A review of neurophysiological testing

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The rapid advances in the technology of, and accumulation of pertinent data in, electrophysiological testing has increased exponentially in the past decade. This is attributable to continued advances in computer technology, biomedical engineering, and now the coregistration of the electrophysiological data with neuroimaging results. Knowledge of normal function and electrophysiological response at rest or on stimulation of the central and peripheral nervous systems is important to the neurosurgeon. Only by a basic understanding of normal and abnormal recordings may diagnoses and localizations be achieved. Intraspinal and intracranial surgical procedures are predicated on nontrauma to the neuraxis. This can be accomplished by performing electrophysiological testing to monitor the function of the spinal and cranial nerves, spinal cord, brainstem, basal ganglia, and cerebrum. If the surgeon cannot delineate critical cortex or pathways, he or she will be unable to avoid these areas in the patient.

KEY WORDS • electrophysiological testing • electroencephalography • magnetoencephalography • evoked potential • computerized tomography imaging • motor pathway stimulation

Sophisticated electrophysiological testing is available to the neurosurgeon for diagnosis and localization. It is imperative that he or she has a basic understanding of neurophysiology and clinical testing of the nervous system as well as recent advances in the field. The purpose of this paper is to review the field of electrophysiological testing and to present the information to the reader in a clear and practical fashion. Recent advances including MEG and functional imaging are presented.

This review is divided into several sections: electromyography and nerve conduction, EEG, MEG, evoked response monitoring, intraoperative testing (including cranial nerve monitoring, basal ganglia and thalamic localization with microelectrode recordings, and cortical mapping), and an overview of neurophysiological testing to help the surgeon protect the spinal cord.

Abbreviations used in this paper: BAEP = brainstem auditory evoked potential; CMAP = compound muscle action potential; CNS = central nervous system; CT = computerized tomography; ECoG = electrocorticography; EEG = electroencephalography; EMG = electromyography; MAP = muscle action potential; MEG = magnetoencephalography; MR = magnetic resonance; PLED = periodic lateralized epileptiform discharge; SD = standard deviation; SSEP = somatosensory evoked potential; STN = subthalamic nucleus; Vim = nucleus ventralis intermedius.

ELECTROMYOGRAPHY AND NERVE CONDUCTION STUDIES

A variety of techniques are available for the electrodiagnosis of the pathologies of muscle, peripheral nerve, and their CNS distribution. Historically, the earliest investigations were performed in the latter portion of the 18th century by Galvani, who published his observations on electricity and muscle contraction. In 1848 Dubois-Raymond discovered the action potential and described it as a negative variation of the standing potential of a nerve related to the conduction of a nerve impulse. Soon thereafter, Hermann von Helmholtz recorded the conduction velocity in a median nerve in a human.¹²⁹

Anatomy and Physiology

The peripheral nerve trunk contains motor, sensory, and autonomic axons, which may be myelinated or unmyelinated; the latter ones have a slower conduction velocity. The resting transmembrane potential of an axon is -80 to -90 mV, based on a membrane impermeable to Na^+ on the outside but permeable to K^+ intracellularly.¹¹⁹ Although the ionic concentrations are equal on either side of the membrane, there are higher concentrations of K^+ on the inside and Na^+ on the outside of the membrane. These

concentrations are maintained by the transmembrane potential and the active transport of Na and K by an energy-dependent pump.¹⁰⁹ When a stimulus is introduced to the axonal membrane, permeability to Na⁺ increases. This brings about a transient change in the transmembrane potential, which increases the conductance of Na⁺ into the cell until the Na⁺ equilibrium potential is reached (30 mV). Once started, the change is rapidly propagated to adjacent inactive portions of the membrane. The propagated disturbance is known as an impulse and the electrical manifestation as the action potential. The creation of this local flow of electrical charge reduces the membrane potential in the inactive regions, increasing permeability of Na⁺ and the flow of current proceeds in continuity. As the initial area repolarizes with Na⁺, conductance low and K⁺ increasing, this process is repeated successfully in both directions from the point of stimulation at a constant velocity and duration, depending on the nerve fibers' excitability and conductive properties.¹²⁹

Conduction has two types: continuous or saltatory. The former occurs in unmyelinated nerve fibers and is continuous and bidirectional. Conduction in a myelinated fiber occurs in a somewhat different manner, however. The myelin sheath, an effective insulator, is covered by a Schwann cell. The cell process is interrupted at intervals up to 2 mm along the length of the fiber, and the axon is uncovered. This is the node of Ranvier. The circuit flow is also bidirectional from one node of Ranvier to the next. The impulse hops from one node to the next (saltatory), resulting in faster conduction. Therefore, large-diameter fibers have faster conduction times (Table 1).⁷⁰

When a mixed nerve is maximally stimulated and recorded monophasically, there is a somewhat irregular contour to the action potential based on the different velocities and thresholds of the nerve (this is called a "compound nerve action potential"). Following the fall of the electri-

cal response in the action potential, there are two low-amplitude, relatively low deflections, based on prior spike activity. The first is a negative after-potential. The nerve is still excitable at this point, and, in fact, may be more easily excitable. Following this, a positive after-potential occurs where the nerve has a high threshold to activation. Both states relate to Na⁺ permeability.³⁹

A motor unit consists of a motor axon and the population of muscle fibers innervated.⁷⁹ The neuromuscular junction is a synapse at which the neural impulse stimulates the release of acetylcholine from the axon terminals, resulting in a brief muscle contraction. The consequent electrical response is the CMAP.⁶ There is release of Ca from the vesicles of the sarcoplasmic reticulum, which facilitates the cross bridging between myosin and actin filament of the myofibrils in the presence of adenosine 5'-triphosphate, resulting in shortening of the muscle.¹⁵⁵

Muscle, as opposed to nerve, may have a heightened response on increased frequency of nerve stimulation. This is called "summation," which may lead to sustained contraction (tetanus). Skeletal muscle may only be activated by motor neurons as opposed to cardiac or smooth muscles.¹⁵⁵ Fast-fatigue and fast-resistant motor units, innervated by large-diameter nerves, are present in the more anaerobically functional muscles. The slow motor unit with smaller-diameter neurons innervate the more aerobically functional muscle fibers.²⁷ When contraction of the muscle occurs, there may be shortening (isotonic), no shortening or external work (isometric), or lengthening when the external force is greater than the muscle contraction.¹⁵⁵

Pathological Features and Testing

Pathological Features. Seddon¹²⁷ described three forms of nerve injury: neurapraxia, axonotmesis, and neurotmesis. In neurapraxia, there is conduction loss without structural change of the axon, and recovery takes place within days or weeks. The conduction velocity, if initially slowed, is probably due to focal demyelination, but should return to normal with remyelination. In chronic entrapment, focal demyelination is due to mechanical forces, but there can be axonal damage. During a complete block, stimulation below the nerve reveals normal excitability. The degree of compression determines the severity of the conduction block, but not the recovery rate.⁵⁵

Axonotmesis results in axonal damage and loss of continuity with wallerian degeneration of the distal segment, followed by denervation-induced muscle atrophy.⁸⁶ Distal excitability is lost in 4 or 5 days, first at the neuromuscular junction and then at the distal nerve segment.⁸⁶ Regeneration occurs slowly at 1 to 3 mm per day.¹⁹

Neurotmesis occurs when the nerve is disrupted. This may happen in varying degrees where perineurium is preserved, but axons and supportive tissue are disrupted nonetheless. Regeneration results in a very poor return of continuity and requires surgical repair. Despite microsurgical repair, functional recovery remains poor based on the axon's ability to select the appropriate target to re-innervate.^{91,137} Conduction velocity may reach 60% in 4 years.⁵⁴ Persistent prolongation of distal latencies indicates a limited number of fibers distally reaching the appropriate target.

