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Neural Adaptations to Resistive Exercise

Mechanisms and Recommendations for Training Practices

David A. Gabriel,[1] *Gary Kamen*[2] and *Gail Frost*[1]

1 Department of Physical Education and Kinesiology, Brock University, St Catharines, Ontario, Canada
2 Department of Exercise Science, University of Massachusetts at Amherst, Amherst, Massachusetts, USA

Contents

Abstract

It is generally accepted that neural factors play an important role in muscle strength gains. This article reviews the neural adaptations in strength, with the goal of laying the foundations for practical applications in sports medicine and rehabilitation.

An increase in muscular strength without noticeable hypertrophy is the first line of evidence for neural involvement in acquisition of muscular strength. The use of surface electromyographic (SEMG) techniques reveal that strength gains in the early phase of a training regimen are associated with an increase in the amplitude of SEMG activity. This has been interpreted as an increase in neural drive, which denotes the magnitude of efferent neural output from the CNS to active muscle fibres. However, SEMG activity is a global measure of muscle activity. Underlying alterations in SEMG activity are changes in motor unit firing patterns as measured by indwelling (wire or needle) electrodes. Some studies have

reported a transient increase in motor unit firing rate. Training-related increases in the rate of tension development have also been linked with an increased probability of doublet firing in individual motor units. A doublet is a very short interspike interval in a motor unit train, and usually occurs at the onset of a muscular contraction. Motor unit synchronisation is another possible mechanism for increases in muscle strength, but has yet to be definitely demonstrated.

There are several lines of evidence for central control of training-related adaptation to resistive exercise. Mental practice using imagined contractions has been shown to increase the excitability of the cortical areas involved in movement and motion planning. However, training using imagined contractions is unlikely to be as effective as physical training, and it may be more applicable to rehabilitation.

Retention of strength gains after dissipation of physiological effects demonstrates a strong practice effect. Bilateral contractions are associated with lower SEMG and strength compared with unilateral contractions of the same muscle group. SEMG magnitude is lower for eccentric contractions than for concentric contractions. However, resistive training can reverse these trends. The last line of evidence presented involves the notion that unilateral resistive exercise of a specific limb will also result in training effects in the unexercised contralateral limb (cross-transfer or cross-education). Peripheral involvement in training-related strength increases is much more uncertain. Changes in the sensory receptors (i.e. Golgi tendon organs) may lead to disinhibition and an increased expression of muscular force.

Agonist muscle activity results in limb movement in the desired direction, while antagonist activity opposes that motion. Both decreases and increases in co-activation of the antagonist have been demonstrated. A reduction in antagonist co-activation would allow increased expression of agonist muscle force, while an increase in antagonist co-activation is important for maintaining the integrity of the joint. Thus far, it is not clear what the CNS will optimise: force production or joint integrity.

The following recommendations are made by the authors based on the existing literature. Motor learning theory and imagined contractions should be incorporated into strength-training practice. Static contractions at greater muscle lengths will transfer across more joint angles. Submaximal eccentric contractions should be used when there are issues of muscle pain, detraining or limb immobilisation. The reversal of antagonists (antagonist-to-agonist) proprioceptive neuromuscular facilitation contraction pattern would be useful to increase the rate of tension development in older adults, thus serving as an important prophylactic in preventing falls. When evaluating the neural changes induced by strength training using EMG recording, antagonist EMG activity should always be measured and evaluated.

1. Rationale for the Existence of a Neural Mechanism

The scientific literature concerning mechanisms underlying strength increases through resistance training is marked largely by references to the role of muscle mass. There are other strength-related changes as well, such as alterations in the contractile characteristics of the muscle,[1,2] and muscle architectural changes.[3] However, that muscle mass is important for muscular strength is indisputable. There is also little question that neural factors play an important role in muscle strength gains.[4-7] This article reviews the neural adaptations in maximal muscle strength, with the goal of laying the foundations for practical applications in sports medicine and rehabilitation.

The role of these neural factors is particularly strong during the early phase of strength training. Although protein synthesis is noticeable after a single strength training session,[8] overt changes in muscle hypertrophy are not observed until the eighth week of exercise training.[9-13] This delay, concurrent with a substantial gain in muscle strength, has led some to suggest that neural factors are important.[12,14]

One of the earliest suggestions that muscle strength gains could be achieved very quickly is found in the physical therapy literature. The mere assessment of maximal knee extensor force results in an increase in muscular strength.[15,16] This rapid increase in muscular strength over a period of days, has since been replicated in the wrist flexors,[17] dorsiflexors,[14] elbow extensors[18] and several intrinsic finger muscles.[19,20]

2. Evidence for Neural Enhancement

Data from surface-recorded electromyographic (SEMG) techniques are dependent on the biophysical characteristics of both muscles and motor neurons.[21-23] As detailed in the following sections, changes in EMG amplitude not accompanied by hypertrophy or changes in membrane characteristics are evidence of alterations in neural drive to the muscle. These alterations in neural drive could be central or peripheral in origin.

