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Muscle Strength and Its Development
New Perspectives

Roger M. Enoka

Departments of Exercise & Sport Sciences and Physiology, University of Arizona, Tucson, Arizona, USA

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Summary

Skeletal muscle undergoes substantial adaptation when it is subjected to a strength training regimen. At one extreme, these effects are manifested as profound morphological changes, such as those exemplified by bodybuilders. However, it is possible to increase strength without any change in muscle size. This dissociation underscores the notion that strength is not solely a property of muscle but rather it is a property of the motor system. The nervous system seems to be of paramount importance for the expression and development of strength. Indeed, it is probable that increases in strength can be achieved without morphological changes in muscle but not without neural adaptations. This review focuses on the role of the nervous system in the development of strength. In the strength literature, 3 topics exemplify the importance of the nervous system in strength development. These 3 topics are considered in detail in the review: electromyostimulation, cross-training effects, and EMG-force relationships. Evidence is presented from several different paradigms emphasising the significant contribution of neural mechanisms to the gains in strength with short term training. Although little is known about the specific neural mechanisms associated with strength training adaptations, the literature emphasises that the measure of human performance known as strength can be influenced by a variety of neurophysiological processes.

Strength is a physiological concept used to refer to one of the output capabilities of the motor system. Like the concepts of fatigue and power, the notion of strength is not something that is limited to the laboratories of physiologists and exercise scientists, rather it exists in the daily activities of both the scientist and the layman. Despite the breadth of interest in this topic, the literature on strength and its development often seems quite contradictory and confusing. Perhaps the major reason for this confusion is the blurring of terminology and concepts among the different groups interested in strength development. If we are to synthesise and advance our knowledge on strength, however, it is critical that we share a concern for the precision of vocabulary, and hence the underlying ideas, related to this topic. Unfortunately, much of the jargon associated with the practice of strength training has permeated the strength literature, to the extent that few scientists can agree on a definition of strength. The lack of precision associated with this sharing of vocabulary by the practitioner and the scientist is unfortunate because it hinders scientific progress on the topic.

In order to evaluate ideas on strength development, it is necessary that we have as a basis a precise definition of strength. What exactly do we mean by the term 'strength'? We can all agree that strength is a measure of human performance. There, however, the agreement probably ends. Weightlifters might define strength in terms of the maximal weight that can be lifted. In contrast, scientists tend to be more specific and describe capabilities such as static strength, dynamic strength, isometric strength, isokinetic strength, isotonic strength, explosive strength and muscle strength. This is clearly an undesirable situation because strength is used in so many contexts that it has become a vague and meaningless term.

Some investigators have recognised this shortcoming and have suggested '... that the term strength be employed to refer to the maximal force a muscle or muscle group can generate at a specified velocity' (Knuttgen & Kraemer 1987). While this restricted definition is an improvement over the range available in the literature, it raises 2 concerns: (a) Without using invasive techniques (e.g. Komi et al. 1987) or complicated EMG-to-force conversion procedures (e.g. Hof & Van den Berg 1981a,b,c,d), how is it possible to measure muscle force?; and (b) if the measurement is made 'at a specified velocity' then strength will be influenced by the dynamic characteristics of muscle described by the force-velocity relationship (Hill 1938). The definition of strength should be one that allows us to make a simple non-invasive measurement and one that minimises the physiological factors that affect the measurement. In accordance with these criteria, Atha (1981) has proposed that strength be defined '... as the ability to develop force against an unyielding resistance in a single contraction of unrestricted duration.'

Based on this simple definition, strength is regarded as the maximal isometric activation of the motor system (see also McDonagh & Davies 1984; Milner-Brown et al. 1986). In the interest of simplicity, the measurement of strength is generally confined to the activity about one joint at a time (cf. Andrews et al. 1987). Although this definition establishes strength as one of the simplest measurements of human performance possible (i.e. isometric and single joint), it is nonetheless the consequence of a complicated interaction among all neuromuscular elements. To a first approximation, these elements can be categorised as neural, muscular and mechanical factors (Enoka 1988b; Rutherford & Jones 1986). The neural factors involve those associated with motor unit activity: recruitment and modulation of discharge frequency. The muscular factors are the size of the muscle(s), as represented by cross-sectional area, and muscle length at the time of measurement. Since the force which an individual exerts on a load depends on the torques acting on the system, particularly muscle torque, the mechanical factors include the moment arms associated with the different forces. Given the maximal-isometric-force definition of strength, it is apparent that a training-induced increase in strength may be caused by several different factors.

The motor system is exquisitely flexible and capable of a great range of outputs. One classic way

of characterising the range of outputs is within the force-length-velocity domain (Hill 1938; Ralston et al. 1947), in which the force that a muscle exerts depends on its length and the rate at which the length changes (i.e. velocity). There is an optimal length at which a muscle can exert its maximal force and, furthermore, the maximal force is affected by whether or not muscle length is constant. The maximal isometric force definition of strength represents one unique point in this domain; the location where muscle length is optimal and not changing. With this standardisation, the only way to evaluate the efficacy of training procedures and devices for increasing strength is to measure strength as a maximal isometric contraction. Thus, strength is not the ability to lift a heavy weight (e.g. Olympic or power weightlifting; Enoka 1988a) or the maximal torque exerted on an isokinetic device. Strength will influence the performance of such tasks but then so will other factors, such as the force-velocity relationship and the timing of activity among different muscles.

The literature on strength and its development is extensive, ranging from the study of training techniques (e.g. electromyostimulation, variable load devices) and their optimal prescription, to the mechanisms triggering protein accumulation, to the neural adaptations that accompany strength training. This review largely focuses on the role of the nervous system in strength development and does not consider the hyperplasia-hypertrophy controversy or the effects of strength training on muscle ultrastructure, contractile proteins or fibre types (for recent reviews on these latter topics: Hoppeler 1986; Matoba & Gollnick 1984; McDonagh & Davies 1984; Swynghedauw 1986; Taylor & Wilkinson 1986). The purpose of this review is to consider 3 statements: (a) strength can be increased by using artificial activation (electrical stimulation) of muscle; (b) the strengthening of one limb increases the strength of the inactive contralateral limb; and (c) strength can be increased without any change in muscle size. The evidence considered in this review will demonstrate that each of these statements is true and, furthermore, that the mechanisms underlying each statement largely remain unknown. In order to achieve these goals, the review focuses on 3 topics: electromyostimulation, cross-training effects, and EMG-force relationships. These topics underscore the complexity of this measure we call strength.

1. Electromyostimulation

The nervous system is known to communicate with muscle at 2 levels. At one level this communication is rapid and electrical in nature, while at the other level it is much slower and has a chemical basis. Both forms of interaction are thought to be important in the developmental and adaptive capabilities of nerve and muscle. The slow chemical interaction comprises neurotrophic transport systems that translocate, in both directions, biochemical material between the cell bodies of neurons and muscle fibres (Alvarez & Torres 1985; Wilson & Stone 1979). Little is known about the role of these mechanisms in the adaptive response of the motor system to strength training (Jasmin et al. 1987, 1988). In contrast, investigators tend to focus on the rapid electrical interaction between nerve and muscle which involves the generation and propagation of action potentials and their measurement as an EMG (Enoka et al. 1988; Loeb & Gans 1986; Rankin et al. 1988).

Scientists have known for about 200 years (Galvani 1792; Jallabert 1748) that it is possible to excite muscle by passing an electric current across the muscle or its peripheral nerve. This capability has been exploited in rehabilitation medicine for most of the twentieth century (Geddes 1984) and as a supplement to normal training procedures for the last 2 decades (Kots 1971; Kots & Hvilon 1971; Kraemer & Mendryk 1982). Such artificial activation of muscle is known as electromyostimulation. The efficacy of electromyostimulation is based on the assumption that the output of the motor system (i.e. excitation to muscles) is insufficient and needs to be supplemented by artificial means. This rationale seems reasonable for rehabilitation paradigms where the function of the nervous system may have been compromised by a traumatic event or some disease process (Bajzek & Jaeger 1987;

Valencic et al. 1986). In contrast, the validity of this insufficiency assumption seems questionable for healthy individuals.

