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EMG IN EXERCISE PHYSIOLOGY AND SPORTS

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19.1 SURFACE EMG FOR STUDYING MUSCLE COORDINATION

Surface EMG is useful in studying many issues of motor control. The issues of co-activation, onset muscle timing, and characterization of exercise will be analyzed in this section under the viewpoint of muscle coordination (see also Chapter 6).

19.1.1 Methodological Issues in Assessing Muscle Coordination

As highlighted in the review of Hug [80], many limitations of sEMG could constrain the appropriateness of EMG in detecting muscle coordination. The limitations of sEMG are intrinsically related to the technique or to signal processing. Intrinsic limitations are that sEMG can be detected only from superficial muscles, provides information related to only a limited volume of the muscle, is dependent on the electrode configuration, can be altered by crosstalk, and is affected by amplitude cancellation.

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Filtering EMG. Regarding signal analysis, one drawback revealed by Hug [80] is the lack of standards in the choice of the low-pass filter to extract the linear envelope of sEMG profile (see Chapter 16). A wide range of low-pass filters have been used in literature, from 3 Hz to 40 Hz [64,190]. Since the choice of the cutoff frequency and of the filter order lead to different EMG pattern, this issue is of considerable importance. A hypothesis is that there should be an "optimal" order and low-pass cutoff frequency for each application (e.g., biofeedback, gait analysis, control of devices).

To increase the signal-to-noise ratio [24], a number of trials or cycles of each task should be averaged. Because of differences in the task durations, time normalization is commonly used to convert time axis in a percentage axis [82,190] (e.g., gait cycle, pedaling cycle, or other tasks).

EMG Normalization. Since comparing sEMG amplitude between different muscles does not provide any information about the degree of activation, normalization of EMG amplitude is recommended. In this case, sEMG activity is expressed as a percentage of a previously recorded reference value, that is, MVC [7,48]. Since muscle activation is dependent on joint angle [156], some studies using MVC normalization methods reported an activity level above 100% in maximal cycling [73] and during a pitch in baseball [89]. To avoid lengthy procedures related to MVC assessment and to face the problem that sEMG amplitude during MVC without training could be 20/40% lower than that obtained after training [75], a number of studies normalized the EMG pattern with respect to the peak obtained in a specific movement [168]. However, this method (named peak dynamic method) does not provide information about the degree of muscle activation. In summary, at this time, there is no agreement for the best normalization procedure [25]; therefore, precise comparison about the degree of muscle activity during a specific task cannot yet be performed.

Assessing Timing Activation. Two additional issues concern the interpretation of coordination regarding the timing of muscle activation. The first is the threshold value chosen to consider a muscle as "active." There are more than ten methods to determine muscle onset: usually a percentage of the peak EMG (for example, 20% of MVC value) or one to five standard deviation above the mean of baseline activity have been used [180]. Another method has been proposed by Merlo et al. [122] and is not based on threshold crossing but on the identification of motor unit action potentials with the use of the continuous wavelet transform. When repetitive or cyclic movements are studied, muscle activation timing is obtained by time normalization and averaging many consecutive cycles. This EMG profile generally depicts the evolution of the amplitude throughout the stride or crank cycle, and its duration is expressed as percentage of total duration of the complete cycle.

Another issue is the so called electromechanical delay, which is the time lag between muscle activation and muscle force production. As reported [148], it ranges from 30 to 100 ms depending on mechanical properties of both tendon and aponeurosis. Hence intersubject timing variability should be taken into account in the interpretation of sEMG onset.

19.1.2 Co-activation

Role of Co-activation. The simplest pattern of activation among a group of muscles is the relation between agonist and antagonist muscles across a joint. Since the biomechanical definition of agonistic and antagonistic muscle does not necessarily coincide with the corresponding anatomical definitions [158], agonist and antagonist muscles are herein respectively defined as the muscle producing a moment coincident with, or opposite to, the direction of the joint moment [5].

Co-activation is defined as antagonist muscle activity occurring during voluntary agonist contraction [157]. The modulation of co-activation is a strategy used by CNS to achieve opposite objectives: maximal force output and stabilization to ensure joint integrity. Although co-activation may impair the full activation of agonist muscle by reciprocal inhibition [186], it can (i) assist ligaments in maintaining joint stability under heavy loads [10] and (ii) provide breaking mechanisms during high-velocity movements [109]. The effect of co-activation is the stiffening of the joint, when a precision or dangerous motor task is required.

Surface EMG Variables to Assess Co-activation. Co-activation level could be expressed as a percentage of a reference EMG values recorded in different conditions: during MVC as agonist in isometric contraction; slow concentric contraction like 15°/s [10]; slow eccentric contraction [1]; the maximum value obtained during a movement. It is worth noting that the eccentric normalization is a proper method since any antagonist activation is an eccentric contraction, a condition related to higher torque and lower EMG activation [93]. Gracies [63] proposed a co-contraction index defined as the ratio of the RMS of a muscle when acting as antagonist to the intended effort to the RMS of the same muscle when acting as an agonist to the opposite effort.

Motor Learning and Co-activation. Co-activation has a protective effect, but could be seen as a counterproductive action because to maximize the torque expressed by a joint it necessary to minimize the activation of the antagonist muscles. As expected, reduced activation of antagonist muscles has been related to the force gains obtained after a strength training period. For instance, in Carolan and Cafarelli [27] a 20% decrease of hamstring co-activation during the early period of training were associated to a 33% increase of net knee extensor torque; that is, the increase in torque exertion depends both on the greater capacity of quadriceps to generate force and on the deactivation of antagonist hamstring muscles.

Reduced level of muscle co-activation is also related to the achievement of motor skill [11]. In order to obtain a more economical coordination strategy [16], a progressive inhibition of unnecessary muscular activity (e.g., co-activation) is obtained during the course of specific task training. For instance, lower co-activation was found in isometric or isokinetic movements across the elbow in skilled tennis player [13] and across the knee in high jumpers [4].

McGuire et al. [118] referred to maximal isometric contractions as a motor skill, not only for the acquisition of muscle strength but also for the variability of force

output. In their study, participants performed three training sessions consisting in 10 rapid maximal contractions of the wrist flexor muscles. Force variability was defined as both the variance of the torque–time curves and the stability of the torque at maximum. The peak-to-peak amplitude of the V-wave was measured to assess changes in neural drive in agonist muscle (see Chapter 12 for details). The primary mechanism to increase maximum strength was a reduction in co-activation because the peak-to-peak amplitude of the V-wave for the agonist remained unchanged with training. The authors argued that during the early stages of training, participants learned to manage "minimally sufficient" levels of co-activation, allowing an increased expression of agonist muscle strength while at the same time providing enough joint stiffness to reduce the variability of force production.

Task Differences. The role of velocity in co-activation is still a topic of discussion: Some studies found a positive relation between angular velocity and antagonist co-activation [66,93]; others did not find any influence [13,79,149]. Only few works investigated the role of co-activation in ballistic sport specific actions. Sbriccoli et al. [173] compared two actions, a "constrained" (isokinetic) vs a "free" (karate front kick) ones, in two groups of elite and amateur karateka. They showed that elite karateka used a more effective tuning of agonist–antagonist muscles. Elite karateka adopted lower co-activation in isokinetic movements and higher co-activation in the front kick with respect to amateurs. Indeed when the movement is safe, such as that performed using an isokinetic machine, they decrease the antagonist activation to increase the net torque expressed; when the co-activation of flexor muscles provides a stronger braking action, which is essential for a correct technique execution, they increase the antagonist activation.

The level of co-activation across a joint is different, depending on which muscle acts as agonist or antagonist. As shown in Bazzucchi et al. [13], when triceps brachii acts as an antagonist of elbow flexion, it shows an activation that is 16% greater than shown by biceps brachii when it is an antagonist of elbow extension. Since the authors clearly discussed both confounding factors such normalization procedure and relative subcutaneous tissue thickness, it is possible to conclude that the observed differences in co-activation are probably due to the different involvement of those muscles in daily activity.

Age Effect. The loss of strength in elderly people, beyond the histological and neuromuscular issues at the level of agonist muscles, could be caused by an ineffective tuning of antagonist muscles. Indeed some studies report an increase in co-activation in elderly compared to young people at the level of elbow and knee joint both in men and women [72,87,94,113] and at the level of ankle in postural balance task [144].

19.1.3 Onset Timing

Many other strategies of the neuromuscular control are aimed to protect joints from potential dangerous situation. Critical is the timing of onset muscle activation prior

(i.e., preparatory activity) or posterior (i.e., latency) to a sudden mechanical event. In general the variable adopted to address these issues is the time difference between a mechanical event occurring at a joint and the muscle onset aimed to protect the joint. As previously mentioned, the method used to define the sEMG threshold of muscle activation is pivotal to detect the muscle onset.

Preparatory Activity. The absorption of impacts resulting from the contact with a landing surface during running, jumping, and landing has been widely addressed in the literature. This topic has an important clinical relevance to appropriately plan and control the absorption of impacts which might injury the muscle–skeletal system [172]. The so-called preparatory, or pre-landing, muscle activation is a neuromuscular strategy occurring before the impact during the downward flight of landing. Lower limb muscles are activated in order to provide the appropriate levels of stiffness of the joints to smoothly absorb the impact of landing. This mechanism is involved partly to counteract latency period due to electromechanical delay [22] and partly to prepare force buildup before toe down in landing [61]. An imbalanced or ineffective neuromuscular recruitment pattern during landing or pivoting maneuvers may lead to increase in injury risk [18]. The preparatory activity has been shown to modulate both EMG amplitude and onset timing on the basis of the landing height and control strategies.

