The effects of ball impact position on shoulder muscle activation during spiking in male volleyball players

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Background: The ball impact position during spiking in volleyball may influence the pattern of activation of shoulder girdle muscles and, therefore, could be a significant risk factor for shoulder injury.

Methods: Activation of 10 muscles in the dominant shoulder was evaluated using surface electromyography (EMG) in 11 male volleyball players, during spiking in a static standing position, with the goal of precisely control the specified ball impact positions, without a run-up or ball setting. The following 4 ball impact positions were evaluated: standard, posterior, medial, and lateral. The EMG amplitude, normalized to the maximal voluntary isometric contraction of the respective muscles, was compared for each phase of the spiking movement between the standard position and the other 3 different impact positions, using the Dunnett test.

Results: The following between-position differences were noted for the deltoid muscle: increased activation of the anterior deltoid during the acceleration phase for the posterior position (P = .041), increase in the posterior deltoid during the acceleration phase for the lateral position (P = .04), and increase in the middle deltoid during the deceleration phase for the posterior position (P = .005).

Conclusion: A posterior or lateral shift in the position of ball impact may cause an increase in the activity of the deltoid muscle that would cause a decrease in the centripetal force of the humeral head through the acceleration and deceleration phases. As such, neuromuscular exercises, combined with strengthening of the rotator cuff muscle, might reduce the risk of shoulder injury during performance of the volleyball spiking movement.

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The primary goal of spiking in volleyball is to hit the ball as hard as possible so as to convey maximum velocity to the ball, with the angular velocity of internal rotation at the shoulder reaching values as high as 4000°-7000°/s.3,10,11 As such, volleyball players experience the same level of stress on their shoulder as athletes performing overhead pitching in baseball, passing in football, or serving in tennis. Therefore, the shoulder joint in volleyball players is vulnerable to acute or overuse injuries due to repeated spiking during training and game performance.22 According to a 16-year surveillance of injury by the National Collegiate Athletic Association Injury Surveillance System (NCAAISS), shoulder injuries are among the leading causes of time lost from training and competition among female volleyball players,1 with injuries to the shoulder accounting for 8%-25% of all volleyball-related injuries.4,26

Most volleyball-related shoulder pathologies (impingement, rotator cuff tendinopathy, atraumatic glenohumeral instability, suprascapular neuropathy) result from chronic (overuse) rather than acute overload.23,27 Inappropriate ball impact position during spiking can further increase the risk of specific injuries (such as a rotator cuff injury or superior labrum tear from anterior to posterior [SLAP] lesion) caused by internal impingement due to excessive external rotation of the shoulder.13 Moreover, different ball impact positions could change the recruitment pattern of glenohumeral and scapular muscles, further predisposing players to shoulder injury.

In our review of the literature regarding shoulder injuries in volleyball players, no research was identified regarding the effect of ball impact position on shoulder muscle activity during spiking. We did identify a single study that quantified the firing patterns of the glenohumeral muscles between the volleyball serve and spiking movements.22 However, there was no research regarding the recruitment patterns of scapular muscle during volleyball spiking.
Despite scapular muscles being important to maintain proper position of the scapula relative to the humerus and the fact that shoulder pain in volleyball players often results from an imbalance of the scapular muscles,\textsuperscript{5,6} Thus, the aim of our study was to elucidate the effect of ball impact position during spiking on the activity patterns of glenohumeral and scapular muscles in male volleyball players. Understanding muscle recruitment patterns of the shoulder complex during spiking would inform the development of muscle-specific treatment and training protocols, which could lower the risk of injury, as well as enhance performance. We hypothesized, a priori, that muscle recruitment pattern of the shoulder girdle would be modified by the ball impact position during spiking.

