Once the practitioner has mastered the basic nerve conduction techniques, it is important to pursue more specialized methods of evaluating the peripheral nervous system. Additionally, nerves requiring needle excitation and less commonly studied nerves are of importance. From time-to-time patients may present with lesions affecting specific sensory branches that yield small amplitude responses or require averaging techniques to better define the desired waveform. With the majority of nerve conduction studies described in this chapter, the difficulty lies not in the inherent technique or nerve, but more so in unfamiliarity. Most, if not all, of the techniques described in this chapter can be mastered with simple practice and repetition.

## Motor Nerve Conduction Studies

### Late Responses

- F-Wave • Physiology of F-Wave Production
- Diagnostic F-Wave Techniques • F-Wave Clinical Utility
- H-Reflex • Physiology of the H-Reflex • Factors Affecting the H-Reflex • Diagnostic H-Reflex Techniques • Peripheral Nervous System Applications • Central Nervous System Applications

### Miscellaneous Techniques

- Residual Latency • Collision Technique • Refractory Period
- Clinical Utility • Sensory and Motor Nerve Refractory Periods • Refractory Periods in Muscle

### Motor Nerve Conduction Studies

- Nerve Root Stimulation • Erb’s Point (Supraventricular) Stimulation • Nerve Root Stimulation: Lumbosacral Plexus Conduction Latencies • Cranial Nerve Conduction Studies

### Nerve Root Stimulation

The purpose of attempting to stimulate the nerve roots and record CMAPs is primarily to evaluate conduction in various proximal nerves and assess neural conduction time across the plexus. Conduction times as opposed to conduction velocities are preferred as it is difficult to accurately measure the neural segment’s length. Nerve root stimulation is a relatively advanced nerve conduction technique and should only be attempted once the fundamentals of more routine procedures are mastered. Nerve roots can be stimulated electrically with needle electrodes and magnetically with a coil over the skin. Because root stimulation with needle electrodes can be done with standard apparatus, this technique is described in the following sections. It is presumed that with monopolar needle stimulation the root is depolarized just proximal to the intervertebral foramen.

### Nerve Roots C5–C6

Because the nerve roots are located under a relatively significant amount of muscle tissue, attempts to localize just one nerve root in a “blind” manner is rather difficult. Additionally, considerable expertise in addition to adjunctive fluoroscopy is required to accurately localize a particular root level. A more simple yet effective approach is to place the stimulating cathode (needle electrodes are required) just lateral to the spinous process (see below) so that it overlies the posterior arch of the cervical vertebrae, thus preventing the needle from piercing the nerve root or other vital neurovascular structures.

**Recording Electrodes.** When stimulating the C5–C6 nerve roots, recordings are typically obtained from the biceps brachii muscle. This muscle allows the practitioner to assess neural
impulses originating in C5–C6 nerve roots traversing the upper trunk, lateral cord, and musculocutaneous nerve.96

**E-1.** A surface E-1 electrode is positioned over the mid-point of the biceps brachii in an attempt to record from the muscle’s motor point, thus resulting in an initial negative deflection. Standard concentric needle electrodes may also be used; however, a more localized recording ensues, limiting the value of the CMAP’s amplitude. The latency with needle electrodes located deep within the muscle are as valid as those recorded with surface electrodes.102

**E-2.** The E-2 recording electrodes is usually a surface electrode positioned on the tendinous insertion of the biceps brachii.

**Stimulation.** Stimulation for all nerve root studies is performed with a monopolar needle serving as the cathode positioned on the posterior arch of the cervical vertebra. In the case of cervical nerve root excitation, the needle electrode is not positioned directly next to neural tissue but somewhat removed from it. A pulse duration of 0.1 ms or more may be required to achieve a supramaximal activation of the cervical nerve root under study. To optimally excite the C5–C6 nerve roots, a monopolar needle is inserted perpendicular to the skin 1 or 2 cm lateral and just inferior to the spinous process of C5 until the posterior spinal arch is encountered (Fig. 6-1). The needle electrode is then withdrawn several millimeters to ensure a volume-conducted spread of the depolarizing stimulus. A needle electrode 50 mm in length is recommended because the depth of needle insertion is usually between 25–40 mm. It is important to maintain the needle perpendicular to the skin surface to avoid directly encountering sensitive neurovascular or lung structures.

A similar needle electrode has been recommended to be inserted contralateral to the side of stimulation and serve as the anode.144 Using a rather strong current intensity may activate both left and right nerve roots, simultaneously allowing one to record from both sides should a two-channel instrument be available. When the contralateral side is examined, then the cathode and anode are reversed. A surface anode also can be positioned several centimeters distal to the needle insertion site should a recording obtained from one side at a time be desired.

**Instrumentation Parameters.** Specific instrumentation settings were not provided; however, similar latencies to those originally obtained should be approximated when routine settings are used.144 A sweep speed of 2 ms/div and sensitivity capable of displaying the entire response on the screen are sufficient to obtain the desired responses. Also, low- and high-frequency filters approximating 10 Hz and greater than or equal to 8 kHz, respectively, are used.

**Reference Values.** The anticipated latency to the biceps brachii muscle from the C5–C6 region is between 4.5 to 6.6 ms with a mean of 5.3 ± 0.4 ms.144 An expected left/right difference less than 0.6 ms is anticipated (Table 6-1).

**Nerve Roots C6–C8**

As previously noted, exact localization of specific nerve roots is difficult because of the overlying muscular tissue and volume-conducted spread of the depolarizing current. The posterior divisions and posterior cord of the brachial plexus can be evaluated by recording from the triceps muscle following C6–C8 nerve root excitation.96

**Recording Electrodes.** Because the motor point of the triceps muscle is rather difficult to locate, one may wish to consider using an intramuscular needle recording electrode. If a needle electrode is chosen, it should be a standard concentric or monopolar needle and inserted deeply into the main bulk of the triceps muscle. Although surface electrodes are capable of recording a response, an initial negative deflection may be difficult to reproduce in all patients.

**E-1.** A standard concentric needle electrode is positioned within the depth of the main bulk of the triceps muscle on the posterior or posterolateral aspect of the arm. This allows one to obtain a clearly recognizable deflection from the baseline whether in the positive or negative direction and should be noted for determining the onset latency. A recording from the triceps muscle permits the practitioner to assess the C6–C8 neural fibers traversing the brachial plexus’ posterior divisions and posterior cord. A surface electrode may be used; however, onset latency determination may be somewhat difficult because of less than distinct deflections from the baseline.

### Table 6-1. Cervical Nerve Root Stimulation

<table>
<thead>
<tr>
<th>Stimulation</th>
<th>Recording</th>
<th>Latency (ms)</th>
<th>L/R (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C5/C6</td>
<td>Biceps brachii</td>
<td>5.3 ± 0.4 (4.5–6.6)</td>
<td>0.0–0.6</td>
</tr>
<tr>
<td>C6/C7/C8</td>
<td>Triceps brachii</td>
<td>5.4 ± 0.4 (4.4–6.1)</td>
<td>0.0–0.6</td>
</tr>
<tr>
<td>C8/T1</td>
<td>Abductor digiti minimi</td>
<td>4.7 ± 0.5 (3.7–5.5)</td>
<td>0.0–0.7</td>
</tr>
</tbody>
</table>

As amplitude is not considered, one may use needle recordings to assess onset latency. The time to the abductor digiti minimi represents the transbrachial plexus latency as calculated by subtracting the axillary latency from the C8/T1 latency.144
If a standard concentric needle electrode is used, the E-2 electrode is the surrounding cannula. In monopolar needle recordings, a surface E-2 should be located on the olecranon.

**Stimulation.** Again, a monopolar needle cathode electrode is inserted perpendicular to the skin surface 1–2 cm lateral and just inferior to the spinous process of C6 and positioned a few millimeters superior to the posterior arch of C6 (Fig. 6-1). A supramaximal stimulation is delivered by optimizing the CMAP recorded from the triceps muscle. An anode can be placed in a similar position contralaterally or ipsilaterally as previously described.

**Instrumentation Parameters.** See C5–C6 nerve root stimulation.

**Reference Values.** A triceps brachii latency between 4.4 to 6.1 ms with a mean of 5.4 ± 0.4 ms is expected in normal individuals (Table 6-1). Additionally, a right-to-left difference of less than 0.6 ms is expected.141

**Nerve Roots C8–T1**

Perhaps one of the more commonly performed nerve root stimulation procedures involves excitation of the C8–T1 nerve roots. This may be a result of the regional diagnostic popularity of C8–T1 root, lower trunk, or medial cord compression secondary to possible anatomic compromise of these structures, i.e., the thoracic outlet syndrome. Although not discussed in detail at this time, evaluation of C8–T1 proximal nerve fiber conduction is one objective electrophysiologic way in which to evaluate possible neural compromise in a patient suspected of having the thoracic outlet syndrome.

**Recording Electrodes.** Locating the recording electrodes on the hand intrinsic muscles, either median- or ulnar-innervated muscles, allows one to assess C8–T1 neural fibers traversing the lower trunk and medial cord of the brachial plexus. Because of the long conduction route, a second proximal stimulation site (see below) is necessary to preferentially consider this segment of the C8–T1 fiber course.

**E-1.** A surface electrode is recommended to be positioned over the motor point of the abductor digiti minimi muscle. It is certainly acceptable to use a standard concentric needle electrode as long as quantitative amplitude measurements are not desired.

**E-2.** The E-2 electrode in a surface recording is placed just distal to the insertion of the muscle (see ulnar nerve conduction). If a standard concentric needle is used, the cannula serves as the E-2 recording electrode.

**Stimulation.** A 50-mm monopolar needle electrode is inserted perpendicular to the skin surface approximately 1 cm distal and lateral to the spinous process of C7 until the posterior bony arch is contacted (Fig. 6-2). The needle cathode is then withdrawn several millimeters. Again, a contralateral needle anode is possible or an ipsilateral surface anode located several centimeters distal to the needle insertion site. The onset latencies for left and right abductor digiti minimi CMAPs are recorded.

A second stimulus is then applied at the axilla on a line 25 cm in length from the mid-sternal notch with the arm abducted 90° and externally rotated (Fig. 6-3). This procedure is repeated for the contralateral limb. The onset latencies to the left and right abductor digiti minimi muscles are recorded to the CMAP’s initial departure from baseline. Onset latencies from axillary stimulation are subtracted from the nerve root excitation latencies to arrive at a transbrachial plexus conduction time.

**Instrumentation Parameters.** See C5–C6 nerve root stimulation.

**Reference Values.** The range of conduction times across the brachial plexus is 3.7–5.5 ms with a mean of 4.7 ± 0.5 ms (Table 6-1). Left-to-right conduction time differences range from 0.0 to 0.7 ms.

**ERB’S POINT (SUPRACLAVICULAR) STIMULATION**

A number of proximal nerves are not amenable to direct neural excitation because of the surrounding musculoskeletal structures. An indirect means is required to assess their integrity.
with respect to potential pathology. Specifically, techniques have been developed to examine a number of these proximal nerves, including long thoracic, suprascapular, axillary, musculocutaneous, and proximal radial nerves. Each of these nerves innervates a muscle that can easily be used to record from; however, the individual nerves are not readily accessible. As a result, it becomes necessary to deliver a rather strong depolarizing pulse to the brachial plexus as a whole in an attempt to activate in a supramaximal manner all of the above-noted nerves. This can be accomplished if the excitation pulse is delivered at Erb’s point.

**Recording Electrodes.** Because of the nature of the stimulus producing depolarization of the brachial plexus as a whole, one can record from multiple muscles simultaneously provided one’s instrument possesses more than one channel. This is advantageous because it limits the number of rather intense shocks delivered to the patient. Also, the original studies described below used intramuscular recordings with standard concentric needle electrodes, thus limiting the utility of amplitudes.

### Long Thoracic Nerve

The long thoracic nerve arises directly from cervical nerve roots C5–C7 and innervates the serratus anterior muscle.95,96 This muscle is of clinical value because its nerve originates proximal to the formation of the brachial plexus and may help to assess the extent of a possible brachial plexus injury. Should this nerve be spared, it suggests that the injury site is distal to the C5–C7 root level.

**Recording Electrodes.** Surface recording electrodes are typically used for this study; however, there is a report of monopolar needle recordings from this muscle.29,111 The use of surface recording electrodes is understandable given the proximity of this muscle to the chest wall and the potential for inadvertently piercing the intercostal space and possibly producing a pneumothorax.

**E-1.** An E-1 surface recording electrode is positioned over the fifth or sixth rib at the midaxillary line. In persons with significant subcutaneous tissue, an intramuscular E-1 may be preferable for recording the response’s latency. If a standard concentric needle is chosen, it is imperative to use proper technique to avoid the possibility of inducing a pneumothorax. The onset latencies to both the supraspinatus and infraspinatus muscles.

**Stimulation.** Stimulation is carried out at Erb’s point with an intensity and pulse duration capable of delivering a supramaximal excitation. A pulse duration between 0.5 and 1.0 ms is usually sufficient to achieve the desired result. Initially, the patient can be requested to turn the head opposite to the side of stimulation, making accurate identification of Erb’s point relatively easy. Once Erb’s point is localized, the patient’s head can be turned slightly toward the stimulus site just past midline. This is necessary to relax the skin and subcutaneous tissues allowing the practitioner to position the cathode (distal) and anode as deeply into the supraclavicular fossa as possible without producing undue patient discomfort. It is anticipated that the entire plexus will be activated, but an attempt should be made to confirm contraction of the serratus anterior by palpating the muscle over the anterolateral aspect of the rib cage.

**Instrumentation Parameters.** See nerve root stimulation.

**Reference Values.** Onset latencies to the initial deflection of the response are in the range of 2.6–4.0 ms (Table 6-2).29,111

### Suprascapular Nerve

The suprascapular nerve originates just distal to the formation of the brachial plexus’ upper trunk and is composed of nerve fibers from C5–C6.96 This nerve then proceeds posteriorly through the suprascapular notch to innervate the supraspinatus muscle. The nerve continues to course around the lateral aspect of the scapula to innervate the infraspinatus muscle. We describe latencies to both the supraspinatus and infraspinatus muscles.

**Recording Electrodes.** The majority of recordings from proximal muscles following Erb’s point stimulation are performed with standard concentric needle electrodes, although it is possible to also use monopolar needles.

**Supraspinatus Muscle.** E-1. A standard concentric needle electrode is located in the suprascapular muscle midway between the medial border of the scapula and the acromion.72 Using obstetric calipers, two separate distances were measured from the center of the cathode/anode at Erb’s point to the recording locations for the supraspinatus muscle. The two mean distances were 8.5 cm and 10.5 cm for respective E-1 locations. This allows the practitioner to choose one of the distances most appropriate to the size of the patient. Surface electrodes are of questionable value for this muscle because of the overlying trapezius muscle that may diminish the CMAP’s magnitude.

**E-2.** When using standard concentric needle electrodes, the cannula serves as the E-2 recording electrode. If a monopolar needle is used as E-2, it should be located several centimeters lateral to E-1.143

**Stimulation.** A surface cathode and anode are located at Erb’s point. The pulse duration should be between 0.5 and 1.0 ms with an intensity capable of producing a supramaximal response. Firm pressure applied at Erb’s point is often necessary to evoke an optimal response. See long thoracic nerve for details of head positioning.

**Instrumentation Parameters.** See nerve root stimulation.

**Reference Values.** The onset latencies to the initial deflection of the response is recorded for either distance chosen (Table 6-2).

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Recording</th>
<th>Latency (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long thoracic</td>
<td>Serratus anterior</td>
<td>2.6–4.0</td>
</tr>
<tr>
<td>Suprascapular</td>
<td>Supraspinatus (8–9 cm)</td>
<td>2.6 ± 0.07</td>
</tr>
<tr>
<td></td>
<td>Supraspinatus (10–11 cm)</td>
<td>2.7 ± 0.07</td>
</tr>
<tr>
<td>Suprascapular</td>
<td>Supraspinatus (7.4–12 cm)</td>
<td>2.7 ± 0.5</td>
</tr>
<tr>
<td>Suprascapular</td>
<td>Infraspinatus (13–15 cm)</td>
<td>3.4 ± 0.09</td>
</tr>
<tr>
<td>Suprascapular</td>
<td>Infraspinatus (16–18 cm)</td>
<td>3.4 ± 0.13</td>
</tr>
<tr>
<td>Musculocutaneous</td>
<td>Infraspinatus (10.6–15 cm)</td>
<td>3.3 ± 0.5</td>
</tr>
<tr>
<td>Axillary</td>
<td>Biceps brachii (19–21 cm)</td>
<td>4.6 ± 0.14</td>
</tr>
<tr>
<td></td>
<td>Biceps brachii (23–25 cm)</td>
<td>4.7 ± 0.15</td>
</tr>
<tr>
<td></td>
<td>Biceps brachii (27–29 cm)</td>
<td>5.0 ± 0.13</td>
</tr>
<tr>
<td>Axillary</td>
<td>Deltoid (15–16 cm)</td>
<td>4.3 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>Deltoid (18–19 cm)</td>
<td>4.4 ± 0.08</td>
</tr>
</tbody>
</table>

Recording performed with standard concentric needle electrodes and amplitudes not recorded.
Infraspinatus Muscle. E-1. A standard concentric needle electrode is inserted into the mid-portion of the infraspinatus muscle 14 cm and 17 cm from the stimulation site at Erb’s point. This distance is measured with obstetric calipers.
E-2. See supraspinatus muscle.
Stimulation. See supraspinatus muscle.
Instrumentation Parameters. See nerve root stimulation.
Reference Values. As for the supraspinatus, the initial onset latency is of interest (Table 6-2).

Musculocutaneous Nerve

The musculocutaneous nerve is the continuation of the brachial plexus’ lateral cord and consists of fibers from nerve roots C5–C6.96 This nerve innervates the coracobrachialis, biceps brachii, and brachialis muscles. It is possible to injure this nerve as a result of shoulder dislocations.

