Treatment of Frozen Shoulder Using Distension Arthrography (Hydrodilatation): A Case Series

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Abstract
Frozen shoulder (adhesive capsulitis) is a common, painful and disabling condition which is typically slow to resolve. Patients with this condition will be seen in every musculoskeletal practitioner’s clinic on a regular basis. There is a wide variety of treatment modalities available, some more effective than others. This article reviews the literature on the aetiology and natural history of the condition, and the common treatments provided. The literature on hydraulic arthrographic capsular distension (hydrodilatation) is reviewed and six cases referred for this treatment from a chiropractic clinic are presented.

Background
Since first described as peri-arthritis scapulo-humerale by Duplay in 1872, the condition commonly known as frozen shoulder continues to present a clinical conundrum for practitioners1. In 1934 Codman labelled the condition “frozen shoulder” and postulated that the underlying pathology was tendonitis of the rotator cuff. Codman went on to state that frozen shoulder constituted “a class of cases which are difficult to define, difficult to treat, and difficult to explain from the point of pathology”2. Over the years this painful and disabling condition has been known varyingly as: adhesive capsulitis, restrictive periarthritis, Duplay disease, frozen shoulder, frozen shoulder syndrome, scapulohumeral periarthritis, adhesive bursitis, obliterative bursitis and arthrofibrosis3. For the purposes of this paper the term frozen shoulder(FS) will be used.

Definition
Primary (idiopathic) FS may be defined as idiopathic shoulder pain of at least one month duration accompanied by increasingly severe limitation of active and passive glenohumeral movements in all ranges of motion in persons who have no identifiable general illness and whose radiographs are entirely normal7. Secondary FS is clinically indistinguishable from primary FS, however in secondary FS, an identifiable disorder such as a rheumatological or neurological disease is present8 or other potential predisposing factors as listed below. Zuckerman and Cuomo subdivided secondary FS into intrinsic, extrinsic and systemic categories9.

Prevalence
FS affects approximately 2 percent of adults, usually between ages 40 and 65 and affects more women than men10 and appears to be more prevalent in patients with heart disease11. Although rare, FS has been reported in children12 and in identical twins13.

FS is found in 10 - 20 percent of diabetics and the incidence appears to be increasing along with that of diabetes14. Within the diabetic population, separation of the diabetic patients into type I and type II diabetics reveals a prevalence of 16 percent and 7 percent, respectively. An increased prevalence of FS was noted in female patients as well as those who had a longer duration of diabetes mellitus however the incidence does not appear to be associated with long-term glycemic control.10

Potential Predisposing Factors
Although no etiological studies have been performed, several potential predisposing factors have been identified in FS, including:
- diabetes10,14
- pulmonary tuberculosis15
- scleroderma15
- a period of enforced immobility resulting from trauma, overuse injuries or surgery16,18

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• genetic predisposition\textsuperscript{25}
• Dupuytren’s disease\textsuperscript{26,27}
• elevated serum lipids\textsuperscript{28}
• heart disease\textsuperscript{11}
• neurotic personality\textsuperscript{29}

While none of these predisposing factors have been formally proven and at least one, neurotic personality, has been refuted\textsuperscript{30} most have sufficient anecdotal evidence to be considered when interviewing a patient with suspected FS.

**Natural History**

It is commonly believed that FS is a self-limiting syndrome divided into three consecutive stages lasting approximately 18 months in total\textsuperscript{11,12}. These stages are: pain, stiffness and recovery. Studies have shown the duration of the disorder to be far more protracted. In a prospective study involving 49 FS patients over a 5-10 year period, Reeves\textsuperscript{33} found that the time to greatest recovery was an average total of 30.1 months and in 30 percent of patients the second shoulder became similarly affected 6 months to 7 years after the first with the second event following a similar chronology to the first.

**Stage 1 - Painful stage: 2 - 9 months.** During this stage the patient experiences increasing pain with movement and pain is often worse at night. There is progressive loss of motion with increasing pain. The pain often becomes disabling and has a significant effect on activities of daily living.