TABLE 1
Types of nerve fibers

Fiber Type & Diameter	Conduction Speed (m/sec)
A fibers: myelinated fibers of somatic nerves	5–120
muscle nerve	
afferent group	
I (12–21 μ)	
II (6–12 μ)	
III (1–6 μ)	
IV (C fiber)	
efferent group	
α motor neuron	
γ motor neuron	
cutaneous nerve	
afferent group	
α (6–17 μ)	
γ (1–6 μ)	
B fibers: myelinated preganglionic fibers of autonomic nerve	3–15
C fibers: unmyelinated fibers of somatic or autonomic nerve	
sC fibers: efferent postganglionic fibers of autonomic nerve	0.7–2.3
drC fibers: afferent fibers of the dorsal root & peripheral nerve	0.6–2.0

Testing. The laboratory testing in a patient involves the needle electrode examination and recording of muscles at rest and with varying degrees of voluntary contraction. Nerve conduction studies are performed at the same time and coordinated with the clinical examination.

The EMG study conducted while a patient is at rest reveals no spontaneous activity except for insertional activity from placement of needle electrodes, which may be monopolar or bipolar. Insertional activity may become prominent in denervation or acute myopathies. Fibrillation potentials are the action potentials of single muscle fibers and occur when the motor axon has been interrupted. They are biphasic or triphasic with initial positive deflection. Their duration is 1 to 5 msec and their amplitude varies from 30 to 50 μ V. There may also be associated sharp waves. These potentials appear in 2 to 4 weeks. In contrast, fasciculation potentials are polyphasic and associated with denervation caused by varying degrees of severity of pathological features, but involve a visible muscle twitch. These twitches originate from motor units. Myotonic discharges are trains of single muscle fiber action potentials.²⁷ Their duration is long and the amplitude and frequency may vary.⁵⁴

Abnormal results on EMG and the degree of denervation are reflected by the amount of spontaneous neurotonic activity. Hence, spontaneous muscle activity is a reflection of the degree of axonal loss. Additionally, voluntary contraction and recruitment of motor units assess denervation and reinnervation.²⁷

Nerve Conduction Studies

Nerve stimulation is performed using surface electrodes, usually made of silver plate (Fig. 1). The stimulating electrodes consist of cathode and an anode. The current flows between the two, and depolarization of the nerve occurs at the cathode, which should be placed closest to the recording site. Some physicians prefer a monopolar electrode as a subcutaneous needle electrode. Stimulation may be constant-voltage, in which the voltage is regulated and the current varies inversely to the impedance, or constant-current in which voltage varies according to impedance but a chosen current reaches the nerve.⁷⁰ Nerves may be stimulated antidromically or orthodromically (Fig. 2).

Stimulus pulses are of square-wave and variable duration. Intensity varies from 10 to 30 mA (100–300 V) and 0.05 to 2 msec duration. Diseased nerves may require longer duration pulse widths and up to 40 to 50 mA (400–500 V) stimulation intensity. Surface electrodes are generally used for recording both the nerve action potential and the compound MAP, generated by stimulating the motor axons. These are averaged, amplified, and displayed by using an oscilloscope with computer storage of data and retrieval.¹²⁹

The motor nerve conduction time equals the latency minus the time for nerve activation, neuromuscular transmission, and MAP. Conduction velocity (m/sec) is expressed by the formula distance between stimulation points/latency (proximal) – latency (distal) (Fig. 3).⁷⁰ This is performed by stimulating at different points along the nerve.

Sensory nerve fibers may be stimulated orthodromical-

TABLE 2
Sensory nerve conduction studies commonly available in the EMG laboratory*

Nerve	Stimulation Site	Recording Site	Normal Values†		
			AMP	DL	CV
median	wrist	fingers 1, 2, & 3	>20.0	<3.3	>50.0
ulnar	wrist	finger 5	>18.0	<3.0	>50.0
ulnar	forearm	dorsum, hand	>18.0	<3.0	
radial	forearm	dorsum, hand	>18.0	<3.0	NA
lateral antebrachial cutaneous	elbow	forearm	>16.0	<2.9	NA
sural	calf	lateral malleolus	>6.0	<4.4	NA
superficial peroneal	shin	lateral malleolus	>6.0	<4.4	NA
saphenous	shin	medial malleolus	>6.0	<4.4	NA

* Reprinted with permission from Youmans JR (ed): **Neurological Surgery**, ed 3. Philadelphia: WB Saunders, 1990. AMP = amplitude (μ V); CV = conduction velocity (m/sec); DL = distal latency (msec); NA = not applicable.

† Normal values are for young adults and vary from laboratory to laboratory. Some normal values in this table are obtained courtesy of the EMG laboratory, The Cleveland Clinic.

ly or antidromically and require amplification two orders of magnitude (10^2) greater than that of motor axons. Sensory latency consists of only the nerve activation and conduction time to the recording electrode. Sensory fibers with large diameters have lower thresholds and conduct faster than most motor fibers (Table 2).⁷⁰

Late responses (that is, latency responses longer than the initial muscle responses) may be recorded by stimulating a mixed nerve with large afferent fibers and recording in the muscle to receive the efferent response. This requires a low intensity stimulus and is called the H-reflex. It requires the monosynaptic reflex to be intact. This is commonly measured in the lower extremity by using the tibial nerve and recording in the soleus muscle. A stronger stimulus will antidromically stimulate the motor axons with a return volley to the muscle. This is the F-response (Fig. 4).¹²⁹

Abnormalities of motor nerve conduction when stimulating between two points are of three types: 1) reduced amplitude with normal to slightly prolonged latency; 2) increased latency with normal amplitude; and 3) absent response. In general, axonal dysfunction results in a reduction in amplitude and demyelination leads to prolonged conduction time.⁷⁰ The distinction between acute partial neurapraxia and early axonotmesis is revealed by a loss of response when stimulating the nerve below a lesion 4 to 5 days later. At this time, the distal axons have lost excitability in the latter type of disease (Table 3).

Entrapment syndromes of the upper extremity most commonly involve median nerve compression at the carpal ligament. There have been findings of prolonged distal motor latency to the abductor pollicis brevis muscle, as well as sensory abnormalities ranging from prolonged latencies to loss of amplitude on stimulation with a ring electrode on the index finger. Differences in peak latencies of 0.4 msec or more are considered abnormal.⁶⁰ The pronator syndrome occurs when the median nerve is compressed between the two heads of the pronator teres muscle. Motor and sensory testing may not always be helpful, but EMG studies may show denervation of abductor pol-

TABLE 3

*Motor nerve conduction studies commonly available in the EMG laboratory**

Nerve	Stimulation Site	Recording Site	Normal Values†		
			AMP	DL	CV
median	wrist, elbow, axilla, SCF	thenar region	>6	<3.9	>51
ulnar	wrist, elbow, axilla, SCF	hypothenar region	>8	<3.0	>51
ulnar	wrist, elbow, axilla, SCF	first dorsal interosseous muscle	>8	<3.8	>51
radial	elbow, spiral groove, SCF	extensor digitorum communis muscle	>6	<3.0	NA
musculocutaneous	axilla, SCF	biceps muscle	>4	<3.5	NA
axillary	axilla, SCF	deltoid muscle	>4	<4.8	NA
peroneal	ankle, fibular head, popliteal fossa	extensor digitorum brevis muscle	>3	<4.8	>41
peroneal	fibular head, popliteal fossa	tibialis anterior muscle	>4	<4.0	>41
posterior tibial	ankle, popliteal fossa	abductor hallucis muscle	>6	<5.6	>41
femoral	inguinal groove	rectus femoris muscle	>4	<6.0	NA
facial	mastoid foramen	facial muscles	NA	NA	NA

* Reprinted with permission from Youmans JR (ed): **Neurological Surgery**, ed 3. Philadelphia: WB Saunders, 1990. SCF = supraclavicular fossa. For a more in depth analysis, see Table 5-2 in Kimura, 2000.