Recording Electrodes. Because of a relatively well defined motor point along a band in the middle of the biceps brachii muscle, surface electrodes can be used to obtain a CMAP.228 It is also possible, however, to use standard concentric needle electrodes (Table 6-2).
E-1. When recording with surface electrodes E-1 is located at the mid-point of the biceps brachii muscle. A standard concentric needle electrodes is placed in the muscle at 20 cm, 24 cm, or 28 cm depending upon the patient’s stature. As for the suprascapular nerve, this distance is measured with obstetric calipers from the Erb’s point stimulation site.
E-2. In the surface recording technique, E-2 is located on the biceps brachii tendon in the antecubital fossa. As previously noted, the cannula of the standard concentric needle electrode serves as E-2.

Stimulation. There are two stimulation techniques for eliciting a CMAP from the biceps brachii muscle. It is possible to either stimulate Erb’s point128 or directly excite the musculocutaneous nerve in the anterior aspect of the axilla.223 The latter stimulation site more selectively activates the musculocutaneous nerve as opposed to activating the entire brachial plexus and may be of use during repetitive stimulation studies obviating the need to excite the entire brachial plexus. Stimulation at Erb’s point is performed as previously noted for the long thoracic and suprascapular nerves.

When attempting to directly excite the musculocutaneous nerve in the arm, one can use either surface or needle stimulation. For surface stimulation, the cathode is positioned close to the insertion of the pectoralis major muscle on the humerus distal and somewhat posterior to its inferior margin, whereas the anode is located proximally. A needle cathode is located between the coracobrachialis tendon laterally and the axillary artery medially just proximal to the latissimus dorsi tendon. Similar parameters for needle stimulation previously noted are again used. The needle anode is located transversely at a distance of 3 cm.

Instrumentation Parameters. See suprascapular nerve.
Reference Values. When using a surface recording electrode the latency is measured to the initial deflection of the response (Table 6-2). Unlike needle electrodes, the amplitude of the surface-recorded CMAP best reflects the summed response of the muscle and may be used for diagnostic purposes. Amplitudes obtained with needle recording should be used with caution regarding any attempt to quantify axonal loss.

If the musculocutaneous nerve is directly excited in the axilla, one can anticipate onset latencies in the range of 1.3–3.6 ms for recording distances of 7–13 cm.223 Peak-to-peak amplitudes recorded with a concentric needle electrode can range between 6 and 32 mV.

Axillary Nerve

The axillary nerve, also called the circumflex nerve, is formed by nerve roots C5–C6.96 This nerve arises from the posterior cord of the brachial plexus. There are two muscles innervated by the axillary nerve: teres minor and deltoid. The relevant anatomy of the axillary nerve is that it courses through the quadrilateral (quadrangular) space, i.e., teres minor superiorly, teres major inferiorly, surgical neck of the humerus laterally, and long head of the triceps muscle medially. This nerve then travels posterolaterally around the humerus to divide into anterior and posterior neural branches to innervate the deltoid muscle. Dislocations or fractures of the humerus may injure the axillary nerve.

Recording Electrodes. The accessibility of the deltoid muscle permits surface-recording electrodes to be used.228 Standard concentric needle electrodes have also been used to record onset latencies.72
E-1. If a surface E-1 electrode is chosen, it should be secured to the most prominent portion of the deltoid muscle in the upper lateral aspect of the arm. The mid-portion of the muscle contains the motor point and a recording from this region should result in a well-defined negative onset. The use of a standard concentric needle electrode requires the needle to be placed deep in the substance of the muscle. As for needle recordings from other proximal muscles, there are several distances measured with obstetric calipers from the point of stimulation to account for different arm lengths. The distances for E-1 placement are 15.5 cm and 18.5 cm.72
E-2. For a surface recording, E-2 is located at the tendinous insertion of the deltoid muscle in the mid-arm area. As previously noted, the cannula is the E-2 electrode for concentric needle recordings.
Stimulation. See long thoracic and suprascapular nerves.
Instrumentation Parameters. See suprascapular nerve.
Reference Values. Onset latencies are similar for both standard concentric needle and surface recordings (Table 6-2). It is important to recall that should a needle recording be used, the needle electrode is placed deep into the substance of the muscle to avoid erroneously long latencies.229 Only surface recordings are optimal for comparing side-to-side amplitudes.

Nerve Root Stimulation: Lumbosacral Plexus Conduction Latencies

It is possible to evaluate conduction across the lumbosacral plexus by stimulating the nerve roots constituting the plexus and subtracting the time of conduction from either the femoral or sciatic nerves. The lumbar plexus is assessed by simultaneously exciting roots L2–L4 and recording a response from the vastus medialis muscle.96 The femoral nerve is depolarized in the infrapatellar space, i.e., teres minor superiorly, teres major inferiorly, surgical neck of the humerus laterally, and long head of the triceps muscle medially. This nerve is then stimulated at the anterolateral aspect of the thigh and a recording from the vastus medialis muscle is obtained. The sciatic nerve is then stimulated at the mid-thigh and a recording from the extensor hallucis longus (EHL) is ob-
Sacral Plexus (L5–S1). E-1-E-2. See sciatic nerve (Chapter 5).

Stimulation (L2–L4). A monopolar needle electrode 75 mm in length is used for the cathode. Approximately 2–2.5 cm lateral to the spinous process of the L4 vertebral body, a needle cathode is inserted perpendicularly to the skin in a sagittal plane (Fig. 6-4). The needle is positioned on the periosteum of the vertebral arch overlying the L4 nerve root. The anode, a similar needle electrode to the cathode, is located in the same position on the contralateral aspect of the body. Stimulation as described above allows one to activate the lumbar nerve roots bilaterally. It is important to rest the tip of the cathode and anode on the posterior bony aspect of the vertebral arch and not the inferior or superior interspaces. Sufficient current is delivered by adjusting both the intensity and pulse duration to achieve a supramaximal response. The patient should be sufficiently warned as this can be uncomfortable.

Stimulation (L5–S1). The same needle cathode and anode used for lumbar stimulation are also used for excitation of L5–S1 nerve roots (Fig. 6-5). In this instance, however, the cathode/anode are inserted perpendicular to the skin surface just medial and a bit caudal to the posterior superior iliac spine. Similar comments noted above for L2–L4 nerve root excitation also apply to activating L5–S1 nerve roots.

Instrumentation Parameters. See femoral and sciatic nerve conduction study instrumentation recommendations (Chapter 5).

Reference Values. Calculated means, ranges, and left/right differences are provided for lumbosacral nerve root stimulation (Table 6-3).

CRANIAL NERVE CONDUCTION STUDIES

Three of the cranial nerves can be readily studied with routine nerve conduction studies previously described for upper and lower limb peripheral nerves. The cranial nerves discussed in this text are: cranial nerve VII (facial nerve), cranial nerve V (trigeminal nerve, afferent component only), and cranial nerve XI (spinal accessory nerve). The techniques discussed are performed with surface stimulation and recordings and of proven value in the authors’ experience.

Cranial Nerve VII (Facial Nerve)

The seventh cranial nerve’s nucleus is located within the central nervous system in the pons. This nerve provides motor innervation to the muscles of facial expression, i.e., all facial muscles except those innervated by the trigeminal nerve (masseter, temporalis, and pterygoid muscles). Additional neural components mediated by the facial nerve include taste sensation to the anterior two-thirds of the tongue (chorda tympani nerve), sensation to a portion of the external ear and soft palate, and

<table>
<thead>
<tr>
<th>Table 6-3. Lumbosacral Nerve Root Stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulation</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>L2/L3/L4 (femoral nerve)</td>
</tr>
<tr>
<td>L5/S1 (sciatic nerve)</td>
</tr>
</tbody>
</table>

The above-noted times represent the latency across the lumbosacral plexus with femoral and sciatic nerve latencies subtracted from the absolute nerve root latencies. As amplitude is not considered, one may use needle recordings to assess onset latency.

Figure 6-4. L2/L3/L4 nerve root stimulation. Needle electrode placement for activation of the L2/L3/L4 nerve roots. Additionally, stimulation of the femoral nerve is depicted for the determination of transplexus conduction times. (From MacLean IC: Spinal nerve stimulation. In AAEM Course B: Nerve Conduction Studies—A review course. Rochester, MN, American Association of Electrodiagnostic Medicine, 1988, with permission.)

Figure 6-5. L5/S1 nerve root stimulation. L5/S1 nerve stimulation is shown along with sciatic nerve activation in order to determine the transplexus conduction times for the L5 and S1 nerve root fibers. (From MacLean IC: Spinal nerve stimulation. In AAEM Course B: Nerve Conduction Studies—A review course. Rochester, MN, American Association of Electrodiagnostic Medicine, 1988, with permission.)
finally the parasympathetic supply to the lacrimal and salivary glands. The anatomic course of the facial nerve can be separated into an intracranial and extracranial portion. Intracranially, the seventh nerve arises from the pons to enter the facial canal via the internal auditory meatus. The facial canal consists of the labyrinthine, tympanic, and mastoid segments of which the labyrinthine is the smallest. The termination of the mastoid segment, stylomastoid foramen, is where the facial nerve exits the skull to begin its extracranial course. After exiting the skull, the nerve enters the substance of the parotid gland and divides into a number of divisions to innervate various muscles of facial expression. These muscles are relatively easy to evaluate with nerve conduction techniques because of their superficial location. Also, the facial nerve can be readily excited anterior to the earlobe.

**Recording Electrodes.** As previously noted, only surface recordings are described as this method provides the best assessment of the total number of muscle fibers excited. Essentially any muscle can be used to record a CMAP following facial nerve activation. This gives the opportunity to selectively measure the different branches of the facial nerve (e.g., zygomatic, mandibular etc.). Facial muscles do not necessarily have well-defined motor points and subsequently may yield CMAPs with an initial positive deflection. One can attempt to reposition the electrodes, but this may not always result in a waveform with an initial negative onset. When this occurs, one is advised to accept the response and measure the onset latency to the beginning of the initial positive deflection. When calculating the amplitude of any CMAP, it is better to measure the potential from the initial negative deflection to the peak of the negative spike. If it is impossible to obtain an initial negative deflection, an initial positive to subsequent negative peak suf- fices. The major value in facial nerve studies with respect to prognosis is comparing side-to-side amplitudes.55,56 Hopefully, both sides of the face have similar appearing potentials for comparison purposes. There may be occasions when one side of the face has a pronounced positive deflection, whereas the contralateral side begins with the expected negative deflection. This poses a significant problem for comparative evaluations. If repositioning the E-1 electrode with the positive deflection does not resolve the problem, one cannot use two morphologically different CMAPs for comparative purposes. All factors being equal (recording electrode position, stimulus location, current pulse width and intensity, and manual pressure on all electrodes), a marked side-to-side amplitude discrepancy of greater than 50% is suspicious. This is a conservative estimate as normal side-to-side variations may reach approximately 3–20%.55,56,103,104 One may wish to proceed to a different muscle in the hope of finding relatively symmetric CMAPs for left and right sides of the face.

A second problem in facial nerve studies is a volume-conducted response from the masseter. When stimulating the facial nerve anterior to the earlobe it is relatively easy to directly activate the masseter muscle. In patients with profound facial nerve loss, a volume-conducted masseter CMAP can coincide with the expected facial nerve response’s position and be mistaken for a facial CMAP. The practitioner must be aware of this potential problem to avoid an erroneous conclusion that there is facial nerve function when indeed this nerve may have undergone complete degeneration. Should this be encountered, it behooves the practitioner to palpate the masseter muscle for a contraction when stimulating the facial nerve. There is also a recommendation to excite the facial nerve as it passes beneath the zygoma, thereby avoiding coexcitation of the masseter muscle.152 Of course, the latency is significantly shortened in this case, but the response is acceptable for side-to-side amplitude comparisons.

**E-1.** The E-1 surface-recording electrode can essentially be placed on any facial muscle desired. Three commonly examined muscles are the orbicularis oris, orbicularis oris, and nasalis (Fig. 6-6). Should the orbicularis oris be chosen for recordings, the E-1 electrode is usually positioned inferior to the eye’s lower canthus aligned with the pupil or at some point laterally to the outer margin of the eye. Some repositioning of the electrode may be required to achieve an initial negative deflection. For orbicularis oris recordings, E-1 is located at the angle of the mouth just lateral to where the upper and lower lips join. The nasalis muscle area is perhaps the easiest region to record from when exciting the facial nerve (Fig. 6-6). It is located by having the patient “crinkle” the nose as if a foul scent has been encountered. The prominent bulge just superior to the lateral nasal ala is the nasalis muscle area. The paretic side should be compared with the normal side in order to properly position the electrode. Recording from the nasalis muscles usually result in the best CMAPs.186

If amplitude is not of interest when performing facial nerve recordings, it is acceptable to use standard concentric needles placed into the muscle under investigation. Relatively sharp onsets of either a positive or negative direction should be obtained. It is important to remember, however, that the amplitude

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**Figure 6-6.** Facial nerve activation. The facial nerve is stimulated either anterior or posterior to the ear (S) with subsequent recording from any facial muscle. In the above diagram a recording from the left nasalis (E-1 : Ra) is depicted with E-2 (Rr) on the superior aspect of the nose away from muscle tissue. We believe the posterior stimulation is preferable. (From Ma DM, Liveson JA: Nerve Conduction Handbook. Philadelphia, F.A. Davis, 1983, with permission.)
obtained with needle recordings is not valid to be used for assessing axonal loss with respect to prognosis.

E-2. A surface E-2 is usually located in an area devoid of muscle if at all possible. The most likely location on the face is on the tip or bridge of the nose as it is mostly cartilage or bone (Fig. 6-6). Although this location is not “electrically silent,” it is a convenient location to assist in differential amplification and common mode rejection. Of course, should a standard concentric needle be used, the cannula serves as E-2.

Ground. As with other nerve conduction techniques, the ground electrode should be located close to E-1 between it and the cathode.

Stimulation. Surface stimulation can be applied to one of two convenient locations. A cathode may be placed either anterior or posterior to the earlobe (Fig. 6-6). Anteriorly, the cathode is pressed into the substance of the parotid gland several centimeters superior to the angle of the mandible. Slight superior/inferior movement may be required to optimally locate the facial nerve. Postauricular activation of the facial nerve is accomplished by positioning the cathode posterior to the neck of the mandible inferior to the mastoid process. Again, it is important to avoid direct masster activation.42-44,74 Despite recommendations in the literature, we strongly believe that all facial nerve stimulations should occur in proximity to the stylomastoid foramen, i.e., behind the ear.

Because there are several parameters one can measure following facial nerve stimulation, the characteristics of the stimulator must be specified. If facial nerve latency or amplitude is of primary interest, then either a constant-current or constant-voltage stimulator with sufficient current intensity capable of delivering a supramaximal response is all that is required. On the other hand, should one wish to measure the amount of current necessary to evoke just a minimal facial muscle contraction, a constant-current stimulator with a pulse width of 0.2–0.5 ms is necessary. It is very easy to stimulate the facial nerve intracranially with a magnetic stimulator. In contradistinction with magnetic root stimulation, it is possible to obtain maximal CMAPs, even with low stimulus strength. The magnetic coil is positioned over the parietal region. The facial nerve is depolarized just at the proximal part of the facial canal.186,199 Attempting to define the minimal amount of current that just produces a minimal twitch of a facial muscle is known as the nerve excitability test (NET).

To perform a NET study, the patient is comfortably positioned with a bright light directed across the side of the face so that sharp shadows are cast by the facial structures to aid in visualizing muscle contraction. The current intensity is slowly increased until a minimal twitch of a facial muscle is observed. The current is recorded and compared with a similar procedure for the unaffected side. The muscles usually observed for this minimal twitch are the orbicularis oris and orbicularis oculi. Of course, any other muscle may be used.

Instrumentation Parameters. Facial muscle CMAPs are considerably smaller than those obtained in the limbs and thus require a sensitivity of about 200–1,000 μV/div. The latency is rather short to the CMAP’s onset necessitating a sweep speed of about 1–2 ms/div. Filter settings are the same as those used for median nerve motor studies. Stimulator parameters are noted above.

Reference Values. Stimulation of the facial nerve anterior to the ear lobe yields a mean onset latency of 3.57 ± 0.35 ms (2.8–4.1 ms). Postauricular stimulation generates a mean onset latency of 3.88 ± 0.36 ms (3.2–4.4 ms).141 When comparing side-to-side amplitudes within the first 2 weeks following a lesion such as Bell’s palsy, sparing of 10% or more of the response compared to the uninvolved side suggests a good prognosis for recovery.55,56 Normal threshold stimulation currents are between 3.0–8.0 mA with a side-to-side difference less than 2.0 mA.125

Cranial Nerve V (Trigeminal Nerve)

That aspect of the trigeminal nerve capable of being examined with peripheral nerve stimulation involves primarily the sensory afferent fibers originating in the supraorbital nerve. This nerve can be located by palpating the supraorbital notch along the medial aspect of the supraorbital ridge. Afferent impulses arising from the cutaneous distribution of this nerve, vertex of skull to supraorbital area, travel to their cell body located in the trigeminal ganglion.60 From this ganglionic region, the action potentials travel into the pontine portion of the central nervous system and apparently diverge into two separate pathways. An oligosynaptic path proceeds superiorly to synapse in the principle sensory nucleus (Fig. 6-7). A second-order pathway then travels caudally to synapse with the facial nerve nucleus causing depolarization of this structure with an ensuing contraction of the orbicularis oculi muscle ipsilateral to the side of excitation. A second pathway from the point of divergence in the rostral pons courses caudally in the lateral medullary plate region a variable distance (Fig. 6-7). At some point in the lower medulla and several synapses later, two separate tracts head superiorly, both ipsilateral and contralateral to the side of stimulation. These two pathways eventually synapse with their respective facial nerve nuclei and induce a contraction of both orbicularis oculi muscles, which is the clinically observed blink.

The above described and presumed pathway describes the electrically induced “blink reflex.”125 Apparently, a relatively strong depolarization of this nerve is required to generate a blink reflex as cutaneous stimulation to the distribution of the supraorbital nerve does not produce the clinically observed blink response. The above-described pathway is believed to be slightly different than that taken by impulses generated with tactile stimulation of the cornea, i.e., the clinical blink reflex.

