Assessment of ultrasonography as a diagnostic tool in shoulder pain and alterations in glenohumeral range of motion in tennis players

Krzysztof Guzowski[1], Artur Stolarczyk[2], Zbigniew Czyrny[1], Anna Dębek[1], Bernard Kranc[1]

[1]Center of Sports Rehabilitation, Warsaw, Poland

[2]Orthopaedic and Rehabilitation Department, Medical University of Warsaw, Warsaw, Poland

Abstract

Introduction: Shoulder pain and alterations in the range of motion are common disorders in tennis players. However, the relation between shoulder structures and these conditions is unknown.

Aim: To evaluate whether, using ultrasonography, one can identify tennis players with shoulder pain and those having specific changes of the range of rotation of the glenohumeral joint.

Material and methods: A total of 66 subjects were assessed through examination of the range of rotation of the glenohumeral joint and ultrasonography.

Results: The study group consisted of 37 people with shoulder pain (24.2 ±8.6 years) and the control group included 29 subjects without shoulder pain (21.9 ±10.8 years). The prevalence of pathologies of the supraspinatus (SSP), infraspinatus (ISP) or subscapularis (SSC) was significantly higher in the study group than in the control group (p = 0.044) but solely for the combined pathologies. The incidence rate of pathological shoulder changes (the SSP, ISP, SSC and the subacromial bursa) was not correlated with the ranges of rotations or intensity of pain. Glenohumeral internal rotation deficit (GIRD), total rotational motion (TROM) deficit and external rotation deficiency (ERD) were independent of pathological shoulder changes, except the significantly higher prevalence of SSP pathologies among subjects with ERD.

Conclusions: Ultrasonography could be helpful in identifying tennis players with painful shoulder having rotator cuff pathologies. However, the ability of the method to identify players having specific changes of the range of rotation of the glenohumeral joint is limited, with the exception of tennis players with ERD having SSP pathologies.

Key words: diagnosis, ultrasonography, shoulder pain, range of motion, tennis, shoulder injuries.

Introduction

According to epidemiologic research, trauma of the upper limb in tennis players represents 30–39% of all injuries [1, 2]. Shoulder injury is the most frequent one and it is typically accompanied by pathology of the supraspinatus and the infraspinatus as well as the impingement syndrome [2]. Between 7% and 30% of all injuries to tennis players are shoulder problems [3, 4].

The prevalence of shoulder pain rises with age, especially in highly developed societies where people perform more and more physical activities as a pastime. Those disciplines which include throwing and overhead activity, such as volleyball, handball and tennis, are the ones which cause injuries most often [3, 4].

Frequently performed tennis strokes starting at a young age impact formation of bones and lead to

Address for correspondence

Krzysztof Guzowski, Center of Sports Rehabilitation, 2 Przechodnia St, 00-100 Warsaw, Poland, e-mail: krzysztofguzowski@vp.pl

a change of tension and rebuilding of the soft tissues. It results in an increase of the range of external rotation (ER) and a decrease of internal rotation (IR) of the glenohumeral joint on the dominant side [5–8].

In 2013 Kibler *et al.* [9] defined a limitation of the range of IR of the glenohumeral joint (glenohumeral internal rotation deficit – GIRD) as asymmetry within IR between the sides which is more than 18°, and an insufficiency of ER (external rotation deficiency – ERD) as a lack of increase of the range of ER on the dominant side of at least 5° in comparison to the non-dominant shoulder.

Wilk *et al.* [10, 11] suggested comparing the sum of IR and ER of the glenohumeral joint (total rotational motion – TROM) of both sides. TROM deficit was defined as the difference between the sides greater than 5°.

There is research evidence showing that specific changes of the range of rotation of the glenohumeral joint are more often observed in overhead athletes feeling shoulder pain [7, 12, 13]. As described in medical literature, the pathologic cascade of the thrower's shoulder starts with acquired asymptomatic posteroinferior capsular contracture (which restricts internal rotation), continues through the phase of painful shoulder (which is often neglected) and could lead to serious structural changes of the rotator cuff and the articular labrum [5]. To the authors' knowledge there is a lack of research analyzing exactly in which structures of the shoulder there are pathological changes in the situation when a tennis player complains about shoulder pain or when one has alterations in the glenohumeral range of motion.

Aim

The aim of this study was to evaluate whether using ultrasound examination we can identify tennis players with shoulder pain and those having specific changes of the range of rotation of the glenohumeral joint.

Material and methods

Subjects

Sixty-six tennis players were examined: 42 (63.6%) men and 24 (36.4%) women.

Cases of shoulder surgery, adhesive capsulitis, dislocation and fracture of the scapula or humeral bone were excluded from the study.

The study received the positive opinion of the Bioethics Committee of the Medical University of Warsaw.

Each of the patients was informed in writing about the study procedure. A patient or, in the case of minors, parents signed consent to undergo the study and filled in a form including exclusion criteria.

Medical history

The standardized Visual Analog Scale (VAS) was used to assess shoulder pain during and after playing tennis. The duration of shoulder pain was also determined.

Range of rotation of the glenohumeral joint

Examination of the range of IR and ER of the glenohumeral joint was performed. Right and left limbs were tested at random. Measurement of IR and ER was performed in a sequence tailored to each subject. The examiner was unaware of the examinee's handedness and the group to which he or she belonged. The measurement was taken at 90° of abduction with a digital inclinometer (PRO 360 Digital Protractor), following the procedures described in previous literature [14–17].

Jobe's test

To identify shoulder pain and weakness we used Jobe's test. The examiner positioned the subject's glenohumeral joints in internal rotation and abduction in scaption (the scapular plane), following the suggestions of the test authors [18, 19]. Next, the examiner pressed the subject's wrists towards the ground with his hands. The subject was supposed to hold the taken position. The test was considered positive when there was pain and/or weakness.