1.1 Effects on Strength

Most recent studies, but not all (Davies et al. 1985; Mohr et al. 1985), have shown that it is possible to induce strength gains with electromyostimulation techniques. This adaptation has been accomplished in both hypotrophic (Godfrey et al. 1979; Wigerstad-Lossing et al. 1988; Williams et al. 1986) and healthy muscle (Boutelle et al. 1985; Cabric & Appell 1987; Cabric et al. 1987, 1988; Cannon & Cafarelli 1987; Currier et al. 1979; Currier & Mann 1983; Duchateau & Hainaut 1988; Eriksson et al. 1981; Laughman et al. 1983; Romero et al. 1982; Selkowitz 1985; Stefanovska & Vodovnik 1985). Furthermore, these increases in strength have been attained with a variety of stimulus parameters (fig. 1) that range from conventional trains of low frequency rectangular pulses (25 to 200Hz, fig. 1a; e.g. Cabric & Appell 1987; Cabric et al. 1987, 1988; Duchateau & Hainaut 1988; Stefanovska & Vodovnik 1985) to trains of high frequency sinusoidal pulses that are modulated at low frequencies (fig. 1c; e.g. Currier & Mann 1983; Laughman et al. 1983; Moreno-Aranda & Seireg 1981a,b,c). The general conclusion to emerge from these studies is that the strength gains associated with electromyostimulation procedures are similar to, but not greater than, those that can be achieved with normal voluntary training. However, since most studies have been of short duration (i.e. less than 5 weeks) and confined to the period when neural adaptations are thought to underlie the increases in strength (Moritani & deVries 1979), it is unclear whether the strength gains with long term electromyostimulation would be superior to voluntary training.

1.2 Electromyostimulation Protocols

Given the success achieved with these techniques, it seems reasonable to assess their relative effectiveness in producing increases in strength.

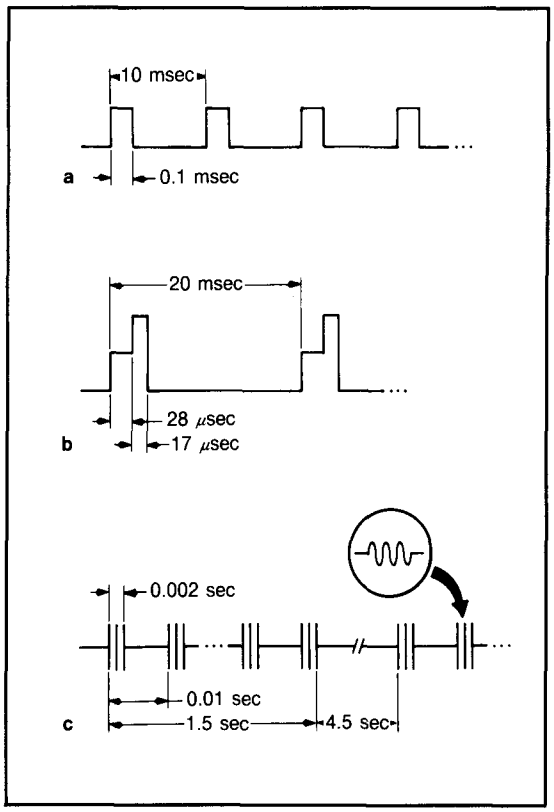

Fig. 1. Selected stimulus regimens used in electromyostimulation. (a) A conventional train of low frequency (100Hz) rectangular stimuli with a pulse width of 0.1 msec; (b) a more complicated low frequency (50Hz) regimen in which the magnitude of the stimulus pulse (width = 0.045 msec) doubles at about the midpoint of its duration. This pattern is produced by the 'high volt galvanic stimulator' (Mohr et al. 1985); (c) a pattern of high frequency stimulation (10 kHz) that is modulated at a low frequency (100Hz). Moreno-Aranda and Seireg (1981a,b,c) have suggested this comprises the optimal electromyostimulation protocol.

This is difficult to evaluate because many of the details associated with electromyostimulation and training protocols, with few exceptions (e.g. Currier & Mann 1983; Laughman et al. 1983; Selkowitz 1985), are not provided. The absence of such information raises doubts about whether or not the failure to observe an increase in strength with electromyostimulation was due to an inadequate training stimulus (Davies et al. 1985; Mohr et al. 1985). In the study by Mohr et al. (1985) the absence of a training effect may well have been due to an in-

sufficient excitation of nerve fibres; that is, the stimulus pulse had a 2-part intensity and a width of 45 μsec (fig. 1b). A pulse duration of 0.5 to 1.0 msec seems optimal for percutaneous stimulation (Hultman et al. 1983; Ranck 1975).

Nonetheless, considerable attention has been directed towards identifying the optimal features of electromyostimulation for inducing strength gains. Interest has spread to both academic and commercial settings and as a result there are a number of commercially available products. In general, however, the commercial products offer less flexibility in manipulating the stimulus parameters making it difficult to determine the combination of parameters (e.g. frequency, pulse width, duration, current) which produces the best result. From the research literature, it is apparent that the criteria that need to be considered in the evaluation of electromyostimulation protocols include:

1. The minimisation of pain and unpleasant sensations is best accomplished by the use of high stimulus frequencies (Moreno-Aranda & Seireg 1981a) and narrow pulse widths (Vodovnik et al. 1965).

2. Maximum force is elicited by frequencies of 50 to 120Hz (Davies et al. 1985; Marsden et al. 1983; Miller et al. 1981).

3. Since the refractory period of the sarcolemma for action potential propagation is 2 to 3 msec, the time between stimuli should be at least 3 msec (Miller et al. 1981). Apparently, however, the refractory period increases with fatigue (Borg et al. 1983) and hence there should be some latitude in the interstimulus interval.

4. The stimulus protocol should comprise a duty cycle (i.e. an active-rest cycle) which will minimise the effects of fatigue (Duchateau & Hainaut 1985).

5. The electrical signal should periodically reverse polarity in order to reduce electrode polarisation (Moreno-Aranda & Seireg 1981a).

6. The magnitude of the elicited force is affected by electrode area and location (Moreno-Aranda & Seireg 1981b).

Based on these considerations, Moreno-Aranda and Seireg (1981a,b,c,) have proposed that the optimal electromyostimulation protocol should have the characteristics shown in figure 1c. In essence, this procedure is based on a high frequency stimulation (10 kHz) that is modulated (i.e. turned on and off) at a lower frequency (100Hz). Using this stimulus, Moreno-Aranda and Seireg reported that the optimum protocol involved the application of the stimulus for 1.5 seconds every 6 seconds for 60 seconds followed by a 60 second rest. The independent evaluation of this protocol by other laboratories would be helpful, especially if applied to elite athletes for extended periods of time (i.e. greater than 5 weeks).

1.3 Physiological Basis of Electromyostimulation

It has been known for some time that when a muscle is activated and the force it exerts is increased, the motor units belonging to the muscle are activated in a rather fixed sequence. This behaviour is known as the orderly recruitment phenomenon (Denny-Brown 1949). The mechanisms underlying this phenomenon include motoneuron size, the organisation of synaptic input on to motoneurons, and the biophysical properties of the motoneuron membrane (Enoka & Stuart 1984; Gustafsson & Pinter 1985). According to this concept of orderly recruitment, motor units are recruited in a sequence that progresses from low threshold (small) to high threshold (large) units.

