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Time	Event	Who
07:30 - 08:00	Refreshments	
08:00	Welcome Message	Dr Doron Sher
	Dupuytrens disease: can it be treated without an operation	Dr Kwan Yeoh
	An approach to low back pain diagnosis	Dr Mel Cusi
	Ankle replacement: The good the bad and the ugly	Dr John Negrine
08.55	Panel Discussion	
	Osteo-Chondral transplantation (OATS)	Dr Ivan Popoff
	Chondral grafting and HTO	Dr Doron Sher
	The National Joint Replacement Registry	Dr Andreas Loefler
10.05	Panel Discussion	
10:30 - 11:00	Morning Tea	
	Physio question	Dr Todd Gothelf
	Assessment of the stiff shoulder	Dr Jerome Goldberg
	The natural history and treatment of capsulitis	Dr Paul Annett
	The non-operative treatment of the osteoarthritic shoulder	Dr John Best
	Management of the post-operative stiff shoulder including arthroscopy	Dr Todd Gothelf
	Arthroplasty of the stiff and arthritic shoulder	Dr Jerome Goldberg
12:15	Panel Discussion	
12:45	Close	



Dupuytrens disease: Can it be treated without an operation?

What is it?

- Fibrotic changes in palmar and digital fascia.
- Progressive, chronic and benign.
- Unknown cause.
- Leads to Dupuytrens contracture.

Prevention

Unknown

Treatment

- Splints
 - o Do not work
- Operative excision of diseased tissue
 - o Fasciectomy
 - Total
 - High morbidity
 - Low recurrence rate
 - Limited
 - The current gold standard
 - Often requires skin flaps or joint release Moderate morbidity
 - Low to moderate recurrence rate



- Needle-based soft tissue release
 - o Needle fasciotomy
 - Low morbidity
 - Moderate to high recurrence rate
 - o Collagenase injection
 - Low morbidity
 - Moderate recurrence rate

What to do for your patient?

Post-procedure physiotherapy

- Wound care
- Maintain the correction, prevent early recurrence
- Restore function

Further reading

- 1. Henry M. Dupuytren's disease: current state of the art. HAND; 2014 Mar;9(1):1–8.
- 2. Eaton C. Dupuytren Disease. In: Green's Operative Hand Surgery. Seventh Edition. Elsevier Health Sciences; 2016. pp. 128–51.



An Approach to Low Back Pain Diagnosis

The assessment of patients with low back pain has been a diagnostic challenge for many years. Only 15% of patients can be offered a definite pathological diagnosis, but inroads have been made in specific diagnosis. It has been estimated that up to 30% on non specific low back pain can arise from the SIJ, and up to 14% form cluneal nerve entrapment We can however reassure the patient by excluding serious spinal pathology, predicting likely progress and offering a rational approach to management.

Given the variety of causes of back pain and that they often present in combination, the concept of diagnostic triage is worth considering.

Diagnostic triage

Back pain can be the result of a multitude of causes, both of a mechanical or a biological nature. Most of them are rare, and a *differential diagnosis list* is not practical in the clinic. A diagnostic triage approach is a more effective way, both for the clinical management of the condition and the reassurance of the patient. A simple -but not simplistic- diagnostic triage (simple backache, nerve root pain or potentially serious spinal pathology) can help to identify more specific causes and therefore plan management better.

Causes of low back pain

Other causes of low back pain

Not to be missed

Injuries			
IV disc			
Apophyseal joints			
Pars interarticularis stress fracture			
S.I.J injury / inflammation			
Paravertebral muscle trigger points			
Disc prolapse / hernia			
Poor postural habits			

- Spondylolisthesis Spinal canal stenosis Insufficiency fractures Osteoarthritis of the spine Rheumatological conditions Genito-urinary conditions Gastrointestinal conditions Cluneal nerve entrapments
- Malignancy Primary Metastatic Osteoid osteoma Multiple myeloma Severe osteoporosis Stress fractures

Table 1: Differential diagnosis of low back pain

Inspection

- 1. Greet in waiting room, observe posture (? standing, seated, and how arises)
- 2. Observe gait as the patient walks into consultation room: look for antalgic gait, decreased stance phase on one side, lateral compensation
- 3. Observe preferred postures during interview: does the patient remain seated comfortably, fidget / shift weight from right to left.
- 4. If seated, what postures does the patient adopt?
 - Erect, erect with lumbar spine correctly placed in the back of the chair
 - Slumped anteriorly, slumped posteriorly (Perched on chair end)
 - Using arms on chair sides to help take weight
 - Weight on left or right ischial tuberosity. Position of feet

Standing

Range of motion

It is difficult to measure range of motion, and to draw conclusions from a deficit, as it can be due to multiple causes. It is important to bare the whole back and look for stiff segments in the general movement, as they can point to a local cause of pain (facet joint strain, muscle spasm, etc). Measure the distance from fingers to floor to assess forward flexion. Normal extension range would allow fingers to reach the posterior aspect of mid thigh. Lateral flexion should see fingers reaching the level of the knee. Rotation takes place mainly at the level of the thoracic spine.



The <u>stork (Gillett) test</u> (stance phase). Place one thumb on PSIS (posterior superior iliac spine) and the other over the spinous process of S2. Ask the patient to flex the opposite hip (raise the knee) to 90°. Both thumbs should remain at the same level. If the thumb over S2 moves inferiorly, the test is positive.

Special tests

The <u>straight leg raise test</u>: lift the heel with a straight knee. It increases neural tension. A normal test would allow nearly 90° of flexion. This can be reduced by hamstring tension or by neural tension (disc herniation). Pain should travel all the way to the foot. Pain in the posterior thigh is usually due to hamstring tightness. A malingerer with have a positive test very early (usually in the first 30°) and at the same time press on the examination couch with the opposite heel.