Neer's test

To identify shoulder pain we also used Neer's test. The examiner used one hand to stabilize the subject's scapula, and the other to perform a passive elevation through flexion and sustaining the glenohumeral joint in internal rotation – according to Neer's description from 1983 [20]. Next, elevation was performed in scaption. The test was considered positive when there was pain.

Krzysztof Guzowski, Artur Stolarczyk, Zbigniew Czyrny, Anna Dębek, Bernard Kranc

Ultrasonography

To assess the rotator cuff, the subacromial bursa and to define the prevalence of subacromial impingement we performed ultrasonography (US) of the dominant shoulder. The radiologist was unaware of the patient's shoulder condition (painful or not painful).

Toshiba Aplio MX apparatus was used in all examinations. The apparatus was equipped with a linear transducer of 5–18 MHz. The examination was performed by three highly qualified ultrasonographers (doctors). The protocol of the examination was the result of the cooperation of all three specialists (Table I).

The examination analyzed the state of the supraspinatus (SSP), infraspinatus (ISP) and subscapularis (SSC) tendons with capsuloligamentous complex. The teres minor was not taken into consideration because its damage is extremely rare in tennis players.

During the examination the specialists assessed pathologies of tendons of the examined muscles (full or partial rupture), capsuloligamentous structures (full or partial rupture), entheses of tendons (scars, erosions or calcific cavities) and entheses of capsuloligamentous structures (scars, erosions or calcific cavities).

In the subacromial bursa the presence of an increased amount of liquid (effusion), synovial swelling and fibrosis was examined as a feature of acute, chronic or post-inflammatory condition.

We used the phrase "pathological shoulder changes" for any deviation from the norm for the SSP, ISP, SSC and the subacromial bursa.

The presence of signs and symptoms of subacromial impingement (such as the greater tubercle of the humerus not being pulled in fully under the acromion or presence of pain) were examined during passive and active abduction of the glenohumeral joint within 0–90°.

Statistical analysis

The calculations were done using the STATISTICA 12.0 PL program (StatSoft, Inc. 2015, Tulsa, USA). Statistically significant results were those where making type I error probability was less than 5% ($p < 0.05$). Data analysis used both parametric and non-parametric tests (depending on analyzed data distribution).

The auxiliary calculations, tables and graphs were prepared with MS Excel (Microsoft Corporation 2010, Redmond, USA).

In reference to detecting the pathology of given shoulder structures we also calculated sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and accuracy together with 95% confidence intervals for Neer's test and Jobe's test.

Results

The subjects were divided into two groups (the study group and the control group).

The study group consisted of 37 players: 22 (59.5%) men and 15 (40.5%) women suffering from shoulder pain, aged 11–40 (mean: 24.2 ±8.6) and who play tennis at least twice a week, for 2 previous years minimum. The group included tennis players experiencing shoul-

Table I. Shoulder ultrasound examination protocol

Sequence	Structure and condition	View	Positioning and comments
1	Subscapularis	Long and short axis	Patient externally rotates the arm with the elbow flexed at 90°
2	Supraspinatus	Long and short axis	Patient takes the posterior position of the arm with the dorsal surface of the hand on the contralateral iliac wing or the palmar surface of the hand on the ipsilateral iliac wing (on the back pocket), which abducts and internally rotates the glenohumeral joint
3	Subacromial bursa	Long axis	The same position as for SSP. Longitudinal view of the SSP with hypoechoic overlying line representing subacromial bursa
4	Infraspinatus	Long axis	The arm in neutral position (hand on thigh) or across the front of the chest with the hand placed over the opposite shoulder. ISP is the only structure examined from a posterior view of the shoulder
5	Subacromial impingement	–	Passive and active abduction of the glenohumeral joint within 0–90° (starting from the neutral position). Assessment is done by moving the transducer over the lateral edge of the acromion

der pain in at least one of the situations during the period of the previous 12 months – while playing tennis and directly after/the day after playing tennis.

The control group consisted of 29 tennis players: 20 (69.0%) men and 9 (31.0%) women without shoulder pain, aged 10–39 (21.9 ±10.8), playing tennis at least twice a week for the previous 2 years or more.

There were no significant differences in age and sex in both groups.

Shoulder pain

In the study group shoulder pain felt during playing tennis was estimated at 3.8 ±2.2 in the VAS (0–10). Fifty-four percent of subjects claimed that the condition was equal or more than 4 in the VAS. Shoulder pain which occurred after playing tennis was estimated at 3.4 ±2.4. 43.2% of tennis players suffered from this kind of pain at the level of 4 or more in the VAS.

Subjects with significant pain were defined as those reporting the level of shoulder pain in the VAS within the range 4–10, both during and after play. Subjects without significant pain were those suffering from shoulder pain in both situations within the range 0–3. In this way we missed some tennis players complaining about pain within the range 4–10 only in one of the mentioned situations, but probably it increased the chance to reveal possible relations.

It was not confirmed that the duration of shoulder pain (< 1 month, 1–3 months or > 3 months) was significantly correlated either with the occurrence of pathological shoulder changes or with the occurrence of features of subacromial impingement (Table II).

On the dominant side Jobe's test was positive among 43.2% of the study group and 0% in the control group. On the non-dominant side the test was negative in all subjects. On the dominant side Neer's test was positive among 54% of the study group and 0% in the control group. On the non-dominant side the test was negative in all subjects (Table III).

Range of motion

Comparing the study and the control groups we identified a statistically significant difference ($p = 0.030$) in the range of IR of the glenohumeral joint on the dominant side: 47.5 ±11.3° and 53.6 ±10.9° respectively. There was a non-significantly ($p = 0.084$) smaller TROM on the dominant side in the study group (146.0 ±17.3°) in comparison to the control group (152.8 ±13.3°) (Table III).