One of the most common injuries in sports is the rupture of the anterior cruciate ligaments (ACL). This injury occurs when high dynamic loading of knee joint are not adequately absorbed by the muscle activation and the passive ligaments structure are subjected to excessive torque [19,110]. In particular, anterior directed shear of the tibia, possibly responsible for ACL injury, should be counteracted not only by the ACL but also by appropriated activation of knee flexor muscles [47]. The timing of non-contact ACL injury ranges between 17 to 50 ms after initial ground contact [105]; within this time lapse, feedback and reactive motor mechanism are too slow to occur. Thus, substantial neural pre-activation of knee flexor muscles just before ground contact seems to be essential [191] during fast dangerous movements such as landing and side cutting. This is consistent with the findings that a reduced pre-activation of knee flexor muscles associated with an elevated activation of knee extensors (which can lead to an augmented anterior shear of the tibia) are two main risk factors in ACL injuries [191].

Latency or Delayed Onset. Besides preparatory activities, the stretch reflex activation could also be seen as a protective joint mechanism. The short latency component of stretch reflex is a mechanism responsible for rapid muscle recruitment elicited by mechanical muscle stretch. The stretch reflex of muscles involved in ankle control could represent a neuromuscular protection mechanism to avoid ankle sprain. This is an important issue since ankle sprain is one of the most common injuries in athlete lower limbs.

The reaction time of evertor muscles to simulated ankle sprain has been advocated to characterize sensorimotor deficit in the so-called "functional ankle instability." The reaction time has been measured as the time lapse between platform tilt and sEMG

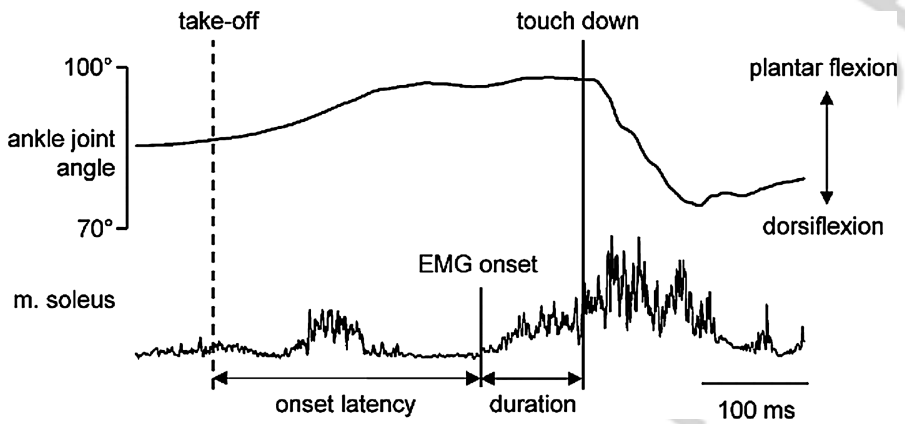

FIGURE 19.1 EMG activity of soleus muscle (**bottom**) and the time course of the ankle joint angle (**top**) during a drop jump from 0.2-m height. Reproduced from Santello [172] with permission.

onset of muscles involved in ankle control, such as peroneus longus, peroneus brevis, tibialis anterior, and extensor digitorum (see Fig. 19.1). Several tilt platforms were used to simulate ankle sprain while remaining within an injury-free range of motion. Some had only one tilt plate, allowing one predetermined limb tilted, and other have two movable plates so that the subject is unaware which side will tilt. Most platforms expose the limb only to supination while others combining plantar flexion and supination, reflecting better the mechanics of joint injury [103]. Using these methods several authors have reported imbalances showing 10–20 ms of delay in the onset latency in subject with functional ankle instability [91,102,103,128]. Thus measuring evertors reaction time in sudden inversion movements has been proposed as a reliable procedure to assess sensorimotor imbalances (Fig. 19.2) [15,103]. However, this issue is still under debate since recent meta-analysis did not find unequivocal impairment in subjects with functional ankle instability [1[illegible]]. Considering the neuromuscular basis of imbalanced muscle latency, it could be assumed that training program can influence the onset timing of muscles involved in ankle sprain. Balance and strength training has been adopted to prevent ankle sprain, but contradictory results on muscle latency were found. While some authors reported neuromuscular training as an effective program to reduce muscle latency [175], others did not [45].

19.2 USE OF sEMG TO CHARACTERIZE TRAINING EXERCISE

sEMG could be used as a useful tool to (a) precisely characterize strength and rehabilitation exercise, (b) know the reciprocal activation of each muscle involved in an exercise, and (c) determine which exercise could be suitable for a specific training aim. Moreover, sEMG could help sport scientists and practitioners to choose the most effective configuration of physical exercises to a specific target. Indeed each exercise could be performed in various forms [182] by modifying the device or parameters

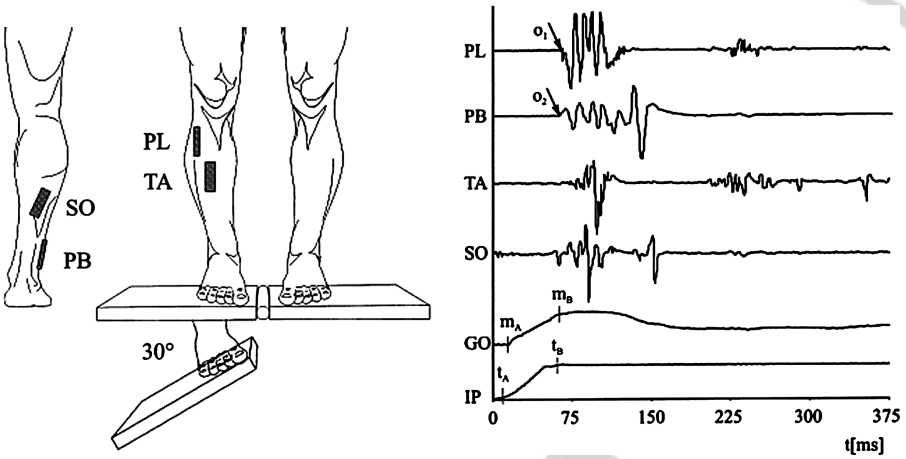

FIGURE 19.2 **Left panel:** Schematic view of the tilting platform. The subject had to stand on the tilting part with more than 95% of body weight on it. When the EMG showed baseline activity, the trapdoor was released (PL, peroneus longus; PB, peroneus brevis; TA, tibialis anterior; SO, soleus). **Right panel:** Representative graph of a single measurement. It shows that rear foot inversion lasts longer than tilting movement. Whether inversion is limited actively by peroneal contraction alone or by passive ligamentous complex cannot be distinguished (in this study, the inversion angle (GO) was not analyzed. Reproduced from Benesch et al. [15] with permission.

such as overload, body position, range of motion of the movement, speed, and grip. Each form corresponds to a different type of training stress and to a different physiological adaptation.

19.2.1 Preferential Activation

The hypothesis that one synergic muscle could be preferentially activated with respect to another one is an interesting topic of discussion. In particular, the relative contribution of synergistic muscle heads belonging to the same muscle group has been widely addressed (see Chapter 5 and the issue of load sharing). Muscle groups such as quadriceps, gastrocnemius, triceps brachii, and biceps femoris has been investigated. For instance, a debate exists around whether in the quadriceps muscle the vastus medialis obliquus (VMO) can be preferentially activated with respect to vastus lateralis (VL) muscle during knee extension exercises.

An imbalance in the activation of VMO and VL may be an etiological factor in the development of patella–femoral pain syndrome [34,116], and VMO atrophy contributes to patella instability [62,150]. To reduce such an imbalance, VMO has been proposed to be strengthened through exercises involving VMO more than other quadriceps muscle [62], and sEMG has been used to verify the relative activation of VMO and VL [178]. To perform this analysis, sEMG amplitude of each muscle head has been assessed during MVC of knee extension and used as reference for successive analysis. The VMO/VL sEMG amplitude ratio during MVC has been taken as

reference. Then, each successive exercise is characterized by its own VMO/VL ratio, where the larger the ratio the greater the activation of VMO.

As sometimes occurs, practice habits are not based on solid methodological studies. Contrary to the widely held belief that training quadriceps at 30° knee flexion mostly activates VMO, recent work of Spairani et al. [179] failed to find a difference in VMO/VL activation by changing the knee angle from 30° to 90°. Moreover, some authors suggested that tibial external rotation [189] and hip adduction [76] enhanced VMO activity while performing a knee extension task. Other exercise alterations such as foot pronation or supination, ankle dorsiflexion, or plantar-flexion [108] have been used in the hope to increase VMO intervention. Irish et al. [85] showed that closed kinetic chain exercise (such as squat and lunge) implied a greater VMO/VL ratio than open kinetic chain exercises (such as pure knee extension). However, in the review of Smith et al. [176], only three studies (with some methodological limitations) out of 20 referred preferential VMO activation by altering lower limb joint orientation or co-activation. Because of these findings, in presence of patella-femoral disorder, it has been recommended to focus on general quadriceps strengthening opposite to specific VMO training.

19.2.2 Multi-articular Muscles

SEMG has been widely used to study muscle coordination during locomotion such as walking, running, and cycling. SEMG is often used to highlight the role of each muscle along the locomotion cycles. Typically up to 12 muscles of hip and inferior limbs are sampled. The pattern of muscle activation during locomotion can be analyzed in terms of activity level and/or activation timing as earlier discussed.

