Differences in Passive Shoulder Range of Motion Between Baseball Players With Neurogenic Thoracic Outlet Syndrome and Matched Healthy Controls

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Background: Neurogenic thoracic outlet syndrome (nTOS) is becoming more recognized as a diagnosis in the throwing athlete. Currently, there is limited information on the clinical presentation and development of nTOS in baseball players.

Purpose: To compare passive shoulder range of motion (ROM) and anatomic humeral retrotorsion (HRT) of baseball players diagnosed with nTOS with a group of healthy, matched controls.

Study Design: Cross-sectional study; Level of evidence, 3.

Methods: A total of 53 adolescent baseball players diagnosed with nTOS (age, 17.2 ± 2.3 years; height, 180.9 ± 10.1 cm; weight, 80.0 ± 13.3 kg) were compared with 53 healthy baseball players (age, 17.2 ± 2.4 years; height, 183.9 ± 9.0 cm; weight, 83.8 ± 11.5 kg). Participants were measured for shoulder internal rotation (IR) and external rotation (ER) ROM and HRT. All measurements were taken bilaterally, and the differences (throwing to nonthrowing arm) were used to calculate mean values for glenohumeral internal rotation difference, glenohumeral external rotation difference (GERD), total rotational motion difference (TRM_{diff}), and anatomic humeral retrotorsion difference. Group comparisons were made between the nTOS and control players using multivariate analysis of variance, and descriptive comparisons were made with independent *t* tests.

Results: There were no significant differences between groups in age, height, weight, or years of experience. Players in the nTOS group had significantly less throwing arm ER compared with controls (103.4° ± 10.4° vs 109.6° ± 7.5°, respectively; $P = .001$) and GERD (3.0° ± 9.2° vs 8.8° ± 9.2°, respectively; $P = .002$). TRM_{diff} was significantly greater in nTOS (–11.1° ± 11.1°) than in controls (–3.7° ± 9.4°) ($P < .001$).

Conclusion: In the current study, adolescent baseball players diagnosed with nTOS were evaluated with shoulder ROM differences when compared with a matched healthy cohort. A loss of throwing arm ER appeared to be the main factor behind shoulder ROM changes in the nTOS group.

Keywords: neurogenic thoracic outlet; baseball; shoulder range of motion

Thoracic outlet syndrome (TOS) is becoming a more recognized diagnosis in the throwing athlete. Some of the first reported cases of thoracic outlet compression in athletes were noted in the late 1970s in 3 baseball players who reported pain with throwing that caused them to halt participation.[20] More recently, there has been a heightened sense of awareness in baseball players who are evaluated with vague arm pain that often goes undiagnosed. These patients oftentimes have seen multiple medical providers and are misdiagnosed, underdiagnosed, or receive a delayed diagnosis secondary to a variation of clinical presentations with a lack of objective data to guide a definitive diagnosis.[10] TOS can be identified by the structure being compressed (artery, vein, or nerve) and is classified as 1 of 3 different types: arterial, venous, or neurogenic.[10,18] The most common type of TOS is neurogenic (nTOS) (>90%), where the brachial plexus becomes compressed because of soft tissue and osseous causes[10,18] or, in the case of baseball players, tractioned during throwing.[21] The repetitive use of the upper extremity in the throwing athlete is believed to contribute to the development of nTOS, where the brachial plexus undergoes repeated tension and/or compression with each throw.

The clinical symptoms of nTOS often include nonradicular distribution of pain, paresthesia, weakness in the upper extremity, cervical pain, and potentially

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occipital headaches.[10,18] In baseball players, arm or upper extremity heaviness is noted during the throwing motion. Additionally, these individuals will report a “loss of control” with their accuracy during throwing, with a subsequent decrease in velocity. In contrast, venous TOS (3%-5% of cases) may present as swelling in the arm, pain and/or an aching sensation in the upper extremity, and paresthesia in the hands and fingers.[18] Although less common (<1%), arterial TOS presentation involves digital ischemia, coldness, paresthesia, and pain in the hand, but pain is typically not found in the shoulder or cervical region. While any of the 3 presentations of TOS are possible in a baseball player, previous literature[1,19,22] supports a greater incidence of nTOS in these athletes.

