Specialty Section on Surgical Neuromonitoring

INTRAOPERATIVE MONITORING OF SEGMENTAL SPINAL NERVE ROOT FUNCTION WITH FREE-RUN AND ELECTRICALLY-TRIGGERED ELECTROMYOGRAPHY AND SPINAL CORD FUNCTION WITH REFLEXES AND F-RESPONSES

A POSITION STATEMENT BY THE AMERICAN SOCIETY OF NEUROPHYSIOLOGICAL MONITORING

Ronald E. Leppanen, Ph.D., D.ABNM, FASNM

ABSTRACT. Background Context. Orthodromic ascending somatosensory evoked potentials and antidromic descending neurogenic somatosensory evoked potentials monitor spinal cord sensory function. Transcranial motor stimulation monitors spinal cord motor function but only activates 4–5% of the motor units innervating a muscle. Therefore, 95–96% of the motor spinal cord systems activating the motor units are not monitored. To provide more comprehensive monitoring, 11 techniques have been developed to monitor motor nerve root and spinal cord motor function. These techniques include: 1. neuromuscular junction monitoring, 2. recording free-run electromyography (EMG) for monitoring segmental spinal nerve root function, 3. electrical stimulation to help determine the correct placement of pedicle screws, 4. electrical impedance testing to help determine the correct placement of pedicle screws, 5. electrical stimulation of motor spinal nerve roots, 6. electrical stimulation to help determine the correct placement of iliosacral screws, 7. recording H-reflexes, 8. recording F-responses, 9. recording the sacral reflex, 10. recording intralimb and interlimb reflexes and 11. recording monosynaptic and polysynaptic reflexes during dorsal root rhizotomy. **Objective.** This paper is the position statement of the American Society of Neurophysiological Monitoring. It is the practice guideline for the intraoperative use of these 11 techniques. **Methods.** This statement is based on information presented at scientific meetings, published in the current scientific and clinical literature, and presented in previously-published guidelines and position statements of various clinical societies. **Results.** These 11 techniques when used in conjunction with somatosensory and transcranial motor evoked potentials provide a multiple-systems approach to spinal cord and nerve root monitoring. **Conclusions.** The techniques reviewed in this paper may be helpful to those wishing to incorporate these techniques into their monitoring program.

KEY WORDS. H-reflex, F-response, electromyography, EMG, intraoperative monitoring, spinal cord, spinal nerve root.

INTRODUCTION

This document presents the American Society of Neurophysiological Monitoring (ASNM) position statement regarding the utilization of free-run and electrically-triggered electromyography (EMG) for monitoring segmental motor nerve root function and spinal cord function with reflexes and F-responses.

This statement is based on information presented at scientific meetings, published in the current scientific and clinical literature, and presented in previously-published guidelines and position statements of various clinical

From the Knoxville Neurology Clinic, 939 Emerald Avenue, Suite 907, Knoxville, Tennessee 37917.
E-mail: Leppanen@AOL.com

societies. This document may not include all possible methodologies and interpretive criteria, nor is it intended to exclude any new alternatives. ASNM provides general information and education materials as a service in furtherance of its non-profit and tax-exempt status.

Traditional studies

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

Since the development of transcranial stimulation of the motor cortex [13], a variety of electrical [14, 15] and magnetic [16] stimulation techniques have been used intraoperatively. These motor techniques provide information about long-tract function, but also provide some information regarding segmental interneurons and anterior gray matter function because successful transmission to the lower motor neuron depends upon the functional integrity of this segmental system [12]. Direct electrical stimulation of the exposed motor cortex activates only about 5% of the motor units in the target muscle. This degree of motor unit activation is the same as conventional transcranial electrical simulation in awake and relaxed subjects [17]. Intraoperative transcranial electrical stimulation activates from 3.0–4.0% of the total muscle fibers and from 3.1–3.9% of the motor units in the abductor hallucis muscle [18]. Monitoring antidromic somatosensory spinal cord activity resulting from electrical spinal cord stimulation and recording of peripheral nerve activity has been reported. These peripherally-recorded descending potentials are called descending neurogenic evoked potentials (DNEPs) [19]. Initially, DNEPs were thought to be mediated by the long spinal cord motor tracts, but recent intraoperative collision studies in which spinal cord stimulation was delayed following unilateral stimulation of the tibial nerve at the ankle demonstrated that DNEPs and SSEPs are mediated through common spinal cord pathways. These studies demonstrated that in idiopathic scoliosis patients, DNEP activity represents antidromic spinal cord somatosensory activity [20–24]. DNEPs are more sensitive for detecting spinal cord sensory compromise than are SSEPs [24]. The recording of DNEPs is the result of the activation of antidromic sensory spinal cord pathways with the peripherally-recorded DNEP nerve action potentials representing 20% of the total peripheral nerve fibers [21]. Recording of motor evoked potentials (MEPs) monitors those systems of the spinal cord that are responsible for mediating the function of muscle strength.

Reflex and F-response studies

The recording of intraoperative reflex and F-response activity can be used to monitor those systems of the spinal cord that are responsible for the control of complex motor behavior [25–29]. Reflex and F-response techniques monitor complex spinal cord function and monitor activity in highly-integrated ascending, descending and spinal interneurons. These techniques also monitor function of the dorsal and ventral roots.

The advantage of the use of intraoperative reflex and F-response recordings to detect the onset of spinal cord or nerve root compromise is that the recordings are single sweep. The reflex recordings are therefore real-time and there is no delay after the onset of spinal cord or nerve root compromise that is present when using averaged evoked potentials. Reflex and F-response recordings provide the surgeon with immediate feedback about spinal cord or nerve root function. They can usually be acquired continually throughout surgery with little or no noticeable patient movement. Additionally, anesthesia is less restrictive than for other methods of monitoring motor cord function. Reflex changes are very sensitive to spinal cord compromise, with reflex changes occurring before ascending and descending spinal cord long-tract recordings change. Intraoperative reflex and F-response studies provide a model for understanding the mechanisms of spinal cord pathophysiology. Intraoperative reflex studies have been used to help identify sensory and motor roots and to help identify which sensory rootlets should be sectioned during selective dorsal root rhizotomy.

Segmental motor nerve root studies

The motor unit consists of the anterior horn cell, its axon and all the muscle fibers innervated by that axon [30]. Segmental motor nerve root monitoring involves monitoring the function of the motor unit axons by recording free-run and electrically-stimulated electromyographic (EMG) activity from the muscle fibers of the motor units.

Summary

Mixed-nerve SSEPs are neither specific nor sensitive to individual nerve root function, and dermatomal SSEPs only

represent nerve root sensory function. Since transcranial electrical stimulation activates from 3.1 to 3.9% of the motor units, this technique results in conduction in only from 3.1 to 3.9% of the motor axons in the motor nerve root and using this technique would not determine what is happening in the remainder of the 96.9 to 96.1% of the motor axons. Therefore, free-run EMG, electrically-triggered EMG, reflexes and F-response techniques have been utilized to monitor motor nerve root and spinal cord function.

These techniques, when combined with SSEP and MEP recordings made sequentially or simultaneously, provide a multiple-systems integrated approach to spinal cord monitoring.

ANESTHETIC TECHNIQUE

Overview

The technique used depends upon what systems are being monitored (i.e., somatosensory evoked potentials, motor evoked potentials, F-responses, H-reflexes, free-run EMG and electrically-evoked EMG). In order to record EMG activity intraoperatively it is critical that the anesthetic technique does not inhibit the activity of the spinal interneurons and the alpha motor neurons. Also, adequate neuromuscular junction (NMJ) transmission must be present in order to record EMG activity. The same anesthetic technique can be used for the recording of extracranial free-run EMG, compound motor action potentials (CMAPs) following spinal cord and nerve root stimulation, and for the recording of monosynaptic and polysynaptic reflexes and F-responses [25, 26, 29, 31].

Neuromuscular junction monitoring

Five methods are available for evaluating NMJ function using evoked responses: measurement of the evoked mechanical response of muscle (mechanomyography), measurement of the evoked electrical response of the muscle (electromyography), measurement of acceleration of the muscle response (acceleromyography), measurement of the evoked electrical response in a piezoelectric film sensor attached to muscle, and sonomyography. There are 5 electrical nerve-stimulation EMG techniques for monitoring neuromuscular junction function. These are the single twitch (T1%), train-of-four ratio (TOF, TR%), tetanus, post-tetanic stimulation, and pulse or double-burst techniques. The TOF technique is most often used for determining the degree of neuromuscular blockade in the operating room. With TOF nerve stimulation a train of

four supramaximal stimuli are given with an interstimulus interval of 0.5 s (2 Hz) for a train-duration of 2 s. When used continuously, the train of stimuli is repeated every 10th to 20th s. In order to activate all the motor fibers innervating a muscle, the intensity of stimulation must be supramaximal. Therefore, the electrical stimulus applied is usually at least 20 to 25% above the intensity needed to produce a maximal muscle response. Stimulation of the skin surface over a nerve is with a monophasic rectangular pulse of between 200 and 300 μs duration. Computerized stimulators use a constant-current output of at least 70 mA. When using subdermal EEG electrodes for stimulation, the amount of current needed to produce a suramaximal response is substantially less than that needed with surface stimulation. Direct muscle stimulation should be avoided, for it bypasses the neuromuscular junction, so that evoked responses may persist in the presence of complete neuromuscular junction blockade. Neuromuscular junction monitoring should be assessed in the same or neighboring muscle groups from which EMG activity is monitored.

The diaphragm is most resistive to neuromuscular junction blockade. The facial muscles are less resistive than the diaphragm, and the muscles of the limbs are less resistive than the facial muscles. Neuromuscular junction blockade develops faster in centrally-located muscles, such as the larynx, jaw and diaphragm, than in more peripherally-located muscles such as the abductor digiti minimi muscle. In addition to developing more quickly, neuromuscular junction blockade in these central regions is less profound and recovers quickly. The pattern of response to TOF stimulation varies with the type of neuromuscular junction blocker administered, because the 2 relaxant types, depolarizing and non-depolarizing agents, have different mechanisms of action.

With TOF stimulation each stimulus in the train causes the muscle to contract (T1, T2, T3, T4). Fade of the amplitude of each of the last 3 responses in relation to the amplitude of the first response is the basis for evaluation. Dividing the amplitude of the fourth response by the amplitude of the first response provides the TOF ratio (T1:T4, TR%). In the control response before administration of a muscle relaxant, all four responses are ideally the same amplitude and the TOF ratio is 1.0. A computer may be used to quantify the relationship of the TOF responses. A mechanical strain gauge or accelerometer may also be used for recording.

Non-depolarizing neuromuscular junction blocking agents are competitive inhibitors of the acetylcholine (ACh) receptors. They compete with ACh for the active, or binding sites on the alpha subunits of the muscle membrane receptors and prevent ACh from depolarizing the membrane. With TOF stimulation the muscle response to stimulation fades over time because of a decrease in the amount of ACh released from the pre-junctional nerve terminal

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

Depolarizing neuromuscular junction agents block neuromuscular junction function by depolarizing the muscle membrane without subsequent re-polarization, so that ACh released by nerve action potentials cannot activate the post-junctional muscle membrane. ACh is rapidly hydrolyzed by acetylcholinesterase and is cleared from the synaptic cleft. In contrast, the depolarizing agent is not susceptible to hydrolysis by acetylcholinesterase and is not removed from the junctional cleft until after it is eliminated from the plasma, which is very slow. Therefore, the effect of the depolarizing agent is prolonged. With a depolarizing neuromuscular junction blocking agent the response to TOF stimulation is different than that seen with the use of a non-depolarizing agent. During a partial depolarizing block, no fade occurs in the TOF response. Ideally the TOF ratio is approximately 1.0. With higher concentrations of depolarizing agents, the four TOF response amplitudes decrease at the same time and equally. Therefore, the TOF ratio cannot be used when a depolarizing agent is used. When it is desirable to quantify the degree of neuromuscular junction blockade with the use of the TOF ratio, non-depolarizing neuromuscular junction blocking agents should be used [32, 33].

Neurophysiological basis for 2 Hz train-of-four stimulation

During neuromuscular junction transmission there is propagation of neural depolarization into the axon terminal.

The depolarization causes voltage-sensitive calcium ions to enter the axon terminal. This calcium facilitates the release of synaptic vesicles which contain ACh. Synaptic vesicles in the terminal axon active zone fuse with the terminal membrane. There are 1,000–30,000 synaptic vesicles in the terminal active zone. A single synaptic vesicle contains approximately 5,000–10,000 molecules of ACh. There are approximately 200,000–400,000 individual synaptic vesicles in most nerve terminals. The active zone vesicles release ACh into the synaptic cleft by exocytosis. When an action potential invades the terminal axon the amount of calcium that enters is sufficient to facilitate the release of approximately 50–100 synaptic vesicles. The ACh diffuses across the synaptic cleft and binds to the ACh receptors. There is depolarization of the post-junctional membrane and a muscle action potential is generated.

