Adhesive Capsulitis: A Proposed Clinical Staging Scheme with Histological Correlation

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ABSTRACT

Background: The literature on the treatment and outcome of adhesive capsulitis is confusing. This is due in part to inconsistencies in determination of the clinical and pathologic stage once the diagnosis is made. Optimal treatment of adhesive capsulitis should be based upon proper staging of the disease. The purpose of the present study is to define the pathologic stages of adhesive capsulitis and to correlate these with clinical and surgical findings to produce a working clinical staging scheme.

Methods: Patients with a clinical diagnosis of primary adhesive capsulitis from 1991 to 2000 without other confounding disease process were retrospectively evaluated. Patients were included who had a pathologic specimen of the capsule and synovium available for review. A chart review was performed to document the clinical stage and a review of biopsy specimens was performed to determine the pathologic stage. The surgeons and pathologist were not aware of the prior histologic or clinical stage during their respective staging. There were 34 females and 16 males with an average age of 50 $\pm$ 8 years.

Results*:* The histologic stages of adhesive capsulitis specimens were described and correlated to the clinical stage of each patient treated. Clinical and histological stages were identical in 47 out of 50 cases: four, stage 1; forty-one, stage 2; two, stage 3. There were two cases in which the clinical and histological stage did not match. These were both stage 2 on histologic analysis and stage 3 on clinical exam. One additional patient biopsy demonstrated a normal histological reading but was clinically diagnosed as stage 2. The data were analyzed using a Spearman's rank correlation. A strong correlation was found between clinical and histological diagnosis of the stage of adhesive capsulitis ($rho = 0.8$, $p < 0.0001$).

Conclusions: We determined that using our clinical, arthroscopic, and histologic descriptions, determination of stage with one method highly predicts the other. When treating adhesive capsulitis, it is important not only to correctly diagnose adhesive capsulitis, but also to correctly stage it. This information will facilitate the staging of adhesive capsulitis, which may lead to more consistent treatment modalities, more effective outcomes, and better consistency within the literature.

Clinical Relevance:

This correlation between the histologic features of adhesive capsulitis and a proposed clinical stage of the condition serves as a basis for a rational approach to the medical and surgical treatment of adhesive capsulitis.

INTRODUCTION

Idiopathic adhesive capsulitis is a condition of unknown etiology characterized by gradual loss of both active and passive glenohumeral motion. Factors associated with adhesive capsulitis include female gender [1], age older than 40 [2], trauma [2], diabetes [3, 4], prolonged immobilization, thyroid disease [5], stroke or myocardial infarction [4], and the presence of autoimmune diseases [6].

As for any other disease process, appropriate treatment should be based upon the pathogenesis and natural history of the disease. For adhesive capsulitis, both remain controversial. Miller and Rockwood [4] found that the majority of their patients (n=50) regained motion with decreased pain after treatment with anti-inflammatory medications, moist heat, and physician-directed rehabilitation. In contrast, Shaffer et al [7] found that 50% of his patients (n=62) treated conservatively had residual stiffness at 7-year follow-up.

There is still disagreement in the literature whether the underlying pathologic process is inflammatory [8, 9] or fibrotic in nature [10]. Recent work by Rodeo et al [11] confirmed that adhesive capsulitis involves both synovial hyperplasia and capsular fibrosis and that cytokines such as transforming growth factor-β and platelet-derived growth factor may be involved in these processes. In addition, matrix bound transforming growth factor-β may act as a persistent stimulus resulting in capsular fibrosis. Therefore, adhesive capsulitis is both an inflammatory and fibrotic condition depending on the stage of the disease [12, 13].

Neviaser and Neviaser [14] described the arthroscopic stages of adhesive capsulitis stressing the importance of an individualized treatment plan based on an understanding of the clinical stages of the disease. In 1994, Hannafin [13] hypothesized a correlation between the arthroscopic stage, the clinical exam, and the histologic appearance of capsular biopsy specimens in a small series of patients with stage 1, 2, and 3 adhesive capsulitis [13]. This was the first report

describing the histologic stages of adhesive capsulitis and the first description of the capsular fibroplasia as found in the latter stages of adhesive capsulitis. The purpose of this paper is to expand upon this initial hypothesis that a correlation exists between the clinical and histological stage of adhesive capsulitis.

