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MUSCULAR ACTIVATION PATTERNS IN SWIMMERS WITH ASYMPTOMATIC SHOULDER JOINT INSTABILITY

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ABSTRACT
Purpose. The aim of this study was to investigate the (electromyographic) EMG patterns of the shoulder muscles in asymptomatic swimmers with AGI – atraumatic glenohumeral instability. AGI, one of the most common shoulder disorders in athletes who perform overhead activities, occurs especially among swimmers. AGI usually provokes shoulder pain and secondary impingement syndrome, which might alter glenohumeral and scapular kinematics and change the activity of the shoulder muscles. Alternatively, pain or functional activities might affect EMG patterns in individuals with AGI. **Methods.** Eight swimmers with AGI and eight healthy swimmers took part in this cross-sectional study. Bilateral and simultaneous shoulder elevations in the scapular plane toward three different target distances were investigated, and the shoulder kinematics and EMG activities of the glenohumeral and scapulothoracic muscles were also collected. **Results.** No differences in the EMG patterns were found between swimmers with and without AGI in terms of the rate of EMG rise and magnitude. **Conclusions.** Shoulder instability does not necessarily affect the modulation of the shoulder muscles in swimmers with AGI. Others factors such as laxity in the capsular structures and ligaments may be one of the primary reasons for pain and instability in these athletes.

Key words: shoulder electromyography, shoulder instability, motor control, impingement syndrome

Introduction

Glenohumeral instability, one of the most common pathologies of the shoulder joint [1], is usually classified as traumatic or atraumatic glenohumeral instability (AGI). AGI generally provokes shoulder pain and secondary impingement syndrome [2], which may lead to movement disorder (kinematic changes) and changes in shoulder muscle activity (EMG changes) [3–5]. For instance, some of the kinematic changes observed in individuals with AGI include altered scapular kinematics such as excessive scapular protraction and anterior tilt [6] or decreased scapular upward rotation [5] during upper-arm elevations.

Studies reporting on the EMG activity of the shoulder joint muscles in individuals with AGI have shown controversial results regarding their latency and magnitude during shoulder elevation movements [4]. For example, such individuals demonstrated lower EMG activity in the anterior and middle deltoid muscles during shoulder flexions and abduction [7]. On the other hand, Illyes and Kiss [4] observed an increase in anterior deltoid muscle activity but no change of the middle deltoid during shoulder elevations. The anterior and middle deltoids, along with the scapular muscles, also showed no alterations in either their onset, duration, or magnitude of activation in recent studies [6, 8]. Several factors may account for the disparity in results presented by these studies such as type of labor activity, etiology (traumatic and atraumatic instability), or differences in symptoms and coexisting injuries (e.g., presence of pain and impingement syndrome in addition to AGI).

On the other hand, Santos et al. [9] found no alteration in the onset and recruitment order of shoulder muscles during arm elevations when controlling for some of these factors (i.e., only swimmers with AGI and without pain symptoms). The fact that no changes in the recruitment order of the shoulder muscles were observed does not rule out the possibility that AGI can generate changes in patterns of the muscles' EMG bursts.

To explore this possibility, we applied the 'speed insensitivity strategy' [10], a simple set of rules used to evaluate EMG muscle activity modulation. This strategy was previously used to explain how the central nervous system (CNS) modulates the muscle activity patterns during performance of constrained [10, 11] and unconstrained [12] single-joint movement. According to these studies, the strategy the CNS applies to control movement performed 'as fast as possible' is to scale the duration of agonist EMG activities and the onset of the antagonist activities according to the target distances, whereas the rising initial phase of the agonist EMG is not sensitive to the target distances.

Therefore, the present study aimed at testing the possibility that AGI without pain symptoms could not provoke changes in shoulder muscle activity patterns, as the identification of these muscular activation patterns may advance current rehabilitative management strategies in swimmers with AGI.

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Material and methods

The experiment included a total of 16 participants from a local university swimming team. We recruited eight swimmers (mean age 20.9 ±1.8 y) with a history of AGI and shoulder impingement syndrome (SIS) and eight swimmers (mean age 20.6 ± 2.4) with no shoulder disease. Each of the two groups was composed of four males and four females. We chose the sample size based on previous studies that showed significant differences in EMG activity between individuals with glenohumeral instability and healthy controls [19, 20]. The calculated power using this sample size and alpha level (0.05) was 0.779. All participants were informed of the purpose and procedures of the study and provided a signed informed consent form approved by the local ethics committee.

Swimmers with a history of SIS were recruited and interviewed. From the interview results, we considered a swimmer to have SIS if she/he referred to pain localized in the proximal anterior-lateral region of the shoulder or during overhead elevation or abduction of the upper-limbs (from 60° to 120° of upper-limb elevation, which characterizes the painful arc of movement). We sent swimmers fulfilling these requirements to a specialized physician at the Campinas State University Medical Center. This physician examined the subjects using a protocol defined by Bak and Fauno [2], which consisted of a clinical assessment that included: (1) anamnese, (2) coracoacromial impingement tests (Neer and Hawkins), (3) glenohumeral instability tests, and (4) general joint hyper mobility tests, as reported in detail in a previous study [9]. Thus, criteria for the AGI group according to the medical examination included: (1) symptoms of SIS within the last six months, (2) no acute inflammatory symptoms or shoulder pain at the moment of the test, and (3) at least one type of moderate or severe AGI (anterior, inferior, or posterior).

