Journal of Clinical Monitoring and Computing (2006) 20: 267–304
DOI: 10.1007/s10877-006-9036-x


*Specialty Section on Surgical Neuromonitoring*

# INTRAOPERATIVE APPLICATIONS OF THE H-REFLEX AND F-RESPONSE: A TUTORIAL

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Leppanen RE. Intraoperative applications of the H-reflex and F-response: a tutorial.
J Clin Monit Comput 2006; 20: 267–304

**ABSTRACT.** Traditional intraoperative monitoring of spinal cord function involves the use of three techniques: 1. Orthodromic ascending somatosensory evoked potentials (SSEPs) and 2. antidromic descending neurogenic somatosensory evoked potentials (DNSSEPs) monitor long-tract sensory function. SSEPs and DNSSEPs do not monitor interneuronal gray matter function. 3. Transcranial motor evoked potentials (TMEPs) monitor descending long-tract motor function and measure interneuronal gray matter function by activating motor neurons. TMEPs activate from 4–5% of the motor neuron pool. When using TMEPs 95–96% of the motor spinal cord systems activating the motor neurons are not monitored. Our ability to interact with our environment involves not only intact sensation and strength, but also complex coordinated motor behavior. Complex coordinated motor behavior is controlled by groups of electrically-coupled spinal cord central pattern generators (CPGs). The components of CPGs are: descending and propriospinal systems, peripheral input, and segmental interneurons. The point-of-control is the level of excitation of interneurons, which is determined by the integrated activity of the other components. Spinal cord injury (SCI) changes segmental reflex gain by uncoupling these components. Changes in gain are detected by recordings from muscles. SSEPs, DNSSEPs and TMEPs provide limited information about the status of CPGs. H-reflexes measure the function of from 20–100% of the motor neuron pool. F-responses measure the function of from 1–5% of the motor neuron pool. H-reflexes and F-responses provide information about the degree of coupling between CPG components. Recording H-reflexes and F-responses together with SSEPs and TMEPs not only monitors spinal cord long-tract function, but also provides a multiple-systems approach that monitors those spinal cord systems that are responsible for the control of complex coordinated motor behavior. The objective of this paper is to describe how H-reflexes and F-responses can be used to monitor complex coordinated motor behavior.

**KEY WORDS.** H-reflex, F-response, electromyography, intraoperative monitoring, spinal cord, central pattern generator, spinal cord injury

## INTRODUCTION

There are 3 goals for intraoperative neurophysiological monitoring. The first is to reduce the risk of neurological complications by detecting insult to neuronal elements. The second is to provide a guide that may affect a surgeon's approach or actions, such as mapping the location of sensory and motor tracts within the spinal cord. The third is to perform studies with enough detail to help understand normal and pathophysiological function. The techniques used for spinal cord monitoring should accomplish these 3 goals.

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Received 19 March 2006. Accepted for publication 22 May 2006.

*Traditional studies*

Somatosensory evoked potentials (SSEPs) have been used to monitor spinal surgery since their intraoperative use was first reported in 1977 [1]. The recording of SSEPs reflects the integrity of spinal cord white matter. Lower-extremity SSEPs are mediated primarily through the dorsal columns [2–5] or dorsal spinocerebellar tracts [6, 7], but there have been reports of the ability to monitor the anterolateral columns and spino-olivo-cerebellar and spino-reticulo-cerebellar tracts [8–11]. Recording of SSEPs provides no information about the condition of the spinal cord gray matter [12] and monitors those systems of the spinal cord that mediate sensation.

Since the development of transcranial stimulation of the motor cortex [13], a variety of electrical [14, 15] and magnetic [16] stimulation techniques have been used intraoperatively. These motor techniques provide information about long-tract function, but also provide some information regarding segmental interneurons and anterior gray-matter function because successful transmission to the lower motor neuron depends upon the functional integrity of this segmental system [12]. Direct electrical stimulation of the exposed motor cortex activates only about 5% of the motor units innervating a target muscle. This degree of motor unit activation is the same as conventional transcranial electrical simulation in awake and relaxed subjects [17]. Intraoperative transcranial electrical stimulation activates from 3.0 to 4.0% of the total muscle fibers and from 3.1 to 3.9% of the motor units in the abductor hallucis muscle [18]. Motor spinal cord function has been monitored by recording action potentials directly from the corticospinal tracts [19, 20]. Two components are recorded following transcranial electrical motor stimulation. The shorter-latency component is the 'D Wave' which is generated by direct activation of the corticospinal neuron at the initial segment. The second component are the 'I Waves' which are generated by indirect activation of the corticospinal neurons by excitation by corticocortical interneurons [21]. Recording of motor evoked potentials (MEPs) monitors those systems of the spinal cord that are responsible for mediating the function of muscle strength.

Monitoring antidromic somatosensory spinal cord activity resulting from electrical spinal cord stimulation and recording of peripheral nerve activity has been reported. These peripherally-recorded descending potentials are called descending neurogenic evoked potentials (DNEPs) [22]. Initially, DNEPs were thought to be mediated by the long spinal cord motor tracts, but recent intraoperative collision studies in which spinal cord stimulation was delayed following unilateral stimulation of the tibial nerve at the ankle demonstrated that DNEPs and SSEPs are mediated through common spinal cord pathways. These studies demonstrated that in idiopathic scoliosis patients, DNEP activity represents antidromic spinal cord somatosensory activity [23–27]. There have been reports of postoperative motor deficits with intact DNEPs [28] and postoperative sensory deficits with normal motor function with absent DNEPs [27]. DNEPs are more sensitive for detecting spinal cord sensory compromise than are SSEPs [27]. The recording of DNEPs is the result of the activation of antidromic sensory spinal cord pathways with the peripherally-recorded DNEP nerve action potentials representing 20 percent of the total peripheral nerve fibers [27]. Since DNEPs represent antidromic somatosensory activity, a more accurate term to use is descending neurogenic somatosensory evoked potentials (DNSSEPs).

Intraoperative spinal cord mapping techniques have been used to identify the neurophysiological location of the dorsal median sulcus and the lateral descending motor spinal cord tracts during myelotomies for intramedullary spinal cord tumor resection. Direct dorsal column spinal cord electrical stimulation for dorsal median sulcus mapping with recording of antidromic descending somatosensory potentials from peripheral nerves has been used to localize the myelotomy site [29, 30]. In addition to activating sensory tracts antidromically, orthodromic SSEPs may be recorded with a miniature multi-electrode with 8 contacts placed over the dorsal spinal cord. The ascending tibial somatosensory potentials are recorded from these 8 contacts and help determine the functional midline corresponding to the dorsal median sulcus [31]. Direct spinal cord stimulation with recording of compound motor potentials has also been used to identify the location of the spinal cord descending motor tracts [29, 32].

*H-reflex and F-response studies*

Our ability to interact with our environment involves not only intact sensation and strength but also complex coordinated motor behavior. Intraoperative spinal cord monitoring should include techniques that monitor those spinal cord systems controlling sensation, strength and coordination. Since TMEPs activate only from 4–5% of the motor neuron pool [17], H-reflexes and F-responses can be used to monitor the function of a greater percentage of the output of the motor neuron pool. H-reflexes measure the function of from 20–100% of the motor neuron pool [33]. F-responses measure the function of from 1–5% of the motor neuron pool. [34]. These techniques may measure the function of the same, different or overlapping populations of the motor neuron pool [35, 36].

The recording of intraoperative reflex and F-response activity can be used to monitor those systems of the spinal

cord that are responsible for the control of complex motor behavior [37–41]. Reflex and F-response techniques monitor complex spinal cord function and monitor activity in highly-integrated ascending, descending and spinal interneurons. These techniques also monitor function of the dorsal and ventral nerve roots.

The advantage of the use of intraoperative H-reflex and F-response recordings to detect the onset of spinal cord or nerve root compromise is that the recordings are single sweep. These recordings are therefore real-time and there is no delay after the onset of spinal cord or nerve root compromise that is present when using averaged evoked potentials. H-reflex and F-response recordings provide the surgeon with immediate feedback about spinal cord or nerve root function. They can be acquired continuously throughout surgery with little or no noticeable patient movement. H-reflexes and F-responses may be monitored in patients in which SSEPs and MEPs cannot be recorded because of a pre-existing neurological deficit. Reflex changes are very sensitive to spinal cord compromise, with reflex changes occurring before or without SSEP changes [37]. Recording H-reflexes and F-responses together with SSEPs and TMEPs not only monitors spinal cord long-tract function, but provides a multiple-systems approach that monitors those spinal cord systems that are responsible for the control of complex coordinated motor behavior. Intraoperative reflex and F-response studies also provide a model for understanding the mechanisms of normal and pathological spinal cord function.

## MECHANISMS OF SPINAL CORD NORMAL AND PATHOPHYSIOLOGICAL FUNCTION

One approach to understanding complex motor behavior is to consider spinal cord integrating function to be controlled by a system of tightly electrically-coupled CPGs. The integrated activity of these spinal cord CPGs is responsible for controlling the stepping mechanism of gait and the coordination of upper- and lower-extremity function [42–46]. Spinal cord CPGs may be thought of as having four components. These components are the segmental interneurons, the descending suprasegmental systems, the propriospinal systems and the peripheral afferent input. The point of control is the level of excitation of interneurons, which is determined by the integrated summated synaptic excitatory and inhibitory effect of the other components on the interneurons and motor neurons. The level of excitation of interneurons determines the level of reflex gain. Sensory afferent and antidromic F-response signals following peripheral nerve stimulation provide the time-locked synchronization of the system. Summated activity from descending spinal cord systems, especially the corticospinal, rubrospinal, vestibulospinal and reticulospinal systems, contribute to controlling the gain set by the interneurons. Vestibulospinal and reticulospinal tracts control proximal function, and rubrospinal and corticospinal tracts control distal lower-extremity function [47]. The gain is also controlled by short, intermediate and long propriospinal systems that control processing at multiple spinal cord levels ipsilaterally and contralaterally. Interaction between the cervical and lumbosacral networks is mediated by propriospinal neurons [44]. The output from the system is through the motor neurons, which is measured by reflex and F-response recordings from muscle. Intraoperative reflex and F-response recordings provide information about the degree of coupling between CPGs. Acute or chronic damage to the peripheral afferent input, the descending suprasegmental systems, the propriospinal systems or the segmental interneurons results in the uncoupling of these components. This changes the level of excitability of the segmental interneurons, which results in a change in the segmental reflex gain. The change in the reflex gain can be detected by changes in reflex processing and F-responses recorded from muscles. Changes in reflex and F-response processing can be used as a monitoring technique to detect acute and chronic compromise of the spinal cord suprasegmentally and segmentally, and of the nerve roots (Figure 1).

Reflex processing can be considered relatively simple. For example, the monosynaptic or oligosynaptic H-reflex involves processing at a single segment of the spinal cord. Additionally, it also involves complex polysynaptic reflexes which involve processing at multiple spinal cord levels. Monosynaptic reflex muscle recordings are of short latency, short duration, simple configuration and high amplitude. These parameters are stable and vary little from one stimulus to the next. Polysynaptic recordings are of longer latency, longer duration, complex configuration and low amplitude. Polysynaptic recordings are not stable and vary from one stimulus to the next [48].

Acute spinal cord transection causes spinal shock that is characterized by complete paralysis, hyporeflexia, loss of sensation and muscle hypotonia caudal to the lesion. SCI disrupts or disinhibits the suprasegmental influence over segmental interneurons mediating pre-synaptic inhibition. The hyporeflexia associated with spinal shock may be due to an increase in the efficacy of pre-synaptic inhibition [49]. Over time pre-synaptic activity decreases, resulting in enhanced spinal reflexes [50]. Observations in cats indicate that rostral acute SCI causes post-synaptic caudal lumbar motor neuron changes. In cats, rostral acute SCI causes hyperpolarization of caudal lumbar segment motor neurons [51–54]. Immediately following rostral spinal cord transection, the monosynaptic reflexes from the medial and lateral

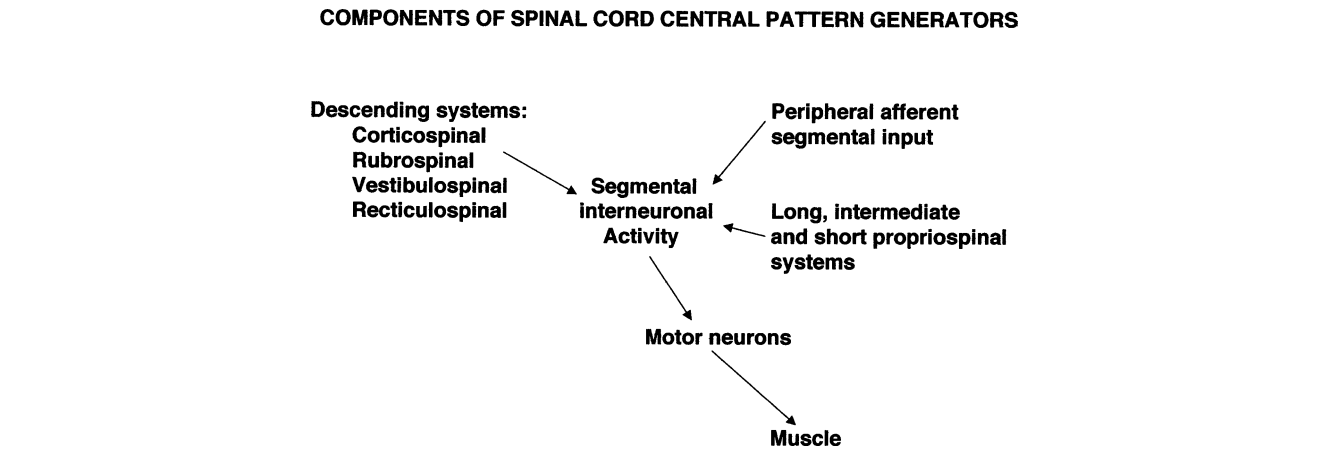


*Fig. 1. Components of the spinal cord central pattern generators. The point-of-control is the level of excitation of segmental interneurons, which is determined by the integrated activity of the other components. H-reflexes and F-responses measure the output through the anterior horn cells (motor neurons).*

gastrocnemius, soleus, posterior biceps and semitendinous muscles are reduced in amplitude or completely absent. During the 6 h following transection there is some recovery of reflex activity [57].

When using cold as a model for acute reversible spinal cord transection, hyperpolarization of caudal motor neurons occurs within 30 s and monosynaptic reflex amplitudes are gradually decreased. These changes persist during the application of cold. Rewarming restores reflex amplitudes to original values in 30 s, and the resting motor neuron membrane potential back to the original value in 1 min [51–53].

The hyperpolarization of motor neurons following spinal cord transection or cooling is thought to be secondary to decreased suprasegmental facilitation of motor neurons, which usually keeps them in a slightly hypopolarized state [51–54]. Fusimotor drive is also depressed early after acute spinal cord injury, for gamma motor neurons are also hyperpolarized [55]. H-reflexes and F-responses reflect the level of excitation of a large percentage of the motor neuron pool in the spinal cord gray matter [33, 34]. These recordings may be very helpful in detecting intraoperative spinal cord ischemic insults, since the level of gray matter excitability is depressed more with ischemia than is posterior column function. In animals the dorsal horn potential that is generated by post-synaptic gray matter activity disappears within 3 to 5 min after cessation of spinal cord perfusion. Posterior column potentials persist for 12 to 15 min [56].

Changes in spinal cord electrophysiological signal processing can be used to help understand the electrophysiological mechanisms occurring during acute SCI [57]. Changes in serial, parallel and oscillatory processing, hyperpolarization, inhibition and disinhibition may be observed. In dogs, the intensity of ultrasound energy needed to change spinal cord function was determined [57]. The dura was exposed from T11 to L3. Progressively-higher levels of ultrasonic energy were delivered to the spinal cord by an ultrasonic aspirator bathed in irrigation fluid suspended 1 cm above but not touching the spinal cord. The ultrasonic energy was delivered between the T11 and L3 vertebrae. Following ankle tibial nerve stimulation, averaged SSEPs were recorded from the exposed sciatic nerve in the thigh, the T11 and L3 dura and the scalp.

In baseline recordings short-latency N1 and 3 late components were recorded from the sciatic nerve. The segmental N2 component representing the level of excitability of post-synaptic segmental gray matter [58] was recorded from the L3 dura. Multiple conducted components were recorded over the T11 dura, representing conduction in progressively-slower conducting ascending parallel spinal cord systems (dorsal spinocerebellar, dorsal column and anterior and lateral spinothalamic tracts) [59, 60]. The N25/P30 components representing activation of neurons in cerebral cortical areas 3a, 3b, 1, 2, 4 [61] were recorded from the scalp (Figure 2). Ultrasonic energy was applied for 1 min at each intensity level. Recordings were made before and after each more-intense application of ultrasonic energy.

Following application of ultrasonic energy in the continuous mode at the 75% intensity level, SCI occurred which was detected by the SSEP recordings (Figure 3). The L3 N2 component amplitude was decreased, the scalp N25/P30 component was absent and synchronized oscillatory activity was present cortically. The L3 N2 amplitude decreased because of suppression of caudal spinal cord gray matter,

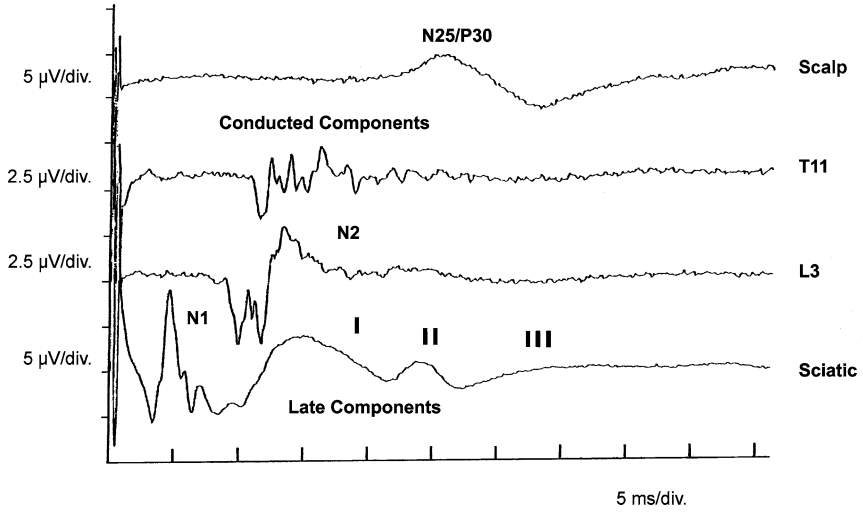


*Fig. 2. Baseline tibial somatosensory components recorded from the sciatic nerve, L3 and T11 dura, and from the scalp.*

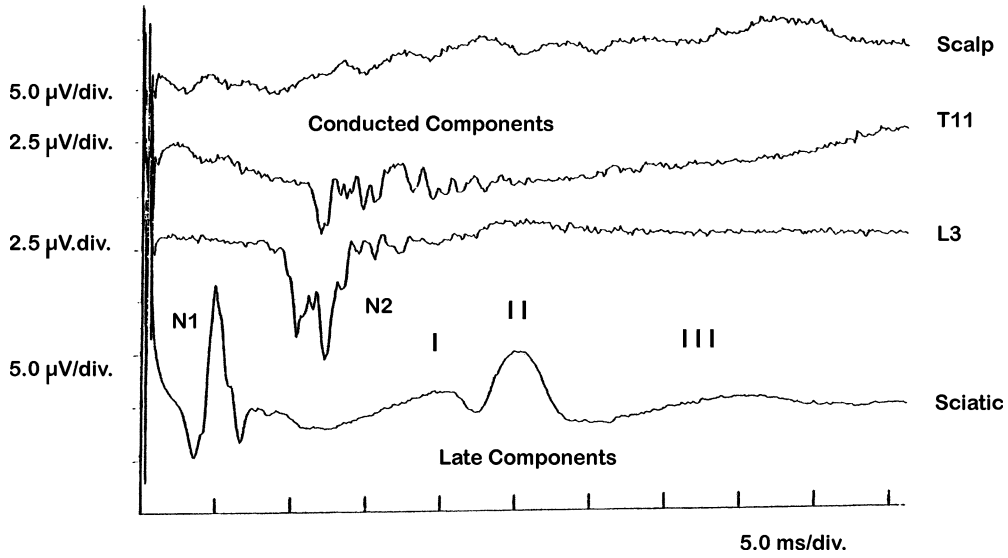


*Fig. 3. Following application of ultrasonic energy in the continuous mode at the 75% intensity level, the L3 N2 amplitude decreased; the scalp N25/P30 component was absent; and synchronized oscillatory activity was present cortically. The first late-sciatic component amplitude decreased, the second increased, and the third late and the N1 components were unchanged.*

either through stimulation of descending spinal cord inhibitory systems or because of interruption of descending spinal cord excitatory systems. This resulted in disinhibition, resulting in increased pre-synaptic inhibition and hyperpolarization of interneurons. The scalp N25/P30 component was absent because of injury to ascending sensory tracts affecting serial processing. Scalp oscillatory activity was present because the cortical neurons were oscillating at their inherent oscillatory frequency. Changes in the amplitude of the first 2 late-sciatic components occurred in parallel, with the suppression of the spinal cord segmental L3 N2 gray matter function. The first late component amplitude decreased and the second increased. No changes occurred in the third late component. No changes occurred in the short-latency N1 sciatic component which represents activity in the primary afferent neurons.