Once the vesicles in close proximity to the prejunctional membrane have released their ACh content, there are fewer vesicles present when compared to prior to the action potential. The ACh released during the first several stimulations is greater than that released during ensuing stimulations. The active zone vesicles are replaced exponentially in 5–10 s after each stimulus. The replacement stores are 1,000 vesicles in the active zone which constitute the immediately-available ACh store, 300,000 vesicles that constitute the mobilization store, and 10,000 vesicles that are the transition compartment. After the release of ACh, calcium diffuses out of the axon in 200 ms. If another action potential invades the axon terminal before the calcium has diffused out of the axon, the calcium will facilitate the release of vesicles to cause muscle contraction even though the number of synaptic vesicles in the terminal active zone has been reduced. With TOF stimulation an interstimulus interval of 500 ms (2 Hz) allows enough time for most of the calcium to diffuse out of the nerve terminal. Faster rates of stimulation with interstimulus intervals less than 200 ms should be avoided, because calcium will accumulate and ACh release will be facilitated despite the depletion of ACh stores. Faster rates of stimulation may also cause depression of neuromuscular junction transmission, because ACh release exceeds the rate of mobilization of ACh, leading to substantial depletion of ACh stores. At slower rates of stimulation with a longer interstimulus interval, calcium accumulation no longer compensates for the depletion of ACh stores. A TOF stimulation rate of 2 Hz provides the most sensitive balance between calcium accumulation and reduction of ACh stores, so that adequate neuromuscular junction function can be maintained and any change in the amplitude of the response is due to the effect of the neuromuscular junction blocking agent [34, 35].

Ideal technique

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

When monitoring the lower extremity, the peroneal nerve may be stimulated at the knee and TOF CMAPs may be recorded from the tibialis anterior muscle. The tibial nerve may be stimulated at the ankle and CMAPs recorded from the abductor hallucis muscle. The tibial nerve may be stimulated in the popliteal fossa and the CMAPs may be recorded from the gastrocnemius muscle. There are reports of recording EMG activity with different levels of neuromuscular blockade, and one technique used is that during surgery the level of muscle relaxation is maintained at 50% or less (computerized recording) with a continuous infusion of a non-depolarizing agent such as atracurium. When monitoring surgical procedures (such as cauda equina tumors) where more sensitive EMG recordings are needed, no muscle relaxants should be used [38].

The effect of neuromuscular junction blockade on direct-stimulation nerve root threshold was examined in 21 roots in 10 patients. Neuromuscular junction blockade below 80% provided nerve root stimulation thresholds similar to thresholds without blockade [39]. Anesthesia is maintained with N_2O 50% or less in O_2, a continuous infusion of alfentanil and 0.5% or less of isoflurane. Higher concentrations of isoflurane have been found to suppress triggered EMG recordings [40, 41]. A continuous infusion of propofol may also be used. When performing transcranial electrical stimulation it is important to use EEG to detect cortical suppression secondary to propofol use, which may make it difficult to record MEPs. With propofol, burst-suppression has been reported to occur with infusion rates from 170 to 500 μg/Kg/min [42].

Effects of different agents on different levels of the neuromuscular system

With double-pulse spinal cord stimulation, propofol did not attenuate CMAPs, but 0.5% halothane, enflurane or isoflurane attenuated CMAPs from 25 to 50% of baseline values [40]. Polysynaptic sacral reflexes have been recorded with propofol, fentanyl and N_2O with no muscle relaxant [43]. H-reflexes and F-responses are used to determine the level of motor neuron excitability intraoperatively. In normal human volunteers, 1–1.5% enflurane was found to decrease H-reflex amplitudes from 35 to 100% of baseline values [44]. Isoflurane and isoflurane plus N_2O were found to decrease H-reflex and F-response amplitude and F-response persistence (number of measurable F-responses divided by the number of stimuli). H-reflex amplitude was decreased to 48.4% of the baseline with 0.6 MAC isoflurane, and to 33.8% of the baseline with 1.2 MAC isoflurane. F-response amplitude and persistence decreased to 52.2 and 44.4% of the baseline respectively at 0.6 MAC isoflurane, and decreased to 33.8 and 21.7% of the baseline respectively at 1.2 MAC isoflurane. With 1.0 MAC isoflurane the H-reflex amplitude was decreased by 32.5, 33.3 and 30.4% of baseline levels at 30, 50 and 70% N_2O, respectively [41]. The results of these studies were not able to determine if the attenuation of H-reflexes and F-responses by inhalation agents was due to a direct inhibition of the 1a fibers, to direct effect on the alpha motor neuron membrane, or to an altered balance between supraspinal excitatory and inhibitory pathways projecting on the alpha motor neuron.

When intraoperative EMG recordings are interpreted it is important to consider the effect of these different agents on the different levels of the neuromuscular system.

MONITORING SEGMENTAL MOTOR NERVE ROOT FUNCTION

Anatomical and neurophysiological basis of segmental motor nerve root monitoring

The motor unit consists of the anterior horn cell, its axon and all the muscle fibers innervated by that axon [30]. Segmental nerve root monitoring involves monitoring the function of the motor unit axon by recording free-run and electrically-stimulated electromyographic (EMG) activity from the muscle fibers of the motor units. The number of nerve fibers innervating a muscle and the number of muscle fibers per motor unit varies from one muscle to another. The lateral rectus muscle of the eye has 1,740 motor

units, 22,000 muscle fibers, and 13 muscle fibers per motor unit. The gastrocnemius muscle has 579 motor units, with 1,730 muscle fibers per motor unit, for a total of over 1,000,000 muscle fibers. Small muscles that are involved in precise movements have fewer muscle fibers per motor unit. The individual muscle fibers of a motor unit are not all grouped together, but occupy more than one fascicle. There is considerable intermingling of muscle fibers from different motor units. The muscle fibers of a motor unit occupy a circular area with a radius of 5 to 11 mm. This area is occupied by from 10 to 30 motor units [45]. The muscle fibers of a motor unit are confined to innervation territories of a neuromuscular compartment. Anatomically the different compartments of the whole muscle have different functions, even though the different compartments share a common tendon for insertion [46].

The neuromuscular junction (end plate) is at the middle of the muscle fiber. The terminal-zone-of-innervation band of end plates corresponds to the motor point, and is the point of lowest threshold for electrical stimulation. The shape of the terminal innervation zone varies with the arrangement of the muscle fibers in different muscles (parallel and circumpennate arrangement). The amplitude of a single muscle fiber discharge may be from 20 to 1,000 μV. The amplitude of the discharge of all the muscle fibers in a motor unit may be from 100 to 3,000 μV [45].

Supramaximal stimulation of a motor nerve innervating a muscle results in the recording of the CMAP, which is the result of activation of all the motor units in that muscle. In the clinical laboratory, the amplitude of the CMAP recorded from the abductor pollicis brevis muscle following stimulation of the median nerve at the wrist is from 5.4 to 30 mV. The amplitude of the CMAP recorded from the abductor digiti minimi muscle following stimulation of the ulnar nerve at the wrist is from 4.0 to 22.0 mV. The amplitude of the CMAP recorded from the abductor hallucis muscle following stimulation of the tibial nerve at the ankle is 5.8–32 mV. The amplitude of the CMAP recorded from the extensor digitorum brevis muscle following stimulation of the deep peroneal nerve at the ankle is 2.6–20.0 mV. In the operating room, these CMAP amplitudes are obliterated or significantly compromised by the use of NMJ blocking agents. When measuring latency, the sharpest rise-time from baseline occurs when the active recording electrode is directly over the motor point. It is therefore necessary to know the location of the motor points of the muscles from which the recordings are made, in order to optimize both amplitude and latency [47].

It is important to take these anatomical and neurophysiological principles into consideration when recording EMG signals in the operating room. When recording EMG activity with needle electrodes, the activity recorded may be the result of activation of only a few motor

units innervating that muscle. Other motor units may be activated but because of the location of the recording electrodes, this activation will go undetected. A monopolar EMG needle records the summated activity of from 9 to 17 muscle fibers [48]. When using a monopolar EMG needle to record from the tibialis anterior muscle, it is important to realize that only 9 to 17 of the total 270,000 muscle fibers in the tibialis anterior muscle are being monitored. The number of muscle fibers that contribute to the electrical activity that is recorded by subdermal EEG electrodes has not been determined. When electrically stimulating a nerve root or part of the plexus or peripheral nerve, it is important to know what the normal amplitude and latency is for the recorded muscle.

Segmental motor nerve root function can be monitored by recording from muscles innervated by the cervical, thoracic, lumbar and sacral spinal nerve roots. The limb muscles are innervated by multiple root levels. The various human myotomal charts are the result of multiple clinical studies and are based on patient history, physical examination, electrodiagnostic evaluations and surgical observations. As a result, there may be substantial differences from one myotomal chart to another, with less than unanimous agreement regarding the myotomal innervation of a given muscle [49]. In addition to paraspinal muscles, some of the commonly-used muscles for these intraoperative recordings are [50]:

Nerve Root		Muscles Innervated
Cervical	C2, C3, C4	Trapezius, Sternocleidomastoid, Spinal portion of the Spinal Accessory Nerve
	C5, C6	Biceps, Deltoid
	C6, C7	Flexor Carpi Radialis
	C8, T1	Abductor Pollicis Brevis, Abductor Digiti Minimi
Thoracic	T5, T6	Upper Rectus Abdominis
	T7, T8	Middle Rectus Abdominis
	T9, T10, T11	Lower Rectus Abdominis
	T12	Inferior Rectus Abdominis
Lumbosacral	L2, L3, L4	Vastus Medialis
	L4, L5, S1	Tibialis Anterior
	L5, S1	Peroneus Longus
Sacral	S1, S2	Gastrocnemius
	S2, S3, S4	External Anal Sphincter

The ventral primary rami of L1–L3 with a branch from T12 and L4 form the lumbar plexus. The ventral primary rami of L4–S3 form the sacral plexus. Normally the L4 nerve is subdivided between the lumbar and sacral plexuses. Occasionally the L3 nerve is the lowest in the lumbar plexus, and at the same time contributes fibers to the sacral plexus. When this occurs, the nerve root contribution to the lumbar plexus is shifted superiorly and is termed pre-fixed. More frequently, the L5 nerve root is divided between the lumbar and sacral plexuses; in this case the nerve root contribution to the lumbar plexus is shifted inferiorly and is termed postfixed [51].

There are three types of extradural lumbar nerve root anomalies, as follows:

Type I. Two separate nerve roots arise from a single dural sheath.

    1A. Two pairs of nerve roots arise from a single dural sleeve.
    2B. A dural sleeve from one level arises adjacent to the next lower level sleeve's site of origin.

Type II. Two nerve roots exit the spinal column from a single neural foramen.

    2A. The foramen is unoccupied.
    2B. There is a nerve root in all foramina, but one has two roots.

Type III. There is communication between individual nerve roots, creating a neural anastomosis [52–56].

These extradural anomalies occur in 8.5% of patients, and intradural communication between lumbar nerve roots occurs in 11–30% of patients [57].

Within the cauda equina the motor fibers are located anteromedial to the posterolateral sensory fibers. A posterior injury may preferentially affect sensory fibers more than motor, and an anterior lesion may damage motor fibers prior to sensory fibers. In the lumbar region the dorsal root ganglia is located immediately inferior to the pedicle 90% of the time. Two percent of the time it is located medial to the pedicle, in the lateral recess. Thirty two percent of the time the lumbar dorsal root ganglia lie directly over the same aspect of the associated intervertebral disc for that respective level [58, 59].

The majority of the time, the C5-T1 nerve roots contribute to the formation of the brachial plexus. The brachial plexus is said to be prefixed when C4 contributes extensively. In this instance the cervical nerve root contribution to the plexus is shifted superiorly by one level, and

T1 contributes minimally. The contribution to the plexus is postfixed when the cervical nerve root contribution is shifted inferiorly by one level, with C5 making a minimal contribution and there being a large contribution from T2 [60, 61].

Free-run EMG

Pathophysiology of proximal spinal nerve root

Free-run EMG recordings are used to detect motor nerve root mechanical activation. Spinal nerve roots are more susceptible to injury than are peripheral nerves [62]. Spinal nerve roots are susceptible to injury by two mechanisms. The first is that proximally the dorsal and ventral roots split into rootlets and minirootlets [63]. The area where this split occurs is the central-peripheral transitional region, which is the point where the nerve root is more susceptible to mechanical injury. The axons at this point are enclosed by a thin root sheath, cerebrospinal fluid and meninges, and lack the protective covering of epineurium and perineurium that is present in peripheral nerve [64]. The second mechanism of injury is that there is an area of hypovascularity at the junction of the proximal and middle one-third of the dorsal and ventral roots. This is the point of anastomosis between the central vasa corona and peripheral segmental vessels. At this point, the nerve roots are more susceptible to mechanical injury [63].

The mechanosensitivity of normal and abnormal dorsal root ganglia and axons has been defined in human and animal models [62]. In a cat model, rapidly-applied mechanical forces induce short-duration (200 ms) impulses in normal nerve root. Static mechanical forces do not induce impulses in normal nerve root. Rapidly- and statically-applied mechanical forces induce long periods (15–30 s) of repetitive impulses in irritated or in regenerating nerves in-continuity. Minimal acute compression of normal dorsal root ganglion induces prolonged (5–25 min) repetitive firing of nerve [65]. When interpreting intraoperative motor nerve root studies, it is important to understand the pathophysiological mechanisms of nerve root injury and to understand the response of normal and pathological nerve to not only different types of mechanical force, but also to electrical stimulation.

Technique

Subdermal EEG needle electrodes are most often used for recording. Monopolar EMG needle electrodes and longer, uncoated stainless steel needle electrodes may also be used. Fine teflon-coated silver wires with the wire exposed at the end that are inserted with a spinal tap needle may also be

used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point of the muscle, and the reference electrode is inserted over tendon or bone. The needles are secured with tape. Reference and active electrodes may be placed over the same muscle. The result is increased specificity.

A range of different high- and low-frequency filters are used. A high-frequency filter of 10–30 KHz and a low-frequency filter of 2–30 Hz are most often used. A low-frequency filter greater than 50 Hz, and a high-frequency filter less than 3 KHz should be avoided [66]. The timebase is from 200 ms to 5 s depending on the desired resolution for the lower and upper extremities. The recordings are made continuously. An adequate number of muscles are recorded from, to represent all myotomal levels of interest. Baseline recordings are made before the start of the surgical procedure to determine any pre-existing nerve root irritation. Recordings are then made continuously throughout the surgical procedure.