† Normal values are for young adults and vary from laboratory to laboratory. Some normal values in this table are courtesy of the EMG laboratory, The Cleveland Clinic.

licis brevis or flexor pollicis longus muscles.¹¹ Sensory findings in the thenar eminence occur in contradistinction to carpal tunnel syndrome. Anterior osseous nerve compression just distal to the pronator teres will affect the flexor pollicis longus, pronator quadratus, and the radial side of the flexor digitorum profundus muscles. Conduction velocities will be normal, but EMG studies will be positive in the aforementioned muscles.

The ulnar nerve is most commonly injured at the elbow where it is superficial in the olecranon groove. Motor conduction velocities measured above, across, and below the elbow reveal a slowing across the elbow (Fig. 5). Patients with motor conduction velocities higher than 40 m/sec may be watched for 6 to 8 weeks, but those with velocities less than 30 m/sec should undergo surgery promptly.⁹⁷ To a lesser extent the ulnar nerve may be entrapped at the wrist or in the Guyon canal. Distal latencies and EMG studies may be abnormal.²

The radial nerve is commonly involved in the spiral groove and supracondylar portion of the humerus as well as in the forearm proximal and distal to the supinator muscle. Motor conduction studies are performed in the forearm and sensory testing is possible by using the superficial radial nerve. Conducting EMG studies of selected muscles innervated by the radial nerve is helpful in localizing the site of compression.¹⁵ Less afflicted nerves in the upper extremity at the shoulder girdle are axillary, musculocutaneous, dorsal scapular, suprascapular, and long thoracic nerves. The presence of abnormalities on EMG of the muscles innervated by the specific nerve, but the absence of paraspinal muscle changes can aid in the diagnosis.³⁶

Thoracic outlet syndrome involves the lower trunk of the brachial plexus, made up of C-8 and T-1 roots, which are compressed by the cervical first rib. Electromyographically demonstrated abnormalities occur in the hand muscles, and sensory conduction from the fifth finger compared with the index finger will show delayed latencies or a reduction in amplitude. Supraclavicular motor stimulation and recording in distally innervated muscles and, more recently, evoked response monitoring together with

recording distally at the Erb point have been found to be helpful.⁹⁸

Entrapment Syndromes in the Lower Extremity

Entrapment syndromes in the lower extremity occur less frequently, but are more likely to be acutely traumatic. The femoral nerve may be injured proximal or distal to the inguinal ligament. Motor and sensory testing by using the saphenous nerve are available. An electromyographically recorded sampling of multiple muscles in the legs may be required to determine involvement of the lumbar plexus.¹³⁸

The sciatic nerve is commonly injured in the gluteal region often by penetrating injury or posterior dislocation of the hip joint. Direct stimulation with the aid of needle electrodes, H-reflex stimuli, and EMG studies facilitate diagnosis and localization of injury to this nerve.¹³⁸

The peroneal nerve enters the anterior compartment of the calf by passing from the popliteal fossa around the neck of the fibula. It is here that the nerve is usually compressed. A reduction in the combined MAP of the extensor digitorum brevis muscle or prolonged latency when stimulating in the popliteal fossa and below the fibular neck and recording distally, coupled with selective EMG studies is helpful in making the diagnosis of peroneal nerve injury at the fibular neck region.¹³⁸

The posterior tibial nerve may be injured in the distal popliteal fossa or chronically compressed in the tarsal tunnel. This compression syndrome can be identified electrically with prolonged latencies of conduction across the retinaculum of the medial malleolus to the abductor hallucis muscle or digiti quinti pedis. Additionally, there will be abnormalities of the intrinsic muscles of the foot on EMG studies.¹⁵³

Presently, MR imaging with the aid of T₁- and T₂-weighted short tau inversion recovery is a useful tool in the evaluation of peripheral nerves.⁵⁸

Cranial nerve testing, particularly that of cranial nerves seven and eight, will be discussed in a separate section.

Cervical and Lumbar Radiculopathies

Cervical and lumbar radiculopathies less commonly require the use of electrodiagnostic studies with the advent of high-quality MR imaging and CT myelography. Nonetheless, separating radiculopathy from more peripheral pathological entities, or assessing which nerve root is the cause of a patient's symptoms may require EMG. Motor and sensory amplitudes are normal, and denervation abnormalities are present in two paraspinal muscles and two extremity muscles common to a cervical or lumbar nerve root. The hand F-response or S-1 root H-reflex may be abnormal.¹⁰⁹

Plexopathies, particularly the brachial ones are commonly affected by trauma (motor vehicle accident, gunshot wound). With this disease entity a combination of EMG-demonstrated abnormalities—reduced sensory amplitudes with normal paraspinal EMG—is found. The additional use of supraclavicular motor stimulation and evoked response testing may be helpful. Intradural root avulsion will have preserved sensory responses. A similar workup for lumbosacral plexopathy is performed.¹⁵²

Polyneuropathies are characterized by symmetrical distal motor and sensory deficits. Motor and sensory velocities and amplitude may be reduced with distal extremity muscle abnormalities on EMG.¹⁵³

Myopathies may have reduced amplitudes of the combined MAPs. With successive stimulation, further decreases occur in amplitude. Motor conduction velocities are normal and EMG-demonstrated abnormalities reveal reduced recruitment and fibrillation.¹⁵⁴

The recent development of magnetic stimulation of the cerebral cortex will be helpful in spinal cord and peripheral nerve diagnoses.¹⁰⁸

ELECTROENCEPHALOGRAPHY STUDIES

Background and Technology

Electroencephalography is a noninvasive method of visualizing the ongoing physiology of the brain by recording the potential differences between two points, one or both of which are located on the scalp. The recorded signals are amplified and displayed, reflecting the movement of electrical charges in the brain.

The electroencephalogram in humans was first used in 1924 by Hans Berger, a professor of psychiatry at the University of Jena in Germany. His first records involved subcutaneous electrodes placed in the area of skull defects, and his first publication appeared in 1929.⁴¹ Several years later he was able to use a galvanometer to amplify these signals. Unfortunately, this significant discovery was unrecognized until 1934, when Adrian and Matthews confirmed the value of his work.³ Gibbs and Lennox were the first to describe the characteristic changes of epilepsy on EEG.²⁰ Also, in 1934 Jasper⁶² started his work on the explanation of the origin of cortical rhythms demonstrated on EEG and their regulation by the thalamus.

Recorded EEG voltages represent the summed postsynaptic potentials from large populations of cortical neurones, which are activated by thalamocortical input. These potentials act as a dipole, with a negative and a positive pole. They are created by the flow of the current (electri-

cal circuit) as a result of postsynaptic input at different points along the neurone (Fig. 6). Biological tissues are three-dimensional electrical conductors and are therefore volume conductors. The potentials are by and large generated by cortical neurones lying parallel to the surface of the scalp so that the single dipole is oriented at a right angle to the cortical surface. Thus it follows that electrodes placed anywhere within the volume will record a potential difference with current flow. Only if the two electrodes lie on the same isopotential line with regard to the current flow will they fail to record a potential difference (Fig. 7).²³ With this in mind, electrodes are placed on the scalp to take advantage of symmetrical coverage in the sagittal and coronal planes (Fig. 8).⁶⁴ Recordings are performed using a bipolar technique in which potential differences are measured between adjacent electrodes in a chain, or potentials at all points are measured against a common reference. The former relies on the localization of a generator on a phase reversal occurring at the same electrode in two montages that are at right angles to each other; in the latter procedure, reliance is placed on amplitude measurements. With monopolar recording, the highest amplitude would be recorded at the F₇ electrode (Fig. 9).

To understand the EEG machine and its recordings, some basic terminology in electrical measurement must be advanced. One coulomb (C) is equal to the charge of 6×10^{18} electrons. One ampere (A) of current is equal to the flow of one coulomb of charge per second (1 C/sec). Current flows from positive to negative potentials, and one joule (J) of energy is expended when one coulomb of charge is moved across a potential of 1 V. Voltage (V) is measured between two points and is the electromotive force. Resistance is measured in joule-seconds per coulombs-squared, or ohms (Ω). One ohm is the resistance (R) that will dissipate one joule of energy when 1 ampere of current (I) flows for one second (Ohm's law is $V = I \times R$).

