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REVIEW ARTICLE

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Considerations for use of the Hoffmann reflex in exercise studies

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Abstract There continues to be great interest in evaluating the adaptive plasticity of the human nervous system in response to exercise training or other interventions. For various reasons, researchers have been interested in estimates of spinal reflex processing in intact human subjects before and after training. A reflex pathway that has been employed in this regard is the Hoffmann (H) reflex. This brief review describes the basic procedure for evoking the H reflex in different muscles. Other sections address methodological issues that affect interpretation of the H reflex. In particular, the role that presynaptic inhibition serves in the modification of the H reflex and how this precludes its use as an unambiguous measure of alpha-motoneuron excitability will be discussed. Applications of the H reflex to study adaptive plasticity in humans is also reviewed, and methodological requirements that should be maintained for accurate interpretation of H reflexes in exercise studies are presented.

Keywords Neural · Plasticity · Training · EMG · Methodology

Introduction

Originally described by Paul Hoffmann in the early 20th century (Hoffmann 1910, 1918) and carrying his name, the Hoffmann (H) reflex is considered to be the electrical analogue of the stretch reflex, but bypasses the effects of gamma motoneurons and of muscle spindle discharge (Brooke et al. 1997a; Schieppati 1987). This reflex has received considerable attention in literature concerning movement control, clinical neurophysiology, and applied physiology. The first objective of this short review is to provide an overview of the methodology used to evoke H reflexes in the muscles that have been most commonly studied. Secondly, the point will be made that the H reflex is not a direct measure of alpha-motoneuron excitability due to the effect of presynaptic inhibition on reflex amplitude. Thirdly, basic methodological requirements for successful use of the H reflex technique are addressed, with a particular emphasis on controlling extraneous factors that can affect H-reflex amplitude. Finally, the use of the H reflex to study the adaptive plasticity arising in spinal reflex pathways as a result of exercise training or detraining is addressed.

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Evoking the H reflex

The technique used to evoke the H reflex involves electrical stimulation of a mixed (i.e., containing both motor and sensory axons) peripheral nerve. Stimulation to evoke the H reflex involves both afferent sensory (from the point of stimulation to the spinal cord) and efferent motor (from the alpha motoneurons in the spinal cord to the neuromuscular junction) arcs as well as a direct (from the point of stimulation to the neuromuscular junction) efferent motor response (M wave). When percutaneous stimulation of increasing intensity is applied, the Ia afferents that innervate muscle spindle sensory receptors, because of their larger diameter, will be recruited before the smaller diameter motor axons (Erlanger and Gasser 1968; Kukulka 1992; Li and Bak 1976). Therefore, the H reflex can be observed with or without an M wave. An H reflex will be recorded if electrical stimulation of the nerve is above threshold for activation of Ia afferents and the afferent terminals are sufficiently depolarized to cause neurotransmitter release at the Ia afferent/alpha-motoneuron synapse. Significant release of a neurotransmitter from the primary afferent

terminals will then result in postsynaptic depolarization of alpha motoneurons. If this postsynaptic depolarization is above threshold, then the alpha motoneurons will fire action potentials that will cause neurotransmitter release at the neuromuscular junction. This will result in depolarization and contraction of the muscle fibers, which will then be recorded as an H reflex in the muscle under study. This is typically recorded, using surface electromyography (EMG) electrodes placed over the muscle of interest, as a population muscle action potential. Increasing the level of electrical stimulation recruits additional Ia afferent and motor axons, thus yielding a larger reflex response and a larger M wave (Magladery 1955). Because of this, measurement of the M wave is often used to monitor stimulus constancy (see section headed "Stimulus constancy"). Shown in Fig. 1 is a cartoon schematic of the basic circuit involved in the H reflex (Fig. 1A) along with experimentally derived data obtained from the soleus muscle (Fig. 1B).

The amplitudes of the H reflex and M wave will both increase fairly linearly with the stimulation intensity until the maximum H reflex (H_{max}), representing the fullest extent of reflex activation, and, at higher stimulation levels, the maximum M wave (M_{max}), representing the maximal muscle activation, are reached. By incrementally increasing stimulation intensity from low levels sufficient to evoke a small H reflex, up to H_{max} and then M_{max}, an H-reflex recruitment curve can be obtained. Shown in Fig. 2 is an example of an H-reflex recruitment curve obtained using the forearm muscle flexor carpi radialis (FCR). With incremental application of stimulation from zero, the following notable parts of the recruitment curve will be observed: H-reflex threshold, M-wave threshold, H_{max}, and M_{max}. For reference, H_{max} and M_{max} are indicated in Fig. 2.

Recruitment of motor units by corticospinal or Ia afferent inputs (as in the H reflex) proceeds in an orderly fashion from smallest to largest according to the "size principle" (Henneman et al. 1965; Somjen et al. 1965). It has been shown that the percentage of motoneurons recruited into the soleus H reflex averages around 50% (range 24–100%; Taborikova 1968). Furthermore, it has been suggested that, again in the soleus, the lower threshold and so-called smaller, "slow" motor units predominate in the human H-reflex response (Buchthal and Schmalbruch 1970), and that recruitment according to stimulus intensity proceeds in an orderly manner from small to large motor units (Awiszus and Feistner 1993). Indeed, based upon computer simulations, Ia-induced effective synaptic currents in small motoneurons may be almost twice as large as those in large motoneurons (Heckman and Binder 1993), thus strengthening orderly motor unit recruitment in the H reflex. However, motor unit recruitment during H-reflex stimulation examined in the human tibialis anterior (TA) muscle appears to be the opposite of that seen in soleus (Semmler and Turker 1994). Whether this represents a functional separation in connections from Ia afferents to flexor and extensor