Materials and methods

Participants

Eleven male collegiate volleyball players (mean age, 22.1 ± 2.1 years; mean height, 173.5 ± 9.7 cm; mean weight, 67.3 ± 11.1 kg), with an average volleyball experience of 6.4 ± 3.3 years and each having >3 years of experience, were enrolled into the study. Among these 11 players, 7 were wing attackers, 2 middle blockers, and 2 setters. We noted that the 2 setters had more than 1 year of prior experience as wing attackers. All players were right arm dominant, and none had a history of shoulder injury resulting in activity restriction.

All players were provided with written documentation about the study and provided their consent to participate. The study was approved by the ethical review board of the Hirosaki University, School of Medicine (EK No. 2016-266), and was performed in agreement with the Declaration of Helsinki.

Electromyography recording

Muscle activity was recorded using surface electromyography (EMG) from the following 10 shoulder and scapular muscles of the dominant (hitting) arm: upper trapezius (UT), middle trapezius (MT), lower trapezius (LT), anterior deltoid (AD), middle deltoid (MD), posterior deltoid (PD), serratus anterior (SA), infraspinatus (ISP), pectoralis major (PM), and teres major (TM). The compact electrode telemeter system was used, consisting of active, reference, and ground electrodes, integrated into a wireless transmitter (ZB-150H; Nihon Kohden, Tokyo, Japan), which transmitted the signals to the host computer for real-time display and storage (WEM-1000, Nihon Kohden, Tokyo, Japan; Fig. 1). The distance between the active and reference electrodes was 10 mm, and the ground electrode was placed at the midpoint between these 2 electrodes. After standard skin preparation using rubbing alcohol, the electrodes were placed on the skin over the center of each muscle, as previously described,\textsuperscript{5,10,25} and secured using doublesided tape.

Surface EMG signals were sampled at 1000 Hz, band-pass filtered (5-Hz low-frequency cutoff and 500-Hz high-frequency cutoff), with the processed signals saved to a computer for offline analysis.\textsuperscript{19,20} A camera was used for motion capture in the lateral view (sampling frequency, 120 frames/s). Motion capture and EMG data were time synchronized, using an electronic pulse, over each phase of the movement (wind-up, cocking, acceleration, deceleration, and follow-through) for analysis.

Testing procedure

Before recording of the spiking movement, resting EMG data were recorded to establish a baseline level of activity for each muscle studied. Subsequently, a 5-second maximum isometric contraction was elicited, using a manual muscle test, for each muscle\textsuperscript{1} to be used as the normalizing values for between-subject comparisons. Participants performed the spiking movement in a static standing position, using a training ball attached to the wall via an elastic cord (MVA400ATTR; Mikasa, Hiroshima, Japan; Fig. 2). The static standing position was selected to ensure precise and consistent positions of ball impact across participants, recognizing that this standardization is not authentic in real-life situations where spiking is incorporated in a jumping motion and the ball is set to the required height by a setter. Spiking was performed for the following 4 different ball impact positions: standard, posterior, medial, and lateral (Fig. 2). Three of these positions (standard, medial, and lateral) simulated the conditions for a left wing spiker performing spikes along the following 3 paths: cross court, straight, and inner court, respectively (Fig. 3). In the standard position, the ball was located 50 cm anterior to the anterior wall of the trunk, and the line between the center of the glenohumeral joint and the hand of the dominant arm was perpendicular to the horizontal line connecting the centers of each glenohumeral joint (Fig. 2, A). In the posterior position, the ball was located 20 cm posterior to the standard position (Fig. 2, B). In the medial and lateral positions, the ball was located 20 cm medial and 20 cm lateral to the standard position, respectively (Fig. 2, C and D). Participants were instructed to perform 3 spikes at each of the 4 ball impact positions, with the order of presentation randomized to avoid systematic effects of fatigue and learning. A 1-minute rest period was provided between trials.
Data reduction