Mammalian motor units can be characterised with a quadripartite classification scheme (Stuart et al. 1984). Based on a physiological test of fatigability and the profile of an unfused tetanus, it is possible to distinguish four types of motor units: S, slow contracting; FR, fast contracting and fatigue resistant; F(int), fast contracting and intermediate fatigability; FF, fast contracting and fatigable (Burke et al. 1973; McDonagh et al. 1980a,b). If we measured any single physiological or biochemical property of motor units, we would find that the values for the parameter would be spread along a continuum for all motor units. However, when the values for several parameters are considered together, motor units tend to cluster into 4 distinct groups (Botterman et al. 1985). According

to the concept of orderly recruitment, type S motor units are activated first and type FF units last. Motor unit types are not recruited as distinct populations, however, but rather there is considerable overlap among the groups during voluntary activation of the muscle. Because of this overlap it is not possible to activate just slow twitch muscle fibres without any fast twitch fibres; both type S and type FR motor units are recruited at low forces because of the substantial overlap in the recruitment ranges of these types (fig. 17-16 in Stuart & Enoka 1983).

When muscle is artificially activated, as with electromyostimulation, the involvement of motor units is quite different from that underlying natural activation. Although the electrodes are placed over the muscle, electrically activating a muscle that has an intact peripheral nervous system results in excitation of intramuscular branches of the nerve and not the muscle fibres directly (Hultman et al. 1983; Mortimer 1984; Moulds et al. 1977). This is because muscle fibres are much less excitable than nerve branches. With activation of the nerve branches, action potentials are elicited in axons, propagated bidirectionally along the axon, transmitted across the neuromuscular junctions, and then propagated along the muscle fibres to activate the contractile machinery. Electromyostimulation, therefore, does not bypass the peripheral nervous system if it is intact. The order of motor unit activation with electrical stimulation depends on at least 3 factors: (a) the diameter of the motor axon (Erlanger & Gasser 1937); (b) the distance between the axon and the active electrode (McComas et al. 1971; Mortimer 1984); and (c) the effect of input to motoneurons from cutaneous afferents that have been activated by the artificial signal (Garnett & Stephens 1981; Kanda et al. 1977). Together, these 3 factors produce a recruitment order during electromyostimulation that is quite different from voluntary activation.

The effect of axon diameter is such that the largest axons have the lowest activation threshold with electrical stimulation (Clamann et al. 1974; Eccles et al. 1958). If an electric current is passed across a muscle, the largest diameter axons will be recruited first, which is the reverse of the natural sequence described by the orderly recruitment phenomenon. This reversal of recruitment order is further compounded by a common anatomical feature of human muscle in which the largest motor units, which have the largest axons, are often located superficially in a muscle (Lexell et al. 1983) and hence closer to the source of electrical stimulation. Futhermore, since electromyostimulation produces an unusual sensation in the stimulated limb it must activate some large afferents and many sensory receptors, including those that detect cutaneous stimuli. Input from cutaneous afferents via reflex activation, if sufficient in magnitude, may cause a reversal in the recruitment order of motor units (Burke et al. 1970; Garnett & Stephens 1981; Kanda et al. 1977; Stephens et al. 1978). On the basis of these 3 physiological effects (i.e. axon diameter, electrode-axon distance and cutaneous input), it seems likely that electromyostimulation is associated with a reversal of the recruitment order of motor units and that it may even preferentially activate the largest motor units that are difficult to activate under voluntary conditions (Cabric et al. 1988).

Trimble (1987) recently evaluated the magnitude of this effect by examining the response of a population of motor units to low intensity electromyostimulation. The technique involved measuring the time-to-peak force of the twitch response elicited by a Hoffmann reflex (Buchthal & Schmalbruch 1976). The Hoffmann reflex is based upon excitation of group Ia afferents that normally causes activation of motor units in the sequence described by orderly recruitment (i.e. smallest to largest; Hugon 1973; Magladery & McDougal 1950). However, in the presence of electromyostimulation, which elicits afferent cutaneous feedback, the time-to-peak force of the twitch response was considerably shorter than either before or after the electromyostimulation. This would occur if a faster contracting group of motor units had been activated by the Hoffmann reflex during the electromyostimulation. This observation provides indirect but compelling evidence that electromyo-

stimulation has a preferential effect on the larger motor units.

1.4 Evidence of Neural Adaptations

Given the physiological basis of electromyostimulation, it is perhaps not surprising that much of the evidence obtained from the study of healthy muscle suggests that the increase in strength with electromyostimulation is largely due to neural adaptations (i.e. training-induced changes in the function of the nervous system). The principal argument given for a neural effect has to do with the length of the training period. Most studies have been performed in less than 5 weeks, typically involving 10 to 15 training sessions. This duration is generally regarded as too short to induce gross morphological changes in muscle (Hakkinen et al. 1985a; Moritani & deVries 1979; Rutherford & Jones 1986). Along these lines, Eriksson et al. (1981) applied electromyostimulation to the quadriceps femoris muscles of subjects for 15 sessions spread over 4 to 5 weeks. They reported that while their subjects increased in strength they did not exhibit any significant changes in muscle enzyme activities, fibre size, or mitochondrial properties. In contrast, Cabric and colleagues (Cabric & Appell 1987; Cabric et al. 1987, 1988) used more intense training which elicited greater increases in strength that were accompanied by increases in limb girth, the number and size of myonuclei, and the average cross-sectional area of the muscle fibres in triceps surae. Similarly, Greathouse et al. (1986) found in rats that short term electromyostimulation can affect the mitochondria, triads, and glycogen content of fast contracting muscle fibres (see also Kernell et al. 1987; Pette 1984; Salmons & Henriksson 1981; Staron & Pette 1987). These data suggest that electromyostimulation can induce both neural and muscular changes, where the assessment of the muscular adaptations has been by direct observation and the neural adaptations by inference.

In addition to the argument based on the time course of the electromyostimulation effect, the neural consequences of electromyostimulation are underscored by 3 further lines of evidence: (a) training intensity; (b) cross-training (i.e. contralateral) effects; and (c) acute effects. Based on a substantive review of the literature, McDonagh and Davies (1984) concluded that an increase in strength with voluntary training techniques requires loads that are at least 66% of maximum. Laughman et al. (1983) trained the quadriceps femoris muscles of 2 groups of subjects, one group with isometric exercises and the other group with electromyostimulation. Although both groups exhibited similar increases in strength (18 and 22%, respectively) after 5 weeks of training, these were accomplished with average training intensities of 78% (isometric) and 33% (electromyostimulation) of maximum. Similarly, Stefanovska and Vodovnik (1985) administered electromyostimulation to subjects for 21 sessions (3 weeks) but only at an intensity that elicited 5% of maximum force. Nonetheless, the subjects achieved significant increases in strength. These discrepancies between electromyostimulation and voluntary intensities can be explained by a combined afferent-mediated effect (i.e. cutaneous feedback) and a preferential activation of larger motor units with electromyostimulation.

In a similar vein, another feature of electromyostimulation is its effect on the non-exercised contralateral limb that accompanies the electromyostimulation delivered to the test limb. The magnitude of this contralateral effect was demonstrated by Howard and Enoka (1987) when they applied electromyostimulation to the quadriceps femoris muscle group of one leg and had subjects exert a maximal isometric force with the other leg. They used 2 groups of subjects who differed as to whether the maximal force for a single leg occurred when 1 or 2 legs were active. Both groups exhibited significant increases in the single leg maximum (5.7% for the 1-leg group and 16.5% for the 2-leg group) when electromyostimulation was applied to the contralateral leg. Thus, the maximum force exerted by a single leg can be increased by the application of electromyostimulation to the inactive contralateral limb. This observation of an increased maximum force questions the concept of a maximum voluntary contraction and suggests that

electromyostimulation can have an effect that is not accessible by voluntary activation.

As observed by Howard and Enoka (1987), electromyostimulation can have a profound effect on strength after a single session. This acute effect has also been demonstrated by Alon (1985) who conducted a study to determine the effect of electrode size on perceptual discrimination between sensory, motor and painful responses. At the conclusion of a single experimental session, Alon (1985) found that, on average, the strength of the quadriceps femoris muscle group for the 14 subjects had increased by 13% of the pre-test maximum. This increase seems larger than can be accounted for simply by habituation to the stimulus over the course of a single session.