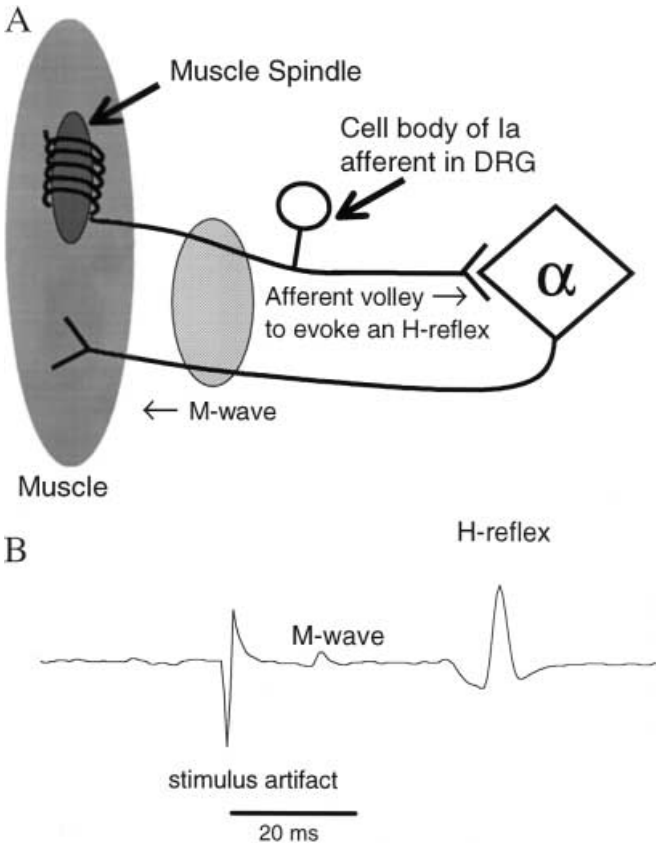

Fig. 1 **A** Simplistic schematic of the spinal processing of the monosynaptic component of the H reflex. The electrical stimulus used to activate the mixed peripheral nerve is shown by the *grey ellipse*. The activation of the nerve is shown to propagate orthodromically in the motor axons to evoke the M wave, and orthodromically in the sensory axons (shown here as group Ia afferents arising from annulospiral endings on the muscle spindle) to evoke the H reflex via a monosynaptic connection to the alpha motoneurons (α). **B** Stimulus-triggered and averaged ($n = 20$ sweeps) H reflexes evoked in the soleus muscle while maintaining a tonic contraction of ≅10% maximum voluntary contraction (data from Zehr and Stein 1999a). (*DRG* Dorsal root ganglion)

muscles or a methodological problem in human motor unit studies (i.e., type-identifying motor units based upon force recruitment thresholds) is presently unclear. Furthermore, in the upper limb, a skewed distribution of Ia effects (wherein larger motor units receive greater Ia inputs) to the motoneurons of intrinsic hand muscles has been identified (Mazzocchio et al. 1995). This may be due to specific adaptations in the human upper limbs necessary for reaching and grasping, and occurring as an outcome of the strong corticospinal connections to the upper limb.

To summarize, it is likely that smaller motoneurons are recruited first when increasing nerve stimulation to evoke the H reflex is applied in many muscles. However, the evidence to support this is far from complete and is mostly inferential, and there may be significant differences between H reflexes in the upper and lower limbs owing to differences in synaptic efficacy and connectivity between motoneuron pools.

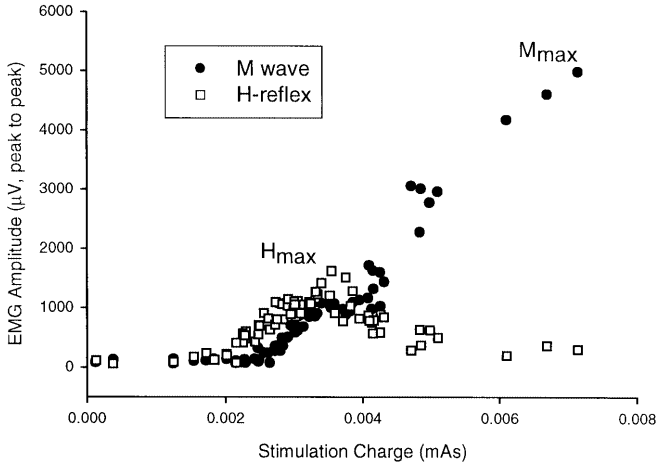

Fig. 2 Sample recruitment curve for M waves and H reflexes in the forearm muscle flexor carpi radialis (*FCR*). Data were obtained while the subject performed a tonic, low-level (≅10% MVC) flexion contraction while in pronation. Note the position of the maximal H and M responses (H_{max} and M_{max}, respectively). (*EMG* Electromyography)

Occurrence of H reflexes in human limb muscles

H reflexes have been evoked in many different muscles of both the upper and lower limbs. The most commonly studied muscle in the lower limb is the soleus, and in the upper limb the FCR. Brief procedures for evoking and recording H reflexes in numerous muscles of the upper and lower limbs that may be of interest to applied physiologists are given in Tables 1 and 2, respectively. Provided in the tables is information regarding the muscle from which the reflex was recorded along with brief details about the electrode placement for EMG recording and nerve stimulation (see also Leis and Trapani 2000). Also given are details of selected references with an emphasis on methodology. Furthermore, both tables show the percentage occurrence of H reflexes in each muscle as determined from the selected references. It should be noted that it is often difficult to evoke an H reflex in many muscles while the subject is at rest (Hultborn and Nielsen 1995; Mazzocchio et al. 1995), but that with voluntary contraction many muscles in which the peripheral nerve is accessible to stimulation will yield H reflexes (Burke et al. 1989). In addition, although there is a large amount of literature regarding H reflexes in the muscles of the trigeminal system, the focus here is restricted to H reflex studies of the limb musculature.

As already mentioned, the muscle that has received considerable attention in studies of the H reflex is the ankle extensor, soleus. An H reflex can be evoked in the soleus muscle by stimulation of the tibial nerve (Hugon 1973). In this case, the H reflex has typically been evoked by placing surface electrodes in either a bipolar configuration over the predicted path of the tibial nerve in the popliteal fossa, or with one electrode in the popliteal fossa and one over the patella. (for a review of further details about evoking the H reflex in the soleus see Brooke et al. 1977a; Capaday 1997; Hugon 1973; for specific details on EMG methodology see Merletti et al. 2001; Zipp 1982). Regardless of the muscle under study, the general procedure is to initially place one electrode over the predicted path of the nerve and then to carefully move the electrodes until the best response (in terms of clarity of H reflex and M wave) is observed. Refer to Tables 1 and 2 for details of recording and stimulation conditions for the muscle of interest.

Is H reflex amplitude a valid measure of alpha motoneuron excitability?

Originally suggested to be a purely monosynaptic reflex, in fact only the rising edge of the H reflex waveform is monosynaptic (Burke et al. 1984; Hultborn and Nielsen 1995; Stein 1995). Burke et al. (1984) estimated the rise times of the composite excitatory postsynaptic potentials (EPSPs) in soleus motoneurons by examining the changes in discharge probability of single motor units activated voluntarily and induced by a subthreshold (for evoking a reflex) tendon tap or electrical stimulation of the tibial nerve. It was suggested that for both stretch and H reflexes in the soleus muscle, the rise time of the composite EPSP had a long duration (≅10 ms) such that oligosynaptic contributions to the later portions of the H reflex waveform are likely to be involved. In particular, inhibitory Ib effects from Golgi tendon organ afferents were suggested as possible confounding contributors to the H-reflex waveform (Burke et al. 1984), although the implications and quantification of this effect require further research. In addition, it has been shown that while the monosynaptic excitation is greater in smaller motoneurons of the FCR and extensor carpi radialis muscles of the forearm, non-monosynaptic excitation seems to be preferentially distributed to larger motor units (Marchand-Pauvert et al. 2000).Thus the H reflex should not be considered as a purely monosynaptic response. However, because of the direct anatomical synaptic contact between Ia afferents and alpha motoneurons, the H reflex has been much studied in motor control research and in exercise and clinical studies in which neural adaptations involving motoneuron excitability is of interest. Unfortunately, it is exactly this issue of motoneuron excitability for which the H reflex became known that is at the heart of continued misinterpretation.