**Stage 2 - Adhesive or Stiffness stage: 4 - 12 months.** During this stage pain begins to diminish, and moving the arm is more comfortable although range of motion is greatly reduced, often as much as 50 percent less than the other arm.

**Stage 3 - Recovery stage: 12 - 42 months.** The condition begins to resolve with gradual restoration of motion. In general the longer the stiffness phase, the longer the recovery phase. In over 50 percent of patients some restriction of motion remains even after greatest recovery but only in a small percentage is this restriction a handicap.\textsuperscript{33}

**Pathophysiology**

The cause of FS has yet to be identified. Radiographs are essentially normal. There are no signs of systemic inflammatory disorder, and other joints remain unaffected. Laboratory tests yield no useful answers. ESR and C reactive protein are not elevated. Tests for immunologic components such as RF, antinuclear antibodies and autoantibodies to smooth muscle, collagen, or cartilage are negative and the frequency of HLAB27 is not increased among FS patients. No known infectious agent has been identified.\textsuperscript{35}

Neviaser and Neviaser described FS as “primarily an inflammatory reaction in the joint capsule that subsequently leads to the formation of adhesions in the axillary fold”. They described four stages of FS. Stage I was labelled the pre-adhesive stage in which there is a fibrinous synovial inflammatory reaction only demonstrable by arthroscopy. Stage II was marked by acute adhesive synovitis with a proliferative synovitis and early adhesion formation. In Stage III, (the stage of maturation) there is less synovitis with loss of the axillary fold. Stage IV they labelled the chronic stage in which adhesions in the axillary fold are fully mature and may in fact obliterate this structure.\textsuperscript{36}

Hannafin\textsuperscript{37} described the arthroscopic findings during three stages of FS as follows:

**Stage 1 findings:** diffuse glenohumeral synovitis, hypertrophic, hypervascular synovitis, rare inflammatory cell infiltrates and a normal underlying capsule.

**Stage 2 findings:** diffuse, pedunculated synovitis, tight capsule with rubbery feel on insertion of the arthroscope, hypertrophic, hypervascular synovitis with perivascular and subsynovial scar, significant fibroplasia and scar formation in the underlying capsule.

**Stage 3 findings:** no hypervascularity, remnants of a fibrotic synovium visible. The capsule feels extremely dense and thick on insertion of the arthroscope and there is a diminished capsular volume.

Recent studies have shown the histological and immunocytochemical findings in FS to be very similar to those in Dupuytren’s disease of the hand, with no inflammation and no synovial involvement\textsuperscript{30}. Contracture of the coracohumeral ligament acts as a check rein to passive glenohumeral movement and external rotation\textsuperscript{26,38}. Later work by Bunker\textsuperscript{39} demonstrated that in patients with FS there is an absence of metalloproteinase-14 when compared to controls which may lead to chronic fibrosis of the joint capsule.

Müller tested the hypothesis that FS syndrome is a reflex sympathetic dystrophy. Müller found several similarities between FS syndrome and an algoneurodystrophic process
including progressive decreased bone mineral density in the affected humerus in comparison to the non-affected side, increased uptake in affected areas in both diseases on radioisotope bone scan, dull night pain, idiopathic and post-traumatic causes and reduced skin temperature in affected areas as demonstrated by thermography. It is worth noting that there is some controversy surrounding the nature of reflex sympathetic dystrophy and whether it is a primary organic disorder or a primary psychogenic disorder associated with achieving some secondary gain.

**Diagnosis**

**History and Examination**

History and physical examination form an important basis for clinical decision-making. While knowledge of a patient’s history does not appear to influence the reliability of clinical tests, such knowledge does focus testing and produces an increased prevalence of positive findings. Patients with a history of worsening shoulder pain with loss of motion of 1 month duration and a physical examination confirming loss of active and passive glenohumeral joint motion may be considered to fit the criteria for FS. Physical examination of such patients should include inspection and palpation of the joints of the shoulder girdle, examination of the cervical spine, range of motion assessment of the shoulder girdle, and provocative testing.

