Time for a new name for frozen shoulder – contracture of the shoulder

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2004 was the 70th anniversary of the introduction by Codman of the term "frozen shoulder". Perhaps, as Roy and colleagues (Roy et al 1982) have suggested, "the term frozen shoulder, which for too long has encouraged doctors to do as little as possible about this common and distressing condition, should be abolished". Their view was shared by Neviaser, who argued that "the misnomer 'frozen shoulder' should be deleted from the medical literature". (Neviaser 1980). For this reason frozen shoulder is termed adhesive capsulitis in the United States of America, although it turns out to have no adhesive and, as shall be seen, it turns out to be a capsular contracture rather than a capsulitis. The Germans call it Steiffschulter, which is honest, but perhaps too basic, and the French "capsulite retractile", although, in fact, the capsule turns out to be contracted rather than retracted. Over the last twenty years a large body of research has been performed in order to understand and treat this common, enigmatic, protracted, painful and disabling condition. Perhaps it is now time to reflect on the progress that has been made over the last two decades into understanding and successfully treating this disease.

Firstly we can define this disease according to its clinical profile, demographics, clinical course, associations, arthroscopic and surgical appearance, microscopic and electron microscopic appearance, molecular cell messenger profile, and finally its genetic basis. Building on this evidence we can argue why it should be treated, ask what the patient wants of treatment and see if evidence exists for rapid, effective and long lasting treatment. Finally we can appeal for a new name for this common, painful and protracted condition and call it what it is, capsular contracture.

Codman coined the term frozen shoulder in 1934. He said it was difficult to define, but then went on to propose a definition which has not been bettered in 70 years. He stated 'this is a condition which comes on slowly with pain over the deltoid insertion, inability to sleep, painful incomplete elevation and external rotation, the restriction of movement being both active and passive, with a normal radiograph, the pain being very trying and yet all patients are able to continue their daily habits and routines'. The problem is that many of these features are shared by other common shoulder conditions such as rotator cuff disease. However there is one clinical feature that distinguishes capsular contracture from all other conditions, restriction of passive external rotation in the face of a normal radiograph. There are only two pathologies that cause limited passive external rotation; firstly damage to the joint surface such as occurs in arthritis, head splitting fractures and locked dislocation (all of which have an abnormal radiograph); and secondly contracture of the ligaments. Since the radiograph is normal this condition can only be due to contracture of the ligaments, quod est demonstratum.

There are many myths about this condition which are handed down from paper to paper without examination of the evidence. A recent metanalysis (Carr) quotes 25 papers on frozen shoulder that studied 935 patients of whom 58% were female. However many of the studies in this metanalysis showed a ratio of 1;1 male;female. The condition is also less common than the usually quoted figure of 2% of the population. This figure was arrived at forty years ago (Hazelman) when shoulder disease was ill understood and frozen shoulder was used as a wastebin diagnosis for any stiff and painful shoulder. When Hazelman performed arthrograms of 36 patients he had diagnosed as frozen shoulder, eleven were found to have complete tears of the rotator cuff. The early arthroscopic studies (Bayley) showed that only 50% of patients diagnosed as having frozen shoulder actually had visual/tactile evidence of the disease. This authors repeated studies on capsular contracture show that it only accounts for 5% of shoulder disease, and since shoulder disease only affects 15% of the population then it would be reasonable to suggest that the real incidence of capsular contracture is about 0.75% of the population. So frozen shoulder is an overused as well as misused term.

Codman stated "even the most protracted cases recover with or without treatment in about two years". Once again this statement has been handed down from author to author without any questioning of the evidence. This has led to the commonly held and false view that this is a benign condition that resolves completely. Many eminent surgeons who have researched this disease have pleaded that complete resolution is not inevitable, but their pleas have fallen upon deaf ears. Simmonds stated 'complete recovery is not my experience' and DePalma stated' it is erroneous to believe that in all instances restoration of function is attained'. Shaffer et al (1992) in the most detailed follow up study in the literature found that at seven years 50% had mild pain, stiffness or both. They found that 60% has measurable restriction of passive mobility and they concluded 'this made us question whether this is a benign self resolving condition'. These findings were confirmed by Griggs et al who stated that 'even amongst the patients who were satisfied, a substantial number were not pain free'. 10% had mild pain at rest, and 27% had mild or moderate pain with activity. 40% of the satisfied patients had abnormal shoulder function. Our own studies at 2-5 years showed that although 86% had an improvement in their level of pain, this did not mean that they had no pain. Only 53% had no pain, 33% had an occasional pain and 14% had marked residual pain. These findings have been confirmed by the largest ever study from Oxford (Hand &Carr) on 273 patients followed for up to 20 years. Using the Oxford Shoulder Score they demonstrated that 40% of their patients had mild to moderate persistent symptoms at seven years.