In summary, short term electromyostimulation protocols, such as those commonly used in rehabilitation medicine, are able to increase strength in healthy muscles. Although the magnitude of the inrease is no greater than that which can be achieved with voluntary training, the increases can be achieved in considerably less time. Four lines of evidence suggest that this increase is due to neural adaptations: time course of adaptation, training intensity, cross-training effects, and acute facilitative effects. There is insufficient evidence to determine the consequences of the long term application of these procedures and its effect on highly trained athletes.

2. Cross-Training Effects

In essence, the neuromuscular apparatus comprises sets of actuators (i.e. muscles) that operate on semirigid links (e.g. forearm, upper arm) to cause them to rotate about one another. The actuators are controlled by the nervous system and, in turn, relay information back to the controllers on the state of the system. Given the unidirectional function of the actuators (i.e. muscles can only pull and not push), a minimum of 2 are required to control a single degree of freedom at the articulation between 2 semirigid links. Such a set of actuators is known as an agonist-antagonist muscle set and much is known about the neurophysiological interactions between them. Much less attention, however, has been directed towards investigating the interactions between agonist-antagonist muscle sets located in different limbs. Indeed, the literature on interlimb interactions, in addition to being more sparse, is also much more convoluted and obscure than that on single limb agonist-antagonist interactions, despite the interest of prominent investigators (Jankowska & Odutola 1980; Perl 1957; Sherrington 1909) in the interlimb effects.

One consistent observation that emerges from this literature, however, is the profound effect that the activities of one limb can have on its contralateral counterpart. This is apparent from a variety of topics that include interlimb timing (Boylls et al. 1984; Miller & van der Meche 1976; Shaffer 1982), interlimb reflex effects (Delwaide et al. 1988; Dietz et al. 1980; Lagasse 1974), neuromuscular synapse formation (Rotshenker 1979), and the expression of myosin isozymes (Srihari et al. 1981). One striking example of the magnitude of interlimb effects was provided by the Srihari et al. (1981) study in which the soleus (slow twitch) muscle of a rabbit hindlimb was cross-innervated by a nerve that normally innervates the fast twitch gastrocnemius muscle. As expected, the soleus muscle in the test limb began to express the myosin light chains and isozyme forms commonly associated with a fast twitch muscle. Unexpectedly, the soleus muscle in the non-operated contralateral limb also, but to a lesser extent, exhibited the same types of changes.

In this review, the interest is in whether these interlimb phenomena might contribute to changes in strength. This possibility has already been encountered in the section on electromyostimulation where the training of one limb was noted to result in a relatively smaller increase in the strength of the untrained contralateral limb (Cabric & Appell 1987; Laughman et al. 1983). This effect, however, has not been observed by all investigators who have used artificial activation (electromyostimulation) of the motor system (Cannon & Cafarelli 1987; Eriksson et al. 1981). In this section, chronic (cross-education) and acute (bilateral deficit) evidence for an interlimb effect related to strength are reviewed.

2.1 Cross-Education

The contralateral effect of chronic motor activity in one limb has been described by several terms including: cross-education, cross-exercise, cross-training and cross-transfer. Of these terms, cross-education seems to have the earliest origin, being attributed to Scripture et al. (1894), and remains in vogue, particularly in the rehabilitation literature. The notion of cross-education arose from psychology in the context of training movement patterns in one limb and having an improvement in performance transferred to the contralateral limb. This remains a viable concept in rehabilitation practices, such as physiotherapy, where deficient limbs and muscles are exercised by manipulation of the contralateral limb (Devine et al. 1981; Gregg et al. 1957; Knott & Voss 1968; Moore 1975; Sills & Olson 1958).

Although not all investigators (Rutherford & Jones 1986; Young et al. 1983,1985) have observed an increase in strength in an untrained contralateral limb when a single limb is strengthened, the cross-education phenomenon had been reported frequently enough to inspire confidence in its existence (Cannon & Cafarelli 1987; Coleman 1969; Hellebrandt et al. 1947; Houston et al. 1983; Komi et al. 1978; Krotkiewski et al. 1979; Lewis et al. 1984; Moritani & deVries 1979; Parker 1985; Smith 1970; Yasuda & Miyamura 1983). The magnitude of the cross-education effect can be quite substantial. Moritani and deVries (1979), for example, trained the elbow flexor muscles of 15 subjects with isometric exercises at an intensity of 67% of maximum. The exercise was performed 10 times, twice daily, 3 days per week. After 8 weeks of training, Moritani and deVries reported an increase in strength of 36.4% in the trained limb and 24.7% in the untrained contralateral limb. Other investigators have not reported such substantial strength gains in the contralateral limb, but they have demonstrated significant increases in strength that are generally in the range of 10 to 30% but always less than the increase in the trained limb (viz. values below the line of identity in fig 2).

Hellebrandt et al. (1947) reported that the magnitude of the cross-education effect was related to the '. . . severity of the effort evoking the response rather than the duration of the exercise'. In surveying the literature, there does seem to be a strong association between the intensity of the training programme and the increase in strength of the untrained contralateral limb. For example, Moritani and deVries (1979) used a reasonably intense regimen and elicited a substantial cross-education effect (24.7%). Parker (1985) trained the quadriceps femoris muscles of subjects with isometric exercises for 4 months at one-half the intensity (i.e. 10 repetitions, 3 times per week) and obtained an increase in contralateral strength of 15%. In contrast, Young et al. (1985) had subjects perform a sustained (60-second) isometric knee extensor exercise at 30% of maximum. The subjects did 7 repetitions of the exercise each day, every day of the week. After 3 weeks, they found no change in the strength of the untrained contralateral limb, probably because of an insufficient intensity (i.e. 30% of maximum). Interestingly, after 8 weeks of this regimen, Young et al. (1985) did observe an increase in the strength of the trained limb but the experimental design was such that they could not test for a cross-education effect. Thus, as proposed by Hellebrandt et al. (1947), there does appear to be a strong association between training intensity and the magnitude of the cross-education effect (fig.2).

While this increased strength of an untrained limb seems curious, an obvious explanation is that the limb does indeed undergo training due to the postural requirements associated with the activity. Some investigators have examined this possibility by recording the EMG activity in other muscles that might be involved in the task (Devine et al. 1981; Panin et al. 1961). During a strength test for a muscle about a single joint, there is widespread activation of other muscles throughout the body. However, the magnitude of the EMG is rather low and quite insufficient to represent the training stimulus for the contralateral limb. Similarly, others have reported no change in muscle fibre areas or enzyme activities in contralateral limbs that exhibit a cross-education effect (Houston et al. 1983). Consequently, it is probable that the cross-educa-