2.1 Neural Drive

Increases in the amplitude of the SEMG signal appear well before increases in muscle size.[12] Numerous other investigators have noted increases in EMG amplitude during strength training,[11,24-32] although some laboratories have failed to observe such an increase.[33,34]

Likewise, disuse results in a decrease in muscle electrical activity. For example, EMG amplitude during maximal effort decreased following a period of limb immobilisation in both human[35-38] and animal models.[39] One interpretation of this finding might be that the decrease in EMG amplitude is caused by a decrease in central drive to the muscle. However, in the case of limb immobilisation, decreases in muscle fibre size could cause a decrease in EMG amplitude. Increases in intramuscular fat and elevated amounts of collagen associated with long-term immobility could also decrease EMG amplitude.[40,41] Further evidence of the role of central drive is seen in the retention of strength values and EMG amplitude during periods of rest. In the case of Gabriel and colleagues,[18,42] elbow strength and EMG amplitude were retained over a 2-week period following a limited number of contractions (i.e. 35) administered on 1 day. Repeated testing at 2-week intervals is a training stimulus wherein subjects 'learn' to maximally activate their muscles.[43] Physiological adaptations to a single test session should be dissipated over a 2-week rest period.[44]

2.1.1 Motor Unit Activation

Studies have shown that humans are unable to fully activate muscle voluntarily,[45-47] but that training improves activation. The general term 'incomplete motor unit activation' has been used to imply possible limitations to either motor unit recruitment or firing rate. Twitch interpolation techniques have been used to measure motor unit activation. While a subject performs a maximal contraction, one or more supramaximal stimuli are delivered to the muscle to activate any muscle fibres not already activated by the voluntary contraction. This twitch interpolation technique generally reveals that full activation of the muscle is not possible during maximal effort: about 2–5% additional force is generally

observed immediately following stimulation.[47] The magnitude of overall muscular activation increases with training,[47] and this improved muscular activation could result in higher discharge rates.

Resistance Training and Motor Unit Discharge Behaviour

Motor Unit Firing Rate

Although the precise relationship between force and firing rate remains unclear, one possible factor that could account for rapid increases in muscular force at the onset of strength training is motor unit firing rate. To date, there are only a limited number of training studies.[2,19,48,49] The influence of resistance exercise training on motor unit firing rates has been investigated during maximal fifth finger abduction force.[19] The abductor digiti minimi is not frequently used during daily activities and is therefore a good candidate for resistance exercise adaptations. Maximal force was tested on two occasions spaced at 1-week intervals. As expected, there were notable strength increases 1 week after the initial test session. More importantly, motor unit firing rate during maximal voluntary contractions (MVC) was elevated in the second test session. The increase in motor unit firing rate, in both the trained hand and the untrained contralateral hand, suggests that it may well be a mechanism for the early increase in muscular strength.

Subjects then underwent a strength-training regimen for 6 weeks. Over the 6-week training period, maximal force increased. However, maximal firing rates actually decreased, so that firing rates at the end of the 6-week training interval were similar to those measured on the first test day. That some neural mechanism is operative was supported by the observation that similar changes were observed in the untrained, contralateral muscle group. Apparently, the neural mechanism producing the early increase in motor unit discharge rate is moderated as other adaptations begin. One possible mechanism for changes seen after initial adaptations may be a reduction in antagonist co-activation.[50]

In a subsequent investigation, firing rates in the vastus lateralis were measured while subjects underwent 6 weeks of resistance exercise training in a large muscle group: the knee extensors.[48] First, muscular force and maximal motor unit firing rates were assessed in two test sessions spaced 48 hours apart. Even this 48-hour interval was sufficient to produce a rapid increase in muscular strength and increases in motor unit firing rate. The increases in motor unit firing rate were correlated with the increases in muscular strength, providing further evidence that rapid, early gains in muscular strength are mediated by whatever mechanism is producing the increase in motor unit firing rate. Six weeks of strength training for the knee extensors increased vastus lateralis motor unit firing rates 15% in younger adults and 49% in older adults during maximal isometric knee extension. Knee extensor strength increased about 35%. The results of these two strength-training studies suggest that a single strength testing session is associated with rapid and/or transient changes in maximal motor unit firing rates. However, the importance of neural drive (as measured by motor unit firing rate) seems to decrease considerably after 6 weeks of exercise training.

Another important finding of the Kamen and Knight[48] study was that motor unit firing rates were unchanged when assessed at 10% and 50% of MVC. Rich and Cafarelli[2] were also unable to detect differences in motor unit firing rates of the vastus lateralis at 50% of MVC after 8 weeks of maximal isometric strength training for knee extensors. It is possible that differences in motor unit firing rate do not manifest themselves at lower levels of force, and testing must include maximal effort contractions.

Doublet Firing

There is some evidence that the pattern of motor unit activation can be just as important as the number of motor units activated or the frequency of activation in producing increases in muscular strength. A single extra spike or a missed spike in a train of motor unit potentials can have a dramatic effect, either increasing or decreasing muscular force.[51] The onset of a muscular contraction is often marked by a short interspike interval in a motor unit train. Such a 'doublet' firing pattern is most frequent when the rate of force development or

the speed of contraction is high.[49] One possible reason for the initial doublet firing is to produce a rapid increase in muscular force within the initial phase of contraction. Van Cutsem et al.[49] have shown that the frequency of short interspike intervals (doublets) increases following exercise training. So it is possible that adaptations in doublet firing occur to produce rapid increases in muscular strength.

