Frozen shoulder

Introduction

Idiopathic frozen shoulder or adhesive capsulitis is a commonly occurring condition characterised by a capsular pathology associated with pain and progressive loss of passive and active movement. It occurs in the general population with an incidence of approximately 2%[1] and of these 20 to 30% will go on to develop the condition bilaterally. It is more common in females[1], age over 40 years[2] and in the non-dominant arm[3]. There is a strong association with diabetes mellitus[4]and to a lesser extent with thyroid disease[5].

The hallmarks of frozen shoulder syndrome were first described by Duplay in 1872[6]. He felt the pain and stiffness noted in these patients was not due to arthritis, but rather, was due to soft tissue pathology of the periarticular structures. In 1934, Codman[7] first proposed the term frozen shoulder. He described a slow onset of pain felt near the insertion of deltoid, inability to sleep on the affected side, painful and restricted elevation and external rotation, with a normal radiological appearance. The loss of passive range of movement, particularly related to external rotation, has remained pivotal to the diagnosis of frozen shoulder. Codman was, however, unable to explain the pathology. Only recently has the pathogenesis become more clearly, but as yet, not fully understood.

Matsen[Matsen, 1994 #10] has defined the condition experienced by patients with stiff shoulder syndrome occurring *de novo* as an idiopathic global limitation of humeroscapular motion resulting from contracture and loss of compliance of the glenohumeral joint capsule. These primary idiopathic frozen shoulder patients make up 5% of all patients with stiff shoulder syndrome [8]. Harryman[9] has highlighted the importance of recognising any previous history of trauma to

categorise patients with stiff shoulders. With a history of trauma or other preceding stimulus, another group can be defined as having a limitation in humeroscapular motion presenting after an injury, low-level trauma or part of an accompanying condition, which results in a contracture of structures participating in the glenohumeral or humeroscapular motion interfaces. This second group can be further subdivided by injury or disease process and is analogous to secondary adhesive capsulitis as proposed by Lundberg[10] in which a known intrinsic, extrinsic or systemic precursor can be identified. Cuomo[11] believes it is inappropriate to include all various aetiologies within secondary frozen shoulder. Frozen shoulders due to systemic disorders such as diabetes mellitus or hypothyroidism only really differ from the idiopathic group in their resistance to treatment and can easily be included as part of this primary group. Intrinsic disorders, such as rotator cuff pathology, merit their own subdivision, as treatment must be directed at dealing with the initiating pathology as well as treating the shoulder stiffness.

A multitude of pathologies can predispose to frozen shoulder[12]. It can follow fractures or dislocations about the shoulder, shoulder, cervical or thoracic surgery or immobilisation following any upper limb surgery. It can occur in association with soft tissue pathologies such as rotator cuff and biceps tendonitis. It can follow any degenerative shoulder joint disease process. It has also been seen with cervical spine disorders, intrathoracic pathology and abdominal pathology. Clearly, as noted by Kozin[13], many of these conditions associate the patient with a period of pain, immobility or both.

Frozen shoulder research has been limited by the heterogeneous nature of the patient groups presenting with shoulder stiffness, as represented by these various definitions. Wiley and co-workers[14] have reported their series of 150 patients with presumed diagnosis of frozen shoulder. After arthroscopic examination only 37

patients were found to have primary frozen shoulder. Other studies have confirmed the importance of arthroscopy in accurately selecting patients for study[15]. Research performed without the control of arthroscopy to exclude other pathologies must therefore be questioned. The better identification of homogeneous patient groups will simplify treatment decisions and make outcomes more predictable[12].