**Imaging**

If imaging evaluation is contemplated, studies should generally be limited to routine shoulder radiographs. Plain film radiography may be performed initially to assess the glenohumeral joint and subacromial space and to rule out frank pathologies such as calcific tendonitis. The joints should be normal or show minimal age-related changes. Bone mineral density may be reduced in the proximal humerus in female sufferers but this appears to resolve over time and is not clinically significant. Some authors have suggested that the diagnosis of FS “must be confirmed by arthrography because it is the most effective means of differentiating the stiff and painful shoulder from an adhesive capsulitis” and others have suggested that arthrography is very useful in distinguishing between idiopathic and secondary adhesive capsulitis. However, arthrography is an invasive procedure with concomitant risks and it is the opinion of the authors that the use of arthrography for diagnosis of FS is somewhat old school and that the diagnosis of FS is readily made at clinical examination by a practitioner skilled in musculoskeletal examination.

Dynamic sonography has been suggested as a valuable tool for diagnosing adhesive capsulitis. In a study involving 23 patients with confirmed adhesive capsulitis at arthrography, 21 showed limitation of movement of the supraspinatus tendon against the acromion of the scapula on sonographic examination. The study found a sensitivity of 91%, specificity of 100% and an accuracy of 92%. The authors suggest that dynamic sonography is a reliable technique for the diagnosis of FS.

The clinical utility of MRI in evaluating patients with FS remains unclear. Emig reported that a joint capsule and synovial thickness greater than 4 mm was a useful criterion for the diagnosis of FS. Manton reported that capsular/synovial thickness and static fluid volume are inconclusive as MR arthrographic signs for establishing a diagnosis of FS while Lee reported that MR arthrography appears to yield useful results for the diagnosis of FS. Connell compared MRI and surgical findings in a group of patients who underwent surgery for adhesive capsulitis and concluded that MRI can identify alterations in the shoulder joint that correspond to abnormalities seen at surgery and therefore MRI may be useful for discriminating adhesive capsulitis from other shoulder abnormalities. These findings are similar to Carrillon’s who reported that MR imaging with gadolinium injection can contribute to the diagnosis of idiopathic FS in difficult cases.

**Differential Diagnosis**

Several conditions have been reported to produce shoulder pain and stiffness. These should be kept in mind when evaluating the patient with suspected FS. Spindler and Dovan described the following conditions which may cause symptoms similar to FS:

- referred pain from cervical spine
- subdiaphragm
- ribs
- sternoclavicular joint
- acromioclavicular joint
- fracture of the distal clavicle
- glenohumeral joint and associated structures
- rotator cuff lesions
- subacromial space pathology
- bicep tendon lesions
- impingement syndrome
- joint instability
- g/h joint dislocation
- labral tears

Less common conditions which have been known to cause symptoms similar to FS are:

- neoplasms
- pneumonia
- neuralgic amyotrophy
Objective Measures
Several objective tools have been developed to assess shoulder disability and response to treatment. In this series the Simple Shoulder Test was chosen because of its ease of use for both patients and practitioners.

Simple Shoulder Test
The Simple Shoulder Test (SST) was developed by the University of Washington Shoulder and Elbow Service as a standardised way of recording shoulder function before and after treatment. The short test requires patients to answer ‘yes’ or ‘no’ to twelve questions derived from the common complaints of persons suffering from shoulder pain. The SST is simple to administer and has demonstrated a high degree of reproducibility and face validity. The SST has been shown to be reliable and responsive to successful treatment of FS.

