The rational use of ultrasound in musculoskeletal medicine

Dr Mike Cleary
President
NZ Association of Musculoskeletal Medicine

Conflict of interest: The Association is seeking members
I don’t have an ultrasound machine
Ultrasound in medicine

The advances in medical imaging go hand in hand with computing developments

36 years ago:
  • Variations on plain x-rays – fears of a silver shortage (cf Rare earth metals now)
  • Nuclear medicine

1977: Hospital Ground Round: an A mode US was demonstrated by Tom Fiddes O&G Professor Ian Donald, University of Glasgow, developed and improved on the devices that he used in pregnancy monitoring fetal development.

1978 “Put a scanner in the works”
The public appeal to get a CT head scanner for neurology at Dunedin Hospital to do away with air encephalograms and other methods of neuroradiolgy
During my O&G diploma 1981 we had to make a case for a fetal US

Now it would be exceptional **not** to have at least one US during a pregnancy.

The accepted use of US in diagnosing vascular conditions such as aortic aneurysms, viscera and in cardiology.

The arrival of MRI:
- Prior to that our Palmerston North patients would fly to Auckland for an MRI

PET scans are the latest imaging on the block
- There is a nuclear reactor adjacent to Wellington Airport to make the isotopes
- At present the place for this in musculoskeletal imaging is unclear other than it is very sensitive (The old story of treating the patient not the imaging report?)
Postgraduate Certificate in Clinician-Performed Ultrasound (PGCertCPU)

**Postgraduate Papers**

**GENX 717 Generalist Medical Echocardiography**

*Principles of generalist performed echocardiography, its application to medical diagnosis and management, especially in an emergency or rural setting to more accurately diagnose and appropriately manage medical problems.*

**GENX 718 Generalist Medical Ultrasound**

*Principles of generalist performed ultrasound, its application to medical diagnosis and management, especially in an emergency or rural setting to more accurately diagnose and appropriately manage medical problems.*

AAA, FAST scan – trauma, gallstones, bladder, early pregnancy, foreign body, hydronephrotic kidney, above knee DVT, pericardial effusion, impaired LV systolic function, volume assessment in the shocked patient, significant mitral incompetence and aortic stenosis.

**Procedures under US guidance** - draining pleural effusions and ascites, insertion of suprapubic catheter, bladder aspiration, difficult vascular access and removal of foreign body.

*(No mention of US for musculoskeletal diagnosis or injections)*
Nurture a working relationship with your musculoskeletal radiologist:  Ring and discuss what is appropriate

**Operator dependent : 23 April**

**INDICATION:**
Right wrist symptoms following injury. ?TFCC complex abnormality  O/E  ECU subluxation

**FINDINGS:**
The tendons around the right wrist are normal.
No subluxation of the ECU was demonstrable on dynamic scanning.

There is increased flow on Doppler in the TFCC, but a tear has not been demonstrated.

**IMPRESSION:**
Inflammatory change in the region of the TFCC, there is no evidence of a tear.
The tendons around the wrist joints show normal appearances.

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**1 July**

**INDICATION:**
?Subluxing extensor carpi ulnaris tendon.

**FINDINGS:**
The extensor carpi ulnaris is situated in the groove in the pronated position. However in **full supination** it does sublux out of the groove.

A tiny amount of fluid is seen in the sheath.
Interestingly the opposite wrist shows a similar degree of subluxation so the significance of this is unclear.
Otherwise the extensor tendons appear normal.
No evidence of tenosynovitis elsewhere.