Pedaling is a standardized movement and could be a useful framework to extract information about the role of mono-articular and bi-articular muscles. Ericson [50] showed that 120-W cycling workload induces higher EMG activity in mono-articular than in bi-articular muscles. Mono-articular muscles such as VL and soleus showed 44% and 32% amplitude of their respective sEMG at MVC, while bi-articular rectus femoris and gastrocnemius lateralis showed 22% and 18%, respectively. In addition, mono- and bi-articular muscles show different timing activations. Indeed, has been reported [169] that both VMO and VL muscles, being mono-articular knee extensors, exhibited a rapid onset and cessation with relatively constant activity among subjects during the down-stroke phase. Conversely the rectus femoris, being bi-articular muscle, demonstrated a more gradual rise and decline. Moreover, Ryan and Gregor [168] noted that the mono-articular muscles (gluteus maximum, VL, VM, tibialis anterior and soleus) play a relatively invariant role as primary power producers. Conversely the bi-articular muscles (biceps femoris, semitentidosus, semimembranosus, rectus femoris, gastrocnemius medialis, and gastrocnemius medialis) behave differently and with greater variability [81]. According to the theory proposed by Ingen Schenau et al. [84] and largely reported in the literature following their study, bi-articular muscles appear to be primarily active in the transfer of energy between joints at critical times in the pedaling cycle and in the control of the direction of force production on the pedal.

19.2.3 Characterizing Strength Training Exercises

sEMG can be also used to characterize muscle activation patterns in strength training exercises. Since each exercise recruits muscles in a specific way, it is possible to focus the activity of a particular muscle and select the right exercise variation. Tomasoni et al. [185] recruited strength-trained athletes to test different forms of bench press exercise (barbell bench press, dumbbell bench press, inclined dumbell bench press). They recorded sEMG from prime mover muscles (pectoralis major, long and lateral head of triceps brachii, anterior deltoid, and serratus anterior) and used the sEMG normalization technique to compare muscles. Athletes exercised at 70% of one repetition maximum, which is the maximum amount of weight that one can lift in a single repetition for a given exercise. The most important finding was that long and lateral heads of triceps brachii were more active (i.e., greater sEMG amplitude relative to their sEMG maximum) in the exercises performed with barbells than in those performed with two dumbbells, while pectoralis major showed comparable activation in the two conditions. It seems that, when passing from the use of dumbells to barbells, the elbow extensors increase their contribution in the upper limbs push.

The same methodology has been used in the literature to quantify the contribution of muscles involved in the exercises not as prime movers but as stabilizers and neutralizer. For instance, Freeman et al. [54] compared different forms of push-ups introducing asymmetric hand placement and labile support surfaces. They measured not only the activations of prime movers upper limbs muscles but also abdominal and lumbar muscles, which act as trunk stabilizers. They noted that asymmetric handstand and ballistic push-up exercise evoked the highest level of muscle activation in the abdominal and back extensor musculature. This is an important issue since the load at the level of lumbar spine in free weight exercises should be considered. Indeed, it can both elicit back pain in some patients or it can be suggested as a therapeutic or prevention intervention in some other cases.

19.2.4 Links Between Coordination and Fatigue in Isometric Task

Muscle fatigue has effects in the coordination and pattern of load sharing among muscles. Tamaki et al. [184] reported the activity of synergistic muscles of triceps surae (soleus, medial and lateral gastrocnemius) during prolonged low-level isometric contraction. During the 210-min time course of each isometric task, alternate activity among synergist muscles of the triceps surae was observed, with some muscles becoming more active while others becoming inactive or less active. The activities seemed to occur complementary: When the lateral gastrocnemius sEMG amplitude increased, the medial gastrocnemius became inactive. It is suggested that alternate activities might facilitate the maintenance of the ankle plantar flexion tasks for as long as 210 minutes. Indeed the interval between occurrences of alternate activity tended to be shorter and more frequent in the second than in the first half of the exercise periods.

Kouzaki and Shinohara [104] confirmed this hypothesis assessing the alternating activity of quadriceps muscles (VM and VL versus rectus femoris) during prolonged isometric knee extension. They found a negative correlation between the frequency of

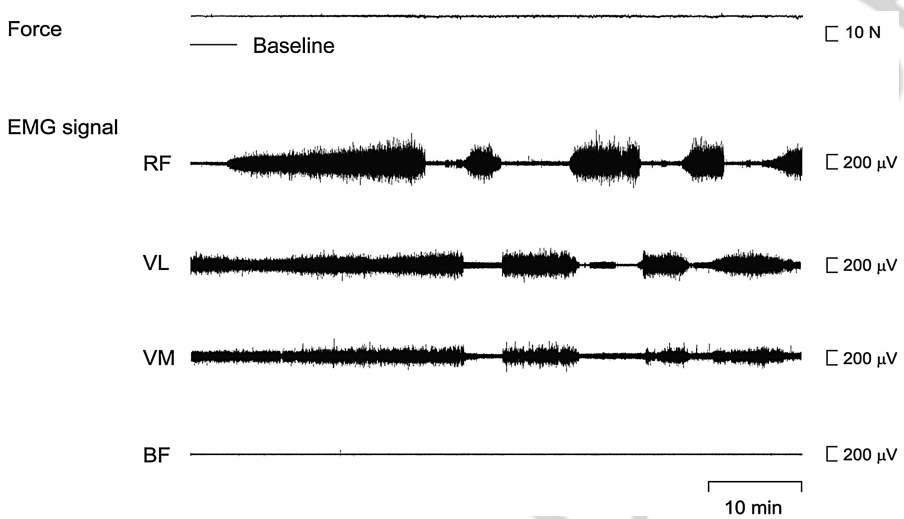

FIGURE 19.3 Representative data demonstrating alternate muscle activity during sustained knee extension at 2.5% of MVC force. Knee extension force, sEMG of rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and biceps femoris long head (BF) are shown. Reproduced from Kouzaki and Shinohara [104] with permission.

alternating activity and mechanical fatigue; that is, the subjects who alternated more frequently the activity of a muscle pair showed lower mechanical fatigue after the fatiguing task (see Fig. 19.3).

19.2.5 Links Between Coordination and Fatigue in Dynamic Task

The occurrence of fatigue induces significant alterations in muscle coordination in all muscles of the kinetic chain involved in the task. For instance, during a fatiguing cycling task [46], quadriceps sEMG amplitude remained constant, whereas hip extensor muscles, gluteus, and biceps femoris increased their sEMG activity by 29% and 15%, respectively. The authors interpreted the increase of activity in gluteus maximus and biceps femoris as an instinctive coordination strategy compensating for potential fatigue and loss of force of the knee extensors (i.e., VL and VM) with a higher moment of the hip extensors. The question of benefits of these adaptations is open to discussion.

Alternating or shifting muscle activity during the time course of a fatiguing task seems to be an optimal strategy to face fatigue. So et al. [177] compared kinematic and EMG data during a 6-minute test in five groups of rowers, from young slow rowers to Olympic athletes. Olympic rowers used an alternating muscle strategy despite the fact that they showed the lower mechanical fluctuation in intensity. Indeed they involved all muscle groups in the beginning of the task, with particular emphasis of back muscles and then switching the emphasis between the quadriceps and the back in the middle of the test, lowering the activity of back muscles and increasing that of quadriceps. Such

shifts may occur without the awareness of an athlete, as an instinctive CNS strategy to cope with fatigue.

In the reported examples the changes of muscle activation pattern during the course of fatigue was unintentional. If alternating and switching are strategies to counteract fatigue, voluntary intervention could indeed provide better control of such mechanisms. For that purpose, direct sEMG biofeedback variables would be useful for improving the activity modulation of muscles pattern.

19.3 TRAINING-INDUCED MUSCLE STRENGTH GAIN: NEURAL FACTORS VERSUS HYPERTROPHY

A motor unit (MU) consists of a motoneuron in the spinal cord and the muscle fibers it innervates [26] (see Fig. 19.4). The number of MUs per muscle in humans may range from about 100 for a small hand muscle to 1000 or more for large limb muscles [74]. It has also been shown that different MUs vary greatly in force generating capacity, that is, a 100-fold or more difference in twitch force [59,181]. In voluntary contractions, force is modulated by a combination of motor unit recruitment and changes in motor unit activation frequency (rate coding) [106,126]. The greater the number of motor units recruited and their discharge frequency, the greater the force will be. During motor unit recruitment the muscle force, when activated at any constant discharge

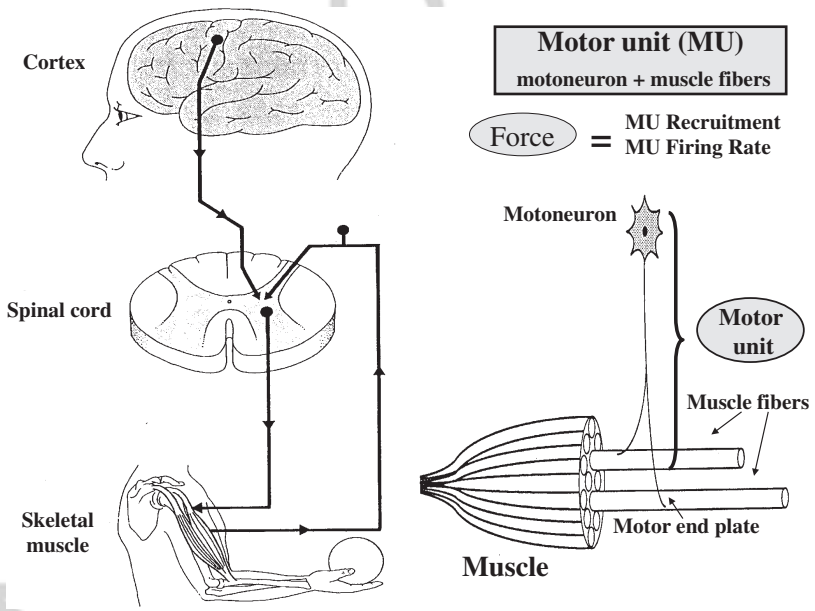

FIGURE 19.4 Schematic representation of a motor unit and its basic components. Modified from Sale [170].

