Relationship between muscle coordination and forehand drive velocity in tennis

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A B S T R A C T

This study aimed at investigating the relationship between trunk and upper limb muscle coordination and stroke velocity during tennis forehand drive. The electromyographic (EMG) activity of ten trunk and dominant upper limb muscles was recorded in 21 male tennis players while performing five series of ten crosscourt forehand drives. The forehand drive velocity ranged from 60% to 100% of individual maximal velocity. The onset, offset and activation level were calculated for each muscle and each player. The analysis of muscle activation order showed no modification in the recruitment pattern regardless of the velocity. However, the increased velocity resulted in earlier activation of the erector spinae, latissimus dorsi and triceps brachii muscles, as well as later deactivation of the erector spinae, biceps brachii and flexor carpi radialis muscles. Finally, a higher level of activation was observed with the velocity increase in the external oblique, latissimus dorsi, middle deltoid, biceps brachii and triceps brachii. These results might bring new knowledge for strength and tennis coaches to improve resistance training protocols in a performance and prophylactic perspective.

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1. Introduction

The forehand drive is a key stroke in modern tennis, as it is the most frequent groundstroke played during matches (Johnson and McHugh, 2006). Understanding its muscular coordination may therefore contribute to enhance tennis player performance.

Overall analysis of tennis forehand drive showed that the trunk and upper limb muscle activation sequence follows a proximal-to-distal sequence, which is independent of the tennis players’ age or skill-level (Rouffet et al., 2009). Both trunk and upper limb muscles temporal activation and deactivation are patterned by muscle groups, and remain unchanged with the increase in racket mass (Rogowski et al., 2009). In addition, the analysis of forehand drive per phase revealed that most dominant shoulder and upper limb muscles are activated at low level during the preparation phase, as compared to the high level during the acceleration phase and the medium one during the follow-through phase (Ryu et al., 1988; Morris et al., 1989). However, previous studies in tennis were made either at high (Rouffet et al., 2009) or sub-maximal speed (Rogowski et al., 2009), and never examined the muscle activity across a wide range of speeds. In other words, the influence of the tennis forehand drive velocity on trunk and upper limb muscular coordination remains unexplored.

The relationship between movement velocity and muscle coordination has been already investigated during single- and multi-joint movements. The velocity increase results in earlier onset of antagonist and/or agonist muscle activity in upper-limb (Lindinger et al., 2009; Marsden et al., 1983) and lower-limb (Neptune et al., 1997). This study also demonstrated that the increasing pedalling rate led to earlier deactivation of the main lower-limb muscles during the crank cycle. These findings can be transposed to the muscle activation onset in tennis movement, but not to the deactivation. Indeed, as hitting a ball needs more slowdown forces than cyclical movement, one may postulate that increasing the kinetic energy of the upper limb-racket system requires a greater slowdown work, and may thus lead to a delayed deactivation to protect joints from hyperextension. Concerning the activation level, either positive linear, quadratic, or lack of any relationship has been observed between activation level and movement velocity depending on the studied muscle (Neptune et al., 1997). When the velocity of movement increased, these relationships have been reported for the activation level of agonist and/or antagonist muscles during mono- and multi-joint upper limb movements performed at slow (Antony and Keir, 2010; Brindle et al., 2006; Marsden et al., 1983) and high velocities (Illyés and Kiss, 2005). These results seem to be applicable to tennis movements, but this needs to be demonstrated.

The present study aimed at observing changes in the trunk and upper limb muscle coordination as a function of stroke velocity during tennis forehand drive. Therefore, this muscle activity study on a continuum of velocity was intended to investigate issues of tennis training performed at sub-maximal velocity. It was...
hypothesized that the recruitment order would not be altered by movement velocity, while earlier activation, later deactivation and increased activation level of some muscles should be observed with increasing stroke velocity.