Supine

<u>Active straight leg raise test</u>. This also tests the integrity of the load transfer through the pelvis. The patient raises one leg about 30° for five seconds, and repeat the other side. If there is pain, inability to raise one leg, rotation of the trunk towards the same side or the patient feels that one leg is 'heavier' than the other, the test is positive, and compensation strategies are at work. These can involve psoas, gluteals, hamstrings and adductors, and produce pain in these areas.

Prone

Palpate in sequence the spinous process of all the vertebrae, and the spaces between them (supraspinous ligament). Surface anatomy tip: L4 is level with the iliac crests, and there is a dip between L5 and the sacrum.

The posterior superior iliac spine is deep to the dimples of Venus, and inferior to it lies the long dorsal sacro-iliac ligament. It can be confused with the lateral border of the sacrum. Tenderness of the ligament on palpation in association with a positive stork and active straight leg raise test indicates SIJ incompetence (poor load transfer).

Neurological screening

The purpose of the neurological examination in the context of a patient with lower back pain is to confirm or exclude nerve root compression, either uni or bilateral.

Sensation

Use body charts, ask the patient to draw areas of pain and altered sensation (pins and needles, numbness, loss of feeling) with pre-arranged symbols (dots, lines, crosses, etc). A dermatomal distribution of symptoms will point to anatomical or pharmacological nerve root irritation.

Power

The main levels to check are L5 and S1.

L5 Check toe extensor strength by asking the supine patient to lift *dorsiflex* his/her great toe against your resistance. Compare both sides. Alternatively, ask the patient to walk on heels. S1 Ask the patient to heel rise up to 10 times on each side, check that the patient is able to do it, and compare ease of heel lift in either side. Alternatively, ask the patient to walk on tip toes.



Reflexes

Knee (L2/3/4) The patellar tap can be done either with the patient sitting on the edge of the couch, or laying supine and bending the knee supported by the examiner's arm. Compare sides

Ankle (S1) The ankle tap can be done either in the supine or sitting position. It can also be done with the patient kneeling on the couch or chair, and the foot gently dorsi-flexed by the examiner opposite hand.



Ankle replacement: "the good, the bad and the ugly"

In Australia, approximately 50,000 hip and 50,000 knee replacements are performed annually with the numbers increasing. At its peak in 2011 there were 274 ankle replacements performed in Australia and in 2015, only 130. This talk will explore the reasons why.

Ankle arthritis is relatively uncommon. It is estimated that 0.75% of the general population will suffer with ankle arthritis compared with 6% of the population suffering from knee arthritis. The most common cause of ankle arthritis is post-traumatic. Primary osteoarthritis is extremely rare. The remainder of the cases consist of inflammatory arthropathies, haemochromatosis, infection, neuropathic arthropathy and tumour.

The historical time-tested treatment for end-stage ankle arthritis has been an ankle fusion/arthrodesis. Unfortunately, ankle fusion does not solve all the patient's problems. When patients who have had ankle fusion undergo gait analysis, they have decreased knee flexion before heel strike, less time in single limb stance, reduced sagittal ground-reaction force (this is only an issue with barefoot walking.)

There is a 5 to 10% incidence of complications following ankle arthrodesis. The commonest complications include malunion, pseudarthrosis, infection and "the big one": arthritis in the surrounding joints.

The paper that got us thinking about this was written in 2001 where 23 patients who had had an ankle arthrodesis were followed for a minimum of 22 years. 100% of the patients had developed arthritis in the surrounding joints. The author commented that there was more pain, disability and limitation of movement when compared with the contralateral limb. None of these 23 patients however had undergone any subsequent surgery.

Ankle arthrodesis is clearly unphysiological. Hip replacement and knee replacement are in the main successful so why not replace ankles?

In the example given, a 61-year-old man with arthritis in the ankle and the talonavicular joint is clearly not a good candidate for an ankle fusion. If one were to fuse his ankle and his talonavicular joint he would finish up with a totally stiff hindfoot, the function mechanically being little better than a below knee amputation.

Ankle replacement is not new, it was tried in the 1970s and was mostly a disaster. Failures occurred because of poor techniques, poor implants and poor patient selection. The procedure was largely abandoned for 30 years by most orthopaedic surgeons. In the 1990s and 2000's there was a renewed interest in ankle replacement largely from the Europeans: Kofoed in Denmark, Bonnin in France and Hintermann in Switzerland.

All of these authors, many of whom visited Australia and presented their work suggested that ankle replacement was a good operation, provided reliable pain relief and was enduring. They all claimed results between 80 and 90% implant survival at 10 years.

The procedure is technically demanding and extremely unforgiving. We felt however that with better techniques, fellowship training and better implants that we could replicate the results of these European surgeons.

Unfortunately, ankle replacement has not been anywhere near as successful as knee or hip replacement.



The relief of pain, the primary goal of joint replacement is unpredictable. Ankle replacement is accompanied by a high reoperation rate in one study 9% at one year and 23% at five years which was twice the reoperation rate of ankle fusion. Peri-prosthetic cysts/osteolysis frequently occurs and whilst many are asymptomatic initially they can cause catastrophic failure of the prosthesis necessitating urgent revision.

The causes of periprosthetic cysts are likely many and not totally clear. Certainly in the hip and the knee, osteolysis and stimulation of osteoclasts can accompany implant wear debris. Stress shielding beneath the components and continuous high fluid joint pressure are certainly likely factors. Pre-existing osteoarthritic cysts are another possible cause. Some ankle joint prostheses seem to be more prone to cysts than others. Some ankle joint surgeons are more prone to reporting cysts than others!