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frequency, is approximately 2–5 kg/cm^2 and in general is relatively independent of species, gender, age, and training status [3].

The electrical activity in a muscle is determined by the number of motor units recruited and their mean discharge frequency of excitation, that is, the same factors that determine muscle force [20,131,138]. Thus, direct proportionality between EMG and force might be expected (see also Chapters 10 and 13). Under certain experimental conditions, this proportionality can be well demonstrated by recording the smoothed rectified or integrated EMG (iEMG)[1] [41,125,129,131], and reproducibility of EMG recordings are remarkably high—for example the test–retest correlation ranging from 0.97 to 0.99 [97,129,131]. However, the change in the surface EMG should not automatically be attributed to changes in either motor unit recruitment or excitation frequencies as the EMG signal amplitude is further influenced by the individual muscle fiber potential, degree of motor unit discharge synchronization, and fatigue [20,21,137,141]. Nonetheless, carefully controlled studies have successfully employed surface EMG recording techniques and demonstrated the usefulness of iEMG as a measure of muscle activation level under a variety of experimental conditions [66,67,69,71,96,129,130,136,170,171].

The above short summary suggests that muscle strength can be modulated by motor unit activity which in turn is under the influence of central motor drive [40]. It is a common observation that repeated testing of the strength of skeletal muscles results in increasing test scores in the absence of measurable muscle hypertrophy [33,41]. Such increasing test scores are typically seen in daily or even weekly retesting at the inception of a muscle strength training regimen. In some cases, several weeks of intensive weight training resulted in significant improvement in strength without a measurable change in girth [41,100]. It has also been shown that when only one limb is trained, the paired untrained limb improves significantly in subsequent retests of strength but without evidence of hypertrophy [33,83,129,130].

In an earlier study of Rasch and Morehouse [164], it was demonstrated that strength gains from a six week training in tests when muscles were employed in a familiar way, but little or no gain in strength was observed when unfamiliar test procedures were employed. These data suggest that the higher scores in strength tests resulting from the training programs reflected largely the acquisition of skill and training-induced alterations in antagonist muscle activity, that is, enhanced reciprocal inhibition that contributes to greater net force production, reduced energy expenditure, and more efficient coordination [167].

All of the above findings support the importance of "neural factors," which, although not yet well-defined, certainly contribute to the display of maximal muscle force which we call strength. On the other hand, a strong relationship has been demonstrated both between absolute strength and the cross-sectional area of the

[1]The term integrated EMG (iEMG) was widely used in the past as a gross measure of muscle activation. It is actually incorrect since it is equal to zero if EMG is not rectified and is always positive and monotonically increasing if EMG is rectified. It was (sometimes until now!) wrongly used instead of the average rectified value (ARV). We decided to herein maintain it only for a matter of historical references. The authors used such a term only when referring and quoting previous papers in which iEMG was used. Such a term should actually be avoided.

muscle [166] and between strength gain and increase in muscle girth or cross-sectional area [83]. It is quite clear, therefore, that human voluntary strength is determined not only by the quantity (muscle cross-sectional area) and quality (muscle fiber types) of the involved muscle mass, but also by the extent to which the muscle mass has been activated (neural factors) (see Moritani [133,134] for reviews).

A reasonable hypothesis for describing the time course of strength gain with respect to its two major determinants is that suggested by De Lorme and Watkins [38], who postulated that: "The initial increase in strength on progressive resistance exercise occurs at a rate far greater than can be accounted for by morphological changes within the muscle. These initial rapid increments in strength noted in normal and disuse-atrophied muscles are, no doubt, due to motor learning. It is impossible to say how much of the strength increase is due to morphological changes within the muscle or to motor learning."

We now have available electromyographic instrumentation and methodology which makes it possible to separate muscle activation level (motor learning) from hypertrophic effects (morphological changes) as described by deVries [41] and Moritani and deVries [129,130]. Figure 19.5 shows a schema for evaluation of percent contributions of neural factors and hypertrophy to the gain of strength output. If strength gain is brought about by "neural factors" such as learning to disinhibit and/or to increase muscle excitation level, then we should expect to see increases in maximal neural activation (iEMG) without any change in force per fiber or motor units innervated as shown in Fig. 19.5a. On the other hand, if strength gain were

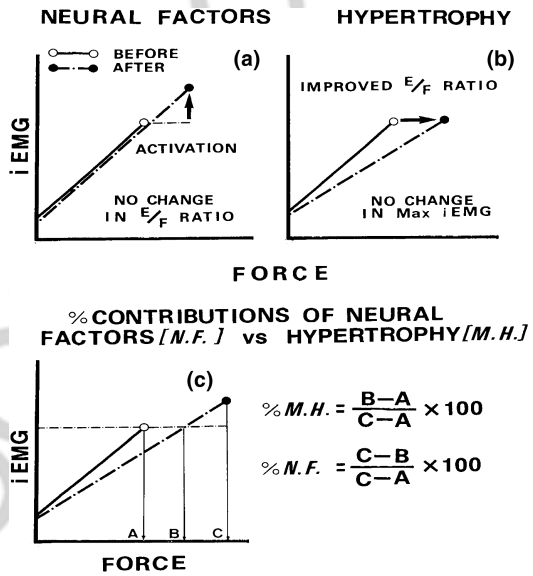

FIGURE 19.5 Schema for evaluation of percent contributions of neural factors and hypertrophy to the gain of strength during the course of muscle training based on Moritani and deVries. Reproduced from Mitchell et al. [128] with permission.

entirely attributable to muscle hypertrophy, then we should expect the results shown in Fig. 19.5b. Here the force per fiber (or per unit activation) is increased by virtue of the hypertrophy, but there is no change in maximal activation (iEMG). Figure 19.5c shows our method for evaluation of the percent contributions of the two components when both factors may be operative in the time course of muscle strength training.

Figure 19.6 illustrates the time course of strength gain with respect to the calculated percent contributions of neural factors and hypertrophy, calculated with the equation in Figure 19.5, during the course of 8-week strength training of the arm flexors of young college students. The results clearly demonstrated that the neural factors played a major role in strength development at early stages of strength gain and then hypertrophic factors gradually dominated over the neural factors for the young subjects in the contribution to the further strength gain (see Moritani and deVries [129,130] for more details). The strength gain seen for the untrained contralateral arm flexors provided further support for the concept of cross education.

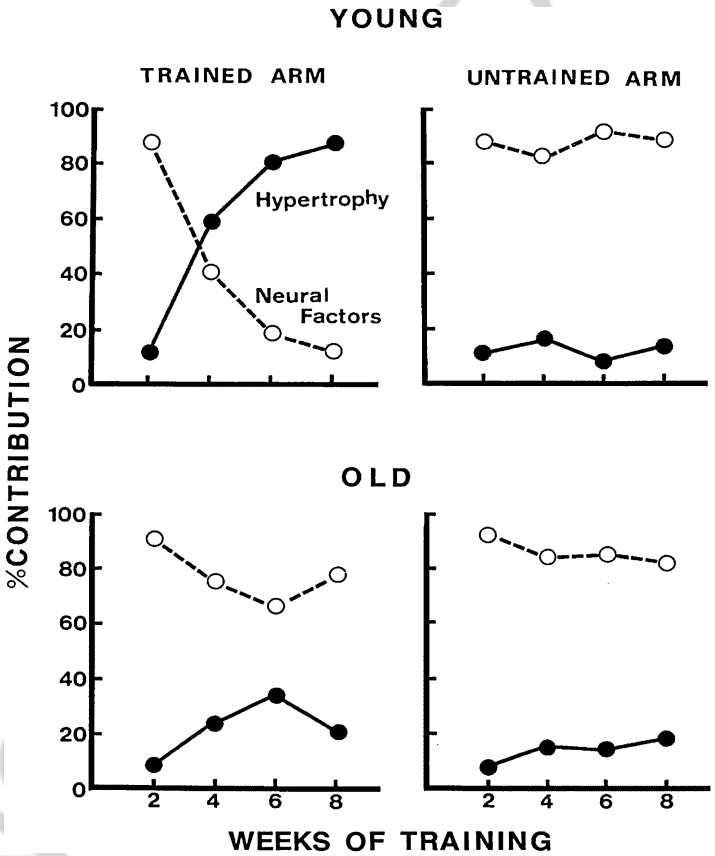

FIGURE 19.6 The time course of strength gain showing the percent contributions of neural factors and hypertrophy in the trained and contralateral untrained arms of young (**above**) and old (**below**) subjects. Reproduced from Moritani and deVries [130] with permission.

It is reasonable to assume that the nature of this cross education effect may entirely rest on the neural factors presumably acting at various levels of the nervous system which could result in increasing the maximal level of muscle activation.

A subsequent study [130] with older men (mean age of 70 years) demonstrated that the old subjects achieved the strength gain by virtue of neural factors as indicated by the increases in the maximal iEMG in the absence of hypertrophy. It is suggested that the training-induced increase in the maximal level of muscle activation (neural factors) through greater motor unit discharge frequency and/or motor unit recruitment may be the only mechanism by which the aged subjects increase their strength in the absence of any significant evidence of hypertrophy. These results are entirely consistent with those reported by Komi et al. [100], who demonstrated that changes in iEMG and force take place almost in parallel during the course of 12 weeks of training.

