H Reflexes and F Waves: Physiology and Clinical Indications

Morris A. Fisher, MD
AAEM MINIMONOGRAPH #13:
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PHYSIOLOGY AND CLINICAL INDICATIONS

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AMERICAN ASSOCIATION
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CME STUDY GUIDE

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EDUCATIONAL OBJECTIVES

Following electric stimulation of peripheral nerves, motoneurons may be activated reflexly (H reflexes) or antidromically (F waves). This minimonograph reviews the physiology and clinical applications of these commonly used electrophysiologic responses. H reflexes and F waves are analyzed from the perspective of both the central and the peripheral nervous system, and it is emphasized that understanding these responses requires consideration of both central and peripheral influences. Established information as well as areas of controversy and theoretical concepts are discussed. This minimonograph is designed to enhance meaningful use and encourage further investigation of H reflexes and F waves.
H reflexes and F waves are clinically useful electrophysiological responses produced by electrical stimulation of peripheral nerves (Figs. 1–3). The former are reflex responses, while F waves result from antidromic activation of motoneurons. Despite this difference, it is reasonable to discuss these responses together. They occur at similar latencies, involve conduction to and from the spinal cord, reflect motoneuron activation, and occur at the interface between the peripheral and central nervous systems. They are also frequently helpful in analyzing similar clinical problems.

H REFLEX

Physiology. A reflex response in calf muscles following submaximal stimulation of the posterior tibial nerve, comparable in latency with the Achilles reflex, was initially described by Hoffman in 1918. In recognition of his original contribution, the response was named the H reflex by Magladery and coworkers.

A characteristic of H reflexes is a “late” response larger than the associated submaximal compound muscle action potential. This can occur only if there is amplification of the motor response centrally due to reflex activation of motoneurons. The reflex arc of the H reflex includes input from large, fast-conducting Ia fibers. The H reflex does not include muscle spindle activation, but the H-reflex arc is similar to that for the spindle-dependent phasic myotatic (“deep tendon”) reflex produced by muscle stretch. The presence of calf H and Achilles reflexes are highly correlated.

Intrathecal electrode recordings have been reported to show a minimum difference between afferent and efferent limbs of the H reflex consistent with a monosynaptic response. Recent intraneural studies, however, have questioned the exclusively monosynaptic nature of either H or phasic myotatic reflexes.

H reflexes are largest at submaximal nerve stimulation and inhibited by stronger stimulation. This inhibition has been attributed to “collision” of orthodromic impulses by antidromic conduction in motor axons. Large H reflexes, however, are obtained from calf muscles even with supramaximal stimulation if the stimuli are timed appropriately with phasic contractions of the muscles. This would not be true if H-reflex inhibition were dependent on fixed differences between afferent and efferent conductions.
“Collision” to be complete must occur distal to the motoneurons. Because rise times for motoneuron excitatory postsynaptic potentials (epspS) are at least 3 ms, and allowance must be made for at least one synapse, H-reflex afferent conduction would have to be at least 4 ms faster than conduction in the fastest efferent fibers for complete inhibition to occur. Such differences in afferent and efferent conduction times for H reflexes have not been established.

Inhibitory interneurons (Renshaw cells) are well described. Renshaw cells are activated by antidromic stimulation, and are distributed widely throughout a motoneuron pool, and discharge more strongly and with shorter latency with increasing stimulus intensity. Such motoneuron inhibition would involve two synapses, while H-reflex activation may involve only one. This difference in the onset of inhibitory in comparison to excitatory effects may be as brief as 0.5 ms and, therefore, well within a reasonable physiological range given motoneuron eppS rise times. Direct motoneuron to motoneuron connections are also present and could contribute to H reflex inhibition. Single fiber EMG studies of H reflexes have supported a process of active inhibition involving inhibitory synapses with stimuli of increasing strength. H-reflex studies themselves have directly demonstrated prominent central inhibition following supramaximal nerve stimulation.

In normal newborns, H reflexes may be widely distributed, but this is no longer true beyond the age of 2 years. Beyond infancy, H reflexes are regularly found only in calf muscles, primarily the soleus, and homologous forearm flexors. They are also frequently present in the quadriceps and occasionally in plantar foot muscles. This more restricted distribution of H reflexes reflects refinement of motoneuron pool activation with central nervous system (CNS) maturation.

