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Review article

Clinical uses of H reflexes of upper and lower limb muscles

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ABSTRACT

H reflexes can be recorded from virtually all muscles that have muscle spindles, but reflex reinforcement may be required for the reflex response to be demonstrable. This can allow conduction across proximal nerve segments and most nerve root segments commonly involved by pathology. Stimulus rate is critical in subjects who are at rest. However the reflex attenuation with higher rates is greatly reduced during a background contraction of the test muscle, with only minor changes in latency if any. In addition the contraction ensures that the reflex response occurs in the desired muscle. Reflex latencies should be corrected for height (or limb length) and age. Because the reflex discharge requires a synchronised volley in group Ia afferents, large increases in reflex latency occur rarely with purely sensory lesions. If the H reflex of soleus, quadriceps femoris or flexor carpi radialis is absent at rest but appears during a voluntary contraction at near-normal latency, there is either low central excitability or a predominantly sensory abnormality. With the former H reflexes will be difficult to elicit throughout the body. If H reflexes can be recorded at rest from muscles for which no reflex can normally be demonstrated, there is good evidence for hyperreflexia. In the context of possible ALS, this is an important finding when there is EMG evidence of chronic partial denervation in that muscle.

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The H reflex was first described in 1910 and 1918 by Paul Hoffmann (hence the name), its basis was investigated in a series of papers by Magladery and colleagues (e.g., Magladery and McDougal, 1950; Magladery et al., 1951), and it was introduced into motor control studies by Paillard in 1955. It has been the subject of numerous reviews since then (e.g., Hugon, 1973; Schiepatti, 1987; Burke et al., 1999; Pierrot-Deseilligny and Mazevet, 2000; Fisher, 1992, 2002; Zehr, 2002; Misiaszek, 2003; Knikou, 2008; Pierrot-Deseilligny and Burke, 2012).

The H reflex is perhaps one of the simplest reflexes that can be recorded in human subjects, and it has become a standard tool in motor control studies. However, the results of some of these studies do not survive critical analysis because they are based on overly simple views of the underlying mechanisms. This review will highlight some aspects of the underlying physiology and a number of misconceptions that are still taught and continue to plague the literature. In *motor control research*, the H reflex has been used too much without a full appreciation that it is actually not the simple "two-neurone reflex" envisaged by Magladery et al. (1951). On the other hand, in *clinical practice* the H reflex has been under-utilised, based on the misconception that it can only be recorded reliably for soleus.

Perhaps the major use for H reflex studies is that they allow the clinician to study conduction across proximal segments (i.e., plexuses and nerve roots) of the peripheral nerve and, in the author's experience, they do so better than alternative techniques. Somatosensory evoked potentials (SEPs) allow sensory axons to be studied. F waves and spinal/nerve root stimulation allow motor axons to be studied. These tests provide different information (purely sensory, purely motor, sensory and motor), and they are therefore not equally sensitive in different pathologies. Other considerations affect the choice of which test should be used in a given situation.

To obtain a reflex response, the afferent volley must be synchronised when it reaches the motoneuron pool, and it must produce a compound excitatory postsynaptic potential (EPSP) sufficient to discharge the motoneurons of lowest threshold. Pathological dispersion of the afferent volley will delay a reflex response and reduce its size. When extreme, dispersion could abolish the reflex response completely even if all afferent axons were capable of conducting. Loss of conducting axons without slowing conduction can also cause a small conduction delay because threshold for the motoneuron discharge is reached later on the compound EPSP. As a result, the reflex response is sensitive to subtle changes, and may be absent when there is no clinically detectable deficit.

1. Underlying physiology

1.1. The H reflex may depend on monosynaptic excitation but it is not an exclusively monosynaptic reflex

Group Ia afferents from the primary endings of muscle spindles form the afferent limb for the H reflex. Ia afferents have a strong monosynaptic excitatory connection with motoneurons of the homonymous pool, but there are also weaker monosynaptic projections to heteronymous (synergistic) pools and there are probably oligosynatic projections to homonymous and synergistic motoneurons (see Pierrot-Deseilligny and Burke, 2012).

