Delayed Anticipatory Muscle Activation in Rotator Cuff Tendinopathy

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Background: Previous research investigating rotator cuff (RC) tendinopathy has usually focused on pathoanatomy. The pathologic response to anticipatory postural adjustments (APAs) has not yet been investigated.

Purpose/Hypothesis: To explore changes in APAs as detected by pre-emptive activation of shoulder muscles during ball catching. It was hypothesized that anticipatory muscle activation (AMA) would be present in the unaffected shoulder but delayed or absent in the affected shoulder in patients with RC tendinopathy.

Study Design: Controlled laboratory study.

Methods: This study included 21 RC tendinopathy patients with a mean age of 49.5 years. Patients were required to grab a ball embedded with an electromyography sensor when it dropped on their hand, and surface electromyography signals were recorded from the infraspinatus, upper trapezius, anterior deltoid, and biceps. The trials utilized 2 balls, weighing 200 g and 500 g. Each ball was used in 2 trials, 1 involving a number count preceding the ball drop (predictable) and the other involving a sudden drop (unpredictable). The onsets of AMA between the affected and unaffected limbs were compared.

Results: Regardless of the experimental condition, significantly delayed AMA onsets were identified in all investigated muscles of the affected side compared with those of the unaffected side, except for the biceps muscle in the 500-g predictable trial. For the infraspinatus, the mean onset time in the 200-g predictable trial was -141.0 ± 60.2 ms on the affected side and -211.9 ± 67.1 ms on the unaffected side ($P < .001$); in the 200-g unpredictable trial this value was -139.5 ± 54.9 ms on the affected side and -199.5 ± 56.2 ms on the unaffected side ($P < .001$).

Conclusion: Delayed AMA was observed in the affected shoulder compared with the unaffected shoulder in patients with RC tendinopathy, not only in the RC muscle but also in the periscapular and upper arm muscles. This may indicate that central hypoexcitability is partly responsible.

Clinical Relevance: The basis for RC tendinopathy treatment should not be limited to the tendon pathoanatomy. Delayed AMA around the shoulder joint could provide insight into potential mechanisms related to the central nervous system.

Keywords: rotator cuff; tendinopathy; anticipatory; postural adjustments; electromyography

Rotator cuff (RC) tendinopathy is a common shoulder issue that causes pain and disability, thus lowering health-related quality of life.[15,28,30] Previous research investigating RC tendinopathy has usually focused on the pathoanatomy at the joint level, such as tissue pathology, altered shoulder biomechanics, and muscular deficit.[8,13,14] Although these studies have increased the understanding of the nature of RC tendinopathy, treatment based on pathoanatomy at the joint level has failed to treat more than a third of patients.[27] Recently, the hypothesis has been raised that reorganization in the brain could explain part of the deficits associated with RC tendinopathy.[21,22,26,29] If some of the deficits experienced by patients with RC tendinopathy stem from neural changes, the clinical approach should be broadened to incorporate interventions, such as sensorimotor training aimed at the reversal of neural changes. Human motor control has been shown to exhibit a feedforward and future-oriented approach.[20,32,34] Anticipatory postural adjustments (APAs) are defined as the activation of postural muscles in a feedforward manner before voluntary movement begins.[33] APAs are presumed to estimate the stability-perturbing forces to be generated by an impending focal action and produce pre-emptive muscle activations to maintain body balance in advance of the perturbation.[1,17] The pathologic response in APAs has usually been investigated in people with low back pain.[10] Delayed muscle onset in response to unexpected perturbations has been observed in people with low back pain compared with those of healthy individuals.[7,16,24] Previous studies have demonstrated that APAs also occur during

ball catching to prepare the upper limb for ball impact.[11,12,31] Upper limb muscles are known to produce bursts of activation between 100 and 200 ms before ball impact.[12] In this context, investigation of APAs in the ball-catching action may reveal changes in onset time of shoulder muscles in people with shoulder pain.

This study aimed to measure the onset of activation of 4 different muscles around the shoulder joint during ball catching. Emphasis was focused on the detection of anticipatory infraspinatus activation in the affected and unaffected shoulders in patients with RC tendinopathy. We hypothesized that anticipatory muscle activation (AMA) would be present in the unaffected shoulder but delayed or absent in the affected shoulder in patients with RC tendinopathy.