A capacitor is a device that stores separate charges. It has two conducting plates situated close together but separated by an insulator. The movement of charges causes a current to flow, but not across the plates. An inductor is made of coils of wire around a magnetic field that is generated by current in the wire itself. The number of turns of wire increases the magnetic field strength of the current and is used to increase the induced voltage. The power source is expressed in joules per second or watts or voltage, and amperage.²³

Originally, vacuum tubes were the active circuit elements used to record and amplify small potentials; now, semiconductors transistors and integrated circuits are used. Note that impedance is a combination of resistance, capacitor reactance, and inductive reactance. These usually can be ignored on EEG.

The EEG machine has 16, 20, 24, 32, or 64 channels for recording output to an oscilloscope, paper, or digital reformatting (Fig. 10). Surface electrodes are usually made of silver or silver and silver chloride; subdermal electrodes usually consist of platinum. The scalp electrodes record potentials with the aid of conductive paste sealed with collodion (Fig. 11). Distal electrodes plug into a jackbox labeled in accordance with the international 10–20 system of electrode designation.^{18,64} From here, inputs are carried

to the montage (or the specific electrode recording arrangement on the scalp) selector board of the EEG machine.⁴⁰ From that point, the EEG signals (μV or mV) are amplified by preamplifiers. The amplification factor is called a "gain." Sensitivity is measured in micro- or millivolts per centimeter, and defines the amount of voltage needed to deflect the EEG pen a given distance. Typical sensitivities used are between 7 and 10 $\mu\text{V}/\text{mm}$. Filters are used to attenuate and discard a low frequency (< 0.5 Hz), and there is a high frequency cutoff of 70 Hz. In addition, a 60-Hz notch filter is required to remove electrical noise. Grounding the patient and machine through the EEG machine is important to prevent ground loops of current between a patient and the machine ground.⁸⁵

Electroencephalographic Patterns

Normal Patterns. Normal electroencephalographic patterns vary with age and level of consciousness.⁵⁶ The following patterns discussed refer to those in an adult. The occipital/alpha rhythm is a bilateral, symmetrical, posterior 8- to 13-Hz pattern with amplitudes of 20 to 60 μV . It is recorded in an awake patient with his or her eyes closed (Fig. 12); it is attenuated by opening of the eyes. Slower rhythms without mixed fast rhythms are abnormal.⁸⁷ The central rhythm pattern seen in an awake, relaxed patient is 7 to 12 Hz and has a comb-and-wicket pattern because of the rounded positive phase and the sharp peak to the negative phase. The amplitude is less intense than that of the alpha rhythm. The rhythm is bilateral, but may be asymmetrical, and is sustained or intermittent. It is attenuated by touch and movement of an extremity, an effect that may be bilateral. At times a more rapid, lower voltage may be seen.⁷² The pattern in the frontocentral area consists of varying frequencies, but resides in the β range (> 13 Hz). The frequency bands may be 18 to 25 Hz, less commonly 4 to 16 Hz, and rarely higher than 35 Hz.⁷² Temporal patterns may reveal varying rhythms: posteriorly in the α range and anteriorly, particularly with drowsiness, at ranges of 5 to 7 Hz.¹⁶

Sleep has four recognizable stages on EEG. Stage 1 occurs during drowsiness when there is a dropout of the α rhythm and the presence of vertex sharp waves. Rapid eye movement sleep, as revealed on electroculograms, occurs in the near-awake state. Slow and β rhythms may alternate. Stage 2 begins as drowsiness deepens and is characterized by spindling at 12 to 15 Hz in the parasagittal area, vertex slow sharp waves, and K-waves that are high-voltage slow waves, followed by a spindle at the vertex (Fig. 13). Stage 3 happens as sleep deepens, slower rhythms occur, and there are fewer K-waves. Stage 4 is a deep sleep and is characterized by delta (2–4 Hz) waves. Sleep, sleep deprivation, hyperventilation, various barbiturate agents, and photic stimulation may be used to activate or accentuate normal and epileptic patterns.¹⁶

Abnormal Patterns. Abnormalities on EEG are indicated by the alteration, attenuation, or disappearance of normal rhythms and the appearance of abnormal rhythms with or without significant changes in normal rhythms. The interpreter of EEG results should first describe the normal and/or dominant background activity in an individual patient. Frequency is measured in Hertz or cycles per second. Mean amplitude should be estimated, and re-

sponse to eye opening and movement should be noted. Next, the abnormalities that do not form part of the background activity are described, including spikes, sharp waves, slow waves, and their amplitude. Furthermore, one must determine whether these abnormalities are focal or symmetrical, synchronizing or episodic. Algorithms exist to quantify slow waves or spikes and their location.⁹⁹ A description of the activation procedures and their results should follow. Third, an opinion should indicate whether the record is abnormal and, if so, to what degree. Last, clinical correlation should be determined.¹⁷

Polymorphic δ (2–4 Hz) waves on EEG are the most common sign of either structural or metabolic pathological features in the brain, although in diffuse gray matter disease there may be paroxysmal slow-wave discharge. The former indicates deafferentation of cortex from thalamus.⁴² Focal lesions such as a neoplasm or an abscess are associated with focal δ waves and desynchronization of background activity. With cerebral infarction, loss of focal activity and PLEDs may occur. Subdural hematomas may produce focal or diffuse slow-wave activity and sometimes reduction in amplitude ipsilateral to the clot.¹ Head injuries produce, based on the level of a patient's consciousness, slowing and disorganization of background activity. Focal damage will produce suppression of activity regionally or hemispherically. Late findings include slow-wave and epileptiform abnormalities.²²

Criteria for the diagnosis of brain death on EEG requires a specific number of electrodes, their resistance and spacing, reduced sensitivities, attention to recording of extracerebral sources, time constants, and length of recording.^{149,158}

Epilepsy and EEG

Epilepsy is a disorder of hyperirritability of cortex and/or thalamus. Epileptiform abnormalities are the most common pathological features requiring electroencephalographic evaluation. Interictal (the time between clinical and electrographic seizures) abnormalities may be recorded by EEG. They consist of spikes (20–70 msec duration), sharp waves (70–200 msec duration), spike and slow-wave complexes, paroxysmal slow waves, and PLEDs. Sharp wave and spikes are asymmetric, with a shorter upswing than fall of the pen. Their occurrence may be transient, occurring singly or in trains at varying frequencies. They should be paroxysmal and distinguished from background activity. There must be an abrupt change in polarity to give them their sharpness. Their duration must be less than 200 msec. The interictal discharges must have a physiological field (Fig. 14).^{17,89}

Ictal (the time during a seizure) EEG activity is characterized by abnormal waveforms and patterns. It is variable in duration, but evolves over time and location. Patients are traditionally monitored by video, and abnormal behavior associated with electrographic changes should occur at the same time as or after the electroencephalographic changes to be caused by these electrical abnormalities, that is, a clinical seizure. Electrographic seizures may occur without a clinical change in behavior, however.¹⁰⁶ The EEG and video monitoring is time-locked to behavior and electrical signals with the aid of an automatic spike or seizure detection algorithm that saves and plays back the previous 2 minutes and the length of the event.^{43–45}

In the neonatal patient, ictal waveforms may be variable and tend to evolve. They may be focal or multifocal and associated with PLEDs, burst suppression, poor background, and high-voltage delta waves. These latter interictal and background findings indicate structural damage to the brain.^{68,69,94}

The present general classification of seizures characterizes them as partial or generalized. Partial seizures may be simple (consciousness not impaired) or complex (consciousness impaired). In the former, ictal and interictal abnormalities are confined to the contralateral hemisphere. In the latter, discharge is unilateral or frequently bilateral and its onset may be obscured by muscle artifact or remoteness of scalp electrodes from the generator. Interictal abnormalities may be unilateral or bilateral and usually have an asynchronous focus.¹⁴⁸