The electrical blink reflex examines the afferent trigeminal tract through the supraorbital nerve and the efferent facial nerve pathway to the orbicularis oculi muscle. It is possible to elicit a blink reflex with excitation of the infraorbital and mental nerves but with significantly less consistency than the supraorbital nerve. Facial muscles other than the orbicularis oculi do not typically yield a consistent blink response. Because of the time resolution of the electrodiagnostic equipment, it is possible to resolve both the early ipsilateral response (R1) and the later bilateral response (R2) (Fig. 6-7). By assessing the presence, absence, or delay of various components of the blink reflex, it is possible with some assurance to localize the lesion’s presumed site. Both central nervous system and peripheral nerve lesion affecting the supraorbital and facial nerves can be investigated with this technique.

Recording Electrodes. The most efficient manner to record the blink reflex is using two channels to detect the three responses generated with stimulation of one supraorbital nerve, specifically, the early ipsilateral R1 and the bilateral delayed R2.113 The ipsilateral E-1 to stimulus records two ipsilateral facial nerve responses, R1 and R2. The contralateral E-1 only records its orbicularis oculi R2. It is also possible to record the blink reflex with only one channel, but more stimuli are required.
E-1. Two E-1 electrodes are located bilaterally on the patient. Each is positioned as if one is performing a facial nerve study to the orbicularis oculi (Fig. 6-8).

E-2. There are a number of positions one may choose for E-2. It is possible to locate E-2 on the temporal region bilaterally or just superior to the nasalis muscle (Fig. 6-8). One can also use a single E-1 electrode placed on the tip of the nose and using a "jumper" cable connect it to both E-2 ports on the instrument's amplifier, i.e., a common reference for both channels.

Ground. The ground electrode can be placed on the chin, forehead, or cheek.

Stimulation. The cathode is positioned directly over the supraorbital notch, i.e., the supraorbital nerve (Fig. 6-8). With the cathode in this location, the anode is directed superiorly and laterally. It is important not to rotate the anode too far medially as the contralateral supraorbital nerve will become activated through anodal break excitation, thus producing bilateral R1 responses and confusing the diagnostic utility of the blink reflex. It may be necessary to rotate the anode about the cathode to optimize the effects of stimulus artifact, which can be a problem because of the close association between the cathode and recording electrode. As long as the above caution is kept in mind regarding anodal break excitation, there should be no difficulty with anode rotation. The stimulation site may be somewhat uncomfortable for patients and a slow stimulus rate of 1 Hz is preferable. Additionally, the stimulator prongs should rest lightly on the supraorbital nerve as this is a rather sensitive structure. Stimulator parameters similar to those used for other peripheral nerves are recommended. The current intensity of the stimulator is slowly increased until stable, reproducible, and maximal R1 and R2 responses are obtained. Because the blink reflex involves a multisynaptic pathway, there is some variability between successive activations of the supraorbital nerve (especially with respect to the R2) and at least 8–10 responses should be elicited with the shortest used for measurement. Following completion of the study on one side, the contralateral side is stimulated and responses recorded. Care should be exercised at all times as it is easy to concentrate on the CRT screen and allow the cathode to slip inferiorly into the patient's eye.

A particularly annoying problem during blink reflex studies is that of stimulus artifact that can obscure the R1 response. To minimize stimulus artifact production in the face it is crucial to remove all makeup, facial oils, and perspiration. This needs to be accomplished for the entire face and not just around the stimulus site as current from the stimulator will follow the path of least resistance and may still arrive at the electrodes prior to the response, resulting in possible R1 contamination. Attention to detail is especially important in attempting to generate optimal blink reflex responses.

Instrumentation Parameters. The R1 and R2 response is relatively small and requires a sensitivity of 50–200 μV/div. The delayed R2 necessitates a sweep speed of 10 ms/div. Filter settings are those used for routine motor studies.

Reference Values. Reference values are provided for both the ipsilateral R1 and R2, as well as the contralateral R2 (Table 6-4). Because of the variability of the responses, three standard

Figure 6-7. Blink reflex pathway. The afferent impulse traverses the supraorbital nerve and then enters the pons to divide into a rostral and caudal pathway. The rostral fibers synapse in the principal sensory nucleus and then descend to synapse with the facial nucleus. The fibers not connecting with the principal sensory nucleus descend in the lateral aspect of the medulla to then send a contralateral and ipsilateral group of fibers rostrally to synapse with both the left and right facial nucleus. The facial nerve then conveys the initial ipsilateral and shorter pathway to generate the R1, while the longer bilateral pathway produces the two R2 waveforms. (From Kimura J: Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice. Philadelphia, F.A. Davis, 1989, with permission.)
deviations are used to compute reference values. Temperature is not routinely measured as the face is usually quite warm. Distance is inconsequential in this study and therefore the above-noted anatomic landmarks are used. One can also combine information from the facial and trigeminal nerves to arrive at an optimal ratio of latencies to the orbicularis oculi muscle. Specifically, the indirect facial response through supraorbital nerve excitation R1 (R) is divided by the facial nerve latency to direct activation of the facial nerve (D) to arrive at R/D (Table 6-4). If R/D exceeds the normal limits because R1 is prolonged but D is normal, a lesion involving the trigeminal nerve is likely present. On the other hand, should the R/D ratio fall below the expected reference values D is prolonged, implying an abnormality of the facial nerve.

The same recording and instrumentation parameters can be used for recording the cornea reflex. The stimulation is easiest done electrically with a cotton thread soaked in saline and positioned on the sclera with anode as a surface electrode near the eye. The main difference from the blink reflex is that the afferent arc consists of thin (A-delta) fibers with a slow conduction. The R1 is normally not present and a bilateral response of 35–50 ms is expected. It is also possible to measure reflexes limited to the sensory and motor part of the trigeminal nerve itself. This can be done by eliciting the masseter tendon reflex by tapping on the chin with a reflex hammer and recording the responses of the masseter muscle bilaterally with surface electrodes. The advantage of this technique in comparison to the clinical examination is that unilateral absence or delay of the reflex can be shown. The last reflex is the masseter inhibitory reflex. The mentalis nerve is stimulated on the left and right with surface electrodes and recording is done bilaterally on the masseter with surface electrodes while maximally clenching the jaws. After unilateral stimulation, two phases of EMG interruption (silent period) on both sides occur at 10–15 and 40–50 ms latencies, respectively.

**Figure 6-8. Blink reflex stimulation.** Stimulation (S) of the right supraorbital nerve that can usually be palpated in the supraorbital notch. Bilateral recording from the orbicularis oculi with E-1 (Rr) and E-2 (Rr) electrodes positioned for optimal recording of the blink reflex responses. (From Ma DM, Liveson JA: Nerve Conduction Handbook. Philadelphia, F.A. Davis, 1983, with permission.)

![Figure 6-8. Blink reflex stimulation.](image)

**Table 6-4. Blink Reflex**

<table>
<thead>
<tr>
<th>Latency (ms)</th>
<th>Amplitude (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral R1</td>
<td>10.6 ± 0.8; &lt; 13.1</td>
</tr>
<tr>
<td>Ipsilateral R2</td>
<td>31.3 ± 3.3; &lt; 41.0</td>
</tr>
<tr>
<td>Contralateral R2</td>
<td>31.6 ± 3.7; &lt; 43.0</td>
</tr>
<tr>
<td>L/R; R1</td>
<td>1.2</td>
</tr>
<tr>
<td>L/R; S; R2</td>
<td>5.0</td>
</tr>
<tr>
<td>L/L; R/R; R2</td>
<td>8.0</td>
</tr>
<tr>
<td>R/D</td>
<td>2.6–4.6</td>
</tr>
</tbody>
</table>

L/R, left-right difference for shortest R1 latencies; L/R/S, left-right difference for R2 responses simultaneously obtained for a particular stimulus; L/L; R/R, R2 differences for the same side obtained with opposite-side stimuli, e.g., R2 latency on the right obtained with right-sided stimulation subtracted from R2 latency on the right obtained with left-sided stimulation; R/D, R1 divided by direct facial nerve response.

**Cranial Nerve XI (Spinal Accessory Nerve)**

The cervical portion of the eleventh cranial or spinal accessory nerve originates from cervical levels C1–C5. Individual nerve rootlets from these cervical segments proceed superiorly, fusing with each sequentially rostral segment until the spinal accessory nerve trunk is formed. It continues to course rostralward entering the cranium through the foramen magnum. While intracranial, this nerve joins with nerve fibers arising from the tentorial nerve to exit the skull by way of the jugular foramen. Once extracranial, the spinal accessory nerve separates from the tenth-nerve fibers to descend into the neck to innervate the sternocleidomastoid and trapezius muscles. The spinal accessory nerve is joined by additional nerve fibers from cervical segments C1–C4 via a communication with the cervical plexus while in the neck region. These fibers preferentially innervate the trapezius muscle after joining the spinal accessory nerve. After innervating the sternocleidomastoid muscle, the spinal accessory nerve is superficial just posterior to the posterior border of this muscle at approximately the muscle’s mid-point. The nerve then continues distally to innervate the trapezius muscle. The superficial location of the spinal accessory nerve posterior to the sternocleidomastoid muscle allows easy access to stimulation. As for previous NCSs, a technique using surface stimulation and recording is preferred.

**Recording Electrodes.** E-1. A surface E-1 electrode is located on the trapezius muscle approximately 5 cm lateral to the C7 spinous process on a line between this structure and the acromion.

**E-2.** This electrode is located over a lower thoracic spinous process. One may also position this electrode on the acromion.

**Ground.** Although one investigator recommends that ground be located on the acromion, positioning it between the stimulus and E-1 is preferred.

**Stimulation.** The cathode is located approximately 1–2 cm posterior to the posterior margin of the sternocleidomastoid muscle mid-way between the mastoid process and the suprasternal notch. This location approximates the superior margin of the thyroid cartilage. The anode is directed superior to the cathode.
Both cathode and anode should be maintained posterior to the sternocleidomastoid muscle as anterior placement may activate the brachial plexus or phrenic nerve. If the brachial plexus is activated, depolarization of the supraspinatus muscle may be mistaken for the trapezius muscle response because of its close proximity. When the spinal accessory nerve is excited, the practitioner should observe contraction of the trapezius muscle resulting in shrugging of the shoulder ipsilateral to the side of stimulation.

Instrumentation Parameters. The relatively short distance between the stimulus and recording sites requires a sweep speed between 1 and 2 ms/div. Other than sweep speed, routine motor nerve conduction study parameters are used.

Reference Values. The spinal accessory nerve should normally generate an onset latency of 1.8–3.0 ms. This is an important technique to master because spinal accessory nerve injuries are common and this technique can be quite productive when performing repetitive nerve stimulation in neuromuscular junction disorders or following lesions due to surgical procedures of the neck.

MISCELLANEOUS TECHNIQUES

A number of specialized nerve conduction techniques may be of clinical assistance under certain circumstances. Occasionally, alternative methods may help to define a particularly challenging diagnosis. The residual latency, collision study, and refractory period are electrophysiologic techniques that electrodiagnostic medicine practitioners should be capable of performing.

RESIDUAL LATENCY

It is well known that nerve conduction velocities in proximal nerve segments are greater than in the distal portion of the nerve. Because NCV in general is directly proportional to axon velocity within a few centimeters of the nerve’s termination compared to a region in the forearm. Consequently, in an upper limb a nerve cannot be expected to conduct with the same velocity as it reaches the distal regions of the limb.40,41 Consequently, in an upper limb a nerve cannot be expected to conduct with the same velocity within a few centimeters of the nerve’s termination compared to a region in the forearm. However, if one were to apply the forearm conduction velocity to the distance over which the distal motor latency were measured, a time difference between the predicted and observed distal motor latency would arise. This difference is referred to as the residual latency (RL).110,129 The concept of residual latency is perhaps best understood by using an example. Let us suppose a median nerve conducts with a velocity of 60 m/s in the forearm and has a distal motor latency of 4.0 ms over an 8-cm segment. If one were to assume that the NCV over the distal 8 cm also was 60 m/s, then the predicted distal motor latency would be 1.3 ms (60 m/s = 8 cm/DML; DML = 1.3 ms). The difference between the predicted and observed DMLs, residual latency, is 2.7 ms. In other words, there is a 2.7-ms discrepancy between the observed and calculated DML. This same principle may be applied to sensory as well as motor nerves only using the distal latency (to initial takeoff of the SNAP) as opposed to the DML. A general formula may be used to determine the residual latency: RL = DL – (cathode to E-1 distance in mm/forearm NCV in mm/ms).

The proposed diagnostic utility of residual latencies is to compare the distal aspect of the nerve segment to the more proximal aspect of the same nerve. Residual latency determinations should theoretically eliminate the intersubject variability of distal segment conduction by providing a smaller standard deviation and tighter normal range than distal latencies for both motor and sensory studies.110,129 For example, let us assume that two individuals have a DML for their right median nerve of 4.0 ms. This DML would be considered normal by most practitioners. There may be diagnostic significance, however, in this DML if one person had a forearm conduction velocity of 65 m/s as compared to the other subject with a forearm NCV of 52 m/s assuming the DML is measured over an 8-cm segment in both individuals. The respective RLs would be 2.8 ms and 2.5 ms. The implication in these findings is that the comparative difference between the predicted and actual DML is larger for the person with a forearm NCV of 65 m/s. This suggests that the distal segment of nerve for the subject with a forearm NCV of 65 m/s is conducting slower than for the individual with the lower proximal NCV. The question then arises as to possible pathology affecting the distal segment of nerve with the larger RL. Normative data are available for both median and ulnar nerves for motor and sensory studies (Table 6-5). Unfortunately, the clinical utility of the RL has only been examined in a limited number of patients and needs further study to assess its true clinical applicability.110,129

COLLISION TECHNIQUE

Most routine studies excite the distal portions of peripheral nerves where they are separated from neighboring nerves by sufficient distances to allow selective neural excitation. Unless one is using large current intensities and durations, a single nerve can usually be examined. The selective delivery of a depolarizing pulse becomes much more difficult when attempting to excite nerves in a proximal location such as the axilla. The close proximity of the median and ulnar nerves often precludes exciting either one individually. The result is a significant depolarization of multiple upper limb muscles with occasional overlap of distal electrical responses. For example, suppose a selective recording from the median-innervated thenar muscles is the desired goal. This should pose no particular problem when activating the median nerve at the wrist or elbow provided excessive current intensities are not used. The difficulty arises if a proximal conduction velocity of the median nerve is desired, i.e., axilla to elbow segment. It is highly probable that axillary stimulation will result in coactivation of both the median and ulnar nerves as well as possibly the radial nerve. The recorded CMAP from the thenar muscles may not be a true reflection of the activity arising solely from the median-innervated thenar muscles. There is a good chance that the observed CMAP reflects not only the median-innervated thenar muscle electrical activity, but may also contain volume-conducted potentials from

<table>
<thead>
<tr>
<th>Table 6-5. Residual Latency (ms)†10,129</th>
<th>Control</th>
<th>Neuropathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulnar nerve (S)</td>
<td>1.3 ± 0.3 (0.8–1.8)</td>
<td>2.4 ± 1.0 (2.0–3.0)</td>
</tr>
<tr>
<td>Ulnar nerve (M)</td>
<td>1.4 ± 0.8 (1.0–1.9)</td>
<td>3.0 ± 0.8 (2.7–3.3)</td>
</tr>
<tr>
<td>Median nerve (S)</td>
<td>1.3 ± 0.3 (0.8–1.8)</td>
<td>3.4 ± 1.2 (2.0–4.0)</td>
</tr>
<tr>
<td>Median nerve (M)</td>
<td>1.5 ± 0.3 (1.0–2.0)</td>
<td>3.3 ± 1.0 (2.7–3.8)</td>
</tr>
<tr>
<td>Median nerve (M)*</td>
<td>1.9 ± 0.2 (1.4–2.5)</td>
<td></td>
</tr>
</tbody>
</table>

S, Sensory RL; M, motor RL
† Median nerve RL (From Kraft GH, Halvorson GA: Median nerve residual latency: normal value and use in diagnosis of carpal tunnel syndrome.Arch Phys Med Rehabil 1983;64:221–226.)
the neighboring ulnar-innervated hand intrinsic muscles such as the first dorsal interosseous (FDI) and adductor pollicis (AP). If the action potentials conducting in the median nerve fibers reach the thenar eminence first, then a correct DML is detected with an appropriate proximal median nerve conduction velocity. The amplitude, however, may be erroneous as it reflects activity from both median- and ulnar-innervated muscles. Depending upon phase interactions of the two potentials, the amplitude may be larger or smaller than anticipated, although it is typically larger. This situation would change if there was preferential slowing of the median nerve conduction across the wrist segment; the fastest-conducting fibers would be prevented from reaching the thenar muscles either by a conduction block or axonal loss.

Stimulation of the median nerve at the wrist and elbow in the above case would accurately reflect this slowing, yielding both a prolonged DML and lower conduction velocity. Remember, even though the distal segment is supposedly removed from nerve conduction velocity determinations for the elbow-to-wrist segment, if the fastest fibers never reach the muscle, then the onset latency of the slower-conducting fibers determines the CMAP’s onset latencies for all stimulus sites and hence the respective conduction velocities.118 When performing the axillary stimulation, it is highly likely that the coactivated ulnar nerve impulses will reach the hand intrinsic muscles prior to the median nerve because of its slowing at the wrist. If the instrument’s sensitivity is relatively low or the ulnar nerve’s nearby muscles happen to coincidentally align their motor point with E-1, then an initial positive deflection is not observed and one may erroneously conclude that the observed CMAP’s negative onset latency reflects median nerve conduction. The prolonged antecubital median nerve latency combined with the shortened axillary median nerve latency results in a rather fast axilla-to-elbow conduction velocity that is not a true reflection of the median nerve’s proximal neural segment conduction. Should a positive deflection be observed with axillary excitation, it is clear that one is not observing median nerve fiber excitation and no conduction velocity should be attempted. Should the positive deflection be used to compute the conduction velocity, a similar situation to that described above results. The question remains, is it possible to examine the proximal segment of the median nerve without contamination from the ulnar nerve?