2. Material and methods

2.1. Participants

Twenty-one male tennis players (mean ± SD: age 23.3 ± 6.6 years; height 178.0 ± 7.7 cm; mass 71.8 ± 10.5 kg; tennis experience 14.2 ± 4.3 years; weekly training 6.8 ± 4.9 h; International Tennis Numbers = 2 and 3) volunteered to participate in this study, which was approved by the local Ethics Committee. The players signed an informed consent and none of them displayed injury on trunk and upper limbs during a six month-period before the study. Two players had an eastern forehand grip, 12 a semi-western, and seven a western one. All players were informed about experimental procedures and tasks, without receiving any information about the objectives of the study and the variables of interest.

2.2. Experimental procedure

After a 15 min standardized warm-up including rallies in the service area, baseline rallies, and series of crosscourt forehand drives, participants performed five series of ten successive cross-court forehand drives in the deuce diagonal, with their own racket and a minimum topspin. Experimental procedures were similar to those described by Rogowski et al. (2009). Players were instructed to play inside a defined zone of the tennis court, and to throw the ball in a target zone (4 × 4 m) which was located in the forehand side of the opposite court. They were asked to increase ball velocity between each series. A 3-min rest period was scheduled between each series to avoid fatigue. All trials took place in the same indoor gymnasium. Tennis balls were thrown with a frequency of 0.3 Hz by a ball machine (Airmatic 104, Pop-Lob, France) located in a standard way in the opposite side, behind the baseline of the tennis court. During the test session, the precision of the ball machine throw was about 40 cm. A radar gun (SR3600, Sports-radar, Homosassa, FL, USA) was placed behind the player to record the ball velocity after the forehand drive impact.

2.3. Electromyographic recordings

During forehand drives, muscle activity in ten selected muscles of the trunk and upper-limb was monitored by surface electromyography (EMG) using surface electrodes (EMG Triode Electrode, Nickel-plated brass, interelectrode distance = 2 cm, Thought Technology, Montreal, Canada). EMG signal was recorded from the skin surface in accordance with SENIAM recommendations (Hermens et al., 2000). Skin surface was shaved, abraded, and cleaned with alcohol swabs before placing the EMG surface electrodes. These were placed on the corresponding muscle belly aligned with the fiber direction, according to international standards (Hermens et al., 2000). Electrode placements were confirmed by analyzing EMG signal to noise ratio during isometric muscle contractions. These included manually resisted trunk extension (ES) and rotation (EO), shoulder extension (LE), horizontal adduction (PM), flexion (AD) and abduction (MD), resisted elbow flexion (BB) and extension (TB), as well as resisted wrist flexion (FCR) and extension (ECR). EMG signals were collected using the Flexcomp Infiniti system (Thought Technology, Montreal, Canada, 2048 Hz). Raw signals were filtered (Butterworth order 4, band pass 10–500 Hz) before calculating root mean square values (EMGrms, 25 ms). Quiet files were recorded to establish the baseline for each muscle. The onset and offset of the EMGrms burst were then detected using the method of the threshold based on mean baseline of quiet files ±3 SD, and described by Kibler et al. (2007). The impact time was determined on EMG recordings using the method described by Rogowski et al. (2009).

According to this study, the synchronised analysis of the EMG and kinematic data showed that peak maximal EMG occurred at the impact. The onset and offset of the EMGrms burst, as well as average EMGrms values, were calculated for the ten studied muscles, for all forehand drives performed by each participant.

2.4. Data analysis

Only successful trials, i.e., when the ball rebounded in the target zone, were considered for subsequent statistical analyses. For each player, maximal ball velocity (MBV) was determined among all successful strokes. Then, velocities were expressed in percentage of the MBV. The activation and deactivation order of the ten studied muscles were expressed in rank. The relationships between the EMG parameter (activation order, timing of muscle activation and deactivation, and activation level), and the increase in ball velocity were evaluated using the Linear Mixed-Effect Model (Pinheiro and Bates, 2000). The general form of the model was:

\[ Y = F[I + a.Lv + b.Qv] + R[I(i) + a(i).Lv + b(i).Qv] + E \]

