# Abnormal synovium in the frozen shoulder: A preliminary report with dynamic magnetic resonance imaging

Kazuya Tamai, MD, PhD, and Minoru Yamato, MD, PhD, *Tochigi, Japan*

*We studied 16 patients (18 shoulders) with frozen shoulders, 8 patients with subacromial impingement syndrome, and 3 healthy volunteers with dynamic magnetic resonance imaging enhanced with gadolinium diethylenetriaminepentaacetic acid. After intravenous contrast was administered, gradient-recalled echo images were obtained in the oblique coronal plane every 11 to 13 seconds for a total period of 4 to 5 minutes. The signal intensity was measured at the periphery of the glenohumeral joint and in the subacromial bursa. The coefficient of enhancement (percent signal increase per second) in the frozen shoulders was 1.33 ± 0.43 (mean ± SD) for the glenohumeral joint and 0.89 ± 0.47 for the subacromial bursa. These values were far greater than those in subacromial impingement syndrome or in the control group, indicating increased blood flow to the synovium in the frozen shoulders. No previous reports have shown a clinical measure related to the pathophysiology of this disease. (J SHOULDER ELBOW SURG 1997;6:534-43.)*

Frozen shoulder (FS) still remains an enigma. Most possibly it is a syndrome derived from several different conditions that could cause constriction of the joint capsule in the course of the disease. Previous pathologic studies have documented chronic inflammation, fibrosis, or both in the synovium and capsule in patients with FS.[18, 20, 21, 26, 31, 36, 42] In most instances, however, these observations were made in patients with a long history of pain and stiffness for which a biopsy could be justified. To our knowledge there has been no noninvasive means that may scrutinize the property of the synovium and capsule of FS. Arthrography[3, 27, 32] and athroscopy[8, 9, 11, 13, 28, 30, 40, 42] may have contributed to this purpose to some extent, but they are invasive and depict only macroscopic changes on the surface of the joint structures. Although magnetic resonance imaging (MRI) is a useful technique in evaluating soft tissue lesions, it has been considered to show no specific abnormalities in patients with FS.[15] Only recently Emig et al.[7] have reported thickening of the joint capsule and synovium on MRI.

The detection of abnormalities in the synovium with MRI has already been achieved in patients with rheumatoid arthritis. Some studies showed that MRI enhanced with intravenous gadolinium diethylenetriaminepentaacetic acid (Gd-DTPA) was helpful to visualize the inflammatory synovium.[1, 4, 14, 19] Others used dynamic imaging after intravenous administration of Gd-DTPA and recorded signal intensity changes in the hypertrophic synovium.[17, 33] We found that the increase in signal intensity measured with dynamic MRI was correlated with histologic findings of the synovium in rheumatoid arthritis.[38] Such knowledge led us to expect that the synovial changes in FS could also be quantified or semiquantified by dynamic MRI enhanced with Gd-DTPA.

This report describes preliminary results of dynamic MRI on 16 patients with a typical clinical course of FS who were examined during the study period of 1991 to 1994. We investigated whether the dynamic MRI findings of the synovium in FS

From the Departments of Orthopaedic Surgery and Radiology, Dokkyo University School of Medicine

Reprint requests. Kazuya Tamai, MD, PhD, Department of Orthopaedic Surgery, Dokkyo University School of Medicine, 880 Kita-kobayashi, Mibu-machi, Shimotsuga-gun, Tochigi 321-02 Japan



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might differ from those in normal shoulders or in subacromial impingement syndrome.

## PATIENTS AND METHODS

**Patients.** We examined 18 shoulders in 16 patients with FS (8 female patients and 8 male patients). All the patients were informed of the purpose of the study and possible side effects of the contrast agent before the imaging. In this study FS was defined as a painful and stiff shoulder that developed in otherwise healthy persons 40 to 70 years of age, had lasted for more than 1 month before examination, and showed less than 135° of passive forward elevation associated with recognizable limitation of external and internal rotation. The additional inclusion criterion for FS was that after diagnosis and imaging, the patient was monitored by us until the affected shoulder became painless with normal or near normal range of motion. The exclusion criteria for FS were a history of definite trauma, fractures, rotator cuff tearing, presence of calcium deposit on x-ray evaluation, rheumatoid arthritis, osteoarthritis, or cervical spine disease. None of the 16 patients had diabetes mellitus, thyroid disease, heart disease, pulmonary disease, or hemiplegia.