The proximal segment of the median nerve can be investigated by using coactivation of both the median and ulnar nerves at appropriately separated time or distance intervals. If a supramaximal stimulus is delivered to the axilla and coincidentally at the wrist to only the ulnar nerve, an interesting electrical event ensues. An early volume-conducted response from the ulnar-innervated hand intrinsic muscles is recorded from E-1 located on the thenar eminence secondary to ulnar nerve stimulation at the wrist. Because the origin of this CMAP is known to arise from the ulnar nerve, it is ignored. The impulse induced at the wrist also conducts proximally along the ulnar nerve. Recall that the axillary impulse is traveling distally in both the ulnar and median nerves. At approximately the mid-arm level, the proximally and distally propagating ulnar impulses collide and annihilate each other. The median nerve impulse, however, continues distally to reach the thenar eminence generating a pure median nerve response. Because the median nerve action potentials originated in the axilla, the CMAP produced is sufficiently delayed in time so as to not overlap with the volume-conducted CMAP generated at the wrist by ulnar nerve excitation. The end result is a pure median nerve CMAP arising solely from axillary excitation. It is then possible to calculate the conduction velocity from this segment involving only the median nerve fibers. Delaying the axillary stimulation slightly compared to that delivered at the wrist results in slightly more separation between the two recorded CMAPs should this be necessary in selected cases. The collision of the two induced ulnar nerve impulses is why the method is known as a collision technique. Of course, the principle of collision can be used for any nerve and not just the ulnar nerve. Additionally, applying collision principles and appropriately separated stimulus intervals, one also can examine slower-conducting nerve fibers by selectively blocking the faster-conducting axons. The collision technique also may be of assistance in selectively blocking conduction in anomalous neural conducting pathways.76,85,100,187

REFRACTORY PERIOD

Immediately following depolarization, that portion of an axon is completely inexcitable and cannot generate an action potential for a brief time. Within the next several milliseconds, the axonal membrane becomes relatively excitable and can produce an action potential, eventually returning to its resting state. It is possible to investigate the axon’s membranous electrical properties by delivering two successive stimuli with varying interstimulus intervals. By convention, the first excitation pulse is referred to as the conditioning stimulus. The second or test stimulus is then delivered at a predetermined interval. This terminology is used because the first excitation conditions the nerve, whereas the second depolarization tests the effect of the first stimulus on the nerve’s voltage-dependent ion gates. That time period after the conditioning excitation during which a test stimulus fails to evoke a response is referred to as the absolute refractory period. A depolarization pulse, irrespective of strength, is incapable of inducing an action potential. At some point in time a test response can generate an action potential but it is smaller than the conditioning response and delayed in latency compared to the anticipated time of observation with respect to when the nerve is activated. At some longer interval following the conditioning stimulus, the test response again resembles the conditioning response regarding appearance latency and amplitude. That segment of time following the absolute refractory period and detection of a test response identical to the conditioning potential is known as the relative refractory time.

The proposed physiologic mechanism generating the two aspects of reduced neural excitability is believed to be sodium inactivation.139 Recall that immediately following activation of voltage-dependent sodium gates, action potential generation, the same voltage-dependent gates close, thus significantly reducing sodium conductance. The closure of sodium gates is an intrinsic property of these proteinaceous channels and they remain closed for a finite period of time irrespective of an additional depolarizing stimulus.

It is important to remember that sodium channel opening is dependent upon a voltage difference and that their opening spans a finite time period. If the voltage applied to a nerve is slowly and progressively increased, it is possible to exceed the threshold level at which an action potential is generated. This occurs because only a few sodium channels are induced to open at a time. As new channels are opened at a slightly greater voltage difference, the previously opened channels are closed or beginning to close. The process of exceeding the nerve’s threshold without action potential production is called accommodation.
On the other hand, just after the passage of an action potential, sodium gate closure or sodium inactivation renders the membrane incapable of sustaining action potential induction. This time of complete inexcitability during which the sodium gates are closed accounts for the absolute refractory period. Sodium gate closure and subsequent opening occur over a finite time in that the gates do not all open and close simultaneously, i.e., this process occurs over a little less than 1 ms. As more and more of these voltage-dependent gates begin to recover from their mandatory inexcitable phase, at some point there is enough potentially excitable gates to again generate an action potential, but one of less magnitude that takes longer to generate the amount of current required to excite the next node of Ranvier, i.e., propagation. A stimulus of sufficient magnitude above the resting state’s previous supramaximal level can induce a synchronous opening of the available sodium gates to produce a relatively small and delayed action potential. With progressively longer interstimulus intervals, more and more sodium gates capable of being excited become available. Correspondingly, less and less current is required to generate an action potential. The increasing number of potentially excitable sodium gates allows threshold to be reached progressively earlier. Also, the larger number of sodium gates allows more current to flow, which in turn produces a larger action potential until the test and conditioning waveforms are the same. The time between sufficient sodium gates to just generate an action potential and enough to produce similar conditioning and test responses is the relative refractory period. Following the relative refractory period is a supernormal period during which the propagating test stimulus conducts at a velocity somewhat greater than normal.

Although the above description is correct, the actual technique requires propagated action potentials to be recorded at a distance from their production site. In other words, there may be a time where an action potential may be produced locally at the region of axonal membrane depolarization but it is of insufficient magnitude to result in propagation. Indeed, this is found to be the case and the time period between the absolute refractory period and the observation of a small and delayed propagating action potential is known as the critical interval of conduction. Of course, this time interval can best be measured with near-nerve microelectrodes. For practical purposes, however, the absolute and relative refractory periods can be conceptualized depending upon the detection or lack of a test stimulus following a conditioning pulse.

Clinical Utility

By investigating the refractory periods of peripheral nerves, it is possible to assess the effects of various disease states. In experimental demyelinating diseases of the peripheral nervous system, experimental allergic neuritis, and diphtheria-induced demyelination, the refractory periods are significantly increased. In demyelination secondary to lyosphosphatidylcholine, refractory periods demonstrated a better correlation with histologic findings than did conduction velocities. Of interest is the finding of abnormal refractory periods in patients with multiple sclerosis, suggesting that peripheral nervous system membrane characteristics may be altered in this disease. Also, in patients with various peripheral neuropathies, the relative refractory period appeared to be a more sensitive indicator of abnormality involving neural structures than the absolute refractory period. On the other hand, hypokalemia has been found to actually shorten the relative refractory period. People with motor neuron diseases also display prolonged refractory periods.

A limited number of investigations have been performed to determine the clinical utility of neural refractory characteristics in disease states. The relative ease with which refractory periods can be applied to the peripheral nervous system with commercially available equipment should allow investigators to pursue this area in the future. Direct muscle stimulation reveals that in muscle suffering from various forms of muscular dystrophy, the absolute and relative refractory periods are reduced compared to normal. Denervated muscle, on the other hand, reveals a prolongation in both the absolute and relative refractory times. The pathophysiology underlying these changes remains to be completely elucidated.

Refractory period observations have been performed in animals for quite some time but this requires removal of the nerve. As this is unacceptable for human studies, a simple yet elegant methodology has been developed that can be performed routinely by most practitioners with the appropriate equipment. The actual methodology requires that one’s instrument have the capability of delivering two stimuli with varying interstimulus intervals. With this type of stimulus delivery, it is relatively straightforward to examine either mixed or pure sensory nerves.

Mixed Nerve Studies. To perform mixed nerve refractory period measurements, the technique of Gilliatt and Willison can be used.

Recording Electrodes. E-1. The E-1 surface recording electrode is located over the median nerve just proximal to the antecubital fossa.

E-2. A surface E-2 electrode is positioned over the insertion of the deltoid on the lateral aspect of the arm.

Ground Electrode. The ground electrode should be secured to the forearm just distal to E-1.

Stimulation. The median nerve is excited at the wrist in a similar manner to that used for routine median nerve motor studies except the cathode is located proximal, i.e., pointing toward E-1. A pulse duration of 0.2 ms may be used. Initially, a minimum threshold and single supramaximal stimulus is delivered. The supramaximal response is then used to determine the optimal recording electrode position for the mixed median nerve waveform.

An instrument with the capability of delivering sequential pair of stimuli from the same cathode at predetermined interstimulus intervals is required. Specifically, it is helpful if interstimulus intervals between two successive stimuli of 0.1 ms can be delivered. A stimulus exceeding the suprathreshold magnitude 4–6 times is delivered at 0.1 ms intervals following the conditioning stimulus to determine the absolute refractory period.

Once the absolute refractory period is determined it is possible to determine the relative refractory period. Beginning at the point when the second response was first detected with the maximal stimulus, a response is attempted at the next 0.1-ms interval. In this instance, however, only enough current is used to produce a detectable response. This procedure continues at increasing intervals until the originally determined baseline stimulus is reached. That stimulus interval between a just visible response at 4–6 times stimulus threshold to the resting value defines the relative refractory period. Continuing to increase the interstimulus interval and measuring minimum stimulus excitation levels allows one to calculate the supranormal period. The time when the original threshold value is required to just elicit a potential defines the cessation of supranormality.
It is important to note that delivery of the high-intensity currents/voltages required to properly study the refractory periods can be quite uncomfortable and not tolerated by all patients. Additionally, proper skin site preparation with commercially available abrasives to reduce impedance is recommended.

**Instrumentation Parameters.** A sweep speed of 1 ms/div and amplifier sensitivity of 20 µV/div should suffice for most persons. Filter settings of 10–20 Hz to 2 kHz will yield detectable responses.

**Reference Values.** The absolute refractory period measured with above technique was found to be less than 0.6–0.7 ms. In other words, the second potential was first observed at an inter-stimulus interval of 0.6–0.7 ms. The relative refractory period lasted between 2.5 and 3.5 ms. Following the relative refractory period, a supranormal time interval extended for 5–8 ms.

**Sensory and Motor Nerve Refractory Periods**

In addition to examining mixed nerves, it is also possible to measure the refractory periods of pure motor and sensory nerves using paired stimuli techniques similar to those noted above. Sensory nerve refractory periods can be calculated by placing stimulating ring electrodes, cathode proximal, on the skin site. In the peroneal nerve, the absolute refractory period measured with above technique was found to be less than 0.6–0.7 ms. In other words, the second potential was first observed at an inter-stimulus interval of 0.6–0.7 ms. The relative refractory period lasted between 2.5 and 3.5 ms. Following the relative refractory period, a supranormal time interval extended for 5–8 ms.

The absolute refractory period for the median and sural sensory nerve fibers was approximately 0.7 ms. In these studies the relative refractory period was assessed by both amplitude and latency criteria. Amplitude criteria suggested relative refractory durations of 5 times the absolute refractory period, whereas latency criteria revealed a length of 3 times the absolute refractory period.

Refractory periods in motor nerves also can be studied; however, the rather long duration of the conditioning CMAP interferes with the necessary latency measurements of the test response. An alternate method of calculating the refractory times other than direct paired stimuli is required. A collision technique (see above) was developed to eliminate the interfering effects of the first stimulus while continuing to investigate the interactions of the conditioning and test responses. For example, surface recordings are obtained over the hypothenar eminence while CMAPs resulting from paired stimuli at the axilla combined with a solitary pulse at the wrist are examined. With this technique, the conditioning stimuli is blocked when it collides with the action potentials propagating toward it from the wrist. The second stimulus from the axilla is then free to propagate to the hypothenar muscle and produce a response provided the nerve is not in the absolute refractory period induced by the axillary conditioning response. The CMAP resulting from wrist stimulation is sufficiently displaced from the axillary CMAP to offer no interference. By appropriately adjusting paired stimuli at the axilla, one is free to investigate the membrane properties regarding refractory characteristics of pure motor nerves in a similar manner used for sensory and mixed nerves. Absolute refractory period for the median and sural sensory nerve fibers was approximately 0.7 ms. In these studies the relative refractory period was assessed by both amplitude and latency criteria. Amplitude criteria suggested relative refractory durations of 5 times the absolute refractory period, whereas latency criteria revealed a length of 3 times the absolute refractory period.

**Refractory Periods in Muscle**

In addition to measuring the refractory periods in nerve, it is also possible to determine the absolute and relative refractory periods in muscle fibers. Using the paired stimulation technique, direct muscle fiber stimulation can be performed while recording from single muscle fibers. The studies reveal that the absolute refractory period in muscle with a stimulation intensity 25–35% above the conditioning stimulus is 4.12 ± 1.73 ms (2.69–8.13 ms). The relative refractory period for muscle fibers is 5.99 ± 2.7 ms (2.88–12.40 ms). A supranormal period also can be observed at 10.19 ± 3.2 ms (4.86–15.7 ms). As for nerve, the waveforms in the relative refractory period are smaller and demonstrate an increase in the rise time and a longer total duration.

**LATE RESPONSES**

Following the CMAP or M response in motor NCS a number of secondary or late responses can be observed on the CRT several milliseconds later. Depending upon the particular physiologic conditions, there are three late responses of interest that are discussed in this section: F-wave, H-reflex, and axon reflex. These three individual waveforms are essential to gain insight into the physiologic mechanisms underlying the peripheral and central nervous systems. Additionally, a number of investigators have proposed various techniques whereby the late responses may be used for diagnostic purposes with regard to pathology involving specific regions of the peripheral nervous system. Each response is discussed in detail and their clinical relevance to particular disease entities is noted during the remainder of this text when appropriate.

**F-WAVE**

In 1950, Magladery and McDougal first detected a small and late response occurring after the CMAP elicited from the peroneal innervated foot muscles and designated it the F-wave (F from foot). The above two investigators noted that the F-wave increased in amplitude and reached a maximum at supramaximal stimulation of the peripheral nerve, varied in amplitude from subject to subject, displayed different morphologies from one stimulus to the next as well as slightly different latencies, and that not all CMAPs were followed by an F-wave (Fig. 6-9). Of interest was the observation that moving the stimulus site from the elbow to the distal forearm resulted in a shortening of the CMAP but a prolongation of the F-wave from 26 ms to 31 ms. The decrease in the CMAP onset latency was expected because the excitation site moved closer to the muscle from which the response originated. The increase in F-wave latency, however, suggested that the neural impulses generating this response had a longer pathway to travel prior to reaching the hypothenar muscles. Additionally, F-waves were noted to be absent when a stimulus was delivered to the ulnar nerve distal to a complete procaine block of the nerve. Faced with these observations, Magledery and McDougal concluded that the F-response could not arise from repetitive firing of the motor nerve, neuromuscular junction, or muscle but must be a delayed potential that first travels centripetally toward the central nervous system and then centrifugally back to the muscle. The F-wave, therefore, somehow involved the central nervous system via motor neuron discharge, either by a backfiring of the anterior horn cells or through a reflex mechanism involving afferent-to-efferent central
investigators assumed that the F-wave was a reflex response mediated through an oligosynaptic or polysynaptic pathway requiring afferent fiber activation. Shortly after the F-wave was first described, a group of investigators suggested that instead of a reflex pathway, the F-wave was produced by a motor neuron activated through an antidromic impulse, i.e., a backfiring mechanism. Sectioning the posterior roots supplying limbs to be examined in both animal and human subjects demonstrated little change in the production of F-waves.

Further support for the lack of a reflex or synapse involved in F-wave production occurred when single-fiber electromyography demonstrated essentially the same delay or jitter (see Chapter 8) from one F-wave to the next as observed in the same muscle fiber. In other words, only one neuromuscular junction or synapse was involved in F-wave generation that was present in the muscle. If a reflex were involved in the F-wave, a synapse interposed between the afferent and efferent neural pathway would be necessary. This synapse would significantly add to the transmission variability from one F-wave firing to the next, thus increasing the jitter. When removal of the anatomic pathways conveying the afferent electrical impulses resulted in F-wave generation, it had to be concluded that the F-wave did not depend on a reflex. The only alternative clearly suggested that following activation of a mixed nerve, a small late response was observed that originated from the antidromic motor impulses propagating centripetally and activating a small population of motor neurons. The limited number of excited motor neurons then generated an impulse that traveled orthodromically in several motor nerves to activate the muscle fibers they innervate. These reactivated motor units were the potentials designated as the F-wave.

Given that F-waves are believed to be generated by an antidromic backfiring of motor neurons, it is reasonable to ask why the F-wave amplitude is significantly less than the previously generated CMAP. When considering the amplitude of the F-wave, it is important to first consider factors that may affect the magnitude of the motor units contributing to the F-wave. The number of muscle fibers and their cross-sectional diameter comprising a particular motor unit and how closely these fibers are arranged in space can influence a potential’s amplitude. The more fibers per motor unit and a given area, the more voltage produced during depolarization and the bigger the F-wave observed. Also, the total number of motor units activated and their temporal dispersion with respect to each other directly affect F-wave amplitude. Several motor units temporally synchronized (superimposed) yield a larger potential than if they were more separated in time. The implication in the relatively small F-wave amplitude compared to the CMAP is that only a small subpopulation of available motor neurons is activated by all of the antidromically propagating motor impulses. An explanation for this assertion is obviously required. Renshaw observed that following dorsal root section in cats, stimulating a motor nerve resulted in the anticipated large antidromic impulse being conducted toward the central nervous system. Recording directly from the same motor nerves revealed a second impulse only 2–3% of the original amplitude that required a central turning around time or delay of approximately 1 ms. These neural impulses correspond to the F-wave response described by Magladary and McDougal even though Renshaw recorded them from the nerve, whereas the F-wave was observed in muscle. In other words, Renshaw documented the neural response responsible for the muscular potential produced by the backfiring neural impulses. In both animal and human investigations, the F-wave is between 1 and 3% of the CMAP, which corresponds nicely to the percentage of total nerves found to be activated and represents roughly 1–2% of the available motor neuron pool. When individual F-waves are examined with needle recording techniques, each F-wave is found to consist of 1–3 motor units, roughly supporting the previously noted data. The actual explanation for the small number of motor neurons activated by an antidromic impulse is poorly understood.

