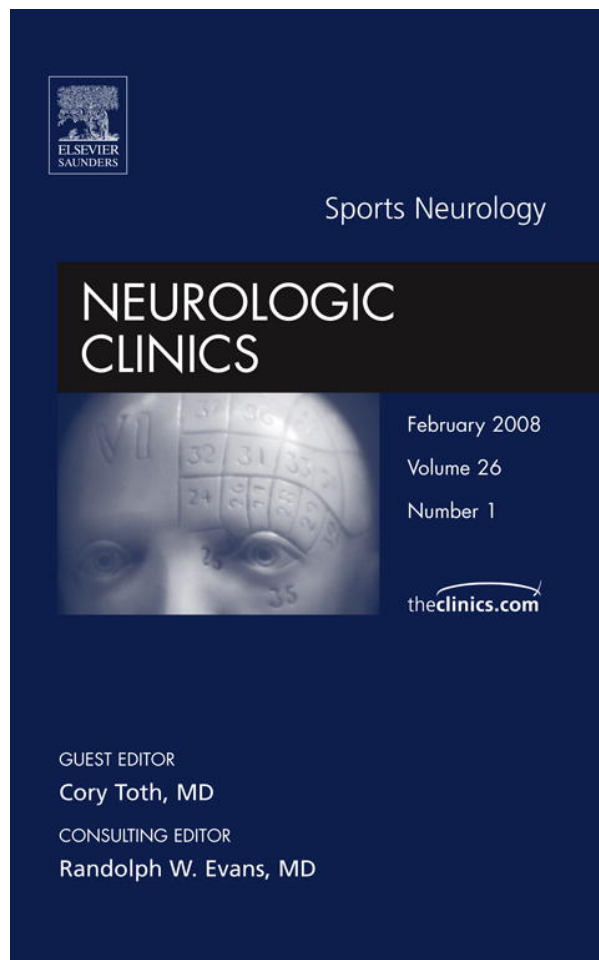


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## Neuromuscular Fatigue in Racquet Sports

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This article describes the physiologic and neural mechanisms that cause neuromuscular fatigue in racquet sports; table tennis, tennis, squash and badminton. In these intermittent and dual activities, performance may be limited as a match progresses because of a reduced central activation, linked to changes in neurotransmitters concentration or in response to afferent sensory feedback. Alternatively, modulation of spinal loop properties may occur because of changes in metabolic or mechanical properties within the muscle. Finally, increased fatigue manifested by mistimed strokes, lower speed, and altered on-court movements may be caused by ionic disturbances and impairments in excitation-contraction coupling properties. These alterations in neuromuscular function contribute to decrease in racquet sports performance observed under fatigue.

### Technical characteristics and physiologic demands of racquet sports

Badminton, squash, and table tennis are among the most popular racquet sports, even if tennis is probably the most widely practiced. Before discussing the potential mechanisms that limit performance, the technical characteristics of these sports and the physiologic strain imposed on the players have to be described. In racquet sports, the activity pattern is intermittent; that is, characterized by repetitions of fast starts and stops and alternating brief periods of exercise at maximal or near maximal intensity, and longer periods of lower intensity [1,2]. Performance arises from complex interaction between technical, tactical, physiologic, and psychologic skills that often

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have to be sustained in hostile environmental conditions. To successfully endure tournament competition, racquet sports players must accelerate, decelerate, change direction, move quickly, maintain balance, and repeatedly generate optimum stroke production. This physiologic strain is influenced by hydration and nutritional status [1–4]. The duration of competition in racquet sports can vary from 30 to 60 minutes in squash and badminton to more than 5 hours in tennis, but average durations of 30 to 90 minutes are common in all racquet sports [2]. In most high-level matches, the rallies last on average between 2 and 15 seconds and the work-to-rest ratio varies between 1.1 and 1.5. Nevertheless, match activity varies widely across racquet sports. In tennis, the mean durations of rally and resting periods are approximately 4 to 8 seconds and 15 to 20 seconds, respectively [1]. The average effective playing time ranges usually between 10% and 30% of the game duration. In squash, the point duration is longer (10–20 seconds) and the resting period is shorter (7–8 seconds); so the effective playing time is 50% to 70% of game duration [5,6]. A summary of the results of several notational analyses of racquet sports is presented in Table 1 [1,2,5–7].

