

## Masterclass

## Frozen shoulder contracture syndrome – Aetiology, diagnosis and management



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## ABSTRACT

Frozen shoulder is a poorly understood condition that typically involves substantial pain, movement restriction, and considerable morbidity. Although function improves overtime, full and pain free range, may not be restored in everyone. Frozen shoulder is also known as adhesive capsulitis, however the evidence for capsular adhesions is refuted and arguably, this term should be abandoned. The aim of this Masterclass is to synthesise evidence to provide a framework for assessment and management for Frozen Shoulder. Although used in the treatment of this condition, manipulation under anaesthetic has been associated with joint damage and may be no more effective than physiotherapy. Capsular release is another surgical procedure that is supported by expert opinion and published case series, but currently high quality research is not available. Recommendations that supervised neglect is preferable to physiotherapy have been based on a quasi-experimental study associated with a high risk of bias. Physiotherapists in the United Kingdom have developed dedicated care pathways that provide; assessment, referral for imaging, education, health screening, ultrasound guided corticosteroid and hydro-distension injections, embedded within physiotherapy rehabilitation. The entire pathway is provided by physiotherapists and evidence exists to support each stage of the pathway. Substantial on-going research is required to better understand; epidemiology, patho-aetiology, assessment, best management, health economics, patient satisfaction and if possible prevention.

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## 1. History and nomenclature

Duplay (1896) described the disabling combination of shoulder pain and restricted movement as péri-arthritis scapulo-humérale, attributing the condition to inflammation of the subacromial bursa. The term peri-arthritis of the shoulder has been used by others, both as a diagnosis and to explain the pathology (Dickson and Crosby, 1932; Wright and Haq, 1976). With the advent of radiographs, calcific deposits were observed, and for a period of time, the pain and stiffness was attributed by some to this newly observed phenomenon (Baer, 1907).

Codman, initially considered the condition to be an 'adherent subacromial bursitis', but after 15 years of clinical observation he rejected this in favour of the term frozen shoulder (Codman, 1934). He believed the condition involved a non-calcifying tendinitis of the rotator cuff, arguing that calcification represented a different

pathology. During a one year period (approximately 1933), Codman treated four people suffering from frozen shoulder, and described the symptoms to consistently involve; slow onset (typically insidious, although trauma or strain may predispose), pain near the insertion of deltoid, inability to sleep on the affected side, painful and incomplete shoulder elevation and external rotation, and, with the exception of possible bone atrophy, normal shoulder radiographs. He added that although the aetiology remained uncertain, and the condition difficult to treat, the disorder would almost certainly resolve. To treat frozen shoulder, Codman advocated hospitalisation, with the arm constrained in elevation for one to two weeks. Patients were permitted to get up, out of bed, once a day to perform pendular exercises.

Lippmann (1943) supported many of Codman's observations, but argued that peri-arthritis or frozen shoulder resulted from inflammation of the long head of biceps tendon that eventuated in firm adhesions of the tendon to the bicipital sheath and bicipital groove. On the basis of intra-operative findings in 12 people, Lippmann argued the condition should be called bicipital tenosynovitis and clinically should be regarded as being similar to de Quervain's disease.

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Soon after this and based on a case series of 10 patients and observations of inflammation, fibrosis and contraction of the shoulder capsule, and with the axillary fold becoming 'adherent' to the humeral head, [Neviasser \(1945\)](#) suggested the term adhesive capsulitis better described the pathology. The adhesion was described as being similar to that of an adhesive plaster applied to the skin. Rotation and manipulation of the humerus was advocated to separate the adherent capsule from the humeral head. Later evidence suggested that thickening and contracture of the glenohumeral joint capsule was associated with frozen shoulder, *without* adhesions to the humerus ([Wiley, 1991](#)). Capsular adhesions have also not been reported in other investigations ([Uitvlugt et al., 1993](#); [Bunker and Anthony, 1995](#)). The term adhesive capsulitis appears not to appropriately describe the condition and arguably should be abandoned.

[Lundberg \(1969\)](#) introduced the terms primary and secondary frozen shoulder, with primary being associated with idiopathic onset and secondary occurring following trauma, and forced inactivity following trauma. Others have further classified secondary frozen shoulder into; intrinsic, extrinsic and systemic categories ([Zuckerman and Rokito, 2011](#)). Conditions such as; calcific tendinitis, rotator cuff and biceps tendinopathy precede intrinsic secondary frozen shoulder. Shoulder surgery may result in iatrogenic intrinsic secondary frozen shoulder. Secondary extrinsic frozen shoulder is diagnosed when the condition is preceded by pathology remote from the shoulder, such as; humeral or clavicular fractures, cervical radiculopathy, ipsilateral breast surgery, chest wall tumour or cerebrovascular accident. Systemic secondary frozen shoulder occurs in the presence of conditions such as; diabetes, thyroid abnormalities, and heart disease. It is important to emphasise that currently a definitive relationship between many of these conditions, and frozen shoulder remains uncertain.