In order to consider the relatively few motor neurons activated following depolarization of an entire mixed nerve, it is first necessary to briefly consider the anatomy of the anterior horn cell. The anterior horn cells concerned with motor function consist of a relatively large soma or main body with several substantial projections emanating from it. One rather large projection is the axon destined to innervate all of the muscle fibers innervated by that motor neuron. The unmyelinated portion of the motor neuron forming the junction between the last myelinated segment of the axon and the main portion of the soma is referred to as the axon hillock. The axon hillock’s threshold for depolarization is approximately one-half that for the remaining portions of the motor neuron. Dendrites are the remaining projections from the soma. In excess of 6,000 synapses with
other dendrites occur over the motor neuron’s soma and generate either excitatory or inhibitory impulses.\textsuperscript{11,198} The net summation of excitatory and inhibitory potentials determines the overall excitability of the motor neuron and whether it generates a depolarization impulse of sufficient magnitude to excite the axon hillock region producing a propagating action potential. Within a short distance distal to the axon hillock, a number of spinal motor neurons possess a \textit{recurrent collateral}, which is a neural branch given off from the axon that proceeds back into the ventral horn of the spinal cord to synapse with inhibitory interneurons known as Renshaw cells (Fig. 6-10). Renshaw cell activation tends to suppress activation of motor neurons they synapse with by generating inhibitory postsynaptic potentials (IPSPs).\textsuperscript{55,198} As an antidromic impulse traverses the axon toward the ventral horn, the axon collateral conveys an action potential to the Renshaw cell, which in turn tends to suppress the motor neurons it synapses with.

The final level of excitability of the motor neuron pool, therefore, is dependent upon multiple excitatory and inhibitory influences from various aspects of the central and peripheral nervous systems.\textsuperscript{176,177} When a mixed peripheral nerve is stimulated with a supramaximal stimulus, the large number of antidromic motor action potentials enter the ventral horn to find the resting membrane potentials of their respective motor neurons’ soma at various levels. Whether a particular motor neuron generates a recurrent discharge depends upon the level of depolarization of the soma and its dendrites. Let us assume that the resting membrane potential of the axon hillock favors depolarization of this region, thus facilitating action potential propagation into the motor neuron soma from an antidromically induced impulse. This action potential then propagates into not only the main portion of the soma but also into the various expanses of the alpha motor neuron’s dendrites (Fig. 6-11). By the time the depolarization has reached the distal portions of the dendrites, the axon hillock has undergone repolarization and is no longer in its refractory period (about 1 ms).\textsuperscript{46,47,48} The negative sinks of the dendrites are causing the ions surrounding the axon hillock to serve as a current source for the dendritic depolarization. This tends to alter the ionic distribution around the axon hillock by decreasing the positive charge on its surface. The transmembrane voltage alteration may induce an action potential to occur at this portion of the axon, thus generating the recurrent backfiring of the motor neuron begetting the subsequently observed F-wave (Fig. 6-11). The critical time period or “window of opportunity” between repolarization of the axon hillock coinciding with soma/dendritic local circuit currents is about 10–30 $\mu$s.\textsuperscript{195}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure6-10.png}
\caption{Renshaw cell activation. The alpha motor neurons possess a recurrent collateral portion of the axon just distal to the axon hillock, which extends to inhibitory interneurons known as Renshaw cells (R). Once the recurrent collaterals activate the Renshaw cell, it in turn synapses with alpha motor neurons to generate inhibitory postsynaptic potentials (–), which suppress firing of these neurons.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure6-11.png}
\caption{Motor neuron “backfiring.” Proposed mechanism of the so-called alpha motor neuron’s “backfiring” to generate an F-wave. \textbf{A}, Initially the action potential enters the axon hillock region and begins depolarization of the anterior horn cell’s soma. Solid arrows are the sodium ions carrying the inwardly directed current while dotted arrows are the internally directed current. \textbf{B}, This depolarization then extends into the dendritic extensions of the motor neuron while the axon hillock is refractory. Because the motor neuron’s dendrites are depolarizing similar to an unmyelinated nerve, i.e., continuous and not saltatory, the axon hillock exits its refractory period while depolarization is still occurring in the dendrites. The dendrites regions of depolarization act as a current sink while the sodium ions surrounding the axon hillock serve as a current source. \textbf{C}, A source current from the region of the axon hillock alters the transmembrane voltage (less positive extracellular) and this tends to depolarize the axon hillock generating an impulse propagating toward the periphery, i.e., an F-wave is then detected.}
\end{figure}
Should the soma’s membrane be depolarized to an extent exceeding that previously described, it and the dendrites will depolarize comparatively early and generate a local circuit current during the refractory period of the axon hillock. This situation results in the failure of F-wave production. On the other hand, the transition between the myelinated portion of the axon and the axon hillock does not favor conduction into the soma because the current distribution is diluted over the nonmyelinated portion of the axon hillock. In other words, the current distribution is not concentrated at a node of Ranvier but spread out over the surface of the axon hillock. If the resting membrane potential of the axon hillock is relatively hyperpolarized because of segmental and suprasegmental influences, action potentials will not cross this region to invade the soma and dendrites. In this case, an F-wave is not produced. The reason only a small number of F-waves are observed, therefore, is because of the required convergence of a number of excitatory and inhibitory influences favoring action potential conduction across the axon hillock with an appropriate temporal delay across the soma and dendrites favoring reactivation of the axon hillock. This situation changes from moment to moment, thereby resulting in a different subpopulation of motor neurons amenable to depolarization by an antidromic means with each ensuing stimulus.

The variable latency of sequentially elicited F-waves may be understood if one considers the motor neuron population producing the individual F-waves. Investigations in both humans and animals reveal that there is a greater chance of F-waves being generated by comparatively larger motor neurons. Larger motor neurons give rise to relatively large axons that have faster conduction velocities than smaller axons from the smaller motor neurons. Also, larger motor neurons innervate more muscle fibers, thus creating larger motor units with larger-magnitude F-waves. The resultant F-waves detected, therefore, preferentially arise from the faster-conducting axons that have a certain diameter range. This diameter distribution yields axons conveying F-waves with slightly different conduction velocities. Since there are very few F-waves that repeat with sequential stimulation (19.5%), a large number of different F-waves are observed from the available pool of motor neurons. The variable latency of F-waves represents the distribution of conduction velocities of axons mediating the recurrent responses.

Of the 1–2% of motor neurons capable of producing an F-wave secondary to segmental and suprasegmental inhibitory influences, one may attempt to understand the preferential bias toward larger motor neurons generating F-waves. First, there is a greater chance of larger motor neurons, through axon collaterals activating Renshaw cells, inhibiting smaller ones. This is because of the faster conduction velocity of antidromic impulses in larger axons (bigger motor neurons) reaching the Renshaw cells before the smaller axons (smaller motor neurons) and exerting a blocking influence on the smaller motor neurons. In a sense, the faster axons compete for optimal levels of resting membrane potentials for recurrent motor neuron excitation with each other, whereas the smaller motor neurons have a reduced chance of generating an F-wave. Also, it is easier for Renshaw cells to inhibit smaller motor neurons as there is less soma membrane to be affected. Secondly, the afferent fibers of smaller motor neurons conduct slightly faster than their corresponding motor fibers. These afferent impulses may reach the spinal cord prior to the antidromically excited motor fibers setting up a reflex response in which the small motor neuron is reflexively excited. The reflex-induced action potential from the small motor neuron would then collide and cancel the antidromic action potential in the proximal segment of the peripheral nervous system or depolarize the soma preventing repolarization. Finally, there is a greater chance of shortening the depolarization time of the soma in smaller motor neurons, possibly because of suprasegmental influences lowering the resting membrane threshold of the smaller motor neuron soma. It is known that smaller motor neurons fire at lower thresholds than larger motor neurons giving rise to the orderly recruitment of motor neurons, i.e., the Hennemann size principle. Smaller motor neurons, therefore, may have resting membrane levels closer to the depolarization threshold compared to larger ones. This may be an important mechanism of recurrent discharge inhibition in smaller motor neurons because recurrent collaterals are found in approximately 70–80% of them, leaving 20–30% without the possibility of recurrent inhibition. Should the threshold be lowered in smaller motor neurons, they will depolarize rather quickly, generating an action potential in the soma-dendrite region and creating a local circuit current incapable of exciting the axon hillock because it is still refractory. These three mechanisms or some combination may be the reason why there is preferential activation of relatively larger motor neurons generating the detectable F-waves.

**Diagnostic F-Wave Techniques**

A number of investigators have developed several interesting methodologies in which the F-wave can be used diagnostically. The basic parameter used by all investigators is the F-wave latency. Because sequential F-wave latencies are variable, innovative strategies have been developed to address this potential problem. Some of the techniques discussed include mean F-wave latencies over various body segments, latency ranges, F-wave conduction velocities, and F-wave latency ratios. Only a few investigations, however, have addressed amplitude for diagnostic purposes.

An F-wave may be obtained from essentially any muscle provided a supramaximal stimulation is used and the amplifier’s sensitivity is sufficient to detect the response. Because of the relative long duration of the CMAP the F-wave may be unrecordable in short nerve segments. The amplifier should be set at approximately 100–200 μV/div to ensure observation of the F-wave. Of course, a sensitivity of this magnitude does not permit one to simultaneously observe the entire CMAP. The rather delayed latency of the F-wave with respect to the CMAP requires a sweep speed of 5 ms/div and 10 ms/div in the upper and lower limbs, respectively. The easiest muscles to record F-waves from are the small intrinsic hand and foot muscles. Because of this, the majority of reference data available pertain to the following muscles: abductor pollicis brevis, abductor digiti minimi, extensor digitorum brevis, and abductor hallucis. Routine recording techniques previously described are used. It is not necessary to relocate the anode distal to the cathode when exciting the nerve as anodal block most likely does not occur. When stimulating the nerve, an optimal stimulus rate is 1 Hz or less.

Slight contraction of the muscle under investigation can facilitate the observation of F-waves should one note a decreased ability to record them. This is most likely mediated through increased motor neuron pool excitability. Caution is required when attempting to facilitate the F-wave because amplitudes may be increased and an H-reflex (see below) may contaminate the desired responses. The effect of facilitation, however, is not consistent. Although difficult to quantify, a reduction in the numbers of F-waves following supramaximal stimulation may indicate pathology.
The rationale for attempting to record the F-wave is multifactorial. Recall that the F-wave is a potential that represents conduction from the site of stimulation to the motor neuron and back to the recording electrode, i.e., both the proximal and distal regions of the peripheral nervous system. As demonstrated above, it is relatively easy to examine conduction in the distal portions of the peripheral nerves. Assessment of proximal conduction becomes technically more demanding and subject to volume conduction effects. The F-wave impulse, however, originates distally where there is less ambiguity of the nerve excited; it also is recorded distally, with little interference from neighboring muscles. Theoretically, the F-wave appears to be the ideal parameter to use to assess proximal conduction. Although there are a number of limitations regarding the F-wave (see below), this concept is generally correct.

F-Wave Latency

As previously stated, the F-wave latency varies from one stimulus to the next (Fig. 6-9). It is important to record a sufficient number of F-waves to ensure analysis of a representative sample of the total available pool of motor neurons producing F-waves. The exact number of F-waves necessary to produce a representative number is unknown. The practicality of available time for F-wave collection during an electrodiagnostic medicine examination also must be considered. A number of investigators recommended obtaining between 10 and 20 F-waves per stimulus site. Although gathering still larger numbers of F-waves may yield a few with shorter latencies, the diagnostic utility versus time consumed becomes prohibitive. F-wave reference data are somewhat variable from one investigator to the next not only because of the inherent variability of the response itself, but also because the latency depends on the stimulus site. As there are no universally accepted standards with respect to distance between the cathode and recording electrodes, the F-wave demonstrates slightly different mean values from one laboratory to the next. For the F-wave latencies reported, the median and ulnar nerves are excited just proximal to the distal wrist crease, whereas the tibial nerve is activated posterior to the superior margin of the medial malleolus and the peroneal nerve just above the ankle region (Table 6-6). The previous locations of an 8 cm standard distance should result in similar mean F-wave latencies. Recommended side-to-side differences for both shortest latency and mean latency are 2.0 ms in the upper limb and 4.0 ms for lower limb intrinsic muscle studies. In general, F-wave latencies are directly related to height and limb length as anticipated given the length of the neural pathway, but there is minimal correlation to age and gender, especially if the median and ulnar nerves are excited just proximal to the distal wrist crease, whereas the tibial nerve is activated posterior to the superior margin of the medial malleolus and the peroneal nerve just above the ankle region.

The F-wave latency is the interval from the onset to the peak of the F-wave. Once the shortest F-wave of a series is obtained, it is possible to determine pathology if a given nerve is injured. The difficulty in using the shortest F-wave is that the study is biased toward one nerve fiber. If there is significant damage to the peripheral nervous system but one or a few of the fastest-conducting fibers survive, then a normal study is declared. A more rational approach is to consider the mean value of a group of F-waves recorded. The mean onset latency of 10 or more F-waves is believed to be more sensitive than only considering the fastest F-wave.

One also may attempt to measure the latencies of a large number of F-waves, 100 or more, and calculate the time difference between the shortest and longest F-waves. This technique has been referred to as F chronodispersion. F chronodispersion reference values for a number of muscles are known: APB: 3.6 ± 1.2 ms; ADM: 3.3 ± 1.1 ms; EDB: 6.4 ± 0.8 ms; and soleus: 2.8 ± 1.1 ms. The major limiting factor in performing the F chronodispersion technique is that 100 F-waves must be acquired in order to obtain a large distribution of latency differences. Patient tolerance and the time required to calculate these data are major drawbacks to routinely using this technique despite its reported sensitivity to pathology.

Occasionally, one may wish to calculate the F-wave latency over a localized proximal segment such as the brachial plexus. Obviously, stimulating the median or ulnar nerve at the wrist includes the entire nerve segment from wrist to spinal cord and back to the muscle. By subtracting the CMAP distal motor latency to wrist stimulation from the shortest F-wave latency and then subtracting an additional 1 ms, a conduction time for the fastest conducting F-wave from wrist to spinal cord and back to the wrist is obtained. It is necessary to reduce the conduction time by 1 ms because this is believed to represent the turnaround time for motor neuron reactivation in the spinal cord. It is important to note that this presumed 1-ms turnaround time has never been documented and obviously presents itself as a potential complicating factor in various techniques using this time frame. Further, dividing this latency by 2 allows one to determine the conduction time from wrist to spinal cord, the central conduction time. In other words, the equation representing this latency is: central conduction time = (F-wave latency − DML − 1 ms)/2. The problem with this method is that a small lesion in a proximal portion of the peripheral nervous system could be diluted out over the spinal cord to wrist distance, thereby reducing the sensitivity of this technique. An alternative method is to stimulate the median or ulnar nerve in the axilla and measure the F-wave over this comparatively shorter segment. Unfortunately, the CMAP and F-wave occur at about the same time, thus obliterating the F-wave. A second stimulation applied at the wrist simultaneously with axillary excitation collides with the orthodromic axillary impulses permitting detection of the axillary F-wave through a collision technique. A simpler method to examine the proximal F-wave latency is to stimulate the desired nerve in the axilla 25 cm from the sternal notch with the arm abducted 90° and the forearm supinated. The shortest F-wave latency from the wrist is then added to the previously obtained CMAP DML from which is subtracted the axillary CMAP latency multiplied by 2 and is called the axillary F-loop latency (AFLL): AFLL = (F-wave + DML) − 2 axillary latency. An axillary F-loop latency in excess of 11.0 ms is considered abnormal. Because this technique involves the fastest F-waves, an attempt was made to increase the sensitivity by averaging 32 F-waves and measuring the averaged F-wave peak latency and inserting this value into the previously defined AFLL equation. Normal values for the median and ulnar nerves were reported as 14.12 ± 0.88 ms and 13.79 ± 0.9 ms, respectively.

F-Wave Conduction Velocity

Once the shortest F-wave of a series is obtained, it is possible to convert this latency into a conduction velocity. There are two major assumptions involved in using F-wave conduction velocities. The first assumption is that the shortest F-wave is detected within the limited number of responses obtained, less than 20, and these correspond to the motor fibers producing the onset latency for the CMAP detected with distal stimulation. It has been clearly demonstrated that the shortest F-wave does not always occur within the first 20 potentials, but may require up to 100 or more responses. The second assumption requires an accurate measurement of the conducting pathway traversed by the impulses generating the F-wave.
is rather easy for the limb, but the difficulty arises when proximal segments across the brachial or lumbosacral plexi are involved. It has been determined in a very limited number of anatomic specimens that measuring from the stimulus site, ankle or popliteal fossa, to the T12 spinous process by way of the greater trochanter approximates rather well the true anatomic length of the tibial nerve.130 The same anatomic verification, however, has not been determined for the upper limb. Although F-wave conduction velocities have been criticized because of the unnecessary addition of a potentially large error due to less than accurate distance measurements,130,243 conduction velocities nevertheless continue to be used. The use of F-wave conduction velocities has been justified on the basis of noting that the difference in latencies between stimulating the peroneal nerve at the ankle and knee while recording from the EDB correspond to the differences in F-wave latencies from these two sites, i.e., 6.5 ms and 6.4 ms, respectively. The implication of this finding is that the shortest-latency motor fibers determining CMAP onset latency correspond to similar fast fibers mediating the shortest F-wave,121,122. In calculating F-wave conduction velocities for upper limb examinations, the distance from the point of stimulation is measured to the C7 spinous process with the arm abducted 90°. The equation used to calculate F-wave velocities for both intrinsic hand and foot muscles is:

\[ \text{F-wave CV (m/s)} = \frac{(\text{distance to T12 or C7 in mm}) \times 2}{(\text{F-wave latency} - \text{CMAP latency} - 1 \text{ ms})} \]

Normal values for both upper and lower limb nerves at multiple stimulation sites are provided (Table 6-6). The F-wave conduction velocity has been reported to be of value in detecting proximal slowing in various disease states affecting the peripheral nervous system.58,114,115 There is some suggestion that using an averaged F-wave latency to calculate F-wave conduction velocities may be of greater sensitivity in detecting abnormality compared to the shortest F-wave latency.59,98 A modification of the F-wave chronodispersion using the distribution of F-wave conduction velocities (F-tacheodispersion) is believed to be a sensitive method of defining peripheral nerve conduction abnormalities but more studies are required to fully evaluate this technique.59

**F-Wave Ratio**

Because of the potential for distance measurement errors in calculating F-wave conduction velocities, an alternative F-wave technique was developed that does not involve distance.49,50 It was determined that if the median or ulnar nerve was stimulated at the elbow region, the time of conduction for the F-wave to the spinal cord was very similar to the latency for direct motor nerve activation from the same site to the muscle, i.e., CMAP onset latency. In other words, the F ratio is close to unity. Similar findings were noted for tibial and peroneal nerve stimulation while recording from the intrinsic foot muscles (Table 6-6).140,120,121 The equation used to determine F ratios is:

\[ \text{F ratio} = \frac{(\text{F-wave latency} - \text{CMAP latency} - 1 \text{ ms})}{2} \]

Although it is possible to calculate F ratios with either more proximal or distal stimulation sites, the variability of data is minimal with elbow and popliteal fossa excitation. Motor nerve and F-wave conduction velocities may both be abnormal yet the F ratio can be within normal limits. This suggests that not only are the peripheral nerves conducting slowly over both the distal and proximal segments, but they are slowed to a similar degree.

