Here is where trigger points come into play





What are Trigger Points & How are They Involved?

- Ubiquitous
- Discrete exquisitely tender nodules in taut bands
- Woven into the warp and weft of the myofascial continuum
- Appear spontaneously after trauma
- Part of the bodies protect and defend mechanism?
- Alarm bells?
- Part of the bodies wisdom?
- Pain Pleasure





Trigger Points 101

1942 by Dr. Janet Travell:

Painful lumps or nodules within tight bands of muscle.

Trigger points all seem to have the following characteristics:

- Pain
- Referred Pain Map remote from origin
- Nodule in Taut band in muscle
- ANS changes
- Cannot be explained by neurological examination – what why are they????





Distinctive Pain Maps – E.g. - Infraspinatus





Trigger Point Essentials

Reduced mechanical efficiency!

- Develop in the *muscle belly*
 - Multi-pennate (several heads) e.g.. deltoid or serratus anterior may have several trigger points.
- Overstimulation of the muscle spindle, becomes sticky and permanently 'switched-on'
- Muscle shorter, fatter and less efficiency:
- Neurovascular Myopathic compression.
- Effect on antagonists?
- Increase the burden of nociceptive input
- Reduced efficiency = increased risk of injury



Figure 3.6: A trigger point showing 100 shortened sarcomeres without nerve stimulus and associated taut band.



Aetiology

Several possible trigger point mechanisms (Dommerholt et al. 2006):

- Low-level muscle contractions
- Uneven intramuscular pressure distribution
- Direct trauma
- Unaccustomed eccentric contractions
- Eccentric contractions in unconditioned muscle
- Maximal or submaximal concentric contractions





What's happening inside the MM: Sarcomere Contraction





Summary of A.N.S. Effects

- Hypersalivation increased saliva
- Epiphora abnormal overflow of tears down the cheek
- Conjunctivitis reddening of the eyes
- Ptosis drooping of the eyelids
- Blurring of vision
- Increased nasal secretion
- Goose bumps/flesh
- GIT Bowel? Unexplored!





G.I.T. & LHB Tendon Trigger points

- Parasympathetic HF/LF waves
- LHB NB Subscapularis fibers
- Usually as phase I goes to phase II
- Feel a heat/temp/energy changeshift
- ALWAYS heralds a major step change in sleep
- Independently reported
- Why? Holding Pattern? Change in Parasympathetic NS? Vagus Nerve? TP related?
- Part of my PhD study





PhD Study

Osteopathic Manipulative Therapy of the Long Head Biceps in Idiopathic Adhesive Capsulitis using Myofascial Trigger Points: effects on Parasympothetic activity to the Gastro Intestinal Tract

Simeon Niel-Asher SPhil BSc (Ost) - PhD Proposal

Protocol (version 4) 04/04/2016

Introduction

Achesive copsulitis (AC) is a common complaint affacting over 2% of adults and up to 11% of bioberics (Tasto, J. 2006)¹. It is encountered by many cinicians (GP's, Rhoumotologists, Orthoppedia Surgeons) and Physical Therapists (Physiotherapists), Osteopaths and Chroproctors, Defining AC is complex and it has aften incorrectly been used as a general diagnosis for shoulder pain and stiffness. The cellology, pathophysiology and treatment of this condition are also subjects of depate. AC offects fermiles slightly more than males, typically between 40 and 60 years (Crubbs 1993), usually the non-dominant arm (Foread, & Collyon 1989) 4, with 12% affacted bilaterally (Widsworth 1986) 5. Other ogents such as depression, immunologic factors, positive and has, AC has been described as an enigma wrapped in a mysley (Lews 2014). Why more women than men? Why women in their 50x9 Why does it present or the non-dominant side?