Swimmers in the healthy control group had no history of severe shoulder pain or injuries, glenohumeral instability, neurological disorder, or any other pathological condition that could impair motor performance. An experienced physical therapist (one of the authors) tested their glenohumeral stability prior to the experimental tasks. None of the recruited asymptomatic swimmers presented with glenohumeral instability.

The angles of the shoulder were quantified using an Optotrack model 3020 motion analysis system (Northern Digital, Canada). Light-emitting diode (LED) markers were fixed on the anterior superior iliac spine (ASIS), anterior aspect of the humeral head (in the direction of the joint center of the shoulder), and on the anterior aspect of the elbow joint (joint line). The three-dimensional coordinates of these LEDs were recorded at 200 frames per second.

Surface electromyography (EMG) from the glenohumeral and scapulothoracic muscles (right side) was recorded for the anterior deltoid (AD), pectoralis major superior portion (PEC), latissimus dorsi (LD), biceps (BIC) and triceps (TRIC) long head and serratus anterior (SER), and upper (UT) and lower (LT) fibers of the trapezius. After the skin was swabbed with alcohol, single differential surface Bagnoli model DEL2.1 electrodes (Delsys, USA) were attached to the muscle bellies in the main direction of the muscle fibers with electrode interface adhesive as outlined in another study [13]. The electrode material was 99.9% silver, and the inter-electrode distance was fixed at 10 mm. After similar skin preparation, a five-centimeter circular reference electrode was attached over the bony protuberance of the 7th cervical vertebrae. The EMG signals in volts were amplified and filtered (common mode rejection ratio at 90 dB, band-pass filtered between 20–450 Hz, gain 2000). EMG data were digitized at 1000 Hz by an ODAU system (Northern Digital, Canada), enabling a 16-bit synchronized collection of analogue and digital data with the Optotrack markers. EMG data were fully rectified and smoothed using a 20-millisecond moving average window.

The task of the participants was to stand upright in the orthostatic position with their arms relaxed at their sides. They were asked to perform bilateral and simultaneous elevations of both shoulders in the scapular plane (30° in front of the body's coronal plane) in three different target distances: 30°, 90°, and 150°. Two vertical metal bars (2.2 m in length with a radius of 2.0 cm) marked with these three target distances were placed in front of the participants (one at each side of the subject) to measure shoulder elevation. The target distances were individually measured and marked at the bar for each swimmer using a standard goniometer. The subjects were required to move the shoulders 'as fast as possible' from the relaxed position to the target position while keeping the elbows extended. Five movement trials were recorded for each experimental condition (30°, 90°, and 150°). The blocks of five trials that were performed for each condition were chosen randomly. Although bilateral movements were performed to obtain better trunk stabilization, we analyzed only the right affected shoulders of the experimental group and the right unaffected shoulders of the control group.

Shoulder angles were measured between two vectors, defined as follows: (1) the first vector was from the humeral head to the elbow joint line and (2) the second vector from the humeral head to the ASIS. The shoulder angle and its first derivative were used for analysis. The angle and velocity of the shoulder joint and the EMG activity of the eight recorded muscles for each trial were plotted on a screen. The initial and final angles were determined in order to obtain the total angular excursion. The maximum peak velocity of the shoulder was also identified. The onset of the agonist (AD, BIC, PEC, LT, UT, and SER) and antagonist (TRIC and LD) muscle activities for the shoulder elevation movement [9] were con-

sidered from the point of the first sustained rise above the baseline using an algorithm implemented in Matlab software (Mathworks, USA). The onset for a specific muscle was defined when the EMG amplitude for that muscle was greater than three standard deviations above the mean of its baseline value for at least 50 ms [9, 14].

The EMG activity of the agonists muscles were then integrated during the 30 milliseconds from their onset. This activity was used to quantify the rate of EMG rise of the agonist muscles ($\int EMG_{30}$) [10–12]. The agonist EMG activities were further integrated from their onset to the time of shoulder peak velocity. The antagonist EMG activities ($\int EMG_{antag}$) were also integrated from their onset until velocity reached zero (approximately at the end of movement). The integrated values of the agonist ($\int EMG_{ag}$) and antagonist ($\int EMG_{antag}$) muscles, based on their velocity profiles, were used to evaluate the magnitude and duration of muscle activity observed during the movement. As was demonstrated by Gottlieb et al. [10] and in other studies [12, 15], the $\int EMG_{30}$ during fast movements is independent of movement distance. However, EMG magnitudes ($\int EMG_{ag}$ and $\int EMG_{antag}$) increase with an increase in target distance [16].

