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REVIEW

Neural control of lengthening contractions

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ABSTRACT

A number of studies over the last few decades have established that the control strategy employed by the nervous system during lengthening (eccentric) differs from those used during shortening (concentric) and isometric contractions. The purpose of this review is to summarize current knowledge on the neural control of lengthening contractions. After a brief discussion of methodological issues that can confound the comparison between lengthening and shortening actions, the review provides evidence that untrained individuals are usually unable to fully activate their muscles during a maximal lengthening contraction and that motor unit activity during submaximal lengthening actions differs from that during shortening actions. Contrary to common knowledge, however, more recent studies have found that the recruitment order of motor units is similar during submaximal shortening and lengthening contractions, but that discharge rate is systematically lower during lengthening actions. Subsequently, the review examines the mechanisms responsible for the specific control of maximal and submaximal lengthening contractions as reported by recent studies on the modulation of cortical and spinal excitability. As similar modulation has been observed regardless of contraction intensity, it appears that spinal and corticospinal excitability are reduced during lengthening compared with shortening and isometric contractions. Nonetheless, the modulation observed during lengthening contractions is mainly attributable to inhibition at the spinal level.

KEY WORDS: Electromyogram, Motor unit, Voluntary activation, Cortical excitability, Spinal excitability

Introduction

The performance capacity of a muscle or a muscle fibre is usually greater when it is lengthened while being activated (eccentric contraction) than when it shortens (concentric contraction) (Edman, 1988; Herzog, 2014; Katz, 1939; Morgan et al., 2000). The realization of this potential *in vivo*, however, depends on the intensity of the motor command sent by the nervous system. Indeed, the magnitude of muscle activation depends on the number of motor units (ensemble comprising a motor neurone and the fibres innervated by its axon) that are recruited and the rate at which they discharge action potentials (Duchateau and Enoka, 2011; Heckman and Enoka, 2012). A number of studies during the last few decades have established that the control strategy employed by the nervous system during a lengthening contraction differs from those used during shortening and isometric contractions (Duchateau and Baudry, 2014; Duchateau and Enoka, 2008; Enoka, 1996).

The current review examines: (1) methodological issues that can confound the comparison between lengthening and shortening actions; (2) the capability of the nervous system to activate the muscle maximally when performing lengthening contractions; (3) the motor unit activity during submaximal lengthening and shortening actions; (4) the difference in the control strategy during submaximal and maximal lengthening contractions; and (5) the potential supraspinal and spinal mechanisms involved in the control of lengthening contractions.

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Methodological issues

A simple way to compare the performance of a muscle during shortening and lengthening contractions is to lift and lower a load. It is easier to lower than to raise the load and, accordingly, muscle activity assessed by surface electromyography (EMG) is usually less during the lengthening phase of the movement. However, two main factors confound the association between muscle activity and muscle force (torque) during such anisometric contractions. First, the greater intrinsic force capacity of the muscle fibres during lengthening contractions (Edman, 1988; Herzog, 2014; Katz, 1939; Morgan et al., 2000) means that less motor unit activity is necessary to achieve a specific absolute force compared with that needed during a shortening contraction. Second, muscle force must be greater than the load to overcome the inertia and lift it with a shortening contraction, whereas muscle force must be less than the load to lower it with a lengthening contraction. As a consequence of these characteristics, less motor unit activity is needed to move a submaximal load with a lengthening contraction than with a shortening contraction. Moreover, the rate of change in muscle length must be similar when comparing the shortening and lengthening phases of a movement due to the influence of movement velocity on central and peripheral afferent feedback, and on the neural activation of muscle (Duchateau and Enoka, 2011).

To avoid these confounding factors when comparing muscle activation during the two anisometric contractions, many studies have used isokinetic dynamometers to control the force and joint angular velocity (Aagaard et al., 2000; Amiridis et al., 1996; Babault et al., 2001; Baudry et al., 2007; Beltman et al., 2004; Duclay and Martin, 2005; Duclay et al., 2011; Grabiner and Owings, 2002; Pasquet et al., 2006; Pinniger et al., 2003; Westing et al., 1990). Nonetheless, the strategy employed by the central nervous system (CNS) when resisting the force imposed by a torque motor can differ slightly from that used when lowering an inertial load to match an imposed trajectory (Duchateau and Enoka, 2008).

Muscle activation during maximal contraction

One of the main questions in the field at the end of the last century was whether or not voluntary activation was sufficient to elicit the maximal force capacity of the muscle during lengthening contractions (Amiridis et al., 1996; Westing et al., 1990). Whereas the peak force that can be evoked from isolated fibres and whole muscles in animals is usually 50–80% greater during lengthening contractions (Edman, 1988; Katz, 1939; Morgan et al., 2000), the peak force achieved in untrained humans during maximal voluntary contraction (MVC) is usually either comparable or only modestly greater (<40%) for lengthening contractions versus isometric or slow shortening contractions. For example, no significant difference