(The SST is distributed free of charge via the internet and may be obtained at: http://www.orthop.washington.edu/shoulder_elbow/technical/shouldertest)

Treatments
Some authors state that pain relief is the main objective of all treatments for FS. However, considering the protracted nature of this disorder and its impact on patients’ functionality, this objective should be refined to early pain relief and functional restoration. The literature is rife with studies reporting on the many treatments for FS. Searches were conducted in MEDLINE, CINAHL and ACMd up to June 2004 using the search terms: frozen shoulder, adhesive capsulitis, treatment, diagnosis, hydrodilatation, and distension arthrography. Table I summarises the FS treatments and outcomes reported in the retrieved literature. Evident from this table is the wide variety of treatments reported. These range from a program of rest, analgesia and gentle exercise to open surgical release. It is important to note that overly vigorous exercises or physical therapy can be counterproductive. Aggressive techniques such as glenohumeral joint manipulation under anaesthesia and open capsulotomy are not without significant risk and should be reserved for the most refractory cases which have failed to respond to less invasive approaches however, there is little evidence to show that such an approach will alter the natural course of FS. Combining supervised physiotherapy with intraarticular steroid injection and home exercises may provide faster improvement in shoulder range of motion however, physiotherapy alone appears to be of little value in the management of FS. Treatment outcomes appear to be less satisfactory in patients with diabetes or when treatment is covered by workers’ compensation. It is beyond the scope of this paper to review all FS treatments. Readers are directed to the 2004 Cochrane Review of Interventions of Shoulder Pain for the evidence of effectiveness of FS treatments including hydrodilatation. The reviewers concluded that there is little evidence to support or refute the efficacy of common interventions for shoulder pain including FS. In addition, they concluded that there is a need for further well designed clinical trials and that more research is needed to establish a uniform method of defining shoulder disorders and developing outcome measures which are valid, reliable and responsive in these study populations.

Choice of treatment approach depends upon the patient’s functional status at the time of presentation. Generally speaking a trial of conservative care is warranted provided the clinician closely monitors progress. If progress stalls or the condition worsens, an alternate approach to treatment should be considered.

This was the approach taken with patients in this case series. All patients had been symptomatic for several months and had failed to respond to a course of physiotherapy prior to referral for hydrodilatation.

The 2004 Cochrane Review of Interventions for Shoulder Pain uncovered no trials of hydrodilatation for FS which met the four criteria for inclusion in the review. There were studies which partially met the criteria but were excluded because of some deficiencies. The following studies were excluded but are worth considering here.

Hsu and Chan reported a prospective study comparing manipulation under anaesthetic and physiotherapy with arthroscopic distension and physiotherapy, and with physiotherapy alone in patients with FS. The authors found that there was no significant difference between distension and manipulation groups in terms of pain, range of motion or function. They reported that clinically, arthroscopic distension offers advantages in terms of less risk of fracture, and affords the opportunity to view the shoulder joint and assess for rotator cuff pathology. This study was not included in the Cochrane Review because it was not a randomised trial.

Jacobs et al compared three FS interventions in a prospective randomised trial. Shoulder distension with air, intra-articular steroid injection, and distension with air and steroid. The authors found intra-articular steroid injection and distension with steroid to be superior to distension alone, but found no significant difference between steroid with distension, and intra-articular steroid injection without...
The Cochrane reviewers excluded this study because changes in pain were reported for the whole population and not by treatment group, and improvement in range of motion and strength was reported by group as a mean change, but with no baseline scores and no standard deviations.

Corbeil et al. conducted a double-blind prospective study which compared intraarticular injection of steroid (n=20) vs hydrodilatation (n=25) and found no significant difference between the two groups. More than 80% of the patients who were experiencing pain at rest and nocturnal pain improved under both treatment regimes. However, because Corbeil et al failed to define ‘adhesive capsulitis’, failed to report standard deviations and ‘p’ value and there was no description of how passive motion of the scapulohumeral joint was measured the study was excluded from the Cochrane Review.

In a paper published subsequent to the aforementioned 2004 Cochrane Review, Buchbinder et al reported on their randomised, double blind trial comparing placebo to shoulder joint distension with normal saline plus corticosteroid. In this study involving 48 subjects the hydrodilatation group demonstrated significantly greater improvement in pain and disability measures over placebo.

