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ORIGINAL RESEARCH REPORT

Is There a Decrease in the Acromiohumeral Distance Among Recreational Overhead Athletes With Rotator Cuff–Related Shoulder Pain?

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Context: Recreational overhead athletes are exposed to high overload, which increases the risk of shoulder injuries. Reduction of the acromiohumeral distance (AHD) is often associated with rotator cuff–related shoulder pain (RCRSP) among the general population. However, the AHD of symptomatic shoulders of recreational athletes has not yet been compared with their asymptomatic shoulders. ***Objective:*** To compare the AHD of a symptomatic to asymptomatic shoulder at rest (0°) and 60° abduction. To establish the relationship between AHD, pain, and functional limitations of recreational athletes with RCRSP. ***Design:*** Cross-sectional study. ***Setting:*** University laboratory. ***Participants:*** A total of 45 recreational overhead athletes with RCRSP were examined. ***Main Outcome Measures:*** The AHD was measured by ultrasonography at 0° and 60° abduction (angles). Shoulder pain was assessed using a numeric pain scale, whereas functional limitations were assessed using the The Disabilities of the Arm, Shoulder, and Hand questionnaire. Differences in the between-shoulders condition (symptomatic and asymptomatic) were determined using 2-way analysis of variance for repeated measures. A Pearson correlation established the relationship between AHD, pain, and functional limitations. ***Results:*** No angles × shoulder condition interactions ($P = .776$) nor shoulder condition effects ($P = .087$) were detected, suggesting no significant differences ($P > .05$) between asymptomatic and symptomatic shoulders in the AHD at 0° or 60°. The AHD at 60° reduced significantly compared with 0° (3.05 [1.36] mm [2.77–3.33], angle effects: $P < .001$). The AHD at 0° and 60° was not correlated with pain or functional limitations ($-.205 \le r \le .210$, $.167 \le P \le .585$). ***Conclusions:*** The AHD of recreational athletes is not decreased in symptomatic shoulders compared with asymptomatic shoulders. Reduction of the AHD in symptomatic shoulders is not associated with an increase in pain or functional limitations of recreational athletes with RCRSP.

Keywords: disabilities, rotator cuff tendinopathy, subacromial impingement syndrome, subacromial space, ultrasonography

The acromiohumeral distance (AHD) is used for estimating the subacromial space.[1] It corresponds to the tangential distance between the bony landmarks of the humeral head and the inferior edge of the acromion.[1–3] The AHD is estimated to range between 9 and 12 mm among healthy shoulders,[1,4] with an inversed correlation to arm elevation,[2] indicating that, from 0° to 100°–120°, as the arm is raised higher, the AHD decreases. Decreased AHD is used to suggest a reduction of the subacromial space.[5] Superior migration of the humeral head with respect to the glenoid cavity is one of the causes of an abnormal reduction of the AHD, compressing the structures crossing this critical zone,[6] such as the subacromial bursa, the rotator cuff (RC) tendons, and the long head of the biceps. These structures are common sites for inflammation and degeneration with shoulder disorders,[7] such as RC-related shoulder pain (RCRSP). In addition, scapular kinematic alterations, such as a limited posterior tilt and upward scapular rotation, have been demonstrated to favor the narrowing of the subacromial space and, consequently, the AHD reduction.[8] Therefore, these alterations in the scapular motion, frequently observed in individuals with RCRSP, may compromise the dimensional integrity of the subacromial space. Given these arguments, it is crucial to better understand the possible underlying mechanisms and coexisting factors that could contribute to the progression of RCRSP to guide clinical decision making.

Recreational athletes of sports requiring repetitive arm elevation >90° are often affected by RCRSP as a result of their overhead activities. Alternating acceleration and deceleration actions expose the athletes to loads of large magnitude of the upper extremity, affecting their shoulder function and predisposing the development of RCRSP.[8] Indeed, RC tendinopathies among athletes are associated with deficits in the activation of scapulothoracic muscles, such as the serratus anterior; the lower trapezius; and the RC muscles, such as the internal and external shoulder rotators muscles.[9] These muscles play an important role in the transverse RC force coupling, maintaining the humeral head well centered in the glenoid fossa during arm elevation and in the coronal force coupling, counteracting the deltoid activation. Therefore, weakness or fatigue of the internal and external muscles may contribute to the compression of subacromial structures.[10,11] A previous study[9] reported a decreased AHD at 0°, 45°, and 60° of abduction of the dominant shoulder in athletes having a glenohumeral internal rotation deficit >15°.[9]

