

# Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses

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**Aagaard, Per, Erik B. Simonsen, Jesper L. Andersen, Peter Magnusson, and Poul Dyhre-Poulsen.** Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. *J Appl Physiol* 92: 2309–2318, 2002. First published March 8, 2002; 10.1152/jappphysiol.01185.2001.—Combined V-wave and Hoffmann (H) reflex measurements were performed during maximal muscle contraction to examine the neural adaptation mechanisms induced by resistance training. The H-reflex can be used to assess the excitability of spinal  $\alpha$ -motoneurons, while also reflecting transmission efficiency (i.e., presynaptic inhibition) in Ia afferent synapses. Furthermore, the V-wave reflects the overall magnitude of efferent motor output from the  $\alpha$ -motoneuron pool because of activation from descending central pathways. Fourteen male subjects participated in 14 wk of resistance training that involved heavy weight-lifting exercises for the muscles of the leg. Evoked V-wave, H-reflex, and maximal M-wave ( $M_{\max}$ ) responses were recorded before and after training in the soleus muscle during maximal isometric ramp contractions. Maximal isometric, concentric, and eccentric muscle strength was measured by use of isokinetic dynamometry. V-wave amplitude increased  $\sim 50\%$  with training ( $P < 0.01$ ) from  $3.19 \pm 0.43$  to  $4.86 \pm 0.43$  mV, or from  $0.308 \pm 0.048$  to  $0.478 \pm 0.034$  when expressed relative to  $M_{\max}$  ( $\pm$  SE). H-reflex amplitude increased  $\sim 20\%$  ( $P < 0.05$ ) from  $5.37 \pm 0.41$  to  $6.24 \pm 0.49$  mV, or from  $0.514 \pm 0.032$  to  $0.609 \pm 0.025$  when normalized to  $M_{\max}$ . In contrast, resting H-reflex amplitude remained unchanged with training ( $0.503 \pm 0.059$  vs.  $0.499 \pm 0.063$ ). Likewise, no change occurred in  $M_{\max}$  ( $10.78 \pm 0.86$  vs.  $10.21 \pm 0.66$  mV). Maximal muscle strength increased 23–30% ( $P < 0.05$ ). In conclusion, increases in evoked V-wave and H-reflex responses were observed during maximal muscle contraction after resistance training. Collectively, the present data suggest that the increase in motoneuronal output induced by resistance training may comprise both supraspinal and spinal adaptation mechanisms (i.e., increased central motor drive, elevated motoneuron excitability, reduced presynaptic inhibition).

M-wave;  $\alpha$ -motoneurons; skeletal muscle

IT IS WELL ESTABLISHED that physical activity that incorporates high muscle tensions, i.e., heavy-resistance

strength training, can lead to an increase in maximal contractile muscle force. However, the specific mechanisms responsible for this adaptation are not fully known. For instance, the increase in maximal contraction force may not solely be explained by increases in muscle cross-sectional area or volume. Rather, an increased “neural drive” to the muscle fibers contributes to the training-induced increase in maximal contractile force, even in the absence of increases in muscle size (27). Thus not only muscle size and muscle phenotype but also neural innervation are important determinants of maximal contractile muscle strength in vivo.

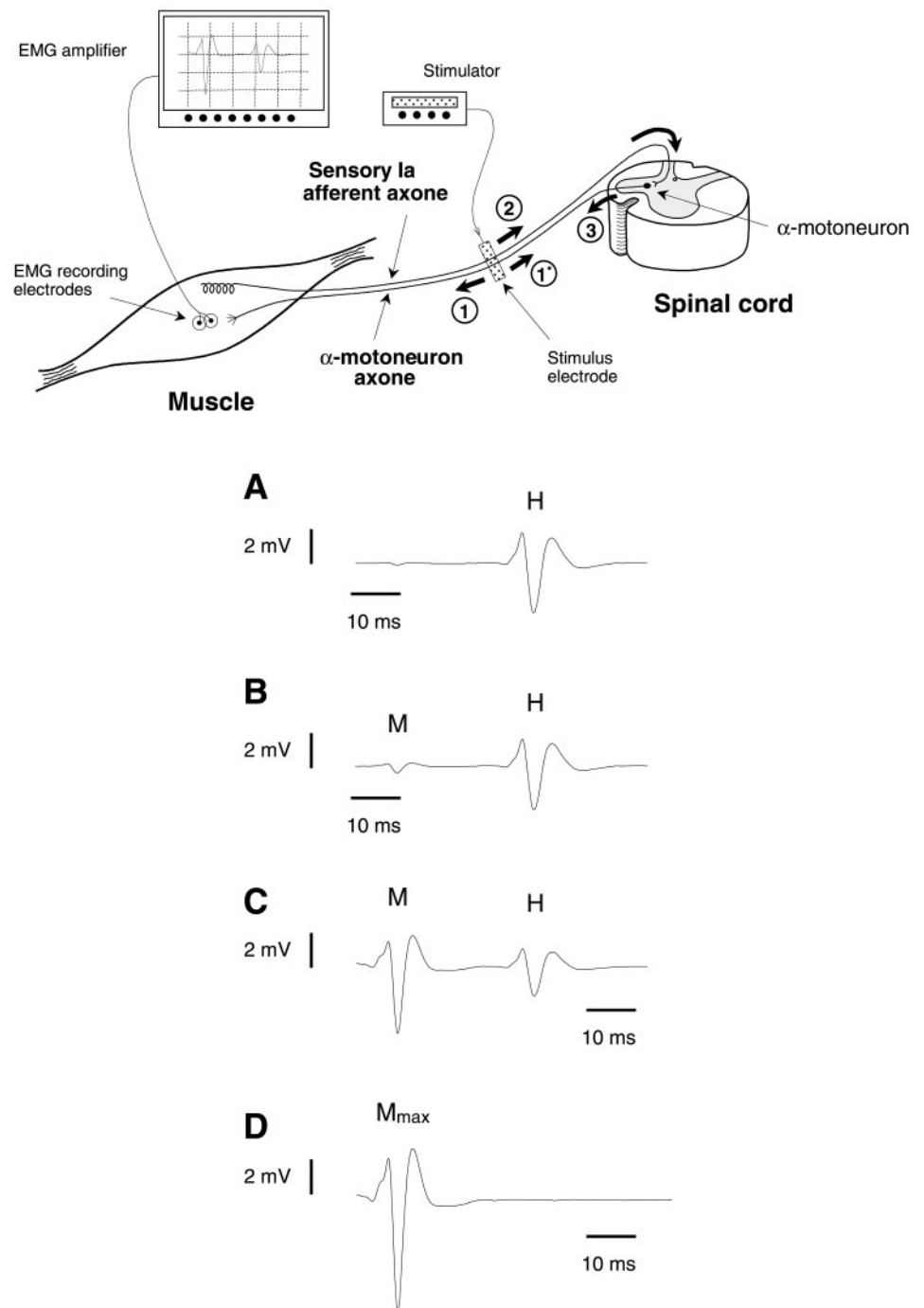
Numerous reports exist of the morphological changes in human skeletal muscle induced by resistance training. Such changes include increases in anatomical muscle cross-sectional area (2, 29) and physiological muscle fiber area (3, 14, 44), increased percentage 2A fibers with a corresponding decrease in 2X fibers (3, 14), and steeper muscle fiber pennation angles (2). Likewise, the neural adaptation induced by resistance training has been addressed with the use of integrated electromyography (EMG) as an indicator for a change in efferent neural drive. Several investigators have reported increases in integrated EMG after resistance training (1, 27, 29), although not consistently demonstrated in all studies (6, 43). Some of this disparity may be explained by the inherent methodological constraints associated with the recording of surface muscle EMG during maximal voluntary contraction (MVC).

