Acetic Acid Iontophoresis and Ultrasound for the Treatment of Calcifying Tendinitis of the Shoulder: A Randomized Control Trial

Marc Perron, BSc, Francine Malouin, PhD

ABSTRACT. Perron M, Malouin F. Acetic acid iontophoresis and ultrasound for the treatment of calcifying tendinitis of the shoulder: a randomized control trial. Arch Phys Med Rehabil 1997;78:379-84.

Objective: To assess the effects of acetic acid iontophoresis (AAI) and ultrasound on calcifying tendinitis of the shoulder, and to determine the relation between changes in the radiological measures of calcium deposit (CD) and shoulder function.

Design: Randomized control trial.

Setting: General community, private practice.

Patients: Twenty-two adults (7 men, 15 women) with a calcifying tendinitis of the shoulder, without associated conditions, stratified according to the type of lesions (X-ray: type I, fleecy appearance; type II, homogeneous), were randomly allocated to an experimental (EXP, $n = 11$) or to a control (CTL, $n = 10$) group.

Interventions: CTL group, no treatment; EXP group, nine treatments including AAI (5% acetic acid solution via the negative electrode, 5mA galvanic current, 20 minutes) followed by continuous ultrasound (0.8w/cm^2, 1MHz, 5 minutes).

Main Outcome Measures: Area and density of the CD, passive shoulder abduction (range of motion [ROM]), pain intensity.

Results: Significant reduction in the area and density of CD (ANCOVA, $p = .01$ and .03) over time in the EXP and CTL groups, but no significant difference between groups for any of the variables measured. The decrease in the area of CD in type I lesions ($n = 5$) was larger (Mann-Whitney U test, $p < .01$) than in type II ($n = 16$) lesions. The relation was stronger ($r_s = .90$) between changes in area and density of CD than between ROM and pain ($r_s = -.67$). Correlations were weak ($r_s = .21$ to .41) between radiological and functional changes.

Conclusion: The reduction in CD area and density likely results from a natural process rather than treatment (AAI and ultrasound); type I lesions (resorptive phase) are more likely to display resorption of the CD than type II lesions (formative phase). Reduction of the CD area does not necessary result in a functional improvement.

From the Clinique de Physiothérapie de Charny (Mr. Perron), Charny, and Physiotherapy Department, Faculty of Medicine, Laval University (Dr. Malouin), Québec, PQ, Canada.

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Reprint requests to Dr. Francine Malouin, Physiotherapy Department, Faculty of Medicine, Laval University, Québec, PQ, Canada G1K 7P4.

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CALCIFYING TENDINITIS of the shoulder is characterized by a reactive calcification that affects the rotator cuff tendons.[1] Patients mainly complain of pain and reduced range of motion (ROM). The magnitude of these problems is variable and depends on the stage of evolution (ie, formative or resorptive) of the condition.[1] In the early formative stage, calcium crystals coalesce in the tendon to form a homogeneous and well-defined deposit. This type II calcium deposit (CD) may impinge against the coracoacromial arch to produce light pain and slight, or no, restriction in ROM. With time, the resorptive stage is initiated, and a type II CD evolves to a type I CD; the latter has a fleecy aspect with a poorly defined periphery. During this stage, patients experience acute pain concomitant with important disabilities. This self-resorptive stage may result in complete disappearance of the CD within a week.[2]

It is clinically recognized that techniques which deal directly with the CD, including surgery and needling, successfully reduce pain and restore shoulder function. However, their use is indicated when the conservative approach is unable to relieve acute pain that markedly interferes with daily activity.[1] Thus, an important part of the early management of calcifying tendinitis of the shoulder is left to physiotherapists who must select optimal therapeutic strategies to improve function.

