Johannes Schramm · Aage R. Møller (Eds.)

Intraoperative Neurophysiologic Monitoring in Neurosurgery

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Professor Dr. JOHANNES SCHRAMM
Neurochirurgische Universitätsklinik
Sigmund-Freud-Str. 25, W-5300 Bonn 1

AAGE R. MØLLER, Ph. D.
Professor of Neurological Surgery
Department of Neurological Surgery
University of Pittsburgh School of Medicine
9402 Presbyterian-University Hospital
230 Lothrop Street, Pittsburgh, PA 15213, USA

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List of Contributors

You will find the address of each first mentioned author at the beginning of the respective contribution

Preface

Wide spread interest has developed in the last few years in the techniques of intraoperative neurophysiological monitoring, and in many centers use of these techniques has become a routine practice in neurosurgery, in otologic, and orthopedic surgery.

At the same time, many other centers have tried to introduce these techniques into their routine and have found it difficult at times to determine which techniques provide results reliable enough for routine monitoring and which require caution in interpretation of results, of perhaps further research, to be useful for monitoring.

The goal ot the International Symposium on Intraoperative Neurophysiologic Monitoring held in Erlangen was to further delineate the true value for daily clinical practice of the neurophysiologic monitoring techniques used in neurosurgery. The purpose of the Symposium was to bring together clinicians – i.e the neurosurgeon or otological surgeons – who might profit from the applicaton of these techniques with their neurophysiological colleagues, who in many countries have the responsibility of supplying intraoperative monitoring services to the clinicians. It was hoped that at the Symposium those neurophysiologists who are interested in introducing such monitoring service at their institutions either on their own initiative or in response to a request by clinicians at their facilities, would gain up-to-date information on the field. This Symposium was therefore planned to bring together specialists from the two fields 1) to assess the current status of neurophysiological monitoring techniques in neurosurgery and otology and 2) to draw a line and strike a balance on what has been achieved to date. Therefore, the present volume should provide a newcomer to the field of intraoperative neurophysiological monitoring the opportunity to learn what has been established in the field and what has still to be further developed.

Because this meeting was held on a small scale with invited speakers from well-known centers only, these articles truly reflect the current status of intraoperative monitoring.

It is our hope that this book will appeal to both surgeons and neurophysiologists, and to those who use intraoperative monitoring of evoked potentials and those who wish to introduce these techniques.

Bonn and Pittsburgh
J. SCHRAMM and A. R. MØLLER

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Completion of this volume would not have been possible without the special help that we have received from two very skilled and talented individuals. Diana Mathis meticulously edited each chapter and Cleat Szczepaniak transcribed and organzied the various chapters into a form that was suitable for typesetting. We also want to thank Dr. Taniguchi for preparing the Subject Index. The Department of Neurological Surgery, University of Pittsburgh School of Medicine, and the Department of Neurosurgery, University Hospital of Bonn, Bonn, provided economic resources for the completion of this book.

Contents

Techniques and Equipment for Intraoperative Monitoring

A. R. MØLLER[1]

Summary

The basis of intraoperative monitoring is discussed. Techniques for intraoperative recording of brainstem auditory evoked potentials (BAEP) and electromyographic (EMG) potentials from muscles that are innervated by cranial motor nerves are described. It is shown that intraoperative recordings of evoked potentials not only are of value in helping to reduce neurological deficits as a result of surgical manipulations, but they can also be used to guide the surgeon in some types of operations.

The main purpose of intraoperative monitoring of evoked potentials is to help reduce complications in the form of neurological deficits. Intraoperative monitoring has also proven useful in a few types of neurosurgical operations. Intraoperative monitoring can be helpful in reducing the risk of neurological deficits if 1) electrical potentials that change when neural injury is imminent can be recorded and evaluated before the injury becomes irreversible, and 2) if the process that caused the injury can be reversed, by surgical or other intervention. Meeting this second requirement means that the step in the operation that caused the injury must be identified, and to meet both of these requirements it must be possible to detect the related change in the recorded potentials with as little delay as possible (Raudzens 1982; Grundy 1982, 1983a,b).

In this chapter some examples will be given of the uses of intraoperative monitoring of evoked potentials in neurosurgical operations. Specifically, methods will be described for preserving hearing in operations in the posterior fossa, and for preserving facial function in acoustic tumor operations. In addition, the use of intraoperative monitoring of evoked potentials to preserve function of several of the cranial motor nerves in operations on large tumors of the skull base will be discussed. Finally, an example will be given of how intraoperatively recorded electromyographic (EMG) potentials can be used to identify the blood vessel compressing the central portion of the facial nerve in patients with hemifacial spasm (HFS). A brief discussion follows of the various techniques that are in use for intraoperative monitoring of evoked potentials, including the equipment needed and requirements of personnel who operate the equipment and interpret the results.

[1] Department of Neurological Surgery, University of Pittsburgh School of Medicine, 230 Lothrop Street, Pittsburgh, PA 15213, USA.

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Monitoring Sensory Evoked Potentials

Often the sensory evoked potentials that are used in intraoperative monitoring can only be recorded from locations that are remote from the generators of the potentials. Such "farfield" potentials are usually of much lower amplitude than background electrical activity such as electroencephalographic (EEG) potentials or spontaneous muscle potentials in patients who are not paralyzed during the operation. In addition, there is usually a considerable amount of electrical interference from equipment in the operating room. Signal averaging is the method commonly used to improve the signal-to-noise ratio so that an interpretable record can be obtained of sensory evoked potentials that have small amplitudes compared with amplitudes of the background noise. This is the case for brainstem auditory evoked potentials (BAEP) and somatosensory evoked potentials (SSEP).

Under ideal conditions, the signal-to-noise ratio increases in proportion to the square root of the number of responses that are added, assuming 1) that the responses to each stimulus are identical and 2) that the noise is random and unrelated to presentation of the stimulus. When the amplitude of the signal is small in relation to the noise, many responses must be added and the signal-to-noise ratio therefore increases slowly. Since there are limitations as to how frequently sensory stimuli can be presented without altering the waveform and amplitude of the response, it may take a considerable time to obtain an interpretable record when the response has a low amplitude relative to the background noise, because the responses to many consecutive stimuli must be added in order to recover the signal from the noisy background. If the response changes during the period when responses are averaged, as it may after an injury, the responses to the different stimuli that are presented will not be identical. When this is the case, the first assumption for the use of signal averaging will not be fulfilled, and the result will be that the waveform of the averaged response will be distorted and may not resemble the waveform of any of the individual responses.

In addition, the background noise is seldom random but instead is sometimes intermittent and often periodic or quasiperiodic. Thus, the second condition mentioned above is also not fulfilled when averaging techniques are applied to intraoperative monitoring.

Because it is imperative that recordings be obtained and interpreted in as short a time as possible when evoked potentials are used in intraoperative monitoring, all technical means to these ends should be employed when evoked potentials are used intraoperatively. Spectral filtering aids averaging in that it increases the signal-to-noise ratio; however, it is not used as often as it might be because it is assumed to cause severe distortion of the waveform of the recorded potentials and, more importantly, to shift the peaks of an evoked potential in an unpredictable manner (Boston and Ainslie 1980; Doyle and Hyde 1981a). Most often, only the electronic filters that are normal components of the amplifiers are used, but the signal-to-noise ratio can be improved considerably and more efficiently by the use of zero-phase digital filters (Doyle and Hyde 1981a; Møller 1983, 1988a,b). Because such filters do not shift the peaks of evoked potentials in time,

much more aggressive spectral filtering can be implemented when zero-phase digital filters rather than electronic filters are used. More aggressive spectral filtering, in turn, can reduce considerably the number of responses that need to be averaged to obtain an interpretable response. Thus, aggressive spectral filtering implemented as zero-phase digital filtering can be a very efficient way to increase the signal-to-noise ratio of evoked potentials without the drawbacks of electronic filtering.

The filtering procedure just described can only be used when digital computers are available, so that the filters used do not need to be physically realizable. Digital filters can be designed to have no phase shift, so that filtering does not shift the different peaks of an evoked potential waveform. Also, digital filtering can be performed after the recorded responses have been averaged, and therefore different types of filtering can easily be applied to the same data when digital filters are used; this cannot be done when electronic filters are used. Digital filters are also more flexible than electronic filters in that digital filters can be designed to enhance certain features of a recorded pattern, such as certain peaks in the response. We have used this feature of digital filters to enhance peaks I, III, and V of BAEP recorded and viewed in the operating room (Møller 1988a). Since the raw averaged responses are stored, these recordings can later be passed through different digital filters to enhance other features of the recordings.

When choosing what characteristics are desired in a filter, it has been common practice to compare the spectrum of the "clean" response with the spectrum of the background noise, and then to choose a filter that interferes minimally with the spectrum of the response and maximally attenuates the background noise. However, the entire spectrum of evoked potentials is not necessarily needed for interpretation of the response. In evaluating BAEP for example, the features of main clinical importance are the latencies of the individual peaks. The raw averaged data, however, contains a considerable amount of spectral energy at lower frequencies that is unimportant for the identification of the individual peaks. Thus, when BAEP are used in intraoperative monitoring, attenuating the energy at low frequencies helps in the correct identification of the various peaks of the BAEP and at the same time increases the signal-to-noise ratio because the low-frequency components of the background noise are also attenuated. However, it is important that the filtering that is used to attenuate low-frequency components not shift the peaks of the potentials in time, and therefore zero-phase digital filtering should be used for this purpose (Boston and Ainslie 1980; Doyle and Hyde 1981a,b; Møller 1988a,b).

When evoked potentials (BAEP and SSEP) are evaluated clinically, replicability of the averaged potentials is usually determined as a measure of how well the averaged potentials represent the responses to the stimuli rather than artifacts or noise of one kind or another. However, obtaining the 2 separate records needed to test response replicability takes time, and in the operating room it is important to achieve quality control in the shortest time possible. A quality control measure that does not take any extra time is comparing the average of all responses with the average obtained by adding every other response and the inverse of the alternate responses (Schimmel 1967; Wong and Bickford 1980; Elberling 1985; Møller

1988a). The response to the stimulus will be cancelled in the latter "± average" and only the noise will remain. Normally, the amplitude of the ordinary averaged response will increase as each new response is added, while the amplitude of the ± average will decrease with the addition of each new response. If the 2 averages remain similar as more and more responses are added, it is an indication that the stimuli that are presented do not produce any response. The ratio between the RMS value of the ordinary average and the ± average is a quantitative measure of the reliability of the obtained averaged responses.