Research on the physiologic demands of racquet sports indicate that these sports place considerable demands on both aerobic and anaerobic pathways, but their relative contributions are controversial [1,2,5]. Estimates of exercise intensity (oxygen uptake, heart rate, or blood lactate concentrations) are described in Table 2 [1,2,5–7]. In racquet sports, the physiologic demand may vary to a large extent, and is influenced by a multitude of factors such as the style of the player, gender, the level and style of the opponent, the surface, the equipment (ie, missile and racquet characteristics), and environmental factors (ie, temperature and humidity) [1,2].

Cardiorespiratory fitness has been traditionally measured by maximal oxygen uptake. Racquet sports players possess moderate (table tennis:  $\sim 50 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ ; tennis:  $\sim 50\text{--}55 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ ) to high (badminton:  $\sim 55 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ ; squash:  $> 60 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ ) aerobic capabilities, similar to those of team-sports players but obviously lower than those of endurance athletes [2]. However, an elevated cardiovascular endurance is a prerequisite attribute to compete at the elite level (ie, fast recovery between points) in all racquet sports but table tennis. Flexibility, muscular endurance, strength, and power, as well as more specific factors such as

Table 1  
Notational analysis in racquet sports

Activity	PD (s)	RD (s)	W:R ratio	EPT (%)	References
Table tennis	3–4	8	1:3	35	[2]
Tennis	5–12	15–20	1:4	20–30	[1–3]
Badminton	4–8	10–16	1:2	40–50	[2,7]
Squash	15–20	8–10	1:1	50–70	[2,5,6]

*Abbreviations:* EPT, Effective playing time; PD, point duration; RD, recovery duration; W:R ratio, work-to-rest ratio.

Table 2  
Typical physiologic strain experienced in racquet sports

Activity	%VO <sub>2max</sub>	%HR <sub>max</sub>	[La] (mmol.l <sup>-1</sup> )	References
Table tennis	–	80–85	2	[2]
Tennis	60–80	70–85	2–4	[1–3]
Badminton	75–85	75–90	3–6	[2,7]
Squash	80–85	85–92	6–10	[2,5,6]

*Abbreviations:* %HR<sub>max</sub>, percentage of maximal heart rate; [La], lactate concentration; %VO<sub>2max</sub>, percentage of maximal oxygen uptake.

acceleration, agility, balance, and response time have also been described as important physical factors in racquet sports. At the elite level, effective stroke production requires rapid on-court movements, explosive force, and the capacity to generate explosive bursts of power. As a consequence, success in the decisive rallies at the end of a long and demanding match can be determined by the ability to repeatedly perform sprints and generate effective powerful strokes. Therefore an important issue is to describe and understand how fatigue limits performance in racquet sports.

### Manifestation of fatigue

A close inspection of the literature reveals that the effects of fatigue on performance in squash, badminton, or table tennis players have received little documentation [2]. During the last decades, several studies have provided scientific evidence to support the observations made by coaches that fatigue impairs performance, as shown by mistimed shots (ie, power and precision) and altered on-court movements (ie, speed, positioning to the ball). In tennis, physiologic perturbations during training and simulated matches and their combined effects on a number of performance variables have been reviewed [3]. Fatigue-inducing protocols have been recently developed to determine the effects of fatigue on stroke production in specific conditions close to those of competition [8,9]. These studies have reported conflicting results regarding change in stroke velocity and accuracy. For example, Davey and colleagues [8] observed a large decrease in the accuracy of shots played (~69% and 30% for ground stroke and serve, respectively) during an exhausting tennis simulation test, whereas accuracy was only slightly reduced (ground stroke) or unchanged (serve) after a 2-hour, on-court strenuous training session [9]. However, the lack of sensitivity and the large variability in selected variables limit considerably the generalization of these findings. Another shortcoming is that fatigue levels experienced by players failed to reflect those recorded in match play (ie, format of the protocol, using a ball machine to administer pre- and post-fatigue on-court skill assessment; Ref. [8]). For example, it is questionable how an intermittent test leading to volitional exhaustion in approximately 35 minutes could induce a degree of physiologic strain comparable to that of actual competition.