The spike movement was subdivided into 5 phases for analysis (wind-up, cocking, acceleration, deceleration, and follow-through) as described in previous studies for the volleyball spike,22 baseball pitch,1 and tennis serve (Fig. 4).15 The wind-up phase is a preparatory phase that begins with shoulder abduction and extension and ends with initiating external shoulder rotation (Fig. 4, A). The cocking phase begins with the initiation of external shoulder rotation and ends with maximal external shoulder rotation (Fig. 4, B). The acceleration phase begins with forceful internal shoulder rotation and ends with ball impact (Fig. 4, C). The deceleration phase begins with ball impact and ends with the upper arm perpendicular to the trunk (Fig. 4, D). The follow-through phase begins when the upper arm is perpendicular to the trunk and ends when all arm motion is complete (Fig. 4, E). The band-pass filtered EMG signals were rectified and smoothed, using a root mean square algorithm. The average activity in a 50-millisecond (ms) window in each phase of the spiking movement was used to compare effects of the 4 different ball positions on muscle activation levels. These average muscle activation levels were normalized to the manual maximal voluntary isometric contraction (MVIC) value obtained at baseline for each muscle. This baseline MVIC value was calculated as the average activation level over a 500-ms window for the highest 5-second manual muscle test for each muscle. The %MVIC value used to compare muscle activity between positions was calculated as the average of 3 trials, in each phase, for each of the 4 ball positions.

Statistical analysis

The %MVIC of the target muscles and the duration of each phase were compared between the standard position and the other 3 ball impact positions using the Dunnett test for multiple comparisons. Statistical analyses were performed using SPSS (version 23 for Macintosh: IBM, Armonk, NY, USA). The level of significance was set at $P < .05$.

Results

Duration of each phase

The mean duration of the volleyball spike movement for the standard position was 1.67 seconds, with the mean for each of the 5 phases as follows (Table I): wind-up, 0.84 ± 0.21 seconds (51% of total spike time); cocking, 0.31 ± 0.13 seconds (18% of total spike time), acceleration, 0.12 ± 0.04 seconds (7% of total spike time); deceleration, 0.09 ± 0.03 seconds (6% of total spike time); and follow-through, 0.30 ± 0.11 seconds (18% of total spike time). A significant difference was identified between the standard and the posterior position only for the deceleration phase, this phase being significantly longer for the posterior (0.15 ± 0.07 s) than for the standard (0.09 ± 0.03 s; $P = .016$) position.

Muscle activation

The %MVIC activation level for the 10 shoulder muscles for the 4 different ball impact positions is reported in Tables II and III. Salient features of the muscle activation profile were as follows. During the wind-up phase, activation of the UT, SA, and AD was >40% MVIC, with the activation of other muscles being <25% MVIC. Activation of the SA and AD was significantly lower for the lateral than for the standard position (SA: 16% ± 9% MVIC and 48% ± 51% MVIC, respectively, $P = .045$; and AD: 25% ± 14% MVIC and 53% ± 48% MVIC, respectively, $P = .031$). During the cocking phase, the LT (52%
± 30% MVIC), SA (58% ± 47% MVIC), and AD (64% ± 43% MVIC) were highly active for the standard position, with all other muscle activation levels being <40% MVIC, with the UT activation being slightly higher at 47% ± 36% MVIC.

Activation of the TM was significantly lower for the medial (8% ± 7% MVIC) than for the standard (29% ± 41% MVIC; \( P = .05 \)) position.

During the acceleration phase, all activation levels increased to >35% MVIC, with the highest levels in the MD (44% ± 40% MVIC), PD (50% ± 36% MVIC), PM (68% ± 33% MVIC), TM (76% ± 55% MVIC), UT (56% ± 49% MVIC), and MT (62% ± 42% MVIC) muscles. A significant difference in muscle activation levels between the standard and posterior position was identified during this phase, with higher activation of the AD for the posterior than for the standard position (74% ± 72% MVIC and 43% ± 33% MVIC, respectively; \( P = .041 \)) and lower activation for the PM (44% ± 29% MVIC and 68% ± 33% MVIC, respectively; \( P = .007 \)). Furthermore, the muscle activation level of the PD was significantly higher for the lateral than for the standard position (80% ± 41% MVIC and 50% ± 36% MVIC, respectively; \( P = .004 \)).