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was observed in peak force between lengthening and isometric contractions in young adults for the knee extensors (Amiridis et al., 1996; Babault et al., 2001; Beltman et al., 2004; Seger and Thorstensson, 2000; Westing et al., 1991), ankle plantar flexors (Pinniger et al., 2000) and elbow flexors (Colson et al., 1999), whereas a slightly greater force was reported during lengthening actions for the knee extensors (Aagaard et al., 2000; Kellis and Baltzopoulos, 1998; Reeves et al., 2009), elbow flexors (Linnamo et al., 2006) and plantar flexors (Duclay et al., 2011). In contrast, the force produced during lengthening contractions by the ankle dorsiflexors is substantially greater (~30–50%) than that during isometric contractions (Klass et al., 2007; Pasquet et al., 2000; Reeves and Narici, 2003). In older adults, the difference in peak force between shortening and lengthening contractions performed at the same velocity is often greater than in young adults, especially for shortening contractions (see Roig et al., 2010). The absence of substantial changes in voluntary activation for older adults suggests that the differential decline in peak force during shortening and lengthening contractions is mainly attributable to adaptations within the muscle (Klass et al., 2005).

The discrepancy between animal and human studies in the relative forces produced during lengthening and isometric or shortening contractions is often ascribed to a neural command that is not sufficient to achieve the intrinsic force capacity of the muscle during maximal lengthening contractions. Three lines of evidence support this point of view. First, when individuals perform maximal isokinetic actions, EMG amplitude recorded with surface electrodes is often less during lengthening contractions than during shortening contractions performed at the same speed (Aagaard et al., 2000; Amiridis et al., 1996; Kellis and Baltzopoulos, 1998; Komi et al., 2000; Tesch et al., 1990; Westing et al., 1991); the difference is even greater when the lengthening contraction is not preceded by a maximal isometric contraction (Komi et al., 2000). Second, the level of voluntary activation assessed by superimposing a single stimulus or a brief train of electrical pulses over the muscle or its motor nerve during the force plateau of a MVC is often, but not always (Babault et al., 2001), depressed during lengthening contractions (Amiridis et al., 1996; Beltman et al., 2004; Westing et al., 1990). For example, Beltman and colleagues (2004) observed a deficit in voluntary activation of 21% during a maximal lengthening contraction at constant speed with the quadriceps femoris compared with a deficit of only 7% and 8% during an isometric and shortening MVC, respectively (Fig. 1). In the ankle dorsiflexors, a muscle group in which full voluntary activation is more easily reached than for other muscles (Belanger and McComas, 1981), the average deficit in voluntary activation during lengthening contraction is very weak (~2%; Klass et al., 2007). Interestingly, the deficit in voluntary activation during lengthening contractions can be reduced with training (Aagaard et al., 2000; Colson et al., 1999) and is abolished in highly trained athletes (Amiridis et al., 1996). Third, the peak discharge rates of motor units in triceps brachii during maximal lengthening contractions are less than those during maximal shortening contraction (Del Valle and Thomas, 2005).

Together, the three sets of results indicate that voluntary activation is often not maximal during lengthening contractions in untrained individuals, but can be increased with practice and training.

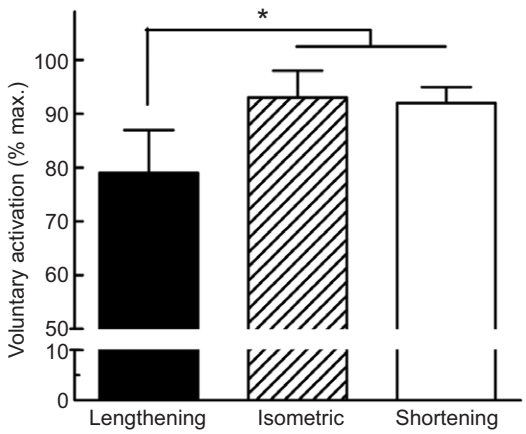

Fig. 1. Voluntary activation level during maximal isometric and anisometric contractions of the knee extensor muscles. To assess voluntary activation, three electrical stimuli (200 µs pulses at 300 Hz) were applied to the femoral nerve during maximal voluntary efforts (isometric and anisometric contractions at 60 deg s^{-1}) and when the muscle was relaxed and the knee joint passively moved. Stimulation was triggered at the same knee angle for the isometric and anisometric contractions and in active and passive conditions. The extra force induced by the electrical stimulation during each type of contraction is expressed relative to the force produced by the same stimulation in the relaxed muscle. The deficit in voluntary activation during lengthening contractions is significantly greater (*P<0.05) than that during isometric and shortening contractions. Data are means±s.e.m. for 8 subjects. Adapted from Beltman et al. (2004).