However, when the background noise is not a stationary random noise but contains periodic or quasiperiodic components, then the results of comparing the ordinary average and the ± average may be misleading. In such cases the repetition rate should be varied randomly around the value that is selected. This will eliminate or at least reduce the possibility that periodic components in the background noise add in phase and therefore show up in the averaged response or in the ± average. The latter will occur when the periodic component in the interference signal has a frequency that is equal to or a submultiple of twice the periodicity of the repetition frequency of the stimulus.

The importance of obtaining an interpretable record in as short a time as possible when evoked potentials are used in intraoperative monitoring has prompted the development of methods other than just recording farfield evoked potentials in the operating room. For example, using potentials recorded directly from the exposed eighth nerve in intraoperative monitoring for the purpose of preserving auditory function in operations where the eighth nerve becomes exposed provides interpretable records in a shorter time than using conventionally recorded BAEP (Møller and Jannetta 1983; Silverstein et al. 1984; Møller 1988a). The compound action potentials (CAP) recorded directly from the eighth nerve have large amplitudes and they can often be viewed directly on an oscilloscope without any averaging, or only a few responses need to be averaged in order to obtain an interpretable response. Such nearfield potentials therefore provide second-by-second information about changes in neural conduction in the auditory nerve. The immediate availability of information about an injury is of importance in preventing permanent injuries because it makes it possible to reverse the step in the operation that caused the injury with very little time lag. Immediate availability of information is also important because it permits identification of exactly which surgical manipulation caused the injury. This is naturally of great importance in developing better surgical methods, but it is also of value in the teaching of residents and fellows.

We have recorded CAP from the exposed eighth nerve routinely for about 8 years using the electrode shown in Fig. 1. This electrode, which is designed for monopolar recording from exposed neural structures, consists of a fine, malleable, Teflon-insulated silver wire (Medwire Corp., 121 South Columbus Ave., Mt. Vernon, New York 10553, U.S.A., Type AG 7/40) to the uninsulated tip of which a small cotton ball is sutured using 5–0 silk suture. When used for recording from the exposed eighth nerve, this electrode is placed in direct contact with the nerve (Fig. 2). The potentials recorded from a normal nerve in a person with normal hearing have a triphasic shape with an initial positivity followed by a large

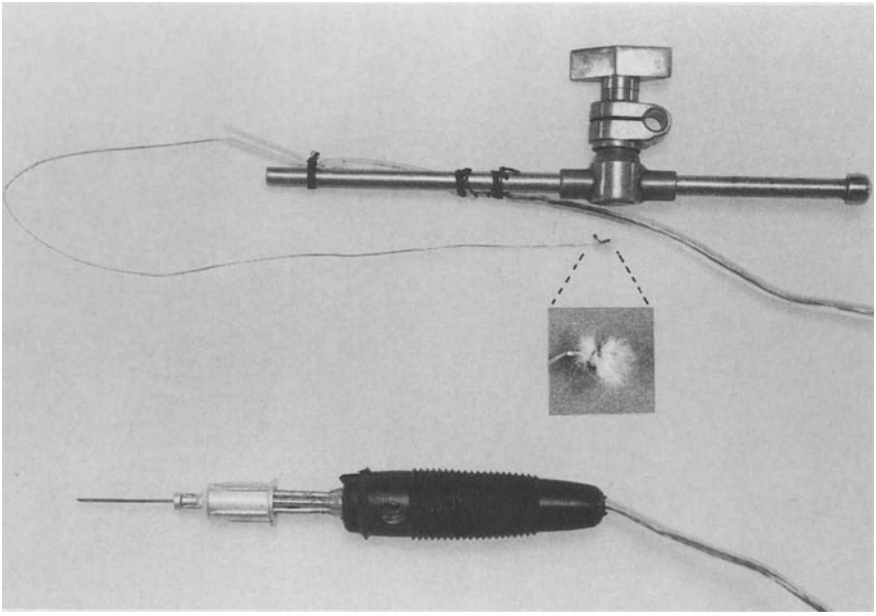

Fig. 1. The electrode used to record from the exposed neural surface

negative peak and then a small positive peak (Fig. 3). If the auditory nerve is injured, for example by the heat of electrocoagulation, its waveform typically changes and the initial positive peak can become the dominating component, thus indicating that there is a partial conduction block in the nerve ("cut-end" potential) (Fig. 4).

We have used the electrode shown in Fig. 1 to monitor auditory function in patients undergoing microvascular decompression (MVD) operations for trigeminal neuralgia (TN), hemifacial spasm (HFS), disabling positional vertigo (DPV), and glossopharyngeal neuralgia (GN) and in operations to remove acoustic tumors. Others have used a similar electrode for recording from the auditory nerve (Silverstein et al. 1984). When recording CAP from the eighth nerve is combined with monitoring of BAEP that is enhanced by digital filtering, the risk of hearing loss can be reduced to less than 1% in MVD operations to relieve TN, HFS, DPV, and tinnitus (Møller and Møller 1989). More complex methods for enhancing signals in noise have been described (Hoke et al. 1984; Sgro et al. 1985 a, b), but these methods, although more efficient than traditional averaging, have not won more general acceptance and use and are not at present available in commercial equipment.

In selecting equipment for intraoperative monitoring, it may be of interest to note that versatility may not be the foremost feature to consider. Rather, reliability and ease of operation of the equipment are the most important features, so that the chances of making mistakes are minimized.

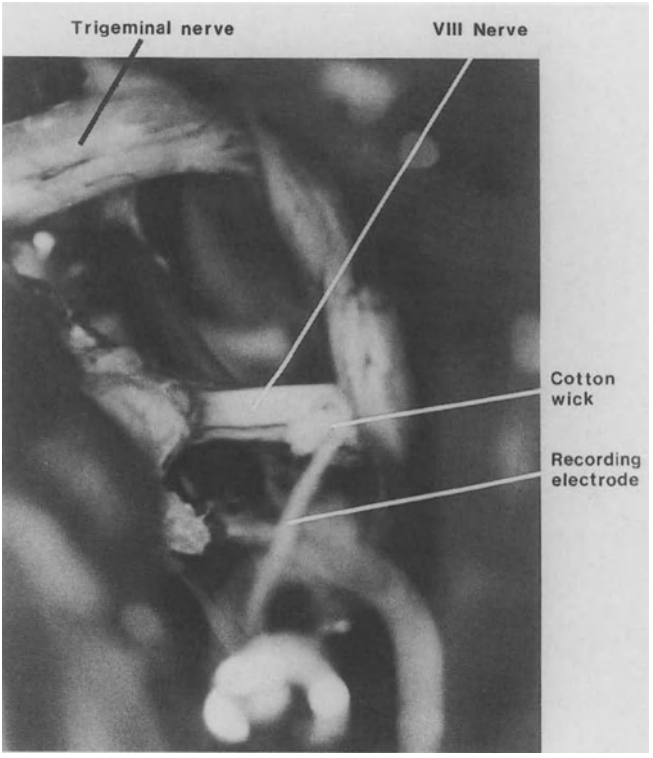

Fig. 2. The electrode in Fig. 1, placed on the exposed eighth nerve. (From Møller 1988a)

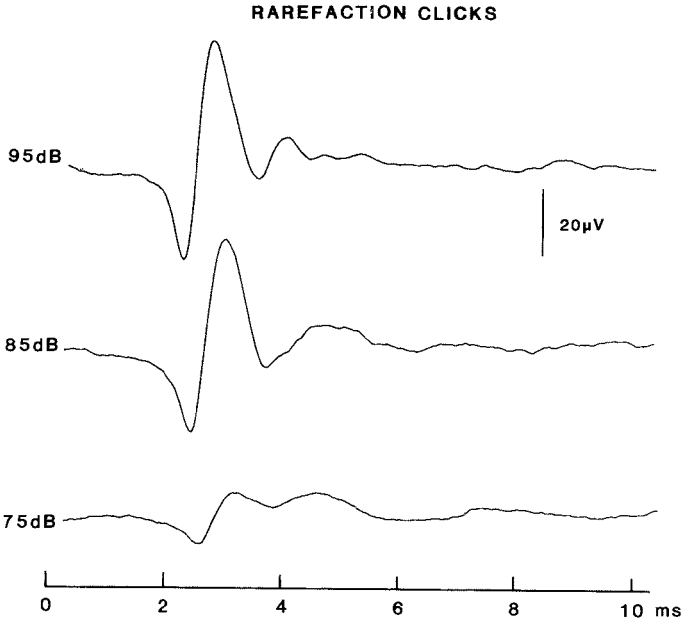

Fig. 3. CAP recorded from the exposed eighth nerve in response to click sounds obtained in a patient with normal hearing

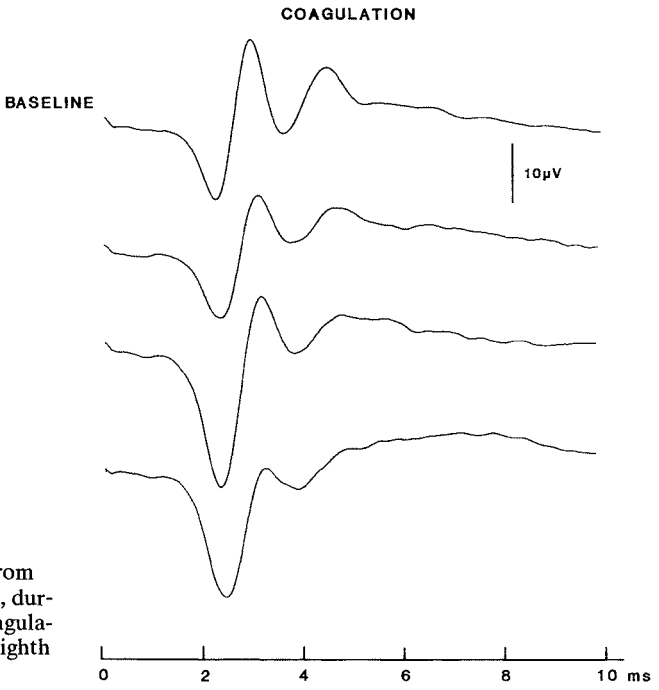

Fig. 4. CAP recorded from the eighth nerve before, during, and after electrocoagulation was done near the eighth nerve

When to Inform the Surgeon of Changes in Evoked Potentials

The changes in evoked potentials that are most indicative of neural injury are changes in the latency. Because these changes usually occur gradually, the question arises as to when the surgeon should be informed that a change has occurred. It has been our practice to inform the surgeon about any change that is greater than the small deviations that normally occur in the response. The occurrence of such small changes does not indicate that permanent sensory deficit is likely to result, but it does indicate to the surgeon that the preceding step in the operation affected the function of the auditory system. When presented with this information, the surgeon has the option to ignore the change or to reverse the step in the operation that led to the change. The latter is an obvious choice if that step is not crucial and can be modified without any negative effects. Even when the surgeon chooses not to modify the operative procedure at the earliest sign of a change in the evoked potentials, knowing which step in the procedure initiated the change is crucial if the change should increase to the point that it indicates risk of a permanent neurological deficit. A change of this magnitude calls for action on the part of the surgeon, but appropriate action can only be taken when the surgeon knows the cause of the change.