Häkkinen et al. [70] demonstrated that the subject trained with high-intensity loads of combined concentric and eccentric contractions showed an accelerated increase in force together with the parallel increase in iEMG during the first 8 weeks of training while showing minor muscle fiber hypertrophy. Greater muscle hypertrophy of both slow- and fast-twitch fibers was observed during the last 8 weeks of training that resulted in further strength gain with no significant change in the iEMG. Subsequent studies [35,37,67–71,86,101,145] confirmed these observations and provided evidence for the concept that in strength training the increase in voluntary neural drive accounts for the larger proportion of the initial strength increment and thereafter both neural adaptation and hypertrophy take place for further increase in strength, with hypertrophy becoming the dominant factor [70,129]. Seynnes et al. [174] have recently investigated the early skeletal muscle hypertrophy and architectural changes in response to a 35-day high-intensity resistance training (RT) program. It was clearly demonstrated that changes in muscle size are detectable after only 3 weeks of RT and that remodeling of muscle architecture precedes gains in muscle cross-sectional area. Muscle hypertrophy seems to contribute to strength gains earlier than previously reported.

Interestingly, there has been some evidence suggesting that strength development can be achieved by involuntary contractions initiated by electrical stimulation [51]. However, these experiments resulted in a considerably smaller strength gain than the values found in normal voluntary training. Since the motor pathways are probably minimally involved in electrical training, it seems likely that a training stimulus resides in the muscle tissue itself and hence the hypertrophic factor is the principal constituent for strength development. Subsequent studies [35,117] have indicated that the muscle training using electrically evoked contractions (80 maximal isometric tetani for 10 s) produced no increase in maximal voluntary strength, suggesting that neural drive has to be present in the training in order to produce large increases in maximal voluntary strength.

Strength training studies are typically carried out for a period of 5–20 weeks and have shown that the early increases in the voluntary strength are associated mainly with neural adaptation while hypertrophy begins to occur at the latter stage of training. Serious athletes, however, train over a period of many months or years. Häkkinen et al. [69] have studied the effects of strength training for 24 weeks with intensities

ranging between 70% and 120% of maximal voluntary force. The increase in strength correlated with significant increase in the neural activation (iEMG) of the leg extensor muscles during the most intensive training months along with significant enlargement of fast-twitch fiber area. During subsequent detraining, a great decrease in the maximal strength was correlated with the decrease in maximal iEMG of the leg extensors. It was suggested that selective training-induced hypertrophy could contribute strength development but muscle hypertrophy may have some limitations during long-term strength training, especially in highly trained subjects. This suggestion has recently been confirmed by a one-year training study indicating the limited potential for strength development in elite strength athletes [71].

On the other hand, there has been evidence indicating that lifelong high-intensity physical activity could potentially mitigate the loss of motor units associated with aging well into the seventh decade of life [155]. We have recently reported that the winner of an international contest to find the world's fastest drummer (WFD) can perform repetitive wrist tapping movements with one hand using a handheld drumstick at 10 Hz, much faster than the maximum tapping frequency of 5–7 Hz in the general population [58]. The WFD showed more rapid sEMG amplitude rise, earlier decline of sEMG activity, and more stable muscle activation time than the non-drummers (NDs) and ordinary drummers (ODs). In addition, there was a significant correlation between the EMG rise rate and the duration of drum training in the group of drummers (i.e., ODs and WFD). Our subsequent spike shape analysis revealed that the WFD had exceptional motor unit activity such as higher motor unit discharge rate, more motor unit recruitment, and/or higher motor unit synchronization to achieve extraordinary fast 10-Hz drumming performance [58]. Interestingly, Claflin et al. [32] have investigated the effects of movement velocity during resistance training on the size and contractile properties of individual muscle fibers from human VL muscles of young (20–30 years old) men and women and older (65–80 years old) men and women. In each group, one-half of the subjects underwent a traditional progressive resistance training (PRT) protocol that involved shortening contractions at low velocities against high loads, while the other half performed a modified PRT protocol that involved contractions at 3.5 times higher velocity against reduced loads. Contrary to their hypothesis, the velocity at which the PRT was performed did not affect the fiber-level outcomes substantially. They concluded that, compared with low-velocity PRT, resistance training performed at velocities up to 3.5 times higher against reduced loads is equally effective for eliciting an adaptive response in type 2 fibers from human skeletal muscle.

19.4 INVESTIGATION OF MUSCLE DAMAGE BY MEANS OF SURFACE EMG

Nearly everyone has experienced delayed-onset muscle soreness (DOMS) at some time; many have suffered from this common ailment on numerous occasions. DOMS is characterized by stiffness, tenderness, and pain during active movements and weakness of the affected musculature. A number of investigators have demonstrated

that the eccentric component of dynamic work plays a critical role in determining the occurrence and severity of exercise-induced muscle soreness [17,55,146]. It has been also demonstrated that type II fibers are predominantly affected by this type of muscular contraction [55,92].

It is well established that eccentric (lengthening) muscle action requires less oxygen and lower amount of ATP than concentric muscle action [36]. Both surface [98] and intramuscular EMG studies [140] have demonstrated that motor unit recruitment patterns are qualitatively similar in both types of contractions, but for a given MU the force at which motor unit recruitment occurs is greater in eccentric muscle action than in either isometric or concentric (shortening) muscle actions. Based on these findings and the results of EMG studies cited earlier, it is most likely that DOMS associated with eccentric component of dynamic exercise might be in part due to high mechanical forces produced by a relatively small number of active MUs which may in turn result in some degree of disturbance in structural proteins in muscle fibers, particularly those of high recruitment threshold MUs.

Despite the fact that DOMS is a well-known phenomenon in sports as well as working life, the exact pathophysiological mechanisms underlying it are still not well understood. According to Armstrong [6], a number of hypotheses may exist to explain the etiology and cellular mechanisms of DOMS. The following model may be proposed: (1) High tension, particularly associated with eccentric muscle action in the contractile and elastic system of the muscle, results in structural damage; (2) muscle cell membrane damage leads to disruption of Ca^{2+} homeostasis in the injured muscle fibers resulting in necrosis that peaks about 2 days post-exercise; and (3) products of macrophage activity and intracellular contents accumulate in the interstitium, which stimulate free nerve endings of group IV sensory neurons in the muscles leading to the sensation of DOMS.

Earlier EMG work by deVries [42,44] demonstrated that symptomatic soreness and tenderness seemed to parallel reduced EMG amplitude. DeVries has thus proposed the spasm theory: DOMS is caused by tonic, localized spasm of motor units as a result of a vicious cycle in which the activity induced ischemia in turn leads to further pain and reflex activity. Later workers have been unable to demonstrate any EMG activity in resting painful muscles [2]. Berry et al. [17] have shown that muscle soreness only occurred in muscles that had contracted eccentrically and did not occur at the time of greatest myoelectric signal changes. Although elevated EMG activity was accompanied with eccentric muscle action, there seems to exist dissociation in the time course of these two parameters [17,136,146]. The data of Newham et al. [146] have also demonstrated that eccentric muscle action has a long lasting effect on the muscle's ability to generate force after exercise. When the quadriceps muscle was stimulated at low frequencies, it was not able to develop the same force as it had under similar conditions before eccentric exercise. On the other hand, high-frequency stimulations elicited similar force before and after such exercise. The underlying mechanism of this so-called "low-frequency fatigue" has been postulated to be impaired excitation-contraction coupling [49] due to reduced release of calcium or possibly because of impaired transmission in the transverse tubular system, as a result of muscle damage in the period of ischemic activity.

19.4.1 Acute Effects of Static Stretching on Muscle Soreness

We conducted a series of studies to determine the physiological effects of static muscle stretching upon DOMS which was induced experimentally by heel raise (10 rep, 10 sets) with a 70% MVC equivalent weight attached on a universal shoulder press equipment or step test [17,136]. Electrophysiological parameters—for example, maximal mass action potential (M-wave), H-wave, and H/M ratio for determination of alpha motoneuron excitability—were measured during standing position (control), 24 h post experimental fatigue, and immediate post static stretching. Changes in the standing EMG signal up to 48 h post experimental fatigue were subjected to frequency power spectral analysis in order to determine the degree of muscle fatigue and resting action potential amplitude.

Surface EMG power spectral analyses revealed that (1) the experimentally induced DOMS was associated with significantly higher resting action potential amplitude and lower mean power frequency, suggesting the existence of some degree of muscle spasm and a possible synchronization of tonic motoneurons [17,136], and (2) static muscle stretching (three sets of 20-s duration) showed immediate and quite noticeable effects of restoring these electrophysiological parameters back towards the control level. Results on alpha motoneuron excitability indicated that there was very little change, if any, in the maximal amplitude of the H waves for the control leg while experimental leg post H wave was markedly reduced by static stretching. The mean relative reduction in the H/M ratio from pre- to post-test for the control and experimental legs was 0.63% ($p > 0.05$) and 21.5% ($p < 0.01$), respectively. These results are entirely consistent with earlier studies [43,44] and further suggest that the inverse myotatic reflex which originates in the Golgi tendon organs (GTO) may be the basis for the relief of DOMS by static stretching (see Fig. 19.7). Since H reflex

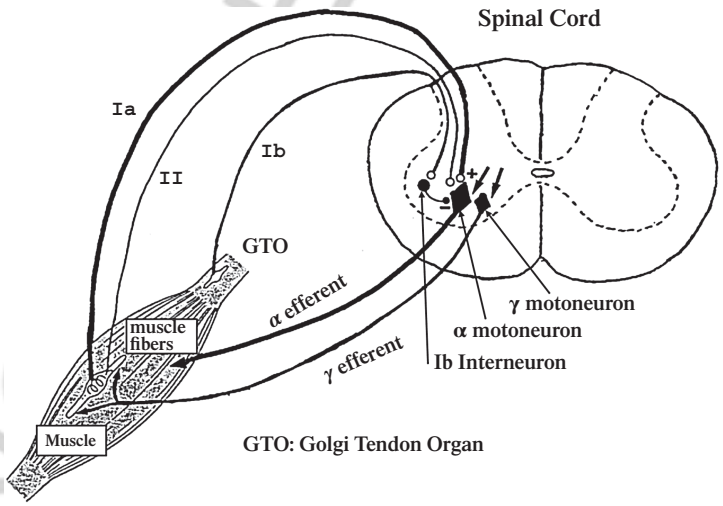

FIGURE 19.7 A simplified schematic representation of basic neural components involved in stretch reflex and Golgi tendon organ Ib inhibition.

involves tonic motor units [119], it is likely that the Ib afferent inhibitory effects from GTO could be mediated through the tonic MUs, thus reducing the evoked H wave amplitude.