A normal free-run EMG response is the absence of activity. The firing pattern consistent with pre-existing nerve root irritation consists of spontaneous activity characterized by low-amplitude, periodically-occurring activity that resembles positive waves and fibrillation potentials. Fasciculation potentials may also be recorded, and may be normal or may be secondary to nerve root irritation. Pathologically-significant mechanically-elicited activity can be characterized as: (1) phasic or a burst pattern consisting of a single potential or non-repetitive asynchronous potentials which are often complex and polyphasic in configuration; and (2) tonic or train activity consisting of periods of prolonged multiple or repetitive synchronously-grouped motor unit discharges that last up to several minutes. Burst potentials rarely represent neural insult, while repetitive discharges are associated with a more serious pathology and may represent neural injury. Burst potentials are associated with direct nerve trauma such as tugging and displacement, free irrigation, electrocautery, and application of soaked pledgets. Train activity is commonly related to sustained traction and compression. When these patterns occur, the surgeon should be notified so that corrective measures can be instituted. The presence of an abnormal free-run EMG signal provides after-the-fact information regarding an acute neurological event. Hopefully, this has been caused by a reversible injury, such as stretching of the nerve root by a retractor, or minor nerve root compression [67]. When pronounced EMG activity that resembles motor unit activity occurs in all muscle groups recorded for a prolonged period of time, the possibility of the anesthesia becoming 'light' and the patient voluntarily contracting the muscles should be considered.

Electrical interference prevents the recording of free-run EMG during the use of cauterization. Nerve root irrita-tion or damage may go undetected during this period of time. During this period of electrical interference, muscle activation may be detected by the use of piezoresistive accelerometers which detect the motion that is associated with muscle activation. The accelerometer recordings are not affected by the electrical interference and are not nerve-root-specific, for they will detect any motion. The accelerometers are taped on the surface of the skin over the desired muscle using the same recording parameters as that used for EMG recordings [68].

Free-run EMG has been used intraoperatively to detect motor nerve root compromise during placement of pedicle screws, decompression for spinal stenosis, correction for scoliosis deformity, radiculopathy secondary to disc herniation and/or spondylosis, and during removal of tumors involving the nerve roots [69–75]. Trapezius muscle free-run EMG may be used to detect compromise of the spinal portion of the accessory nerve in the cervical spinal canal, and may be used to detect adequacy of decompression of the accessory nerve in the cervical spinal canal [74].

Electrically stimulated

Pedicle screw stimulation

Background. Pedicular fixation of the lumbar spine has become an accepted technique in spinal fusion surgery. A significant advantage to using the pedicle is the rigidity it provides for fixation of the vertebral motion segment. However, there is considerable potential morbidity associated with incorrect placement of the screws into the vertebral pedicles. Neural structures are close to the pedicle and incorrect placement of the screws can lead to postoperative neurological deficits or radicular pain. Neurological deficits caused by inaccurate pedicle screw placement have been documented in clinical studies [76–78].

Insertion of screws into the pedicles is essentially a "blind" technique with radiographic assistance. Most authors recommend the use of intraoperative radiographs or fluoroscopy to aid in correct screw placement [79–83]. However, several studies have demonstrated that radiographs are unreliable in determining accurate screw position [77, 79, 83]. Various techniques to verify correct placement have been proposed, including radiographs and direct inspection of the pedicle. While radiographs are valuable in verifying correct screw placement, an unacceptable false-negative rate of 14.5% using radiography for incorrectly-placed screws was found [77]. Direct inspection of the pedicle offers the best means for verification of screw placement, but results in additional cost and morbidity to the patient.

Incorrect placement of the pedicle screw can compromise the stability of the fixation or worse, produce a neurological deficit or radiculopathy. Two of 7 neurological deficits were believed to be related to misplaced screws in a series of 124 patients who underwent posterior spinal fusion with variable screw plate fixation [78]. In one report, postoperative neurological complications occurred in 11% of 57 patients undergoing pedicle screw plate fixation of the lumbar spine, with most being attributed to direct nerve root impingement by the screw [76]. In cadavers, incorrect screw placement occurred in 21% of cases, and 92% of these failures represented cortical perforations into the canal [77].

Intraoperative SSEPs have been used to monitor lumbosacral nerve root function as a means of preventing neural deficits, determining adequacy of decompression and as a means of predicting postoperative outcome. Studies have been performed with peripheral mixed-nerve stimulation [84, 85]. Because multiple nerve roots contribute to the cortical SSEP it is possible to damage one nerve root without a significant change in the cortical potentials. Dermatomal SSEPs have been used to improve the sensitivity and specificity for detecting single nerve root dysfunction [86–90] for a variety of surgical procedures. Dermatomal SSEPs have also been used to monitor single nerve root function during intrapedicular fixation of the lumbosacral spine [90]. Unfortunately, it is possible for a pedicle screw to breach the cortex and lie next to a nerve root without compressing it. Such a condition may not be detected with dermatomal monitoring, but could conceivably lead to nerve root irritation and postoperative radiculopathy. To improve the sensitivity and specificity for detecting potential pedicle screw compromise of nerve root function, intraoperative electrical CMAP evoked techniques have been developed [31, 69, 91–95]. Also free-run mechanically-elicited EMG techniques are used.

Technique: Recording. Needle electrodes are most often used for recording subdermal EEG. Monopolar EMG needle electrodes, and longer uncoated stainless steel needle electrodes may also be used. Fine Teflon-coated silver wires with the wire exposed at the end, which are inserted with a spinal tap needle, may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point of the muscle and the reference electrode is inserted over tendon or bone. The needles are secured with tape. EMG should be acquired from those myotomes whose nerve roots innervate above and below each pedicle to be tested. For lumbosacral (L2–S2) nerve root monitoring, the vastus medialis, tibialis anterior, peroneus longus and medial head of the gastrocnemius muscles can be used. A range of different high- and low-frequency filters are used. A high-frequency filter of 10–30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [66]. Recordings are single-sweep.

Stimulation. Constant current or constant voltage may be used for stimulation. With constant current the duration is 0.2 ms and the rate is 1–3 Hz. If partial neuromuscular blockade is used, a stimulation rate faster than 1 Hz may result in decay of the CMAP from one stimulus to the next. Therefore, a faster rate of stimulation should be avoided when using partial neuromuscular blockade. The cathode is attached to the screw or drill-bit with a gas-sterilized alligator clip, and the anode is a larger dispersive electrode secured over the posterior shoulder area. This results in monopolar stimulation without paraspinal muscle contraction. The constant current EMG threshold for drill-bits that do not breach the pedicle cortex averages 30.4 mA (range 8.5–53.0 mA, SD 13.9 mA). For screws the average is 24.0 mA (range 9.0–60.0 mA, SD 11.9 mA). The constant voltage evoked EMG threshold for drill-bits that do not breach the cortex averages 65.9 V (range 15.0–100.0 V, SD 26.4 V). For screws the average is 52.5 V (range 22.0–110.0 V, SD 49.4 V). Constant voltage is more variable than constant current; therefore constant current stimulation should be used for drill-bit and screw stimulation. A constant current threshold of at or less than 6.0 mA for screw and drill-bit stimulation is considered to be abnormal and a sign that a drill-bit or screw has breached the cortex, is adjacent to neuronal tissue, and potentially can cause damage. The current is gradually increased until threshold is reached [31]. Others have used constant current thresholds of less than 10 mA [89, 95], less than 4 mA [96] and less than 11 mA [85] as indicating abnormal thresholds. When screws are electrically evaluated during placement in patients with previous surgery where scar tissue is present, the threshold for both drill-bit and screw stimulation may be raised [31]. Stimulating screws that are immersed in irrigation fluid may cause current shunting and raise the threshold, resulting in false-negative findings [97]. If the nerve root can be visualized in the surgical field, a control technique involves comparing the pedicle screw threshold to direct nerve root threshold.

Correlation. Evoked EMG is 93% sensitive in identifying misplaced hardware [31]. The sensitivity of radiography is only 63% [31]. In 36 patients, electrically-evoked EMG demonstrated malpositioned hardware in 13 of 239 instances of hardware insertion (5.4%) [31]. In 5 of the 13 instances of electrically-identified malpositioned hardware, radiography was normal and incorrect placement was confirmed by direct inspection of the pedicle wall [31]. Ten screws were repositioned. Three screws had a defect in the

pedicle wall with the threads of the screws not in contact with the nerve root. These screws were left in place, and in these as well as any of the other patients, no postoperative neurological deficit was present [31]. A threshold of less than 6.0 mA indicates that the screw is in direct contact with the nerve root [31]. An L4 drill-bit significantly breached the cortex superiorly, with its position being far removed from the adjacent nerve root, and the threshold was normal [31]. Therefore, it is possible for a screw to breach the cortex and still have a normal electrical threshold if the screw is not touching the nerve root. Transient free-run mechanically-elicited EMG that was related to a surgical event was present in 10 of 29 patients [31]. Lower-extremity dermatomal SSEPs accurately identified the correct level or levels of involvement in 20 of 29 patients, but there were no intraoperative changes in these signals as well as in the posterior tibial SSEPs [31]. Eleven percent of 102 screw placements involved sites where a defect was missed by palpation alone, but was identified by electrical stimulation combined with palpation and visualization [39]. The efficacy of pedicle screw stimulation to avoid nerve root damage during screw placement was evaluated in 662 patients, in whom 3,409 pedicle screws were tested by electrical stimulation [98]. A threshold of less than 10 mA was considered to be abnormal, and 133 (3.9%) screws in 102 (15.4%) patients had a threshold less than 10 mA [98]. Of the 133 screws, 82 were left in place, 18 were removed and redirected, and 33 were removed and not replaced. All 21 of the screws with a threshold less than 5 mA were removed. Nerve root irritation during decompression was detected with free-run EMG recordings in 25 patients [98]. There were no new postoperative neurological deficits [98].

Other pedicle evaluation techniques. In one study a gas–sterilized alligator clip was attached to a ball-tip probe which acted as the cathode and the anode was a self-adhesive bovie pad placed on the skin between the scapulas [92]. Monophasic constant current of 0.2 ms with a rate of 3 Hz was used [92]. The probe was insulated with Teflon except for the tip. The probe was advanced into the pedicle hole and a searching current of 7.0 mA was used [92]. If a perforation was present in the wall and was adjacent to motor axons, the 7.0 mA would be threshold for these axons and evoked EMG would be recorded [92].

Electrical impedance testing as an alternative approach to electrical screw stimulation has been proposed [99]. The average vertebral impedance was 400 ohms (range 244–556 ohms) at the insertion and decreased to 100 ohms (range 78–122 ohms) at maximum probe penetration. Vertebral impedance less than 58 ohms indicated a 100% likelihood of cortical perforation [99]. Impedance measurements, electrically-elicited EMG and mechanically-elicited

free-run EMG were recorded in 20 patients undergoing surgery for spinal degeneration [100]. Electrically-elicited EMG was more sensitive to detecting wall breakthrough than were impedance measurements [100]. Mechanically-elicited EMG was present in only one pedicle. The recommended method for monitoring pedicle screw insertion was combined electrically- and mechanically-elicited EMG [100].

The use of electrically-elicited EMG for thoracic screw placement has been reported in a sheep model [101]. A threshold of 10 volts or less was found to have a sensitivity of 94% and specificity of 90% [101]. In humans, 87 thoracic screws (T1–T12) were stimulated in 22 patients [102]. Electrically-elicited EMG was recorded from the flexor carpi ulnaris, intercostals and abdominal muscles. Thresholds greater than 11 mA represented correct placement in 97.5% of the screws [102]. Thresholds less than 11 mA were associated with an increased rate of vertebral penetration [102]. Five screws (5.7%) showed penetration on postoperative CT scans [102]. Six screws (6.9%) had stimulation thresholds at or less than 11 mA, 3 of which showed cortical breakthrough [102]. 81 screws had thresholds higher than 11 mA [102]. 79 (97.5%) of these were within the vertebra [102]. There were no postoperative complications [102].

Nerve root stimulation

Introduction. Intraoperative motor nerve root stimulation can be used to map the cauda equina to help identify motor axons versus non-neural tissue (tumor), to differentiate between motor and sensory roots, and to determine what vertebral or segmental level a motor root is. When scar tissue is present from previous surgical procedures, electrical stimulation can be used to help identify where the motor axons lie within the scar tissue. With plexus tumors or traumatic lesions, nerve root stimulation can help to determine which neural structures have continuity peripherally.

Technique. The threshold for lumbar and sacral nerve roots was determined with the use of a flush-tip stimulator (0.5 mm tip diameter) as the cathode, and the constant current threshold for nerve root stimulation averaged 2.2 mA (range 0.2–5.7 mA) [31]. The duration was 0.2 ms and the rate was 1.0 Hz [31]. The anode was a larger dispersive electrode secured over the posterior shoulder area [31].

The threshold for lumbar and sacral nerve roots was determined with a ball-tip probe that was insulated by Teflon except for the tip which served as the cathode, and the constant current threshold for nerve root stimulation averaged 2.1 mA (range 1.2–3.8 mA) [39]. The anode was a self-adhesive bovie pad placed on the skin between the scapulae. The duration was 0.2 ms and the rate was 3 Hz [39].

The motor nerves were identified during cauda equina surgery during 25 operations [73]. The operations performed were release of tethered spinal cord, removal of sacral lipomas, teratomas, myxopapillary ependymomas, astrocytomas and neurofibromas [73]. Constant current of 1–3 mA was connected to a standard bipolar cautery forceps for bipolar stimulation [73]. The duration was 0.2 ms and the rate was 2 Hz [73].