These limitations have guided investigators to evaluate the effects of fatigue on performance during simulated match conditions. For example, Mitchell and colleagues [10] have reported that fatigue after a 3-hour tennis match is manifested by a decreased velocity of the serve and longer time to complete tennis pattern shuttle-runs. Using a similar approach, Girard and colleagues [11] recently reported progressive reductions in maximal voluntary strength ( $\sim 10\%$ – $13\%$  in quadriceps) and leg stiffness highly correlated with increases in perceived exertion and muscle soreness throughout a 3-hour tennis match, whereas explosive strength was maintained and decreased only after the exercise. As recently outlined by Hornery and colleagues [3], these studies are also limited by a lack of knowledge regarding mechanisms underlying potential fatigue. Finally, it is important to keep in mind that several factors, including the environmental conditions (hot/humid), dehydration status, and intake of carbohydrate and caffeine, have been shown to affect racquet sports performance and therefore the extent of muscle fatigue experienced by players [3,4].

### **Defining and quantifying neuromuscular fatigue**

Fatigue is a complex phenomenon and has been a major research topic for exercise scientists for the last half century. Neuromuscular fatigue refers to a transient reduction in the maximal force capacity of the muscle, and is measured objectively by an acute reduction of performance during exercise [12]. The inability to produce and maintain the required force can be attributed to several potential mechanisms occurring within cortical regions to muscular contractile elements, with each of these stages as a possible limiting factor [13]. Traditionally, studies of neuromuscular fatigue during exercise have focused on alteration in the recruitment of motor units or muscles themselves [12]. In reality, it is best to use the concepts of central fatigue (ie, an exercise-induced decrease of muscle force caused by a reduction in recruitment) and peripheral fatigue (ie, decrease in force caused by a decrease in muscle fiber contractility induced predominantly by metabolic events within the muscle). However, it is not within the scope of this article to describe in detail the central and peripheral aspects of fatigue. The reader is referred to the excellent reviews of Gandevia [14] and Fitts [15].

Reliable assessment of neuromuscular fatigue can be studied in humans using percutaneous stimulation of nerve or muscle, although this method sometimes causes discomfort [12,16,17]. This method usually associates electromyography (EMG) and measurements of voluntary and evoked forces. It is possible to superimpose an electrical or magnetic stimulation on the motor nerve (ie, tibial nerve for the soleus muscle) or to perform transcranial magnetic stimulations to evaluate the level of central fatigue of a subject [12]. In short, the twitch superimposed to a maximal voluntary contraction (MVC) is compared with the twitch evoked on the relaxed muscle in order to

calculate the level of voluntary activation (twitch interpolation technique Ref. [18]). If the electrical stimulus elicits an increase in force greater than MVC, then there is reduced motor drive and central fatigue has occurred. Electrical or magnetic stimulation can also be evoked on the relaxed muscle to explore peripheral fatigue (ie, amplitude of the mechanical response). By analyzing the changes in the integrated electromyographic signal (iEMG), or root mean square (RMS), and the compound action potential (M-wave) during voluntary and evoked contractions, respectively, EMG can also be considered as an acceptable noninvasive method of measuring neuromuscular fatigue [12]. With such indices it is possible to clarify whether a decrease in MVC force is completely attributable to the loss of muscle contractile properties, or whether central drive may contribute to this decrease. This method has been applied to isometric contractions more frequently than to dynamic ones of muscles groups of both upper and lower limbs [14]. In addition, peak MVC force is commonly assessed in humans to characterize fatigue, probably because it is an easily measurable variable under relatively standardized conditions, which ultimately allow valuable comparisons between studies [17]. In racquet sports, one issue to be resolved when assessing functional impairment of fatigued muscles is that the dynamic parameters such as the rate of force development, usually analyzed in other sports, does not reflect the specific fatigue that is induced by repeated accelerations-decelerations of brief durations in order to reach maximum velocity. Therefore one may underestimate the functional muscle impairment (ie, ability to generate fast movements) by analyzing force parameters only. Furthermore, the large variability in the contraction types, the muscle groups tested, and the testing protocols (ie, nature of the exercise, ergometers, fatigue variables) complicate the interpretation of mechanisms underlying neuromuscular fatigue for a given task [17]. However, a good reliability of measurements used for characterizing central (ie, voluntary activation level or EMG) and peripheral (ie, M-wave properties or peak twitch and peak doublet torques) fatigue was recently described by Place and colleagues [19].