With Y, one of the EMG parameters for one muscle, F indicating the fixed effect composed of the intercept (I), linear (Lv) and quadratic (Qv) variables of the ball velocity, and a and b, the associated coefficients. R denotes the random effect composed of the intercept (I(i)), linear (Lv) and quadratic (Qv) effect of the ball velocity for the participant i, a(i), b(i), the associated coefficients, and E, residual error term. Hence, this kind of model may lead to a fixed effect, which is representative of the whole studied sample, and/or a random effect, which refers to individual strategies. Then, for each EMG parameter in each muscle, nine models, corresponding to different degrees (0, 1, and 2) of the polynomial for the fixed and random effects, were computed. The choice of the best model was based on the Akaike’s Information Criterion (Akaike, 1974) and the significance of the fixed and/or random coefficient(s). Fixed effects were tested using conditional t-tests and random effects by Likelihood Ratio Tests, LRT (Pinheiro and Bates, 2000). Linear mixed-effect models were performed using the package “nlme” in the free software R (R.2.7.2., R Foundation for Statistical Computing, Vienna, Austria). For all statistical tests, coefficients were deemed significant at p < 0.05.

3. Results

3.1. Ball velocity

Ball velocities after forehand drive impact ranged from 15.6 to 38.9 ms⁻¹ in the whole sample of participants. The individual relative ball velocity varied from 23% to 50% of individual maximal ball velocity, with a mean variation of 40% for the whole of the sample.

3.2. Activation and deactivation order

No significant relationship was found between the EMGrms burst onset rank and increase in ball velocity, except for the ECR muscle. In the ECR activation rank model (Fig. 1), the significant

quadratic effect of the ball velocity in the fixed \( b = -18.9, \ t = -2.05, \ df = 474, \ p = 0.04 \) and random terms (LRT = 3.90, df = 7, \( p = 0.05 \)) showed that the ECR activation rank increased until about 80% of the relative ball velocity and decreased until MBV; these variations were more or less slight according to the participant considered.

No significant relationship was found between the EMGrms burst offset rank and increase in ball velocity, except for the FCR muscle (Fig. 2). The quadratic effect of the ball velocity in the fixed term of the FCR deactivation rank model \( b = 13.25, \ t = 2.61, \ df = 474, \ p = 0.009 \) showed that the FCR deactivation rank decreased slightly until 75% of the relative ball velocity, and then increased until MBV in all players.

### 3.3. EMG burst onset

No significant relationship was observed between the ball velocity and EMGrms burst onset for OE, AD, MD, BB, and FCR muscles. Significant relationships were observed for the ES, LD (Fig. 3), PM, TB, and ECR muscles. The significant ball-velocity linear-effect in the fixed term of the ES EMGrms burst onset model \( a = -0.17, \ t = -2.43, \ df = 475, \ p = 0.02 \) pointed out that all players activated earlier before impact their ES muscle when the ball velocity increased. As shown by Fig. 3, for the LD EMGrms burst onset model, the linear effect of the ball velocity in the fixed term \( a = -0.42, \ t = -2.96, \ df = 475, \ p = 0.003 \) indicated a negative linear relationship between the increase in ball velocity and the LD burst onset. However, the presence of the ball-velocity linear-effect in the random term of the model (LRT = 10.23, df = 5, \( p = 0.001 \)) revealed that 18 players recruited earlier before impact their LD muscle, while the three others recruited it nearer the impact when the ball velocity increased.

Considering the PM EMGrms burst onset model, the ball-velocity linear effect in the random term (LRT = 11.59, df = 4, \( p < 0.001 \)) yielded an individual relationship between PM activation and ball velocity. Indeed, three groups of players were distinguished; the first group displayed a delay in the PM EMGrms burst onset along with the velocity, the second one showed an inverse relationship, while the third displayed a constant PM EMGrms burst onset despite the ball velocity increased. Considering the TB burst onset model, the ball-velocity quadratic-effect in the fixed
term ($b = -2.91, t = -3.31, df = 474, p = 0.001$) revealed that, in all players, the TB muscle was recruited closer to the ball impact for the low increase in ball velocity, while the TB muscle was recruited earlier before impact when the ball velocity reached its maximal values. However, the presence of ball-velocity linear-effect in the random term ($LRT = 22.06, df = 5, p < 0.0001$) indicated that the inversion in the relationship slope, corresponding to the point for which the TB burst onset was closest to the ball impact, was related to individual behaviour and ranged between 50% and 70% of the MBV. Finally, the ball-velocity quadratic effect in the random term of the ECR burst onset model ($LRT = 4.66, df = 5, p = 0.03$) showed that 12 players recruited their ECR muscle nearer to the ball impact when the ball velocity increased from 50% to 70% of the maximal velocity, and earlier before impact when the ball velocity rose up to 100%. The inverse relationship was observed in the other nine players.