Due to the direct synaptic connection of Ia afferents and alpha motoneurons it has been tempting for researchers to assume that the H reflex represents faithfully the excitability of the motoneuron pool under study. Angel and Hofmann (1963) were amongst the original advocates of using the H reflex in this way, and this continues to the present day (see recent examples Dishman and Bulbulian 2000; Hopkins et al. 2000a, b, 2001; Kalmar and Cafarelli 1999). However, the synaptic connection between Ia afferents and alpha motoneurons is itself subject to modification. It is sensitive to

Table 1 Details on H-reflex recording from some muscles of the upper limb. Where possible, the H-reflex percentage occurrence has been taken from the specific references cited. Note that a general reference for the anatomical location of recording and stimulation sites is Leis and Trapani (2000). For reference, the spinal nerve root for each nerve stimulated is given in parentheses. (*OP* Opponens pollicis, *APB* abductor pollicis brevis, *Na* not available)

Muscle	EMG recording	Nerve stimulation	H reflex (%)	Specific references
Flexor carpi radialis	One-third distance on a line from the medial epicondyle of the humerus to the radial styloid: wrist flexion with radial deviation will assist in location	Median nerve (C6 and C7): cathode in the cubital fossa, just above the elbow under the curve of biceps brachii muscle	73–100	(Aymard et al. 2000; Baldissera et al. 1987; Burke et al. 1989; Day et al. 1984; Delwaide et al. 1988; Deschuytere et al. 1976; Garcia et al. 1979; Inghilleri et al. 2000; Jabre 1981; Jusic et al. 1995; Marchand-Pauvert et al. 2000; Panizza et al. 1989; Rossi-Durand et al. 1999; Sabatino et al. 1992; Sabbahi and Khalil 1990a)
Flexor digitorum superficialis	7–9 cm distal to the biceps tendon and 2–3 cm medial to the ventral midline; finger flexion at proximal joints with distal joints in extension will cause activation	Median nerve (C7, C8, T1): see above	50–73	(Abbruzzese et al. 1996a; Baldissera et al. 2001; Jusic et al. 1995)
Thenar: (OP, APB)	OP: Obliquely at the midpoint of the first metacarpal shaft just lateral to the APB muscle; opposition of the thumb to the little finger to activate. APB: Over the APB muscle and the other midway along the proximal phallanx of the thumb; abduction of the thumb to activate	Median nerve (C8 and T1): Cathode attached to the skin and the anode positioned on the dorsal surface of the wrist, or stimulation at the proximal crease of the wrist.	0–21	(Abbruzzese et al. 1996a; Burke et al. 1989; Cowan et al. 1986; Jusic et al. 1990, 1995)
Brachioradialis	Electrodes placed on the belly of the brachioradialis or 2–3 cm lateral to the biceps tendon; flexion of the forearm in the neutral position activates the muscle.	Radial nerve (C5 and C6): distal stimulation 5 cm over the lateral humeral epicondyle with cathode oriented proximally.	16–37	(Abbruzzese et al. 1994; Jusic et al. 1995)
Extensor Carpi Radialis	≅One-fifth of the distance on a line from the lateral epicondyle of the humerus to the second metacarpal; extension and radial deviation at the wrist performed with finger flexion will assist in location	Radial nerve (C5-C7): 3–6 cm proximal on the lateral aspect of the arm where the nerve leaves the spiral groove, or mid-forearm between the ulna and radius	20–50	(Baldissera et al. 1987; Day et al. 1984; Katz et al. 1993; Marchand-Pauvert et al. 2000)
Extensor digitorum communis	Middle of the forearm between the ulna and the radius; extension of fingers 2–5 will activate	Radial nerve (C7 and C8): see above	0–72	(Garcia et al. 1979)
Flexor carpi ulnaris	5–8 cm distal to the medial epicondyle along a line connecting the medial epicondyle and pisiform bone; wrist flexion with ulnar deviation to activate	Ulnar nerve (C8 and T1): Proximal to the cubital tunnel or at the level of the axilla, above the elbow or just above the medial humeral epicondyle	26–47	(Jusic et al. 1995)
Abductor digiti minimi	One-quarter of the distance from the ulnar styloid to the distal phalange of the little finger; abduction of the little finger will activate	Ulnar nerve (C8 and T1): stimulate at wrist	Na	(Mazzocchio et al. 1995)

mechanisms that cause changes in the presynaptic inhibition of Ia afferent transmission and that directly affect neurotransmitter release at the Ia/alpha-motoneuron synapse. This aspect of the H reflex has been known for some time (Capaday and Stein 1989; Morin et al. 1984; Schieppati 1987), but has been more clearly defined, discussed and reviewed recently (see reviews in Brooke et al. 1997a; Stein 1995).

Despite the above caution, there are scenarios in which changes in H-reflex excitability could reflect alpha-motoneuron excitability. For example, if a subject maintained the same postural orientation, the same intent, the same level of muscle activation, and was not moving, and then a short-latency pathway [e.g., condition (C)–test (T) interval, or delay between a stimulus intended to alter activity in the pathway and the actual

Table 2 Details on H-reflex recording in some muscles of the lower limb. Where possible, the H-reflex percentage occurrence has been taken from the specific references cited. Note that a general reference for anatomical location of recording and stimulation sites is Leis and Trapani (2000). For reference, the spinal nerve root for each nerve stimulated is given in parentheses. (*EMG* Electromyography, *VM* vastus medialis, *RF* rectus femoris, *MG* medial gastrocnemius, *AHP* abductor hallucis pollicis, *FDB* flexor digitorum brevis)

