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Concord	47-49 Burwood Road CONCORD NSW 2137	Tel Fax	**02 9744 2666** **02 9744 3706**	**Doctors Consulting here** Dr Todd Gothelf Dr Paul Mason Dr John Negrine Dr Rodney Pattinson Dr Doron Sher Dr Kwan Yeoh
Hurstville	Level 7 Waratah Private 29-31 Dora Street HURSTVILLE NSW 2220	Tel Fax	**02 9580 6066** **02 9580 0890**	**Doctors Consulting here** Dr Paul Annett Dr Jerome Goldberg Dr Todd Gothelf Dr Andreas Loefler Dr John Negrine Dr Rodney Pattinson Dr Ivan Popoff Dr Allen Turnbull Dr Kwan Yeoh
Penrith	Suite 5B 119-121 Lethbridge St PENRITH NSW 2750	Tel Fax	**02 4721 7799** **02 4721 7997**	**Doctors Consulting here** Dr Todd Gothelf Dr Kwan Yeoh
Randwick	160 Belmore Road RANDWICK NSW 2031	Tel Fax	**02 9399 5333** **02 9398 8673**	**Doctors Consulting here** Dr John Best Dr Jerome Goldberg Dr Todd Gothelf Dr Paul Mason Dr Andreas Loefler Dr John Negrine Dr Rodney Pattinson Dr Ivan Popoff Dr Doron Sher

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Time	Event	Who
07:30 – 08:00	Refreshments	
08:00	Welcome Message	Dr Doron Sher
	Non-Operative Management of Knee Osteoarthritis	Dr John Best
	Surgical Treatment of Knee Arthritis	Dr Doron Sher
	Achilles Tendon Disorders – The Ageing Athlete	Dr John Negrine
09.10	Panel Discussion	
	Flexor Tendon Injuries in the hand	Dr Kwan Yeoh
	Chronic Exertional Compartment Syndrome & Lower Leg Pain	Dr Paul Annett
	Low Back Pain–aetiology and variant presentations	Dr Paul Mason
10.30	Panel Discussion	
10.50-11.20	**Morning Tea**	
	Physio question, education	Dr Todd Gothelf
	The Rotator Cuff – the science behind the disease	Dr Jerome Goldberg
	Reverse Total Shoulder Arthroplasty (RTSA)	Dr Ivan Popoff
	Return to Work and Sport After Rotator Cuff Repair	Dr Todd Gothelf
12.30	Panel Discussion	
13.00	Close	

NOTES:

Dr John P Best
B Med, Dip Sports Med (London), FACSP, FFSEM
Sport & Exercise Medicine Physician

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The Non-Operative Management of Knee Osteoarthritis (KJOA)

Prevalence and burden of disease: Osteoarthritis of the Knee (KJOA) is a chronic disease characterised by joint pain, swelling, reduced mobility and an altered quality of life (qOL). It is most common in people over 55 years. There is a 45% lifetime risk of developing KJOA, increasing to 65% in obese individuals. Increasing obesity and longevity combines to produce a growing prevalence of KJOA requiring early knee arthroplasty. Good non-operative management may allow patients to delay primary surgery (and therefore possible revision surgery) and enjoy an improved qOL.

Understanding of the Pathogenesis: KJOA is an imbalance between anabolism and catabolism of articular cartilage. Pathological findings with KJOA include the upregulation of substances such as matrix metalloproteinases (MMPs), a proliferation of inflammatory cytokines and ultimately a failure of extracellular matrix proteins to resist compressive forces and maintain tensile properties of the tissue. From the age of 50 years, knee articular cartilage deteriorates by the rate of 4-5% per decade; greater with obese patients.

The Principles of Non-Operative Managements of KJOA: Genetic factors aside, the table below may act as a 'check-list' to ensure that one has considered the various issues in your care of your patients. A practical expansion of these principles as "seven key steps" for good care follows.

Lifestyle Factors	Chemical Factors
Weight management	Analgesia / Inflammation – oral, topical
Load Management	Supplements and non-prescription meds
Exercise and rehabilitation	Analgesia / Inflammation - injections
Eating patterns and diet	Injections to improve loading
Psychological and behavioural factors	Injections claiming to offer regeneration

Non-Operative Management – Seven Key Steps: It is important to individualise the management for each patient and not be tempted to run through a set paradigm. For example, the advances with weight reduction and the role of eating patterns causing inflammation has provided great frontiers.

1. **Make a clear diagnosis, grade the severity and understand your patient.** Confirming the diagnosis with a careful history, examination and appropriate imaging is required. Many patients have undergone previous treatments and may be discouraged or fearful. In your history always ask about levels of pain (including night pain), function (walking tolerance and ADLs), current medications and recent treatments. Common functional tests include- functional strength and squat ability; a six-minute walk; negotiating stairs. Also take time to explore the patient's hopes and expectations.
2. **Manage pain and inflammation well.** In conjunction with patient education, Analgesia and anti-inflammatory treatments have a role. Analgesia may be achieved simply with paracetamol use and the episodic use of non-steroidal anti-inflammatory medications which may reduce night pain. Always check with the patients GP.
   Intra-articular corticosteroid injections are appropriate with existing knee joint synovitis. Cortisone injections offer patients 80% relief for 3-12 weeks. This period of reduced pain may offer a window for better activity and rehabilitation. One may explore non-prescription items such as oral supplements with Fish Oil (Omega-3FA) being the authors most recommended.

Dr John P Best
B Med, Dip Sports Med (London), FACSP, FFSEM
Sport & Exercise Medicine Physician

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The use of Platelet Rich Plasma (PRP) has existed for almost twenty years. Results remain inconclusive with modest studies showing a benefit in 70% of patients; which may be anti-inflammatory in nature rather that regenerative. There is no standard PRP delivery system with large cost variations noted.