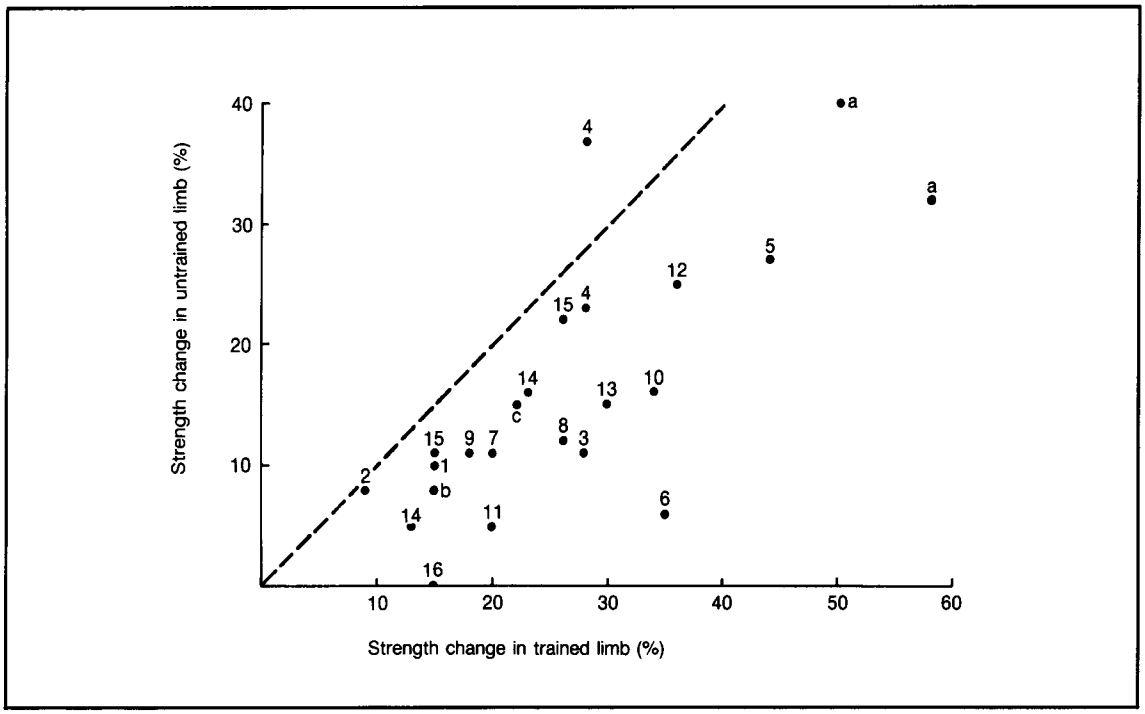

Fig. 2. Range of associations between increases in strength for the trained and the untrained limbs. Strength was measured as the maximal isometric force and was increased by a variety of training programmes in several different muscles. The figure represents a survey of the values reported in the literature for voluntary (1 to 16) and electromyostimulation (a,b,c) training: 1 = Cannon and Cafarelli (1987); 2 = Coleman (1969); 3 = Davies et al. (1985); 4 = Hellebrandt et al. (1947); 5 = Houston et al. (1983); 6 = Jones Rutherford (1987); 7 = Komi et al. (1978); 8 = Krotkiewski et al. (1979); 9 = Laughman et al. (1983); 10 = Lewis et al. (1984); 11 = Milner-Brown et al. (1975); 12 = Moritani and deVries (1979); 13 = Parker (1985); 14 = Smith (1970); 15 = Yasuda and Miyamura (1983); 16 = Young et al. (1983); a = Cabric and Appell (1987); b = Cannon and Cafarelli (1987); c = Laughman et al. (1983). The line of identity indicates that the increase in strength in the trained limb was always greater than that for the untrained limb.

tion phenomenon reflects a centrally located neural adaptation, like changes in the interneuronal networks between limbs.

2.2 Bilateral Deficit

In contrast to the facilitative interlimb effects observed with cross-education, acute features of bilateral interactions have largely revealed a deficit in strength. Early investigators, who designed experiments based on the cross-education literature, expected a facilitation of strength during bilateral activation (Henry & Smith 1961; Kroll 1965). Instead, Henry and Smith (1961) reported a 3% strength decrease in the dominant hand during a hand-grip strength test when the non-dominant hand was concurrently performing a maximal hand-grip. This decrement in strength has subsequently been substantiated by a number of investigators using such muscles as the finger flexors, elbow extensors and flexors, knee extensors and leg (hip, knee, and ankle) extensors (Coyle et al. 1981; Howard & Enoka 1987; Ohtsuki 1981, 1983; Rube et al. 1980; Rube & Secher 1981; Secher 1975; Secher et al. 1978; Vandervoort et al. 1984). The magnitude of the bilateral deficit is generally in the range of 5 to 25% of maximal unilateral strength; that is, the strength of a particular muscle group in one limb is 5 to 25% less when the contralateral limb is concurrently performing a maximal activation.

As with cross-education, the most obvious explanation for the bilateral deficit is a mechanical

effect in that with 2 limbs maximally active the postural demands on the remainder of the body are proportionally greater and detract from the maximal output of the test muscle group. However, since the bilateral decrement in force is accompanied by a parallel decline in EMG (Howard & Enoka 1987; Ohtsuki 1981, 1983; Vandervoort et al. 1984), the principal mechanism underlying the bilateral deficit, as with cross-education, must have a neural rather than a mechanical basis. In an attempt to delineate the mechanisms accounting for the bilateral deficit (i.e. declines in force and EMG), Secher et al. (1978) used pharmacological agents which were thought to selectively inactivate slow and fast twitch muscle fibres. Based on a comparison of strength during unilateral and bilateral tasks, Secher et al. (1978) concluded that the bilateral deficit was due to a diminished contribution of slow twitch muscle fibres. Subsequently, however, Vandervoort et al. (1984) expressed concern over the selectivity and precision of the pharmacological agents used by Secher et al. (1978), and were able to demonstrate with standard physiological tests (i.e. force-velocity relationship and fatigability) that the bilateral deficit was most likely due to a failure to activate all the fast twitch muscle fibres.

In a later study, Vandervoort et al. (1987) suggested that variation in the magnitude of the bilateral deficit was due to differences in the familiarity of the various muscle groups with concurrent bilateral activation. For example, we tend to use our legs less frequently in a concurrent mode (e.g. vertical jump) and more often in a reciprocal manner (e.g. locomotion), while our arms commonly experience both modes. Based on this rationale, the smallest (3%; Henry & Smith 1961) and even nonexistent bilateral deficits (Vandervoort et al. 1987) have been reported for arm muscles, while the largest deficit has been observed with the knee extensors (25%; Secher et al. 1978). Futhermore, among the arm muscles those used most often (e.g. elbow flexors) exhibit the least bilateral deficit (6 to 8%) compared with the less frequently used antagonist muscles (e.g. 19 to 25% for the elbow extensors; Ohtuski 1983). However, the neural mechanisms subserving this bilateral phenomenon seem confined to the concurrent activation of bilaterally homologous muscles. There is no bilateral deficit when antagonist muscles (e.g. right elbow flexors and left elbow extensors; Ohtsuki 1983) or muscles in different limbs (e.g. left elbow flexors and right knee extensors; Howard 1987) concurrently perform strength tests.

Futhermore support for the notion that the magnitude of the bilateral deficit is related to the familiarity of the subject with the task has been provided by testing different populations of subjects. Secher (1975) related the strength capabilities of rowers to their level of expertise and found that the knee extensors of the elite rowers did not exhibit a bilateral deficit while less capable rowers did produce a deficit. Similarly, Howard and Enoka (1987) reported a bilateral deficit in the knee extensors of control subjects and elite cyclists (who train their legs in a reciprocal manner) but a bilateral facilitation for weightlifters. The latter observation means that the maximal strength for each limb of the weightlifters is only realised during 2-legged efforts. These data raise the possibility that bilateral deficits might be mutable with training. Rube et al. (1980) examined this possibility and suggested that most of the changes in a bilateral deficit were due to habituation rather than to training. Similarly, Coyle et al. (1981) reported comparable increases in 1- and 2-legged strength with 2-legged training and hence no change in the bilateral deficit. In contrast, Howard (1987) trained subjects with either 1- or 2-legged regimens for 3 weeks at 3 times each week and found a training-induced removal of the bilateral deficit for the group that trained with 2 legs. This observation by Howard (1987) suggests that the bilateral deficit is indeed mutable and argues strongly in favour of the principle of specificity in the design of strength programmes. However, this issue awaits further study.

In summary, the neural interactions between limbs are quite potent and have received relatively little attention in the neurophysiological literature. The potency of these effects is underscored by 2 strength-related topics: cross-education and bilateral deficit. Studies of cross-education reveal that

unilateral training of one limb can result in an increase in the strength of the untrained contralateral limb. In contrast, when bilateral homologous muscles are maximally activated, there is a decrement in the strength of each limb due to neural limitations associated with a 2-limb task. However, this bilateral deficit is mutable and with the appropriate training the deficit may become a facilitation such that the strength of a limb is greatest when two, rather than a single, limbs are maximally activated.