Although the effect of resistance training on muscle morphology has received considerable examination, less is known about the specific neural mechanisms responsible for the training-induced increase in maximal muscle strength. Furthermore, only a few studies have studied evoked spinal motoneuron responses to examine more closely the adaptive change in neural function induced by resistance training. The H (Hoffmann) reflex may be useful to assess motoneuron excitability in vivo (16, 39), while also reflecting presyn-

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Fig. 1. Hoffmann (H) reflex. When the tibial nerve is stimulated percutaneously by a short-lasting low-intensity electrical current, action potentials are elicited selectively in the axons of the sensory Ia afferents because of their large axon diameter (*response 2; top*). The evoked action potentials propagate to the spinal cord, where they give rise to excitatory postsynaptic potentials, in turn eliciting action potentials, which travel in the  $\alpha$ -motoneuron axons toward the muscle (*response 3*). Subsequently, with a latency of 30–40 ms, the volley of efferent action potentials is recorded in the muscle as an H-reflex (A). Using gradually higher stimulus intensity will cause action potentials to occur also in the thinner axons of the  $\alpha$ -motoneurons (*response 1; top*), traveling to the muscle as a direct M-response (M-wave, B). At the same time, action potentials propagate antidromically in the  $\alpha$ -motoneuron axons toward the spinal cord (*response 1\**) to collide with action potentials of the evoked reflex response (*response 3*), thereby resulting in a partial cancellation of the reflex response. In consequence, the M-wave is seen to increase with increase in stimulus intensity while at the same time the H-reflex amplitude gradually decreases because of the increase in antidromic collision (C and D). At a given stimulus intensity, an elevated H-reflex response will indicate the excitability of  $\alpha$ -motoneurons to have increased (and/or presynaptic inhibition to have decreased). At very high (i.e., supramaximal) stimulus intensity, orthodromic and antidromic action potentials will occur in all motoneuron axons, the former giving rise to a maximal M-wave ( $M_{max}$ ), whereas the latter cause a complete cancellation of the H reflex volley due to antidromic collision occurring in all axon fibers (D). More details are given in MATERIAL AND METHODS. EMG, electromyogram.



aptic inhibition of Ia afferent synapses (18, 30) (Fig. 1). We have previously examined the modulation in H-reflex excitability during walking, running, and jumping to address the integration of afferent sensory inflow and efferent motor output during natural movement in humans (11, 12, 40, 41). The so-called V-wave, which is an electrophysiological variant of the H-reflex, can be recorded during maximal voluntary motor efforts (17, 24, 46). The evoked V-wave response may be used to reflect the level of efferent neural drive from spinal  $\alpha$ -motoneurons during maximal muscle contraction (46) (Fig. 2).

In the present study, evoked V-wave and H-reflex responses were obtained in the soleus muscle to address the neural adaptation induced by intense heavy-resistance strength training. Previous studies on a cross-sectional basis have demonstrated elevated H-reflex excitability in endurance athletes compared with power and sprint athletes (8, 22, 34). Likewise, increased V-wave responses were observed in sprint athletes and weight lifters compared with sedentary subjects (38, 47). In contrast, lower H-reflex amplitudes and reduced H-reflex gains were observed in ballet dancers compared with physical education students