To our knowledge, no study has clearly demonstrated the efficacy of physiotherapy protocols for calcifying tendinitis of the shoulder. Nevertheless, it is generally accepted in physiotherapy practice that light ROM exercices[1] and electroanalgesia contribute to maintaining shoulder amplitude and relieve pain. Another potential approach is to reduce the CD by acetic acid iontophoresis (AAI). This treatment is particularly attractive because it could decrease the size of the CD and therefore improve shoulder function by relieving the impingment in the subacromial space. AAI was first introduced in 1955 by Psaki and Carroll,[3] who reported partial or complete disappearance of CDs and functional improvement in 9 of 12 patients (75%) treated for 3 to 4 months with AAI followed by electrical stimulation of surrounding muscles. More recently, Fortin[4] reported a reduction of CDs in 55% of 135 patients treated for 6 weeks by AAI, electrical stimulation, and superficial heat. In these two uncontrolled studies, limited information was provided regarding the methods used for the measurement of the dependent variables. As such, the practice of AAI has not been validated and the proposed beneficial effects of AAI on calcifying tendinitis of the shoulder need to be confirmed in controlled studies using reliable and valid outcome measures.

The mechanisms of action of AAI remain obscure. To what extent the acetate ion can be successfully introduced into the tissues by mean of iontophoresis or bind to the CD is unknown. On the other hand, it has been shown that iontophoresis can effectively introduce other substances through the skin. Indeed, animal studies have confirmed the presence of ionized drugs in the deep tissue layers,[5] and clinical trials[6-9] have documented the effectiveness of analgesic or anti-inflammatory drugs using iontophoresis. Based on these results, it is possible that the

acetate ion is introduced by iontophoresis to help in the resorption of the CD; however, no experimental evidence is available to support this contention.

The use of ultrasound to promote resorption of the CD and improve function has been examined in a control study.[10] Since reduction of the CD was found in patients of both groups, it appears that the ultrasound had no specific effects. Unfortunately, the lack of statistical analysis and of information on the methods used to measure the CD reduction limit the interpretation of the results. It is known, however, that ultrasound increases the kinetic energy of the molecules, augments cell permeability, and enhances drug diffusion.[11] Because of these thermal and mechanical effects, ultrasound could enhance the penetration of the acetate ion following iontophoresis, creating an effect similar to phonophoresis. It could thereby act as an excellent adjunct to AAI.

In this study, we examined the specific effects of AAI combined with ultrasound in the treatment of calcifying tendinitis of the shoulder in a randomized control trial, using objective measures of the CD (area and density) and shoulder function (ROM and pain). A second goal was to determine the relationship between radiological and functional changes.

METHODS

Subjects. The patients included in this study were adults (men and women) with a confirmed diagnosis of symptomatic calcifying tendinitis of the shoulder. The area of the CD had to be 50mm^2 or larger. Subjects were excluded if they presented with associated conditions such as systemic disease or cervical lesions producing shoulder pain, or x-rays were contraindicated for their conditions, or they received secondary benefits (ie, workman compensation).

Selection and randomization procedures. From June 1990 to December 1994, patients with a symptomatic diagnosis of calcifying tendinitis of the shoulder were referred to our clinic by physicians and physiotherapists from the Quebec City area. The first step in the selection process was to confirm the eligibility of the patients on the basis of nonradiological inclusion criteria. Patients who agreed to take part in the study were requested to sign a consent form. The final step in the selection procedure was the radiological evaluation to confirm that the CD was larger than 50mm^2 and to determine the type of the lesions (type I or type II). To control for the stage of the condition, patients were stratified by lesion type. The patients in each stratum were then randomly assigned to the experimental (EXP) or control (CTL) groups.

Experimental design. Patients were evaluated (radiological and functional evaluations) four times: at baseline and thereafter weekly for 3 weeks. In the EXP group, patients were evaluated after the 3rd (end of week 1), 6th (end of week 2) and 9th (end of week 3) treatments of the 3-week physiotherapy protocol; the CTL group was evaluated at corresponding time periods.

Evaluation procedures. The testing session at baseline was part of the selection procedure. All testing sessions comprised a radiological evaluation followed by a functional evaluation. The radiological evaluations were carried out in the same hospital using procedures standardized by the participating radiologist. With the patient lying supine and the upper limb placed parallel to the trunk, one exposure was taken for each of three incidences: external rotation, neutral, and internal rotation as previously described.[1,2] Since none of the patients had any significant restriction of shoulder rotation, position was easily standardized, and there was no need for goniometric measurements to obtain similar incidence of exposures at each testing session. X-ray parameters (depth of penetration) were constant for each patient. The functional evaluation consisted of measuring, with the patient in the supine position, the range of passive abduction to the point where pain was induced. ROM was measured with a manual goniometer and passive shoulder abduction was performed with the elbow extended. Care was taken to allow natural humeral rotation during passive abduction. To prevent exacerbation of the underlying inflammatory process, only shoulder abduction was assessed. The movement of abduction was chosen because subacromial impingment occurs particularly with abduction[12]; therefore, this movement is a good indicator of the evolution of calcifying tendinitis of the shoulder. The degree of pain induced by passive abduction was scored using the Present Pain Index scale,[13] which ranges from 0 to 5; passive abduction of the shoulder was repeated 3 times.