The fraction of a soleus motoneuron pool activated in an H reflex is usually about 50%, but can be as high as 100%. The ratio of the peak-to-peak maximum H reflex to maximum M-amplitude ratio (H/M) provides a easily obtained measure of motoneuron pool activation, and therefore, excitability. Although there is considerable variability in H/M ratios, the H/M ratio for calf H reflexes is normally less than 0.7.

Because H reflexes involve activation of a portion of a segmental motoneuron pool, it is not surprising they are enhanced by maneuvers which increase motoneuron pool excitability.
reflexes are found with contraction or posttetanic potentiation in muscles where they are not normally present without such facilitation, i.e., small hand muscles. H reflexes are affected by changes in segmental CNS activity, and these responses have been used to study patterns of central reflex organization. H-reflex studies have shown results consistent with the reflex effects of both group Ia and group II afferents. Myotatic arcs between proximal and distal muscles also affect H reflexes. Detailed study of these functional myotatic arcs using H reflexes has indicated patterns of presynaptic inhibition of Ia fibers consistent with functional needs.

Characteristic excitability or recovery curves can be defined for H reflexes if test stimuli are given at varying intervals after a conditioning stimulus. These patterns vary with the level of stimulation (Fig. 4) and are not clearly established until 1 year of age.

H reflexes are inhibited by vibration, probably in part due to vibration-induced activation of large afferent fibers with resultant peripheral "busy line" interference and in part due to central presynaptic inhibition of afferent input. Activation of spindle receptors in antagonistic muscles may have a role in the usual vibratory inhibition of H reflexes.

**Technique.** In general, H-reflex testing can be performed using recording techniques and stimulation sites similar to that for a motor latency. The stimulus cathode should be proximal to avoid anodal block. Stimulation pulses of long duration, i.e., 1 ms, are used to preferentially activate large sen-
sory fibers. Stimulus frequency should be 0.2 Hz or less in order to avoid any effects on a response from a prior stimulus.

H reflexes are routinely recorded with the muscle at rest. Contraction of the recording muscle will enhance H reflexes. This can facilitate identification of H reflexes, and therefore, at times, be clinically useful.

Calf H reflexes are usually readily evaluated using percutaneous stimulation and surface recording. The posterior tibial nerve is stimulated in the popliteal fossa. Bipolar stimulation is usually adequate, but stimulation using an anode with a large surface area at the patella can decrease stimulus artifact and provide a more discrete cathodal excitation of the nerve in the popliteal fossa. Recordings should be made from the soleus. Although techniques vary, a standard and convenient location for the active electrode is medial to the tibia one-half the distance between the stimulation site and the medial malleolus. It is preferable that the indifferent electrode be placed on the Achilles tendon rather than the muscle, since this results in larger amplitude responses.

H reflexes in the forearm are recorded from the flexor carpi radialis (FCR) muscle. These responses can be obtained with surface recordings, but needle recording is a standard technique which can produce better defined responses. The recording electrode is placed at the junction of the upper one-third and lower two-thirds of the distance between the medial epicondyle and the radial styloid. The median nerve is stimulated percutaneously in the cubital fossa.

Uses. The upper limit of normal for calf H reflexes is 35 ms; that for FCR H reflexes 20 ms. Normal latencies for these responses are directly related to age, leg or arm length, and height. Use of these types of data enhances the sensitivity of H-reflex latency studies. Upper limits of normal for side-to-side latency differences are 1.5 ms for calf H reflexes and 1.0 ms for FCR H reflexes. Data from our laboratory indicate that the upper limit of normal for the amplitude difference between sides for surface recorded calf H reflexes is fourfold while the maximal comparable difference for needle recorded FCR H reflexes has been reported as threefold.

Calf and FCR H reflexes are found in the overwhelming majority of normal subjects. As with the physiologically similar phasic myotatic reflexes, however, it is important to recognize that symmetrically absent H reflexes are not necessarily abnormal and that the percentage of absent responses increases in the elderly.

H reflexes can be a sensitive test for polyneuropathies. Because H reflexes involve conduction in proximal fibers, H reflexes are a valuable technique for defining proximal nerve injury and may be abnormal even when more distal studies are unremarkable. H-reflex abnormalities, for example, can be an early, isolated, and thus important finding in patients with idiopathic polyneuropathy (Guillain–Barré syndrome). H reflexes can also be abnormal in plexopathies as well as radiculopathies. FCR H reflexes may be abnormal with C-6 or C-7 root injury.