Pain and Shiffness

Handhord² has suggested that AC potients fit into two groups, "poin predominant" and "stiffness predominant". One of the many questions here is what is the source of the pain and where is the stiffness coming from? Is it on antalgic posture or are other biomechanical or biochemical mechanisms of play? Certainly we know that immunocytachemical and histological analysis (of biopsies) of the copsule show a prependerance of fibrablesis, chronic inflammatory cells particularly most cells, if cells. 3 cells and macrophages (Hand et al 2008) 3. But the bioeps is also implicated: There is also a possible link to Duppytrens contracture of the hand (Bunker 1995)?.

ANS & Homeostasis





Manual TP Techniques VS Dry Needling

- NAT = ICT & DSM
- Mechano-Receptor stimulation golgi tendon, Spindles, Paccini corpuscles, Extra-spindle sarcolemma and myofascia
- Nociceptive input
- Requires a moderate skill level of palpation – wait/feel for change
- Different TP effect to DN/IMS
- Tried NAT sequence with DN/IMS = doesn't work
- There IS a place for IMS but from session seven onwards non acute





Fingers Thumbs or Elbows? Chose your weapons carefully





Figure 4.1: (a) Flat-finger palpation, (b) pincer palpation.



Lets get into it?



Niel Asher Technique



What is NAT?



- Developed in 1999 to treat FSS
- Advanced trigger point technique using ICT and DSM in specific repeatable algorithms
- Deliberately utilizes automatic spinal reflexes
- Trigger points = Nociceptive inputs which have a neuroplastic effect
- Nociception is the language of the nervous system - The alarm bell
- Deliberate neuromuscular sequence
- Spinal cord reflexes such as RI
- Utilizes neuroplasticity
- Evidence Based



Prospective Observational Multi-Center Study on NAT

IJOM – June 2014



Study Characteristics

- 154 patients with globally restricted AROM & PROM
- UK, USA & Israel independent and post tt analyses intention to treat model
- Outcome measures = AROM using goniometer & Pain using VAS
- Average age 54.2, 60:40 females, 8.2 months duration, exclusion criteria

Conclusions: "All patients demonstrated a significant improvement in AROM for both flexion and abduction. The data supports the notion that NAT is autonomously reproducible. Average number of treatments was seven, average of 11 weeks with an average 12 degree improvement in AROM per treatment visit. NAT expedites both pain reduction and increased mobility for adhesive capsulitis over and above the natural history".





"There was a large and statistically significant reduction in the VAS pain score between baseline and post-treatment assessments. There was a significant improvement in AROM abduction and flexion across time irrespective of the phase of illness (acute / stiff / resolving)".



NAT Evidence Base

Journal Rheumatology 2003 - RCT



Study design

- Hospital Study
- Placebo controlled design
- 3 groups = Ost, PT & Placebo?
- 30 patients = 10 per group
- 6 sessions over 9 weeks
- Mean duration 8.4 months
- Osteopathy group = more acute
- Data analyzed by research PhD physio
- Follow up at 6 months
- Outcomes AROM, SPADI, S&P
- "A larger study is warranted (N=99)"



RCT Results - Active ROM



Improved Range Of Motion

Cambridge University Hospitals

Significantly Increased Active Range of Motion

Range of motion improved by **52.6**° degrees for the Niel-Asher Technique[™] compared to **24**° degrees for standard physiotherapy and 0.8° degrees for placebo. This was a statistically significant result (p<0.002).



RCT Results - SPADI



Cambridge University Hospitals

80% Reduction in Pain Approached Significance

Pain improved by 38.7 points for the Niel-Asher Technique[™] compared to 19.9 points for physiotherapy and 22.8 points for placebo. In fact those patients treated with the Niel-Asher Technique[™] reported an 80% decrease in pain over 9 weeks! This result approached statistical significance (p=0.07).



RCT Results - Strength & Power



Significantly Increased Strength and Power

- Treatment with the Niel-Asher Technique[™] demonstrated a significant increase in strength and power (Newton metres per second as measured by a cybex dynamometer), even though no exercises were given.
- In this study the physiotherapy group did not improve at all and the placebo group decreased in strength and power! (p=0.043). Why? Niel-Asher Technique[™] works directly on the brain to improve shoulder muscle co-ordination via nociceptive inputs.







