Treatment
- Worth attempting aspiration with a 16G needle despite high recurrence rate (90%)
- Surgery indicated for symptomatic ganglions.

Osteoarthritis of the thumb

Pathology
- Part of primary generalised osteoarthritis (OA) with Heberden's nodes of DIP joints
- First carpometacarpal (CMC) joint is involved with loss of cartilage
- Results in bone-on-bone deformity
- Very common problem
- Affects 1 in 4 postmenopausal women.

History
- Pain – base of thumb
- Difficulty opening jars/taps/ door handles
- Weakness of pinch grip.

Examination
- Almost always associated with other stigmata of primary generalised OA, including Heberden's nodes of DIP joints
- Subluxation and osteophyte formation cause prominence of thumb base
- Called 'shoulder sign'
- Crank and grind test very painful
- Can get fixed adduction deformity of thumb with compensatory hyperextension at MP joint – so-called Z-thumb.

Special investigations

Radiographs are diagnostic and should include the fully pronated or Roberts' view.

Treatment
- Splints
- Simple analgesics
- Cortisone injection into joint (NB: sterile technique)
- Surgery for failed conservative management
- Excision of trapezium bone with ligament stabilisation is often performed with predictably good results.

Summary
- Five conditions comprise the bulk of primary hand disorders.
- Carpal tunnel syndrome has a very typical presenting picture and special tests are usually not necessary.
- Conservative measures are usually unsuccessful and surgery is eventually required (90%).
- De Quervain's tendonitis is a cause of severe pain on the dorso-radial aspect of the distal forearm.
- Surprisingly there is very little to find except for a positive Finkelstein test.
- Cortisone is highly effective.
- Triggering of the digits responds well to cortisone injections.
- Ganglions are fluid-filled sacs associated with joints or tendon sheath.
- Arthritis at the base of the thumb is the most common site for primary osteoarthritis.
- One in four postmenopausal women will be afflicted, but less than one-third of these will be symptomatic.

Current concepts in rotator cuff management

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This article will look at two aspects of the current management of the rotator cuff tear. The first will be the most recent evidence-based approach and the second will be the experimental biological approach.

The risks, outcomes and the ability to undertake treatment by either the conservative or the surgical route need to be understood by the patient and treating physician.

Conservative treatment comes with the concern that we may miss the boat in terms of tendon healing. Several studies have shown that the longer the tear has been present the more likely it is that there will be irreversible changes.[1] These changes include rotator cuff muscle atrophy and fatty changes (Figs 1 and 2), as well as changes in collagen fibre composition, which do not reverse with successful repair. The tear may progress with time and larger tears have been shown to have higher failure rates with surgery. Therefore it is preferable to intervene prior to these changes occurring.

The concern in the surgical group is the morbidity of the surgery and the long rehabilitation required for tendon healing. This requires 6 weeks in a shoulder immobiliser followed by another 6 weeks to regain range of motion before starting the strengthening programme that allows them to return to full function at 6 months after surgery. For the elderly or those at anaesthetic risk, the questions that need to be asked are whether the surgery is necessary and whether the patients can safely be treated conservatively.

Fig. 1. Sagittal MRI cut of the rotator cuff muscles. The dotted line showing the 'Y of the scapula body and fossae showing normal bulk of the rotator cuff muscles: 1=supraspinatus, 2=subscapularis, 3=infraspinatus. Compare with Fig. 2.

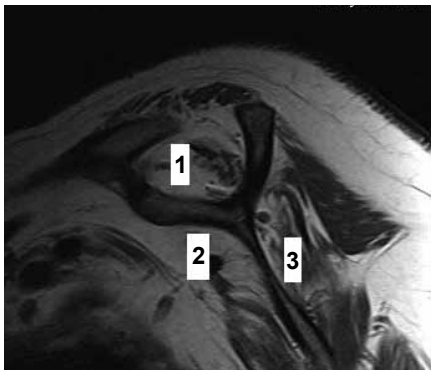

Fig. 2. Sagittal MRI cut at the level of the fossa showing marked atrophy of supraspinatus (1), subscapularis (2) and infraspinatus(3) in a massive long-standing tear. There is 'white' fat replacing the atrophied dark muscle. Compare with Fig. 1.