It seems that the more rigid the shoulder is at the time of hydrodilatation, the less likely it is that full movement will be restored. However, even if rigidity is unchanged following hydrodilatation, pain relief is still achievable.

Hydrodilatation

Hydrodilatation or as it is more accurately known, distension arthrography, generally involves inserting a needle into the joint capsule under local anaesthesia using fluoroscopic control and dilating the contracted joint capsule with a mixture of long acting anaesthetic, cortisone and saline. Some studies have used air instead of saline while others have excluded the use of steroid. However, hydrodilatation with air results in a “squelch” when the shoulder is subsequently moved which patients find unpleasant. Furthermore, it is the experience of one of the authors (RB) that because of the inherent compressibility of air, distension of a contracted capsule is more difficult that if saline is used.

| Treatment                                        | Outcome                                                                                           | References |
|--------------------------------------------------|---------------------------------------------------------------------------------------------------|------------|
| 1. rest, analgesia, motion exercises, moist heat | Equal to natural history. Too rapid an increase in activity may prolong the disorder.            | 5, 62, 63, 65. |
| 2. physiotherapy and mobilization                | Lacks good clinical studies. Overly aggressive mobilization may prolong the disorder.            | 64-70.     |
| 3. oral corticosteroids                          | Decrease in pain but no change in range of movement.                                              | 71.        |
| 4. corticosteroid injection                      | Usually decreased pain but not much improvement in range of movement. Pain relief is moderate:    | 72-75.     |
|                                                  | subacromial injection may be as effective as intra-articular injection.                           |            |
| 5. glenohumeral joint manipulation with and      | Marked improvement in range of movement. Adverse outcomes include humerus fracture, g/h joint   | 10, 61, 76-81. |
| without anaesthesia with                        | dislocation, neurovascular injury, articular cartilage injury.                                    |            |
| 6. arthroscopic capsular release                 | Marked improvement in range of movement. Significant post-procedure pain reported.               | 31, 80, 82-84. |
| 7. open surgical release                        | Good improvement in range of motion. Reserved for FS cases which have failed to respond to less   | 80, 85, 86. |
|                                                  | invasive procedures. Should be used with caution in insulin dependent diabetics.                 |            |
| 8. suprascapular nerve block with intra-articular| Reduction of pain and increase in range of movement. No controlled clinical studies performed.   | 87.        |
| injections                                        |                                                                                                  |            |
| 9,10. capsular hydrodilatation with and without  | Significant pain reduction and moderate improvement in range of movement. The procedure is      | 73, 76, 88-107. |
| steroid injection, and with TENS                 | moderately painful. Significant reduction in procedural pain is reported with the use of TENS     |            |
|                                                  | during hydrodilatation. FS stage may be important in predicting degree of improvement.             |            |
| 11. Japanese herbal medicine                     | Improvement in range of movement in 2 patients. Lacks any good clinical studies.                  | 108.       |
| 12. Bowen technique                              | Lacks good clinical studies.                                                                     | 109.       |
| 13. acupuncture and exercise                     | Randomized controlled trial with 35 patients. Improved functional mobility and decreased pain     | 110.       |
|                                                  | in treatment group.                                                                             |            |
| 14. osteopathic treatment                        | Pilot study comparing osteopathic treatment and physiotherapy. Both improved range of motion and  | 111.       |
|                                                  | gave pain relief.                                                                               |            |
| 15. chiropractic treatment                       | Single case report. Lacks good clinical studies.                                                 | 112.       |
In the series by Bell et al. (101), distension was continued until capsular rupture occurred. Depending upon the contracted state of the joint capsule, this usually occurred when between 10 ml and 55 ml of normal saline had been injected. On occasion, rupture was not achieved until 100 ml had been injected. Rupture usually occurred through the subscapularis bursa and occasionally down the bicep sheath. In some cases, the pain of the procedure was so severe that it had to be terminated before capsular rupture was achieved (101). Rizk reported that pain relief was not achieved when rupture occurred at the distal bicipital sheath (94).