In Japan and China frozen shoulder is known as *the fifty year old shoulder*. This reflects the mean age of onset of the condition ([Lundberg, 1969](#)). The condition has been termed; *frozen shoulder syndrome* ([Lundberg, 1969](#); [Yang et al., 2007](#)), and [Bunker \(2009\)](#) recommended the term *contracture of the shoulder* arguing this best incorporates the clinical and histological presentation. The multiple nomenclature used to describe this condition reflects poor understanding of the pathoetiology, with the term frozen shoulder being described as a "waste-can diagnosis" ([Neviasser and Neviasser, 1987](#)) as it is often applied to any stiff and painful shoulder. In addition, the appellation frozen shoulder suggests the shoulder will eventually thaw, without the need for treatment. Not only may this belief lead to complacency, it may also be incorrect, as ongoing symptoms, 11 years post onset, have been reported ([Shaffer et al., 1992](#)). The recommendation for using the term contracture of the shoulder ([Bunker, 2009](#)) may be more suitable, but this may not reflect the often severe pain experienced with this condition, and as such, frozen shoulder contracture syndrome (FSCS), may more appropriately describe the condition.

Earlier clinical, operative and histological findings, often based on observational inferences from small studies with uncertain inclusion and exclusion criteria, have frequently been reported in later publications without critique of the quality of the earlier evidence, and as such, many 'truisms' relating to FSCS have become integrated into current clinical practice and this 'evidence' is often used to inform management and patient education. In fact, there is no certainty that women are affected more than men, and the true incidence and prevalence of FSCS remains unknown, as is certainty that the non-dominant side is more frequently involved than the dominant side. There is no certainty that onset relates to menopause in woman, or personality type (peri-arthritis personality). The number of people being affected bilaterally is often cited as 1 in

6 (17%), but this, alongside the belief that relapse in the same shoulder does not occur, also remain definitively unsubstantiated.

## 2. Pathoetiology

The normal intra-articular volume of the glenohumeral joint has been reported to be between 15 and 35 cc and in FSCS the volume may reduce to 5–6 cc ([Lundberg, 1969](#)). [Neviasser \(1945\)](#) described an inflammatory (hence capsulitis) process, [Lundberg \(1969\)](#) did not report significant numbers of inflammatory cells, a finding supported by others ([Bunker, 1997, 2009](#)). However, others have suggested that the pathology associated with FSCS involves a chronic inflammatory response with fibroblastic proliferation ([Hand et al., 2007](#)).

[Lundberg \(1969\)](#) described the capsular changes to resemble Dupuytren's contracture. In addition, he reported that osteopenia was commonly observed in the humeral head of the affected side. Histological investigations of the coracohumeral ligament revealed nodules and laminae of dense tissue reported to be mature type III collagen, with a proliferation of fibroblasts and myofibroblasts (cells associated with contractile scar tissue). These histological and immunochemical changes have also been reported in Dupuytren's contracture. There has been a recent suggestion of an association between Propionibacterium acnes and frozen shoulder ([Boyd et al., 2014](#)). If proven, this may lead to a change in the understanding and management of this condition.

A summary of reported abnormalities include; thickening and fibrosis of the rotator interval, obliteration and scarring of the subscapular recess (area between biceps and subscapularis), neovascularity, increased cytokine concentrations, contraction of the anterior and inferior capsule (axillary recess), reduced joint volume, contraction and fibrosis of the coracohumeral ligament, proliferation of fibroblasts and myofibroblasts, presence of contractile proteins, and uncertainty regarding inflammatory changes. Adhesions of the capsule to the humeral head do not occur. The contracted tissue resembles Dupuytren's contracture. Neovascularity is present in the earlier stages of the disease and is found in the rotator interval, superior capsule, posterior capsule and the infra-glenoid recess ([De Palma, 1952](#); [Lundberg, 1969](#); [Ozaki et al., 1989](#); [Neer et al., 1992](#); [Bunker and Anthony, 1995](#); [Bunker, 1997](#); [Handa et al., 2003](#); [Ryu et al., 2006](#); [Uthoff and Boileau, 2007](#); [Bunker, 2009](#)).