Motor Unit Synchronisation

Another pattern of motor unit activation that might result in augmented force involves the simultaneous activation of numerous motor units, termed 'synchronisation'. In an early study, Milner-Brown et al.[52] used an SEMG technique to study motor unit synchronisation in the thenar muscles of British bus drivers. Using a resistance exercise training protocol to observe the influence of training on motor unit synchronisation, they concluded that motor unit synchronisation increases with exercise training. However, the validity of the SEMG technique to study motor unit synchronisation has since been questioned.[53] Semmler and Nordstrom[54] observed differences in motor unit synchronisation between strength-trained subjects, musicians and controls, with a protocol that measured motor unit discharges directly. The degree of motor unit synchronisation increased in order, from untrained to skilled (musicians) to strength-trained subjects.

Mechanisms Underlying Changes in Motor Unit Discharge Behaviour

Spinal Mechanisms

It is possible that changes in the intrinsic characteristics of motoneurons are responsible for the enhanced motor unit activation observed during the early phases of strength training. Motoneuron excitability has been reported to be greater in well trained athletes[55] and if training improves motoneuron excitability, we might expect motor unit discharge rates to increase with training. However, greater motoneuron excitability following resistance exercise has yet to be demonstrated in a definitive way.

Motoneuron excitability is frequently assessed using the Hoffman (H) reflex, which measures the amplitude of the evoked response produced by stimulating large diameter afferent nerve fibres.[56] Learning experiments utilising operant conditioning demonstrate that the excitability of the spinal stretch reflex can be either enhanced or diminished, depending on the command given to the subject.[57-59]

Spinal reflexes are affected by both training[60] and detraining.[61] It has been suggested that changes in motoneuron excitability during movement are mediated by presynaptic inhibition.[62,63] Motoneuron firing (through recurrent collaterals) produces a facilitation of spinal inhibitory interneurons called Renshaw cells. These Renshaw cells synapse on the motoneuron, providing a potential limit to motor unit firing rate, and also serving to 'shape' the characteristics of purposeful movements.[64] Renshaw inhibition can be moderated by both spinal and supraspinal centres. Although Earles et al.[65] found greater recurrent inhibition in power-trained athletes than in endurance-trained athletes, there is no direct evidence of training-related adaptations.

The literature includes both cross-sectional comparisons between differently trained individuals[65-69] as well as training studies to monitor the actual adaptation.[26,70] It is important to recognise that the H-reflex is not only affected by presynaptic inhibition,[71] but alterations may also reflect training-related adaptations in motoneuron properties.[21,22,72] Interpretations of these H-reflex studies should, therefore, be made within this context.

Sale et al.[70] measured an H-reflex variant called the V-wave (V1). The V-wave is an H-reflex recorded during a muscle contraction; it therefore incorporates the central descending input from higher motor centres to the spinal motoneuron pool.[25,73] When recorded in selected muscles of the hand and lower limb, sprinters and weight-lifters demonstrated elevated V-wave (V1) amplitudes relative to untrained control subjects.[52,74] Increased V-wave amplitudes have also been observed in response to strength training,[25,70] indicating elevated neural drive in descending corticospinal pathways, increased motoneuron excitability and/or decreased presynaptic inhibition of Ia afferents.[4]

Other possible mechanisms include activity-related changes in synaptic efficiency.[75] The histo-

chemical and morphological characteristics of motoneurons are affected by physical activity.[76,77] Hypertrophic changes also occur in motoneuron axons following periods of increased use.[78-80] This may result in faster motor nerve conduction velocities.[81]

Adaptations in reflex excitability in older men (65–80 years) following a 14-week strength training programme, as assessed by the maximum Hoffman reflex amplitude to maximum wave amplitude (H_{max}-to-M_{max}) ratio, were found to be absent.[82] The authors concluded that adaptability was impaired by age-related (degenerative) changes in the neuromuscular system. However, a methodological consideration is that the H-reflex was recorded at rest. Previous studies have reported that the H-reflex measured during actual muscle contraction (V-wave) increased with training, whereas resting H-reflex amplitude remained unchanged.[25,83] These findings strongly advocate that the neural adaptation to strength training should be evaluated based on H-reflex recordings obtained during actual muscle contractions, and not solely on measurements obtained at rest.

Descending Activation

There are several different lines of evidence that weight training can increase descending activation from the motor cortex. The first of these involves motor imagery, which highlights the role of the motor cortex in resistive exercise. Motor imagery is a type of mental practice – i.e. practicing a task in the absence of movement. It is logical to expect that the corticospinal pathway is activated to a greater degree during muscle contraction compared with imagery, since no motoneurons are activated during imagery. Transcranial magnetic stimulation (TMS), functional magnetic resonance imaging (fMRI), and event-related potential studies (ERPS) have all demonstrated that cortical areas are involved in motor imagery to the same degree as in movement.[84-88] Ranganathan and colleagues[89] concluded that mental training enhances the cortical output signal, which drives the muscles to a higher activation level and increases strength.