For shoulder arthrography a posterior approach to the shoulder is preferred. Typically the axillary pouch is fibrosed in FS making an anterior approach technically more difficult. It has also been our experience that a posterior approach is more readily tolerated by the patient.

In our series the procedure was performed by one of the authors (RB), a skilled radiologist. Initially, skin anaesthesia was performed using a sterile technique and a 22 gauge spinal needle passed into the joint under fluoroscopic control. Needle position was confirmed by injection of 5 ml of non-ionic contrast material (Omnipaque 300) (see figure I). This was followed by 5 ml of Marcaim long acting anaesthetic and 2 ml of Celestone Chronodose. Twenty to 30 ml of refrigerated sterile saline was then instilled, making 42 ml the maximum total injected volume into the shoulder joint. Even though capsular rupture was not the clinical objective in our series, evidence of rupture down the bicep tendon sheath is noted in figure II.

**Case Reports**

Over an eight month period, four patients attending a chiropractic clinic were diagnosed with FS. The diagnosis was made based on their history and physical examination findings. Each patient was referred for hydrodilatation treatment. Where practical, a Simple Shoulder Test was administered prior to hydrodilatation and at varying intervals following.

1) 74 Year Old Male

SV, a 74 year old male was referred by a general medical practitioner for treatment of left shoulder pain. SV had been suffering severe left shoulder pain since suffering a CVA with left sided hemiparesis 13 months previously. He had no prior history of shoulder pain. There was point tenderness over the greater tuberosity of the left humerus. Passive range of motion was grossly reduced and painful in all ranges of glenohumeral motion. An SST was not administered for obvious reasons. SV’s shoulder pain interfered with sleep while the reduced range of movement presented difficulties in personal hygiene and dressing. Crepitus was noted in the glenohumeral joint in the limited range of movement available. Plain radiographs of the left shoulder demonstrated degenerative sclerosis of the upper cortex of the greater tuberosity of the humerus. No soft tissue calcification was present. Minor degenerative lipping was present in the glenohumeral joint. The acromioclavicular joint was normal. Previous treatment consisted of physiotherapy and occupational therapy three times per week for most of the prior 12 months with no resultant increase in range of movement or diminution in pain. SV was considered a candidate for hydrodilatation treatment. This was performed using 8 ml of Marcaim, 2 mls of 2% Lignocaine, 5 ml of contrast material and 2 ml of Celestone chronodose. A further 5 ml of saline was then injected. The joint capsule remained intact. There were no complications noted at the time of procedure.

![Pre-distention contrast](image-url)
The immediate result was a marked improvement in glenohumeral joint movement in all ranges. SV reported that the procedure was quite uncomfortable but that he had slept through the night without being awakened with shoulder pain for the first time since his CVA. SV received physiotherapy treatment at the local hospital for three weeks following the hydrodilatation procedure. He chose to cease these treatments because of the discomfort involved and lack of obvious improvement in shoulder range of motion. SV attended the chiropractic clinic for seven treatments over a four month period after which he was discharged from care. Treatment consisted of 6 minutes of ultrasound (2.5 wcm2 @ 45%) to the glenohumeral joint, mobilisation of the glenohumeral joint and the scapulothoracic joint followed by 10 minutes of moist heat application (HeatTreat™). There was a gradual increase in range of movement during the first two months of treatment to approximately 50% of normal. SV’s range of motion did not change in the ensuing two months. SV’s sleep remained undisturbed by shoulder pain and there was a marked improvement in the ease with which he and his carer were able to perform his normal daily activities related to hygiene and dressing.

