2001

Amateur Volleyball Attackers Competing Despite Shoulder Pain: Analysis of Play Habits, Anthropometric Data, and Specific Pathologies

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Recommended Citation

Published in Physical Therapy in Sport, 2001; 2:112-122. doi: doi:10.1054/ptsp.2000.0045 http://www.sciencedirect.com/science/article/pii/S1466853X00900452

Original Research

Amateur volleyball attackers competing despite shoulder pain: analysis of play habits, anthropometric data, and specific pathologies

Ryan P. Jacobson and Cindy J. Benson

Objectives: Our purpose was to collect and analyze information on spiking and playing habits, anthropometric data and shoulder pathologies among competitive volleyball players with spiking-related shoulder pain, in order to gain new understanding of the unique task of spiking. Design: Each subject completed a four-page questionnaire and a standard musculoskeletal shoulder examination. Information from these was integrated into a database and analyzed. Significant findings and associations were reported. Setting/participants: Thirty amateur competitive volleyball attackers (21 males, nine females) were recruited from three tournaments. Results: Seven subjects reported that they could spike without pain only when spiking across their body. Wilcoxon signed-ranks tests revealed significant dominant side scapular lateralization ($P = 0.02$). Twenty-five subjects presented with anterior glenohumeral capsular laxity and/or posterior capsular tightness in the dominant shoulder. Moderation correlation ($r = 0.58$, $P < 0.001$) was found between female gender and the presentation of biceps involvement with anterior capsular laxity. Conclusions: A subset of attackers could only spike the ball across their body without pain. We hypothesized that this motion requires less glenohumeral internal rotation versus other ball placements, thereby decreasing impingement. Significant comorbidity of biceps involvement and anterior laxity found in female spikers may be attributed to kinematic differences versus males. Further investigation into these is warranted.

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Study approved by the University of Puget Sound Institutional Review Board

Introduction

Unlike most overhead sports, spiking a volleyball requires the athlete to strike and accelerate a ball using an overhead arm swing while the body is in mid-air without closed-chain support (Fig. 1). Controlled ball velocity and direction are necessary for success, and the attacker's dominant shoulder girdle becomes highly specialized for the task (Kugler et al. 1996). In addition to requiring task-specific strength and skilled timing, successful spiking involves coordination with teammates and, thus, last-second adaptability. An attacker must quickly adjust the direction and timing of his approach and arm swing to the location and loft of the set (Eom & Schutz 1992). Such demands on the shoulder complex predispose the attacker to painful shoulder pathology (Briner & Kacmar 1997; Kugler et al. 1996; Rokito et al. 1998). It is possible that predisposition to injury is modulated by technique – that is, an individual might have either an increased or decreased risk of developing shoulder pain depending on their spiking kinematics. While no investigation to date has demonstrated such a relationship, Coleman et al. (1993) found the kinematics of

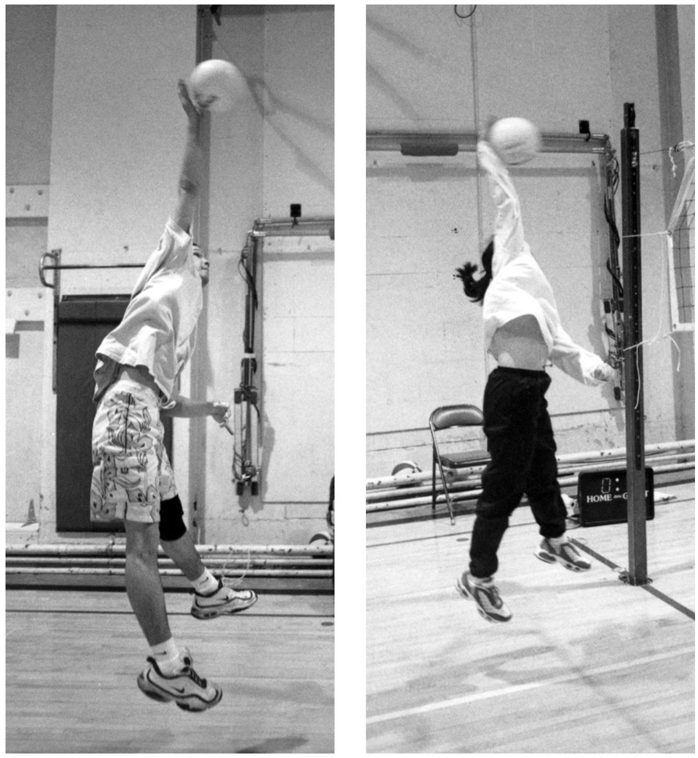

Fig. 1 Examples of two amateur competitive volleyball attackers, illustrating mid-air ball impact without closed-chain support.

the spiking motion to vary notably between 10 professional male attackers. This variability (demonstrated with high-speed cinematography) contrasts with the kinematics of overhead pitching which tend to be highly similar across individuals.