It has long been recognized that H reflexes may be abnormally widespread in patients with CNS lesions and upper motor neuron (UMN) signs. An abnormal distribution of H reflexes, such as in the tibialis anterior or small hand muscles, therefore has direct clinical utility by demonstrating a disordered central motor system state. H reflexes in the tibialis anterior have been used as evidence for damage to developing CNS pathways in phenylketonurics, and tibialis anterior H reflexes have been found with a high frequency in patients with Huntington's disease as well as first generation "at risk" offspring.

In patients with hemiplegia, there is decreased potentiation of H reflexes with muscle contraction consistent with decreased background motoneuron facilitation. At the same time, H/M ratios tend to be increased in patients with CNS lesions and UMN signs consistent with increased central motoneuron pool excitability. Conversely, H reflexes during cataplexy are depressed. Changes in H reflexes after CNS lesions are time dependent. Increased H/M ratios which develop during the weeks to months following a cerebrovascular lesion are temporally related to the appearance of features of the UMN syndrome such as increase in tone, increase in reflexes, and extensor plantar responses. In patients with chronic UMN lesions, vibratory inhibition of H reflexes is less than expected possibly due to decreased presynaptic inhibition. In contrast, vibratory inhibition of H reflexes may be enhanced in patients with acute cerebral lesions. H reflexes are relatively well preserved acutely after spinal shock at a time when both Achilles reflexes and H-reflex recovery curves are depressed.

Within several months after central injury, H-reflex excitability curves can show an abnor-
nally rapid pattern of recovery associated with increased H/M ratios (Fig. 4). These patterns differ from those reported in patients with Parkinsonian rigidity or cerebellar hypotonia. Depressed H-reflex recovery curves consistent with decreased motoneuron excitability have been found in hyperkinetic children.

Studies of recovery curves as well as patterns of reciprocal inhibition of forearm flexor H reflexes in patients with various types of dystonia have defined physiological abnormalities even in clinically normal parts of the body. Because patients with chronic UMN syndromes have H reflexes in preterminal muscles, H-reflex analysis of patterns of reciprocal activity in these patients can often be performed with relative ease. In such patients, depression of the preterminal H reflex with weak stimulation of the tibial nerve has been found consistent with enhanced reciprocal inhibition of flexor muscles by extensors. In patients with chronic long tract motor dysfunction, altered patterns of reflex activity have been found in flexor and extensor muscles of both the arms and legs.

H reflex analysis of recurrent inhibition in patients with UMN lesions has indicated disruption of supraspinal inhibitory control in the abnormalities of movement in patients with UMN syndromes.

Finally, for those future-oriented, H reflexes can be used to monitor alteration in motoneuron excitability with space travel.

**F Response**

** Physiology.** F waves are low amplitude, ubiquitous responses, and somewhat variable in amplitude, latency, and configuration.

F waves are so named because they were originally recorded in small foot muscles. They were also initially considered reflex in nature. This view was subsequently challenged, particularly by the observation that motor units in F waves were present only if the same units were present in the direct motor response. The antidromic origin of F waves has been confirmed by the presence of F waves in deafferented animals and man as well as single fiber EMG analysis indicating that an F response requires direct activation of a motor axon.

Motoneurons are activated by depolarization at the low threshold initial segment and subsequent invasion of the soma. This is true whether the stimulus is orthodromic or antidromic. The shortest F-wave latencies are comparable even if usually 1 to 2 ms longer than H reflex latencies. In contrast to H reflexes, F waves are most prominent, and therefore, routinely elicited at high levels of stimulation. In addition, F waves must ultimately pass in an orthodromic fashion through the axonal initial segment which has been discharged by the preceding antidromic impulse. Because of this, the effect of altered motor neuron pool excitability on F waves is variable. If the motoneuron “backfired” is at a high level of excitability, i.e., already relatively depolarized, then the resultant motoneuron activation will occur rapidly and the resultant orthodromic axonal discharge will present at the initial segment at a time when it is still refractory. Increased motor neuron pool excitability could then result in decreased prominence of F waves, and this is consistent with observations in both animals and man. As a result, the effects of agonist and antagonist contraction are less consistent with F waves than H reflexes, and can vary with the physiological organization of a particular muscle.

F waves may not appear after each stimulus, are variable in configuration, and are low in amplitude (Fig. 3). This occurs because individual motoneurons are activated infrequently with antidromic stimulation and there are usually no more than several motor units in an F wave.

A priori, one would expect an F-wave latency range comparable to the normal range for conduction in motor axons. Although the subject remains controversial, there is circumstantial and direct evidence which argues for selective discharge of larger motoneurons in F waves. Renshaw cells inhibit larger motoneurons the least and Renshaw-cell activation provides a physiological model for selective discharge of larger motoneurons in F waves.