It is well-established that the magnitude and the etiology of fatigue depend on the exercise (task-dependency) [20]. During racquet play, fatigue develops as the duration and intensity of physical exertion increase. Transient episodes of muscular fatigue generally occur after several consecutive intense rallies. As a result of this combination of repeated sprints and the prolonged pattern of exercise, the ability to maintain exercise intensity in racquet sports represents a unique challenge. During real competitions the changes in the sequence of movement are complex and unpredictable, and are widely affected by the level and game of the opponent [1], so this variability imposes unique physiologic demands. Therefore, it is likely that the mechanisms underlying fatigue are different among the different racquet games.

The neuromuscular adaptation to fatigue has been widely studied in conditions of voluntary isometric muscle actions (ie, Duchateau and colleagues

[21]) and electrical stimulation (ie, Boerio and colleagues [22]). Much less is known, however, about the effects of real exercise such as racquet games on the neuromuscular function. This lack of direct scientific information on racquet games-specific fatigue is only partly compensated for by interpretation of data from laboratory studies or of other intermittent activities (ie, team sports). The objective of the next two sections is to carry out an integrative review of the available literature on possible causes (peripheral and central) limiting muscular performance during racquet sports competitions.

## **Peripheral fatigue**

### *Muscle activity*

At the peripheral level, surface EMG recordings during evoked contractions have been used to indirectly explore neuromuscular fatigue [12]. In human experiments, the M-wave is commonly used as an index of neuromuscular transmission and action potential propagation in muscle fibers [23]. During intense short-term activities, reductions in ionic ( $\text{Na}^+$  and  $\text{K}^+$ ) trans-membrane gradients may occur, resulting in a decreased M-wave amplitude [24]. Briefly, the increased intracellular  $\text{Na}^+$  and reduced intracellular  $\text{K}^+$  could be attributed to insufficient activation of the  $\text{Na}^+/\text{K}^+$  muscle pumps [24,25]. After prolonged continuous exercises [26], changes in M-wave characteristics (ie, decreased amplitude, increased duration) are rather modest, suggesting that sarcolemmal excitability does not play a fundamental role in limiting performance in these activities. In accordance with these findings, no significant change in M-wave of both vastii has been observed during a 3-hour tennis match play [27]. Rather, fatigue related to  $\text{Na}^+/\text{K}^+$  disruptions might be a transient phenomenon, which would explain the temporary loss of performance (ie, incapacity to repeat powerful movements) associated with periods of repeated intense exercise bouts with incomplete recovery periods, as typically observed in racquet sports. Correspondingly, Mohr and colleagues [28] have recently postulated that temporary fatigue during an intense soccer match could be linked to an accumulation of  $\text{K}^+$  in the muscle interstitium.

### *Excitation-contraction coupling*

The excitation-contraction (E-C) coupling process is the sequence of movement that starts with release of acetylcholine at the neuromuscular junction and ends with the release of  $\text{Ca}^{2+}$  from the sarcoplasmic reticulum (SR) [29]. Despite the influence of stiffness alteration of the muscle-tendon complex on the evoked twitch mechanical response, it has been proposed to study the changes in the shape of this parameter to determine whether impaired intracellular  $\text{Ca}^{2+}$  regulation by the SR is implicated in the fatigue process [25]. In examining modifications of single twitches (quadriceps, Ref. [27]; soleus, Ref. [30]) during a 3-hour tennis match play, a reduced