MATERIALS AND METHODS

The Institutional Review Board at the Hospital for Special Surgery approved the study protocol. The first 15 patients were those previously diagnosed with adhesive capsulitis using clinical, arthroscopic, and histologic criteria by the primary author (JH) from 1991 to 1994. The remaining patients were identified by searching the Department of Pathology database for patients with a clinical *or* histological diagnosis of adhesive capsulitis. A search performed from 1995 through 2000 identified 215 patients who had been given the diagnosis of adhesive capsulitis clinically *or* histologically. These specimens were reviewed with our Pathologist (ED) for definitive characteristics of adhesive capsulitis. Any specimen which had confounding features of trauma or degenerative joint disease was excluded. Any patient who had a concomitant repair procedure (rotator cuff, labral repair) was excluded. These 35 patients were felt to represent idiopathic adhesive capsulitis. No patients with secondary adhesive capsulitis related to arthritis, labral or rotator cuff injury were evaluated in this study.

The pathologist (ED) and both orthopedic surgeons (JH, DF) described and staged the 50 histologic specimens according to the initial criteria proposed [13] without knowledge of the clinical status of the patients. Chart review was then performed to identify the clinical stage, which was determined by noting the exam-under-anesthesia (EUA) and arthroscopic characteristics (photos and descriptions). Clinical staging was defined according to the following criteria:

Stage	Duration of Symptoms (months)	ROM after injection or EUA	Histology	Arthroscopic Picture
1	0-3	Full	Hyperplasia of lining, chronic inflammation, hypervascularity	Hypertrophic vascular synovitis
2	3-9	Partial	Increase in inflammatory cells (lymphocytes); progressive fibrosis	Dense, proliferative hypervascular synovitis
3	9-14	No improvement	No inflammation; well organized, collagenous tissue	Residual filmy synovial layer with thickened capsule; no hypervascularity

Stage 1: EUA demonstrates normal motion (in contrast to pre-operative exam) and arthroscopic exam reveals hypertrophic vascular synovitis coating the entire capsular lining (Figure 1A).

Stage 2: Partial recovery of range-of-motion (ROM) is obtained when comparing EUA and pre-operative exam. Arthroscopic examination reveals a dense, proliferative hypervascular synovitis (Figure 1B).

Stage 3: ROM is unchanged when EUA is compared with pre-operative exam. Arthroscopic examination shows a residual filmy synovial layer with or without patches of synovial thickening without hypervascularity (Figure 1C).

In addition to the clinical stage, the patient's age, surgeon, and procedure performed were recorded. The procedures included manipulation under anesthesia (MUA), capsular release (CR), synovectomy, subacromial decompression, and debridement. There were 34 females and 16 males with an average age of 50 $\pm$ 8 years. The patients were treated by six orthopaedic surgeons.

RESULTS

Within the continuum of the proposed clinical stages and despite the absence of well defined, discrete boundaries, consistencies in the histologic characteristics made it possible to allow staging of each specimen[12] based on the presence, degree, and extent of changes in inflammation, vascularity, and capsular sclerosis or "fibroplasia".

There were four ***Stage 1*** cases. In this stage, the synovial features included hyperplasia of the lining layer, usually a slight degree of chronic inflammation (Figure 2A), and hypervascularity of small, delicate blood vessels in the superficial region (Figure 3A). Fibrosis was not generally a feature and the deeper tissues appeared normal. In three out of four stage 1 specimens (75%) inflammatory infiltrates were identified.

There were 43 ***Stage 2*** cases. In this stage, the inflammatory changes showed an increase in intensity compared to the stage 1 cases reaching a focally prominent degree of inflammation extending into the perivascular tissues (Figure 2B) and trailing off to a slight degree of inflammation in the later cases. In all cases, the inflammation was almost exclusively composed of small, round lymphocytes (Figure 2C).

Changes affecting the blood vessels also showed gradual changes related to the degree of perivascular fibrosis, which ranged from slight (Figure 3B) to dense (Figures 3C and 3D). As

seen in these images, as the degree of fibrosis increased, the vessels at first appeared to be held open and then to be constricted toward closure. The endothelial cells in the constricted vessels bulged into the lumen producing a hob-nail appearance.

