# Frozen Shoulder

*Webinar – Simon Lambert and Simeon Niel-Asher 20th August 2014*

## Summary Notes

*(see end for list of abbreviations)*

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- Simeon Niel-Asher is a UK osteopath, author of *The Concise Book of Trigger Points (Lotus Publishing UK and North Atlantic Books, Ca)* and developer of the Niel-Asher Technique for treating Frozen Shoulder. The *Evening Standard* newspaper named him as one of the top ten osteopaths in London, and he's involved in treating, research, writing, and teaching throughout Europe, the Middle East and the USA. He can be contacted through his clinic in Highgate, London on +44 (0)208 347 6160.

- Simeon's Slide Notes on Adhesive Capsulitis/Frozen Shoulder

  - Aetiology and pathology of Adhesive capsulitis (AC):
    - Affects 3-5% of 40 – 70 year olds (Grubbs 1993)
    - Affects women more than men (60:40) - ibid
    - The average length of symptoms is 30 months (alternative views that FS lasts about 18 months were based on a single study of 15 patients). Loss of RoM remains in 70% sufferers.
    - It is up to 10 times more common in diabetics (note: IDD renders cells susceptible to pH changes – therefore sets up patient to fail the healing process)
    - Typically it affects the non-dominant shoulder first
    - The pathology is still not clear but there is some evidence connecting the histopathological findings of Dupuytrens contracture (Hand et al 2007).
    - The four main theories are:
      a) Postural
      b) Hormonal
      c) Genetic
      d) Auto-immune (mechanism not understood)
    - FSS has four clinical phases (Reeves 1975):
      I. an early pre-adhesive stage where it may be indistinguishable from other shoulder conditions (such as impingement)
      II. a freezing stage characterized by rapid loss of range GHJ and severe night pain
      III. a frozen phase characterized by reduced range (stuck) but reduced pain
      IV. a (resolving) thawing stage

  - Current treatment pathways:

- Adhesive Capsulitis is commonly clinically encountered by doctors, surgeons and manual therapists. Whilst the natural history is well documented there has been little research published that supports either surgery or physical therapy as an intervention.
- Drug: NSAIDs, oral cortisone, HCZ injections
- Distension/Hydrolisation under ultrasound (Fareed & Gallivan 1989)
- Surgical: Debridement, decompression, removing LHB, MUA {adhesions?}
- Hands-on: to increase RoM, treat secondary or underlying adaptations and or compensations (Wadsworth et a 1986)
- Osteopathic: Treating the whole person? Niel-Asher technique (NAT) is different, based on a trigger point based soft tissue algorithm (attached)
- To date no treatment has reliably demonstrated a reduction in the severity or in the duration of symptoms (DTB)
- It has been suggested that benign neglect is the best pathway (Dierks et al 2004)

## Clinical presentation

- Patient in a great deal of pain
- Global loss of AROM > 50% in all directions – capsular pattern?
- Scared and tired (can't sleep on side), highly motivated to seek relief
- Loss of shoulder function impacts many areas of life – fitness, hygiene..
- Often tried many therapies/interventions
- Clinical tests and investigations often not helpful other than excluding
- What is the body telling us and how and why should we listen?

## NAT Research – Prospective Multicenter Study - IJOM 2014[1]

- 154 consecutive patients with (adhesive capsulitis) pain, stiffness and globally restricted gleno-humeral mobility shoulder for more than three months
- Mean duration of symptoms at presentation was 9 months
- Mean patient age of 54.2 years
- Patients were treated by 4 private therapists in 3 independent clinics (USA, Israel and UK) with collaboration taking place after all data were collected

## IJOM: Outcomes (see attachment)

- Significant change in active range of motion (AROM) Flexion of the gleno-humeral joint
- Significant change in active range of motion (AROM) Abduction of the gleno-humeral joint
- Significant reduction in pain
- Mean improvement in AROM of 12 degrees per session
- Mean number of sessions was seven