Methods to Reduce Risk of Loss of Facial Function in Operations on Acoustic Tumors

For many years, surgeons have sought to preserve facial function in operations to remove acoustic tumors by identifying the facial nerve via electrical stimulation: an electrical current is applied to tissues and a response is looked for in the facial muscles. Various methods for detecting the contractions of the facial muscles have been used. The earliest, observation of the face by an assistant or the anesthesiologist, has now been largely replaced by recording of the EMG activity in face muscles (Delgado et al. 1979; Møller and Jannetta 1984a; Prass and Lueders 1986) or by sensing the movements of the face using electronic transducers (Sugita and Kobayashi 1982).

We have used EMG recordings during operations for acoustic tumors. In addition to displaying the recorded potentials on an oscilloscope, we also make the EMG potentials audible so that the operating surgeon can hear directly when the face muscles contract (Møller and Jannetta 1984a, 1985a; Møller 1988a). The stimulation consists of rectangular impulses of 100-μs duration generated by a low-impedance source and applied through a hand-held monopolar electrode (Fig. 5). With this electrode it is easy to probe the operative area, and the use of a low-impedance electrical source makes the stimulation of neural tissue less dependent on whether the field is wet or dry, because even though current is shunted through cerebrospinal fluid, current flow through any given part of the tissue will be less affected than would have been the case had a constant-current source been used.

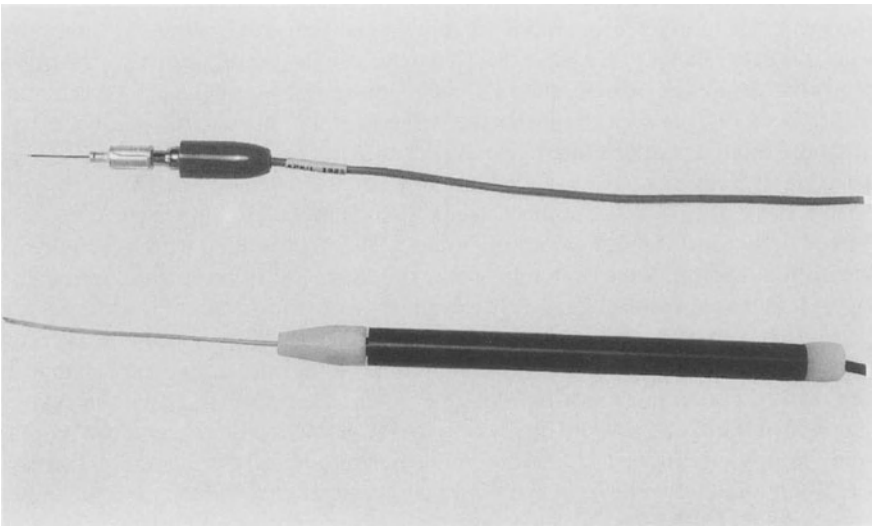

Fig. 5. Handheld monopolar stimulating electrode and hypodermic needle used as return electrode. (From Møller 1988a)

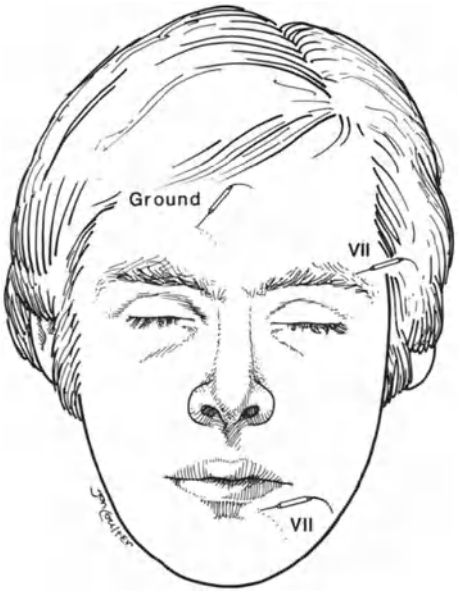

Fig. 6. Schematic illustration of the placement of electrodes for recording EMG potentials from face muscles. The electrodes are subdermal platinum needles (Grass Instruments Co., Type E2). (From Møller 1988a)

Since there is no need to determine which part of the facial nerve is being activated, EMG potentials are recorded differentially between electrodes placed in the upper face and the lower face (Fig. 6). This electrode arrangement permits EMG recorded from muscles of the entire face to be displayed on a single channel. Because the recorded potentials are made audible, and the near-constant voltage characteristic of the source of stimulation makes it largely unnecessary to adjust the stimulus strength, the surgeon can quickly probe many locations in the tumor for the presence of the facial nerve and can remove portions of the tumor that give no response. These portions can then be removed without risk of injuring the facial nerve.

In this type of intraoperative monitoring, EMG are recorded from one entire half of the face. Further, the anatomical changes that may occur with large tumors can make it difficult to distinguish between the facial and the trigeminal nerve on an anatomical basis. Thus, the surgeon may mistake an EMG response from the mastication muscles to stimulation of the trigeminal nerve for an EMG response from the facial muscles to stimulation of the facial nerve. It is, however, easy to discriminate between responses generated by the facial nerve and those generated by the trigeminal nerve if the response latency can be determined: the latter has a much longer latency than the former (approximately 1.5 ms versus 5 ms) (see Fig. 7). Displaying the responses on an oscilloscope will enable one to distinguish whether the responses were evoked by stimulation of the facial or the trigeminal nerve.

Monitoring spontaneous facial muscle activity (in the absence of electrical stimulation) is of great value when the facial nerve is not directly visible. If the nerve is stretched as a result of retraction it may be injured, and such an injury will

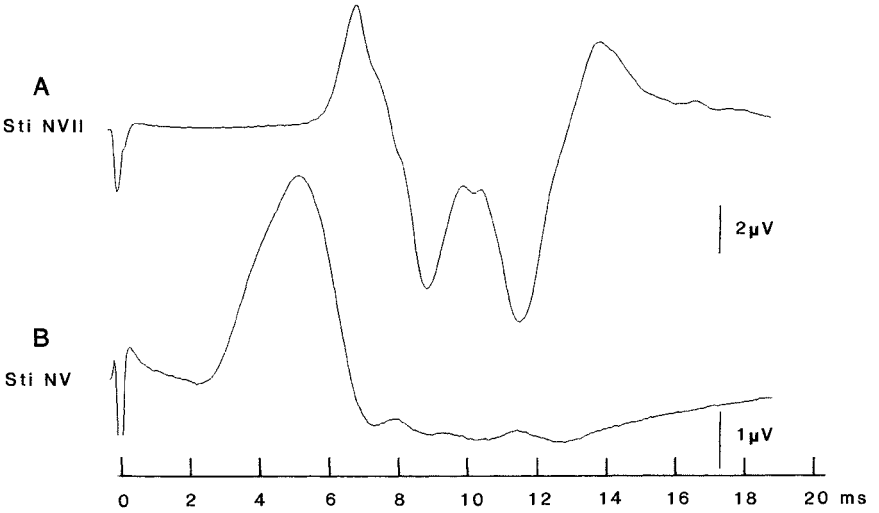

Fig. 7. Typical recording from electrodes placed in the face as shown in Fig. 6. **A** Obtained when the intracranial portion of the facial nerve was stimulated electrically with 100-μs rectangular impulses at about 0.8 V; **B** Obtained when the intracranial portion of the motor portion of the fifth cranial nerve was stimulated electrically with similar pulses.

normally be associated with facial muscle activity that can be identified and brought to the surgeon's attention. The surgeon is thus warned that the nerve has been stretched. Continuous monitoring of facial muscle activity is particularly important when the last small pieces of tumor are removed from the facial nerve. Injury to the facial nerve will usually manifest itself by facial muscle activity that can take the form of single impulses of activity or bursts of periodic activity, the latter being indicative of a more severe injury (Møller and Jannetta 1984a; Prass and Lueders 1986). When such muscle activity is monitored continuously and made audible to the surgeon, it provides the surgeon very useful feedback in the delicate work of removing the last portions of tumor affecting a facial nerve that was injured preoperatively by the tumor.

Monitoring Other Cranial Motor Nerves

In operations to manage large skull base tumors, some of which may invade the cavernous sinus, all cranial motor nerves can be at risk. Our work has shown that monitoring EMG recorded intraoperatively from the muscles innervated by cranial nerves III, IV, V, VI, and XII, in ways similar to that just described for the facial nerve, can reduce the risk of neurological deficits as a result of surgical injury to these nerves (Sekhar and Møller 1986; Møller 1987, 1988a, 1989).