The age range of the patients enrolled in this study was 41 to 70 years (mean 56 years), and the range of the duration of the disease before imaging was 1 to 18 months (mean 7 months). In 14 patients with unilateral involvement the right shoulder was affected in 5 and the left in 9. The other two patients presented with bilateral FS and had images of both shoulders obtained separately with an interval of 2 to 3 days. In all the patients the degree of pain and stiffness of the affected shoulder was assessed on the day of imaging according to a scoring system provided by the Japanese Orthopedic Association.[37] The shoulders studied showed a pain score of 5 to 20 (mean 11, full mark 30) and a range-of-motion score of 11 to 19 (mean 14, full mark 30). All the patients were treated with physiotherapy such as local warmth and self-assisted gentle motion exercises supported by the occasional use of nonsteroidal antiinflammatory drugs. Such a treatment regimen had been performed for at least 3 weeks before imaging. No patients had received intraarticular or subacromial injections of corticosteroids within 3 months before imaging. As described, all the patients were monitored until clinical remission was ensured; the overall symptomatic period ranged from 11 to 27 months (mean 18 months).

Control imaging was performed on three shoulders in three healthy volunteers (all male; 16, 40, and 50 years, respectively) who had no history of shoulder pain and no shoulder symptoms. In addition, to compare with FS, we obtained images of eight shoulders in eight patients (three men and five women; age range 22 to 33 years) who had subacromial impingement syndrome of stage I or II according to Neer's classification.[25] All of these patients reported pain localized to the anterolateral aspect of the shoulder that occurred with overhead activities, but there was no restriction of passive range of motion. The pain was relieved by an injection of 5 ml lidocaine into the subacromial bursa, which was performed after MRI. None of the eight shoulders showed signs of rotator cuff tearing on sonography or arthrography.

MRI was repeated on four shoulders in three patients with FS. Three of the four shoulders had the second imaging 5 to 8 months after the first one, when the clinical symptoms showed substantial improvement. In the remaining one shoulder the first MRI was performed at the time when the patient reported shoulder pain but the range of motion was not restricted. We included this patient in this study because he thereafter had limitation of motion, and 6 months later, when the second MRI was obtained, his symptoms fulfilled the inclusion criteria for FS described previously.

**MRI.** The imaging was carried out with a 1.5 Tesla MRI unit (SMT 150GUX, Shimadzu Medical Co. Ltd., Kyoto, Japan) equipped with a flexible coil. The patients were supine in the unit with the arms at side and the shoulders in neutral rotation. $T_1$- (repetition time [TR], 500 msec; echo time [TE], 20 msec), proton density- (TR, 2000 msec; TE, 20 msec), and $T_2$-weighted (TR, 2000 msec; TE, 80 to 90 msec) spin echo (SE) images were obtained in the oblique coronal plane.

Then the gradient-recalled echo (GRE) technique (TR, 45 to 50 msec; TE, 9 to 10 msec; flip angle, 40° to 50° [in several joints, 70° to 80°]) was used to obtain an image through the center of the humeral head determined on $T_1$-weighted SE images. Other imaging parameters included a 15 cm field of view, a 256 × 192 matrix, and 4 mm section thickness with a 1 mm intersection gap. After the first nonenhanced GRE image, Gd-DTPA (0.1 mmol/kg body weight) was administered via the cubital vein opposite the shoulder studied by a bolus injection over a period of 10 to 15 seconds. The second GRE image was taken at the time when Gd-DTPA injection was started. Serial GRE images were then acquired every 11 to 13 seconds for a total period of 4 to 5 minutes (Figure 1).

**Evaluation of static SE images.** We evaluated rotator cuff morphologic characteristics, subdeltoid/subacromial fat, and subdeltoid/subacromial fluid according to the scoring system of Zlatkin et al.[16, 45] In addition, the absence and presence of fluid in the glenohumeral joint was scored on a scale of 0 and 1, respectively; in this study only a thin intraarticular rim without distention of the axillary pouch was regarded as normal (scored as 0).

**Evaluation of dynamic GRE images.** Mean signal intensity values of the synovial surface were determined with a circular region of interest (ROI) set in the enhancing rim of a GRE image taken 120 to 140 seconds after Gd-DTPA injection (Figure 2, *A*). A total of five ROIs were used; each contained an area of 4 to 10 $mm^2$ and included 12 to 29 pixels. Two ROIs were set in the glenohumeral joint (GHJ) close to the upper and lower corner of the glenoid. The other three ROIs were set in the subacromial bursa (SAB) along the outer surface of the rotator cuff: beneath the acromion, over the greater tuberosity, and approximately the mid-point between them. The signal intensity values in each area were then plotted as a function of time with an implemented evaluation program.