After including the side of measurement the analysis of variance in the intragroup scheme showed a significant difference between IR and ER ($p < 0.001$) – IR was bigger on the non-dominant side, while ER was bigger on the dominant side (Figure 1).

The GIRD, TROM deficit and ERD prevalence did not relate to the group ($p > 0.05$). The GIRD, TROM deficit and ERD were identified in 17%, 58% and 55% of tennis players respectively (Table IV).

Ultrasonography findings

It was observed that 72.7% of subjects had pathological shoulder changes. The maximum number of pathological shoulder changes was 4 (mean: 1.73; SD = 0.89; median = 1). Any pathologies visible in ultrasonography of the SSP, ISP or SSC along with their tendons, entheses and capsuloligamentous structures were more often identified in the study group (59.5% of the subjects) than in the control group (34.5%). It was a statistically significant difference ($p = 0.044$). There was a non-significantly higher incidence of pathologies of the ISP in the study group than in the control group ($p = 0.072$). Pathologies of the entheses of capsuloligamentous structures were present in 28 subjects while pathologies of the entheses of tendons were present in 9 subjects. There were 2 partial ruptures of the tendons (SSP and SSC) and also 2 partial ruptures of the capsuloligamentous structures (ISP and SSC) at any place apart from the entheses. Further examination of the entheses pathologies revealed 38 scars

Table II. Relationship between pathological changes of the shoulder and features of subacromial impingement occurrence vs. duration of shoulder pain; Spearman's rho correlation coefficient

Variable	Duration of shoulder pain	
	rho	P-value
Incidence rate	0.203	0.102
SSP	0.181	0.145
ISP	0.197	0.113
SSC	0.151	0.227
Bursa	−0.062	0.621
Impingement	0.174	0.163

Krzysztof Guzowski, Artur Stolarczyk, Zbigniew Czyrny, Anna Dębek, Bernard Kranc

Table III. Relations between values, prevalence and incidence of studied features vs. group

Variable	Tennis players without shoulder pain ($n = 29$)	Tennis players with shoulder pain ($n = 37$)	P-value
Internal rotation, mean ± SD [°]:			
Dominant side	53.6 ±10.9	47.5 ±11.3	0.03
Non-dominant side	61.5 ±10.5	59.3 ±12.2	0.45
External rotation, mean ± SD [°]:			
Dominant side	99.2 ±11.8	98.5 ±10.9	0.801
Non-dominant side	94.8 ±10.2	92.6 ±12.6	0.444
Sum of rotations, mean ± SD [°]:			
Dominant side	152.8 ±13.3	146.0 ±17.3	0.084
Non-dominant side	156.3 ±13.5	151.9 ±18.9	0.3
Subjects with pathological shoulder changes, n (% of group)	24 (65.5)	29 (78.4)	0.444
Subjects with SSP, ISP or SSC pathology, n (% of group)	10 (34.5)	22 (59.5)	0.044
Subjects with SSP pathology, n (% of group)	4 (13.8)	8 (21.6)	0.413
Subjects with ISP pathology, n (% of group)	6 (20.7)	15 (40.5)	0.086
Subjects with SSC pathology, n (% of group)	8 (27.6)	14 (37.8)	0.381
Subjects with bursa pathology, n (% of group)	13 (44.8)	14 (37.8)	0.567
Pathology of entheses of tendons/subject, mean ± SD	0.07 ±0.26	0.24 ±0.55	0.13
Pathology of the entheses of capsuloligamentous structures/subject, mean ± SD	0.62 ±0.98	0.78 ±1.00	0.518
Subjects with pathology of entheses of tendons, n (% of group)	2 (6.9)	7 (18.9)	0.279
Subjects with pathology of entheses of capsuloligamentous structures, n (% of group)	10 (34.5)	18 (48.7)	0.248
SSP pathology/subject, mean ± SD	0.17 ±0.47	0.22 ±0.42	0.69
ISP pathology/subject, mean ± SD	0.21 ±0.41	0.43 ±0.55	0.072
SSC pathology/subject, mean ± SD	0.31 ±0.54	0.49 ±0.80	0.315
Subjects with lack of full abduction, n (% of group)	0 (0.0)	0 (0.0)	–
Subjects with painful abduction, n (% of group)	0 (0.0)	3 (8.1)	0.080
Subjects with full rupture of the tendon or capsuloligamentous structure, n (% of group)	0 (0.0)	0 (0.0)	–
Jobe – dominant side, n (% of group)	0 (0.0)	16 (43.2)	< 0.001
Jobe – non-dominant side, n (% of group)	0 (0.0)	0 (0.0)	–
Neer – dominant side, n (% of group)	0 (0.0)	20 (54.0)	< 0.001
Neer – non-dominant side, n (% of group)	0 (0.0)	0 (0.0)	–

M – mean, SD – standard deviation, n – quantity of population.

and 25 erosions. A calcific cavity was present in 1 person. Within the entheses of capsuloligamentous structures 52 and within the entheses of tendons 11 changes were diagnosed. None of the subjects was identified to have a full rupture of the tendon or capsuloligamentous structure. Pathologies within the bursa occurred in 37.8% of the study group members and 44.8% of the control group. We diag-

nosed 21 cases of fibrosis and 13 cases of increased amount of liquid or swelling. None of the subjects had subacromial impingement understood as the lack of full abduction (the greater tubercle of the humerus not being pulled in fully under the acromion). Passive and active abduction of the glenohumeral joint performed during the examination of the subacromial impingement under ultrasonography guidance was painful in 3 cases of the study group and in none in the control group ($p > 0.05$) (Table III).

The incidence rate of pathological shoulder changes was positively correlated with ageing but was not correlated with Neer's and Jobe's test results, the ranges of rotations of the glenohumeral joint, the time span of the pain or the intensity of the pain (Table V).