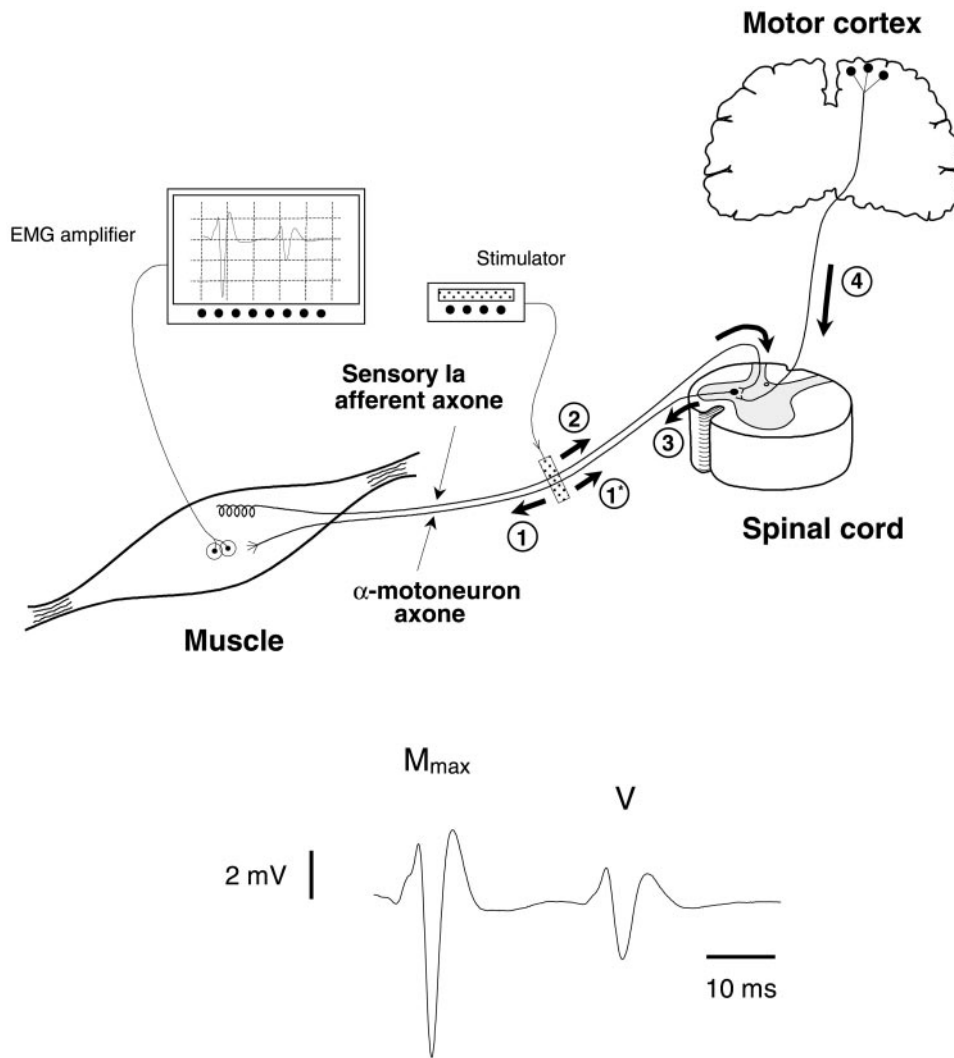


Fig. 2. Evoked V-wave potentials. When supramaximal nerve stimulation is applied during maximal voluntary muscle contraction, the efferent motor impulses generated because of activation of motoneurons via central descending pathways (nerve impulses 4 → 3) will collide with the antidromic action potentials (*response 1\**), thus allowing a part of the evoked reflex response to pass to the muscle (2 → 3 → V wave; typical recording shown at *bottom*). Although actually constituted by a volley of H-reflex impulses, this evoked potential is denoted a V-wave to indicate its presence during volitional contraction but absence during rest (17, 46). With increase in efferent motoneuron activity (i.e., increased recruitment and/or elevated firing frequency), the volley of antidromic action potentials will be increasingly cancelled because of increased collision with volitional motor impulses in the  $\alpha$ -motoneuron axons. Consequently, this results in a rise in the peak-to-peak amplitude of the evoked V wave. Thus training-induced alterations in efferent motoneuronal activity will be reflected by a corresponding change in  $V/M_{\max}$ . More details are given in MATERIALS AND METHODS.

(28, 31). However, such cross-sectional comparisons are difficult to interpret because both V-wave amplitude and H-reflex excitability may also be influenced by anatomical or genetic differences and not by the level of physical activity alone. Only a single longitudinal study has examined the neural effects of resistance training on the basis of the recording of evoked spinal reflex responses (37). However, whereas V-wave amplitude was found to increase in response to 9–21 wk of resistance training, no measurements were performed on the corresponding change in H-reflex excitability or maximal contractile strength (37).

The purpose of the present study, therefore, was to employ combined longitudinal measurements of evoked V-wave and H-reflex responses to examine the neural adaptation induced by intensive heavy-resistance strength training.

#### MATERIALS AND METHODS

**Subjects.** Fourteen male subjects volunteered to participate in the study (body mass  $70.3 \pm 3.6$  kg, height  $178.6 \pm 5.0$  cm, age  $25.3 \pm 4.7$  yr, means  $\pm$  SD). All subjects gave their informed consent to the procedures of the study. None of the

subjects had previously participated in systematic resistance training. The conditions of the study were approved by the local ethics committee.

**Electromyography.** After careful preparation of the skin (shaving, abrasion, and cleaning with alcohol), pairs of surface electrodes (Medicotest Q-10-A, 2-cm interelectrode distance) were placed at the soleus ~13 cm above the calcaneus and below the muscle fibers of the gastrocnemius, at the tibialis anterior ~10 cm below the caput fibulae, and at the gastrocnemius medial and lateral heads ~7 cm below the caput fibulae. The actual electrode positions were carefully measured in each subject to control that pre- and posttraining recording sites were identical. The EMG electrodes were connected directly to small custom-built preamplifiers (input impedance 80 M $\Omega$ ) taped to the skin (11, 40, 41). The EMG signals were led through shielded wires to custom-built differential amplifiers with a frequency response of 10–10,000 Hz and common mode rejection ratio >100 dB. The preamplifiers lowered the impedance, which effectively prevented movement artifacts.

**H-reflex recordings.** The H-reflex is elicited by electrical stimulation of the peripheral nerve, i.e., tibial nerve in the popliteal fossa for the soleus muscle, thereby bypassing the influence of muscle spindle sensitivity and  $\gamma$ -activation of intrafusal fibers. As a result, the H-reflex response can be



used to assess spinal motoneuron excitability and transmission efficacy in Ia afferent synapses (Fig. 1). The soleus muscle is especially convenient for measurement of the H-reflex because the large-diameter Ia afferents and small-diameter  $\alpha$ -motoneuron axons differ considerably in size (39). It is possible, therefore, to electrically stimulate the thickest axonal fibers (i.e., the Ia afferents) selectively by using low-stimulus intensity. Additional increase in stimulus intensity causes action potentials to be elicited also in the slightly thinner axons of the  $\alpha$ -motoneurons.

As previously described in detail (11, 12, 40, 41), the soleus H-reflex was elicited by percutaneous stimulation of the tibial nerve by use of an AgCl cathode (Medicotest Q-10-A) in the popliteal fossa and a 40-mm-diameter anode placed over the patella. The optimum site of stimulation was first located by a hand-held stimulation probe on the basis of the criterion that the soleus Ia afferents could be stimulated selectively at low stimulus intensities. Subsequently, the stimulation electrode was firmly affixed to this site with rigid straps and taping. The stimulus consisted of a 1-ms square pulse delivered by a custom-built constant-current stimulator. To compare and interpret changes in evoked H-reflex responses with training, it is necessary to ensure that the effective stimulus strength remains invariant between recording sessions, because the size of the H-reflex is heavily modulated by even small changes in stimulus intensity. Therefore, when repeated H-reflex measurements are performed it is an advantage to use a stimulation intensity that produce M-wave responses corresponding to a constant percentage of the maximal M-wave ( $M_{\max}$ ) to ensure that the same number of motor axons are recruited in each trial, which indicates that stimulus intensity to the efferent nerve is also kept constant (12, 40, 41). Thus, during the present H-reflex measurements, effective stimulus strength was controlled by adjusting stimulation intensity so that the peak-to-peak amplitude of the direct M-wave response was  $20 \pm 2.5\%$  of  $M_{\max}$  (11, 12, 40, 41). The M-wave amplitude was measured on-line and displayed (expressed relative to  $M_{\max}$ ) on a personal computer screen after each ramp contraction. This made it possible to continuously adjust stimulation intensity, to ensure that the intended target level ( $20\% M_{\max}$ ) was maintained in all ramp contractions. In each subject,  $M_{\max}$  was determined by gradually increasing stimulus strength to the point of no further visual increase in the peak-to-peak amplitude of the M-wave, with a subsequent doubling in stimulation intensity during the actual measurement of  $M_{\max}$  (40, 41).