During the deceleration phase, muscle activation was generally above 30% MVIC, with the highest activation in the ISP muscle (62% ± 34% MVIC) in the standard position. Activation of the ISP was significantly lower for the posterior (44% ± 18% MVIC) than for the standard (62% ± 34% MVIC; \( P = .043 \)) position. In addition, activation of the MD was significantly higher for the lateral (66% ± 31% MVIC) than for the standard (37% ± 24% MVIC; \( P = .005 \)) position.

### Table 1

Duration(s) of each phase during the spiking movement for the 4 different ball impact positions

| Position  | Phase | Total time |
|-----------|-------|------------|
|           | Wind-up | Cocking | Acceleration | Deceleration | Follow-through |
| Standard  | 0.84 ± 0.21 | 0.31 ± 0.13 | 0.12 ± 0.04 | 0.09 ± 0.03\* | 0.30 ± 0.11 | 1.67 ± 0.33 |
| Posterior | 0.84 ± 0.25 | 0.26 ± 0.08 | 0.10 ± 0.03 | 0.15 ± 0.07\* | 0.38 ± 0.14 | 1.73 ± 0.26 |
| Lateral   | 0.79 ± 0.24 | 0.26 ± 0.10 | 0.11 ± 0.04 | 0.11 ± 0.03 | 0.26 ± 0.05 | 1.54 ± 0.29 |

\* Significant difference between the standard position and the posterior position (\( P < .05 \)).

Figure 4 The spike is divided into the following 5 phases: (A) wind-up, (B) cocking, (C) acceleration, (D) deceleration, and (E) follow-through phases.
### Table II
Electromyographic activity of the scapular muscles during spiking (% MVIC, mean ± SD)

| Muscle          | Phase          | Wind-up | Cocking | Acceleration | Deceleration | Follow-through |
|-----------------|----------------|---------|---------|--------------|--------------|----------------|
| Upper trapezius | Standard position | 40 ± 34 | 47 ± 36 | 56 ± 49 | 39 ± 23 | 26 ± 16 |
|                 | Posterior position | 52 ± 23 | 67 ± 51 | 88 ± 56 | 41 ± 37 | 30 ± 18 |
|                 | Medial position   | 44 ± 19 | 59 ± 46 | 61 ± 55 | 28 ± 27 | 18 ± 13 |
|                 | Lateral position  | 40 ± 17 | 62 ± 81 | 52 ± 39 | 41 ± 27 | 28 ± 17 |
| Middle trapezius| Standard position | 28 ± 35 | 38 ± 49 | 62 ± 42 | 25 ± 17 | 21 ± 19 |
|                 | Posterior position | 31 ± 46 | 33 ± 28 | 74 ± 48 | 27 ± 19 | 18 ± 11 |
|                 | Medial position   | 26 ± 19 | 41 ± 37 | 32 ± 26 | 17 ± 15 | 10 ± 7 |
|                 | Lateral position  | 28 ± 23 | 49 ± 65 | 63 ± 19 | 31 ± 20 | 21 ± 9 |
| Lower trapezius | Standard position | 37 ± 25 | 52 ± 30 | 37 ± 27 | 15 ± 13 | 11 ± 9 |
|                 | Posterior position | 39 ± 23 | 66 ± 47 | 52 ± 41 | 19 ± 18 | 18 ± 19 |
|                 | Medial position   | 27 ± 16 | 50 ± 39 | 38 ± 34 | 13 ± 11 | 8 ± 6 |
|                 | Lateral position  | 28 ± 18 | 51 ± 28 | 53 ± 45 | 22 ± 26 | 11 ± 10 |
| Serratus anterior| Standard position | 48 ± 51 | 58 ± 47 | 42 ± 36 | 43 ± 22 | 30 ± 22 |
|                 | Posterior position | 43 ± 11 | 55 ± 29 | 61 ± 51 | 32 ± 22 | 31 ± 19 |
|                 | Medial position   | 17 ± 9  | 35 ± 48 | 68 ± 68 | 54 ± 32 | 41 ± 29 |
|                 | Lateral position  | 16 ± 9  | 50 ± 48 | 36 ± 34 | 36 ± 25 | 24 ± 13 |

