Lower trapezius weakness and shoulder complex biomechanics during the tennis serve

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Abstract:

PURPOSE: This study aimed to assess the effect of lower trapezius (LT) weakness on humeral and scapular kinematics and shoulder muscle activity during the tennis serve. **METHODS:** Fifteen competitive male tennis players (age: 23.8 ± 3.4 years; height: 182.8 ± 6.7 cm; mass: 76.6 ± 8.7 kg; tennis experience: 15.6 ± 4.9 years) performed two tennis serves before and after selective fatigue of the LT (25-min electric muscle stimulation). During each tennis serve, racket, humeral and scapular kinematics and the activity of 13 shoulder muscles were recorded using an optoelectronic system synchronized with indwelling and surface electromyography. The serve was split into five phases, i.e., early and late cocking, acceleration, early and late follow-through. **RESULTS:** Selective fatigue led to a 22.5 ± 10.4% strength decrease but did not alter maximum racket speed and humerothoracic joint kinematics. However, increased scapular upward rotation was observed in the acceleration ($p=0.02$) and early follow-through ($p=0.01$) phases. Decreased muscular activity was observed during the early cocking phase for the LT ($p=0.01$), during the acceleration phase for the LT ($p=0.01$), anterior deltoid ($p=0.03$), pectoralis major ($p=0.04$) and subscapularis ($p=0.03$), and during the early follow-through phase for the anterior deltoid ($p=0.03$) and LT ($p=0.04$). **CONCLUSION:** LT weakness altered neither serve velocity nor humerothoracic joint kinematics, but impaired scapulothoracic kinematics and anterior shoulder muscle activation. Such alterations may reduce the subacromial space and jeopardize humeral head stability. These findings shed new light on the consequences of LT weakness, highlighting the importance of monitoring and strengthening this muscle in overhead athletes.

Key words: overhead sport, muscle activity, humerothoracic joint, scapulothoracic joint

Introduction

Chronic shoulder injuries affect 20 to 50% of both junior and adult tennis players (1, 2). These injuries commonly involve scapular dyskinesis, which is an alteration of the physiological movement or positioning of the scapula (3). Causes of scapular dyskinesis include nerve palsy (long thoracic and spinal accessory), muscle stiffness (pectoralis minor, biceps sort head), glenohumeral internal rotation deficit related to tightness of the posterior soft tissues, or alterations in the periscapular muscles (3). However, it is weakness, fatigue or delay in activation of the lower trapezius muscle (LT) that are thought to be the primary causes of scapular dyskinesis (3). Yet exactly how such LT alterations affect tennis skills remains unclear. Fatigue is defined as a physiological process that decreases the muscle's ability to produce force and alters its contraction timing (4). Most fatigue studies use a specific task to target one muscle or one muscle group (5, 6). However, such tasks also fatigue the surrounding muscles, due to co-contraction and synergistic processes, as well as the other muscles involved in the kinetic chain (7). To better target one single muscle, therefore, electric muscle stimulation is often used (8, 9). With the electrodes placed close to the muscle tendons, the stimulation acts on the intramuscular axons triggering the contraction (10). The high metabolic demand from such contractions leads to peripheral fatigue, thereby reducing the muscle's ability to produce force (10). Electric muscle stimulation can be considered an appropriate way to produce muscle weakness and thus to observe its impact on tennis skills.

The tennis serve is the most frequent stroke during a game, representing 45 to 60% of all strokes (11). During the cocking phase of the tennis serve, the LT acts concentrically to posteriorly tilt and externally rotate the scapula, (7, 12), while the humerus is abducted to 90° and at maximum external rotation (12). During the follow-through phase, the LT also acts eccentrically to restrain scapular anterior tilt and internal rotation (7, 12). In addition, the tennis serve is known to engender a high risk of shoulder injury because of: *(i)* the physiological contact between the greater tuberosity of the humeral head and the posterosuperior part of the scapular glenoid at the end of the cocking phase (13, 14); and *(ii)* the high constraint applied to stabilize the shoulder complex when upper-limb/racket motion is decelerated, *i.e.* during the follow-through phase (15). It has been shown that overhead athletes with an impingement sign present decreased LT activation and increased upper trapezius activation (16, 17). Such intramuscular imbalance may limit scapular external rotation and posterior tilt (3, 17). Performing a tennis serve with a weak LT may thus affect scapular kinematics, potentially resulting in increased pressure on the tuberosity-glenoid contact (18) and anterior translation of the humeral head (12). However, to the best of our knowledge, the effect of LT weakness on shoulder complex biomechanics during the tennis serve has never been studied. Knowledge of such biomechanical effects could shed light on shoulder pathomechanics as related to LT weakness, suggesting strategies to improve injury prevention in tennis.

This study aimed to assess the influence of LT weakness on humeral and scapular kinematics and on shoulder muscle activity during a tennis serve. We hypothesized that decreased LT strength (caused by selective fatigue) would be compensated for by increased upper trapezius activity. As the LT is mainly involved in the cocking and follow-through phases, this change in the upper-lower trapezius force couple would result in altered scapular internal/external rotation and anterior/posterior tilt during both phases.

Methods

Participants

Fifteen competitive male tennis players (age: 23.8 ± 3.4 years; height: 182.8 ± 6.7 cm; mass: 76.6 ± 8.7 kg; tennis experience: 15.6 ± 4.9 years; weekly tennis training: 4.4 ± 1.8 hours, Canadian ranking: 4 to 6; International Tennis Number: 2 to 4 according to the system of equivalent rankings of the International Tennis Federation) gave their written informed consent to participate in this study, which was approved by the ethics committee of the 'Université de Montréal' (16-165-CERES-D). Inclusion criteria were being an adult competitive player with an International Tennis Number rating better than 6. Exclusion criteria were having shoulder problems in the last six months or heart problems.

Experimental design

The experiment was structured in two sessions scheduled at least 48h apart to allow full recovery: 1) familiarization with electric muscle stimulation on the dominant LT to determine the maximum intensity of the fatigue task that the player could tolerate and 2) testing. During the testing session, participants performed two sets of 13 maximum voluntary isometric contractions (MVIC), a static position, a series of setup trials (included a static position with the arm abducted to 90° in the scapular plane and with the arm fully abducted, lateral tilt of the trunk, rotation of the trunk, flexion/extension of the trunk, flexion/extension of the elbow, pronation/supination of the forearm, rotation of the wrist, flexion/extension of the wrist and abduction/adduction of the wrist) to locate the joint centers. They performed two set of two tennis serves. Before a first set of two serves, players performed a 10-15 min warm-up in line with their own routine. To familiarize them with the study conditions, they also repeated a few racket strokes in the camera field of view (width: 4 m, length: 5 m; height: 3 m). Players then performed the fatigue task targeting their dominant LT, and immediately afterwards performed a second set of two tennis serves. Two tennis serves ensure a short delay between the end of the electrostimulation period and the end of motions of interest to limit the recovery of lower trapezius strength.