Maximal isometric plantar flexor contractions were performed in a modified KinCom dynamometer (Kinetic Communicator, Chattecx, Chattanooga, TN). Subjects were seated in a rigid chair with a hip joint angle of  $90^\circ$ . Ankle joint angle was  $88^\circ$  ( $2^\circ$  dorsiflexion relative to neutral position,  $90^\circ$ ). The knee was  $90^\circ$  flexed and rigidly supported by a solid custom-built steel frame cushioned with a stiff vacuum cast adapted to fit the individual shape of the thigh. At the start of the experiment, maximal voluntary isometric plantar flexor contraction strength (i.e., MVC) was determined with subjects typically performing four to six attempts separated by 60 s of rest. Subsequently, an M-H calibration curve was established during rest by systematically varying stimulation intensity from zero to a level that elicited a direct  $M_{\max}$  by using a total of 80 stimulation points or more (see Ref. 41, their Fig. 5). On the basis of these data points, the maximal H-reflex response ( $H_{\max}$ ) expressed relative to  $M_{\max}$  (i.e.,  $H_{\max}/M_{\max}$ ) was determined to provide a measure of resting H-reflex excitability (22, 31, 33, 34, 49). After the M-H calibration procedure, H-reflex recordings were obtained in the soleus muscle during maximal voluntary isometric con-

traction of the plantar flexors. Specific subject positioning and details of the dynamometer are given below (*Measurement of maximal dynamic muscle strength*). The subjects were asked to gradually increase the contraction level from 0 to 100% MVC in 2-s isometric force ramps of maximal voluntary effort. Each subject performed a total of 10 maximal ramp contractions. In each maximal ramp contraction, an electrical stimulus (1-ms square pulse) was applied to the tibial nerve (popliteal fossa) at the instant the ankle plantar flexor moment exceeded 90% MVC. This was achieved by continuously feeding the moment signal to a custom made computer program written in ASYST that instantaneously triggered the pulse stimulator when reaching a force level of 90% MVC. It should be noted that the subjects were carefully instructed to exert maximal voluntary muscle force (i.e., intending to reach 100% MVC) in each ramp contraction. As described above, stimulus intensity was adjusted to give a direct M-response amplitude of  $\sim 20\% M_{\max}$ . Subsequently, maximal peak-to-peak H-reflex amplitude was determined among the ramp contractions with an M-wave amplitude ranging between 17.5 and 22.5% of  $M_{\max}$ . A 45-s rest period was used between trials to prevent any postcontraction depression of the H-reflex response as otherwise reported when interstimulus intervals shorter than 8–15 s were used in resting conditions (10, 13, 35). It should be recognized, however, that the postcontraction suppression in H-reflex amplitude observed during rest is fully abolished when the H-reflex is elicited onto ongoing muscle contraction (5, 35).

Acceptable intrasession and intersession reliability has previously been demonstrated for the soleus H-reflex when obtained at rest (15).

*V-wave recordings.* Briefly, the origin of the V-wave resides on the following mechanisms: When a supramaximal electrical stimulus is applied to the tibial nerve during ongoing soleus muscle contraction, it will elicit action potentials in all Ia afferents and  $\alpha$ -motor axons, the latter traveling to the muscle, where it is recorded as an  $M_{\max}$ . At the same time, action potentials will propagate antidromically toward the spinal cord in every single  $\alpha$ -motor axon (Fig. 2). During this passage, the antidromic action potentials collide with orthodromic motor action potentials generated as a result of the descending voluntary input to the pool of motoneurons. Collision between these antidromic and orthodromic nerve impulses results in a cancellation of the two signals, and as a result the H-reflex volley is allowed to pass to the muscle, where it is recorded as a so-called V-wave (17, 42, 46; Fig. 2). An increased number of voluntary motor impulses results in an increased incidence of antidromic collision, which allows more motoneuron axons to be cleared for passage of the evoked reflex response in turn manifested by an increase in V-wave amplitude (46) (Fig. 2). Consequently, the peak-to-peak amplitude of the V-wave expressed relative to that of the maximal M-wave ( $V/M_{\max}$ ) reflects the amount and frequency of efferent nerve impulses traveling in  $\alpha$ -motoneuron axons (46). In other words,  $V/M_{\max}$  may be taken to reflect the magnitude of efferent motoneuronal output during voluntary muscle activation.

Similarly to the experimental protocol used for the H-reflex recordings, subjects were asked to perform 2-s isometric force ramps of maximal voluntary effort. During the ramp contraction, a supramaximal stimulus (1-ms square pulse) was applied to the tibial nerve at the instant of reaching (and exceeding) 90% MVC. Ten trials were performed, and the maximal peak-to-peak V-wave amplitude was determined in the trials of M-wave amplitudes  $\geq 95\% M_{\max}$  (typically 2–4) (Fig. 5).

Acceptable test-retest reliability values have previously been reported for the evoked V response (37).

**Measurement of maximal dynamic muscle strength.** Maximal isokinetic plantar flexor moments were obtained during slow (30°/s) concentric and eccentric muscle contraction performed in a modified KinCom dynamometer. Subjects were positioned as previously described (see *H-reflex recordings*, *V-wave recordings*).

Range of ankle joint excursion was  $-15^\circ$  (plantar) to  $20^\circ$  (dorsal) relative to a  $90^\circ$  ankle angle (neutral position). All plantar flexor moments were obtained at  $90^\circ$  ankle angle. After a 10-min warm-up subjects performed four to six sub-maximal plantar flexion ramps in the dynamometer, followed by a number of force ramps at maximal voluntary effort. Visual feedback of the exerted plantar moment was provided on-line to the subjects on a personal computer screen. This allowed subjects to perform repeated trials (separated by 45-s rest periods) until no further improvements in peak moment could be detected (1). Typically five to seven trials were performed to fulfill this goal.