Ankle replacement is quite unforgiving of deformity. Although varus or valgus deformities of over 15° can be treated I believe the failure rate in these patients is far higher. If there is any deformity in the foot such as cavus or severe planus then it needs to be corrected at the same time.

No surgeon would sensibly replace the ankle of a 25-year-old man who injured it in a motorcycle collision. Rheumatoid patients who are low demand are probably better candidates. The problem of ankle arthritis in young patients is largely unsolved. Whilst a fusion will give them pain relief they will likely live long enough for the surrounding joints to become arthritic and likely symptomatic. Various other procedures have been tried such as distraction arthroplasty with variable results. At present the role of the injectables such as stem cells and PRP are no longer controversial we know they don't work!

References:

- 22 year follow-up of ankle fusion JBJS (Am) 83A: 219. 2001
- Peri-prosthetic cysts Foot. Ankle Int. 35.(1) 14. 2014
- Comparison reoperation rates ankle arthrodesis versus ankle fusion. JBJS (Am) 89:2143-2149. 2007.
- Results of 722 Hintermann ankles. JBJS (Am) 93:1175-1183. 2013.
- Grafting of cysts. Foot. Ankle Int. 37:157-164. 2016



Osteo–Chondral Transplantation (OATS)

Articular cartilage is a highly complex structure composed of:

- chondrocytes,
- collagen (predominantly type 2, but many other types present),
- proteoglycans,
- non collagenous proteins and
- water

4 distinct layers: superficial, middle deep and calcified zone

Chondrocytes, 5% wet weight, are responsible for synthesis and maintenance. Including production of collagen, proteoglycans and enzymes.

Collagen type 2 fibers – orientated horizontal at surface and vertical in deeper layers Create small pores on surface for controlled egress and ingress of water. Increase in thickness in deeper layers. Bound to the sub-chondral bone in the calcified layer

Proteoglycans trapped in collagen matrix exert high osmotic pressure holding water

Water 75% weight

Visco-elastic construct in which the osmotic effect of the proteoglycans combined with the small pore size slows the egress of water from the cartilage resulting in hydraulic cushioning during compression.

The low friction surface is a combination of the horizontal orientation of superficial collagen fibers, production of a fluid film with compression and the orientation of phospholipids to the superficial collagen layer creating an electrostatic effect.

Articular cartilage has no blood, nerve or lymphatic supply lesions which don't penetrate the calcified zone have no healing potential whereas those that do only create fibro cartilage.

Surgical procedures to restore the articular surface are of two types; reparative or restorative.

- Reparative techniques result in fibrocartilage formation, or at best fibro/ hyaline hybrids.
- Restorative techniques result in articular cartilage filling the defect.

The only restorative technique available at present is Osteo-chondral transplantation either autologous or allograft.

- Advantages of OATS type procedures are reliable production of articular cartilage, single procedure, non-restricted weight bearing and relatively quick recovery.
- Disadvantages are limited donor material, painful (especially young patients, usually need 1-2 days in hospital) and only femoral defects can be treated (poor access to tibial articular surface.)



The Unhappy ACL

Despite ACL reduction surgery reliably restoring knee stability in about 90% of patients, the return to pre injury levels of activity, patient satisfaction and knee function remains poor in a large subgroup of patients.

Patient psychology in particularly in response to injury, surgery and subsequent rehabilitation plays an important role in the return to normal function.

An ACL tear is a devastating injury for any athlete, given the need for surgery, prolonged rehabilitation and time off sporting activities.

This has a profound effect on the athlete's psychological well being which has a reciprocal effect on the patient's physical recovery.

Patient's response to injury, self-efficacy, locus of control, athletic identification and mood states have a direct effect on a patient's adherence to rehabilitation and eventual desire or indeed ability to return to preinjury activity levels.

Susceptible patients should be identified early and referred to appropriate psychological management in order to maximize their recovery.

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CASTLEREAGH IMAGING



Medial Opening Wedge HTO

Osteotomy means to break a bone and then set it at a different angle. Alterations of knee alignment lead to redistribution of load within the joint. Varus deformity results in greater loading of the medial compartment and valgus deformity in greater loading of the lateral compartment. This inequality in the distribution increases with the degree of axis deviation, proximity to the joint line, and amount of applied force. The deformity of a Varus malalignment is mostly commonly located in the proximal tibia (primary varus). In symptomatic patients this primary varus is made worse by medial meniscus loss or medial cartilage wear (secondary varus).

The idea of an osteotomy is to transfer load from diseased, arthritic areas of the joint to areas with relatively intact, healthy cartilage. In patients with cartilage replacement procedures and a frontal plane deformity, an osteotomy reduces compression of the regenerated cartilage areas, avoiding the risk of early failure of these procedures.

The valgus opening-wedge high tibial osteotomy (HTO) changes the loading of the tibiofemoral joint. The degenerated medial compartment cartilage is unloaded, resulting in a relief of pain and a delay of cartilage damage. The best indication is moderate single compartment arthritis combined with malalignment of the leg (in the frontal plane). Ideally the patient should not be older than 65, should not be severely overweight, and have a range of-motion of at least 0 - 120°. A small flexion contracture may be acceptable if a change in the tibial slope is included in the surgical plan. A medial opening wedge HTO should not be used if leg lengthening is contraindicated or if the medial soft tissue coverage is compromised. The lateral compartment must be intact with no lateral joint line pain, and no xray signs of lateral osteoarthritis.

There is far less morbidity from a medial opening wedge osteotomy than from the older closing wedge techniques. Conversion to a knee replacement is simpler and more reliable and there is less risk of nerve injury. Smaller corrections are possible and these days we are intervening earlier to prevent more severe disease states.