Dupuytren's contracture is classified under a group of tissue pathologies known as fibromatoses. There appears to be a high incidence of Dupuytren's contracture in people with FSCS ([Smith et al., 2001](#); [Degreef et al., 2008](#)). Both FSCS and Dupuytren's contracture may occur without an identifiable precipitating event, and both are more common in people with diabetes ([Smith et al., 2012](#)).

Raised serum lipid levels have been reported both in FSCS ([Salek et al., 2010](#)) and in people with Dupuytren's contracture ([Sanderson et al., 1992](#)). However, this association, as well as the relationship between FSCS and thyroid disease and heart disease, is less certain ([Smith et al., 2012](#)).

Significantly greater plasma levels of substance P were reported in people who developed FSCS following shoulder surgery than those that didn't ([Franceschi et al., 2008](#)). In addition, substance P has been reported to accelerate angiogenesis and hypercellularity in tendon ([Andersson et al., 2011](#); [Backman et al., 2011](#)). Substance P in combination with interleukin 1 $\alpha$  have also been shown to promote angiogenesis ([Fan et al., 1993](#)) and high concentrations of interleukins have been identified in the capsule and subacromial bursa of people with FSCS ([Lho et al., 2013](#)). It is possible that these substances are involved in the pathogenesis of FSCS, and directing treatment at the neovascularity may contribute

to reducing symptoms associated with the condition. The expression of angiogenesis has been reported to be reduced by heat (Eikesdal et al., 2002). This may be one reason why shortwave diathermy (SWD) and stretching were found to have beneficial effect in people with FSCS (Leung and Cheing, 2008). Research investigating the effect of heating on the expression of neovascularity and concomitant influence of symptoms would add to the evidence required to more effectively treat FSCS.

### 3. Epidemiology and natural history

Although published data suggest FSCS occurs in 2%–5% of the population (Neviasser and Hannafin, 2010), the actual lifetime prevalence and annual incidence of FSCS remains uncertain. Different diagnostic criteria used in epidemiological analyses is one reason for this. Risk factors for FSCS appear to include; diabetes, family history and possibly hypothyroidism (Smith et al., 2012; Wang et al., 2013), genetic predisposition (Hirschhorn and Schmidt, 2000; Hakim et al., 2003; Smith et al., 2012) and ethnicity, as one study reported that being born or having parents and grandparents born in the British Isles increases the risk (Wang et al., 2013). FSCS is typically described as passing through three stages (i) frozen or pain, (ii) freezing or stiffness and (iii) thawing or recovery phase (Reeves, 1975). Others have described a four stage process (Neviasser and Neviasser, 1987). The average duration of FSCS being 30.1 months (range 12–42 months). A longer frozen phase may be associated with a longer thawing phase (Reeves, 1975). Although, Codman (1934) stated that recovery will occur and should be expected, this may not be correct, with 50% of people diagnosed with FSCS experiencing pain and/or stiffness at an average seven years post onset (Shaffer et al., 1992). Another study reported 41% of people suffering FSCS had ongoing symptoms with functional loss at an average 4.4 years (range, 2–20 years). Of these, 94% reported mild and 6% reported severe symptoms (Hand et al., 2008). These differences may represent; different diagnostic criteria, different assessment and outcome measures, varying severity of the condition, or development of concomitant pathologies.

### 4. Diagnosis

There is no definitive gold standard test to diagnose FSCS and diagnosis is based upon; (i) clinical examination, (ii) exclusion of other pathologies and (iii) normal glenohumeral radiographs. What constitutes a positive clinical examination is equivocal. Cyriax and Cyriax (1993) recommended a pattern of progressively restricted joint movement attributed to capsular restriction to diagnose FSCS. A consensus recommendation involved restriction of active and

passive shoulder movements with normal shoulder radiographs (with the exception of osteopenia of the humeral head and calcific tendinosis (Zuckerman and Rokito, 2011). The simplest clinical diagnostic criterion involves; an equal restriction of active and passive glenohumeral external rotation and an essentially normal shoulder radiograph (Bunker, 2009). Research is required to validate this approach. Radiographs are required to exclude other conditions (locked dislocations, arthritis, fractures, avascular necrosis, osteosarcomas) that may painfully restrict movement and masquerade as FSCS. Arriving at a diagnosis of FSCS may take time, due to multiple conditions that may present with increasing pain.