Although there is some understanding of the signs and symptoms with which these patients may be evaluated, there is a lack of information regarding the overall objective clinical profile of these overhead athletes at the time of diagnosis. Earlier work in baseball players with a diagnosis of an ulnar collateral ligament (UCL) tear has presented objective data such as deficits in shoulder external rotation (ER) range of motion (ROM),[6,7] shoulder strength,[8] humeral retrotorsion,[11] and lower extremity balance.[6] Each of these studies helped to define the expected objective profile of a baseball player with UCL dysfunction. Clinically, the recognition of objective data, such as changes in shoulder ROM in a thrower, may be important to help define the diagnosis and guide impairment-based treatment. Therefore, the aim of this study was to compare the shoulder ROM characteristics of baseball players with a diagnosis of nTOS to a group of age-, activity-, and position-matched healthy controls without nTOS. The primary hypothesis was that baseball players with a diagnosis of nTOS would have a greater loss of passive shoulder ER ROM on their throwing arm when compared with the throwing arm of healthy controls. A secondary hypothesis was that baseball players with nTOS would demonstrate greater side-to-side total rotational motion (TRM) differences (TRM_{diff}) compared with the control group.

METHODS

The research procedures for this study were approved by an institutional review board. A total of 106 male competitive high school and collegiate baseball players volunteered to participate in this study during a 30-month time frame from 2015 to 2017. Participants eligible for the nTOS group were identified during regularly scheduled visits to the participating physician and/or physical therapist for

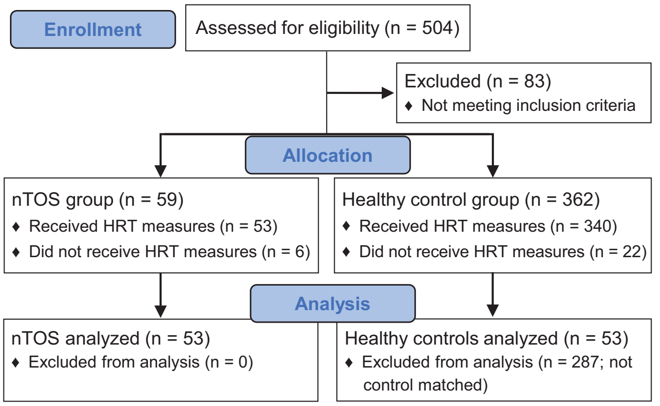

Figure 1. CONSORT (Consolidated Standards of Reporting Trials) flow diagram. HRT, anatomic humeral retrotorsion; nTOS, neurogenic thoracic outlet syndrome.

shoulder pain. Eligible healthy control participants were recruited from local high schools and colleges. For both the nTOS and the control groups, participants were considered for study participation if they were a baseball player between the ages of 14 and 22 years.

Players were included in the nTOS group if they were diagnosed with nTOS and (1) their ability to throw was affected by the injury, (2) they were unable to continue participating in baseball at the level before nTOS diagnosis, (3) their reported past history and clinical examination results were positive for symptoms of nTOS, and (4) they were attempting to return to sports at a competitive level. Players were excluded if they (1) had a previous diagnosis of nTOS, (2) had a previous shoulder surgery for labral or rotator cuff involvement, (3) did not undergo anatomic humeral retrotorsion (HRT) measurements, and (4) did not plan to return to baseball after treatment. The same exclusion criteria were applied to participants in the control group. Participants were enrolled into and gave consent for the study by an investigator in the outpatient sports medicine facility once they were confirmed to have met the inclusion and exclusion criteria (Figure 1). If the participant was a minor, child assent and parental permission were obtained.

A total of 53 baseball players with a diagnosis of nTOS were compared with 53 age-, experience-, and position-matched healthy baseball players. Of the participants diagnosed with nTOS, secondary diagnoses included shoulder impingement (57%), dynamic posterior shoulder instability (9%), Little Leaguer shoulder (5%), shoulder labral tear

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Ethical approval for this study was obtained from Texas Health Resources.

(8%), biceps tunnel disease (2%), and those with no secondary injury (19%).