METHODS

Study Design

This study was designed to compare the AMA between bilateral shoulders in patients with RC tendinopathy. The study protocol was approved by our institutional review board, and informed consent was obtained from all patients. As there were no previous studies investigating AMA in patients with tendinopathy, we conducted a power analysis for the hypothesis by virtue of a difference in AMA of the infraspinatus during a 200-g ball drop in our preliminary data. Given a mean difference of 0.055, a minimum of 17 participants were required to reject the null hypothesis at alpha = .05 (2-tailed) with a power of 0.95. The present study included 5 male and 16 female patients.

Patient Selection

All patients had undergone ultrasonography or magnetic resonance imaging to diagnose RC diseases in the affected shoulders. Only patients with posterosuperior RC tendinopathy and positive findings on Neer and Hawkins tests were included in this study. Exclusion criteria were as follows: (1) shoulder pain for <6 months; (2) previous neurological or musculoskeletal surgeries on the neck, back, and upper extremities; (3) pain or discomfort on the contralateral shoulder; (4) positive findings in physical examinations to evaluate RC diseases on the contralateral shoulder; (5) underlying systemic diseases such as rheumatoid arthritis, hyper- or hypothyroidism, or diabetes mellitus; and (6) difficulty in carrying out imposing tasks.
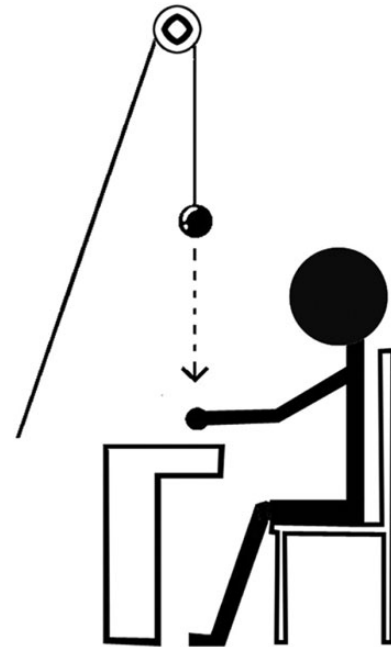
Figure 1. A ball embedded with an electromyography sensor was dropped from a height of 0.75 m. The participant grabbed the ball in the sitting position with the examined forearm on a table.

Clinical Assessments

Five clinical measures were evaluated before the electromyography (EMG) procedure in this study: the visual analog scale (VAS) pain score, the American Shoulder and Elbow Surgeons (ASES) score, the Constant score, and muscle strength in abduction and external rotation.

Procedures

We prepared 200-g and 500-g balls, each with a diameter of 10 cm, in which an EMG sensor was embedded. The 2 balls were made visually identical by concealing them with black elastic covers. A string was attached to each ball and extended to a loop mounted on the wall above the patient's head and down to the examiner's hand. Patients sat in an armless chair and placed the examined forearm over a pad on a table with the palm up. When the examiner released the string, the patient grabbed the ball falling from a height of 0.75 m directly above the hand (Figure 1). We recorded the ball impact instant using the EMG system. Time zero

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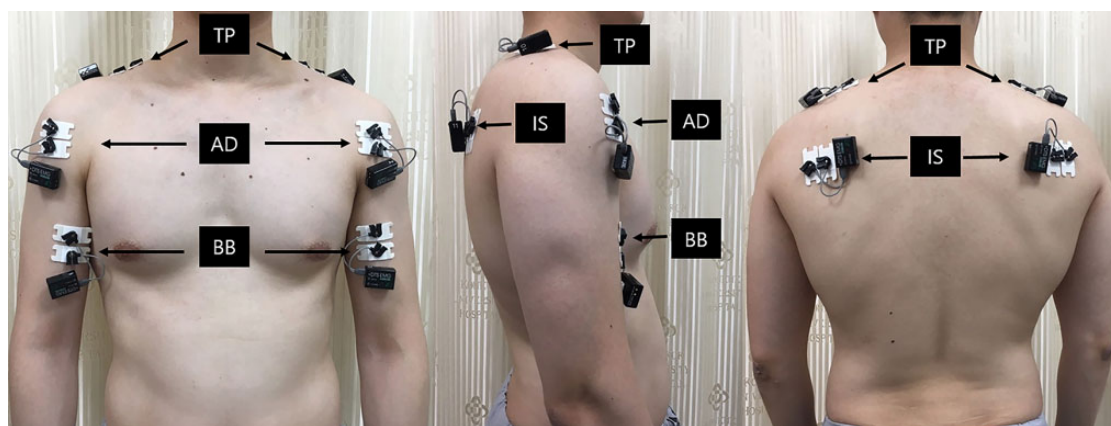†Department of Orthopaedic Surgery, Konkuk University School of Medicine, Seoul, Republic of Korea.
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Figure 2. Locations of electromyography electrodes for each muscle. AD, anterior deltoid muscle; BB, biceps brachii; IS, infraspinatus muscle; TP, upper trapezius muscle.