Muscle activation during submaximal contraction

In 1996, Enoka suggested in a review paper that lengthening contractions require a unique activation strategy by the nervous system (Enoka, 1996). His conclusion was based on experimental evidence that included a study in which the discharge of single motor units was recorded during a movement that involved lifting and lowering submaximal inertial loads (15–20% MVC force) with the plantar flexor muscles to match a defined trajectory (Nardone et al., 1989). This pioneering study reported that 15% and 50% of the motor units recorded in the soleus and gastrocnemii, respectively, were only recruited during lengthening contractions. These units had high recruitment thresholds and their activation was accompanied by the derecruitment of other units that were active during the shortening phase of the movement. This selective recruitment of high-threshold motor units (presumably fast-contracting muscle units) during the lengthening contractions occurred most often at faster angular velocities, which led Nardone and colleagues (1989) to suggest that such changes in task requirements may involve an adjustment in the recruitment order of motor units. Although another paper reported the occasional selective recruitment of high-threshold motor units (3 out of 21 units) in the first dorsal interosseous when lifting and lowering submaximal inertial loads (~15% MVC force; Howell et al., 1995), most subsequent studies have not found any systematic differences in motor unit recruitment between anisometric contractions when lifting and lowering relatively light inertial loads (≤20% MVC force; Bawa and Jones, 1999; Garland et al., 1996; Laidlaw et al., 2000; Søgaard, 1995; Søgaard et al., 1996; Stotz and Bawa, 2001) or assisting and resisting a torque motor with submaximal forces (≤50% of maximum; Altenburg et al., 2009; Pasquet et al., 2006; Stotz and Bawa, 2001).

As suggested by Bawa and Jones (1999), factors such as small stretches due to oscillations in muscle length during the braking phase (lengthening phase) or a slight variation in joint position at the transition between the shortening and lengthening phases may contribute to the occasional recruitment/derecruitment of some motor units. A slight shift in posture may also either change the force

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vector within the muscle and activate motor units from different muscular compartments (ter Haar Romeny et al., 1982), especially for bi-articular muscles such as the gastrocnemii (Nardone and Schieppati, 1988), or modify the relative contribution of synergistic muscles (Nakazawa et al., 1993; Nardone et al., 1989). To standardize the kinematics of dynamic contractions and reduce its influence on the discharge pattern of motor units, Pasquet and colleagues (2006) compared shortening and lengthening dorsiflexion actions when a torque motor was used to control ankle velocity. Each anisometric contraction began from an isometric contraction and a similar change in absolute force was compared. Ultrasonography was used to control the rate of change in muscle fascicle length during the shortening and lengthening actions. Except during the early transition between the isometric and anisometric phases of the task, muscle fascicle length changed linearly when the ankle joint moved over a 20 deg range of motion around the neutral position for contractions at target forces between 5% and 30% of maximum (Fig. 2A). The magnitude of the change in fascicle length and the average velocity did not differ significantly between shortening and lengthening actions. Under these conditions, motor units that were active during the shortening contraction were always active during the subsequent lengthening contraction. Furthermore, motor units that were recruited during the shortening contraction (high-threshold units), to compensate for the loss of force produced by the reduction in muscle length, were always derecruited first during the following lengthening contraction (Fig. 3). Moreover, motor units that were recruited or derecruited during the anisometric contractions had high recruitment thresholds during gradual, linear changes in force when performing isometric contractions, in agreement with the size principle (Duchateau and Enoka, 2011; Heckman and Enoka, 2012; Henneman, 1957).

Task requirements during anisometric contractions also influence the modulation of discharge rate. As the net muscle force must exceed the load during shortening contractions and be less than the load during lengthening contractions, most studies have reported that discharge rate declines when lowering an inertial load but not when lifting the load (Del Valle and Thomas, 2005; Kallio et al., 2013; Laidlaw et al., 2000; Semmler et al., 2002; Søgaard, 1995; Stotz and Bawa, 2001; Tax et al., 1989). A similar behaviour was observed when resisting instead of assisting a torque motor for a comparable change either in relative force (Altenburg et al., 2009) or in absolute force and fascicle length between the anisometric contractions (Pasquet et al., 2006). For example, discharge rate was nearly constant throughout the entire range of motion when performing a lengthening contraction, whereas it increased progressively during shortening contractions and reached greater average values than during lengthening contractions (Fig. 2B). The greatest difference in discharge rate between the two anisometric contractions, which was observed at short muscle lengths, was presumably due to an increased neural drive that was required to compensate for the reduced force capacity of muscle at that length.

In conclusion, most studies have found that the recruitment order of motor units is similar during submaximal shortening and lengthening contractions and consistent with the size principle. Nonetheless, additional studies are needed to examine whether the changes in recruitment order observed occasionally during lengthening actions performed in some conditions (fast contraction along a prescribed trajectory) are due to a specific strategy related to the task being performed or are solely the result of the mechanical conditions encountered by the muscle during its contraction. In contrast, the decline in the discharge rate of motor units during lengthening contractions is a consistent finding regardless of the type of load (inertial load or torque motor) and the intensity of contraction. Together, these observations indicate that the neural drive discharged by the spinal cord is less for lengthening than for shortening contractions. The lesser recruitment and discharge rate of motor units during lengthening contractions relative to shortening contractions performed with a similar absolute