peak twitch torque has been found to occur, suggesting a failure of the E-C coupling. Although early research focused on acidosis as the most likely cause of muscular fatigue, recent evidence provides substantial support for an increase in inorganic phosphate (Pi) having a key role [31]. Pi appears to interfere with muscle function because its entry into the SR induces a  $\text{Ca}^{2+}$ -Pi precipitation, and hence decreases the  $\text{Ca}^{2+}$  available for release. This would in turn attenuate the binding of  $\text{Ca}^{2+}$  to troponin C. Fewer cross-bridges would be formed between actin and myosin molecules, and hence a lower force or power would be generated. Low-frequency fatigue shown by a large decrease of force following stimulation at low frequency (ie, 20 Hz) concomitant with small or no change in force evoked by a stimulation at high frequency (ie, 80 Hz) has been shown to be related to a reduction in  $\text{Ca}^{2+}$  release, and is generally considered to reflect E-C coupling failure [32]. Muscular damage following exertion and low-frequency fatigue are connected. Low-frequency fatigue has been observed after prolonged tennis playing [27]. This is not surprising considering the amount of stretch-shortening cycle movements (ie, serves, jumps, or changes of direction) and eccentric contractions (ie, flexions or breakages), which may induce some structural myofibrillar impairment.

#### *Metabolic energy supply*

Several factors including decreased phosphocreatine (PCr) availability, increased muscle acidity, decreased muscle carbohydrate (glycogen) stores, or a low blood glucose level have also been suggested as causes of fatigue at the muscle level. These are beyond the scope of this article, but they are presented briefly here because they may also contribute to fatigue.

#### *Phosphocreatine availability*

After a bout of intense/maximal work, a decrease in PCr stores occurs, and the complete replenishment can last between 3 and 5 minutes [33]. The recovery time between two consecutive efforts in racquets games is largely smaller (see Table 1), and thus PCr stores are only partially restored between rallies, leading to a progressive depletion during a match. In soccer, decreased muscle PCr was significantly correlated with impairment in sprint ability [28]. Conversely, performance in intense intermittent exercise was improved after a period of creatine supplementation [28]. It is doubtful, however, that the PCr depletion is a predominant factor of fatigue, because muscle ATP rarely falls below 60% of pre-exercise levels during exhaustive exercise [34].

#### *Acidosis*

There are some strong correlations between muscle (skinned fibers) fatigue (ie, reduced isometric force and shortening velocity), and the presence of high lactate ([La]) or low pH concentrations [35,36]; however, recent

studies have challenged the role of acidosis as a direct cause of muscle fatigue [31]. Generally, [La] levels (see Table 2) and hence muscle acidity are thought to remain low (table tennis, tennis) to moderate (badminton, squash) in racquet sports competitions [2] and to contribute little to fatigue. Temporary fatigue during a game (ie, a series of consecutive intense rallies), however, may be related to high [La] or muscle acidosis. Nevertheless, it is noteworthy that [La] measured infrequently can only reflect the exercise intensity immediately before sampling. In addition, results linking acidosis and performance in racquet sports are sometimes conflicting. For example, it has been reported that performance in repeated-sprints can be maintained despite increased muscle acidity [37]. Taken together, these findings suggest that acidosis is probably not the most important cause of neuromuscular fatigue in intermittent activities. However, further studies are required to determine the role of acidosis in racquet games.

### *Muscle glycogen*

The close association between fatigue and glycogen depletion during prolonged continuous exercise is well-documented [38]. Although conflicting results have been reported (ie, [37]), there is good evidence that low muscular and hepatic glycogen concentrations play an important role in limiting performance during long-term high-intensity intermittent exercises. For example, soccer players who have low pre-match muscle glycogen spent more time walking and less time sprinting than their counterparts who had a higher muscle glycogen content [38]. However, there is little direct information concerning the rate and extent of muscle glycogen depletion during racquet sports. Carbohydrate intake has been found to improve explosive strength (Sargent jump) after 2 hours of tennis [39]. These findings suggest an unexplored relationship between low muscle glycogen levels and fatigue during prolonged racquet sports matches. In addition, it is known that glycogen concentrations directly influence the metabolism of neurotransmitters involved in the central component of fatigue [40].

### *Glucose*

In tennis, blood glucose concentration does not decrease significantly [10], and in most cases actually increases [41,42]. Hypoglycemia (ie, low blood glucose) per se is therefore unlikely to be a factor in fatigue during tennis and probably more generally in the other racquet sports.