### 3.4. EMG burst offset

No significant relationship was observed between the ball velocity and EMGrms burst offset in EO, LD, PM, and ECR muscles. Significant relationships were observed for the ES, AD, MD, BB (Fig. 4), TB, and FCR muscles. The significant ball-velocity linear effect in the fixed term of the ES EMG burst offset model ($a = 0.09, t = 2.73, df = 475, p = 0.007$) pointed out that all players deactivated their ES muscle later, when the ball velocity increased. For the BB EMG burst offset model (Fig. 4), the linear effect of the ball velocity in the fixed term ($a = 0.28, t = 4.02, df = 475, p < 0.001$) showed that, in all players, the BB muscle was deactivated later after impact when the ball velocity increased.

Considering the FCR EMGrms burst offset model, the ball-velocity quadratic effect in the fixed term ($b = 0.83, t = 1.86, df = 474, p = 0.06$) indicated in all players that the FCR muscle stopped its activation later after impact from about 60–70% of the MBV with the ball velocity increase. The ball-velocity quadratic effect in the random term of the model in AD ($LRT = 22.06, df = 5, p < 0.0001$), MD ($LRT = 10.35, df = 5, p = 0.001$) and TB ($LRT = 23.04, df = 5, p < 0.0001$) muscles revealed an individual relationship between muscle deactivation and ball velocity. Some players deactivated these muscles later after impact for MBV ranged from 50% to 80%, and closer to the impact for higher velocities. The inverse relationship was observed in the other players.

### 3.5. Activation level

No significant relationship was observed between the ball velocity and EMGrms values in ES, PM, AD, FCR, and ECR muscles. Significant relationships were observed for the EO, LD, MD, BB, and TB (Fig. 5). The ball-velocity linear effect in the fixed term of the EO EMGrms values model ($a = 109.9, t = 4.11, df = 475, p = 0.0001$) indicated a linear relationship between the EO EMGrms values and the increase in ball velocity. However, the presence of the ball-velocity linear-effect in the random term of the model ($LRT = 10.54, df = 5, p = 0.001$) pointed out that this relationship was positive in 20 players and negative in one. For the LD EMGrms values model, the linear effect of the ball velocity in the fixed term ($a = 165.2, t = 4.00, df = 475, p = 0.001$) indicated a linear relationship between the LD EMGrms values and the ball velocity increase in most players. Nevertheless, the ball-velocity quadratic effect in the random term of the model ($LRT = 4.63, df = 6, p = 0.03$) revealed individual shape of the relationship. Half of players displayed a constant LD level until about 70% of MBV, then an increase for higher velocity. The inverse relationship was observed in the other half, i.e., increase in LD level then constant level. The ball-velocity linear effect in the fixed term of the MD EMGrms values model ($a = 96.7, t = 3.85, df = 475, p < 0.001$) indicated a linear relationship between the MD EMGrms values and the ball velocity increase in all players. But the presence of the ball-velocity linear-effect in the random term of the model ($LRT = 13.88, df = 5, p < 0.001$) highlighted that the MD activation level increased in 17 players and was constant in four players with the velocity increase. Considering the BB EMGrms values model, the ball-velocity linear effect in the fixed term ($a = 494.3, t = 1.94, df = 474, p = 0.05$) and the random term of the model ($LRT = 9.51, df = 6, p = 0.002$) showed that...
18 players gradually activated their BB muscle along with the increase in ball velocity, while three participants displayed a inverse relationship. For the TB burst EMGrms values model (Fig. 5), the linear effect of the ball velocity in the fixed term ($\alpha = 133.1$, $t = 2.98$, $df = 475$, $p = 0.001$) and in the random term of the model (LRT = 34.93, df = 5, $p < 0.0001$) indicated that the TB activation level increased linearly in 19 participants and decreased slightly in two participants with the increase in ball velocity.