Also in this stage, changes in the capsular tissue became apparent and went through a similar degree of transformation as did the blood vessels. In the early cases the capsular tissue showed increasing *in situ* fibrosis of a disorganized nature with at first, a low degree of cellularity (Figure 4A). In the intermediate stages, this fibroplasia became more intense and the cellularity more dense (Figure 4B). The cytologic atypia present in these fibroblasts was characterized by increased variations in size, shape and nuclear staining intensity, imparting a pseudosarcomatous and fibromatosis-like appearance to the tissue. Following this, it appeared that the cellularity decreased and the fibrosis remained dense, but somewhat less disorganized (Figure 4C).

There were two ***Stage 3*** cases. A small amount of pliable and loose synovium was present in these cases, inflammation was not a feature. The vessels were embedded in dense connective tissue, causing obliteration of some of the lumens (Figure 3E). The capsular tissue shows much denser collagenous tissue with fewer cells present within fairly well organized, densely packed collagen fibers that had a delicately crimped appearance in polarized light (Figures 4D and 4E). This last feature imparted a fibromatosis-like appearance to the tissue,

The clinical staging and histologic staging were then compared. The clinical and histologic stage matched in 47 out of 50 cases: four, stage 1; forty-one, stage 2; and two, stage 3. In three cases the clinical and histologic stages did not correlate. In two cases, a histologic stage 2 and a clinical stage 3 were noted. In one patient, a clinical diagnosis of stage 2 with a biopsy revealing normal shoulder capsule was present. The data was analyzed using a Spearman's rank

correlation. A strong correlation was found between diagnosing the stage of adhesive capsulitis using clinical exam and histological criteria (rho = 0.8, $p < 0.0001$).

DISCUSSION

The literature on the treatment and outcome of adhesive capsulitis is confusing. This is due in part to inconsistencies in staging and confusion in the literature as to whether adhesive capsulitis is an inflammatory or sclerosing condition. If it can be assumed that optimal treatment of adhesive capsulitis is based upon proper staging of the disease, then treatments deemed effective for stage 1 and early stage 2 disease, in which inflammation is present, are unlikely to be successful for later stages of adhesive capsulitis given the progression from inflammatory synovitis to capsular fibrosis. We have described the histological stages in detail and have shown that they correlate to the clinical stage of the disease. Thus, the stage can be diagnosed with higher accuracy which may lead to more consistent treatment, more effective outcomes, and better consistency within the literature.

There were three cases in which the histologic stage did not match the clinical stage. Two were late stage 2 by histology and stage 3 clinically. Since the disease process is a continuum spreading from the rotator interval superiorly and inferiorly, it may be the representative biopsy captured a portion of the capsule earlier in the disease process. Another possibility is that physician was biased by the lack of pain reported by the patient. This could be a distinguishing factor between a late “active” stage 2 and a early stage 3 disease process. The real outlier is the clinical stage 2 which had “normal” histology. However, it is possible that in an early stage 2, a portion of the “normal” synovium existed and was biopsied.

This is the first published study describing the stage specific histologic characteristics of adhesive capsulitis and the first to show a statistically significant correlation between clinical and histologic stage in patients with adhesive capsulitis.

The limitations of our study are those inherent in retrospective chart review. In addition, it would have been helpful to include clinical duration of symptoms; however, this data was precisely available for only a third of the patients.

As mentioned, the treatment protocols in the literature can be confusing since many treat the diagnosis in general without regard to stage. Treatment options documented in the literature include: benign neglect [4], supervised physical therapy [1, 15, 16], nonsteroidal anti-inflammatory medications [17], oral corticosteroids [18], intraarticular injections [15, 19, 20], distention arthrography [21], closed manipulation [22-25], open surgical release [26], and arthroscopic capsular release [25, 27, 28].

Over many years of observation, the senior author (JH) has developed a protocol for clinical identification of the stage of adhesive capsulitis and a stage based approach to treatment.

In *Stage 1*, symptoms have been present for less than 3 months. Patients will present with pain and loss of motion. After injection with local anesthetic or with examination under anesthesia (EUA), range of motion (ROM) approaches normal limits. This improvement is assumed to result from the removal of pain due to inflammation without the structural limitations imposed by fibrosis as suggested by the presence of inflammation and the lack of fibrosis apparent in the histologic samples.