We further investigated the physiological effects of static stretching upon DOMS in conjunction with the spinal alpha motoneuron pool excitability and peripheral muscle blood flow in seven healthy male subjects. All subjects performed heel raises (30 rep, 5 sets) with 20-kg load 24 hours prior to testing. Electrophysiological measurements included the Hoffman reflex amplitude (H amplitude) as a measure of spinal alpha motoneuron pool excitability. The directly evoked muscle action potential (M-wave) remained constant for each subject throughout the experiments. Blood flow measurement was performed by near-infrared spectroscopy (NIRS) with venous occlusion technique. In the experimental condition (EXP), those measurements were obtained before and after static muscle stretching (35 s, 3 sets) under experimentally induced muscle soreness. During the control condition (CON), the same measurements were made before and after standing rest for a period of 4 min. The order of the experimental treatments (EXP or CON) was chosen at random.

Figure 19.8 represents a typical set of H-reflex data obtained 24 h after experimentally induced muscle soreness prior to muscle stretching and immediately after muscle stretching. The data clearly indicated that, for the same elicited M-wave, H-reflex amplitude was considerably reduced after muscle stretching. Group data demonstrated that the static stretching brought about a statistically significant reduction in the H/M ratio (23.5%, $p < 0.01$) of the EXP conditions while no such changes were observed in CON trials. These changes were accompanied by nearly 78.5% increase ($p < 0.01$) in blood flow after stretching the gastrocnemius muscle with the experimentally induced soreness. These finding was entirely consistent with earlier studies, suggesting that the inverse myotatic reflex (Ib inhibition) may be the basis for the relief of muscle soreness by static stretching. The increase in blood flow after stretching found in the present study suggested that static stretching could bring about a relief of spasm, which could have caused local muscles ischemia and pain (see Fig. 19.9). Our data strongly suggest that static stretching plays a significant role in

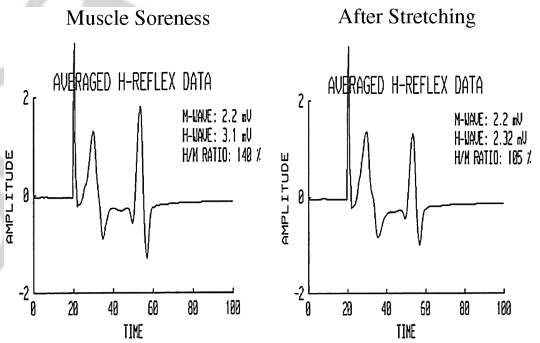

FIGURE 19.8 A typical set of H-reflex data during the experimentally induced muscle soreness and after static muscle stretching.

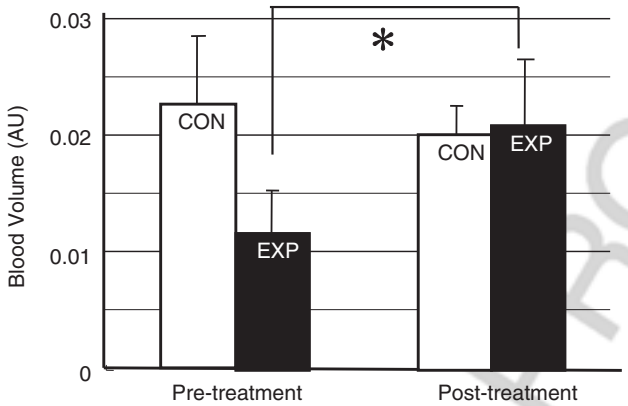

FIGURE 19.9 Blood flow (volume, arbitrary units) changes during the experimentally induced muscle soreness and after static muscle stretching.

relief of DOMS by reducing spinal motoneuron pool excitability and enhancing muscle blood flow.

19.4.2 Fusimotor Sensitivity After Prolonged Stretch-Shortening Cycle Exercise

Evidences have been presented that both short- and long-duration fatiguing exercises lead to deterioration in neuromuscular performance (exhaustive exercise and intensive effort [147], as well as long-lasting low-intensity effort [8]. The underlying mechanisms which mediate modifications of reflex activity after repeated stretch-shortening cycle (SSC) exercise remain an open question. To elucidate which factor is more influential on the stretch reflex reduction after repeated SSC, T-reflex was employed in this study. T-reflex, which is elicited by tendon tap and shares reflex pathway (e.g., alpha-motoneuron pool) with H-reflex except for spindle-mediated fusimotor component, can help to factor out modification of spindle and fusimotor activity when compared to H-reflex. We therefore performed comparison of T-reflex and H-reflex of the triceps surae before, immediately after, 2 h after, and 24 h after two hours of exhaustive running in terms of EMG activity and impact force on the tendon. Five consecutive EMG responses of T-reflex and H-reflex were averaged and analyzed for peak-to-peak amplitude. Results revealed that immediately after the running, T- and H-wave amplitudes were significantly depressed while maximal M-wave remained constant. On the other hand, 2 h after the running, H-reflex amplitudes showed clear-cut rising ($p < 0.001$) and, by contrast, the T-reflex amplitude did not show such a significant elevation. All the EMG amplitudes returned to the pre-exercise level in 24 h.

The impact force on the Achilles tendon (coefficient of rebound force) showed a reduction immediately after the running ($p < 0.05$) and recovered in 24 h. The difference between H- and T-reflex amplitudes 2 h after the exhaustive running might suggest that the sensitivity of fusimotor activity was reduced by 2 h of running. Furthermore, the reduced impact force might reflect deteriorated stiffness regulation

of muscle–tendon complex. This may also suggest the degradation of spindle activity. These results support the hypothesis that stretch reflex reduction might be attributed to disfacilitation of alpha motoneuron pool caused by degradation of spindle-mediated fusimotor support and/or fatigue of the muscle spindle itself due to the possible depletion of intrafusal muscle glycogen [8,9].

19.5 RELATIONSHIPS BETWEEN EMG FEATURES AND MUSCLE FIBER FEATURES

Fiber composition is usually investigated by biopsy and histochemical analysis. Johnson et al. [90], analyzed 36 muscles during an autopsy study on six male cadavers (aged 17–30 years) and provided the percentage of type I and type II fibers found in each muscle. This is probably the most cited paper about this issue, but other authors focused their efforts on fewer muscles and a greater number of subjects producing more reliable data [56,60,151,183,192].

The information obtained from bioptical specimens are actually not representative of the muscle as a whole, thus the need of repeated sampling decreases the subjects' compliance supporting the validation of alternative noninvasive methods of fiber type estimate. Hence the main issue is to assess if it is possible to extract, from a wider portion of muscle and using superficial electrodes, information related to the histological properties of human muscles.

A number of physiological parameters were considered in the past to be related to muscle fiber types and motoneurons for their noninvasive assessment. The amplitude estimators (ARV, RMS) and the power spectrum estimators (MNF, MDF) of the recorded surface EMG signals (see Chapters 4, 5, and 10), and the muscle fiber conduction velocity (CV, see Chapter 5) were shown to be related to the pH decrease due to the increment of metabolites produced during a fatiguing contraction.

The "size principle" described by Henneman and Mendell [74] was first proposed based upon results from cat motoneurons, strong evidence has been presented that in muscle contraction there is a specific sequence of recruitment in order of increasing motoneurons and motor unit size [39,106,126,138]. Earlier studies have demonstrated in humans that, for a muscle group with mainly type I slow-twitch fibers (adductor pollicis), rate coding plays a prominent role in force modulation [126,138]. On the contrary, in a muscle group composed of both type I and II fast-twitch fibers, motor unit recruitment seems to be the major mechanism for generating extra force above 40% to 50% of MVC [39,106,138].