**IMPRESSION:**
There is symmetrical subluxation of the extensor carpi ulnaris tendons.
No injection was performed.
**INDICATION: MRI**  May 2013   
Painful ulnar aspect of wrist. Suspected TFCC or ECU pathology.  
**FINDINGS:**  
The triangular fibrocartilage has some horizontal bands of hyperintensity consistent with delamination but does not appear disrupted. There is neutral ulnar variance.  
The **extensor carpi ulnaris tendon does show some hyperintensity** as it traverses the ulnar styloid but remains in the groove in this neutral position.  
The other extensor tendons appear normal.  
The proximal surface of the lunate shows some signal change and there is a small focal area of cartilage loss.  
The features suggest ulnar lunate impingement. The scapholunate ligament is intact and the lunatotriquetral is intact.  
Alignment of carpal bones is otherwise normal.  
The extrinsic wrist ligaments appear normal and articular cartilage is preserved elsewhere throughout the wrist. The carpal tunnel, median and ulnar nerves appear normal.  
**IMPRESSION:**  
Delamination of TFCC without disruption.  
ECU tendinopathy.  
Probable mild ulnar lunate impingement.

**INDICATION:** July 2013   
?Subluxing extensor carpi ulnaris tendon. MRI showed positive ulnar variance with ulnar lunate impingement.  
**FINDINGS:**  
The extensor carpi ulnaris is situated in the groove in the pronated position. However in full supination it does sublux out of the groove.  
A tiny amount of fluid is seen in the sheath. Interestingly the opposite wrist shows a similar degree of subluxation so the significance of this is unclear. Otherwise the extensor tendons appear normal.  
No evidence of tenosynovitis elsewhere.  
**IMPRESSION:**  
There is symmetrical subluxation of the extensor carpi ulnaris tendons. No injection was performed.
Rational use of shoulder ultrasound

Can we make a valid clinical diagnosis?

1998 Australian Guidelines; 2007 and 2008 reviews agree with the 2003 ACC Shoulder Guidelines:

“There is no evidence that any specific test is both valid and reliable for the diagnosis of shoulder injuries”
Infancy

- Developmental Dysplasia of the hip - DDH:
  - Bilateral in 20% of cases

Infants at increased risk of DDH:
- Family history in a first degree relative
- Firstborn infant -
- Breech presentation especially if delivered vaginally – all elective LSCS now?
- Infants with poor muscle tone or neurological problems

If they are significantly at risk perform a screening US at 6 weeks.

US is more difficult and less accurate after 3/12 of age
- ‘Uneven skin creases’ should not on its own be a justification of investigation, rather – If there limited abduction of the hip in flexion at 6/52 then orthopaedic referral is required.
Lumps and bumps

Less than 1% of those submitted for pathological analysis are malignant.

UK criteria for urgent referral of a soft tissue mass:

- Soft tissue mass > 5cm (golf ball
- Painful lump
- A soft tissue mass that is increasing in size or appearance
- A lump of any size that is deep to the muscle fascia
- Recurrence of a lump after a previous incision

In a large study from the UK, US examination of 80% of soft tissue masses referred from primary care could be classed as benign and no further imaging was needed.
The most common findings in soft tissue masses referred for assessment at tertiary centres are

- Lipoma
- Normal tissue
- Ganglion or cyst
- Inflammatory lesion
- A-V malformation
# Double pathology - 36 year old call centre worker

<table>
<thead>
<tr>
<th>6 May 2013  Plain x-ray</th>
<th>1 July 2013 Dynamic US</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INDICATION:</strong></td>
<td><strong>INDICATION:</strong></td>
</tr>
<tr>
<td>• Injury to the right elbow in a fall 1/2/2013. Persisting pain.</td>
<td>• Injury to right elbow. Tender lateral aspect.</td>
</tr>
<tr>
<td><strong>FINDINGS:</strong></td>
<td><strong>FINDINGS:</strong></td>
</tr>
<tr>
<td>• There is no effusion. The joint space is normal medially and laterally. There is no fracture or periosteal reaction.</td>
<td>There is extensive thickening of the common extensor origin with some hypoechoic bands within it. Marked hypervascularity is seen with colour Doppler in this tendon. There is no effusion of the elbow joint. The biceps and triceps appear normal. In addition there is oedema of the ulnar nerve. This subluxes across the medial epicondyle in full flexion and this appears to be associated with pain and tingling. The common flexor origin appears intact. The triceps insertion appears normal.</td>
</tr>
<tr>
<td>• The common flexor and common extensor tendon origins are normal.</td>
<td><strong>IMPRESSION:</strong></td>
</tr>
<tr>
<td><strong>IMPRESSION:</strong></td>
<td>1. Severe common extensor origin tendinopathy.</td>
</tr>
<tr>
<td>• There is no bony or joint abnormality.</td>
<td>2. Subluxing ulnar nerve with ulnar neuritis.</td>
</tr>
</tbody>
</table>
Teninopathy/Tendinosis/Tendinitis