Natural History

The pathological stages of primary frozen shoulder

One of the most contentious issues with regard to frozen shoulder is in defining the exact natural history. It has traditionally been regarded as a self-limiting condition, which universally settles over a variable time course[16]. Codman himself believed even the most protracted cases recover with or without treatment in about two years. Reeves[17] has performed a prospective longitudinal study of the natural history in 41 patients. Three sequential stages were noted. An early painful stage was described lasting between 10 and 36 weeks. This was followed by an intermediate stiff or frozen stage lasting 4 to 12 months. Finally a recovery stage was described lasting from 5 to more than 24 months. Reeves noted all but 3 of the patients ultimately obtained useful function in the affected limb although 25 patients showed some residual signs of mild range of motion limitation. The overall impression of frozen shoulder was that it was a self-limiting disorder and that most patients would be expected to recover within 2.5 years. Another important observation of Reeves was that the length of the painful period could be related directly to the length of the recovery period.

This self-limiting nature of frozen shoulder has been challenged by other workers[18 19]. Shaffer and co-workers have followed up a cohort of 62 patients with frozen shoulder for a mean time period of 7 years. They found that 50% of patients still had either stiffness or mild pain effecting the shoulder and 60% of

patients demonstrated some loss of expected range of movement particularly in external rotation. Thus though producing little functional disability half of patients remained symptomatic. This has led them to question whether frozen shoulder should be regarded as a self-limiting condition.

The stages of adhesive capsulitis have been further investigated by Hannafin et al[20]. They have correlated the clinical appearance and histology of patients with their arthroscopic stage as defined by Neviaser[3]. Using this information they have produced a comprehensive classification of adhesive capsulitis divided into 4 stages, which will be summarised.

In stage 1, symptoms have been present for less than 3 months and consist of an aching pain exacerbated at extremes of range of movement. Patients demonstrate a mild loss of forward flexion, abduction and internal and external rotation, which invariably resolves on administration of local anaesthetic as most of the loss of motion is due to synovitis rather than capsular contraction. Arthroscopy shows a hypertrophic, vascular synovitis and biopsy confirms the hypervascular nature of the synovium and normal morphological structure of the underlying capsule.

In stage 2, "the freezing stage", symptoms have been present for 3 to 9 months. Significant loss of all movements is present which is persistent even under anaesthesia. At arthroscopy, it is frequently difficult to introduce the arthroscope due to a loss in capsular volume and there is often a diffuse, pedunculated, hypervascular synovitis seen. Biopsy again shows dense, proliferative, hypervascular synovitis but additionally there is perivascular scar formation and capsular fibroplasias with deposition of disorganized collagen fibrils and a hypercellular appearance. Inflammatory infiltrates are not seen.

In stage 3, "the frozen stage", symptoms have been present from 9 to 14 months. Patients often report a previous painful phase, which has substantially settled leaving a relatively painless but stiffened glenohumeral joint. Arthroscopic appearance demonstrates patchy synovial thickening without hypervascularity and biopsy shows dense, hypercellular collagenous tissue. In stage 4, "the thawing stage", gradual improvement occurs between 15 and 24 months. There is little pain and as capsular remodelling occurs there is progressive increase in range of movement. Arthroscopic and histological correlation has not been investigated as surgery is unusual at this phase.

Other workers have reported on the pathological appearance of frozen shoulder, principally during open surgery for stage 3 disease. The macroscopic observations by Neviaser[21] described "a thickened contracted capsule peeled from the humeral head like an adhesive plaster from skin." These observations, leading him to coin the term 'adhesive capsulitis', remain true today. DePalma[18] observed that the corocohumeral ligament is converted into a tough, inelastic band of fibrous tissue spanning the interval between the corocoid process and the tuberosities of the humerus. He felt this structure is thus converted into a powerful check-rein. He therefore described division of this abnormal ligament to allow early restoration of movement.

Lundberg[10] was first to report the similarity in pathology between frozen shoulder and Dupuytren's disease, observing the prominence of fibroblast-type cells in both tissues. This link has been confirmed in a study by Bunker who identified a highly vascular, cellular, collagenous tissue as the principle lesion. They were, however, unable to show any significant inflammatory component in their histological investigations. They also commented that the synovium appeared inactive though the presence of an initial inflammatory stage cannot be excluded

as none of the specimens were taken from patients in the early stage of the disease.

Aetiology of primary frozen shoulder.