Evaluators. The standardized radiological evaluation was carried out by technicians under the supervision of the participating radiologist, who was blind to group assignment. Analysis of the X-ray films was carried out by the same radiologist throughout the study. Four physiotherapists participated in the functional evaluations, but each patient was reevaluated by the same physiotherapist. Evaluators were unaware of the group assignment, and the patients were reminded not to make any statement that would unblind the evaluators.

Treatment regimens. Patients in both groups were asked to avoid activities requiring overhead arm movements or repetitive tasks with the involved shoulder. They were authorized to use analgesic medication as necessary. Icing, warming, or massages were not allowed. As an incentive to restrain the activity level of the involved shoulder, patients were asked to keep a log book of their daily activities; medication was also monitored. Patients in the CTL group were given no treatment; they were seen weekly for radiologic and functional evaluations only. Patients in the EXP group were given three treatments weekly, for 3 consecutive weeks (ie, nine treatment sessions). Treatments were provided by trained physiotherapists in a private clinic and patients paid regular fees to avoid secondary benefits. Treatment consists of administering AAI followed by ultrasound. For the administration of the AAI and ultrasound, patients were seated on a treatment table with the backrest inclined 30° backward; the involved shoulder was allowed to fall outside the treatment table to obtain a shoulder extension of 30° and to provide full exposure of the rotator cuff. A 48-cm^2 carbon rubber electrode was connected to the negative pole (active electrode) as specified in previous studies[3,4,11,14] for AAI. The cathode was then inserted into a sponge soaked in a 5% acetic acid solution[14] and fixed to the area to be treated with an elastic bandage. The hand of the uninvolved arm was placed away from the anode (indifferent electrode) in a bath of tap water. A Dynatron 406[a] was used to produce a galvanic current; current amplitude was set to 5mA, which corresponds to a current density of less than 1mA per square inch as recommended for iontophoresis.[16] Iontophoresis was administered during 20 minutes as previously described.[5,9,14] Following AAI, continuous ultrasound using a Sonopuls 434[a] was applied over the same area; the patient was maintained in the same position. The frequency of ultrasound was set at 1Mhz to reach 2 to 4 cm in depth,[11,17] and the ultrasound was delivered during 5 minutes at an intensity of .8W/cm^2.

Data reduction and statistical analyses. The area and the density of the CD were measured. Each X-ray film was examined by the radiologist and the density of the CD was scored on a scale from 0 (no trace) to 4 (maximal density). The periphery of the CD was also traced onto a transparency. To ensure a fair comparison over time, for each patient, the film incidence with superimposable bony landmarks and the largest CD was

Table 1: Subject Characteristics

	Experimental Group (n = 11)	Control Group (n = 10)
Age (yrs)*	43 (32-57)	40 (33-50)
Gender	M, 4; F, 7	M, 2; F, 8
Type of lesion	I, 3; II, 8	I, 2; II, 8
Onset (mos)*	45 (0.2-180)	31 (0.5-120)
Shoulder	Dominant, 3	Dominant, 7

* Mean (range).

chosen for measurements. The area of the CD was estimated using a manual planimeter. For all variables, the mean of three measurements was used in the final analysis. Descriptive statistics (means and standard deviations) were used for both radiological and functional variables. Differences over time between the EXP and CTL groups, in shoulder function and CD, were determined using an analysis of covariance (ANCOVA) for repeated measures. The effect of the type of lesion on the radiological and functional change scores in percent (post-pre/pre × 100) was studied using the Mann-Whitney U test. Finally, the Spearman correlation coefficient was used to study the relationship between radiological and functional changes. The level of statistical significance was set at .05.