Generalized seizures are associated with the initial involvement of both hemispheres, consciousness may be impaired, and this impairment may be the initial symptom. Motor involvement is bilateral. Ictal changes on EEG are also bilateral. Interictal recordings may be normal in the genetic/idiopathic group, and the secondary group will have associated interictal and background abnormalities. Brief absence seizures have 3-second spike-and-wave discharges. An atypical absence of seizures may demonstrate an irregular pattern with interictal background changes. Myoclonic seizures have polyspike-and-wave ictal change. Clonic seizures are characterized by fast activity and slow waves. Tonic seizures have low-voltage fast activity. Tonic-clonic seizures have a rhythm of 10 Hz followed by a slow wave, and atonic seizures have polyspike-and-wave discharges.^{29,133}

Electrocorticography involves obtaining the EEG recordings directly from the brain surface. This is performed by placing electrode grids or an array of electrodes with cotton wicks or carbon balls (Fig. 15). Amplitudes are such that sensitivities must be increased. Usually interictal data are recorded. Stimulation with after-discharge to delineate seizure focus has limited value. After-discharge may occur from less than 1 to 90 msec after stimulation.¹¹² Activation with thiopental or methohexital may be used. The value of ECoG lies in tailored resections of the temporal lobe and lesional extratemporal resection.^{80,88}

Intracranial recordings obtained to delineate a seizure focus, that is, lateralization and localization, may best be performed preoperatively with chronic video-EEG recording by using the appropriate depth or strip and grid electrodes. The onset of focal medial temporal lobe seizure activity is characterized by attenuation in background amplitude and a rapid buildup of low-voltage beta rhythms evolving to 18- to 25-Hz high-amplitude discharges. A more regional onset may have attenuation in background followed by rhythmic 4- to 12-Hz discharges. In the frontal lobe and extratemporal cortex, a change in interictal activity followed by a buildup of polyspike or spike-and-wave discharges can occur.^{76,132} Postictal attenuation and slow-wave activity may be lateralizing.¹¹⁸

MAGNETOENCEPHALOGRAPHY

Magnetoencephalography has developed during the last decade.¹⁰⁷ It is a noninvasive equivalent to EEG and pro-

vides better spatial delineation of surface electromagnetic data, particularly that which is brain activated, that is, evoked response signals.¹²¹

To record the magnetic fields induced by electrical activity of the brain requires an instrument sensitive to the signals and their frequency. The instrument presently used is a superconductivity quantum interference device, or SQUID magnetometer.³³ It measures small variations in magnetic flux that gives rise to an electrical current (Josephson effect).⁶⁵ The Biot-Savart law states that a magnetic field due to a small electric current, such as that from cerebral neurons, varies as the inverse square of the distance of the current source and directly with the current and the sine of the angle between the directions of the current and the vector leading to it. The cerebral magnetic field generated is on the order of 10–12 t, far less than the earth's magnetic field.¹²¹

The components of the SQUID consist of a detection coil that senses the magnetic field and transforms it into an electrical current that becomes the signal of the field, and an input coil that transforms the resulting current into a magnetic flux. The superconductivity current is then influenced by the magnetic flux. The ring may be biased at one weak link (Josephson effect) by a radiofrequency current or by a direct current in two weak links (Fig. 16). The signal is improved by further electronic manipulation and then stored for display and further analysis. The SQUID amplifier and detection coils are superconductivity devices and require some type of refrigerant (liquid helium). The SQUID ring is made of niobium and is cooled below 23°K when immersed in a bath of helium at 4.2°K (Fig. 17).¹²¹ The SQUID is then housed in a Dewar that insulates the outside ambient temperature from the super coolant. In addition, it is transparent to magnetic fields (Fig. 18). Magnetic and eddy current shielding is mandatory. Originally, by increasing the number and type of detector coils, only seven channels were available for recording. This first instrument had to be moved around the patient's head for recording; with an increasing number of channels, the instrument required shifting from one hemisphere to the other.^{21,71}

Both EEG and MEG record electromagnetic signals in the range of milliseconds, whereas neuroimaging, which depicts anatomy, and positron or single-photon emission CT scanning, which measures blood flow or metabolism, record time in a range of minutes. The electrical potentials measured on EEG are volume potentials and are altered by dura mater, skull, and scalp. Magnetoencephalography is not influenced by these structures. Whereas the EEG measures a potential difference, MEG measures an absolute value that is perpendicular to the electrical current. This indicates that the magnetic fields are generated from an intracellular compartment. Furthermore, MEG measures the tangential dipole (Fig. 19).⁶⁷ Using a spherical head model, EEG or evoked response source will be rotated 90° to the magnetic field. The combined use of MEG and closely spaced electrodes for EEG will result in greater knowledge of both the tangential and radial components of the physiological signals (Fig. 20). On balance, MEG has fewer problems with cortical spatial localizations, but remains problematic with deep sources because of quick decay of the magnetic field.¹²¹

Laboratory data on the localization of action potentials in peripheral nerve physiology have provided insight into the spatial domain of the area of depolarization. The magnetic dipole is founded on the right hand rule that states that the fingers lie in the direction of the magnetic flow and the thumb points to the direction of propagation. Using room temperature sensors (toroids) that surround the nerve, investigators have studied postinjury regeneration. The nonuniform propagation has its clinical counterpart in the neuroma-in-continuity in humans.¹⁵¹

In neurological studies, MEG can reveal spreading depression in migraine attacks¹⁰⁴ and polymorphic delta waves in cerebral infarction,¹⁴⁶ but its special promise resides in magnetic source imaging⁴ and its combined use with EEG in seizure detection.^{137,139} In the latter use, MEG requires fewer correction factors, but EEG detects more basal current orientations and allows for chronic monitoring. Although MEG is more expensive, it provides a simpler picture; the reverse is true for EEG. In patients with nonlesional temporal lobe epilepsy MEG may help differentiate medial compared with lateral onset and spatial relationships between the lesion and irritative zone (Fig. 21). In patients with neocortical epilepsy MEG seems to be more sensitive than EEG.⁷ Furthermore, the merging of MEG and EEG with MR imaging will help delineate areas of epileptic activity and their relationship to lesional abnormalities and functionally important areas.^{121,136}

Using event-related stimuli and MEG, colocalized with MR imaging, new insight has been gained into motor, speech, visual, and auditory physiology. More recently these same techniques have been used to identify neural plasticity (Figs. 22 and 23).⁵²

EVOKED POTENTIALS

Evoked potentials are gross potentials extracted from EEG studies by the averaging of stimulus-locked signals. They are the CNS's response to a sensory stimulus delivered peripherally.¹³ The use of this methodology for the diagnosis of acute and chronic pathological features along the neuraxis, including cognitive deficits, is based on the original findings of Dawson²⁴ and substantiated in laboratory animals by Donaghey and Numoto.²⁸ The potentials complement EEG studies and coupled with MEG are co-registered by fiducials to MR imaging for source localization. Neurosurgeons most commonly use SSEP monitoring in posterior fossa and spinal surgery, the former being accompanied by BAEPs. Visual evoked potentials are used in selected intracranial surgeries around the optic chiasm and in prelesion recording during pallidotomy to avoid the optic tract.¹⁴³ In performing this type of electrophysiological testing, the pathway for the stimulus to enter must be preliminarily evaluated, that is, vision, hearing, and peripheral nerve/spinal cord function. Evoked potential monitoring differs from EEG in that the signals have a low amplitude (μ V) and the averaging of a large number of trials must be performed to obtain good waveform resolution.