4. Discussion

The present study focused on the influence of the forehand drive velocity on the activation order, timing and level of trunk and upper-limb muscles. The main results showed that the muscle recruitment order was not substantially modified by changes in movement velocity, which had a significant influence on the EMG rms burst activation/deactivation timing, and activation level of some muscles.

The participants performed crosscourt forehand drives with a mean variation of 40% in ball velocity. According to Elliott et al. (1989), the ball velocity following the impact is related to the racket velocity at the time of impact in similar playing conditions. Considering the low ball velocity variability of the machine (less than 0.83 m s$^{-1}$), ball velocity variations after impact might be interpreted as a modification in players’ movement velocity. In addition, the analysis of the movement velocity effect on both the activation and deactivation sequences of trunk and upper limb muscles showed stability in muscle recruitment patterns. Significant models were found only for the ECR activation and FCR deactivation, but conducted to slight variations in both muscle ranks (Figs. 1 and 2). As the trunk and upper limb muscle activation was patterned into two groups, whereas the muscle deactivation sequence was performed into three groups (Rogowski et al., 2009), the alterations in activation/deactivation order are small enough to be considered as negligible (Figs. 1 and 2). Consequently, the increase in stroke velocity elicited negligible modifications in the muscle recruitment order, suggesting that the function of the studied muscles was maintained with increasing movement velocity in expert tennis players.

Increasing stroke velocity influenced the onset activation of five (ES, LD, TB, PM, and ECR) out of 10 muscles. Accordingly, a significant earlier activation onset before impact was observed in ES, LD, and TB along with the increase in velocity. In line with the “activation dynamics hypothesis” (Neptune et al., 1997), such muscular adaptation might contribute to ensure that these muscles produce their peak force adequately and at the right time during movements with higher velocity. In tennis, the stroke velocity increase might lead to a reduction of the total movement time, which required an earlier activation for some muscles, in order to reach an optimal head racket velocity at the impact. The velocity is likely to differentially influence the temporal patterns of muscles, with regard to their functional role during the forehand drive. The increase in velocity elicits stronger loads in trunk muscles and spine, which may explain the earlier activation of ES muscle. During powerful strokes, the stability of lower trunk remains essential to initiate the stroke and prevent low back pain (Reid et al., 2003). LD and TB muscles are activated before impact to provide greater stiffness of the muscle–tendon complex to stabilize upper limb joints at impact (Rouffet et al., 2009) and to anticipate the racket/upper limb slowdown during the follow-through phase (Rogowski et al., 2011). Their earlier activation observed for the velocities near MBV might be explained by a more vigorous backswing to stretch the front-body muscles, and then the effects of the stretch–shortening cycle on these front-body muscles would be optimized to achieve high stroke velocity (Elliott, 2003). PM and ECR activation onsets were modelled only by linear or quadratic effects in the random term of the model, hence suggesting that players applied individual strategies to increase their movement velocity, probably in relation with their technical and morphological characteristics. For example, the kind of forehand preparation and the height of the player may influence the capacity of adaptation to the temporal constrain. This inter-individual variability was also observed in a recent study in cycling (McGhie and Ettema, 2011).