To compare the EMG integrals ($\int EMG_{30}$, $\int EMG_{ag}$, and $\int EMG_{antag}$) across individuals, the EMG muscle activities were normalized in the following manner: first, the correspondent baseline integral values (identical time windows) were subtracted from the EMG integrals. The baseline of each muscle was calculated by integrating the EMG activities from the first 200 ms of recorded data before the movement onset. Second, we divided the EMG integral of each subject by its absolute highest EMG integral value (individually obtained) across the three experimental conditions [17, 18].

Two-way analysis of variance (ANOVA) was used to determine the effects of group (AGI vs. control), angular distance (30°, 90°, and 150°), and interaction (group and distance) for each dependent variable: shoulder angle, velocity, and the calculated EMG integrals ($\int EMG30$, $\int EMG_{ag}$, and $\int EMG_{antag}$) of each muscle. The Bonferroni post-hoc test was used for pairwise comparisons when a significant difference was found across the main factors. For statistical analysis, SPSS v. 15 statistical software (IBM, USA) was used.

Results

The clinical examination of the right shoulder of the AGI group resulted in negative coracoaromial impingement tests for all subjects. Six swimmers with AGI

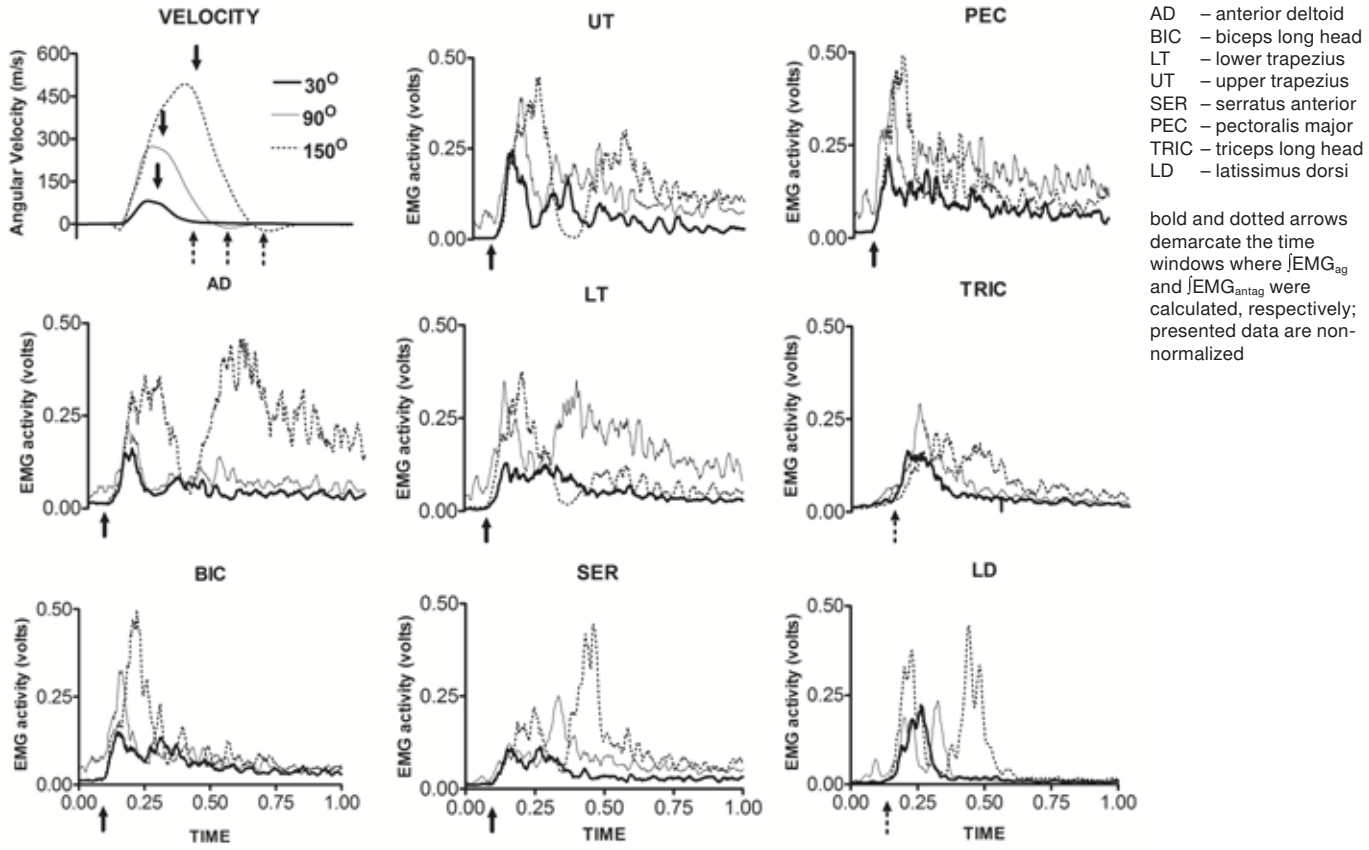


Figure 1. The mean velocity and EMG profiles recorded during five shoulder elevations toward the three target distances (30° – thick solid line, 90° – thin solid line, 150° – dashed line) for a swimmer with AGI; movement was stopped when it reached the target