3. **Assess eating patterns and aim for a healthy BMI.** The most impressive results in the reduction of KJOA symptoms and improved function are through weight loss. There is a dose-response effect with weight loss on KJOA. In overweight and obese patients, achieving as little as 5% weight loss offers reduced symptoms and improved function. In patients losing up to 20% body weight the results are dramatic. For example, if a 55 yo male weighing 97kg (BMI 29.9, Obesity I) were to lose 14kg (= weight becomes 85kg, BMI 26.2 = borderline overweight), this results in a >50% reduction of knee pain!!! The other consideration with diet is the reality that some patterns of eating are very "pro-inflammatory", in particular high carbohydrate eating plans, which may contribute to joint inflammation.
4. **Manage load and exercise safely.** Creating a variety of alternate ways to exercise helps patients with KJOA. The avoidance of lunges, squatting beyond 90° and hard surface impact training are all beneficial. The author believes that Hyaluronic Acid injections have a role in certain patients through improving load tolerance with existing articular cartilage through the improvement of viscoelastic properties. Braces and orthotic devices will be discussed briefly.
5. **Perform key exercises.** The EULAR reference is a very good review of effective exercise protocols. In summary, modified impact aerobic exercise is recommended. Resistance training of key muscle groups improves joint mobility and unloads the arthritic joint. Inexperienced patients may need to commence at low levels (e.g. 30% 1RM per body area) with the key aspects agreed as 2-3 sessions per week, 2-4 sets of 8-12 reps and the avoidance of deep squatting and lunging.
   Neuro-motor improvement with balance exercise and activities such as Tai Chi, one – legged standing and some forms of Yoga have been studied. The frequency is recommended as 2-3 times per week, up to 30 mins per session.
6. **Have a review plan, set goals and pursue behaviour change techniques**. For most patients KJOA is a journey which often pre-occupies their life. The appreciation of psychological factors is significant. Re-assessment and resetting of goals are essential and consider strategies to boost compliance (e.g. follow-up phone call, email, closer supervision, biometric measuring devices).
7. **Keep up to date with research.** Patients suffering KJOA are a vulnerable group. They are frequently awake at night asking "there must be something new out there?" Perhaps they having given up on managing their obesity – please remind them of the impressive data available. The role of diet, understanding MMPs and supplements such as Boron are showing promise.
   The area of regenerative therapies may be the way forward. There is no current TGA approval for KJOA treatment using stem cell (bone marrow or fat derived) therapies. Remind patients that this is a procedure – it is not a non-operative treatment.

Summary: KJOA can create severe disability with altered quality of life. Following prompt diagnosis and patient education, the non-operative treatment should be holistic and involve pharmacological management, load management (including weight loss as required) and judicious physical therapy.

References:
RACGP. Guideline for the management of knee and hip OA. 2nd ed. 2018
European League Against Rheumatism (EULAR) recommendations for physical activity in people with inflammatory arthritis and OA. Rausch Osthoff et al. Ann Rheum Dis 2018 – 213585

Dr Doron Sher
M.B., B.S. (NSW), M.Biomed.E., F.R.A.C.S. (Ortho.)
Knee, Elbow, Shoulder Surgery

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Surgical Treatment of Knee Arthritis

Arthroscopy:
The role of arthroscopy to treat arthritis is controversial. Most people will agree that the success rate of physiotherapy and corticosteroid injections is about the same as the success rate of surgery after about 3 months of treatment.

About 70% of patients in both groups will do quite well.

Of the patients that undergo surgery that do not do well up to 15% will progress to total knee replacement within one year.

Of the patients that did not do well with non-operative treatment, about 70% will respond well to arthroscopic debridement (the same as the first group discussed above).

The presence of mechanical symptoms and an unstable meniscal tear at surgery is predictive of a better outcome.

Younger patients with mechanical symptoms from a meniscal tear with normal alignment and without tibial chondral lesions are likely to benefit (even if it is short lived) from arthroscopy.

High Tibial Osteotomy:
The principle of an HTO is to redirect the mechanical loading from the damaged area of the joint to the relatively well-preserved compartment. This is an operation that is designed to fail with time. The ‘less damaged’ compartment is now taking virtually the whole body’s weight and wears out over time.

In most cases, the weight- bearing axis is shifted from a worn medial compartment and passed through the healthier articular cartilage of the lateral side. Generally speaking, this allows patients to remain active without major activity modification. While many people manage to play sport or work in manual jobs, long distance running is not recommended.

The most common indication is treatment of isolated medial compartment arthritis in a young, active patient. HTO should be avoided if there is damage to the articular cartilage on the lateral side of the knee or if there has been a prior lateral meniscectomy.

Results are not as good in the presence of inflammatory arthritis, patellofemoral arthritis or in those over 65 years of age. Range of motion less than 90 degrees and obesity are contraindications to the operation and the patient needs to understand that the leg will be in valgus from a cosmetic perspective.

HTO can also be used to treat instability of the knee with changes to the slope of the tibia being used to compensate for ACL or PCL deficiency.

The alignment correction can be made by removing bone from the lateral side or adding bone to the medial side.

Converting an opening wedge HTO to a total knee replacement is quite straightforward with the results being almost as good as having a primary total knee replacement. This may require a bone graft and a longer period of restricted weight bearing than the closing wedge technique.

Converting a lateral closing wedge HTO to a total knee replacement can be somewhat challenging and the patients often end with scarring and shortening of the patella tendon which can affect the function of the prosthetic knee.

Lateral compartment arthritis can be treated with a distal femoral osteotomy but this is a much bigger procedure and rarely lasts more than 10 years.

Unicompartmental Knee Replacement:
There was a time when it was debated whether a patient should have a uni knee or an HTO but in fact they really should not be considered on the same patient as their indications are totally different.
While the concept of a uni knee replacement is good, they must be used judiciously. The results of converting a uni knee to a total knee are actually not very good. It is almost the same as the result of doing a revision of a standard total knee replacement (less patient satisfaction, poorer ROM).