Two minutes after these changes occurred, the L3 N2 amplitude increased above baseline, the scalp activity was absent and the T11 conducted latencies were prolonged. The first late-sciatic component amplitude increased and the second decreased, in parallel with the increase in amplitude of the L3 N2 component. No changes occurred

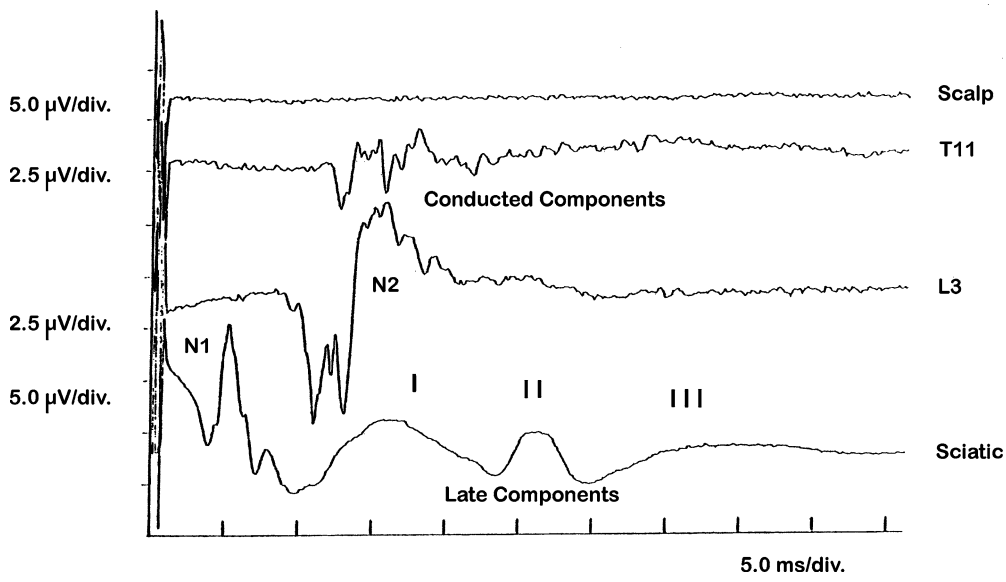


*Fig. 4. The L3 N2 amplitude increased to greater than baseline, the scalp activity is absent, and the T11 conducted latencies are increased. The first late-sciatic component amplitude is increased, and the second decreased. The third late and the N1 components were unchanged.*

in the third late component. No changes occurred in the short-latency N1 sciatic recording. The L3 N2 amplitude increased either because of compromise of a descending spinal cord inhibitory system, resulting in disinhibition and excitation of caudal gray matter function, or because of stimulation of some descending spinal cord excitatory system. The T11 conducted component latencies increased because of additional injury to ascending sensory tracts affecting parallel processing (Figure 4).

The spinal cord was then totally transected between the T11 and L3 levels. The L3 N2 amplitude was further increased and the T11 conducted components were absent. Oscillatory activity was present in the L3, T11 and scalp recordings. The amplitude of the last 2 late-sciatic components increased in parallel with the increase in amplitude of the L3 N2 component. No changes occurred in the first late or N1 components. Oscillatory activity was present cortically and in the L3 and T11 recordings because the spinal cord central pattern generator components were uncoupled and oscillating at their inherent oscillatory frequencies, and the cortical neurons were oscillating at their inherent oscillatory frequencies. The L3 N2 amplitude increased because of further disinhibition, resulting in increased interneuronal excitation (Figure 5).

These recordings in dogs demonstrate that acute SCI causes changes in the different types of spinal cord signal processing. They help in the understanding of the electrophysiological mechanisms occurring during acute SCI. With partial acute spinal cord injury there is inhibition and decreased excitability of caudal interneuronal activity. In a few minutes this may then be followed by disinhibition and increased excitability of caudal interneuronal activity. With total transection of the spinal cord, there is further immediate disinhibition and further increased excitability of caudal interneuronal activity. These caudal changes occur because of injury to descending spinal cord systems that have control over the level of excitability of caudal segmental interneurons. Changes in the late-sciatic components occur in parallel to the caudal interneuronal changes and they reflect the level of gray matter excitability. They demonstrate that late peripheral nerve recordings can be used to monitor the level of gray matter excitability, which controls the level of reflex gain. These peripheral recordings can be used to detect suprasegmental acute injury to the spinal cord.

## ELECTROPHYSIOLOGY OF H-REFLEXES AND F-RESPONSES

Following electrical stimulation of a mixed sensory–motor peripheral nerve the compound motor action potential (CMAP) or M-wave is recorded from the peripherally innervated muscle. The M-wave is the result of orthodromic motor conduction from the point of stimulation to the muscle. In addition to the short latency M-wave three late responses can be recorded: H-reflex, F-response and A-wave (axon reflex) [62]. Intraoperatively the gastrocnemius and flexor carpi radialis H-reflexes are most commonly used. The abductor digiti minimi, abductor pollicis

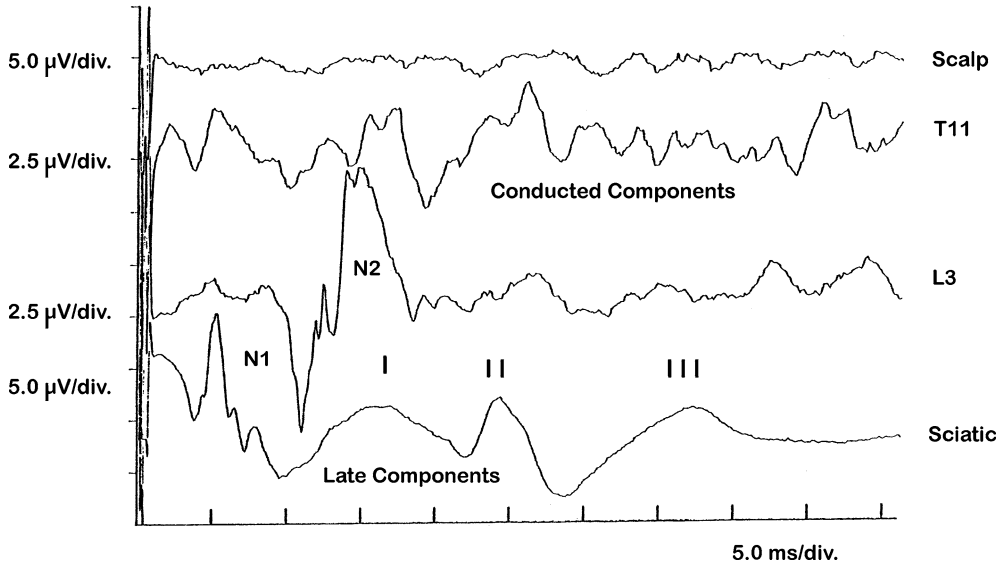


*Fig. 5. The L3 N2 amplitude further increased, the T11 conducted components were absent, and oscillatory activity was present in the L3, T11 and scalp recordings. The amplitude of the last 2 late-sciatic components increased and no changes occurred in the first late or N1 components.*

brevis, tibialis anterior, gastrocnemius and abductor hallucis F-responses are used most often intraoperatively.

## H-REFLEX

### *Background*

The H-reflex is believed to be a CMAP recorded from muscle following electrical afferent activation of a monosynaptic reflex. The afferent pathway involves electrical activation of the large 1a nerve fibers originating from muscle. After entering the dorsal horn of the spinal cord the 1a fibers synapse with the motor neurons. The efferent pathway involves orthodromic motor conduction through motor fibers in the same homologous spinal segment as the afferent pathway (62). In normal newborns H-reflexes may be recorded from many widely distributed muscles. After 2 years of age they are only present in the gastrocnemius, soleus and flexor carpi radialis muscles. The more restricted distribution of H-reflexes in adults reflects refinement of motor neuron pool activation with central nervous system maturation. In the adult they are also frequently found in the quadriceps and plantar foot muscles [63–65].

The H-reflex was first described by Hoffman in 1918 [66] and characterized more in the 1950s [67]. The reflex is most easily recorded from the gastrocnemius muscle following stimulation of the tibial nerve in the popliteal fossa. The fast conducting, low threshold 1a fibers are activated with long duration (1 ms) low intensity stimulation. The stimulation rate is 0.5 Hz. The intensity of stimulation is slowly increased. Low intensity stimulation activates the 1a fibers before the motor fibers so at low intensity of stimulation the H-reflex appears before the M-wave. The H-reflex CMAP is usually of a biphasic or triphasic configuration (Figure 6a and b). The 1a fibers are activated first either because they have a lower threshold than motor fibers to long duration stimulation and/or because they are located anatomically more superficial than the motor fibers in the popliteal fossa [68].

As the intensity of stimulation is increased a greater percentage of the motor neuron pool is activated and the H-reflex amplitude increases [62]. In the awake human when recording the gastrocnemius H-reflex the percentage of the motor neuron pool activated averaged 50% (range: 24.0–100%) [33]. The motor neurons recruited with increasing intensity of stimulation obey the size principle. Low-force motor neurons are recruited with low intensity of stimulation and high-force motor neurons are recruited with higher intensity of stimulation [36].

The H-reflex amplitude usually peaks at or just before the M-wave becomes present. Further increases in stimulation intensity result in a steady increase in the M-wave amplitude. When the M-wave no longer increases in amplitude the H-reflex is usually replaced by the F-response. The F-response is not a reflex. Following supramaximal stimulation of a mixed nerve antidromic motor nerve impulses

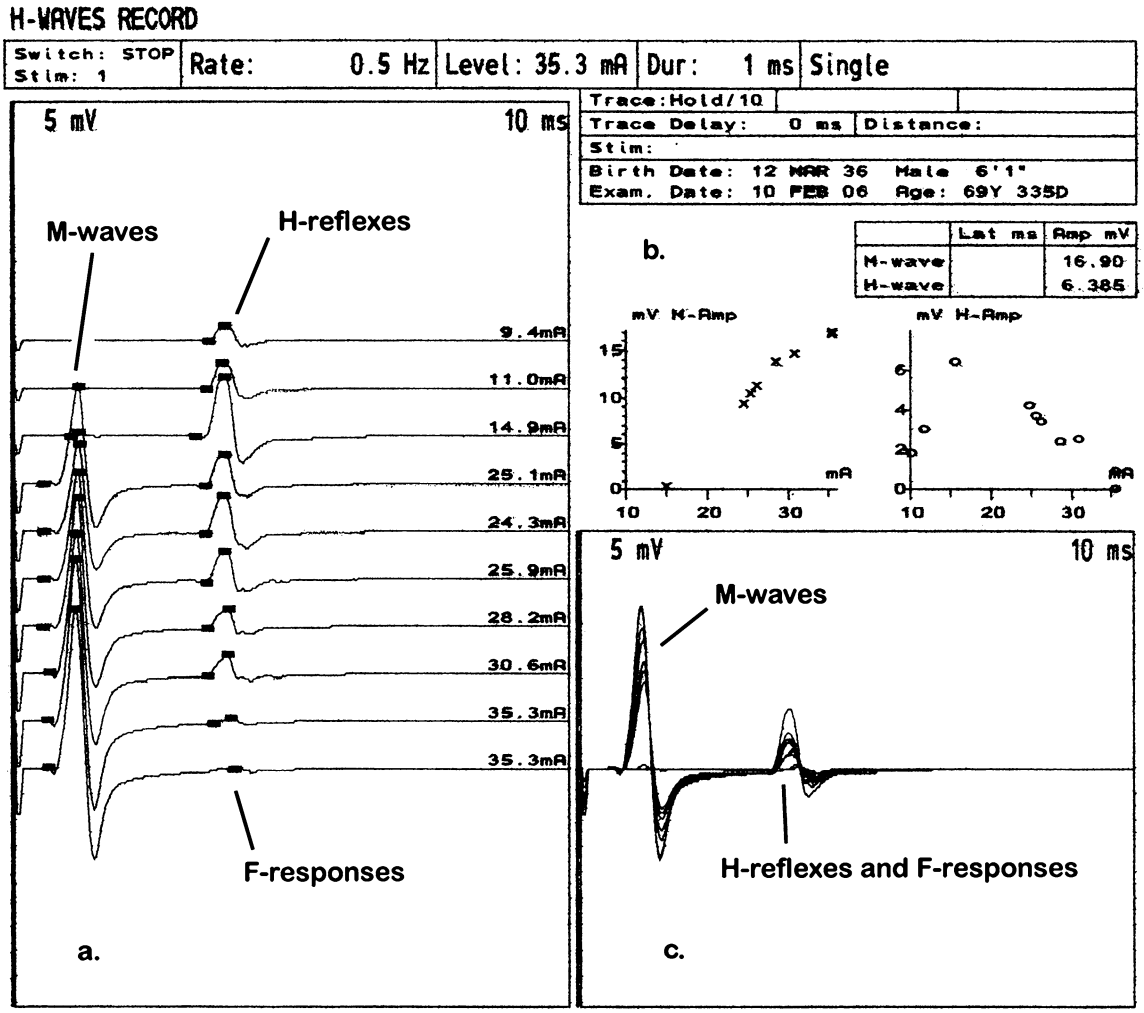


*Fig. 6. Gastrocnemius H-reflex. a and b: At low-intensity stimulation the H-reflex appears first and the amplitude peaks when the M-wave appears. Higher-intensity stimulation results in the M-wave amplitude increasing and the H-reflex is replaced by the F-response. c: The H-reflex is reproducible and of short latency, short duration and simple configuration.*

are conducted proximally to the ventral horn where they activate from 1 to 5% of the motor neurons. This is followed by orthodromic conduction through motor fibers and the F-response is recorded from muscle [69] (Figure 6a and b). Maximal stimulation of a motor nerve innervating a muscle results in the recording of the CMAP which is the result of activation of all the motor units in that muscle. A maximal stimulation intensity is achieved when the CMAP amplitude no longer increases with increased stimulus intensity. Supramaximal intensity is achieved when the stimulation intensity is increased to 25% above the maximal intensity [63]. When the stimulus intensity is held constant from one stimulus to the next, the H-reflex is of short latency, short duration, simple configuration and constant amplitude [62] (Figure 6c). To determine if a CMAP is a H-reflex the amplitude should exceed the M-wave amplitude and the configuration and latency should be the same from one stimulus to the next [62]. Changes in the effect of the central facilitation and inhibition on the motor neurons may change the pattern of how the H-reflex is activated. With increased presynaptic inhibition and hyperpolarization of motor neurons the M-wave may be present before the H-reflex and the H-reflex amplitude may be smaller than the M-wave amplitude. With decreased presynaptic inhibition the H-reflex may be robust and it may not be possible to suppress and replace it with the F-response.

The H-reflex has been thought of as a monosynaptic reflex. The central conduction time between dorsal and ventral roots reveals only enough time for one synapse, i.e., between 0.5 to 1.0 ms (62). There is also evidence

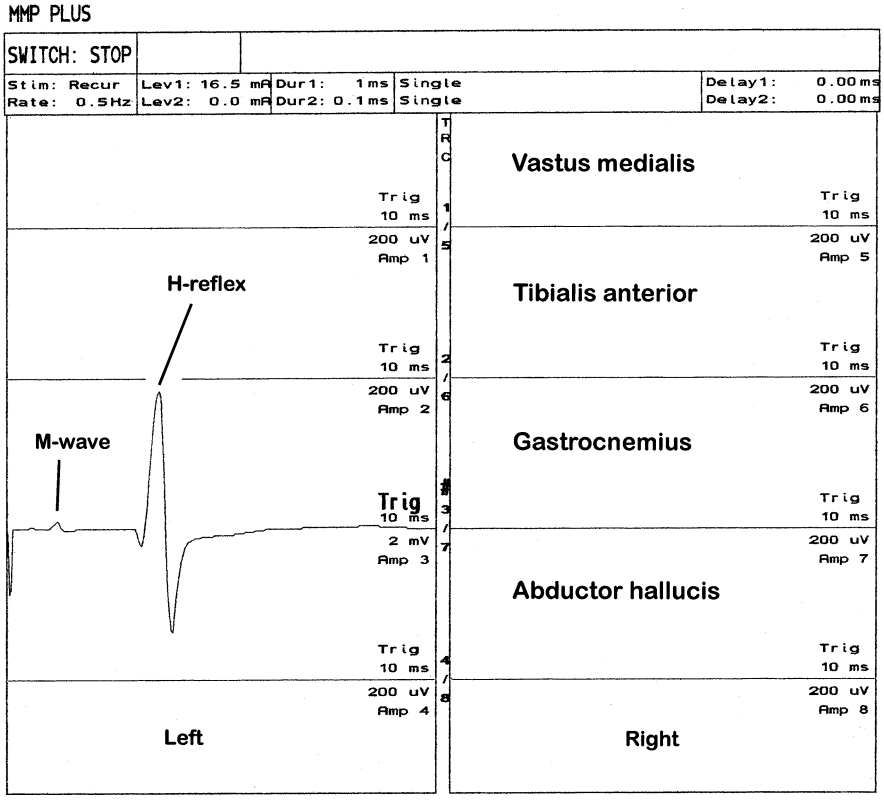


*Fig. 7. Intraoperative left gastrocnemius homonymous monosynaptic H-reflex. The left tibial nerve was stimulated in the popliteal fossa in a 13-year-old female with neuromuscular scoliosis.*

to indicate that the H-reflex is an oligosynaptic reflex. Low threshold motor neurons may be activated by a single (monosynaptic) synapse through the fastest 1a afferents. Higher threshold motor neurons may be activated through several (oligosynaptic) synapses by the fastest 1a afferents and through single synapses by slower 1a afferents [62].

There are two mechanisms that are thought to be responsible for the reduction in H-reflex amplitude and the replacement of the H-reflex with the F-response. The first is that supramaximal peripheral stimulation of a mixed sensory-motor nerve results in the antidromic motor and orthodromic 1a sensory signals reaching the dorsal and ventral roots at the same time. The antidromic motor action potentials invade the motor neurons and depolarize them. After traversing the reflex arc the 1a sensory fiber action potentials are not able activate the motor neurons because they are still in a depolarized state. The second is that a recurrent collateral arises from the motor neuron just distal to the axon hillock. Conduction through these collaterals activates the Renshaw cells in the gray matter. As an antidromic impulse traverses the axon toward the ventral horn the axon collateral is activated. Renshaw cells generate inhibitory postsynaptic potentials that inhibit the motor neurons. This also decreases the ability of 1a activity to activate motor neurons [62].

In the lower extremity of man there are two types of prewired monosynaptic 1a H-reflex connections. These are homonymous (homosynaptic) and heteronymous (heterosynaptic) H-reflexes. They are both part of the functional synergistic spinal cord CPGs. Both types may be recorded in the operating room. Homonymous monosynaptic H-reflexes are H-reflexes that are recorded from muscles that are innervated by the same nerve root as the 1a activated sensory fibers. The gastrocnemius H-reflex is an example of a homonymous monosynaptic H-reflex (Figure 7). 1a sensory action potentials may also make monosynaptic connections with motor neurons at spinal cord levels other than the 1a sensory segmental level. As a result of this activation H-reflexes may be recorded from muscles having segmental innervation other than the 1a segmental afferent activation. These H-reflexes are called heteronymous monosynaptic H-reflexes (Figure 8).