## **Central fatigue**

### *Evidence of central activation failure*

There is a growing body of literature to support a predominant association between fatigue in racquet sports and reduced neural drive to muscle [27,30,43]. The role of central fatigue in neuromuscular perturbations can

be studied using the twitch interpolation technique, the ratio of the EMG signal during MVC normalized to the M-wave amplitude or the comparison of torques achieved with maximal voluntary and electrically-induced contractions [12,26]. Using a combination of these different methods on two lower limb muscle groups (ie, quadriceps and soleus muscles), it has been shown that central activation deficit occurs progressively during a 3-hour tennis match [27,30]. Such central activation failure has been observed by several authors during the latest stages of prolonged running and cycling exercises performed at a fixed intensity [26], and more recently after repeated cycling sprints [44]. If different types of exercise (intense versus prolonged) produce acute changes in muscular performance, however, the magnitude of these changes differs according to the type of contraction involved, the muscle groups involved, and the exercise duration and intensity; a phenomenon termed “task-dependency” [20]. Thus, it is likely that the mechanisms underlying fatigue in the different racquet games are different. Future experimentation should therefore be designed to investigate the role of fatigue in the impairment of performance in badminton, table tennis, or squash. Based on their central governor model, St Clair Gibson and Noakes [45] proposed that during self-paced, continuous exercise, the central nervous system continuously modifies the pace as part of a complex, nonlinear dynamic system. This model predicts that the ultimate control of exercise performance resides in the brain’s ability to vary the work rate and the metabolic demand by altering the number of skeletal muscle motor units recruited during exercise, with the end point of the exercise acting as the reference point. In the study by Girard and colleagues [27], the progressive reduction in normalized EMG and muscle activation level as the exercise progressed could be interpreted as a protective mechanism of the neuromuscular system [14]. However, “pacing” does not make sense in racquet sports because the games are not predictable and are influenced by numerous factors, including the level and style of the opponents [1]. Collectively, these results indicate that a reduced number of motor units are voluntarily recruited by the subjects, or that the maximal discharge rate from active motor units is not attained [46] in the latter stages of an exhausting tennis match [27,30]. Nevertheless, the ability to identify central and peripheral fatigue is complicated by the facts that both supraspinal and spinal mechanisms are involved, and that spinal fatigue involves both positive and negative influences of afferent sensory feedbacks.

### *Supraspinal fatigue*

Changes in neurotransmitter concentrations and flux have been suggested to be involved in central fatigue (ie, supraspinal fatigue). Increase in serotonin, dopamine, and acetylcholine concentrations in the brain may reduce the rate of central neural drive, which negatively influences the excitement and recruitment of skeletal muscles [47]. It has been also clearly shown that an

increase in the ratio of free tryptophan:branched chain amino acids (BCAA) in the circulation could affect prolonged performance via an increased concentration of free tryptophan (the serotonin precursor) in the brain [48]. Struder and colleagues [43] have postulated that the fatigue-induced reduction in performance in racquet sports players has a supraspinal origin: indeed, 4 hours of singles tennis increased the free tryptophan:BCAA ratio more than 2.5-fold. In addition, it is thought that these changes in neurotransmitters concentration have an important effect on arousal, lethargy, sleepiness, and mood state [48], which in turn may influence the cognitive and sensorial variables (ie, perception of effort, stress management, tactical choices) during decisive rallies. Interestingly, it has been suggested that nutritional status (especially the carbohydrate metabolism) affects neurotransmitter metabolism, such as with an increase in carbohydrate intake attenuating fatigue-related increases in serotonin [40]. In short, carbohydrate supplementation before or during exercise aims to limit depletion of muscle glycogen stores by attenuating the rise in free fatty acids, which in turn contributes to limiting the augmentation of some precursors of central fatigue (ie, plasma-free tryptophan:BCAA ratio). Thus, it is not surprising that carbohydrate supplementation is an efficient strategy to reduce fatigue after several hours of tennis play, even if performance variables (ie, ground stroke accuracy, number of games won and lost per match) remained unaffected by the treatment [40]. More recently, measuring handgrip force (ie, a muscle not involved in the fatiguing exercise) has also been proposed as a method of further exploration of the potential existence of supraspinal fatigue after prolonged exercise involving lower limbs [26]. However, no changes were observed in grip strength of the nondominant arm during a 3-hour tennis match suggesting that loss of cortical excitability per se is probably not the only cause of central fatigue [30].