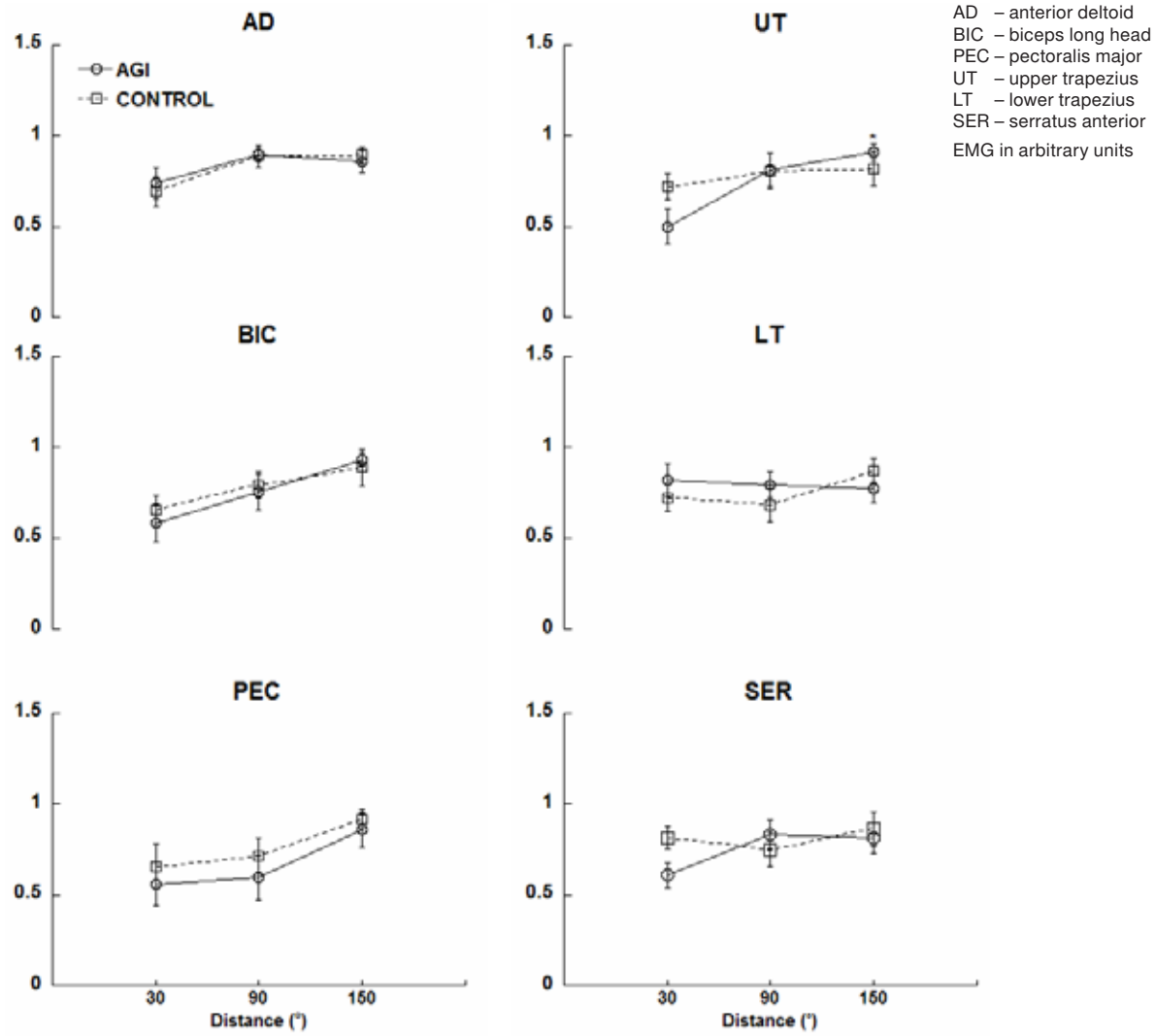


Figure 2. Initial rate of EMG rise of agonist muscles (means of $\int EMG_{30}$ and standard error) for AGI (solid line with open circle) and control groups (dashed line with open square) during shoulder elevations to the three target distances (30°, 90°, 150°)