To monitor cranial nerves II, IV, and VI we record EMG potentials from the extraocular muscles; to monitor the motor portion of cranial nerve V we record

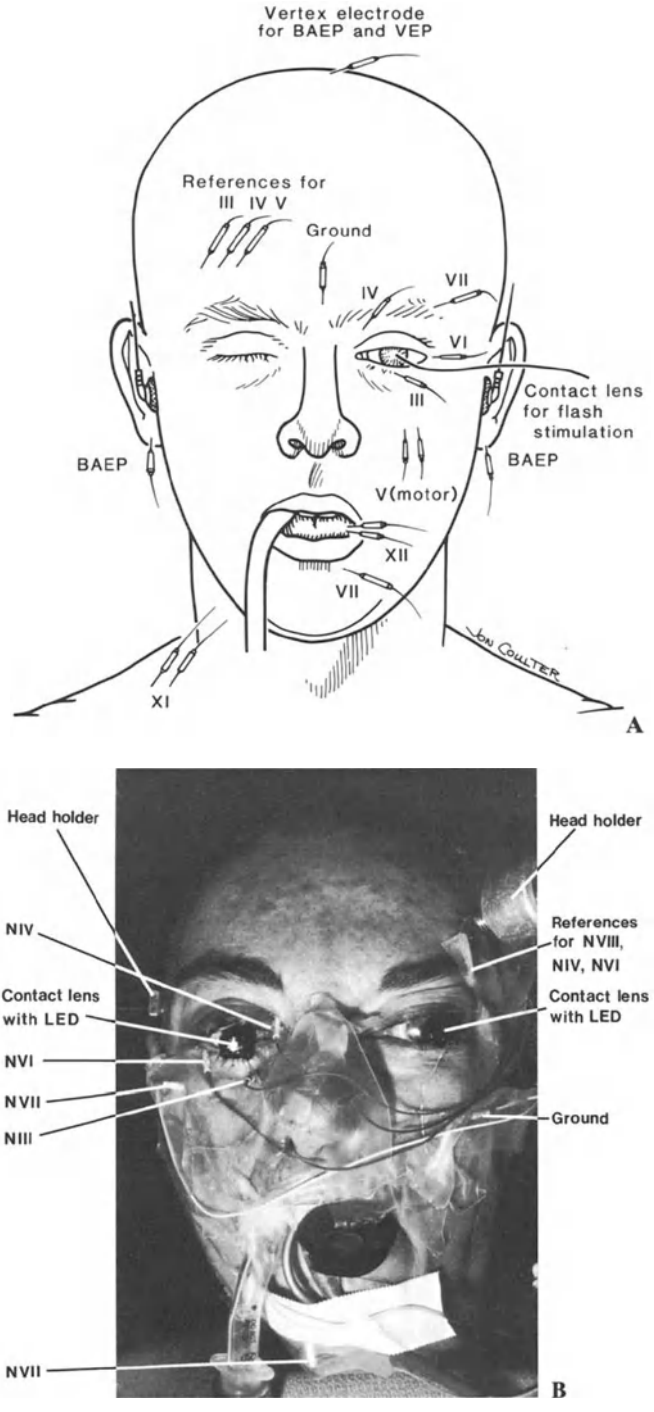

Fig. 8. A Placement of electrodes to record EMG potentials from the extraocular muscles, facial muscles, masseter muscles, and the tongue, representing the responses from cranial nerves III, IV, VII, XII, and the motor portion of V. Note that the recordings from the extraocular muscles are monopolar, with the reference electrode placed on the opposite side. This is to avoid contamination of the response by facial muscle activity. Responses from the facial muscles, masseter muscle, and the tongue are all bipolar, as they were recorded with pairs of needle electrodes. The same type of subdermal needle electrodes as are shown in Fig. 6 were used. **B** Patient with electrodes in place. This patient also had contact lenses with light-emitting diodes attached to stimulate the eyes for visual evoked potentials. (From Møller 1988a)

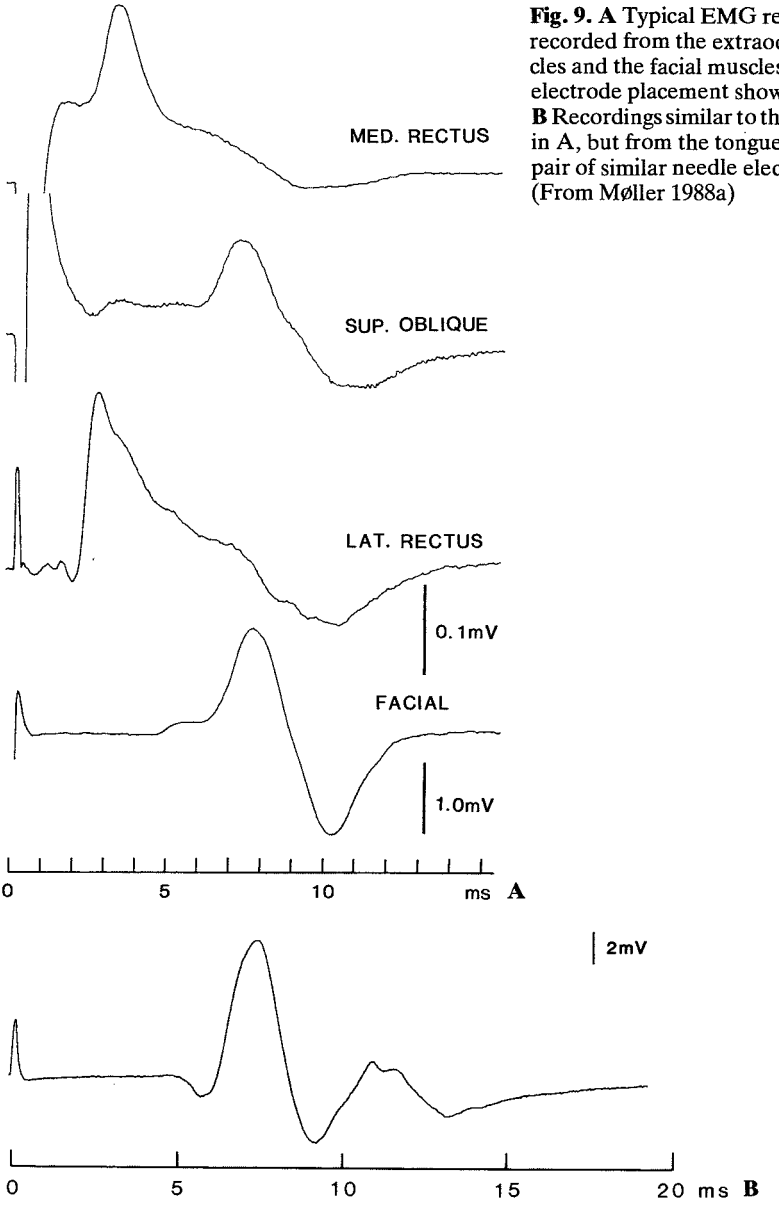

Fig. 9. A Typical EMG responses recorded from the extraocular muscles and the facial muscles using the electrode placement shown in Fig. 8. **B** Recordings similar to those shown in A, but from the tongue using a pair of similar needle electrodes. (From Møller 1988a)

from the masseter muscle; to monitor cranial nerve VII we record from the facial muscles, as described above; and to monitor cranial nerve XII we record EMG potentials from the tongue. The electrode placements we use are shown in Fig. 8. Subdermal needle electrodes (Grass Instruments Co., 101 Old Colony Ave., P.O. Box 516, Quincy, Massachusetts 02169, U.S.A., Type E2) are placed in the

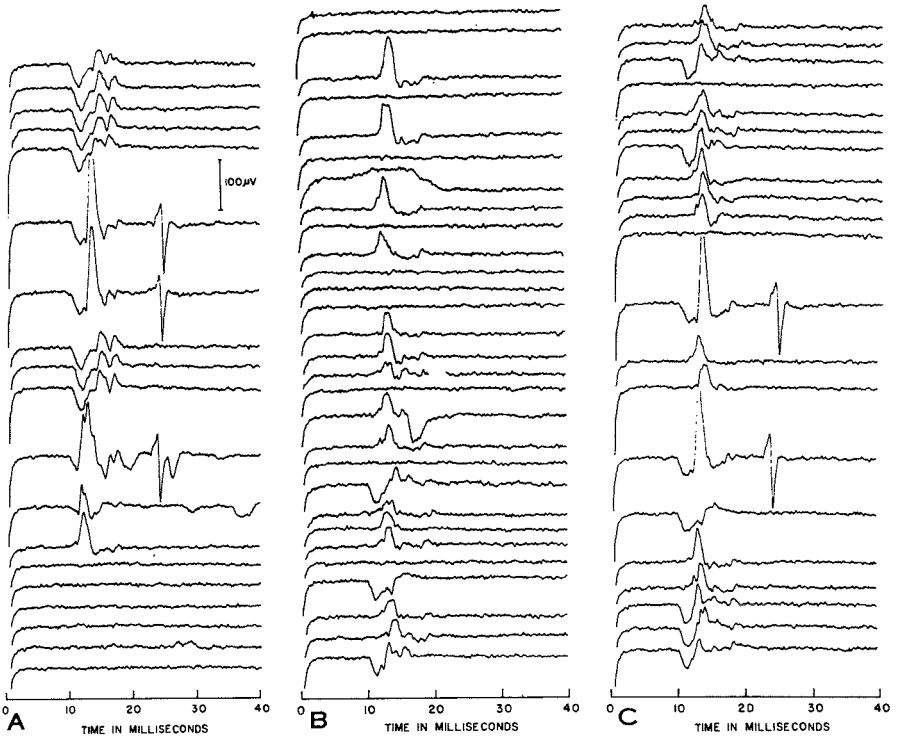

Fig. 10. Typical abnormal muscle responses recorded from the mentalis muscle in response to stimulation of the zygomatic branch of the facial nerve obtained during a MVD operation to relieve HFS. The stimulation was presented at a rate of 10 pps and the graph shows consecutive recordings *(beginning at top)*. When the offending blood vessel was lifted off the nerve, the response disappeared *(middle of column B)*, and it reappeared when the vessel fell back on the nerve *(top of column C)*. There was an interval of 10 seconds between the end of column B and column C. (From Møller and Jannetta 1985b)

extraocular muscles percutaneously to obtain a monopolar recording. The reference electrodes are placed on the forehead on the other side to avoid contamination with EMG from face muscles on the operated side (Fig. 8 A, B). Two similar electrodes, placed nearby, are used to obtain a bipolar recording from the masseter muscle and a similar arrangement is used for recording from the tongue. The EMG recordings from the face muscles are made in the same way as was described earlier in this chapter for recording EMG in operations for acoustic tumors. The respective cranial nerves can be stimulated intracranially using a handheld monopolar stimulating electrode similar to that used for monitoring facial function (Fig. 5) in operations to remove acoustic tumors.