Mental practice training involving imagined muscle contractions was first studied by Bowers,[90] who used the term 'autosuggested muscle contractions'. He developed a system of exercising muscle by imagining positive and vivid, strong muscular contractions. Individuals further imagined the muscles contracting against the resistance of a very heavy weight. During the exercise, subjects were in a relaxed state of deep concentration but not in a state of hypnosis. Autosuggested contractions were shown to result in a rapid increase in elbow flexion strength and biceps brachii SEMG during the first half of a 6-week study, but these measures later plateaued. In contrast, maximal effort isometric contractions of the elbow flexors continued to produce gains in strength and SEMG of the elbow flexors throughout the duration of the study. Yue and Cole[91] also observed benefits in muscular strength attributed to mental practice training, although contradicting evidence has also been presented.[92] More recently, Ranganathan et al.[89] resolved some of the controversy by demonstrating that imaginary training was more effective in the fifth finger abductor than in the elbow extensors. Cortical potentials obtained using EEG techniques were greater in amplitude after 'mental MVC' training. Imaginary training is unlikely to be as effective as physical training, and it may be more applicable to rehabilitation.

Changes in the bilateral deficit phenomenon also point toward alterations in descending activation. The force generated by two limbs simultaneously is generally less than the sum of the forces produced by the two limbs, separately. The lower force level generated during simultaneous bilateral contraction has been attributed to interhemispheric inhibition, which ultimately translates to reduced neural drive.[93] Resistance training has been shown to actually shift the bilateral deficit in a positive direction.[94,95] That is, the force generated by two limbs simultaneously is greater than the sum of the forces produced by either limb.

2.1.2 Cross-Transfer

Activation of the contralateral heteronymous muscle group is particularly effective in facilitating the motoneuron pool. For example, brief muscle contraction of the knee flexors increases maximal force of the contralateral knee extensors,[96] but mus-

cle contraction involving the knee extensors impedes contralateral knee extension muscular force output.[97,98] These studies suggest that exercise training involving contralateral heteronymous muscle groups (e.g. left wrist extensors enhancing muscular strength in right wrist flexors) might produce a potent training effect in the untrained muscle group. It is interesting to note that training with electrical stimulation can also result in contralateral limb strength gains.

The notion that contralateral limb exercise might produce long-term benefits, a phenomenon called 'cross-education' or 'cross-transfer', is actually over a century old,[99] although the mechanism remains unclear. Early physical rehabilitation techniques frequently used bilateral reciprocal exercise for spastic hemiplegic cerebral palsy patients.[100] In healthy individuals, 8 weeks of isometric exercise training can increase the strength of the contralateral elbow flexors by as much as 25%.[12] This cross transfer of strength phenomenon is actually quite effective in both the upper limbs[101,102] and the lower limbs.[103-105]

The increase in maximal force produced by contralateral resistance exercise training is generally greater following training using dynamic rather than static, isometric contractions.[106,107] Muscle lengthening exercise is particularly effective,[108] resulting in strength gains in the contralateral limb as high as 77%,[31] although these eccentric contractions may also produce muscle soreness.[109,110] Thus, it is clear that increases in muscular force can be obtained by resistance exercise training involving the contralateral limb.

It could be argued that the marked contralateral training effects observed with muscle lengthening exercises (eccentric exercises) are explained by the effect of learning, induced by the initial strength test that included maximal voluntary eccentric contractions – also in the contralateral untrained leg. However, Hortobágyi et al.[31] were quite careful to include pre-test sessions to control for the learning effect and document reliability. Moreover, the use of a control group also helps minimise potential learning effects.

What is the Mechanism for Cross-Transfer?

The increase in strength of the contralateral muscle group is usually not associated with an increase in limb girth,[90,107] suggesting a possible neural origin for the strength gains. Indeed, the existence of a neural factor in this 'cross transfer' of muscular strength phenomenon is suggested by the absence of a change in muscle fibre area or enzymatic activity,[111] and an enhancement of myoelectric activity in the unexercised muscle group.[112] In the absence of physiological or biochemical change in the muscle, increased SEMG activity is good evidence of a change in neural drive to the muscle.

The force enhancement from contralateral resistance exercise training could result from facilitation of suprasegmental neurons activated during either an ipsilateral or a contralateral contraction.[112] We have long known that muscle activation results in activation of cortical neurons related to contralateral homologous muscle groups.[113] Mental practice training has also been effective in producing strength gains in the contralateral muscle group,[91] and this could also be attributed to increased activation of cortical neurons.

Alternatively, the cross-transfer effect could result from an alteration in a segmental pathway involving reciprocal inhibition or presynaptic inhibition, or increases in contralateral motoneuron excitability caused by cutaneous receptor activation.[114] Whatever the mechanism for the cross-transfer effect, it is clear that there is a need to document the efficacy of this treatment to attenuate functional disuse atrophy.