Although many clinical studies have been conducted to investigate the relationship between the AHD and RCRSP,[12–14] information regarding AHD in symptomatic overhead recreational athletes is still scarce. To the best of our knowledge, no studies have assessed the AHD and the relationship among AHD, shoulder pain, and functional limitations in symptomatic recreational athletes clinically

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diagnosed with RCRSP. Therefore, the aim of this study was (1) to compare the AHD at rest (0°) and 60° of active shoulder abduction in the frontal plane between the symptomatic and asymptomatic shoulder of recreational overhead athletes with unilateral RCRSP and (2) to determine the relationship between AHD, pain, and functional limitations among this population. The reliability of AHD measurements at rest and 60° of shoulder abduction was also evaluated to confirm the reliability of our procedures.

As kinematic scapular alterations are common features observed in overhead athletes, favoring a reduction of the subacromial space,[15] the authors hypothesized that the symptomatic shoulder of recreational athletes would have smaller AHD at 60° than the asymptomatic shoulder. As the reduction of the AHD could result in inherent compression of the subacromial structures, moderate correlations ($r > .60$) between AHD/pain and AHD/functional limitations are hypothesized at 60° of shoulder abduction.

Methods

Study Design

This study followed a cross-sectional design and was conducted in accordance with the STrengthening the Reporting of OBservational studies in Epidemiology statements. All participants were evaluated in a single session conducted at the Center for Interdisciplinary Research in Rehabilitation and Social Integration in Quebec City, Canada.

Participants

A total of 45 recreational athletes (25 men and 21 women), aged between 20 and 53 years old, with a unilateral RCRSP, regardless of upper-extremity dominance, were examined. The participants were recruited from a mailing list of students and employees from Université Laval, Canada. To be included, the participants had to regularly practice (at least twice a week) in a sport that requires repetitive overhead movements, such as badminton, tennis, volleyball, basketball, baseball, climbing, and so forth. In addition, the participants had to present with at least 1 positive sign in each category of criterion: (1) Neer or Hawkins–Kennedy impingement sign[16]; (2) painful arc of movement during shoulder flexion or abduction; and (3) pain on resisted external rotation, abduction, or empty can test.[16] Therefore, the participants should present 3 positive clinical findings to be considered as having RCRSP. Upper-extremity dominance was not considered, as most participants (68.9%) were affected on the dominant side. Thus, no participant with a symptomatic nondominant side played a sport that requires repetitive movements predominantly on the dominant side. The exclusion criteria included the following: (1) diagnosis of adhesive capsulitis,[17] (2) previous history of shoulder surgery, (3) history of glenohumeral luxation within the last 12 months or any fractures of the shoulder girdle, (4) shoulder pain reproduced by cervical movements, and (5) clinical sign of RC full-thickness tears (lag signs).[18,19]

The Sectorial Rehabilitation and Social Integration Research Ethics Committee of the CIUSSS-CN granted ethical approval, which complies with the Declaration of Helsinki for human research.

Procedures

All participants provided signed written informed consent prior to enrolling in the study. Thereafter, the eligibility criteria were confirmed, and the participants completed a sociodemographic questionnaire. The study outcomes were assessed as follows: (1) functional limitations, (2) pain intensity, (3) AHD at rest (0°), and (4) AHD at 60° of shoulder abduction. The procedures for the data collection followed the same order for all participants. The asymptomatic shoulder was always assessed prior to the symptomatic shoulder.

In an effort to reduce the risk of bias, all outcome assessments were conducted by an independent evaluator not involved in data analysis. In addition, standardized procedures were used for each outcome measurement.

Outcome Measures

Functional Limitations. Functional limitations were assessed using the Canadian-French version of the The Disabilities of the Arm, Shoulder, and Hand questionnaire (*Quick*DASH),[20] which is a shortened version of the 30-item DASH instrument.[21] The *Quick*-DASH consists of 11 items addressing the level of difficulty when performing daily activities and the severity of the symptoms.[20,21] Each item is rated from 1 to 5 points (minor to major difficulty). A total score of 100 points indicates the most severe disability.[20,21]

Pain Intensity. Given that the *Quick*DASH has few questions related to the pain assessments, a numeric pain scale was used to assess the level of pain intensity experienced by the participants. The participants rated their most severe pain within the last 24 hours on an 11-point scale (0 = no pain; 10 = worst pain).