3. EMG-Force Relationship

Although the maximal force which a muscle can exert is directly related to its cross-sectional area, there is a poor correlation between increases in strength and muscle size (Howald 1985; Ikai & Fukanaga 1970; Jones & Rutherford 1987; Luthi et al. 1986; MacDougall 1986; Young et al. 1983). This dissociation between strength and size occurs because strength is not solely a property of muscle but rather it is considered a property of the motor system. Strength is affected by an interaction of neural, mechanical and muscular factors (Enoka 1988b; Howard et al. 1985; Rutherford & Jones 1986). The essential features of this interaction are schematised in figure 3. In this scheme, the nervous system is partitioned into 3 compartments which correspond to functional roles subserved by different neural elements during the elaboration of movement (Enoka & Stuart 1985; Feldman & Grillner 1983; Hasan et al. 1985). These compartments of the tripartite model interact with one another and with the musculoskeletal system. The expression of strength involves the generation of a command by the high-level controller (central command) that is transformed into an appropriate sequence of muscle activations (motor programme) by the low-level controller and subsequently transmitted to the requisite muscles. During a sustained task, such as a strength test, these commands from the high- and low-level controller may also be modified by feedback from either peripheral sensory receptors or the high-level controller. The maximal, isometric output of the system (i.e. its strength) is measured as the force exerted by the musculoskeletal elements against a force transducer.

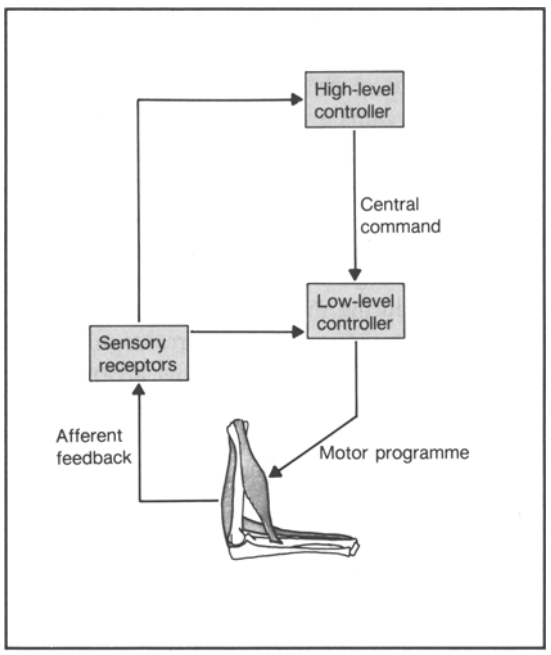

Fig. 3. A schematic of the motor system. The nervous system is represented as a tripartite model which interacts with itself and with the musculoskeletal system. The tripartite model is a useful conceptual framework for the functional roles subserved by the various neural elements during the elaboration of movement. The central programme for a task is located in the low level controller, which corresponds anatomically to the spinal cord or brainstem, depending on the task. The task-related output of the low-level controller is referred to as a motor programme. The activity of the low-level controller is initiated and sustained by descending signals (central command) from the high-level controller (i.e. the supraspinal centres) and modified by afferent feedback from peripheral sensory receptors. The commands issued by the nervous system, including those associated with the expression of strength, impinge on the musculoskeletal system and in turn are altered (feedback) by the activity induced in the system. We characterise the brief, maximal, isometric output of the system as its strength. Although strength is measured as the maximal force exerted by the musculoskeletal system, it is altered by the activity of the nervous system in addition to the mechanics of the musculoskeletal system.

The observation that strength and muscle cross-sectional area do not change in parallel suggests that increases in strength are not accompanied by

linear increases in all elements of the system. Indeed, this appears to be the case. The general consensus is, particularly with naive subjects, that initial strength gains are due to neural adaptations while later increases in strength are largely the result of muscle hypertrophy (Ikai & Fukanaga 1970; Moritani & deVries 1979). Thus, the association between changes in strength and muscle size is lowest at the beginning of a training programme and highest during the later stages. The dissociation between training-induced increases in strength and muscle size (i.e. cross-sectional area) may be explained by an enhancement of the EMG or an improvement in the efficacy of the force transmitted from individual sarcomeres to the skeletal system.

3.1 Force Transmission

One possible explanation for the increase in strength without any change in the cross-sectional area of muscle might be that training induces an increase in the force that muscle can exert per unit of cross-sectional area, i.e. an increase in the specific tension of muscle. This might be accomplished in either of two ways; by increasing the volume density of contractile proteins or by increasing the intercellular connective tissue matrix among muscle fibres and hence the proportion of sarcomere force that is transmitted to the skeletal system. Computed tomography scans have revealed a small but consistent increase in radiological density as a consequence of strength training (Horber et al. 1985; Jones & Rutherford 1987). This increase may be due to an increase in the density of myofilaments, a decrease in fat content, or an increase in the proportion of connective tissue. However, since MacDougall (1986) has found that prolonged strength training induces a 16% increase in the cross-sectional area of myofibrils which is not accompanied by a change in the density of thick filaments, it appears unlikely that specific tension is altered by varying myofilament density (see also Howald 1985; Luthi et al. 1986).

Several lines of evidence, however, suggest that the quality and quantity of connective tissue structures are affected by training and that these adaptations may influence specific tension. Firstly, one paradox in the specific tension literature that supports this postulate is the difference between values obtained from single fibre *vs* motor unit measurements. Based on single fibre values (skinned fibre preparation), there appears to be no difference in the specific tension of type I and type II muscle fibres (24.5 *vs* 24.3 N/cm^2, respectively) of the cat medial gastrocnemius (Lucas et al. 1987). In contrast, motor unit studies (Bodine et al. 1987; Burke & Tsairis 1973; McDonagh et al. 1980b), which involve the electrical activation of a motoneuron or a ventral root axon, report 3- to 5-fold differences in specific tension between type S (slow twitch) and type F (fast twitch) motor units (6 *vs* 24 N/cm^2). One explanation for this discrepancy is that the force measured with the *in situ* preparation (i.e. the motor unit studies) is affected by the layers of connective tissue in which the muscle fibres are embedded. This connective tissue effect seems possible given the observation that the concentration of endomysial collagen is significantly greater for slow twitch muscle fibres compared with fast twitch fibres (Kovanen et al. 1984a). Certainly, the presence of connective tissue structures (e.g. fascia) is known to have a significant effect on the force transmitted to the tendon (Borg & Caulfield 1980; Garfin et al. 1981). Undoubtedly, more will become known about this effect as the complexities of muscle architecture are unravelled (Barrett 1962; Loeb et al. 1987).

Secondly, the properties of connective tissue are known to be mutable with training (Suominen et al. 1980; Tipton et al. 1975; Woo et al. 1981). In skeletal muscle, these adaptations are manifested as increased tensile strength and with the effect being greater in slow contracting (e.g. soleus) compared to fast contracting (e.g. rectus femoris) muscle (Kovanen et al. 1984b). With endurance-type training, the increased strength of connective tissue seems to be related to variation in the number of collagen cross-links (Kovanen et al. 1984b). Such changes in connective tissue strength may improve the transmission of force from individual sarcomeres to the skeletal system; that is, less of the sar-

comere force may be dissipated by surrounding tissues. However, there do not appear to be any data on the adaptations associated with strength training.

Thirdly, training adaptations are specific to the exercise stress which induces them (e.g. Dons et al. 1979; Duchateau & Hainaut 1984; Häkkinen & Komi 1986; McCafferty & Horvath 1977; Rosler et al. 1986; Sale & MacDougall 1981). Among these effects is the observation that strength training results in increases in strength but no change in peak power production (Rutherford et al. 1986). Since power is the product of force and velocity and training causes increases in force (i.e. as indicated by an increase in the cross-sectional area of muscle fibres; Goldspink 1985; Häkkinen et al. 1985a,b; MacDougall 1986; Thorstensson et al. 1976), then strength training must elicit a decrease in the maximum velocity of whole-muscle shortening. The reduction in velocity could be accounted for by a decrease in the effective length of muscle fibres, such as might be accomplished by an increase in the quality (i.e. type of collagen) or quantity of the connective tissue matrix surrounding skeletal muscle fibres. In addition, the 3-fold increase in the number of split or partially fused myofibrils following strength training (MacDougall 1986) might contribute to the more secure mechanical coupling of the contractile proteins.

