# A Stiff, Painful Shoulder Is *Not* Adhesive Capsuiitis by Dr. Warren I. Hammer

I often hear fellow practitioners brag about how they cured an adhesive capsulitis / frozen shoulder (ADC) in one or two visits. The shoulder they treated may have been stiff and painful, but it was not by definition a primary adhesive capsulitis. Based on the pathology of primary adhesive capsulitis, I am extremely doubtful that anyone has ever cured it in two visits using a non-operative method, as the results of even operative and manipulations under anesthesia are still questionable. At this stage of knowledge, not only is the cause of ADC unknown, but there is also no treatment that **passes prospective and controlled investigations**.[1]

ADC is "characterized by a painful, gradual loss of both active and passive glenohumeral motion resulting from progressive fibrosis and ultimate contracture of the glenohumeral joint capsule."1 It is a progressive disease with definite clinical stages and once the capsule has become fibrotic, scarred and contracted, it is difficult to reverse this pathology in short order.

A primary ADC is a capsular inflammation and fibrosis of the whole capsule by itself not related to other lesions. A secondary ADC usually occurs at the same time with other causes of a stiff shoulder such as extra-articular pathology, rather than capsular pathology. Other causes of a stiff and painful shoulder fall in the category of rotator cuff, calcific tendinitis, and glenohumeral and acromioclavicular arthritis.

A distinguishing feature of the stiffness is that a capsular limitation of motion is only present in the primary type. But of course, there can be an overlap between primary and secondary, since for example, 62 percent of patients with stage 2 ADC often have **partial-thickness tears found on MRI**.[2] This should not be surprising since most people over 50 have non-symptomatic partial-thickness tears.

**According to Neviaser**,[1] it is possible to diagnose ADC based on symptoms and physical examination. They still describe four stages in the progression of the disease; however, it is very difficult to be absolutely sure that a patient has a stage 1 ADC. Stage 1 is characterized by night pain referred to the deltoid insertion, symptoms less than three months duration, and capsular pain on deep palpation or passive stretch. There is an empty end feel at the end-range of motion that causes a patient to order the examiner to stop the stretch, even though the examiner has not reached the end range. In this stage, normal capsular tissue is found on biopsy, although there are inflammatory cells and hypertrophic synovitis.

I was happy to see Neviaser mention an early loss of external rotation as a hallmark of ADC that is not usually found in other shoulder disease processes. The author sites Cyriax,[3] who made that statement in his textbook written in 1962. For years, when a patient had shoulder stiffness and limited end range in external rotation with a minimal harder end-feel than normal, based on my knowledge of Cyriax's methods, I have always suspected ADC and have been right more often than not.

In stage 2 (freezing stage), the night pain is worse and there is increased stiffness. Active and passive motion is restricted in forward flexion, abduction, internal and of course external rotation. The frozen stage continues into stage 3 and there is significant stiffness and loss of motion. Pain is evident at the end range of motion and there is less night pain. Patients only complain of shoulder pain at end ranges at which the capsule is under stretch and are pain free for most motions.

The passive range of motion examination reveals a hard end-feel mechanical block. This stage is present for about nine to 15 months. Arthroscopically, there is a complete loss of the axillary fold and biopsy reveals hypercellular collagenous tissue, similar to other fibrotic conditions.

In stage 4, the "thawing" chronic stage, there is less pain than the other stages, with about the same range-of-motion loss as stage 3. "Thawing" means there is a very gradual increase in range of motion. There are no reported biopsies in this stage and arthroscopy reveals fully mature adhesions that make

**identification of intra-articular structures difficult**.[1] X-rays at any stage are negative except for a disuse osteopenia.

While many state that ADC is self-limiting, there are no adequate natural histories of the condition in the literature. Some patients have returned to normal function after two years with analgesics and reassurance; others have responded to moist heat and anti-inflammatory medications; some with home exercise alone; and there are reports that 50 percent of patients have **residual mild pain and decreased motion** at 4-7-year follow-up.[4]

Non-operative care for ADC, consisting of physical therapy and NSAIDs, has not been shown to alter the natural course of the disease.1 Oral and intra-articular steroids, while providing early relief, also have not been shown to alter the course of the disease. **Griggs**,[5] **in a level 4 study** of stage 2 ADC, used a specific four-direction stretching program and found 90 percent of patients achieved a satisfactory outcome. Others recommend gentle stretching and active motion within the pain-free range to help. **Levine**,[6] **in a level 4 review**, identified patients who failed to improve or were worse after four months as most likely to fail non-operative treatment.

In stage 1, it is necessary to minimize the inflammation and the pain, re-establish normal scapulohumeral rhythm and perform exercises in the pain-free zone. In stage 2, cane exercises emphasizing internal and external rotation range of motion and active exercise in the scapular plane are useful. **Stages 3 and 4 require** heat and warm-up of tissues followed by prolonged, low-load stretching and strengthening of the scapular and cuff muscles.[7] Even though the literature lacks adequate studies, there are also patients who have responded to manipulation under anesthesia, hydrodilation, arthroscopy and open release.

*References*


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