Muscle	EMG recording	Nerve stimulation	H reflex (%)	Specific references
Quadriceps (VM, RF)	Electrodes on the belly of the VM or RF; knee extension to activate	Femoral nerve (L2–L4): Stimulation site found a line between the anterior superior iliac spine and upper tip of patella, or 5–7 cm above the patella in the anteromedial thigh, also been found by stimulating the inguinal region	100	(Hultborn et al. 1987a, b; Jusic et al. 1995; Sabbahi and Khalil 1990b)
Tibialis anterior	One-half of the distance between the tibial tubercle and the ankle; dorsiflexion to activate	Deep peroneal nerve (L4 and L5): Behind the head of the fibula or proximal to the head of fibula	0–11	(Brooke et al. 1997b; Cowan et al. 1986; Garcia et al. 1979; Jusic et al. 1995; Pierrot-Deseilligny et al. 1981b)
Biceps femoris	One-third to midway along a line connecting the fibular head with the ischial tuberosity; knee flexion to activate	Sciatic nerve (tibial division, L5, S1 and S2): Proximal ischiadicus stimulation at the gluteal sulcus	97–100	(Pierrot-Deseilligny et al. 1981a; Jusic et al. 1995)
Semitendinosus	One-third to midway along a line connecting the tendon with the ischial tuberosity; knee flexion plus hip extension to activate	Sciatic nerve (L5, S1 and S2): see above	93–100	(Jusic et al. 1995)
Soleus	One half of the distance between the mid-popliteal crease and the medial malleolus, or just distal to the belly of the MG, medial to the Achilles tendon; plantar flexion to activate	Tibial nerve (S1 and S2): Stimulation at the popliteal fossa	77–100	(Aymard et al. 2000; Cowan et al. 1986; Garcia et al. 1979; Hultborn et al. 1987a, b; Jusic et al. 1995; Kasai and Komiyama 1996; Kawanishi et al. 1999; Rossi-Durand et al. 1999; Sabbahi et al. 1990; Zehr and Stein 1999a; Zehr et al. 2001)
Intrinsic muscles of the foot (AHP, FDB)	AHP: one-third of the distance from the calcaneous to the big toe on the medial side of the foot; abduction of the big toe will activate. FDB: one-half of the distance from the distal phalanges to the calcaneous; flexion of the four lesser toes will activate	Medial plantar nerve (from Tibial nerve S1 and S2): Stimulation at the medial malleolus	Na	(Abbruzzese et al. 1996b; Burke et al. 1989; Ellrich et al. 1998)

activation of the pathway, of ≅2 ms], such as reciprocal inhibition, was tested by antagonist nerve stimulation, the alterations in excitability would reflect alpha-motoneuron excitability. The important point is that it is dangerous to interpret changes in H-reflex size as changes in motoneuron excitability. The primary reason for this is the effect of presynaptic inhibition (PSI).

PSI of the H reflex

PSI of spinal monosynaptic reflexes was initially described in the cat in 1957 (Eccles et al. 1962; Frank and Fourtes 1957), and has since received considerable experimental attention (for review see Rudomin and Schmidt 1999). PSI is mediated by the action of an inhibitory interneuron (using gamma aminobutyric acid as the neurotransmitter; Rudomin and Schmidt 1999) acting on the Ia afferent terminals, leading to a reduction in neurotransmitter release and a concomitant reduction in motoneuron depolarization induced by Ia activity. Thus, afferent transmission can be altered without a corresponding effect on the postsynaptic (e.g., motoneuron) membrane. Frank and Fourtes (1957) demonstrated that in the presence of PSI there was no change in the postsynaptic membrane potential, despite activity in the Ia afferents. Furthermore, the motoneurons remained receptive to other inputs that were unaffected by PSI. This provided conclusive evidence that PSI could selectively alter transmission in a monosynaptic reflex pathway, and it has recently been demonstrated that this mechanism is selective enough to affect different collaterals from the same muscle spindle afferent (Rudomin et al. 1998).

It is for this reason that one cannot determine unambiguously the level of alpha-motoneuron excitability by measuring an H reflex. PSI can alter the afferent signal that actually evokes the H reflex and thus can lead to a separate pattern of modulation of reflex and motoneuron excitability (a property of the postsynaptic cell membrane). This holds for reflexes in many muscles, but has been most clearly illustrated in the H reflexes of leg extensor muscles such as the soleus. This observation is illustrated in Fig. 3, in which a cartoon schematic representing PSI is shown (A, top) along with the corresponding inhibition of the H reflex (B, bottom). In panel B of this figure, the H reflex has been presynaptically inhibited by conditioning with CP nerve stimulation (at a C–T interval of 100 ms; see section "Increasing PSI") to increase PSI and is shown superimposed on the control H-reflex waveform. Note that conditioning the soleus H reflex with prior CP nerve stimulation at C–T intervals of 50–120 ms is considered to increase PSI (Capaday et al. 1995; Iles 1996; Morin et al. 1984; Zehr and Stein 1999a). In both cases (e.g., the control H reflex and that elicited in the presence of increased PSI), the level of motoneuron pool excitability (as measured using the background EMG activation level of the soleus muscle) was held at a constant level by voluntary contraction. The change in H-reflex amplitude shown in Fig. 3B represents a change in PSI and not a change in motoneuron excitability.

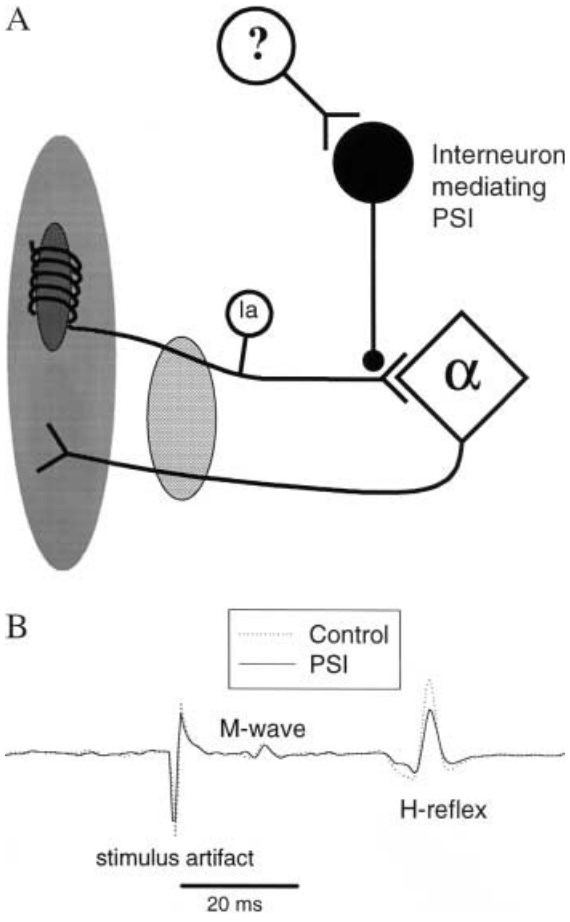

Fig. 3 **A** Schematic of presynaptic inhibition (*PSI*) of the H-reflex pathway. The transmission from Ia afferents (*Ia*) onto alpha motoneurons is shown to be presynaptically inhibited via axo-axonal action of an inhibitory interneuron (*filled neuron*). The PSI interneuron is activated from non-specific sources, as indicated by the excitatory interneuron at the top of the figure (indicated by *?*). Other details are as found in Fig. 1A. **B** The effect of PSI on the soleus H-reflex pathway. The H reflex is shown to be presynaptically inhibited (*solid line*) as compared to the control (*dashed line*) reflex. Note that the soleus muscle was tonically contracted to ≅10% MVC and that the amplitude of the M wave was similar in both conditions. Furthermore, the level of activation in the antagonist motor pool was constant (not shown) and the postural orientation of the subject was the same in both conditions. Thus, the reduction in amplitude of the soleus H reflex was due to PSI. Data from Zehr and Stein (1999a)

What factors affect PSI of the H reflex?