Kinematics

Players were equipped with 33 reflective skin markers (pelvis: 4; thorax: 6; clavicle: 2; scapula: 5; arm: 4; forearm: 4; wrist: 4; hand: 4) according to Jackon et al.'s (19) kinematics model (Figure 1). In line with the findings of Blache, Dumas, Lundberg and Begon (20) about shoulder soft tissue artefact, the most affected markers of the Jackon et al.'s (19) kinematics model were removed to simplify the model. This marker set included anatomical markers positioned on bony landmarks to define the biomechanical model and technical markers positioned on skin areas that minimize soft tissue artifacts. Six additional markers were attached to the racket-head and one marker to the non-dominant hand, to determine performance and the beginning of the serve motion, respectively. The reflective marker positions were recorded by an 18-camera motion analysis system (VICON T20S & T40S, Oxford, UK) at 300 Hz during static position and tennis serves. Before and after selective fatigue of the dominant LT, participants performed two flat serves without a ball (for in-lab safety reasons) at maximum speed, having been instructed to reproduce the technique they used on the tennis court.

A 25 degree-of-freedom (DOF) kinematics model was personalized using the static and setup trials: global-pelvis and thoracopelvic joints (6 DoF each), sternoclavicular and acromioclavicular (3 DoF each), glenohumeral (3 DoF), elbow and wrist (2 DoF each). The centers of rotation of the wrist joint and between the pelvis and the thorax were located using the SCoRE algorithm (21) and elbow flexion and supination axes using the SARA algorithm (22), while the sternoclavicular, acromioclavicular and glenohumeral joint centers were defined according to anatomical bony landmarks (23). Finally, following Michaud et al. (2017), a scapulothoracic pseudo-joint constraint was modeled as a point-to-ellipsoid contact (barycenter of the Angulus Acromialis, Angulus Inferior and Trigonum Spinae) where a thorax-sized ellipsoid was fitted to the area covered by the scapula. Briefly, the ellipsoid radii were obtained by minimizing the sum of the quadratic distances between the scapula landmarks (angulus inferior and trigonum spinae) and the ellipsoid envelope for three arm positions (rest, abducted to 90°, fully abducted). Moreover, the most lateral point of the mediolateral axis of the ellipsoid had to be the most lateral point of the thorax and the origin of center of the ellipsoid had to coincide with the origin of the thorax coordinate system (24).

Humerothoracic and scapulothoracic rotation matrices, usually used to determine shoulder kinematics in tennis (12), were calculated from the sternoclavicular, acromioclavicular and glenohumeral joint angles. Then the Cardan angles were extracted to define the joint rotation axes, with X the antero-posterior direction, Y the lateral direction and Z the infero-superior direction. The humerothoracic joint rotations were, successively, abduction(-)/adduction(+), flexion(+)/extension(-) and internal(+)/external(-) rotation, according to the YXZ rotation sequence (25). The scapulothoracic joint rotations were internal(+)/external(-) rotation, upward(-)/downward(+) rotation and anterior(-)/posterior(+) tilt, and were defined using the rotation sequence ZYX (26).

Electromyography

Supraspinatus, infraspinatus and subscapularis activities were measured using indwelling electromyography (EMG) sampled at 2000 Hz (Figure 1). Under sterile conditions, intramuscular paired hook-fine-wire electrodes (30 mm x 27 ga, Natus Neurology, Middleton, WI, USA) were inserted according to Kadaba et al. (27) and Morris et al. (28). The activities of the anterior, posterior and middle deltoids, biceps, triceps, pectoralis major, upper and lower trapezius, latissimus dorsi, and the serratus anterior were recorded using surface EMG sampled at 2000 Hz. After shaving and cleaning the skin with alcohol, sensors (Delsys Trigno Wireless EMG, Natick, MA, USA; inter-electrode distance of 10 mm) were placed according to the SENIAM recommendations over the muscle belly (Figure 1). Thirteen maximum voluntary isometric contractions (MVIC) were performed to assess the maximum voluntary activation of each muscle (29). Participants performed two 5-s contractions for each MVIC with 30 s rest between contractions. The raw EMG signal was filtered using a 4th order Butterworth bandpass filter (15- 500 Hz for the surface EMG and 15- 800 Hz for the indwelling EMG). The EMG envelope values were calculated using a 250-ms sliding window Root Mean Square for the MVIC and a 50-ms sliding window for the serve. For each muscle, the envelopes were normalized by the maximum muscle voluntary activation obtained over the two repetitions of 13 MVIC tests (29). The envelope of every muscle was integrated (iEMG) over the different phase durations (i.e., early and late cocking, acceleration and early and late follow-through; see section *serve analysis*) for statistical analysis.

Insert Figure 1

Fatigue Task

The fatigue task was performed after the first set of serves. A musculoskeletal electric stimulator (Compex mi-Sport; DJO Global, Vista, CA) was used to generate a decrease in LT strength due to peripheral fatigue. Participants were in a supine position, humerus abducted to 140° and at maximum external rotation. Bipolar electrodes (5 cm x 5 cm) were placed on the skin over the extremities of the LT, close to the lateral side of the seventh thoracic spinous process and close to the medial part of the inferior angle of the scapula, in line with Bdaiwi et al. (9). The current width pulse was 300 μs, and the frequency was 40 Hz. The session lasted 25 minutes. Current intensity was increased every two minutes during the first half of the session. Once the maximum intensity determined as tolerable for the players from the first session was reached, current intensity was kept constant. Mean intensity was 79.5 ± 60.5 mA (range: 48 to 120 mA). To monitor strength decrease, players performed a one-repetition lower trapezius isometric strength test (30) before and immediately after the fatigue task, and after the second set of serves. For the strength tests, players were in a supine position, humerus abducted to 140° and at maximum external rotation, with elbow fully extended. Strength was measured using a hand-held dynamometer (HHD; microFET2; Hoggan Health Industries Inc, West Jordan, Utah, USA) placed on the lateral aspect of the distal radius while the players exerted extension against the resistance of the experimenter.