There is extensive thickening of the common extensor origin with some hypoechoic bands within it. Marked hypervascularity is seen with colour Doppler in this tendon.

There is no effusion of the elbow joint.

Continuum model – concept of staging tendinopathies
97% of tendons that rupture have degenerative tendinopathy.

Two thirds of tendons that rupture are pain-free before rupture.

Pain can occur anywhere along the tendinopathy continuum which is confusing for us but consistent with the well known dissociation between pain and pathology in tendinopathy.

Tendons that appear normal on imaging can be painful and tendons that are abnormal on imaging can be pain free.

Evidence favours the neoinnervation that accompanies neovascularisation as the structure responsible for pain.

The “other cause” of tendon pain has proved elusive (Like O.A pain)
There is a substantial body of evidence indicating that ongoing tendon degeneration is an active process with involvement of many aspects of inflammation-mediated responses. Scott et al 2013

**Brain drugs of the future** BMJ 1998; 317 1698 - 701
Susan Greenfield (The promiscuous transmitter)
One transmitter may be linked to many disorders, and one disorder to many transmitters
Classic transmitters have nonclassic modulatory functions too
Substances such as nitric oxide and acetylcholinesterase have unexpected signalling properties
ABSTRACT:

It is currently widely accepted among clinicians that chronic tendinopathy is caused by a degenerative process devoid of inflammation.

Current treatment strategies are focused on physical treatments, peri-tendinous or intra-tendinous injections of blood or blood products and interruption of painful stimuli.

Results have been at best, moderately good and at worst a failure.

The evidence for non-inflammatory degenerative processes alone as the cause of tendinopathy is surprisingly weak.

There is convincing evidence that the inflammatory response is a key component of chronic tendinopathy. **Newer anti-inflammatory modalities may provide alternative potential opportunities in treating chronic tendinopathies and should be explored further.**
Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy

J L Cook,¹ C R Purdam²


Figure 1  Rotator cuff tendinopathy: a model for the continuum of pathology. Dotted arrows, potentially reversible; solid two-directional arrows, reversible; solid single-directional arrows, irreversible; dotted single-directional arrows, irreversible without going through an intermediate step.
<table>
<thead>
<tr>
<th></th>
<th>Reactive Tendon</th>
<th>Dysrepair</th>
<th>Degenerative</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Histology</strong></td>
<td>Cells numbers increase</td>
<td>Similar to reactive but greater matrix breakdown</td>
<td>Areas of cell death from apoptosis, “exhaustion”</td>
</tr>
<tr>
<td></td>
<td>They tend to be “chondroid”</td>
<td>Increased chondroid cells as well as myoblasts</td>
<td>Resultant acellularity</td>
</tr>
<tr>
<td></td>
<td>More proteins made</td>
<td>Focal discontinuity of the collagen fibres</td>
<td>Areas of disordered matrix</td>
</tr>
<tr>
<td></td>
<td>More water bound</td>
<td>Further protein so swelling</td>
<td>lacking collagen filled with vessels, matrix breakdown products</td>
</tr>
<tr>
<td></td>
<td>Collagen fibres separated</td>
<td>Neurovascular ISQ</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neurovascular ISQ</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>US Imaging</strong></td>
<td>Fusiform swelling</td>
<td>Discontinuity of fascicles</td>
<td>Hypoechoic regions</td>
</tr>
<tr>
<td></td>
<td>Intact collagen fascicles</td>
<td>Small areas hypoehchogenicity</td>
<td>Few reflections from collagen fascicles</td>
</tr>
<tr>
<td></td>
<td>Diffuse hypoehchogenicity between fascicles</td>
<td>Doppler shows increased vascularity</td>
<td>Numerous large vessels</td>
</tr>
<tr>
<td></td>
<td>Minimal change</td>
<td>Swollen, increased signal</td>
<td>Increased tendon size &amp; intratendinous signal</td>
</tr>
<tr>
<td><strong>MRI</strong></td>
<td>Minimal change</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Clinical</strong></td>
<td>Acute overload from unaccustomed use “Start up Pains”</td>
<td>The chronically overloaded young tendon</td>
<td>Generally older person</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Seen across a spectrum of ages and load environments</td>
<td>recurrent pain</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronically overloaded tendon in the younger person or elite athlete</td>
<td></td>
</tr>
</tbody>
</table>
Why this emphasis on tendinopathy in the context of rational use of US?