Iliosacral screw stimulation for sacroiliac dislocation

Iliosacral screw fixation is used for operative stabilization of disruption of the iliosacral joint. Whether screws are inserted percutaneously with closed reduction or after open reduction, the optimum intraosseous screw course is close to the fifth lumbar and sacral nerve roots as well as the spinal canal [103, 104]. One of the potential hazards of this technique is misdirection of the guide-wire, drill-bit or screw that results in injury of the nerve root or compromise of the spinal canal. The incidence of iatrogenic neural injury with this technique has been reported to be from 1 to 10% [105, 106].

An electrically evoked drill-bit, screw technique with recording of CMAPs has been developed to detect when these devices approximate neuronal tissue [107]. A threshold less than 8.0 mA for drill-bit and less than 6.5 mA for screw stimulation indicates that these devices approximate neuronal tissue and should be redirected. Constant current monopolar, monophasic square-wave stimuli of 0.2 ms duration at 3 Hz was used [107]. The cathode was attached to the drill-bit or screw with a sterile alligator clip and the anode was a subdermal EEG needle electrode inserted subcutaneously. Neuromuscular blockade was minimal and 3 or 4 of the TOF responses were present. Spontaneous EMG activity was also recorded. EMG was recorded from the tibialis anterior and gastrocnemius muscles. In addition to these muscles, EMG activity may also be recorded from the vastus medialis and peroneus longus muscles, for CMAPs may be present in these muscles when they are not evident in the others. The drill-bits and screws were periodically stimulated while being inserted. Four of 51 screws were redirected because evoked EMG indicated approximation to the nerve roots [107]. Therefore, 4 of 27 patients were potentially prevented from developing neural damage using this technique [107]. No free-run EMG changes were present [107].

ELECTROPHYSIOLOGY OF MONOSYNAPTIC AND POLYSYNAPTIC REFLEXES AND F-RESPONSES

One approach to understanding complex spinal cord function is to consider the integrating function to be controlled by a system of tightly electrically coupled central pattern generators (CPGs). The integrated activity of these spinal cord CPGs is responsible for controlling the stepping mechanism of gait and the coordination of upper- and lower-extremity function [108–110]. Reflex recordings provide information about the degree of coupling between CPGs. Acute and chronic damage to the system produces uncoupling of CPGs resulting in a change in the reflex gain; because of the change in the reflex gain, this uncoupling of CPGs can be detected by changes in reflex recordings. Spinal cord CPGs may be thought of as having four components. These components are the segmental interneurons, the descending suprasegmental systems, the short, intermediate and long propriospinal systems and the peripheral afferent input. Intraoperative reflex recordings provide information about the interaction between these components of this system. The point of control that determines the reflex gain of the system is the segmental interneurons. Sensory afferent signals following peripheral nerve stimulation provide the time-locked synchronization of the system. Summated activity in descending spinal cord systems, especially the corticospinal, rubrospinal, vestibulospinal and reticulospinal systems, contribute to controlling the gain set by the interneurons. Short, intermediate and long propriospinal systems control processing at multiple spinal cord levels ipsilaterally and contralaterally. The output from the system is through the anterior horn cells, and is measured by reflex recordings from muscle. A disturbance of the peripheral afferent input, the descending suprasegmental systems, the propriospinal systems or the segmental interneurons results in the uncoupling of these components and a change in the reflex gain that can be detected by changes in reflex processing and F-responses.

Reflex processing can be considered relatively simple, for example, the monosynaptic or oligosynaptic stretch reflex which involves processing at a single segment of the spinal cord. However, it also involves complex polysynaptic reflexes which involve processing at multiple spinal cord levels. Monosynaptic reflex muscle recordings are of short latency, short duration, simple configuration and high amplitude. These parameters are stable and vary little from one stimulus to the next. Polysynaptic recordings are of longer latency, longer duration, complex configuration and low amplitude. Polysynaptic recordings are not stable and vary from one stimulus to the next [111].

MONOSYNAPTIC H-REFLEX

Background

In normal adults the monosynaptic or oligosynaptic H-reflexes are regularly found in the gastrocnemius muscle

and the flexor carpi radialis muscle. They are also frequently found in the quadriceps and plantar foot muscles. The gastrocnemius and flexor carpi radialis H-reflexes are most commonly used intraoperatively. Afferent conduction is through large 1a low-threshold fast-conducting fibers and efferent conduction is through the alpha motor neurons. The H-reflex is recorded following long-duration (1 ms), low-intensity stimulation which activates low-threshold, fast-conducting 1a afferent fibers. H-reflex amplitudes are maximal at submaximal stimulation, and are inhibited as the stimulus is increased. In addition to the H-reflex, the direct muscle response (M-wave) can be recorded. H-reflex stimulus intensity is submaximal for the M-wave. To confirm the response is an H-reflex the amplitude should exceed the M-wave amplitude, and the configuration and latency should be the same from one stimulus to the next [112]. At times H-reflex amplitude may be less than the M-wave amplitude. As the intensity of stimulation is increased a progressively larger percentage of the motor neuron pool is activated, resulting in a progressive increase in the H-reflex amplitude. When the M-wave amplitude is maximal further increases in the stimulus intensity result in the inhibition of the H-reflex, and the F-response becomes present [112]. When recording the gastrocnemius H-reflex in the awake human, the percentage of the motor neuron pool activated averaged 50% (range: 24.0–100%) [65]. Homonymous monosynaptic H-reflexes are H-reflexes that are recorded from muscles that are innervated by the same nerve root as the 1a-activated sensory fibers. The gastrocnemius H-reflex is an example of a homonymous monosynaptic H-reflex. 1a sensory action potentials may also make monosynaptic connections with motor neurons at spinal cord levels other than the 1a sensory segmental level. As a result of this activation, H-reflexes may be recorded from muscles having segmental innervation other than the 1a segmental afferent activation. These H-reflexes are called heteronymous H-reflexes [113]. The gain of these monosynaptic reflexes is influenced by the level of presynaptic inhibition of the segmental interneurons. In CNS lesions with upper motor neuron signs, presynaptic inhibition is decreased and heteronymous H-reflexes may become present in muscles where they are not usually elicited. This provides evidence for a disordered central motor system state [114–116].

Gastrocnemius H-reflex

In the lower extremity the H-reflex can be recorded from the gastrocnemius and soleus muscle following electrical stimulation of the posterior tibial nerve in the popliteal fossa [117]. This reflex is mediated by segmental S1 afferent and efferent activity [114]. The gastrocnemius H-reflex latency varies with leg length, but is usually less than 35 ms [111]. Intraoperatively onset latencies may be greater due to decreased limb temperature. Gastrocnemius H-reflex parameters that have been monitored are H-reflex amplitude, latency and the H:M ratio. Right–left amplitude and latency differences are also used.

Gastrocnemius H-reflex technique

For recording, subdermal EEG needle electrodes are inserted in the medial head of both gastrocnemius muscles. The H-reflex may also be recorded in the calf muscle from the soleus muscle. The technique for recording the H-reflex from the soleus muscle is the same as that for recording from the gastrocnemius muscle, except that the recording electrodes are placed over the mid-dorsal line of the leg with the active electrode 4 cm above the point where the 2 heads of the gastrocnemius muscle join the Achilles tendon. The reference electrode is placed 3 cm distal to the active electrode [117]. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used.

Fine Teflon coated silver wires with the wire exposed at the end, which are inserted with a spinal tap needle may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point and the reference electrode is inserted over tendon or bone. Recording electrodes may be placed in a bipolar derivation with the active and reference electrodes in the muscle. The needles are secured with tape. A range of different high- and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [66]. The time base is 100 ms. Recordings are single-sweep. Stimulation is with needle or surface electrodes. The anode is placed in the medial distal area of the popliteal fossa and the cathode approximately 4 to 5 cm lateral and proximal. The stimulation rate is 0.5 Hz and the stimulus duration is 1 s. The stimulus intensity is adjusted so that the H-reflex amplitude is maximal. The most effective stimulus intensity is chosen such that any increase or decrease in stimulus intensity results in a decrease in the H-reflex amplitude. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure. Any variability in latency and amplitude should be noted in the baseline recordings.

In addition to monitoring peripheral nerve, segmental spinal nerve root and spinal cord function, these reflexes can be used to monitor the function of a variety of

suprasegmental descending spinal cord systems that control the S1 segmental interneurons [118].

Flexor carpi radialis H-reflex

In the upper extremity the flexor carpi radialis H-reflex can be recorded following electrical stimulation of the median nerve in the axilla or at the elbow. This reflex is mediated by segmental C6/C7 afferent and efferent activity. The latency of the flexor carpi radialis H-reflex varies with the arm length, but is usually less than 20 msec [119]. Flexor carpi radialis H-reflex parameters that have been monitored are H-reflex amplitude, latency and the H:M ratio. Right–left latency and amplitude differences are also used.

Flexor carpi radialis H-reflex technique

For recording, subdermal EEG needle electrodes are inserted in the flexor carpi radialis muscles. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine Teflon coated silver wires with the wire exposed at the end, which are inserted with a spinal tap needle may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point and the reference electrode is inserted distally over tendon or bone. Recording electrodes may be placed in a bipolar derivation with the active and reference electrodes in the muscle. The needles are secured with tape. A range of different high- and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [66]. The time base is 50 ms. Recordings are single-sweep. Stimulation is with needle electrodes spaced 2 cm apart unilaterally medially in the axilla at 0.5 Hz and of 1 s duration. The cathode is proximal. The stimulus intensity is adjusted so that the H-reflex amplitude is maximal. The most effective stimulus intensity is chosen such that any increase or decrease in stimulus intensity results in a decrease in the H-reflex amplitude. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure. Any variability in latency and amplitude should be noted in the baseline recordings.

In addition to monitoring peripheral nerve, segmental spinal nerve root and spinal cord function, these reflexes can be used to monitor the function of a variety of suprasegmental descending spinal cord systems that control the C6/C7 segmental interneurons.

F-RESPONSE

Neurophysiological basis

Upper- and lower-extremity F-responses have been used clinically to detect abnormalities of the lower motor neuron and to detect a disordered central motor system state [120]. The F-response is not a reflex, but following stimulation of a peripheral nerve, represents antidromic motor conduction that activates approximately 1–5% of the alpha motor neurons in the anterior horn. This results in orthodromic conduction to the muscle. F-responses are usually composed of no more than several motor units. They are variable in latency and configuration and low in amplitude, being from 1 to 5% of the maximum M-wave amplitude. F-responses can be recorded from any muscle. They can be recorded at submaximal stimulation, but are most prominent with supramaximal stimulation. Both afferent and efferent components of the F-response follow the same alpha motor neurons. F-responses and H-reflexes have similar latencies and are at times confused. The F-response varies in amplitude, latency and configuration from one stimulus to the next, while the H-reflex does not. The H-reflex is usually inhibited at higher intensities of stimulation, while the F-response is not. There is direct evidence for selective discharge of the larger motor neurons in F-responses. Lesser inhibition of larger motor neurons by inhibitory Renshaw cell interneurons provides a physiological model for such selective discharge. Because of these F-response properties, F-response recordings may not represent the function of the entire motor neuron pool [121]. Supra F-response stimulation does not inhibit the F-response but activates higher-threshold slower-conducting group II, III and IV fibers. Because of disinhibition of alpha motor neurons F-responses are more prominent in chronic upper motor neuron lesions. They are also of higher amplitude when lower motor neuron re-innervation occurs [112, 122–125].

The A-wave is a late response that is present with constant latency and amplitude. Low intensity stimulation elicits the A-wave and it is usually blocked by higher intensity of stimulation. A-wave latency is between the CMAP and the F-response latency, or it exceeds the F-response latency. It may also appear with a latency between the M- wave and H-reflex latency, or the latency may exceed the H-reflex latency. A-wave amplitude is less than the H-reflex amplitude. The A-wave should not be confused with the H-reflex or F-response. The A-wave is generated by peripheral nerve changes rather than changes in central nervous system signal processing. The physiology of the A-wave is that there is peripheral neural damage with the presence of a collateral sprout from a proximal point of damage to the

muscle. When the antidromic impulse reaches the point of damage, a portion of the electrical impulse proceeds distally along the collateral sprout and a small portion of the muscle is activated. Depending upon the nerve stimulated, A-waves may be normal or abnormal [126].

Normal values

Intraoperatively F-responses are usually recorded from the abductor digiti minimi muscle following stimulation of the ulnar nerve at the wrist, the abductor pollicis brevis muscle following stimulation of the median nerve at the wrist, the abductor hallucis muscle following stimulation of the tibial nerve at the ankle, and from the extensor digitorum brevis muscle following stimulation of the deep peroneal nerve at the ankle. The F-response latency in the upper extremity varies with arm length, but is usually less than 31 ms when recorded from the abductor pollicis brevis muscle and less than 32 ms when recorded from the abductor digiti minimi muscle. The F-response latency in the lower extremity varies with height, but is usually less than 58 ms when recorded from the abductor hallucis and extensor digitorum brevis muscles [112].

F-response technique

For recording, subdermal EEG needle electrodes are inserted into the muscles. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine Teflon coated silver wires with the wire exposed at the end, which are inserted with a spinal tap needle may also be used for recordings when the subcutaneous tissue is thick. The active electrode is inserted at the motor point and the reference electrode is inserted distally over tendon or bone. The needles are secured with tape. A range of different high- and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [66]. The time-base in the upper extremity is 50 ms, and is 100 ms in the lower extremity. Recordings are single-sweep. Stimulation is with surface or needle electrodes spaced 2 cm apart over the nerve being monitored, at 1.0 Hz and of 0.2 ms duration. Stimulus intensity is at a supramaximal level for the presence of the F-response. F-response parameters that have been monitored are minimum, maximum and mean latency, amplitude and persistence. Persistence is the percentage of F-responses present with successive stimuli. Clinically a persistence less than 50% is considered to be abnormal. Right–left differences are also monitored [29, 30, 127]. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure. The degree of variability of F-response parameters should be noted in baseline recordings.