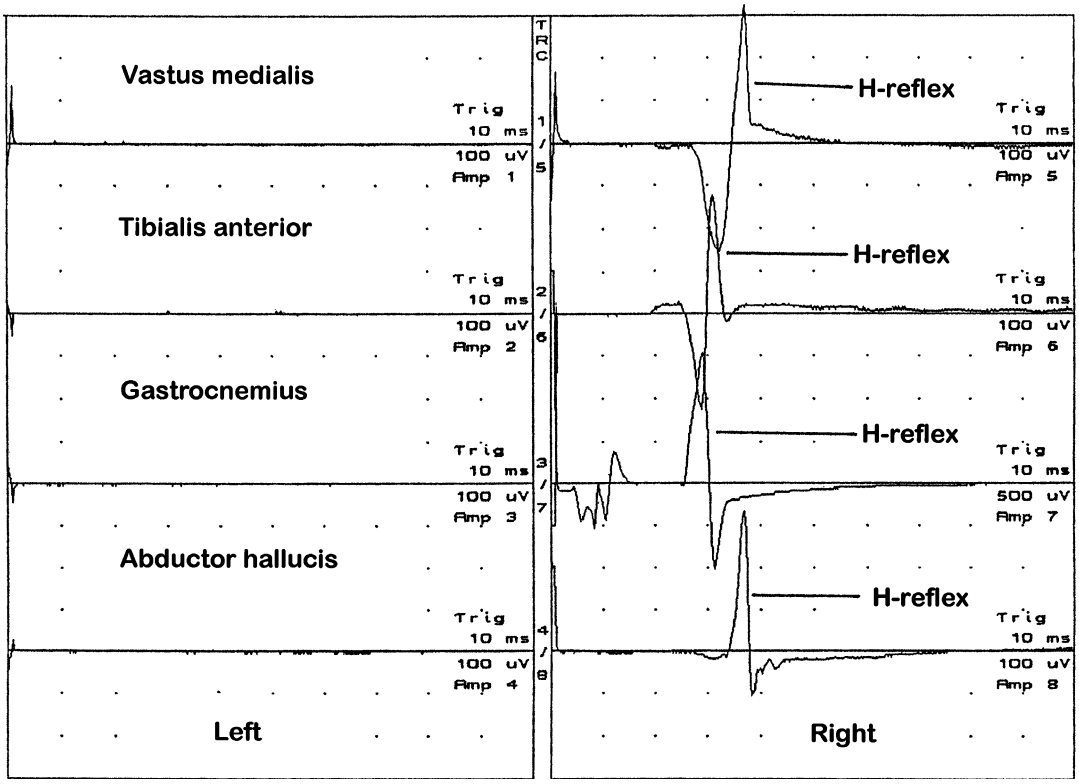


*Fig. 8. Baseline intraoperative H-reflexes. In a 12-year-old female with idiopathic scoliosis the right tibial nerve was stimulated in the popliteal fossa. The monosynaptic homonymous H-reflex is present in the gastrocnemius muscle, and heteronymous H-reflexes are present in the vastus medialis, tibialis anterior and abductor hallucis muscles.*

In man heteronymous connections normally exist between ankle and knee muscles. Functionally heteronymous connections provide coupling between muscles operating at different joints. These transjoint monosynaptic connections are important for maintaining equilibrium during bipedal stance and during gait. Intraoperatively heteronymous H-reflexes are inhibited by presynaptic inhibition. When presynaptic inhibition is decreased heteronymous H-reflexes may become present [70].

Heteronymous monosynaptic connections have been studied in the awake human. Examples of heteronymous connections are that when electrically stimulating the tibial nerve innervation of the soleus muscle heteronymous H-reflexes may be recorded from the gastrocnemius, peroneus brevis, quadriceps, biceps femoris and semitendinous muscles. When stimulating the femoral nerve innervation of the quadriceps muscle heteronymous H-reflexes may be recorded from the soleus, gastrocnemius, peroneus brevis and tibialis anterior muscles. Heteronymous connections may be bidirectional [70]. Stimulating the femoral innervation of the quadriceps muscle results in recording heteronymous H-reflexes from the gastrocnemius muscle, and stimulating the tibial innervation of the gastrocnemius muscle results in recording the heteronymous H-reflex from the quadriceps muscle. Heteronymous connections may be unidirectional. Stimulating the femoral innervation of the quadriceps muscle results in recording the heteronymous H-reflex from the tibialis anterior muscle but when stimulating the peroneal innervation of the tibialis anterior muscle the heteronymous H-reflex cannot be recorded from the quadriceps muscle [70].

The presence of H-reflexes in muscles where they are not usually recorded can be an indication of a suprasegmental central nervous system lesion that results in a decreased effect of presynaptic inhibition on motor neurons. The abnormal distribution of H-reflexes in the adult such as in the tibialis anterior and intrinsic hand muscles may indicate a disordered central motor system state. These changes occur because of uncoupling of the different components of CPGs [71–74]. An example of this uncoupling is a 55 year-old female who had intermittent numbness and tingling in both hands and left neck and right shoulder pain. Upper-extremity nerve conduction studies

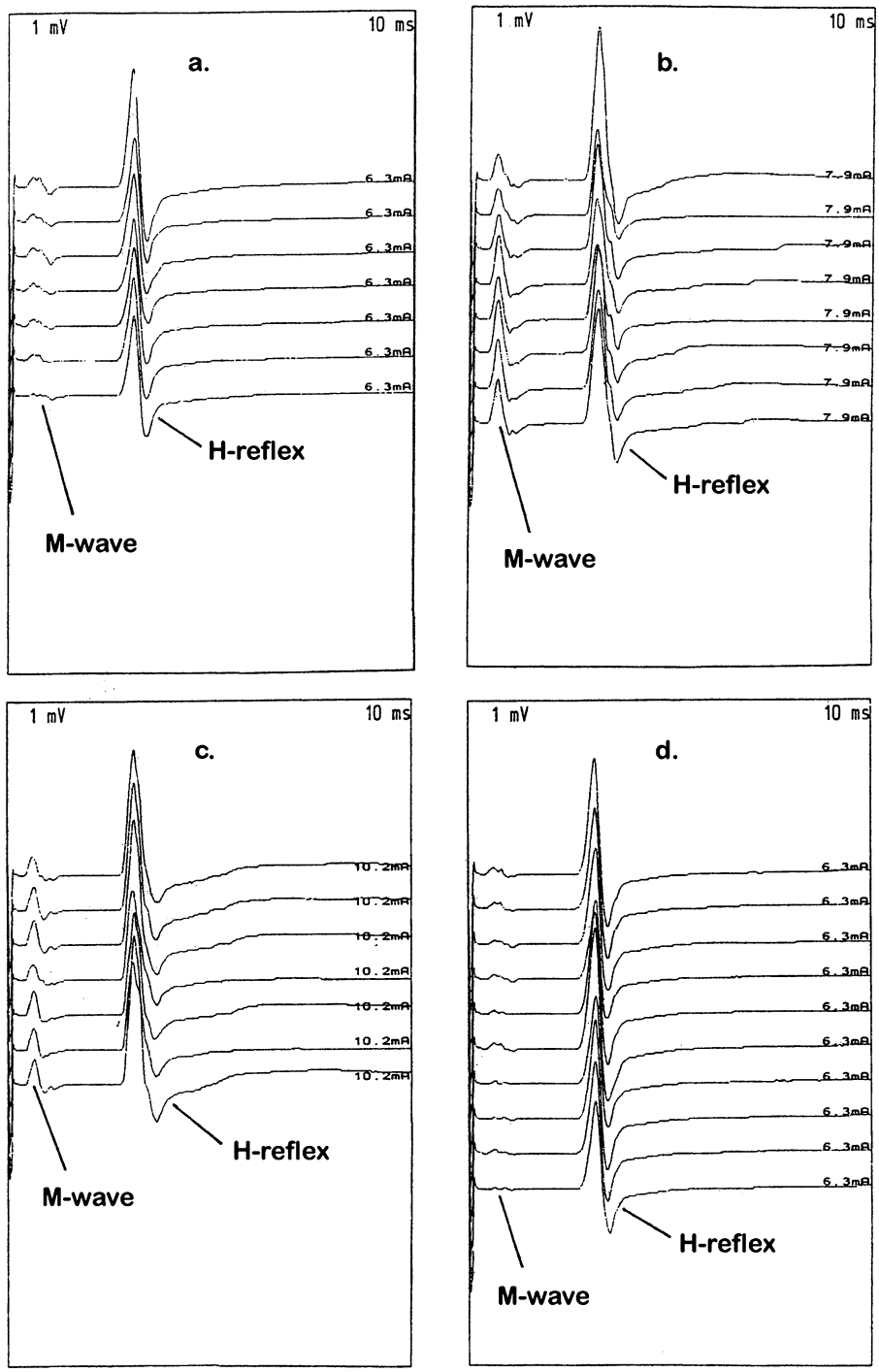


*Fig. 9. H-reflexes are present in the abductor pollicis brevis (a: left, b: right) and abductor digiti minimi (c: left, d: right) muscles following median and ulnar nerve wrist stimulation. Subsequent imaging studies revealed flattening of the spinal cord with myelomalacia at C4-C5. The patient was referred to neurosurgery.*

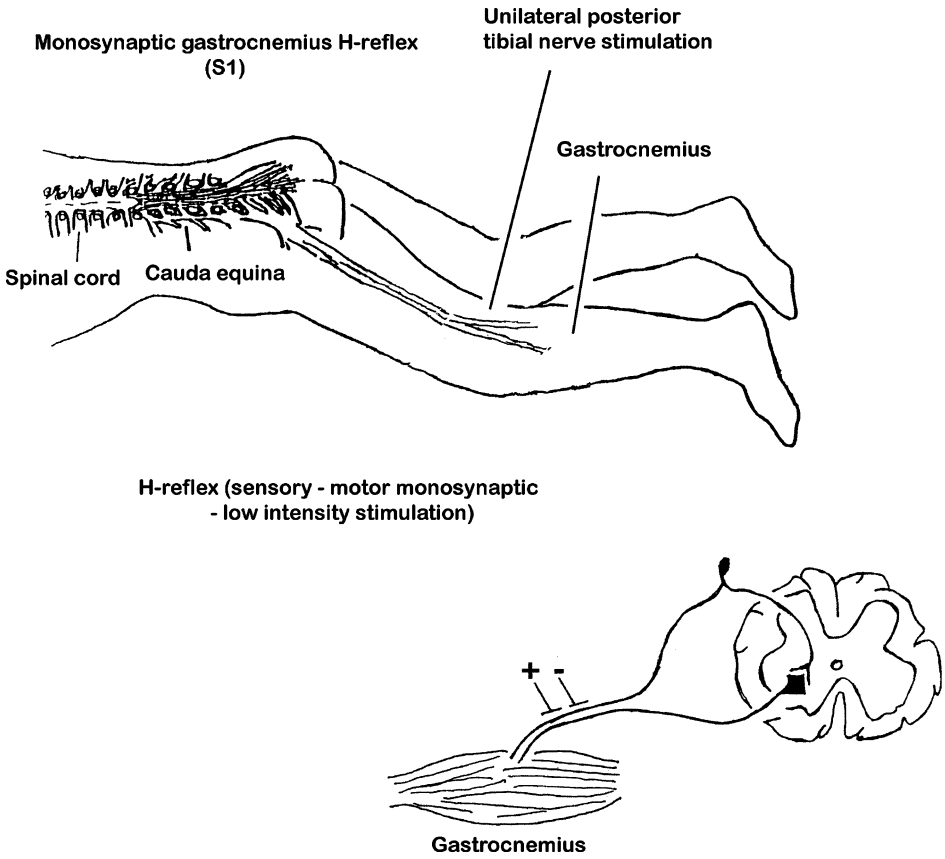


*Fig. 10. Technique for recording the gastrocnemius H-reflex. The tibial nerve is stimulated in the popliteal fossa and the gastrocnemius M-wave and H-reflex are recorded. This is mediated at the S1 level.*

and electromyographic studies were normal. An abnormal finding was that H-reflexes were present bilaterally in the abductor pollicis brevis and abductor digiti minimi muscles following wrist stimulation of the median and ulnar nerves. This suggested the presence of a suprasegmental lesion that caused uncoupling of cervical motor neurons. Imaging studies showed a flattening of the cervical spinal cord and magnetic imaging signal changes consistent with myelomalasia at C4–C5 [74] (Figure 9).

In addition to 1a afferents having connections directly with the motor neuron pool that results in recording homonymous and heteronymous H-reflexes a portion of 1a activity ascends through the dorsal spinocerebellar tract to synapse in the cerebellum and reticular formation. From these structures descending fibers through the vestibulospinal tract facilitate the spinal motor neuron pool. A dual stimulation technique in awake humans with varying the time interval between a conditioning and test stimulus was used to identify the presence of this long latency loop. The H-reflex was facilitated with a latency of between 150–200 ms [75].

## *Gastrocnemius H-reflex*

### *Gastrocnemius H-reflex normal parameters*

In the lower extremity the H-reflex can be recorded from the gastrocnemius and soleus muscle following electrical stimulation of the posterior tibial nerve in the popliteal fossa [76]. This reflex is mediated by segmental S1 afferent and efferent activity [71]. Intraoperative normal parameters have not been established. Normal parameters established for clinical studies may serve as a guide for intraoperative studies. Clinically the gastrocnemius H-reflex latency varies with age and leg length and has a mean latency of 28.9 $\pm$ 2.7 ms in awake man. A regression equation may be used to calculate the expected latency for each individual: H-reflex (ms) = 9.14 + 0.46 (leg length in cm) + 0.1 (age in years). A normogram based upon this equation is available as a reference. [77]. Clinically the normal side-to-side amplitude difference between the ages of 21 and 67 years of age may reach 60% [78]. The upper limits of normal clinical side-to-side latency difference is 1.5 ms [63]. When measuring

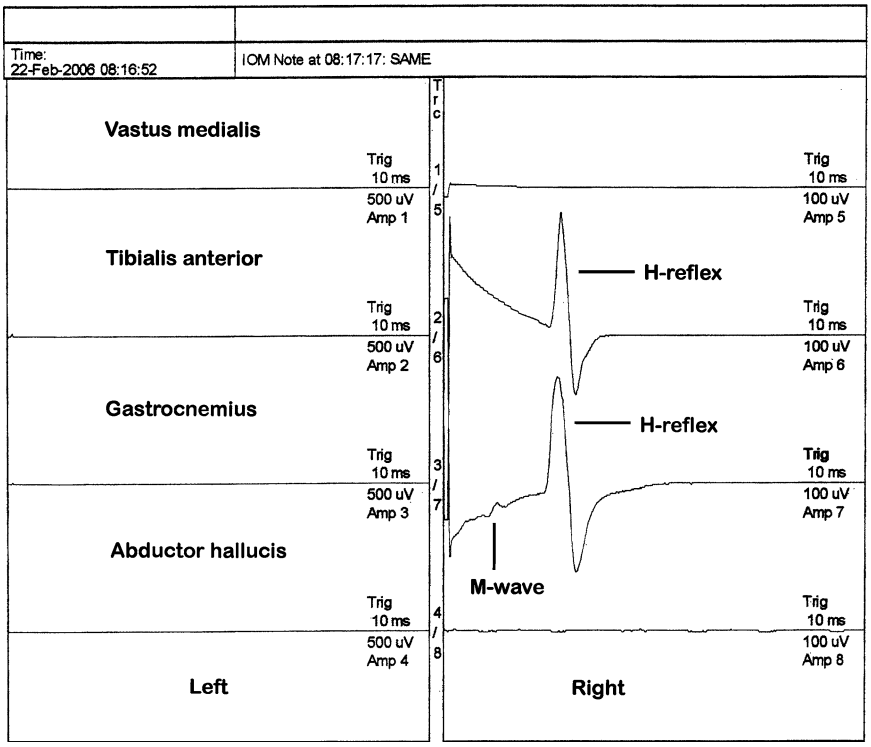


*Fig. 11. These are intraoperative baseline right homonymous gastrocnemius and heteronymous tibialis anterior monosynaptic H-reflexes recorded in a 14-year-old female with idiopathic scoliosis. The right tibial nerve is stimulated in the popliteal fossa.*

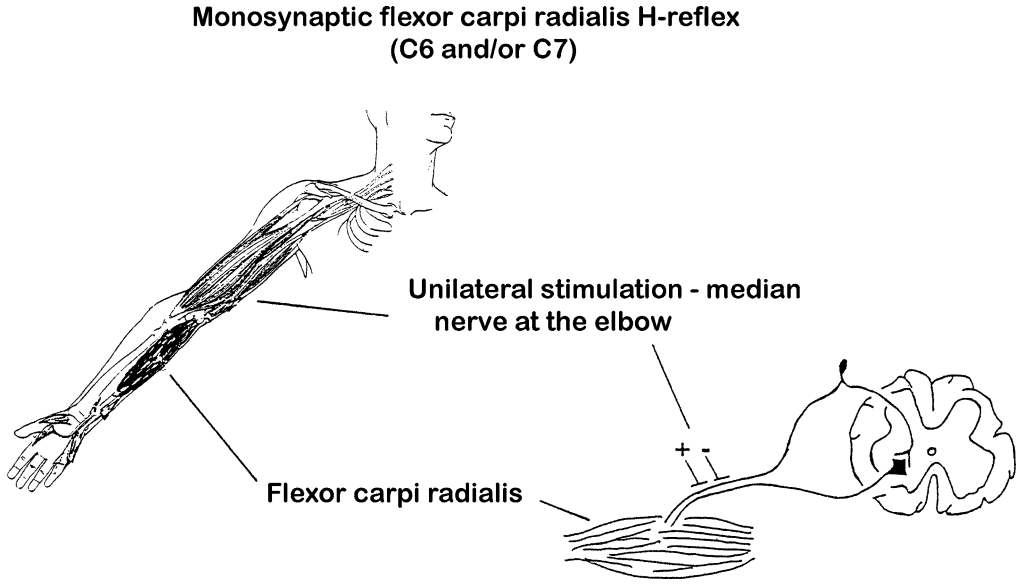


*Fig. 12. The flexor carpi radialis H-reflex is recorded by stimulating the median nerve over the distal medial upper arm, or over the anterior medial elbow, and recording the M-wave and H-reflex compound muscle action potential (CMAP). This is a sensory-motor monosynaptic reflex that is mediated at the C6 and/or C7 segmental level.*

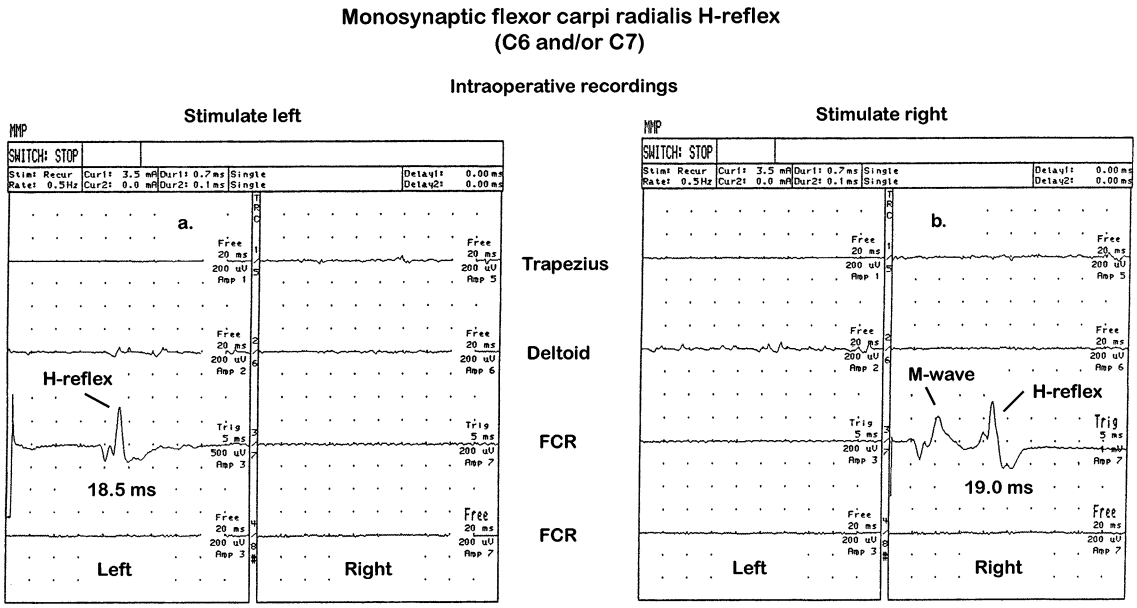


*Fig. 13. These are baseline intraoperative flexor carpi radialis H-reflexes with stimulation on the left (a) and right (b). Bilateral free-run EMG activity is recorded from the trapezius, deltoid and flexor carpi radialis muscles. Electrically-triggered EMG is recorded bilaterally from the flexor carpi radialis muscles. Baseline left C5 and/or C6 nerve root irritation is present, which can be seen as EMG activity in the left deltoid muscle.*

latency the most concise departure from the baseline occurs when the active recording electrode is over the motor point. It is therefore necessary to know the location of the motor point of the gastrocnemius muscle [79]. The H/M ratio is a measure of H-reflex motor neuron pool activation or excitation. It is calculated by dividing the maximum H-reflex amplitude by the maximum M-wave amplitude. It is normally less than 0.7 [63]. Intraoperatively onset latencies may be greater due to decreased limb temperature. In the operating room H-reflex and M-wave amplitudes that are recorded in the clinical setting may be reduced by the use of neuromuscular junction (NMJ) blocking agents. Intraoperatively the gastrocnemius H-reflex parameters that have been monitored are H-reflex amplitude, latency and the H/M ratio. Right–left amplitude and latency differences are also used.