Ideally the patient should be over 60yrs old, low demand, not have contractures or instability and have isolated medial compartment arthritis. This is completely different from the active and/or unstable patient for whom we perform an HTO. Many patients return to their desired level of sport but this is usually not something that involves contact or impact loading.

Total Knee Replacement:
Knee replacements these days should comfortably last 10-15 years. The revision rate does tend to rise from about 5% at 10 years to about 15% at 15 years in some series.

Return to sport after TKR

The prosthesis is made of metal and plastic and is not designed to withstand impact loading. Low impact exercise, cycling, golf, walking, swimming and doubles tennis almost certainly are acceptable sports to perform. If you already know how to do them then skiing and ice skating are not unreasonable but activities such as soccer, rugby, basketball, gymnastics, jogging and squash are best avoided.

Arthrodesis:
Fusing the knee is a very last resort these days. It is rarely used except as a salvage operation after a catastrophic event like an infection or major muscle or nerve injury.

The challenge when treating a young person with arthritis is that the prosthesis needs to last longer and is subject to higher loads and stresses than would be applied by an older person. Joint replacement in a younger patient should only be performed once all non operative measures such as NSAIDS, weight loss, exercise, activity modification and injections have been exhausted.

NOTES:

Dr John Negrine
M.B., B.S. (Syd), F.R.A.C.S. F.A. Ortho. A.
Adult Foot & Ankle Surgery

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Achilles tendon disorders – The ageing athlete

We in foot and ankle practice are seeing a veritable explosion in the incidence of Achilles tendon disorders. Whilst these conditions are certainly more common in sports people (Mafulli states that the incidence of Achilles tendon injuries in runners is ten times that in age matched controls) they are by no means confined to athletes, indeed in a study of 58 patients one third did not participate in any vigorous sporting activity at all.

Patients are running faster, longer and until they are older. With life expectancy now in excess of 80 in this country, as Bernard Salt (demographer) put it, “the medical profession has gifted this generation 20 years of active life.”

In this 20 years patients expect to maintain full activity. It is indeed a boom time for our joint replacement colleagues! (100,000 hip and knee replacements performed annually in Australia)

Achilles tendon problems are basically overuse or rupture. They occur at the midsubstance (where most ruptures occur) or at the insertion. Keep in mind that seronegative arthropathy can also cause Achilles symptoms.

Terms can be confusing so that we prefer to refer to the majority of patients as suffering “tendinopathy” or “tendinosis” rather than “tendonitis” which implies an inflammation (as mostly the condition is not inflammatory based on biopsies of patients with chronic Achilles symptoms).

Clinical presentation: The most consistent symptom complained of is pain. Typically start up (first thing in the morning of after resting for a period and then getting up). Many runners feel that when the tendon warms up the pain improves only to return when they cool down. In more serious cases patients are unable to walk because of pain. On examination observe swelling of the tendon or its insertion. Crepitus around the tendon occurs with paratenon conditions.

Investigations: The diagnosis is usually obvious from the history and clinical examination though investigations will yield additional information. Plain x- rays show the Achilles insertion and the presence or absence of intratendinous calcification (although many calcifications are present in asymptomatic patients). Ultrasound can demonstrate intra tendinous areas of degeneration and will show the width of the tendon. MRI will demonstrate bone and more importantly soft tissue and can help to differentiate a mechanical from an inflammatory cause. It will most reliably predict the findings at surgery.

Treatment: As with many musculoskeletal conditions we initially treat patients non-surgically though in the case of Achilles tendinopathy we know that a proportion of patients (30%) will fail non-surgical treatments based on longterm follow up studies.

Treatments for tendinopathies are wide ranging, and include but are not limited to orthotic devices, physiotherapy eccentric loading, laser treatment, ultrasound, extracorporeal shock wave therapy, NSAIDs, peritendinous corticosteroid injections, platelet rich plasma injections, whole blood injections, stem cell injections, bone marrow injections, autologous tenocyte injections, high volume saline injection, sclerosant injection and surgery. Naturally when so many different treatments are available no single treatment will help all patients.

In an attempt to make sense of these newer injectable remedies George Holmes et.al published a review of 9 randomised controlled trials. The study included 312 Achilles tendons. The interventions of interest included platelet-rich plasma (n = 54), autologous blood injection (n = 40), sclerosing agents (n = 72), protease inhibitors (n = 26), hemodialysate (n = 60), corticosteroids (n = 52), and prolotherapy (n = 20). Only 1 study met the criteria for a high-quality randomized controlled trial. All of the studies were designated as having a low quality of evidence. While some studies showed statistically significant effects of the treatment modalities, often studies revealed that certain injectables were no better than a placebo.

Similarly: Cochrane review they found 18 studies, which included 732 participants. Seven studies included athletes only. Study participants in the individual studies were mainly young to middle aged adults. Insufficient evidence for injectables to be recommended in painful Achilles tendons.

Conclusion: More work needed.

What do I do?

Mid substance tendinosis: rest in a walking boot, heavy load eccentric strengthening, never steroid injections. Surgically I attach the flexor hallucis muscle to the anterior surface of the Achilles tendon to give it vascularity and assist in the healing.

Insertional tendinopathy: (often calcific): rest in a walking boot, shock wave lithotripsy, never steroid injections.

Surgically: take down the Achilles insertion, remove the calcium and the dorsal 5mm of the calcaneus and re-attach the tendon. 6 weeks in plaster non-weightbearing and 4 weeks in a walking boot. Long recovery so reserved for patients that fail non-surgical remedies.