(T_0; moment of ball impact on the hand) was estimated from the signal of the EMG sensor embedded in the ball. When the ball made contact with the hand, a clear signal was produced.[31] This study combined 2 types of trials with each of the balls, 1 involving a number count signaling when the ball would fall (predictable) and the other involving a sudden drop without counting (unpredictable). Before measuring EMG, all patients were given several trials to practice catching a falling ball without any pain. Therefore, a total of 4 trials each were conducted for the affected and unaffected sides. For each trial, the ball-release procedure was performed 4 times, and the mean value of the AMA onset time was used for the analysis.

EMG and Work Data

Surface EMG signals were recorded from the infraspinatus, upper trapezius, anterior deltoid, and biceps using a Telemyo DTS Desk Receiver (Noraxon USA). Four muscles were selected because they are well-activated during ball catching in the sitting posture, with the forearm extended on a table. The placement of electrodes for each muscle was determined in line with recommendations reported in the literature.[5,6] To minimize skin impedance, we prepared the skin surface by cleaning the area with an alcohol swab before placing the electrodes. Bipolar surface EMG electrodes with an interelectrode spacing of 2 cm were placed over the muscle belly in alignment with the muscle fibers (Figure 2). All data processing was performed using MR3 software (Version 3; Noraxon USA). The signals were amplified with a gain of 400, noise <1 μV, and common mode rejection ratio of 100 dB. They were sampled at 1500 Hz and filtered with a bandwidth of 10 to 500 Hz. To construct a linear envelope, we performed full-wave rectification. A Lancosh finite impulse response digital filter (Noraxon USA) was used to filter the raw signal. The bandpass filter frequency was between 10 and 350 Hz. We processed the EMG data into the root mean square value in 200-ms windows. The AMA for a specific muscle was defined as the instant lasting for at least 50 ms when its

EMG amplitude was greater than the mean ± 2 SD of its baseline value, measured from –500 to –400 ms.[2]

Statistical Analysis

Data are presented as mean ± SD. Comparison of AMA between the affected and unaffected shoulders was performed using paired Student t tests. One-way repeated-measures analysis of variance (ANOVA) was performed to detect differences in AMA between the trials for each shoulder. Statistical analysis was performed using SPSS software (Version 17.0; SPSS). The level of significance was set at $P < .05$.

RESULTS

The mean age of the 21 study participants was 49.5 years, and the mean height, weight, and body mass index were 162.9 cm, 62.1 kg, and 23.2 kg/m^2, respectively. The dominant shoulders were involved in 12 of 21 patients, while the remaining 9 shoulders were nondominant. The patients' mean VAS pain score, ASES score, Constant score, and muscle strength in abduction and external rotation were 5.6, 52.9, 62.0, 13.4, and 7.5 kg, respectively.

Predictable Ball-Drop Trials

The difference in AMA onset between the affected and unaffected shoulders was statistically significant for all muscles in the predictable 200-g and 500-g trials except for the biceps muscle in the 500-g trial (Table 1 and Figure 3). The mean onset time across the 4 muscles was as follows: 200 g, affected: 0.42 ± 0.04 ms, unaffected: 0.41 ± 0.05 ms; 500 g, affected: 0.38 ± 0.64 ms, unaffected: 0.39 ± 0.02 ms.

Unpredictable Ball-Drop Trials

Similar to the predictable trial, significant differences were noted in all of the unpredictable ball-drop trials between the affected and unaffected shoulders (Table 2

and Figure 4). The mean onset times across the 4 muscles were as follows: 200 g, affected: 0.43 ± 0.05 ms, unaffected: 0.42 ± 0.03 ms; 500 g, affected: 0.73 ± 1.22 ms, unaffected: 0.37 ± 0.03 ms. Although the difference between the sides for the biceps in the 500-g trial was statistically significant, the *P* value was near .05 (*P* = .049).