2) 39 Year Old Female

PW, a 39 year old female process worker was referred by a general medical practitioner for management of shoulder pain of 3 months duration involving her dominant left side. Onset was insidious. Prior history included a work related rotator cuff injury approximately 9 years previously resulting in a residual reduction in range of motion but no significant reduction in functionality. The presenting left shoulder pain was deemed by her general medical practitioner to be work related and consequently hers was a workers’ compensation case. Examination revealed grossly restricted and painful active and passive ranges of left glenohumeral joint motion. Plain radiographs revealed calcification in the subscapularis tendon which was believed to be related to the previous injury. PW answered ‘no’ to questions 11/12 on the initial SST evaluation. PW rated her shoulder pain as 9/10 on a 10 point VAS.

PW was taking prescription NSAIDs and had been receiving physiotherapy treatment for six weeks prior to presentation without an increase in range of motion or decrease in pain.

PW was referred for hydrodilatation which was performed using 12 ml of saline, 2 ml of 0.5% Marcain and 1 ml of Celestone Chronodose. No immediate complications were noted at the time of procedure.

PW was reviewed the day following the procedure. She reported that although the procedure itself was quite painful, she had experienced an immediate reduction in shoulder pain and that she had slept well the night of the procedure. Both active and passive ranges of glenohumeral joint motion were increased by approximately 60%. PW was treated using a combination of ultrasound (3wcm2 @ 50%), glenohumeral joint mobilisation, interferential current (80 - 160 Hz continuous) to the glenohumeral joint and cervical spinal manipulation. In addition PW was given a home exercise program of stretching, light resistance band exercises and heat treatment (HeatTreat™). PW returned to modified work duties after the first week of the procedure. These duties were increased to pre-injury status within one month of the procedure. After six weeks PW’s range of motion was near full with some grabbing shoulder pain noted when working overhead or moving the arm rapidly. A second SST was administered at this time. PW answered ‘yes’ to 11/12. Twelve weeks post-procedure PW returned to her general medical practitioner complaining of residual shoulder pain (VAS 3/10) and was subsequently referred for physiotherapy treatment under workers’ compensation.
3) 49 Year Old Male

SO, a 49 year old security officer who worked in a hospital setting had been attending for supportive chiropractic care since 1991. In August 2001, during the course of a regular consultation, he mentioned that he was experiencing right shoulder pain which had been diagnosed as an impingement syndrome and was being treated by the hospital's physiotherapist. Three months later, when SO next attended for chiropractic care, he was questioned about his shoulder and indicated that he continued to have physiotherapy but that the condition was not improving. SO’s shoulder was examined and he was found to have severely and painfully restricted active and passive range of glenohumeral joint motion. An SST was administered and SO answered ‘no’ to all twelve questions. It was suggested that the diagnosis of impingement syndrome was incorrect and that he in fact was suffering with FS. He was referred for hydrodilatation treatment at the hospital where he worked to be followed by physiotherapy. SO reported that the procedure was very painful but that he experienced immediate and lasting relief from his shoulder pain and a marked increase in both passive and active shoulder movement. He curtailed the physiotherapy after eight weeks because of the pain caused by that treatment and the apparent lack of progress. Twelve months following the procedure his right shoulder range of motion was full and he was unrestricted in his activities. An SST was administered at this time and SO answered ‘yes’ to all twelve questions. At this time, and for no apparent reason, he developed FS on the left side. An SST at this time yielded ‘no’ answers to 11/12 questions. SO was referred for hydrodilatation treatment without any post-procedure treatment. As he had experienced with the first hydrodilatation, the procedure was very painful but there was an immediate and significant reduction in his shoulder pain and a marked increase in range of movement. An SST administered at 4 weeks yielded ‘yes’ answers to 9/12 questions. Twelve months later and with no post-procedure physiotherapy, the range of movement in the second shoulder remains full and he is performing all activities involving his shoulders without restriction. Affirmative answers are given to all 12 SST questions.