A number of mechanisms have been proposed to explain the aetiology of primary frozen shoulder. Early suggestions localising the pathology to the rotator cuff-bursa interface have been dismissed[6 7]. Most workers agree that the defining pathology is a capsular fibrosis. However, the exact cause of this fibrosis is not clearly understood. An inflammatory process has been suggested based on the histological observations of synovial or sub-synovial inflammatory reactions[10 19]. This in turn has led investigators to attempt to identify a particular inflammatory stimulus in frozen primary shoulder. Bulgen and co-workers have found increased levels of circulating immune complexes together with an elevation of serum C-reactive protein level. This is suggestive of an autoimmune type IV reaction[22]. Reduced serum IgA levels have also been noted in patients with frozen shoulder, which have persisted after recovery[23]. Unfortunately, further studies have failed to confirm this work[24]. The absence of identified autoantibodies and the lack of polyarthropathy in association with frozen shoulder makes an autoimmune basis unlikely. An infective stimulus, whether viral, bacterial or fungal, is unlikely[25] as patients do not report a prodromal illness or systemic symptoms. There is also no evidence to link frozen shoulder causally with crystal arthropathy, reactive arthropathy or haemarthrosis[25]. Bunker and co-workers[15] have recently investigated the link between frozen shoulder and fibromatoses such as Dupuytren's disease. Clonal chromosomal abnormalities have been found in many of these conditions. Bunker has detected chromosomal abnormalities in some frozen shoulder patients similar to those found in Dupuytren's disease, namely trisomy 7 and trisomy 8[8]. Both these conditions appear to be linked to each other and to a high prevalence of diabetes mellitus. In addition, these workers have found an association between elevated serum lipid levels and frozen shoulder[26].

Serum lipid levels are also elevated in patients with Dupuytren's disease[27]. These facts add further weight to the hypothesis that idiopathic frozen shoulder is a variant of the fibromatoses.

Further work by Rodeo[28] has investigated the role of cytokines in the development of Frozen shoulder. They have identified an elevation of both platelet-derived growth factor (PDGF) and transforming growth factor β (TGFβ) in the early stages of frozen shoulder. PDGF has been shown to stimulate fibroblast proliferation and TGFβ promotes extracellular matrix production leading to fibrosis. The stages of frozen shoulder, as already described, can be linked to the development of fibrosis. Hannafin and co-workers[20] have hypothesised that the initial hypervascular synovitis provokes a progressive fibroblastic response in the underlying capsule, which leads to progressive fibroplasias, thickening and contracture. These stages are modulated by the action of TGFβ and PDGF by both paracrine and autocrine abnormal upregulation.

A recent link between protease inhibitors and the genesis of frozen shoulders has also been proposed. In work by Grasland and co-workers, 8 patients with HIV were identified as developing frozen shoulder, bilateral in 4 patients, after treatment with the protease inhibitor, indinavir[29]. This may suggest a link between the inhibition of metallaproteinases and the development of abnormal fibrosis within the joint.

Abnormal biomechanics

Differences between primary frozen shoulder and post-traumatic stiff shoulder

Movement at the shoulder occurs between the two articular surfaces of the glenohumeral joint and between the scapula and thoracic wall in a ratio of 2:1[30]. Movement at the glenohumeral joint is facilitated by the mutual sliding of a set of bursal-lined surfaces, which include the deep sides of deltoid, the acromion, the corocoid process and its tendons against the proximal humerus, rotator cuff and long head of biceps. Matsen and Romeo[31] have coined the term humeroscapular motion interface (HSMI) to describe this 'articulation'. A further term, the scapulothoracic motion interface (STMI) has also been proposed to describe the bursal lined surface between the scapula blade and thoracic wall. In a glenohumeral joint with healthy articular surfaces, stiffness can occur with 3 scenarios; (1) contractures producing shortening of capsule, ligament or muscle-tendon units, (2) adhesions between gliding structures such as the cuff and biceps tendon, (3) adhesions crossing the HSMI. Clearly, the first scenario can explain primary frozen shoulder pathology, with its recognised capsular contractures. It is known that the capsule is essentially lax during motion in the mid-range[32]. The distinct ligamentous structures of the capsule only really become significantly tense at the end-range extremes of motion. Different regions of the joint capsule have specific functions in limiting particular end-range motions. It is well recognised that the inferior glenohumeral ligament functions as a check- rein for external rotation in abduction. By contrast, ligamentous structures within the rotator interval are more important in limiting external rotation in the adducted arm. Thus we can link the findings of loss of rotation in neutral with the observation of tight, contracted structures in the rotator interval.