Acromiohumeral Distance. The AHD examination was undertaken using an ultrasound scanner (Logic *e*9; GE Healthcare, Milwaukee, WI) with a 4 to 15 MHz linear array probe. Ultrasound imaging is a valid[22] and reliable (intraclass correlation coefficient [ICC] > .75)[1] method for estimating the AHD, regardless of the experience of the evaluator.[4]

In our study, the AHD measurements were taken at rest (0°) and 60° of shoulder abduction in both symptomatic and asymptomatic shoulders, using standardized ultrasonography parameters. Although painful symptoms are most often reported between 70° and 110° of arm elevation,[23] there is a known difficulty in visualizing, simultaneously, both structures that define the AHD (acromion and humeral head) above 60° of shoulder abduction. For

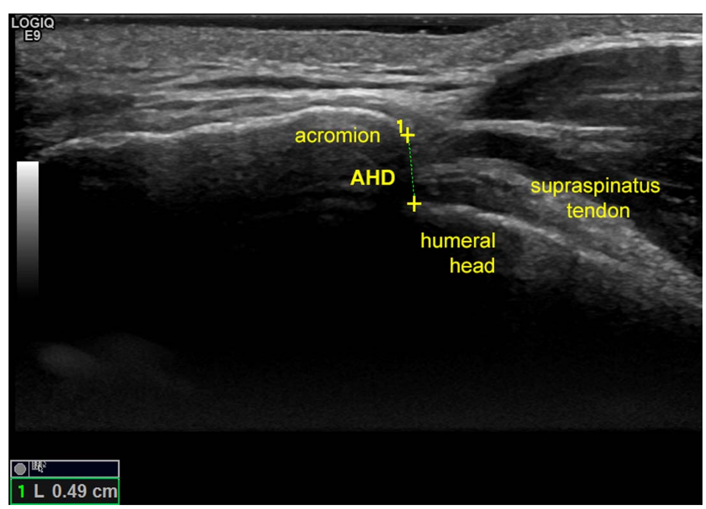

Figure 1 — Ultrasonography of the AHD at 60° of shoulder abduction in the frontal plane. The dotted line indicates the AHD. AHD indicates acromiohumeral distance.

this reason, the AHD was examined at 60° of arm elevation (Figure 1). Placement of the probe followed the procedures described by Desmeules et al.[2] To record the AHD images, participants were seated with the arm in a neutral position, elbow flexed at 90°, and forearm in a neutral position resting on a pillow on their lap. To restrain the arm elevation to 60° abduction at maximum, a belt fixed to a custom-made chair was attached to the proximal forearm. The participants were instructed to actively maintain the belt slightly stretched out during the AHD image acquisition to keep the shoulder at 60° of abduction. The angle of interest was confirmed with a universal inclinometer. The measurements were performed twice with an interval of at least 20 seconds between trials. The recorded images were analyzed online. The mean of the 2 trials was used for statistical analysis.

Sample Size

The sample size was determined based on the mean difference (0.51 mm, $P < .05$) of AHD at 60° abduction between the asymptomatic and symptomatic shoulders of individuals with RCRSP, previously reported.[24] Considering the following parameters (G*Power [version 3.1.9.2][25], *t* tests, difference between 2 dependent means [matched pairs]; $\alpha = .05$, power $[1-\beta] = 0.95$, effect size [ES] = 0.654), at least 33 individuals with RCRSP was sufficient to ensure the robustness of the results. The authors highlight that the sample size calculation was performed before recruitment of the participants, expecting a normal distribution of the AHD data.

Statistical Analyses

All data analyses were performed using SPSS Statistics for Windows (version 20; IBM Corp, Armonk, NY). The Kolmogorov–Smirnov test was used to detect the normal distribution of the AHD data. A 2-way analysis of variance for repeated measures (general linear model; SPSS [version 20, IBM Corp, Armonk, NY]) was used to compare the AHD (2 angles [0° and 60° of abduction]) in 2 shoulder conditions (symptomatic and asymptomatic).

The Pearson correlation coefficient was used to evaluate the relationship between the AHD to functional limitations (*Quick*DASH) and pain intensity (numeric pain scale). The alpha criterion was always set at 5%.