Dr Kwan Yeoh
M.B., B.S. (Hons) (Syd), F.R.A.C.S. (Ortho)
Hand, Wrist, Upper Limb & General Orthopaedics

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Flexor tendon injuries in the hand

ANATOMICAL REVIEW

- Flexor tendons
  - Flexor digitorum superficialis and flexor digitorum profundus
- Pulley system
  - Annular pulleys
  - Cruciate pulleys
- Neurovascular bundle

CHRONIC INJURIES

Tenosynovitis and trigger finger

- Inflammatory and mechanical causes.
- Mechanical = Tendon entrapment tenosynovitis
  ↳ Trigger finger
- Non-operative treatment
  - Splinting
  - Steroid injection: Risk of tendon rupture
- Operative treatment
  - Surgical release
- Post-operative treatment
  - Early use as tolerated

ACUTE INJURIES

Tendon laceration

- Commonly missed!
  - Surgical exploration of all hand lacerations
  - If delayed presentation, careful examination of each joint & sensation
  - Delayed treatment takes longer, more expensive, has worse outcomes
- Treatment always operative
- Post-operative treatment
  - Promote tendon excursion; limit tendon force
  - Start within few days post-operatively

- In conjunction with treating surgeon
- Splint & controlled movement
- Strengthening at 10 weeks

Closed tendon rupture

- Commonly missed
  - Due to forced finger flexion against resistance
  - Careful examination
    - Lack of active DIP joint flexion
    - Tenderness along flexor tendon path
- Treatment always operative
- Post-operative treatment
  - Similar to tendon laceration above

Flexor tendon pulley rupture

- Rare injury, but easily missed
  - Due to forced finger flexion against resistance
  - Careful examination
    - Swelling, tenderness along flexor tendon path
    - Weak active finger flexion
    - Bowstringing when severe
  - Imaging
    - XR; ultrasound; MRI
- Non-operative treatment
  - Swelling control; range of motion; tendon gliding
  - Pulley ring; taping; other orthoses
- Operative treatment
  - Pulley reconstruction
- Post-operative treatment
  - Early range of motion with pressure over pulley reconstruction
  - Protect for 3 months

FURTHER READING

1. Seiler JG III. Chapter 6 – Flexor Tendon Injury. In: Green's Operative Hand Surgery, 7th edn. Elsevier Inc; 2016. pp. 183-230.
2. Tang JB. Recent evolutions in flexor tendon repairs and rehabilitation. J Hand Surg (Eur). 2018 Jun;43(5):469-73.
3. King EA, Lien JR. Flexor Tendon Pulley Injuries in Rock Climbers. Hand Clin. 2017 Feb;33(1):141-8.

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Dr Paul Annett
M.B.,B.S,(Hons I) FACSP,
Sport & Exercise Medicine Physician

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Chronic Exertional Compartment Syndrome & Lower Leg Pain

Lower leg pain is not uncommon in the running athlete. Studies have shown that it is second only to the knee in causing lower leg pain.
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There are numerous causes of lower leg pain. The most common of these include tibial periostitis (the old 'shin splints'), stress fractures and chronic exertional compartment syndromes. Popliteal artery entrapment is a less common cause of pain although should be considered.

Tibial periostitis is an inflammation of the attachment of the deep calf (most likely the soleus muscle) to the medial tibial border. It generally comes about with increased training load on top of intrinsic factors such as poor foot biomechanics (particularly over-pronation), and calf inflexibility.

A stress fracture is defined as the failure of normal bone to cope with abnormal loads. It generally occurs in the tibia at the junction of the upper 2/3 and the lower 1/3. Similar factors may cause a tibial stress fracture that cause tibial periostitis, and there may be a continuum and overlap between the 2 conditions.

Chronic exertional compartment syndrome is a condition where the fascia or covering of a muscle group becomes excessively stiff. A muscle normally swells with activity due to increased blood flow. The fascia should stretch to accommodate this. If the fascia becomes stiff and non-compliant– and this may occur due to ageing, genetics or trauma – then the swelling muscle becomes constricted. The blood supply then becomes compromised, causing worsening pain with continued exercise.

The features of these conditions may vary both on history and examination. Tibial periostitis pain may initially warm up with activity, and there is no pain after a few minutes of running. There may, however, be considerable and prolonged post-activity pain, even the next morning. In contrast, compartment syndrome is usually painless for the first 5-10 minutes of activity (depending on its severity) and slowly worsens throughout the run. Usually it is severe enough to make the runner stop, but generally subsides within a few minutes.
Stress fracture pain is largely insidious and progressive, occurring initially after exercise only, then progressing to pain during activity and even rest and night pain.

Examination by an appropriately trained doctor, such as a sports physician, is important in the diagnosis of lower leg pain. The site and degree of tenderness and other associated findings may help to confirm the diagnosis. Examination post exercise in compartment syndrome may demonstrate a significant increase in muscle tension or even associated muscle herniation, which is a diagnostic finding.

Investigation of the problem is generally required to make a diagnosis and confirm your doctors' clinical suspicions. At least this may take the form of a simple x-ray, or may require more specific tests such as a bone scan (nuclear medicine) or an MRI. If a compartment syndrome is suspected the there may be a requirement to perform an invasive test known as a compartment pressure measurement. This involves placing a needle attached to a pressure gauge into the affected compartment and obtaining a reading both before and after exercise. The results of this may determine if this condition is present and what treatment may be required.

Dr Paul Annett
M.B.,B.S,(Hons I) FACSP,
Sport & Exercise Medicine Physician

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Treatment of the different lower leg complaints may be similar. It generally includes a period of rest with 'hands on' physiotherapy, a rehabilitation program of stretching and strengthening exercises, biomechanical assessment and occasionally an orthotic prescription.

In the case of compartment syndrome, surgery may be required. This involves surgical division of the fascial covering of the affected compartment. It is a generally successful procedure in the hands of a skilled surgeon

Appropriate assessment and advice from a practitioner with experience in this area can greatly shorten the time to diagnosis and institution of appropriate management, short-circuiting a lengthy period of inactivity.