*Gastrocnemius H-reflex stimulation and recording techniques*

For recording subdermal electroencephalographic (EEG) needle electrodes are inserted in the medial head of the gastrocnemius muscle. The H-reflex may also be recorded in the calf from the soleus muscle. The technique for recording the H-reflex from the soleus muscle is the same as that for recording from the gastrocnemius muscle only that the recording electrodes are placed over the mid-dorsal line of the leg with the active electrode 4 cm above the point where the 2 heads of the gastrocnemius muscle join the Achilles tendon. The reference electrode is placed 3 cm distal to the active electrode [76]. Monopolar electromyographic (EMG) needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine teflon coated silver wires with the wire exposed at the end that are inserted with a spinal tap needle may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point of the gastrocnemius muscle and the reference electrode is inserted over tendon or bone. The needles are secured with tape. A range of different high and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [80]. The time base is 100 ms. Recordings are single sweep. Stimulation is with needle or surface electrodes. The cathode is placed proximally in the popliteal fossa between the tendons of the medial and lateral hamstring muscles. The anode is placed 2 to 4 cm distal to the cathode. The stimulation rate is 0.5 Hz and the stimulus duration is 1.0 s. The stimulus intensity is adjusted so that the H-reflex amplitude is maximal. The most effective stimulus intensity is chosen such that any increase or decrease in stimulus intensity results in a decrease in the H-reflex amplitude. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure. Any variability

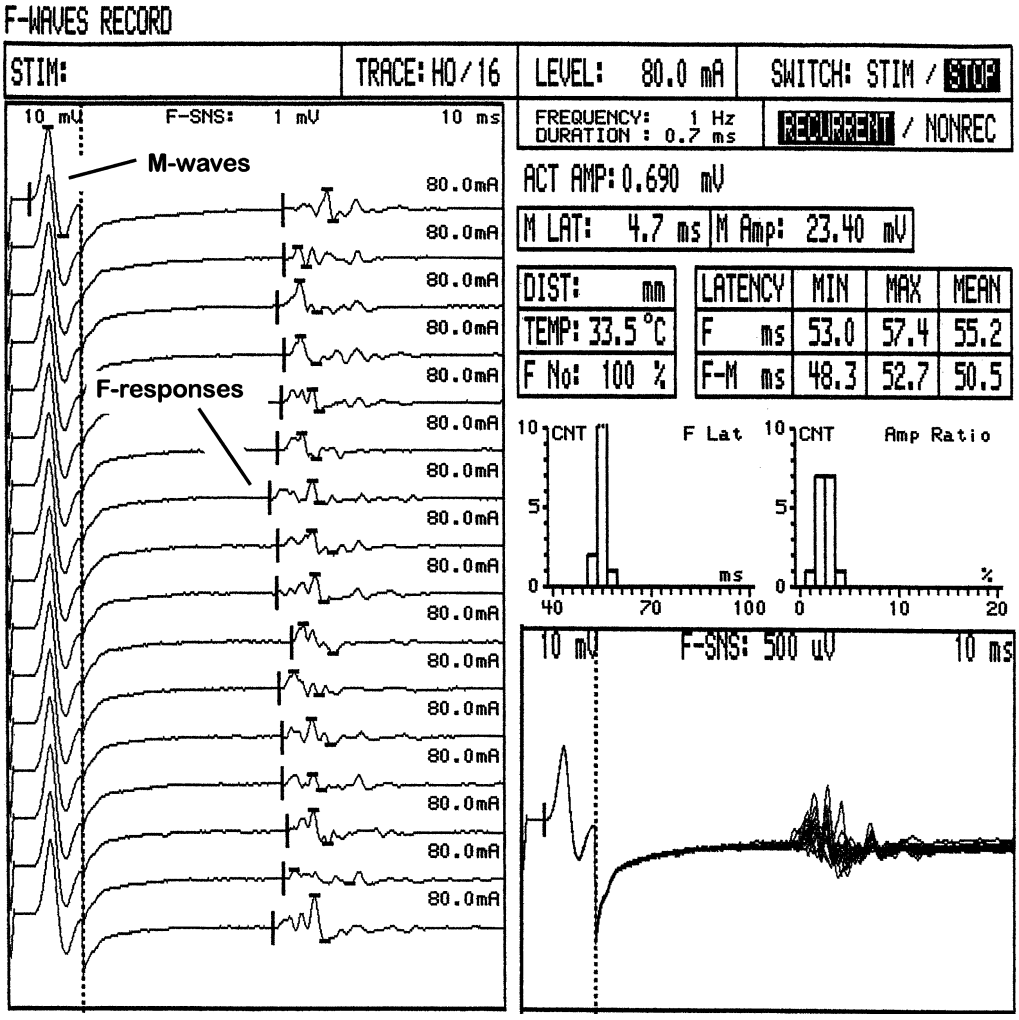


*Fig. 14. These are 16 F-responses recorded from the abductor hallucis muscle following stimulation of the tibial nerve at the ankle. With each stimulus, the F-response latency, amplitude, duration and configuration change.*

in latency and amplitude should be noted in the baseline recordings.

In addition to recording H-reflexes from the gastrocnemius muscle recordings may also be made bilaterally and simultaneously from the vastus medialis, tibialis anterior and abductor hallucis muscles. Recording from these muscle will allow for the detection of heteronymous H-reflexes. This also allows for the monitoring proximal vestibulospinal and reticulospinal controlled motor neurons and distal rubrospinal and corticospinal controlled motor neurons [47], (Figures 10 and 11).

The gastrocnemius H-reflex may be used to monitor peripheral tibial nerve, proximal sciatic nerve, sensory and motor S1 nerve root and S1 segmental spinal cord function. These reflexes can also be used to monitor the function of a variety of suprasegmental descending spinal cord systems that control the S1 segmental interneurons [81]. The ability to record lower-extremity H-reflexes may be affected by pre-existing pathology such as a generalized polyneuropathy, plexopathy or radiculopathy. H-reflexes may be absent, latencies may be prolonged, amplitudes may be decreased and the CMAP configuration may change. Amplitudes may also be decreased with the presence of a myopathy.

### *Flexor carpi radialis H-reflex*

#### *Flexor carpi radialis H-reflex background and normal parameters*

In the upper extremity the flexor carpi radialis H-reflex can be recorded following electrical stimulation of the median nerve over the distal medial upper arm or over the

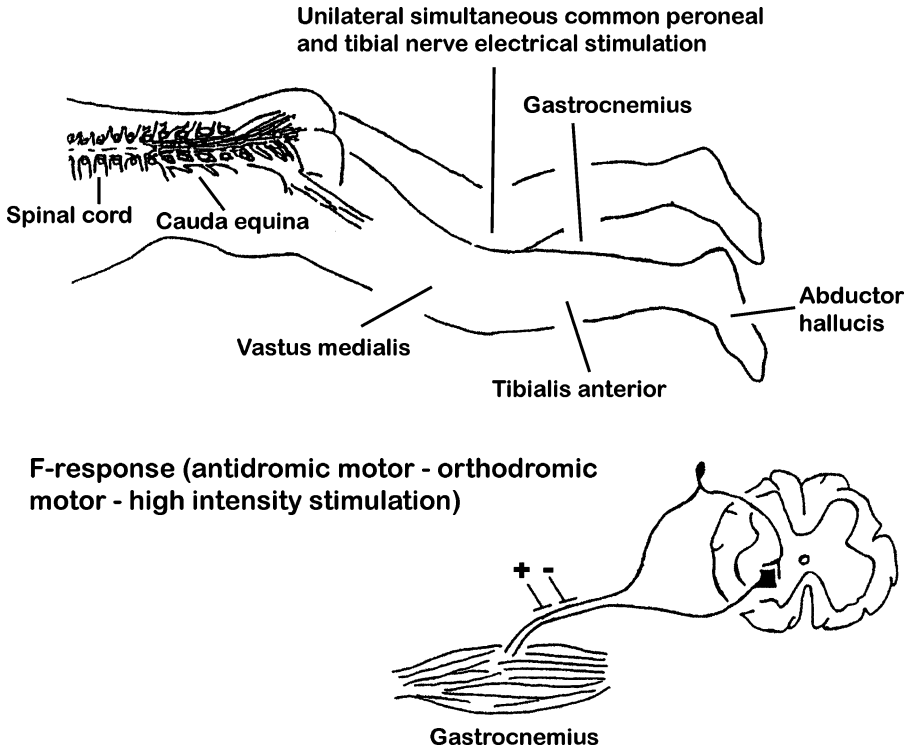


*Fig. 15. Technique for recording lower-extremity F-responses. The peroneal and tibial nerves are unilaterally stimulated in the popliteal fossa, and F-responses are recorded from the tibialis anterior, gastrocnemius and abductor hallucis muscles.*

anterior medial elbow. This reflex is mediated by segmental C6/C7 afferent and efferent activity. Intraoperative normal parameters have not been established. Normal parameters established for clinical studies may serve as a guide for intraoperative studies. Clinically the flexor carpi radialis H-reflex latency varies with the arm length. When measuring latency the most concise departure from the baseline occurs when the active recording electrode is over the motor point. It is therefore necessary to know the location of the motor point of the flexor carpi radialis muscle [79]. In awake humans the mean latency is 17.07 ± 1.77 ms. The interlatency time (ILT) is calculated by subtracting the M-wave from the H-reflex latency. The ILT mean latency is 14.5 ± 1.8 ms. The maximum side-to-side H-reflex latency difference is 0.002 ± 0.42 ms. The maximum side-to-side ITL latency is 0.11 ± 0.44 ms. A regression equation is used to calculate the expected H-reflex latency: H-reflex (ms) = 0.29 + 0.195 × arm-length in cm. The equation for the ILT is: −2.08 + 0.1878 × arm-length in cm. A normogram based upon these equations is available as a reference. The arm length is measured from the tip of the 3rd finger to the C6 spinous process with the arm pronated and the shoulder abducted to 90° [82, 83]. Intraoperatively onset latencies may be greater due to decreased limb temperature. Flexor carpi radialis H-reflex parameters that have been monitored are H-reflex amplitude, latency and the H/M ratio. Right–left latency and amplitude differences are also used.

*Flexor carpi radialis H-reflex stimulation and recording techniques*

For recording subdermal EEG needle electrodes are inserted in the flexor carpi radialis muscles. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine Teflon coated silver wires with the wire exposed at the end that are inserted with a spinal tap needle may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point and the reference electrode is inserted distally over tendon or bone. The needles are secured with tape. A range of different high and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [80]. The time base is 50 ms. Recordings are single sweep. Stimulation is with needle electrodes spaced 2 cm apart unilaterally over

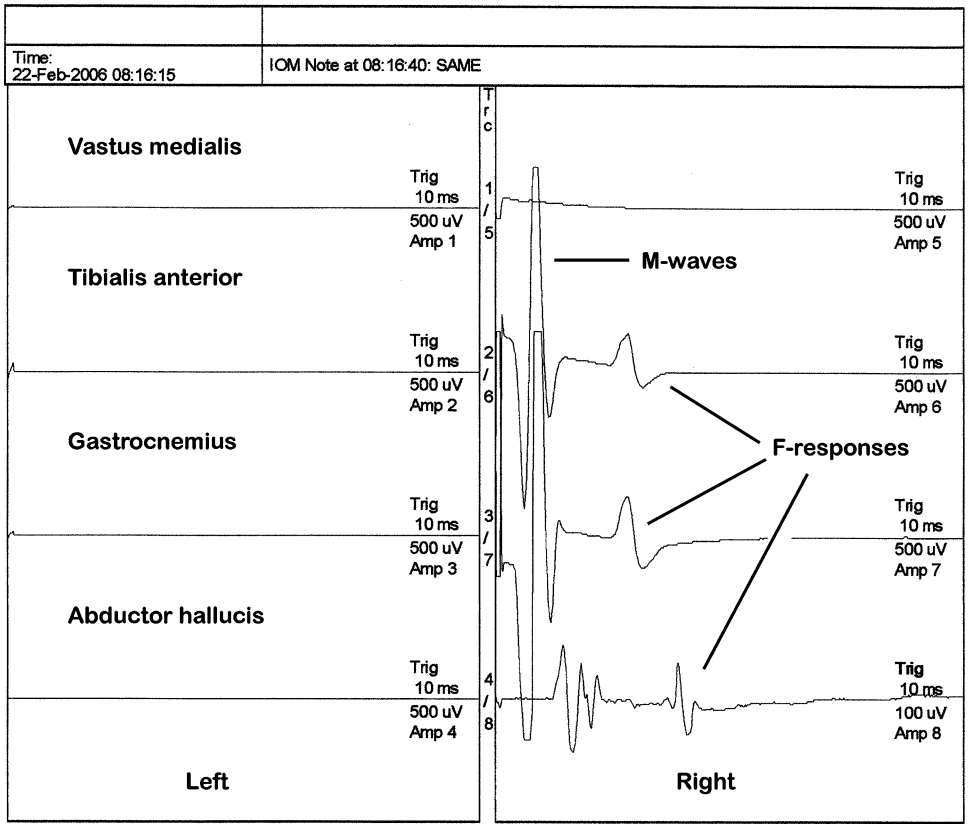


*Fig. 16. Baseline right lower-extremity F-responses recorded in a 14-year-old female with idiopathic scoliosis. The recordings are made from the same patient as in Figure 11, only the right popliteal stimulation intensity is increased to a supramaximal level to elicit F-responses.*

the distal medial upper arm or over the anterior medial elbow at 0.5 Hz and 1.0 s duration. The cathode is proximal. The stimulus intensity is adjusted so that the H-reflex amplitude is maximal. The most effective stimulus intensity is chosen such that any increase or decrease in stimulus intensity results in a decrease in the H-reflex amplitude. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure. In the operating room H-reflex amplitudes that are recorded in the clinical setting may be reduced by the use of NMJ blocking agents. Any variability in latency and amplitude should be noted in the baseline recordings (Figures 12 and 13).

Flexor carpi radialis H-reflexes may be used to monitor median peripheral nerve, brachial plexus and segmental sensory and motor spinal nerve root and spinal cord function. These reflexes can also be used to monitor the function of a variety of suprasegmental descending spinal cord systems that control the C6/C7 segmental interneurons. The ability to record flexor carpi radialis H-reflexes may be affected by pre-existing pathology such as a generalized polyneuropathy, plexopathy or radiculopathy. H-reflexes may be absent, latencies may be prolonged, amplitudes may be decreased and the CMAP configuration may change. Amplitudes may also be decreased with the presence of a myopathy process.

## F-RESPONSE

### *Background*

The F-response is not a reflex but following supramaximal stimulation of a peripheral nerve antidromic motor action potentials are conducted proximally. These antidromic motor action potentials invade the motor neurons in the ventral gray matter and 1–5% of the motor neurons are activated. This is followed by orthodromic conduction through the same motor fibers and the F-response is recorded from muscle. The estimated delay for activation or backfiring of motor neurons is 1.0 ms [34]. The F-response is preceded by the shorter latency M-wave which is the result of conduction from the point of stimulation to the peripheral muscle. These are called F-responses because they

were originally recorded from the intrinsic foot muscles (67). F-responses are composed of the summated electrical activity generated by from 1 to 5 motor units. The F-response amplitude, latency, configuration, and duration vary from one stimulus to the next. These parameters vary because each time the antidromic motor action potentials invade the motor neurons, a different population of motor neurons is activated and these have different conduction characteristics. The amplitude of the F-response is from 1 to 5% of the M-wave amplitude (Figure 14).

F-responses can be recorded from any muscle. They can be recorded at submaximal stimulation but are most prominent with supramaximal stimulation. Both afferent and efferent components of the F-response follow the same motor neurons. F-responses and H-reflexes have similar latencies and are at times confused. The F-response varies in amplitude, latency, duration, and configuration form one stimulus to the next while the H-reflex does not. The H-reflex CMAP is biphasic or triphasic while the configuration of the F-response is usually complex. When the stimulation intensity is held constant the amplitude of the H-reflex does not vary from one stimulus to the next. The H-reflex is usually inhibited at higher intensities of stimulation while the F-response is not [62]. The subpopulation of the motor neuron pool activated by the F-response and H-reflex are not the same [84]. The motor neurons recruited by the H-reflex obey the Henneman size principle [85, 86], recruiting low-force low threshold motor neurons with low intensity stimulation and higher-force high threshold motor neurons with more intense stimulation. F-responses are recruited from the entire motor neuron pool without regard to recruitment thresholds [36]. F-responses have been found to be better at detecting inhibition than facilitation of motor neurons [36]. There is direct evidence for selective discharge of the larger motor neurons in F-responses. The larger motor neurons are inhibited less by Renshaw cell interneurons because the density of the termination of these cells is less on the larger motor neurons. Renshaw cell termination on smaller motor neurons is denser and therefore smaller motor neurons are inhibited more than the larger ones. The larger motor neurons have a faster conduction velocity and antidromic action potentials in these motor neurons will activate the Renshaw cells before action potentials in smaller slower conducting motor neurons. The larger motor neurons will activate those Renshaw cells that inhibit the smaller motor neuron pool and the smaller motor neurons will not be able to generate an F-response when an antidromic motor action potential invades the motor neuron pool. The smaller motor neurons also have a lower resting membrane threshold for activation than larger motor neurons. This is thought to be secondary to suprasegmental systems maintaining them at a lower threshold [87, 88]. Because of this activity recurrent collaterals will depolarize the smaller motor neurons quickly, generating an action potential in the somadendrite region creating a local current circuit. This results in the small motor neuron being refractory when the antidromic motor nerve action potential reaches the axon hillock. Excitation at the axon hillock will not occur and a F-response will not be generated [62]. Orthodromic 1a action potentials may also depolarize the small motor neurons reflexly. Antidromic motor action potentials may not be able to depolarize these motor neurons because they may still be in a refractory state. Because of these F-response properties, F-response recordings may not represent the function of the entire motor neuron pool [89]. Supra F-response stimulation does not inhibit the F-response but activates higher threshold slower conducting group II, III and IV fibers [62].

With chronic upper motor neuron lesions disinhibition of the motor neuron pool may occur. The amplitude and duration of the F-response may increase because a greater percentage of the motor neuron pool is activated by each antidromic impulse. The latencies may be prolonged because the smaller slower conducting motor neurons are activated due to increased central excitability, while the larger motor neurons are blocked by too rapid activation. F-response amplitudes are also increased following reinnervation of a lower motor neuron lesion. With reinnervation there are a greater number of muscle fibers per motor unit. This results in greater summated electrical activity contributing to each F-response amplitude [34, 62, 63].