For quantitative characterization of the signal intensity time curves, we determined a regression line by the least squares method to fit the data from the first approximately 120 seconds of imaging (Figure 2, *B*). The slope of this regression line was regarded as a coefficient of enhancement (CE). This assertion was based on such observations that the initial increase of signal intensity after Gd-DTPA administration was basically linear in all the shoulders studied, that the signal intensity usually reached a plateau 150 to 180 seconds after contrast injection, and that more rapid initial increase resulted in a greater value of maximum signal intensity obtained in each imaging. Actually, in this study the CE was given in percent signal increase per second with the precontrast signal intensity in each ROI as the baseline, considering the variety of raw signal intensity among the individuals and among the measurement sites. The CE of GHJ ($CE_{GHJ}$) and SAB ($CE_{SAB}$) in each shoulder was calculated by averaging the values from two and three ROIs, respectively. In addition, we repeated the calculation in two shoulders with FS, changing the size and location of ROIs to ascertain the reproducibility of the measurements.

**Statistical analyses.** The regression slope (CE) in each data series of dynamic MRI was tested against the null hypothesis (CE = 0) to prove the enhancement. We used the *t* test to compare the average CE between two groups of patients and the paired *t* test to compare the $CE_{GHJ}$ with $CE_{SAB}$ in a group of patients. For testing the linear correlation Pearson's correlation coefficient was calculated. In each test the null hypothesis was rejected at the 95% confidence level; a $p$ level $<0.05$ was significant.

## RESULTS

**Static SE images.** All of the shoulders in the control group appeared normal except for an indistinct subacromial fat plane in one. The shoulders with subacromial impingement syndrome showed an increase in tendon signal in four of eight, absence of subacromial fat plane in five of eight, and subacromial fluid in one of eight. None of the shoulders in these two groups had glenohumeral fluid collection (Table I).

The findings in the FS group varied among individuals. Abnormalities of the tendon were noted in 12 of 18, of which 11 showed increased signal intensity only (scored as 1), whereas the remaining one had localized thickening of the tendon in addition to some signal intensity changes (scored as 2). No shoulders showed discontinuity of the tendon. Subdeltoid/subacromial fluid was noted in 6 of 18. Glenohumeral fluid was present in 6 of 18, of which 5 showed concomitant subdeltoid/subacromial fluid (Table I).

**Dynamic GRE images.** Reproducibility tests in two sample shoulders revealed that the signal intensity measurements depended on the size and location of the ROIs. However, as far as the placement of ROIs within the enhancing rim was strictly followed, the calculated CE values did not differ more than 6% if every ROI was changed as much as 50% of its size or relocated a distance of its diameter away.

The shoulders in the control group showed a slight increase in signal intensity in the periphery of the synovial cavity after Gd-DTPA administration. Overall, the subacromial impingement syndrome showed greater enhancement compared with the control group, but the degree of enhancement