Despite a variety of spiking techniques among players, Kugler et al. (1996) found certain significant anatomical adaptations of the dominant side – specifically shoulder girdle depression and scapular lateralization – in 30 volleyball attackers, 15 with shoulder pain and 15 without. The 15 attackers in this study who were experiencing shoulder pain were found, based on diagnostic ultrasound, to have underlying pathology including bursitis, involvement of supraspinatus, biceps tendinitis, and instability. Many of these players reported the cause as 'unknown', implying insidious onset of symptoms. Repetitive spiking has been associated with rotator cuff tendinitis (Shafle 1993), although many competitive volleyball players never experience shoulder pain despite years of play. Schafle believes that sore shoulders usually result from 'technique error' (i.e. kinematics), though it is likely that an individual's musculoskeletal integrity, their anatomical adaptations, and the demands of their specific play environment also play an important part.

Compared to overhead throwers, relatively little has been published on shoulder kinematics or pathology specifically related to volleyball spiking. For example, a recent Medline search of the literature in English from 1975 to 2000 returned 123 articles with the keywords 'baseball' 'shoulder', but only 24 with 'volleyball' 'shoulder' (a majority of which do not specifically investigate shoulder pain and/or kinematics related to spiking). With so few investigations of shoulder pathology in volleyball attackers, many questions remain unanswered. For example, in light of the notable variation observed in spiking kinematics, is it possible that certain spiking techniques might be more likely to result in injury? How do anthropometric data measurements (e.g. shoulder girdle depression, scapular lateralization, etc.) and spiking habits correlate with shoulder pathology in this population? Are

Table 1 Means ± standard deviations for subjects' age, height, standing vertical jump height, and play frequency* ($n = 30$)

	Age (year)	Height (m)	Standing vertical (m)	Days per week	Hours per day
All subjects	27.0 (5.1)	1.82 (0.08)	0.53 (0.11)	2.4 (0.86)	3.5 (2.0)
Males ($n = 21$)	27.7 (5.5)	1.84 (0.07)	0.58 (0.09)	2.6 (0.93)	3.3 (1.8)
Females ($n = 9$)	25.4 (3.8)	1.77 (0.07)	0.41 (0.06)	2.1 (0.60)	3.9 (2.6)

*Based on combined practice and game time per self-report.

findings in this group similar to the typical shoulder findings reported in overhead pitching studies? Answers to these questions may help guide the clinician in determining attacker rehabilitation needs, especially in terms of player technique modification.

The purpose of this study was to collect and analyze information on playing and spiking habits, anthropometric data, and shoulder pathologies in amateur volleyball players who were competing despite concurrent symptomatology associated with spiking. It was anticipated that such analysis would create new understanding of the unique task of spiking a volleyball, thus leading to viable hypotheses to guide future, more focused investigation.

Methods

Subjects were recruited on a volunteer basis during three separate amateur volleyball tournaments at Northwest Volleyball Center in Tukwila, WA, part of the USA Volleyball™ Puget Sound Region, in December 1998 and January 1999. A total of 40 different teams from Washington, Oregon, and British Columbia competed in these tournaments. Inclusion criteria were: (1) 18–40 years of age, (2) currently participating in competitive volleyball, and (3) having painful symptomotology of the shoulder region which is associated with spiking a volleyball. Exclusion criteria were: (1) having arm, shoulder, neck, back or trunk problems as a result of non-volleyball-related causes, and (2) having any medical problems that would be exacerbated by a musculoskeletal assessment of the upper trunk, neck, and shoulders. Thirty subjects, 21 males and nine females, met all criteria and completed the study. Informed consent was obtained and all subject rights were protected.