**Training.** Details of the training regime have been reported previously (3). In brief, progressive heavy-resistance strength training was performed for 14 wk in a total of 38 sessions. All training was surveyed and supervised by the authors of the study. The primary training exercise performed was seated calf raises. Other obligatory leg training exercises were hack squats, incline leg press, isolated knee extension, and hamstring curls. Four (*weeks 1–10*) or five (*weeks 11–14*) sets were performed for each exercise. Training loads ranged between 3 and 10 repetition maximum load (RM), except for the first 10 days (4 sessions), in which lower loading was used (10–12 RM). Very heavy loadings (4–6 RM) and increased number of sets (ensuring unchanged total work load) were used in the final 4 wk of the study.

**Statistics.** Data are given as means  $\pm$  SE unless otherwise stated. All pre- to posttraining changes were evaluated by use of the Wilcoxon signed-rank test for paired samples (two-tailed, 0.05 level of significance).

## RESULTS

**Evoked motoneuron potentials.** A short-latency V-wave response was elicited when the tibial nerve was stimulated with supramaximal intensity during maximal isometric contraction (Fig. 2). The latency of the evoked V-wave was consistently identical between successive sweeps (Figs. 3–5). Of the 10 sweeps obtained in each subject pre- and posttraining, peak-to-peak V amplitude was determined for the sweep demonstrating the largest M-wave amplitude (Fig. 4). Typical pre- and posttraining V-wave responses are shown in Fig. 5.

Resistance training resulted in a significant increase in normalized V-wave amplitude ( $V/M_{\max}$ ) rising from a pretraining value of  $0.308 \pm 0.048$  to  $0.478 \pm 0.034$  posttraining, with an average relative increase of 55% ( $P < 0.01$ , Fig. 6). Expressed in absolute units, the peak-to-peak amplitude of the V-wave increased from  $3.19 \pm 0.43$  to  $4.86 \pm 0.43$  mV ( $P < 0.01$ ).

H-reflex excitability measured during the maximal isometric ramp contractions also increased with training, as evidenced by an increase in normalized H-reflex amplitude ( $H/M_{\max}$ ) from  $0.514 \pm 0.032$  to  $0.609 \pm 0.025$  ( $P < 0.05$ , Fig. 6), which corresponded to an average relative increase of 19%. When expressed in absolute units, the peak-to-peak amplitude of the H-

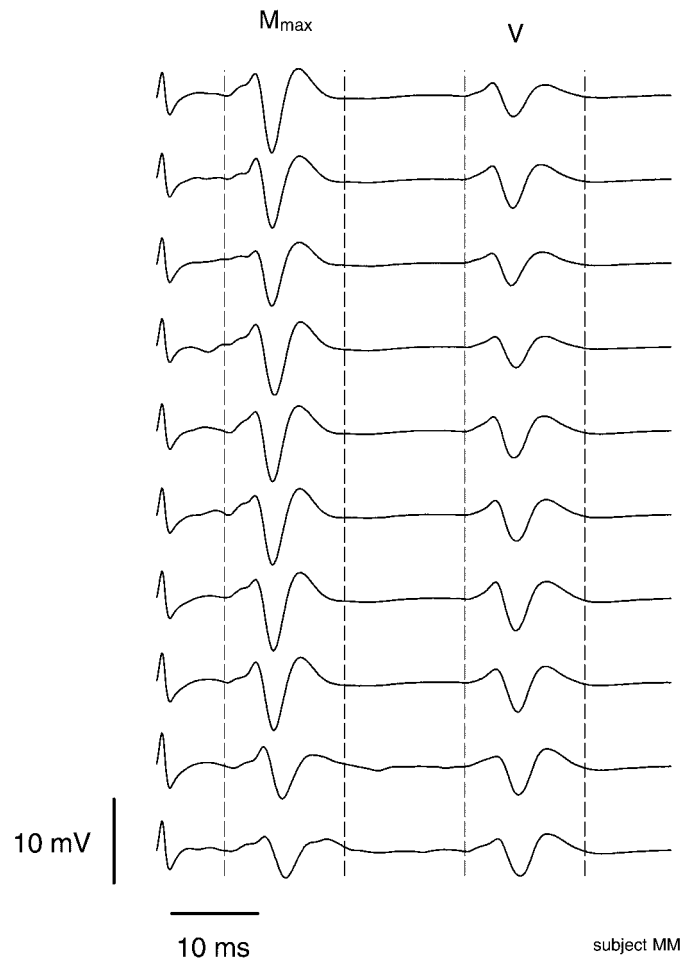


Fig. 3. Evoked V-wave responses recorded in a representative subject before training during 10 repeated isometric muscle contraction ramps separated by  $\sim 45$ -s pause. Dotted vertical lines show the intervals chosen for determination of the peak-to-peak amplitude of the direct  $M_{\max}$  and V-wave, respectively.

reflex increased from  $5.37 \pm 0.41$  to  $6.24 \pm 0.49$  mV ( $P < 0.05$ ). Resting H-reflex amplitude, determined as  $H_{\max}/M_{\max}$ , did not change with training ( $0.503 \pm 0.059$  vs.  $0.499 \pm 0.063$ ). Likewise, at  $H_{\max}$  the peak-to-peak amplitude of the direct M-wave response did not differ before and after training ( $0.103 \pm 0.024$  and  $0.116 \pm 0.021$ , expressed relative to  $M_{\max}$ ).

The maximal M-wave amplitude recorded during supramaximal nerve stimulation ( $M_{\max}$ ) was unchanged with training ( $10.78 \pm 0.86$  and  $10.21 \pm 0.66$  mV).

**Maximal muscle strength.** Maximal concentric muscle strength increased 23% from  $112.2 \pm 16.7$  to  $137.6 \pm 10.8$  Nm ( $P < 0.05$ ). Maximal eccentric muscle strength increased 30% from  $135.5 \pm 18.6$  to  $175.6 \pm 8.2$  Nm ( $P < 0.05$ ). MVC determined in the maximal isometric ramp contractions was found to increase from  $120.4 \pm 14.9$  to  $158.5 \pm 19.8$  Nm ( $P < 0.05$ ), corresponding to an increase of 20%.