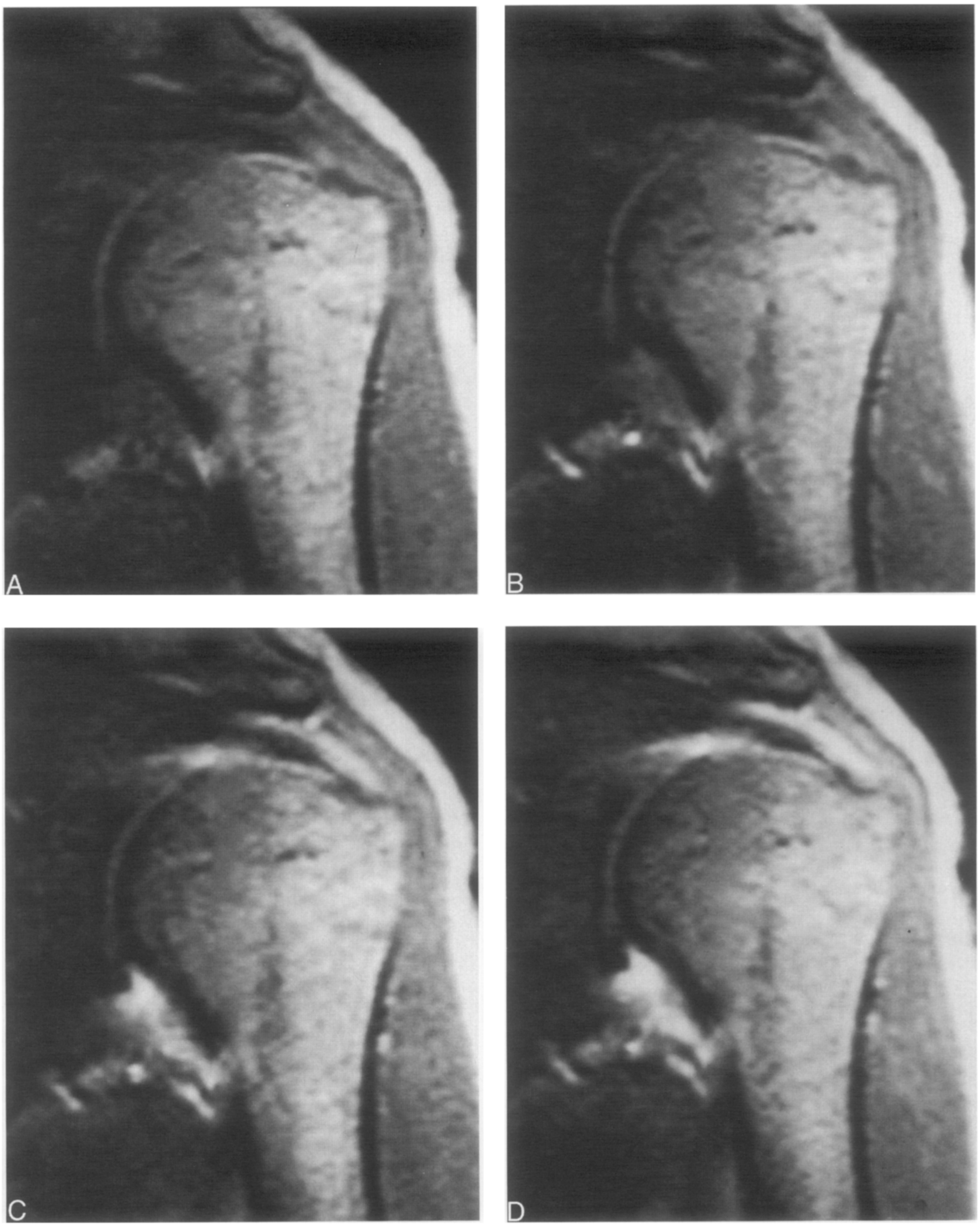


**Figure 1** Dynamic magnetic resonance imaging enhanced with Gd-DTPA. Dynamic gradient-recalled echo images obtained before **(A)**, 33 **(B)**, 66 **(C)**, and 110 **(D)** seconds after Gd-DTPA administration Dramatic increase in signal intensity in glenohumeral joint and subacromial bursa is noted.

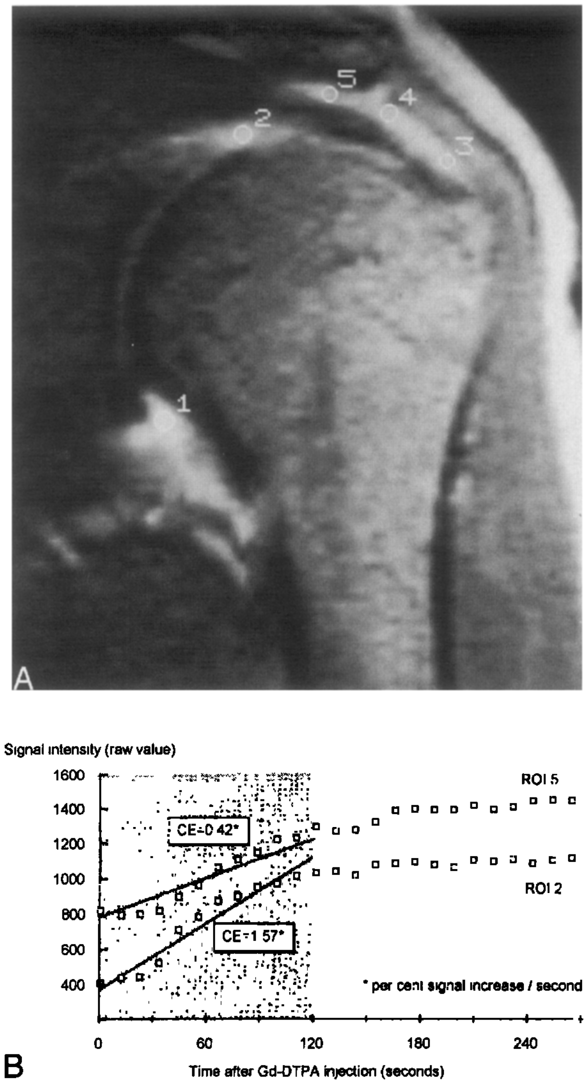


**Figure 2** Example of quantitative evaluation of dynamic gradient-recalled echo images. **A**, Two regions of interest (Nos. 1 and 2) were set in glenohumeral joint and three (Nos. 3, 4, and 5) in subacromial bursa in image obtained 121 seconds after Gd-DTPA administration. See text for detailed location of regions of interest. **B**, Graph showing signal intensity-time curves obtained from two regions of interest Data from first 120 seconds were used to calculate regression line, slope of which was regarded as coefficient of enhancement *(CE)* See text for method of calculation of CE.

greatly varied among individuals ($CE_{GHJ}$ = 0.32 ± 0.29 [mean ± SD], $CE_{SAB}$ = 0.40 ± 0.37). No significant difference was noted between $CE_{GHJ}$ and $CE_{SAB}$ (Table I).