The following discussions of visual, brainstem auditory, and somatosensory evoked response protocols are based on the American EEG Society guidelines.³⁸

Visual evoked responses are a result of patterned or unpatterned stimuli. The latter are used in patients unable

to fixate on the stimuli. Additionally, flash stimuli are used with goggles, particularly in the operating room. Response wave peaks are N75, P100, and N145. The P100 is the most reliable response and considered the standard recording. Between four and 10 stimuli are presented per second, with a luminance of 8.2 candela/m.²⁴ The visual angle will dictate macular vision or a larger portion of retina stimulated. Typically, a 32° field with a 50-minute arc check is a large field. The patient is no closer than 70 cm to the stimulus screen. Analysis time is 250 msec, with a system bandpass of 1 to 100 Hz. The P100 latency should replicate within 2.5 msec, with 100 to 200 stimuli per response. The recording electrodes are placed left, middle, and right occipital to midfrontal and a fourth channel is situated midfrontal to ear/mastoid (Fig. 24). Latency criteria, either ipsilateral or interocular, should not exceed 2.5 to 3 SDs higher than the mean. Criteria for an abnormal amplitude are its absence, its marked reduction, or an abnormally high interocular ratio. Clinically, the P100 peak latency and amplitude are the most useful measurement in half field patterned stimuli. At the time of testing an electroretinogram (electrode periorcular and one scleral) can be obtained to differentiate retinal from optic pathway pathological features.

Brainstem auditory evoked potentials consisting of five peaks are to be differentiated on the electrocochleogram. There is some controversy regarding the second peak of the BAEP, that is, whether this is an action potential of the auditory nerve (Fig. 25). The convention has been that the peaks occurring over 10 to 15 msec localize peak 1 to the eighth cranial nerve; peak 2, to the cochlear nucleus; peak 3, to the superior olivary complex; and peaks 4 and 5 merge to the lateral lemniscus and inferior colliculus. The stimulus is created with broad-band clicks generated from 100-Hz rectangular pulses by using a standard audiometric speaker. For intraoperative use, small ear inserts can be used. Stimulus rates between 8 and 10 per second are best suited for delineating the various peaks. A bandpass filter 10 to 30 and 2500 to 3000 Hz, and 12 to 24 dB octave for the low and high frequencies, respectively, are used. Results of 1000 to 4000 trials should be averaged to obtain good waveform for 10 to 15 msec. Electrodes are placed at Cz and at each earlobe or mastoid. Criteria for clinically significant abnormalities are latencies of peak, inter-

TABLE 4
Normative latency values of BAEP*

90 dB Above SL clicks (msec)		Interwave latency (msec)	
Wave	Mean $\pm$ SD	Waves	Mean $\pm$ SD
I	1.88 $\pm$ 0.09	I–II	1.19 $\pm$ 0.21
II	3.07 $\pm$ 0.17	I–III	2.14 $\pm$ 0.19
III	4.01 $\pm$ 0.16	I–IV/V	3.46 $\pm$ 0.27
IV/V	5.36 $\pm$ 0.21	I–V	4.09 $\pm$ 0.24
IV	5.23 $\pm$ 0.17	III–V	1.93 $\pm$ 0.26
V	5.93 $\pm$ 0.25	V–VI	1.60 $\pm$ 0.28
VI	7.50 $\pm$ 0.26		

* SL = sensation level. Reprinted with permission from Feblot P, Uziel A: Detection of acoustic neuromas with brainstem auditory evoked potentials. Comparison between cochlear and retrocochlear abnormalities, in Courjon J, Mauguire F, Revol M (eds): **Clinical Application of Evoked Potentials in Neurology**. New York: Raven Press, 1982.

peak latencies or amplitude ratios more than 2.5 to 3 SDs from the mean (Table 4). During surgery of the posterior fossa, either intra- or extraaxial, the absence of all waves or Waves IV and V or prolonged internal latencies of Waves I to III or Waves III to V is highly abnormal. Interaural comparisons are important. Testing of BAEPs is helpful outside the operating room in the critical care unit in evaluating patients with head injuries (Fig. 26) or when MR imaging studies are not clear in delineating invasive tumors of the brainstem.³⁴ Additionally, late evoked potentials on imaging studies have been used to evaluate cognitive disorders.¹¹⁵

Somatosensory evoked potentials are elicited by stimulating peripheral nerves transcutaneously by using a pulse width of 100 to 300 msec and the rate of 3 to 5 Hz. A pass bandwidth of 30 to 300 Hz is used. The recording should last 40 msec for the median nerve and 60 msec for the posterior tibial nerve. A designation of the components follows: EP is the propagated volley under Erb point, N13 is the stationary cervical potential recorded referentially from the dorsal neck, P14 is a subcortically generated far-field potential from scalp electrodes whose source is believed to be in the medulla, N18 is similar to P14 but localized to the brainstem thalamus, and N20 reflects activation of the cortical somatosensory area. Electrodes on the scalp are placed at C3 and halfway between P3 as well for C4 and P4, and referential recording areas. The absence of any obligate waveforms or the prolongation of interpeak latencies more than 2.5 to 3 SDs greater than the mean is abnormal (Fig. 27).

In the lower extremity, LP is a stationary lumbar potential, and N34 is a subcortical far-field potential generated from the upper brainstem, and P37 reveals activation of the primary sensorimotor cortex. Recording loci are set at T-12 as a reference and at other locations similar to those used with upper-extremity SSEPs. Criteria for abnormalities include the absence of obligate waveforms and the prolongation of the LP-P37 interpeak latency 2.5 to 3 SDs greater than the mean (Fig. 28).

Somatosensory evoked potentials are an important part of monitoring of the neuraxis in spinal cord or posterior fossa surgery (Fig. 29). Additionally, dermatomes may be a source of stimulus for entrance into the CNS. As with BAEPs, SSEPs may be a helpful tool in evaluating the comatose patient given that these potentials are not altered by coma or barbiturate agents (Fig. 30). Reversal of the N20-P20 has been used to identify the central sulcus, but one must be aware of the peculiar anatomy of the sulcus in each patient or the presence of a tumor appearing in the sulcus (Fig. 31).⁷⁵

ELECTROPHYSIOLOGICAL TESTING FOR INTRACRANIAL PROCEDURES

Posterior Fossa and Cranial Nerves

In performing intracranial surgery that requires prolonged dissection and retraction of vital centers and cranial nerves, intraoperative electrophysiological monitoring of these structures is mandatory to assist the surgeon in localization as well as in assessing the extent of the manipulation of these structures. Cranial nerves two to 12 can be monitored by evoked response potentials (visual

evoked potentials, BAEPs), triggered EMG, or a combination of the two. Except for cranial nerves two and eight, the remaining nerves can be monitored by compound MAP triggered by manipulation or direct nerve stimulation. The muscles innervated by a particular cranial nerve are recorded using small subdermal electrodes or surface electrodes around the orbit of the extraocular muscles. Methodology of evoked response monitoring has been discussed earlier in detail. Before embarking on evoked potential monitoring, testing of the visual apparatus for acuity, visual field perimetry, and color recognition should be performed. Likewise, the cochlear and vestibular system should be evaluated. The combination of SSEPs and BAEPs is helpful in assessing brainstem function during posterior fossa surgery; the use of SSEPs is also helpful in supratentorial surgery (Fig. 32).