Contractures are, however, not limited to the rotator interval. Isolated contractures of other capsulo-ligamentous structures can occur, leading to unusual patterns of motion loss [25 33]. Isolated posterior capsular contracture has been linked to abnormal gleno-humeral motion. With a significant posterior contracture, the humeral head can be translated antero-superiorly during motion, leading to compression of the rotator cuff beneath the coracoacromial arch and thus impingement[32]. In the stiff shoulder related to capsular contracture, there is therefore potential to perform limited release of tight structures. Clearly, if the procedure is to be successful, the correct structures limiting movement must be identified.

The pathomechanics of some secondary frozen shoulders or post-traumatic stiff shoulders may require different explanation. Neviaser and Neviaser[3] have identified a group of patients with stiff shoulders, occurring after trauma, who appear not to have any capsular contracture. To explain these findings, we can look to the HSMI. If adhesions secondarily to trauma or surgery cross the HSMI then movement will be limited dependent on the position and extent of the involvement. It is not known whether these patients settle with gradual improvement, as in most patients with primary frozen shoulder. Treatments aimed at the capsular structures are clearly misplaced in this group of patients and are likely to fail. In practice, however, it can be difficult to distinguish between the various groups of patients. There is almost certainly considerable overlap between the conditions and potential for one to progress to the other.

Diagnosis and Investigations

History

Primary or idiopathic frozen shoulder is diagnosed from the history and investigation. As has been stated, it is a diagnosis of exclusion after other causes of painful shoulder stiffness are precluded. As part of the assessment, an attempt should be made to define in which stage the disease is presenting. This is invaluable in informing the patient about their individual prognosis and best treatment. Codman's original description of the condition is still valid[7]. Pain, in the early stages, can be severe usually radiating to the deltoid insertion. It is worse on attempting movement of the effected shoulder but can also be present at rest, invariably interfering with sleep. The patient often notices a gradual loss of motion and in particular movements overhead and behind the back become difficult. Patients with rotator cuff pathology can also present with such complaints. This can lead to difficulties in diagnosis. Enquires should be directed at identifying any co-existent pathology, which might lead to a diagnosis of secondary frozen shoulder. Patients should be questioned about past injuries. A history of major injury or surgery is easy to define. Most patients can, however associate some minor trauma with the onset of symptoms and the ability to identify the relevance of this can be difficult. Previous surgery predisposing to frozen shoulder is not limited only to operations on the shoulder. Axillary node dissections and neck dissections are often seen to predispose to frozen shoulder particularly when associated with radiotherapy[34] [35]. Cardiac surgery and neurosurgery are also regarded as being high risk with regard to development of frozen shoulder[36] [37]. An incidence of adhesive capsulitis of 3.3% has been estimated in male post-cardiac surgery patients[38]. The link between all these stimuli may be a period of immobility associated with pain. A prospective study of frozen shoulder development in neurosurgical patients has been performed. The condition occurred in 25% of patients and was linked, in particular, with impaired

consciousness and hemiparesis. Other neurological conditions have been linked to frozen shoulder and include Parkinson's disease and Brachial neuritis. In addition, the cervical spine should always be considered. Patients with cervical spine intervertebral disc degeneration have been shown to be at greater risk of developing frozen shoulder[10]. Clearly, the importance of therapist guided rehabilitative mobilisation, in all these high-risk groups, cannot be over emphasised. Enquires should also be made with regard to diabetes and thyroid disorders. Bridgman has found an incidence of almost 11% in diabetic patients contrasted to an incidence of 2% in a non-diabetic control group[4]. Also abnormal glucose tolerance tests have been observed in 28% of patients presenting with frozen shoulder[39].