The rotator interval is a region readily observed as part of the sequence of ultrasound examination of the shoulder. It is bounded medially by the subscapularis tendon and laterally by the supraspinatus tendon and contains a cross sectional view of the biceps tendon between these tendons. The coracohumeral ligament is located in the lateral aspect of the rotator interval, above the capsule. There may be a role for diagnostic ultrasound in the diagnosis and staging of FSCS. In an investigation of 30 people diagnosed with the FSCS, neovascularity was identified in the rotator interval in 26 patients (87%). In these 26, symptoms had been present for less than one year and in the other four, who had experienced symptoms for a longer period, no neovascularity was observed. Ten people without symptoms served as a control and no neovascularity was observed in this group (Lee et al., 2005). The sonographer was not blinded to the participants group, and this may have introduced bias when examining the participants, as the identification and interpretation of neovascularity is largely operator dependent. However, this ultrasound finding may contribute to the development of improved staging of treatments for this condition. Fig. 1 depicts the rotator interval of the left shoulder and demonstrates an area of neovascularity adjacent to the long head of biceps tendon.

### 5. Management

As described FSCS is typically classified into three or four stages (Reeves, 1975; Neviasser and Neviasser, 1987). It is the authors preference that following diagnosis to divide the condition clinically into two stages; (i) more pain than stiff, and (ii) more stiff than pain.

Once the diagnosis is established the first stage in management involves patient education. People suffering from FSCS typically want to know; what is the problem?, what has caused the problem?, why them?, how long will it last?, what treatments are available?, how effective are the available treatments?, and what are the expected outcomes? It is incumbent upon every health professional to present this information in an unbiased and patient focussed manner. In order to do this, the clinician must be aware of

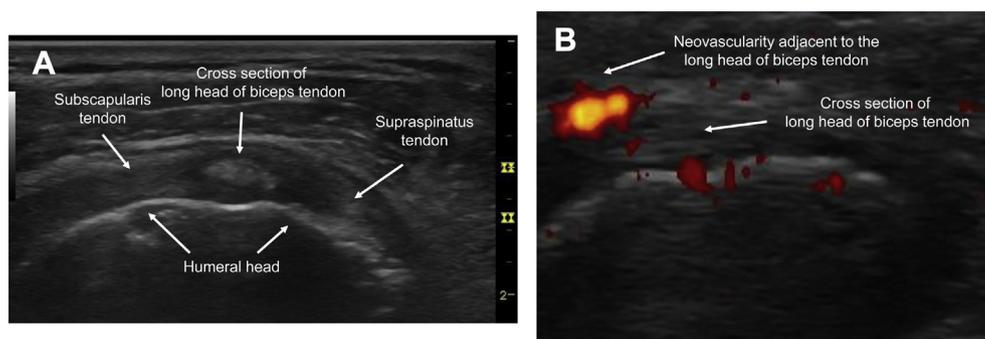


Fig. 1. Rotator interval. 1A: Grey scale ultrasound scan of the rotator interval of the left shoulder. 1B: Neovascularity adjacent to the long head of biceps tendon in 56 year old woman with FSCS.

the current research evidence, the quality of that evidence, and its generalizability.

People suffering FSCS are frequently informed that the condition will get better without treatment and 'supervised neglect' is preferable to physiotherapy. This was the conclusion reported in an investigation employing a two-arm controlled design (Diercks and Stevens, 2004). Although described by the authors as a randomised study the fact that; no allocation concealment was described, no definitive description of how the quasi-randomisation process was conducted, that there was an unequal distribution of men and women and no blinding of assessors, suggests that this study was associated with a high risk of bias and as such, recommendations made from this investigation must be made with caution. In addition, when informing the patient that natural history studies suggest an average duration of 30.1 months, many may want a faster resolution of the pain and restricted movement, than would be afforded by supervised neglect.

Using ICD-9 code 726.0, and a database of 2370 people diagnosed with FSCS, outcome findings suggested that joint mobilization and exercise were associated with better outcomes, and ultrasound and massage with poorer results (Jewell et al., 2009). A limitation of these data is that the inclusion and exclusion criteria to diagnose FSCS are not available.

Other studies have also found in favour of mobilisation. In a study of 100 people diagnosed with FSCS receiving an average of 20 treatments over 12 weeks, both high grade and low grade glenohumeral inferior, anterior and posterior mobilisation techniques demonstrated significant improvements over 12 months with a trend for greater improvement (pain and movement) in the high grade group (Vermeulen et al., 2006). However, as a control group was not included in the design, the findings may have mirrored natural improvement. Mid-range mobilisation techniques were clinically less effective than end range mobilisation techniques and mobilisation with movement procedures over a 12 week period in 28 people with FSCS, and mobilisation with movement may be more effective in improving scapulo-humeral rhythm (Yang et al., 2007). A specific soft tissue massage procedure (Neil-Asher technique) demonstrated improvement in shoulder abduction range when compared with a group receiving manual therapy and exercise and a placebo group (Wies et al., 2003). Although randomised, this small study ( $n = 27$ ) only reported short term (12 week) data. In addition, no significant difference was reported for any of the groups for the main outcome measure, the Shoulder Pain and Disability Index (SPADI). More research is required as the clinical importance of increased abduction range in isolation, need be interpreted cautiously.