2.1.3 Adaptations in Force Sensation

Activation of the Golgi tendon organs results in autogenic inhibition of the agonist via Ib inhibitory interneurons and facilitation of the antagonist via inhibition of the Ia inhibitory interneuron (disinhibition). The same circuitry is used for both cutaneous and joint afferents, and it is difficult to distinguish their effects. These receptors serve as a protective mechanism to prevent the generation of harmful muscular forces. Changes in input-output properties of any one of these proprioceptors with weight training may result in disinhibition and an increased

expression of muscular forces.[14] For example, the results of Aagaard et al.[24] suggested that Ib afferent feedback (autogenous inhibitory feedback) to the spinal motoneuron pool was down-regulated as an effect of heavy-resistance strength training, perhaps due to modulation via supraspinal pathways. It is important to point out that whether weight training results in a change in receptor morphology and activation, or there is an alteration in descending control signals that directly affect the Renshaw cells is unclear. There is no evidence to date that weight training causes either effect. Current research efforts are directed at methodologies to disentangle the peripheral mechanism.[25]

2.2 Agonist-Antagonist Interaction

Contraction of a muscle acting as an agonist results in limb movement in the desired direction. A muscle acting as an antagonist opposes the motion of the agonist. Alterations in antagonist muscle activity are one of the most misunderstood adaptations associated with strength training. It is generally accepted that training-related increases in muscle strength are associated with a reduction in antagonist co-activation, a view that is entrenched in exercise science.[115,116] The tenet is based on an historically powerful teleological explanation that has only recently been questioned. That is, antagonist muscle activity is associated with a force that opposes the desired direction of motion. Decreases in antagonist co-activation would therefore allow agonist muscle strength to manifest itself unimpeded by contraction of the opposing muscle group.[14] This theory has been reinforced over time by the common clinical observation that increased antagonist co-activation is associated with a maladaptive response following stroke.[117] In contrast, orthopaedic research has demonstrated that a strong, well balanced antagonistic musculature surrounding the joint is also necessary to distribute pressure evenly across the articulating surfaces, and avoid a focal stress point that would lead to wear – a potential factor in degenerative joint disease.[118-120]

2.2.1 Evidence of Antagonist Activity

To date, there is only one study that has definitively demonstrated a reduction in antagonist co-activation in response to resistive training. Carolan and Cafarelli[50] studied the effect of an 8-week strength-training regimen on MVC of the knee extensors and SEMG activity of the vastus lateralis and biceps femoris. There was a 32.8% increase in maximal isometric knee extension strength without a concomitant rise in vastus lateralis SEMG. The SEMG activity of the biceps femoris was normalised to maximal SEMG activity of that same muscle when it was acting as an agonist during maximal isometric knee flexion. There was a decrease from 14.9% to 11.5% of maximal biceps femoris SEMG. The authors also determined the SEMG to force relationship for the biceps femoris, and the decrease corresponded to a decrease from 25% to 20% of flexion MVC. This provides strong support for the notion of increased expression of agonist (vastus lateralis) muscle force due to a reduction in antagonist (biceps femoris) co-contraction. Neither knee extension nor maximal SEMG of the biceps femoris changed during strength training. Thus, it is very difficult to refute these findings on any methodological basis.

These findings are, however, not without question. Gabriel and Kroll[42] examined the effects of a fatigue protocol consisting of 30 maximal isometric elbow flexion strength trials. There were five test sessions at 2-week intervals to assess retention of practice effects. There was a significant increase in maximal isometric elbow flexion strength, averaged across the 30 trials. The 30-trial mean for both biceps and triceps brachii SEMG activity also increased. Increased co-activation has been observed for the elbow extensors using isometric contractions,[18] and for the elbow flexors during an isotonic training regimen.[121] Taken together, whenever subjects train to become stronger or faster, there is a concomitant rise in antagonist co-activity. In each case, the raw (non-normalised) SEMG scores of the antagonist muscle were used for analysis. Contradictory findings related to antagonist co-activation may, therefore, be due to ipsative versus normative scaling of SEMG activity. Expressing SEMG activi-

ty in normalised units can introduce bias in the statistical analysis of the data as opposed to using the raw SEMG scores.[122] This may or may not necessarily be the case in the study by Carolan and Cafarelli.[50]

Isometric contractions are a useful model for studying training-related changes in antagonist co-activation, because the SEMG-force relationship is relatively straightforward.[123] However, resistive training involves stretch-shortening contractions, which affects the generalisability of the studies presented thus far. Several research groups have examined the effects of a training regimen consisting of eccentric contractions on the torque-velocity profile.[30,31,124,125] The studies reported a significant increase in the overall magnitude and shape of the torque-velocity curve and in the amplitude of agonist SEMG activity at each velocity. Two of the studies also recorded antagonist SEMG and found no change in co-activation.[124,125]