### *F-response normal parameters*

Intraoperatively F-responses are usually recorded from the abductor digiti minimi muscle following stimulation of the ulnar nerve at the wrist, the abductor pollicis brevis muscle following stimulation of the median nerve at the wrist, the abductor hallucis muscle following stimulation of the tibial nerve at the ankle and from the extensor digitorum brevis muscle following stimulation of the deep peroneal nerve at the ankle. Intraoperative normal parameters have not been established for recording F-responses. Normal parameters established for clinical studies may serve as a guide for intraoperative studies. F-response parameters that have been monitored are minimum, maximum and mean latency, right–left latency differences, amplitude, F/M amplitude ratio and persistence [18, 40, 41, 90].

F-response latencies are usually reported using the minimum onset latency but the mean and maximum latencies are also used. The F-response latency varies with arm length and height. The minimum onset latency mean and standard deviation (SD) and upper limits of normal are:

| | Mean ± SD | Upper limits of normal |
|---|---|---|
| Median | 26.6 ± 2.2 | 31 ms |
| Ulnar | 27.6 ± 2.2 | 32 ms |
| Peroneal | 48.4 ± 4.0 | 56 ms |
| Tibial | 47.7 ± 5.0 | 58 ms |

Normograms based upon height are available to determine the expected minimum latency for each of these nerves. The maximum latency differences between right and left for these nerves are:

| | Mean ± SD | Upper limits of normal |
|---|---|---|
| Median | 0.95 ± 0.67 | 2.3 ms |
| Ulnar | 1.00 ± 0.83 | 2.7 ms |
| Peroneal | 3.5 ± 1.42 | 1.03 ms |
| Tibial | 1.40 ± 1.04 | 3.5 ms [34] |

The number of F-responses recorded to adequately identify the minimum latency is usually 20. But the shortest latency F-response may not occur in the first 20 F-responses and may require 100 or more responses to be recorded [36, 62]. When measuring latency the most concise departure from the baseline occurs when the active recording electrode is over the motor point. It is therefore necessary to know the location of the motor points of the muscles recorded from [79].

The amplitude of the F-response fluctuates between 1 and 5% of the amplitude of the CMAP in normal controls [89]. Maximal stimulation of a motor nerve innervating a muscle results in the recording of the CMAP which is the result of activation of all the motor units in that muscle. A maximal stimulation intensity is achieved when the CMAP amplitude no longer increases with increased stimulus intensity. Supramaximal intensity is achieved when the stimulation intensity is increased to 25% above the maximal intensity [63]. In the clinical laboratory the amplitude of the CMAP recorded with maximal stimulation is:

| Nerve | Muscle | Range (mV) |
|---|---|---|
| Median | abductor pollicis brevis | 5.4–30 |
| Ulnar | abductor digiti minimi | 4.0–22.0 |
| Peroneal | extensor digitorum brevis | 2.6–20.0 |
| Tibial | abductor hallucis | 5.8–32 [47] |

The F-response amplitude median, SD and range parameters are:

| | | |
|---|---|---|
| Ulnar | 206 ± 92 | 13–1940 $\mu$V |
| Peroneal | 141 ± 71 | 10–1471 $\mu$V [89] |

Area measurements of the CMAP and complex configured F-response may be a more accurate measure of the muscle activity contributing to these signals than are peak-to-peak amplitude measurements [91]. In the operating room these CMAP amplitudes that are recorded in the clinical setting may be reduced by the use of NMJ blocking agents.

The ratio of F-response amplitudes to that of the associated M-wave amplitudes (F/M ratio) is a measure of the proportion of a motor neuron pool activated by the antidromic motor impulse. This is calculated as the mean of the amplitudes of the F-responses divided by the mean of the M-wave amplitudes both measured peak-to-peak. The mean and SD F/M ratio for the abductor pollicis brevis is 2.2 ± 1.0% and 2.5 ± 1.2% for the soleus muscles with a upper limit of normal of 5.0% [63]

F-response persistence is a measure of the excitability of the motor neuron pool. Persistence is the number recorded F-responses divided by the number of stimuli. Clinically a persistence less than 50% is considered to be abnormal [92]. The sample size needed to adequately detect pathophysiological persistent changes is 20 for the median and ulnar nerves, 10 for the tibial nerve and 40 for the peroneal nerve [89]. There is some variability in normal persistence reported by different authors. Normal persistence for the abductor pollicis brevis, adductor digiti minimi, soleus and abductor hallucis muscles is 80–90% and 30–40% for the tibialis anterior, extensor digitorum brevis and extensor digitorum communis muscles [63]. Normal persistence was found to be: median: 60–100%, ulnar: 70–100 with a mean of 92%, peroneal: 17–100 with a mean of 60% and the tibial was almost always 100% [89].

### *F-response stimulation and recording techniques*

For recording subdermal EEG needle electrodes are inserted into the muscles. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine teflon coated silver wires with the wire exposed at the end that are inserted with a spinal tap needle may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point and the reference electrode is inserted distally over tendon or bone. The needles are secured with tape. A range of different high and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency

filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [80]. The timebase in the upper extremity is 50 ms and 100 ms in the lower extremity. Recordings are single sweep. Lower-extremity F-responses are recorded from the same muscles as used for recording lower-extremity H-reflexes (vastus medialis, gastrocnemius, tibialis anterior and abductor hallucis muscles). Stimulation is with surface or needle electrodes spaced 2 cm apart over the nerve being stimulated at 1.0 Hz and a 0.2–0.3 ms duration. For lower-extremity recordings the tibial and peroneal nerves are simultaneously stimulated in the popliteal fossa. Stimulus intensity is at a supramaximal level for the presence of the F-response. The cathode is proximal. If the anode is proximal there is a theoretical possibility that anodal blocking may occur. Proximal anodal stimulation may hyperpolarize the nerve resulting in the blocking of the antidromic motor nerve action potential produced by distal cathodal stimulation [92]. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure. Intraoperative F-response parameters may vary due to decreased limb temperature. The degree of variability of F-response parameters should be noted in baseline recordings. (Figures 15 and 16).

The ability to record F-responses may be affected by pre-existing pathology such as a generalized polyneuropathy, entrapment neuropathy, plexopathy or radiculopathy. F-responses may be absent, latencies may be prolonged, amplitudes may be decreased and the configuration may change. Amplitudes may also be decreased with the presence of a myopathy.

Upper and lower-extremity F-responses have been used clinically to detect abnormalities of the lower motor neuron and to detect a disordered central motor system state [93].

F-responses have been recorded intraoperatively to monitor peripheral motor nerve function during total hip surgery [90] and during removal of tumors of the nerve roots proximally. Lower-extremity F-responses have been used to detect suprasegmental injury of the cervical spinal cord [40].

## A-WAVE

The A-wave is a late motor response that is present with constant latency, configuration and amplitude. Low intensity stimulation elicits the A-wave and it is usually blocked by higher intensity of stimulation. A-wave latency is between the CMAP and the F-response latency or exceeds the F-response latency. It may also appear with a latency between the M-wave and H-reflex latency or the latency may exceed the H-reflex latency. A-wave amplitude is less than the H-reflex amplitude. The A-wave should not be confused with the H-reflex or F-response. The A-wave is generated by peripheral nerve changes rather than changes in central nervous system signal processing. The physiology of the A-wave is that there is peripheral neural damage with the presence of a collateral sprout from a proximal point of damaged motor nerve. The collateral sprout innervates muscle. When the antidromic impulse reaches the point of damage a portion of the electrical impulse proceeds distally along the collateral sprout and a small portion of the muscle is activated. Depending upon the nerve stimulated A-waves may be normal or abnormal [62], (Figure 17).

## ANESTHETIC TECHNIQUE

### *Background*

In order to record H-reflexes and F-responses intraoperatively it is critical that the anesthetic technique does not inhibit the activity of the spinal interneurons and the segmental motor neurons. Also, adequate NMJ transmission must be present. H-reflexes and F-responses are usually monitored during the recording of SSEPs, MEPs, polysynaptic reflexes and free-run EMG. The anesthetic technique used must allow for the recording of all these signals with maximum sensitivity [14, 38, 39, 41, 94, 95].

### *Neuromuscular junction monitoring*

There are 5 electrical nerve stimulation EMG techniques for monitoring NMJ function. These are the single twitch (T1%), train-of-four ratio (TOF, TR%), tetanus, post-tetanic stimulation and pulse or double-burst techniques. The TOF technique is most often used for determining the degree of neuromuscular blockade in the operating room. With TOF nerve stimulation a train of 4 supramaximal stimuli are given with an interstimulus interval of 0.5 s (2 Hz) for a train duration of 2 s. When used continuously the train of stimuli are repeated every 10th to 20th second. In order to activate all the motor fibers innervating a muscle the intensity of stimulation must be supramaximal. Therefore, the electrical stimulus applied is 25% above the intensity needed to produce a maximal M-wave response. Stimulation of the skin surface over a nerve is with a monophasic rectangular pulse of between 200 and 300 us duration. Computerized stimulators use a constant current output of at least 70 mA. When using subdermal EEG electrodes for stimulation the amount of current needed to produce a supramaximal response is substantially less than that needed with surface stimulation. Direct muscle stimulation should be avoided for it bypasses

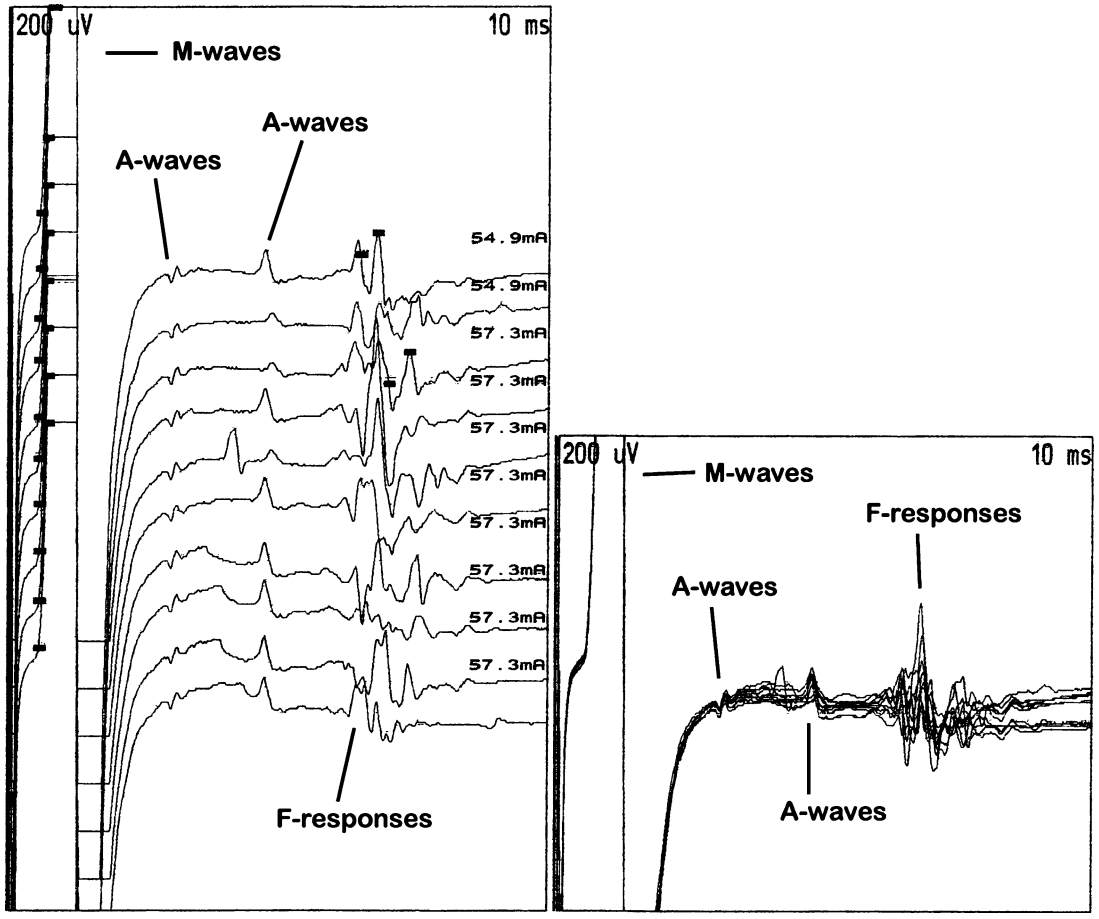


*Fig. 17. These are A-waves and F-responses recorded from the abductor hallucis muscle following stimulation of the tibial nerve at the ankle. A-wave recordings remain stable, while F-responses vary from one stimulus to the next.*

the NMJ so that evoked responses may persist in the presence of complete NMJ blockade. NMJ monitoring should be assessed in the same or neighboring muscle groups from which EMG activity is monitored from. The diaphragm is most resistive to NMJ blockade. The facial muscles are less resistive than the diaphragm and the muscles of the limbs are less resistive than the facial muscles. NMJ blockade develops faster in centrally located muscles, such as the larynx, jaw and diaphragm, than in more peripherally located muscles such as the abductor digiti minimi muscle. In addition to developing more quickly, neuromuscular blockade in these central regions is less profound and recovers quickly. The pattern of response to TOF stimulation varies with the type of neuromuscular blocker administered because the 2 relaxant types, depolarizing and non-depolarizing agents have different mechanisms of action.

With TOF stimulation each stimulus in the train causes the muscle to contract (T1, T2, T3, T4). Fade of the amplitude of each of the last 3 responses in relation to the amplitude of the first response is the basis for evaluation. Dividing the amplitude of the fourth response by the amplitude of the first response provides the TOF ratio (T1:T4, TR%). In the control response before administration of a muscle relaxant all 4 responses are ideally the same amplitude and the TOF ratio is 1.0. A computer may be used to quantify the relationship of the TOF responses. A mechanical strain gauge or accelerometer may also be used for recording. Non-depolarizing NMJ blocking agents are competitive inhibitors of the acetylcholine (ACh) receptors. They compete with ACh for the active, or binding sites on the alpha subunits of the muscle membrane receptors and prevent ACh from depolarizing the membrane. With TOF stimulation the muscle response to stimulation fades over time because of a decrease in the amount of ACh released from the prejunctional nerve terminal with successive stimuli. The amplitude of the fourth response is decreased relative to the first response because the lesser

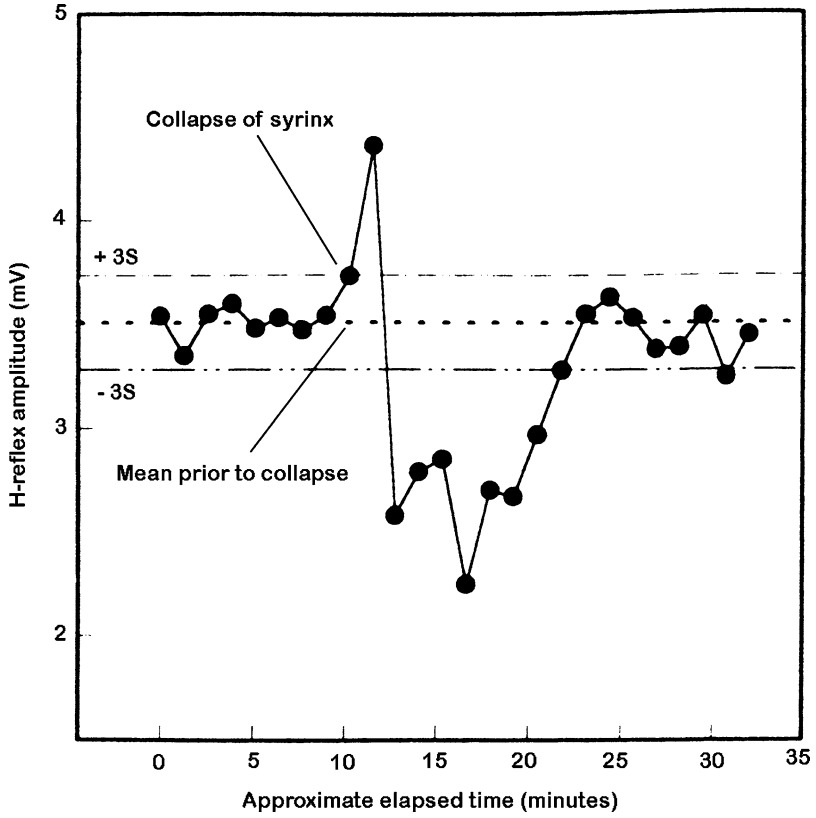


*Fig. 18. Cervical myelotomy for syringomyelia in a 27-year-old woman. Collapse of the syrinx was accompanied by a transient increase in the H-reflex amplitude, followed by suppression of the H-reflex. Stimulus intensity was held constant (22.0 mA). This intensity proved subthreshold for motor fibers and the M wave. Concentrations of anesthetic agents were also held constant (ET isoflurane 0.63–0.67%; ET $N_20$ 51.4–52.0%). Postoperatively, no deterioration in function was noted. Reproduced with permission from Leis et al (1996).*

amount of ACh that is released into the synaptic cleft with the fourth stimulus cannot overcome the competitive block readily. The degree of fade is inversely proportional to the degree of blockade. As a non-depolarizing agent becomes more effective, a sequential loss of twitches is observed and each loss is related to the amplitude of the first twitch (T1). T4 disappears when the amplitude of T1 is about 25% of the control value and a 75% blockade is present. T3 disappears in addition to T4 when the amplitude of T1 is about 20% of the control value and a 80% blockade is present. T2 disappears in addition to T4 and T3 when the amplitude of T1 is about 10% of the control value and a 90% blockade is present. When T1 disappears and all 4 responses are absent a 100% blockade is considered to be present. If the patient's anesthesia becomes light with 60% or greater blockade the patient should not be able to lift the arms over the head. When blockade is 40% the patient may be able to lift the head for 3 s. When blockade is between 25 and 30% the patient may be able to lift the head for 5 s and complete recovery occurs when blockade is 20%.

Depolarizing NMJ agents block NMJ function by depolarizing the muscle membrane without subsequent repolarization so that ACh released by nerve action potentials cannot activate the post-junctional muscle membrane. ACh is rapidly hydrolyzed by acetylcholinesterase and is cleared from the synaptic cleft. In contrast, the depolarizing agent is not susceptible to hydrolysis by acetylcholinesterase and is not removed from the junctional cleft until after it is eliminated from the plasma which is very slow. Therefore, the effect of the depolarizing agent is prolonged. With a depolarizing neuromuscular blocking agent the response to TOF stimulation is different than that seen with the use of a non-depolarizing agent. During a partial depolarizing block, no fade occurs in the TOF response. Ideally the TOF ratio is approximately 1.0. With higher concentrations of depolarizing agents the 4 response amplitudes decrease at the same time and equally. Therefore, the TOF ratio cannot be used when a depolarizing agent is used. When it is desirable to quantify the degree of neuromuscular blockade with the use of the TOF ratio non-depolarizing NMJ blocking agents should be used [96, 97].