### *Spinal fatigue*

As previously stated, it is well-established that racquet sport performance may be associated with peripheral perturbations (ie, increase in metabolites, acidosis, lesser energy supply). Despite the direct effects that selected metabolites might have on muscle contractility [15], it is also believed that neural-mediated afferent feedbacks from the muscle (ie, fusimotor system disfacilitation and presynaptic inhibition) play a role in the inhibition of motoneuron excitability [49]. Two major hypotheses have been proposed to explain the mechanisms responsible for the reduced neural drive to the muscle. The first hypothesis involves the decreased facilitation of the  $\alpha$ -motoneuron pool concomitant to a progressive withdrawal of spinal-mediated fusimotor support [50]. The second hypothesis speculates that reduced neural activation depends on the reflex response from the contracting muscle itself to metabolic or mechanical changes [51]. An increased inhibitory drive to the  $\alpha$ -motoneuron pool, probably mediated by small-diameter Group III and IV

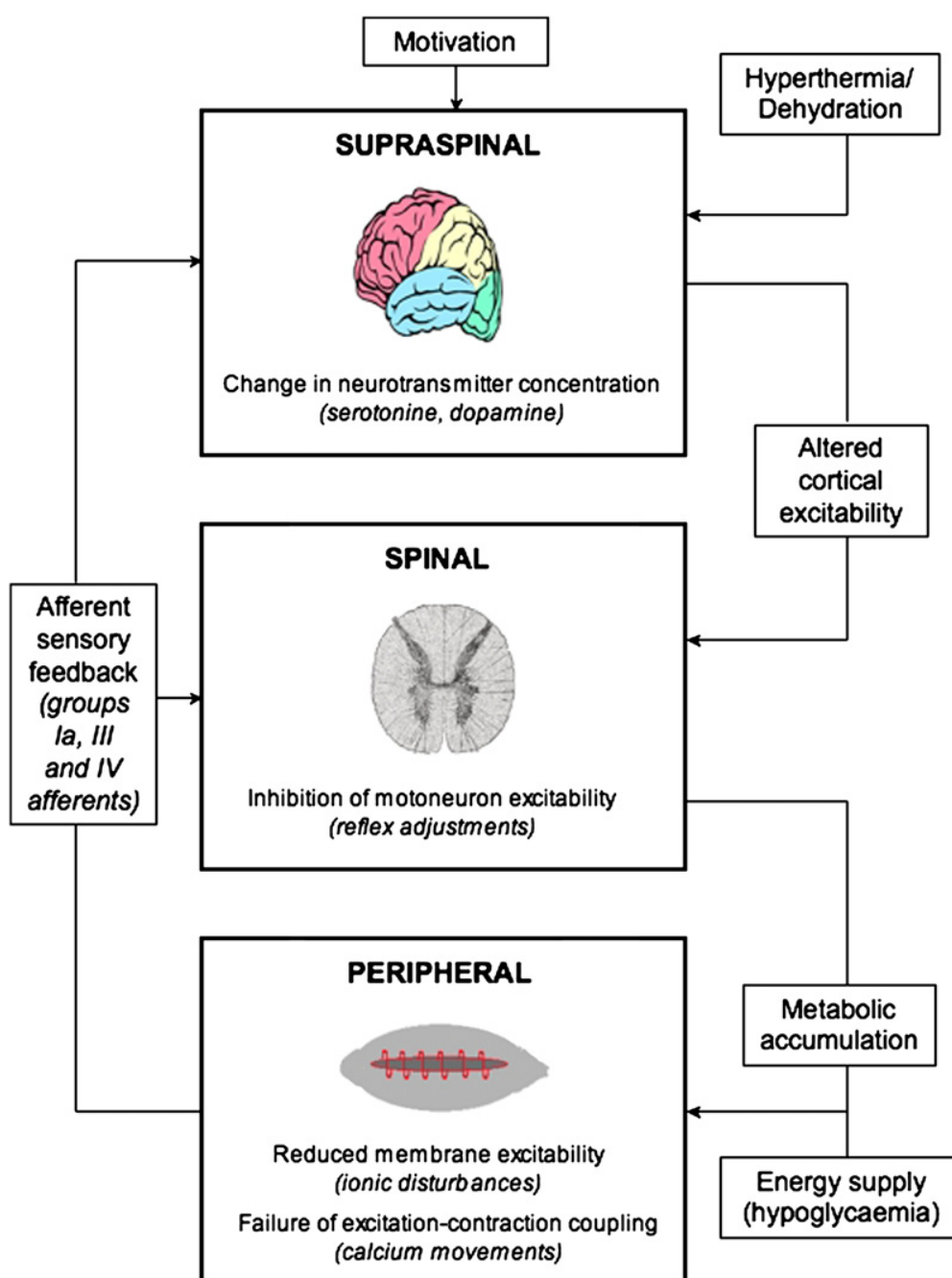


Fig. 1. Potential mechanisms underlying neuromuscular fatigue in racquet sports. Exercise performance may be limited (as match progresses) because of a reduced central activation, linked to changes in neurotransmitter metabolism or in response to afferent sensory feedbacks. Alternatively, modulation of spinal loop properties (ie, inhibition of motoneuron excitability) may occur because of changes in metabolic or mechanical properties within the muscle. Finally, the temporary fatigue observed after a series of consecutive intense rallies may be caused predominantly by a reduction in sarcolemmal excitability because of ionic disturbances and alterations in excitation contraction coupling (low-frequency fatigue).

muscle afferents, contributes to the reduction in voluntary drive (at both spinal and supraspinal levels). Regarding racquet sports performance, this latter hypothesis is supported by a recent study [30] examining the excitability of spinal reflex loops by using combined measurements of evoked Hoffmann (H) reflex (at rest) and first volitional (V) wave (during contraction; ie, an