The relation between the pathologies and the range of rotation of the glenohumeral joint

In the context of the range of rotation of the glenohumeral joint and the prevalence of pathologies of the SSP, ISP or SSC we did not find significant differences ($p > 0.05$) between the groups of tennis players with significant shoulder pain (VAS 4–10) and those without significant shoulder pain (VAS 0–3) (Table VI).

The occurrence of GIRD and TROM deficit was independent of pathological shoulder changes. The occurrence of ERD was independent of most pathological shoulder changes. We only observed a significantly higher percentage of people with SSP pathologies in the ERD group (Table VII).

The Mann-Whitney U test analysis did not confirm the dependence between the occurrence of pathologies of the SSP, ISP or SSC and range of internal and external rotation on the dominant side. The pathologies of the subacromial bursa did not influence the differences within external and internal rotation on the dominant side (Table VIII).
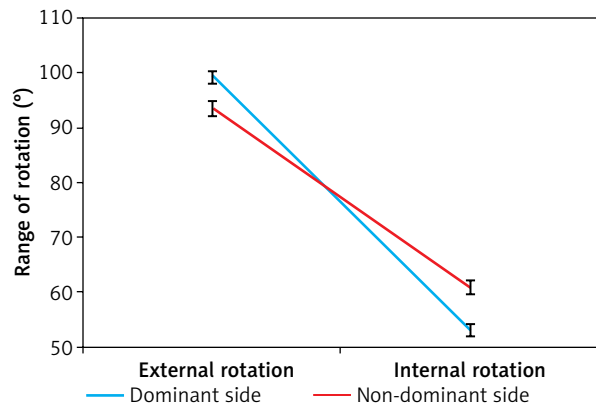

Figure 1. Range of external and internal rotation (± SE) depending on the side (dominant or non-dominant)

Diagnostic value of Jobe's test and Neer's test – the assessment

The following parameters were taken into consideration when assessing the diagnostic value of both tests: sensitivity, specificity, positive predictive value, negative predictive value and test accuracy. The dominant side was analyzed. Ultrasonography was used to identify any shoulder pathologies. Both tests are assessed below.

In Jobe's test, sensitivity (describing the test ability to detect pathology) had the highest score for the pathology of tendon entheses (44.4%) and the lowest for the pathology of capsuloligamentous structure entheses (25.0%). Specificity did not reveal differences between given structures; a slightly higher result was found for the pathology of SSP, ISP or SSC. The lowest positive predictive value was found for the pathology of tendon entheses (25%). For the remaining pathologies the positive prognostic value was similar.

The assessment of the diagnostic value was also supported by the evaluated accuracy parameter, which indicates the percentage of the correct test classification for the given structures. The accuracy

Table IV. Relations between GIRD, TROM deficit and ERD prevalence vs. group

Variable	Tennis players without shoulder pain	Tennis players with shoulder pain	General
Subjects with GIRD, n (% of group)	3 (10.3[a])	8 (21.6[a])	11 (16.70)
Subjects with TROM deficit, n (% of group)	14 (48.3[a])	24 (64.9[a])	38 (57.60)
Subjects with ERD, n (% of group)	15 (51.7[a])	21 (56.8[a])	36 (54.50)

Each letter in the superscript means a subset of category of the group, whose proportions of the column within each line do not differ statistically significantly from one another at the level of 0.05.

Krzysztof Guzowski, Artur Stolarczyk, Zbigniew Czyrny, Anna Dębek, Bernard Kranc

Table V. Correlation between Neer's and Jobe's test results, ranges of rotations, age, time span of the pain and intensity of the pain vs. incidence rate of pathological shoulder changes (number of pathological shoulder changes per subject); Pearson's correlation coefficient (r)

Variable	Incidence rate
Neer D	0.098
Jobe D	0.0109
Internal rotation D	−0.163
External rotation D	−0.054
Sum of rotations D	−0.157
Age	0.385*
Time span of pain	0.149
Pain during play	0.117
Pain after play	0.084

*$p < 0.05$, D – dominant side.

index was crucial for the assessment of the diagnostic value. The highest accuracy was found for tendon entheses pathology (74.2%) – such a high result may be explained with the high negative prognostic value (90%). The remaining structures showed similar test accuracy. Table IX summarizes the results of Jobe's test.

For Neer's test as far as sensitivity is concerned the highest score was found for the pathology of the SSP, ISP or SSC (34.4%) and the lowest for the pathology of tendon entheses (22.2%). We note that the differences here were not as apparent as in the case of Jobe's test. Considering specificity, the differences between the given structures were not significant with scores over 66%. The pathology of tendon entheses had the lowest positive prognostic value (10%) and the highest value was found for the pathology of the SSP, ISP or SSC (55%).

The highest accuracy was for the pathology of the entheses of the tendon (62.1%) – the result may be explained with the high percentage of the negative prognostic value (84.8%). For the remaining struc-

Table VI. Relation between range of rotation of the glenohumeral joint on dominant and non-dominant sides and prevalence of pathology of SSP, ISP or SSC vs. occurrence of significant pain among tennis players with shoulder pain

Variable	Tennis players with shoulder pain		
	VAS 0–3 ($n = 13$)	VAS 4–10 ($n = 12$)	*P*-value
Internal rotation, mean ± SD [°]	46.9 ±11.0	48.6 ±11.9	0.972
External rotation, mean ± SD [°]	101.5 ±10.5	96.6 ±8.7	0.369
Sums of rotations, mean ± SD [°]	148.4 ±16.3	145.2 ±15.2	0.580
Subjects with SSP, ISP or SSC pathology, n (% of group)	8 (61.5)	8 (66.7)	1.000

Table VII. Relation between pathological shoulder changes prevalence vs. GIRD, TROM deficit and ERD occurrence

Variable	GIRD			TROM deficit			ERD		
	No	Yes	General	No	Yes	General	No	Yes	General
Subjects with SSP pathology (% of group)	14.0[a]	23.1[a]	15.20	15.2[a]	15.1[a]	15.20	6.4[a]	23.1[b]	15.20
Subjects with ISP pathology (% of group)	30.2[a]	46.2[a]	32.30	39.1[a]	26.4[a]	32.30	27.7[a]	36.5[a]	32.30
Subjects with SSC pathology (% of group)	30.2[a]	46.2[a]	32.30	34.8[a]	30.2[a]	32.30	23.4[a]	40.4[a]	32.30
Subjects with bursa pathology (% of group)	39.5[a]	38.5[a]	39.40	37.0[a]	41.5[a]	39.40	34.0[a]	44.2[a]	39.40

Each letter in the superscript means a subset of the category of GIRD, TROM deficit and ERD, whose proportions of the column within each line do not differ statistically significantly from one another at the level of 0.05.