Examination

Examination should commence with an assessment of the cervical spine to exclude pathology. Both upper limbs should be properly exposed and formally examined Assessing the passive and active shoulder range of movement and defining the relative contributions from both the glenohumeral and scapulothoracic articulations is pivotal in both grading the severity of the condition and also analysing future response to treatment. Movements examined to a firm end point should include forward flexion, abduction and internal and external rotation both in neutral and abduction. It is possible to fix the scapular by holding the inferior pole of the scapular with the examiner's index finger and thumb. The examiner's other arm can then be used to assess glenohumeral motion with the scapulothoracic articulation excluded. Most researchers in this area have suggested a decrease in range of motion of about 50% as a criterion for diagnosis of frozen shoulder[9]. If motion loss is not global then the exact planes of movement should be defined. Loss of external rotation in abduction would suggest a contracture in the antero-inferior capsule whereas loss of external rotation in adduction is more indicative of a contracture in the rotator

interval. Loss of internal rotation in either adduction or abduction suggests posterior capsule contracture. Movements within the examined range of motion should be smooth and free from crepitus. As an addition, a lignocaine injection can be performed to attempt to distinguish the loss of movement associated with pain seen in impingement from the fixed decrease in movement noted in frozen shoulder. Such injections can also be useful in helping to define disease stage[20].

Investigations

No specific haematological tests are diagnostic for frozen shoulder. Routine haematological and biochemistry tests, though usually normal, should be performed. A fasting blood glucose assessment is a sensible screening test useful in identifying patients with impaired glucose tolerance presenting with shoulder stiffness[39]. It is also useful to perform an erthrocyte sedimentation rate, which may be elevated in up to 20% of patients[1].

A full series of shoulder radiographs should be obtained including true antero-posterior, scapular lateral and axillary views. These images should show, by definition, a normal joint appearance. Loss of bone density associated with disuse can be observed but a diagnosis of idiopathic frozen shoulder is excluded if any other pathology is identified. Bone and soft tissue tumours can occasionally mimic the presentation of frozen shoulder and plain radiographs are useful for their identification. Bone scan is also useful in identifying reflex sympathetic dystrophy. If there is any suspicion of the presence of unusual pathology then a bone scan should supplement the plain radiography. Diphosphonate bone scans have demonstrated increased uptake in 90% of patients with frozen shoulder[40]. Scans have also demonstrated 50% increased activity on the contralateral "unaffected" side. Bone scan activity is however of no use in predicting disease severity or prognosis. Dynamic ultrasonography has been used in frozen shoulder[41] to demonstrate a constant limitation in the sliding movement of the supraspinatus

tendon against the scapula. Magnetic resonance imaging has been used to demonstrate an increase in the thickness of the capsule in patients with frozen shoulder[42]. Neither of these modalities are useful in the routine management of frozen shoulder. They are however useful in the investigation of those patients suspected of having a shoulder frozen secondarily to some other intrinsic pathology.

Arthrography has also been used in the assessment of frozen shoulder. Neviaser pioneered the use of arthrography in the assessment of the frozen joint.[43]. The joint is typically decreased in volume to between 10 and 12 mls with obliteration of the axillary recess and subscapularis bursa. Though useful for diagnosing frozen shoulder, arthrography cannot be used to differentiate between the primary and secondary forms of the disease[40]. It is also of no use in predicting the extent and rate of recovery.

Treatment

The natural history of frozen shoulder has already been presented. Typically it is divided into 4 stages, lasting at least 18 months and often with incomplete symptom resolution. Treatment should be aimed at pain relief, improving the quality of the recovery and reducing the time taken to achieve this recovery. High-risk groups with regard to frozen shoulder include patients undergoing shoulder, arm and cardio-thoracic surgery and neurosurgery. Early mobilisation is of great importance in the prevention of shoulder stiffness symptoms. However most patients present well into the 2nd stage with an already significantly stiffened shoulder. The first priority in treating patients is to control the pain. Without good pain relief rehabilitation will be inadequate and poorly tolerated. Patients should be commenced on a non-steroidal anti-inflammatory medication provided there is no contra-indication to its use. Other analgesics can be added in

for use in patients with severe resistant pain. Opiate analgesics should be avoided as dependency is a risk with their use.