As described, SWD may be beneficial in the management of FSCS. In a small study of 30 people with FSCS randomised to (i) SWD and stretching (ii) superficial heating and stretching and (iii) stretching alone, 12 sessions of SWD and stretching over 4 weeks produced better range of movement results than the other 2 groups (Leung and Cheing, 2008). Twelve treatments of laser therapy administered over 12 weeks combined with home exercises may also be beneficial (Stergioulas, 2008).

Although acupuncture is frequently used to treat musculoskeletal conditions, the evidence for its effectiveness remains uncertain, and its use controversial (e.g. DC's Improbable Science <http://www.dcsience.net/?p=6089>, Which? <http://www.which.co.uk/news/2014/01/five-surprising-facts-about-health-treatments-350088/>). With respect to FSCS; one RCT, deemed to be of high quality (Sun et al., 2001), and two systematic reviews (Favejee et al., 2011; Jain and Sharma, in press) concluded that moderate short term benefit may result from acupuncture combined with exercise. Another recent systematic review concluded the evidence supporting the use of acupuncture in the treatment of FSCS remains

uncertain (Maund et al., 2012). At best the research evidence to support the use of acupuncture in the treatment of FSCS remains equivocal.

In the United Kingdom, physiotherapists have been performing injections to treat musculoskeletal conditions since the mid 1990's and performing ultrasound guided procedures, including ultrasound guided hydrodistension procedures (O'Connaire and Lewis, 2011). The reasons for this evolution in health provision have recently been discussed (Gamlin et al., in press).

Although popular and commonly recommended, the evidence for the use of corticosteroid (CS) injections in the treatment of FSCS is not definitive.

A summary of published literature suggests that CS injections may reduce pain and improve function in the short term (Carette et al., 2003; Blanchard et al., 2010; Maund et al., 2012), and that the benefit may be enhanced in both short term and medium term when guided intra-articular CS injections are combined with physiotherapy (Carette et al., 2003; Maund et al., 2012). In those with FSCS for less than one year, the best outcomes were reported in the group randomised to receive image guided injections and physiotherapy, which involved 12, 1-h sessions over 4 weeks (Carette et al., 2003).

In an investigation of ultrasound guided intra-articular injections and using the SPADI, shoulder pain scores and range of movement as outcome measures, the dose of CSs and the benefit of analgesics in isolation were compared. Significant benefits were reported in the CS group when compared with the analgesic group. As no difference was detected between the high and low dose CS groups, low dose CS injections were recommended (Yoon et al., 2013).

In an attempt to restore restricted glenohumeral movement, intra-articular injections have been used for more than 80 years. Payr in 1931 was described as attempting to distend the contracted capsule using intra-articular injections (Lundberg, 1969). Current practice involves using imaging guidance, such as ultrasound, to distend the capsule. Ultrasound guided hydrodistension is a procedure that involves injecting large volumes of sodium chloride into the glenohumeral joint, with an aim of distending the contracted capsule (Fig. 2).

The procedure is typically performed as an in-office technique where patients are screened for contra-indications and special precautions to the procedure, and are informed of the risks and benefits and post procedure course. Following consent and performance of the procedure, patients are monitored for 15 min and then are free to go home (O'Connaire and Lewis, 2011). The procedure is associated with a numbers needed to treat (NNT) for pain reduction of 2 and a NNT for improved function of 3 (Buchbinder et al., 2008). Improvement in shoulder range of movement has been reported when manual therapy and exercise are performed after the procedure (Buchbinder et al., 2007). Considerably more research is necessary to further understand the value of hydrodistension in managing FSCS.

Operative procedures are also used in FSCS. Manipulation under anaesthetic (MUA) involves scapular stabilisation with shoulder flexion, abduction and adduction, followed by maximal shoulder internal and external rotation. Tearing of the contracted capsule may be felt or heard. Although a routine procedure, the evidence supporting MUA is equivocal.