**F-Wave Amplitudes and Persistence**

In disorders in which the central excitability of the motor neuron pool is decreased, one could anticipate both a reduced number and smaller amplitude of F-waves. This has been found to be the case in patients examined immediately following a unilateral stroke.58 Excitation of the cerebellum also can decrease F-wave amplitude and persistence.58,66 On the other hand, in patients with chronic myelopathies and spasticity, F-wave persistence and magnitude are increased commensurate with the elevated excitability of the motor neuron pool.51 The latencies of F-waves in patients with upper motor neuron lesions, however, may be prolonged secondary to the unmasking of smaller motor neurons (slower peripheral conduction) while the larger ones are blocked secondary to rapid depolarization.56,61 It is possible

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**Table 6-6. F-Wave Reference Values**

<table>
<thead>
<tr>
<th>Number of Nerves Tested</th>
<th>Site of Stimulation</th>
<th>M latency (msec)</th>
<th>F latency (msec)</th>
<th>F ratio (F – M – 1)/2M</th>
<th>F ratio (R)</th>
<th>MNCV between Two Stimulus Sites (m/sec)</th>
<th>FWCV from Cord to Site (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>66 Median nervesa</td>
<td>Wrist</td>
<td>3.5 ± 0.5</td>
<td>29.1 ± 2.3</td>
<td>1.04 ± 0.08</td>
<td>1.01 ± 0.07</td>
<td>56.0 ± 5.0</td>
<td>62.2 ± 5.2</td>
</tr>
<tr>
<td></td>
<td>Elbow</td>
<td>7.8 ± 0.8</td>
<td>24.8 ± 2.0</td>
<td>1.04 ± 0.08</td>
<td>1.01 ± 0.07</td>
<td>56.0 ± 5.0</td>
<td>62.2 ± 5.2</td>
</tr>
<tr>
<td></td>
<td>Axilla</td>
<td>11.3 ± 1.0</td>
<td>21.7 ± 2.8</td>
<td>1.04 ± 0.08</td>
<td>1.01 ± 0.07</td>
<td>56.0 ± 5.0</td>
<td>62.2 ± 5.2</td>
</tr>
<tr>
<td>66 Ulnar nervesb</td>
<td>Wrist</td>
<td>2.9 ± 0.5</td>
<td>30.5 ± 3.0</td>
<td>1.40 ± 0.11</td>
<td>0.99 ± 0.09</td>
<td>55.9 ± 5.1</td>
<td>58.2 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>Below elbow</td>
<td>6.7 ± 0.7</td>
<td>26.0 ± 2.0</td>
<td>1.40 ± 0.11</td>
<td>0.99 ± 0.09</td>
<td>55.9 ± 5.1</td>
<td>58.2 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>Above elbow</td>
<td>9.2 ± 0.9</td>
<td>23.5 ± 2.0</td>
<td>1.40 ± 0.11</td>
<td>0.99 ± 0.09</td>
<td>55.9 ± 5.1</td>
<td>58.2 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>Axilla</td>
<td>11.2 ± 1.0</td>
<td>21.9 ± 1.9</td>
<td>1.40 ± 0.11</td>
<td>0.99 ± 0.09</td>
<td>55.9 ± 5.1</td>
<td>58.2 ± 2.9</td>
</tr>
<tr>
<td>66 Peroneal nerves</td>
<td>Ankle</td>
<td>4.5 ± 0.9</td>
<td>51.3 ± 4.7</td>
<td>1.11 ± 0.09</td>
<td>1.02 ± 0.09</td>
<td>49.4 ± 3.8</td>
<td>51.3 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>12.9 ± 1.4</td>
<td>42.7 ± 4.0</td>
<td>1.11 ± 0.09</td>
<td>1.02 ± 0.09</td>
<td>49.4 ± 3.8</td>
<td>51.3 ± 2.9</td>
</tr>
<tr>
<td>66 Tibial nerves</td>
<td>Ankle</td>
<td>4.1 ± 0.6</td>
<td>52.3 ± 4.3</td>
<td>1.17 ± 0.10</td>
<td>1.00 ± 0.10</td>
<td>46.8 ± 3.4</td>
<td>51.3 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>12.8 ± 1.3</td>
<td>12.8 ± 1.3</td>
<td>1.17 ± 0.10</td>
<td>1.00 ± 0.10</td>
<td>46.8 ± 3.4</td>
<td>51.3 ± 2.9</td>
</tr>
</tbody>
</table>

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*a F wave was elicited by axillary stimulation in 42 of 66 nerves.
*b Middle segment across elbow was tested in 34 of 66 nerves.
to calculate the ratio of the F-wave to that of the corresponding CMAP (F/M ratio) in an attempt to measure the amount of the motor neuron pool activated. Because of the variability of the F-wave amplitude, mean amplitudes calculated from a series of F-waves appears to be the most reasonable method. The clinical utility of F/M measurements in routine electrodiagnostic medicine examinations remains to be demonstrated.

**F-Wave Clinical Utility**

The clinical utility of various F-wave techniques is by no means universally agreed upon by even a minority of practitioners. As a result, the authors will exercise their prerogative based on clinical experience and a review of the literature that some readers may disagree with.

As noted above, the F-wave is a very long conduction pathway with a variable response latency from one stimulus to the next. This is clearly a physiologic disadvantage in attempting to localize a lesion to a focal region of the nervous system, particularly in mild disease. This so-called disadvantage, however, can be used to an advantage in some disorders. In our opinion, the very fact that the F-wave is traversing the peripheral nerve twice can be used to a diagnostic advantage in detecting an early disease process that is diffusely distributed along the nerve and may not be detected by assessing a focal neural segment. One such disease entity is diabetic neuropathy. Although appropriate reservations have been raised regarding the true sensitivity of F-waves in diagnosing early peripheral nerve disease, there is a sound physiologic basis for considering the use of F-waves in attempting to define if there is a generalized mild process affecting at least the motor aspects of the peripheral nervous system.

In addition to a mild diffuse peripheral nerve process, a proximal lesion in the neighborhood of the brachial plexus or more rostral (root level) may be amenable to diagnosis by the use of F-waves. These “proximal” lesions are limited to two disorders, and under specific conditions. The first is Guillain-Barré syndrome, in which a reduced number or absence of F-waves may be observed secondary to an early and significant blockade of action potential propagation across the root region. This may be the only abnormality noted early on in some but not all patients with this disorder. We do not mean to imply that this is the most sensitive technique for diagnosing this entity, but rather that the practitioner should not forget to address these regions of the nervous system in a patient that may be presenting in an atypical manner. A second possible disorder in which F-waves may be of assistance in alerting the clinician to a particular disease is multifocal motor neuropathy with conduction block. Again, it should not be concluded that an absence or reduced number of F-waves is diagnostic of this disease, but rather that many practitioners do not routinely study the peripheral nervous system from the root to the axilla in all patients presenting with limb weakness. Conduction studies of the arm and forearm may be normal, whereas the F-waves may be reduced in number or absent. This finding should suggest that the region between the root and axilla should be studied. No doubt fibrillation potentials may be detected in distal muscles, but the F-wave can help direct a more “focal” exam of the relatively proximal neural segments. Having said the above, not all proximal lesions are particularly amenable to F-wave studies. For example, cervical and lumbosacral radiculopathies certainly can result in abnormal F-wave studies, however, most of these patients have more localizing findings on needle electromyography. This is understandable since the majority of muscles are innervated by more than one root and most radiculopathies do not produce complete obliteration of all the nerve fibers in a nerve root. It is not surprising, therefore, that F-waves are usually abnormal in radiculopathies only when significant unilevel or multilevel root disease is present. Although F-waves can be used to study focal demyelinating lesions, it is not our contention that focal entrapment neuropathies such as carpal tunnel syndrome should be routinely assessed by F-wave studies. As always, it is incumbent upon the practitioners to become familiar with a particular technique and its available literature, and then assess whether the technique is of value in their patient population.

**H-REFLEX**

A stimulus applied to the tibial nerve with a magnitude that is subthreshold for a direct motor response usually produces a late response when recording from the gastrocnemius-soleus muscles in the neighborhood of 30 ms (Fig. 6-12). This potential was first described by Hoffmann in 1918. Magledary and McDougal performed in-depth electrophysiologic investigations of this late response by the 1950s and they designated this potential the H-reflex in honor of Hoffmann. In studying both the H-reflex and the F-wave, Magledary and McDougal defined a number of ways to distinguish between these two responses with similar latencies (Table 6-7). A number of clinical applications have been developed using the H-reflex. Prior to discussing how the H-reflex may be employed in the diagnosis of potential pathology affecting the peripheral and central nervous systems, it is necessary to discuss the physiology of the H-reflex.

![Figure 6-12. H-reflex](image-url)
Physiology of the H-Reflex

The H-reflex is believed to be a CMAP arising from an electrical afferent activation of a monosynaptic reflex arc. The afferent pathway of the H-reflex involves electrical activation of the large Ia afferent nerve fibers originating from muscle. After entering the dorsal horn of the spinal cord, the Ia afferents synapse with the alpha motor neurons innervating that muscle. This afferent motor impulse traverses the motor nerves to result in a CMAP. The complete reflex arc, therefore, is mediated by orthodromic sensory and orthodromic motor neural conduction.

The H-reflex is most easily elicited by stimulating the tibial nerve at the popliteal fossa with a relatively long-duration stimulus and an intensity that is subthreshold for motor nerve stimulation. Recordings are typically performed from the gastrocnemius-soleus muscles. The intensity of the current is initially set at zero. As the stimulus intensity is slowly increased, the H-reflex is first noted to appear with a small amplitude and duration approximating 30 ms (Fig. 6-12). Continued elevation of the stimulus results in a progressively larger-amplitude H-reflex. The magnitude of the H-reflex usually peaks at or just prior to the observation of a direct CMAP or M-response from the gastrocnemius-soleus muscles. Further increases in the current intensity results in a continually increasing M-response but a steadily declining H-reflex amplitude. When the M-response approaches a maximum and its amplitude no longer increases, the H-reflex is usually replaced by an F-wave.

The recommendation of a stimulus duration between 0.5 ms and 1.0 ms is made because the relatively longer current durations are believed to preferentially activate the large sensory compared to smaller motor fibers. Excitation of the large Ia afferent fibers is desired in order to initiate the reflex arc. An alternative explanation uses an anatomic location of sensory compared to motor fiber within the nerve. Selective activation of the anterior as opposed to posterior aspects of the tibial nerve in both the human and cat reveal that the motor fibers are primarily located anteriorly. Additionally, the motor and sensory fibers are found to be approximately of the same size and should have the same threshold of activation. Sensory fibers are activated first because they are more superficial and located closer to the cathode. Although this may be partially responsible for activation of the sensory fibers prior to motor fibers, it does not address the capability of comparatively longer-duration impulses at the same location to selectively activate sensory before motor fibers. The most likely explanation is probably a combination of lower threshold levels in sensory compared to motor fiber within the nerve. Selective activation of the anterior as opposed to posterior aspects of the tibial nerve in both the human and cat reveal that the motor fibers are primarily located anteriorly. Additionally, the motor and sensory fibers are found to be approximately of the same size and should have the same threshold of activation. Sensory fibers are activated first because they are more superficial and located closer to the cathode. Although this may be partially responsible for activation of the sensory fibers prior to motor fibers, it does not address the capability of comparatively longer-duration impulses at the same location to selectively activate sensory before motor fibers. The most likely explanation is probably a combination of lower threshold levels in sensory fibers to longer current durations and the more posterior or superficial position of sensory fibers in the tibial nerve at the popliteal fossa. Following activation of the large Ia afferents at the knee, the impulse propagates superiorly to enter the dorsal horn of the spinal cord, where it synapses with the alpha motor neurons innervating that muscle.

As previously stated, the H-reflex is believed to be a monosynaptic reflex arc. This belief is founded upon several experimental observations. Sectioning of the dorsal root permanently obliterates any trace of the H-reflex. Investigations regarding the time necessary to conduct through the possible central connections between the dorsal and ventral root revealed only enough time for one synapse, i.e., between 0.5 and 1.0 ms. The Ia afferents that synapse with a homonymous alpha motor neuron tend to depolarize the soma’s resting membrane potential through a transmitter substance by increasing the permeability of small cations such as Na⁺ and K⁺. The release of a transmitter with the capability of depolarizing the soma’s membrane is said to generate an excitatory postsynaptic potentials (EPSPs). The rise time of the EPSP approximates 3.6 ms. It is necessary for multiple Ia afferents to each contribute an EPSP in a rather synchronous volley given the above-noted rise time to sufficiently depolarize the motor neuron to threshold to generate an efferent motor impulse. Single-fiber electromyography studies reveal that the amount of variability between successive H-reflex responses is rather large and requires an interposed synapse in addition to the neuromuscular junction. The amount of afferent stimulus required to generate the reflex contraction of the homonymous muscle (H-reflex) is dependent upon several factors.

Slowly increasing the magnitude of the electrical stimulus results in a progressively larger H-reflex (Fig. 6-12). This is the result of activating more Ia afferents with each impulse, thereby recruiting more alpha motor neurons. This process continues until the H-reflex begins to decline. The maximum H-reflex amplitude may be compared to the maximum CMAP response obtained by direct excitation of the peripheral nerve to estimate the percentage of the motor neuron pool activated through the reflex response. It has been determined that 24–100% of the motor neuron pool may participate in the H-reflex.

Table 6-7. Distinguishing Characteristics of F-Wave vs. H-Reflex

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>F-Wave</th>
<th>H-Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presumed response</td>
<td>Motor neuron</td>
<td>Monosynaptic reflex arc</td>
</tr>
<tr>
<td><strong>Afferent path</strong></td>
<td>α motor fibers</td>
<td>Ia afferents</td>
</tr>
<tr>
<td><strong>Efferent path</strong></td>
<td>α motor fibers</td>
<td>α motor fibers</td>
</tr>
<tr>
<td><strong>Muscle</strong></td>
<td>All skeletal muscles</td>
<td>Gastrocnemius-soleus Flexor carpi radialis</td>
</tr>
<tr>
<td><strong>Stimulus threshold</strong></td>
<td>Supramaximal</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Morphology</strong></td>
<td>Variable</td>
<td>Constant at low stimulus rates</td>
</tr>
<tr>
<td><strong>Magnitude (compared to maximal CMAP)</strong></td>
<td>&lt; 5%</td>
<td>Same or smaller</td>
</tr>
<tr>
<td><strong>Jitter</strong></td>
<td>&gt; CMAP; &lt; H-reflex</td>
<td>&gt;&gt; F-wave</td>
</tr>
<tr>
<td><strong>Response to increasing stimulus intensity</strong></td>
<td>More persistent</td>
<td>Disappears</td>
</tr>
<tr>
<td><strong>Response to agonist</strong></td>
<td>Slight increase</td>
<td>Appears in muscles not displaying H-reflex at rest</td>
</tr>
</tbody>
</table>

* Refers to muscle at rest where response can usually be elicited. Modified from Lachman et al and Magledary and McDougal.
the threshold level. Additionally, there may be motor neurons that were not previously depolarized that can now fire because less EPSP summation is required. The suprasegmental influence of central facilitation introduces a potentially significant variable when attempting to quantitate the H-reflex amplitude for diagnostic purposes. Each individual possesses his or her own level of central nervous system activity with respect to the necessary amount of transmitter directed EPSPs required to potentiate alpha motor neuron discharge.

The progressive reduction and eventual disappearance of the H-reflex with continued elevation in stimulus strength is poorly understood, but may be a result of several factors. A mechanism initially proposed relies upon the H-reflex impulses colliding with the antidromic action potentials propagating in the motor nerves just distal to the alpha motor neurons.153,239 Remember that action potentials are generated at the same location in both sensory and motor fibers, e.g., the popliteal fossa for an H-reflex recorded from the gastrocnemius-soleus muscle. If the H-reflex impulses are to collide with the antidromic motor action potentials somewhere at the level of the ventral roots or distally, sensory action potentials obviously have to conduct significantly faster than the motor impulses in order to traverse and exit the central nervous system. Recall that about 4 ms is required for the EPSP to reach its peak plus an additional 1 ms for synaptic transmission. A difference of about 5 ms is the required separation between the sensory and motor conduction times. Histologic examination of the peripheral nerves concerned has revealed similar diameters for both motor and sensory nerves suggesting that they have similar conduction velocities.153 Despite the well-accepted opinion that sensory fibers generally conduct faster than motor fibers, this has not been found in careful investigation of afferent and efferent conduction velocities.40,41,52,153,217 Although it may be possible for fast-conducting Ia afferents that have activated the lower threshold and relatively smaller slower-conducting alpha motor neurons to collide at the ventral root region, this may not be the complete explanation for all motor neurons. The fast-conducting antidromic motor potentials reach the ventral root at essentially the same time the afferent impulses arrive at the dorsal root. Additionally, by strongly contracting the muscle, an H-reflex reappears despite a strong stimulus, thereby proving collision is not a major component of H-reflex suppression. Therefore another explanation of this phenomenon is necessary.