Really just to make you aware of the issues when it comes to interpreting the imaging reports and whether the report helps you in the management of the patient in front of you.

There is no clinical gold standard when it comes to the diagnosis of tendinopathies (Jill Cook).

Many astute practitioners have been caught out.
You don’t have the time
You don’t feel confident to do the injection
Do you know the efficacy of the injection?
You can distance yourself from the treatment decision
You distance yourself from adverse effects
The technology implies a better treatment outcome
Cost to patient and your costs (Materials and time)
Delays in patent treatment: jeopardise job, more pain
1998
The Australian National Musculoskeletal Medicine Initiative
“...objective evidence of the utility of physical examination tests is either inadequate or non existent... ...clinical examination is not, in general, scientifically based”

2003
Page 6, ACC Shoulder Guidelines
“There is no evidence that any specific test is both valid and reliable for the diagnosis of shoulder injuries”

2008:
“Currently, almost without exception, there is a lack of clarity with regard to whether common tests used in clinical examination are useful in differentially diagnosing pathologies of the shoulder”

Physical examination tests of the shoulder: A SR. BJSM
Systematic Review

Lewis J and Tennent D
(2007)
How effective are our
diagnostic tests for
rotator cuff pathology?
Evidence Based Sports Medicine
(2nd edition).
MacAuley D and Best T (Eds).

“It is not possible to make a
definitive diagnosis with the
clinical tests currently in use”
Reason 1: Morphology

- Majority of anatomical text books describe tendons as distinct structures
  
  (Basmajian 1975, Williams et al 1995)

- RC tendons fuse to form a common insertion (aponeurosis) on the humeral tuberosities
  
  (Clark and Harryman 1992)
Historically, musculoskeletal assessment of the shoulder has been based around a premise that it is possible to isolate individual structures and apply a mechanical procedure that either compresses or stretches the tissue of interest or requires it to contract. However, it is unlikely that any test would not stretch or compress adjacent structures or cause them to contract during the procedure. Without doubt this is one of the reasons why more recent studies on the sensitivity, specificity and predictive accuracy of tests1–4 have concluded that, although they have a high sensitivity and reproduce symptoms, they have an associated low specificity which substantially reduces their utility in deriving a specific diagnosis. As such the commonly used orthopaedic special tests should be thought of as pain or symptom provocation tests, without the ability to contribute to a structural diagnosis. ...

...The ability to achieve an accurate structural diagnosis is further challenged by the poor correlation between radiological imaging and symptoms. Using MRI, Frost et al21 reported that structural pathology in the rotator cuff in 42 individuals with a clinical diagnosis of subacromial impingement syndrome was similar to that in 31 age-matched asymptomatic individuals. Similar findings have been reported by others investigating the relationship between radiograms, diagnostic ultrasound (US) and MRI, and symptoms.22–26
“Neural Blockade for Diagnosis and Prognosis”,


“Even though diagnostic blocks are motivated by a desire to obtain specific convincing data, the procedure is also inevitably a complex social interaction.