Similarly, muscle fatigability during a sustained isometric contraction, as reflected by the progressive recruitment of new motor units and by the increase of iEMG in time, is also dependent upon muscle fiber type composition (see Fig. 19.10). Earlier EMG studies indicated that ARV of the surface EMG increased progressively as a function of time during sustained muscular contraction with a constant force output [43,135]. The work of deVries [43] and Viitasalo and Komi [187] suggested that EMG fatigue curves (iEMG versus time) could provide a measure of motor unit fatigability. Furthermore, Komi and Tesch [99] demonstrated that human muscles

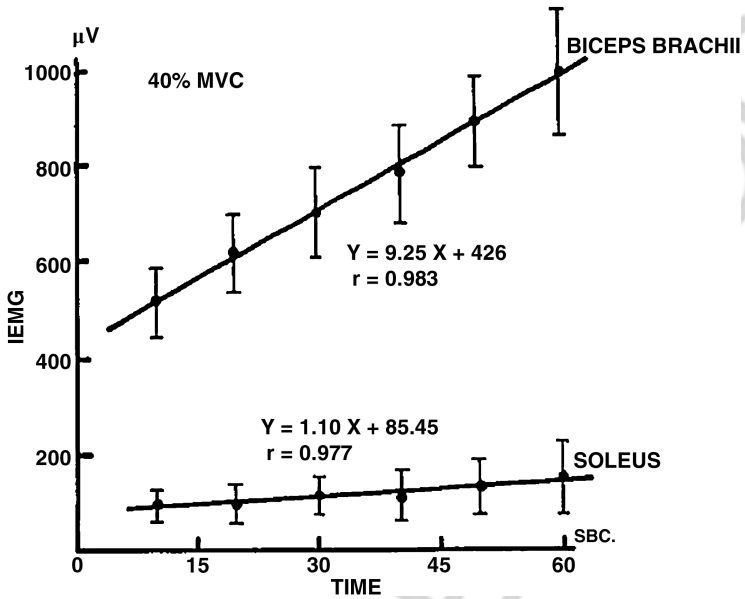

FIGURE 19.10 The mean iEMG data as a function of time at 40% of maximal voluntary contraction (MVC) for the biceps brachii and soleus muscles. Reproduced from Moritani [134] with permission.

characterized by a predominance of fast-twitch fibers showed a greater susceptibility to fatigue, and this was reflected by a sharp decline in force output as well as by a pronounced decrease in the mean power frequency of EMG power spectrum. These data suggested a possibility that muscle fiber features could be well represented by EMG signal characteristics during contractions at varying force levels and muscle fatigue. Figures 19.10 and 19.11 represent a typical set of iEMG and power spectra obtained from the biceps brachii and soleus muscles during sustained isometric contractions at 40% MVC, respectively.

Earlier studies regarding changes of EMG power spectral parameters, such as mean or median spectral frequency (MNF, MDF) and the level of muscle contractions, are somewhat contradictory. For example, a series of studies by Petrofsky and Lind [153,154] showed no systematic relationship between tension levels and MNF for hand grip muscles. Hagberg and Ericson [65] demonstrated in the elbow flexors that MNF increased with contraction strength at low contraction levels but became independent of contraction level above 25–30% MVC, whereas Muro et al. [143] and Broman et al. [23] demonstrated almost linear increases in MNF with force of contraction up to near MVC levels. These different results might be at least in part due to differences in the muscle groups studied, electrode size, and interelectrode distance which could act as various low-pass filters, muscle fiber types [138,141], and underlying motor unit firing statistics and action potential conduction velocity [23].

Figure 19.12 shows EMG signal features demonstrating the influence of electrode size and interelectrode distance upon the amplitude and frequency components (see

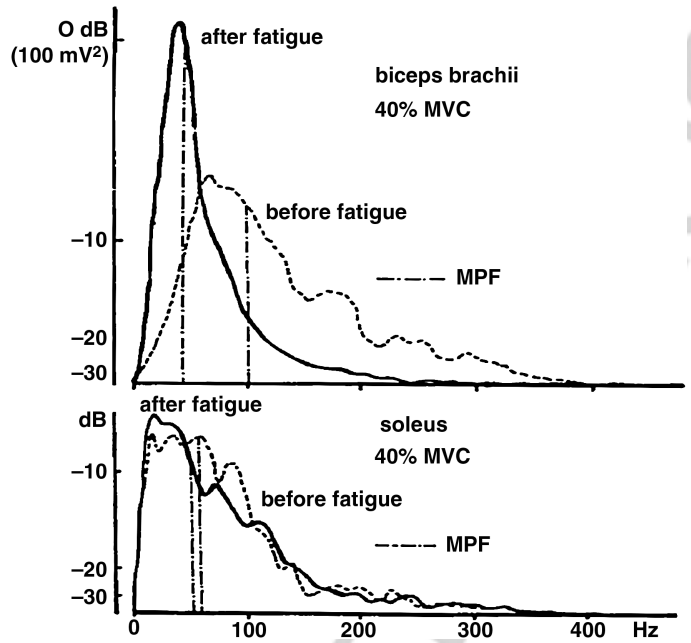

FIGURE 19.11 A typical set of sEMG frequency power spectral changes for the biceps brachii and soleus muscles before and after muscle fatigue at 40% of MVC. Reproduced from Moritani [134] with permission.

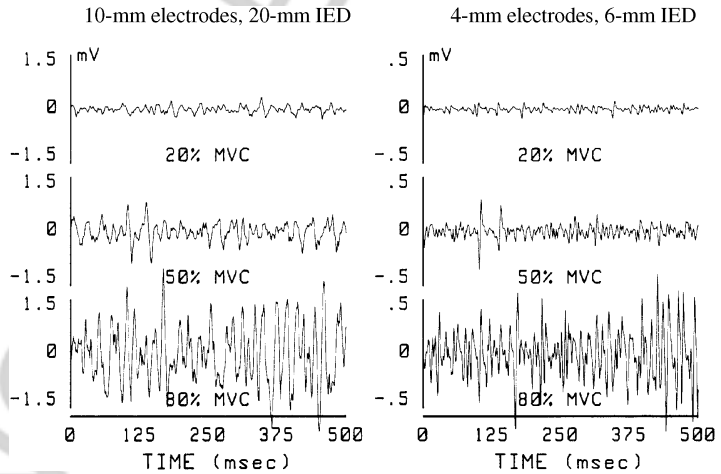

FIGURE 19.12 Biceps brachii muscle EMG signal features obtained simultaneously from large (10-mm electrode diameter, 20-mm interelectrode distance) and small (4-mm electrode diameter, 6-mm interelectrode distance) bipolar silver/silver chloride electrodes.

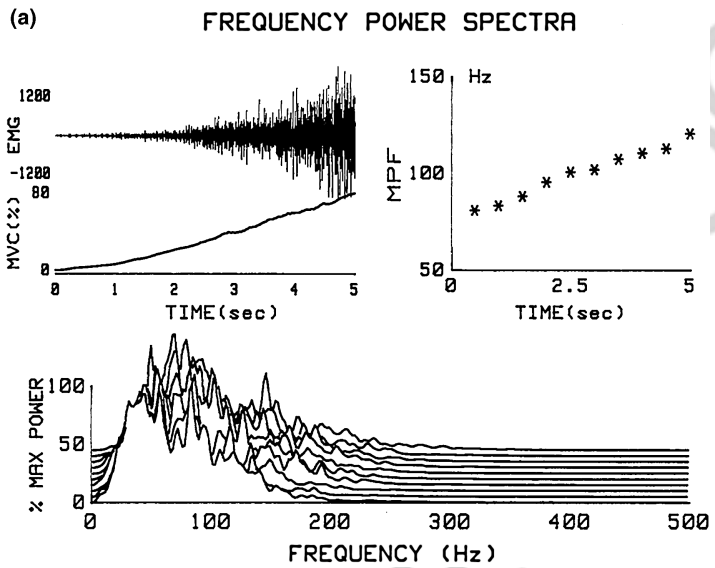

FIGURE 19.13 A typical set of computer outputs showing raw sEMG, force, normalized power spectra and mean power frequency (MPF) plots during ramp force output for a normal subject **(a)** and a highly trained power lifter **(b)**. Reproduced from Moritani et al. [137] with permission.

also Chapter 2). These data were obtained from the biceps brachii muscle contracting at three levels of MVC while recording the EMG signals simultaneously from large (10-mm electrode diameter, 20-mm interelectrode distance) and small (4-mm electrode diameter, 6-mm interelectrode distance) bipolar silver/silver chloride electrodes. Note the marked differences in both amplitude and frequency features of the signals (see also Chapters 2 and 3). Our data demonstrated that surface EMG with larger electrodes and wider interelectrode distance revealed no systematic increases in MNFs, but showed progressive increases in EMG amplitude. The power spectral data obtained from the same muscle with small electrodes and narrow interelectrode distance demonstrated a large and significant increase in MNFs during the ramp contraction up to 80% of MVC [132] (see Fig. 19.13a). For comparison, the results obtained from one of the United States representatives for the 1984 World Power Lifting championship are shown in Fig. 19.13b. These results are entirely consistent with those reported by Petrofsky and Lind [153,154].

In the last decade, however, it was shown that, even if conduction velocity, amplitude, and MNF of sEMG are somehow related to the type of recruited motor units, a large number of additional factors blur the phenomenon. A more extensive discussion of these factors is provided in Chapter 10. Amplitude and spectral features are actually related to the number and type of recruited motor units, whose relative position and depth within the muscle cannot be assessed, and no correction for the filtering effect of the volume conductor thickness can be adopted at this time. Thus the filtering effect due to the tissue between each active motor unit and the recording

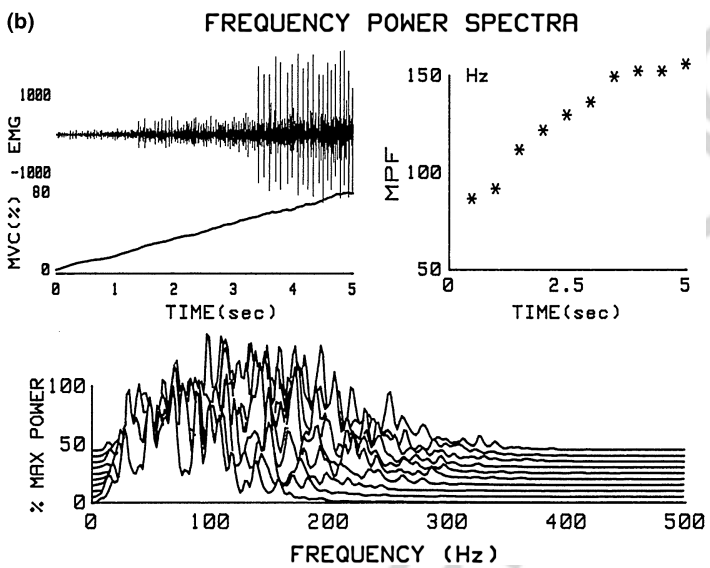

FIGURE 19.13 (*Continued*)

site plays a role which can be accounted for only in particularly controlled experimental conditions. Other factors are discussed in Chapter 10.