Intrarater reliability for the AHD measurements was calculated using the intraclass correlation coefficient (ICC, 2-mixed model, 95% CI) through a comparison of the 2 AHD measurements for each angle of interest. Reliability coefficients were classified as follows: "poor reliability" for ICC < .50, "moderate" for ICC ≥ .50 and <.75, "good" for ICC ≥ .75 and <.90, and "excellent" for ICC > .90.[26] The standard error of measurement ($SEM = SD \times \sqrt{1 - ICC}$) and the minimal detectable change with 95% CI ($MDC_{95\%} = 1.96 \times SEM \times \sqrt{2}$) were also calculated.

Results

The demographic characteristics are presented in Table 1. All participants examined for eligibility (N = 45) were enrolled in the study. The intrarater reliability of the AHD measurements was excellent. The ICC, SEM, and $MDC_{95\%}$ for both the asymptomatic and symptomatic shoulders can be viewed in Table 2. The mean AHD measurements at 0° and 60° of shoulder abduction for both the asymptomatic and symptomatic shoulders can be viewed in Table 3, as well as the mean of the AHD reduction during arm elevation from 0° to 60° of shoulder abduction.

Table 1 Characteristics of the Participants (N = 45)

	Mean (SD) [Min–Max] / n (%)
Demographic data	
Age, y	28.7 (7.7) [20–53]
Sex (men), n (%)	24 (53.3)
Height, cm	1.75 (0.12) [1.52–1.98]
Weight, kg	72.6 (13.8) [48.0–105.0]
Dominance (right), n (%)	40 (88.9)
Dominant shoulder affected, n (%)	31 (68.9)
Use of medication, n (%)	7 (15.6)
Symptoms of RC tendinopathy	
Duration of symptoms, mo	24.2 (28.0)
Pain intensity (NPS)	4.6 (2.2) [1.0–9.0]
Functional limitations (*Quick*DASH)	28.2 (10.6)

Abbreviations: NPS, numeric pain scale; *Quick*DASH, The Disabilities of the Arm, Shoulder, and Hand questionnaire; RC, rotator cuff. Note: Data are expressed as mean (SD) [minimum–maximum]. Continuous variables: *t* tests; categorical variables: Fischer exact probability tests. Medication used included: antacid (1), antidepressant (1), anti-inflammatory (1), antipsychotic (2), hormonal regulator (1), and immunosuppressant (1).

Table 2 The Intrarater Reliability, SEM, and MDC

	ICC (95% CI)	SEM, mm	$MDC_{95\%}$, mm
Symptomatic			
At rest (0°)	.993 (.988 to .996)	0.18	0.51
At 60° abduction	.990 (.981 to .994)	0.23	0.64
Asymptomatic			
At rest (0°)	.987 (.976 to .993)	0.27	0.74
At 60° abduction	.989 (.980 to .994)	0.22	0.62

Abbreviations: CI, confidence interval; ICC, intraclass correlation coefficient; $MDC_{95\%}$, minimal detectable change with 95% CI; SEM, standard error of measurement.

Effects of Angles (0° and 60°) and Shoulder Condition (Symptomatic and Asymptomatic)

The data analyses showed no effect of the shoulder condition ($P = .087$) and no interactions between the angles × shoulder condition ($P = .776$). However, the effects of the shoulder angles ($P < .001$) were detected; the AHD was reduced significantly during arm elevation from 0° to 60° of the shoulder abduction (3.05 [1.36] mm [95% CI, 2.77 to 3.33]).

Correlation of the AHD With Pain or Functional Limitations

The AHD did not present a significant relationship to functional limitations (*Quick*DASH) or pain intensity (numeric pain scale) at 0° ($-.205 \le r \le .135$; $.178 \le P \le .377$) or at 60° of abduction ($-.084 \le r \le .210$; $.167 \le P \le .585$) (Table 4).

Discussion

Our results indicate that recreational overhead athletes present a similar AHD in symptomatic and asymptomatic shoulders at both

Table 3 Group Mean Scores for AHD

	Asymptomatic	Symptomatic	Mean difference (95% CI)	Standard error mean	*P* value
AHD, mm					
At rest (0°)	11.16 (2.35)	11.49 (2.20)	0.33 (−0.05 to 0.71)	0.189	.088
At 60° abduction	8.14 (2.14)	8.40 (2.29)	0.26 (−0.18 to 0.71)	0.215	.234
Angle effects					
Mean difference (mm) from 0° to 60° (95% CI)	3.02* (2.62 to 3.42)	3.08* (2.66 to 3.50)			

Abbreviations: AHD, acromiohumeral distance; CI, confidence interval. Note: Data are expressed as mean (SD) or (95% CI).
*Statistically significant difference ($P < .001$).