## Randomized Placebo Controlled study 2003

- Addenbrookes hospital (UK) 2000-2003: demonstrated similar results for range of motion in a similar timeframe (see attached chart)
- 30 patients treated – 10 per group

[1] A review of this paper has been posted on the Academy website at: http://academyofphysicalmedicine.co.uk/research/adhesive-capsulitis/

- 6 sessions over 9 weeks
- NAT = Significantly improved AROM over and above placebo and PT ($p<0.002$)
- NAT – largest clinical improvement in SPADI
- NAT – significant improvement in strength and power over and above PT and placebo

➢ What is NAT?
- The Niel-Asher Technique (NAT) involves a deliberate, specific algorithm of manipulations to the muscular and ligamentous apparatus of the GHJ based around a five-step treatment protocol
- Simeon estimates that 5-7% patients require a surgical opinion, usually the complex pathologies – secondary FS (eg diabetes)
- Treatment sessions last between 25-40 (average of 30) minutes in duration; the technique is performed on all patients in sequential order on each visit
- NAT is an advanced trigger point technique utilizing the trigger points as neural inputs to change cortical output. Practitioner is guided by both palpation and patient feedback of referral patterns.
- Within the five steps of the NAT protocol there are two main types of techniques employed which have been slightly modified; deep stroking massage (step one) and compression of trigger points (steps two, four and five)
- These techniques are described in the work of Travell and Simons (1999)
- Technique can be taught remotely
- Much quicker resolution in more chronic cases
- Can be practised in any position, but difficult. Easiest supine and side-lying.
- Can be used on any age patient: no thrusts involved
- NAT deliberately uses:
  - Post isometric relaxation (PIR)
  - Reciprocal inhibition (RI)
  - Triangulation
  - Pain gate

• Diagnosis of FS:

➢ Many diagnoses of FS are incorrect: "frozen shoulder lasting 3-4 months" is ***not*** FS. GPs may use RCD or FS as convenient terms for shoulder problems in general.
➢ Often there has been a prior injury resulting in tendon/bursa/biceps/AC problem, leading to *local* contracture.
➢ Possible to predict patients likely to have FS: PH, neck problems CSP (spondylosis almost universal at age 70, so correlation with FS not conclusive). Pt with lower cervical, sub-axial, monolateral, radiculopathy has far higher risk of developing FS.
➢ Simeon noted that there is a palpable band of tissue which is felt on the lateral aspect of the humerus:
- In the midline in FS
- Posteriorly in RCD
- Anteriorly in impingement

➢ The Codman Criteria are generally accepted (in the orthopaedic world) as definitive:
- Global restriction of shoulder movement.
- Idiopathic etiology.
- Usually painful at the outset.

        - Normal x-ray.
        - Limitation of external rotation and elevation.
    - Important to distinguish between:
        - Shoulder that is injured
        - Shoulder that's inflamed
        - FS (onset of symptoms is sudden)

- Types of FS (according to Simeon):
    - Anterior: most common. On palpation, most painful structures are anterior, eg biceps tendon
    - Lateral: includes an element of sub-acromial/RC involvement
    - Posterior: involves triceps tendinopathy. Rare, usually associated with overhead activity such as gym training

- Histopathology:
    - Main cell involved is the fibroblast, originally thought to become myofibroblastic – contractile
    - RNOH study showed this not to be the case in FS:
        - fibroblasts do not respond in te same way as those in Dupuytren's contracture, or those in mature scar tissue
        - not an inflammatory process (but Simeon reports having detected active, inflammatory phases in the capsule)
        - process is an inexorable contracture until the shoulder is stuck globally
    - Via arthroscope, FS tissue can be 1cm thick, where it should be 1.5mm. Can take 45 minutes to cut.
    - No evidence of inflammatory markers in FS, but hyperaemia does exist, probably in response to a neural insult