Fourthly, Walsh et al. (1978) examined the compensatory hypertrophy induced in the medial gastrocnemius muscle of the cat hindlimb by removal or denervation of the synergist muscles. These investigators were interested in the effects of this procedure on the properties of the 4 motor unit types [FF, F(int), FR, and S]. Following 14 to 32 weeks of compensatory hypertrophy, the medial gastrocnemius muscle increased its weight significantly and the maximum tetanic force of all 4 motor unit types increased substantially. In the animal examined, however, there was no associated increase in the cross-sectional area of the different unit types. One factor contributing to this dissociation between an increased force and a constant size may have been a change in the specific tension of the motor units.

Taken together, these observations suggest that part of the dissociation between increases in strength and the cross-sectional area of muscle may be due to an increase in the specific tension of muscle. This variation in specific tension does not seem to be related to a change in the density of contractile proteins but rather to an improvement in the transmission of force from myofibrils to the skeletal system.

3.2 EMG Enhancement

The most common conclusion concerning the dissociation between changes in strength and muscle size is that training has induced some form of neural adaptation. This assertion is generally based on the magnitude of the maximal rectified and filtered EMG during a maximal isometric task that is performed before and after a strength training programme (Häkkinen & Komi 1983b; Häkkinen et al. 1985a; Komi et al. 1978; Moritani & deVries 1979). The mechanisms underlying the changes in EMG, however, are difficult to deduce because the EMG is a complicated, summated signal that represents the extracellular voltage-time measure of the excitation provided by the nervous system for muscle. The interpretation of the EMG is difficult with any degree of confidence (Denny-Brown 1949; Hof 1984; Loeb & Gans 1986; Perry & Bekey 1981). In a strength training paradigm, the interpretation of the EMG is made more difficult by the need to compare measurements before and after training. However, with appropriate attention to detail it appears possible to obtain reasonably reliable long term EMG measurements (Cannon & Cafarelli 1987; Chapman & Belanger 1977; Moritani & deVries 1979).

The evidence for changes in EMG that accompany strength training has been diverse, ranging from no effect (Cannon & Cafarelli 1987; Thorstensson et al. 1976) to substantial increases in the maximal rectified and integrated EMG (38%; Komi et al. 1978). Undoubtedly this variation reflects differences in such factors as the intensity of training programmes, test muscles (e.g. hand *vs* thigh muscles), whether or not the task required the sub-

ject to maintain balance, the extent to which the electrode sampled whole-muscle activity, and the measure of EMG from several muscles involved in the task (i.e. presuming that the task was not controlled by a single muscle). Despite this diversity, a number of reports have documented an EMG effect that includes a training-induced increase in the maximal rectified and integrated EMG during a maximal isometric contraction (Häkkinen & Komi 1983a; Häkkinen et al. 1985a; Komi et al. 1978; Moritani & deVries 1979) and post-training decrease in the EMG associated with a constant submaximal force (Häkkinen & Komi 1983a; Häkkinen et al. 1985a; Moritani & deVries 1979). The month-to-month variation in the maximal EMG is much more sensitive to variations in the training intensity than is the change in strength (Häkkinen et al. 1985a). For example, towards the end of the training programme the maximal EMG may decline while the individual continues to gain or maintain strength (Häkkinen & Komi 1983a; 1986). Furthermore, the adaptation among the muscles within one group (e.g. rectus femoris, vastus lateralis, vastus medialis) can be quite different (Häkkinen & Komi 1983a; Häkkinen et al. 1985a).

Alternatively, an argument for neural adaptations can be made on the basis of the electromyostimulation literature. For example, Young et al. (1985) trained the triceps surae muscles of subjects for 8 weeks with voluntary training techniques. Prior to training, artificial activation (50Hz) of the muscle group by passing current between electrodes located over gastrocnemius elicited a force that was 80% of the maximal voluntary value (i.e. 80% of its strength). The isometric training programme resulted in a 27% increase in strength (i.e. voluntary), but no change in the maximal force that could be artificially elicited. Based on such evidence, it seems that gains from short term strength training may not be associated with the intrinsic capacity of muscle to exert force (see also Cannon & Cafarelli 1987; Davies et al. 1985; Duchateau & Hainaut 1988; McDonagh et al. 1983).

There is sufficient evidence to suggest that the neural adaptations encompass all 3 elements of the tripartite model (fig. 3). At the level of the low-level controller, neural adaptations have been shown by eliciting the Hoffmann reflex and comparing the magnitude of the response (a compound muscle action potential) obtained during rest to that elicited during a maximal voluntary contraction (Sale et al. 1982, 1983a,b). Since the Hoffmann reflex is an indirect measure of the excitability of the low-level controller, it is greater during a maximal voluntary contraction. The increased Hoffmann reflex during voluntary activity is referred to as reflex potentiation. It has been shown that reflex potentiation is greater in weightlifters than in control subjects (Milner-Brown et al. 1975; Sale et al. 1983b) and that this effect is exhibited in most (Milner-Brown et al. 1975; Sale et al. 1983a,b) but not all muscles (Sale et al. 1982, 1983b). One consequence of this increase in excitability of the low-level controller would be that for a given central command from the high-level controller the output of the low-level controller (i.e. as measured by the EMG) might be increased.

Accompanying this increase in the excitability of the low-level controller, it appears that strength training also effects changes in EMG by varying motor unit activity, either by varying the number of active motor units or by changing the rate and timing of the action potentials discharged by the motoneurons. Normally the action potentials discharged by a motoneuron are temporally unrelated to those generated by other units; for this reason the action potential trains of active motor units are described as asynchronous (Gel'fand et al. 1963; Taylor 1962). However, during some slow movements (Loeb et al. 1988) and during strong contractions there is an increase in the degree of synchrony between action potential trains of motor units (Person & Kudina 1968). This synchrony has been shown to result in an increase in the EMG (Weytjens & van Steenberghe 1984). Furthermore, strength training increases the synchronisation between action potential trains (Milner-Brown et al. 1975). One of the consequences of the increased synchronisation is an increase in the rate at which maximal force can be achieved (Miller et al. 1981). It is difficult to determine which compartment of the tripartite model is responsible for the change

in synchronisation, although Milner-Brown et al. (1975) argue that since the reflex potentiation exhibited by the weightlifters involved the longer latency responses to the percutaneous nerve stimulation, the effect was probably dominated by the high-level controller.

There seem to be at least 2 issues concerning the adaptability of the high-level controller to strength training: the magnitude of the central command and learning the task. It is apparently difficult to maximally activate a muscle (Woods et al. 1987), due presumably to insufficiency of the central command, but it can be accomplished if the subjects are motivated (Bigland-Ritchie 1984) and with practice (Jones & Rutherford 1987). These assessments have generally been based on the twitch-interpolation technique (Denny-Brown 1949; Merton 1954) which involves the supramaximal activation of the nerve to a muscle with a single shock to determine if the maximal voluntary force can be artificially supplemented. If the shock does not elicit a discernible twitch response, then the muscle is regarded as maximally active. One limitation of this technique is that with larger muscles it is difficult to artificially activate the entire muscle and hence be secure in the interpretation of the response (e.g. Rutherford & Jones 1986). Based on this technique, however, Belanger and McComas (1981) found that a group of 28 subjects were able to maximally activate the tibialis anterior muscle but about half of the subjects could not achieve full activation of the triceps surae muscle group. This inability could presumably be overcome with an adequate strength training programme that resulted in either an increase in the central command or an improved transformation of the central command within the low-level controller.