Many factors affect PSI of the H-reflex pathway (see recent reviews by Brooke et al. 1997a; Capaday 1997; Pierrot-Deseilligny 1997; Pierrot-Deseilligny and Meunier 1998; Stein 1995). These factors include, but are not limited to, afferent feedback from other peripheral receptors (e.g., muscle spindles, Golgi tendon organs, cutaneous mechano receptors) and descending supraspinal commands. The main point here is not to highlight each possible pathway, but rather to note that the effect of many of these factors can be assumed to be controlled by maintaining the posture and intention of the subject. For example, in exercise studies, use of the H reflex typically involves pre- and post-training measurement or serial measurement over time. Therefore, in order to reduce or minimize extraneous factors that can alter H-reflex amplitude, it is important that the reflex be evoked under the same conditions (including the postural orientation and "set" of the subject). This is of even more importance when movement is involved because it is known that movement induces tremendous modulation in H-reflexes of many muscles (see section "Modulation of H reflexes with posture and movement").

In this regard, a clinical example pertinent to the effect of PSI of Ia transmission on H-reflex amplitude is that of the Jendrassik maneuver. Originally demonstrated as a remote facilitation of stretch reflexes in the lower limb after forearm extensor muscle contraction and jaw clenching (Jendrassik 1883), the facilitation of H reflexes was later also shown (Bussel et al. 1978; Landau and Clare 1964). Once the influence of the fusimotor system had been disproved (Hagbarth et al. 1975), for some time it was thought that motoneuron excitability was altered by the Jendrassik maneuver. This was shown by others to be incorrect, and presynaptic mechanisms were proposed (Dowman and Wolpaw 1988). Recent evidence supports the assertion that a modulation of PSI is the likely mechanism that underlies this clinically useful test (Zehr and Stein 1999a).

Basic procedures to study PSI of the H reflex

Depending upon the focus of the research, it may be of interest to examine specific changes in reflex pathways that result from exercise training. Specific changes in the PSI of Ia afferent transmission and alterations of H-reflex amplitude would be an example of this and serves as a better alternative than attempting to address motoneuron excitability with this reflex. There are two procedures that can be used readily to study PSI of the H reflex. One is to apply an input that will increase PSI and the other is to apply one that will reduce PSI. Although this approach can theoretically be applied to many muscles in which an H reflex can be observed, the following discussion refers to the soleus H reflex.

Increasing PSI

PSI can be increased by stimulation of the nerves supplying the antagonist muscle (or by vibration of the muscle belly or antagonist muscle tendon; see later). In the case of the soleus, this involves stimulation of the CP nerve (Zehr and Stein 1999a; originally described by Capaday et al. 1995; Iles 1996; Morin et al. 1984). In this paradigm, the CP is typically stimulated with a single 1.0-ms pulse at 1–1.5 times the threshold for evoking a motor response in the TA muscle. This stimulation strongly activates Group I afferents in the CP nerve that project to and mediate PSI of the soleus monosynaptic Ia afferent pathway. The soleus H reflex is then evoked by stimulating the tibial nerve after a delay of ≅80–120 ms. Suppression of the H-reflex amplitude at these latencies is due to PSI (Eccles et al. 1962; Iles and Roberts 1987).

Reducing PSI

Another way to alter the level of PSI is to apply an input that should lead to facilitation in the H-reflex pathway and then determine if it is reduced by another input stimulus to increase PSI. Subthreshold electrical stimulation of the tibial nerve (to induce a homonymous facilitation of the soleus H reflex) is strongly affected by vibration of the TA muscle (to increase discharge of the Ia afferents that affect PSI of Ia afferents in the tibial nerve supplying the soleus, such as described above for electrical stimulation; Morin et al. 1984). However, there is concern over the extent to which the vibration and subthreshold (for evoking an H reflex) tibial nerve stimulation can evaluate excitation in this pathway. Thus, this same group proposed a method for assessing PSI in humans by inducing heteronymous facilitation of the soleus H reflex by femoral nerve stimulation (Hultborn et al. 1987a, b). It was demonstrated (and confirmed in the cat) that alterations in the soleus H reflex due to the afferent volley evoked by stimulation of the femoral nerve represented alterations in PSI of the soleus H-reflex pathway. Thus, one can use femoral nerve stimulation as a test for presynaptic inhibition. In this case, the extent of PSI is measured by the amount that presynaptic facilitation from femoral nerve stimulation is reduced.

Other factors that influence H-reflex amplitude

Presynaptic modulation of the H reflex is a major factor that must be considered when interpreting H-reflex data, but other factors (which may also relate to or interact with PSI) must be considered when evoking and interpreting the H reflex. There is extensive modulation of transmission in the H-reflex pathway. As mentioned earlier, peripheral feedback from muscle spindle receptors, Golgi tendon organs, cutaneous afferents, joint afferents, and vestibular inputs can all affect the amplitude of the H reflex (for review see Schieppati 1987). Thus, careful control over these peripheral inputs must be maintained by monitoring the posture of the subjects and the contraction of other muscles. In addition to those factors, the effect of depolarization toward threshold in the motoneuron pool and consistent nerve stimulation must be considered.

Level of motoneuron pool depolarization (background excitability)

In human subjects, motoneuron pool activation can be estimated by measuring and controlling the background level of muscle contraction through the monitoring of surface EMG recordings. In general, the amplitude of the H reflex will increase linearly with the number of motoneurons recruited in the target motoneuron pool (Burke et al. 1989; Matthews 1986; Schieppati 1987). However, there are non-linear aspects to this relationship (Funase and Miles 1999; Stein 1995). For example, it was shown that, in triceps surae, H-reflex amplitude increased linearly with background EMG in most subjects, but that in some, reflex amplitude increased only up to ≅50% of maximum voluntary isometric contraction (MVC) whereafter it plateaued or declined (Loscher et al. 1996). Thus, when comparing the amplitude of the H reflex across conditions or serially in time, the reflexes must be evoked when the target muscle is activated to the same relative level. In the example shown in Fig. 3B, the H reflexes were sampled when the soleus muscle was voluntarily contracted to the same relative level (about 10% MVC), thus allowing for direct comparison across conditions.