Whereas the physician may seek pathophysiologic information, the patient may be looking for reassurance, confirmation of their suspicions or proof to persuade doubting family members, certification of their disability for legal or financial reasons, or may simply wish to please the physician...

...Patients obtain relief from placebos administered during acute pain approximately one third of the time, but obtain relief from chronic pain in approximately two thirds of cases after administration of placebo... ...Injections like surgery are especially potent placebos compared to pills...

...Compelling evidence with regard to placebo responses leads to the conclusion that the ambiguity created by these responses is a major impediment to the valid use of neural blockade for diagnosis.”

(You get 30% for free !)
95% of RC pathology caused by the acromion

Neer 1972

All clinical tests will stretch and/or compress the subacromial bursa
Reducing the tests specificity and ability to differentiate structures

Rising incidence of acromioplasty

NY State, USA
1996: 30.0 per 100,000
2006: 101.9 per 100,000
= 254.4% increase in 11 years

At around US$5000 per procedure
US$100,000,000

Is the effect of subacromial decompression primarily denervation of the bursa?

Bursectomy with and without acromioplasty has the same effect
(Budoff et al 2005, Henkus et al 2009)

...so why remove the acromion???
Is the main benefit of an acromioplasty Relative Rest?

Return to work:
- Non manual workers 6/52
- 85% manual workers 12/52

Return to driving:
- Average – 29 days (range 2-220)

Return to work:
- Non manual workers 9 days (range 2-39)
- Manual workers 3/52 (range 2-72)

Return to driving:
- Median – 13 days (range 2-53)

- Codeine phosphate 30mg
- + 1gm paracetamol 4x/day for 2/52

Mcclelland et al 2005 ANZ J Surg
Charalambos et al 2010 Shoulder & Elbow

Conclusions:
1. Brits are stronger than Aussies?!? (2012 Olympics suggest maybe?!?)
2. Relative rest (and analgesics) may be the mechanism of benefit
Does imaging give validity to a diagnosis?

It’s more of the same

Sadly, many structural lesions are present in asymptomatic subjects

You doubtless have patients with gross deficiencies in their rotator cuff who have painless shoulder function
US diagnosis of RC disease

90 asymptomatic subjects
- with no history of shoulder problems

(Milgrom et al 1995 JBJS 77-B)

<table>
<thead>
<tr>
<th>Decade</th>
<th>n</th>
</tr>
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<tbody>
<tr>
<td>4th decade</td>
<td>18</td>
</tr>
<tr>
<td>5th decade</td>
<td>18</td>
</tr>
<tr>
<td>6th decade</td>
<td>18</td>
</tr>
<tr>
<td>7th decade</td>
<td>13</td>
</tr>
<tr>
<td>8th decade</td>
<td>18</td>
</tr>
<tr>
<td>8th to 9th decade</td>
<td>10</td>
</tr>
</tbody>
</table>

The incidence of asymptomatic tears ↑ with age

- Dominant side pathology = non-dominant side pathology
- AFTER 5th decade 55% incidence of asymptomatic FTT

“Treatment should be based on clinical and NOT on imaging findings”
Why the bursa??