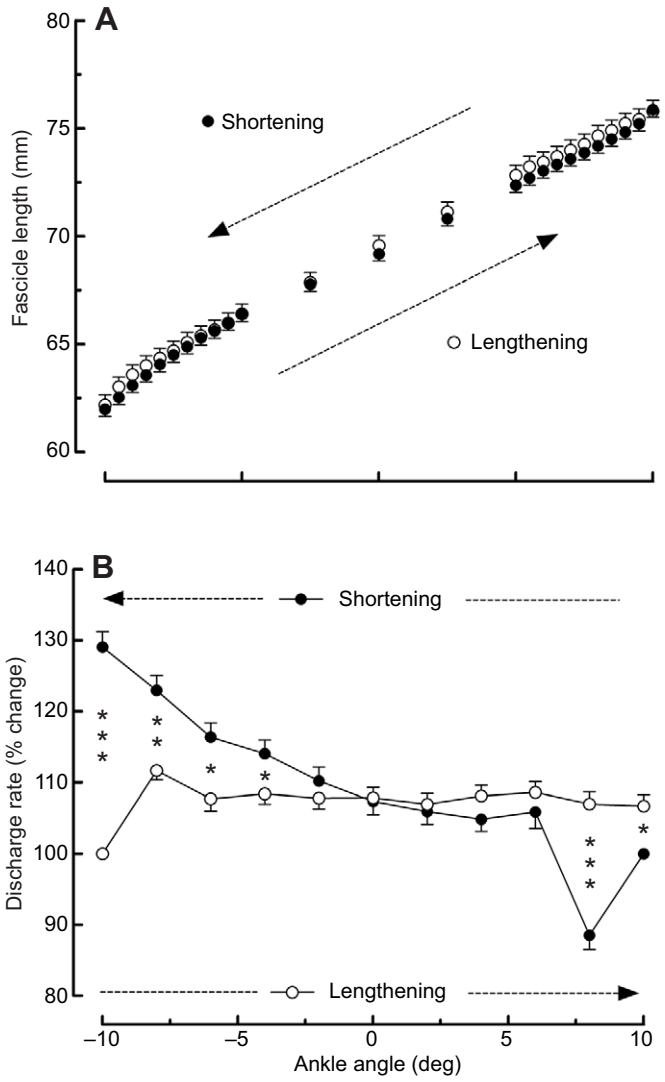

Fig. 2. Change in muscle fascicle length and modulation of motor unit discharge rate during anisometric contractions. Data were recorded in the tibialis anterior as the dorsiflexor muscles performed 2 s shortening and lengthening contractions against a torque motor over a 20 deg range of motion around the neutral position (0 deg). The shortening contraction began from an ankle angle of 10 deg (long length), whereas the lengthening contraction began from −10 deg (short length). (A) Changes in fascicle length (means±s.e.m.) of the tibialis anterior averaged across subjects (N=8) and over contraction intensities corresponding to the force recorded during motor unit recordings in B. Note that fascicle length changes nearly linearly and similarly for shortening and lengthening contractions. (B) Mean±s.e.m. (N=63) discharge rate of motor units in tibialis anterior. Each value, expressed as a percentage of the discharge rate recorded during the initial isometric contraction, was averaged over 0.2 s bins for all motor units and computed across contraction intensities. Note the increase in discharge rate when the muscle progressively shortened and the absence of modulation during lengthening contractions. Asterisks indicate significant differences between the two anisometric contractions (*P<0.05, **P<0.01, ***P<0.001). Data are from Pasquet et al. (2006).

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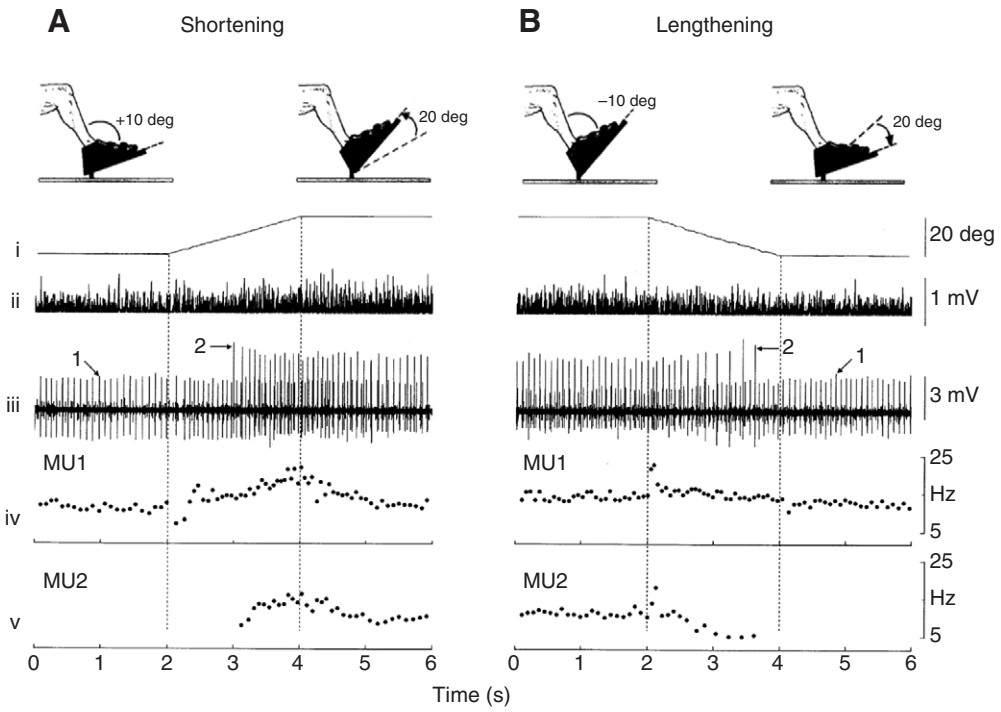