RESULTS

Forty-five patients were screened from June 1990 to December 1994. Table 1 summarizes data for 21 of 22 patients (49%) enrolled in the study. Although all 22 patients completed the study, results from one patient were rejected because the incidence of X-ray films taken at each evaluation did not allow a fair comparison of the CD area. The analysis included 21 patients who were randomized to the EXP (n = 11) or CTL (n = 10) groups. The type II lesions occurred more frequently (n = 16: 8 in each group), than type I lesions (n = 5: 3 and 2 in the EXP and CTL groups, respectively). Patient age and gender were comparable for the two treatment groups. The duration of symptoms varied markedly in both groups (Table 1). The median was 18 and 24 months, respectively, for the EXP and CTL groups, and there was no statistical difference between groups for the duration of symptoms.

Effect of Treatment

Figure 1 illustrates the mean values (and SD) for the radiological variables (area and density of the CD) in the EXP and CTL groups during the 3-week study. At baseline, the mean area and density of the CD were similar in both groups. Thereafter, there is a clear reduction in the area (p = .01) and density (p = .03) of the CD compared to baseline in both treatment groups; there was no difference between EXP and CTL groups. Mean values (and SD) for shoulder function over time for both groups are shown in figure 2. The ROM of passive abduction tended to increase (p = .07) and pain was slightly reduced but this trend was similar in both groups. Statistical differences were not detected between the EXP and CTL groups.

The mean percent change over time (post-pre/pre) in area of the CD was 20% (±29%) and 36% (±43%), respectively, for the EXP and CTL groups, while the gain in passive abduction was, respectively, 36% (±21%) and 36% (±69%). The large standard deviations, however, indicate that the percent change across subjects within each group was quite variable. These results led us to investigate the effect of the type of lesion, which is a factor sensitive to time and may help in understanding the present results.

Influence of Lesion Type

We stratified for the type of lesion; therefore, patients with type I or type II CD lesions were equally distributed in each group. Because a statistical difference was not detected between EXP and CTL groups, patients were then distributed according to lesion type irrespective of the treatment group. Figure 3 illustrates the percent change over time (post-pre/pre) for the area of the CD and ROM of passive shoulder abduction for type I and type II groups. The percent change in CD area in the type I group was greater (p = .01) than in the type II group. The difference for shoulder abduction was not significant. The influence of lesion type is further illustrated in figure 4, which shows individual percent reductions in area of CD. On the left, note that in patients with type I (squares) lesions, the changes ranged from 30% to 100%, without distinction between patients in the EXP (open squares) or CTL (filled squares) groups. In patients with type II lesions (triangles), the changes were generally (12 of 16) less than 30%; 3 of the 4 patients with changes above 40% were from the CTL (filled triangles) group.

To further determine how type I and type II groups compared in terms of functional recovery, statistical analyses were repeated following inclusion in the type I group of the four patients who evolved from an initial type II lesions to type I lesions (identified by an asterix on fig 4). As such, in the second analysis the type I group includes all patients (n = 9) with a decrease in the area of the CD. As shown in the bar graphs of