Serve analysis

The barycenter of the six markers of the racket-head, coinciding with its center, was computed and used to calculate racket-head velocity throughout the tennis serve. The pre-impact maximum velocity of each serve was defined as the performance factor (31). Four key events in the tennis serve were determined based on joint kinematics: 1) the beginning of the movement, corresponding to the time when the non-dominant hand was at the same height as the xiphoid process; 2) the end of the cocking phase, defined as the maximum humeral external rotation; 3) the impact (T_{impact}=0 s), defined as the maximum racket elevation (15); and 4) the end of the movement, corresponding to the minimum humeral adduction (12). The humerothoracic and scapulothroracic joint rotations at each key event were collected for subsequent analysis. According to these four events, three phases were defined: the cocking phase from the beginning of the movement to the maximum humeral external rotation, the acceleration phase from the maximum humeral external rotation to the impact, and the follow-through phase from the impact to the end of the movement. The cocking and follow-through phases were also divided into two subphases as proposed by Morris, Jobe, Perry, Pink and Healy (32): early (0-75%) and late (75-100%) cocking; early (0-25%) and late (25-100%) follow-through. Overall, therefore, the tennis serve was divided into 5 phases according to 6 events (4 key events and the 2 subordinate events). For each phase, duration, iEMG, minimum and maximum humerothoracic and scapulothroracic joint rotations and the timing of these extreme rotations were calculated and used for data analysis.

Data reduction

Data for two players were removed from the analysis because some EMG signal recordings failed after the fatigue task. In the end, data for 13 players were used in the analysis, which included 11 players monitored with indwelling EMG (two players did not agree to it).

Statistical analysis

After testing the normality and homoscedasticity of the values, paired t-tests were applied to compare outcome measures before and after selective fatigue of the lower trapezius. The effect size (ES) was calculated and interpreted according to the Cohen scale (1988) (33) (small: 0.2, medium: 0.5, and large: 0.8), and level of significance was set at $p \leq 0.05$. All statistical analyses were performed using SPSS 11.0 software (SPSS, Inc., Chicago, IL, USA).

Results

The paired t-test showed a significant decrease in LT strength values (Table 1), of 22.5 ± 10.4%, after electric muscle stimulation (ES = 1.26, large effect; p = 0.001). After the second set of serves, the decrease in LT strength was 4.0 ± 15.2%, not significantly different from the initial value. No significant differences were found for serve duration and performance (Table 1) before and after LT selective fatigue.

Insert Table 1

A representative participant's humerothoracic and scapulothoracic joint kinematics during the tennis serve, before and after selective fatigue of the lower trapezius, are available in Supplemental Digital Content 1 (see Figure, SDC 1, humerothoracic and scapulothoracic joint kinematics during the tennis serve). Angle values at the key events can be found in

Supplemental Digital Content 2 (see Table, SDC 2, humerothoracic and scapulothoracic angles at tennis serve key events). At impact, the humerus relative to the thorax was more flexed (ES = 0.63, medium-to-large effect; p = 0.02) after the fatigue task (Figure 2). At the end of motion, the humerus relative to the thorax was less internally rotated (ES = 0.71, medium-to-large effect; p = 0.01), while the scapula relative to the thorax was more downwardly rotated (ES = 0.71, medium-to-large effect; p = 0.01) after the fatigue task. No other significant differences were found for humerothoracic and scapulothoracic joint rotations at the tennis serve key events (Figure 2, Supplemental Digital Content 2). The extreme values for shoulder joint kinematics (Table 2) were affected following the fatigue task. The humerus relative to the thorax was significantly more flexed during the acceleration (ES = 0.60, medium-to-large effect; p = 0.03) and early follow-through phases (ES = 0.59, medium; p = 0.03), while the scapula relative to the thorax was significantly more upwardly rotated (ES = 0.59, medium effect; p = 0.02 and ES = 0.67, medium-to-large effect; p = 0.01, respectively). No significant differences were found in the timing of the extreme values for shoulder kinematics (Table 2).

Insert Figure 2 and Table 2

A representative participant’s EMG signal envelopes during the tennis serve, both before and after LT selective fatigue, are available in Supplemental Digital Content 3 (see Figure, SDC 3, EMG signal envelopes of 13 shoulder muscles during the tennis serve). During the early cocking phase (Figure 3), the iEMG of the LT was significantly lower (ES = 0.53, medium effect; p = 0.04), while the biceps was significantly more activated (ES = 0.58 medium effect; p = 0.04) after LT selective fatigue. During the late cocking phase (Figure 3), iEMG values decreased significantly for the posterior deltoid (ES = 0.67, medium-to-large effect; p = 0.04) and the pectoralis major (ES = 0.64, medium-to-large effect; p = 0.02) after LT selective fatigue. During the acceleration phase (Figure 3), iEMG values were significantly lower after LT selective fatigue for the LT (ES = 0.98, large effect; p = 0.01), the latissimus dorsi (ES = 0.53, medium effect; p = 0.04), the subscapularis (ES = 0.87, large effect; p = 0.03), the anterior deltoid (ES = 0.62, medium-to-large effect; p = 0.03), the pectoralis major (ES = 0.54, medium effect; p = 0.04), and the triceps (ES = 0.68, medium-to-large effect; p = 0.03). During the early follow-through phase (Figure 3), iEMG values were significantly lower after LT selective fatigue for the LT (ES = 0.51, medium effect; p = 0.04), the posterior deltoid (ES = 0.66, medium-to-large effect; p = 0.04), the anterior deltoid (ES = 0.63, medium-to-large effect; p = 0.03), the biceps (ES = 0.82, large effect; p=0.01), and the triceps (ES = 0.68, medium-to-large effect; p = 0.03). No differences were found for the other muscles.

Insert Figure 3 here

Discussion

The aim of this study was to investigate whether LT weakness generated by electric muscle stimulation affected scapular and humeral kinematics and shoulder muscle activity during the tennis serve. We found that LT weakness did not alter serve performance, humerothoracic and scapulothoracic joint rotations at the key events, nor humerothoracic joint kinematics during motion. However, weakness decreased LT activation, thereby altering scapulothoracic joint kinematics and leading to a decrease in anterior shoulder muscle activation during the acceleration and follow-through phases.