Chondral Grafting and Rehabilitation

Unfortunately chondral cartilage has no blood supply and no capacity for self repair. More significant osteochondral injuries that penetrate the subchondral bone can lead to fibrous or fibrocartilageneous healing. This led to a procedure called Marrow stimulation or microfracture where holes are drilled into the subchondral bone to allow the adjacent marrow (rich in blood cells and pluripotent cells) to fill the overlying chondral defect and hopefully allow it to heal. While this works well it does not work for everyone and is not always long lasting. *Cartilage surgery should be targeted at reducing symptoms and on improving functional daily activities rather than as a method of returning to high-level sports participation for competitive athletes with chondral damage.*

BST-CarGel is a chitosan-based medical device which is mixed with autologous whole blood and applied to a microfractured cartilage lesion. The hope is that it will physically stabilize the clot that forms and guide and enhance marrow-derived repair. Chitosan is a linear polysaccharide made by treating the chitin shells of shrimp and other crustaceans with an alkaline substance, like sodium hydroxide. It can be used in agriculture as a seed treatment and bio-pesticide, helping plants to fight off fungal infections. In winemaking it can be used as a fining agent, also helping to prevent spoilage. In industry, it can be used in a selfhealing polyurethane paint coating.



In medicine, it may be useful in bandages to reduce bleeding and as an antibacterial agent; it can also be used to help deliver drugs through the skin. The haemostatic agent works by an interaction between the cell membrane of erythrocytes (negative charge) and the protonated chitosan (positive charge) leading to involvement of platelets and rapid thrombus formation. Several very promising studies have been done and while they do not show significant clinical benefits at 12 months BST-CarGel treatment resulted in greater lesion filling and superior repair tissue quality compared with micro fracture treatment alone. The hope is that this will lead to a more durable long term result.

The primary goals for a rehabilitation program are to allow local adaptation and remodelling of the repair with return to function. The problem is that each injury and surgery is different so standardised protocols are not very useful. The main components of any rehabilitation program are: progressive weight bearing, restoration of range of motion and finally muscle control, proprioceptive retraining, and strengthening.

Since the repair site is at its most vulnerable during the first 3 months it is important to avoid impact as well as excessive loading and shearing forces during this time. Since shear forces have to be minimized, the size and location of the defect have to be known because during several activities only parts of the femur/tibia are articulating. For example, the posterior aspect of the medial femur condyle contacts the tibia between 90° and 120°; therefore, loading in positions between 0° and 80° of knee flexion are unlikely to damage a graft in this area.

Neuromuscular control and retraining involves varying movement speed from slow movements that target the feedback system in the early stages of rehabilitation through progressions to quick movements that focus more on retraining the feed-forward system in the later stages of rehabilitation.

Hydrotherapy - Exercises under water produce lower electromyography (EMG) activity during isometric and dynamic conditions when compared to similar exercises on land, thereby leading to lower joint forces. Research has shown that an early and intensive application of hydrotherapy for improving coordination and strength during rehabilitation is helpful. In addition, moving in water gives patients a "feeling of freedom" which seems to be an important psychological advantage.

Therapeutic Ultrasound and Laser - Low-intensity pulsed ultrasound (LIPUS) and low-level laser therapy have been proposed as providing appropriate stimuli for the acceleration of chondrogenesis. The initial findings are quite promising.

Neuromuscular electrical stimulation is mainly useful in patients who are poorly motivated, have long-term muscle weakness, or are slow responders (active exercises work just as well in the majority of people).

Cycling - Increases in the cycling workload result in a significant increase in knee loadmoments and compressive and shear forces, but increases in the pedalling rate do not appear to affect the maximum knee load-moment. Use low resistance stationary bike when ROM good enough.

Low-impact exercise on elliptical trainers, cross-trainers and ski trainers are closed kinetic chain activities but the implications for loading on the knee joint are not fully understood and must be used with caution early on.



Generally, low-impact sports and exercise such as swimming, cycling, and golf can usually be resumed within 6 months. Racquet sports, team sports, martial arts, and running range from an earliest postoperative return at 12 months up to 18 months.

Rehabilitation following a CarGel begins the day after the surgery. The knee is splinted in extension for 24 hours to allow the clot to mature. Depending on the location of the lesion weight bearing may be possible and CPM might be used after that. The frequency, duration, and ROM will depend on the location and size of the lesion. Active and passive motion is also utilized to facilitate the integration of the graft into the surrounding articular cartilage and subchondral bone.

Most patients will be on crutches for 6 weeks as the medial femoral condyle is the most commonly injured site. If the procedure is combined with a high tibial osteotomy, 3 months total crutch use may be required with partial weight bearing for 6 weeks.

The goal is to eventually provide cyclical compression and decompression to allow mechanical stimulation of the graft to promote chondrocyte growth. This includes reducing pain and oedema, addressing soft tissue adaptive changes, restoring muscle strength and function, and gradually including progressive resisted exercise to allow a return to the prior level of function.



The National Joint Replacement Registry

Physiotherapists are well aware of the benefits of joint replacement, which enables patients to return to work and sport and an almost normal life. Physiotherapists will also be aware of changing technology and of the fact that not all joint replacements are equal. How then can we assess the quality of prostheses and look at long term outcomes? The Australian Joint Replacement Registry collects data on all joint replacements performed in this country and provides post market surveillance.

The Registry was set up in 1999. All public and private hospitals in Australia participate. Surgeon participation is voluntary and currently 100% of orthopaedic surgeons contribute their cases to the registry. Patients can opt out, but to date very few patients have refused to participate. The registry is governed and protected by Federal Quality Assurance Legislation. The registry is funded by the federal government, but owned by the Australian Orthopaedic Association.