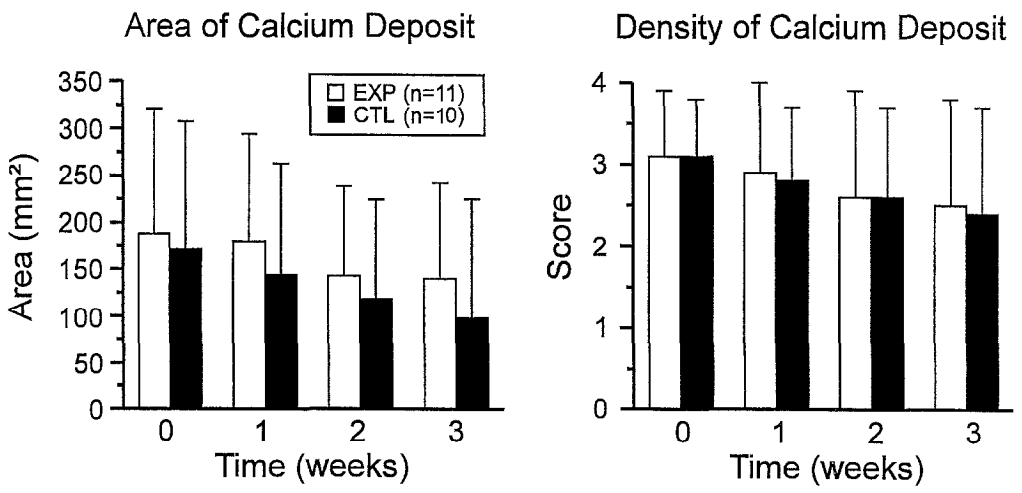

Fig 1. Mean values in area and density of calcium deposit for patients in the experimental (EXP) and control (CTL) groups, at baseline (week 0) and thereafter at 1-week intervals for 3 weeks. Significant reduction in area (p = .01) and density (p = .03) was observed over time in both groups. Vertical bars indicate one standard deviation.

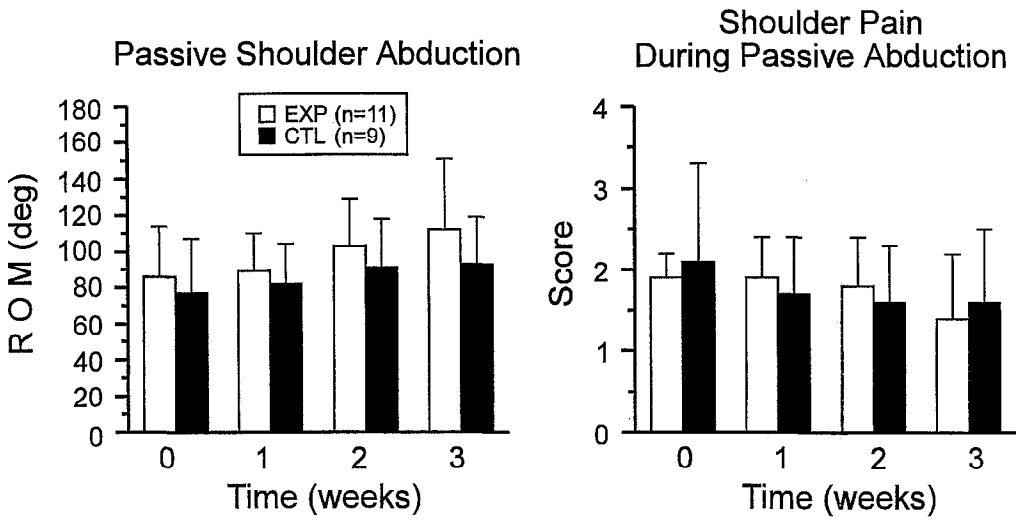

Fig 2. Mean values in the range of motion (ROM) during passive abduction to the point of pain and pain intensity in the experimental (EXP) and control (CTL) groups, at baseline (week 0) and thereafter at 1-week intervals for 3 weeks. Nonsignificant gain (p = .07) in ROM was found in both groups and pain was slightly reduced in both groups. Vertical bars indicate one standard deviation.

figure 5, this analysis emphasized the differences (p = .0002) in the changes of CD area between the type I and type II groups, and further demonstrates that the type of lesion does not influence the gain in shoulder abduction.