Table 1 gives means and standard deviations for age, height, standing vertical jump height, and frequency of play for the 30 subjects. On the player questionnaire, two subjects reported spending only 0–10% of their total playing time spiking, 14 reported 11–25%, 13 reported 26–40%, and one reported >40%. Based on self-assessment, eight subjects reported that they were competitive attackers in level AA competition (the highest division of play), 16 reported level A (next highest), and six reported level BB (third highest).

The primary author developed and pilot tested a four-page player questionnaire surveying player habits related to spiking and subjective information related to shoulder pain. All participants completed this questionnaire.

Subjects were then given a 15–20 min standard musculoskeletal shoulder examination, a clearing screen of the cervical spine and, if periscapular symptoms, a clearing screen of the corresponding thoracic region (Fig. 2). The second author, who has completed the Kaiser Physical Therapy Residency Program in Advanced Orthopedic Manual Therapy (Hayward, California) and has 20 years of clinical experience working with orthopaedic populations, performed all examinations. The evaluating physical therapist knew that all subjects had shoulder pain but was blind to the results of the player questionnaire. Prior to the objective examination, the evaluator did ask each subject to describe the area of symptoms and to describe the specific complaints in order to determine indications of severity, irritability, nature or stage that would limit the vigour of the objective examination.

The following tests were routinely performed and assessed according to published guidelines: Empty Can (Jobe & Moynes 1982), Speed's (Magee 1997), Crossed-Arm Adduction (Boublik & Hawkins 1993), Hawkins–Kennedy

Objective Shoulder Exam for Volleyball Attackers

Involved side R_____ L_____ Bilat ____
Dominant side R_____ L_____
Description of Symptoms

Empty Can Weakness___________ Pain/Location_________________________

Speeds Pain/Location ____

Cross-over sup shld______ ant shld ______ post shld ______

Hawkins-Kennedy / Neer _______________ / _________________

O'Brien (PRN) _____________________________

Load and Shift______________________________

		R	L
Functional ROM (Scratch test)			
	HBB ER	______	______
	HBB IR	______	______
Mod Lateral Scap Slide	**0° ABD**	______	______
	90° ABD/IR	______	______
	150° ABD	______	______

Apprehension (supine)________________________
Ant drawer / Relocation_____________________ /__________________________
Post drawer ________________________________
Clunk_____________________________________

Cervical Central & Unilat PAs _______________________ / Thoracic PRN______
Ipsilat Mid/Low Cervical Quadrant ____________________

Palpation: AC ______ SC_______ Coracoid _______ Biceps tendon _______
Gr. Tuberosity ____ Subdeltoid bursa ____ Post capsule/SIT _____
Other under area of Sx ____________________________________

Fig. 2 Objective examination used on all subjects.

Impingement (Hawkins & Kennedy 1980), Neer Impingement (Neer & Welsh 1977), O'Brien (Konin et al. 1997), Load and Shift (Hawkins & Mohtadi 1991), Apprehension and Relocation (Jobe & Bradley 1989), Anterior Drawer and Posterior Drawer (Gerber & Ganz 1984), and Clunk (Andrews & Gillogly 1985). The cervical screen was designed to reproduce symptoms referred to the shoulder and consisted of the Lower Cervical Quadrant test for foraminal provocation and central and medially-directed unilateral posterior-to-anterior pressures at a Grade IV+ from C4 through T1 as described by Maitland (Maitland 1986). We modified the

Lateral Scapular Slide test (Davies & Dickoff-Hoffman 1993; Kibler 1991) such that the horizontal distance from the inferior angle of the scapula to the corresponding thoracic spinous process was measured bilaterally in three positions: arms resting at sides, 90° of glenohumeral (GH) abduction with internal rotation, and 150° of GH abduction. Bilateral Apley's Scratch test (Konin et al. 1997) for functional combined GH external rotation and scapular motion was recorded as the distance from the spinous process of C7 to the tip of the subject's thumb maximally sliding down the vertebral column. Similarly, combined GH internal rotation with scapular motion was measured as the distance from the spinous process of L5 to the tip of the subject's thumb maximally sliding up the vertebral column.