Table 4 Relationship Between the AHD of the Symptomatic Shoulder and the Outcomes of Interest

	Pearson (*r*)	*P* value
AHD at rest (0°)		
vs functional limitations (*Quick*DASH)	.135	.377
vs pain (NPS)	−.205	.178
AHD at 60° abduction		
vs functional limitations (*Quick*DASH)	.210	.167
vs pain (NPS)	−.084	.585

Abbreviations: AHD, acromiohumeral distance; NPS, numeric pain scale; *Quick*-DASH, The Disabilities of the Arm, Shoulder, and Hand questionnaire; *r*, Pearson correlations coefficients.

angles tested (0° and 60° of abduction). The AHD decreased similarly with arm elevation to 60° of abduction in both the symptomatic and asymptomatic shoulders (Table 3). The absence of statistically or clinically significant differences between the symptomatic and asymptomatic shoulders at rest ($\Delta AHD_{0°} = 0.33$ mm, $P = .088$) was not surprising, seeing as deficits related to RCRSP are best observed during dynamic movements rather than a static position.[10] This result corroborates previous studies,[2,12] which demonstrated no significant differences at 0° when symptomatic shoulders were compared with healthy shoulders. Contrastingly, the absence of significant differences at 60° of abduction is noteworthy, as this angle of arm elevation corresponds to the moment when the supraspinatus tendon crosses the subacromial space, resulting in less AHD width due to the proximity between the greater tuberosity and the acromion.[27] Differences in the AHD between the symptomatic and asymptomatic shoulders were not demonstrated at this critical angle ($\Delta AHD_{60°} = 0.26$ mm, $P = .234$). Clinically, this is an important finding, which suggests that RCRSP does not necessarily impact the AHD.

Substantially, asymptomatic overhead athletes present greater scapular upward rotation,[28] which may drive the acromion away from the humerus. This statement is true when dominant shoulders are assessed. Notwithstanding, it makes room for inquiring whether the symptomatic shoulder could have increased its AHD to allow for greater clearance of the subacromial structures, to adapt to the sport-specific needs through enhanced scapular neuromuscular control,[7] ultimately avoiding exacerbated pain during sports gestures. In our study, the symptomatic and asymptomatic shoulders were assessed regardless of upper-extremity dominance. Therefore, these are speculations that need further examination.

Another explanation is possible. It has been reported that balance between shoulder mobility and stability is necessary to optimize overhead sport-specific gestures.[5] As all participants were actively involved in activities requiring repetitive use of both shoulders, overtime postural alterations in the contralateral shoulder (asymptomatic) may likely have occurred naturally as a strategy to maintain a bilateral balance between shoulders. It has not been ascertained yet whether this could make the asymptomatic shoulder more susceptible to further tendinopathy.

It has been argued that athletes present with sport-specific adaptations of the shoulder,[29,30] due to the best performance of shoulder external and internal rotators and scapular stabilizers muscles, when compared with the general population.[11,31] Although this argument has merit when evaluating shoulder functioning,[31] suggesting an increase in AHD among athletes, further evidence is needed to confirm this theory. Moreover, it has been demonstrated that shoulder muscle activity may affect the AHD.[32,33] However, strength and activation of the shoulder musculature were not quantified within this study. Therefore, further studies should focus on the activation of RC and scapulothoracic muscles, particularly those that participate in humeral head depression during arm elevation, such as the teres minor, infraspinatus, subscapular, pectoral minor, and long head of the biceps, to better understand their implications on the AHD.

Currently, a single study[24] has compared the AHD (at rest and 60° of scaption) among affected, nonaffected shoulders of individuals with RCRSP. Similarly to our study, Navarro-Ledesma and Luque-Suarez[24] found no differences between the affected and nonaffected shoulder in the AHD at rest (9.46 vs 9.65 mm, $P > .05$). However, divergent from our results, their findings indicated that the affected shoulder presented greater AHD at 60° of scaption (0.51 mm [0.12 to 0.90], $P < .01$) than the nonaffected shoulder of the same participants.[24] While these authors examined the AHD in the scapular plane in the general population, the authors examined the AHD in the frontal plane of recreational athletes who regularly participate in sports requiring repetitive overhead movements. This methodological difference may explain the divergent results between the 2 studies.