Fig. 3. Modulation of motor unit discharge rate during submaximal shortening and lengthening contractions. The discharge rate and recruitment of two motor units (MU1 and MU2) in tibialis anterior of one subject during an initial isometric contraction and the subsequent shortening (A) and lengthening (B) contractions with the dorsiflexor muscles. The vertical dotted lines indicate the beginning and end of each movement. The traces indicate, successively, angular ankle displacement (i), rectified surface EMG (ii) and intramuscular EMG (iii) of the tibialis anterior, and the instantaneous discharge rate of MU1 (iv) and MU2 (v). MU2 was recruited during the course of the shortening contraction and derecruited during the subsequent lengthening contraction. At the transition from the initial isometric contraction to the anisometric contraction there is either a transient decrease (shortening contraction) or increase (lengthening contraction) in discharge rate due to an unloading reflex or stretch reflex, respectively (iv,v). Note that there is greater modulation of discharge rate for both motor units during the shortening contraction than during the lengthening contraction. Adapted from Pasquet et al. (2006).

load (torque) is consistent with the lower fatigability usually observed during slow lengthening contractions in young adults (Pasquet et al., 2000; Tesch et al., 1990).

Control of maximal and submaximal lengthening contractions

The tension-regulating inhibitory hypothesis

An often-evoked suggestion for the lower voluntary activation during maximal lengthening contractions is the intervention of a tension-regulating mechanism intended to protect the muscle–tendon unit against excessive tension (Amiridis et al., 1996; Del Valle and Thomas, 2005; Gruber et al., 2009; Seger and Thorstensson, 2000; Westing et al., 1990, 1991). Despite earlier animal and human experiments having shown that the Golgi tendon organs (Ib inhibition; see Houk and Henneman, 1967; Priori et al., 1998) are not responsible for the 'clasp-knife' phenomenon triggered at high muscle forces (Rymer et al., 1979), the proponents of this protective strategy assume that the inhibitory action of the Golgi tendon organs is to depress the responsiveness of the motor neurones, thereby limiting the force produced by the muscle–tendon unit. However, Pinniger and colleagues (2000) observed that the normalized force–velocity relationship of the plantar flexor muscles was similar during maximal and submaximal (30% of isometric MVC) lengthening contractions, but not when the muscle was activated by electrical stimulation. Under these conditions, the evoked force was substantially greater during lengthening contractions than during isometric contractions. The force depression during voluntary lengthening actions, therefore, is not limited to maximal contractions but is evident during the voluntary control of lengthening contractions. Moreover, EMG amplitude is depressed during a maximal isometric contraction that precedes the change in muscle length of a subsequent lengthening contraction (Grabiner and Owings, 2002).

Taken together, these findings cast doubt on the concept of tension-related inhibitory control (Pinniger et al., 2000; Duclay et al., 2014) and raise uncertainty about the mechanisms responsible for the modulation of muscle activation during both submaximal and maximal lengthening contractions (Duchateau and Baudry, 2014). The lower motor unit discharge rates during both maximal (Del Valle and Thomas, 2005) and submaximal (Altenburg et al., 2009; Kallio et al., 2013; Laidlaw et al., 2000; Pasquet et al., 2006; Semmler et al., 2002; Søgaard, 1995; Stotz and Bawa, 2001; Tax et al., 1989) lengthening contractions relative to shortening contractions suggests the involvement of either supraspinal or spinal constraints that limit the neural drive to muscle.

Modulation of cortical and spinal excitability

The modulation of cortical pathways in humans during a motor task can be assessed non-invasively with a painless electrophysiological method known as transcranial magnetic stimulation (TMS). The recording of motor-evoked potentials (MEPs) from surface EMG in response to a single TMS pulse indicates the responsiveness of the corticospinal tract during muscle activity (Fig. 4A; Rossini et al., 2015). The amplitude of the MEP can be influenced by changes at both the cortical and spinal level. In addition, the duration of the silent period in the ongoing EMG activity that follows the MEP provides an index of intracortical inhibition when it lasts longer than 100 ms (Inghilleri et al., 1993).