Mirroring typical practice in the clinic setting, the evaluator was allowed to perform other related tests if results were positive for any of the protocol tests. After all data was collected, the authors pooled information from each questionnaire and corresponding examination to identify the most likely site or sites of involvement. In all cases where subjects had been given a previous diagnosis based upon physician evaluation with imaging studies, the current examination findings were still suggestive of the same problem, so the prior medical diagnosis was used.

Data from both the player questionnaire and the musculoskeletal examination were integrated, coded, and entered into a spreadsheet on SPSS 7.5 (Statistical Product & Service Solutions Inc., Chicago, IL) from which all means and standard deviations were calculated. This large pool of data was then analyzed for differences (i.e. for dominant versus non-dominant measures) and for correlations. Notable, statistically significant results are reported herein along with pertinent raw data, and conclusions are based on these results. Specific statistical tests used were as follows. Mean values for dominant versus non-dominant side anthropometric data were compared on SPSS 7.5 using the Wilcoxon signed-ranks test. Correlation coefficients and significance levels for the relationship between gender and biceps involvement, gender and biceps involvement with anterior capsular laxity, and posterior capsular tightness and impingement were also calculated on SPSS 7.5 using the phi coefficient. All remaining correlation coefficients and significance levels were calculated using either the point biserial or rank biserial coefficients, per the appropriate equations (Portney & Watkins 1993). The significance level for all statistical tests was set at $P < 0.05$ prior to data collection.

Results

Pain

As shown in Table 2, the most common pathological findings were suggestive of instability, impingement, and biceps involvement with a pattern of comorbidity. Reported pain intensities using a 0–100 visual analog scale for 'the most pain experienced due to spiking' ranged from 16–74 ($|x| = 45.9$, SD = 18.2). Eighteen subjects reported having to take time off from playing volleyball for periods ranging from 7–365 days ($|x| = 96$, SD = 98) due to shoulder pain. Also, 23 subjects reported that their pain limits how hard they can spike. The time since the onset of symptoms ranged from 2–180 months ($|x| = 40$, SD = 39). Only five subjects reported that their pain resulted from a specific event. In all of these cases, the subject had dived for a ball and, at the time of the examination, demonstrated clinical findings

Table 2 Pathologies and specific sites found in subjects with number of subjects in parentheses (males, females) as determined on examination by an orthopedic physical therapist*

Category	Specific site
Instability	Anteriorly (6, 6) Inferiorly (2, 0)θ Posteriorly (1, 0)
Impingement	Biceps (2, 5) Posterior cuff (5, 2) Superior cuff (4, 1)
Biceps involvement	Biceps tendon (6, 6)
Other	AC joint involvement (3, 1) Labrum (2, 1)ϕ Posterior cuff mm strain (2, 0) C5–C6 referral (1, 0) T5–T6 facet (0, 1)

*Evaluator was unable to determine site in one subject. θ, positive Sulcus sign; ϕ, one with positive O'Brien test and prior Dx of SLAP. Two with positive Clunk test and complaints to match.

Refer to the volleyball court diagram below to answer #17, #18, & #19.

17) When spiking from the left front (**L**) position, is there any area on the court that you can hit to *WITHOUT* feeling pain*?* (Circle all that apply.)

1 **2** **3** I have felt pain hitting to **all** areas.

18) When spiking from the middle front (**M**) position, is there any area on the court that you can hit to *WITHOUT* feeling pain*?* (Circle all that apply.)

1 **2** **3** I have felt pain hitting to **all** areas.

19) When spiking from the right front (**R**) position, is there any area on the court that you can hit to *WITHOUT* feeling pain*?* (Circle all that apply.)

1 **2** **3** I have felt pain hitting to **all** areas.

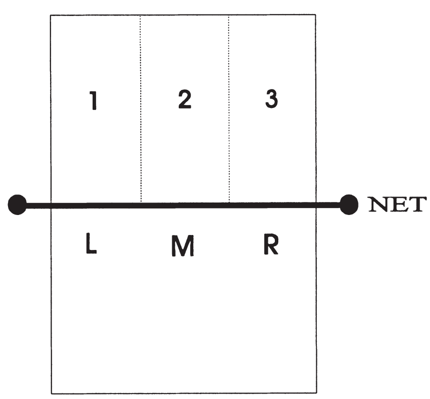

Fig. 3 Section from the player questionnaire used to determine under what conditions, if any, subjects could spike a volleyball without feeling shoulder pain.

indicative of either acromioclavicular joint involvement or a superior labral tear. Table 3 lists the personal strategies and/or professional treatment subjects utilized for pain control.