The treatment of the synovitis found in *Stage 1* is treated with an intraarticular steroid injection (80mg depomedrol, 5cc 1% lidocaine, and 3cc 0.25% marcaine). The patient is reexamined in two weeks and if rest and night pain have not resolved a second injection may be

given. A home stretching and strengthening program is begun. The patient is followed until resolution of symptoms.

Binder et al found that patients undergoing PT mobilization only in the first six weeks had greater restriction of motion than those receiving steroid injections, ice, or no specific therapy[1].

In *stage 2* (the "freezing" stage) symptoms are present for 3 to 9 months with progressive loss of motion, rest and night pain. After injection with local anesthetic or with examination under anesthesia, the pain is relieved but only partial ROM is obtained, reflecting the capsular contracture in response to the painful synovitis. Again, this combination of clinical observations is corroborated by the findings of reducing inflammation and increasing fibrosis around the blood vessels and involving the capsular tissue as the clinical status of the disease progresses through time and severity.

In *Stage 2*, both the synovitis and capsular contracture must be addressed for successful treatment. An injection is administered as above and home or supervised therapy is begun to restore normal glenohumeral and scapulothoracic kinematics and strength of rotator cuff and scapular stabilizers. The patient is reexamined at 2 weeks to assure significant relief of pain (increased ROM may not be discernable at this point) and then every 6 weeks to monitor recovery of ROM. If adequate pain relief or recovery of motion is not satisfactory after 3 to 6 months of conservative treatment, capsular release and manipulation under anesthesia can be considered.

Bulgen et al evaluated 42 patients in stage 1 and 2 (painful shoulders) and found that steroid injections initiated in stage 1 (before 3 months) versus stage 2 (after 3 months) produced

similar end results. In addition, the severity of restriction gave no indication of the final outcome[15].

In *stage 3* (the "frozen" stage), the symptoms are present for 9-14 months, and have changed over time. The shoulder is pain free at rest but increasingly stiff with pain at end range of motion. To the extent that inflammation may be the cause of pain at rest, the absence of inflammation and the presence of the dense capsular sclerosis in the tissues of these patients could very well explain the lack of rest-pain and the general stiffness of the joint. Since the inflammatory component appears to have resolved, steroid injection to treat patients in this stage of the disease is not warranted. Patients can be treated with home exercises and physical therapy to restore capsular mobility. Arthroscopic capsular release with manipulation under anesthesia is our treatment of choice if the patient is not responding to conservative therapy.

Stage 4 (the "thawing" stage) is characterized by the slow, steady recovery of ROM resulting from capsular remodeling in response to movement. No arthroscopic or histologic data are available for these patients as they rarely undergo surgery.

This self-limiting process typically takes approximately two years to complete without intervention. We hypothesize that the natural history of adhesive capsulitis can be explained by the histologic findings beginning with the inflammation. Release of cytokines would appear to initiate the process of synovial hyperplasia and is likely responsible for the associated pain (stage 1) which, in turn, evoke the fibrotic response seen in stage 2 [11]. Collagen deposition begins in the perivascular tissue in the superficial synovium, possibly related to high cytokine concentration. Scarring progressively extends into the subsynovium and capsule. As the perivascular scarring progresses during stage 2, the vessels, which are patent early in stage 2, begin to decrease in internal diameter later in stage 2, due to the contraction of perivascular scar.

The vascular constriction may then interfere with the flow of cytokines locally and more globally in the synovium. Involution of the vessels may provide a plausible explanation for the self-limited nature of this condition. With resolution of the synovitis and disappearance of the cytokine stimulus to the capsular cells, the capsular fibroplasia becomes less active and the collagenous tissue remodels according to the improved ROM allowing motion to return toward normal.

The histologic observation that inflammation is present in the clinically early stages of adhesive capsulitis gives support for the belief that the condition is essentially and initially an inflammatory one. The subsequent sclerosis (progressing from mild through increasingly active fibroblastic behavior ("fibroplasia") involving the blood vessels and the capsular tissue) supports the observations that cytokines are functional and explains why loss of motion eventually becomes the clinically significant feature of adhesive capsulitis. Finally, in the latest clinical stages of the disease, the absence of inflammation and the progressive reorganization of the collagenous matrix support the general observation of slow improvement in the clinical state. The histologic features therefore support a classification scheme that is based on the theory of initial inflammation followed by fibrosis and slow resolution over time. Likewise the histologic findings support treatment regimens that emphasize targeting inflammation early and addressing structural considerations later in the disease.