Conservative therapy

Injections

Hannafin and Chiaia have presented a rational for the use of an injection of steroid and local anaesthetic into the glenohumeral joint [44]. If after administration pain and range of motion are both improved then a diagnosis of stage 1 disease can be made. However if pain is improved but motion is unimproved then a diagnosis of stage 2 disease is made. It has been suggested that intra-articular steroid injections are more effective if administered in the early stage of the disease. Neviaser suggested that the presence of steroids can have little effect on established scar or contracture[45]. This view is supported by Hazelman[46] who has reviewed the effect of intra-articular steroids using duration of symptoms at injection as the major variable. It appears patients injected within 3 months of the start of symptoms have a significantly accelerated improvement compared with those patients injected more than 5 months after symptom commencement. If this is the case, then staging the patients is important in any study analysing outcomes following treatment with intra-articular steroids. Intra-articular steroid therapy is not benign. Detrimental effects on tendon structure and function have been reported following their use[47]. There is also a small but substantial risk of infection introduction associated with steroid injection. These factors have prompted several studies to determine their efficacy. Bulgen[48] has performed a randomised study of steroids, physiotherapy, ice and benign neglect. Initially the steroid group responded to treatment best but no significant difference was found at final long-term review. In another prospective, randomised study with observer blinding, local steroid injections were found to be as effective as physiotherapy alone or in combination[49]. Such injections were considered to accelerate recovery in the most cost-effective manner. Thus the use of steroids in the early stages of

the disease does appear to have a role in speeding recovery. However, the use of multiple steroid injections over a protracted period of time cannot be supported. It should also be noted that the delivery intra-articular injection of steroids without imaging control is at best unpredictable[50]. In our unit we routinely use ultrasound control to ensure correct positioning of injections both above and beneath the cuff.

Physiotherapy

The importance and effectiveness of physical therapies as applied to frozen shoulder has been highlighted[44]. Miller, Wirth and Rockwood have presented their review of 50 patients treated with home therapy, moist heat and anti-inflammatory medications under close supervision by a physician[51]. At review all patients regained significant ROM and returned to activities of daily living without pain. The objective of such physical therapies is to restore function by reducing inflammation and pain and thus allowing the reestablishment of normal shoulder mechanics. Historically, they have taken the form of simple repetitive stretching exercises and have been shown to effective in the vast majority of cases[52]. In the early stage, gentle pain-free mobilization using the opposite arm can be used to decrease nociceptive input to break the cycle of pain and muscle spasm. Exercises should progress to include ROM and pendulum movements to increase the pain-free range of forward elevation, external rotation, internal rotation and cross-body adduction. Wherever possible this should be home-based and self-directed after a single instructional session and occasional reviews with a physiotherapist. In the resistant case, the therapist can adopt a more active role possibly involving the use of hydrotherapy, which provides an environment for passive assisted exercises. Other modalities can also be introduced including TENS, cryotherapy and ultrasound, all of which may act to decrease pain perception. In a recent study, the effectiveness of a stretching-exercise program was investigated[53]. Sixty-four patients (90%) reported a satisfactory outcome

with only 5 patients (7%) proceeding to either MUA or arthroscopic release. Patients with more severe pain and functional limitations before treatment had relatively worse outcomes but the early use of more interventional therapies could not be supported. Most patients will have significant improvement by 12 to 16 weeks[44]. If this is not apparent or there is deterioration then consideration should be given to more interventional modalities.