TABLE 1
AMA Onset Times for the Predictable Ball-Drop Trials[a]

Muscle	AMA Onset, ms		
	Affected Side	Unaffected Side	*P* Value
200-g trial			
Infraspinatus	−141.0 ± 60.2	−211.9 ± 67.1	**<.001**
Upper trapezius	−118.6 ± 80.8	−170.5 ± 89.4	**.024**
Anterior deltoid	−129.0 ± 83.8	−202.4 ± 88.5	**.002**
Biceps brachii	−97.6 ± 60.4	−142.4 ± 78.7	**.007**
500-g trial			
Infraspinatus	−142.6 ± 39.3	−183.2 ± 58.3	**.003**
Upper trapezius	−111.6 ± 52.7	−165.3 ± 63.2	**<.001**
Anterior deltoid	−140.5 ± 50.2	−179.5 ± 63.5	**.040**
Biceps brachii	−113.2 ± 52.8	−141.6 ± 65.5	.087

[a]Data are reported as mean ± SD. Bolded *P* values indicate a statistically significant difference between sides (*P* < .05). AMA, anticipatory muscle activation.

Influence of Experimental Conditions

One-way repeated-measures ANOVA indicated no statistical difference in AMA among the 4 trials (200 g predictable and unpredictable and 500 g predictable and unpredictable) for the affected and unaffected shoulders.

DISCUSSION

This study aimed to investigate differences in the onset of anticipatory activation of 4 muscles around the shoulder joint between the affected and unaffected shoulders in patients with RC tendinopathy. The key finding of this study was that, regardless of experimental conditions (ball weight or prediction of ball drop), delayed onsets were identified in all investigated muscles of the affected side in contrast to those of the unaffected side. This indicates that the feedforward mechanism is altered in the regulation of response during anticipated conditions in patients with RC tendinopathy. Four muscles were selected because they are well-activated during ball catching in the sitting posture, with the forearm extended on a table. Therefore, these muscles were chosen to demonstrate APAs in our experimental setting.

Motor-control alterations have been suggested as a reason for the periscapular and RC muscle activity changes that are associated with impingement syndrome.[9] Delayed APAs may reflect altered motor control of the shoulder joint in patients with RC tendinopathy. Research on how APAs
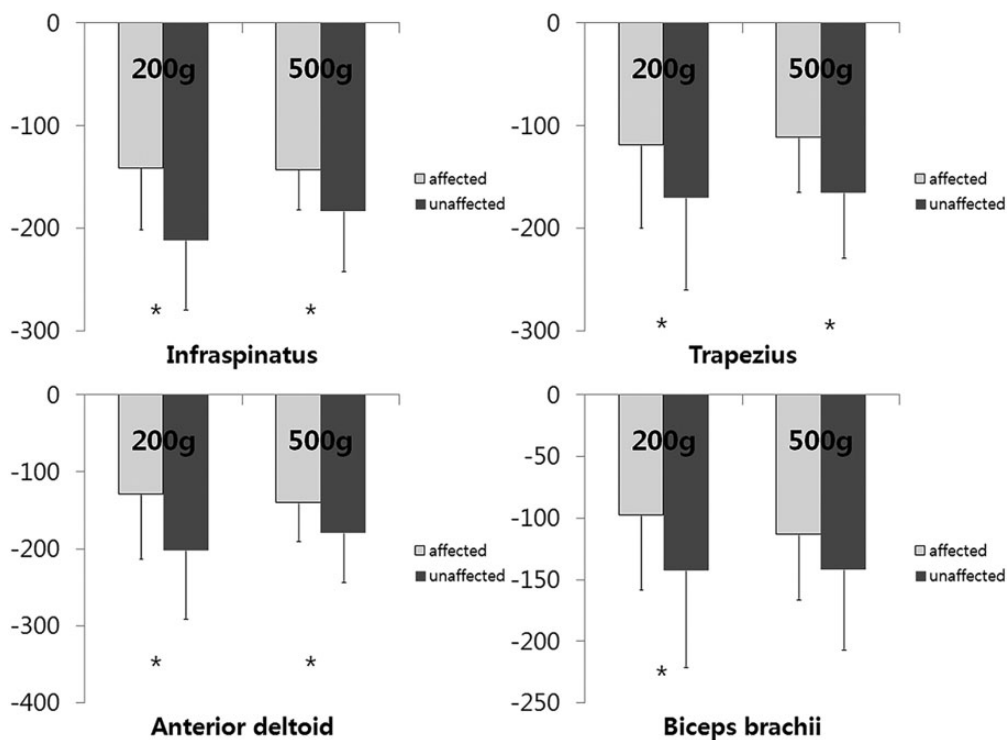
Figure 3. Anticipatory muscle activations: responses of predictable trials. *Statistically significant difference between the affected and unaffected sides (*P* < .05).