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LEGENDS TO FIGURES

Figure 1A: This arthroscopic photograph of a case of Stage 1 disease shows the diffuse glenohumeral synovitis, which usually is most pronounced at the anterior-superior capsule.

Figure 1B: This arthroscopic photograph of a case of Stage 2 disease shows a diffuse, pedunculated synovitis ("Christmas tree synovitis").

Figure 1C: This arthroscopic photograph of a case of Stage 3 disease shows a residual filmy synovial layer with patches of synovial thickening without hypervascularity.

Figure 2A: This photomicrograph of a case of Stage 1 disease shows hyperplasia of the lining layer with sparse inflammation around the superficial blood vessels without fibrosis of the intervening tissue. (x25 magnification)

Figure 2B: This photomicrograph of a case of early Stage 2 disease shows more diffuse perivascular inflammation extending into the intervening tissue. (x20 magnification)

Figure 2C: This photomicrograph of a case of early Stage 2 disease shows the inflammation to be composed of small round lymphocytes. (x40 magnification)

Figure 3A: This photomicrograph of a case of Stage 1 disease shows the hyperplasia of the small, delicate blood vessels in the superficial synovium not accompanied by increased fibrosis. (x25 magnification)

Figure 3B: This photomicrograph of a case of early Stage 2 disease shows the vascular hyperplasia with slightly increased perivascular fibrosis that appears to "hold" some vessels open while causing "constriction" of others. Note that the intervening

tissue does not have increased fibrosis and that the endothelial cells in constricted vessels have a hob-nail appearance. (x25 magnification)

Figure 3C: This photomicrograph of an established case of Stage 2 disease shows more extensive fibrosis with constriction of the vessels and the same endothelial appearance of the endothelial cell as seen in some of the vessels in figure 3B. (x20 magnification)

Figure 3D: This photomicrograph in polarized light of the same field shown in Figure 3C shows the presence of increased connective tissue around and between the blood vessels. (x20 magnification)

Figure 3E: This photomicrograph of a case of late Stage 2 / Stage 3 disease shows dense perivascular fibrosis of the superficial synovium. (x25 magnification)

Figure 4A: This photomicrograph of a case of Stage 2 disease shows disorganized fibroplasia of the capsular tissue with a low level of cellularity, but with some degree of cytologic atypia characterized by the variation in nuclear profiles apparent even at this magnification. (x10 magnification)

Figure 4B: This photomicrograph of a case of Stage 2 disease shows intense fibroplasia present as dense fibrosis, increased cellularity, and a moderate degree of cytologic atypia of the fibroblasts (variation in size, shape and staining intensity) resulting in a pseudosarcomatous, fibromatosis-like appearance. (x25 magnification)

Figure 4C: This photomicrograph of a case of late Stage 2 disease shows more orderly arrangement of the collagen with a lesser degree of cellularity compared to earlier cases in Stage 2. (x25 magnification)

Figure 4D: This photomicrograph of a case of Stage 3 disease shows dense fibrosis with fewer cells arranged with a greater degree of organization. (x25 magnification)

Figure 4E: This photomicrograph in polarized light of the same field shown in Figure 4E shows the crimped nature of the fairly well organized. densely packed collagen fibers, imparting more of a fibromatosis-like appearance. (x20 magnification)

REFERENCES

1. **Binder AI, Bulgen DY, Hazleman BL, Roberts S. Frozen shoulder: a long-term prospective study. Ann Rheum Dis 1984;43(3):361-4.**
2. **LLoyd-Roberts G, French P. Periarthritis of the shoulder: a study of the disease and its treatment. Br Med J 1959;1:1569-74.**
3. **Bridgman JF. Periarthritis of the shoulder and diabetes mellitus. Ann Rheum Dis 1972;31(1):69-71.**
4. **Miller MD, Wirth MA, Rockwood CA, Jr. Thawing the frozen shoulder: the "patient" patient. Orthopedics 1996;19(10):849-53.**
5. **Bowman CA, Jeffcoate WJ, Pattrick M, Doherty M. Bilateral adhesive capsulitis, oligoarthritis and proximal myopathy as presentation of hypothyroidism. Br J Rheumatol 1988;27(1):62-4.**
6. **Bulgen DY, Binder A, Hazleman BL, Park JR. Immunological studies in frozen shoulder. J Rheumatol 1982;9(6):893-8.**
7. **Shaffer B, Tibone JE, Kerlan RK. Frozen shoulder. A long-term follow-up. J Bone Joint Surg Am 1992;74(5):738-46.**
8. **Wiley AM. Arthroscopic appearance of frozen shoulder. Arthroscopy 1991;7(2):138-43.**
9. **Neviaser TJ. Arthroscopy of the shoulder. Orthop Clin North Am 1987;18(3):361-72.**
10. **Bunker TD, Reilly J, Baird KS, Hamblen DL. Expression of growth factors, cytokines and matrix metalloproteinases in frozen shoulder. J Bone Joint Surg Br 2000;82(5):768-73.**