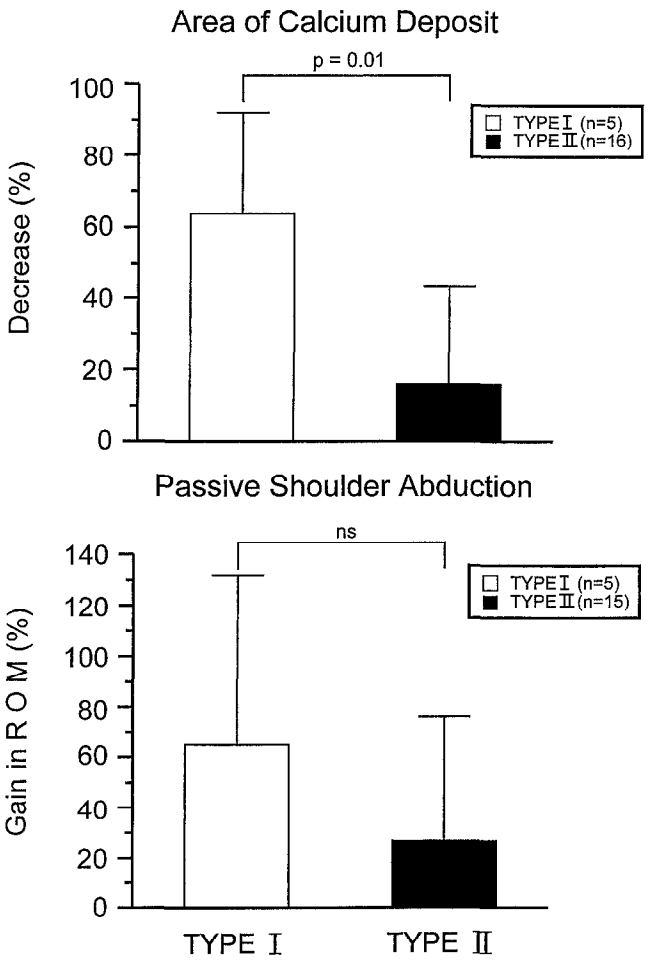

Fig 3. Mean percent decrease in calcium deposit area and percent gain in ROM over the 3-week period in type I and type II groups. Vertical bars indicate one standard deviation.

Correlation Between the Radiological and Functional Variables

Spearman correlation coefficients for radiological (area and density of the CD) and functional (ROM of passive abduction and pain) variables are summarized in table 2. The strongest correlations ($p < .003$) were found between area and density of CD (r_s = .90) and between ROM and pain (r_s = −.67). Weaker correlations (r_s = −.25 to .41) were found between radiological (area or density) and functional (ROM and pain) variables. These results indicate that when the area of the CD is reduced, density is also expected to decrease, and that a gain in shoulder ROM is associated with a reduced level of pain. On the other hand, a gain in shoulder ROM does not lead to a reduction in CD area and vice-versa. In fact, the relationships between radiological and functional variables at baseline and after treatment were very weak (r_s = .25 to .31).

DISCUSSION

Results of the present study indicate that after nine treatments with AAI and ultrasound, patients (EXP) with calcifying tendinitis of the shoulder did not show more resorption of the CD or a better recovery of shoulder function than patients with no treatment other than rest (CTL). Findings that resorption of the CD was significant over time in both patient groups suggest that the changes in size and density of the CD resulted from some natural process. On the basis of our findings and within the parameters of the present study, the combined use of AAI and ultrasound cannot be recommended for the treatment of calcifying tendinitis of the shoulder.

In this study, 43% (9 of 21) of the patients displayed some reduction (larger than 30%) in the CD area. This incidence is close to the 55% reported for a group of 135 patients diagnosed with calcifying tendinitis of the shoulder who had received AAI for 6 weeks.[4] Others have reported a reduction of the CD in 75% (9 of 12) of patients administered AAI for 3 to 4 months.[3] In light of the present study, resorption of the CD reported in previous uncontrolled studies most likely resulted from the natural evolution of the CD and can hardly be attributed to AAI. The larger incidence of patients with resorption of CD in previous studies[3,4] could be explained by several factors, such as time (both the number of treatments and the time elapsed between evaluations were longer), methods of measurement, and type of lesions. Factors such as time and type of lesions will influence the magnitude of the CD resorption measured, whereas criteria