The maximum racket speed of competitive tennis players when serving can be close to 80 km/h (31). In the laboratory, without a ball, our competitive players served at a similar racket speed (85±4 km/h). Our first set of tennis serves presented humeral and scapular kinematics and electromyographic patterns (Figure 2, Figure 3, and Figure A1) similar to those described in the literature. During the cocking phase, the infraspinatus and the posterior deltoid acted to externally rotate the humerus to 125°, while the supraspinatus and middle deltoid acted to abduct the humerus to 100° (7, 12, 34). At the same time, the scapula was slightly externally rotated, upwardly rotated to 30° and posteriorly tilted due to the actions of the serratus anterior, upper and lower trapezii (7, 12, 34). During the acceleration phase, the pectoralis major, latissimus dorsi, and subscapularis muscles were activated to internally rotate the humerus (7, 12, 34). The serratus anterior was also activated to internally rotate the scapula to 33° (7, 12, 34). The other shoulder muscles acted to stabilize the shoulder joints (7). Finally, during the early follow-through phase, the shoulder muscles continued their activations to internally rotate and adduct the humerus as well as to internally and downwardly rotate and posteriorly tilt the scapula (7, 12). As the laboratory conditions did not seem to significantly influence tennis serve performance and kinematics (35), our serves before LT selective fatigue can be considered an adequate representation of the serve in competitive tennis players. We therefore assume that the alterations in humeral and scapular kinematics and shoulder muscle activity observed after the fatigue task were due to the LT weakness it induced.

LT weakness is the principal cause of scapular dyskinesia, commonly involved in shoulder injuries (3, 17). The weakness of a single muscle can be simulated using selective fatigue, which reduces the targeted muscle's ability to produce strength (4, 8). After electric muscle stimulation, we observed a decrease in LT strength of 22.5 ± 10.4% which was reduced to 4.0 ± 15.2% after the second set of serves. This large intersubject variability could be explained by the range of electrostimulation current intensities, which were determined according to each players' discomfort tolerance. Particpants' recovery in LT strength was almsost complete after the post-test (i.e. about 5 min after the fatigue task), as already reported after high intensity excercices (36). The electrostimulation commonly simulates the muscular effort of a high intensity exercise, but the motor unit recruitement differs from a natural contraction (10).

Indeed, with the electrostimulation, the motor unit recruitment is randomized, superficial and fixed, while in the natural contraction, it is organized and adaptive (10). During the post-test, the neuromuscular system may be reorganized by recruiting motor units, which were not involved during the electrostimulation task. This could then explain the fast observed strength recovery. The strength decrease that remained at the end of our post-test was however similar to the 4% decrease reported after a series of overhead performances, such as after a baseball game (99 ± 29 throws) (6). We then considered that the second set of tennis serves, i.e. after electric stimulation, was performed with a weaker LT than the first set.

Despite LT weakness, the players sustained their performance and maintained their humerothoracic and scapulothoracic joint rotations during tennis serve key events, as well as their humerothoracic joint kinematics throughout the tennis serve. Previous EMG studies (7, 12) showed that during this motion, the LT acts to externally rotate and posteriorly tilt the scapula during the late cocking phase and helps decelerate the arm-racket complex during the follow-through phase by controlling and limiting the internal and upward rotation and anterior tilt of the scapula. In the weakness condition, LT activation was low throughout the tennis serve. A decrease in LT activation is commonly compensated for by an increase in upper trapezius activation during arm abduction or arm rotation (16, 17). However, this adaptation was not observed during the tennis serve: upper trapezius activity was not modified. Decreased LT activation coupled with unchanged upper trapezius activation may alter scapular kinematics (17). We also found increased scapular upward rotation during both the acceleration and the early follow-though phases. According to Ludewig & Reynolds (37), such an increase is thought to be beneficial, increasing the subacromial space when the upper limbs are elevated, as in the acceleration phase. However, according to Karduna et al. (38), it may also reduce the subacromial space when the humerus is abducted to 90° and maximally internally rotated, as in the follow-through phase. Consequently, performing the follow-through phase under LT weakness may place the shoulder at higher risk of rotator cuff tendon impingement between the acromion and the humeral head. Furthermore, in our study, the decrease in LT activation was accompanied by reduced activations of other shoulder muscles: posterior (posterior deltoid and triceps brachialis) and anterior (anterior deltoid, latissimus dorsi, pectoralis major, subscapularis, and biceps brachialis). Since most of these muscles are involved in the humeral head’s active stability through their co-contraction (39), their reduced activations could jeopardize humeral head stability, potentially resulting in shoulder impingement (40). With repeated serves during tennis training and competition, the potential decrease in the subacromial space and loss of humeral head stability may increase the risk of rotator cuff tendinopathy (3, 18, 38). Consequently, performing repeated serves with a weak LT may alter scapular kinematics and humeral head stability and potentially jeopardize rotator cuff tendon integrity.

One limitation to this study is the fact that selective fatigue was induced through electric muscle stimulation, which could trigger non-physiological contractions (10) that may affect muscle functions differently from a physiological contraction. Consequently, the results obtained from simulated LT weakness may differ slightly from those that would be observed with natural LT weakness. Furthermore, the maximum intensity of electrostimulation tolerable by each player was determined after a unique familiarization session, resulting in a large variability in current intensity (48 to 120 mA) used for the fatigue task. This variability may explain the large inter-subject variability observed in LT strength decrease. More familiarization sessions should be included in future studies to determine the maximum intensity tolerable for player with more accuracy. A second limitation concerns the study’s lack of clinical assessment of scapular dyskinesia, tendinopathies and impingements, shoulder problems related to LT weakness and which could alter shoulder biomechanics. The test-retest procedure used in this study should minimize the impact of shoulder problems, since each player acted as his own control. Finally, our laboratory conditions meant that the serves were performed without a ball, which may unconsciously influence serve performance. However, this study was the first to assess the effect of LT weakness on humerothoracic and scapulothoracic joint kinematics and shoulder muscle activation during sport-specific motion. It would be valuable to corroborate our results by assessing the shoulder complex kinematics and muscular activity during overhead sport-specific motion in a symptomatic population. Future studies using musculoskeletal modeling may be useful to evaluate the relationship between loss of humeral head stability, change in humeral head translation and LT weakness.