The bursa is innervated
Aszmann et al (1996)

Many free nerve endings and substances to stimulate

- Cytokines
  - IL-1β, TNF-α, VEGF, IL-6
- MMPs (matrix metalloproteinases)
  - MMP1, MMP9
- Cyclo-oxygenase enzymes
  - Cox 1, Cox 2
- Neuropeptides
  - Substance P
  - IL-1, IL-6, MMP1, MMP9, TNF-α, Cox 1, Cox 2

Potential mediators of inflammation and may have a catabolic effect on tendon

Correlation between pain scores and chemicals
Gotoh et al (2001)

Bursectomy may be as beneficial as acromioplasty and bursectomy
Henkus et al (2009)
MRI and RC pathology

*n* = 96 asymptomatic subjects (Sher et al 1995 JBJS 77A)

- **Dominant shoulder scanned**
- **Questionnaires used to exclude history of shoulder pathology**

<table>
<thead>
<tr>
<th>40-60 years</th>
<th>&gt; 60 years (n=46)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 in 4 (28%) structural pathology</td>
<td>1 in 2 (54%) structural pathology</td>
</tr>
<tr>
<td>1 (4%) FTT</td>
<td>13 (28%) FTT</td>
</tr>
<tr>
<td>6 (24%) PTT</td>
<td>12 (26%) PTT</td>
</tr>
</tbody>
</table>

The presence of a RC tear does not correlate with painful shoulder dysfunction.
Subacromial Impingement

- Incidence of tears increased with age
- Cause of RC disease is degeneration
- Majority of partial thickness tears are either intra-tendinous or articular side tears.
- Tensile forces transmitted through CAL → acromial spurs and CAL changes (spur is 2ndry)

Loehr and Uhthoff (Orthopaedic Trans 11: 237, 1987) -306 RC cadaver
Ozaki et al (JBJS 70A: 1224-1230, 1988) -200 RC (n=100) cadaver
Ogata and Uhthoff (Clin Orth 254: 39-49, 1990) -76 RC (n=38) cadaver
If the acromion is responsible then there should be a correlation between acromial shape, symptoms and outcome

Research investigations have failed to demonstrate this relationship

- No significant correlation between acromial morphology (shape) and rotator cuff pathology in patients with shoulder pain over 50 years

- No clinical correlation between acromial morphology (shape) and rotator cuff pathology and symptoms in 59 asymptomatic people
  "Surgeons should interpret hooked acromia and rotator cuff tears with caution"

- At 6 months post surgery (n=55) no clinical correlation between acromial morphology (shape) and rotator cuff pathology and symptoms and outcome
  Snow et al (2009) Shoulder & Elbow
What is the treatment for a Reactive Tendinopathy?

Initial: **Relative Rest**

Progression if required: **Relative Rest** +

**Ibuprofen**

- NSAID may have a detrimental effect on tendon repair.
- Ibuprofen (and, indomethacin and naproxen sodium) inhibit expression of aggrecan.
- Ibuprofen (and, celecoxib) down regulate cellular response
- Ibuprofen may be preferred as it may not have a detrimental effect on ultimate tendon repair.

What is the treatment for a Reactive Tendinopathy?

Stage 1: Relative Rest

Stage 2: Relative Rest + Ibuprofen

Stage 3: Relative Rest + Injection Therapy
How to inject?
Ultrasound guided versus landmarked guided injections

Randomised to
Group 1  (n= 20) Blinded Technique: (20mg triamcinolone)
Group 2  (n= 21) Guided Technique: (20mg triamcinolone)

Outcome measurements
50% improvement in symptoms
Shoulder Function Scale, VAS (pain), Active and passive ROM at 6/52

Landmark guided:  
1 / 20  
(5%)

Ultrasound guided:  
11 / 21  
(52%)

Accurate versus inaccurate subacromial bursal injections

33 subjects received subacromial injection
- Non traumatic lateral shoulder pain / pain with abduction
- +ve H&K, +ve Neer, inability to lie on shoulder

Group 1 \((n=33)\) Following injection immediately T2 MRI (fat saturated)

Outcome measurements
Constant Score, Simple Shoulder Score, VAS (pain and impairment) at 2 hours and at 6/52 (blinded)

Results
Of 33 shoulder RC infiltrated in 17 subjects
Only isolated bursal injections lead to a decrease in pain

Best practice suggests injections should be performed as guided procedures.

Volumes need to be considered / Post injection advice essential.