MVIC, maximal voluntary isometric contraction; SD, standard deviation.

* Significant difference between the standard position and the anterior position (P < .05).

### Table III
Electromyographic activity of the glenohumeral muscles during spiking (% MVIC, mean ± SD)

| Muscle          | Phase          | Wind-up | Cocking | Acceleration | Deceleration | Follow-through |
|-----------------|----------------|---------|---------|--------------|--------------|----------------|
| Anterior deltoid| Standard position | 53 ± 48 | 64 ± 43 | 43 ± 33 | 50 ± 28 | 42 ± 19 |
|                 | Posterior position | 57 ± 18 | 63 ± 40 | 74 ± 72 | 58 ± 27 | 49 ± 42 |
|                 | Medial position   | 28 ± 17 | 52 ± 46 | 53 ± 50 | 67 ± 41 | 35 ± 16 |
|                 | Lateral position  | 25 ± 14 | 58 ± 50 | 40 ± 50 | 52 ± 31 | 33 ± 23 |
|                 | P value           | .031    |         | .041       |             |                |
| Middle deltoid  | Standard position | 29 ± 15 | 37 ± 22 | 44 ± 40 | 37 ± 24 | 33 ± 13 |
|                 | Posterior position | 32 ± 18 | 47 ± 45 | 60 ± 33 | 40 ± 29 | 35 ± 23 |
|                 | Medial position   | 28 ± 15 | 43 ± 19 | 33 ± 41 | 34 ± 21 | 26 ± 24 |
|                 | Lateral position  | 26 ± 15 | 49 ± 28 | 50 ± 32 | 66 ± 31 | 39 ± 19 |
|                 | P value           | .005    |         |            |             |                |
| Posterior deltoid| Standard position | 6 ± 4  | 7 ± 6   | 50 ± 36 | 21 ± 14 | 18 ± 13 |
|                 | Posterior position | 9 ± 10 | 12 ± 7  | 55 ± 35 | 18 ± 11 | 19 ± 18 |
|                 | Medial position   | 9 ± 8  | 12 ± 9  | 21 ± 15 | 21 ± 18 | 17 ± 24 |
|                 | Lateral position  | 8 ± 7  | 14 ± 14 | 80 ± 41 | 39 ± 24 | 27 ± 17 |
|                 | P value           | .04     |         |            |             |                |
| Infraspinatus   | Standard position | 27 ± 19 | 28 ± 18 | 46 ± 34 | 62 ± 34 | 55 ± 42 |
|                 | Posterior position | 27 ± 13 | 29 ± 18 | 44 ± 12 | 44 ± 18 | 46 ± 21 |
|                 | Medial position   | 20 ± 14 | 22 ± 23 | 33 ± 12 | 57 ± 29 | 42 ± 28 |
|                 | Lateral position  | 19 ± 12 | 27 ± 24 | 62 ± 40 | 48 ± 31 | 36 ± 23 |
|                 | P value           | .043    |         | .019       |             |                |
| Pectoralis major| Standard position | 28 ± 53 | 13 ± 17 | 68 ± 33 | 20 ± 28 | 18 ± 25 |
|                 | Posterior position | 8 ± 1  | 14 ± 9  | 44 ± 29 | 11 ± 8  | 11 ± 10 |
|                 | Medial position   | 9 ± 8  | 9 ± 14  | 51 ± 30 | 26 ± 31 | 26 ± 34 |
|                 | Lateral position  | 7 ± 7  | 13 ± 20 | 69 ± 27 | 26 ± 31 | 15 ± 17 |
|                 | P value           | .007    |         |            |             |                |
| Teres major     | Standard position | 25 ± 41 | 29 ± 41 | 76 ± 55 | 29 ± 36 | 25 ± 35 |
|                 | Posterior position | 11 ± 12 | 13 ± 13 | 51 ± 19 | 29 ± 52 | 16 ± 14 |
|                 | Medial position   | 7 ± 8  | 8 ± 7   | 64 ± 29 | 29 ± 29 | 24 ± 34 |
|                 | Lateral position  | 6 ± 5  | 9 ± 7   | 72 ± 30 | 19 ± 17 | 20 ± 20 |
|                 | P value           | .05     |         |            |             |                |