No differences were reported at 1.5, 3, 6 and 12 months, between MUA and exercise compared with physiotherapy exercises alone, in a randomised clinical trial of 127 people diagnosed with FSCS (Kivimaki et al., 2007). Iatrogenic intra-articular damage following MUA has also been reported, including; haemarthrosis, SLAP lesions, partial thickness tears of subscapularis, osteochondral defects and labral detachment (Loew et al., 2005).

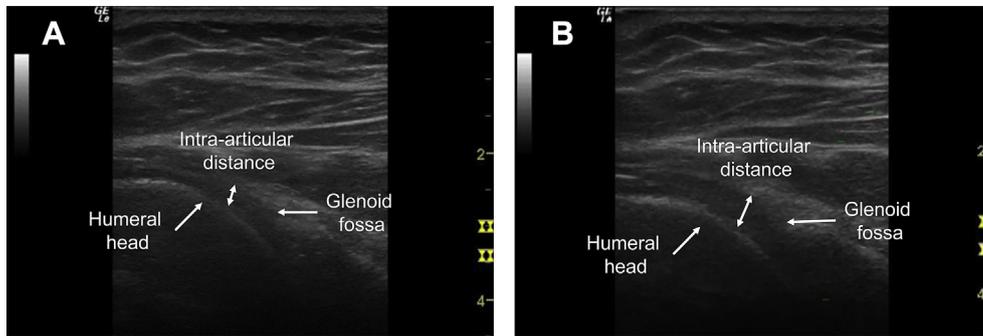


Fig. 2. Hydrodistension of the glenohumeral joint. 2A: Pre-distension and 2B: after 30 mls NaCl injected into joint under ultrasound guidance.

Arthroscopic capsular release involves combinations of; glenohumeral joint distension, capsular debridement, ligament splitting, loosening of adhesions, shoulder movement, shoulder manipulation, and post-surgical physiotherapy. Following the surgery, patients

usually need to take one week or longer off work. Currently the procedure is not supported by randomised controlled clinical trials and conclusions have been based on expert opinion and published case series, with an associated risk of bias (Dattani et al., 2013;

Table 1

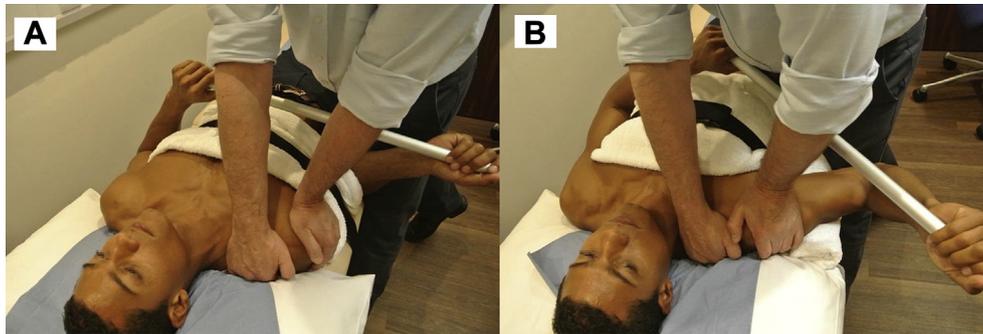
Frozen shoulder contraction syndrome: Injection therapy and physiotherapy care pathway.