Table VIII. Relation between range of internal and external rotation vs. occurrence of pathologies of SSP, ISP, SSC and subacromial bursa

Variable		No pathologies		Pathologies		Mann-Whitney *U* test	
		Mean	Standard deviation	Mean	Standard deviation	Z	P-value
Internal rotation	SSP	99.41	11.47	95.92	10.14	0.83	> 0.05
	ISP	98.31	11.14	99.76	11.70	0.04	> 0.05
	SSC	100.07	11.37	96.18	10.80	1.48	> 0.05
	Bursa	49.92	11.11	50.59	12.14	1.12	> 0.05
External rotation	SSP	51.07	11.82	46.25	9.02	1.51	> 0.05
	ISP	50.58	11.82	49.38	10.86	1.05	> 0.05
	SSC	50.34	11.84	49.91	10.91	0.03	> 0.05
	Bursa	100.00	11.24	97.00	11.23	0.20	> 0.05

Table IX. Assessment of diagnostic value of Jobe's test

Jobe's test	Bursa pathology	SSP/ISP/SSC pathology	Pathology of entheses of tendons	Pathology of entheses of capsuloligamentous structures
Sensitivity % (95% CI)	25.9 (21.0–30.8)	31.3 (26.4–36.2)	44.4 (39.5–49.3)	25.0 (20.1–29.9)
Specificity % (95% CI)	79.5 (74.6–84.4)	82.4 (77.5–87.3)	79.0 (74.1–83.9)	76.3 (71.4–81.2)
PPV % (95% CI)	46.7 (41.8–51.6)	62.5 (57.6–67.4)	25.0 (20.1–29.9)	43.8 (38.9–48.7)
NPV % (95% CI)	60.8 (55.9–65.7)	56.0 (51.1–60.9)	90.0 (85.1–94.9)	58.0 (53.1–62.9)
Accuracy % (95% CI)	57.6 (52.7–62.5)	57.6 (52.7–62.5)	74.2 (69.3–79.1)	54.6 (49.7–59.5)

Table X. Assessment of diagnostic value of Neer's test

Neer's test	Bursa pathology	SSP/ISP/SSC pathology	Pathology of entheses of tendons	Pathology of entheses of capsuloligamentous structures
Sensitivity % (95% CI)	25.9 (22.0–29.8)	34.4 (30.5–38.3)	22.2 (18.3–26.1)	32.1 (28.2–36.0)
Specificity % (95% CI)	66.7 (62.8–70.6)	73.5 (69.6–77.4)	68.4 (64.5–72.3)	71.1 (67.2–75.0)
PPV % (95% CI)	35.0 (31.1–38.9)	55.0 (51.1–58.9)	10.0 (6.1–13.9)	45.0 (41.1–48.9)
NPV % (95% CI)	56.5 (52.6–60.4)	54.4 (50.5–58.3)	84.8 (80.9–88.7)	58.7 (54.8–62.6)
Accuracy % (95% CI)	50.0 (46.1–53.9)	54.6 (50.7–58.5)	62.1 (58.2–66.0)	54.6 (50.7–58.5)

tures the accuracy of the test was similar. Table X summarizes the results of Neer's test.

Discussion

This study was performed in a group of tennis players to determine whether US can identify painful shoulders and specific alterations in the range of motion.

An important issue in the context of accuracy is the interpretation of the ultrasonography examination result by a radiologist. The study of Scheel *et al.* [21] assessed the interobserver reliability among independent radiologists. The modified κ factor of

agreement (proposed by Craig [22]) for the shoulder examination was estimated to be 0.76, which was considered a good result. It was concluded that the different interpretation of the image was due to a lack of standards both in the scanning techniques as well as in the description of the results (definitions of pathological findings). In our current study both factors were reduced because the diagnosticians were in fact not independent: they consulted and agreed upon the same protocol, and, in general, shared the same diagnostic practices. We believe that, altogether, such correlation between the examiners positively influenced the consistency of the results.

Considering the facts that none of the subjects was identified with a full rupture of the tendon or capsuloligamentous structure and the incidence rate of the pathological shoulder changes rose with age, it is suggested that the pathologies in this area of the body develop slowly in tennis players.

Rotator cuff is a multilayer structure built of interweaving fibers of tendons and capsuloligamentous complex. The outer tendon part plays the dynamic role while the inner capsuloligamentous layer controls the joint statically. The thickness of both is similar. The pathological changes of both layers may have a negative influence on the rotator cuff and the glenohumeral joint function. Thus, during ultrasound examination they should be identified and diagnosed separately [23–25].

Most damage of the rotator cuff was on the joint side (capsuloligamentous structures), which is in line with previous results from the literature [26–29]. More precisely, the pathological changes occurred most often in the region of the entheses of the capsuloligamentous structures. It casts doubt on Neer's hypothesis stating that 95% of all the pathologies of the rotator cuff are due to the irritation by the anteroinferior aspect of the acromion [30]. If this statement is correct, damage of the rotator cuff should occur mainly on the bursa side, so the tendons would be the structures most exposed to injury. A more probable genesis of the pathologies of the structures on the joint side in tennis players is the overload, meaning the inner factors (compressive and shear forces) which cause the degenerative changes (vascularisation disorders, fiber disorientation, fat infiltration, increase in the number of cells) [26, 31, 32]. Ultrasonic findings of a hypoechoic area correlate with the histologic findings described above [33, 34]. It is also worthwhile noting that in

such cases as rotator cuff tendinosis, tears may develop due to tendon weakness [35, 36].