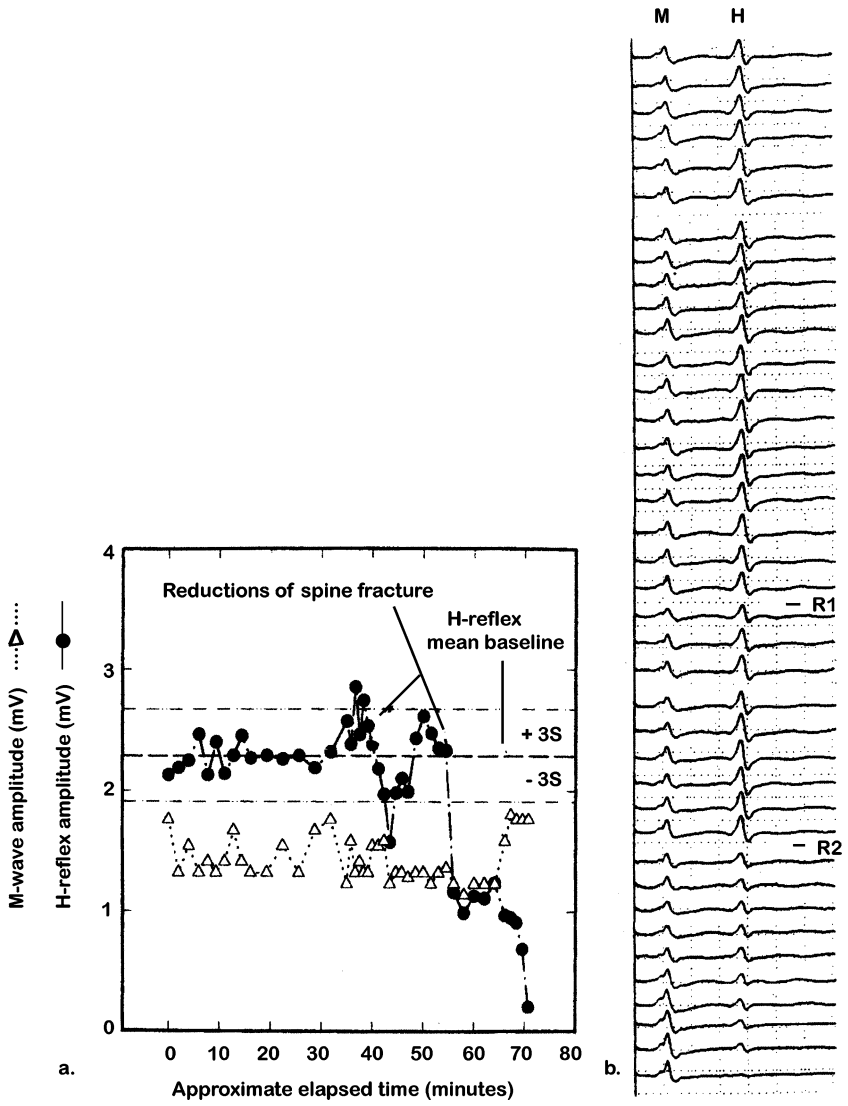


*Fig. 19. (a) M-wave and H-reflex responses (graphical data) during thoracic spine surgery in a 24-year-old man. Reduction of a T8 fracture resulted in prolonged suppression of the H-reflex. Stimulus intensity was maintained at 34.0 mA. Minor fluctuations in the M wave were not responsible for the H-reflex changes; M-wave mean amplitude for data points 0 to 15 was 1.47 ± 0.17 mV, compared with 1.34 ± 0.13 mV for data points 21 to 36 during the reductions. Concentrations of anesthetic agents were altered only slightly (ET isoflurane, 1.34-1.41%; ET $N_2$0 54-45%). Postoperatively, the patient developed severe weakness in lower extremities. (b) M-wave (M) and H-reflex responses (H, actual waveforms) during thoracic spine surgery in a 24-year-old man. R1 denotes time of first attempt to reduce the fracture. R2 denotes time of second reduction. Note marked suppression of the H-reflex after the second reduction. Sweep 10 ms/division; gain 2 mV/division. Reproduced with permission from Leis et al (1996).*

*Anesthetic effects on spinal cord CPGs*

H-reflexes and F-responses measure the level of motor neuron excitability [63, 92]. Studies performed to determine the concentration of anesthetic agents needed to prevent movement in surgical patients have used H-reflexes and F-responses to measure the effect of these agents on the level of excitability of the motor neuron pool [39, 98–105].

Soleus H-reflexes were used to determine the level of motor neuron excitability intraoperatively. In 10 normal human volunteers 1.0–1.5% enflurane was found to decrease H-reflex amplitudes from 35 to 100% of baseline values [99].

The polysynaptic bulbocavernosus sacral reflex was recorded in 119 patients. The dorsal penile or clitoral nerve was electrically stimulated and reflex activity was recorded

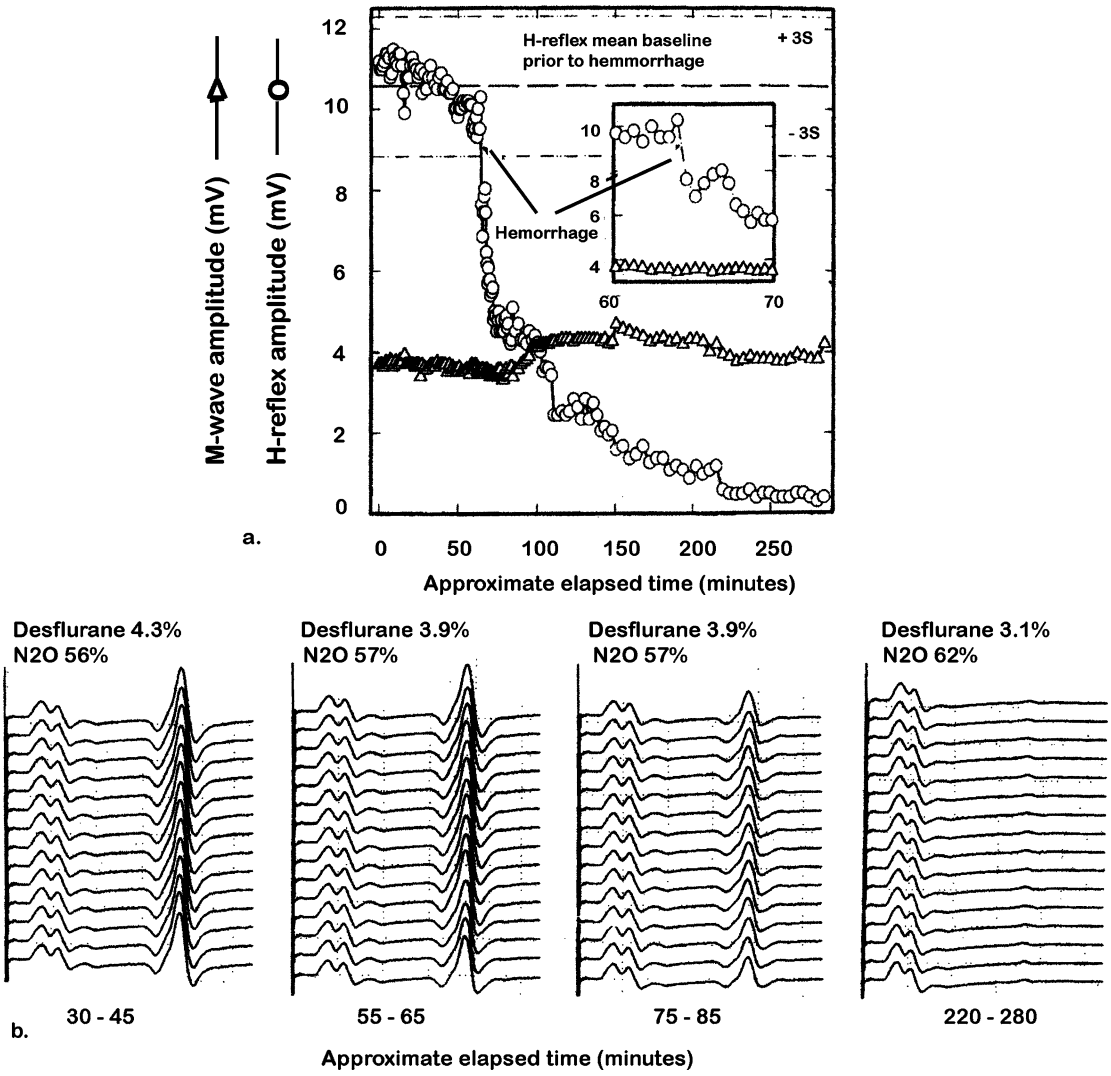


*Fig. 20. (a) M-wave and H-reflex responses (graphical data) during cervical myelotomy for post-traumatic syringomyelia in a 71-year-old man. Spinal cord hemorrhage was associated with an immediate reduction in the H-reflex amplitude, followed by a steady decline and disappearance of the response. Stimulus intensity was maintained at 36.0 mA, resulting in stable M-wave responses. Postoperatively, the patient developed severe weakness in his lower extremities. (b) Representative M-wave and H-reflex responses (actual waveforms at times indicated) during cervical myelotomy for post-traumatic syringomyelia in a 71-year-old man. Minimum alveolar concentrations of anesthetic agents decreased during the 280-minute window of data acquisition (ET desflurane fell from 5.0 to 3.1% during this period; ET $N_20$ was maintained from 55 to 62%). Sweep 10ms/division. Reproduced with permission from Leis et al (1996).*

with hooked wire electrodes from the anal sphincter muscles. No NMJ blocking agents were present. Patients were anesthetized with propofol and fentanyl. Reflex activity was suppressed when 1.27% isoflurane or 60% nitrous oxide was added [95].

The effect of isoflurane alone and isoflurane with $N_20$ on the soleus H-reflex was studied under general anesthesia in 25 patients. No NJM blocking agents were used. 23 of these patients had consistently measurable stable H-reflexes with baseline amplitudes varying from 3.43–11.97 mV. The addition of 0.68% isoflurane decreased the amplitude to 48% of the baseline. Increasing the isoflurane to 1.37% decreased the amplitude to 33.8% of baseline. The combination of 0.81% isoflurane with 30% $N_20$ decreased the amplitude to 66.2% of baseline. The combination of 0.37% isoflurane with 70% $N_20$ decreased the amplitude to 30.4% of the baseline. They reported a linear relationship between isoflurane concentration and H-reflex amplitude. There was a linear relationship between $N_20$ concentration and H-reflex amplitude. They concluded that the H-reflexes may be recorded with a combination of isoflurane and $N_20$. The H-reflex is maximally stable within the range

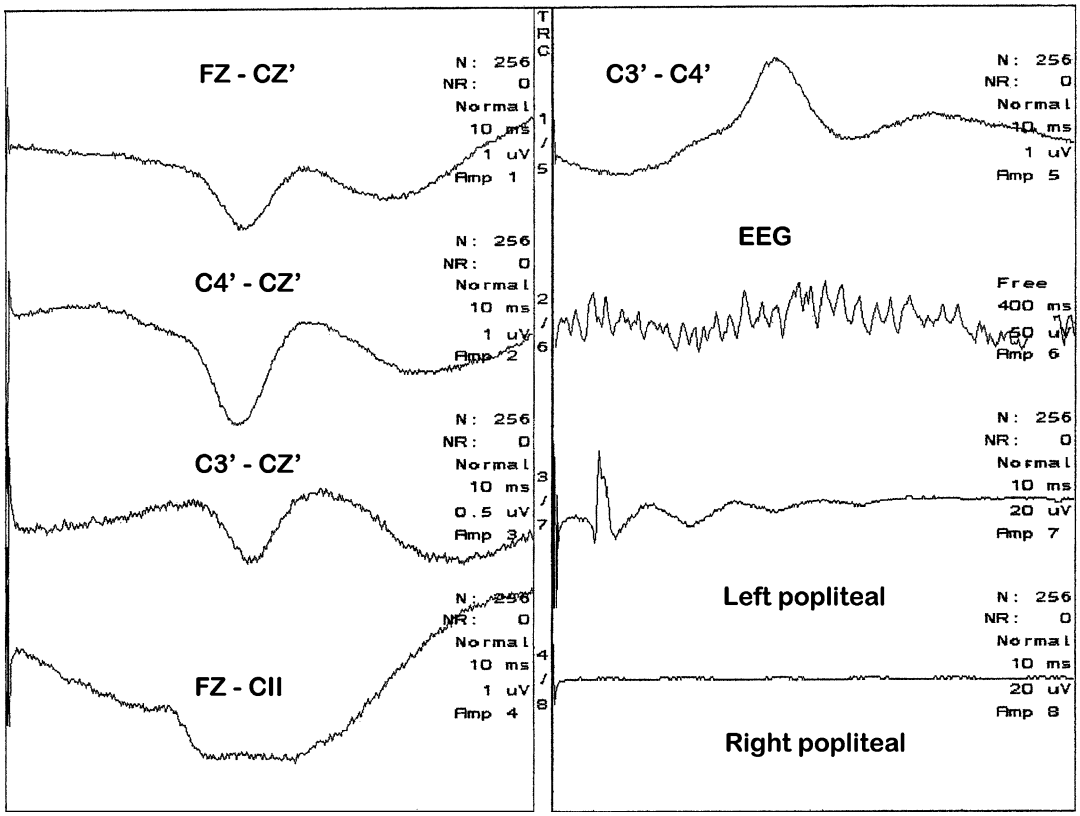


*Fig. 21. Baseline left posterior tibial nerve somatosensory evoked potentials. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

of anesthetic concentrations used to achieve surgical immobility [39].

The effect of isoflurane and $N_20$ on spinal motor neuron excitability was studied in 8 adult patients by monitoring soleus H-reflexes and abductor hallucis F-responses. No NMJ blocking agents were used. H-reflex amplitude was decreased to 48.4 ± 18.6% of the baseline with 0.6 MAC isoflurane and to 33.8 ± 19.1% with 1.2 MAC isoflurane. Minimum alveolar concentration (MAC) is defined as the alveolar concentration of an anesthetic agent that prevents purposeful movement in 50% of patients in response to a surgical stimulus. F-response amplitude and persistence decreased to 52.2 ± 22.8% and 44.4 ± 26.0% of the baseline at 0.6 MAC isoflurane and to 33.8 ± 26.0% and 21.7 ± 22.8% at 1.2 MAC. With 1.0 MAC isoflurane the H-reflex amplitude was decreased by 32.5 ± 19.2%, 33.3 ± 20.8% and 30.4 ± 23.5% of baseline levels at 30%, 50% and 70% nitrous oxide respectively [98].

In 12 adult patients a comparison of the effects of isoflurane on transcranial electrical activated MEPs and F-responses were studied. Anesthesia was maintained with 60% $N_20$, 100 $\mu$g/Kg/min of propofol and supplementary fentanyl, 0.5–1.0 $\mu$g/Kg. Recordings were made before and after adding 0.5% isoflurane. Baseline MEP amplitudes (median, 205 $\mu$V, 25th–75th percentiles, 120–338 $\mu$V), F-response amplitudes (median, 100 $\mu$V, 25th–75th percentiles, 64.2–137.5 $\mu$V) and F-response persistence (59 ± 29%) were decreased to 0.0 $\mu$V (0–15 $\mu$V), 49 $\mu$V (12.4–99.6 $\mu$V) and 30 ± 31% respectively by 0.5% isoflurane. The MEPs were suppressed more than the F-responses. No NMJ blocking agents were used [100].

Soleus H-reflexes and abductor hallucis F-responses were monitored to determine the effect of hyperventilation and hypoventilation on motor neuron excitability during isoflurane anesthesia. H-reflex and F-responses were recorded before and after changing the $ETCO_2$ concentration. Anesthesia was maintained with 0.8% isoflurane and no muscle relaxants were used. An $ETCO_2$ of 25 mm Hg decreased the pre-anesthetic H-reflex amplitude from 6.8 ± 2.7 mV to 4.0 ± 2.0 mV and to 2.0 ± 2.2 mV at an $ETCO_2$ of 45 mm Hg. F-response persistence decreased from the pre-anesthetic value of 100% to 77 ± 24% at an $ETCO_2$ of 25 mm Hg and to 61 ± 19% at an $ETCO_2$ of 45 mm Hg. They concluded that

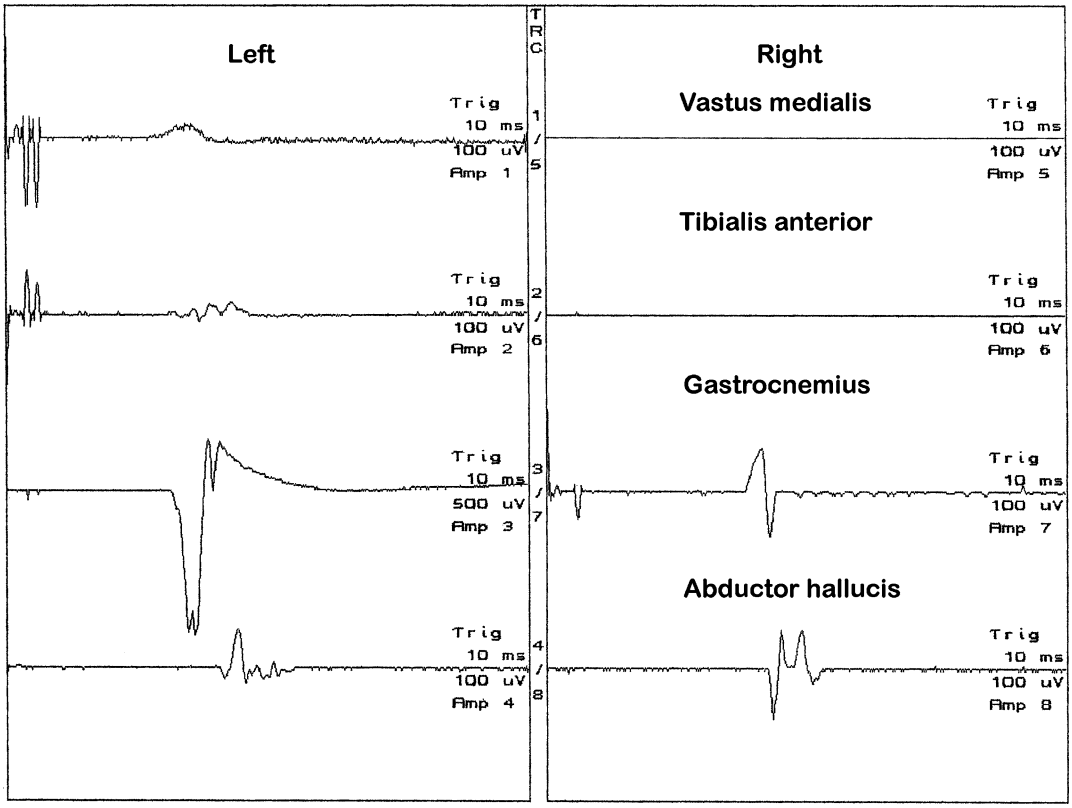


*Fig. 22. Baseline lower-extremity transcranial electrical motor evoked potentials; 224 V, anode – right. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

hyperventilation and hypoventilation effects motor neuron excitability and may affect the probability of patient movement during surgery [101].

The median nerve F-responses were monitored to determine the effect of propofol, ketamine and fentanyl on motor neuron excitability. No NMJ blocking agents were used. Propofol decreased the F-response persistence from $77.5 \pm 15.2\%$ to $40.9 \pm 16.8\%$. Ketamine and fentanyl had no significant effects on F-response persistence [102].

The effects of propofol on the soleus H-reflex were studied in 33 patients. No NMJ blocking agents were used. H-reflexes were recorded before administration of anesthetics and after an initial dose of 2 $\mu$g/Kg over 1 min followed by a continuous infusion at a rate of 167 $\mu$g/Kg/min for 10 min. Measurements were also made after infusing propofol to a blood level of 6 $\mu$g/ml and 9 $\mu$g/ml. The initial dose of 2 mg/Kg decreased the H-reflex amplitude and H/M ratio. The following 10 min infusion did not further decrease these values. The 6 $\mu$g/Kg injection did not change the H-reflex amplitude and H/M ratio. The 9 $\mu$g/Kg injection decreased the amplitude and H/M ratio. They recommended that the propofol induction dose be 1.0–2.5 $\mu$g/Kg and this should be followed by an infusion of from 100–200 $\mu$g/Kg/min. They concluded that the immobility during propofol anesthesia is not caused by a depression of spinal motor neuron circuit excitability. Propofol does not decrease axon conduction peripherally nor transmission at the neuromuscular junction [103].

In 12 patients during sevoflurane anesthesia the predictive power of the soleus H-reflex for detecting movement was compared to EEG Bispectral index and spectral edge frequency. Sevoflurane (1.43 to 1.77%) caused a gradual reduction in H-reflex amplitude without a change in latency and no changes in the M–wave latency or amplitude. H-reflexes predicted motor responses to noxious stimulation while Bispectral Index and spectral edge frequency were not different from chance alone. Noxious electrical stimulation over the forearm increased the H-reflex amplitude but the EEG parameters did not change. Therefore, electrical noxious stimulation may lead to spinal arousal without activation of thalamocortical circuits. More intense noxious stimulation may cause more generalized arousal [104].