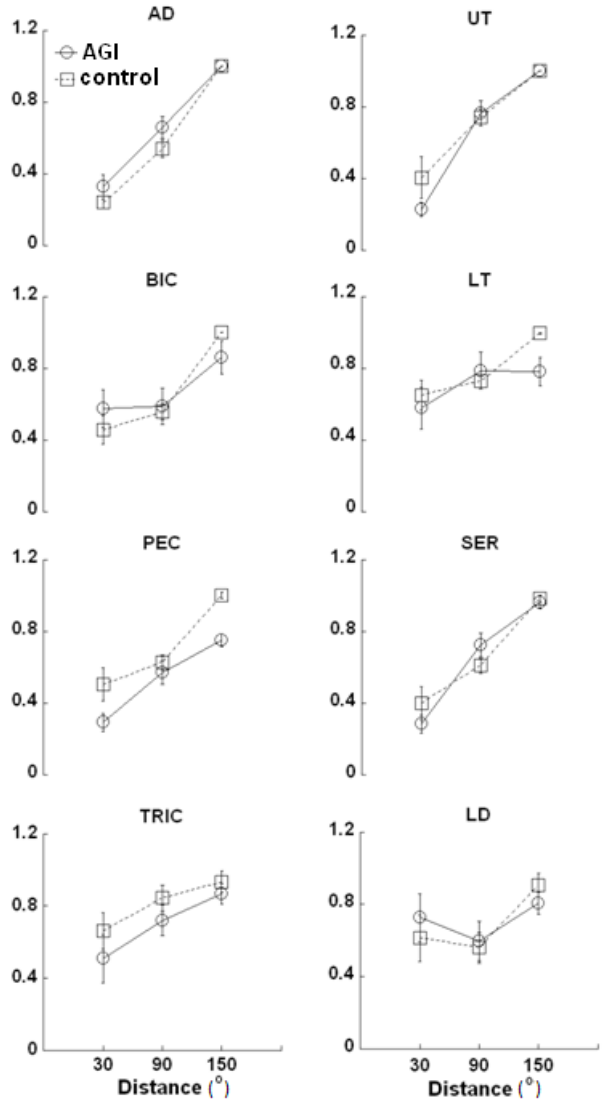
demonstrated multidirectional instability, one inferior laxity, and one anterior laxity. Three swimmers tested positive for general hyper-mobility.

Figure 1 depicts the mean velocity and the EMG activities of the five trials for one swimmer with AGI. The figure displays the glenohumeral (AD, BIC, and PEC) and scapulothoracic (LT, UT, and SER) agonist and antagonist (TRIC and LD) muscle activities during shoulder elevations to the three different target distances. Note that both angular velocity and EMG activity increased with distance and bell-shape profiles characterized the velocities.

Both groups performed the shoulder elevations using similar movement kinematics. The angular excursion and the angular velocity increased gradually and significantly according to the angular distance for both groups ($p < 0.01$). The angular excursions for the three angular distances of 30°, 90°, and 150° were, respectively, 25 ± 6°, 70 ± 7°, and 117 ± 8° for the AGI group and 21 ± 7°, 70 ± 11°, and 117 ± 8° for the control group. The movement velocities from the shorter (30°) to the longer (150°) distance were 155 ± 30°/s, 361 ± 43°/s, and 465 ± 47°/s for the control group and 166 ± 57°/s, 328 ± 49°/s, and 500 ± 41°/s for the experimental (AGI) group. No significant differences in these variables were found between the groups.

Figure 2 depicts the rate of EMG rise of the agonist muscles at the glenohumeral and scapulothoracic joints. Two-way ANOVA showed no significant difference of $\int EMG_{30}$ between groups for all tested agonist muscles (AD, BIC, PEC, UT, LT, and SER). However, the angular distance affected the BIC and LT muscles. Post-hoc analysis revealed the $\int EMG_{30}$ to be significantly smaller at 30° than in the 150° distance for both muscles for AGI individuals only ($p < 0.01$ and $p = 0.01$, respectively). Except for these muscles, $\int EMG_{30}$ showed no significant difference across the distances for either group.

Both groups performed the shoulder elevations using



AD – anterior deltoid; BIC – biceps long head; PEC – pectoralis major; TRIC – triceps long head; DT – descendens trapezius; LT – lower trapezius; UT – upper trapezius; SER – serratus anterior; LD – latissimus dorsi; EMG in arbitrary units

Figure 3. Magnitude of agonist and antagonist muscle activation (means of $\int EMG_{ag}$ and $\int EMG_{antag}$ and standard error) for the AGI (solid line with open circle) and control groups (dashed line with open square) during shoulder elevations to the three target distances (30°, 90°, 150°)

a similar magnitude of EMG muscle activity (Fig. 3). ANOVA revealed no significant group effect either for the $\int EMG_{ag}$ (AD, BIC, PEC, UT, LT, and SER) or for the $\int EMG_{antag}$ (TRIC and LD). On the other hand, the angular distance significantly affected all muscles. The AD ($p < 0.01$), UT ($p < 0.01$), and SER ($p < 0.01$) muscles demonstrated significant increases in the $\int EMG_{ag}$ for all three target distances by both groups as revealed by post-hoc analysis. Although the BIC, AT, TRIC, PEC, and LD muscles showed significant differences across the distances ($p < 0.01$, $p < 0.01$, $p < 0.01$, $p < 0.01$, $p = 0.02$, respectively), they did not change activity progressively across all three conditions as seen for AD, UT, and SER. For example, the $\int EMG_{ag}$ for the BIC muscle differed significantly only between the 30° and 90° conditions for the control group ($p < 0.01$) and between the 90° and 150° positions for the AGI group ($p = 0.02$). For the AGI group, the $\int EMG_{ag}$ of the LT muscle showed no significant difference across the conditions. For the control group, the LT $\int EMG_{ag}$ was significantly greater at the 150° position in comparison with the 90° and 30° conditions ($p < 0.01$ and $p < 0.01$, respectively). For the PEC muscles, the $\int EMG_{ag}$ was significantly different only between 90° and 150° positions in the experimental group (AGI). For this muscle in the control group, the $\int EMG_{ag}$ was greater in the 150° condition than in the 30° position ($p = 0.02$).