Distension Arthrography

Distension arthrography or brisement is a hydrostatic technique were fluid is insufflated into the gleno-humeral joint to produce a stretching or rupture of the capsule[10]. During the procedure, incremental injections of fluid lead to a progressive increase in the intra-articular pressure to greater than 800mmHg up to a maximum of 1500mmHg[9]. Disruption of the capsule occurs at either the biceps tendon sheath or the sub coracoid bursa. Investigators have reported distension arthrography to be a reliable, safe and effective technique for treating frozen shoulder[54]. Other workers have not found such effective results. Harryman and co-workers have observed that treating a disease effecting the whole capsule by simply rupturing a small area of the anterior capsule without specifically reducing inflammation or lengthening the contracted capsule is unlikely to be effective in severe frozen shoulder[9].

Interventional Therapies

Manipulation under anaesthesia

The most difficult decision with regard to frozen shoulder management is, if and when, to progress to operative treatment, in what is regarded by some as a self-limiting condition. Neviaser and Neviaser have stated that operative intervention should be avoided while a patient is still experiencing severe pain and stiffness[3]. They believe such a circumstance is indicative of on-going inflammation and that surgery at this stage is likely to worsen the condition. In contrast to this view,

Harrymann as stated his indications to perform a gentle manipulation under anaesthesia is in the patient unable to perform exercises due to severe pain, usually following a failed intra-articular steroid and local anaesthetic injection[9]. This discordance remains and will only be resolved by future prospective studies of the management of patients with painful, stiff shoulders. In addition, Harryman suggested manipulation in patients with increasing stiffness after 12 weeks of physiotherapy or no improvement following 18-24 weeks of physiotherapy.

Contraindications to the use of closed manipulation have been summarised by Hannafin[44]. These include the patient with osteopaenia, or in the presence of fractures, neurological injury, reflex sympathetic dystrophy and instability. Manipulation is also relatively contraindicated following recent surgical repair of soft tissues around the shoulder due to the risk of disruption of the repair. The technique of closed manipulation for the treatment of frozen shoulder has been previously described by Haines and Hargadon[55]. In our unit, we perform manipulation under a general anaesthetic with a scalene block. The block is advantageous in allowing immediate physiotherapy or Continuous Passive Motion to be performed in the first few hours following manipulation. Prime importance during MUA is given to holding the humerus as near to the proximal end as possible to reduce the lever arm acting through the bone and thus limiting the risk of fracture. The scapular is stabilised with one hand cupped over the point of the shoulder. Return of motion is achieved by the use of steady, controlled force addressing first flexion, then adduction to stretch the posterior capsule, then abduction with both internal and external rotation. There is usually an audible release of soft tissues particularly with return of flexion and force should not be increased if such an audible release is absent[10].

In a review of the literature the reported results of shoulder manipulation alone or in combination with steroid injection were found to be extremely variable[9].

Those showing significant improvement at 3 months ranged from 25 –90% with a mean of 70% improved at 6 months. In a randomised study of 30 patients manipulation and steroid injection significantly improved movement and pain compared to patients treated with steroid alone[56]. In addition, Hill and Bogumill have stated their study observation that MUA is a safe means to treat frozen shoulder and significantly shorten the course of the disease. Opponents of the use of MUA in the main do not question effectiveness but are more critical of the associated damage that they feel can occur during the procedure. Provided the clinician is aware of the contraindications to the use of MUA, it can be an effective therapy for frozen shoulder not responding to physiotherapy. As has been stated, MUA has a limited role in the treatment of post-surgical shoulder stiffness.

Arthroscopic Release

As has been described[3] [43], arthroscopy has a role in the staging of frozen shoulder. The arthrosopic stages have been correlated to clinical examination and histological appearance by Hannafin[20]. The best clinical research in the area of frozen shoulder has included arthroscopy to stage the disease. This then allows the more effective comparison of data from different studies. The lack of appropriate staging of the disease in previous studies has limited the value of such comparison[44]. Arthroscopy performed prior to MUA can yield useful diagnostic information allowing identification of associated pathology such as labral or cuff tears. The treatment of some associated pathology can also be addressed through the arthroscope. Arthroscopy performed immediately after MUA has identified intra-articular haemarthrosis, avulsion of the inferior capsule adjacent to or peripheral to the labrum, tears in the rotator interval capsule with occasional labral avulsions or capsular tears anterosuperiorly or anteroinferiorly[9] [57]. Such extensive but variable damage may explain the sometimes poor results seen with MUA[58].