MVIC, maximal voluntary isometric contraction; SD, standard deviation.

* Significant difference between the standard position and the anterior position (P < .05).

† Significant difference between the standard position and the posterior position (P < .05).

‡ Significant difference between the standard position and the medial position (P < .05).
The overall muscle activity was lower in the follow-through phase (<30% MVIC) than for the other 4 phases of the spike movement in the standard position, with higher activation of the AD (42% ± 19% MVIC), ISP (55% ± 42% MVIC), and MD (33% ± 13% MVIC). Activation of the ISP was significantly lower for the lateral (36% ± 23% MVIC) than for the standard (55% ± 42% MVIC; P = .019) position.

Discussion

As of this writing, several studies have reported on the EMG of shoulder girdle muscles during overhead pitching actions to elucidate the pathophysiology of shoulder injury,18,20,12,14 but with only 1 study having investigated the mechanism of injury related to the volleyball spike.20 Our study revealed significant effects of the ball impact position on the recruitment of shoulder girdle muscles during spiking in male volleyball players. Specifically, positioning of the ball impact posterior to the standard position significantly increased the activation level of the AD during the acceleration phase. In addition, a ball impact position located lateral to the standard position significantly increased the activation level of the PD during the acceleration phase and of the MD during the deceleration phase.

Glenohumeral muscle activation

Our results are generally in agreement with those of Rokito et al.22 who investigated the patterns of shoulder muscle recruitment during spiking in male volleyball players, with differences in specific details explained by differences in the type of EMG electrode used (surface vs. fine wire electrodes) and in the spiking position (static standing vs. jumping).

Rokito et al22 described the function of 4 of the 8 muscles included in our analysis (AD, ISP, TM, and the PM). During the wind-up phase, the AD assists with rapid elevation of the arm while the ISP initiates external rotation. Internal rotators, such as the PM, are also active to help stabilize the humeral head in the glenoid fossa. During the cocking phase, the AD maintains the arm in an elevated position, as the ISP produces a rapid external rotation of the shoulder, which helps to unload the anterior capsule as the humeral head translates anteriorly. The relatively high activity of the PM would offer some protection against this anterior translation of the humeral head through an eccentric contraction of the anterior shoulder musculature, which would control the rate velocity of shoulder external rotation.9 During the acceleration phase, the internal rotators (TM and PM) generate their highest activity to accelerate the arm into internal rotation of the shoulder, accelerating the arm forward and providing a stabilizing posterior restraint to anterior translation of the humeral head.9 Simultaneously, activity of the PD and TM peaked to rapidly extend the arm and the elbow, respectively. EMG investigations of the baseball pitch,7 football throw,11 and tennis serve23 have shown similar patterns of muscle activity. In this overhead position of the arm, we identified an increase in the activation of ISP from the cocking (28% ± 18% MVIC) to the acceleration (46% ± 34% MVIC) phase. By contrast, Rokito et al12 reported a decrease in the activation level of the ISP from the cocking (49% ± 16% MVIC) to the acceleration (27% ± 18% MVIC) phase, whereas the TM maintained a higher level of activation (51% ± 24% MVIC) to provide a restraint to the rapid acceleration of the humerus. After ball contact, the excess kinetic energy of the arm is dissipated through a deceleration of the upper extremity, from the deceleration to the follow-through phase, as in the tennis serve12 and after ball release in the baseball pitch.21,22 During the deceleration phase, we observed a higher activation of the ISP (62% ± 34% MVIC) than previously reported (38% ± 19% MVIC).22