The significant differences in the $\int EMG_{antag}$ for the TRIC muscle were determined by the difference in $\int EMG_{antag}$ between the 30° and 90° target distances in the AGI group, in which the latter was significantly greater than the former ($p = 0.03$). Similarly, the $\int EMG_{antag}$ of the LD muscle was also significantly greater at the 150° than at the 90° distance for the AGI group ($p = 0.01$).

Discussion

This study intended to further investigate the patterns of EMG activity in a specific group of individuals (swimmers) who share a similar pathology, i.e., AGI. We included only individuals who presented no shoulder pain to minimize any pain symptoms that might induce bias in the EMG activity of the muscles [21]. Previous EMG investigations found no significant difference in the time and order of recruitment of the shoulder muscles between individuals with AGI with no pain symptoms and their healthy counterparts [9]. The present study extended these observations by showing that AGI alone was insufficient to modify the muscle activation strategy used by the CNS to perform fast overhead movements towards different target distances.

Both groups of individuals used the 'speed insensitivity strategy' [10] to modulate shoulder muscle activities. First, both groups initially activated the shoulder agonist muscles with a similar rise rate across the different target distances, which indicates a similar amount of recruited motor units despite the changes in movement excursion. The only exception to this rule were the BIC and LT muscles for the AGI group. Second, the EMG activity of the humeral and scapular muscles was scaled with the target distances, allowing the individuals to move faster for longer distances (Fig. 3). Third, the latency between the beginning of agonist and antagonist muscle activation increased with the target distances [9]. Thus, the simple set of rules used to describe the modulation of EMG activity during voluntary elbow movements [12] can also explain the modulation of scapular and glenohumeral muscles in the present experiment (Fig. 2 and 3).

Previous studies observed an increase in the magnitude of muscle activity with a progressive increase in the shoulder angular excursion. For instance, LT, UT, SER, and levator scapulae activities for healthy individuals increased gradually as the positions of shoulder elevation at the scapular plane became larger (0°, 90°, and 140°) [22]. Matias and Pascoal [6] also observed concomitant increases with target distances and EMG activity of the glenohumeral (AD) and scapular muscles (LT, UT, and SER) in individuals with glenohumeral instability. These results parallel the findings of the present study, showing that both groups of individuals used a similar strategy to modulate the agonist and antagonist EMG bursts of activity with target distances. Thus, AGI per se may not be enough to charge the motor control system to the point where one would expect to observe group differences during the performance of free unloaded overhead movements. The lack of group differences in time and order of recruitment of the shoulder muscles between individuals with and without shoulder instability observed in a previous study, further support this explanation [9], where the CNS did not change strategies in order to activate muscles in the presence of AGI.

On the other hand, in a previous study, it was observed decreased EMG activity in the PEC, LD, SER, and subscapularis muscles and increased EMG activity of the supraspinatus and BIC muscles in individuals with AGI during pitching [3]. In addition, previous studies also observed changes in EMG activity in individuals with multidirectional instability during different isokinetic shoulder movements [4]. Particularly, it was found that EMG activity of the anterior AD, BIC and TRIC decreased while the EMG activity of the infraspinatus increased for the group with shoulder instability compared with a control group [4]. In contrast, Barden et al. [8] found no significant difference in EMG amplitudes during isotonic concentric/eccentric movements of the shoulder joint in individuals with multidirectional instability. Therefore, certain movements such as pitching and isokinetic movements (at constant velocity) may possibly require greater stability and higher muscle torque at the shoulder joint. The latter movement, for example, requires excessive force at the beginning of the movement [8]. This may compromise joint stability to a level that would affect the generation and modulation of the EMG activity of these muscles. Thus, we cannot rule out the possibility that additional loads or velocity for whichever movement could be detrimental for individuals with joint instability to the level of influencing the EMG and kinematic strategies in which the overhead shoulder movements are performed.

We should note that the present study investigated only individuals with shoulder AGI without pain and measured only the superficial muscles of the shoulder joint. The discrepancies between this study and others that showed alterations in EMG activity might, in part, relate to different experimental designs or the nature of shoulder instability. For example, subjects with traumatic shoulder instability might have decreased afferent input from the joint due to severe lesions of the shoulder's capsular structures provoked by a shoulder dislocation [23]. Thus, this may affect neuromuscular control of shoulder movements and increase instability of the joint [24]. None of the individuals in our study had previously experienced severe trauma or dislocation at the shoulder; hence, the afferent information from the shoulder joint might be intact or less affected. Consequently, they did not show alterations in the recruitment [9], EMG rate, and magnitude of activation of the shoulder muscles.