In contrast, all the shoulders with FS showed obvious enhancement ($CE_{GHJ}$ = 1.33 ± 0.43, $CE_{SAB}$ = 0.89 ± 0.47). These values were significantly ($p < 0.001$, $p < 0.05$, respectively) greater than their counterparts in subacromial impingement syndrome. In addition, $CE_{GHJ}$ was significantly ($p < 0.01$) greater than $CE_{SAB}$ in patients with FS (Table I).

When the clinical features of FS were correlated with the enhancement in the synovial cavity, a shorter duration of disease before imaging resulted in a greater $CE_{GHJ}$ ($r = -0.751$, $p < 0.001$) (Figure 3). However, no significant correlation was noted between the duration of disease and $CE_{SAB}$.

The CE was also tested if it depended on the scores of static SE images (tendon abnormalities, subdeltoid/subacromial fluid, glenohumeral fluid, and total Zlatkin score). Both the $CE_{GHJ}$ and $CE_{SAB}$ were independent from these scores, however.

**Longitudinal study.** When the second MRI in the four shoulders with FS was compared with the first one, there was no discernible difference in the findings of static SE images. On the other hand, the dynamic imaging recorded a decrease in $CE_{GHJ}$ and $CE_{SAB}$ in the three shoulders that had the second MRI after the improvement of symptoms and an increase in $CE_{GHJ}$ in the shoulder that had the second MRI after the progression of the disease (Table I).

## DISCUSSION

The increase in signal intensity on MRI enhanced with Gd-DTPA has been considered to be a result of perfusion dynamics of this contrast agent. In the musculoskeletal system there is no barrier between blood vessels and the extracellular space.[2] In addition, because the joint fluid is an ultrafiltrate of plasma, it is comparable to an extension of the extracellular space.[10, 43] Thus intravenously injected Gd-DTPA freely diffuses into the interstitial space of the synovium and then into the joint fluid because of its small molecular weight.[41, 44] The rate of diffusion varies depending on the blood flow to the synovium and the bulk flow of fluid from the capillary bed to the joint space.[2, 43]

It has been shown that the periphery of the joint cavity enhances with intravenous Gd-DTPA in rheumatoid arthritis.[1, 4, 14, 17, 19, 33, 38, 44] The early enhancement of the rheumatoid synovium results from increased blood flow to the synovium and an enlarged interstitial space caused by hypertrophy of the synovium. The periphery of the joint cavity may also enhance with Gd-DTPA in posttraumatic conditions, in which the enhancement appears as a thin band of contrast material mimicking the