Alternatively, the H reflex can be evoked while the target muscle is electromyographically silent. Indeed, many researchers have measured the H reflex in quiescent muscles. An advantage of this approach is that cortical effects on spinal interneurons that are affected by voluntary contraction (such as those mediating recurrent inhibition) are likely to be less active and will not

confound the results (Katz and Pierrot-Deseilligny 1999; Pierrot-Deseilligny 1997). However, at rest the relative state of depolarization of the motoneuron pool (which is sensitive to many inputs) is not known. Furthermore, contraction can usefully reduce the variability in both the latency and amplitude of the H reflex (Burke et al. 1989; Funase and Miles 1999). Thus, it is recommended that the H reflex is evoked upon a tonic level of background muscle activation whenever possible (see Pierrot-Deseilligny 1997 for a contrary view).

Stimulus constancy

Assuming a consistent level of presynaptic and postsynaptic inhibition or facilitation, the amplitude of the H reflex will vary directly with the afferent volley arriving at the Ia afferent-motoneuron synapse. In an experiment it is critical that the synaptic input received by the alpha motoneurons is constant. That is, the same number of Ia afferent axons must be activated by electrical stimulation under different conditions. In human experiments the H reflex is typically evoked with surface electrodes placed over the peripheral nerve. Movement of the stimulating electrodes or of the nerve relative to the electrodes (e.g., during postural change or continuous movement) will alter the relative activation of the Ia afferent axons, thereby leading to changes in the H reflex, independent of changes in synaptic efficacy or other factors (for more detail see Brooke et al. 1997a). To safeguard against this methodological error, H reflexes should be evoked at an intensity of stimulation that also evokes a direct muscle response (i.e., the M wave). It is also suggested that constant-current stimulation, as opposed to constant-voltage stimulation, be employed to minimize the effect of time-dependent changes in skin-electrode impedance. The size of the M wave can then be monitored as a means of estimating and controlling stimulus consistency. However, activity-dependent changes in axonal excitability should be considered when using this as a control (Burke and Gandevia 1999).

Effect of reflex size

It has been demonstrated that the susceptibility of the H reflex to conditioning depends upon the size of the reflex itself (Crone et al. 1990). Thus, when comparing across different conditions or over time, the same relative afferent input must be compared. To help with this it is useful to obtain M-wave/H-wave (M/H) recruitment curves for different experimental conditions (Baldissera et al. 2000; Zehr and Stein 1999a; Zehr et al. 2001). In Fig. 4, M/H recruitment on the ascending limb of the curve (to the maximal H-reflex amplitude) was obtained from the soleus in a condition that reduced PSI (facilitation via the Jendrassik maneuver), a condition that increased PSI (CP nerve stimulation with a C–T interval of 100 ms), and during control with no reflex

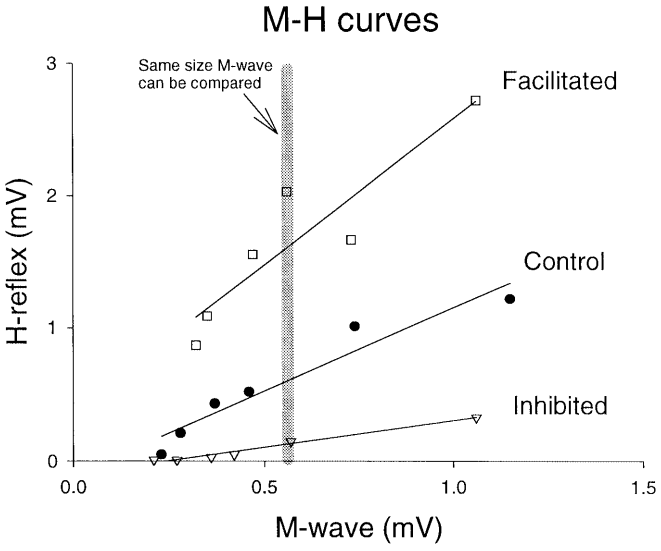

Fig. 4 Evaluating conditioning of the H reflex using an M/H curve to control for the effect of test reflex amplitude and M-wave size. Plotted are averaged data from one subject in whom control (*filled circles*), facilitated (*open squares*), and inhibited (*open triangles*) H reflexes were obtained from the soleus muscle. Significant regression lines indicating the ascending limb of the M/H curve are indicated for each condition. The *vertical grey line* shows an M-wave amplitude at which reflex conditioning can be seen and compared across conditions. Note that in all cases the subject was attempting to hold the soleus EMG contraction at a stable and constant level equal to ≅10% MVC. Modified data from Zehr and Stein (1999a)

conditioning (Zehr and Stein 1999a). Control for the effect of different reflex amplitudes and controlling for M-wave size can be obtained by comparing reflex amplitudes at the position indicated by the vertical grey line shown in Fig. 4. Alternatively, one can obtain many different M/H pairs and group together those with similar M-wave sizes and take an average (Zehr et al. 2001). In either case one can thus control for reflex conditioning at different reflex sizes as well as for the amplitude of stimulation as expressed by the M-wave amplitude, provided the relative threshold of Ia afferents and motor axons remains constant. When using this procedure, researchers can select the desired reflex size (usually expressed as a percentage of the maximum M-wave response; see later). This is often in the range of 15–25% of the maximum M wave (Crone et al. 1990). Depending upon the question under study, this factor, or the relative amplitude of the response, may have greater or lesser importance. However, irrespective of the question under study, it is suggested that researchers should always acquire full recruitment curves. In this way, the best control over the effect of reflex size can be implemented.

Post-activation depression and stimulus timing

Repetitive activation of the Ia afferent pathway can lead to reductions in neurotransmitter stores, and thus

neurotransmitter release at the alpha motoneurons (Hultborn et al. 1996; Voigt and Sinkjaer 1998). This "postactivation depression" can persist for many seconds after the activation of the Ia afferents. Furthermore, it has been shown that random alternation of test (T) and conditioned (C) stimuli (e.g., TTCTCCCTTC) in evoking H reflexes is a superior method to repetitive, constant-frequency stimulation (Fournier et al. 1984). In addition, even when evoking an unconditioned H reflex, it is recommended that random stimulation with an interval of no shorter than 3 s be used to evoke the H reflex (at least in the leg; this may be shorter in the arm; Rossi-Durand et al. 1999).