In addition, few studies have examined the AHD of an athletic population. Vanderstukken et al[30] reported that healthy hockey players had similar AHD at rest (0°) to matched nonathletic individuals (11.64 [1.44] vs 11.58 [1.58] mm, $P > .05$). Similarly, Wang et al[7] did not report significant differences in the AHD at rest or 90° of scaption of both injured or uninjured baseball players compared with healthy controls ($P > .05$). Concerning the AHD at 60° of abduction, Silva et al[15] reported no significant differences between healthy young tennis players compared with matched healthy controls (7.19 [1.55] vs 7.62 [1.52] mm, $P > .05$). Thomas et al[29] found no significant differences in the AHD at rest (12.07 [1.95] vs 11.96 [1.85] mm, $P > .05$) and 90° of shoulder abduction (12.85 [2.35] vs 12.77 [2.35] mm, $P > .05$) between the dominant and nondominant upper extremity of healthy young baseball players. It is important to highlight that our study compared shoulders bilaterally from a single group, whereas the aforementioned studies compared different groups[7,15,24,30] or compared the dominant to the nondominant side in healthy participants,[29] or compared athletes to a nonathletic population.[7,15,30] Differing methodologies implemented by these studies, such as varied angles, protocols, and measurement techniques, as well as diverging sample characteristics, may hamper a comparative analysis between the outlined studies addressing AHD and our results.

Correlations

Considering that RCRSP has a multifactorial etiology, the absence of a relationship between AHD and functional limitations suggests that some individuals with symptomatic RCRSP may not present a reduced AHD. Reciprocally, individuals with reduced AHD may not have RCRSP. Our results corroborate Navarro-Ledesma et al,[34] who reported no significant correlation between AHD and disabilities, quantified using the Shoulder Pain and Disability Index (at 0°, $r = -.215$; at 60°, $r = -.148$). Withal, pain intensity was not correlated either to AHD (at rest: $r = -.044$; at 60°: $r = .135$), indicating that painful symptoms experienced by individuals with RCRSP do not indicate a clear reduction in AHD.

Study Limitations

First, it is important to take into consideration that the AHD is a 2D measurement, whereas the subacromial space is a 3D area. This divergence may be a limitation in visualizing the undersurface of the acromion, and it may impact the calculation of the subacromial space.[34] Second, it is important to recognize that the AHD measurements were not blinded. Thus, the evaluator was aware of which shoulder was symptomatic or asymptomatic. Third, although the authors evaluated a critical angle (60°) of arm elevation in abduction, the AHD was measured in a static position. This may have influenced our results, as the deficits caused by RCRSP are best evidenced during dynamic movements.[10] Fourth, RCRSP was diagnosed based on the clinical exam since the combination of positive results from the clinical diagnostic tests used as the inclusion criteria has values ≥0.74 for sensitivity and specificity for RCRSP.[35] Magnetic resonance imaging and ultrasound were not used for diagnostic purposes. Therefore, RC tendons and the bursa width were not taken into consideration during the AHD measurements. This is an important issue that may have impacted the subacromial space width.[36] Finally, our results should be interpreted with caution, as our study design does not infer the cause–effect relation between AHD, pain, and functional limitations.

Clinical Implications

It is undeniable that AHD measurements can yield relevant contributions to the diagnostic evaluation and rehabilitation of RCRSP, besides having important implications in the determination of treatment goals in overhead athletes with impingement symptoms. Many interventions have focused on the normalization of the AHD to optimize the rehabilitation and minimize residual impairments of RCRSP. However, clinicians should be aware that, although reduced AHD is a common feature leading to painful symptoms of RCRSP, it does not mean that all symptomatic individuals will present with a reduction of the AHD. Thus, clinicians should use therapeutic approaches that aim to treat intrinsic alterations of the RC tendons, instead of focusing solely on increasing the AHD. As our results did not reveal differences in AHD between asymptomatic and symptomatic shoulders, managing the complexity of RCRSP as a localized problem may not be the optimal strategy. Clinicians should give attention to a wholistic approach, such as central sensitization. Indeed, it remains unclear at this time whether the alterations in the AHD are part of a physiological maladaptive loading mechanism associated with a chronicization process. Interventions such as sensorimotor training seem to be a relevant option for improving tissue load tolerance and, consequently, overall shoulder function.

Conclusions

The AHD of recreational athletes is not less in the symptomatic shoulder compared with the asymptomatic shoulder. The reduction of the AHD is associated with neither an increase in pain nor the functional limitations of recreational athletes with RCRSP.

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