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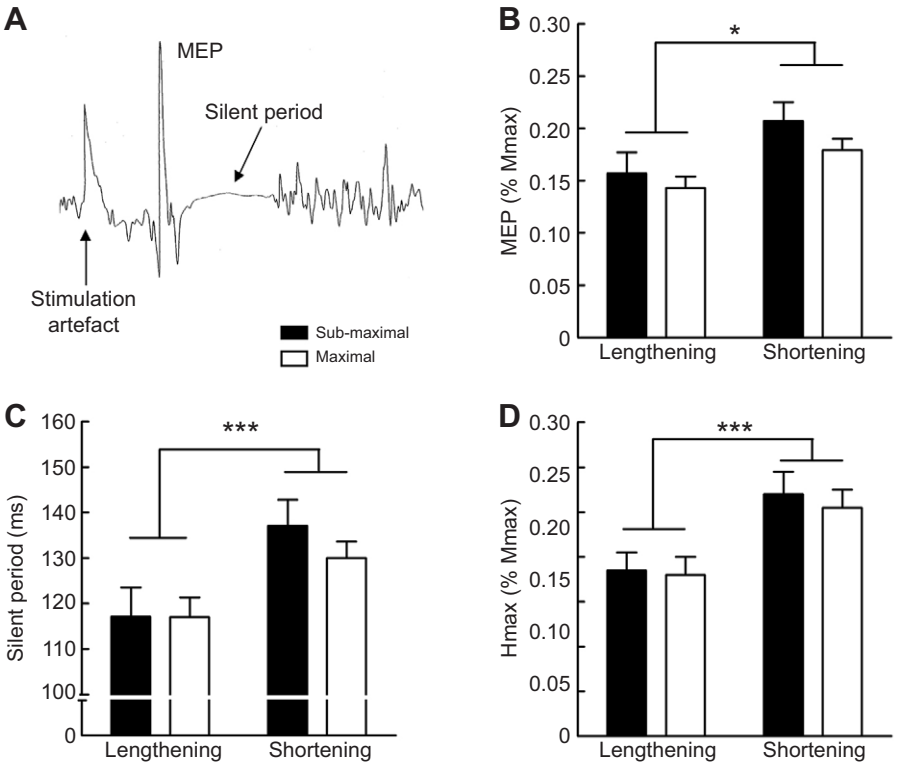

Fig. 4. Modulation of corticospinal and spinal responsiveness during submaximal and maximal shortening and lengthening contractions. (A) A motor-evoked potential (MEP) in the soleus of one subject induced by transcranial magnetic stimulation (TMS) and the corresponding silent period in the ongoing EMG activity. The duration of the silent period was measured from the stimulus artefact to the return of continuous EMG activity. (B,C) The modulation of MEP amplitude (B) and the duration of the silent period (C). (D) The modulation of H-reflex amplitude (Hmax). The MEP and Hmax are normalized to the corresponding maximal M-wave (Mmax) obtained in response to a supramaximal electrical stimulus. Data are means ±s.e.m. for 11 subjects. Note the similar modulation of all parameters for maximal and submaximal lengthening contractions compared with shortening contraction. Asterisks indicate significant differences between lengthening and shortening contractions (*P<0.05, ***P<0.001). Data are from Duclay et al. (2014).

The size of the MEP in response to TMS is reduced for many muscles during lengthening contractions compared with shortening or isometric contractions. The reduction has been reported both when resisting an imposed force at a constant velocity (Duclay et al., 2011; Gruber et al., 2009) and when lowering an inertial load even with the background EMG activity matched, a method that controls partly for differences in the recruitment gain in the motor neurone pool (Pierrot-Deseilligny and Burke, 2012), during both types of anisometric contractions with the elbow flexor (Abbruzzese et al., 1994; Sekiguchi et al., 2001) and soleus (Sekiguchi et al., 2003) muscles. With this approach, MEP amplitude is reduced in the soleus during a maximal lengthening contraction of the plantar flexors compared with a maximal shortening contraction (Fig. 4B; Duclay et al., 2011). Furthermore, the duration of the silent period is significantly briefer for lengthening than for shortening contractions (Fig. 4C). Interestingly, the reduction in MEP amplitude and in silent period duration during the lengthening contractions is of a similar magnitude for maximal and submaximal (50% MVC) contractions (Duclay et al., 2014).

Several studies have shown that spinal mechanisms contribute to the reduction in activation during lengthening contractions. A classic method used to assess the modulation of spinal pathways in humans under different conditions is to record the Hoffmann (H) reflex (Pierrot-Deseilligny and Burke, 2012). H-reflex amplitude, which is elicited by the electrical stimulation of the Ia afferents originating from muscle spindles, can be modulated by changes in the effectiveness of Ia synaptic input and the responsiveness of the motor neurones. H-reflex amplitude is typically depressed during lengthening contraction for both submaximal contractions with inertial loads and isokinetic actions (Abbruzzese et al., 1994; Nordlund et al., 2002; Romanò and Schieppati, 1987; Sekiguchi et al., 2003) and maximal isokinetic actions (Duclay and Martin, 2005; Duclay et al., 2011, 2014). For example, Duclay and colleagues (2014) found that H-reflex amplitude in soleus was lower during lengthening contractions than during shortening contractions for both submaximal (50% MVC) and maximal contractions with the plantar flexor muscles. Moreover, the reduction in soleus H-reflex amplitude was similar for both maximal and submaximal contractions (Fig. 4D).

Although the H-reflex results and those obtained with TMS indicate that both spinal and supraspinal mechanisms are involved in the modulation of muscle activation during lengthening contractions, the relative decrease was greater for soleus H-reflex amplitude than for MEP amplitude, for both submaximal and maximal lengthening contractions, which indicates a greater influence on the excitability of the spinal pathway than on the corticospinal tract (Duclay et al., 2014). The similar modulation of spinal and corticospinal responsiveness regardless of contraction intensity further argues against the hypothesis of a tension-regulating inhibitory mechanism that limits muscle activation during maximal lengthening contractions.