Important structures

Please Note

- Lateral myofascia of UEX & Deltoid
- Teres Minor
- Teres Major
- Long head Biceps
- Subscapularis
- Infraspinatus



NAT Key Muscles - Biceps Brachii - LHB



ORIGIN

- Short head: tip of corocoid process of scapula.
- Long head: supraglenoid tubercle of scapula.

INSERTION

- Posterior part of radial tuberosity.
- Bicipital aponeurosis, which leads into deep fascia on medial aspect of forearm.

ACTION

• Flexes elbow joint. Supinates forearm. (It has been described as the muscle that puts in the corkscrew and pulls out the cork.) Weakly flexes arm at shoulder joint.

ANTAGONIST: Triceps brachii.

NERVE

• Musculocutaneous nerve, C5, 6.

HOW MANY HEADS?????



NAT Key Muscles - LHB



© 2005 Primal Pictures





Long Head of Biceps Tendon

- Originates from supraglenoid tubercle of scapula and the superior glenoid labrum,
- · Intraarticular portion that passes over the humeral head
- Exits the glenohumeral joint through the bicipital groove where it becomes extraarticular.
- Approximately 9 cm long and 5–6 mm in diameter
- Slides passively on the humeral head during abduction and rotation.
- Slides up to 18 mm in and out of the glenohumeral joint during forward flexion and internal rotation.
- Four types: entirely posterior, posterior dominant, equal, and entirely anterior.
- Sliding portion is relatively Avascular the rest is supplied by the brachial artery.
- Musculocutaneous (Motor) Nerve + a rich network of sympathetic fibres

	Vangsness (%)	Tuoheti (%)	
Entirely posterior	22	28	
Posterior dominant	33	55	
Equal	37	17	
Entirely anterior	8	0	





Transverse Humeral ligament



- Distinct structure 14mm long
 14mm wide
- Trapezoidal between lesser and greater tuberosities
- Continuous with the RCT
- Two tissue Layers
 - Superficial Fibrous fascial covering
 - Deep from Subscapularis, Supraspinatus and the CHL – blends with cuff
- Free nerve endings (pain)
- No mechanoreceptors



How many? Supernumerary heads and the Vinculum

Supernumerary Heads

Vincula (approx 23%)



Figure 1: Third head of biceps brachii 1: Biceps common tendon, 2: Brachialis, 3: Brachioradialis, 4: Deltoid, 5: Forearm

Figure 2 Photograph of gross anatomic dissection of vinculum. Natice single strand to eff, abliquely crossing joint and exiting bioioital groove.

Bilateral asymmetry, multivariant morphology in 23% population up to 5 heads – supernumerary heads – both LHB and SHB. Petchprapa et al (2010)



LHB & FSS

- Surgeons/arthroscopy reports – mushroom clouds/ blushes of tenosynovitis on the LHB sheath
- Chronic granuloma? With villous or 'fronded' appearance but no IG markers (Bunker et al 1997)
- Reactive tendinopathy subscap bursa at origin of LHB?
- Responds beautifully to NAT





The Rotator Interval

Rotator Interval



Fig. 11.1 Right shoulder from anterolateral with inkmarks outlining the borders of the supraspinatus (SS) and subscapularis (Sub). The lateral and anterior portions of the deltoid have been detached exposing the anterior part of the humerus. The rotator interval extends from the coracoid (c) to lay over the long head of the biceps tendon (BT), which has been exposed in its groove

Triangular Space

- Medially is the base of the Coraccoid P
- Superior is the base of the Supraspinatus tendon
- Inferior is the border of the Subscapularis tendon
- Laterally is the transverse humeral ligament (Brodie)
- Anatomical variations
- Controversy as to anatomy and function



Don't Forget the Coracobrachialis





Key Muscles - Subscapularis



ORIGIN

• Subscapular fossa and groove along lateral border of anterior surface of scapula.