The performance of shoulder elevations offered additional evidence of intact shoulder neuromuscular control in asymptomatic swimmers with AGI. Both groups achieved similar distances and movement velocities during the experimental tasks. Although both groups of swimmers seemed to undershoot the targets (see results), this was not the case. The difference between manual goniometry and the motion analysis system determined the differences between the target distances and those reached by the swimmers. For the latter, the shoulder marker displaced progressively to the medial and superior positions as the target increased, resulting in differences in the angles (goniometer vs. motion analysis system) for further distances.

Finally, this study used three variables to measure the ability of the CNS to activate the shoulder muscles during fast voluntary movements, i.e., $\int EMG_{30}$, $\int EMG_{ag}$, and $\int EMG_{antag}$. Both groups (experimental and control) demonstrated muscle activation at a similar rate of EMG rise ($\int EMG_{30}$) and magnitude ($\int EMG_{ag}$, and $\int EMG_{antag}$). This suggests that AGI by itself did not modify the way in which the CNS scales the strength of muscle activation during voluntary movements. Sciascia et al. [25] recorded needle and surface EMG activity in various shoulder muscles during scaption, prone horizontal abduction, prone external rotation, push-ups, and shoulder rehabilitation exercises. The authors reported a similar pattern of muscle activity among individuals who have multidirectional instability, anterior instability, generalized laxity, or a healthy shoulder. For this reason they recommended these shoulder-strengthening exercises for patients with shoulder instability.

In contrast, past studies investigating AGI in cadaveric models suggest muscular imbalance as a possible important factor of shoulder instability [26]. Nonetheless, in swimmers with AGI, this may be not the case. It is possible that the deficiency of the static structures of the shoulder (capsular and ligament laxity)– perhaps acquired by repetitive loading [27] – plays a larger role in developing inflammation and pain in individuals with AGI. Indeed, shoulder tasks that require greater joint stability, such as swimming, might require supplementary muscle activation to avoid or overcome the deficiencies of the shoulder joint's static structures. Further

kinematic and electromyographic studies involving healthy and symptomatic swimmers with AGI performing more demanding tasks may further clarify the causes of instability and the tendency to develop SIS and pain.

Some of the limitations of this study are mostly connected with the generalizability of the results given the fact that research was conducted with only eight swimmers with a history of AGI and shoulder impingement syndrome (SIS). The use of a larger sample size could allow for the detection of group differences. Nonetheless, the calculated power of the sample size used in the present study was 0.779 (see Material and methods). Therefore, we believe that the lack of observed group differences in the way the shoulder muscles are activated and modulated is robust and may not require a larger sample size to confirm these results.

Conclusions

We conclude that atraumatic shoulder instability does not necessarily affect the patterns of muscular activities. Deficiencies in the static structures of the shoulder joint may account more for the predisposition of swimmers to develop shoulder pain and impingement syndrome.