Testing

The diagnosis of nTOS was made by a fellowship-trained, board-certified orthopaedic sports medicine surgeon (J.E.C.) with over 30 years of baseball medicine experience and included a comprehensive evaluation of both subjective and objective data. A subjective medical history was taken to include description, location, onset, and duration of signs and symptoms, as well as the effects on baseball performance and overall quality of life. The objective testing consisted of a clinical algorithm of TOS special tests (Adson test, modified Roos test, Full Abduction External Rotation Overhead Activity test), scapulothoracic assessments, upper limb neural tension tests, and shoulder ROM. Participants testing positive for nTOS reported some of the following symptoms: re-creation of symptoms with special tests, vague arm pain with throwing, paresthesia in throwing arm, neck pain, shoulder pain (to include trapezius and supraclavicular pain), arm heaviness or "dead arm," cramping in throwing arm hand, intermittent night arm pain, and a decline in baseball performance (loss of throwing velocity and control). These reports of symptoms are consistent with previous literature describing nTOS.[10,18]

In the nTOS participants, passive shoulder ROM testing was performed at their initial visit to the outpatient sports medicine facility. In addition, each nTOS participant completed a Kerlan Jobe Orthopaedic Clinic (KJOC) shoulder and elbow outcome form at the time of testing. Control participants were measured before their season using the same methods as the nTOS group. Before testing, reliability standards were established in pilot testing (20 participants) among those physical therapists participating in measurements for shoulder internal rotation (IR) (intraclass correlation coefficient[4] $[ICC]_{2,k}$, 0.97; SEM, 1.6) and ER ($ICC_{2,k}$, 0.97; SEM, 1.51). The shoulder ROM measurements have been previously described in the literature.[7,24] For glenohumeral joint ER, the participant was positioned supine with the arm abducted to 90° in the scapular plane. The scapula was stabilized by the therapist, and the arm was taken to the end of available ROM of the glenohumeral joint. This was defined as the point before the participant's scapula moved under the stabilizing hand. Measurements were taken using a digital inclinometer on the ulnar side of the forearm, with the instrument pressed firmly against the ulna.[12] For IR, the positioning of the participant was the same as for ER, but while the scapula was stabilized, the arm was moved into IR until the end range was reached or scapular motion was felt beneath the therapist's hand.

To minimize variability with HRT measurements, the primary investigator (J.C.G.) performed all the measurements, and intrarater reliability standards were established in pilot testing for HRT ($ICC_{3,1}$, 0.993; SEM, 2.77). During setup for HRT measurements, the participant was positioned supine with 90° of shoulder abduction and elbow flexion. The primary examiner used 1 hand to apply the diagnostic ultrasound head over the anterior aspect of the shoulder at the deepest point in the bicipital groove and in the plane of the treatment table.[12] This position was verified with a bubble level and aligned perpendicular with the long axis of the humerus in the frontal plane. The primary examiner's other hand was used to rotate the forearm until the bicipital groove appeared in the center of the ultrasound image and the apexes of the greater and lesser tubercles were parallel to the horizontal plane (Figure 2).

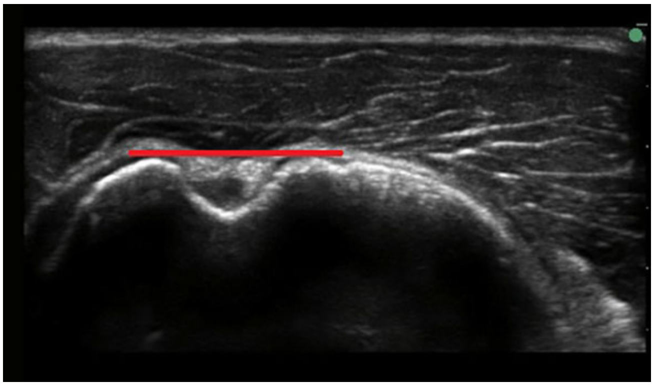

Figure 2. Ultrasound image of anatomic humeral retrotorsion measurement. The tuberosities were aligned while taking the measurement for anatomic humeral retrotorsion (line).

A transparent grid with horizontal lines was used to help determine the parallel positioning of the tubercles. When the greater and lesser tubercles were determined to be parallel, the second examiner used a digital inclinometer to measure the amount of humeral torsion. Two trials were completed for each arm (throwing and nonthrowing) while the primary examiner was blinded from the measured torsion value. The measures were averaged to obtain throwing HRT, nonthrowing HRT, and HRT limb differences (HRT_{diff}) (throwing HRT – nonthrowing HRT).

For this study, glenohumeral internal rotation difference (GIRD) was defined as a difference of IR of the throwing arm in relation to the nonthrowing arm and was calculated for both groups. Likewise, glenohumeral external rotation difference (GERD) was defined as a difference of ER of the throwing arm in relation to the nonthrowing arm. TRM was determined based on the combination of shoulder ER and IR of the participant's throwing arm in comparison with the nonthrowing arm. Side-to-side differences for GIRD, GERD, TRM, and HRT were calculated in each group and used for analysis.