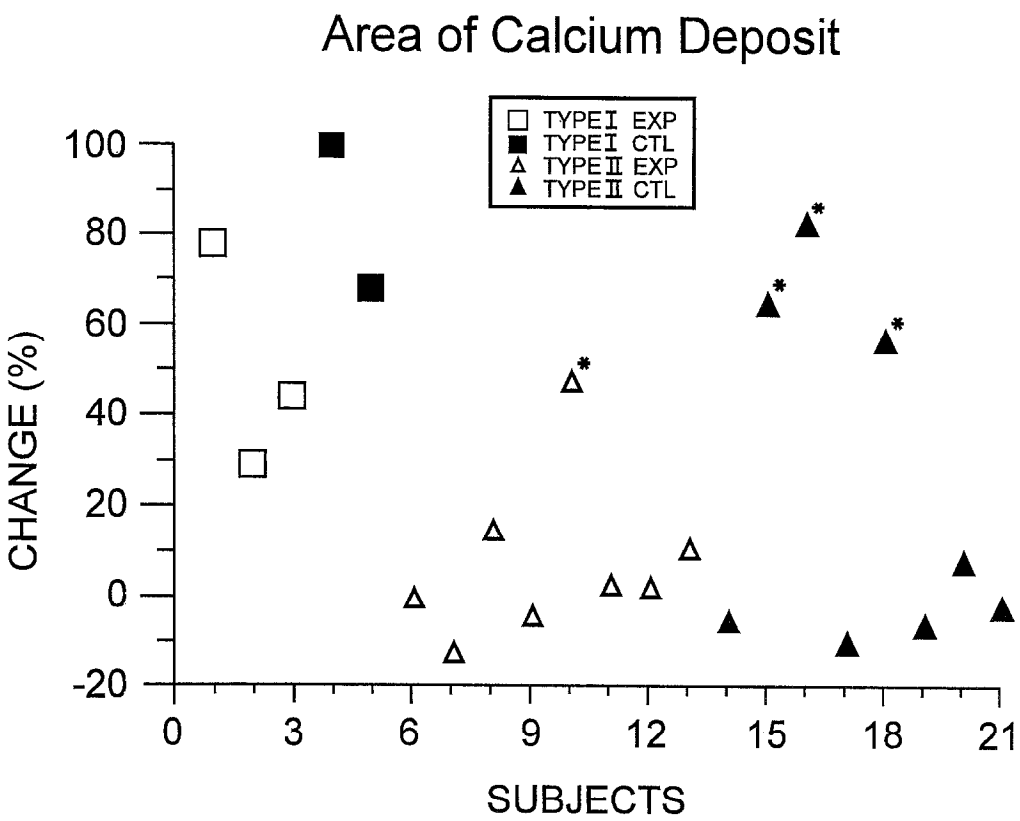

Fig 4. Individual percent change in the area of calcium deposit (CD). On the left, patients (n = 5) with type I lesions (squares), on the right, patients (n = 16) with type II lesions (triangles). Triangles with an asterix indicate the patients whose CD evolved from a type II to a type I lesion. Note that the patients with type I and type II lesions were equally distributed in the experimental (open symbols) and control (filled symbols) groups.

and type of measures will influence the incidence of CD resorption. These methodological differences and the absence of control groups in previous studies[3,4] likely explain why our conclusions are at variance with those of others who have advocated using AAI to reduce CD in the management of calcifying tendinitis of the shoulder.

The rationale underlying the use of AAI remains unclear. To our knowledge, there is no scientific evidence on the capacity of this molecule to be delivered through the skin or to bind with heterogeneous materials that make up the CD.[2] Moreover, the reduced capacity of acetic acid to dissociate in water[18] may be partly responsible for the lack of effect of AAI on CD resorption. It would be of interest to study the effect of other molecules that can easily dissociate in their solvant, such a property being critical for successful iontophoresis. The effect of ultrasound as an adjunct to AAI remains unknown. Results of the present study, however, confirm earlier findings that ultrasound does not promote resorption of the CD.[10] Lastly, there are no data suggesting that ultrasound after AAI might mask the effects of AAI.

An important finding of the present study is that, in addition to time, the type of lesion affects the magnitude of CD resorption and that stratification for the type of lesion may be critical to future clinical trials. In all patients with a type I lesion the CD was resorbed; the decrease in CD area ranged from 35% to 100%, with a mean of 63.3% (±21.7%). In contrast, in patients with a type II lesion, the mean decrease in CD area was 16% (±30%); this slight decrease occurred primarily in the four patients with a lesion that evolved from type II to type I (Figure 4). These results support the findings of others[1,2] to the effect that the appearance of the CD evolves from type II to type I. In this study, results from X-ray films obtained at 3-week intervals further documented that this evolution could occur within a relatively short period. Clinically, the type of calcification observed on X-ray film should assist in the prognosis of calcifying of the shoulder and therefore guide the management of this condition.