Let us assume that a suprathreshold stimulus is delivered to the tibial nerve in the popliteal fossa, thus activating the large Ia afferents as well as the large motor fibers conducting at similar velocities. In this case, the alpha motor neurons are activated by the antidromic impulse as discussed previously for the F-wave.112 Additionally, the recurrent collaterals mediate Renshaw cell inhibition of the alpha motor neuron pool. By the time the EPSP and synaptic transmission of H-reflex impulses have converged on the alpha motor neuron, it is most likely no longer capable of depolarizing the previously activated anterior horn cells at lower stimulus levels because they are either in a refractory state from F-wave generation or under the influence of Renshaw inhibition. It is possible to detect an H-reflex following inhibition through a supramaximal stimulus if the patient contracts the agonist muscles. This finding strongly argues against collision as a reason for obliteration of the H-reflex. Muscle contraction should not affect whether antidromic impulses collide with the orthodromic H-reflex. It appears that the major component of H-reflex suppression with supramaximal stimulus delivery is based on the balance between central facilitatory and inhibitory influences. The progressive decline of the H-reflex and replacement by an F-wave at sequentially greater current intensities is likely a combination of collision, refractory alpha motor neurons, and Renshaw inhibition of the motor neuron pool (6-12).234,235

Factors Affecting the H-reflex

As previously stated, the magnitude of the H-reflex is subject to the amount of current delivered to the nerve, ratio of Ia afferents to motor fibers activated, interpersonal differences in suprasegmental facilitatory influences, and level of muscle contraction.85 The H-reflex is commonly observed only in the gastrocnemius-soleus and flexor carpi radialis muscles. The detection of H-reflexes in these two muscles may not be observed in all normal individuals, particularly in the elderly. Contraction of the muscle from which the H-reflex is recorded and its agonists may allow an H-reflex to be recorded from where it was previously absent. Again, this is because of facilitatory influences decreasing the difference between the alpha motor neuron’s resting membrane and threshold levels. Muscle contraction is found to not alter the latency of the response significantly.25 Contracting the antagonist muscles tends to suppress the appearance of the H-reflex.77,78,95,153 It is also possible through contraction of a muscle to observe H-reflexes where they are not routinely detected.172 For example, through muscular contraction, H-reflexes have been detected in the tibialis anterior, abductor pollicis brevis, extensor digitorum communis, foot intrinsic muscles, and flexor digitorum profundus.25,54,70,174 Patients with spasticity resulting from various upper motor neuron lesions may produce H-reflexes in muscles other than the soleus muscles.131,146,215 Also, in newborns, an H-reflex may often be seen in the small muscles of the hands and feet, and usually disappears by 1 year of age.13,94,216

In addition to contraction of the agonist muscles, it is also possible to facilitate the H-reflex with a Jendrassik maneuver by asking the patient to forcefully make a fist. This is significantly less effective in facilitating an H-reflex than a contraction of the agonist muscles.153 Significant potentiation of the H-reflex can occur with post-tetanic stimulation.135 It is possible to generate an H-reflex with a subthreshold response after a tetanus of 100–500 Hz for 20 seconds lasting about 10–40 seconds. The mechanism is unclear but is believed to involve presynaptic facilitation.85

H-reflex amplitude may be affected by factors other than antagonist muscle contraction.172 Forceful active or passive ankle flexion and contraction also can markedly reduce the tibial nerve’s H-reflex magnitude.53,153 Mild passive stretching of the gastrocnemius-soleus muscle can either facilitate or inhibit the H-reflex.214,236 Vibration is an effective way to suppress the H-reflex.9,20,35,231 Applying a vibrating stimulus to the Achilles tendon in the limb under investigation results in depression of the H-reflex that may outlast the duration of the vibration by several hundred milliseconds.205 The mechanism of H-reflex suppression is unknown but may involve presynaptic inhibition through primary spindle afferent firing or neurotransmitter depletion.30,214 It is also possible to suppress the H-reflex with stimuli delivered at several Hz.25

Diagnostic H-Reflex Techniques

There are two major clinical applications of the H-reflex. First, the H-reflex may be used to evaluate the status of the peripheral nervous system with respect to proximal peripheral nerve conduction and potential entrapment of nerve roots, e.g.,
The H-reflex is a triphasic initially positive potential (Fig. 6-12). It is preferentially detecting the response from the soleus muscle and not over its motor point. A characteristic appearance of the H-reflex is a variable latency and morphology. Should not be recorded if it is smaller than the M-response or the appearance of an F-wave (Fig. 6-12). The H-reflex demonstrates a very stable onset latency from one response to the next. This is quite different than the rather variable F-wave latency with each consecutive stimulus. Continued elevation of the current strength characteristically results in the disappearance of the H-reflex and the appearance of an F-wave (Fig. 6-12). The H-reflex should not be recorded if it is smaller than the M-response or demonstrates a variable latency and morphology.

H-Reflex: Gastrocnemius-Soleus Muscle

Recording Electrodes. Surface recording electrodes are preferred for this technique as they were used for the development of the accompanying reference data. Because amplitudes are not used, needle recordings to document H-reflex latencies are acceptable provided the same technique is used for both left and right measurements.

The patient is positioned comfortably in the prone position with the feet off the edge of the plinth. It is often helpful to place a pillow beneath the legs to cause slight knee flexion. Both left and right H-reflexes can be obtained in this position with little difficulty. It is necessary to record the H-reflexes from both lower limbs as left/right latency comparisons often are quite helpful.

E-1. The E-1 recording electrode is located by first flexing the leg and drawing a line across the popliteal fossa. A line connecting the mid-popliteal fossa with the proximal flare of the medial malleolus is bisected for the E-1 electrode location. This site typically approximates the musculotendinous junction of the gastrocnemius muscle.

E-2. An E-2 electrode is secured to the distal portion of the Achilles tendon just proximal to its insertion on the calcaneus.

Ground. A ground electrode is positioned just proximal to E-1.

Stimulation. The cathode is placed in the mid-popliteal fossa with the anode distal. A pulse duration of between 0.5 ms and 1.0 ms is recommended. The current intensity is slowly increased until the stimulus just activates the large Ia afferent fibers without concomitant activation of the motor fibers or is just threshold for only a few motor fibers. The stimuli should be delivered at a rate of 1 stimulus every 2–3 seconds to avoid suppressing the H-reflex through central mechanisms. It is often necessary to move the stimulating electrodes either slightly medially or laterally to optimize the cathode directly over the tibial nerve. Care should be taken to avoid proceeding too far laterally as the peroneal nerve may be excited. This is relatively easy to define as the foot no longer plantarflexes but dorsiflexes with each stimulus.

Some investigators prefer to use a needle cathode, requiring less stimulus. In this instance, a large electrode is secured to the patella to serve as the anode. Using a similar pulse width to that noted above should not damage the nerve as a subthreshold stimulus is used. It is our experience that should surface stimulation fail to elicit an H-reflex, needle excitation often results in a demonstrable response. Further study, however, is required comparing the optimal stimulus parameters for needle and surface stimulation.

As stated above, the stimulus is slowly increased until a response with a latency approximating 30 ms is detected. The current level is increased in small increments until a motor response is just noted. One should then optimize the H-reflex by decreasing or minimally increasing the current intensity until the H-reflex magnitude is maximized. Several responses are observed at this stimulus level to ensure a reproducible and stable response. The latency is then recorded to the initial departure of the H-reflex from the baseline. This is typically although not always a positive deflection, most likely because the electrode is preferentially detecting the response from the soleus muscle and not over its motor point. A characteristic appearance of the H-reflex is a triphasic initially positive potential (Fig. 6-12). It is not unusual, however, to observe an initially negative H-reflex. In either case, the initial baseline departure denotes the correct latency measurement. An H-reflex demonstrates a very stable onset latency from one response to the next. This is quite different than the rather variable F-wave latency with each consecutive stimulus. Continued elevation of the current strength characteristically results in the disappearance of the H-reflex and the appearance of an F-wave (Fig. 6-12). The H-reflex should not be recorded if it is smaller than the M-response or demonstrates a variable latency and morphology.

Figure 6-13. H-reflex nomogram. A nomogram for predicting the H-reflex provided the patient’s age and leg length (see text for proper measurement) are known. (From Braddom RL, Johnson EW: Standardization of H reflex and diagnostic use in S1 radiculopathy. Arch Phys Med Rehabil 1974;55:161–166, with permission.)
Instrumentation Parameters. A sweep speed of 10 ms/div is optimal for lower limb H-reflex examinations. Although a sweep speed of 5 ms/div can be used, it is possible for this response to be greater than 50 ms in a particularly tall individual or in a person with a peripheral neuropathy. An amplifier sensitivity of 200 µV/div to 500 µV/div usually suffices for most responses. Filter settings commensurate with routine motor studies are recommended.

Reference Values. The technique of Braddom and Johnson is suggested as it represents a standardized method for obtaining H-reflex latencies. H-reflexes are found to correlate highly with both age and leg length. A regression equation may be used to predict the optimal H-reflex latency: H-reflex (ms) = 9.14 + 0.46(leg length–cm) + 0.1(age–yrs). A nomogram based upon this equation also may be used in the clinical setting as a quick reference (Fig. 6-13). When using the nomogram, a line connecting the patient’s age and leg length is constructed and where it crosses the middle set of values predicts the H-reflex latency. A mean H-reflex latency of 29.8 ± 2.74 ms is found for a normal population. In this study a side-to-side difference of 1.5 ms is predictive of an S1 radiculopathy. Some investigators use as little as a 1.0 ms difference. In persons aged 60–88 years, an H-reflex can be obtained in up to 92% of persons with a side-to-side latency difference of 1.8 ms. The normal side-to-side amplitude differences in persons between 21 and 67 years can reach 60% (see Additional Comments for further discussion).

Additional Comments. Should one have difficulty eliciting an H-reflex, slight voluntary contraction of the muscle examined may facilitate its detection. Although this should not significantly affect the latency, it may be advisable to also use slight voluntary contraction on the contralateral side, particularly if the left/right differences approach significance.

Caution should be exercised in attempting to use the H-reflex amplitude for diagnostic purposes as it is highly variable and subject to central nervous system influences. The amplitude is also quite sensitive to electrode placement and may noticeably change within just a few centimeters. If side-to-side amplitudes are to be compared, it is very important to exactly reproduce the electrodes’ locations since different electrode positions can result in considerable amplitude variations. As noted above, several investigations have attempted to define the degree of side-to-side amplitude variation in reference populations. When comparing the smaller of the two responses to the larger, an amplitude ratio (smallest/largest between left and right responses) smaller than 0.4 or 0.5 is considered an abnormal finding and suggestive of possible pathology affecting the fibers conveying the response. Another study found an amplitude ratio (right side/left side) of greater than 1.8 (unaffected side/affected side) to indicate an abnormality. In our opinion, a shortcoming of these studies is a failure to fully explore the effect of central facilitation on the “normal” side-to-side amplitude range. Specifically, in persons with a considerable side-to-side difference no apparent attempt was made to facilitate the response and see if the side-to-side difference would diminish. This is critical since patients with back pain and no radicular axonal/ demyelinating lesion may split the painful side and inadvertently contract the affected side’s foot dorsiflexors ever so slightly, resulting in diminished H-reflex amplitude. As noted above, the effect of antagonist muscles on the H-reflex is well documented. Therefore, side-to-side amplitude differences for the H-reflex may be of diagnostic use, but the presently available studies are unconvincing regarding the most appropriate amplitude values to use clinically. Obviously, any side-to-side amplitude comparisons are of questionable validity in bilateral disease.

When using left/right criteria as sensitive as 1.1 ms, it is advisable to always use standardized distances to assist in reproducibility. Using anatomic landmarks may result in undue latency differences and predispose one to false-positive or false-negative results.

Just as for F-waves, it is possible to calculate the H-reflex conduction velocity for the proximal tibial nerve segment. A maximal H-reflex is obtained using the above noted technique. One then elicits a maximal M-response for supramaximal excitation of the tibial nerve from the same site of stimulation used for the H-reflex, i.e., the anode is rotated proximally. A time of 1 ms is subtracted to account for central synaptic delay. The distance measured is from the popliteal fossa stimulation site to the T11 spinous process. The formula used to calculate this proximal conduction over afferent sensory and effector motor fibers is: H-reflex CV (m/s) = (distance popliteal fossa to T11 × 2)/(H-reflex latency – M latency – 1 ms). This technique can document slowing of proximal conduction velocities in various peripheral neuropathies, such as diabetic polyneuropathy and uremic neuropathy. Of course, the same precautions regarding distance measurements noted for the F-wave also apply for the H-reflex.

H-Reflex: S1 Central Loop

The traditionally performed H-reflex to the lower limb uses a very long pathway reducing its ability to localize a lesion strictly to the S1 nerve root. As result, an attempt has been made to reduce the pathway over which an H-reflex can be obtained in the hopes of localizing a lesion to the S1 nerve root. This technique is promising, but requires larger studies to fully assess the sensitivity and specificity of the S1 central loop latency.

Recording Electrodes. The recording and ground electrodes are positioned in the same manner as for the H-reflex obtained through tibial nerve stimulation (see above).

Stimulation. A monopolar needle electrode serves as the cathode and is inserted 1 cm medial to the posterior superior iliac spine perpendicular to the patient’s frontal plane, and a surface anode is affixed to the anterior superior iliac spine. The cathode is gently inserted until it contacts the sacrum and is then withdrawn slightly. A stimulus pulse duration of 1 ms is used with a maximal pulse delivery of 0.5 Hz. The current is slowly increased until a combined direct motor (M) and indirect H-reflex (H) is obtained.
Instrument Parameters. The instrument’s sweep speed is set to 5 ms/div with high- and low-frequency filter settings of 10 kHz and 20 Hz. A gain of 0.5 to 1.0 mV/div is used to best visualize the response.

Reference Values. An H to M latency difference of 7 ± 0.3 ms is anticipated in healthy individuals (Fig. 6-14). A latency of 8.0 ms or greater is considered indicative of a lesion affecting the proximal S1 conducting pathway.

H-Reflex: Flexor Carpi Radialis

Recording Electrodes. It is possible to record an H-reflex from the FCR in most normal individuals. This muscle is innervated by C6 and C7 nerve roots and thus the H-reflex may be of assistance in assessing the neurophysiologic status of these two nerve roots and possibly conduction through the brachial plexus.38,67,196

E-1. A surface E-1 electrode is positioned over the belly of the FCR. This site is located one-third the distance from the medial epicondyle to the radial styloid.106

E-2. An E-2 is positioned over the brachioradialis muscle.

Ground. The ground should be secured to the skin just proximal to E-1.

Stimulation. The subject may be either supine or sitting with the elbow slightly flexed. The cathode is located over the median nerve at the antecubital fossa with the anode distal. A pulse width of 0.5–1.0 ms is optimal and the current intensity is slowly increased until the H-reflex is maximized with an absent or minimally present FCR CMAP. The presence of an H-reflex should be verified by increasing the stimulus intensity and observing for a disappearance of the response and replacement by an F-wave. The stimuli delivery should not exceed 0.5 Hz. Slight contraction of the FCR may be necessary to detect an H-reflex in some individuals.

Instrumentation Parameters. A sweep speed of 5 ms/div with a sensitivity of 0.5–1.0 mV/div and routine motor conduction filters should produce clearly recognizable H-reflexes.

Reference Values. One may anticipate a latency to the initial baseline deflection of 15.9 ± 1.5 ms.106 A side-to-side difference of 0.4 ± 0.3 ms is expected.

H-Reflex: Quadriceps Muscle

Recording Electrodes. Recording the H-reflex from the quadriceps muscle is somewhat more challenging than either the gastrocnemius-soleus or FCR muscles. The femoral nerve is a rather deep structure and difficult to activate with surface stimulation. Because of this, the current intensity is hard to incrementally deliver and there is little gradation between an absent and present direct M-response. The utility of a quadriceps H-reflex may be of assistance in the L3/L4 nerve root compromise.3,62

E-1. An E-1 electrode may be located over the main muscle bulk of the vastus medialis, vastus lateralis, or rectus femoris.

E-2. The patella is a convenient site for E-2.

Ground. This electrode should be situated just proximal to E-1.

Stimulation. The femoral nerve is excited in the inguinal region just distal to the inguinal ligament about the region of the femoral artery. Cathodal placement is distal to the anode. A pulse duration of 0.5–1.0 ms is delivered at less than 0.5 Hz with an intensity capable of producing an H-reflex with little, if any, M-response.

Instrumentation Parameters. The same instrumentation setup previously described for the FCR muscle can be used.

Reference Values. Onset H-reflex latencies to the rectus femoris, vastus medialis, and vastus lateralis muscles are described as 17.7 ± 1.8 ms, 18.4 ± 1.8 ms and 18.1 ± 1.7 ms, respectively.109 It is important to note that this response may not be obtainable even in normal individuals. Slight voluntary contraction of the quadriceps and a Jendrasik maneuver may assist in the facilitation of the response.

Central Nervous System Applications

Because a portion of the H-reflex involves the central nervous system, it is subject to both segmental and suprasegmental influences.6,58,71,135 This is best demonstrated by facilitation of an H-reflex with contraction of the muscle under investigation and inhibition with antagonist muscle contraction. It should be possible, therefore, to indirectly investigate various aspects of the central nervous system with reflex responses by studying the H-reflex in both health and disease.149,158 One common method of using the H-reflex for this purpose involves a technique of conditioning and test stimuli similar to refractory period experiments.

The central motor neuron pool excitability can be investigated by using a dual stimulation technique to document the H-reflex excitability or recovery curve. The recording electrodes are positioned as noted above for peripheral nerve techniques. A stimulator is secured to the tibial nerve at the popliteal fossa, which has the capability of delivering square wave stimuli 1.0 ms in duration with a variable interval between successive impulses. The initial or conditioning stimulus is delivered at or just below threshold for eliciting an H-reflex. This impulse generates a number of Ia afferent action potentials that enter the central nervous system over the reflex arc to condition the alpha motor neuron pool without causing a motor impulse. A second or test stimulus is then given through the same stimulating electrodes at a level sufficient to evoke a minimal direct M-response. Of course, this neural activation also should yield enough current to activate a large number of Ia afferents with the capability of generating an H-reflex independent of the conditioning impulse. The amplitude of the H-reflex produced by the test stimulus at increasing time intervals from the conditioning stimulus is then plotted for each sequential time interval and describes a characteristic shape reflecting central nervous system segmental and suprasegmental interactions.

