



# Neuromuscular fatigue in tennis: Mind over muscle?

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## ABSTRACT

To successfully endure tournament competition, tennis players must accelerate, decelerate, change direction, move quickly, maintain balance and repeatedly generate optimum stroke production during several hours. This review paper outlines how fatigue manifests during prolonged match play and discusses the neural and muscular factors that may compromise tennis skill and performance. Scientific findings are presented as the basis for practical recommendations to help players to better resist fatigue during intense and extended matches.

**Key words:** Neuromuscular function, Tiredness, Performance alteration.

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## INTRODUCTION

Muscle fatigue is often quantified as a reduction in the maximal force that a muscle can exert, but its aetiology is complex, especially under conditions of high-intensity intermittent exercise involving the whole body such as playing tennis. The inability to produce and/ or maintain the required force, which has the potential to alter on- court movement and stroke production, can be attributed to several potential mechanisms occurring within cortical regions to muscular contractile elements. However, the neuromuscular adjustments to fatigue in tennis have received little attention until recently.

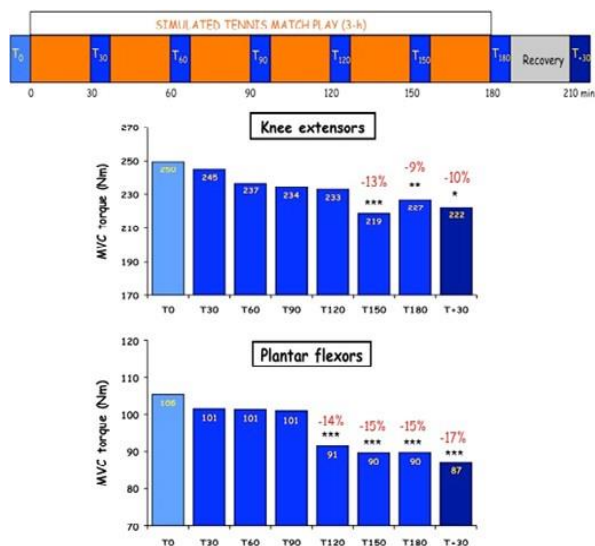
### Manifestation of fatigue - Activity-specific protocols

During the last decades, several studies have provided scientific evidences to support the observations made by coaches that fatigue impairs performance as shown by mis-timed shots (i.e. power and precision) and altered on-court movements (i.e. speed, positioning to the ball). Fatigue-inducing protocols have been developed to determine the effects of fatigue on stroke production in specific conditions close to competition (Davey et al. 2002; Hornery et al. 2007a; Vergauwen et al. 1998). These studies have reported conflicting results regarding change in stroke velocity and accuracy. For example, Davey et al. (2002) observed a large decrease in the accuracy of shots played (69% and 30% for groundstroke and serve, respectively) during an exhausting tennis simulation test, whereas conversely the accuracy was only slightly reduced (groundstroke) or unchanged (serve) after a two hour on-court strenuous training session (Vergauwen et al. 1998). However, the lack of sensitivity and the large variability in selected variables limit considerably the generalisation of these findings. Another shortcoming is that fatigue levels experienced by players failed to reflect those recorded in match play (i.e. format of the

protocol, using a ball machine to administer pre- and post-fatigue on-court skill assessment (Davey et al. 2002). For example, it is questionable how an intermittent test leading to volitional exhaustion in 35 minutes could induce a comparable degree of physiological strain as in actual competitions.

### Matchplay

To overcome these limitations, several investigators have evaluated the effects of fatigue on performance during simulated match conditions. For example, Mitchell et al. (1992) reported that fatigue after a three hour tennis match is manifested by a decreased velocity of the serve and longer time to complete tennis pattern shuttle- runs. Girard et al. (2006) recently reported progressive reductions in maximal voluntary strength (10-13% in quadriceps) and leg stiffness highly correlated with increases in perceived exertion and muscle soreness throughout a three hour tennis match, whereas explosive strength was maintained and decreased only after the exercise. Immediately after the exercise, similar (-15%) in strength loss was observed for plantar flexors using the same match protocol (Girard et al. 2011). However, the time course of these adaptations differs between knee extensors (progressive) and plantar flexors (biphasic pattern with a marked loss after 90 min) muscles (Figure 1).



**Figure 1.** Maximal isometric voluntary contraction (MVC) torque of the knee extensor and plantar flexor muscles before (T0), during (T30: 30th min; T60: 60th minute; T90: 90th min; T120: 120th min; T150: 150th min), immediately after (T180), and 30 minutes after (T+30) a three hour tennis match (see Girard et al. 2008 and Girard et al. 2011).