F waves frequently arise from an unstable baseline, and there may be superimposed axon reflexes. An additional uncertainty is the central “turnaround” time, i.e., the time between the start of antidromic activation and subsequent orthodromic discharge. This is commonly said to be close to 1 ms based on a statement of Eccles, but this has never been directly illustrated. In invertebrates, these central delays can be orders of magnitude greater. In humans, identical F waves have been recorded from foot muscles with latency variations of 3 ms.

For these reasons, there has been discussion about the number of F waves needed for adequate sampling. There are no fixed answers. Fifty to 100 or more have been used in clinical re-
ports.\textsuperscript{6,78,100,138} At the same time, minimal latencies from only three to five F waves yield values only about 1 ms longer than averaging 150 to 200 responses.\textsuperscript{83} For routine clinical purposes, there has developed a general consensus in the literature that analysis of 10 to 20 F waves represents a reasonable balance between feasibility—including patient tolerance—and adequate data. Whatever the uncertainties, the normal variability of F latencies of about 10% is comparable with the range of error for measurements of other commonly used electrophysiological responses.\textsuperscript{52,117}

Latencies are the most frequently reported parameters of F waves. F-waves latencies have been directly related to height, limb length, and, to a lesser degree, age.\textsuperscript{37,62,101} The sensitivity of individual F-latency measurements is increased by using data which consider these variables. F latencies are usually reported as minimal latencies, but the use of mean rather than minimal F-wave latencies minimizes the errors inherent in a single latency measurement.\textsuperscript{37} Because F latencies may not always be normally distributed, median latency values have been suggested as a more accurate description of F-response latencies.\textsuperscript{102} This, however, introduces a complexity which does not seem warranted for routine clinical use, including the need to record more F waves and the difficulty of statistical analysis.

Proximal in comparison with distal conduction can be evaluated by comparing F to M latencies.\textsuperscript{31,32} F-wave latencies can be used to estimate F-wave conduction velocities\textsuperscript{79} as well as proximal conduction times.\textsuperscript{65} As individual measurements, however, they are less accurate than latency values alone because of additional errors of measurement added to those already present with F-wave latencies themselves.\textsuperscript{139}

Parameters of F waves other than latency can add to the depth of F-response analysis. The difference between minimal and maximal latencies in a series of F waves (i.e., F chronodispersion\textsuperscript{94}) provides a measure of the range of conductions in the F waves. F-wave durations and amplitudes are related to both the size and number of motor units in a particular F wave.\textsuperscript{33} F persistence (i.e., the number of measurable F responses divided by the number of stimuli) indicates the antidromic excitability of a particular motoneuron pool. Analyzing the recurrence of individual motor units in a series of F waves measures the selectivity of F-response discharge. The ratio of F-wave amplitudes to that of the associated M waves (i.e., F/M ratios) is a measure of the proportion of a motoneuron pool activated by the antidromic stimulation. Given the variability of F-wave discharge, the routine use of mean F amplitudes has seemed preferable to using maximum F amplitudes for calculating F/M ratios.\textsuperscript{30,35,36,41,47,48} Because there may be a tendency for skewness in F-amplitude recordings, median F/M amplitudes have been recommended.\textsuperscript{96,101} As with median F latencies, the use of median F amplitudes introduces complexities which do not seem warranted for routine clinical use.

F-wave recovery curves can be defined by measuring F-wave amplitudes or persistences following preceding stimuli at varying intervals.\textsuperscript{84} Curves with similarities to those found with H reflexes are present.

As with H reflexes, vibration inhibits F waves.\textsuperscript{114} This observation indicates that vibration causes at least some postspinal changes.

F waves can help define CNS physiology. F-wave studies, for example, have shown changes consistent with activation of group II afferents\textsuperscript{21} as well as relative increased excitability of physiological flexor in comparison to extensor muscles.\textsuperscript{30}

**Technique.** F waves are most prominent with high levels of stimulation. A conventional stimulus intensity is 25% above supramaximal. Unlike H reflexes, long stimulus durations are not indicated, because there is no reason to preferentially activate large afferent fibers. Stimulus rate should be no faster than 0.5 Hz in order to avoid the effects of an earlier stimulus on a subsequent response.\textsuperscript{81} The cathode should be proximal to avoid anodal block. Adequate display of F waves usually requires an amplifier gain of 200 or 500 \( \mu V/\text{div} \) and a sweep of 5 to 10 ms/div. As such, one must evaluate the associated shorter latency and larger M wave using different recording parameters. Clearly identifiable F waves usually require responses greater or equal to 20 \( \mu V \) in peak-to-peak amplitude. Data from 10 identifiable, sequential F waves are suitable for most routine clinical work. The repetition of individual F responses requires at least 20 F waves for adequate evaluation.\textsuperscript{98,102}