Complicating the issue further is the fact that new data are emerging that suggest older (≈70 years) individuals have different antagonist co-activation strategies.[27] Macaluso et al.[126] assessed maximal isometric contractions of the knee extensors and flexors of the dominant leg using a KinCom isokinetic dynamometer. Older females were reported to have lower levels of knee extension torque and smaller contractile muscle volume, as measured by MRI, but greater co-activation of the hamstring muscles than younger females.[126] Häkkinen and colleagues[28] had middle-aged (40 years) and older (70 years) male and female subjects engage in a 6-month heavy-resistance training programme for the knee extensors. All groups exhibited a significant increase in knee extension strength and SEMG activity of the quadriceps, but they differed with respect to co-activation of the hamstrings muscle group. The middle-aged groups (males and females) remained unchanged with respect to antagonist co-activation while the older participants (males and females) exhibited a significant reduction.[28]

An additional finding from Häkkinen et al.[28] was that elderly female subjects had significantly higher hamstring co-activation prior to training than the other groups. After training, the magnitude of hamstring co-activation for the elderly females was then reduced to reach the levels observed in the other subject groups. It may be speculated that the elderly female subjects showed excessive hamstring co-activation prior to training to provide increased stabilisation of the legs, which likely reflects that these specific subjects were particularly uncomfortable with the leg press test procedure. Thus, it is not clear at this time what the CNS will optimise: force production or joint integrity. It is reasonable to speculate that the CNS compromises between these entities, which likely lies closer to ensuring joint integrity (by increasing joint stiffness via elevated antagonist co-activation) in situations of uncertainty about the motor task and/or in unstable movement conditions.

2.2.2 Methodological Considerations

Normalisation is a recurring theme in the explanation of differences between training-related studies of antagonist co-activation. Normalisation is believed to be important to minimise error associated with day-to-day variations in electrode placement, skin impedance and intra- and inter-subject variability in SEMG magnitude.[127-129] Normalisation is supposed to allow for a comparison between studies, and improve the reliability of the SEMG measures. However, several studies have demonstrated that strict methodological controls with respect to electrode placement, skin preparation and subject test position can result in very high intra-class reliability coefficients.[130-133]

Another reason for normalisation is to account for the biomechanics of dynamic contractions. The biomechanics of a dynamic contraction are more complex than an isometric (static) condition. Force exerted by the muscle changes throughout the range of motion due to the force-velocity and length-velocity relationships. The resulting torque also varies as a function of the changing muscle moment arm. Moreover, there is a displacement of muscle fibres underneath the surface electrodes. All these factors can affect the SEMG-to-force relationship. Kellis and Baltzopoulis[134] compared two methods of normalisation for analysing SEMG during dy-

namic contractions. The first method involved normalising SEMG activity with respect to maximal isometric contraction of the muscle when it was acting as an agonist. The second method was supposed to be more parsimonious with respect to muscle action, muscle length and angular velocity. While the agonist muscle shortens, the antagonist muscle is lengthening, which is an eccentric action. This is especially important, as eccentric and concentric contractions exhibit different motor unit firing patterns and SEMG magnitude.[135,136] The authors therefore normalised antagonist SEMG relative to values recorded when the muscle was acting as an agonist during eccentric actions, at the velocities used for testing. The two methods of normalisation resulted in very different conclusions about antagonist muscle function.

A more sophisticated method of studying coactivation patterns between antagonistic muscle groups uses a coefficient of cross-correlation between the agonist and antagonist SEMG signals.[137,138] The cross-correlation coefficient ranges from 0 to 1. A high cross-correlation coefficient indicates that the two muscles have similar firing rates and motor unit action potential shapes. However, findings based on the cross-correlation coefficient are susceptible to error associated with volume-conducted muscle activity, termed cross-talk. The onus is on the investigator to demonstrate through methodological controls that muscles have been sufficiently isolated. Even most careful studies admit there is some degree of cross-talk. There is disagreement among modelling studies, however, as to the level of contamination.[138-140]

3. Recommendations

In a clinical setting, the therapist is charged with restoring function that has been lost or impaired through illness or injury. Rehabilitation can require restoration of not only strength, but also range of motion, balance and coordination. The functional needs of the patient should determine the exercises and activities prescribed, including the mode of strength training used. Strength declines rapidly when muscles are immobilised or inactive.[141] However, limited joint range of motion is often necessary immediately following acute injuries or surgical procedures, when pain, swelling or crepitus is present, or if insufficient healing of an injury has occurred.

3.1 Motor Learning

If neural adaptations to resistive exercise have a motor learning component, then findings from this body of literature should be taken into consideration when prescribing training programmes. This includes timing of inter-session intervals. There may be a trade-off between the time allotted for recuperative rest and the need to practice the task for skill learning. For example, repeated maximal eccentric contractions often involve significant amounts of muscle damage and may require as much as 72 hours before the next session.[142,143] Autosuggested contractions (i.e. imagined) may then be employed during the rest interval to maintain skills specific to the resistive exercise. Zijdewind and colleagues[144] recently demonstrated that imagery motor training increased plantar flexion torque during limb immobilisation. Remarkably, the strength gains were greater than a low-intensity training group and a control group.