NOTES:

Dr Paul Mason
FACSEP, M.B.B.S.(Hons), Bachelor Physiotherapy, Master Occupational Health

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Low back pain – aetiology and variant presentations

Lumbo-sacral pathology: the great mimic: It is well known that "back related hamstring" pain is often referred from the lumbo-sacral spine. Less commonly appreciated is that sacral region pain and lateral hip pain are also commonly caused by lumbo-sacral pathology. This is one reason why "greater trochanteric pain syndrome" and "sacro-iliac joint dysfunction" are often especially troublesome to treat.

Types of pain: Pain can arise in three broad categories from the lumbo-sacral spine, nociceptive, radicular and somatic referred. The resulting pain experience from each is subject to modulation by central processing, potentially leading to central sensitisation in some individuals. While it is commonly thought that "non-specific low back pain" has no pathoanatomical cause, there is a compelling body of evidence that, in concert with central processing, nociceptive causes exist.

Historical research on local nociception: One reason why this body of evidence is not more widely known is that much of it was published in the pre-digital era, with the methodologies utilised unlikely to receive approval of contemporary ethics committees. In 1949 Gunnar Wiberg published a case series on 200 patients operated on while awake under the cover of local anaesthesia only down to the ligamentum flavum. This meant he was able to expose the inter-vertebral disc and assess the pain response to irritation. He found that he was able to reproduce pain over the lumbar spine and sacrum in nearly all cases. This is consistent with the modern anatomical knowledge that the periphery of the annulus fibrosus contains nociceptive fibres. More recently, in 1991 Kuslich published the results of 193 patients operated on while awake under the cover of local anaesthetic. This study remains the most comprehensive of its nature to date, with multiple anatomical structures assessed for pain response by both blunt compression using forceps and low voltage electrical stimulation. The assessment of tissues was extensive, including skin, fat, fascia, supraspinous ligament, interspinous ligament, spinous process, muscle, lamina, facet joint capsule, facet synovium, nerve root, dura, compressed and normal nerve roots, annulus fibrosus of the disc, nucleus of the disc, and vertebral end plates. In 2/3 of patients, stimulation of the annulus fibrosis always reproduced pain which was similar to the pre-operative pain. In contrast, many structures to which pain is often attributed in the spine were found to be insensitive, including muscle, fascia and bone. In fact, it was noted that fascia could often be cut without anaesthesia.

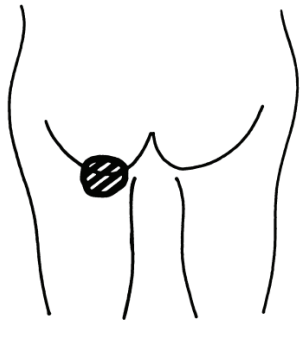
These two papers, while not meeting modern research standards, provide compelling evidence of the nociceptive contribution of the annulus fibrosis to non-specific low back pain. Given that research of this nature is unlikely to be conducted in future, the findings of these papers should be carefully considered before concluding non-specific low back pain has no pathoanatomical cause.

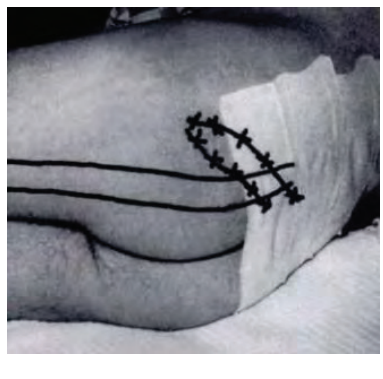
Historical research on referred pain: In 1958, Smyth published a paper based on a different methodology, which again would be unlikely to be approved by an ethics committee today. A series of patients with sciatic symptoms were operated on, after which nylon thread was wrapped around or passed through various anatomical structures. This included nerve roots, dura mater, ligamentum flavum, interspinous ligament and annulus fibrosis. The wounds were then closed, with the free ends of the nylon thread protruding through the skin. In most subjects, after 10-14 days, these threads were then tractioned, and the patients' pain response documented. In keeping with the previously mentioned papers, irritation of the annulus was found to reproduce 'ordinary back ache'.

Of more interest however, Smyth and colleagues were able to demonstrate two clear descriptions of referred pain. This included radicular pain, usually described as a shooting or electrical quality, and somatic referred pain, often described as a deep and intense ache. This was well described by the experience of one patient, who on gentle stimulation of a nerve root, experienced a “deep, boring, aching, unpleasant sensation felt diffusely in the soft tissue”, which then as pressure was increased felt “a sharp pain which ran, as if down a line, down the extremity”. This pattern of pain will be familiar to experienced therapists whose patients often not a period of aching pain prior to the onset of radicular symptoms. Hence it is apparent the nerve root irritation may often present as an aching pain, this somatic referred pain likely arising from stimulation of small innervating fibres to the dural sleeve around the nerve roots.

While the presentation of radicular pain is rarely confused, the diagnosis of somatic referred pain is not always straight forward. Nonetheless, there are observable patterns in the somatic referred pain pattern associated with irritation of nerve roots, which can aid diagnosis. Frequently L5 nerve root irritation produces pain over the sacrum, often extending to the posterolateral aspect of the hip. In contrast, the somatic referred pattern from S1 nerve root irritation is often felt in the buttock, often extending down the posterior thigh. It must be emphasised that this pain is qualitatively different to that of radicular pain, which is often described as of a shooting or electrical quality.

The images below provide examples of the somatic referred pain pattern from the S1 nerve root (left picture) and the L5 nerve root (area between crosses) (right picture)(Smyth 1958).





Conditions that are often confused with somatic pain arising from the lumbosacral spine include sacro-iliac joint pain, greater trochanteric pain syndrome (trochanteric bursitis), ischial tuberosity, hamstring strain or tendinopathy, and coccydynia. If there is any doubt about a diagnosis, CT guided corticosteroid injections targeting individual spinal nerves can be useful. Accurate diagnosis allows the therapist to provide targeted care specific to the pathology.