Intraoperative use of F-responses

F-responses have been recorded intraoperatively to monitor peripheral motor nerve function during total hip surgery [123] and during removal of tumors of the nerve roots proximally. Lower-extremity F-responses have been used to detect suprasegmental injury of the cervical spinal cord [29].

INTRAOPERATIVE H-REFLEX AND F-RESPONSE ABNORMALITIES

Severe acute spinal cord injury that leads to spinal shock results in suppression of H-reflexes and F-responses [128]. This suppression is produced by hyperpolarization of caudal motor neurons that animal studies have demonstrated occurs within seconds after spinal cord injury [129, 130]. Partial acute spinal cord injury that does not result in spinal shock leads to hyperpolarization of caudal motor neurons resulting in absent F-responses or decreased F-response persistence, and H-reflex amplitude may be decreased. Absent F-responses or decreased F-response persistence may continue for up to two weeks following acute spinal cord injury. H-reflex changes associated with acute spinal cord injury will return to normal in several days after injury [128]. Transient intraoperative F-response and H-reflex amplitude reductions less than 50% and transient changes in F-response persistence have not resulted in a postoperative neurological deficit. When amplitude suppression exceeds 90% and persists throughout surgery, the patients have had a profound postoperative neurological deficit [28].

In dogs acute spinal cord injury initially caused inhibition of the gray matter activity caudal to the site of injury. Within minutes this was followed by disinhibition with increased activity of caudal gray matter function. Parallel changes occurred in peripheral sciatic nerve reflex recordings following spinal cord injury. These peripheral reflex changes reflected the level of spinal cord gray matter function. These peripheral reflex recordings demonstrated that peripheral reflexes can be used to detect acute spinal cord injury [131].

With acute spinal cord injury H-reflexes and F-responses may be better predictors of postoperative motor function than are transcranial electrical MEPs. A patient with acute intraoperative T8 spinal cord injury had lower-extremity weakness postoperatively. The MEPs became absent during surgery. Lower-extremity H-reflex amplitudes were

decreased from 47–97%. F-response persistence was decreased to 65 and 70%, compared to 100% before spinal cord injury. The preservation of some H-reflex and F-response activity was a better predictor of postoperative motor function than were MEPs [132].

In 278 pediatric spine surgeries gastrocnemius H-reflex and SSEP monitoring were used for monitoring spinal cord function. Combined H-reflex and SSEP monitoring improved reliability compared to either procedure alone. H-reflexes exhibited more changes than SSEPs. These changes reflected changes in spinal cord gray matter function related to acidosis and changes in hematocrit and blood pressure [133].

Clinically, F-responses have been used to study segmental spinal cord function where intramedullar motor neurons or anterior nerve roots are damaged. The excitability of F-responses is influenced by spinal shock in acute spinal cord injury. Fifty percent of acute cervical spinal cord injury patients had a complete loss of median and ulnar F-responses during spinal shock (mean 2.6 weeks post-trauma). After 6 months all patients regained F-responses. F-response persistence correlated with the severity of the motor neuron lesion, while the mean latency did not differ from healthy subjects [134].

POLYSYNAPTIC REFLEXES

Sacral reflex

Three polysynaptic sacral reflexes (bulbocavernosus: pudendal-bulbocavernosus, vesicourethral: pudendal-external urethral sphincter and vesicoanal: pudendal-external anal sphincter) can be recorded [125]. The vesicoanal reflex can be easily recorded intraoperatively. The vesicoanal sacral reflex which is recorded from the external anal sphincter muscle following electrical stimulation of the pudendal nerves can be used to monitor S2, S3 and S4 segmental afferent and efferent activity and interneuronal activity [43]. The sacral reflex can be activated by unilateral stimulation of the pudendal nerves [135]. There is some variability of the sacral nerve roots involved in the afferent component of this reflex. Most pudendal afferent activity is carried bilaterally in the S2 (60.5%) and S3 (35.5%) roots, although a single (18.0%) sacral root or single unilateral root (7.6%) may be responsible for most pudendal afferent activity [136, 137]. The efferent component of this reflex is through the pudendal nerve supply of the external anal sphincter muscle. The majority of pudendal efferent activity is derived mainly from the second sacral nerve root [138]. These afferent and efferent anatomical variations need to be taken into consideration when interpreting intraoperative sacral reflex recordings.

Normal values

The sacral reflex in awake nonanesthetized normal subjects has two components: an early component having a latency of 30 msec and a longer latency component having a latency of 50 msec. The early component is thought to be oligosynaptic since it does not habituate. The longer latency component is obtainable with stronger electrical stimulation. Intraoperatively it is possible to usually record both components [43, 135].

Sacral reflex technique

Two channels of single-sweep sacral reflex activity are recorded simultaneously bilaterally from the external anal sphincter muscles. Bipolar recordings are made, with two subdermal EEG needle electrodes inserted in each side of the external anal sphincter muscle. A range of different high- and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [66]. In the male, the pudendal nerve is stimulated unilaterally using subdermal EEG needle electrodes with the cathode at the base of the penis and the anode over the lateral aspect of the penis. In the female, the cathode on each side is just lateral to the clitoris and the anode just posterior between the major and minor labial folds. Monophasic constant current of 10 to 20 mA, 0.1 msec duration, from 0.2 to 2.0 Hz is used. The reflex can be facilitated with paired stimulation (3 ms ISI, 0.1 ms duration). Mean baseline latency, duration and amplitude parameters are noted for unilateral stimulation. Baseline recordings are made with the patient anesthetized before the start of the surgical procedure.

Intraoperative application of sacral reflexes

Sacral reflexes can be used for real-time monitoring for surgeries such as reduction of spinal fractures [118], removal of tumors, release of tethered spinal cord, etc. where the conus medullaris or sacral nerve roots in the cauda equina are at risk of injury.

The sacral reflex should be able to be used to monitor the function of a variety of suprasegmental descending spinal cord systems that control the S2, S3 and S4 segmental interneurons, although this has not yet been reported.

Intralimb and interlimb lower extremity reflexes

Background

Intralimb (ipsilateral) and interlimb (contralateral) lower-extremity reflexes [26, 27] are abnormal reflexes that are recorded simultaneously from 4 muscle groups in each lower extremity following unilateral simultaneous stimulation of the tibial and common peroneal nerves in the popliteal fossa [26, 27]. This technique not only monitors segmental afferent and efferent activity at the L4, L5 and S1, S2 levels and L4, L5 and S1, S2 interneuronal function, but also monitors complex spinal cord polysynaptic processing which involves multiple spinal cord levels. These reflexes can be recorded from not only ipsilateral (intralimb) and contralateral (interlimb) lower-extremity muscles, but the distribution of the reflex activity effects proximal and distal muscles differently. The explanation for this proximal–distal distribution is that since vestibulospinal and reticulospinal pathways control proximal lower-extremity muscles [139], proximal lower-extremity changes may represent a suprasegmental compromise of these descending vestibulospinal and reticulospinal pathways. Rubrospinal and corticospinal pathways control distal lower-extremity function [139], and therefore distal lower-extremity reflex changes represent a suprasegmental compromise of these descending rubrospinal and corticospinal pathways.

Intralimb and interlimb reflexes with spinal cord pathology

Abnormal intralimb and interlimb polysynaptic reflexes are present in the intraoperative baseline recordings because a pre-existing neurological deficit exists. Chronic spinal cord compromise uncouples the spinal cord CPGs due to disinhibition and polysynaptic reflexes will be present during the baseline intraoperative recordings. Baseline intraoperative lower-extremity intralimb and interlimb reflex activity was present in patients with idiopathic scoliosis. The presence of this reflex activity demonstrates the presence of abnormal reflex processing in these patients. These reflexes are present in idiopathic scoliosis patients either because of congenital changes in spinal cord signal processing or because of the effect of the spinal curvature on the spinal cord. These reflexes are not present in intraoperative baseline recordings in neurologically normal patients in which the spinal cord CPGs have not been uncoupled [30]. With intraoperative acute spinal cord compromise, previously absent reflexes may become present with the abnormal uncoupling of the spinal cord CPGs. During the monitoring of cervical spine surgery patients with cervical myelopathy and lower-extremity symptoms, intraoperative baseline lower-extremity intralimb and interlimb reflex

activity is present, while those without myelopathy do not have lower-extremity polysynaptic activity present. Both groups may have transient intraoperative lower-extremity intralimb and interlimb reflex changes present with manipulation of the cervical spine without changes in SSEP components [26]. Intraoperatively these reflexes indicate acute spinal cord compromise when there is a change in the pre-existing reflex pattern or the presence of reflex activity that was previously absent. High intensity of stimulation is needed to activate these reflexes with acute or chronic spinal cord involvement, which is consistent with activation of the flexor afferent reflex system.

Intralimb and interlimb reflexes with nerve root damage

These reflexes are present in intraoperative baseline recordings when chronic nerve root damage is present. Chronic nerve root damage results in a decreased threshold for activation of intralimb and interlimb reflexes with a lower intensity of stimulation. Chronic nerve root damage changes the pattern of afferent activity and uncouples the spinal cord CPGs, resulting in the presence of baseline intraoperative intralimb and interlimb reflex activity.

Clinical correlation

These reflex changes occur secondary to acute or chronic spinal cord compromise and are due to a combination of a disruption of the suprasegmental descending influence over segmental interneurons mediating presynaptic inhibition, which results in disinhibition of segmental interneurons [40]; and hyperpolarization of caudal motor neurons [29, 130]. Transient asynchronous polysynaptic reflex changes have not been associated with a postoperative neurological deficit, while persistent high-amplitude synchronous reflex changes have been associated with a postoperative lower-extremity deficit [27].

In chronic cervical spinal cord injured patients, changes in polysynaptic reflex processing between the upper and lower extremities have been recorded in a clinical setting [119]. This upper- and lower-extremity uncoupling has not been reported intraoperatively.

Intralimb and interlimb lower-extremity reflexes – technique

EMG recordings are made in a bipolar fashion with subdermal EEG needle electrodes. A range of different high- and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should

be avoided [66]. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine Teflon coated silver wires that have the tip bare, which are inserted with a spinal tap needle can be used for recordings from deep muscles. The active needle is inserted at the motor point of each muscle and the reference needle is inserted distally over tendon or bone. The needles are secured with tape.

Eight channels of intralimb and interlimb reflex EMG activity are recorded simultaneously bilaterally from the lower extremities. When monitoring the function of the spinal cord, reflex activity is recorded from the vastus medialis, tibialis anterior, gastrocnemius and abductor hallucis muscles. When monitoring nerve root function, reflex activity is recorded bilaterally from the vastus medialis, tibialis anterior, peroneus longus and gastrocnemius muscles. When monitoring the function of the cauda equina when lower sacral nerve roots are at risk, reflex activity is recorded from the external anal sphincter, vastus medialis, tibialis anterior and gastrocnemius muscles. Eight channels of single-sweep triggered reflex activity are recorded simultaneously following unilateral bipolar simultaneous electrical stimulation of the common peroneal and tibial mixed nerves just proximal to the knee in the popliteal fossa. Stimulation is through subdermal EEG needle electrodes or surface electrodes. The anode is placed in the medial distal area of the popliteal fossa, and the cathode is placed approximately 4 to 5 cm lateral and proximal. Constant current of 10 to 80 mA with duration of 1.0 msec and a rate of 0.2 Hz is used. The duration of the activity is variable and the timebase is adjusted to record the total duration of the reflex activity (100–5000 msec). Baseline recordings are made with the patient anesthetized before the start of the surgical procedure.

RECORDING OF MONOSYNAPTIC AND POLYSYNAPTIC REFLEXES DURING SELECTIVE DORSAL ROOT RHIZOTOMY

Background

Monosynaptic and polysynaptic reflexes are recorded during selective dorsal root rhizotomy to determine which sensory rootlets contribute the most to the facilitation of hyperactive spinal cord reflex processing. Rootlets are selectively surgically sectioned to reduce spasticity and increase function in patients with spastic cerebral palsy. Those sensory rootlets sectioned may be identified by intraoperative electrical stimulation, and the type of EMG pattern recorded determines which rootlets contribute the most to spasticity [140–142]. Selective dorsal root rhizotomy has been found to improve postoperative function, with 80%

of patients showing improved walking and 90% a reduction of muscle tone. Improved muscle tone in the upper extremities and improved speech occurred in from 60 to 70% of the patients following lumbosacral dorsal root rhizotomy. Random rootlet sectioning without intraoperative monitoring has been performed but good outcome data is not available [143].

Technique

EMG reflex activity can be recorded from the lower and upper extremities, the face and neck. EMG recordings are made most frequently in a bipolar fashion with subdermal EEG needle electrodes. Monopolar EMG needle electrodes and longer uncoated stainless steel needle electrodes may also be used. Fine Teflon coated silver wires that have the tip bare, which are inserted with a spinal tap needle can be used for recordings from deep muscles. The active needle is inserted at the motor point of each muscle and the reference needle is inserted distally over tendon or bone. The needles are secured with tape. A range of different high- and low-frequency filters are used. A high-frequency filter of 10 to 30 KHz and a low-frequency filter of 2 to 30 Hz are most often used. A low-frequency filter greater than 50 Hz and a high-frequency filter less than 3 KHz should be avoided [66]. Sixteen channels of single-sweep recordings are needed to detect all levels of suprasegmental reflex spread. In the lower extremities the following muscles may be recorded from: adductor longus (L2–L4), vastus medialis (L2–L4), gastrocnemius (S1–S2), tibialis anterior (L4-S1), abductor hallucis (S2–S3) and external anal sphincter (S2–S4). In the upper extremity the following muscles may be recorded from: deltoid, biceps, flexor carpi radialis, extensor digitorum communis and abductor pollicis brevis. In addition, the orbicularis oris, orbicularis oculi, sternocleidomastoid and trapezius muscles can be recorded from. In the lower extremity muscles selected for recording must have myotomal representation from L2 through S3. If possible, those muscles that have the greatest spasticity clinically should be recorded from.