Muscle belly-tendon recordings are standard for hand and foot muscles with the recording cathode placed over the motor point. For calf F responses, we prefer muscle belly recordings, because this decreases extraneous muscle activity when recording the low-amplitude F waves. The active electrode is placed as for H reflexes.

F waves are routinely recorded with the muscle
relaxed. Slight voluntary contraction can enhance F waves, and this can be helpful in their identification. At the same time, muscle contraction will alter F-wave parameters such as amplitude and increase the possibility of contamination by H reflexes.\textsuperscript{36,130}

F waves are diffuse in distribution, but recordings from proximal muscles are difficult, because the low-amplitude F waves are then superimposed on the associated M wave. F waves are therefore routinely only recorded from muscles of the hand, foot, and leg with standard stimulation sites at the wrist, ankle, and knee, respectively. F waves can be recorded in hand muscles stimulating in the axilla using collision techniques, whereby the orthodromic M wave from the axilla is blocked by colliding with antidromic impulses from the wrist.\textsuperscript{70}

F waves should be recorded such that each individual response, as well as the associated maximum M wave, are available for analysis. In our laboratory, for each series of F waves, we routinely note minimal and mean F latency, F chronodispersion, F persistence, and the F/M amplitude ratio. The latter is calculated as the mean of the amplitudes of the F waves (in microvolts) divided by the M-wave amplitude (in millivolts), both measured peak-to-peak.

Upper limits of normal for minimal F latencies in our laboratory are 31, 36, and 61 ms when recording from hand, calf, and foot muscles, respectively. Mean latencies are about 2 to 3 ms longer. Side-to-side differences of 2 ms are meaningful for both minimal and mean values when recording from the hand, 3 ms from the calf, and 4 ms from the foot. Tables of normal latency values are listed in standard texts.\textsuperscript{71} Normal data for minimal F-wave latencies related to height are available for small foot muscles\textsuperscript{437} as well as regression equation relating minimal latencies to age and height recording from the abductor pollicis brevis (APB) and abductor digiti minimal (ADM).\textsuperscript{101} Regression equations for F-wave latencies from these muscles as well as the calf (soleus) are also available relating both minimal and mean latencies to age and limb length.\textsuperscript{37} Based on evidence for better reproducibility, we routinely use mean rather than minimal F latencies for determining normality or abnormality of individual F-latency measurements. When recording from hand muscles, values 3 ms greater than predicted based on regression equations are abnormal, 2 ms borderline; comparable values for the soleus are 4 and 3 ms.

The highest reported normal values for F chronodispersion (mean ± SD) recording from the APB are 3.6 ± 1.2\textsuperscript{31}, from the ADM, 3.3 ± 1.1\textsuperscript{101}; from the soleus, 2.8 ± 1.1\textsuperscript{37}; and, from the extensor digitorum brevis (EDB), 6.4 ± 0.8\textsuperscript{54}. Normal F/M ratios in our laboratory, based on mean F amplitudes, are 2.2 ± 1.0% from the APB and 2.5 ± 1.2% from the soleus, equivalent to an upper limit of normal of about 5%. Mean values for F persistencies recording from the APB, ADM, soleus, and abductor hallucis are about 0.8 to 0.9, while in the antigravity antagonist tibialis anterior, EDB, and extensor digitorum communis muscles these values are about 0.3 to 0.4, but the range of normal is high for individual measurements.\textsuperscript{135,138} The normal maximum frequency of an individual response in a series of F waves from hand muscles is about 10\%,\textsuperscript{98,138} but the percentage of responses in which repeater waves may be seen is higher.\textsuperscript{78} A frequency for individual F waves as high as 58\% has been noted recording from the EDB (mean 21.5\%).\textsuperscript{102}

**Uses.** F latencies can be a sensitive measure in polyneuropathies. F latencies have been abnormal even when other measures of distal motor nerve conduction have been unremarkable.\textsuperscript{1,2,17,72,76,132} F waves are slowed in amyotrophic lateral sclerosis (ALS), comparable with the degree of slowing of motor conduction velocity. F-wave conduction studies have been normal in limb-girdle dystrophy\textsuperscript{75} but abnormal in myotonic dystrophy. The latter is consistent with other electrophysiological evidence for peripheral nerve dysfunction in these patients.\textsuperscript{83}