## DISCUSSION

In the present study, evoked V-wave and H-reflex responses were recorded during maximal muscle con-

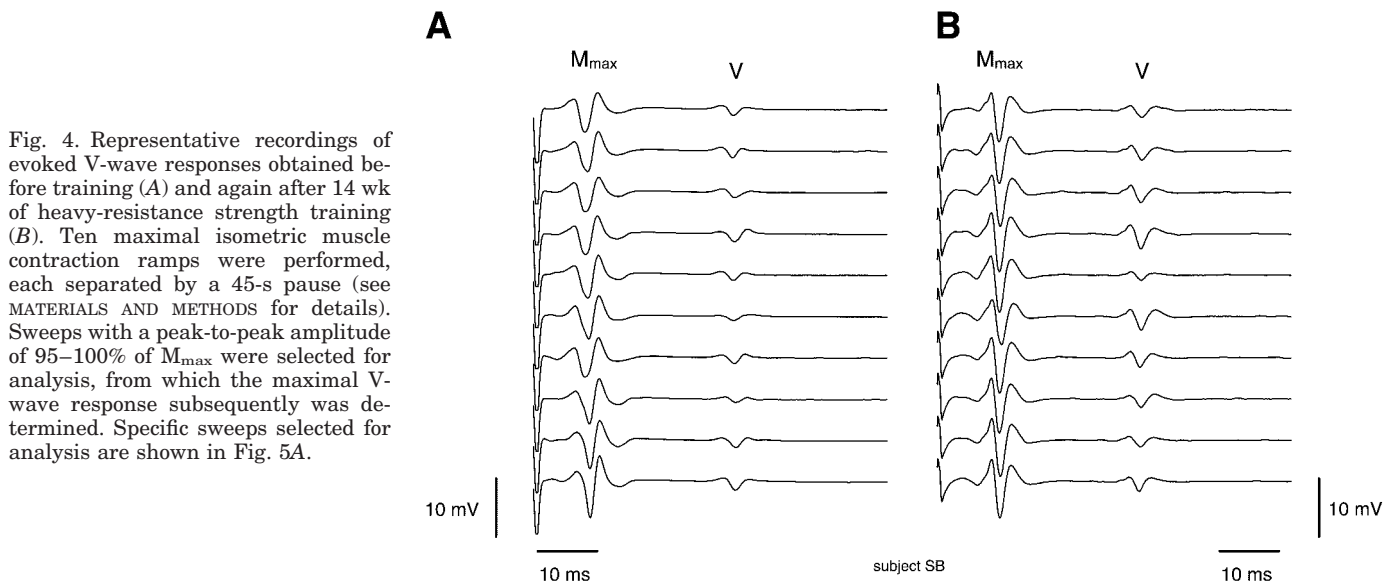


Fig. 4. Representative recordings of evoked V-wave responses obtained before training (A) and again after 14 wk of heavy-resistance strength training (B). Ten maximal isometric muscle contraction ramps were performed, each separated by a 45-s pause (see MATERIALS AND METHODS for details). Sweeps with a peak-to-peak amplitude of 95–100% of  $M_{\max}$  were selected for analysis, from which the maximal V-wave response subsequently was determined. Specific sweeps selected for analysis are shown in Fig. 5A.

traction to examine neural adaptive changes induced by resistance training. Elevated V-wave and H-reflex amplitudes were observed in response to 14 wk of progressive heavy-resistance strength training. Collec-

tively, these findings were taken to reflect adaptive neural alterations at both spinal and supraspinal levels, potentially involving changes in  $\alpha$ -motoneuron excitability and descending motor drive.

*Changes in evoked V-wave responses with resistance training.* During maximal muscle contraction V-waves were evoked by applying supramaximal stimulation to the peripheral nerve. The V-wave consists of a volley of H-reflex impulses that are allowed to reach the muscle because of the removal of antidromic impulses by collision with efferent nerve impulses generated by the voluntary motor effort (17, 46) (Fig. 2). An increase in the level of efferent motor output will give rise to a proportional increase in the probability of antidromic collision (46). In consequence, the V-wave can be used to reflect the magnitude of efferent  $\alpha$ -motoneuron out-

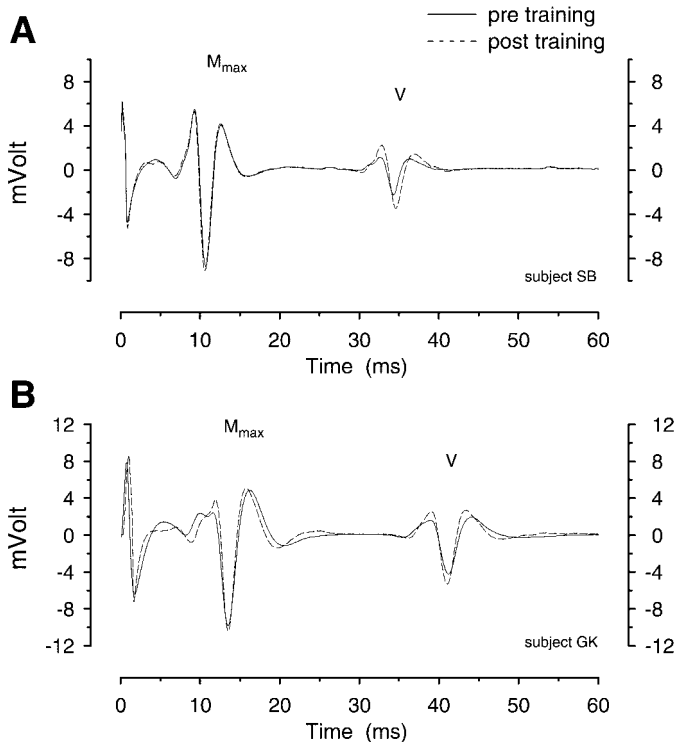


Fig. 5. Evoked V-wave responses selected for analysis before and after 14 wk of heavy-resistance strength training. For *subject A* (same subject as in Fig. 4), pre- and post-V-wave amplitudes were 2.92 and 5.76 mV, respectively.  $M_{\max}$  amplitudes were 14.96 and 14.39 mV, respectively. Normalized V-wave responses ( $V/M_{\max}$ ) increased from 0.195 to 0.400 with training. *Subject B* demonstrated V-wave amplitudes of 6.17 and 7.29 mV pre- and posttraining, respectively. Corresponding  $M_{\max}$  amplitudes were 14.72 and 14.08 mV before and after the period of training. Consequently  $V/M_{\max}$  increased from 0.418 to 0.519 with training.