Normalization to the maximum motor response

To compare H reflexes between subjects and conditions, reflex amplitude is often normalized to the maximum evoked motor response. That is, the amplitudes of both the H reflex and the M wave are normalized to the largest M wave that could be evoked during stimulation. However, when movement is occurring, the maximum M wave can vary considerably throughout an experiment and at different limb positions (Simonsen and Dyhre-Poulsen 1999; Simonsen et al. 1995). The theoretical basis for this lies with changes in the mechanical orientation (e.g., pennation angle) of the underlying muscle fibers that occur with movement during muscle activation (Gerilovsky et al. 1989). Furthermore, it is possible that there may be some Na^+/K^+ pump rundown with repeated activation that may affect M-wave amplitude (Nielsen and Clausen 2000). Finally, it has also been demonstrated that profound reductions in M_{max} amplitude can occur across the time course of an experiment, even in the absence of movement or excessive activity (Crone et al. 1999). However, the precise mechanism for this reduction remains elusive. Therefore, it is critical that a maximal M wave be evoked in each condition and in each position of a movement to be used as a reference for that condition or position throughout an experiment.

The H reflex and adaptive plasticity in human movement and exercise studies

In numerous reports, the H reflex has been studied as a window into evaluating changes in human neuromuscular function arising from exercise training. Evaluation of this "adaptive plasticity" has taken many different forms. The ability of the spinal cord to experience this "learning" was a controversial issue for many years. However, an extremely effective model that established the extensive capabilities of the spinal cord to demonstrate adaptive plasticity was shown in the monkey and rat. An elegant series of studies was published by Wolpaw and colleagues (see for review Wolpaw and Tennisen 2001). The initial investigation of the adaptive plasticity of a "simple" spinal reflex was conducted in the primate stretch-reflex pathway (Wolpaw 1983; Wolpaw et al. 1983a, b, c). The ability of behavioral conditioning to change H-reflex amplitude was demonstrated subsequently (Wolpaw 1987). Additional measures were made in further studies that allowed for exact measurement of the underlying mechanisms involved in the adaptation, for example training-induced changes in motoneuron properties (including rheobase, input resistance, and axonal conduction velocity) (Carp and Wolpaw 1994, 1995) and morphological changes in the terminals of sensory afferents (Feng-Chen and Wolpaw 1996) associated with the H-reflex conditioning. These studies established that adaptive plasticity could be induced in the primate spinal cord and that it could be examined by using the H reflex. These observations have been replicated and expanded in a rodent model (reviewed in Wolpaw and Tennisen 2001).

Estimation of adaptive plasticity in humans

Despite the work in the primate and rodent model identified earlier, similar studies of adaptive plasticity in H-reflex pathways occurring as a result of exercise training in humans are much less common. The following review is a review of some of the few studies that have addressed these issues in human subjects (for an earlier review see Zehr and Sale 1994).

Cross-sectional studies

The first study to identify a possible change in H-reflex excitability induced by exercise training showed that subjects who had trained for explosive movements or so-called "anaerobic sports" (e.g., sprinters) had a significantly lower maximum H reflex to maximum M wave ratio in the soleus (see also Funase et al. 1994), whereas endurance-trained subjects had larger ratios (Rochcongar et al. 1979). A later more detailed study by Casabona et al. (1990) showed that the H_{max}:M_{max} ratio was significantly lower in volleyball players and sprinters (e.g., those trained for explosive movement) than in nontrained subjects. Later, it was shown that both H_{max}:M_{max} ratio and disynaptic reciprocal inhibition (from ankle flexor TA onto the soleus) were increased in trained athletes when compared to sedentary control subjects (Nielsen et al. 1993). Interestingly, well-trained ballet dancers were shown to have reduced H_{max}:M_{max} ratios and disynaptic reciprocal inhibition in comparison to the aerobically trained and control subjects. Thus, it was suggested that there are very specific adaptations in motor control mechanisms that arise as a result of training. However, it has been difficult to identify with certainty the locus of the underlying changes, and it is not possible to fully dissociate intrinsic genetic endowment from actual training adaptations in such cross-sectional studies.

Exercise training studies

Adaptive plasticity in human stretch reflexes was demonstrated before experiments on the H reflex were conducted (Evatt et al. 1989; Wolf and Segal 1990). For the soleus H reflex, Perot et al. (1991) demonstrated that the H_{max}:M_{max} ratio was reduced after 8 weeks of endurance training. The first training study to look at voluntary H-reflex plasticity used a paradigm in which subjects were instructed to suppress the reflex response in order to minimize the perturbation of the reflex contraction to their standing balance (Trimble and Koceja 1994). It was demonstrated that subjects could, over the course of a single day of such "training", reduce the amplitude of the soleus H reflex by ≅26%. Furthermore, this reduction in H-reflex amplitude persisted while standing naturally. In a more recent study by Voigt et al. (1998), after 4 weeks of hopping training, subjects showed a reduction in H-reflex inhibition (≅40% of the pre-training standing control value). Interestingly, this more "chronic" training adaptation occurs in the opposite direction to the earlier studies. That is, there is a reduction in the pre-existing inhibition of the soleus H reflex. Voigt et al. (1998) suggest that short-term changes in H-reflex amplitude (such as that observed by Trimble and Koceja 1994) disappear during prolonged training. Finally, the effects of long-term bed rest on the H_{max}:M_{max} ratio has also been examined as an estimate of "detraining" of the H reflex (Yamanaka et al. 1999). It was shown that after 20 days of head-down bed rest, soleus H_{max}:M_{max} ratios were reduced from ≅63% to ≅27%.

"Detraining" effect of aging on the soleus H reflex

A few studies have examined the effects of aging on the human H reflex. Koceja et al. (1995) examined changes in the H_{max}:M_{max} ratio in the soleus muscle when young (≅24 years old) and old (≅76 years old) subjects changed postures from lying prone to standing. It was shown that while young subjects had a much smaller H_{max}:M_{max} ratio during standing compared to walking (presumably due to an increase in Ia PSI in standing; see schematic in Fig. 5), older subjects generally failed to show a modulation. It was later shown that older (≅76 years old) subjects could actually show increased Ia PSI (that is an increase in the H_{max}:M_{max} ratio) when moving from a prone to a standing posture (Angulo-Kinzler et al. 1998). Interestingly, when estimates of Ia PSI of the soleus H reflex have been conducted in aging subjects, it has been shown that Ia PSI tends to increase with age, thus leading to a reduction in H-reflex amplitude (Koceja and Mynark 2000; Morita et al. 1995).

Thus, there is evidence to support chronic adaptive plasticity in the human H reflex. A further issue is the extent and significance of short-term H reflex changes seen during movement.