References

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

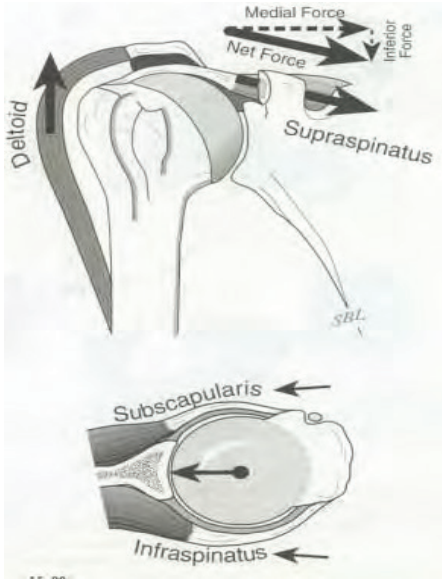
ORTHOSPORTS

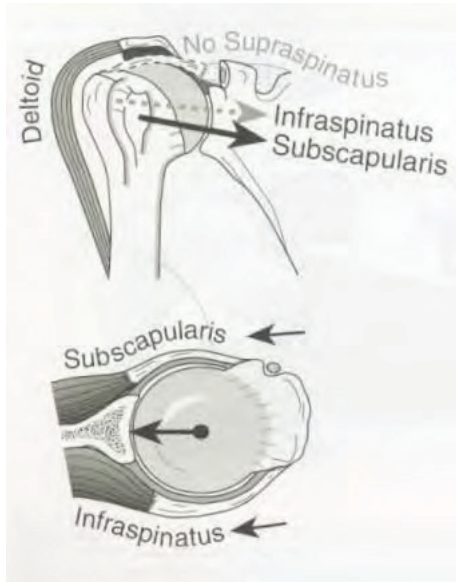
The rotator cuff-the science behind the disease

Rotator cuff tears are extremely common with at least 50% of 70-year-old people having full thickness tears. It should be noted, however, that the vast majority of these patients are asymptomatic. Studies show that larger tears get bigger with time and nonoperative management can provide a good outcome for certain patients. In addition in 40% of surgical cases the rotator cuff does not heal.

It is important to understand the pathology, and be able to select those patients who will benefit from non-operative treatment as opposed to surgical treatment. When surgery is contemplated one must be able to select the right procedure for that patient.

The concept of "force couples" helps one understand the rationale behind the different types of treatment.





The aim of the rotator cuff is to hold the humeral head adjacent to the glenoid and allowed deltoid to elevate the arm. When for example, there is an extremely large tear of supraspinatus and infraspinatus, the humeral head rides up under the acromion, and the deltoid loses its lever arm and cannot efficiently elevate the arm. If the rotator cuff in the transverse plane, that is subscapularis and infraspinatus, are intact, that is the force couples are intact, then even with the tear in supraspinatus, the transverse muscles are able to hold the humeral head against the glenoid, and this allows the deltoid to elevate the arm.

Degeneration, and subsequent tearing of the rotator cuff can be divided into two causes:

1. Mechanical
    a. acromial shape and distal clavicular hook
    b. mechanical overload
    c. trauma

2. Biological
    a. inflammatory cells
        i. macrophages regulate this process and release cytokines and other enzymes
    b. degradation of extracellular matrix
    c. death of tenocytes
    d. collagen disruption
    e. tendon tears
    f. muscle degeneration and atrophy

Patients present with pain, especially with elevation of the arm at night, as well as loss of function. There is often a history of trauma. Clinical examination can reveal wasting, loss of movement and particularly loss of external rotation power.

Plain x-rays are required to assess bone morphology and to exclude other causes of shoulder pain such as calcific tendinitis and tumours. Loss of greater tuberosity to acromial distance indicates a large and long-standing rotator cuff tear. Ultrasounds are quite inaccurate with some studies showing an accuracy rate of only 40%. An MR arthrogram is the investigation of choice. An MRI without contrast can be misinterpreted.

Non-operative treatment, is ideal for older people with good function who have a low demand on their shoulders. The aim is to reduce the inflammation and balance the force couples with

- cortisone injections
- anti-inflammatory tablets
- physical therapy
- activity modification

Operative treatment is recommended for younger and very active people, those people with a considerable functional deficit, and people who fail non-operative treatment. Treatment options include

- arthroscopic shoulder surgery incorporating a rotator cuff repair, an acromioplasty and biceps surgery if indicated
- open rotator cuff surgery which may include use of a graft or muscle transfer
- superior capsular reconstruction
- Reverse total shoulder replacement

It should be noted that even though arthroscopic rotator cuff repair has a 90% patient satisfaction, only about 60% of patients achieve a watertight repair. Poor prognostic factors include large tears, degenerate tears, smokers and diabetics.

Even with the successful outcome patients never have a normal shoulder. Studies have shown that the structural strength following rotator cuff repair is 50% of normal, and the material quality of the rotator cuff is 10% to 20% of normal. There is some question as to whether patients who undergo such surgery should return to heavy work or sporting activities that load the shoulder.

NOTES:

Dr Ivan Popoff
BPhEd (1986), MBChB (1991), F.R.A.C.S. (Ortho.)
Shoulder, Knee and Elbow Surgery

ORTHOSPORTS

Reverse Total Shoulder Arthroplasty (RTSA)

Initially designed to treat rotator cuff arthropathy, RTSA are now used to treat a wide variety of shoulder problems and their use is increasing. In Australia in 2017, 19,929 were performed, an increase of 4,148 from the previous year.

The most common conditions they are used for are:

- OA (45%),
- Cuff arthropathy (35%)
- Fracture (15%)
- Other conditions include AVN, inflammatory arthropathy, instability and tumors.