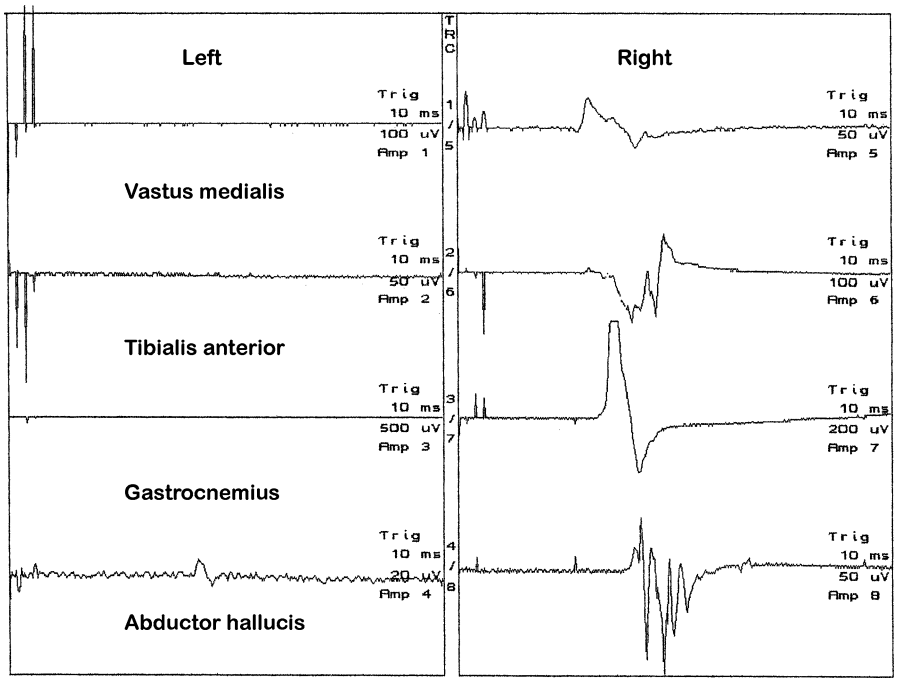


*Fig. 23. Baseline lower-extremity transcranial electrical motor evoked potentials; 224 V, anode – left. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

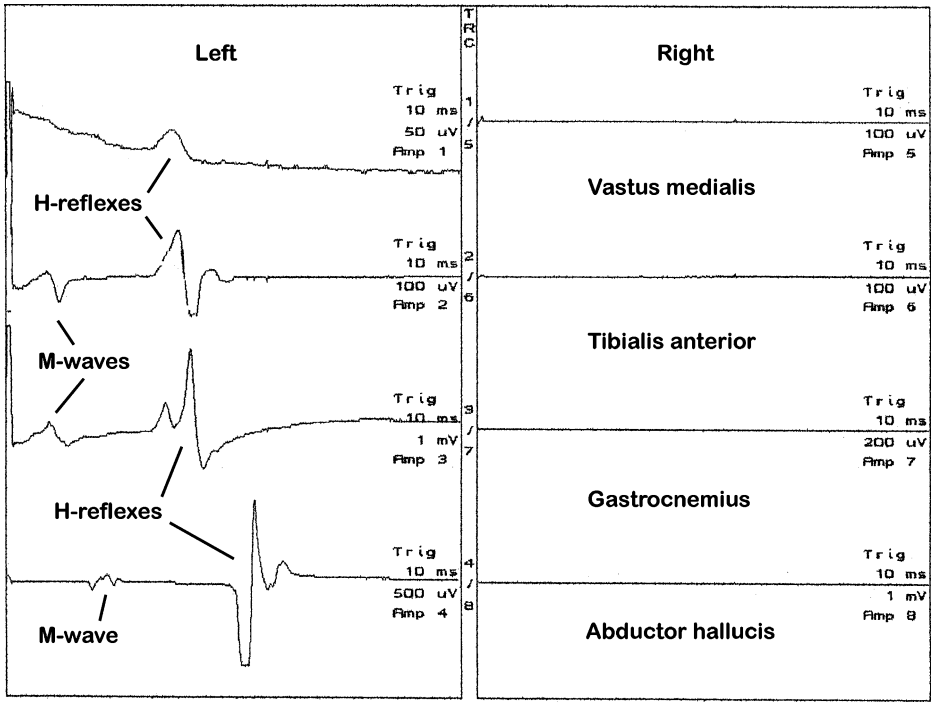


*Fig. 24. Baseline left lower-extremity H-reflexes. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

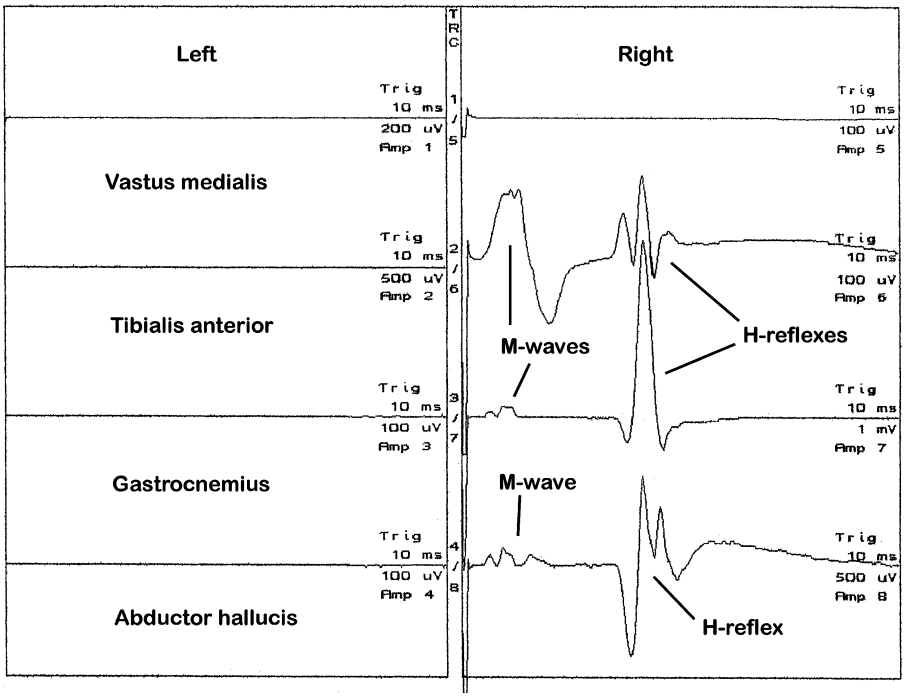


*Fig. 25. Baseline right lower-extremity H-reflexes. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

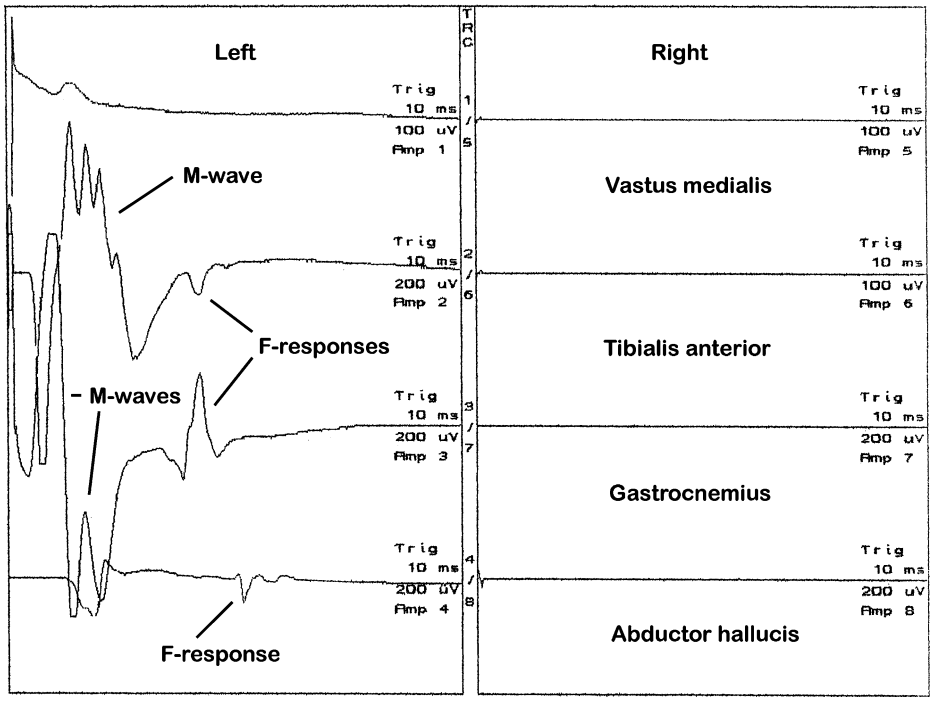


*Fig. 26. Baseline left lower-extremity F-responses. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

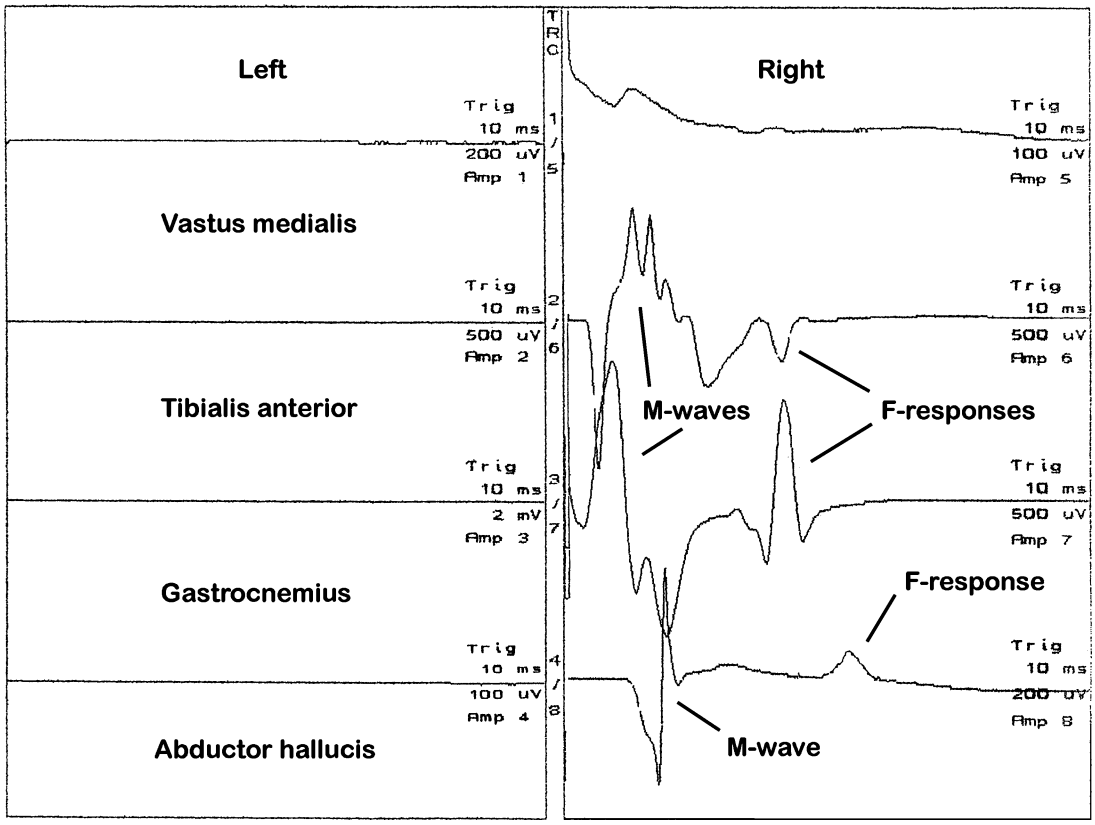


*Fig. 27. Baseline right lower-extremity F-responses. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

The lamprey has been used as a model for studying locomotor systems because they share anatomical, functional and pharmacologic properties with that of mammals. Using lamprey spinal cords the effect of isoflurane on activity and intersegmental coordination of spinal interneuronal locomotion generating circuits was studied. With isolated application of isoflurane to the middle spinal cord compartment there was a dose dependent reduction in locomotor activity in the middle, caudal and rostral compartments. They concluded that isoflurane decreases the coordinated activity of CPGs in the spinal cord [105].

The results of these studies were not able to determine if the attenuation of H-reflexes and F-responses by inhalation agents was due to a direct inhibition of the 1a fibers, to direct effect on the motor neuron membrane or to an altered balance between supraspinal excitatory and inhibitory pathways projecting on the motor neuron pool. When intraoperative H-reflex and F-response recordings are interpreted it is important to consider the effect of these different agents on the different levels of the neuromuscular system.

*Intraoperative monitoring technique*

After the patient has been anesthetized and before the administration of NMJ blocking agents used for intubation baseline train-of-four (TOF) CMAP recordings should be made. When it is desirable to record baseline H-reflex and F-response activity immediately after intubation a short acting NMJ blocking agent such as succinylcholine chloride (a depolarizing agent) may be used. Succinylcholine chloride should not be used when there is a risk of developing malignant hyperthermia [36]. Twenty-five percent recovery of NMJ function with succinylcholine occurs with a mean of 7.6 min [37]. The effects of these agents on NMJ function recovery must be considered in baseline H-reflex and F-response recordings. TOF recordings may be made with a computerized NMJ monitor from the hypothenar eminence of the hand after ulnar nerve stimulation at the wrist. Most of our understanding of intraoperative NMJ monitoring is the result of studies involving stimulating the ulnar nerve and recording CMAPs from the hypothenar eminence. In addition to monitoring ulnar

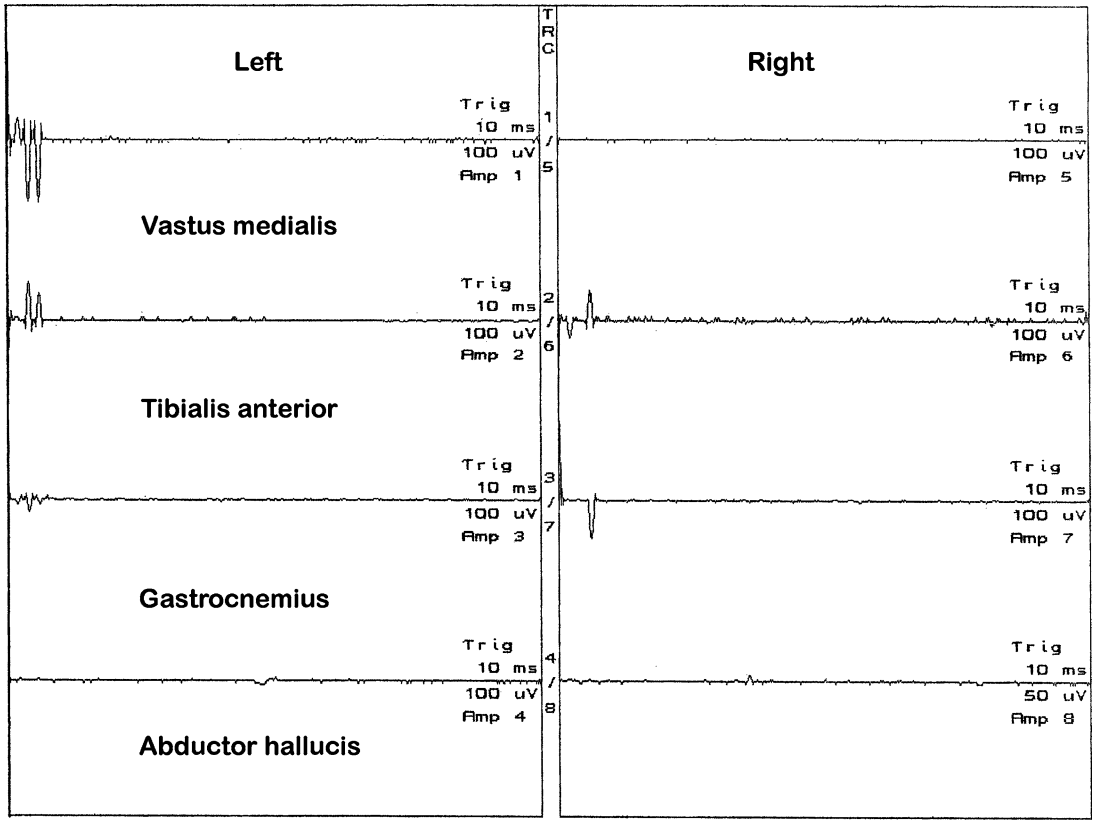


*Fig. 28. Lower-extremity motor evoked potentials became absent during removal of a calcified disk; 233 V, anode – left. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

nerve function it is most appropriate to also record TOF activity from a muscle in the extremity where EMG activity is being monitored. When monitoring the lower extremity the peroneal nerve may be stimulated at the knee and TOF CMAPs may be recorded from the tibialis anterior muscle. The tibial nerve may be stimulated at the ankle and CMAPs recorded from the abductor hallucis muscle. The tibial nerve may be stimulated in the popliteal fossa and the CMAPs may be recorded from the gastrocnemius muscle.

A maintenance anesthetic monitoring technique that allows for the recording of SSEPs, MEPs, H-reflexes, F-responses, polysynaptic reflexes and free-run EMG with maximum sensitivity is:

1. Nitrous oxide: 50% or less
2. Isoflurane: none or 0.2 to 0.5%
3. Propofol: less than 200 $\mu$g/Kg/min
4. Fentanyl: continuous infusion
5. Muscle relaxants: none

Monitoring EEG to determine the level of cortical excitability is helpful when interpreting SSEP and MEP signals. A single EEG recording channel (O2-FZ) provides a measure of generalized cortical excitability (5 s timebase, 0.2–100 Hz filter settings, 50 $\mu$V sensitivity). Propofol infusion rates from 170 to 500 $\mu$g/Kg/min can cause burst suppression [108]. This depth of anesthesia can result in a reduction in amplitude of cortical SSEP components and MEPs.

## CLINICAL CORRELATION

Our understanding of the mechanisms involved in acute complete and partial SCI in humans is derived from studies of the normal and abnormal electrophysiology of the spinal cord CPG components in humans and animals. Our understanding of how H-reflex and F-response changes correlate with the patient's postoperative status is derived from the following intraoperative studies.

Soleus H-reflexes and abductor hallucis F-responses were recorded in 32 patients during spinal cord surgery. In 6, an abrupt fall in H-reflex amplitude beyond 3 SD from the baseline or significant drop in F-response persistence

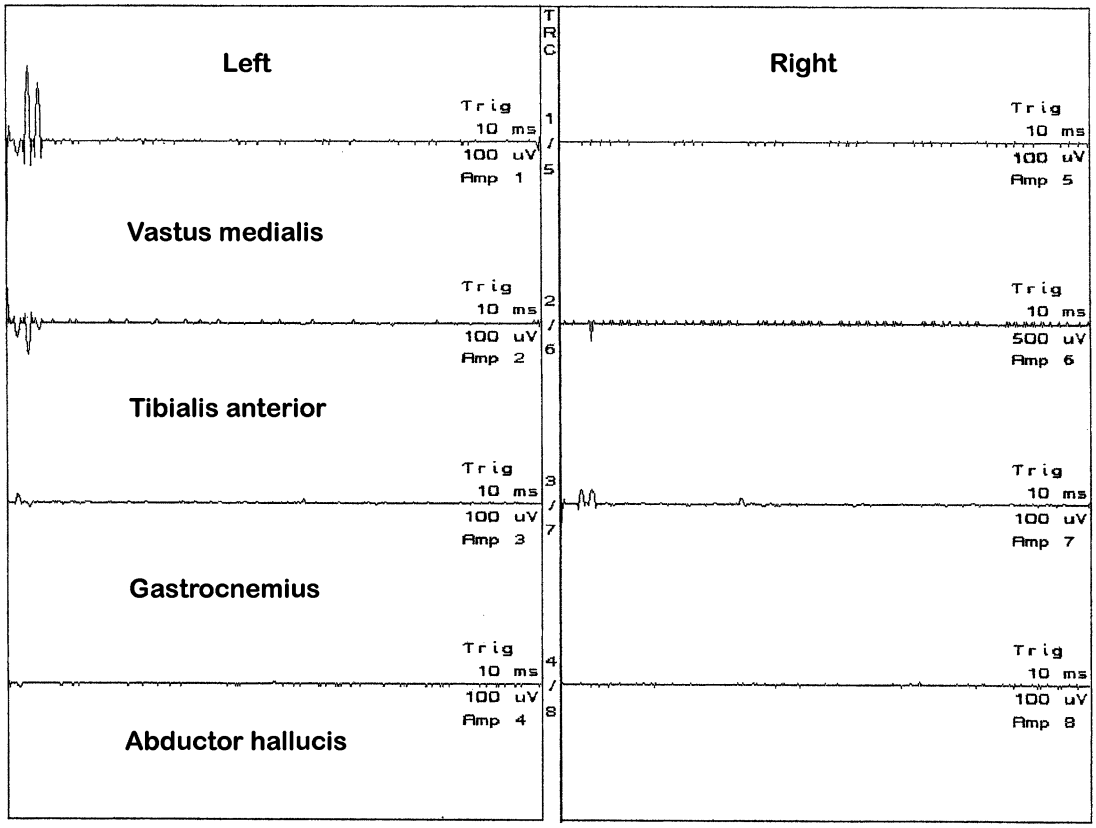


*Fig. 29. Lower-extremity motor evoked potentials became absent during removal of a calcified disk; 233 V, anode – right. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

coincided to perturbation or injury to the spinal cord. Suppression was transient in 4 patients with less than a 50% drop in H-reflex amplitude or abductor hallucis F-response persistence. None of these patients developed a new postoperative neurological deficit. Suppression exceeded 90% of the baseline values and persisted through surgery in 2 patients. Both patients had profound postoperative neurological deficits. The author concluded that rostral SCI suppresses H-reflexes and F-responses. The degree of suppression reflects the severity of injury. The mechanism responsible for these changes is thought to be hyperpolarization of caudal motor neurons which occurs within seconds of injury [40].