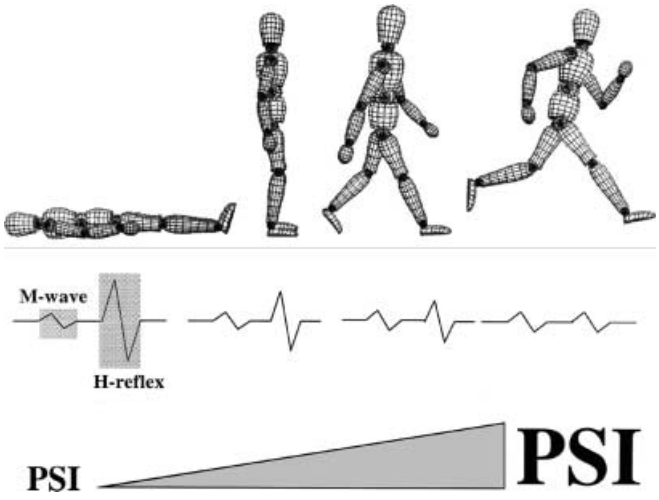

Fig. 5 Changes in PSI cause changes in H-reflex amplitude that depend upon the motor task. The cartoon subject depicted in the top row is shown performing four different motor tasks with corresponding decreases in H-reflex amplitude (*middle*) and increases in PSI (*bottom*). Note that the M wave is held constant and that M-wave and H-reflex amplitude would be normalized to M_{max} for each condition across motor tasks to ensure stimulus constancy and proper comparison. The relative effects of PSI and size of H-reflex amplitude are meant to represent a summary of observations from the literature and not actual data

Modulation of H reflexes with posture and movement

In the literature concerning understanding the mechanisms of human reflex function, H-reflex amplitude has been observed to change between motor tasks, such as standing and walking, as well as within a motor task, such as the swing versus stance phases of walking (for reviews see Brooke et al. 1997a; Stein 1995; Zehr and Stein 1999b). Ia PSI has received significant attention in these studies and has been identified as a major mechanism involved in the control and fine-tuning of afferent feedback from the leg during locomotor tasks (Brooke et al. 1997a; Capaday and Stein 1986; Crenna and Frigo 1987), and in the hand during arm movements (Aimonetti et al. 1999, 2000a, b; Rossi-Durand et al. 1999). In addition, it has been demonstrated that a significant modulation (inhibition) of the soleus H reflex occurs when changing posture from lying, to sitting, to standing (Angulo-Kinzler et al. 1998; Goulart et al. 2000; Koceja et al. 1993, 1995; Mynark and Koceja 1997; Mynark et al. 1997).

Illustrated in Fig. 5 is a schematic representing the effect of changing motor task on PSI of the H reflex. The example shown relates to locomotor and postural tasks and the H-reflex amplitude of leg extensor muscles, but the general concept of task-dependent changes in PSI and H-reflex amplitude may be broadly applicable. As the posture of the cartoon figure is shown to change (left to right at the top of Fig. 5) from lying down, to standing, to walking, and then to running, PSI of Ia transmission is steadily increased (shown schematically at the bottom of Fig. 5). The effect on H-reflex amplitude is shown in the middle of the figure (the M wave is

shown as having the same amplitude to monitor stimulus efficacy as would be maintained in a real experiment and would be obtained by normalization to M_{max} for each condition). Note that the reflex amplitude is steadily reduced across the motor tasks (task-dependent reflex modulation; Stein and Capaday 1988).

Interestingly, while rapid changes in PSI have been suggested to play strong roles in H-reflex modulation during movement, it has also been suggested that chronic alterations in the level of Ia PSI may act as the substrate underlying the training-induced adaptations in H-reflex amplitude described earlier (Voigt et al. 1998). An important point that arises when taking into account task-dependent reflex modulation is that since the nervous system is capable of expressing a given reflex according to the motor task, an attempt to evaluate the training adaptation should also be evoked under the same conditions as training. That is, reflexes are modulated according to function (for locomotion and reflex function see Zehr and Stein 1999b for review). For example, if one wants to evaluate the extent to which a certain type of jumping training causes a change in the soleus H reflex, the reflex amplitude should be measured during the jumping task used for training. This is very similar to the "specificity of training" hypothesis proposed by Sale and MacDougall (1981). Although the response can be evoked at rest or during tonic contraction, it may reveal very little of the functional utility of any adaptive plasticity.

Recommendations and conclusions

It must be mentioned that many of the factors outlined above may not act in isolation, but rather may interact in ways that may yield effects in other neural circuits such as reciprocal (Crone and Nielsen 1994) and recurrent (Katz and Pierrot-Deseilligny 1999) inhibition, which will affect H-reflex amplitude. The following recommendations are suggested to limit the effect of extraneous inputs on the H reflex. Where relevant, examples are included for application in an exercise training study.

1. H reflexes should be evoked upon a background level of muscle activation to ensure a similar level of motoneuron excitability. For example, it is suggested that the reflexes be evoked while subject holds a similar level of activation (e.g., 10% maximal voluntary activation) in the target muscle. It is thus important that maximal voluntary EMG activation measures are made both pre- and post-training. In addition to monitoring the target muscle EMG, antagonist muscle EMG levels should also be measured and controlled to restrict the confounding influence of such factors as reciprocal inhibition (see above).
2. H reflexes should be evoked with a sufficient level of stimulation to provide a corresponding M wave to help ensure stimulus constancy. Furthermore, a similar M-wave amplitude should be maintained and used for comparison across different conditions.
3. Maximal M waves used for normalization of the H reflex should be evoked in each condition where H reflexes are evoked to avoid time-dependent or movement-dependent changes. For example, the maximal M-wave amplitude should be determined before and after training and the control M-wave size should be used as a percentage of the pre- and post-training maximal M wave, respectively. If different conditions or postures are evaluated, a maximal M wave should be evoked under each condition to use in normalization.
4. The size of the reflex that is evoked must be the same relative size used in pre- and post-training measurements to account for non-linearities in the H-reflex recruitment curve. For example, with reference to the previous two points, the relative sizes of the M wave and H reflex may have changed due to the training regimen, and therefore one must control for changes in this relationship by using normalization values that correspond to the pre- or post-training interval. To control for this effect, it is strongly suggested that full M/H recruitment curves are obtained in all conditions.
5. The behavioral state as well as the posture of the subject must be the same when all measurements are taken to control for the task-dependency of reflex modulation. For example, if a well-lit room and a specialized apparatus for maintaining posture were used during measurement before training, these should be maintained for the post-training measurements. Moreover, if the attempt is to evaluate adaptive plasticity due to an exercise-training intervention, the H reflex should be sampled during the performance of the training exercise.
6. Stimulation to evoke the H reflex should be performed randomly and not more frequently than with a 3-s repeat to avoid post-activation depression.
7. When examining the conditioning effect of another input on the H reflex, randomly alternate the conditioned (C) and test (T) stimulation.

The main objective of this review was to outline the requirements for careful use of the H-reflex technique. If the conditions described above are met, the H reflex can certainly be used as another useful tool in evaluating the changes in human reflex pathways and the plasticity of the neuromuscular system. It is hoped that applied physiologists will integrate the methodological suggestions outlined here into future research to improve the accuracy and validity of interpretation of nervous system function and adaptability to training.

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