Now let us examine the evidence starting with the cornerstone of evidence based medicine, pathology. The arthroscopic appearance is specific and spectacular. Firstly there is evidence of capsular contracture with a reduced joint volume. The capsule is thickened and difficult to penetrate with the arthroscope and the joint difficult to navigate due to capsular contracture. The spectacular feature is angiogenesis. This is most marked in the rotator interval area. As the disease progresses into the stiff and contracted phase the angiogenesis declines and thick white scar can be seen and palpated within the capsule. The early studies of the pathology in capsular contracture of the shoulder showed dense collagen causing fibrosis and marked vascularity in the capsule. Lundberg made the first link between this condition and the other contractile diseases such as Dupuytren's disease, for he noted that the histology of the shoulder capsule showed compact or dense collagen with many cells that were fibroblasts. Hannafin et al took biopsies at arthroscopy and showed that in the first phase there was a hypervascular appearance of the synovium with underlying normal capsular architecture; in stage two there was perivascular scar formation and extensive scar formation in the underlying capsule and in the final phase extensive capsular fibroplasias. This was confirmed (Bunker & Anthony) in 1995 where, using immunocytochemistry, the cells were shown to be mainly fibroblasts with some transformation to the contractile myofibroblast. These authors demonstrated a mass of type III collagen laid down in bands and nodules looking, once again, very similar to the histology of Dupuytren's disease. Further confirmation has come from Killian et al (2001) who showed a significant increase in fibroblasts, and under electron microscopy a loss of collagen fibril order and four-fold increase in fibre diameter. In summary all the histological evidence to date shows that this is a capsular contracture of the shoulder.

The question then arises as to why this should happen? What precipitates the angiogenesis, and what orders the fibroblasts to accumulate and lay down colla-

gen? Cell messengers (cytokines and growth factors) control these cellular responses and several groups have looked at cell messengers in capsular contracture. Hamada, in Japan, has found increased levels of vascular endothelial growth factor (VEGF) in stiff shoulders that may account for the angiogenesis that is seen at arthroscopy. Rodeo et al found raised levels of TGF beta and PDGF and suggested that these may act as a perpetual stimulus to fibrosis. Bunker et al found elevated levels of fibrogenic growth factors (FGF) and this work has been elegantly confirmed by Colville's group who took joint fluid from patients with capsular contracture and found that this tissue caused a 5000% increase in in-vitro fibroblast proliferation compared with control groups

On the cellular level one may question why the scar that is laid down is not quickly remodelled as in normal healing? Bunker et al looked at the question of remodelling that is mediated by MMPs (Matrix metalloproteinases). They found an absence of MMP 14 and an elevation of the MMP inhibitor TIMP. Hutchinson et al actually treated patients with end stage gastric cancer with Marimastat, a synthetic TIMP and found that within four months half their patients developed stiff shoulders and a quarter developed Dupuytren's disease. When the Marimastat was stopped the disease regressed.

There has been very little work done on the genetics of capsular contracture. Family studies are difficult to do on conditions where two thirds of those affected return to normal. Bunker et al found that patients with capsular contracture had a normal karyotype, that is they were 46xy if male and 46xx if female, but the cells from the shoulder capsule showed some clonal chromosomal changes. These changes were numerical trisomy of chromosomes 7 and 8, a similar finding to that in other studies of Dupuytren's disease.

Three questions now need to be asked and answered. Firstly do patients want to be treated? Secondly want do they desire of treatment? Finally can we deliver that?

Do patients want to be treated? They certainly do. The pain of capsular contracture (frozen shoulder) is severe, night pain is worse, night awakening is universal, sleep deprivation is constant, and these symptoms persist for months and months on end. Many doctors say that there is no point treating frozen shoulder for it gets better in 18 months to two years. This is patronizing in the extreme. How would you react to a being told that your severe pain and night awakening was not worth treating for it would get better in two years? This is akin to a woman in labour being told that there was no need for pain relief because the pain would go once the baby was out!