## FACTORS RESPONSIBLE FOR FATIGUE

### Defining and quantifying neuromuscular fatigue

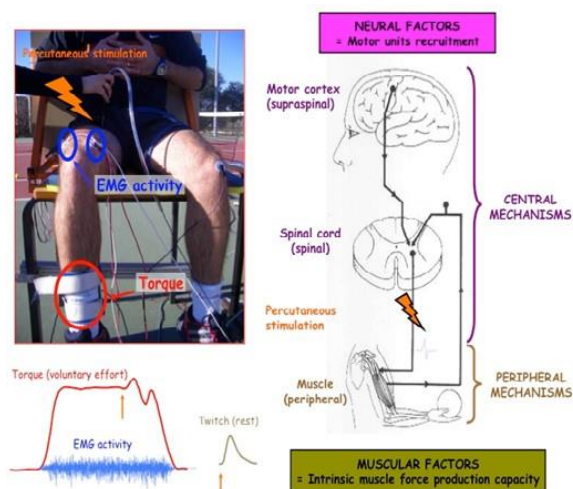
Fatigue is a complex phenomenon, which aetiology depends on the characteristics of the task performed (task dependency principle). The inability to produce/maintain the required force can be attributed to several potential mechanisms from cortical region (neural factors) to contractile elements (muscular factors). Each of these stages is a possible limiting factor for force production and eventually on-court performance. The traditional approach used to identify the causes of muscle fatigue has been to distinguish 'central' (i.e. an exercise-induced decrease of muscle force due to a reduction in recruitment) and 'peripheral' (i.e. decrease in force due to a decrease in muscle fibre contractility induced predominantly by metabolic events within the muscle) mechanisms. This can be performed by applying an electrical stimulus to the peripheral nerve (tibial or femoral nerve) and analysing changes in electromyograms (EMG), voluntary and evoked forces (Figure 2). By using this approach, it has been possible to show that both nervous (impaired muscle activation) and contractile (muscle contractility) mechanisms contribute to the alteration in neuromuscular function as match progresses after three hour tennis (Girard et al. 2008; Girard et al. 2011).

### Central vs. peripheral fatigue mechanisms

Reduced central activation has been linked to changes in neurotransmitters metabolism or in response to afferent sensory feedbacks (i.e. inhibition of motoneuron excitability), possibly due to changes in metabolic and/or mechanical properties within the muscle (Gandevia, 2001). Practically, a suboptimal neural drive to the muscle might impair the rate of force development (i.e. ability to reach higher levels of muscle force within the initial phase of muscle contraction) believed to

be a crucial determinant of fast limb movements. Several factors including decreased phosphocreatine availability, increased muscle acidity, decreased muscle carbohydrate (glycogen) stores or a low blood glucose level have been suggested as causes of fatigue at the muscle level (Fitts, 1994). Fatigue observed temporarily after the periods of exhausting exercise as long or consecutive intense rallies is probably linked directly to disturbances in muscle ion homeostasis, impaired excitation of the sarcolemma (increase in extra-cellular potassium) or accumulation of metabolites (i.e. phosphocreatine, lactate).

**Figure 2.** Reliable assessment of muscle fatigue can be studied using the neuro-stimulation technique. By comparing the twitch superimposed to a maximal voluntary contraction and the twitch evoked on the relaxed muscle (i.e. femoral nerve percutaneous, supra-maximal stimulation), the twitch interpolation technique in conjunction with surface



electromyography (EMG) can be a reliable non-invasive technique to characterise muscle activation (neural factors). In examining modification of resting twitch it is possible to clarify whether a loss of muscle contractile properties (muscular factors) can also contribute to the impaired neuromuscular function (see Girard and Millet, 2008).

### Additional homeostatic perturbations

Hot environments and dehydration are worsening fatigue factors whereas carbohydrate supplementation before or during competitions would eventually contribute to delay fatigue (Hornery et al. 2007b). When tennis is played in the heat, it is likely that the attainment of a high core temperature (> 38.5°C) can impair central nervous system function, resulting in a reduced level of central cognitive or neural drive to the muscle (e.g. wrong tactical choices, strength losses). Leg stiffness regulation is believed to be another important determinant in the optimisation of locomotor performance (sprint running). The progressive reduction in leg stiffness throughout three hour tennis match play suggests that some of the observed fatigue-induced decrements in on-court tennis movements might be partially explained by alterations in the mechano-characteristics of the muscle-tendon complex (Girard et al. 2006). Moreover, Hornery et al. (2007c) reported a significant increase in circulating creatine kinase, which appears to be indicative of acute muscle damage. Thus, muscle damage invoked by the numerous eccentric contractions associated

with on-court-movements might represent an important factor underlying the fatigue observed during tennis.



## CONCLUSION

Fatigue impairs tennis performance, and can be manifested by mistimed shots, altered on-court movements, wrong cognitive (i.e. tactical) choices. The aetiology of muscle fatigue is a complex phenomenon (i.e. distinction between temporary fatigue and fatigue occurring in the final stage of a competition) that might involve impairment in both neural (suboptimal muscle activation) and contractile (accumulation of metabolites) processes.

## Practical applications

- Neural factors are largely responsible for the training-induced strength gains after electromyostimulation and/or resistance programs. By respecting the movement patterns and specific demands of the game (Bennie and Hrysomallis, 2005), such training modalities could be efficient in improving tennis-related variables and in delaying 'central' fatigue.
- Tennis players are required to repeatedly generate large amounts of power during explosive stroke actions and fast on-court movements. Therefore, improving the structural (e.g. hypertrophic adaptations) and biochemical processes (e.g. regulation of plasma potassium, twitch contractile properties) at the muscle level is expected to offer an advantage for fatigue resistance (Behm and St Pierre, 1998).
- The use of pre-fatiguing situation (e.g. bouncing, in-depth jumping, plyometric exercises, medicine-ball) followed by on-court high-intensity interval-training would be efficient for reducing the observed impairment in muscle contractility (excitation-contraction uncoupling).

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