In the same way the frequency content of the surface EMG signal is somehow related to the recruited motor unit pool and it was demonstrated, both in animal model [108] and in humans [14,60,99,115], that its time course during fatiguing contractions shows a steeper decrease if the muscle is characterized by more fast than slow fibers. Nevertheless, even in this case, a recent debate described a number of confounding factors (depth of motor unit within the volume conductor, properties of the volume conductor layers, pinnation angle, motor unit synchronization, detection system geometry, among others) which strongly blur the relationship between sEMG properties and muscle fiber type constituency [53]. For these reasons (even if with particular caution), muscle fiber conduction velocity (CV) seems the most suitable candidate to relate, under a physiological framework, the modifications in EMG signals with both the motor unit pool histochemical characteristics and cross-section fiber size [127].

Hopf et al. [78] electrically evoked single twitches in human biceps brachii muscle estimating both the contraction times (defined as the time from the onset of the deflection to the peak) and the muscle fiber conduction velocity (using invasive technique). A negative correlation between contraction times of the elicited twitches and muscle fiber conduction velocity was found (see Fig. 19.14). In the same direction, Sadoyama et al. [169] showed a strong correlation between different fiber type relative area in biopsies and conduction velocities estimated during voluntary contractions from two different groups of athletes (sprinters vs distance runners) (see Fig. 19.15). Identical findings were observed in the work of Rainoldi et al. [161] in

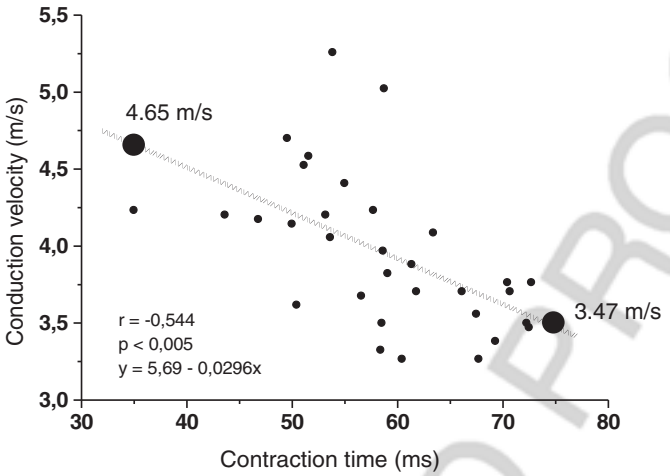

FIGURE 19.14 Diagram showing the first experimental evidence of correlation between muscle fiber contraction times and conduction velocity in electrically evoked contractions. Modified from Hopf et al. [78].

which CV rate of changes during fatiguing contractions were found to be significantly different between sprinters and long-distance runners, matching the expected fiber type composition. A further confirmation of such an approach was provided in the work of Rainoldi et al. [160], where surface EMG signals were recorded from the vastus medialis longus, vastus medialis obliquus, and VL muscles during isometric

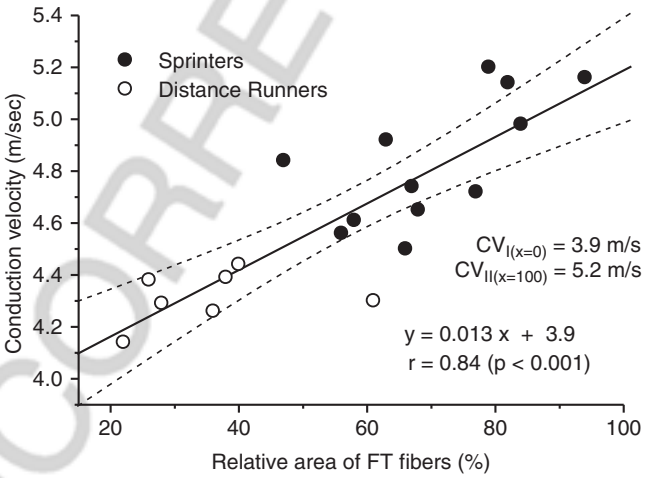

FIGURE 19.15 Correlation between relative areas of fast twitch fibers and conduction velocities estimated during voluntary contractions from two different groups of athletes (sprinters versus distance runners) which can reasonably be considered at the edges of the possible phenotype distribution. Modified from Sadoyama et al. [169].

knee extension contractions; differences in CV initial values among the three muscles were found in agreement with histological evidences provided in the literature.

As known, oxygen distribution in exercising skeletal muscle is regulated by oxygen transport in the blood vessels, as well as by oxygen diffusion and consumption in the tissue [110], hence the amount of oxygen supply is one of the pivotal factor affecting contraction and causing fatigue [77]. In their work, Casale et al. [29] concluded that acute exposure to hypobaric hypoxia did not significantly affect the muscle-fiber membrane properties (no peripheral effect) but impacted on motor-unit control properties (central control strategies for adaptation). Hence, the lack of oxygen induced by high altitude induced central effects resulting in the recruitment of more MUs oxygen-independent than those used at sea level to maintain the requested force task. This finding demonstrated that it is possible to assess, also in clinical settings, if variations in sEMG manifestations of fatigue are central or peripheral adaptations by means of two different contraction modalities, namely voluntary or electrically induced [30]. To reach the same goal—that is, to distinguish between peripheral and central effects of fatigue—a different approach was proposed by Mesin et al. [124] in which a bidimensional vector based on CV estimate and sEMG signal fractal dimension provided selective sensitivity to peripheral or central fatigue.

Moreover, estimates of CV initial values and of CV rates of change (that is, myoelectric manifestations of fatigue) reflect differences, with respect to a control group, due to conditioning [28,95,123,161,163] and pathologies [30,52,111,120,152, 162], aging [28,121] as evidence of changes/alterations in fiber types.

A recent finding further confirmed the role of oxygen in modulating fatigue and motor unit recruitment in a group of patients affected by chronic obstructive pulmonary disease (COPD). Such a pathology is characterized by persistently poor airflow [31] and by a number of side effects. Among others, lower limb muscles of COPD patients showed a significant redistribution of fiber type ratios characterized by a reduction in the proportion of type I fibers with a global shifting towards type II fiber type [114,188]. In a recent protocol [165] based on a prolonged isometric contraction of quadriceps at 70% of MVC, COPD and healthy age-matched subjects were compared in terms of myoelectric manifestations of fatigue (Fig. 19.16). The greater proportion of type II muscle fibers in COPD led to a greater rate of EMG fatigue measured as a greater decrease of CV over time.

A specific protocol was recently proposed as a noninvasive technique to distinguish between two extremely different phenotypes highlighting the effect of oxygen availability in endurance and power trained athletes. As described by Rainoldi and Gazzoni [159], intermittent (3 s of contraction and 1 s of rest in between contractions) and continuous contractions (no rest and same total workload) were proposed to a group of endurance and a group of power trained athletes. Findings showed that while no differences were observed in power-trained athletes, passing from intermittent to continuous contractions, fatigue (estimated by the normalized slope of CV) increased by 200% in the endurance group due to the lack of availability of oxygen.

All these findings seem to confirm that, in carefully controlled experimental conditions, it is possible to correlate modifications of sEMG variables with different muscular phenotypes or muscular adaptation processes. Such an approach seems now

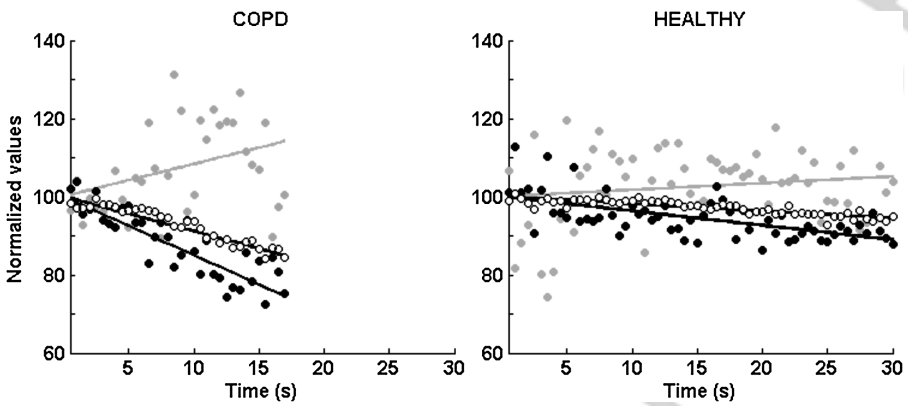

FIGURE 19.16 Fatigue plot diagrams of two subjects: COPD patient (**left panel**) and healthy adult (**right panel**). Time courses of ARV (*gray circle*), MNF (*black circle*), and CV (*white circle*) are represented for each epoch (0.5-s length) of the isometric contraction at 70% of MVC. Each variable is normalized with respect to its initial value, and the slope of the regression line represents an index of myoelectric fatigue: The greater the slope of sEMG variables, the greater the manifestations of fatigue. Modified from Rinaldo et al. [165].

finally ready to be used as a further tool for neuromuscular adaptations monitoring since it is noninvasive and repeatable.

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