The purpose of the registry is to collect demographic data on patients and detailed information about prostheses. It provides an independent mechanism of audit for hospitals and for surgeons. Revision is the primary endpoint of failure. The registry records the collective experience with joint replacements and provides feedback in order to reduce the revision rate of prostheses in our patients.

Joint replacement is complex and there are many reasons, why a prosthesis may fail. In general, there are patient factors, factors related to the surgery, and technical aspects of the prosthesis, which may lead to a revision. Fracture, infection, mechanical loosening and wear debris are the common reasons of failure. The high revision rates of the Metal on Metal hips was one recent example of unforeseen failure of a type of prostheses, which was first identified by the Australian registry.

The registry now has data on more than 1.1 Million joint replacements, consisting mostly of hips and knees. The data is reported in various ways. There are Kaplan Meier survivorship graphs, which show the number of prostheses, which have not yet been replaced. Both hip and knee replacements have excellent results, with a 95% 10year survivorship. Results can also be expressed as cumulative percentage revision rates. These can then be compared between different prostheses, age groups, and technical aspects. The registry also calculates so called hazard ratios, or the likelihood of requiring a revision over a certain number of years.

The registry names and reports those prostheses, which have a higher than anticipated rate of revision. The registry will look at the details and try to ascertain, which factors might contribute to the failure of a prosthesis. This is a detailed process, involving a group of arthroplasty specialist. Once a prosthesis has been identified as having a revision rate of two or more times the average, the prosthesis is reported to the Therapeutic Goods Administration.

The Australian Joint Replacement Registry is linked to the International Society of Arthroplasty Registries. Whilst the general purpose of all registries is similar, each registry collects slightly different data. The collaboration of the registries gives external validity to the process, allows pooling of data and sharing of information, all of which is to the benefit of our patients.



Since 2008, the Australian registry has been linking the surgeon to each joint replacement. The registry is able to give detailed feedback to each surgeon. The so called Surgeon Portal lists all cases of joint replacement and all revisions, even if the revisions have been done by another surgeon. The reasons for the revisions are listed and the surgeons revision rate is compared to the national average. In addition, each surgeon can request a more detailed report. Surgeons are encouraged to discuss their data with colleagues in order to improve their practice.

The Australian registry was the first to notice problems with the ASR Metal on Metal prosthesis and reported this to the TGA. The registry has also flagged problems with other prostheses, such as the use of resurfacing, especially in women. Resurfacing has declined from over 8% to about 2% of hip replacements per year. Modular prostheses, which have an exchangeable neck have a high revision rate. The use of Austin Moore prostheses has almost stopped. It is also interesting to note that none of the prostheses, which were introduced in the last 10 years, have performed better than those we had in the past.

Each year, the registry focuses on a particular issue. This year's report looks at primary total hip replacement in patients under 55. Approximately 13% of THR are performed in younger patients. There is a much higher incidence of hip dysplasia, avascular necrosis, inflammatory disease, and tumours in these patients, while the older age groups have a higher percentage of osteoarthritis and fracture. Younger patients are generally more active, causing more wear of the prostheses, but the cumulative revision rate for all bearing surfaces in patients under 55 is 12.7% at 15 years, or conversely a survivorship of 87.3%, which is much better than telling patients, that hips will last only 10 years.

One of the criticisms of the Australian registry is lack of patient reported outcomes. The New Zealand registry collects such data in perhaps 20% of cases. Outcome data is important, but very difficult to collect, as patients' function and the effects of comorbidities vary a great deal. A patient may have a perfectly good knee replacement, but is unable to walk fast or far due to a cardiac condition. To collect outcome data at regular intervals is expensive. It also introduces an element of feeling good or satisfied, which is highly subjective and may not reflect the function of the prosthesis, but more the expectation of the patient.

Our registry has started to collect additional data, such as the ASA score, which is a measure of comorbidities and surgical risk. Since 2015 the registry also records the BMI. 60% of patients having a hip replacement are normal or pre-obese, while 60% of those having a knee replacement are pre-obese or obese. It will take several years before the registry can report an association of obesity and failure of hip and knee replacements.

The Australian Joint Replacement Registry now has 15 year data. Although the number of joint replacement have risen significantly, the revision rates for hip and knee replacements have been dropping. It is estimated that the registry has effected a saving of \$600 Million to the Australian community, and that figure does not account for the saving in pain and suffering. The registry has achieved this by showing better long term outcomes, taking into account prostheses, patient and technical factors.

The annual report of the registry is a public document. There are detailed reports on the various types of replacements. There is also a lay summary. Physiotherapists may be interested in some of the data. The registry will also provide individual reports for research into joint replacement surgery.

Reference: Annual Report of the Australian Orthopaedic Association National Joint Replacement Registry 2016



ASSESSMENT OF THE STIFF SHOULDER

DEFINITION - loss of both active and passive motion

CAUSES

- adhesive capsulitis
- osteoarthritis
- post operative
- dislocations but usually a locked posterior dislocation
- tumour
- hysteria

HISTORY

- age
- hand dominance
- occupation
- recreational activities and sports
- previous shoulder operations
- medical history-arthritis, diabetes, and thyroid disease
- family history
- trauma
- length of symptoms
- location of pain
- exacerbating and relieving factors
- night discomfort
- loss of movement or power
- neurological symptoms

CONFUSION WITH CERVICAL SPINE & OVERUSE SYMPTOMS

- Posterior shoulder pain
- Not related to shoulder movement
- Pain radiates to forearm and hand
- Paraesthesia
- Occupational overuse

PHYSICAL EXAMINATION

INSPECTION

PALPATION

- Sternoclavicular jt
- Clavicle
- Acromioclavicular jt
- Acromion
- Greater tuberosity
- Coracoid

Dr Jerome Goldberg M.B., B.S., F.R.A.C.S., F.A. Ortho. A. Shoulder Surgery



RHYTHM

- Smooth
- Synchronous
- look for "hitching"

RANGE OF MOTION

- Active elevation
- Passive elevation
- Passive E.R.
- Passive I.R.
- look for GH crepitus
- look for same active and passive elevation (usually 90 degrees) plus complete loss of E.R.