3.2 Static Activity

Isometric exercise produces muscle tension without joint movement. It can provide a strength base for dynamic exercise. Isometric, or static, strength is essential for the performance of activities of daily living and many sports skills, when a stable base is required for effective movement of the limbs. In a rehabilitation setting, this type of exercise is useful when joint motion is uncomfortable or contraindicated, when joints are immobilised, or when weakness exists at a specific point in the joint range of motion.[145]

There is disagreement in the literature as to whether isometric training performed at one joint angle will affect force production at angles other than the one used for training.[146,147] Several studies have found that strength gains are limited to ±10° around the angle of the isometric training posi-

tion.[148] In contrast, Bandy and Hanten[149] reported at least a 30° transfer of strength regardless of the length of the muscle, and at least a 75° transfer of strength after exercise in the lengthened position (90°), in subjects who trained the knee extensors isometrically at 30°, 60° or 90° of knee flexion for 8 weeks. This suggests that if pain, swelling or surgical constraints limit initial rehabilitation to isometric exercise, training with the muscles in a lengthened position would be more likely to increase strength throughout the joint range of motion.

It should also be noted that, at the knee joint, isometric exercise at certain joint angles has been shown to produce a sizeable amount of strain in the ligamentous structures. Isolated loading of the quadriceps muscle group results in significant anterior displacement of the tibia.[150] Even when the quadriceps and hamstring muscle groups are activated simultaneously, and the hamstrings act synergistically with the anterior cruciate ligament (ACL), strain in the ACL is significantly higher than during passive normal motion from full extension to 30º of flexion.[151,152] If the isometric exercise is being used in a programme to increase strength in muscles surrounding a joint after ligament injury or repair, care must be taken when choosing the exercise positions.

The manner in which the isometric contraction is performed also has implications for training. A gradual build-up of force is typically recommended to minimise pain and discomfort.[153] However, if the individual can perform brisk, rapid contractions wherein force increases very quickly, the adaptations are quite different and specific. Training using rapid maximal isometric contractions results in an increase in the rate of force development. That is, the force development phase is shorter in duration.[26,154,155] This has important implications for exercise prescription for the elderly. The rate of force development by the muscles may be the single most important factor in allowing older adults to respond adequately to a balance perturbation. The response time of older individuals is sufficient, but the build-up of force within the muscle takes too long to allow them to regain balance.[156]

3.3 Eccentric Activity

Eccentric (lengthening) contractions are characterised by greater force at a given angular velocity than concentric contractions, and a higher incidence of delayed onset muscle soreness after exercise. In the early stages of rehabilitation, if joint motion is possible but the muscles surrounding the joint are weak, eccentric exercise may be easier than concentric exercise: for example, controlled lowering of a limb with gravity rather than a shortening contraction against gravity. Fewer motor units must fire to control the same load eccentrically as concentrically, so less effort is required. Eccentric contractions have been shown to have a greater effect on type II fibres than concentric contractions.[30] Since these are the muscle fibres that decrease in area subsequent to either detraining or limb immobilisation,[141] submaximal eccentric contractions should be included as part of therapeutic resistive exercise. However, because some motor units may not be activated during submaximal eccentric contractions, it is important to include maximal eccentric exercise at certain points in the programme, allowing sufficient time for recuperation, i.e. 4–7 days between maximal exercise bouts.

In an experiment by Holder-Powell and Rutherford[157] to elicit maximal voluntary eccentric muscle force using an isokinetic dynamometer, a 50° range of motion at the knee (45–95° flexion) produced higher values than an 80° range (15–95°). As well as producing higher eccentric torque values, the reduced testing range seemed to decrease patellofemoral discomfort and apprehension in patients with a previous knee injury. In healthy subjects, the authors suggest that this range may reduce inhibitory neural activity from receptors in muscle, tendon or joints, though this idea remains to be tested. This has applications for both eccentric training programmes and also evaluation of patients with patellofemoral conditions.

Resistive training using eccentric actions of the muscle can alter the entire torque-velocity relationship, in able-bodied individuals. There is an increase in the magnitude of torque at each velocity, but the eccentric portion of the curve also changes shape. At

the same time, the SEMG activity observed during eccentric muscle action increases to a level that matches the isometric portion of the curve.[31,32,124,125]

3.4 Dynamic Activity

As an extension of his studies on reciprocal inhibition, Sherrington[158] used an electrical stimulus to elicit a crossed extensor reflex at regular intervals in the spinal dog. During one of the intervals, a flexion reflex was induced and the following crossed extensor reflex returned with increased intensity. Sherrington[158] called the phenomenon a 'rebound contraction' and suggested that it was produced by the physiological law of 'successive spinal induction'. The same response can be seen between ipsilateral antagonist muscle groups. Kabat[159] applied Sherrington's[158] principles to develop exercise routines to aid in the removal of motor deficits. The proprioceptive neuromuscular facilitation (PNF) system advanced by Kabat[159] utilises the principle of successive induction through the reversal of antagonists technique. This technique requires a contraction of the antagonist immediately before a contraction of the intended agonist (i.e. the muscle that is targeted for facilitation).