What do patients desire of treatment? They want the pain to go. They want the pain to go NOW. If not now they want the pain to go AS SOON AS POSSIBLE. They want to be able to sleep. They want to be able to sleep tonight, please. And it would be a bonus if their movement could return, at least to a functional level.

Finally do we have a treatment that can deliver immediate and long lasting freedom from pain, return of a normal sleep pattern, and a functional range of movement? The short answer is yes, not for everyone, not always immediately, but for the majority arthroscopic release can deliver this package. Before discussing arthroscopic release we should examine the evidence behind other forms of treatment.

Steroids have been shown in four randomised prospective controlled studies to have no benefit over home exercises. However all four papers can be severely criticised as they studied painful stiff shoulders, in other words primary and secondary frozen shoulder so many of the patients would have had other shoulder disease. The best of these papers from Hazelman's unit did arthrograms of the study group and 11 of 36 had cuff tears, yet were kept in the study! This is a recurring criticism of so many papers on capsular contracture, the diagnosis is wrong.

The best paper on physiotherapy is that of Diercks et al that showed that intensive physiotherapy prolonged the natural history of the disease from 15 months to 24 months and achieved a lower Constant Score of 76 compared to 87 in the control group who did home exercises. Once again we must stress that what the patients want is not for their disease to be prolonged from 15 to 24 months, but for it to end TODAY.

Manipulation under anaesthetic has been the mainstay of surgical treatment for decades. There are many studies showing good and relatively rapid improvement. Sneppen et al showed that 75% of their patients attained a near normal range of movement, 79% were relieved of pain and 75% returned to work within 9 weeks.

Arthroscopic release, in the hands of the expert shoulder surgeon, has transformed the management of capsular contracture. Many of the studies can be criticised for purporting to show the results of treating capsular contracture when the index group was

actually made up of any stiff shoulder including fractures, cuff disease and post-surgical stiff shoulders and then pooling the results. For instance one paper started with 1720 stiff shoulders of which only 11 had an arthroscopic release for primary adhesive capsulitis. Four articles are worthy of study. Ogilvie Harris et al (1995) compared the results of manipulation versus arthroscopic release in their hands. Although both groups gained the same substantial improvement in range of motion the arthroscopic group had significantly better pain relief and function, to the extent that twice as many were graded excellent. The following year J.P.Warner (1996) showed a 49º increase in elevation, 42º increase in external rotation and improvement in Constant Scores from 13 to 77/100. Harryman and Matsen published a year later (1997) and demonstrated fantastic results. The range of motion went from 41% of the opposite side to 78% on the first postoperative day and 93% at the end of the study. Before surgery 6% could sleep and after 73%. They were the first to show the dramatic speed of recovery following treatment, which is the very thing that patients want. Berghs et al (2004) confirmed this with a dramatic improvement on day one post surgery in 36% and 88% improvement within 2 weeks. Pain improved from 3.6/15 to 12.6/15 and the partial Constant Scores from 20/75 to 62/75. There were no complications in three of these studies, but one transient axillary neurapraxia in the Harryman study. Arthroscopic release appears to show great promise for it delivers what the patient wants; relief of pain, undisturbed nights and improved function TODAY, or if not today THIS WEEK, in the majority of people, with minimally invasive, keyhole day-case surgery.

Over the last twenty years we have come a lot further in our understanding of this disease. The clinical findings are of a restriction of passive mobility of the shoulder brought on by capsular contracture. At arthroscopic and open surgery the capsule can be seen and felt to be thickened and contracted. Histology shows the capsule to be thickened and contracted. The cells present are fibroblasts that lay down scar, and myofibroblasts that contract scar. Cell messengers that lead to scarring are present, and the inhibitor of remodelling MMPs is strongly present. Giving this inhibitor to humans causes a capsular contracture and in some also leads to Dupuytren's contracture in the hand. The evidence is conclusive, frozen shoulder is a capsular contracture. Twenty years of research have brought an understanding of the cause or causes of this common, disabling, protracted, and painful condition so that we can now offer our patients a benign day-case effective and evidence based method of treatment. Perhaps the day has also come for a new name for frozen shoulder. Let us call it what it is, contracture of the shoulder.