- Surgical intervention:
    - Can be very effective, especially in treating pain
    - It is possible that reported surgical success in treating FS with, eg, MUA, may be due to incorrect diagnosis.
    - If surgery done during the process of contracture, the contracture invariable returns, usually without pain (suggests that surgery is denervating the tissues)
    - Surgery appropriate in mature phase of FS (about 6 months)
    - If shoulder capable of $>0^{o}$ ER, then physical therapy more appropriate
    - MUA does not work (20% complications, including fractured neck of humerus, fractured glenoid fossa, torn biceps – 10 times what is acceptable in normal surgery). But it's cheap and easy!
    - Hydrolisation/hydrodilatation unlikely to be successful in *true* FS

- Lack of ER is caused by:
    - Fractured shoulder
    - Arthritic shoulder
    - FS
    - Posteriorly unstable displaced shoulder
    - Xray relevance:
        - determine whether or not arthritic, fractured or dislocated

- if normal in 50+yo, probably FS

- LHB:
  - Simeon finds LHB involved in many shoulder pathologies
  - Only provides 15% power in elbow flexion
  - Sometimes removed surgically (as though vestigial).
  - Simon observed that if it served no purpose it would have disappeared. May be useful in as a stabiliser. Acts as a bridge between supination of forearm, ER and elevation of shoulder – useful for gesture.
  - Does get inflamed, but is packed with Substance P receptors: useful in stability
  - Because intracapsular, any inflammatory exudate can contribute to FS

- Use of NAT in other shoulder pathologies:
  - 95% shoulder problems respond to physical therapy
  - NAT good for arthritis, impingement – particularly where body's response is to internally rotate shoulder
  - Less useful with calcific tendonitis, but very effective once symptoms have calmed down

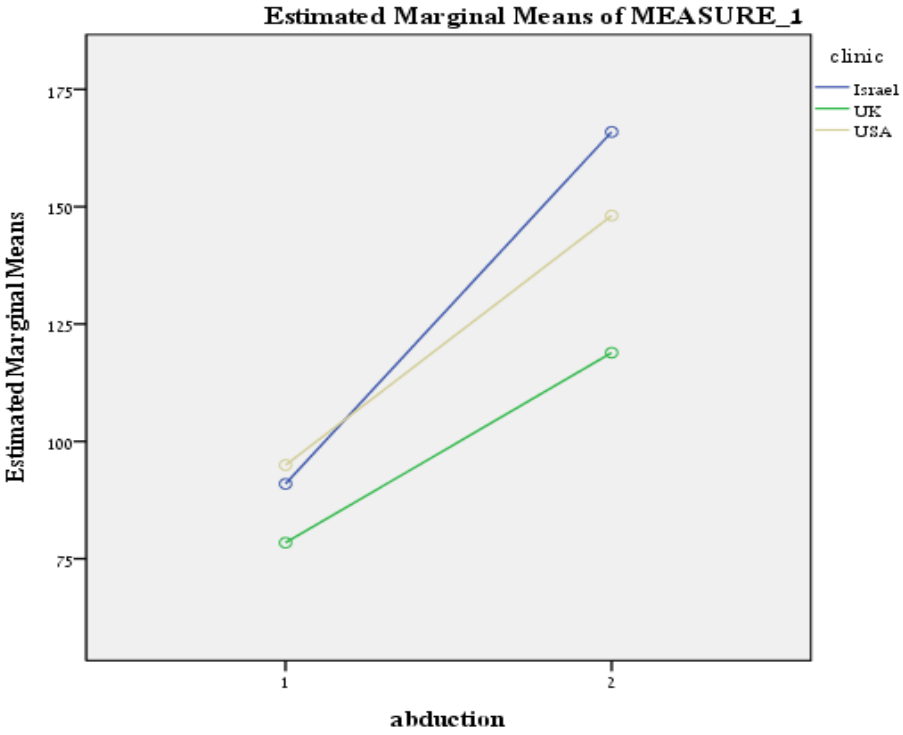
- Is joint manipulation useful in FS in conjunction with FS?
  - Not for FS itself
  - Very relevant after FS resolves in dealing with thoracic and other mechanical compensations