The analysis of activation offset revealed that increasing the stroke velocity resulted in delayed deactivation of ES, BB, and FCR muscles. As expected, these results differed from those reported in cyclical movement (Neptune et al., 1997). These differences could be explained by the need for greater slowdown work in rapid tennis movements. Indeed, higher velocities lead to increase the kinetic energy, which needs to be dissipated at the end of the movement. Higher kinetic energy amount is essential while increasing ball velocity, but requires a greater stabilisation and slowdown time to allow upper limb joint protection and effective movement control. The ES muscle was contracted longer after impact, due to its slowdown function in trunk rotation, stopping the rotation on the longitudinal axis and keeping the body from rotating away from the opponent (Reid et al., 2003). The later deactivation of the BB muscle after the impact was consistent with the internal arm rotation and elbow flexion observed during the follow-through phase. As well, the delayed deactivation of the FCR might be explained by the need for gripping firmly the racket during the slowdown phase. Various relationships were observed between velocity and EMG burst offset in DA, DM, and TB muscles depending on the players. Some players activated later after the impact while other showed a stable deactivation, or closer to the impact. In the former case, the chief of both deltoid and TB muscles would contribute to stabilize and slow down the upper limb/racket system, while in other players the slowdown may be based on a different muscle strategy.

During the forehand drive, the activation level of five muscles (EO, LD, MD, BB, and TB) significantly increased along with the stroke velocity. The increase of agonist muscles contribution would promote a higher acceleration of the upper limb/racket system in order to improve the ball velocity after impact. Because EO mainly contributes to rotate the trunk during the acceleration phase, a greater level of activation might support the critical role of the trunk in increasing the velocity of the forehand drive (Elliott, 2003). As reported in the overhead throwing (Illýés and Kiss, 2005), a greater MD activation level was observed along with movement velocity during tennis forehand drive. This increase could allow a better control of the upper limb adduction/abduction, with regard with the downward/upward racket trajectory during the shot forward swing (Rogowski et al., 2011) and proper stability of the shoulder joint to be ensured. Considering the arm segment, the BB muscle activity increased along with the velocity that could be explained by the plural functions of the internal rotator, the elbow and shoulder flexor, and the forearm stabilisation during the forehand drive (Morris et al., 1989). As far as the antagonist muscles are concerned, the greater TB and LD activation level along with the increase in velocity might be explained by the co-activation phenomenon during the acceleration phase and by the slowdown of upper limb/racket system after impact. In contributing to stiffen and stabilize the joint, the co-activation would guaranty joint integrity. This activation pattern slowdown the movements (Bazzucchi et al., 2008) before reaching maximal amplitudes that may injure joint structures. In addition, no relationship was found between stroke velocity and the activation level of ECR and FCR muscles. As players were instructed to play without spin, forearm muscles would be recruited only to stabilize the wrist as a rigid extension of the racket (Morris et al., 1989) and grip firmly the racket handle, whatever the shot velocity.

The main limitation concerned the lack of a synchronized kinematic analysis. Indeed, this kind of analysis would allow the relationships between muscle coordination and velocity in relation to each
forehand drive phase to be identified. However, this study provides new insight regarding the muscular coordination during tennis forehand drive and promotes new training objectives for strength and tennis coaches in performance and prophylactic perspectives.

5. Conclusion

This study pointed out that the EMG burst timing and the amplitude of muscle activity was adjusted to the velocity of the stroke. Some parameters of muscular coordination were not altered by the increase of the forehand drive velocity, while others were adjusted to preserve the efficiency of the stroke. Increasing velocity had very slight effects on the order of muscle group activation and deactivation that confirmed the interest of technical training at sub-maximal speed. Indeed, this kind of specific training does not stand in the way of development of effective muscle recruitment pattern. Otherwise, ES, LD and TB muscles were activated earlier before the impact in order to anticipate the stroke velocity increase, and to adapt to the decrease of movement time. Additionally, delayed deactivations were also observed in ES, BB, and FCR muscles to contribute more extensively to the slowdown of the racket. The activation level increased in half of the studied muscles, which played a critical role to allow and compensate for the increase in stroke velocity. Interestingly, the statistical method used in this study underlined general muscle adaptations as well as different individual strategies. Altogether, the sensitivity of ES, LD, BB, and TB muscles support the interest of specific training for these muscles in tennis players.

6. Conflict of interest

The authors have no conflict of interest to declare.

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References


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