Conclusion

Despite the weakness of the lower trapezius, players still achieved a fast tennis serve while maintaining their humeral kinematics. However, they presented a decrease in lower trapezius activation that may alter scapular kinematics and anterior shoulder muscle activation during the acceleration and follow-through phases. Such alteration could reduce the subacromial space and humeral head active stability, hence jeopardizing rotator cuff tendon integrity under intensive competition and training. These findings afford coaches and clinicians insights into the possible shoulder-complex pathomechanics related to weakness in scapular stabilizer muscles. They also underline the importance of monitoring and strengthening the lower trapezius muscle in overhead athletes.

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Conflict of interest

The authors declare no conflict of interest.

The authors also declare that the result of the present study do not constitute endorsement by ACSM

The authors declare that the results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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Figure caption

Table 1: Mean ± standard error of maximum isometric strength of the lower trapezius, duration of serve phases and maximum racket speed before (pre) and after (post) selective fatigue of the lower trapezius. Significant difference represented in bold.

	Pre	Post	P
Lower trapezius strength (N)	**44.8 ± 4.9**	**34.3 ± 3.2**	**0.001**
Duration of the serve (s)	1.28 ± 0.05	1.24 ± 0.05	0.07
Duration of the early cocking phase (s)	0.64 ± 0.03	0.65 ± 0.03	0.46
Duration of the late cocking phase (s)	0.21 ± 0.01	0.22 ± 0.01	0.46
Duration of the acceleration phase (s)	0.09 ± 0.01	0.08 ± 0.01	0.11
Duration of the early follow-through phase (s)	0.08 ± 0.01	0.08 ± 0.01	0.07
Duration of the late follow-through phase (s)	0.25 ± 0.02	0.23 ± 0.02	0.08
Maximum racket speed (km/h)	84.6 ± 4.2	82.7 ± 5.1	0.19

Table 2 Mean ± standard error of the extreme orientations (in degrees) of the humerothoracic (HT) and scapulotohoracic (ST) joints and their timing (in seconds relative to the impact $t_{impact}=0$ s) in the different tennis phases before (pre) and after (post) selective fatigue of the lower trapezius; bold represents a significant difference before and after fatigue at $p<0.05$.

Phases				Maximum	p	timing (s)	p	minimum	p	timing (s)*	p
Late cocking	HT	Abduction(-)/adduction(+)	Pre	-71.8 ± 4.1	0.41	-0.24 ± 0.02	0.27	-100.4 ± 4.5	0.12	-0.12 ± 0.02	0.48
			Post	-71.5 ± 4.7		-0.24 ± 0.01		-98.8 ± 5.1		-0.12 ± 0.02	
		Flexion(+)/extension(-)	Pre	36.1 ± 4.8	0.08	-0.09 ± 0.01	0.30	0.0 ± 4.6	0.10	-0.29 ± 0.01	0.07
			Post	39.3 ± 4.7		-0.09± 0.01		2.4 ± 4.3		-0.25 ± 0.03	
		Internal(+)/external(-) rotation	Pre	-64.1 ± 4.9	0.21	-0.29 ± 0.01	0.31	-125.5 ± 6.8	0.25	-0.09 ± 0.01	0.12
			Post	-66.5 ± 5.9		-0.29 ± 0.01		-123.9 ± 6.8		-0.12 ± 0.03	
	ST	Internal(+)/external(-) rotation	Pre	27.7 ± 2.9	0.15	-0.13 ± 0.02	0.13	18.9 ± 2.7	0.25	-0.23 ± 0.01	0.49
			Post	29.1 ± 2.8		-0.11 ± 0.01		19.3 ± 2.7		-0.23 ± 0.02	
		Upward(-)/downward(+) rotation	Pre	-22.8 ± 4.4	0.47	-0.23 ± 0.02	0.33	-34.4 ± 3.2	0.38	-0.16 ± 0.02	0.16
			Post	-22.9 ± 4.5		-0.23 ± 0.02		-34.3 ± 2.7		-0.14 ± 0.02	
		Anterior(-)/posterior(+) tilt	Pre	5.2 ± 2.8	0.38	-0.11 ± 0.01	0.15	-11.7 ± 2.5	0.25	-0.27 ± 0.01	0.43
			Post	4.8 ± 3.1		-0.10 ± 0.01		-11.0 ± 2.4		-0.27 ± 0.01	
Acceleration	HT	Abduction(-)/adduction(+)	Pre	-88.8 ± 6.3	0.37	-0.03 ± 0.01	0.41	-102.7 ± 4.8	0.22	-0.05 ± 0.01	0.40
			Post	-87.9 ± 6.9		-0.03 ± 0.01		-104.9 ± 3.9		-0.05 ± 0.01	
		Flexion(+)/extension(-)	Pre	**42.4 ± 4.7**	**0.02**	-0.02 ± 0.01	0.47	34.7 ± 4.9	0.06	-0.06 ± 0.01	0.32
			Post	**45.3 ± 4.8**		-0.02 ± 0.01		37.8 ± 4.8		-0.06 ± 0.01	
		Internal(+)/external(-) rotation	Pre	-72.2 ± 10.8	0.19	-0.00 ± 0.00	0.43	-125.5 ± 6.8	0.24	-0.08 ± 0.01	0.40
			Post	-67.6 ± 12.6		-0.00 ± 0.00		-123.9 ± 6.8		-0.09 ± 0.01	
	ST	Internal(+)/external(-) rotation	Pre	36.5 ± 2.6	0.30	-0.03 ± 0.01	0.37	24.8 ± 2.3	0.13	-0.05 ± 0.01	0.35
			Post	36.8 ± 2.6		-0.02 ± 0.01		26.0 ± 2.5		-0.06 ± 0.01	
		Upward(-)/downward(+) rotation	Pre	**-24.9 ± 3.4**	**0.02**	-0.03 ± 0.01	0.48	-34.9 ± 2.8	0.42	-0.06 ± 0.01	0.32
			Post	**-27.5 ± 2.8**		-0.03 ±0.01		-34.7 ± 2.6		-0.05 ± 0.01	
		Anterior(-)/posterior(+) tilt	Pre	9.0 ± 2.7	0.23	-0.04 ± 0.01	0.28	1.1 ± 2.8	0.38	-0.06 ± 0.01	0.38
			Post	9.4 ± 2.6		-0.03 ± 0.01		1.5 ± 3.1		-0.06 ± 0.1	
Follow-through	HT	Abduction(-)/adduction(+)	Pre	-71.2 ± 10.3	0.26	0.06± 0.01	0.37	-98.8 ± 6.6	0.47	0.03 ± 0.01	0.35
			Post	-73.7 ± 9.3		0.06 ± 0.01		-98.5 ± 7.1		0.03 ± 0.01	
		Flexion(+)/extension(-)	Pre	53.0 ± 3.6	0.16	0.07 ± 0.01	0.28	**36.9 ± 4.9**	**0.03**	0.02 ± 0.01	0.47
			Post	55.4 ± 3.8		0.07 ± 0.01		**41.0 ± 5.0**		0.02 ± 0.01	
		Internal(+)/external(-) rotation	Pre	19.0 ± 13.1	0.41	0.08 ± 0.01	0.43	-72.7 ± 11.1	0.16	0.00 ± 0.00	0.38
			Post	17.1 ± 10.8		0.08 ± 0.01		-67.6 ± 12.6		0.00 ± 0.00	
	ST		Pre	40.9 ± 2.4	0.36	0.06 ± 0.01	0.11	29.2 ± 2.8	0.33	0.03 ± 0.01	0.30