An alternative approach to a possible training-induced central command effect was provided by Young et al. (1985). In this study, subjects trained 1 leg daily for 8 weeks with 7 to 15 repetitions of a 60-second isometric contraction at an intensity of 30% of maximum. After 3 weeks, the subjects began training the contralateral leg with rhythmic 3-second maximal contractions. The 2 regimens produced increases in strength of 30.2% for the 30%

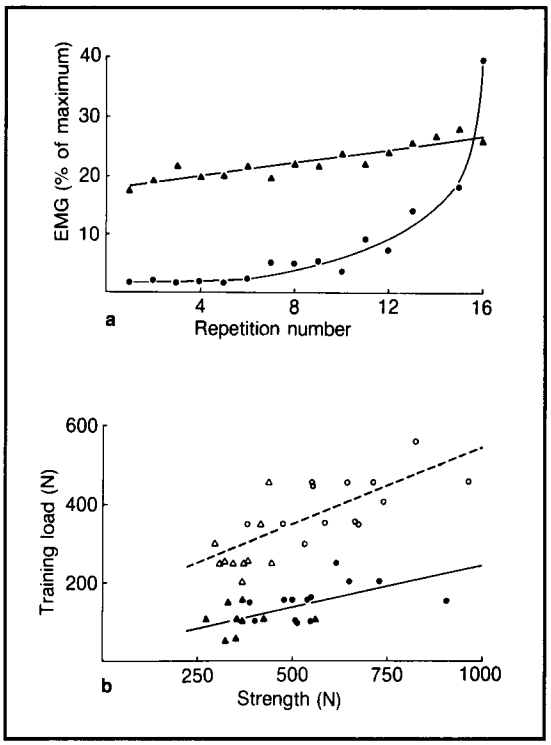

Fig 4. EMG changes associated with strength. (a) Magnitude of the rectified and integrated EMG of one subject for 16 repetitions of a 60-second isometric contraction in which the target force was 30% of maximum. To repeatedly attain the target force, the subject had to increase the EMG of soleus (▲) and medial gastrocnemius (●) over the course of each training session. Redrawn from Young et al. (1985). (b) Relationship between strength (maximal isometric force) and training load for 20 subjects before (-, ▲, ●; r = 0.61) and after (---, △, ○; r = 0.74) a 12-week strength training programme (males = ○, ●; females = △, ▲). Redrawn from Rutherford and Jones (1986).

force group and 26.6% for the maximal force group. As summarised by McDonagh and Davies (1984), the consensus opinion in the strength literature is that muscle must be activated at an intensity of at least 66% of maximum before there will be an increase in strength. It is curious, therefore, that the subjects of Young et al. (1985) could increase their strength at a training intensity that was 30% of maximum. A probable explanation for the adaptation is outlined in fig 4a. Although the subjects perceived the training goal as a target force that was 30% of maximum, achievement of this goal during one training session required a progres-

sively larger EMG (Enoka & Stuart 1985; Jones & Hunter 1983; Seals & Enoka 1988). Thus, neural drive to the muscles increased over the course of a training session. Young et al. (1985) suggest that it is the level of the neural drive during training rather than the size of the load that is the stimulus for increasing strength.

The notion that neural adaptations might underlie the increases in strength following moderate intensity training is intriguing because it raises the issue of the mechanisms responsible for increased strength. It could, for example, involve a modification of the central command itself or the manner in which it is processed in the low-level controller. Both of these possibilities are embodied in the suggestion that some strength gains are due to an improved performance of the task due to learning or altered coordination among the musculature. Rutherford and Jones (1986) addressed this issue by training 3 groups of subjects for 12 weeks; each group trained with a task that had different postural requirements (i.e. minimal to substantial balance requirements) and involvement of musculature (i.e. 1 *vs* 2 legs). One group performed a unilateral isometric task and produced the greatest (40%) increase in strength. The other 2 groups did dynamic exercises (anisometric) and they had strength gains of 15 and 20%. However, the training loads of the 2 dynamic exercise groups increased by 170 and 200%, respectively, with most of this increase occurring during the initial training period. For one of the dynamic exercise groups, Rutherford and Jones (1986) examined the relationship between strength and training load before and after training (fig. 4b). The linear relationship shifted upward with training such that following training the subjects could lift heavier loads for a given strength of the quadriceps femoris muscles. Rutherford and Jones (1986) interpret this observation as evidence for a training-induced improvement in coordination. It would be of interest to conduct a more extensive electromyographic and kinematic analysis in order to determine the basis of the change in coordination.

In contrast to learning and coordination effects, there appear to be minimal sensory effects associated with strength training (Cafarelli 1988). Häkkinen and Komi (1986), for example, elicited tendon-tap reflexes in experienced weightlifters before and after 24 weeks of strenuous training. They reported no training effect on reflex latency, electromechanical delay, or peak twitch force despite a reduction in the amplitude of the reflex EMG. Similarly, Cannon and Cafarelli (1987) found that 5 weeks of voluntary strength training did not alter force sensation. Curiously, however, they noted a decrement in force sensation among subjects who underwent a programme of electromyostimulation. Apparently, the matching of central sensory adaptations with improvement in strength requires the participation of the nervous system in normal, voluntary training procedures.

The final line of evidence on the contribution of neural adaptations to strength development has to do with the training-induced changes in excitable membranes. Kereshi et al. (1983) have reported that conduction velocity of fibres in the biceps brachii muscle of bodybuilders was significantly faster than that for control subjects (5.5 *vs* 2.8 m/sec). Similarly, following 14 to 32 weeks of exposure to the compensatory hypertrophy model, Walsh et al. (1978) found that the most consistent adaptation of all 4 motor unit types in the medial gastrocnemius muscles, along with an increase in strength, was a significant increase in axonal conduction velocity. Although it is uncertain what benefits would accrue with a change in conduction velocity these two reports emphasise the adaptability of excitable membranes to moderate or long term strength training.

In summary, there are a number of reports which provide reasonable support for the suggestion that neural adaptations contribute to strength gains. These observations are based on changes in the EMG that accompany strength training and the demonstration of a number of neural processes that may be associated with the change in EMG. However, definitive evidence on the mechanisms underlying the neural adaptations remains elusive and the prospect for future studies.

4. Conclusions

The following is a list of main points derived from the discussion:

Strength is defined as the maximal, voluntary, isometric force. The magnitude of this value is determined by neural, mechanical and muscular factors.

Electromyostimulation techniques can elicit strength gains in healthy muscle that are comparable to those that can be achieved with voluntary training.

The optimal electromyostimulation protocol may involve high frequency stimulation (ca. 10 kHz) that is modulated at a lower frequency (ca. 100Hz).

The recruitment order of motor units during electromyostimulation differs from that for voluntary activation. Electromyostimulation appears to preferentially activate the largest motor units that are difficult to train under voluntary conditions.

The short term effects of electromyostimulation on strength seem to be based on neural adaptations. However, more intense and longer duration regimens can elicit morphological changes in muscle.

Unilateral strength training usually results in an increase in strength of the untrained contralateral limb. This phenomenon is referred to as cross-education and has been shown to occur following training with both voluntary and electromyostimulation techniques.

The cross-education effect is based on a central neural adaptation.

Acute bilateral interactions generally result in a decrement of strength compared to the maximal, unilateral strength of the limb. This bilateral deficit is largely due to the expression of inhibitory interlimb neural effects that result in a failure to activate all of the fast twitch motor units.

Bilateral interactions are mutable with training and may result in a bilateral facilitation, rather than a bilateral deficit, with appropriate training.

The observation that strength and muscle size do not change in parallel suggests either an increase in the specific tension of muscle or an enhancement of the excitation (EMG) provided by the nervous system to muscle.

Strength training does not cause an increase in specific tension due to an increased density of contractile proteins but it may increase specific tension through improvement of force transmission from the active sarcomeres and muscle fibres to the skeletal system.

Strength training induces changes in the EMG that are generally interpreted as evidence of neural adaptations. The neural mechanisms that may contribute to the EMG effect include reflex potentiation, motor unit synchronisation, improved coordination, and learning.

The development of strength is a complex process that often yields the expected morphological changes in muscle but always involves an adaptation in the neural mechanisms underlying its expression.

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Author's address: Dr *Roger M. Enoka*, McKale Center, Room 228-V, University of Arizona, Tucson, AZ 85721 (USA).