Potential mechanisms underlying the control of lengthening contractions

Supraspinal mechanisms

Few studies have investigated the modulation of cortical output during lengthening contractions. One approach that has been used is to probe changes in the levels of cortical inhibition and facilitation with the paired-pulse TMS technique (Kujirai et al., 1993). The level of inhibition can be estimated from the magnitude of the depression of the response elicited by a second pulse (test pulse) that follows a subthreshold pulse (conditioning pulse) at a brief interstimulus interval (1–5 ms); this is known as short-interval intracortical inhibition. Conversely, the level of facilitation can be estimated from the magnitude of the increase in amplitude of the second response after a longer interstimulus interval (7–20 ms); this measure is known as intracortical facilitation. With this approach, Howatson and colleagues (2011) found that short-interval

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intracortical inhibition was significantly reduced, whereas intracortical facilitation was increased in the ipsilateral motor cortex during lengthening contractions, but not during shortening contractions. These observations, in addition to the reduced duration of the silent period (Duclay et al., 2011, 2014), suggest that cortical responsiveness is augmented in both contralateral and ipsilateral motor cortices during lengthening contractions relative to shortening contractions and that the modulation of networks involved in intracortical and interhemispheric connections varies with the type of contraction.

These observations are consistent with the study by Gruber and colleagues (2009) that compared the size of an MEP elicited by TMS and one elicited by stimulation of the cervicomedullary junction (CMEP) in the elbow flexor muscles. As the CMEP does not involve cortical neurones but instead indicates the responsiveness of motor neurones (Nielsen and Petersen, 1994; Taylor, 2006), the 21% greater MEP/CMEP ratio during lengthening contractions relative to isometric contractions suggests an augmented cortical responsiveness during lengthening contractions. These findings are consistent with those of Fang and colleagues (2001, 2004), wherein the movement-related cortical potential recorded by electroencephalography was greater during the lowering phase relative to the lifting phase of a movement in which the elbow flexor muscles displaced a submaximal load and when subjects performed constant-velocity anisometric MVCs. With the exception of the work by Hahn and colleagues (2012), most studies have found that a greater brain area is involved in the control of lengthening contractions than other types of contractions regardless of load type (inertial load or torque motor). The greater responsiveness of the motor cortex during lengthening contractions has been interpreted as indicating extra excitatory descending drive to compensate for spinal inhibition (Gruber et al., 2009). It is uncertain whether such modulation involves the activation of different cortical areas during lengthening and shortening contractions, as suggested by functional MRI (Kwon and Park, 2011) and by the selective modulation of descending pathways mediating spinal presynaptic inhibition during lengthening contractions but not during isometric and shortening contractions (Grosprêtre et al., 2014).

Spinal mechanisms

The greater decrease in the amplitude of the CMEP elicited by electrical stimulation of the cervicomedullary junction relative to the MEP evoked by TMS during lengthening contractions has been attributed to mechanisms that influence the input and output at the motor neurone level (Gruber et al., 2009). Various presynaptic and postsynaptic mechanisms of the motor neurone may contribute to the reduction in responsiveness during lengthening contractions.

A depression of the H-reflex amplitude during lengthening contractions has been reported frequently for different load types (inertial load and torque motor) during submaximal (Abbruzzese et al., 1994; Duclay et al., 2014; Nordlund et al., 2002; Romanò and Schieppati, 1987; Sekiguchi et al., 2003) and maximal (Duclay and Martin, 2005; Duclay et al., 2011, 2014) contractions. Because H-reflex amplitude is also depressed during passive lengthening (Duclay and Martin, 2005; Duclay et al., 2011; Nordlund et al., 2002; Pinniger et al., 2001), and voluntary lengthening contractions are accompanied by an increase in the amount of facilitatory feedback from muscle spindles (Burke et al., 1978; Hulliger et al., 1985), some of the spinal modulation during lengthening contractions has been attributed to mechanisms located at the presynaptic side of the motor neurones (Abbruzzese et al., 1994; Duclay and Martin, 2005; Duclay et al., 2011, 2014; Grosprêtre et al., 2014; Romanò and Schieppati, 1987). Presynaptic inhibition can be produced by two mechanisms: homosynaptic post-activation depression due to an activity-dependent reduction in neurotransmitter release at the Ia terminals (Hultborn et al., 1987) and primary afferent depolarization through inhibitory interneurones that mediates Ia presynaptic inhibition (Rudomin and Schmidt, 1999). The latter mechanism is controlled centrally and modulated continuously during muscle activity (Fig. 5; for more details, see Pierrot-Deseilligny and Burke, 2012). Both inhibitory mechanisms may reduce the responsiveness of the motor neurone pool during lengthening contractions through disfacilitation, but homosynaptic post-activation depression appears to have a greater influence at rest than during muscle contraction (Petersen et al., 2007). Consistent with a more dominant role of presynaptic inhibition by primary afferent depolarization during lengthening contractions, Grosprêtre and colleagues (2014) conditioned the H-reflex with a subthreshold stimulation of the motor cortex area and found that descending pathways appear to control spinal inhibition during lengthening contractions.