Prominent slowing of proximal F-wave conduction in comparison with distal motor nerve conduction studies has been found in patients with Guillain–Barré syndrome consistent with primary involvement of the central segment of a nerve in these patients.\textsuperscript{73} Proximally predominant abnormalities have not been found in uremia,\textsuperscript{34,93} diabetes mellitus,\textsuperscript{74} or Charcot–Marie–Tooth disease.\textsuperscript{70,93}

F latencies may be prolonged in proximal nerve or root injury.\textsuperscript{91,136} F-wave latencies from hand muscles may be slowed in syringomyelia, possibly due to disturbed function of roots as well anterior horn cells.\textsuperscript{96} Soleus F-wave latencies are abnormal in about 80% of patients with S1 radiculopathies.\textsuperscript{46} In general, slowing of F waves in the presence of normal distal motor conduction points to a proximal nerve or root injury requiring further electrodiagnostic or other clinical amplification.

A sensitivity to parameters of F waves other
than latency can add to the clinical value of F-response analysis. F waves may be absent and/or persistences decreased even in the presence of normal distal motor conduction. This can occur in the Guillain–Barré syndrome as well as in proximal nerve or root injury and ALS. In ALS, this decrease may be related to motoneuron loss, because it is correlated with the decrease in M-response amplitude.

The number of identical responses in a series of F waves may be increased with neurogenic atrophy consistent with a decreased number of motoneurons capable of responding to antidromic stimulation. Increased identical responses have also been reported in ALS and cervical myeloradiculopathies. These findings may be partially related to the increased discharge of responding motoneurons in patients with UMN syndromes. An increase in the percentage of responses following 100 supramaximal stimuli-containing recurring (i.e., "repeater") F waves has been found a sensitive measure of the carpal tunnel syndrome.

F chronodispersion may be prolonged in polyneuropathies. F chronodispersion tends to be larger in nerves with demyelinating in contrast to axonal injury and also tends to be relatively decreased with conduction block. F chronodispersion recorded from calf muscles has been found increased after standing compared to prior resting studies in patients with lumbosacral radiculopathies. This would be compatible with dynamic changes in nerve function affecting F-wave conduction.

A fasciculation may be followed by its own F wave. Analysis of these responses have supported a distal axonal origin for the vast majority of fasciculations.

Single fiber studies in patients with increased tone and reflexes indicate that antidromically activated motoneurons fire more frequently than normals. These same studies indicate that, in these patients, a frequently backfired motoneuron will discharge less frequently with activation by muscle contraction, while those motoneurons discharging infrequently will increase their firing rate. These observations are consistent with F-wave studies in deafferented animals.

In patients with CNS lesions, the normal relative prominence of F waves in resting extensor in comparison to flexor muscles may be disrupted. F-response amplitudes and persistences are decreased in clinically involved limbs compatible with a decreased central excitability state in patients studied early after unilateral cerebrovascular lesions when decreased tone and reflexes are common findings. F amplitudes and persistence can also be decreased by cerebellar stimulation consistent with cerebellar inhibitory outflow. By contrast, F persistences and average F as well as F/M ratios are increased in patients with well-established "spasticity." Huge F waves—as large as 75% of M-wave amplitudes—have been found in chronic tetanus. These findings were associated with a shortened or absent silent period, which has been attributed to failure of Renshaw-cell inhibition, and thereby indirectly support a role for Renshaw-cell activity in F-wave discharge.

In patients with upper motor neuron syndromes in comparison to normals, F-response latencies may be prolonged while durations and amplitudes are increased. These data are consistent with more but smaller, slower conducting motor units discharging due to increased central excitability, while larger motoneurons are blocked because of too rapid activation. Correlations between F-wave latencies, durations, and amplitudes are also disturbed in patients with motor disorders of central origin, consistent with altered patterns of motor unit discharge in these patients.

Finally, there is the promise that analysis of F waves may provide insight into the interaction between the central and peripheral nervous system. F/M amplitude ratios are increased in patients with polyneuropathy as well as spastic hyperreflexia. Log F/M values are normally directly correlated with neuromuscular efficiency as defined by twitch tension/M-wave amplitudes. This relationship is disturbed most prominently in patients with CNS, but also in patients with peripheral nerve dysfunction.

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