It is assumed that high activation of the rotator cuff muscles generates a large compressive force to resist shoulder distraction and, thus, stabilize the humerus within the glenoid, as previously reported in overhead throwing motions, such as baseball pitching and football passing during this phase.8,24 However, we must consider differences in activation of the posterior muscles in the deceleration phase between the volleyball spike and the baseball pitch. Activation of the TM (34% ± 13% MVIC) and LD (20% ± 21% MVIC) during spiking22 is markedly lower than during the deceleration phase of the baseball pitch (84% ± 52% MVIC and 59% ± 35% MVIC, respectively).22

Immediately after ball release in the baseball pitch, the arm travels at a much higher velocity than after ball impact in the volleyball spike, requiring higher posterior torsional forces during the deceleration phase of the pitch to rapidly slow the motion of the arm.7 In contrast, for the volleyball spike, when the hand impacts the ball, the ball itself generates an equal and opposite force that slows the forward movement of the hand. The much slower arm movement in the deceleration phase of the spike, compared with the pitch, results in lower loading of the shoulder during the volleyball spike than the baseball pitch, requiring less restraining force to be generated by the posterior muscles and, hence, lower peak values to the histology and posterior muscles. Specifically, the higher follow-through phase, the activity of each muscle continues to decrease as the arm returns to its resting position by the player’s side, as previously reported,9 finally coming to rest, as described for the baseball pitch12 and the tennis serve.15 The follow-through for the volleyball spike appears to be a noncritical phase in terms of performance, with the remainder of the kinetic energy being naturally dissipated as the motion is completed.9

Scapular muscle activation

When comparing our data to a previous study on overhead baseball pitch,7 the pattern of recruitment of the scapular muscles (UT, MT, LT, and SA) was similar for the volleyball spike, from the wind-up to the follow-through phases. In the cocking phase, our findings of a higher activation of the SA muscle, in combination with a moderate activation of the UT, MT, and LT, to upwardly rotate and elevate the scapula and abduct the shoulder as the arm is initially brought overhead is comparable to the pattern reported by DiGiovine et al1 for the overhead baseball pitch. Specifically, DiGiovine et al recorded an activation level of the SA of 106% ± 56% MVIC on the fourth rib and 69% ± 32% MVIC on the sixth rib, with our values being comparable at 58% ± 18% MVIC on the sixth rib. In the acceleration phase, the activity of these 4 muscles remained generally high (>40% MVIC) to accelerate the arm forward. A moderate to high activation of these muscles was sustained during the deceleration phase to control scapular elevation, projection, and rotation.

The effect of ball impact position on EMG

Compared with the standard position, the posterior position of ball impact was associated with an increase in activity of the AD and a decrease in the activity of the PM during the acceleration phase, as well as a decrease in the activation level of the ISP during the deceleration phase. This change in muscle activation reflects the necessity of placing the shoulder in a position of maximal horizontal extension, elevation, and external rotation when the ball impact position is shifted posteriorly.13 Specifically, the higher activity of the AD maintains the elevated position of the humerus during the acceleration phase, as the associated elongation of the PM would explain the decrease in the activity level of this muscle. Furthermore, eccentric contraction of the fibers of the ISP muscle
may decrease during the deceleration phase because the attacker would not be able to hit the ball as hard from a posterior position compared with the standard position. A medial shift in the position of ball impact significantly decreased activation of the TM, compared with the standard position. This decrease likely reflects the elongation in the muscle fibers of the TM that is associated with a medial shift in the position of ball impact, relative to the standard position, and a decrease in concentric contraction during the cocking phase.