Identification of sensory and motor roots

In addition to anatomical considerations, the sensory and motor roots are identified by electrical parameters. Constant current and constant voltage stimulation has been used. With constant current stimulation the duration is 0.1 ms and the rate of stimulation is 1 Hz. The sensory and motor root bundles at each spinal level are stimulated and are identified by threshold differences. When using a handheld bipolar hooked electrode, the motor roots have a

constant current threshold of less than 1.0 mA and the sensory root threshold is from 2–10 mA [68] or 5–20 mA [69]. Bipolar stimulation is used with an interelectrode distance of 0.5–1.0 cm. The S1 root is usually the first identified and is usually the first to be tested. During stimulation the root is held by the surgeon clear of cerebrospinal fluid and the root is held without tension [144]. The cathode is distal and the intensity is increased until the threshold is reached. The CMAPs recorded help to determine which root has been stimulated. The timebase is 100 milliseconds. It is important to identify the S2 dorsal root to prevent damage to those sensory fibers subserving bladder reflexes [137]. The sacral sensory nerve roots that contribute to the pudendal nerve may be identified in the cauda equina by stimulation of the pudendal nerves distally and recording in a bipolar fashion from the sacral sensory roots in the surgical field [145]. The electrophysiological technique used for the identification of sensory and motor nerve roots may block true reflex responses. Gentle retraction of the dorsal rootlets during electrical evaluation may produce a conduction block of the reflex afferents. Electrical stimulation distal to the conduction block may stimulate adjacent ventral roots with the recording of nonreflex motor responses [146].

Identification of hyperactive sensory rootlets

After the innervation pattern and threshold of the dorsal root has been determined, the root is carefully subdivided into from 2 to 7 smaller rootlets [147]. The threshold for each rootlet may once again be determined. Each rootlet is next stimulated at 50 Hz for 1 s to determine which rootlets are hyperactive and need to be sectioned. The duration of each pulse in the 50 Hz train is 0.1 ms and the timebase is 1–2 s. This stimulation intensity is either at the single-pulse threshold or is reduced by 25 to 30%. The type and distribution of the reflex EMG pattern is noted for each sensory rootlet stimulated. Bilaterally the rootlets of the dorsal roots from L2 to S2 are stimulated with this technique. In addition to recording CMAPs the pattern of contraction is observed and palpated by the neurophysiologist.

The response of each rootlet is graded as: 0 – single discharge, 1 – sustained, ipsilateral same myotome, 2 – sustained, ipsilateral same and ipsilateral adjacent myotome, 3 – sustained, many levels ipsilaterally and 4 – sustained, ipsilateral and contralateral and suprasegmental spread. The type of EMG discharge is noted as: decremental, squared, decremental-squared, incremental, multiphasic, clonic and sustained. Clonic, incremental, multiphasic and sustained discharges are considered criteria for rootlet sectioning and rootlets with a grade of 3 or 4 are usually sectioned. From

25 to 80% of the rootlets at each level are sectioned. Criteria for sectioning rootlets are based upon clinical and electrical observations [142, 147].

Free-run EMG recordings

Free-run EMG activity may be recorded during the laminectomy and during manipulation of the nerve roots to help prevent damage to the roots secondary to tension on the roots.

SAFETY AND DISINFECTION

Disinfection procedures for personnel, equipment and electrodes should be consistent with those detailed in "Infection Control and the Electrodiagnostic Department: 1994 Guidelines" [148] as well as the policy and procedures of the individual institution. In particular:

1. Sterile areas should always be respected and non-sterile personnel should minimize their activity around those areas.
2. Proper surgical attire should be worn, including scrubs, hats, masks, and shoe covers.
3. Brain monitors and ancillary equipment such as cables and the electrode jackbox should be cleaned with a high-level disinfectant after use.
4. All equipment used in the OR should be properly isolated and protected in some manner from contamination or exposure to body fluids.
5. Gloves should be routinely worn when in high-risk areas such as the ICU and OR arenas, particularly when touching patients with wounds, bloody areas, and other secretions.
6. Gloves should also be worn when handling any neuromonitoring item soiled by bodily fluids.
7. Hand washing before and after patient contact should be done with a hospital-approved antimicrobial preparation.
8. Disposable subdermal needle electrodes should be used for recording and disposed of in the appropriate manner for sharp objects.
9. Reusable needle electrodes should be washed IN A BIOACTIVE ENZYMATIC DETERGENT, soaked in Clorox (1:10 solution) for 10–15 min, packaged and taken for STEAM STERILIZATION, TYPICALLY FOR 1 HOUR AT 120 °C AT 15 PSI.
10. Intraoperative neuromonitoring personnel should adhere to standard precautions which guard against the risk of and provide inoculation against Hepatitis B.

CREDENTIALS AND STAFFING PRACTICE PATTERNS

Staffing models for intraoperative neurophysiological monitoring (IOM) vary greatly across institutions. The ASNM recognizes the importance of appropriately qualified IOM personnel and refers the reader to a separate position statement regarding this sensitive issue. However, prior to finalizing our positions on staffing, the ASNM believes that the following statements may assist institutions and individuals in evaluating IOM personnel qualifications.

IOM may be divided into two levels of service delivery: professional and technical. Individuals performing or supervising IOM services should have gained appropriate education, training, and experience prior to practicing in a clinical setting. The ASNM recommends certification by the American Board of Neurophysiologic Monitoring (ABNM), or its equivalent, as a measure of professional-level qualification. Criteria for ABNM certification include: (1) an advanced degree: Masters, Ph.D., M.D., or D.O.; (2) documented clinical experience with the requirement of at least 300 monitored cases over a minimum of three years; (3) surgeon attestations regarding monitoring experience; and (4) the passing of two examinations, one written and the other oral. The ASNM recommends the Certification in Intraoperative Monitoring (CNIM), or its equivalent, sponsored by the American Board of Registry for Electroneurodiagnostic Technologists (ABRET) as a measure of technical-level qualification. Criteria for ABRET certification include: (1) a high school degree and healthcare credential or bachelor's degree; (2) documented clinical experience with the requirement of at least 100 cases; (3) the passing of a written examination; and (4) attestation by a supervising physician as to eligibility. In addition to appropriate credentials, the ASNM recognizes the value of continuing education, as well as the development of institutional policies and procedures including scope-of-practice, duties related to both technical and professional aspects of practice, and interpersonal communications.

DOCUMENTATION

Intraoperative neurophysiological records should include detailed information such as demographic data, diagnosis, type of surgery, equipment used, neuromonitoring procedures, personnel, anesthesia used, and the patient's vital signs (e.g., heart rate, blood pressure, temperature, etc.). Great care should be taken to acquire, print and store on disk artifact-free data before, during and after various routine and critical surgical events. A written log should be kept of all acquired signals and all events related to surgery. All communication to the surgeon or other personnel in the O.R. regarding the status of the neurophysiological signals should be documented. All changes in the patient's care such as changes in the anesthetic technique should be documented.

DEFINITIONS

Introduction

Based upon scientific studies, case studies and the expert opinion of those in the intraoperative monitoring field, these techniques are given evidence ratings and a strength-of-practice rating [149].

Quality of evidence ratings

Class I.   Evidence provided by one or more well-designed prospective blinded, controlled studies.
Class II. Evidence provided by one or more well-designed clinical studies such as case control, cohort studies, etc.
Class III. Evidence provided by expert opinion, non-randomized historical controls or case reports of one or more.

Strength-of-recommendation ratings

Type A. Strong positive recommendation, based on Class I evidence, or overwhelming Class II evidence.
Type B. Positive recommendation, based on Class II evidence.
Type C. Positive recommendation, based on strong consensus of Class III evidence.
Type D. Negative recommendation, based on inconclusive or conflicting Class II evidence.
Type E. Negative recommendation, based on evidence of ineffectiveness or lack of efficacy.

Standards

Standards are generally-accepted principles for patient management that reflect a high degree of clinical certainty.

Guidelines

Guidelines are recommendations for patient management that may identify a particular strategy or range of management strategies that reflect moderate clinical certainty.

Practice options or advisories

Practice options or advisories are other strategies for patient management for which there is some favorable evidence, but for which the community still considers this an option to be decided upon by individual practitioners.

Practice parameters

Practice parameters are results in the form of one or more specific recommendations from a scientifically based analysis of a specific clinical problem.

SUMMARY

Neuromuscular junction monitoring

The use of neuromuscular junction monitoring when using EMG monitoring is a practice guideline and is a valuable adjunct to monitoring of reflexes, spontaneous EMG, or stimulated EMG (Type C recommendation).

Free-run EMG

The use of free-run EMG for the monitoring of segmental motor spinal nerve root function is a practice guideline and is of value during surgical procedures which place motor nerve roots at risk (Type C recommendation).

Electrical stimulation of pedicle screws

The use of electrical stimulation to help determine correct placement of spinal pedicle screws is a practice guideline that is of value for determining appropriate screw placement (Type C recommendation).

Electrical impedance testing

The use of electrical impedance testing to help determine correct placement of pedicle screws is not recommended (Type D recommendation: based upon inconclusive or conflicting Class II evidence).

Electrical stimulation of spinal motor nerve roots

The use of electrical stimulation of spinal motor nerve roots is a practice guideline of established value (Type B recommendation). Spinal nerve root stimulation can be used to identify motor axons in the cauda equina during tumor removal, to determine which motor axons have continuity peripherally during surgery for plexus tumors or traumatic lesions, and to determine nerve root threshold during evaluation of pedicle screw placement.

Electrical stimulation of iliosacral screws

The use of electrical stimulation to help determine the correct placement of iliosacral screws is a practice guideline and is of value in assessing correct screw placement (Type B recommendation).

H-reflexes

The use of H-reflexes is a practice option (Type C recommendation).

F-responses

The use of F-responses is a practice option (Type C recommendation).

The sacral reflex

The use of the sacral reflex is a practice option (Type C recommendation).

Intralimb and interlimb reflexes

The use of intralimb and interlimb reflexes is a practice option (Type C recommendation).

Monosynaptic and polysynaptic reflexes

The use of monosynaptic and polysynaptic reflexes during selective dorsal root rhizotomy is a practice option (Type C recommendation).

REFERENCES

1. Nash CL, Lorig RA, Schatzinger LA, Brown RH. Spinal cord monitoring during operative treatment of the spine. Clin Orthop Relat Res 1977; 126: 100–105.
2. Cohen AR, Young W, Ransohoff J. Intraspinal localization of the somatosensory evoked potential. Neurosurgery 1981; 9(2): 157–162.
3. Cusick JF, Myklebust JF, Larson SJ, Sances A Jr. Spinal cord potentials in the primate: Neural substrate. J Neurosurg 1978; 49(4): 551–557.
4. Larson SJ, Sances A Jr., Christenson PC. Evoked somatosensory potentials in man. Arch Neurol 1966; 15(1): 88–93.
5. Macon JB, Poletti CE, Sweet WH, Ojemann RG, Zervas NT. Conducted somatosensory evoked potentials during spinal surgery. Part 2: Clinical applications. J Neurosurg 1982; 57(3): 354–359.

6. Jones SJ, Edgar MA, Ransford AO. Sensory nerve conduction in the human spinal cord: Epidural recordings made during spinal cord surgery. J Neurol Neurosurg Psychiatry 1982; 45: 446–452.

7. York DH. Somatosensory evoked potentials in man: Differentiation of spinal pathways responsible for conduction from forelimbs vs hindlimb. Prog Neurobiol 1985; 25(1): 1–25.

8. Powers SK, Bolger CA, Edwards MS. Spinal cord pathways mediating somatosensory evoked potentials. J Neurosurg 1982; 57(4): 472–482.

9. Simpson RK Jr., Blackburn JG, Martin HF III, Katz S. Peripheral nerve fibers and spinal cord pathway contribution to the somatosensory evoked potential. Exp Neurol 1981; 73(3): 700–715.

10. Leppanen RE, Tyler W. Evidence for parallel spinal cord conduction by dissociation of spinal and cortical somatosensory components: Case study. J Clin Neurophysiol 2000; 17(5): 531.

11. Hurlbert RJ, Fehlings MG, Moncada MS. Use of sensory-evoked potentials recorded from the human occiput for intraoperative physiologic monitoring of the spinal cord. Spine 1995; 20(21): 2318–2327.

12. Dimitrijevic MR. Clinical neurophysiology of neural stimulation. In: Ducker TD, Brown RH, eds. Neurophysiology and standards of spinal cord monitoring. New York: Springer-Verlag, 1988: 11–15.

13. Merton PA, Morton HB. Stimulation of the cerebral cortex in the intact human subject. Nature 1980; 285(5762): 227.

14. Jellinek D, Jewkes D, Symon L. Noninvasive intraoperative monitoring of motor evoked potentials under propofol anesthesia: Effects of spinal surgery on the amplitude and latency of motor evoked potentials. Neurosurgery 1991; 29(4): 551–557.

15. Levy WJ. Use of motor evoked potentials as a monitoring tool. In: Stimulation of the brain and spinal cord: Fundamentals and clinical applications. New York: Alan R. Liss, Inc., 1988: 275–296.

16. Edmonds HL Jr., Paloheimo MP, Backman MH, Johnson JR, Holt RT, Shields CB. Transcranial magnetic motor evoked potentials (tcMMEP) for functional monitoring of motor pathways during scoliosis surgery. Spine 1989; 14(7): 683–686.