Three questions in the player questionnaire concerned areas on the court which attackers could target when spiking without feeling shoulder pain (Fig. 3). Seven of the 30 subjects reported that they were able to spike without pain *only* when spiking the ball to an area on the court such that their arm swing was directed across their body. An example would be a right-handed attacker spiking the volleyball without pain from the right front position to area 1 (Fig. 3, question 19). Eighteen subjects reported feeling pain when hitting to all areas. One subject reported feeling no pain *only* when spiking straight ahead, and the other four subjects showed no specific patterns.

Anthropometric data

Table 4 compares the mean Lateral Scapular Slide Test values for dominant and non-dominant sides both with the subjects' arms resting at their sides and at 150° abduction. Table 4 also compares the mean Apley's Scratch

Table 3 Personal strategies used and professional treatment sought for shoulder pain (number of subjects utilizing each resource in parentheses

Personal strategy	Professional treatment sought
NSAID (15)	Physical therapy (8)
Ice (9)	Massage therapy (2)
Massage (3)	Athletic training (2)
Stretching (3)	Physician services (2)
Heating pack (1)	Cortisone shot (1)
Sport cream (1)	
Muscle relaxants (1)	

NSAID = non-steroidal anti-inflammatory drug.

Test results within subjects for the dominant versus non-dominant side.

Based on the Load and Shift Test of the dominant shoulder, 15 of the 30 subjects demonstrated posterior capsular tightness combined with at least Grade I anterior capsular laxity (i.e. forward translation of the humeral head greater than 25% of its diameter). Furthermore, eight subjects demonstrated only posterior tightness, three demonstrated only Grade I anterior laxity, three were normal, and one could actively sublux his GH joint posteriorly and inferiorly.

Associations

Looking at specific dominant shoulder pathologies, no correlation (r = 0.12; phi coefficient) was found between the presence of posterior capsular tightness and impingement. Likewise, no correlation (r = −0.13; rank biserial coefficient) was found between the degree of anterior laxity and the presence of biceps involvement.

In terms of gender, six out of nine females (67%) had biceps involvement, and five of these six also demonstrated at least Grade I anterior laxity. Conversely, six out of 21 males (29%) had biceps involvement, and only one of these six also demonstrated anterior laxity. Fair correlation (r = 0.36, $P = 0.03$; phi coefficient) was found between female gender and the presence of biceps involvement. Moderate correlation (r = 0.58; $P < 0.001$; phi coefficient) was found between female gender and the presence of biceps involvement with comorbid anterior laxity. No other correlation between gender and shoulder pathology was found.

Discussion

We studied a population of amateur volleyball attackers who competed in tournaments despite having shoulder pain related to spiking. We were surprised by the significant signs and symptoms which players were willing to 'put up with'. Shoulder pathology in attackers is typically insidious in onset, resulting from many years of repetitive overuse (Briner & Kacmar 1997; Schafle 1993). Shoulder injury incidence rates recorded in treatment centres are relatively low, when compared with traumatic injuries of the fingers and ankles (Solgard et al. 1995; Watkins & Green 1992). Perhaps many attackers do not seek treatment for their shoulders. When asked about medical care, one subject responded 'Are you kidding? I don't have insurance. I'm a farmer.' In fact, 25 out of 30 of our subjects reported shoulder pain of insidious onset, and 18 had never sought any form of professional treatment despite an average 40 months since onset of symptoms. Therefore, it was appropriate to

Table 4 Means ± standard deviations for various anthropometric data within subjects, dominant versus non-dominant side ($n = 30$)

Measure	Dominant (cm)	Non-dominant (cm)	d-nd (cm)	P
Lat Scap Slide 0°	13.0 (1.9)	12.4 (1.8)	0.6	0.02*
Lat Scap Slide 150°	16.7 (1.9)	16.2 (1.8)	0.5	0.05
Apley with ER	5.9 (4.0)	7.4 (3.1)	−1.5	0.02*
Apley with IR	30.8 (6.6)	37.9 (6.0)	−7.1	<0.001*

*Difference is statistically significant for $P < 0.05$ (Wilcoxon signed-ranks test[13]). d-nd = difference between means (dominant-non-dominant)). Lat Scap Slide 0° = Lateral Scapular Slide Test 0° with arms resting at sides. Lat Scap Slide 150° = Lateral Scapular Slide Test with arms abducted to 150°. Apley ER = Apley's Scratch test with external rotation component. Apley IR = Apley's Scratch test with internal rotation component.

study subjects with shoulder pain at competitive volleyball tournaments in order to obtain a more broad range of attacker complaints than is reflected by data from medical treatment facilities.