In a rotator cuff arthropathy, the presence of a large tear in the cuff results in loss of the deltoid-rotator cuff force couple. Unbalanced deltoid activity results in the humeral head being pulled superiorly causing eccentric loading at the glenohumeral joint resulting in mechanical wear. As the proximal migration increases the humeral head starts eroding into the acromion causing abrasion of the articular cartilage as well as releasing hydroxyapatite crystals from the acromion, resulting in a vigorous inflammatory response and biochemical dissolution of the articular cartilage. The functional effects, of the loss of the deltoid – rotator cuff force couple, is a marked disturbance in shoulder mechanics and in it's most severe presentation pseudo-paralysis.

The initial treatment for a cuff arthropathy is always non-operative, patients often respond well to physiotherapy, corticosteroid injections, NSAIDS and simple analgesics. However, prior to the development of RTSA there was no satisfactory surgical treatment for those patients.

Hemiarthoplasty resulted in variable pain relief and no improvement in function, total shoulder replacement (TSA) was better for relief of pain but the glenoid component failed due to the proximal migration of the humeral head and cyclical eccentric loading. Early attempts at prevention of proximal humeral migration failed.

Grammont in the mid1980s developed the first functional RTSA by medialising the centre of rotation to the Glenoid surface, rotational forces at the prosthesis- bone interface were turned into compression and a stable prosthesis. This also resulted in an increase in the deltoid moment arm. The function of the rotator cuff w.r.t. elevation was taken over by the geometry of the prosthesis.

This is however not true of rotation particularly external rotation. RTSA stability is a combination of prosthesis geometry, orientation and soft tissue tension. Soft tissue tension is mainly achieved by inferior displacement of the humerus. One of the results of this is a change in orientation of the posterior deltoid minimising its effectiveness as a potential external rotator. Usually to get satisfactory external rotation patients generally require part of their posterior cuff to be functional. Patients who have lost teres minor may need a latissimus dorsi transfer at the time of RTSA for a functional shoulder.

Results of RTSA are generally very good with predictable relief of pain and restoration of a functional but not normal shoulder. The 10 year survival rate is greater than 90% with some drop off in shoulder scores after around 8 years.

Dr Ivan Popoff
BPhEd (1986), MBChB (1991), F.R.A.C.S. (Ortho.)
Shoulder, Knee and Elbow Surgery

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RTSA in Osteoarthritis:
Indications are the condition of the rotator cuff and Glenoid orientation.
Rotator cuff failure is the leading cause for revision in TSA. If the OA patient's rotator cuff is torn or in poor condition it is often prudent to opt for a RTSA to minimize the need for repeat surgery.

Osteoarthritis of the shoulder is primarily a posterior joint disease, with advancing disease retroversion of the glenoid developing with fixed posterior subluxation of the humeral head. A RTSA gives the most predictable outcome in this scenario.

RTSA in fracture:
Management of 4 part proximal humeral fractures with hemi arthroplasty gives variable results due to non-union and or reabsorption of the tuberosities and subsequent loss of cuff and shoulder function. With RTSA, functional elevation is maintained even in the presence of tuberosity non-union.

Complications:

- Neurological: Normally traction related and transient
- Inter operative fracture: Usually not problematic if identified and treated at time of surgery, can be cause of early loosening
- Infection: Incidence 1-10%, more common in Rhumatoids
- Scapula notching: Caused by inferior impingement of humeral prosthesis on scapula neck, if severe may result in Glenoid loosening
- Dislocation: Most common complication, usually occurs early, first time closed reduction sling for 4/52 if reoccurs or closed reduction not possible, then revision surgery
- Acromial Fracture: Due to increased tension in deltoid on osteoporotic acromion. Sudden onset superior shoulder pain, tender acromion may need CT or bone scan if not seen on x ray, treat 4/52 in sling occasionally need ORIF

Dr Todd Gothelf
MD (USA), FRACS, FAAOS, Dip ABOS
Foot, Ankle, Shoulder Surgery

ORTHOSPORTS

Return to Work and Sport After Rotator Cuff Repair

Rotator cuff tears result in weakness and dysfunction of the shoulder, and patients can experience considerable pain and disability as a result. Surgical repair is generally accepted as appropriate treatment for symptomatic full thickness rotator cuff tears, with greater than 90% success rates.

The concept of "success rate" is one that is subjective to each individual patient. Expectations will vary depending upon varying demands activity levels. Success for some may be that they are simply able to drive, walk the dog, or do the laundry or cooking. Far more challenging is to meet the expectations of workers or athletes. The higher demands of these patients may lead to a lower success rate when there is a failure of returning to one's original level of work or sport.

Return to Sport

The following articles review return to sport after rotator cuff tears. All studies generally report about an 80% return to the same sport. There were varying ranges of return to the same level, from 50% to 80%.