The most obvious change in the recruitment of the shoulder muscles was observed when the ball impact position was moved laterally, compared with the standard position. Specifically, we observed a significant increase in the activation of the PD and MD, compared with the standard position, during both the acceleration and deceleration phases. These findings indicate the greater ease in performing shoulder extension, with higher impact forces transferred to the ball, when the ball impact position is moved laterally, close to the “zero” (or scapular plane) position. In contrast, a significant decrease in activation of the SA and AD was observed during the wind-up phase and in the ISP during the follow-through phase. It is likely that contraction of the SA and AD is not needed to elevate the arm when the ball impact position is shifted laterally, from the standard position, through the wind-up phase. Moreover, the glenohumeral joint was in a more internally rotated position for this laterally shifted position of ball impact, compared with the standard position, during the follow-through phase; thus, eccentric contraction of the ISP may decrease.

Overall, our results indicate that a posterior or lateral shift in the position of ball impact may cause an increase in the activity of the deltoid muscle, which would cause a decrease in the centripetal force of the humeral head through the acceleration and deceleration phases. As such, neuromuscular exercises, combined with strengthening of the rotator cuff muscle, might reduce the risk of shoulder injury during performance of the volleyball spiking movement.

**Duration of each phase in volleyball spiking**

With regard to the effect of the ball impact position on the relative duration of the 5 phases of the spiking movement, the mean duration of the deceleration phase was longer for the posterior than the ball impact standard position. Posterior displacement of the ball impact position may shorten the distance the hand needs to travel to contact the ball, which would shorten the duration of the acceleration phase and, thus, prolong the deceleration phase. Of note, the mean duration of the wind-up phase in our study (0.84 ± 0.21 seconds) was more than twice the duration reported by Rokito et al (0.37 ± 0.05 seconds), whereas the duration of the other 4 phases of the spike were comparable. As a rapid movement of shoulder flexion is required in the wind-up phase to produce a vertical force against gravity to jump higher, the duration in the wind-up phase in Rokito et al’s study, which simulated a real spike, including a run-up, would naturally be shorter than for the static standing position in our study.

**Limitations**

The limitations of our study need to be acknowledged. First, the sample size of 11 male volleyball players was small. Moreover, as shown in Tables II and III, there was variability in performance on the spike movement, as shown by the high standard deviations in Tables II and III. This variability might reflect differences in the positions played, including middle spiker and setters. We presented the standard deviation in each cell, as well as the mean value, to prevent overestimating of our data. Despite this large variation, we did identify significant effects of changing the ball impact position on the pattern of activation of scapular and glenohumeral muscles. Second, performance of the volleyball spike in a static standing position does not simulate the real spiking movement, which is integrated into a jumping motion. Third, we could not measure activation of the supraspinatus or subscapularis muscles with the type of surface electrodes that we used. Further biomechanical studies are needed to include simulation of a real spiking movement, including the run-up, and analysis of all other structures of the kinetic chain, from the lower limbs to the shoulder, to clearly elucidate the impact of the spike movement on the shoulder joint. Moreover, it is also necessary to evaluate the activity of the rotator cuff muscles, which must absorb, through eccentric activity, the kinetic energy that is not transmitted to the ball.

**Conclusion**

Based on our findings of increased activation of the deltoid muscle with a posterior or lateral shift in the position of ball impact, which would decrease the centripetal force of the humeral head through the acceleration and deceleration phases of the spiking movement, we propose that neuromuscular exercises, in combination with strengthening of the rotator cuff muscle, could be beneficial in lowering the risk of shoulder injury in wing attackers in volleyball.

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