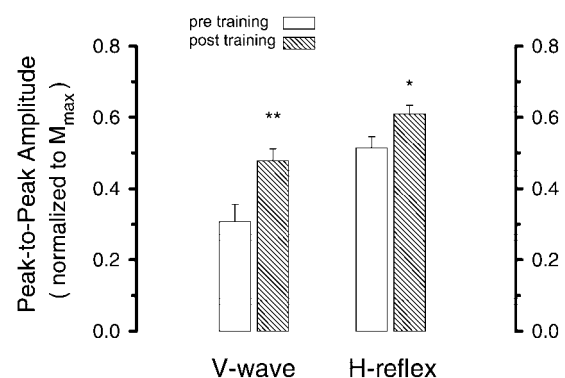


Fig. 6. Group mean peak-to-peak V-wave and H-reflex amplitude expressed relative to  $M_{\max}$  measured during maximal isometric muscle contraction before and after 14 wk of heavy-resistance strength training (open and hatched bars, respectively). Error bars, SE. Pretraining < posttraining: \*\* $P < 0.01$ , \* $P < 0.05$ . The increase in V-wave response observed with training reflects an enhanced neuronal output of spinal motoneurons during maximal muscle contraction after the period of heavy-resistance strength training. The training-induced increase in H-reflex amplitude further suggests that the increase in neuronal output was in part caused by a rise in motoneuron excitability.



put during voluntary muscle activation (cf. Fig. 2). It should be noted that the supramaximal level of nerve stimulation used during recording of the V-wave causes massive excitation of all Ia afferent axons in the peripheral nerve. As a result, the evoked V-wave response will recruit both large and small motoneurons, whereas the H-reflex primarily rely on the pool of smaller motoneurons (see *Changes in H-reflex excitability with resistance training*).

The present study demonstrated a marked increase in V-wave amplitude in response to the regime of heavy-resistance strength training, indicating an enhanced neural drive in descending corticospinal pathways with a corresponding increase in motoneuron excitability, although a decrease in presynaptic inhibition could have contributed as well.  $M_{\max}$  amplitude was found to remain unchanged after training, in accordance with previous reports (36, 37, 48, 49). Consequently, identical changes in absolute and normalized ( $V/M_{\max}$ ) V-wave amplitude were observed after the period of training. Previous findings exist of elevated V-wave amplitudes in the hand and lower limb muscles of sprinters and weight lifters compared with untrained control subjects (26, 38, 47). By use of a longitudinal study design, a 49% increase in V-wave amplitude (recorded as  $V/M_{\max}$ ) was reported in response to 9–21 wk of resistance training (37). The present data appear to verify these earlier findings by demonstrating a 55% increase in V-wave amplitude (Fig. 6) after 14 wk of heavy-resistance strength training. Furthermore, an increase in H-reflex amplitude also was observed after the period of training (Fig. 6). Collectively, the above alterations in evoked motoneuron potentials indicated an enhanced neural drive in descending corticospinal pathways, elevated motoneuron excitability, and/or alterations in presynaptic inhibition to have occurred with training (discussed in detail below). These neural changes were accompanied by significant increases in maximal contractile muscle strength.

The V-wave can be used to indicate the magnitude of antidromic clearing caused by voluntary motor impulses, thereby reflecting the traffic of efferent impulses (i.e., their number and frequency) in  $\alpha$ -motoneuron axons during voluntary muscle activation. Thus the present increase in V-wave amplitude observed with resistance training was likely caused by an increase in motoneuron firing frequency and/or increased motoneuron recruitment, because both result in a direct proportional increase in the probability of antidromic collision. Upton and co-workers (46) formulated this into a mathematical expression, which may be useful to further examine these two factors and their potential contribution to the changes seen with training. Rearranging their original equation, the magnitude of the V-amplitude is given by

$$V/M_{\max} = (H_E \cdot H_V \cdot f \cdot t) \quad (1)$$

with  $H_E$  representing the proportion of motoneurons excited by the Ia afferents and discharging in the ensuing volley of H-reflex impulses (assuming that no motoneurons are involved in "late" H reflex responses,

i.e., after antidromic impulses have died away) and  $H_V$  representing the proportion of  $H_E$  motoneurons participating in voluntary contraction,  $f$  denoting the discharge frequency of  $H_V$  motoneurons during voluntary activation (discharge impulses per second), and  $t$  denoting the time for impulses to travel between the spinal cord and the stimulating electrode, and with the recurrent discharge due to antidromic invasion of the motoneuron assumed to be zero (i.e., assuming no F waves to exist). In Eq. 1, the product  $f \cdot t$  is set to 1.0 for firing frequencies exceeding  $1/t$  to denote that further increase in  $f$  does not result in a corresponding increase in  $V$  (i.e.,  $V/M_{\max}$  can never exceed 1.0). The time available for impulse conduction,  $t$ , can be determined from the latency of the H-reflex by adding the time for synaptic transmission ( $\sim 1$  ms) and approximating conduction velocity to be similar in the motor axons and Ia afferent fibers. This yields

$$L_H = 2t + L_M + 1 \text{ ms} \quad (2)$$

where  $L_H$  and  $L_M$  denote the latency time for the H-reflex and direct M-response, respectively. Using average values for  $L_H$  and  $L_M$  of 32–35 ms and 5 ms, respectively, yields a conduction time  $t$  of 13–15 ms. Because of the level of supramaximal stimulus intensity a value of 1 was assumed for  $H_E$  (46). By using Eq. 1 above, values of  $H_V$  and  $f$  can be derived that meet the pre- and posttraining values presently observed for  $V/M_{\max}$ .

As calculated by Eq. 1, a pretraining firing rate  $f$  of 24–28 Hz and value for  $H_V$  of 0.85–0.90 (leaving room for additional motoneuron recruitment with training) would yield the value of 0.31 observed for  $V/M_{\max}$  before training. Alternatively, if full motoneuron recruitment before training is assumed, a firing rate  $f$  of 19–22 Hz and  $H_V$  of 1.0 would also result in a  $V/M_{\max}$  value of 0.31. Posttraining, the observed  $V/M_{\max}$  value of 0.49 is found to be compatible with a firing rate  $f$  of 33–37 Hz, if full motoneuron recruitment is assumed ( $H_V = 1.0$ ). Consequently, it is likely that the increase in V-wave amplitude observed with training reflects an increased motoneuron firing frequency. In support of this notion, maximal motoneuron firing frequency has previously been found to increase in response to resistance training (20, 32, 48).