Assessment	Stage I to early stage II (Pain > stiffness) corticosteroid injection and physiotherapy	Late stage II to stage III: (Stiffness > pain) hydro-distension and physiotherapy
<ul style="list-style-type: none"> <li>Sexes are affected equally with approximate peak onset of 52 years for men and 55 for women.</li> <li>Full patient interview and physical examination. Screen for yellow and red flags and monitor during treatment.</li> <li>Equal restriction of active and passive external rotation.</li> <li>Radiographs normal</li> <li>Organise blood tests to screen for diabetes and other related health conditions.</li> <li>Outcome measures Consider: ROM   Pain   Function   SPADI   DASH   Quick-DASH   EQ-5D-5L   PGIC   Patient Satisfaction</li> <li>Fully inform patient. Determine care pathway based on evidence and patients values. Consider following as possible pathway.</li> </ul>	<ul style="list-style-type: none"> <li>Screen for contraindications and special precautions for an intra-articular small volume cortico-steroid and lidocaine injection.</li> <li>If no CIs/SPs, explain risks and benefits, gain consent, proceed with ultrasound guided small volume cortico-steroid and lidocaine injection</li> </ul> <p>(Strong evidence short term and moderate evidence medium term)</p> <ul style="list-style-type: none"> <li>Perform the injection as an ultrasound guided procedure (or use another imaging guidance). No radiation associated with ultrasound.</li> <li>RCTs have not demonstrated a difference between location, dosage and volume of injection</li> <li>During first week gentle self-assisted and active movements</li> <li>2nd to 4th week, mobilisation, soft tissue massage, passive movements, self-assisted movements, exercises as tolerated.</li> <li>Consider re-injecting at 4/52 if pain not under control. No more than 3 times in one year and at least one month between injections.</li> <li>Injection procedures may overlap and be performed concomitantly.</li> </ul> <p>If unable to inject consider: [need to screen for CIs]</p> <ul style="list-style-type: none"> <li>Laser, mobilisation, soft tissue massage, passive movements, acupuncture, exercise, short-wave diathermy</li> <li>Oral steroid and suprascapular nerve blocks are also procedures associated with clinical benefit.</li> </ul> <p>Main References:  <a href="#">Favejee et al. (2011)</a>. Frozen shoulder: the effectiveness of conservative and surgical interventions. <i>BJSM</i>. 45:49–56.  <a href="#">Maund E et al.</a> Management of frozen shoulder: a systematic review and cost-effectiveness analysis. <i>Health Technol Assess</i>. 2012; 16(11):1–264. Epub 2012/03/13.</p>	<ul style="list-style-type: none"> <li>Screen for CI and SP to image guided GHJ intra-articular lidocaine and large volume NaCl injection. (ie hydro-distension)</li> <li>Consider USGI as no radiation associated with ultrasound.</li> <li>If no CIs/SPs, explain risks and benefits, gain consent, proceed with hydro-distension procedure if: <ul style="list-style-type: none"> <li>ROM not restored from CS and lidocaine injection, or</li> <li>1st presentation to clinic when in pain &gt; stiffness stage</li> </ul> </li> </ul> <p>(Moderate evidence; NNT-pain: 2, NNT-ROM/function: 3)</p> <ul style="list-style-type: none"> <li>Immediately following the procedure: Passive movements and PNF.</li> <li>Instruct home programme involving regular (hourly if possible) active movements, self-assisted active movements and stretching on day of procedure and regularly on following days.</li> <li>In clinic: mobilisation, PNF, progress home programme.</li> <li>If required repeat hydro-distension at one week.</li> <li>Injection procedures may overlap and be performed concomitantly.</li> </ul> <p>Main References:  <a href="#">Buchbinder et al. (2008)</a> Arthrographic distension for adhesive capsulitis (frozen shoulder) (Review), The Cochrane database of systematic reviews. 2008(1):CD007005.  <a href="#">Favejee et al. (2011)</a>. Frozen shoulder: the effectiveness of conservative and surgical interventions. <i>BJSM</i>. 45:49–56</p> <p>Notes:</p> <ul style="list-style-type: none"> <li>Monitor outcome measures.</li> <li>Ensure adequate physiotherapy is embedded within care pathway.</li> <li>For physiotherapists not currently providing ultrasound guided injections, develop care pathways with orthopaedic and radiology colleagues.</li> <li>In refractory cases and if improvement not as expected, consider orthopaedic referral for opinion on capsular release.</li> </ul>

Legend: ROM (range of movement), PGIC (Patient Global Impression of Change), CI (contra-indication), SP (special precaution), GHJ (glenohumeral joint), CS (cortico-steroid), USGI (ultrasound guided injection), NaCl (Sodium Chloride), PNF (proprioceptive neuromuscular facilitation), SPADI (Shoulder Pain and Disability Index), DASH (Disability of the Arm Shoulder and Hand), Quick-DASH (Quick-Disability of the Arm Shoulder and Hand), NNT (Numbers needed to treat for benefit).

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References: Range of movement (Valentine and Lewis, 2006); SPADI (Tveita et al., 2008); DASH | quick-DASH (Beaton et al., 2005; Kelley et al., 2013); EQ-5D (Maund et al., 2012).



**Fig. 3.** Antero-posterior shoulder mobilisation for the stiff > pain stage. Antero-posterior passive shoulder mobilisations may initially be performed with the humerus stabilised with a towel and mobilisation belt (3A). Using a handle, the patient concurrently holds the shoulder in external rotation or performs physiological external rotation mobilisation simultaneously with the mobilisation technique. 3B: Progression may be made by stabilising the scapula as more humeral abduction becomes available.

Fernandes, 2013). Further research is required to better understand the clinical benefit of capsular release as the quality of the evidence supporting this practice is currently low (Grant et al., 2013).