References

1. Bigliani L.U., Codd T.P., Connor P.M., Levine W.N., Littlefield M.A., Hershon S.J., Shoulder motion and laxity in the professional baseball player. *Am J Sports Med*, 1997, 25 (5), 609–613, doi: 10.1177/036354659702500504.
2. Bak K., Fauno P., Clinical findings in competitive swimmers with shoulder pain. *Am J Sports Med*, 1997, 25 (2), 254–260, doi:10.1177/036354659702500221.
3. Glousman R., Jobe F., Tibone J., Moynes D., Antonelli D., Perry J., Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg Am*, 1988, 70 (2), 220–226.
4. Illyes A., Kiss R.M., Electromyographic analysis in patients with multidirectional shoulder instability during pull, forward punch, elevation and overhead throw. *Knee Surg Sports Traumatol Arthrosc*, 2007, 15 (5), 624–631, doi: 10.1007/s00167-006-0163-1.
5. Ogston J.B., Ludewig P.M., Differences in 3-dimensional shoulder kinematics between persons with multidirectional instability and asymptomatic controls. *Am J Sports Med*, 2007, 35 (8), 1361–1370, doi: 10.1177/0363546507300820.
6. Matias R., Pascoal A.G., The unstable shoulder in arm elevation: A three-dimensional and electromyographic study in subjects with glenohumeral instability. *Clin Biomech (Bristol, Avon)*, 2006, 21 Suppl 1, S52–58, doi: 10.1016/j.clinbiomech.2005.09.014.
7. Kronberg M., Brostrom L.A., Nemeth G., Differences in shoulder muscle activity between patients with generalized joint laxity and normal controls. *Clin Orthop*, 1991, 269, 181–192, doi: 10.1097/00003086-199108000-00026.
8. Barden J.M., Balyk R., Raso V.J., Moreau M., Bagnall K., Atypical shoulder muscle activation in multidirectional instability. *Clin Neurophysiol*. 2005, 116 (8), 1846–1857, doi: 10.1016/j.clinph.2005.04.019.
9. Santos M.J., Belangero W.D., Almeida G.L., The effect of joint instability on latency and recruitment order of the shoulder muscles. *J. Electromyography and Kinesiology*. 2007, 17 (2), 167–175, doi:10.1016/j.jelekin.2006.01.010.
10. Gottlieb G.L., Corcos D.M., Agarwal G.C., Organizing principles for single-joint movements: I. A speed-insensitive strategy. *J Neurophysiol*, 1989, 62 (2), 342–357.
11. Corcos D.M., Gottlieb G.L., Agarwal G.C., Organizing principles for single-joint movements. II. A speed-sensitive strategy. *J Neurophysiol*, 1989, 62 (2), 358–368.
12. Almeida G.L., Hong D.A., Corcos D., Gottlieb G.L., Organizing principles for voluntary movement: extending single-joint rules. *J Neurophysiol*, 1995, 74 (4), 1374–1381.
13. Basmajian J.V., Electromyography – dynamic gross anatomy: A review. *Am J Anat*, 1980, 159 (3), 245–260, doi: 10.1002/aja.1001590302.
14. Santos M.J., Kanekar N., Aruin A.S., The role of anticipatory postural adjustments in compensatory control of posture: 1. Electromyographic analysis. *J Electromyogr Kinesiol*, 2010, 20 (3), 388–397, doi:10.1016/j.jelekin.2009.06.006.
15. Gottlieb G.L., Muscle activation patterns during two types of voluntary single-joint movement. *J Neurophysiol*, 1998, 80 (4), 1860–1867.
16. Gottlieb G.L., Corcos D.M., Agarwal G.C., Strategies for the control of voluntary movements with one mechanical degree of freedom. *Behav Brain Sci*, 1989, 12 (2), 189–210, doi: 10.1017/S0140525X00048238.
17. Santos M.J., Aruin A.S., Role of lateral muscles and body orientation in feedforward postural control. *Exp Brain Res*, 2008, 184 (4), 547–559, doi: 10.1007/s00221-007-1123-9.
18. 1Shiratori T., Latash M.L., Anticipatory postural adjustments during load catching by standing subjects. *Clin Neurophysiol*, 2001, 112 (7), 1250–1265, doi: 10.1016/S1388-2457(01)00553-3.
19. Hundza S.R., Zehr E.P., Muscle activation and cutaneous reflex modulation during rhythmic and discrete arm tasks in orthopaedic shoulder instability. *Exp Brain Res*, 2007, 179 (3), 339–351, doi: 10.1007/s00221-006-0793-z.
20. Morris A.D., Kemp G.J., Frostick S.P., Shoulder electromyography in multidirectional instability. *J Shoulder Elbow Surg*, 2004, 13 (1), 24–29, doi: 10.1016/j.jse.2003.09.005.
21. Pink M., Jobe F.W., Perry J., Browne A., Scovazzo M.L., Kerrigan J., The painful shoulder during the butterfly stroke. An electromyographic and cinematographic analysis of twelve muscles. *Clin Orthop Relat Res*, 1993, 288, 60–72, doi: 10.1097/00003086-199303000-00008.
22. Ludewig P.M., Cook T.M., Nawoczenski D.A., Three-dimensional scapular orientation and muscle activity at selected positions of humeral elevation. *J Orthop Sports Phys Ther*, 1996, 24 (2), 57–65.
23. Guanche C., Knatt T., Solomonow M., Lu Y., Baratta R. The synergistic action of the capsule and the shoulder muscles. *Am J Sports Med*, 1995, 23 (3), 301–306, doi: 10.1177/036354659502300308.
24. Riemann B.L., Lephart S.M., The Sensorimotor System, Part II: The Role of Proprioception in Motor Control and Functional Joint Stability. *J Athl Train*, 2002, 37 (1), 80–84.

25. Sciascia A., Kuschinsky N., Nitz A.J., Mair S.D., Uhl T.L., Electromyographical comparison of four common shoulder exercises in unstable and stable shoulders. *Rehabil Res Pract*, 2012, 783–824, doi:10.1155/2012/783824.
26. Apreleva M., Hasselman C.T., Debski R.E., Fu F.H., Woo S.L.-Y., Warner J.J.P., A dynamic analysis of glenohumeral motion after simulated capsulolabral injury. A cadaver model. *J Bone Joint Surg Am*, 1998, 80 (4), 474–480.
27. Pollock R.G., Wang V.M., Bucchieri J.S., Cohen N.P., Huang C.-Y., Pawluk R.J. et al., Effects of repetitive subfailure strains on the mechanical behavior of the inferior glenohumeral ligament. *J Shoulder Elbow Surg*, 2000, 9 (5), 427–435, doi: 10.1067/mse.2000.108388.
28. Smith C.J., Type I and Type II errors: what are they and why do they matter? *Phlebology*, 2012, 27 (4), 199–200, doi:10.1258/phleb.2012.012j04.

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