Indirect experimental evidence suggests, however, that postsynaptic inhibitory mechanisms may also influence spinal excitability (Duclay et al., 2011). The main potential postsynaptic mechanisms include Ib inhibition (Golgi tendon organs), reciprocal inhibition and recurrent (Renshaw) inhibition (Fig. 5; Pierrot-Deseilligny and Burke, 2012). Although Golgi tendon organs may contribute to the modulation of spinal excitability through Ib

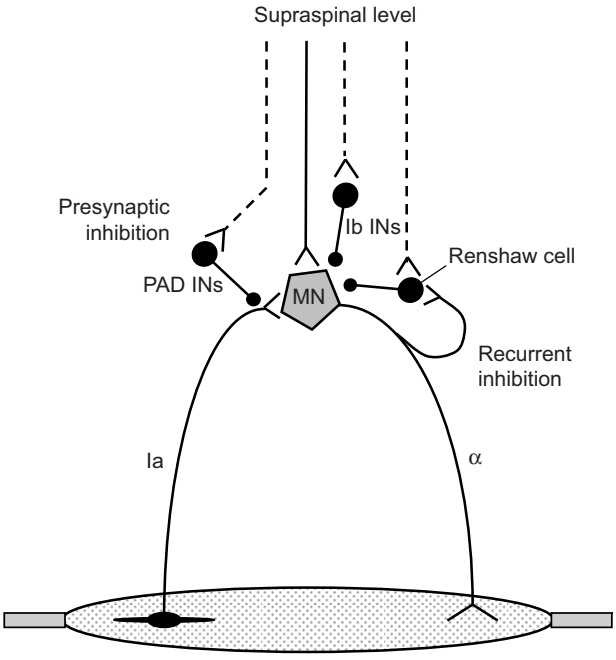

Fig. 5. Schematic diagram of the main spinal networks likely to modulate motor neurone excitability during lengthening contractions. The descending motor command (continuous line) to the motor neurone (MN) pool can be modulated at the spinal level by changes in synaptic inputs from peripheral afferents and descending pathways. The potential modulators include the primary afferent depolarization interneurones (PAD INs), Renshaw cell (recurrent inhibition) and Ib interneurones (Ib INs). PAD interneurones act at a presynaptic level and contribute to disfacilitation of the MN pool, whereas recurrent inhibition and Ib inhibition act at a postsynaptic level and may reduce the responsiveness of the MN pool. For more details, including the sensory afferents that project onto the inhibitory interneurones, see Pierrot-Deseilligny and Burke (2012).

interneurones, the similar depression of muscle activation at low and high forces (Duclay et al., 2014; Pinniger et al., 2000) suggests that other spinal mechanisms are involved in the specific adjustments observed during lengthening contractions. As for Ib inhibition, reciprocal inhibition does not seem to play a major role in contributing to differences in spinal excitability between lengthening and shortening contractions. For example, most studies have found no substantial difference in the level of antagonist co-activation during the two anisometric contractions (Aagaard et al., 2000; Amiridis et al., 1996; Duclay et al., 2011; Pasquet et al., 2006; Pinniger et al., 2003). In contrast, excitability of the motor neurone pool may be modulated through Renshaw cells (recurrent inhibition; Fig. 5) that may reduce motor unit discharge rate during lengthening contractions (Del Valle and Thomas, 2005; Laidlaw et al., 2000; Pasquet et al., 2006; Semmler et al., 2002; Søgaard et al., 1996; Stotz and Bawa, 2001; Tax et al., 1989). For example, animal experiments have shown that descending pathways can modulate recurrent inhibition and thereby motor unit discharge rate (Baldissera et al., 1981), which could serve as a variable gain regulator for motor output (Hultborn et al., 1979). Such control seems plausible given that the amount of recurrent inhibition differs during co-activation and flexion–extension movements (Nielsen and Pierrot-Deseilligny, 1996). However, this possibility needs to be substantiated with experimental data.

Conclusions

The amount of motor unit activity usually differs during shortening and lengthening contractions, regardless of whether the task involves lifting an inertial load or pushing against a torque motor. In untrained individuals, voluntary activation during lengthening MVCs is usually less than that measured during shortening contractions. Nonetheless, the difference between the two types of anisometric contractions differs across muscle, the biomechanical requirements of the task, and the adaptations that accompany ageing and training. The deficit in voluntary activation, however, is not the consequence of an inhibitory mechanism related to the degree of tension produced by the muscle. The neural drive to muscle for lengthening contractions has similar qualities for both submaximal and maximal contraction, suggesting that the CNS generates a specific activation strategy for lengthening contractions (Enoka, 1996). Moreover, mechanisms located at both supraspinal and spinal levels are involved in generating the specific modulation observed during lengthening contractions, but the inhibition mainly occurs at the spinal level as mediated by descending pathways from supraspinal centres (Fig. 5). Although mechanisms located on both the presynaptic and postsynaptic sides of the motor neurone can influence motor output, the dominant mechanism remains unknown.

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