Typical EMG potentials recorded from the extraocular muscles in response to such stimulation are shown in Fig. 9 A, and potentials from the tongue are shown in Fig. 9 B. These potentials are of large amplitude, and they can easily be visualized on an oscilloscope or made audible as feedback for the surgeon as he or

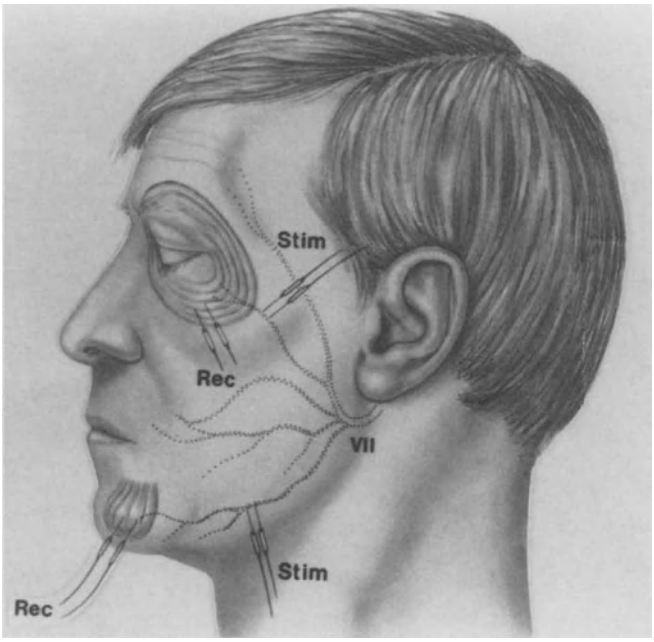

Fig. 11. Electrode placement used for recording EMG potentials in patients undergoing microvascular decompression operations for HFS. (From Møller 1988a)

she searches for the different nerves or seeks to detect injury to these nerves. Using the same type of electrical stimulation, it is easy to find the respective nerves even in cases where the anatomy is grossly distorted by the tumor. Continuous monitoring of the EMG activity in all these muscles makes it possible to detect the occurrence of injury to the respective motor nerves, using techniques similar to those described for monitoring facial function. Making the EMG audible greatly facilitates the detection of such injury activity, and it also serves as a convenient way for the surgeon to obtain feedback when using electrical stimulation to localize specific cranial nerves.

The spinal accessory nerve (CN XI) can be monitored by recording EMG from the scapular muscles, but we have used this technique only occasionally because maximal stimulation of the accessory nerve will result in a very strong contraction of the respective muscles with the attendant risk of injury to the tendons and joints involved.

Using Electrophysiological Recordings to Guide the Surgeon in the Operation

In one type of neurosurgical operation, namely microvascular decompression (MVD) of the facial nerve to treat hemifacial spasm (HFS), it has been possible to use intraoperatively recorded potentials to guide the surgeon in the operation and to assure that the therapeutic goal of the operation has been achieved, before

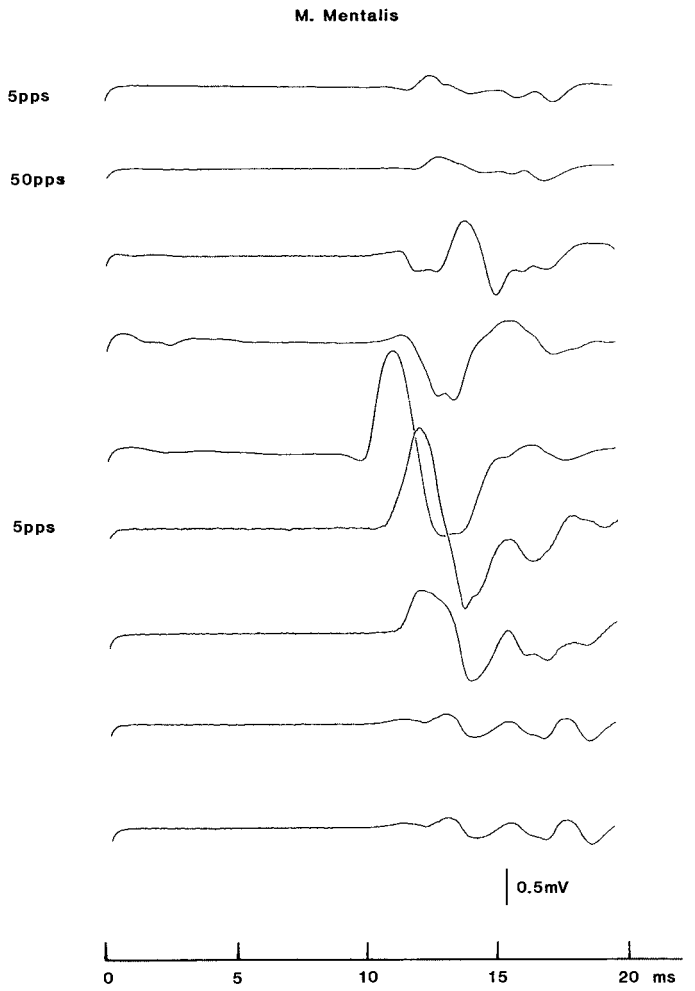

Fig. 12. Response from the mentalis muscle to stimulation of the temporal branch of the facial nerve in a patient undergoing MVD to relieve HFS. Note the increase in amplitude of the response when the repetition rate of the stimulation is increased from 5 pps *(top tracing)* to 50 pps *(middle 4 tracings)*. This increase remains for a short time after the stimulus rate is returned to 5 pps *(lower 4 tracings)*

the termination of the operation (Møller and Jannetta 1987; Møller 1988a). HFS is caused by a blood vessel lying in firm contact with the facial nerve in its intracranial course where it is covered with central myelin, just before it enters the brainstem. In patients with this disorder, an abnormal muscle response can be evoked by electrically stimulating a branch of the facial nerve and recording from muscles that are innervated by other branches of the facial nerve (Esslen 1957; Møller and Jannetta 1984b, 1985b). This abnormal muscle response in patients with HFS disappears instantly when the offending blood vessel is moved off the

facial nerve (Møller and Jannetta 1985b) (Fig. 10). When this abnormal muscle response is elicited by stimulation of the zygomatic (or temporal) branch of the facial nerve and recorded from the mentalis muscle (Fig. 11), it occurs with a latency of about 10 ms (as seen in Fig. 10). The response is enhanced by stimulation at a high rate (e.g., 50 pps) (Møller and Jannetta 1986 a, b) (Fig. 12). The utilization of intraoperative electrophysiological recordings in MVD operations of the facial nerve in patients with HFS is based on the above mentioned finding that the abnormal muscle response disappears when the offending blood vessel is lifted off the nerve. This makes it possible to identify which blood vessel is causing the spasm and also to verify that the facial nerve has been appropriately decompressed before the operation is terminated. Routine intraoperative monitoring of this abnormal muscle response has reduced the percentage of patients needing reoperation after MVD for HFS. It has also decreased the operating time because it has made it possible for the surgeon to identify exactly the particular blood vessel causing the HFS in any individual patient.

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Technical Parameters, Artifacts, and Quality Assessment of Intraoperative Evoked Potentials

M. R. NUWER[1]

Summary

Monitoring personnel should consider techniques to be flexible. A variety of options are available for specific evoked potential (EP) techniques. Somewhat different techniques should be applied whenever monitoring situations require such variation in technique. Informed users should understand the various ways in which such flexibility of technique can be helpful.

Stimulus site, type, rate, recording site, filter, and other technical parameters can be altered to best suit a particular patient's circumstances. Averaging can also be performed in several ways. During the baseline portion of an operation, the monitoring team should choose the technique that best suits the particular patient and procedure.

Artifacts and other technical problems are common in the operating room. The monitoring personnel should understand their own equipment and the artifacts present in the operating room so that they can identify the technical problems and minimize them. The monitoring team can assess the adequacy of their own recordings by applying some general quality-assurance standards, assessing reproducibility, and noisiness of the recordings. The overall technical goal of monitoring should be the rapid production of high-quality EP signals, while minimizing baseline variability in latency and amplitude of the major EP peaks. Knowledgeable users should be able to achieve this technical goal in most cases.

Monitoring personnel should consider techniques to be flexible. The particular methods employed should vary for different applications and for different patients, in ways that optimize the sensitivity and specificity of the techniques in specific circumstances. Informed users of intraoperative monitoring should understand the ways in which such flexibility can help. Each also needs to understand the limitations and disadvantages of the various approaches to monitoring. The goal should always be to provide the patient with the best possible monitoring, based upon the knowledgeable application of general rules and past experience in performing these techniques. The flexibility must include variation of the sites of stimulation, the type and rate of stimulation, sites of recording, filters, and

Department of Neurology, University of California, Los Angeles, Reed Neurological Research Center, 710 Westwood Plaza, Los Angeles, CA 90024, USA.

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other technical parameters. Even the process of averaging can be performed in a variety of ways. Artifacts and other technical problems need to be well understood, so that they can be avoided or at least minimized. Monitoring teams should be encouraged to interact regularly with the surgical and anesthesiology teams in each surgical case, learning to adapt the monitoring procedure to the patient and circumstances as needed. The present chapter outlines some general principles that can be used in setting technical parameters and in identification and elimination of artifacts. The monitoring team should also be able to criticize its own results, as a form of quality assurance. The latter should be undertaken as a way gradually to improve the performance of the monitoring team. This chapter outlines some general guidelines for assessing the quality of the evoked potentials (EP) obtained intraoperatively.

Technical Parameters

Rate

There is a conflict inherent in choosing a stimulus rate. At very fast rates of stimulation, EP amplitude becomes attenuated and the EP itself becomes broad and ill-defined. At such a fast rate, an EP is hard to find and measure, and measurements are subject to substantial error because of background noise. Because of this, slow rates of stimulation are best for providing the highest quality EP peaks. Yet in the operating room setting, there is an opposite need. The EP needs to be produced as fast as possible. A tradeoff needs to be found, providing EP as fast as possible without causing undue loss of EP amplitude and clarity.

A compromise rate can be found in most circumstances. For lower extremity somatosensory monitoring, this rate is often around 5 stimuli per second on each leg. (Nuwer and Dawson, 1984a). A faster rate can be used for upper extremity somatosensory testing because the upper extremity cortical somatosensory evoked potentials (SSEP) are taller and sharper. Rates around 10/s can often be tolerated for this pathway, without causing too great an attenuation or blurring of the EP. For brainstem auditory evoked potentials, the tradeoff is often found around 30/s for each ear (Levine 1988).