MUSCLE POWER

PHYSICAL EXAMINATION Neurological exam Vascular exam Ligamentous laxity Cervical spine exam

SPECIAL TESTS

- impingement /rotator cuff
- instability tests
- special tests- adduction, speeds, adsons
- injection tests

RADIOLOGY

- Plain xray ESSENTIAL
- ultrasound not recommended
- CT scan if bony pathology suspected
- MRA only if diagnosis in doubt or if surgery contemplated

JOINT REPLACEMENT SURGERY

Joint replacement surgery is the last option in the patient with severe pain, loss of movement, and significant interference with the activities of daily living.

Shoulder replacement surgery does not give as good an outcome as hip or knee replacement surgery. As a general rule, pain relief is excellent, but restoration of range of motion is fair only. Patients must be warned that they will never achieve a full range of motion, but they will achieve the ability to perform most activities of daily living.



In addition, shoulder replacement surgery has a higher complication rate than hip or knee replacement surgery. Reverse shoulder replacement surgery has a documented complication rate of up to 30%. Shoulder replacement surgery also has a limited longevity, and patients are often unable to resume their sporting activities. With respect to primary shoulder replacement, the main long-term complication is rotator cuff dysfunction that occurs due to age-related wear and tear.

The surgeon requires, as well as a complete history and full examination, plain x-rays, and MR arthrogram, and in the case of severe bone loss a CT scan.

The surgeon can then decide whether to proceed with the primary total shoulder replacement or whether a reverse shoulder replacement is more appropriate. The reverse shoulder replacement is best in patients who have torn rotator cuffs, significant abnormalities of glenoid erosion, and older patients with degenerative rotator cuffs that are likely to tear in the future.

Post operatively, patients require sling immobilisation for six weeks. If passive exercises are commenced within that six week period, care must be taken not to push external rotation movements which have the potential to tear the Subscapularis. After six weeks a gentle active program can be commenced, but patients need to avoid lifting more than a few kilograms for six months while the rotator cuff around the replacement heals. The post operative programs must be individualised for each patient, depending on the type of replacement done and the state of the rotator cuff.



Adhesive Capsulitis

Introduction:

Adhesive capsulitis or 'frozen shoulder' is a common and often disabling ailment. It is a condition of uncertain aetiology that is characterized by clinically significant restriction of active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder.

It affects 2-5% of the population and is more common in women than men. The incidence peaks in the 5th and 6th decades. It may be bilateral in 10-20% of cases, but usually occurs sequentially.

Etiology:

As previously mentioned the cause of adhesive capsulitis is unknown. It is generally considered to be a self-limiting condition, with signs and symptoms resolving in 90% of patients over a 12-24 month period. Whilst autoimmune causes have been considered, there is a lack of aspects of these diseases, including systemic symptoms and positive serological markers. There is a clear association between adhesive capsulitis and diabetes, dyslipidaemia and thyroid disorders. Blood tests should always be organized to exclude these conditions. Likewise, Parkinson's disease needs to be considered. Capsulitis may also occur after trauma and surgery, especially if there is upper limb immobilization.

Diagnosis:

Historically adhesive capsulitis runs through 3 overlapping phases. The initial phase is the pain phase, which may last from 2-9 months. It generally has an insidious onset and is typically worse at night. As it progresses the pain may become constant and can be quite severe. As range of motion is maintained it may be difficult to distinguish from other painful conditions of the shoulder, such as impingement. The pain is worse with sudden and end of range movements and will often refer down the arm.

The second phase is the freezing phase, which may last from 4-12 months. Pain may persist into this phase, although it may diminish. The loss on movement is capsular in nature, thus affecting all planes on motion, although rotation is generally more affected.

The final phase is the thawing or resolution phase, which may last from between 5-24 months. In this phase pain slowly resolves and range of motion returns. Whilst most patients have a resolution of the stiffness, 40% may have some mild ongoing loss of motion and less than 10% have more clinically significant restriction.

Examination:

Physical examination is essential to exclude other common causes of shoulder pain including impingement, instability or AC joint arthrosis. Clinically the loss of motion is capsular, and thus affecting all planes of motion. The most readily identifiable of these is loss of passive external rotation range. In capsulitis the loss of motion actively and passively is equivalent, where as in impingement passive motion will be maintained even if active motion is restricted by pain. It may be difficult to distinguish the two conditions in phase one where there is pain but not yet loss of motion.



Investigation:

The diagnosis of a frozen shoulder is typically a clinical one. All patients presenting with adhesive capsulitis require a plain X-Ray. This may identify glenohumeral osteoarthritis, an important mimic of a frozen shoulder, or AVN. It will also exclude nasty pathology such as a tumor. An ultrasound is unhelpful, and potentially confounds the diagnosis when it shows fluid in the sub-acromial bursa, which is a common finding. MRI scanning is generally reserved for cases where the diagnosis is unclear or there has been a lack of response to appropriate treatment. The addition of intra-articular contrast may show capsular restriction.

Treatment:

The treatment of adhesive capsulitis is expectant as the majority of patients with this condition will improve. It is rarely a surgical condition unless the patient is unhappy to see out the natural history of the condition or there is persistent stiffness over an 18-month period.

