?

3. Metabolism

For the last two decades there has been an explosion in the understanding of metabolic health and it affects the MSK system. I explain to patients that 'metabolism refers to all the different chemical reactions that occur in the body to convert or use energy. It is complex and requires enzymes, proteins, hormones and other chemicals working together.'

An individual's metabolism will influence important general health and MSK functions. From a general health perspective metabolic syndrome - MetS - (which includes insulin resistance) is a paramount consideration. Other common metabolic concerns include

bone metabolism, inflammatory responses and healing/recovery capacity. Metabolic disorders includes conditions such as gout (uric acid retention) or haemachromatosis (iron retention with very high ferritin levels). These conditions contribute to joint pain, synovitis, chondral degeneration and tendinopathy. The latter may be partly due to microcirculation abnormalities.

A small group of patients will have hereditary metabolic disorders which may be glycogen storage disorders, mitochondrial diseases or enzymes disorders. These are the patients who are somewhat atypical – fatigue with muscle cramping, inability to exercise well and a strong family history of similar symptoms. Genetic testing and sometimes muscle biopsy is required in specialised centres.

The common questions I ask here are…"with your health and the health of your biological family…."

- Is there anyone with sugar diabetes or pre-diabetes?
- Does gout or haemachromatosis exist?
- Are there people in your family who have trouble exercising?

4. Body Morphology – composition and measurement tools

It is important to be able to communicate well with patients about weight and body morphology. There are ways of doing this without embarrassing our patients. This is probably the least accessible area of conversation for your patients unless you know them well and the consultation is behind a closed door.

The use of the body mass index (BMI) has been a long-standing research tool to measure health outcomes but there are some limitations with this. In certain athletic groups where muscle mass is high it is possible to score a BMI in the overweight or obese range. Other indices such as waist circumference in males and arm span to height ratios have been used for health markers as well as DEXA scanning for body composition.

Our patients need to know that there are weight ranges and body morphology which will help optimise their health and quality of life. Conditions such as osteoarthritis (particularly to the lumbar spine and lower limbs), tendon disorders and bone health are all relevant in this discussion.

For patients who are overweight or underweight I start with – "would you mind if we talk about food, weight and your health? "I would then ask:

- Tell about your relationship with food. Are their food groups you avoid?
- Is there an ideal physique you would enjoy?
- What do your family members look like?
- For patients who need to lose weight I ask…have you done this before? What has worked for you?"

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5. Summary - adding health targets with MSK injury management

As we manage our patients MSK needs we need to consider their genetics, metabolism and weight / morphology. The latter is the most sensitive but the former may be done through sensitive history taking over a few of consultations. If done well they will feel that you know them better and care for them well.

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PRPP Injection

Platelet rich plasma protein (PRPP) injections have become a more widely accepted treatment alternative for managing chronic tendinopathy and also degenerative joint disease. This paper will provide an overview of the basic science and indications for PRPP injection.

PRPP is defined as a solution that has a concentration of platelets above the baseline of whole blood (150-400 X10 9/litre). Platelets are the cells of healing and repair. They contain over 1100 proteins including growth factors, immune system messengers, enzymes, enzyme inhibitors, and other bioactive compounds contained in the alpha granules that are involved in various aspects of tissue repair. Commercial preparation systems can achieve 3-5 times normal platelet concentrations.

PRPP solutions aim to harness this 'healing potential' of blood to stimulate a regenerative process in tendons and potentially articular cartilage.

Whilst the pathogenesis of tendon problems is not universally understood, it usually involves overload in a degenerative structure followed by an aberrant microvascular response known as angiofibroblastic hyperplasia.

Tendon conditions are well described to heal slowly, sometimes taking 12-18 months to improve, much of this being attributable to poor blood supply and failure to stimulate the process of tissue repair. These prolonged time frames are not always acceptable to a professional athlete, manual worker or even a 'weekend warrior'.

Standard treatment of tendinopathy usually involves relative rest, anti-inflammatory measures, physiotherapy with an exercise based rehabilitation program, and consideration of a local cortisone injection. Whilst this approach is successful in many patients, a small proportion do not improve and require alternate treatment. Historically this has involved surgery, which is not always universally helpful.

Biologic therapies have been available in other fields of medicine for a number of years, and have stimulated a lot of interest in in filling the gap between standard conservative and surgical treatment. Initial studies involved the injection of whole blood around tendons with a view to stimulating a healing response. In one study, 22/26 patients with tennis elbow improved after a single autologous blood injection for tennis elbow (1).

The logical next step involved concentrating the active component of the blood, the platelets, whilst removing the parts that were not directly useful for healing, mainly the red blood cells and excess plasma. Injection of this 'platelet rich plasma' or PRP, should theoretically enhance tissue healing in chronic tendon conditions.

There are many different ways PRP may be prepared, leading to variable platelet counts. Some authors attribute this as a reason for lack of scientific evidence of PRP efficacy, depending on the preparation used. Ideally platelet concentrations should be

greater than 4 times baseline, reaching 1000 X10 9/L. This is most reliably achieved using a commercial kit for preparation. Ideally an anti-coaggulant such as citrate-dextrose should be added to the whole blood to prevent activation of the platelets and subsequent clotting until they are delivered into the required area, a process which occurs rapidly within minutes in the absence of an anti-coaggulant. Recent studies have shown that a leukocyte rich preparation may be more effective than a leukocyte poor on tendon healing. This part of the plasma is located in the 'buffy-coat', and can be produced only with the use of commercial preparation systems. (2)

Where possible the PRP should be delivered to the affected area with imaging guidance, such as ultrasound, to maximize the accuracy of delivery.

On the whole, PRP injection is a safe procedure. The use of the patients own blood does make this a true 'natural therapy'. The main side-effect is with local pain around the injection site. The severity and duration is variable, but generally lasts for a few days (coinciding with a period of acute inflammation) and will often require stronger pain killers (non-steroidal anti-inflammatory medications should be avoided). Some patients can develop pain for a few weeks post injection. Ice is initially helpful. Pain is generally worse in tight tissue spaces such as the common extensor origin. Other side-effects could include local infection, which is very uncommon.

The PRP injection generally takes 2-3 weeks to take effect, but it will often take 6 weeks before pain will improve significantly. There is a role for repeating the injection at around the 3 month mark if initial improvement has occurred, but plateaued. Uncommonly a third injection may be required

PRPP injections are not universally beneficial. The success rate for various tendon conditions in papers of variable quality is overall around the 75% mark. The literature surrounding PRP injections has become more robust over the last few years.. One higher quality randomized double blind study looking at PRPP injection versus cortisone injection for chronic tennis elbow found a 75% improvement rate in the PRPP group at 12 months compared to a 49% improvement in the cortisone group (3).A recent study looking at PRPP injection versus normal saline injection for osteoarthritis of the knee showed a significant improvement in pain in the PRP group with a deterioration of symptoms in the saline group at 6 months (4). A randomized double-blind trial for PRPP versus cortisone injections in the treatment of gluteal tendonopathy suggested an 82% improvement at 12 weeks in the PRPP group compared to 56% in the corticosteroid group. (5)

In summary, PRP injections seem to be a reasonable treatment option for tendinopathy that bridges the void between standard conservative management and surgical options that are not universally successful. It may also improve pain in the management of osteoarthritis. The basic science of PRP use in soft tissue healing is sound and there does appear to be supportive laboratory and in vitro studies, as well as clinical evidence for its use in treating tendinopathy and degenerative joint disease. There are few downsides apart from post injection pain. Whilst success is not universal, on average PRPP injections are helpful in around 75% of patients as a collective group.

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