For an individual patient, the best stimulus rate is hard to predict. Older patients tolerate fast rates less well, and often need somewhat slower rates of stimulation to produce adequate EP. Patients with pre-existing nervous system impairment tolerate fast rates less well. Young, otherwise healthy subjects often tolerate fast rates quite well. The monitoring team should take the opportunity to investigate the effect of rate on an individual patient whenever possible, or when time permits. Often this can be done during the baseline portion of an operation, when no significant surgical intervention is yet undertaken. The experience gained during this baseline or early portion of the operation can help set the stimulus rates in a way that optimizes the speed at which EP can be produced during subsequent portions of the monitoring. Such testing can be done by increasing or decreasing the rate of stimulation in 10 to 20% increments until the best tradeoff is found. It should be pointed out that the best tradeoff is not at the point

at which the amplitude is maximal, but rather at the point at which the amplitude is somewhat attenuated but still well above background noise.

Also, flexibility is called for during subsequent portions of the operation. If an EP becomes difficult to find because of increasing background noise or decreasing signal amplitude, slowing the rate of stimulation can be helpful to create a better, more well-defined EP. The disadvantage is that a greater amount of time is then necessary to produce each successive EP.

Filters

Just as rate should be set individually for a particular patient and circumstance, the particular settings of filters also should be considered flexible. The filters should be set to provide the best compromise between ideal EP and minimal reproduction of background noise. Oftentimes, this requires using relatively restricted rather than full-range amplifier filter settings. For example, Fig. 1 shows the effect of increasing the low-frequency cutoff frequency when recording lower extremity SSEP. Using a relatively low-frequency filter setting of 1 Hz, the EP has maximal amplitude, but the background noise is also very high in amplitude. Increasing the low-frequency cutoff frequency to 15 Hz causes a slight reduction in amplitude of the EP along with a moderate reduction in overall amplitude of the background noise. Increasing the filter setting still further to 30 Hz causes a further reduction in background noise, making the principal EP peaks more clearly reproducible and better separated from noise. In this particular case, 30 Hz turns out to be the best setting for monitoring. Increasing the low filter setting still further, to 75 Hz, causes a further reduction in amplitude of the background noise, along with substantial attenuation of the principal EP itself. Recognizing that background noise can increase and decrease in the course of the operation, 75 Hz is probably too high a setting for the low filter in this circumstance. The monitoring team should take advantage of the baseline portion or early portion of an operation to investigate the relative effectiveness of filter changes on the particular patient in the individual circumstances. The relationship between the filter settings and EP amplitude should be investigated, and the best tradeoff identified for the individual patient. The goal of monitoring is to find a good, reproducible, early EP that is stable throughout the course of an operation, except of course for any surgical complications that may occur. Finding this best stable early EP may well require setting the filters higher in some patients and lower in other patients. If one uses 30 Hz as the best starting point for a lower extremity SSEP cortical recording, the initial recordings should provide the information needed to determine whether a filter change could be advantageous. If the initial EP seen are relatively low in amplitude and broad, lowering the lower filter setting may well be the most practical way to improve the overall EP amplitude. However, this is at the risk of increasing the background noise, which in turn might mean that a larger sample size is needed to obtain an adequate EP. If the initial EP obtained at the 30 Hz low filter setting is tall and sharp, then a higher setting of the low filter may well suffice to reduce the background noise further, while still maintaining an adequate EP amplitude.

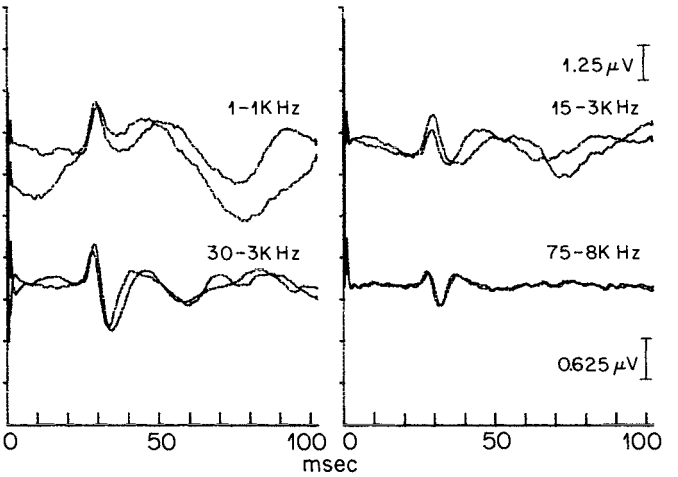

Fig. 1. Effects of four different filter settings used during intraoperative recording in one patient, taken from a single scalp channel (CZ-PZ). Variability is greatest in the 1-Hz channel and is reduced by higher filter settings. Each pair of EPs is a typical set of 2 consecutive recordings. The amplitude scale is doubled for the lower 2 EPs. (From Nuwer and Dawson 1984)

The monitoring team should consider changes in the upper filter settings as well, although the high-frequency cutoff frequency filter tends to have less effect on background noise than do low filter settings.

The 50- or 60-Hz notch filter is used to eliminate line noise. This filter can be useful in some types of monitoring such as for brainstem auditory evoked potential (BAEP) testing. However, it can cause a substantial new artifact if used with SSEP testing. It should be used with caution for intraoperatively monitoring any SSEP. Fig. 2 shows an example of the kind of "ringing" artifact that can be present, an exponentially damped sinusoid caused by the stimulus artifact in the notch filter. The ringing artifact from most notch filters has peaks at 16.6 ms and 33.3 ms for a 60-Hz notch filter, and at 20 and 40 ms for a 50-Hz notch filter. Such peaks can be mistaken for an EP. This ist especially true for SSEP, which tend to have peaks in the neighborhood of 16 to 20 ms for upper extremity tests and 33 to 40 ms for lower extremity tests. An unwary monitoring team might mistake these for an EP, and notice that they remained completely stable over time, providing a false sense of security. In fact, such an artifact can be present even in the absence of any functioning nervous system pathways. The notch filters can also cause a separate kind of problem. They can actually make the main EP peaks themselves disappear, especially for SSEP. These EP peaks have a basic frequency around 50 to 60 Hz. As a result, the main frequency component of the EP peak is greatly attenuated by the notch filter. An example of this is shown in Fig. 2,A. The basic lesson to be learned from these findings is that intraoperative monitoring teams should avoid using the notch filter. Instead, the source of the 50- or 60-Hz artifact should be identified and eliminated. This requires a search among the various

pieces of electrical equipment present in the room. It should be noted that a piece of equipment can emit high-amplitude line noise even when it is turned off just by virtue of grounding problems in that piece of equipment. As a result, the monitoring team needs to unplug pieces of equipment to check them as the source of the line interference, rather than just throwing the power switch off. Figure 3 shows an example of the kind of interference that may arise from equipment that is plugged in even though the equipment is not turned on, a source of interference that is not widely appreciated.

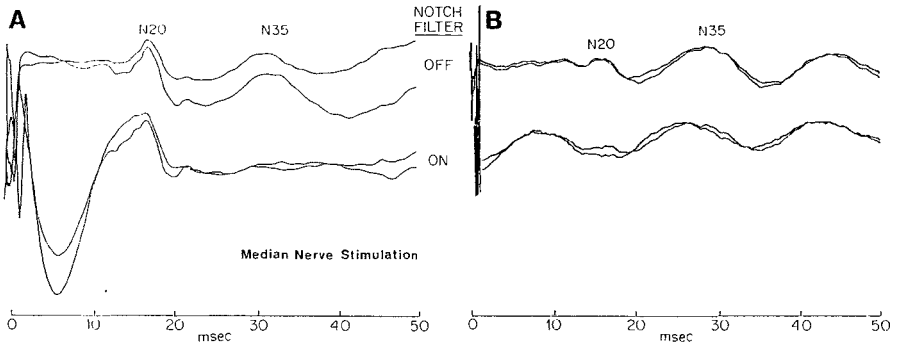

Fig. 2. A, B. The effect of the 60-Hz notch filter. **A:** The notch filter here has caused a large, brief ringing that has 1 peak at 16.6 ms, partly obscuring the N_{20} peak. It also attenuated the N_{35} peak, which has a width of about 17 ms (e.g., the N_{35} EP lies at about 60 Hz on a spectral frequency band). **B:** On a different EP machine, the ringing is more prolonged, with peaks every 17 ms. The 60-Hz ringing (bottom trace) resembles a real SSEP, but it can persist even when the actual SSEP has disappeared. The prominence and periodicity of this ringing differ among machines. (From Nuwer 1986)

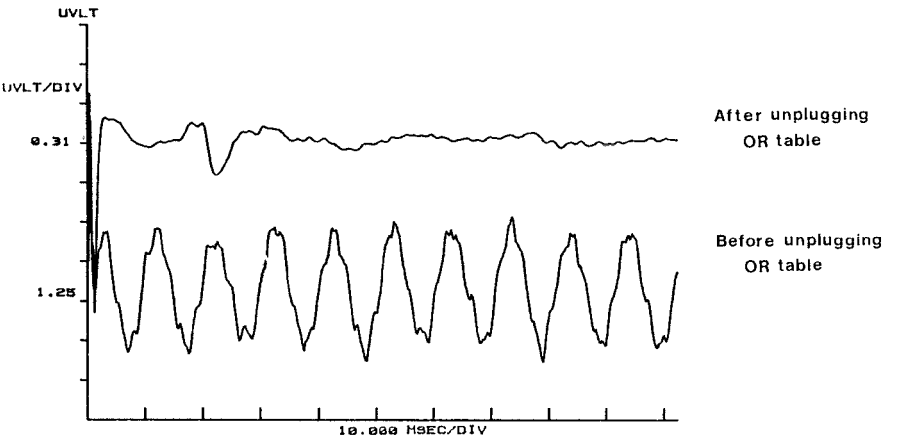

Fig. 3. An example of rhythmical electrical noise related to main power line currents. The artifact is at 100 Hz, instead of 60 Hz. In this case, the signal was broadcast from the operating room table's electric motor, even though the motor was not in use at the time. The artifact was abolished by unplugging the power cord leading from the table to the wall outlet. (From Nuwer 1986)

Stimulus and Recording Sites

Stimulus and recording sites also need to be chosen based on individual patient circumstances. Stimulus sites chosen most often for somatosensory tests are the wrists and ankles. The recording sites used have varied considerably among various reports. One needs to remember to record both rostral and caudal to the surgical site for spinal cord monitoring. The caudal responses can be obtained from around the lumbar spine or over the sciatic nerve. The more rostral recordings can be made most conveniently from the scalp. Or, spinal recordings can be made from electrodes placed within the surgical field or inserted near the spinal cord at the neck, or even passed into the cervical esophagus (Tamaki et al. 1981; Jones et al. 1982; Lueders et al. 1982; Harper and Daube 1989). Various techniques have their own advantages and disadvantages. A good monitoring team should understand the options that are available and make use of the best recording techniques for each individual patient.