1. Antoni and Klouche, et al. Return to Recreational Sport and Clinical Outcomes with at least 2 years follow-up after arthroscopic repair of rotator cuff tears. Orthopaedics and Traumatology: Surgery and Research 102(2016) 563-567.
    a. In patients involved in upper limb sports (tennis, swimming, golf) 86% returned to the same sport. Mean time to return to sports was 6 months. Nearly 80% returned at the same or higher level.
2. S Klouche, N Lefevre. Return to Sport After Rotator Cuff Repair: A Systematic Review and Meta-analysis. American Journal Sports Medicine, 2016.
    a. 25 studies reviewed, 860 patients treated with rotator cuff repair with mean 3.5 years.
    b. Overall return to sport was 85%, with 66% at equivalent level of play after 4 to 17 months. Of the professional and competitive athletes, 50% returned to the same level of play.
3. S Bhatia. JA Greenspoon. Two-Year Outcomes After Arthroscopic Rotator Cuff Repair in Recreational Athletes Older Than 70 Years. Am Journal Sports Med 2015.
    a. 49 shoulders with mean age of 73 years, mean follow up 3.6 years.
    b. All patients had significant improvements in outcome. 77% were able to return to their sport at a similar level of intensity.
4. D Liem, S Lichtenberg. Arthroscopic Rotator Cuff Repair in Overhead-throwing Athletes. Am Journal Sports Medicine, 2008.
    a. 21 overhead throwing athletes after rotator cuff repair were followed up for 2 years, and with MRI.
    b. Results- Re-tear rate was 24%, but sporting activity was not influenced by repair integrity.
    c. All patients returned to their overhead throwing sport an average of 6 months after surgery.
    d. Participation and duration were not significantly lower than prior to injury. Patients estimated their activity level to be 91% their non-injured condition significantly better than 35% of their activity level prior to surgery.
5. B Sonnery-Cottet, TB Edwards, et al. Rotator Cuff Tears in Middle-Aged Tennis Players: Results of Surgical Treatment. Am Journal Sports Med, 2002.
    a. 51 tennis players were reviewed after rotator cuff repair, with average 4 years follow up.
    b. 47 (92%) patients were satisfied with their results.
    c. 40 (80%) patients were able to return to tennis at an average 10 months after surgery.

Dr Todd Gothelf
MD (USA), FRACS, FAAOS, Dip ABOS
Foot, Ankle, Shoulder Surgery

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Return to Work

Rotator cuff tears are one of the most common disabilities experienced by worker's compensation patients. A rotator cuff disorder ranks second only to back and neck pain in frequency of occurrence in the workplace. Most of these patients are relatively young, male, labourers. Return to work can be particularly challenging for these patients. The studies below report that only 60% of worker's return to their previous work duties after a rotator cuff tear and surgery.

1. H Razmjou, S Lincoln. The effect of expedited rotator cuff surgery in injured workers: a case-control study. J Shoulder Elbow Surg, 26, 1196-1202. 2017.
    a. Showed Injured workers who underwent an expedited rotator cuff surgery reported less disability and had a more successful return to work after surgery than injured workers who waited longer for specialist assessment and surgery within a public health care system.
2. L Nove-Josserand, JP Liotard, G Walch. Occupational Outcome after surgery in patients with a rotator cuff tear due to a work-related injury or occupational disease. A series of 262 cases. Orthoapedics & Traumatology: Surgery & Research, (2011) 97, 361-366.
    a. Return to work occurred in 60% of cases.
    b. Factors that prevented return to work:
        i. Retirement- 14%
        ii. Unrelated medical condition 10%
        iii. Outcome of the operated shoulder 16%.
        iv. Age had an impact on return to work.
    c. Type of work and nature of injury did NOT affect return to work but did affect time away from work.
    d. Age was a decisive factor for return to work. Retirement was most common choice in age over 55.
    e. Conclusion: A pre-operative evaluation of the patient's probability of return to work shoulder be done based on occupational and injury features. Management can be improved knowing the factors and conditions that influence return to work.
3. R Balyk, C Luciak-Corea. Do Outcomes Differ after Rotator Cuff Repair for Patients Receiving Worker's Compensation. Clin Ortho and Rel Research, 2008.
    a. WC patients were younger, more likely to smoke, have a traumatic injury, and to undergo surgery within 6 months.
    b. WC had a lower recovery for all outcomes. However, when considering factors that are more common in WC patients i.e. smoking, large tears, Outcomes between WC and non-WC were more similar.
4. R F Henn, L Kang. Patients with Workers' Compensation Claims Have Worse Outcomes After Rotator Cuff Repair. JBJS 2008.
    a. Results: Patients with WC were significantly younger, had greater work demands, lower marital rates, education levels, and preoperative expectations for the outcome. WC had significantly lower preop scores. These patients had worse outcomes one year postop.
    b. Conclusion: Patients with WC claims report worse outcomes, even after controlling for confounding factors. This study supports that WC patients do worse overall regardless of comorbidities.

NOTES:

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Concord	47-49 Burwood Road CONCORD NSW 2137	Tel Fax	**02 9744 2666** **02 9744 3706**	**Doctors Consulting here** Dr Todd Gothelf Dr Paul Mason Dr John Negrine Dr Rodney Pattinson Dr Doron Sher Dr Kwan Yeoh
Hurstville	Level 7 Waratah Private 29-31 Dora Street HURSTVILLE NSW 2220	Tel Fax	**02 9580 6066** **02 9580 0890**	**Doctors Consulting here** Dr Paul Annett Dr Jerome Goldberg Dr Todd Gothelf Dr Andreas Loefler Dr John Negrine Dr Rodney Pattinson Dr Ivan Popoff Dr Allen Turnbull Dr Kwan Yeoh
Penrith	Suite 5B 119-121 Lethbridge St PENRITH NSW 2750	Tel Fax	**4721 7799** **4721 7997**	**Doctors Consulting here** Dr Todd Gothelf Dr Kwan Yeoh
Randwick	160 Belmore Road RANDWICK NSW 2031	Tel Fax	**02 9399 5333** **02 9398 8673**	**Doctors Consulting here** Dr John Best Dr Jerome Goldberg Dr Todd Gothelf Dr Andreas Loefler Dr Paul Mason Dr John Negrine Dr Rodney Pattinson Dr Ivan Popoff Dr Doron Sher

www.orthosports.com.au

47-49 Burwood Rd
CONCORD NSW 2137
Tel: 02 9744 2666
Fax: 02 9744 3706

Lvl 7, 29-31 Dora Street
HURSTVILLE NSW 2220
Tel: 02 9580 6066
Fax: 02 9580 0890

119-121 Lethbridge St
PENRITH NSW 2750
Tel: 02 4721 7799
Fax: 02 4721 7997

160 Belmore Rd
RANDWICK NSW 2031
Tel: 02 9399 5333
Fax: 02 9398 8673