A lot of specialists claim that joint side rotator cuff damage is due to a conflict between the SSP and ISP versus the posterosuperior glenoid rim, known as the posterior or internal impingement syndrome [29, 37–39]. It was proved that tightness of the posteroinferior capsule of the glenohumeral joint increased the glenohumeral contact pressure at maximum ER, causing forceful internal impingement [40].

The results of the ultrasound examination suggest that subacromial impingement does not occur in tennis players as often as most specialists think or it happens only during fast, rapid movements of the upper limb which occur on the tennis court but are impossible to simulate in the physician's office. It is likely that subacromial impingement can be observed in ultrasonography only at an advanced stage.

The pathologies in the subacromial bursa occurred almost as often as in the rotator cuff and with the same prevalence in both groups. The most common finding in the bursa was fibrosis, which could indicate a post-inflammatory condition. This in turn could be the cause of the pain complaints of the shoulder during the last 12 months.

It was suggested by other authors that the stiffness, shortening and scar formation of the posteroinferior area of the glenohumeral joint (joint capsule, ligaments, muscles and fascia) caused the migration of the humeral head, which in turn might lessen the subacromial space and increase the peel-back forces on the labrum while performing the serve [5, 41–44]. Such a phenomenon as limitation of the glenohumeral joint movement, which itself is not a pathology, may cause overload and damage of the shoulder structures.

It was also proved that not every decrease of IR was pathological and did not have to correlate with an increased likelihood of injury of the shoulder. In overhead athletes GIRD on the dominant side is a natural phenomenon to be expected [12, 15]. It is highly probable that such physiological limitation of IR on the dominant side in tennis players is linked to the increase of the angle of retroversion of the humeral head. Probably pathological GIRD is linked to the tightness of soft tissues. The studies and observations of Burkhart et al. [45] suggested that as long as the limitation of IR was lower or equal in the gain of ER the throwing shoulder had unaltered kinematics and it functioned correctly.

The current study confirms that a common feature for the tennis players with and without shoulder pain is that they have significantly less IR and more ER on the dominant side as compared to the non-dominant. It indicates that this change in the range of motion could be tennis-related. The results do not unambiguously link GIRD with shoulder pain, as other authors proposed [7, 13], but still it reveals that the range of IR on the dominant side is significantly smaller in tennis players with shoulder pain. In the study group there was non-significantly smaller TROM on the dominant side as compared to the control group. We conclude that prevalence of GIRD, TROM deficit and ERD were independent of the group and that the ranges of the rotations were independent of the occurrence of significant pain.

Based on the results, the relation between the shoulder structure and the range of motion is not clear. There was no correlation between the incidence rate of pathological shoulder changes and the ranges of rotations of the glenohumeral joint. The range of internal and external rotation on the dominant side was the same in the people with SSP, ISP, SSC or bursa pathologies as in the people without it. The occurrence of GIRD, TROM deficit and ERD was independent of the prevalence of pathological shoulder changes. There was only one exception: a higher percentage of people with SSP pathologies was present among the group with ERD. Therefore, in tennis players special attention should be paid to mobilization of the range of ER on the dominant side.

Jobe's test is used in the diagnostics of damage of the supraspinatus [31]. The methodology of performing Jobe's test needs the subject's muscle force to keep the upper limbs still. As the entheses of tendons may undergo more strain during muscle contraction than non-contractile capsuloligamentous structures, it is not surprising that this study presented the highest sensitivity (44.4%) and accuracy (74.2%) compared to pathologies of other structures.

Neer's test is the tool universally used to identify subacromial impingement. In our study Neer's test was predominantly positive on the dominant side (54% of subjects from the study group). Subacromial impingement, understood as the greater tubercle of the humerus not being pulled in fully under the acromion during abduction, was not identified in anyone in the ultrasonography examination (only 3 subjects had features of impingement in the form of pain during abduction). Hence, subacromial impingement

was more often identified using orthopedic tests than in ultrasonography examination. A possible explanation is that Neer's test is more challenging for the soft tissues (applying extrinsic pressing force after reaching the ending position in full elevation) than ultrasonography (active and passive abduction of the glenohumeral joint within 0–90°) when trying to identify subacromial impingement.

The accuracy of Neer's tests for the pathology of bursa, SSP, ISP or SSC and entheses of tendons or capsuloligamentous structures was similar for all (between 50.0% and 62.1%). This result may be explained by the fact that all of these tissues are compressed by the acromion and/or the glenoid rim in the ending position of the test and could be the source of the reported pain [46].

Clinical reviews report that the currently practiced shoulder clinical tests target either sensitivity or specificity, and those having both parameters at a high level are lacking [47, 48]. In our study, the sensitivity of Neer's and Jobe's tests was lower than the specificity, which shows an ability to identify healthy people rather than those with pathologies (both tests were negative in all subjects from the control group). Due to the low accuracy of the clinical tests for rotator cuff and subacromial impingement, these tests are insufficient to state the correct diagnosis. It is advisable to use ultrasonography for a more precise diagnosis.