Averaging Paradigms

Advanced types of averaging should be considered. Routine stimulation of 1 limb is straightforward but inefficient. Current monitoring technology is able to handle more complex situations. Several types of monitoring paradigms are currently used. Synchronous parallel averaging is the technique of stimulating at 2 separate sites simultaneously, allowing separate analysis of the 2 stimulated pathways because the EP show up at different places. For upper extremity SSEP, stimulation is to both wrists simultaneously, and recordings can be made separately over the 2 sides of the scalp, allowing separation of the results at that level. Asynchronous parallel averaging is the technique of stimulating at 2 sites, but alternating or interleaving the 2 separate stimulations. For lower extremity SSEP, this is done by alternately stimulating the 2 legs. With 5/s stimulation of each leg, overall stimulation is done at 10/s – 5/s on the left leg and 5/s on the right leg, separating the 2 by equal 100-ms increments. This allows separate stimulation of the 2 legs, with separate recordings at the scalp identifying the early P_{37} potential for each leg separately. This requires an averaging device sufficiently modern and automated to allow recording of each leg's P_{37} in separate averaging bins. Separate monitoring from each leg is preferable to simultaneous stimulation of both legs for spinal cord monitoring, because some complications can affect for just one half of the spinal cord (Molaie 1986).

Moving averages are also popular. A paradigm for moving averages is outlined in Table 1. Using moving averages, the monitoring team is updated on the EP several times faster than it would be with routine averaging. The moving average has the disadvantage that it combines old and new information, so that each new update is not completely new compared to the previous EP. In the busy setting of the operating room, however, the monitoring team often appreciates the quickest update possible and so the moving average has become a helpful tool.

Some advanced techniques of averaging will undoubtedly be found helpful in the future. The two-dimensional analysis system employed by Sgro and col-

Table 1. Moving averages and asynchronous parallel averaging

The Moving Average Paradigm
For i = 1 to ...,
Ave (i) = Average of the most recent 100 EP trials
Moving Average (i) =
Ave (i) + Ave (i − 1) + Ave (i − 2) + Ave (i − 3) + Ave (i − 5)
Note that the moving average display is updated 5 times as often as a traditional average, when calculated this way.

The Asynchronous Parallel Averaging Paradigm
For time t = 1 to ...,
At even-numbered t:
Stimulate left leg
Record from scalp or spine
Update left leg averages and displays
At odd-numbered t:
Stimulate right leg
Record from scalp or spine
Update right leg averages and displays
If t represents tenths of seconds, then each leg is stimulated separately at 5/s.

leagues (1985) allows a new EP to be produced in as little as 8 repetitions of the stimulus. For stimulation at 4/s, this would allow for a new EP to be produced every 2 seconds. Although the mathematical and computational design of such a filter and averaging techniques have already been developed, actual production of the equipment is awaiting the introduction of very fast central processing units (CPU) for intraoperative monitoring or specially designed integrated circuitry that can carry out the complex calculations needed in an acceptably short time. We can, however, look forward to having such advanced averaging techniques for intraoperative monitoring in the future.

Artifacts and Quality Assessment

Artifact Identification and Elimination

The monitoring team needs to beware of a variety of sources of artifact. In general, these need to be understood sufficiently well that the monitoring team can eliminate the source of the artifact whenever possible. Eliminating an artifact at its source is far superior to just attenuating the artifact with filters or taking additional samples to average. There are a variety of artifacts common to the operating room setting. These include muscle, movement, and line noise artifacts, among others. Muscle artifacts are best dealt with by administering neuromuscular blocking agents, when possible. Of course, the new appearance of muscle artifact during an operation may indicate that the patient is anesthetized too

Table 2. Procedures to help decrease intraoperative artifacts

Remove grease and abrade skin before applying disc scalp electrodes.
Glue electrodes down with collodion.
If electrodes are on overnight, re-gel and abrade scalp again in the operating room.
Keep electrode impedances at approximately 2,000 ohms.
Use short electrode wires.
Use short interelectrode distances between pairs of recording electrodes.
Braid the recording electrode wires together.
Have back-up stimulus and recording electrodes available and already in place on the patient.
Keep recording and stimulating wires and cords far away from each other.
Do not cross cables or wires over other cables, especially power cables.
Do not kick, jar, or sway the wires.
Keep the low filter above 1 Hz whenever possible.
Unplug unused equipment.
Avoid appliances with 2-pronged power plugs (ungrounded).
Stop averaging whenever amplifiers are blocking (e.g., after electrocautery).
Adjust sensitivity so that some trials cause artifact rejection.
Use enough neuromuscular junction blocking agents.

lightly. Movement artifacts may be a problem in certain types of procedures, but are obviously less of a problem in procedures performed with an operating microscope. Line artifacts have been discussed already. Table 2 lists procedures that sometimes can help to reduce background noise in the operating room.

Electrocautery devices and other high-frequency, high-intensity devices cause too much electrical noise to allow monitoring during their use. Instead, monitoring devices should be connected to electromagnetic triggers that will discontinue monitoring while these devices are being used. Such an electromagnetic trigger can be placed on the foot switch used by the surgeon to turn on electrocautery or similar devices, or direct connections can be made from the cautery devices to the monitoring apparatus. Some recording amplifiers become completely saturated by high-amplitude artifacts, and will fail to operate normally during the 5 to 15 seconds after each burst of high-intensity artifact. In such cases, when "flat lines" are averaged the amplitude of the recorded EP can be distorted so that they appear much smaller than they really are. The knowledgeable monitoring team will be sufficiently familiar with their own equipment that they can identify these technical problems related to the use of electrocautery or similar devices. Simultaneous display of the raw, single-trial signals is the principal way to identify this problem, as well as a technique of major importance in identification of artifacts in general. Monitoring teams should be strongly encouraged to use equipment that allows the simultaneous observation of the raw, single-trial data and the current averaged EP.

Quality Assessment

Every monitoring team needs to be able to assess the quality of the EP they obtain. Figure 4 shows how such an assessment can be done, by comparing the

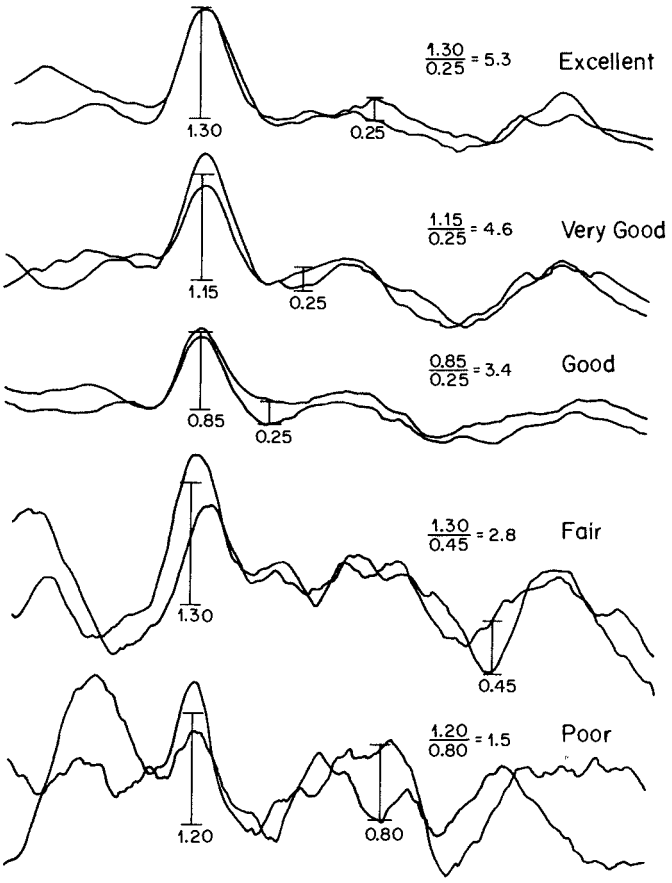

Fig. 4. Assessment of reproducibility or quality of EP obtained using the ratio (quotient) of signal-to-noise. Reproducibility is a key feature distinguishing EP from background noise and artifact. (From Nuwer 1986)

amplitude of the signal to the amplitude of the noise. A scale representing this ratio can be used as a quality assessment tool in critiquing operating room performance (Nuwer 1986). If the quotient of noise amplitude divided by signal amplitude is less than 2, EP quality is poor, i.e. very noisy. If the quotient is between 2 and 3, the EP quality should be considered fair. A quotient between 3 and 4 is good, and between 4 and 5 is very good. Excellent quality is a quotient greater than 5, although it is not unusual to obtain a quotient greater than 10. Those performing intraoperative monitoring should aim for the latter.

In addition to calculating the amplitude ratio of the EP they obtain, the monitoring team should assess the reproducibility of the EP during baseline or stable portions of the operation. One should expect the latency values to vary by at most 5%, and preferably less than 2%. Baseline amplitude measurements for the EP should vary by at most 30%, and preferably less than 10%. However, not everything that is reproducible is an EP. Sometimes artifacts themselves can be time locked to the stimulus, such as the line filter ringing artifacts shown in Fig. 2.

Some stimulus artifacts are also quite time locked. Reproducibility can be considered a necessary feature for good quality EP, but it is not by itself sufficient for identification of an EP. Let the EP reader beware!

During the course of intraoperative monitoring, the monitoring team should be able to track the several variables measuring the quality of the EP: the quotient of noise amplitude and EP amplitude, the latency variability, and the amplitude variability. During the early baseline portions of an operation, these 3 measures certainly would need to be checked. If they are found to be poor, attempts should be made to improve them.