Data Analysis

All statistical analyses were conducted with SPSS Statistics software (Version 25.0; IBM Corp). A priori statistical power analysis was performed using throwing arm ER as the primary outcome and determined that a total of 56 (28 in the nTOS group and 28 in the control group) participants would be needed to detect statistical significance with a moderate effect size based on an 80% power calculation. One-way multivariate analysis of variance was used for group comparisons between nTOS and controls for the main variables of throwing arm ER, GIRD, GERD, TRM_{diff},

TABLE 1
Participant Information[a]

	nTOS (n = 53)	Control (n = 53)	*P* Value
Age	17.2 ± 2.3	17.2 ± 2.4	>.99
Throwing limb			>.99
Right	47 (88.7)	47 (88.7)	
Left	6 (11.3)	6 (11.3)	
Height, cm	180.9 ± 10.1	183.9 ± 9.0	.166
Weight, kg	80.0 ± 13.3	83.8 ± 11.5	.133
Years of experience	12.7 ± 2.7	12.3 ± 2.9	.424

[a]Data are expressed as mean ± SD or n (%). nTOS, neurogenic thoracic outlet syndrome.

and HRT_{diff}. Separate univariate tests were conducted for each dependent variable once a significant interaction was determined. Independent *t* tests were run to compare descriptive statistics of age, height, weight, and years of playing experience between groups. Finally, for descriptive purposes, separate independent *t* tests were run for throwing arm IR, TRM, and HRT and nonthrowing arm variables. Statistical significance was set at $P < .05$.

RESULTS

There were no significant differences between groups for age, height, weight, or years of experience (Table 1). The nTOS group was made up of 69.8% pitchers, 22.6% infielders, 3.7% outfielders, and 3.7% catchers. Control participants consisted of 69.8% pitchers, 22.6% infielders, 1.9% outfielders, and 5.6% catchers. In the nTOS group, the mean duration of symptoms was 7.1 ± 10.9 months, and the average KJOC was 44.9 ± 13.2.

There was a significant main effect for group, $F(4,99) = 5.79$, $P < .001$. The nTOS cohort had significantly less throwing arm ER (nTOS: 103.4° ± 10.4°, control: 109.6° ± 7.5°; $P = .001$) and GERD (nTOS: 3.0° ± 9.2°, control: 8.8° ± 9.2°; $P = .002$) than the control cohort. In addition, TRM_{diff} was significantly greater in the nTOS group (–11.1° ± 11.1°) compared with the control group (–3.7° ± 9.4°) ($P < .001$) (Table 2).

TABLE 2
Range of Motion Comparison Between the nTOS and Control Groups[a]

	nTOS (n = 53)	Control (n = 53)	*P* Value
Throwing arm			
IR	24.5 ± 7.9	24.8 ± 8.8	.846
ER	103.4 ± 10.4	109.6 ± 7.5	**.001**
TRM	127.9 ± 13.2	134.5 ± 10.6	**.006**
HRT	14.7 ± 9.4	15.2 ± 9.8	.789
Nonthrowing arm			
IR	38.6 ± 7.3	37.3 ± 11.2	.472
ER	100.4 ± 11.3	100.9 ± 9.8	.812
TRM	138.9 ± 11.7	138.1 ± 12.3	.72
HRT	30.4 ± 10.0	32.1 ± 12.4	.44
TRM difference	–11.1 ± 11.1	–3.7 ± 9.4	**<.001**
HRT difference	–15.7 ± 10.2	–16.9 ± 9.3	.528
GERD	3.0 ± 9.2	8.8 ± 9.2	**.002**
GIRD	–14.1 ± 8.5	–12.5 ± 10.0	.366

[a]Data are expressed in degrees mean ± SD. Bolded *P* values indicate statistically a significant difference between groups ($P < .05$). ER, external rotation; GERD, glenohumeral external rotation difference; GIRD, glenohumeral internal rotation difference; HRT, anatomic humeral retrotorsion; IR, internal rotation; nTOS, neurogenic thoracic outlet syndrome; TRM, total rotational motion.