Radiological and Functional Correlations

The results of this study show that there was a very high and significant correlation between changes in the area and density of CD, indicating that as the size of the CD decreases so does its density. These results support the idea that during the resorptive stage, there is also a decrease in the density of the calcific material that migrates from the tendon to the subacromial space[1]; such transformations eventually result in the fleecy appearance of the CD that characterizes type I lesions. Thus, changes in either the area or density of CD could be used to determine the stage of evolution of the CD. Another finding that has a clinical impact is the weak relationship between changes in radiological measures and shoulder function. Present results indicate that the ROM does not necessary improve with a resorption of the CD, which suggests that factors other than the size of the CD are likely associated with the functional limitations observed with calcifying tendinitis of the shoulder. For instance, congestion in the subacromial space and/or disuse resulting from inflammatory processes could be responsible for the reduced function seen in patients with calcifying tendinitis of the shoulder.

In conclusion, this study clearly demonstrates that AAI and ultrasound do not promote the resorption of CD and do not improve shoulder function in patients with calcifying tendinitis of the shoulder. These results and the lack of rationale for using AAI strongly support the need to look for other noninvasive approaches in the management of calcifying tendinitis of the

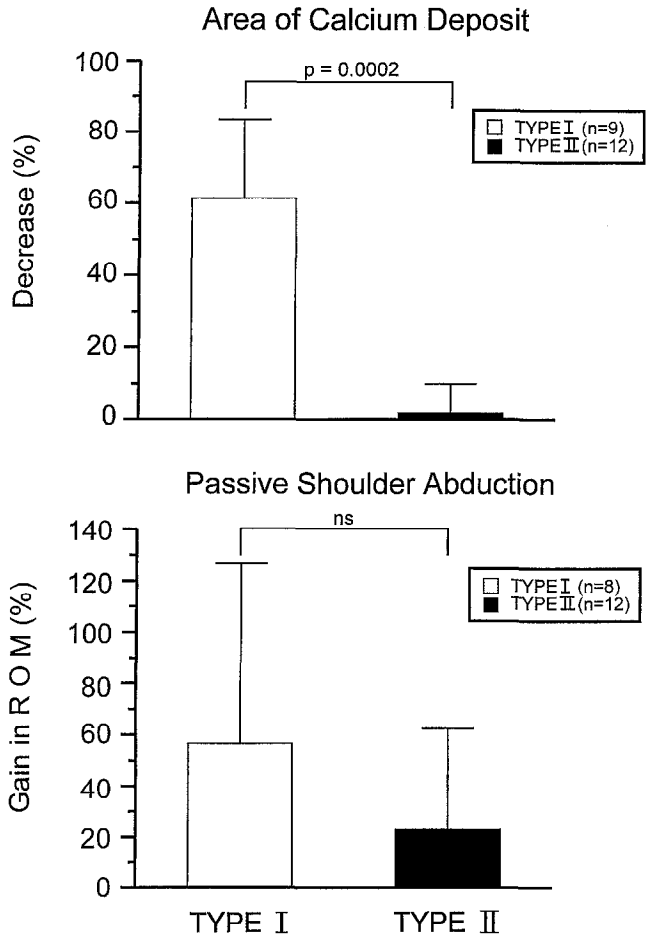

Fig 5. Mean percent decrease in calcium deposit (CD) area and percent gain in ROM over the 3-week period in type I and type II groups. Type I group (n = 9) includes the four patients whose CD evolved from type II to type I lesion. Vertical bars indicate one standard deviation.

shoulder. In the meantime, perhaps the most appropriate approach would be to use ice, transcutaneous electrical nerve stimulation, or antiinflammatory drugs to control acute symptoms generally seen with type I lesions.[1] These could be combined with procedures (ROM exercise and mobilization) to progressively restore ROM. When subacute or chronic symptoms are concomitant with a confirmed type II lesion, procedures to reduce impingment of the CD against the coracoacromial arch (postural control, accessory mobilization, muscle stretching and strengthening) could be considered.

Table 2: Spearman Correlation Coefficients (n = 21)

	Area	Density	ROM	Pain
Area	—	.90*	−.25	.39
Density		—	−.23	.41
ROM			—	−.64*
Pain				—

* $p < .003$.

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a. B.V. Enraf-Nonius Delft, Röntgenweg 1—PO Box 483, 2600AL Delft, The Netherlands.