of the shoulder joint are affected under RC tendinopathy is scarce. Studies so far have measured muscle activities during active arm movements in response to external signals. For instance, Phadke et al[23] introduced the relative latency concept to evaluate feedforward activation of periscapular muscles. They calculated relative latency as a difference of examined muscle latency relative to that of the anterior deltoid, which is the primary mover. They found a relatively earlier onset of the upper trapezius in participants

TABLE 2
AMA Onset Times for the Unpredictable Ball-Drop Trials[a]

| Muscle | AMA Onset, ms | | |
	Affected Side	Unaffected Side	*P* Value
200-g trial			
Infraspinatus	−139.5 ± 54.9	−199.5 ± 56.2	<**.001**
Upper trapezius	−109.5 ± 73.8	−160.0 ± 78.6	**.009**
Anterior deltoid	−138.6 ± 68.7	−188.1 ± 79.7	**.008**
Biceps brachii	−89.0 ± 46.1	−156.2 ± 73.1	<**.001**
500-g trial			
Infraspinatus	−129.5 ± 42.0	−178.4 ± 62.3	**.001**
Upper trapezius	−112.6 ± 48.7	−159.5 ± 75.7	**.014**
Anterior deltoid	−132.1 ± 49.5	−198.9 ± 85.8	**.003**
Biceps brachii	−109.5 ± 39.9	−142.1 ± 55.8	**.049**

[a]Data are reported as mean ± SD. Bolded *P* values indicate a statistically significant difference between sides (*P* < .05). AMA, anticipatory muscle activation.

with impingement syndrome compared with that of healthy participants. However, in that study, the absolute onset of muscle activation was delayed in all investigated muscles (upper trapezius, serratus anterior, and anterior deltoid) except the lower trapezius in participants with impingement syndrome in contrast to those of healthy participants. Moraes et al[19] also noted delayed absolute onsets of the upper, middle, and lower trapezius and the serratus anterior of the affected side in contrast to those of the unaffected side during active elevation in participants with impingement syndrome.

Our experimental setting was different from those of the above studies in that we measured muscle activities while maintaining balance against an external force. Discussing the difference between AMAs, which was evaluated in this study, and muscle latency during active movement evaluated in other studies is beyond the scope of this study. What is relevant to this study is that anticipatory shoulder muscle activities were delayed in impingement syndrome. Because of the specificity of our experiment, we did not investigate certain functional muscle groups separately, such as RC muscles or periscapular muscles, as recruitment pattern change was not the main concern of our research.

Overall, delayed AMAs around the shoulder joint may indicate a problem with the control function of the central nervous system. Studies on central reorganization in RC diseases may provide a clue to understanding our results of delayed APAs. In recent decades, there has been an increasing amount of research suggesting that maladaptive
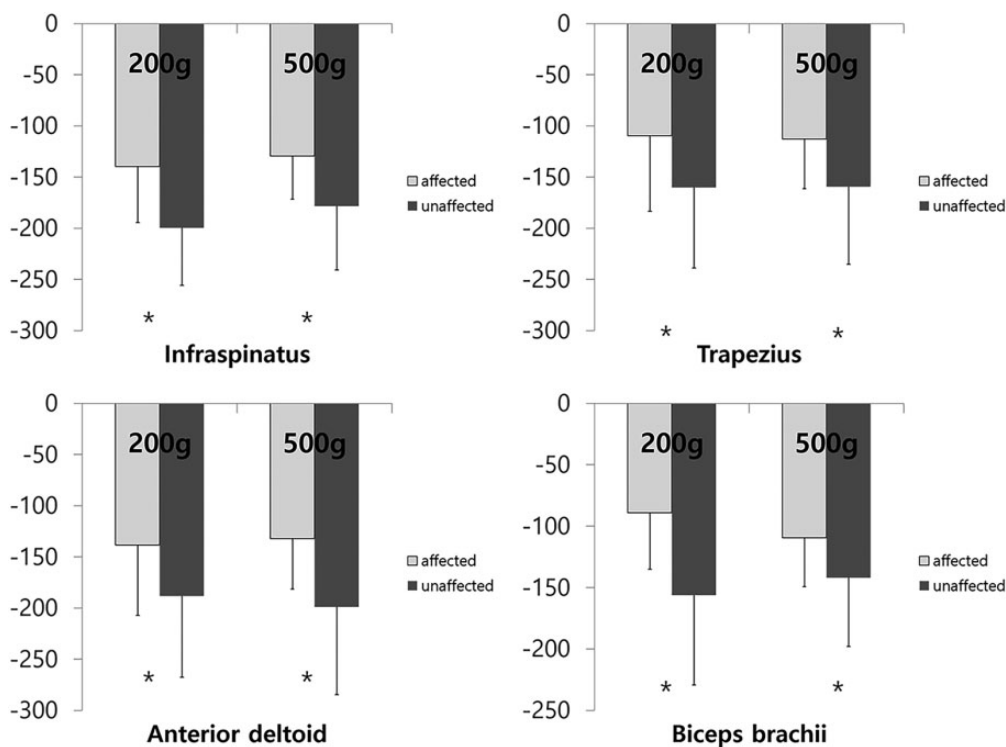