17. Taniguchi M, Cedzich C, Schramm. Modification of cortical stimulation for motor evoked potentials under general anesthesia: technical description. Neurosurgery 1993; 32(2): 219–226.

18. Leppanen RE, et al. Intraoperative interaction of descending transcranial electrical motor evoked potentials and ascending somatosensory evoked potentials and F-responses. Presented at: The Twelfth Annual Meeting of the American Society of Neurophysiological Monitoring. May 11, 2001, New Orleans, Louisiana.

19. Owen JH, Bridwell KH, Grubb R, Jenny A, Allen B, Padberg AM, Shimon SM. The clinical application of neurogenic motor evoked potentials to monitor spinal cord function during surgery. Spine 1991; 16(8 Suppl): S385–S390.

20. Leppanen RE, Madigan R, Sears C, Maguire J, Wallace S, Captain J. Intraoperative collision studies demonstrate descending spinal cord stimulated evoked potentials and ascending somatosensory evoked potentials are mediated through common pathways. Presented at: The Annual Meeting of the American Society of Neurophysiological Monitoring, May 2, 1997, Chicago, Illinois.

21. Leppanen RE. Is the neurogenic motor evoked potential really a motor response? Presented at: The Annual Meeting of the American Academy of Clinical Neurophysiology, January 28, 1999, Sante Fe, New Mexico.

22. Leppanen RE, Madigan R, Sears C, Maguire J, Wallace S, Captain J. Intraoperative collision studies demonstrate descending spinal cord stimulated evoked potentials and ascending somatosensory evoked potentials are mediated through common pathways. Clin Neurophysiol 1999; 110: 2265.

23. Toleikis JR, Skelly JP, Carlvin AO, Burkus JK. Spinally elicited peripheral nerve responses are sensory rather than motor. Clin Neurophysiol 2000; 111(4): 736–742.

24. Leppanen RE. Faces of spine care. From the electrodiagnostics lab. Descending neurogenic evoked potentials. Spine J 2004; 4(6): 713–716.

25. Leppanen RE, Maguire J, Wallace S, et al. Intraoperative recording of long-latency lower extremity reflexes for the detection of suprasegmentally altered complex spinal cord electrophysiological processing. Presented at: The Southern EEG Society Meeting, May, 1992, Richmond, Virginia.

26. Leppanen RE, Maguire J, Wallace S, Madigan R, Draper V. Intraoperative lower extremity reflex muscle activity as an adjunct to conventional somatosensory-evoked potentials and descending neurogenic monitoring in idiopathic scoliosis. Spine 1995; 20(17): 1872–1877.

27. Leis AA, Zhou HH, Mehta M, Harkey HL 3rd, Paske WC. Behavior of the H-reflex in humans following mechanical perturbation or injury to rostral spinal cord. Muscle Nerve 1996; 19(11): 1373–1382.

28. Leis AA. Physiology of acute spinal cord injury (SCI) in humans. I. Behavior of the H-reflex and F-wave immediately following injury to rostral spinal cord in humans. Presented at: The Annual Meeting of the American Academy of Clinical Neurophysiology, June 20–22, 1996, Chicago, IL.

29. Maguire J, Wallace S, Madigan R, Leppanen RE, Draper V. Intraoperative long-latency reflex activity in idiopathic scoliosis demonstrates abnormal central processing: A possible etiology for idiopathic scoliosis. Spine 1993; 18(12): 1621–1626.

30. Goodgold J, Eberstein A. Anatomy of nerve and muscle – a review. In: Electrodiagnosis of neuromuscular diseases. Baltimore: Williams & Wilkins, 1983: 2.

31. Maguire J, Wallace S, Madigan R, Leppanen R, Draper V. Evaluation of intrapedicular screw position using intraoperative evoked electromyography. Spine 1995; 20(9): 1068–1074.

32. Collins VJ. Principles of anesthesia: General and regional anesthesia, 3rd ed., Vol. 2. Philadelphia: Lea & Febiger, 1993: 850–937.

33. Viby-Mogensen J. Neuromuscular monitoring. In: Miller RD, ed. Anesthesia, 6th ed. New York: Elsevier Churchhill Livingston, 2005: 1551–1570.

34. Kimura J. Anatomy and physiology of the neuromuscular junction. In: Electrodiagnosis in diseases of nerve and muscle: Principles and practice. Philadelphia: F.A. Davis, 1983: 171–188.

35. Dumitru D. Electrodiagnostic medicine. Baltimore: Mosby, 1995: 929–1029.

36. Gronert GA, Antognini JF, Pessah IN. Malignant hyperthermia. In: Miller RD, ed. Anesthesia, 5th ed. Philadelphia: Churchill Livingstone, 2000: 1033–1052.

37. Hayes A, Breslin D, Reid J, Mirakhur RK. Comparison of recovery following rapacuronium, with and without

neostigmine, and succinylcholine. Anaesthesia 2000; 55(9): 859–863.

38. Calancie B, Madsen P, Lebwohl N. Stimulus-evoked EMG monitoring during transpedicular lumbosacral spine instrumentation: Initial clinical results. Spine 1994; 19(24): 2780–2786.

39. Minahan RE, Riley III LH, Lukaczyk T, Cohen DB, Kostuik JP. The effect of neuromuscular blockade on pedicle screw stimulation thresholds. Spine 2000; 25(19): 2526–2530.

40. Yamada H, Transfeldt EE, Tamaki T, Nishiura H, Taylor BA, Torres F, Iaizzo PA. General anesthetic effects on compound muscle action potentials elicited by single or dual spinal cord stimulation. J Spinal Disord 1995; 8(2): 157–162.

41. Zhou HH, Mehta M, Leis AA. Spinal cord motoneuron excitability during isoflurane and nitrous oxide anesthesia. Anesthesiology 1997; 86(2): 302–307.

42. Ravussin P, Tribolet N. Total intravenous anesthesia with propofol for burst suppression in cerebral aneurysm surgery: Preliminary report of 42 patients. Neurosurgery 1993; 32(2): 236–240.

43. Deletis V, Vodusek DB. Intraoperative recording of the bulbocavernosus reflex. Neurosurgery 1997; 40(1): 88–93.

44. Mavroudakis N, Vandesteene A, Brunko E, Defevrimont M, Zegers de Beyl D. Spinal and brain-stem SEPs and H-reflex during enflurane anesthesia. Electroencephalogr Clin Neurophysiol 1994; 92(1): 82–85.

45. Mecheise K. The motor unit. In: Notermans SLH, ed. Current practice of clinical electromyography. Amsterdam: Elsevier, 1984: 83–117.

46. English AW, Wolf SL, Segal RL. Compartmentalization of muscles and their motor nuclei: The partitioning hypothesis. Phys Ther. 1993; 73(12): 857–867.

47. Ma MD, Liveson JA. Nerve conduction handbook. Philadelphia: F. A. Davis, 1983.

48. Basmajian JV, DeLuca CJ. Muscles alive. Their functions revealed by electromyography, 5th ed. Baltimore: Williams & Wilkins, 1985: 43.

49. Dumitru D. Electrodiagnostic medicine. Radiculopathies. Baltimore: Mosby, 1995: 523–584.

50. Myology. In: Warwick R, Williams PL, eds. Gray's Anatomy, 35th ed. Philadelphia: W.B. Saunders Company, 1973: 474–585.

51. Neurology. Lumbar ventral rami. In: Warwick R, Williams PL, eds. Gray's Anatomy, 35th ed. Philadelphia: W.B. Saunders Company, 1973: 1050.

52. Bouchard JM, Copty M, Langelier R. Preoperative diagnosis of conjoined roots anomaly with herniated lumbar disks. Surg Neurol 1978; 10(4): 229–231.

53. Cannon BW, Hunter SE, Picaza JA. Nerve-root anomalies in lumbar-disc surgery. J Neurosurg 1962; 19: 208–214.

54. McElvenny RT. Anomalies of the lumbar spinal cord and nerve roots. Clin Orthop Relat Res 1956; 2(8): 61–70.

55. Neidre A, MacNab I. Anomalies of the lumbosacral nerve roots: Review of 16 cases and classification. Spine 1983; 8(3): 294–299.

56. Rask MR. Anomalous lumbosacral nerve roots associated with spondylolisthesis. Surg Neurol 1977; 8(2): 139–140.

57. Bogduck N, Twomey LT. Nerves of the lumbar spine. In: Clinical anatomy of the lumbar spine, 2nd ed. New York: Churchill Livingstone, 1991: 111–112.

58. Cohen MS, Wall EJ, Brown RA, Rydevik B, Garfin SR. Cauda equina anatomy II: Extrathecal nerve roots and dorsal root ganglia. Spine 1990; 15(12): 1248–1251.

59. Wall EJ, Cohen MS, Massie JB, Rydevik B, Garfin SR. Cauda equina anatomy I: Intrathecal nerve root organization. Spine 1990; 15(12): 1244–1247.

60. Bogduk N, Twomey LT. Clinical anatomy of the lumbar spine, 2nd ed. Melbourne: Churchill Livingstone, 1991.

61. Leffert RD. Brachial plexus injuries. New York: Churchill Livingstone, 1985.

62. Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984; 9(1): 7–15.

63. Olmarker K. Spinal nerve root compression: Nutrition and function of the porcine cauda equina compressed in vivo. Acta Orth Scand Supp 1991; 242(62): 1–27.

64. Berthold CH, Carlstedt T, Corneliuson O. Anatomy of the nerve root at the central-peripheral transitional region. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. Peripheral neuropathy. Philadelphia: W.B. Saunders, 1984: 156–170.

65. Howe JF, Loeser JD, Calvin WH. Mechanosensitivity of dorsal root ganglia and chronically injured axons: A physiological basis for the radicular pain of nerve root compression. Pain 1977; 3(1): 25–41.

66. Dumitru D. Electrodiagnosis medicine. Needle electromyography. Baltimore: Mosby, 1995: 213.

67. Isley MR, Pearlman RC, Wadsworth JS. Recent advances in intraoperative neuromonitoring of spinal cord function: Pedicle screw stimulation techniques. Am J END Technol 1997; 37: 93–126.

68. Leppanen RE. Monitoring the motor system via spontaneous activity and reflex arc techniques. Presented at: Neurophysiology: Intraoperative Monitoring, Chicago Institute of Neurosurgery and Neuroresearch, October 21, 1994, Chicago, Illinois.

69. Taborikova H, Sax DS. Motoneurone pool and the H-reflex. J Neurol Neurosurg Psychiat 1968; 31: 354–361.

70. Beatty RM, McGuire P, Moroney JM, Holladay FP. Continuous intraoperative electromyographic recording during spinal surgery. J Neurosurg 1995; 82(3): 401–405.

71. Holland NR, Kostuik JP. Continuous electromyoghraphic monitoring to detect nerve root injury during thoracolumbar scoliosis surgery. Spine 1997; 22(21): 2547–2550.

72. Owen JH, Kostuik JP, Gornet M, Petr M, Skelly J, Smoes C, Szymanski J, Townes J, Wolfe F. The use of mechanically elicited electromyograms to protect nerve roots during surgery for spinal degeneration. Spine 1994; 19(15): 1704–1710.

73. Legatt AD, Schroeder CE, Gill B, Goodrich JT. Electrical stimulation and multichannel EMG recording for identification of functional neural tissue during cauda equina surgery. Childs Nerv Syst 1992; 8(4): 185–189.

74. Leppanen RE. Intraoperative monitoring of trapezius function. From the electrodiagnostics lab . . . , Faces of spine care. Spine J 2003; 3(3): 248.

75. Fan D, Schwartz DM, Vaccaro AR, Hillibrand AS, Albert TJ. Intraoperative neurophysiologic detection of iatrogenic C5 nerve root injury during laminectomy for cervical compression myelopathy. Spine 2002; 27(22): 2499–2502.

76. Matsuzaki H, Toiyama Y, Matsumoto F, Hoshino M, Kiuchi T, Toriyama S. Problems and solutions of pedicle screw

plate fixation of lumbar spine. Spine 1990; 15: 1159–1165.

77. Weinstein JN, Spratt KF, Spengler D, Brick C, Reid S. Spinal pedicle fixation: Reliability and validity of roentgenogram-based assessment and surgical factors on successful screw placement. Spine 1988; 13: 1012–1018.

78. West JL III, Ogilvie JW, Bradford DS. Complications of variable screw plate screw fixation. Spine 1991; 16: 576–579.

79. Krag MH, Beynnon BD, Pope MH, Frymoyer JW, Haugh LD, Weaver DL. An internal fixator for posterior application to short segments of the thoracic, lumbar or lumbosacral spine. Design and testing. Clin Orthop Relat Res 1986; 203: 75–98.

80. Louis R. Fusion of the lumbar and sacral spine by internal fixation with screws and plates. Clin Orthop Relat Res 1986; 203: 18–33.

81. Luque ER. Interpeduncular segmental fixation. Clin Orthop Relat Res 1986; 203: 54–57.

82. Magerl FP. Stabilization of the lower thoracic and lumbar spine with external skeletal fixation. Clin Orthop Relat Res 1984; 189: 125–141.

83. Whitecloud TS, Skalley TC, Cook SD, Morgan EL. Roentgenographic measurement of pedicle screw penetration. Clin Orthop Relat Res 1989; 245: 57–68.

84. Gepstein R, Brown MD. Somatosensory evoked potentials in lumbar nerve root decompression. Clin Orthop Relat Res 1989; 245: 69–71.

85. Keim HA, Hajdu M, Gonzalez EG, Brand L, Balasubramanian E. Somatosensory evoked potentials as an aid in the diagnosis and Intraoperative management of spinal stenosis. Spine 1985; 10(4): 338–344.