	Internal(+)/external(-) rotation	Post	41.3 ± 2.6		0.05 ± 0.01		29.6 ± 2.9		0.03 ± 0.01	
	Upward(-)/downward(+) rotation	Pre	**-18.3 ± 3.5**	**0.01**	0.06 ± 0.01	0.41	-31.4 ± 3.4	0.43	0.02 ± 0.01	0.14
		Post	**-21.1 ± 3.4**		0.06 ± 0.01		-31.6 ± 3.1		0.02 ± 0.01	
	Anterior(-)/posterior(+) tilt	Pre	11.9 ± 3.4	0.30	0.05 ± 0.01	0.13	-0.2 ± 2.9	0.13	0.04 ± 0.01	0.42
		Post	11.3 ± 3.3		0.04 ± 0.01		0.9 ± 2.9		0.04 ± 0.01	

Note: * time resolution was 0.003 s.

Figure 1: Positioning of the 33 reflective markers and the electromyography electrodes with (1) biceps, (2) serratus anterior, (3) major pectoralis, (4) upper trapezius, (5) anterior deltoid, (6) middle deltoid, (7) triceps, (8) posterior deltoid, (9) latissimus dorsi, (10) lower trapezius, (11) subscapularis, (12) infraspinatus, and (13) supraspinatus muscles.

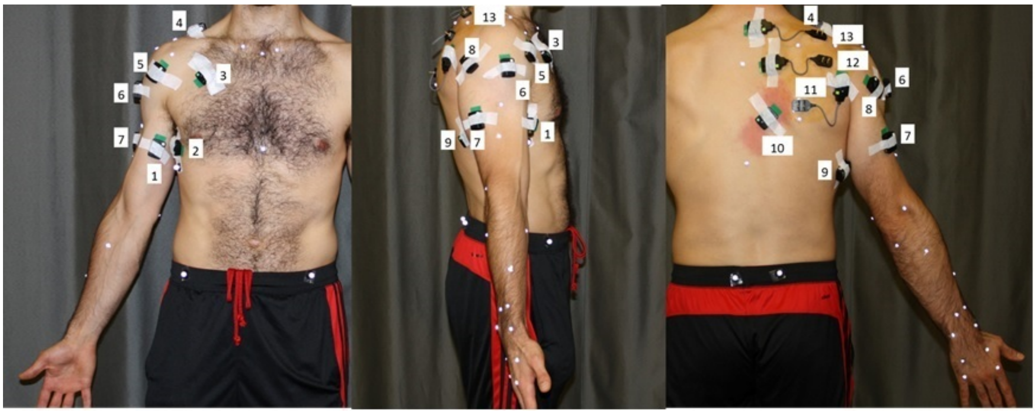


Figure 2: Humerothoracic (HT) and scapulothoracic (ST) joints before (in white) and after (in black) selective fatigue of the lower trapezius at the six serve events, with * for a significant difference at p<0.05