Hearing may be evaluated with pure-tone audiometric testing in the range of 250 to 8000 Hz. Speech sounds are located in the middle frequency range of 500, 1000, and 2000 Hz.⁶⁶ Intensity is measured in decibels; zero is barely audible. The scale is logarithmic to the base 10. Hearing thresholds of 0 to 20 dB are normal; 20 to 40 dB indicates mild hearing loss; 40 to 55 dB indicates moderate hearing loss; 55 to 70 dB indicates moderately severe loss; 70 to 90 dB indicates severe loss; and 90 dB and higher indicate profound loss.¹⁴⁵ Speech reception is performed with two-syllable words at softer levels until only 50% of the words can be heard. This should approach pure-tone thresholds in speech frequencies. Speech discrimination involves the presentation of phonetically balanced words well above the speech-reception thresholds. The list contains 50 monosyllabic words. Patients with sensorineural loss will demonstrate decreased speech discrimination ability.^{66,144}

Through its peripheral neuroepithelial structures, the vestibular system signals the brainstem, cerebellum, and higher centers on the position of the head and retunes the body musculature. The otolith organs perceive linear acceleration, whereas the semicircular canals signal angular acceleration and deceleration. Normally, the bilateral system balances input to the CNS. The vestibular activation produces a deviation of the eyes (nystagmus) with a slow movement, whereas the central stimulus produces a rapid return. Abnormalities may be evaluated with electronystagmography testing by using surface electrodes laterally around the orbit and midline of the head. Spontaneous nystagmus with the patient's eyes closed or fixated and during positional changes and caloric stimulation may be performed.⁵⁰ The slow component is noted on the side of the destructive or cold-water irrigated ear (Figs. 33 and 34).⁸

The nerves most commonly monitored during surgery are cranial nerves seven and eight. In 1893 Krause⁷³ noted that low-current electrical stimulation of the seventh cranial nerve produced contractions in the facial muscles during sectioning of the eighth cranial nerve for tinnitus. In 1979 Delgado and colleagues²⁵ reported on the first series of patients who had undergone intraoperative EMG monitoring of facial nerve stimulation during cerebellopontine angle surgery. This procedure enables the surgeon to locate and preserve anatomically and functionally the seventh cranial nerve during tumor removal. The motor units of the facial muscles may be activated by electrical or me-

chanical stimulation, leading to a compound MAP between 0.1 and 2 mV. Subdermal needle electrodes are placed in the orbicularis oculi and oralis, nasalis, and mentalis. Spontaneous motor unit discharges and those triggered by direct stimulation are recorded. There are four basic types of spontaneous EMG responses.^{25,92,117,157} The first is a brief nonrepetitive polyphasic discharge from brief contact with the nerve (Fig. 34). Note, however, that sustained repetitive, asynchronous motor unit discharges (trains) are a result of nerve irritation that is usually a result of traction of the nerve fibers. Manipulation should be discontinued and warm saline irrigation performed. If the irritation is minor, the nerve quiets within 1 to 2 minutes. A highly traumatized nerve may produce a sputtering audio discharge. Clonic (burst and silence) or multiphasic responses of increasing and decreasing electrical activity are characteristic of impending nerve injury. Direct electrical stimulation is the most important method of assessing nerve location and integrity. Initially, one explores the capsule by using 2 mA of monopolar stimulation. If there is no response, surgery may proceed. Once the nerve is identified, the current is reduced to 0.05 to 0.2 mA. At the end of the procedure, thresholds for CMAP are established from distal to proximal directions along the nerve.¹²³ Anatomical preservation in most large series exceeds 90%, but physiological preservation may be 73%, depending on the size of the tumor.^{57,120,150} There is a correlation between threshold stimulation proximally being equivalent to the distal nerve; in these patients, 88% maintained function.¹⁵ The lower threshold paralleled the higher degree of function.^{96,128}

Cochlear nerve monitoring of action potentials in the setting of a large tumor is quite difficult or impossible, but may be performed in smaller tumors (Fig. 35).⁹³ Thus, from a practical standpoint, BAEP monitoring is more widely used. The BAEP consists of five or more peaks within 10 msec of high-intensity (80–100 db per sound level) repetitive clicks at a rate of 17 to 21 per second. The response is averaged over a number of trials (100–2000). Waves I, III, and V have the most clinical significance. In healthy volunteers the latency between Waves I and V is 4.5 msec.³² In small tumors, that latency may be greater than 5 msec, whereas large tumors may only show Wave I. During surgery, a change in amplitude, latency, or morphology represents insult to the nerve. Matthies and Samii⁹⁰ found that the loss of Waves I, III, and V was correlated with hearing loss; Wave III was sensitive and Wave V was specific. When choosing candidates for hearing preservation, patients should have hearing levels characterized by a speech reception threshold less than 30 dB and a speech discrimination sensitivity greater than 70%.¹⁵⁶ MacDonald, et al.,⁸⁴ reported an inverse correlation between extension into the fundus of the internal auditory canal. In addition, Ojemann found that extension of 0.5 to 18 mm into the posterior fossa correlated negatively.¹⁰³

Basal Ganglia

With the advent of cerebral stereotaxy in humans in 1947 by Spiegel and Wycis,^{134,135} localization with air encephalography (and later ventriculography) was still

somewhat inaccurate despite early brain atlases.^{122,141} Nonetheless, autopsy results in patients who had previously undergone stereotactic operations for Parkinson disease revealed adequate location and correlated with efficacy.⁵¹ Ventriculography has been abandoned for MR imaging or MR imaging/CT scanning fusion images.⁷⁷ Error remains, however, particularly in the z axis.¹²

Initially, macrostimulation was relied on to increase tremor and/or to reveal abnormal signs of suboptimal location of an electrode such as contralateral numbness or forced contraction of the upper extremity. This was performed using a lesioning electrode 1.1 mm in diameter with a 3-mm exposed tip (Radionics, Burlington, MA). This would reflect the nearness to structures not to be lesioned, but gave no indication of exact location of the area to be lesioned.

Impedance monitoring, which measures resistance to an electrical current, gives crude differentiation of cerebral structures of nearly the same density. In 1961 Albe-Fessard, et al.,⁵ reported the use of low-impedance microelectrodes to record evoked field potentials. Later, in 1968 Jasper and Bertrand⁶³ used high-impedance electrodes, but the signal-to-noise ratio was poor. Today, technology has facilitated more clean single-unit recordings. This has permitted mapping of the motor and sensory nucleus of the thalamus and now the globus pallidus and subthalamic area.⁶¹

Microelectrodes made of tungsten or platinum/iridium with a tip diameter of 2 to 4 μm and an impedance of 0.5 to 1 $\text{M}\Omega$ are advanced in protective stainless steel-guided tubes to 20 mm above the target. They are then advanced in small increments to the target by using a hydraulic drive. The signals are amplified and filtered (200–5000 Hz). They are displayed on an oscilloscope and audio monitor. These signals can be digitized and stored. Microelectrode stimulation is applied at 300 Hz, 0.2-msec width, 1 to 100 μA at 1- to 4-second trains.⁶¹

The Vim is a preferred site for lesioning or chronic stimulation in patients with parkinsonian, essential, or cerebellar tremor.¹⁰⁰ The target is one half the width of the third ventricle plus 11.5 mm, rostral to the posterior commissure according to the Guiot's quadrilateral space bounded by a line parallel to one half the height of the thalamus and the anterior commissure–posterior commissure line divided into twelfths (extending forward to the ventrooralis posterior), and 1.5 to 3 mm above the intercommissural plane (Fig. 36).^{47,142} Cell responses with spontaneous high frequency and amplitude, which may or may not be synchronous with tremor, are recorded. In addition, irregular tremor-independent cellular discharge can occur (Fig. 37). The Vim, anterior to the ventrocaudal nucleus (somatosensory) of the thalamus, has cells that are responsive to joint motion (kinesthetic cells; Fig. 38). The arm area is represented medial to the leg. Microelectrode stimulation produces paresthesia on the contralateral side of the body and suggests the electrode is posterior in tactile nucleus. Therefore, the electrode should be moved forward at least 2 mm. Evoked response recordings, particularly those obtained from afferent fibers (muscle spindle) are helpful (Fig. 39). Microelectrode stimulation of the internal capsule is performed to confirm the lateral boundary of a target.¹⁴²

Pollak and colleagues¹¹⁶ have popularized subthalamic stimulation for the disabling akinesia of Parkinson disease. The starting target is 12 mm lateral and 2 to 4 mm posterior to the midcommissural point and 3 mm below the anterior commissural–posterior commissural line. The cell responses recorded in the STN are high amplitude, with some variation in activity at 25 to 45 Hz. Firing of neurones can be modulated by passive or active motion. The arm is represented lateral to the leg. There is, on passing into the substantia nigra, a higher rate of firing at 60 to 90 Hz, which is more regular (Fig. 40).⁵⁹