The cornerstones of treatment are pain relief and improving range of motion. Pain relief may be achieved using paracetamol based medications regularly, possibly with the addition of codeine. Long acting opioids should be used sparingly. Anti-inflammatory preparations may also be helpful, after taking their side-effect profile into account. Oral steroids may be used, and have been shown to have positive effects within the first 6 weeks. Their side-effect profile needs to be considered carefully. Physiotherapy with a home exercise program will help to improve function in patients with capsulitis. Both range of motion and scapular and rotator cuff strengthening are essential.

Interventional treatment may involve performing an intra-articular cortisone injection alone or with the addition of normal saline, to create a hydrodistension effect. Cortisone provides a potent anti-inflammatory effect inside the joint whilst the addition of normal saline will theoretically stretch the restricted joint capsule. The literature suggests that cortisone injection plus hydrodistension will improve pain in the short term (6 weeks) and disability in the short to medium term (12 weeks). Similar effects may be obtained with an intra-articular cortisone injection on its own. Range of motion will be improved with the combination of intra-articular cortisone and post-injection physiotherapy.

Surgery is generally only required for prolonged stiffness and may include a manipulation under anaesthesia followed by an arthroscopic capsuloraphy.

Key Points:

- Frozen shoulder is an idiopathic condition
- It is generally considered to be self-limiting, although time frames may be prolonged
- There are 3 phases of the condition 'Freezing, frozen and thawing'
- Treatment is generally non-operative and aimed at analgesia and improving range of motion
- Interventional treatment may include intra-articular cortisone injections with or without the addition of a normal saline hydrodistension
- Surgery is only considered in recalcitrant cases



The Non-Operative Management of Gleno-Humeral Osteoarthritis (GHOA)

Prevalence, causes and classification:

GHOA has a multi-factorial aetiology. Although less common that tibio-femoral, patellofemoral, hip, interphalangeal and facet joint osteoarthritis, the level of disability and dysfunction has a significant impact on the patient.

The significant consequences of pain, movement restriction and reduced physical capacity arise due to the small surface contact area of the GHJ. This also explains why major dysfunction may occur with relatively minor X-ray changes.

GHOA is more common in females than males presenting in patients >50years. Although 15% of patients over 65 years of age will demonstrate X-ray changes of GHOA, the clinical presentation is variable.

In terms of causation a simple classification approach is:

- i) Primary familial and genetic predisposition
- ii) Secondary typically this is post-traumatic following instability, fracture, rotator cuff tears or as overuse from heavy manual work / resistance training.
- iii) Other this includes infection, chemical causes (e.g. gout), deposition disorders (e.g. haemochromatosis) and inflammatory disorders.

Non-Operative Management:

The American Academy of Orthopaedic Surgeons published a comprehensive review of the non-operative management of GHOA in 2010. Little has changed since then. Treatments have been adapted from hip and knee OA management practices and literature. Evidence-based non-operative treatments are limited. A paradigm with careful and reasonable time frames underpins the treatment process and as per the management of OA in other joints, the treatment is multi-pronged.

The three areas of non-operative management are pharmacological, load management and physical therapy. These should be part of a combined management plan.

a) Pharmacological

Analgesia and anti-inflammatory treatments have a role. Analgesia may be achieved simply with paracetamol use and the episodic use of non-steroidal anti-inflammatories particularly for the use of night pain. Opioid analgesia is required with more severe pain (e.g. codeine). Patients always need to consult with their GP to assess for an interaction with other medications or illnesses.

Intra-articular corticosteroid injections are best performed under imaging. Results are variable. In general, up to 65% of patients report pain reduction and improved sleep for 6-12 weeks. Results are best if the patient protects the shoulder with careful load management in the first 10 days and commences pain free capsular stretches at this time (see below).

Viscosupplemtataion (hyaluronic acid) has not been proven to be of benefit. One industrysponsored study reports 55% pain reduction for 3 months.

Regenerative therapies and other. Platelet rich plasma and stem cell therapies are not proven to be effective. Oral supplements are variable (e.g. fish oil).



b) Load management

In terms of patient education, responsibility and also the long-term health of the GHJ, this aspect of management is huge. Most patients will establish a pattern of pain with overload (*"its worse after playing tennis"*) or they will notice a functional deficit (*"I'm struggling to swim freestyle"*). As with other areas of OA all pain producing activities should be ceased which may be a permanent requirement.

For patients still performing resistance training this means ceasing resistance training in overhead positions (eg: shoulder press) and weight-bearing in abduction and external rotation (bench/chest press, push ups). Other forms of resistance training may be tolerated and it is unknown if they accelerate the degenerative change (eg: arm curls, pulldowns, seated pull).

c) Physical therapy

It should be noted that there is little evidence in the role of physical therapy to improve the symptoms and function with GHOA. Intuitively and anecdotally physical therapy is worth pursuing in most patients. Patients with an intact rotator cuff and mild to moderate disease may respond best.

The following need to be considered when physical therapy is offered:

- i) <u>Is the humeral head 'centred'</u>? GHOA generally commences with posterior glenoid degeneration. Any activity which compresses the posterior GHJ will accelerate this process. Assess range of motion from 0-90° abduction palpating humeral head positioning. Establish if the patient is able to actively control their humeral head positioning. This requires experience.
- ii) <u>Where is the main capsular restriction</u>? Test range of motion as above. Aim to correct capsular restriction with pain free stretches. Assisted passive stretches with the patient in complete control. Patients tend to report more ease performing stretches following a shower, exercise or heat application.
- iii) <u>Where is most of the weakness</u>? Patients with GHOA have significant deltoid wasting but often maintain good isometric rotator cuff strength. Maintaining this with isometric exercises and band resistance exercises is worthwhile. Some patients report comfort performing these using a small abduction pillow. This is also an option with capsular stretches. Patients with medium to large rotator cuff tears benefit from the use of pulley systems to maintain ROM and deltoid function but should avoid resistance exercises in the abduction position.
- iv) What are the secondary musculoskeletal problems? Most patients with GHOA have associated myofascial pain secondary to movement abnormalities and chronic muscle shortening. This may occur globally although usually there are one or two 'trigger points' trapezius, pectoralis major, teres and deep to the posterolateral shoulder. Establishing what assists patients to have some temporary relief is important heat, local massage, trigger point therapy, dry needling, acupuncture there is great variability in this area.