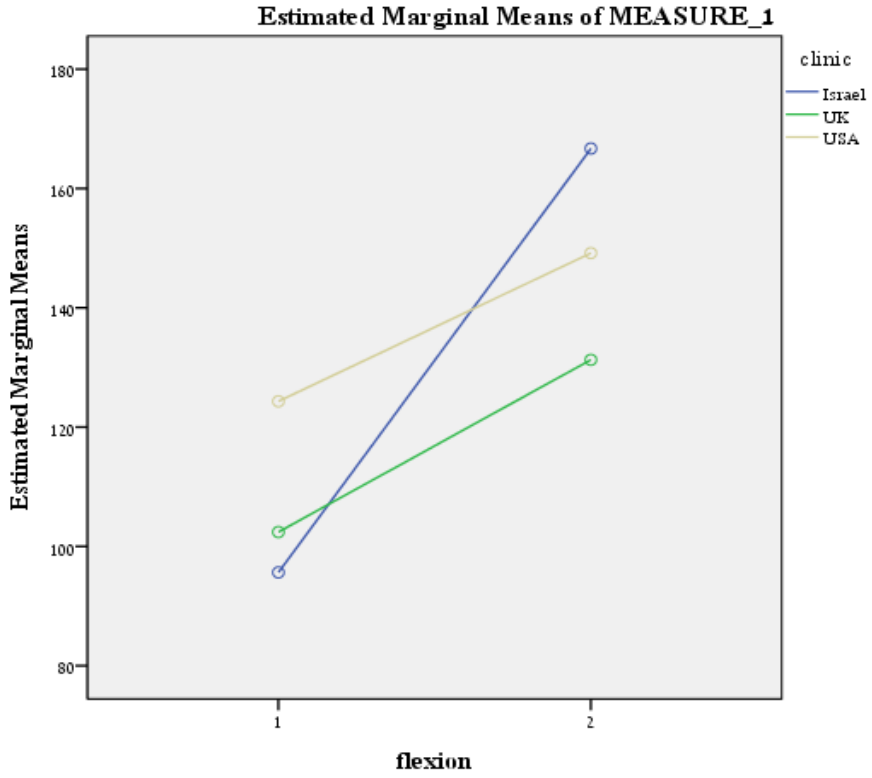
- The future in orthopaedics?
  - Molecular biology and genetic therapy opening up great potential in manipulating diseases such as OA
  - Infection control globally is biggest challenge for orthopaedics
  - Osteoporosis increasing with ageing population (0.5m in UK over age of 90 with osteoporosis and/or OA)
  - So most advances in orthopaedics are not orthopaedic – it's about addressing disease

List of Abbreviations:

| | |
|---|---|
| AC | Acromioclavicular |
| ARoM | Active Range of Motion |
| ER | External Rotation |
| FSS | Frozen Shoulder Syndrome |
| GHJ | Glenohumeral Joint |
| HCZ | Hydrocortisone |
| IDD | Insulin Dependent Diabetes |
| LHB | Long Head of Biceps |
| MUA | Manipulation Under Anaesthetic |
| NAT | Niel Asher Technique |
| NSAID | Non-Steroidal Anti-Inflammatory (drug) |
| OA | Osteoarthritis |
| PH | Previous History |
| PIR | Post Isometric Relaxation |
| PT | Physiotherapy |
| RC | Rotator Cuff |
| RCD | Rotator Cuff Dysfunction |
| RI | Reciprocal Inhibition |
| ROM | Range of Motion |
| SPADI | Shoulder Pain and Disability Index |
| UCL | University College London |
| yo | year(s) old |

## NAT Research – Prospective Multicenter Study - IJOM 2014
## Changes in Active flexion and Abduction over time



[Addenbrookes Hospital: Randomized placebo controlled study 2003]