11. Rodeo SA, Hannafin JA, Tom J, Warren RF, Wickiewicz TL. Immunolocalization of cytokines and their receptors in adhesive capsulitis of the shoulder. J Orthop Res 1997;15(3):427-36.

12. Hannafin JA, Chiaia TA. Adhesive capsulitis. A treatment approach. Clin Orthop 2000(372):95-109.

13. Hannafin JA, E.F. D, Wickiewicz TL, Warren RF. Adhesive Capsulitis: Capsular fibroplasia of the glenohumeral joint. J Shoulder Elbow Surg 1994;3(Suppl):5.

14. Neviaser RJ, Neviaser TJ. The frozen shoulder. Diagnosis and management. Clin Orthop 1987(223):59-64.

15. Bulgen DY, Binder AI, Hazleman BL, Dutton J, Roberts S. Frozen shoulder: prospective clinical study with an evaluation of three treatment regimens. Ann Rheum Dis 1984;43(3):353-60.

16. Mao CY, Jaw WC, Cheng HC. Frozen shoulder: correlation between the response to physical therapy and follow-up shoulder arthrography. Arch Phys Med Rehabil 1997;78(8):857-9.

17. Rhind V, Downie WW, Bird HA, Wright V, Engler C. Naproxen and indomethacin in periarthritis of the shoulder. Rheumatol Rehabil 1982;21(1):51-3.

18. Binder A, Hazleman BL, Parr G, Roberts S. A controlled study of oral prednisone in frozen shoulder. Br J Rheumatol 1986;25:288-92.

19. Steinbrocker O, Argyros TG. Frozen shoulder: treatment by local injections of depot corticosteroids. Arch Phys Med Rehabil 1974;55(5):209-13.

20. Dacre JE, Beeney N, Scott DL. Injections and physiotherapy for the painful stiff shoulder. Ann Rheum Dis 1989;48(4):322-5.

21. Laroche M, Ighilahriz O, Moulinier L, Constantin A, Cantagrel A, Mazieres B. Adhesive capsulitis of the shoulder: an open study of 40 cases treated by joint distention during arthrography followed by an intraarticular corticosteroid injection and immediate physical therapy. Rev Rhum Engl Ed 1998;65(5):313-9.

22. Andersen NH, Sojbjerg JO, Johannsen HV, Sneppen O. Frozen shoulder: arthroscopy and manipulation under general anesthesia and early passive motion. J Shoulder Elbow Surg 1998;7(3):218-22.

23. Haines JF, Hargadon EJ. Manipulation as the primary treatment of the frozen shoulder. J R Coll Surg Edinb 1982;27(5):271-5.

24. Helbig B, Wagner P, Dohler R. Mobilization of frozen shoulder under general anaesthesia. Acta Orthop Belg 1983;49(1-2):267-74.

25. Ogilvie-Harris DJ, Biggs DJ, Fitsialos DP, MacKay M. The resistant frozen shoulder. Manipulation versus arthroscopic release. Clin Orthop 1995(319):238-48.

26. Kieras DM, Matsen III FA. Open release in the management of refractory frozen shoulder. Orthop Trans 1991;15:801.

27. Ogilvie-Harris DJ, Myerthall S. The diabetic frozen shoulder: arthroscopic release. Arthroscopy 1997;13(1):1-8.

28. Segmuller HE, Taylor DE, Hogan CS, Saies AD, Hayes MG. Arthroscopic treatment of adhesive capsulitis. J Shoulder Elbow Surg 1995;4(6):403-8.