DISCUSSION

The findings from the current study are consistent with the original hypothesis that baseball players with a diagnosis of nTOS would have a significantly greater loss of throwing arm shoulder ER when compared with baseball players who were healthy. Additionally, TRM_{diff} in the nTOS group was greater compared with the healthy controls, with the loss of throwing arm ER being the main contributing factor to this difference. This loss of throwing arm ER is likely due to an irritated brachial plexus that can occur from the repeated stresses experienced during throwing. These results are not surprising, and the current loss of 6.2° of throwing arm ER is similar to previous data in baseball players with a diagnosis of a UCL tear (6.4° and 7.1°).[6,7] The biomechanics of the throwing motion likely play a role in explaining previously reported throwing arm ER loss[6,7] and also give insight into the current findings in the nTOS population.

Earlier biomechanical work has demonstrated that throwers who have greater shoulder ER during the throwing motion are able to create increased ball velocity.[23] As such, the shoulder experiences high forces during the late cocking phase of the throwing motion,[5] as maximum shoulder ER combined with increased shoulder horizontal abduction leads to anterior shear forces and potential for injury.[21] Additionally, previous work on 3-dimensional kinematics of the throwing motion demonstrates that opening the pelvis too early at stride foot contact is related to increased trunk tilt to the side of the nonthrowing arm.[14] When this contralateral trunk tilt becomes excessive at maximum shoulder ER, it is associated with increased throwing arm proximal shoulder joint forces.[15] Therefore, from a throwing performance standpoint, if the throwing arm "lags" or experiences increased horizontal abduction combined with maximum shoulder ER and excessive contralateral trunk tilt, it is possible that the adjacent soft tissue (including the brachial plexus originating from the cervical spine) would also undergo increased stress due to the increasing joint loads/forces incurred with these pathomechanics.

The baseball players in the current study who were diagnosed with nTOS demonstrate a throwing arm deficit of approximately 6° when compared with the throwing arm of the healthy controls (Figure 3).

We hypothesize that one of the explanations for this loss of shoulder ROM is the fact that these individuals with nTOS present with an upregulated (ie, hypersensitive) nervous system secondary to the repeated high forces to these neural tissues. This may include peripheral neuropathic pain with altered levels of excitability in the nervous

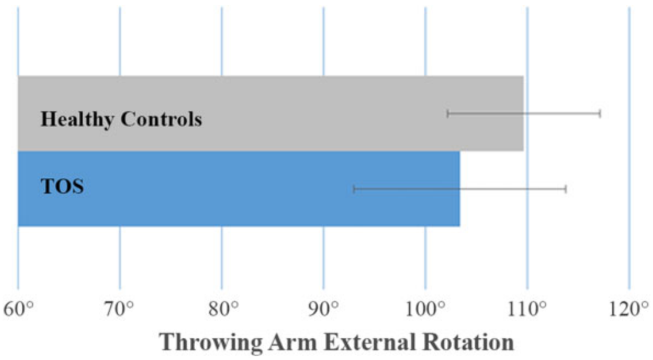

Figure 3. Comparison of throwing arm shoulder external rotation between the study groups. TOS, thoracic outlet syndrome.

system presenting as pain and paresthesia.[13] As a result, it is plausible that these athletes are guarding secondary to pain, thus limiting the amount of shoulder ER during measurements. The mean duration of symptoms in the nTOS group was 7.1 ± 10.9 months, which suggests that the clinical presentation was chronic in nature.

Consistent with previous reports[10] regarding diagnosis of thoracic outlet, the baseball players in the current study reported seeing multiple providers with misdiagnosis, underdiagnosis, and/or delayed diagnosis before being diagnosed with nTOS by the surgeon investigator (J.E.C.) in our study. As such, the delay in proper diagnosis may have contributed to neuromuscular adaptations at the shoulder (ie, decreased shoulder ER) in an effort to minimize stresses across the neural tissues during the throwing motion. This idea of reducing shoulder ER as a biomechanical adaptation has previously been reported in baseball players with a UCL injury[7] and may present similarly in this group of baseball players with nTOS.

In addition to neuromuscular adaptations as a possible factor for the loss of throwing arm ER in the nTOS group, changes in shoulder ROM in this population should be considered within the context of HRT. Earlier work examining the role of osseous changes in the humerus has demonstrated that as HRT increases, there is a shift toward increased shoulder ER,[3,9,16,17] followed by a subsequent decrease in shoulder IR.[3,16,17] There were no significant differences in GIRD between nTOS and healthy controls in the present data set, which is similar to previously published findings in baseball players with a UCL tear.[6,7] In the current study, despite the fact that throwing arm HRT and IR values were similar between the nTOS and control groups, throwing arm ER was different (less). These results suggest that based on the amount of throwing arm HRT in the nTOS group, one would expect to see values of shoulder ER similar to those in the control group. Thus, the loss of throwing arm ER is not related to osseous changes, but more than likely due to previously suggested neuromuscular adaptations or soft tissue changes.