Lower-extremity H-reflexes were recorded in 31 patients during spine or spinal cord surgery. A significant change in amplitude was considered significant if it exceeded 3 SD of the mean post-anesthetic baseline. In 6 patients there was a significant decrease in H-reflex amplitude. In each case the onset of H-reflex suppression coincided with a potentially injurious event. In one case involving a cervical myelotomy for decompression of a large syrinx the syrinx collapsed producing immediate fluctuation in the H-reflex amplitude. The amplitude recovered to the baseline level 9 min later (Figure 18). Postoperatively no neurological deficit was noted. Another case involved mechanical reduction of a T-8 spinal fracture. Manipulation of the spine before reduction resulted in increased variability in H-reflexes. The first attempt to reduce the fracture produced a transient fall in H-reflex amplitude. The next reduction resulted in a pronounced reduction in amplitude to less than 10% of baseline (Figure 19a,b). The H-reflex remained suppressed until the end of surgery. Postoperatively the patient had severe motor and sensory deficits in both legs that were not present preoperatively. A third case involved a cervical myelotomy for decompression of a posttraumatic syrinx. The operation was complicated by cervical cord hemorrhage which was followed by reduction H-reflex amplitude (Figure 20a and b). The amplitude steadily declined and the H-reflex disappeared and remained absent. The patient awoke with profound weakness in both lower extremities. The changes in these patients demonstrated that H-reflex changes occur immediately at the time of spinal injury. H-reflex changes may be reversible and reflect the severity of SCI [39].

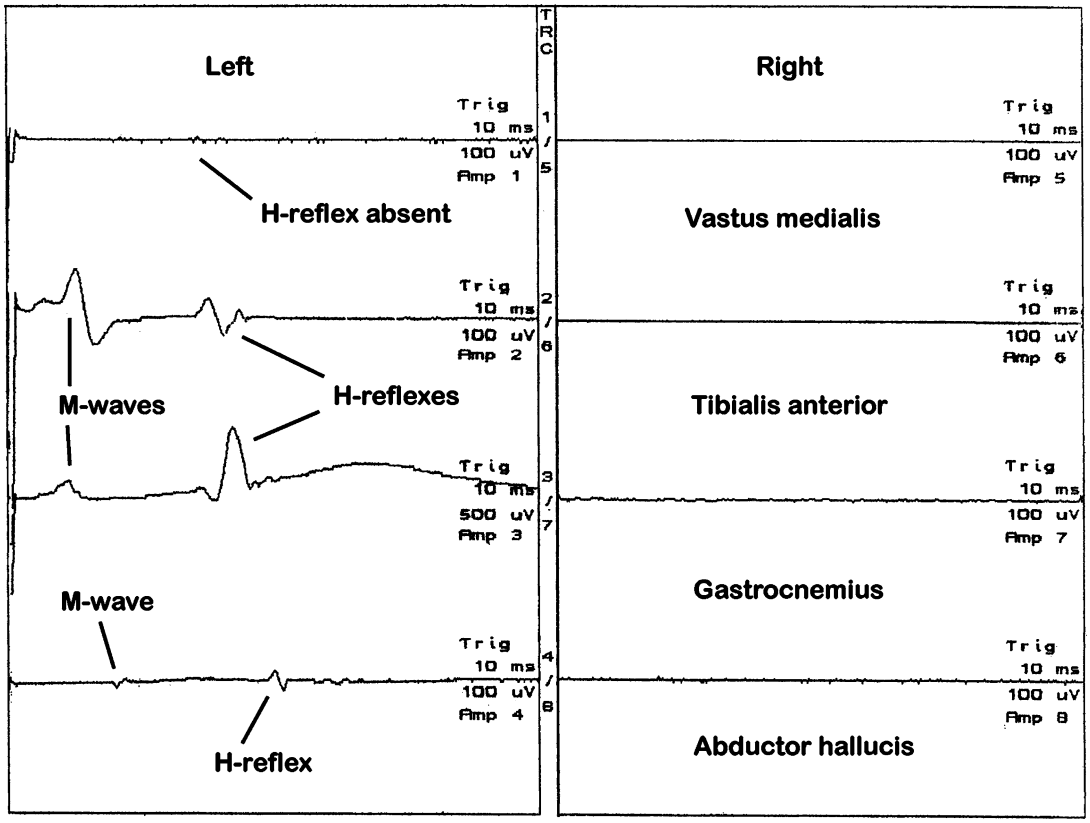


*Fig. 30. During removal of the calcified disk, the left lower-extremity vastus medialis H-reflex became absent, and the tibialis anterior, gastrocnemius and abductor hallucis H-relfex amplitudes were decreased. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

H-reflexes may indicate intact spinal cord function with changes in SSEPs. Soleus H-reflexes and tibial SSEPs were monitored in a patient during T7–T12 laminectomy for spinal stenosis. During laminectomy the left SSEP became absent and the right was transiently reduced. No H-reflex changes occurred. Postoperatively no lower-extremity motor deficits were present and no new sensory deficits [56].

In 278 pediatric spine surgeries gastrocnemius H-reflex and SSEP monitoring were used for monitoring spinal cord function. Combined H-reflex and SSEP monitoring improved the reliability for detecting spinal cord compromise compared to either procedure alone. H-reflexes exhibited more changes than SSEPs. These changes reflected changes in spinal cord gray matter function related to acidosis and changes in hematocrit and blood pressure [109].

In a clinical setting soleus H-reflexes and abductor hallucis F-responses were recorded in 14 patients following SCI that resulted in either partial injury without spinal shock or injury with spinal shock. Deep tendon reflexes following tap of the Achilles and patellar tendons were evaluated also. Patients were evaluated within 24 h of injury and on day 10, 20 and 30 post-injury. F-responses were absent in patients with spinal shock, reduced in persistence in patients with acute injury without spinal shock and normal in persistence in patients with chronic injury. F-response changes persisted up to 2 weeks following SCI. H-reflexes were absent or markedly suppressed in patients with spinal shock within 24 h of injury but recovered to normal amplitude within several days of the injury. Deep tendon reflexes were proportionally more depressed in spinal shock than H-reflexes. This demonstrated dissociation between electrically and mechanically induced reflexes during spinal shock. The observation that the stretch reflex is more depressed than the H-reflex is consistent with depressed fusimotor drive with SCI [55].

A 63 year-old female presented clinically with right T8 dermatomal and bilateral lower-extremity pain. Imaging studies revealed a large T8-9 herniated disk which was causing spinal stenosis with cord compression. The patient was treated with a bilateral T8–9 laminectomy with left far lateral costovertebral exposure for disk removal. Baseline intraoperative tibial somatosensory evoked potentials were

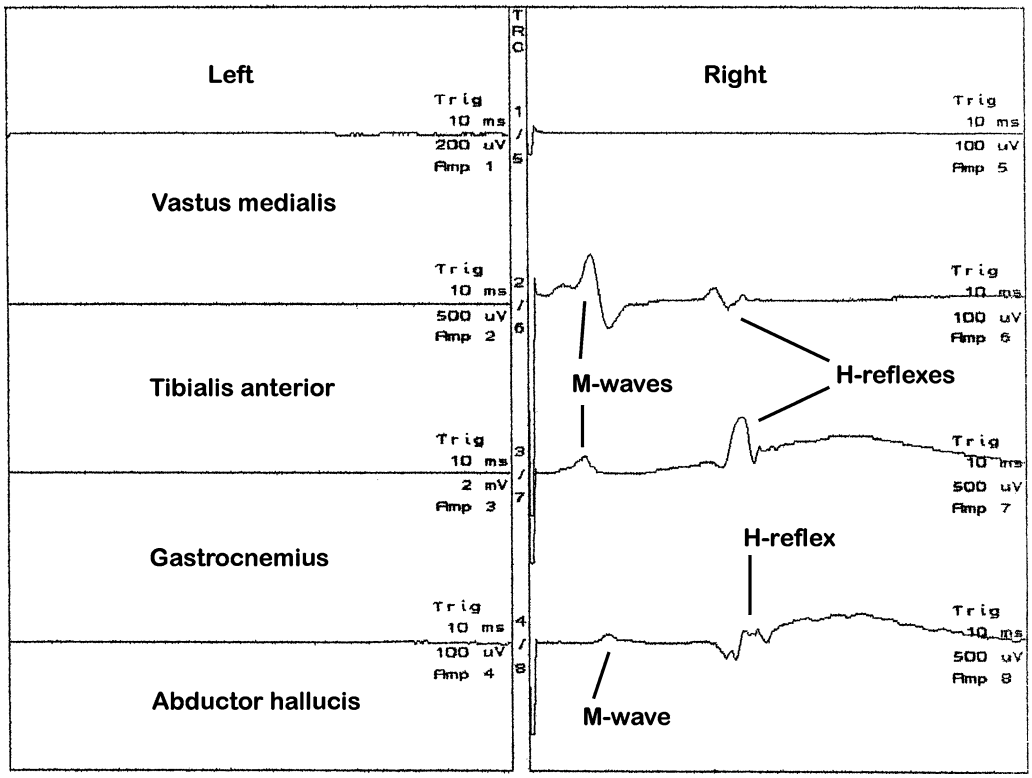


*Fig. 31. During removal of the calcified disk, the right lower-extremity tibialis anterior, gastrocnemius and abductor hallucis H-relfex amplitudes were decreased. 63-year-old female who was surgically treated with bilateral T8–T9 laminectomies with far lateral costovertebral exposures for removal of a large T8–T9 herniated disk.*

bilaterally normal (Figure 21). Baseline transcranial lower-extremity motor evoked potentials were present. With the scalp stimulation with the anode on the right motor evoked potentials were present in the left vastus medialis and tibialis anterior muscles and bilaterally in the gastrocnemius and abductor hallucis muscles (Figure 22). With the scalp stimulating anode on the left motor evoked potentials were present in the right vastus medialis, tibialis anterior, gastrocnemius and bilaterally in the abductor hallucis muscles (Figure 23). Following low intensity stimulation in the left popliteal fossa baseline H-reflexes were present in the left vastus medialis, tibialis anterior, gastrocnemius and abductor hallucis muscles (Figure 24) and with stimulation on the right were present in the tibialis anterior, gastrocnemius and abductor hallucis muscles (Figure 25). Following supramaximal stimulation unilaterally in the popliteal fossa baseline F-responses were present bilaterally in the tibialis anterior, gastrocnemius and abductor hallucis muscles (Figure 26, Figure 27).

During removal of the calcified disk the lower-extremity motor evoked potentials became absent with left scalp anodal stimulation (Figure 28) and with right scalp anodal stimulation (Figure 29). The left vastus medialis H-reflex became absent and the left tibialis anterior, gastrocnemius and abductor hallucis H-reflex amplitudes were decreased 47, 61 and 97% respectively (Figure 30). The right tibialis anterior, gastrocnemius and abductor hallucis H-reflex amplitudes were decreased 75, 88 and 97% respectively (Figure 31). The F-responses were present with 35% of the stimuli on the left and with 30% of the stimuli on the right. Baseline F-responses were present bilaterally with each stimulus. There were no changes in the tibial somatosensory evoked potentials. Post-operatively the patient's lower-extremity sensory function was normal. The lower-extremity pain was gone but pain was still present in the right T8 distribution. The lower extremities were weak, she could not stand and could not perform coordinated lower-extremity functions. Her strength improved and she was able to walk with assistance upon discharge 12 days after surgery. The 1.0 to 5.0% [17, 18] of the motor neuron pool activated by transcranial electrical stimulation was lost. The 24 to 100% [33] of the motor neuron pool activated by the H-reflexes was decreased from 47 to 100 percent. The 1.0 to 5.0% [34] of the motor neuron

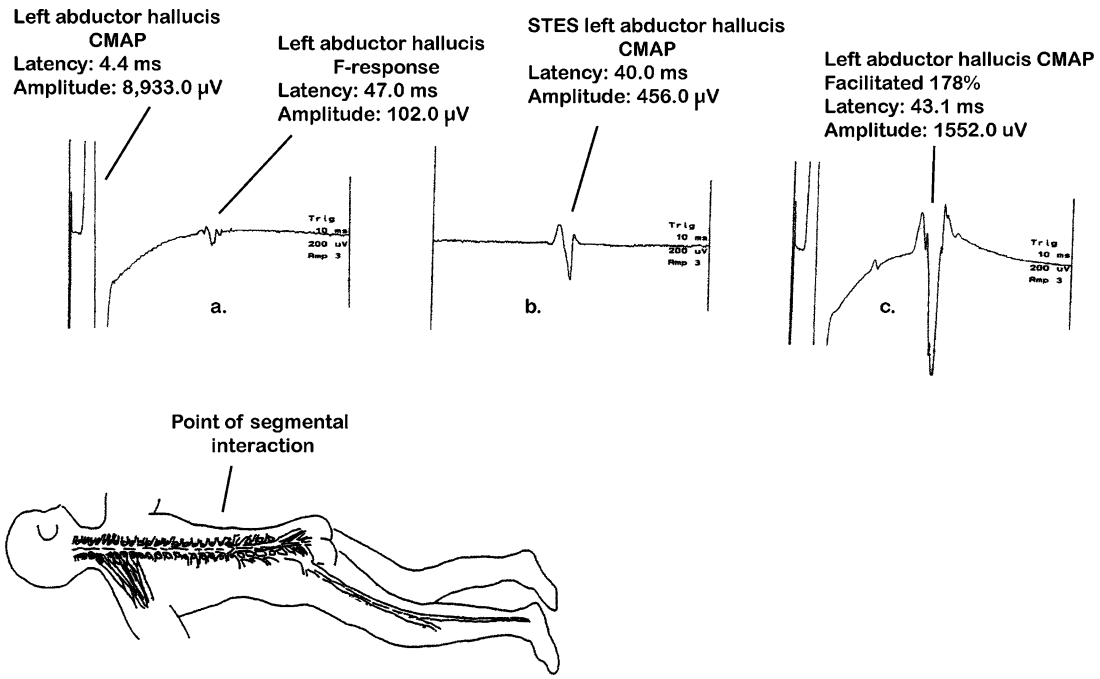


*Fig. 32. 'Facilitated Motor Neuron Monitoring'. In a 16-year-old female with idiopathic scoliosis, intraoperative supramaximal transcranial electrical stimulation (STES) is delayed 6.8 ms after supramaximal tibial nerve ankle stimulation, so that ascending and descending signals meet at the lumbosacral spinal cord segmental level. Interaction occurs and abductor hallucis motor neurons are facilitated. The abductor hallucis compound muscle action potential (CMAP) amplitude is facilitated 178% compared to the amplitude recorded without such interaction. a. Abductor hallucis F-response recorded following tibial nerve ankle stimulation. b. Abductor hallucis CMAP recorded following STES. c. Facilitated abductor hallucis CMAP recorded following simultaneous tibial nerve ankle and STES.*

pool activated by the F-responses was deceased by 65 and 70%.

The pattern of change in these motor systems in this patient indicates that these techniques may activate the same, different or overlapping populations of the motor neuron pool. Since the transcranial motor evoked potentials became absent and there was preservation of some H-reflex and F-response function perhaps H-reflex and F-response changes are better predictors of postoperative motor function than are transcranial motor evoked potentials. The H-reflex and F-response changes correlated with the patient's post-operative motor function while the transcranial evoked potentials did not [110].

*Clinical correlation summary*

1. If H-reflex amplitude and F-response persistence decrease less than 50% of baseline no postoperative deficit was observed [40].
2. Persistent H-reflex and F-response suppression greater than 90% correlates with the presence of a postoperative neurological deficit [40].
3. Transient H-reflex changes are not associated with a postoperative deficit [39].
4. Slow suppression of H-reflexes may be secondary to ischemic spinal cord compromise [39].
5. An abrupt suppression of H-reflexes and F-responses may be associated with mechanical injury to the spinal cord [39, 40, 110].
6. The degree of suppression of H-reflexes and F-responses reflects the severity of SCI [39, 40].
7. Hyperpolarization of caudal motor neurons occurs immediately upon SCI [39].
8. Compared to normal the interaction of motor neurons with H-reflex [71–74] and F-response [34, 62, 63] recordings is different with chronic spinal cord compromise. The sensitivity of the H-reflex and F-response to detecting acute SCI in the presence of a chronic upper motor neuron lesion has not been determined.
9. Combined H-reflex and SSEP monitoring improved the reliability of detecting spinal cord compromise compared to either procedure alone. H-reflexes exhibited more changes than SSEPs. These changes reflected changes in spinal cord gray matter function

related to acidosis and changes in hematocrit and blood pressure [109].

## SUMMARY

The results of animal studies [51–57] and intraoperative observations in humans [39, 40, 109, 110] have demonstrated that suprasegmental SCI changes the interaction of caudal CPG components. Changes in the interaction of caudal CPG components can be detected by reflex and F-response recordings. In humans H-reflex and F-response recordings can be used to alert surgeons about possible impending spinal cord damage. Ischemic [39], mechanical [39, 40, 110] and physiological [109] effects on the spinal cord can be detected with these recordings. H-reflex and F-response changes correlate with the postoperative status of patients.

H-reflexes and F-responses activate different populations of motor neurons [36, 62, 84–86]. MEPs interact with H-reflex and F-response activated motor neuron populations. Low intensity transcranial stimulation activates small motor neurons and inhibits H-reflex and facilitates F-response activated motor neurons. High intensity transcranial stimulation activates large motor neurons and inhibits H-reflex and F-response activated motor neurons [91]. MEPs may be more sensitive to detecting acute SCI than are H-reflexes and F-responses. H-reflexes and F-responses may be better predictors of postoperative motor function [110].

Monitoring spinal cord function using 'Facilitated Motor Neuron Monitoring' may be the most sensitive means of detecting acute SCI. This technique involves timing the transcranial motor and F-response activation of motor neurons such that facilitation occurs. The increased excitability of the motor neurons results in the peripheral MEP and F-response recordings being synchronized and amplified. Facilitation of abductor hallucis CMAPs was observed in 11 extremities in 7 patients during surgery for correction of idiopathic scoliosis [18]. The interval between electrical transcranial motor stimulation and tibial nerve stimulation was controlled so that F-response and transcranial stimulation activated motor neurons simultaneously. The mean percent increase in abductor hallucis MEP and F-response amplitude was: Left: 324.9 $\pm$ 50.9, range: 178-419%, Right: 456.5 $\pm$ 198, range: 244-685% [18]. Subtle changes in spinal cord function may be identified with greater sensitivity using facilitation of motor neurons [18] (Figure 32).

The practical application of H-reflexes and F-responses as intraoperative monitoring tools provides intraoperative neurophysiologists with a means of measuring the effects of acute SCI on different populations of motor neurons and the effect of SCI on the interaction of these different motor neuron populations. This provides neurophysiologists with means of monitoring those spinal cord systems that control complex motor behavior.

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