electrophysiological variant of the H-reflex) responses during the course (each 30 minutes) of a 3-hour tennis match play. These reflexes reflect the efficiency (ie, estimate of  $\alpha$ -motoneuron excitability) of the transmission in Ia afferent- $\alpha$ -motoneuron synapses (principally H-reflex) and the level of efferent and descending neural drive (mostly V-wave), and these evoked potentials are both influenced by reciprocal inhibition and recurrent inhibitions [52]. Significant reductions in H- and V-wave responses (normalized to the M-wave) were observed as the tennis match progressed, suggesting an inhibition of the spinal motoneurons (ie, modulation of spinal loop properties) during this prolonged intermittent exercise [30]. Similarly, in the other racquet sports a decrease in motoneuron excitability in response to metabolic disruptions remains a potential fatigue factor affecting the ability to fully activate the synergistic musculature (ie, ability to generate explosive power within a few seconds) (see Table 2). Nevertheless, future studies are needed to quantify the relative importance of the possible reflex adjustments in the neural origin of performance decrease in all racquet sports. Overall, these findings emphasize the possible role of the peripheral reflex pathways as a probable origin of the reduction in central efferent neural command in racquet sports. In addition, this suggests that the reflex may act as a protective mechanism to preserve force-generating capabilities and possibly avoid irreversible cellular damages (Fig. 1) [45].

## Summary

Fatigue impairs racquet sports performance, and can be manifested as mistimed shots, altered on-court movements, and incorrect cognitive (ie, tactical) choices. The etiology of muscle fatigue in racquet sports is a complex phenomenon that may involve impairment in both neural and contractile processes (see Fig. 1). Temporary fatigue observed after a series of consecutive intense rallies may be caused predominantly by a reduction in sarcolemmal excitability because of ionic disturbances and alterations in excitation contraction coupling (low-frequency fatigue). Future research should investigate if electromyostimulation [53] and resistance training [54] can improve racquet sports-related variables and delay central fatigue. Over time, research into fatigue-related mechanisms in athletes should be expanded to include other forms of sport.

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