Previous reports suggest that it is difficult to elicit training-induced changes in the V-wave when recorded in the muscles of the hand, i.e., hypothenar (26, 36). This finding suggests that the range of neural adaptation may differ between skeletal muscles designed for highly coordinated grasping tasks and muscles responsible for propulsive force generation, respectively. As another methodological concern, the V-wave could be confounded by recurrent F-wave responses. In resting conditions, F-waves may occasionally occur during supramaximal nerve stimulation, presumably because of a small recurrent  $\alpha$ -motoneuron discharge produced by the volley of incoming antidromic action potentials (23, 25). However, both the occurrence and latency of the F-wave varies considerably (5, 19, 25), whereas the V-wave response is highly consistent with a latency

identical to that of the H-reflex (17, 42, 46) (cf. Figs. 3–5). Moreover, in the human soleus muscle the amplitude of the V-wave is sizably greater than that of the F-wave, typically differing by a factor 10 or more (17, 46). It is not likely, therefore, that the change in V-wave amplitude observed in the present study was caused by a varying involvement of the F-wave.

*Changes in H-reflex excitability with resistance training.* For the first time, H-reflex measurements were used to evaluate the neural adaptation induced by resistance training. An elevated H-reflex excitability was observed during maximal voluntary muscle contraction in response to 14 wk of heavy-resistance strength training (Fig. 6), suggesting that  $\alpha$ -motoneuron excitability had increased. The training-induced rise in H-reflex excitability may have been caused by an increase in descending motor drive from higher centers (cf. *Changes in evoked V-wave responses with resistance training*), although reduced presynaptic inhibition of Ia afferents cannot be excluded.

Training-induced changes in H-reflex excitability have previously been reported in animal models as well as in humans, although the number of longitudinal studies is limited. H-reflex excitability has been shown to increase or decrease in monkeys and rats exposed to either long-term positive or negative operant conditioning paradigms (7, 9, 50), demonstrating that considerable adaptive plasticity exists for the H-reflex pathway. In accordance with this notion,  $H_{\max}/M_{\max}$  was reported to decrease in rats after training that involved stretch-shortening exercise (4).

An increase in  $H_{\max}/M_{\max}$  was observed in the soleus muscle after endurance training (33) and intensive hopping training (49) in humans. This increase in H-reflex excitability might represent a beneficial adaptation because it allows subjects to take increased advantage of the excitatory Ia inflow caused by stretch of muscle spindles before and during the initial phase of ground contact. Because of the relatively long latency of the intact stretch reflex (45–60 ms), the increase in reflex-potentiated muscle force would occur primarily in the late ground contact phase, in which it could contribute to the generation of propulsive force and limb acceleration. Conversely, a decrease in soleus H-reflex gain has been reported after short-term balance board training (45). This reduction in H-reflex excitability may also represent a beneficial adaptation, possibly reflecting an increase in presynaptic inhibition of Ia afferents as a result of reciprocal inhibition mechanisms associated with cocontraction of the tibialis and soleus muscles. In fact, it would seem optimal to have suppressed stretch-reflex responses in the tibialis-soleus muscles during postural balance tasks to prevent the occurrence of reflex-mediated joint oscillation. Accordingly, soleus H-reflex excitability appears to be markedly suppressed in beam walking compared with normal walking (21).

When recorded during rest, the soleus H-reflex amplitude (determined as  $H_{\max}/M_{\max}$ ) was greater in endurance athletes than in athletes trained for anaerobic or explosive sports (8, 22, 34). It cannot be excluded,

however, that these findings were the result of subject differences in muscle fiber composition, because at low stimulation intensity the excitatory postsynaptic potentials caused by the Ia afferent volley exert a relatively stronger excitatory influence on the smallest spinal motoneurons that innervate the population of slow-twitch type I muscle fibers (16). Reduced soleus H-reflex excitability was observed in professional ballet dancers compared with physically active young men, which was suggested to reflect elevated levels of presynaptic inhibition in the dancers (31) although differences in muscle fiber composition could not be ruled out. Interestingly, Mynark and Kocejka (28) found no difference in soleus H-reflex amplitude between trained dancers and controls during standing or prone rest, whereas the gain of the H-reflex (i.e., the ratio of H-reflex to background EMG) was lower in the dancers during isometric muscle contraction at 10, 20, and 30% MVC performed in a standing position. These data indicate not only that spinal excitatory and inhibitory pathways can be modulated to adapt to the contraction related demands placed on the system (28), but also that H-reflex measurements obtained during rest does not adequately reflect neuromuscular function and performance during actual muscle contraction. The latter notion is further supported by the results of the present study and those of Voigt et al. (49), who demonstrated that, whereas resting  $H_{\max}/M_{\max}$  remained unchanged in response to training, H-reflex excitability measured during active muscle contraction actually increased.

Taken together, the above findings strongly suggest that training-induced changes in neural motor function should be evaluated by use of H-reflex recordings obtained during actual muscle contraction and not rely solely on measurements obtained in resting conditions. Moreover, H-reflex data obtained during maximal muscle contraction, as performed in the present study, likely represent a more functional estimate of the training-induced change in motoneuron excitability (including presynaptic inhibition) than that achieved by measurements of the H-reflex at rest.

In conclusion, evoked V-wave and H-reflex responses were observed to increase during maximal muscle contraction after a regime of heavy-resistance strength training, reflecting a substantial rise in efferent motor output of spinal motoneurons during maximal voluntary muscle contraction. Collectively, these data support the notion that neural adaptation occurs at both supraspinal and spinal levels, involving an enhanced drive in descending pathways from higher motor centers as well as increased motoneuron excitability and/or changes in presynaptic Ia afferent inhibition, respectively.

In contrast to the changes seen during maximal muscle contraction, the H-reflex response obtained at rest ( $H_{\max}/M_{\max}$ ) remained unchanged with training. Consequently, evoked reflex responses should be obtained during actual contraction and not solely include measurements obtained at rest.



Importantly, the potential problem of ensuring identical pre- and posttraining recording conditions when measuring conventional muscle EMG was eliminated with the present V-wave and H-reflex measurements, because all evoked reflex amplitudes were controlled and expressed relative to the amplitude of the maximal direct M-wave.

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