4) 49 Year Old Female

SA, a 49 year old florist, presented with a chief complaint of insidious onset pain and restricted movement in her non-dominant left shoulder. The problem had been present for six months. An initial SST yielded negative answers to 11/12 questions. There was no prior history of such pain. SA had been receiving physiotherapy treatment for a period of six weeks however her shoulder condition continued to worsen. Examination revealed painful and severely restricted active and passive glenohumeral joint movement in all ranges. Plain radiographs revealed no bony or articular abnormality. There was no capsular soft tissue calcification demonstrated.

SA was referred for hydrodilatation treatment. This was performed in accordance with the procedure described above and shown in Figures I & II. SA kept detailed diary notes for the first seven days following hydrodilatation treatment. The procedure was described as very painful. She was acutely aware of the increasing pressure and intensifying pain in her shoulder as hydrodilatation proceeded. The procedure was terminated at the point when she felt as though her shoulder “was at explosion level”. When she sat up after the procedure there was a feeling of a weight falling down her arm but, even though her arm felt “heavy”, her shoulder “had a sense of freedom”. Examination immediately following hydrodilatation showed greatly increased passive and active ranges of glenohumeral joint motion. These increases were present in all ranges and the movements did not elicit the severe grabbing pain which had been present prior to the procedure. During the following eight hours SA’s arm pain increased but was manageable with OTC analgesics. She reported sleeping for 5 hours continuously and awakening the next morning “absolutely pain free” and with no feeling of heaviness in her arm. SA experienced undisturbed sleep for up to 9 hours per night following the first night. Over the course of the first week SA attended clinic for three treatment sessions which involved interventional current to the glenohumeral joint (100-160 Hz continuous - 15 minutes), mobilisation of the glenohumeral joint, cervical spine manipulations and application of moist heat to the glenohumeral joint (HeatTreat™ 15 minutes). A home rehabilitation program was devised which included shoulder stretches, and moist heat application. SA was instructed to perform the stretches twice daily. One month later, resistance work (yellow Thera-Band™) and pulley work were introduced. An SST was administered at this time. This test yield ‘yes’ answers to 8/12 questions while ‘no’ answers were given to 4/12 questions.

Over the course of the year following hydrodilatation, SA’s shoulder range of motion continued to improve with external humeral rotation being the last range to approach that of her other shoulder. There was a slight loss of motion in all ranges and some shoulder pain following a minor motor vehicle accident eight months after the procedure. These symptoms fully subsided within two weeks. SA reported that she ceased performing pulley exercises after about three months because they seemed to be causing some residual shoulder pain. At the last follow-up, twenty-three months following hydrodilatation, SA reports that she is performing all activities without restriction. The only residual symptom is a feeling...
of weakness in her left shoulder when working with her arms overhead. This has necessitated modifying her lifestyle somewhat but is not perceived as a disability. An SST at this time yielded ‘yes’ answers to all questions but one.

Discussion
The literature on FS reveals that it is a relatively common disorder which, left untreated, will result in significant pain and disability for a period of up to 42 months after onset.

Treatments for FS reported in the literature range from a program of rest, analgesia and motion exercises to open surgical release. The available literature is not overly helpful in assisting clinical decision making with regard to FS treatment. The Cochrane reviewers determined that no conclusions can be drawn regarding the efficacy of the interventions studied for adhesive capsulitis. It is worth noting however, that Buchbinder et al published a randomised, double blind placebo controlled trial which supports the use of hydrodilatation for FS and that there are at least 21 other studies reporting on over 700 shoulders which draws one to conclude that hydrodilatation is safe, yields immediate and lasting results in terms of pain relief and improved ranges of motion and is a cost-effective intervention in the management of FS.

The case series presented supports the position that management of the frozen shoulder using capsular hydrodilatation can reduce FS pain immediately and reduce disability from a period of months to a period of weeks. As hydrodilatation can reduce FS pain immediately and reduce disability from a period of up to 42 months after onset.

The literature on FS reveals that it is a relatively common disorder which, left untreated, will result in significant pain and disability for a period of up to 42 months after onset.

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