During pallidotomy, the initial target is situated 20 to 22 mm lateral, and 2 to 3 mm anterior to the midpoint of the intercommissural line and 3 to 5 mm below the anterior commissural–posterior commissural line.⁷⁴ Characteristically, cells in the globus pallidus externus are of two types: one with low frequency (10–20 Hz) and the other with a higher frequency (30–60 Hz). Both are irregular and interrupted by pauses. The globus pallidus internus has neurones firing at a regular rate between 20 to 200 Hz (mean 80 Hz). Cells responsive to passive or active motion can be identified (Fig. 41).¹⁴⁷ The leg responses are represented, to some degree, more dorsally.²⁶ Tremor-synchronous cells have been identified.¹⁴⁰

Alternatively, semi-microelectrode recording with more sophisticated online computer analysis may be performed using Fourier transform, which is similarly used in locating neural generators in EEG studies.⁵³ Postlesioning recordings are obtained (Fig. 42). Furthermore, wavelet and complexity techniques coupled with clinical features can be incorporated into training a neural network to predict outcome and hazard for lesioning at any recorded locus along the electrode tract.⁴⁹

Functional Mapping

In this decade cortical localization has become a preoperative noninvasive methodology with intraoperative confirmation. This is due to the advances in computer imaging (functional MR imaging) and evoked responses to MEG- or EEG-triggered functional MR imaging.^{30,110} Transcranial magnetic stimulation has been developed during the past decade to be highly localizing and combined with neuroimaging to study connectivity of neural networks.¹⁰⁸ The pulse of the magnetic field passes through the cranium and induces an electrical current. The effect may be inhibitory or excitatory and similar to the effect of direct electrical stimulation as described by Penfield and Roberts.¹¹³ For localization of transcranial magnetic stimulation during EEG and/or MEG the recording positions on the scalp along with fiducial markers can be digitized. The fiducials can then be identified on subsequent MR images, and then coregistration of the results of the two studies can be performed.³⁷ Vision, speech, and motor activities can thus be mapped.

Functional MR imaging involves the activation of brain areas by using event-related techniques, magnetic stimulation, or naturally occurring events such as seizure or spike detection EEG to increased blood flow and change in blood oxyhemoglobin and deoxyhemoglobin levels. This method uses a T₂-weighted echo-planar imaging procedure that is sensitive to the blood oxygenation level-dependent contrast.¹³⁰ These techniques have been used for localizations of seizure focus and speech lateral-

ization in patients undergoing presurgical evaluation.^{35,131} Even with the advances in neuroimaging and manipulation of electromagnetic data, direct cortical stimulation in the awake patient remains the gold standard; however, using triggered EMG in selected muscles, the motor cortex can be mapped in an anesthetized patient (Fig. 43). The monumental work by Penfield and colleagues^{111,112} details this field. Motor mapping can supplement SSEP recording with subdural electrode strips or grids (Fig. 44). This may be correlated with neuronavigational techniques.⁹ In recent years Ojemann, et al.,¹⁰¹ have expanded our knowledge of cortical localization of language (Fig. 45). In a more practical way, Haglund, et al.,⁴⁸ reported that resection of brain and tumor margin extending to less than 1 cm of identified language cortex will, with a high probability, produce a postoperative language deficit.

Cortical or subcortical white matter pathway stimulation involves the use of an electrical generator to deliver biphasic current through monopolar or bipolar electrodes to the cortex for 4 seconds at 60 Hz (0.5– to 1 msec duration, voltage 1 V or 2 mA for initial threshold; Fig. 46). Identifying the motor cortex is accomplished by direct stimulation and the consequent production of motor movements. Applying stimulation to the premotor cortex may produce arrest of muscle activity or change in muscle tone. Furthermore, stimulation of the postcentral gyrus produces paresthesias in the contralateral limb or side of the body. Initially, the face area lends itself very well to localization of the sensorimotor area (Fig. 47).¹¹⁴ The current should then be increased to response or after-discharge levels and then reduced 2 mA below the after-discharge threshold. After performing face area localization, language cortical areas are identified by noting interruptions in speech, counting, or naming during stimulation.¹¹³ Speech arrest is more likely to be related to a language disturbance when it is associated with misnaming, preservation, confusion in number counting, or a retained ability to talk.⁸² Reading and memory areas may also be identified using similar techniques.¹⁰² Electroencephalography will have been performed first. Stimulation techniques for localization of an epileptic focus producing after-discharge (Fig. 48) are no longer used as often—given the more sophisticated imaging and EEG studies that can be performed preoperatively—but may indicate the epileptogenic region. Interictal epileptiform sharp waves or spikes are noted; based on their location and type of surgery to be performed, the areas they encompass are resected, particularly in the frontal and extratemporal cortices. The value of postoperative ECoG is dictated by the cortices involved and perilesional cortex.^{10,31,118}

SPINAL CORD MONITORING

Spinal cord monitoring is commonplace today, particularly in complex spine cases, for example, those requiring scoliosis, decompression, or reconstructive spine surgeries. Circumstances that require monitoring include impending or actual neurological deficit or endangered vascular supply to the spinal cord. The techniques available for assessing nerve root and spinal cord function are monitoring of SSEPs, dermatomal evoked potentials, triggered EMG, and motor evoked potentials.

Somatosensory evoked potentials reflect posterior col-

umner conduction. Neural structures of stimulation include the posterior tibial, median, and ulnar nerves. The latter is unique in its limited entrance into the spinal cord and role as an effective monitor of the brachial plexus in the patient while in a particular position during surgery.¹²⁵ Spinal cord compression may be increased by turning, positioning, or extending the neck (Fig. 49).¹²⁴ The SSEPs are sensitive to the inhalation of anesthetic agents; thus the use of narcotics such as fentanyl, propofol, and midazolam are appropriate.

Monitoring of nerve roots can be performed by stimulating a dermatomal field. It is most effective when undertaken in patients with recent nerve root compromise so that with decompression, improved amplitudes may be seen. Chronic nerve deficits or acute surgical nerve root injuries will not reflect change with adequate decompression. Furthermore, spinal cord deficit may prevent entrance of afferent volleys. Triggered EMG is helpful in revealing intraoperative nerve root injury from pedicle screw placement. The screw may be stimulated and the CMAP is recorded. If the screw encroaches on the nerve, a CMAP will be recorded at low thresholds (< 5 mA).⁷⁸ Alternatively, the muscles innervated by the nerve root can be recorded on EMG. An irritated nerve will be reflected by CMAP with a high reliability.¹⁰⁵

More satisfactory monitoring of the ventral portion of the spinal cord can be achieved using transcranial electrical or magnetic stimulation, which induces an electrical current within cerebral tissues of the motor cortex and activates the corticospinal tract.¹⁴ The CMAP will be recorded at the tibialis anterior muscle (Fig. 50).⁴⁶ This method of recording has been evaluated in laboratory animals.⁹⁵ From a practical standpoint, transcranial electrical stimulation is preferred by using constant-voltage or current stimulators with a single or multiple pulses lasting as long as 0.5 msec. Note that side effects occur rarely.⁸³

Last, a decision algorithm based on neurophysiological monitoring during complex spine surgery is a useful decision-making tool (Fig. 51).¹²⁶

CONCLUSIONS

A basic knowledge of neurophysiology permits understanding of electrophysiological testing as well as its normal and abnormal values. The evolving fields of computer science and electrical engineering have facilitated new methods of cortical stimulation (transcranial magnetic or electrical, magnification and stimulation of evoked signals, and source localization). The latter signals can be coregistered with CT images. Thus, preoperative mapping may be useful in strategic surgical planning and may be correlated with intraoperative pathological features and mapping. Evoked potential monitoring coupled with central and peripheral evoked motor stimulation has led to a higher degree of safety in patients undergoing intraspinal and posterior fossa surgery. Intraoperative recording stimulation facilitates MR-directed placement of therapeutic electrodes in basal ganglia, the thalamus, and the subthalamic region. We await further advances, particularly in source imaging.

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