Summary:

Although less common than other forms of osteoarthritis, gleno-humeral osteoarthritis can create severe disability with altered quality of life. Following prompt diagnosis and patient education, non-operative treatment should involve pharmacological management, load management and judicious physical therapy.

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The Surgical Approach To Stiffness of the Shoulder

Background

With the advent of arthroscopic surgery, there has been a paradigm shift in the management of stiffness of the shoulder. The arthroscope has allowed the ability to successfully release the shoulder to improve motion whereas open surgery had its difficulties. Arthroscopic surgery allows us the ability to perform surgery without violating multiple tissue planes, thus, minimizing the chance of creating scar tissue. As a result, the arthroscopic release of the stiff shoulder is generally an effective operation and satisfying to most patients.

Stiffness After Surgery- Basic Science

As with the idiopathic stiff shoulder, it seems that an inflammatory response occurs that initiates the stiff process. Studies have shown that this inflammatory response is higher after open surgery. Shinoda et al. demonstrated a 3-fold increase in interleukin-6 levels post-operatively in the open group compared to the arthroscopic group. Francheschi et al investigated the levels of substance P in the shoulder joint after arthroscopic rotator cuff repair. They reported a 3-fold increase in the levels of substance P in patients in whom postoperative stiffness developed.

The Benefits of Immobilisation

Previous school of thought about stiffness after rotator cuff surgery led to a philosophy of early range of motion to avoid the evils of stiffness. In 1985, Cofield, made a statement: "The use of physiotherapy is no longer controversial. Early passive range of motion exercises of the shoulder both prevent adhesions and protect the repair." This statement made sense at the time, as post-operative stiffness was one of the most dreadful complications after rotator cuff repair, as without arthroscopy, there was no effective way to combat the stiffness problem.

Fast forward to today, we have seen benefits from limiting early range of motion in preventing stiffness and assisting in rotator cuff repair. Peltz et al looked at the effect of post-operative passive range of motion on rotator cuff healing in a rat model. They found that immediate passive range of motion actually led to increased stiffness compared with an immobilisation protocol. Gimbel et al. found that immobilisation led to enhanced mechanical properties of repaired rat supraspinatus tendons.

Human studies have also demonstrated that limiting early range of motion does not effect the outcome of stiffness. In addition, it may help the healing properties of the rotator cuff. Huberty et al reviewed their series of 490 arthroscopic rotator cuff repairs. Their post-operative rehabilitation protocol is one of limited range of motion, only allowing passive external rotation for the first six weeks. They had an overall incidence of 5% stiffness. They recognised that certain pathologies resulted in increased risk of stiffness: single tendon tears, partial tear repairs, calcific tendinitis, adhesive capsulitis, and SLAP or labral tears. In these patients they initiated a program of early passive overhead stretches by means of closed-chain table slides along with passive ER on day 1. During a second look arthroscopy in 24 patients for stiffness, they found that the rotator cuff tear was completely healed in 23 patients, implying that perhaps the inflammatory response that caused the stiffness contributed to cuff healing. They concluded that early immoblisation does not contribute to overall stiffness after surgery, and that it may enhance rotator cuff healing.



Parsons et al evaluated their limited ROM protocol and stiffness. Patients underwent sling immobilisation for six weeks after arthroscopic rotator cuff repair. 23% of patients were considered stiff at the six-week mark. After one year, both the stiff patients and non-stiff patients restored their motion to a similar, comparable range. MRI was used to assess the rotator cuff, and it was found that the stiff group had a higher rate of rotator cuff healing. They concluded that there may be a benefit to early immobilisation for rotator cuff healing. Lee et al. compared two rehabilitation protocols on ROM and healing rates after arthroscopic rotator cuff repair. They found that there was no difference at one year in range of motion in both groups of early aggressive passive range of motion versus limited early passive motion. However, the early aggressive group had 23% re-tears, while 8.8% had re-tears in the limited early passive motion group. They concluded that aggressive early motion may increase the possibility of failure at the repaired cuff, and more limited rehab does not change the final outcome.

Surgical Results of Arthroscopic Capsular Release

A recent current concepts review of Frozen Shoulder reviewed current evidence in the treatment of Primary Idiopathic Stiff Shoulder. They recommended that arthroscopic release and MUA should be considered after 9 months of activity-limiting stiffness. Arthroscopic release has been shown to be effective in improving range of motion, and the satisfaction rate is approximately 90% in most studies. Release is usually done in the chronic or second phase of a frozen shoulder. Manipulation under anaesthesia alone improves shoulder elevation and abduction, but rotational improvement is limited as surgeon's are concerned about forceful rotation. Arthroscopic capsular release will allow restoration in all planes.

Surgical Results of Arthroscopic Release in patients with OA

Arthritis of the shoulder can cause a loss of range of motion from direct mechanical blockage by osteophytes, effusion, and swollen synovium, or from a secondary capsular contracture. Arthroscopic debridement and capsular release has been reported to improve range of motion, and has satisfactory results. The procedure has been considered for the young, active patients with high demands who may want to delay total shoulder replacement. Generally, patients with arthritis have been shown to do better with total shoulder replacement in the long term.

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