The best indication for the use of arthroscopy in the treatment of frozen shoulder is for the small group of patients continuing to have motion loss even after MUA. The advantages of this technique are its ability to perform precise, selective capsular releases, which can allow further MUA, if it is required, in a very controlled manner. It also has a role in the post-operative stiff shoulder lessening the risk of fracture or re-tearing of repaired soft tissues. However, patients with extensive extra-articular adhesions are probably better suited to open release. The technique, performed in either the lateral decubitus or beach chair positions, is invariably accompanied by difficulty in inserting the arthroscope into the joint. This is due to the capsular contracture and decrease in joint volume. A gentle MUA can facilitate entry, but will compromise view due to the associated, inevitable bleeding that ensues. The biceps is identified and a second working portal sited just beneath this laterally. A release is performed from the axilla of the biceps tendon to the upper border of the subscapularis tendon, thus freeing the rotator interval. This release normally reverses the loss in external rotation and can be completed by a gentle MUA. Further isolated releases of anterior or posterior capsule can be performed if more specific isolated contractures are apparent. Release of a posterior contracture, in the patient with limited cross-adduction, is a specific example of this circumstance. To complete the procedure the sub-acromial space is visualised and any further adhesions identified here are released.

Warner and co-workers have reviewed their experience with arthroscopic capsular release in 23 patients with resistant frozen shoulder[59]. The procedure was followed by 48 hours of physical therapy using an indwelling scalene block. All movements increased significantly and were within a mean of 7° of the values for the contralateral, normal shoulder. These improvements were obtained without complications. Ogilvie-Harris and co-workers have reviewed manipulation versus arthroscopic release in 40 patients with resistant frozen shoulder[58]. All patients

had experienced symptoms and functional loss despite 1 year of non-operative treatment. In the first 20 patients, MUA was performed in association with arthroscopy. In the second 20 patients, arthroscopic division of contracted structures was performed. At independent assessment, between 2 and 5 years follow-up there was no difference in ROM but there was significantly better pain relief and restoration of function in the arthroscopically released group. These co-workers have observed that following 1year of adequate non-operative treatment, in the patients still having significant stiffness, arthroscopic release offers the best chance of an excellent long-term result. They also commented that this is especially so in patients with diabetes mellitus. This has been our experience with regard to our own practice. Clearly in some circumstances, a simple MUA appears overly aggressive, leading to disruption of normal as well as pathological tissue. The ultimate result is thus less than optimal. Some patients have an excellent result following MUA. The answer to the question, 'which patients can be treated simply with an MUA and which patients require an arthroscopic release?' remains unclear.

Open Release

As with arthroscopic release, the objective with open release to free up any adhesions present. It gives the surgeon the ability to easily free up both intra- and extra-articular structures. It is especially useful in treating patients after unsuccessful arthroscopic release and after previous surgical repairs where manipulation might lead to disruption. It is particularly useful in situations where adhesions are predominantly in the HSMI and is thus often indicated in the post-traumatic surgical stiff shoulder. Unfortunately, open release is associated with increased post-operative pain when compared with either MUA or arthroscopic release and this can inhibit early mobilisation. The procedure is normally performed through a delto-pectoral approach. Structures released usually include subacromial and subdeltoid bursal adhesions, the corocohumeral ligament and

the rotator interval[18 60] although a complete perilabral capsular release around some or all of the glenoid can be performed[58]. Ozaki and co-workers have used open release to treat 17 patients with resistant frozen shoulder. They identified the major tethering structure as a contracture of the corocohumeral ligament and release of this restored motion in all patients. Thus Open release can be recommended as an effective therapy in the most resistant forms of frozen shoulder. Such cases are fortunately rare. Open release is also very valuable in the management of post-surgical shoulder stiffness not responding to simple physiotherapy.

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