Evidence-based medicine

The American Academy of Orthopaedic Surgeons' review of the best evidence-based

Table I. Evidence-based recommendations from the American Academy of Orthopaedic Surgeons[2]

- Asymptomatic tears do not require surgery
- There is weak evidence for early repair of traumatic tears
- Surgical outcomes are worse with increasing age, workman's compensation cases and where the MRI findings show significant fatty changes and atrophy
- Routine acromioplasty with repair is not proven
- Weak evidence that debridement for massive tears gives adequate long-term relief
- No difference in outcomes with different surgical techniques (open surgery or arthroscopic)
- Exercise and NSAIDs work in patients with impingement
- Porcine grafts do not work in rotator cuff tears

Table II. No evidence to support or refute these recommendations

- Exercise programmes in the treatment for rotator cuff tear
- NSAIDs and other forms of conservative treatment in rotator cuff tears
- The role of cortisone in patients with a tear or impingement
- Outcomes are worse in smokers and those with diabetes or spinal problems
- Xenograft or allograft material used in repairs

Table III. Poor prognostic factors for conservative treatment

- Tears greater than 1 cm
- Symptoms longer than 1 year
- Severe weakness or functional impairment on initial presentation
- Bilateral rotator cuff tears
- Decreased active ROM preoperatively also has been associated with poor outcome after operative treatment of rotator cuff tear
- Steroid injections preoperatively have a higher failure rate after surgery

medicine available was published earlier this year.[2] Table I lists their recommendations where there is evidence in the literature.[2] Table II lists situations where there is no convincing evidence either way.[2] Table III lists the poor prognostic factors for conservative treatment.[3]

The above paper and Wolf's review paper summarises what most surgeons are practising.[3] These reviews suggest that symptomatic tears can be safely treated conservatively for 6 - 12 weeks, unless there is weakness on examination, bilateral tears, symptoms of more than a year or tear size *less* than 1 cm. There is evidence for early repair if there is trauma. Multiple steroid injections are detrimental.

The biological approach

As genetic influences on the development of tears have been recognised, research is now directed to using this as an intervention. The approach to preventing failed rotator cuff surgery is now being directed to biological strategies at the cellular and molecular level. The reason for this is that despite the mechanical approach of surgery with improved sutures, anchors, surgical technique and grafts there still remains failure of tendon healing of 11 - 94%.[4]

Professor Andrew Carr's group from Oxford has shown that in patients with a painful rotator cuff tear, 62% of their siblings had a tear when compared with 22.1% in the control group. They also showed the rate of progression of the tear was 16.1% in the sibling group, compared with 1.5% in the control group.[5] Does this mean that patients who have a family history should have earlier surgery to prevent progression?

Lawrence started their research by using mesenchymal cells in a rat model, but found no difference in healing rates. These rates improved once these cells were genetically modified (transduced with adenoviral mediated scleraxis) by a transcription factor that is believed to direct tendon development during embryogenesis.[6]

At the molecular level, Millar and his group have shown in a rodent model of tendinopathy that there is upregulation of pro-inflammatory cytokines and apoptotic genes. They also showed significantly increased levels of cytokine and apoptotic genes in human supraspinatus and subscapularis tendon harvested from patients with rotator cuff tears. This allows research to be directed at neutralising the cytokines by using antibodies, etc.[7]

Kovacevic and colleagues carried out a review in which they looked at their own results using bone morphogenic proteins (BMPs) and at other investigators' use of biological interventions, confirming improved healing and strength in the tendons of rats and sheep.[8] These are now being applied in clinical studies with a Level 1 study looking at platelet-rich plasma (PRP), which showed reduced pain in the first postoperative month and improved healing in grade 1 and 2 tears.[9]

As one can see from the above, we are possibly reaching a ceiling with respect to mechanical treatment of rotator cuff tears and that the future belongs to a biological solution to guarantee a successful repair.

References available at www.cmej.org.za

Testing for elbow instability

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The elbow is one of the most stable joints due to the congruity of the articular surfaces and the soft tissues, which consist of the medial and lateral collateral ligaments (MCL and LCL) and muscles crossing the elbow.[1,2]

Unlike in the shoulder, the ligaments tend to heal well following dislocations and therefore recurrent instability is unusual. However, it is postulated that the LCL does not heal quite as well as the MCL following trauma and lateral instability is therefore less rare than the medial side. This results in posterolateral rotatory instability, where the radial head subluxes posteriorly off the capitellum. Iatrogenic injury and subsequent instability may also occur following surgery, for example tennis elbow release.[1,3]

History

Medial collateral ligament insufficiency usually presents with pain. Patients will complain of medial sided elbow pain when throwing or with similar type actions. In acute injuries patients often feel a sharp pain