Evidence based care pathways for FSCS have been developed in the United Kingdom that are entirely led and performed by physiotherapists that encompass; clinical assessment, imaging, education, ultrasound guided cortico-steroid injection and hydrodistension procedures, embedded within a physiotherapy rehabilitation programme (O'Connaire and Lewis, 2011). It would be of value to appropriately compare these physiotherapy led programmes to other procedures such as; MUA and capsular release, for; clinical outcomes, adverse events, economic analysis, time off work and patient satisfaction. A suggested care pathway summary based on the physiotherapy led programme is presented in Table 1. Following a complete patient history, appropriate outcome measurements and physical examination; diagnosis is made on the basis of an equal restriction of active and passive shoulder external rotation range, a normal radiograph and exclusion of other potential sources of shoulder pain. Education detailing; an overview of the condition, expected natural history and outcome, treatment alternatives - including risks, benefits and expected outcomes, are considered in conjunction with the patient's values and beliefs, short and long term aims and expectations, health status and co-morbidities. Following this an individual may choose; a 'wait and see' approach supported by home based activity appropriate for the individual and the stage of the condition; clinically based physiotherapy, or a referral for an orthopaedic opinion. Others may wish to follow a combined injection and physiotherapy pathway (Table 1). Each individual must be appropriately screened for contraindications and precautions before an injection should be considered. If deemed not to be appropriate the

reasons for this need be documented, communicated and an alternative care pathway agreed. Depending on the stage of the presentation, injection therapy may involve a single intervention or combinations of corticosteroid, analgesic and hydrodistension procedures. Following an injection, patients are required to wait for a minimum of 15 min to monitor any side effects related to the procedures and are provided with post injection information and advice leaflets. Following a corticosteroid procedure, individuals are typically requested to perform regular self-assisted or slow pendular shoulder exercises within the tolerance of pain and this may be progressed as able. Following hydrodistension and with the individual's consent, passive and active physiological movements are initiated to maximise gains made during the injection procedure. The patient is instructed to continue with regular home exercises to maintain the gains. Clinically, physiotherapy may involve manual therapy procedures directed at the soft and joint tissues primarily aiming to further modulate pain and progress physiological movements as able. Techniques such as antero-posterior (AP) shoulder mobilisation procedures may be initiated to determine benefit. Anecdotally, simultaneously combining passive shoulder AP mobilisations while the patient performs physiological external rotation of the shoulder (with the aid of a walking stick or broom handle) may enhance the effect of AP mobilisations provided in isolation. It may also be beneficial to use a seat belt or large towel firmly wrapped around the upper chest (but not the arms) in an attempt to stabilise the scapula before performing mobilisation procedures.

Additionally inferior shoulder mobilisations may be attempted by placing the upper limb at the end of the hand behind back range and stabilising the scapula before attempting the procedure (Figs. 3 and 4). A variety of proprioceptive-neuro-facilitation procedures



**Fig. 4.** Inferior shoulder mobilisation for the stiff > pain stage. 4A: With the scapula stabilised and the hand placed at the end of the available 'hand behind back' range of movement the physiotherapist performs inferior shoulder mobilisation. The effectiveness may be enhanced by using a mobilisation belt positioned on a towel over the forearm and around the therapist's waist. Neurovascular compromise must be carefully monitored. 4B: As more 'hand behind back' range becomes available the technique is progressed.

(PNF) may also enhance treatment effectiveness. These anecdotal observations need be tested in appropriate research trials. Determining which clinical treatments to offer and combination(s) of injection therapy currently depends largely on the individual's presenting symptoms, speed of progression and goals.

## 6. Conclusions

The pain and movement restriction that is the hallmark of FSCS is typically associated with substantial morbidity. Although the condition improves over time, full pain free range of movement, may not be restored in everyone. Additionally, it is likely that most people suffering from FSCS would prefer resolution if possible, more quickly than the average 30.1 months. Physiotherapists in the United Kingdom have set up dedicated care pathways that provide; assessment, referral for imaging, education, health screening, ultrasound guided corticosteroid and hydro-distension injections, embedded within physiotherapy rehabilitation. The entire care pathway is provided and performed by physiotherapists and evidence (of varying levels of quality) exists to support each stage of the pathway. Substantial on-going research is required to better understand; epidemiology, patho-aetiology, diagnosis and assessment, best management, health economics, patient satisfaction and if possible prevention. The term adhesive capsulitis does not appear to correctly reflect the pathology, which is more appropriately described by the term, contraction of the shoulder, but this may not reflect the substantial pain associated with this condition, and as such the term frozen shoulder contracture syndrome, may be a more appropriate description.

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