INSERTION

- Lesser tubercle of humerus. Capsule of shoulder joint
- Biceps transverse ligament

ACTION

 As a rotator cuff muscle, stabilizes glenohumeral joint, mainly preventing head of humerus being pulled upward by deltoid, biceps, and long head of triceps. Medially rotates humerus.

ANTAGONIST

Subscapularis, Teres Minor

NERVE

• Upper and lower subscapular nerves, C5, 6, 7, from posterior cord of brachial plexus.



Subscapularis - 3 functional parts 3 Innervations





Practical - Part Two





The case history

Onset and history

Which phase?

Primary or secondary?

May follow trauma such as surgery, fracture/dislocation, post-mastectomy or FOOSH.

May follow emotional trauma such as divorce or bereavement

- Any previous treatments and did they help?
- Any investigations? X-Rays, MRI and Ultrasound can rule out co-existing pathology





The case history

aggravating:

• putting on/taking off shirt/coat/jumper

 \cdot reaching for the back pocket

 \cdot combing the hair

 \cdot reaching for the back seat of the car

 \cdot changing gears

 \cdot reaching to unlock the back seat of the car

 \cdot picking up a case from the back seat

 \cdot reaching behind the back to do up the bra

• washing under the opposite armpit

 \cdot wiping after defecating





Practise recording the range of movement at the shoulder on three other people. Take each reading twice. Compare your results with those of your group. Where inconsistencies arise, try to determine where the inaccuracies are occurring.

Move- ment	No.1	No. 1	No. 2	'No. 2	No. 3	No. 3
Flexion		1			i i	
Extension						
Abduction				0		
Scaption						
External Rotation						



Diagnosis

Side?:

Phase?:

- pre phase l
- phase I
- phase II

Pattern?:

- ant
- post
- lat (or combination)

other comments?:

- flexion
- extension
- abduction





Areas of dysfunction:

(Check biceps groove for tenderness, check triceps, feel for insertion of supraspinatus (cuff), feel for insertion of deltoid, feel biceps short head, feel relationship with pec major & min. check teres group) BE GENTLE.

ANTERIOR

POSTERIOR

LATERAL





The basic moves

- Generalized Beginning
- STEP 1
- STEP 2
- STEP 3
- STEP 4
- STEP 5
- Generalized Ending





NAT involves a combination of 5 simple components. This is what I call the 1-2-3-4-5. Other steps are added, but these form the basis of the technique. These procedures **MUST** be done in the right order. Other STEPS will later be added to the 1-2-3-4-5. These other STEPS are determined by the case history; we will be exploring these on day 3.

The other steps are added to the 1-2-3-4, the order of the other steps is determined by your case history; so make sure you have filled it in before you start treatment.



Pre step one – With the patient sitting apply gentle soft tissue massage to neck and shoulders introducing the concept of tender/trigger point compression and "pain maps" to the patient via pressure on the upper trapezius tender/trigger point.

The patient is encouraged to relax into the pain and not to fight it.











Step 2









Step 4





Central & Lateral bowstringing





Medial bowstringing and SHB/Pec Min modification













Once finished, ask the patient to stand and perform shoulder rotations anteriorly and posteriorly. Then gently test passive range of motion (ROM) – (Flexion in Scaption) supporting the affected arm and note the ROM. Ask the patient to get dressed and then re-check and note passive and active ROM and any subjective changes in, strength and power.

As treatment progresses (session seven onwards) other modalities may be employed such as soft tissue techniques, electrotherapy, needling and high velocity, low amplitude manipulation to the cervical and thoracic spine and other areas.





5 Steps to freedom algorithm

20imson Net-Asher 2007 Not be photocopying, foxing, securing Transmission or reproduction





STEP 1

STEP 5



Step 1: 3 strokes upwards only

Step 2: Pause on the Teres minor trigger point

Step 3: Gentle passive Circumduction

Step 4: Long head Biceps inhibition thro' the chronic inflammation

Step 5: Infraspinatus medial border trigger point