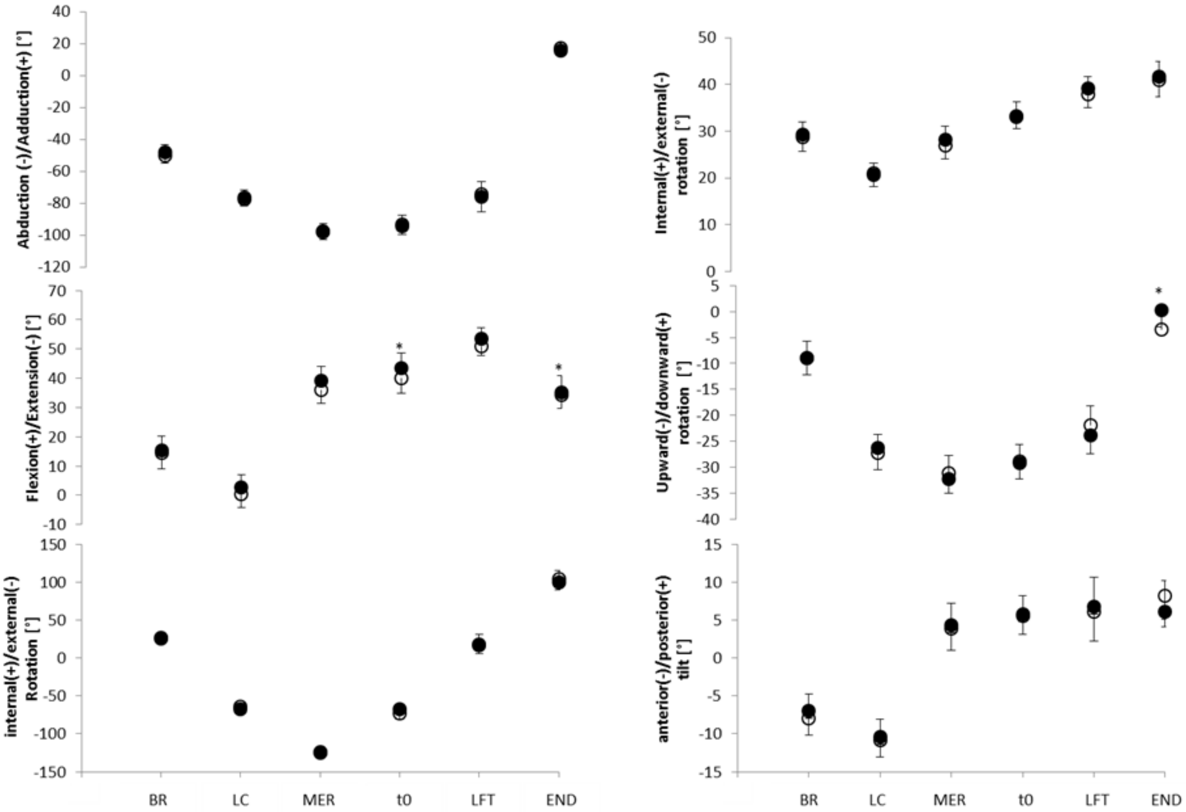
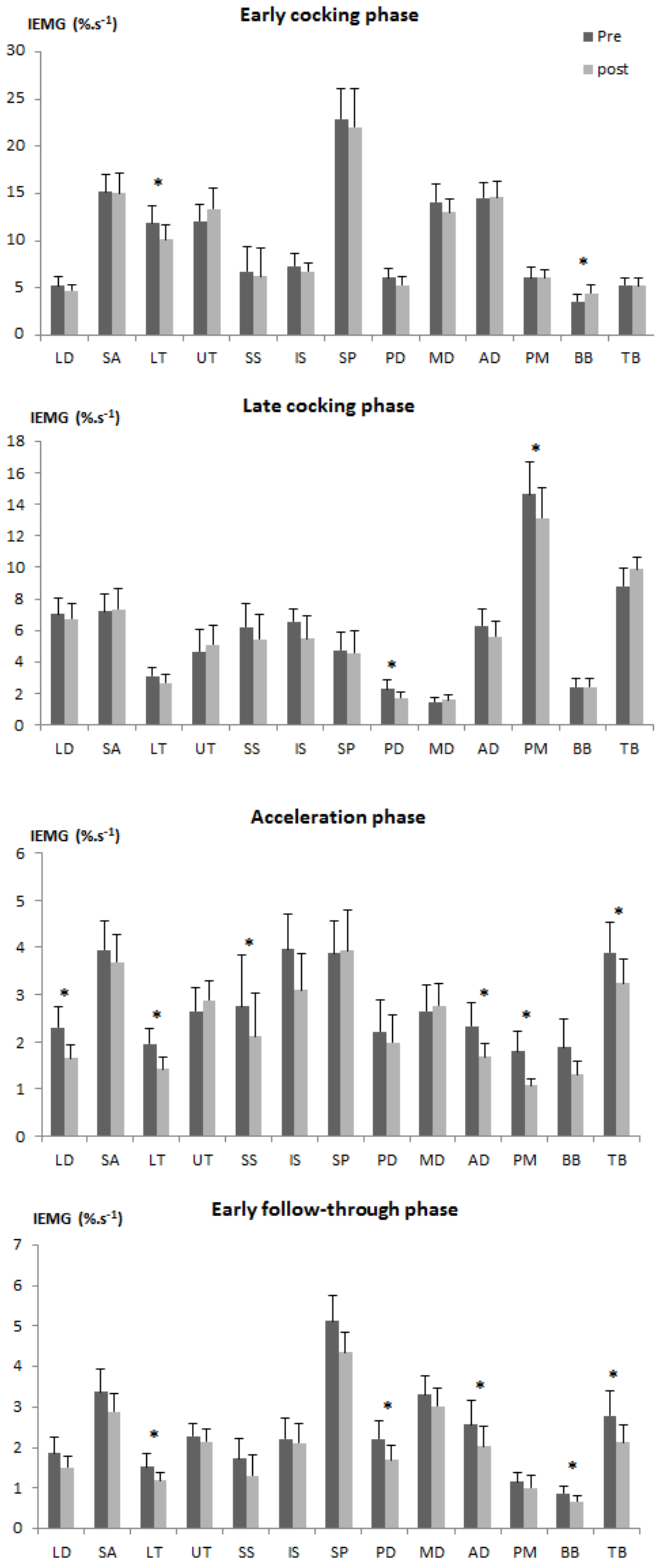
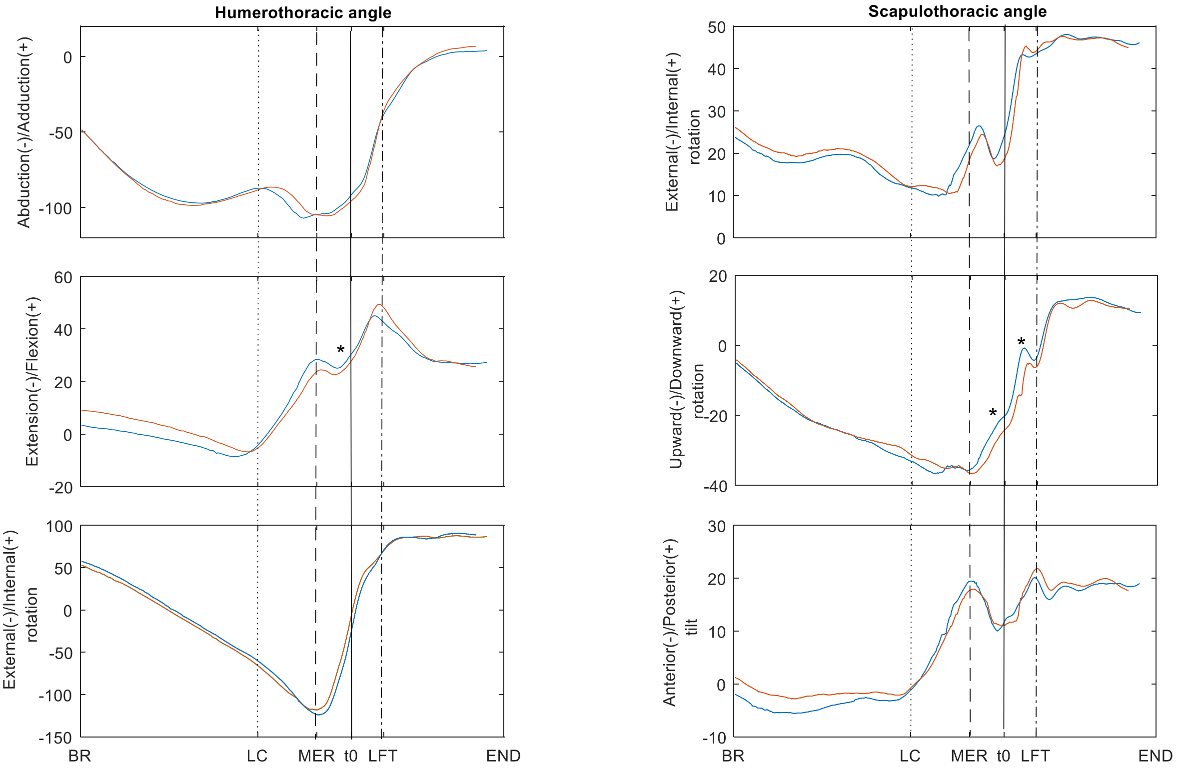