While it was not the purpose of this study to investigate the short- or long-term outcomes of baseball players with nTOS, earlier work[1,19] has highlighted treatment options in participants who have been diagnosed with nTOS with comparable ages and activities (baseball) with the current study. In 232 competitive athletes with nTOS (mean age, 19 years; range, 13-67 years) undergoing first rib resection with scalenectomy, 63% of those played baseball or softball.[19] Similarly, of 27 high-performance athletes diagnosed with nTOS (average age, 19 years; range, 14-32 years) and seeking either conservative or surgical care, 15% of those included baseball players.[1] Conversely, the current study does not describe treatment options but does provide an objective clinical presentation of adolescent baseball players (age, 17.2 ± 2.3 years) who have been diagnosed with nTOS. These objective data can be used to guide clinical decision making based on the loss of throwing arm ER, which, in our experience, is often related to biomechanical alterations in the kinetic chain. Baseball players with nTOS are likely to have throwing arm ER loss, either as a result of having nTOS or as a cause of nTOS. Identification of the cause of throwing arm ER loss followed by restoration of that ROM should be considered an important part of the rehabilitation process. With this in mind, physical therapy treatment specific to nTOS may begin with efforts to decrease the sensitivity of the nervous system through postural corrections, neural mobilizations, and joint mobilizations of the cervical and thoracic spine. This algorithm has previously been described in the literature with noted success.[1,2] Once the nervous system has been calmed, focus can be shifted to restoration of scapular and rotator cuff strengthening and neuromuscular control, with an end goal of analyzing biomechanical movement patterns that may have contributed to the development of nTOS.

Furthermore, at present there is a lack of data on the use of gabapentin or other similar medications to treat nTOS as part of a conservative rehabilitation algorithm. Because of this, we cannot currently recommend the use of nerve pain–relieving medications in the treatment of nTOS in the population of baseball players described in the present study; however, future research on the potential effectiveness of such medications is warranted.

Limitations

The current study did not examine throwing mechanics and therefore cannot suggest a cause-and-effect relationship in this population; however, it is not unreasonable to believe a possible relationship exists, and this idea of thinking is consistent with our experience of working with this population. Nevertheless, further work examining this association between throwing biomechanics and nTOS is warranted. Likewise, the presence of secondary diagnoses such as shoulder impingement, dynamic posterior shoulder instability, Little Leaguer shoulder, shoulder labral tear, and biceps tunnel disease may have also contributed to altered throwing biomechanics, neuromuscular adaptations, and subsequent nTOS in this population. While some imaging was performed in the workup of these participants, it is believed that the diagnosis of nTOS is challenging and dependent upon clinical history, presentation, and physical examination of the patient.[10] As such, the diagnosis of nTOS in the current study was based on the clinical examination of a fellowship-trained, board-certified orthopaedic sports medicine surgeon who has extensive experience with nTOS in the baseball population (J.E.C.). The participants in this study displayed documented thoracic outlet compression that resulted in symptoms consistent with mild (tingling/numbness occurs within 10 seconds of Full Abduction External Rotation Overhead Activity test) to severe (reproduction of symptoms such as pain and numbness/tingling within 10 seconds of Full Abduction External Rotation Overhead Activity test) nTOS. Last, the self-reported symptoms of the current participants may be biased based on their ability to recall when the symptoms began, the overall intensity, and the time points in which they experienced pain during the throwing motion. Great care was taken to obtain an accurate history to account for the variety of possibilities of pain contributors, and the pattern of descriptions that emerged across all participants was consistent with previously reported symptoms of nTOS.[1,2,10,18,22]

CONCLUSION

Adolescent baseball players diagnosed with nTOS present with shoulder ROM deficits when compared with a matched healthy cohort. A loss of throwing arm ER appears to be the main factor behind shoulder ROM changes in the nTOS group. These findings provide an objective clinical presentation for adolescent baseball players diagnosed with nTOS that may help to guide clinical decision making for an appropriate course of treatment. However, further research is likely warranted to further verify and reproduce findings.

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