**Table I** Profile of the patients and the results of imaging

| | | | | Clinical presentation | | | Spin echo image† | | | | | | | Dynamic imaging | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | JOA score | | | Subdeltoid | | Subacromial | | | | | |
| Case | Side | Age (yr) | Sex | Duration (mo)* | Pain | ROM | Tendon | Fat | Fluid | Fat | Fluid | Total score‡ | GHJ fluid | $CE_{GHJ}$ | $CE_{SAB}$ |
| Control | | | | | | | | | | | | | | | |
| 1 | R | 40 | M | | | | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0.16 | 0.15 |
| 2 | R | 16 | M | | | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.27 | 0.20 |
| 3 | R | 50 | M | | | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.02 | 0.17 |
| Subacromial impingement syndrome | | | | | | | | | | | | | | | |
| 1 | R | 24 | F | 1 | 20 | 24 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1.03 | 1.25 |
| 2 | R | 28 | F | 6 | 15 | 30 | 1 | 0 | 0 | 1 | 0 | 2 | 0 | 0.12 | 0.17 |
| 3 | R | 22 | F | 5 | 15 | 30 | 1 | 0 | 0 | 1 | 0 | 2 | 0 | 0.28 | 0.35 |
| 4 | L | 31 | M | 1 | 20 | 27 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0.15 | 0.39 |
| 5 | R | 33 | F | 8 | 15 | 27 | 1 | 0 | 0 | 1 | 0 | 2 | 0 | 0.44 | 0.68 |
| 6 | L | 24 | M | 2 | 20 | 27 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0.31 | 0.18 |
| 7 | L | 26 | M | 4 | 25 | 30 | 1 | 0 | 0 | 0 | 1 | 2 | 0 | 0.13 | 0.03 |
| 8 | L | 23 | F | 1 | 20 | 24 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.11 | 0.12 |
| Mean | | 26 | | 3.5 | 19 | 27 | 0.50 | 0.00 | 0.00 | 0.63 | 0.13 | 1.25 | 0.00 | 0.32 | 0.40 |
| SD | | 4 | | 2.5 | 3.3 | 2.3 | 0.50 | 0.00 | 0.00 | 0.48 | 0.33 | 0.83 | 0.00 | 0.29 | 0.37 |
| Frozen shoulder | | | | | | | | | | | | | | | |
| 1 | L | 57 | F | 18 | 20 | 14 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0.60 | 0.31 |
| 2 | R | 54 | F | 5 | 10 | 19 | 1 | 0 | 0 | 0 | 1 | 2 | 1 | 1.64 | 0.65 |
| 3 | R | 57 | F | 18 | 5 | 13 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0.95 | 0.71 |
| | L | | | 4 | 10 | 13 | 1 | 0 | 0 | 1 | 0 | 2 | 0 | 1.53 | 0.69 |
| 4 | L | 58 | M | 7 | 10 | 14 | 0 | 0 | 1 | 1 | 1 | 3 | 1 | 0.87 | 1.00 |
| 5 | L | 66 | F | 3 | 10 | 14 | 1 | 0 | 1 | 0 | 1 | 3 | 1 | 1.84 | 1.30 |
| 6 | R | 52 | M | 1 | 10 | 16 | 1 | 0 | 0 | 1 | 0 | 2 | 0 | 1.90 | 0.68 |
| 7 | R | 49 | M | 2 | 10 | 13 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 2.07 | 1.15 |
| 8 | R | 54 | M | 3 | 5 | 14 | 1 | 0 | 0 | 0 | 1 | 2 | 1 | 0.97 | 1.13 |
| 9 | L | 41 | F | 14 | 10 | 11 | 1 | 1 | 0 | 0 | 0 | 2 | 0 | 0.91 | 0.62 |
| 10 | L | 56 | F | 7 | 15 | 14 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.91 | 1.15 |
| | R | | | 12 | 15 | 14 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0.90 | 0.84 |
| 11 | L | 49 | F | 4 | 10 | 14 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1.59 | 1.05 |
| 12 | L | 49 | M | 8 | 10 | 19 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1.20 | 0.28 |
| 13 | L | 70 | M | 3 | 10 | 13 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1.91 | 0.27 |
| 14 | R | 55 | F | 7 | 15 | 14 | 2 | 0 | 1 | 0 | 0 | 3 | 0 | 1.54 | 1.09 |
| 15 | L | 56 | M | 6 | 10 | 11 | 1 | 1 | 0 | 1 | 0 | 3 | 0 | 1.52 | 2.38 |
| 16 | L | 69 | M | 6 | 5 | 19 | 1 | 0 | 0 | 0 | 1 | 2 | 1 | 1.06 | 0.72 |
| Mean | | 56 | | 7.1 | 11 | 14 | 0.72 | 0.11 | 0.17 | 0.28 | 0.28 | 1.56 | 0.33 | 1.33 | 0.89 |
| SD | | 7 | | 5.0 | 3.7 | 2.3 | 0.56 | 0.31 | 0.37 | 0.45 | 0.45 | 1.07 | 0.47 | 0.43 | 0.47 |
| Frozen shoulder (longitudinal study) | | | | | | | | | | | | | | | |
| 9 | L | 41 | F | 14 | 10 | 11 | 1 | 1 | 0 | 0 | 0 | 2 | 0 | 0.91 | 0.62 |
| | | | | 22 | 20 | 19 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0.56 | 0.17 |
| 10 | R | 56 | F | 12 | 15 | 14 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0.90 | 0.84 |
| | | | | 17 | 20 | 24 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0.70 | 0.72 |
| | L | 56 | F | 7 | 15 | 14 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.91 | 1.15 |
| | | | | 12 | 20 | 21 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.39 | 0.52 |
| 12 | L | 49 | M | 2 | 10 | 27 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.32 | 0.28 |
| | | | | 8 | 10 | 19 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1.20 | 0.28 |

*JOA*, Japanese Orthopedic Association; *ROM*, range of motion; *GHJ*, glenohumeral joint; $CE_{GHJ}$, coefficient of enhancement of the glenohumeral joint; $CE_{SAB}$, coefficient of enhancement of the subacromial bursa.
*Duration of disease before imaging.
†Evaluated according to the Zlatkin's system.
‡Total Zlatkin's score.