Within a few milliseconds of the conditioning response, the H-reflex magnitude is maximal (Fig. 6-15). Increasing the time interval between conditioning and test stimulus results in a progressive amplitude decline of the H-reflex to a minimum value at approximately 75 ms. At interstimulus intervals between 100 and 200 ms the amplitude of the H-reflex again increases, peaking at about 150–200 ms. As the time interval between the conditioning and test stimulus continues to increase, the H-reflex amplitude demonstrates a second decline, reaching a minimum value at 200–400 ms. The H-reflex amplitude reveals a slow and progressive increase as the interstimulus time delay approaches 1000 ms. This H-reflex recovery curve is a graphic representation of the various central nervous system interactions all converging upon the alpha motor neuron pool and reflects the CNS’s normal physiologic state. Although the exact mechanism generating the above noted curve is unknown, an assumption based upon what little is known about the peripheral/central nervous system provides some insight into the various interactions resulting in the detected H-reflex recovery curve.

The initial H-reflex response is believed to result from the conditioning stimulus’ EPSPs generated by the Ia afferent input.
Taborikova H, Sax DS: Conditioning of H-reflexes by a preceding sub-
long-loop facilitation as shown above, and (3) depletion of transmitter
conditioning factors, i.e., (1) local EPSP (see text) on motoneuron, (2)
activation in the H-reflex magnitude because it provides EPSPs that
in a slightly depolarized state, thereby facilitating an H-reflex
activation of the muscle fibers also possess a motor innervation by small anterior
motor innervation to the intrafusal muscle fibers is provided by one
larger myelinated Ia nerve that wraps around the center of the
muscle fibers several times and is called the annulospiral
ending or primary sensory ending. Stretching of the extrafusal
muscle leads to a concomitant lengthening of the intrafusal
fibers, which in turn activates the annulospiral ending sending
impulses toward the central nervous system along the Ia affer-
ents. The Ia afferents cause EPSPs to be produced in the
homonymous alpha motor neurons, resulting in a contraction of the
muscle stretched. The intensity of muscle contraction de-
pends upon the number and degree to which the annulospiral
endings have been activated. The above description is the clas-

cic reflex arc familiar to all. Intrafusal muscle fibers also are in-
nervated by a second class of sensory fibers, group II fibers,
which form secondary muscle spindle endings or flower spray
endings. Because they do not directly participate in the reflex
arc the details of their function are not discussed. The intrafusal
muscle fibers also possess a motor innervation by small anterior
horn cells referred to as gamma motor neurons. Unlike the
alpha motor neurons with peripheral nerve fibers in the range of
12–21 \( \mu \)m, the gamma efferent diameters approximate 2–8 \( \mu \)m.
Activation of the gamma fibers also can cause the annulospiral
ending to contract, thus potentiating a contraction of the extra-
fusal muscle fibers. The gamma fibers, therefore, exert a power-
ful influence on the output of the muscle spindle, which
modulates the type of contraction resulting from a tendon tap.

The electrical activity associated with muscle contraction arising from a tendon tap can be recorded and displays various
latencies depending upon the length of the afferent and efferent
pathway. The impulse traverses the Ia afferents and alpha motor
neurons noted above. The H-reflex has been considered the
electrical equivalent of the tendon reflex. An important distinc-
tion between the H-reflex and the muscular contraction of a ten-
don tap is that the H-reflex directly activates the large Ia afferents

Specifically, the effects of the conditioning stimulus cause the
Ia afferent-induced EPSPs to occur that have a rise time of sev-
eral milliseconds. The duration of the rise time produces a facili-
	yatory effect that is still present when the test stimulus' Ia
afferents arrive. They find the alpha motor neuron pool already
in a slightly depolarized state, thereby facilitating an H-reflex
from the test stimulus. It is important to realize, however, that
the subthreshold conditioning stimulus has been depleting a Ia
afferent subpopulation of neurotransmitter substance. Experi-
mental results have demonstrated that the subthreshold stimulus
activates about 50% of the Ia afferents, hence accounting for the
50% reduction in the H-reflex by 75 ms.209 Once the stimulus
interval has increased beyond the facilitatory effect of the Ia a-
ferents, the full effect of the transmitter depletion becomes de-
tectable, reaching a maximum at about 75 ms. Despite the
above-noted depletion of neurotransmitter, a second elevation in
the H-reflex is noted peaking at about 200 ms. A long-loop fa-
cilitatory pathway traversing up to and back down the brainstem
has been proposed to account for this increase in H-reflex mag-
nitude. (Fig. 6-15) Some part of the Ia afferent input to the
central nervous system not only directly synapses with the
motor neuron pool, but also ascends through the dorsal spinal
cerebellar tract to involve the cerebellum and reticular sub-

ance. From these structures descending fibers may be con-
veyed through the vestibulospinal tract to also facilitate firing of
the motor neuron pool. It is proposed that the long loop reflex
time course coincides with and accounts for the second ele-
vation in the H-reflex magnitude because it provides EPSPs that
slightly depolarize the motor neuron pool when the condition-
ing stimulus of 200 ms reaches the anterior horn cells. Once the
time course of the long-loop reflex EPSPs has been exceeded,
the neurotransmitter depletion effect of the direct Ia afferent
pathway again becomes manifest. The H-reflex then does not
recover until the neurotransmitter from the initially depleted fa-
cilitatory inputs have been replenished, i.e., greater than 1 second.

This technique has been used to investigate the recovery curves
in normal newborns and patients with Parkinson’s disease,
spinal cord injury, and dystonia.155,165 Continued work is neces-
sary to clearly elucidate the pathways involved in the H-reflex
covery curve and its potential clinical applications.

**Tendon Reflex**

It is possible to record the electrical activity associated with
the reflex contraction of a muscle induced by acutely stretching
a tendon through percussion. A modified reflex hammer is re-
quired with the ability to initiate the instrument’s cathode ray
tube sweep. Surface or needle electrodes can then record the
reflex contraction of the muscle following the mechanical
energy delivered to the muscle’s tendon. In brief, the tendon tap
stretches its muscle, which in turn activates the muscle spindle.

The muscle spindle is a specialized structure consisting of a
connective tissue capsule surrounding several types of muscle
fibers referred to as intrafusal muscle fibers.11,198 The intrafusal
muscle fibers are approximately 15–30 \( \mu \)m in diameter and 4–7
mm long as opposed to the commonly thought-of muscle tissue,
extrafusal muscle fibers, which are 50-100 \( \mu \)m in diameter and
several millimeters to many centimeters in length. Sensory
innervation to the intrafusal muscle fibers is provided by one
larger myelinated Ia nerve that wraps around the center of the
muscle fibers several times and is called the annulospiral
ending or primary sensory ending. Stretching of the extrafusal
muscle leads to a concomitant lengthening of the intrafusal
fibers, which in turn activates the annulospiral ending sending
impulses toward the central nervous system along the Ia affer-
ents. The Ia afferents cause EPSPs to be produced in the

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**Figure 6-15.** Postulated pathways mediating the H-reflex’s magnitude as elicited with conditioning stimuli. **A**. Solid line signifies that 2 of 4 Ia afferent fibers were excited that in turn monosynaptically synapse with a motor neuron (MN) but were subthreshold for evok-
ing a response. This same Ia volley simultaneously proceeded rostral-
ward up the dorsal column tracts (DSTC) to the various structures
depicted in the circle. The vestibulospinal tract (VST) then conveys
these volleys to activate the motor neuron, forming the long-loop reflex. **B**. The continuous line reveals the time course of the condition-
ing of the observed H-reflex by the subthreshold H-reflex stimulus. It is suggested that the H-reflex amplitude curve is a composite of three
conditioning factors, i.e., (1) local EPSP (see text) on motoneuron, (2)
long-loop facilitation as shown above, and (3) depletion of transmitter
in the synapses activated by the conditioning stimulus. (From Taborikova H, Sax DS: Conditioning of H-reflexes by a preceding sub-
Figure 6-16. Axon reflex. A, Series of axon reflex responses designated 1, 2, and 3 demonstrating a constant latency. Note that the axon reflex can either proceed or follow the F-wave. B, Diagrammatic explanation of the axon reflex (see text). (From Kimura J: Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice. Philadelphia, F.A. Davis, 1989, with permission.)

in the nerve and bypasses the intrafusal muscle fibers. This view, however, may be an oversimplification. The above-noted “classic” description of the interaction between the Ia afferents and alpha/gamma motor system may be less well understood than previously thought. Human studies reveal that the fusimotor drive of the gamma system is not necessary to elicit a tendon reflex. The ease with which a tendon reflex can be obtained may be more related to the “central excitability” state of the alpha motor neurons than the gamma system’s influence on the muscle spindle. The muscle’s response to a constant tendon tap varies, suggesting that the central nervous system’s segmental and suprasegmental influences are important and necessary factors affecting the reflex response. The H-reflex demonstrates the same independence of the fusimotor system that the tendon reflex does. Additionally, the afferent volley induced by electrical stimulation for the H-reflex is a synchronized volley, whereas the tendon tap afferent volley is considerably more dispersed in time. EPSP rise times from tendon percussion are 8.3 ± 2.5 ms, whereas those from the H-reflex are 3.5–5.5 ms. The combination of dispersed action potential volleys and longer rise times for the tendon tap reflex suggest that it may not travel the same central pathways to achieve the muscle contraction observed with the H-reflex. There is sufficient time in tendon tap reflex activity, temporal dispersion of action potential volleys combined with relatively long EPSPs, to traverse disynaptic and trisynaptic pathways centrally. This also applies to a lesser degree to the H-reflex. These findings suggest that the classic acceptance of a monosynaptic reflex arc for all fibers activated by either a tendon tap or H-reflex traveling a single synapse may be inaccurate. A more plausible explanation suggests that the motor neurons of lowest threshold may be activated by a monosynaptic arc through the fastest Ia afferents, whereas motor neurons of higher threshold fire through several synapses initiated by the fastest Ia afferents and single synapses of relatively slower Ia afferents. This central combination of synapses and rate offferent conduction serve to yield a more synchronous output independent of the fusimotor system.

Axon Reflex

A response may occasionally be observed with a constant latency between that of the CMAP and the F-wave or exceeding the F-wave, particularly from the small muscle of the hand and rarely from the intrinsic foot muscles (Fig. 6-16). The potential is referred to as an axon reflex or A-wave. This intermediate potential is usually elicited with a submaximal stimulus and displays a constant latency, unlike the varying F-wave. The presumed physiology of the axon reflex involves some type of neural damage with a collateral sprout from a proximal aspect of the nerve to the muscle. A submaximal stimulus distal to the collateral’s branch point activates a significant portion of the nerve but spares some fibers. The orthodromic impulses results in a less than maximal CMAP while the antidromic impulse proceeds proximally to encounter the branch point. A portion of the electrical impulse proceeds distally along the collateral while the remainder of the impulse continues into the CNS. The action potentials in the collateral sprout then activate a small portion of the muscle resulting in the axon reflex response. The major antidromic impulse backfires in the motor neuron pool to then yield the F-wave. Increasing the stimulus intensity eliminates the axon reflex because the entire nerve is now depolarized, including the aberrant collateral branch, thus resulting in a synchronous activation of the entire nerve. The antidromic impulses are induced in both the collateral branch and the main nerve to collide proximally, thus eliminating the delayed axon reflex action potential. Rarely, one may note that the axon reflex persists despite a supramaximal stimulus. In this instance the mechanism for the axon reflex is obscure but may occur because the branch generating the axon reflex is somehow isolated from the impulse either through connective tissue, electrical “shielding” from muscle or bone, or some other poorly understood reason.

It is also possible to localize the site of the collateral sprout producing the axon reflex by slowly moving the site of neural depolarization to sequentially more proximal locations. As the site of neural activation moves more proximally, the latency of the CMAP increases, whereas the axon reflex latency decreases because the branch point is closer to the cathode. When the axon reflex disappears, the stimulus is now just proximal to the branch point. Should the branch point depart the damaged nerve at a site more distal than the lesion, localization is not possible. This is rather obvious as more proximal stimulation does not result in an absent axon reflex, indicating a more distal branching. In this case, one can determine the collateral sprout’s conduction velocity. Of course, should an immature collateral be present, its conduction velocity can be considerably slower than that of the main portion of the nerve. In this instance, it is possible for the axon reflex to follow instead of precede the F-wave. The presence
of an axon reflex is simply a nonspecific indication that the nerve has most likely experienced some type of chronic insult that resulted in a collateral sprout. An axon reflex can be seen in multiple chronic neuropathies such as radiculopathies, plexopathies, peripheral neuropathies, and motor neuron disease. It is possible to observe axon reflexes in some normal persons with routine nerve conduction studies because of possible subclinical nerve injury or some other ill-defined reason. Axon reflexes, however, are a common occurrence in normal individuals when examined with single-fiber electromyography. Caution should be exercised when a late potential is observed. In addition to the possibility of detecting an F-wave, H-reflex, or axon reflex, there are a number of potentials following a stimulus that are none of the above. Additional “late” potentials can be seen with neural stimulation because of repetitive firing of the nerve trunk, slowly conducting poorly myelinated nerves, ephaptic conduction of injured nerves, intramuscular ephaptic conduction loops (complex repetitive discharge), an axon loop, or other unexplained electrical pathways.

An A-wave can be observed in many different types of lesion including polyneuropathy, radiculopathy, motor neuron disease, Guillain-Barré syndrome, plexopathies, myopathies, and focal nerve injuries. It is rare, however, to observe an axon reflex response in normal individuals with the exception of an occasional A-wave detected in the foot intrinsic muscles innervated by the tibial nerve. Therefore, documenting an A-wave in any response other than from the tibial nerve is considered indicative of pathology by some authors.

Sympathetic Skin Response

All of the previously described nerve conduction techniques examine either sensory or motor nerve fibers or both. The peripheral nervous system, however, not only contains sensory and motor fibers, but autonomic nerves as well. The majority of nerve conduction studies only evaluate the fastest-conducting fibers and do not consider the more slowly conducting myelinated or unmyelinated fibers, e.g., the sympathetic fibers contained within the peripheral nerves.

Initially, the autonomic nervous system could only be investigated electrophysiologically with the insertion of a fine needle recording electrode into the substance of a peripheral autonomic nerve and recording the ensuing electrical activity, i.e., microneurography. Although microneurography is capable of distinguishing between the sympathetic fibers conveying impulses controlling intramuscular blood supply and sympathetic nerves influencing cutaneous blood vessels and sweat glands, the technique is quite demanding and limited primarily to research facilities. A simple method of evaluating sympathetic skin activity is the galvanic skin response. Following an emotional or noxious stimulus the sudomotor activity mediated by the sympathetic nervous system results in an alteration in the skin’s resistance to an electrical current. It is possible to use commonly available electrodiagnostic medicine equipment to examine a similar response mediated by the sympathetic system, i.e., the sympathetic skin response.

**Recording Electrodes.** The sympathetic skin response is relatively easy to perform and should be tried by beginning practitioners. Commonly available surface recording electrodes are used. Only a few of the possible nerve techniques are described but essentially any aspect of an limb may be used.

**E-1.** For upper limb median nerve studies an E-1 recording electrode is secured to the palmar surface of the hand. In the lower limb, an E-1 electrode can be positioned to the dorsum of the foot for peroneal nerve stimulation and on the foot’s plantar aspect for tibial nerve excitation.

**E-2.** In the upper limb, E-2 is located on the dorsum of the hand. For peroneal nerve stimulation, the plantar aspect of the foot is used, whereas the foot’s dorsum is appropriate for tibial nerve activation.

**Ground.** This electrode is positioned just proximal to the E-1 electrode with respect to the cathode’s location.

**Stimulation.** The stimulator is positioned in the routine manner over the desired median nerve at the wrist and peroneal and tibial nerves at the ankle. A stimulus is applied to each nerve with a pulse width of 0.1 ms and a current intensity approximating 10–20 mA or enough to elicit a slightly uncomfortable sensation. Because the response readily habituates, the stimuli are delivered at irregular intervals over several minutes. There should be a pause of several seconds between successive stimuli. About 10 stimuli are recommended as each response can vary somewhat and only responses that are consistent are selected for analysis.

It is possible to investigate the conduction velocity of the fibers mediating the sympathetic skin response (Fig. 6-17). A second stimulus site at a more proximal location is selected once a satisfactory distal potential is obtained. The same parameters are used for proximal stimulation as described above.

**Instrumentation Parameters.** A sweep speed of 500 ms/div is optimal as this response is mediated by slowly conducting C fibers and substantial time is required to resolve the sympathetic skin response. Of paramount importance is the bandwidth selected for this technique. A low-frequency filter of 0.5 Hz or less is necessary as the recorded potential has significant low frequency components. The high frequency filter should approximate 2,000 Hz. An amplifier sensitivity of 200–1,000 µV/div usually suffices for most individuals.

**Reference Values.** The onset latencies for upper and lower limb studies are 1.39 ± 0.07 seconds and 1.88 ± 0.11 seconds, respectively. Conduction velocities in the upper limb approximate 1.57 ± 0.11 m/s and for the lower limb are 1.02 ± 0.07 m/s. Amplitudes are 806 ± 322 µV and 640 ± 276 µV in the upper and lower extremities, respectively.

**Additional Comments.** Although the actual technique of eliciting a sympathetic skin response is relatively easy, the actual response can be quite variable. Habituation of the response also can be a problem. The practitioner should be prepared for considerable amplitude variation from one response to the next. It is important to have the patient relaxed and
respond to as little external stimuli as possible between successive stimuli. Temperature can have a significant effect on the response and should be monitored throughout the procedure with a recommended temperature for upper and lower limb studies similar to that of routine nerve conduction studies. A correction factor of 0.088 seconds/°C is recommended. Preliminary investigations in patients with various peripheral neuropathies suggest that the sympathetic skin response may be of some assistance in evaluating autonomic fibers in these diseases. Continued studies with this technique are required to adequately determine its clinical utility.

CONCLUSION

The specialized nerve conduction techniques discussed in this chapter are complementary to those described in the previous chapter. Although a number of the more specialized methods of investigating the nervous system may not be used in most patients routinely, it is nevertheless important for the practitioner to be familiar with both the methodology and utility of these techniques in order to know when their use is most efficacious. These techniques should be practiced until proficiency is achieved so that an absent response can be confidently declared abnormal as opposed to its absence being a result of technical difficulty or a lack of skill. The actual utility of the special nerve conduction is discussed throughout the remainder of the text with respect to specific disorders.

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Chapter 6 SPECIAL NERVE CONDUCTION TECHNIQUES — 255


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