Lewis [49] observed no correlation between the patients' complaints and the structural pathology identified with the imaging tests. The results of the current study confirm this observation to some extent. The prevalence of pathologies of the rotator cuff was higher in the study group than in the control group. On the other hand, the p-value for this relation (0.044) was close to the cut-off value for significance and the difference was significant only for the combined pathologies. The prevalence and also the incidence of SSP pathology, ISP pathology and SSC pathology (when analyzed separately) were not significantly different in tennis players with and without shoulder pain. No significant differences were observed in the prevalence of pathological shoulder changes, bursa pathologies and ultrasonographic features of subacromial impingement in tennis players with shoulder pain and those without pain. Another surprising result is the lack of correlation between the incidence rate of pathological shoulder changes versus the intensity of pain during

Krzysztof Guzowski, Artur Stolarczyk, Zbigniew Czyrny, Anna Dębek, Bernard Kranc

and after play, the time span of the pain experienced and also the results of Neer's and Jobe's tests. Furthermore, no significant differences were observed between the tennis players with significant shoulder pain (VAS ≥ 4) versus those without any significant pain (VAS ≤ 3), in the context of the prevalence of pathologies of the SSP, ISP or SSC.

In tennis players shoulder pain and changes of the ranges of rotation of the glenohumeral joint do not have to be caused by structural pathological changes. There was a considerable number of subjects in the group with shoulder pain without any pathological shoulder changes or features of subacromial impingement observed in ultrasonography. It is possible that among those tennis players the source of the pain was a functional problem, for example myofascial trigger points. This might be the missing link in the diagnostic process of pain complaints and decreased function. Active trigger points could imitate damage of the muscles which are overloaded and have increased stiffness, which in turn may explain the limitation of the range of motion [50–53].

Some limitations of the present study should be considered. Firstly the sample size is small, and thus it is difficult to draw a general conclusion. Secondly it is recommended to examine more structures in ultrasonography (for example the long head of the biceps and the acromioclavicular joint).

Conclusions

We examined ultrasonography as a tool in identifying painful shoulder (taking into consideration the presence, intensity and duration of the pain) and alterations of the range of rotation of the glenohumeral joint among tennis players.

We conclude that ultrasonography could be helpful in detecting tennis players with painful shoulder having SSP, ISP or SSC pathologies. However, it seems that the ability of the method to identify players having specific changes of the range of rotation of the glenohumeral joint is limited, with the exception of tennis players with ERD having SSP pathologies.

In ultrasonography it is possible to identify features of even small inflammation (effusion, synovial swelling) and degenerative changes of the shoulder (scars, erosions or calcific cavities), which might be asymptomatic. Accordingly, US diagnostics could be used to adjust training loads for a tennis player early, that is before serious structural pathological changes develop, such as full rotator cuff tear.

In order to confirm the present results it is necessary to conduct prospective and randomized studies with larger groups over a period of several years.

Acknowledgments

Conflict of interest

The authors declare no conflict of interest.

References

1. Krenn M. Epidemiology of injuries in tennis. Med Sci Tennis 1997; 2: 3.
2. Montalvan B, Parier J, Gires A, et al. Results of three years medical surveillance of the International Championships at Roland Garros. Med Sci Tennis 2004; 9: 2.
3. Ellenbecker TS, Roetert EP: Sport – specific training for tennis. In: The Scientific and Clinical Application of Elastic Resistance. Page P, Ellenbecker TS (eds.). Human Kinetics 2003; 211-20.
4. Kibler WB. Pathophysiology of tennis injuries – an overview. In: Tennis. Renström PA (ed.). Blackwell; 2002; 147-54.
5. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part I: pathoanatomy and biomechanics. Arthrosccopy 2003; 19: 404-20.
6. Kibler WB. Glenohumeral internal rotation deficit (GIRD) in tennis players. Med Sci Tennis 2006; 11: 13.
7. Vad VB, Altchek DW. Range of motion deficits in professional tennis players. Med Sci Tennis 2001; 6: 7.
8. Van der Hoeven H, Kibler WB. Shoulder injuries in tennis players. Br J Sports Med 2006; 40: 435-40.
9. Kibler WB, Kuhn JE, Wilk K, et al. The disabled throwing shoulder: spectrum of pathology – 10-year update. Arthroscopy 2013; 29: 141-61.
10. Wilk KE, Meister K, Andrews JR. Current concepts in the rehabilitation of the overhead throwing athlete. Am J Sports Med 2002; 30: 136-51.
11. Wilk KE, Macrina LC, Fleisig GS, et al. Correlation of glenohumeral internal rotation deficit and total rotational motion to shoulder injuries in professional baseball pitchers. Am J Sports Med 2011; 39: 329-35.
12. Wilk KE, Macrina LC, Fleisig GS. Correlation of shoulder range of motion and shoulder injuries in professional baseball pitchers: a 8 year prospective study. Presented at 2013 AOSSM Annual Conference; July 2013.
13. Vad VB, Gebeh A, Dines D, et al. Hip and shoulder internal rotation range of motion deficits in professional tennis players. Med Sci Tennis 2002; 7: 18.
14. Awan R, Smith J, Boon AJ. Measuring shoulder internal rotation range of motion: a comparison of 3 techniques. Arch Phys Med Rehabil 2002; 83: 1229-34.
15. Manske R, Wilk KE, Davies G, et al. Glenohumeral motion deficits: friend or foe? Int J Sports Phys Ther 2013; 8: 537-53.

16. Norkin CC, White DJ. Measurement of Joint Motion: A Guide to Goniometry. 3rd edn. F.A. Davis Company, Philadelphia 2003.

17. Wilk KE, Reinold MM, Macrina LC, et al. Glenohumeral internal rotation measurements differ depending on stabilization techniques. Sports Health 2009; 12: 131-6.

18. Jobe FW, Jobe CM. Painful athletic injuries of the shoulder. Clin Orthop 1983; 173: 117-24.

19. Jobe FW, Moynes DR. Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries. Am J Sports Med 1982; 10: 336-9.

20. Neer CS 2nd. Impingement lesions. Clin Orthop 1983; 173: 70-7.

21. Scheel A, Schmidt W, Hermann K, et al. Interobserver reliability of rheumatologists performing musculoskeletal ultrasonography: results from a EULAR "Train the trainers" course. Ann Rheum Dis 2005; 64: 1043-9.