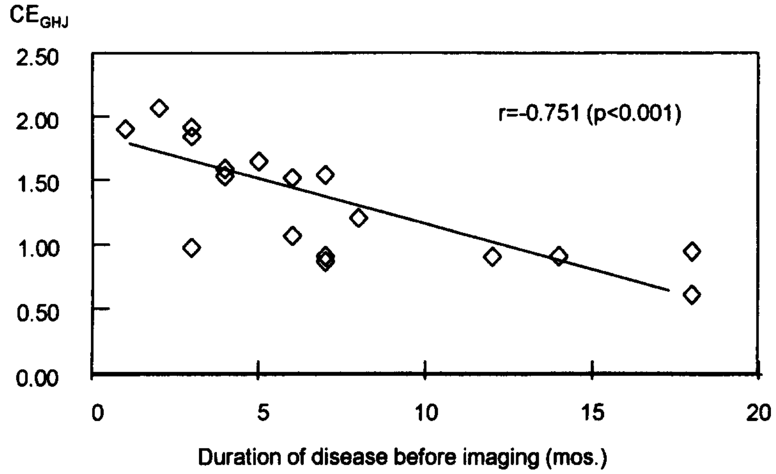


**Figure 3** Graph showing negative correlation of coefficient of enhancement in glenohumeral joint ($CE_{GHJ}$) with duration of disease before imaging in patients with frozen shoulder.

contour of the synovium unless the joint is mobilized.[6, 43] This phenomenon is a result of physiological passage of Gd-DTPA from capillaries to the joint fluid.

Our study has demonstrated obvious enhancement of the glenohumeral synovium in patients with FS clearly distinguishable from that of normal shoulders. Among the previous histologic studies on FS, Neviaser[26] observed reparative inflammatory changes in the subsynovial layer associated with vascular dilatation. McLaughlin[21] found a nonspecific proliferative synovitis of the glenohumeral joint, whereas Ozaki et al.[31] recognized hyalinization and fibrinoid degeneration of the rotator interval. Wiley[42] reported nonspecific vasculitis in the joint capsule. Kumagai et al.[18] described vascular dilatation in the deep layer of the synovium. Moreover, recent arthroscopic observations of FS have shown synovial changes in the glenohumeral joint such as villous hypertrophy,[11] fibrinous reaction,[28] increased vascularity,[9, 11, 28, 40] synovitis[8, 13, 30] or synovitis with patchy vascular reaction,[42] and thickening of the synovium.[28] Taking these together it seems that there are inflammatory or degenerative changes in the glenohumeral joint with this disease associated with increased vascularity in the synovium, the subsynovial layer, or both. Therefore we presume that the enhancement observed in this study was mainly caused by increased blood flow in and around the synovial tissue, even if a small amount of peripheral enhancement was attributable to "normal" passage of the contrast material into the joint fluid.

Before this study we believed that the enhancement would be much more subtle in FS than rheumatoid arthritis. However, the enhancement of the glenohumeral joint observed in this study (CE = 1.33 ± 0.43) was only slightly lower compared with our result on rheumatoid arthritis[38] (CE = 1.52 ± 0.77 when the data were converted). Others reported greater enhancement in rheumatoid synovium. In the study of Reiser et al.[33] the CE for pannus tissue was approximately 1.8 when converted from their original data, supposing the enhancement had occurred linearly as a function of time. Based on the results of König et al.,[17] the CE could be calculated as approximately 2.3 for hypervascular and 1.5 for slightly hypervascular pannus, whereas it was 0.23 for fibrous pannus. The observations of Munk et al.[23] on rheumatoid shoulders showed an average CE of approximately 2.2 when recalculated. It seems that the unexpected similarity of the results between our two series is at least partly the result of the patient population in our previous study[38]; we had obtained images of patients with advanced rheumatoid arthritis including those having "burned out" synovitis, whose enhancement was particularly low. We now postulate that in general the enhancement of the glenohumeral joint with FS is lower than that of rheumatoid arthritis but may occasionally exceed the level of rheumatoid synovium with less active inflammation.

This study also revealed that the presence of glenohumeral fluid noted in SE images did not result in greater enhancement. Although the distention of the jolt capsule with effusion accelerates the transsynovial exchange of small molecules, it may hinder the passage of Gd-DTPA by increasing the intraarticular pressure and compressing the synovial microcirculation.[35] Drapé et al.[6] observed insignificant increase in enhancement in the presence of joint effusion. Subsynovial fibrosis, which has been shown to be a usual concomitant of FS,[20, 26, 31] may also hinder the passage of contrast material. However, we were unable to test the latter possibility because of the lack of histologic specimens from our patients.