Pain

As described in the results above, seven subjects reported that they were able to spike without pain *only* when it involved swinging their arm across their body. However, no subjects reported that they could spike the ball without pain to an area on the court that was ipsilateral to their spiking arm, such as when a right-handed attacker spikes the volleyball from the left front position to area 3 (Fig. 3, question 17).

Why is it that spiking a volleyball across the body did not exacerbate some attackers' symptoms as easily as spiking to the side ipsilateral to the spiking arm? In order for an attacker to direct the volleyball to the side ipsilateral to the spiking arm, he or she must pronote the forearm and internally rotate the GH joint upon contact with the ball. Internal rotation brings the greater tubercle of the humerus under the coracoacromial arch and can exacerbate impingement pain upon arm elevation. Since an attacker abducts the GH joint up to 150° when spiking (Coleman et al. 1993), it seems likely that spiking to the ipsilateral side would result in impingement. On the other hand, spiking the volleyball to the side contralateral to the spiking arm, such that the arm swing is directed across the body, does not involve such marked GH internal rotation. Subacromial impingement would thus be less likely to occur at ball contact. In addition, the continued horizontal adduction of the GH joint as the arm is decelerated across the body might provide extra anterior support to help limit anterior translation of the humeral head in the glenoid fossa. Such translation has been described related to arm deceleration in baseball pitchers with shoulder pathology (Meister & Andrews 1993). Since anterior laxity of the shoulder is related to injury of the glenoid labrum and can lead to impingement of anterior structures such as the tendon of the long head of the biceps (Kamkar et al. 1993; Meister & Andrews 1993), extra support to limit anterior translation might serve to minimize symptoms during spiking. Further investigation into what role arm swing kinematics have in the irritation of shoulder symptomatology could lead to the development of guidelines for safely returning to full-time spiking following injury.

Anthropometric data

Regarding scapular stabilization, Kibler (1991) describes the original Lateral Scapular Slide Test measurements with the glenohumeral joint abducted in three positions: 0°, 45° and 90° with internal rotation (Kibler 1991). Subsequently, Davies & Dickoff-Hoffman in 1993 described the Modified Lateral Scapular Slide using two additional positions of abduction: 120° and 150°. These were added based on the observation that often patients complain of pain when in an overhead position. However, no statistically significant change in scapular position from one arm position to the next was demonstrated. We therefore chose 0° GH abduction as an indicator of resting scapular position, and added 90° with internal rotation and 150° as positions related to spiking.

Our subject population demonstrated statistically significant mean lateralization of the dominant scapula ($|x| = 0.6$ cm) relative to the position of the non-dominant scapula. An average of 0.9 cm lateralization of the dominant scapula versus the non-dominant side for volleyball attackers has been previously reported (Kugler et al.1996). Similar findings have been described among baseball pitchers and indicate a functional scapular instability related to throwing (Kamkar et al. 1993; Pappas et al. 1985). In both throwing and spiking, scapular stability is a prerequisite to proper scapulohumeral rhythm as well as to the strength and timing of the motion itself. A protracted scapular resting position may lengthen the rhomboid and middle trapezius muscles into a range of insufficiency. The resulting weakness in eccentric scapular stabilization during GH elevation could contribute to subsequent secondary impingement (Kamkar et al. 1993).

In addition to scapular lateralization, subjects in our study demonstrated a statistically significant limitation on the dominant side for both Apley's Scratch tests. A previous study using Apley's Scratch tests demonstrated a significant limitation of dominant side internal rotation with scapular motion versus non-dominant in attackers with painful shoulders, but not in combined scapular motion and external rotation (Kugler et al. 1996). It has been reported that attackers with painful shoulders typically have not only a limited arc of pure internal rotation ROM, but also an excessive arc of pure external rotation ROM (Briner & Kacmar 1997). Because Apley Scratch tests involve not only pure glenohumeral rotation but also motion at the sternoclavicular, acromioclavicular and scapulothoracic joints, we cannot draw conclusions about isolated glenohumeral motion in our subject population.