22. Craig RT. Generalization of Scott's index of intercoder agreement. Public Opin Q 1981; 45: 260-4.

23. Czyrny Z. Anatomia diagnostyczna i diagnostyka patologii strefy entezy pierścienia rotatorów. J Ultrason 2012; 12: 178-87.

24. Pouliart N, Somers K, Eid S, et al. Variations in the superior capsuloligamentous complex and description of a new ligament. J Shoulder Elbow Surg 2007; 16: 821-36.

25. Pouliart N, Somers K, Gagey O. Arthroscopic glenohumeral folds and microscopic glenohumeral ligaments: the fasciculus obliquus is the missing link. J Shoulder Elbow Surg 2008; 17: 418-30.

26. Hashimoto T, Nobuhara K, Hamada T. Pathologic evidence of degeneration as a primary cause of rotator cuff tear. Clin Orthop Relat Res 2003; 415: 111-20.

27. Jobe CM. Posterior superior glenoid impingement: expanded spectrum. Arthroscopy 1995; 11: 530-7.

28. Morgan CD, Burkhart SS, Palmeri M, et al. Type II SLAP lesions: three subtypes and their relationship to superior instability and rotator cuff tears. Arthroscopy 1998; 14: 553-65.

29. Ogata S, Uhthoff HK. Acromial enthesopathy and rotator cuff tear. A radiologic and histologic postmortem investigation of the coracoacromial arch. Clin Orthop Relat Res 1990; 254: 39-48.

30. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am 1972; 54: 41-50.

31. Lewis JS. Rotator cuff tendinopathy. Br J Sports Med 2009; 43: 236-41.

32. Lewis JS. Rotator cuff tendinopathy: a model for the continuum of pathology and related management. Br J Sports Med 2010; 44: 918-23.

33. Davies SG, Baudouin CJ, King JB, et al. Ultrasound, computed tomography and magnetic resonance imaging in patellar tendinitis. Clin Radiol 1991; 43: 52-6.

34. Paoloni JA, Appleyard RC, Nelson J, et al. Topical nitric oxide application in the treatment of chronic extensor tendinosis at the elbow: a randomized, double-blinded, placebo-controlled clinical trial. Am J Sports Med 2003; 31: 915-20.

35. de Jesus JO, Parker L, Frangos AJ, et al. Accuracy of MRI, MR arthrography, and ultrasound in the diagnosis of rotator cuff tears: a meta-analysis. Am J Roentgenology 2009; 192: 1701-7.

36. Gaitini D, Militianu D, Nachtigal A, et al. Imaging of the shoulder. In: Musculoskeletal Ultrasound with MRI Correlations. Dogra V, Gaitini D (eds.). Thieme Medical Publishers, Stuttgart, New York 2010; 1-21.

37. Karelse A, De Beer J. Internal impingement – the pathological cascade. Med Sci Tennis 2007; 12: 28.

38. Noël E, Walch G. The postero-superior impingement of the glenoid and partial rotator cuff ruptures. Med Sci Tennis 1999; 4: 3.

39. Reilly P, Emery R. Shoulder pain in tennis players: the role of impingement and rotator cuff tears. Med Sci Tennis 2004; 9: 8-9.

40. Mihata T, Gates J, McGarry MH, et al. Effect of posterior shoulder tightness on internal impingement in a cadaveric model of throwing. Knee Surg Sports Traumatol Arthrosc 2015; 23: 548-54.

41. Clabbers KM, Kelly JD, Bader D, et al. Effect of posterior capsule tightness on glenohumeral translation in the late-cocking phase of pitching. J Sport Rehabil 2007; 16: 41-9.

42. Matsen FA III, Arntz CT. Subacromial impingement. In: The Shoulder. Rockwood CA Jr, Matsen FA III (eds.). WB Saunders, Philadelphia 1990; 623-46.

43. Tyler TF, Nicholas SJ, Roy T, et al. Quantification of posteriori capsular tightness and motion loss in patients with shoulder impingement. Am J Sports Med 2000; 28: 668-73.

44. Van der Hoeven H. Injury of the superior labrum in overhead athletes. Med Sci Tennis 2004; 9: 18-19.

45. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part III: the SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. Arthroscopy 2003; 19: 641-61.

46. Valadie AL 3rd, Jobe CM, Pink MM, et al. Anatomy of provocative tests for impingement syndrome of the shoulder. J Shoulder Elbow Surg 2000; 9: 36-46.

47. Dinnes J, Loveman E, McIntyre L, et al. The effectiveness of diagnostic tests for the assessment of shoulder pain due to soft tissue disorders: a systematic review. Health Technol Assess 2003; 7: iii, 1-166.

48. Longo UG, Berton A, Ahrens PM, et al. Clinical tests for the diagnosis of rotator cuff disease. Sports Med Arthrosc 2011; 19: 266-78.

49. Lewis JS. Rotator cuff tendinopathy/subacromial impingement syndrome: is it time for a new method of assessment? Br J Sports Med 2009; 43: 259-64.

50. Chen Q, Bensamoun S, Basford JR, et al. Identification and quantification of myofascial taut bands with magnetic resonance elastography. Arch Phys Med Rehabil 2007; 88: 1658-61.

51. Sikdar S, Shah JP, Gebreab T, et al. Novel applications of ultrasound technology to visualize and characterize myofascial trigger points and surrounding soft tissue. Arch Phys Med Rehabil 2009; 90: 1829-38.

52. Simons DG, Travell JG, Simons LS. Myofascial Pain and Dysfunction: the Trigger Point Manual. Vol. 1 – Upper Half of Body. 2nd edn. Lippincott Williams & Wilkins, Philadepphia 1999.

53. Macdonald AJ. Abnormally tender muscle regions and associated painful movements. Pain 1980; 8: 197-205.