Figure 4. Anticipatory muscle activations: responses of unpredictable trials. *Statistically significant difference between the affected and unaffected sides (*P* < .05).

reorganization of the brain is a prominent characteristic in the cause of chronic musculoskeletal pain. Regarding RC diseases, 2 studies have reported alterations in the corticospinal excitability of shoulder muscles. Ngomo et al[22] found interhemispheric asymmetry of the infraspinatus active motor threshold in patients with RC tendinopathy. A higher intensity of stimulation was required to evoke infraspinatus muscle response on the affected side compared with that of the unaffected side. Another study on adaptive changes in the motor cortex revealed corticospinal hypoexcitability of the deltoid muscle during voluntary motion in patients with chronic full-thickness RC tears.[3] The authors attributed it to altered afferent input from the glenohumeral joint, which causes a central reprogramming of the cortical excitability and representation of the deltoid muscle. Despite the limited number of these findings, it is still presumable that central hypoexcitability is partly responsible for delayed APAs in shoulder muscles. Through these studies, it is necessary to consider rehabilitation with brain activity. Rio et al[25] investigated an efficient way of rehabilitation by investigating the correlation between pain and cortical inhibition. They found that a reduction in pain was paralleled by a reduction in cortical inhibition. The authors recommended isometric exercise using 70% of maximal voluntary isometric contraction as a more ideal rehabilitation protocol compared with isotonic exercise based on a reduction in cortical inhibition. Until now, no studies on brain activities have investigated rehabilitation protocol for shoulder joint. Future studies should be conducted to find an ideal shoulder rehabilitation protocol that exploits brain activities.

There are several limitations of this study that warrant review. First, there was no control group comprising participants with painless shoulders. We designed the study in accordance with the interhemispheric asymmetry of the infraspinatus active motor threshold in patients with RC tendinopathy. Since the experiment was conducted in static posture, there was little possibility that interaction across shoulders would affect the outcomes. Evaluation of differences between affected and unaffected shoulders may provide clues to the maladaptive reorganization of the brain. Second, we adopted surface EMG to investigate muscle activation, whereby activation of supraspinatus muscle could not be directly measured. Instead, the infraspinatus muscle, which is easy to measure because it is not covered by other muscles, was investigated to reduce patients' resistance caused by needle EMG investigation. Mochizuki et al[18] reported that the greater tuberosity is actually occupied by a substantial amount of the infraspinatus tendon. It is best to measure the supraspinatus activation invasively. However, measuring infraspinatus activation may alternatively be considered in the clinical setting. Third, intergroup differences in all evaluated muscles were small, and the standard deviations were wide, although these are statistically significant. A concern may be raised as to whether they are meaningful clinically. Actually, it has been suggested to use task-specific performance to overcome small intergroup differences in impingement syndrome.[4] We think a drop-ball apparatus is useful to measure muscle activations, whether anticipatory or not,

without brain reaction caused by pain anxiety in impingement syndrome. Future studies will be needed to clarify whether these delayed APAs reflect pathologic tendon conditions or brain reorganization.

CONCLUSION

APAs were observed in all investigated muscles. APAs in the affected shoulder were delayed in contrast to those of the unaffected shoulder in patients with RC tendinopathy. Delayed onsets occurred not only in the RC muscle but also in the periscapular and upper arm muscles, which may imply that central hypoexcitability is partly associated with delayed APAs in shoulder muscles.

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