Another commonality in our sample was the finding of posterior capsular tightness and/or anterior capsular laxity. Only four of the 30 subjects did *not* demonstrate at least one of these findings during the Load and Shift Test. Posterior tightness has been previously described in the shoulders of attackers, regardless of pain (Kugler et al. 1996). However, dominant side anterior laxity, as seen in 18 subjects here, has not been reported in the population of volleyball attackers. Anterior laxity of the dominant GH joint capsule is common in overhead pitchers (Ticker et al. 1995; Wilk & Arrigo 1993), as is posterior tightness (Kamkar et al. 1993; Pappas et al. 1985; Wilk & Arrigo 1993).

Associations

Secondary impingement has been described as a direct sequel of both posterior tightness and anterior laxity (Kamkar et al. 1993; Meister & Andrews 1993; Ticker et al. 1995; Wilk & Arrigo 1993). Typically, a tight posterior GH capsule can cause abnormal translation of the humeral head superiorly and/or anteriorly and result in impingement of structures under the coracoacromial arch during elevation. In the present sample, however, no correlation was found between posterior tightness and a positive impingement sign. In this regard, it is possible that our sample of only 30 is not representative of the average population of amateur volleyball attackers. However, it seems worth noting that over half of the subjects with posterior tightness (13 out of 23) were negative for shoulder impingement signs.

Our study demonstrated moderate correlation ($r = 0.58$, $P < 0.001$; phi coefficient) between female gender and the presence of anterior laxity with concurrent biceps involvement. The association of anterior capsular laxity and anterior impingement has been described in baseball pitchers and is evident during either arm cocking or deceleration when dynamic muscular stabilization is not adequate (Meister & Andrews 1993; Ticker et al. 1995). This can cause wear and tear on anterior structures, including the long head of the biceps tendon, resulting in anterior impingement or tendinitis (Meister & Andrews 1993). However, only females in our study demonstrated this association. Perhaps there are specific kinematic aspects of repetitive overhead spiking in amateur female attackers that might selectively predispose them to such a pattern of injury. Anecdotally, the primary author has played competitive volleyball for eight years and has noted that amateur male attackers often utilize different spiking mechanics than females. Males tend to jump higher, generate more ball velocity, and spike the ball in a more downward direction into the opposing team's court. Future research exploring associations between gender, shoulder pathology and spiking mechanics in a larger sample size is needed.

Limitations

Several limitations of this study have already been discussed: the small sample size, the inability to draw conclusions about isolated glenohumeral rotation when a combined movement measure (i.e. Apley's Scratch test) is used, and the accuracy of determining the specific site of shoulder pathology based upon clinical musculoskeletal assessment alone. A final limitation concerns the applicability of findings to all volleyball attackers. Our subjects were participating in competitive volleyball despite shoulder symptoms. While 77% of these subjects reported that their pain limited how hard they could spike and 67% had at some point lost playing time, the results might not apply to attackers who are currently sidelined with more acute symptoms.

Conclusions

Within the limitations of this investigation, the following main conclusions were made:

1. A readily identifiable population of amateur volleyball attackers exists which is competing despite related shoulder signs and symptoms and might not be reflected in epidemiological data from medical treatment facilities.
2. A notable subset of attackers could spike across the body without pain. We hypothesized that this was due to decreased need for GH internal rotation upon contact with the ball, thereby avoiding impingement.
3. Significant comorbidity of anterior laxity and biceps involvement was found in females but not in males. We hypothesized that females typically demonstrate different spiking techniques and lower jump heights than males, suggesting an association between kinematics and shoulder pathology.
4. The kinematics of spiking a volleyball are unique when compared with other overhead sports because the athlete must accelerate a ball *while in mid-air without closed-chain support*. A better understanding of spiking kinematics and of technique differences between males and females, as well as the relationships between anatomy, play habits, kinematics, and pathologies could guide the clinician in attacker-specific shoulder rehabilitation.

Acknowledgement

Special thanks to Kathie Hummel-Berry, PT, MEd, PhD Epidemiology, for her critical input on development and pilot testing of the player questionnaire, as well as on the study design.

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