86. Cohen BA, Huizenga BA. Dermatomal monitoring for surgical correction of spondylolisthesis: A case report. Spine 1988; 13(10): 1125–1128.

87. Cohen BA, Major MR, Huizenga BA. Predictability of adequacy of spinal root decompression using evoked potentials. Spine 1991; 16(8 Suppl): S379–S384.

88. Herron LD, Trippi AC, Gonyeau M. Intraoperative use of dermatomal somatosensory-evoked potentials in lumbar stenosis surgery. Spine 1987; 12(4): 379–383.

89. Owen JH, Padberg AM, Spahr-Holland L, Bridwell KH, Keppler L, Steffee AD. Clinical correlation between degenerative spine disease and dermatomal somatosensory-evoked potentials in humans. Spine 1991; 16(6 Suppl): S201–S205.

90. Toleikis JR, Carlvin AO, Shapiro DE, Schafer MF. The use of dermatomal somatosensory evoked potentials(DSSEPs) to monitor surgical procedures involving intrapedicular fixation of the lumbo-sacral spine. Presented at: The American Electroencephalographic Society Annual Meeting, December 12, 1991, Philadelphia, Pennsylvania.

91. Calancie B, Lebwohl N, Madsen P, Klose KJ. Intraoperative evoked EMG monitoring in an animal model: A new technique for evaluating pedicle screw placement. Spine 1992; 17(10): 1229–1235.

92. Calancie B, Madsen P, Lebwohl N. Stimulus-evoked EMG monitoring during transpedicular lumbosacral spine instrumentation. Initial clinical results. Spine 1994; 19(24): 2780–2786.

93. Clements DH, Morledge DE, Martin WH, Betz RR. Evoked and spontaneous electromyography to evaluate lumbosacral pedicle screw placement. Spine 1996; 21(5): 600–604.

94. Darden BV II, Wood KE, Hatley MK, Owen JH, Kostuik J. Evaluation of pedicle screw insertion monitored by intraoperative evoked electromyography. J Spinal Disord 1996; 9(1): 8–16.

95. Glassman SD, Dimar JR, Puno RM, Johnson JR, Shields CB, Linden RD. A prospective analysis of intraoperative electromyographic monitoring of pedicle screw placement with computed tomographic scan configuration. Spine 1995; 20(12): 1375–1379.

96. Lenke LG, Padberg AM, Russo MH, Bridwell KH, Gelb DE. Triggered electromyographic threshold for accuracy of pedicle screw placement: An animal model and clinical correlation. Spine 1995; 20: 1585–1591.

97. Skelly JP, Toleikis JR, Carlvin AO. Pedicle screw stimulation in a fluid environment. Presented at: The Tenth Annual Meeting of the American Society of Neurophysiological Monitoring, May 1, 1999, Denver, Colorado.

98. Toleikis JR, Skelly JP, Carlvin AO, Toleikis SC, Bernard TN, Burkus JK, Burr ME, Dorchak JD, Goldman MS, Walsh TR. The usefulness of electrical stimulation for assessing pedicle screw placements. J Spinal Disord 2000; 13(4): 283–289.

99. Myers BS, Hasty CC, Floberg DR, Hoffman RD, Leone BJ, Richardson WJ. Measurement of vertebral cortical integrity during pedicle exploration for intrapedicular fixation. Spine 1995; 20: 144–148.

100. Darden BV II, Owen JH, Hatley MK, Kostuik J, Tooke SM. A comparison of impedance and electromyogram measurements in detecting the presence of pedicle wall breakthrough. Spine 1998; 23: 256–262.

101. Danesh-Clough T, Taylor P, Hodgson B, Walton M. The use of evoked EMG in detecting misplaced thoracolumbar pedicle screws. Spine 2001; 26(12): 1313–1316.

102. Shi YB, Binette M, Martin WH, Pearson JM, Hart RA. Electrical stimulation for intraoperative evaluation of thoracic pedicle screw placement. Spine 2003; 28(6): 595–601.

103. Kellam JP, McLaren A, Tile M. Internal fixation. In: Tile M, ed. Fractures of the pelvis and acetabulum, 2nd ed. Baltimore: Williams & Wilkins, 1995: 150–199.

104. Templeman D, Schmidt A, Freese J, Weisman I. Proximity of iliosacral screws to neurovascular structures after internal fixation. Clin Orthop Relat Res 1996; 329: 194–198.

105. Matta JM, Tornetta P III. Internal fixation of unstable pelvic ring injuries. Clin Orthop Relat Res 1996; 329: 129–140.

106. Webb LX, de Araujo W, Donofrio P, Santos C, Walker FO, Olympio MA, Haygood T. Continuous EMG monitoring for placement of percutaneous iliosacral screws. J Orthop Trauma. 2000; 14(4): 245–254.

107. Moed BR, Ahmad BK, Craig JG, Jacobson GP, Anders MJ. Intraoperative monitoring with stimulus-evoked electromyography during placement of iliosacral screws: An initial clinical study. J Bone Joint Surg Am. 1998; 80(4): 537–546.

108. Grillner S. Control of locomotion in bipeds, tetrapods and fish. In: Brookhart JM, Mountcastle VB, eds. Handbook of physiology. The nervous system, Vol. II, Part 2, Motor control. Baltimore: Williams & Wilkins, 1981: 1179–1236.

109. Anderson B, Binder M. Spinal and supraspinal control of movement and posture. In: Patton HD, Fuchs AF, Hillie B, Scher AM, Steiner R, eds. Textbook of physiology: Excitable cells and neurophysiology. Philadelphia: W.B. Saunders Co., 1988: 563–581.

110. Binder M. Peripheral motor control: Spinal reflex actions of muscle, joint and cutaneous receptors. In: Patton HD, Fuchs AF, Hillie B, Scher AM, Steiner R, eds. Textbook of physiology: Excitable cells and neurophysiology. Philadelphia: W.B. Saunders Co., 1988: 522–548.

111. Lloyd DPC. Reflex action in relation to pattern and source of afferent stimulation. J Neurophysiology 1943; 6: 111–120.

112. Kimura J. Principles of nerve conduction studies. Electrodiagnosis in diseases of nerve and muscle: Principles and practice. Philadelphia: F.A. Davis, 1983: 83–104.

113. Meunier S, Pierrot-Deseillgny E, Simonetta M. Pattern of monosynaptic heteronymous 1a connections in the human lower limb. Exp Brain Res 1993; 96(3): 534–544.

114. Magladery JW, Porter WE, Park AM, Teasdall RD. Electrophysiological studies of nerve and reflex activity in normal man. IV. Two-neurone reflexes and identification of cortical action potentials from spinal roots and cord. Bull Johns Hopkins Hosp. 1951; 88(6): 499–519.

115. Magladery JW, Teasdall RD, Park AM, Languth HW. Electrophysiological studies of reflex activity in patients with lesions of the nervous system. I. A comparison of spinal motoneurone excitability following afferent nerve volleys in normal persons and patients with upper motor neurone lesions. Bull Johns Hopkins Hosp. 1952; 91(4): 219–44.

116. Leppanen RE. H-reflexes in the hand muscles following cervical spinal cord disease. From the electrodiagnostic lab . . . , Faces of spine care. Spine J 2003; 3(5): 405.

117. Hugon M. Proprioceptive reflexes and the H-reflex. Methodology of Hoffman reflexes in man. In: Desmedt JE, ed. New developments in electromyography and clinical neurophysiology. Basel: Karger, 1973: 277–293.

118. Leppanen RE. Intraoperative reflexes can be used to monitor nerve root and spinal cord gray matter function. From the electrodiagnostic lab . . . , Faces of spine care. Spine J 2004; 4(4): 480–481.

119. Schimsheimer RJ, de Visser BW, Kemp B. The flexor carpi radialis H-reflex in lesions of the sixth and seventh cervical nerve roots. J Neurol Neurosurg Psychiatry 1985; 48(5): 445–449.

120. Roth G, Soichot P. Cancellation of single F wave by double stimulation in case of chronic denervation. Electroencephalogr Clin Neurophysiol. 1995; 97(3): 155–158.

121. Panayiotopoulos CP, Chroni E. F-waves in clinical neurophysiology: A review, methodological issues and overall value in peripheral neuropathies. Electroencephalogr Clin Neurophysiol. 1996; 101(5): 365–374.

122. Binder MD. Peripheral motor control: Spinal reflex actions of muscle, joint and cutaneous receptors. In: Textbook of physiology: Excitable cells and neurophysiology. Philadelphia: WB Saunders, 1988: 543.

123. Hagbarth KE: Post-tetanic potentiation of myotatic reflexes in man. J Neurol Neurosurg Psychiatry 1962; 25: 1–10.

124. Weber RJ. Motor and sensory conduction and entrapment syndromes. In: EW Johnson, ed. Practical electromyography, 2nd ed. Baltimore: Williams & Wilkins, 1988: 117.

125. Oh SJ. Anatomical and physiological basis for electromyography studies. In: Clinical electromyography: Nerve conduction studies, 2nd ed. Baltimore: Williams & Wilkins, 1993: 51.

126. Dumitru D. Electrodiagnostic medicine. Special nerve conduction techniques. Baltimore: Mosby, 1995: 203–204.

127. Schoenfeldt R, Groce R, Laurenzi B. Motor system monitoring during joint replacement operations. Neurosurgery 1987; 20(1): 197–198.

128. Leis AA, Kronenberg MF, Stetkarova I, Paske WC, Stokic DS. Spinal motoneuron excitability after acute spinal cord injury in humans. Neurology 1996; 47(1): 231–237.

129. Barnes CD, Joynt RJ, Schottelius BA. Motoneuron resting potentials in spinal shock. Am J Physiol. 1962; 203: 1113–1116.

130. Walmsley B, Tracey DJ. The effect of transection and cold block of the spinal cord on synaptic transmission between 1a afferents and motoneurones. Neuroscience 1983; 9(2): 445–451.

131. Leppanen RE. Spinal cord injury changes caudal segmental spinal cord excitability resulting in changes to spinal cord signal processing and modulation of late response recordings. Spine J 2005; 5(1): 115–117.

132. Leppanen RE. From the electrodiagnostics lab: Where transcranial stimulation, H-reflexes and F-responses monitor cord function intraoperatively. Spine J 2004; 4(5): 601–603.

133. Hicks GE. The reliability and specificity of the Hoffman's Reflex during pediatric spinal instrumentations. J Clin Monit Comput 2004; 18(3): 210.

134. Curt A, Keck ME, Dietz V. Clinical value of F-wave recordings in traumatic cervical spinal cord injury. Electroencephalogr Clin Neurophysiol 1997; 105(3): 189–193.

135. Rechthand E. Bilateral bulbocavernosus reflexes: Crossing of nerve pathways or artifact? Muscle Nerve 1997; 20(5): 616–618.

136. Deletis V, Vodusek DB, Abbott R, Epstein FJ, Turndorf H. Intraoperative monitoring of the dorsal sacral roots: Minimizing the risk of iatrogenic micturition disorders. Neurosurgery 1992; 30(1): 72–75.

137. Huang JC, Deletis V, Vodusek DB, Abbott R. Preservation of pudendal afferents in sacral rhizotomies. Neurosurgery 1997; 41(2): 411–415.

138. Corman ML. Physiologic and anatomical bases of continence. In: Colon and rectal surgery, 3rd ed. Philadelphia: J.B. Lippincott, 1993: 193.

139. Ghez C. The control of movement. In: Kandel ER, Schwartz JH, Jessell TM, eds. Principles of neural science, 3rd ed. East Norwalk, Connecticut: Appleton and Lange, 1991: 533–547.

140. Fasano VA, Barolat-Romana G, Zeme S, Squazzi A. Electrophysiological assessment of spinal circuits in spasticity by direct dorsal root stimulation. Neurosurgery. 1979; 4(2): 146–151.

141. Peacock WJ, Arens LJ, Berman B. Cerebral palsy spasticity. Selective posterior rhizotomy. Pediatr Neurosci 1987; 13(2): 61–66.

142. Staudt LA, Nuwer MR, Peacock WJ. Intraoperative monitoring during selective posterior rhizotomy: Technique and patient outcome. Electroencephalogr Clin Neurophysiol. 1995; 97(6): 296–309.

143. Peacock WJ, Staudt LA. Spasticity in cerebral palsy and the selective posterior rhizotomy procedure. J Child Neurol 1990; 5(3): 179–185.

144. Harper CM, Nelson KR. Intraoperative electrophysiological monitoring in Children. J Clin Neurophysiol 1992; 9(3): 342–356.

145. Lang FF, Deletis V, Cohen HW, Velasquez L, Abbott R. Inclusion of the S2 dorsal rootlets in functional posterior rhizotomy for spasticity in children with cerebral palsy. Neurosurgery 1994; 34(5): 847–853; discussion 853.

146. Logigian EL, Soriano SG, Herrmann DN, Madsen JR. Gentle dorsal root retraction and dissection can cause areflexia: Implications for intraoperative monitoring during "selective" partial dorsal root rhizotomy. Muscle Nerve 2001; 24(10): 1352–1358.

147. Park TS, Gaffney PE, Kaufman BA, Molleston MC. Selective lumbosacral dorsal rhizotomy immediately caudal to the conus medullaris for cerebral palsy. Neurosurgery 1993; 33(5): 929–933; discussion 933–934.

148. Infection control and the electrodiagnostic department; 1994 Guidelines. Am J EEG Technol 1995; 35: 3–36.

149. Nuwer M. Assessment of digital EEG, quantitative EEG, and EEG brain mapping: Report of the American Academy of Neurology and the American Clinical Neurophysiology Society. Neurology 1997; 49(1): 277–292.