It is not possible to stimulate Ia afferents in isolation. Group Ib afferents from Golgi tendon organs are of the same size and, even with weak stimuli, they will be activated equally effectively. Assuming that conduction across the Ib inhibitory interneuron (now termed the "non-reciprocal group I inhibitory interneuron") is intact, group Ib inhibition will begin in the motoneuron pool 0.5–1.0 ms after the onset of group Ia excitation, this delay being that required to cross an interneuron. This would not matter if the group Ia excitation was brief but, based on probable conduction velocities (Macefield et al., 1989; Pierrot-Deseilligny and Burke, 2012), the slowest group Ia afferents from soleus would reach soleus motoneurons 5–7.5 ms after the fastest (Pierrot-Deseilligny and Burke, 2012). There is therefore ample opportunity for Ib inhibition to truncate the Ia excitation and limit the size of the reflex response. There is now evidence that this does occur (see below).

That an inhibitory volley that reaches the motoneuron 1 ms after the onset of excitation will truncate the excitation is well established in the cat (Araki et al., 1960). There is evidence for this in human subjects. Although the Ia excitatory input lasts some 5–7.5 ms, the rising phase of the compound group I EPSP in human soleus motoneurons is remarkably brief: estimated to be only 1–2 ms (Birnbaum and Ashby, 1982; Burke et al., 1983, 1984). Group Ib inhibition due to medial gastrocnemius Ib afferents begins only 1 ms after the onset of monosynaptic excitation in soleus motoneurons (Pierrot-Deseilligny et al., 1981), so that the net effect on soleus motoneurons will be the sum of opposite Ia and Ib effects. It has been shown for quadriceps femoris that changes in transmission of the Ib component of the group I volley across the Ib inhibitory interneuron can change the size of the H reflex of quadriceps (Marchand-Pauvert et al., 2002). Accordingly, while the excitation underlying the H reflex may be largely monosynaptic, the reflex discharge will be determined by the balance between Ia excitation and Ib inhibition and will vary with the excitability of the Ib inhibitory interneuron.

1.2. The H reflex and the tendon jerk

The H reflex is generally considered the electrical equivalent of the tendon jerk, differing only in that the H reflex bypasses muscle spindle mechanisms. This belief has led to comparisons of the two reflexes as a means of assessing the sensitivity of muscle spindle endings and, by inference, the level of fusimotor drive. The assumption that the only significant difference between the reflexes involves muscle spindle mechanisms ignores other well-established differences (Table 1), and has led to many invalid conclusions in the literature (see Burke, 1983; Pierrot-Deseilligny and Burke, 2012).

The excitatory pathway underlying the H reflex and tendon jerk may be the same, but the impulse traffic over that pathway differs. However, this does not invalidate the usefulness of timed tendon jerk studies in clinical neurophysiological studies, particularly when studying muscle groups for which it is technically difficult to record the H reflex.

1.3. Factors limiting the size of the H reflex

Each Ia afferent has an excitatory projection to almost every motoneuron in the homonymous pool (Mendell and Henneman, 1971). However, it is impossible for the soleus H reflex to involve all motoneurons in the motoneuron pool (Fig. 1). There are many reasons for this (Table 2). First, although the mean threshold for group Ia afferents is much lower (~0.6×) than the threshold for the most excitable soleus motor axon (="MT"), the individual thresholds vary greatly, and it is not possible to recruit all Ia afferents unless the stimulus approaches 4× MT (Gracies et al., 1994). Second, pre-motoneuronal gating of the Ia afferent volley may preclude activation of some motoneurons. This can be due to classical "presynaptic inhibition" associated with depolarisation of the primary afferent terminals at $GABA_A$-mediated synapses or depletion of transmitter in Ia terminals when they are activated repetitively. The latter causes post-activation depression (also termed "homosynaptic" depression) of the H reflex. This phenomenon is important in clinical practice: it underlies the need for low