We observed that in FS the enhancement of the glenohumeral joint was greater than that of the subacromial bursa (CE = 1.33 ± 0.43 vs 0.89 ± 0.47). There are two possible reasons for this. First, the subacromial bursa with FS may have less remarkable abnormalities. In fact, several studies failed to find edema, cellular infiltration, and vascular proliferation in the bursa.[21, 22, 34, 39] In the series of Neviaser[26] 3 of his 10 cases with FS had a histologically normal bursa, whereas only one had normal glenohumeral synovium. In their book on arthroscopy Esch and Baker[8] stated that subacromial bursoscopy findings varied from normal to proliferative chronic bursitis. It seems that the subacromial bursa with FS is often normal or near normal, in contrast with the glenohumeral joint, which in most instances shows substantial disease. Second, the lower enhancement in the bursa may be related to the measurement of the signal intensity. One or two ROIs in the subacromial bursa were placed in an area that was initially relatively high in signal intensity. The signal intensity in this area may possibly be an average of the fat and the synovium. Because the fat is less vascular than the synovium, the real CE of the bursal synovium may be greater than that obtained in this study.

We also observed that the subacromial bursa showed greater enhancement in FS than in subacromial impingement syndrome (CE = 0.89 ± 0.47 vs 0.40 ± 0.37). According to Neer[25] there is edema and hemorrhage in the subacromial bursa in stage I impingement and fibrosis and thickening in stage II. These changes, if present, could have affected the perfusion dynamics of Gd-DTPA, leading to early peripheral enhancement of the bursa. Unexpectedly, however, the enhancement of our patients with impingement was generally low with a few exceptions. This result may partly be explained by the following speculations. (1) Because the subacromial impingement syndrome is a clinical diagnosis, the disease in the bursa may differ from what has been described. (2) The bursal abnormality in stage I impingement can be reversible[25] and may have improved with rest before the imaging. This and other issues on the pathophysiology of subacromial impingement syndrome are the subject of another review and are not discussed further here.

It is important to recognize that MRI findings may vary depending on the timing of imaging. Clinically, FS contains the well-known three phases: freezing, frozen, and thawing.[5, 24] With arthroscopy Neviaser and Neviaser[28] documented four stages of this disease consisting of preadhesive, acute, mature, and chronic. Viewed from the perfusion property of Gd-DTPA, it is natural to assume that the thawing phase, or the chronic stage, would show less enhancement compared with the earlier, more active phases. In fact, $CE_{GHJ}$ showed a negative correlation with the duration of disease in our cross-sectional study. Furthermore in our longitudinal study there was a trend that the enhancement was associated with improvement or exacerbation of the symptoms, although the number of the patients was too small. These results suggest that the enhancement in dynamic MRI may be a reflection of the disease process of FS.

In addition to the lack of surgical proof, there are several disadvantages in this study. First, in this protocol only one section can be viewed continuously over time. We selected the oblique coronal rather than the oblique sagittal or axial plane for measuring enhancement, because we initially thought that the supraspinatus tendon and its insertion might also enhance in dynamic MRI. Perhaps the best way to identify the pathologic area of the shoulder joint synovium is taking images through two planes alternating, for example, the oblique coronal and the oblique sagittal planes. The second problem is the accuracy of measurement. Even if all the ROIs are exactly positioned in the anatomic structure, a partial volume effect cannot be disregarded, particularly when the enhancement is low. In this context only a high value of CE (subjectively more than approximately 0.5) may make sense. The third disadvantage is the study population. Because this study was primarily aimed to characterize the dynamic MRI findings of FS, we analyzed the data from those patients whose clinical course proved typical. Consequently this study did not include very mild or intractable cases in which the enhancement pattern may be different from that described here.

Nevertheless, our results are consistent with the idea that FS is at least partly a disease of the synovium involving the glenohumeral joint and less predominantly the subacromial bursa. Although the enhancement of the synovium with Gd-DTPA is not specific to FS, this study has shown a minimally invasive means that may enable quantification of the synovial abnormalities in FS, which hitherto have never been estimated in the clinical setting. In the near future dynamic imaging may become an adjunct for choosing a method of treatment. For example, when the enhancement of the synovium is sufficiently high, the use of oral nonsteroidal antiinflammatory drugs or of an appropriate dose of intraarticular steroids may be justified. On the other hand, these agents may not be effective when the enhancement is low. In addition, a patient with prolonged limitation of motion despite low enhancement may have reason to undergo surgery such as arthroscopic capsular release.[12, 29] Naturally this study does not resolve how much enhancement is critical in using those treatment options. Further observations are required to confirm or refute this speculation.

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