Figure 3 Mean ± standard error of the normalized integrated electromyography signal (in %) of the latissimus dorsi (LD), serratus anterior (SA), lower trapezius (LT), upper trapezius (UT), subscapularis (SS), infraspinatus (IP), supraspinatus (SP), posterior deltoid (PD), middle deltoid (MD), anterior deltoid (AD), major pectoralis (PM), biceps (BB), and triceps (TB) during the early (duration: 0.65 ± 0.03 s) and late cocking (0.22 ± 0.01 s), acceleration (0.09 ± 0.01 s) and early follow-through (0.08 ± 0.01 s) phases of the tennis serve before (dark grey) and after (light grey) LT selective fatigue, with * for significant difference before and after fatigue at $p < 0.05$


Early cocking phase
IEMG (%.s-1)
Pre
post
30
25
20
15
10
5
0
*
*
LD
SA
LT
UT
SS
IS
SP
PD
MD
AD
PM
BB
TB
Late cocking phase
IEMG (%.s-1)
18
16
14
12
10
8
6
4
2
0
*
*
LD
SA
LT
UT
SS
IS
SP
PD
MD
AD
PM
BB
TB
Acceleration phase
IEMG (%.s-1)
6
5
4
3
2
1
0
*
*
*
*
*
*
LD
SA
LT
UT
SS
IS
SP
PD
MD
AD
PM
BB
TB
Early follow-through phase
IEMG (%.s-1)
7
6
5
4
3
2
1
0
*
*
*
*
*
LD
SA
LT
UT
SS
IS
SP
PD
MD
AD
PM
BB
TB

List of Supplemental Digital Content

Supplemental Digital Content 1 (.docx): Humerothoracic and Scapulothoracic joint kinematics in degrees during the tennis serve before (blue) and after (red) selective fatigue of the lower trapezius for a representative player.



Supplemental Digital Content 2 (.docx): Mean ± standard error of the orientations (in degrees) of the humerothoracic (HT) and scapulothoracic (ST) joints before (pre) and after (post) selective fatigue of the lower trapezius at the six serve events, with bold for a significant difference at p<0.05

HT			Abduction(-) adduction(+)	Flexion(+) extension(-)	Internal(+) external(-) rotation
	Beginning of motion	Pre	-50.1 ± 4.6	14.6 ± 5.7	25.9 ± 6.9
		Post	-48.1 ± 4.8	15.2 ± 4.9	27.6 ± 8.5
		p	0.19	0.35	0.26
	Beginning of Late cocking	Pre	-77.3 ± 4.7	0.5 ± 4.6	-64.1 ± 4.9
		Post	-76.4 ± 4.8	2.6 ± 4.3	-66.8 ± 6.0
		p	0.28	0.12	0.18
	MER[1]	Pre	-97.5 ± 5.1	36.1 ± 4.8	-125.5 ± 6.8
		Post	-98.0 ± 5.1	39.3 ± 4.7	-123.9 ± 6.8
		p	0.39	0.08	0.24
	Impact	Pre	-93.1 ± 6.3	**39.9 ± 5.1**	-72.5 ± 10.9
		Post	-94.4 ± 6.8	**43.6 ± 5.0**	-67.6 ± 12.6
		p	0.38	**0.02**	0.17
	End of Early follow-through	Pre	-74.4 ± 11.1	50.8 ± 2.9	18.2 ± 12.9
		Post	-76.2 ± 9.9	53.6 ± 3.8	16.9 ± 10.8
		p	0.34	0.12	0.44
	End of motion	Pre	17.1 ± 3.8	34.3 ± 4.7	**105.1 ± 10.2**
		Post	15.9 ± 4.5	35.1 ± 5.7	**99.9 ± 9.6**
		p	0.30	0.36	**0.01**
ST			Internal(+) external(-) rotation	Upward(-) downward(+) rotation	Anterior(-) posterior(+) tilt
	Beginning of motion	Pre	28.8 ± 3.1	-8.9 ± 3.1	-10.8 ± 2.3
		Post	29.3 ± 2.7	-9.0 ± 3.2	-10.4 ± 2.4
		p	0.17	0.22	0.16
	Beginning of Late cocking	Pre	21.1 ± 2.9	-27.2 ± 3.5	-10.8 ± 2.3
		Post	20.6 ± 2.5	-26.3 ± 4.1	-10.4 ± 2.4
		p	0.24	0.22	0.35
	MER[1]	Pre	26.9 ± 2.9	-31.1 ± 3.4	3.9 ± 2.9
		Post	28.3 ± 2.7	-32.2 ± 2.7	4.4 ± 3.1
		p	0.19	0.22	0.33
	Impact	Pre	33.1 ± 2.6	-28.9 ± 3.3	5.6 ± 2.5
		Post	33.3 ± 3.0	-29.2 ± 3.0	5.8 ± 2.7
		p	0.45	0.35	0.47
	End of Early follow-through	Pre	37.9 ± 2.9	-21.8 ± 3.6	6.2 ± 3.9
		Post	39.1 ± 2.6	-23.8 ± 3.5	6.8 ± 3.5
		p	0.26	0.10	0.32
	End of motion	Pre	40.9 ± 3.5	**-3.4 ± 3.2**	8.2 ± 4.1
		Post	41.6 ± 3.3	**0.2 ± 3.2**	6.2 ± 4.6
		p	0.33	**0.01**	0.17

Note: [1]MER for humeral Maximum External Rotation

Supplemental Digital Content 3 (.docx): Envelopes of the electromyography signal normalized by maximum activation recorded during isometric maximum voluntary contractions (IMVC) for the anterior, middle, posterior deltoid, biceps, triceps, upper trapezius, pectoralis, infraspinatus, supraspinatus, subscapularis, latissimus dorsi, lower trapezius and serratus anterior muscles during the tennis serve, before (blue) and after (red) selective fatigue of the lower trapezius, for a representative player. The follow-through phase was colored in grey because the kinematics and the EMG signals were not analyzed during this phase. Note: BR stands for ball release, LC: beginning of the late cocking phase, MER: humeral maximum external rotation, t0: impact, LFT: beginning of the late follow-through phase