During an isometric reversal of antagonists, the mechanism for a facilitated response is theorised to involve the Golgi tendon organs. The Golgi tendon organs send afferent fibres through the posterior spinal root to fire upon anterior horn cells in the spinal cord. Kabat[159] hypothesised that this afferent input could be used to facilitate the recruitment of subliminal fringe anterior horn cells. When activated, the Golgi tendon organs are responsible for autogenic inhibition of the contracting muscle and facilitation of the antagonist.[160] Thus, before the central command arrives at the α-motoneuron pool to actively recruit the intended agonist, the membrane threshold has been lowered by proprioceptive input from the contracting antagonist. Although the basic neurophysiological mechanism responsible for a facilitated muscular contraction has been shown to be operative,[161,162] evidence of potentiated muscular strength is equivocal.[18,163]

Maximal concentric force appears to be facilitated by a brief contraction of the contralateral antagonist muscle group.[96] The effect on force produced of contracting the ipsilateral antagonist is not so clear. Bohannon[164] found no difference in peak isokinetic knee extensor torque at 60° and 120°/sec between groups of healthy subjects performing: (i) maximal concentric knee extension followed by passive knee flexion; and (ii) maximal concentric knee extension followed by maximal concentric flexion. However, in a subsequent study of patients with hemiparesis secondary to an intracranial lesion (cerebrovascular accident, tumour or closed head injury), peak knee extension torque was significantly greater during repeated knee extension-flexion reversals at 60°/sec than during separated knee extension-flexion movements.[165] The apparent disagreement between study results was explained by the nature of the subjects. It was suggested that patients with hemiparesis have knee extensor weakness, an inability to generate dynamic knee flexion values in excess of those produced by the weight of the leg, and, possibly, altered motor control. In making the clinical recommendation that, for patients with similar injuries, knee extension-flexion exercise be performed without a pause between reversals, Bohannon et al.[165] also point out that there is a great deal of variability in this patient group and, ideally, clinicians should test each patient to determine what treatment is best.

Grabiner[166] examined the effect of graded conditioning contractions of the knee flexors on maximum force produced by the knee extensors at a higher velocity (250°/sec) than those used in previously reported studies. Maximum force did not change, but the rate of force development increased significantly with the intensity of the conditioning contraction. An explanation for these findings was that the conditioning contractions increased the number of large-threshold motor units activated or caused an increase in the maximum discharge rate of the involved motor units. Gabriel et al.[163] also studied the effects of a conditioning contraction of the antagonist and observed the same effects for the maximum rate of force development. However, the

absence of any alterations in SEMG activity pointed towards a mechanism that involved the mechanical properties of muscle rather than motor control. In either case, the antagonist-to-agonist contraction pattern enhanced muscle dynamics. The antagonist-to-agonist contraction pattern may prove ideal for older adults, to ameliorate the natural decline in contractile performance as it pertains to balance maintenance.

Research clearly shows that bilateral transfer can occur as a result of strength training. The increase in maximal force produced by contralateral resistance exercise training is generally greater following training using dynamic rather than static, isometric contractions.[106,107] Muscle lengthening exercise is particularly effective, resulting in strength gains in the contralateral limb as high as 77%,[31] although these eccentric contractions may also produce muscle soreness.[109,110] In this case, submaximal contractions should be emphasised.

3.5 Co-Activation

Significant hamstring co-activity has been demonstrated during closed kinetic chain exercises for the quadriceps. For this reason, closed kinetic chain exercises are thought to be beneficial for enhancing joint stability.[167] However, it is important to keep in mind that training-related increases in muscular strength may or may not result in greater co-activation.[120] Monitoring muscle strength is necessary[168] but is not sufficient for assessing muscle balance. If co-activation is beneficial, then it should also be monitored using SEMG activity. A great deal of work is necessary to determine what level of co-activation is beneficial. Musculoskeletal modelling and some type of SEMG-to-force calibration are necessary to understand the functional effects of co-activation.[134,169-171] It is also important to take into consideration sex- and age-related factors in antagonist muscle function when prescribing a resistive exercise programme.

4. Conclusions

Muscular adaptations that contribute to force enhancement are well documented. We now have evidence that neural adaptations also contribute to increases in muscular strength. Neural mechanisms discussed in this review include alterations in agonist-antagonist coactivation, increases in motor unit firing rates, and changes in descending drive to the motoneurons. However, there are numerous questions that remain to be resolved. How does motor unit synchronisation or doublet firing contribute to muscular force enhancement? What spinal circuitry sites (i.e. Renshaw cell inhibition or Ib inhibition) change in response to exercise training? Might there be changes in peripheral sites, such as receptor sensitivity, that contribute to enhanced muscular force? Might there be alternative techniques (such as imagery training) that contribute to muscular force increases? Future research needs to be aimed at resolving these issues and how they translate to practical application.

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Correspondence and offprints: Dr *David A. Gabriel*, Department of Physical Education and Kinesiology, Brock University, 500 Glenridge Avenue, St Catharines, ON, L2S 3A1, Canada.
E-mail: dgabriel@brocku.ca