A knowledgeable monitoring team will vary their rates, filters, and recording electrode sites and will eliminate artifacts well enough to obtain high-quality EP during monitoring. This can be done even when EPs are produced every minute or so. When only poor-quality signals can be obtained, the monitoring team needs to do a thorough evaluation of the situation, looking for ways to produce better signal quality. The rapid production of high-quality signals should be a major technical goal in all cases.

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An Automatic Peak Detection and Evaluation Program for Somatosensory Evoked Potential (SSEP) Monitoring

K. SCHWERDTFEGER[1]

Summary

An almost fully automatized program for evaluation of somatosensory evoked potentials (SSEP) is described. It is suitable for intraoperative monitoring of SSEP as well as for continuous recording in the intensive care unit. The principles of peak detection, the reliability of peak amplitude and latency measurements, and possibilities to reduce the complex information of evoked potentials to a few parameters are shown. As yet, there has been little clinical experience; however, in practice, a good applicability of the softeware could be shown.

Introduction

For neurosurgeons who wish to monitor their patients electrophysiologically but whose staffs lack training in monitoring and interpreting results, a computer program that evaluates monitoring results automatically is a necessity. Such a program is mandatory because it is the only way the neurosurgeon can perform both surgical and monitoring tasks. But even when monitoring can be shared among an electrophysiologist and the surgical team, such a program will be of benefit if it provides results more quickly and is more reliable than visual evaluation. Finally, monitoring around the clock, in the intensive care unit (ICU) for example, is greatly facilitated if the results can be condensed and presented in such a way that they can be interpreted by everyone on the ICU staff.

In developing the software for automatic evaluation of monitoring results, we therefore sought to fulfill the following:

a) Interaction with the computer is kept to a minimum so that this interaction can be delegated to inexperienced staff.
b) Only the most important parameters are monitored and only the most useful information is displayed in the results.
c) When the value of a parameter changes, the difference between the previous and present values is checked for significance.
d) Monitoring results are displayed in a nearly self-explanatory way.

The computer program we describe was designed to monitor median nerve evoked potentials, but with some modification it could be applied to monitoring other modalities as well.

[1] Abteilung für Neurochirurgie der Universität des Saarlandes, W-6650 Homburg/Saar, FRG.

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Stimulation and Recording Technique

The median nerve is stimulated at the wrist with an intensity adjusted to evoke a twitch of the thumb. The interstimulus interval is varied randomly between 303 and 363 ms, corresponding to an average stimulus frequency of 3 Hz. Recordings are made from three points: from Erb's point, from the spinous process of the second cervical vertebra, and from the scalp above the somesthetic cortex (i.e., 2 cm posterior to the C3, respectively, C4- location of the international 10–20 system). In all cases a frontopolar reference was used. Either stainless steel needles or small stainless steel skin staples (Schwerdtfeger and Ludt 1986) are used for electrodes. Signals are amplified 100,000 times (Toennies DA IIR) with filter settings of 0.5 Hz to 3 kHz and then fed to a microcomputer (described in the next section).

Digital Processing

The microcomputer system we use (Nase Industrie – Elektronik) is an 8-bit processor with CP/M Plus-Version 3.0. The analog signals are fed to an analog-digital converter with 12-bit resolution and a digitizing rate of 10 kHz per channel. The computer programs are written in Fortran, are written in assembler language for subroutines that handle time-critical parts of the system.

Each sweep is composed of a prestimulus epoch of 12.8 ms and a poststimulus epoch of 51.2 ms. Before averaging, each signal is passed through artifact rejection algorithms to eliminate responses contaminated by noise of large amplitude. The total number of responses that are averaged is adjustable and depends upon the amount of noise contamination. Generally, the signal-to-noise ratio is relatively high when the patient is under anesthesia, so 128 to 256 responses are usually all that need to be averaged. For unconscious but restless patients in the ICU, however, at least 1,024 responses must be averaged.

Eight subaverages are obtained for each cycle, and these are averaged to obtain the grand average, as shown in Fig. 1. The technique of subaveraging has two advantages:

1) When the program is looking for the peaks of an evoked response, a presumed peak can be differentiated from background noise by its reproducibility. In fact, it must be present in at least 6 of the 8 subaverages to be accepted. Noise and perhaps natural variability as well cause the peak parameters (latency and amplitude) to vary among the subaverages. For this reason, an expected latency range is defined (shaded area in Fig. 1) and this range rather than a peak value is used to test for reproducibility of the parameters. The peak detection algorithm itself is very simple and is based on searching for zero-crossings of the first derivative of the signal. However, the presence of residual, especially high-frequency noise and a wide expected latency range may lead to the acceptance of pseudo-peaks. Therefore energy at frequencies with wave lengths shorter than the expected range must be removed from the signal, and this is done by digital filtering. More details concerning software design and a discussion of the disadvantages of digital filtering will be given

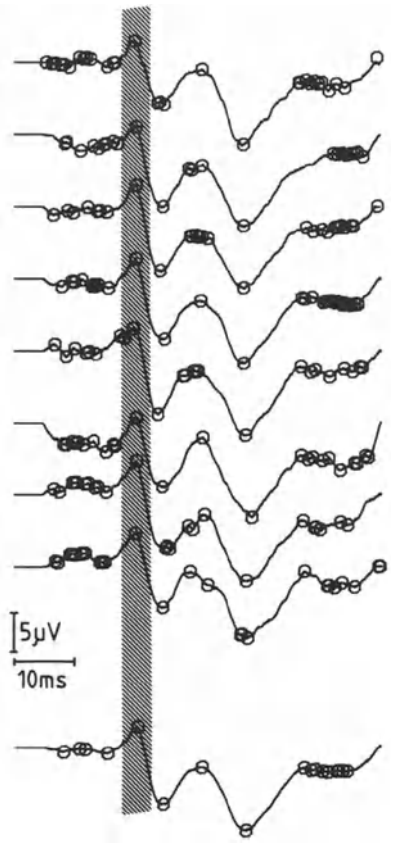


Fig. 1. The principle of automatic peak identification. The cortical SSEP shown – 8 subaverages and the grand average *(bottom tracing)* – resulted from stimulation of the right median nerve and averaging of $8 \times 64 = 512$ sweeps. The maxima and minima (determined mathematically as zero crossings of the first derivative of the signal) are indicated by circles. Each peak of the grand average was tested for reproducibility as explained in the text. The expected latency (range) is indicated by the shaded area

elsewhere (Schwerdtfeger et al., in preparation). What must be noted for the present discussion is that digital filtering may introduce signal distortion and therefore corrections must be made when such filtering is used.

2) While there is variability among the subaverages, as just mentioned, this variability can be quantified, thus allowing us to check differences between subsequent recordings by means of a statistical test, i.e., the Wilcoxon-Whitney-Mann test. This has proved to be a powerful tool when it must be decided whether a peak latency increase occurs by chance (i.e., as a result of severe noise contamination) or is an indicator of beginning brain deterioration.

Clinical Testing

In developing this program, we tested a number of patients. Each patient gave informed consent to the experimental procedure. Four patients were monitored during operations to manage lumbar disc prolapse, 7 patients during open resection of supra- or infratentorial lesions (4 meningiomas, 2 metastases, and 1

angioma), and 2 patients were monitored during carotid endarectomy at the bifurcation.

At the start of each monitoring session we record the reference somatosensory evoked potential (SSEP). For intraoperative monitoring the reference recording is obtained after the anesthetic drugs have reached a steady state and before the operation starts. When monitoring has been continued postoperatively in the ICU, an SSEP recording was obtained the day before the operation and served as the reference recording for postoperative monitoring (Fig. 2). Peaks (marked by an open square in Fig. 2) are automatically identified by the computer program. They must be identified with waves of the cortical primary complex – N_{20} and P_{27} – when monitoring from the cortex, the main spinal component – N_{13} – when recording from the cervical area, or the plexus potential, called $N_{10}e$, which can be recorded at Erb's point. Identification of these peaks is facilitated by numbering so only the corresponding numbers have to be entered. In all subsequent sessions, the computer program will make this association automatically (Fig. 3 A).

The identified waves are used to calculate the following parameters: the plexus-spinal conduction time (PSCT), which is the latency difference between N_{13} and the plexus potential; the central conduction time (CCT); the latency difference between N_{20} and N_{13}; the latency of the cortical P_{27} wave; and the amplitude ratio of N_{20} and N_{13}, called the central amplitude ratio (CAR). The patient's reference and intraoperative values (the latter expressed as differences from the reference values) are depicted on a trend diagram (Fig. 3 B). The trend diagram is displayed

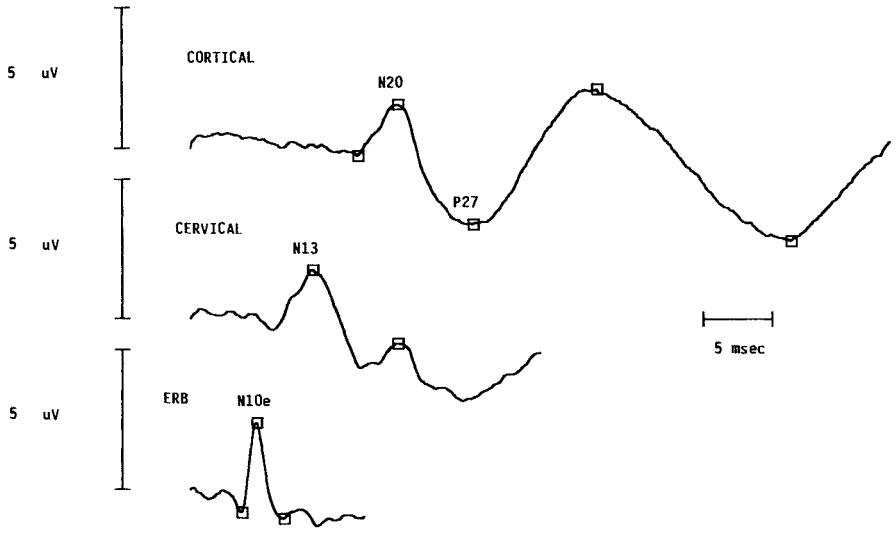


Fig. 2. SSEP recorded the evening before the operation on a patient with a large temporoparietal angioma on the left. The right median nerve was stimulated and recordings were made from Erb's point *(bottom tracing)*, from the spine at C2 level *(middle tracing)* and from the cortex at C3′ *(top tracing)*. The peaks used by the computer program are indicated by open squares. Each is the grand average of 8 × 64 responses

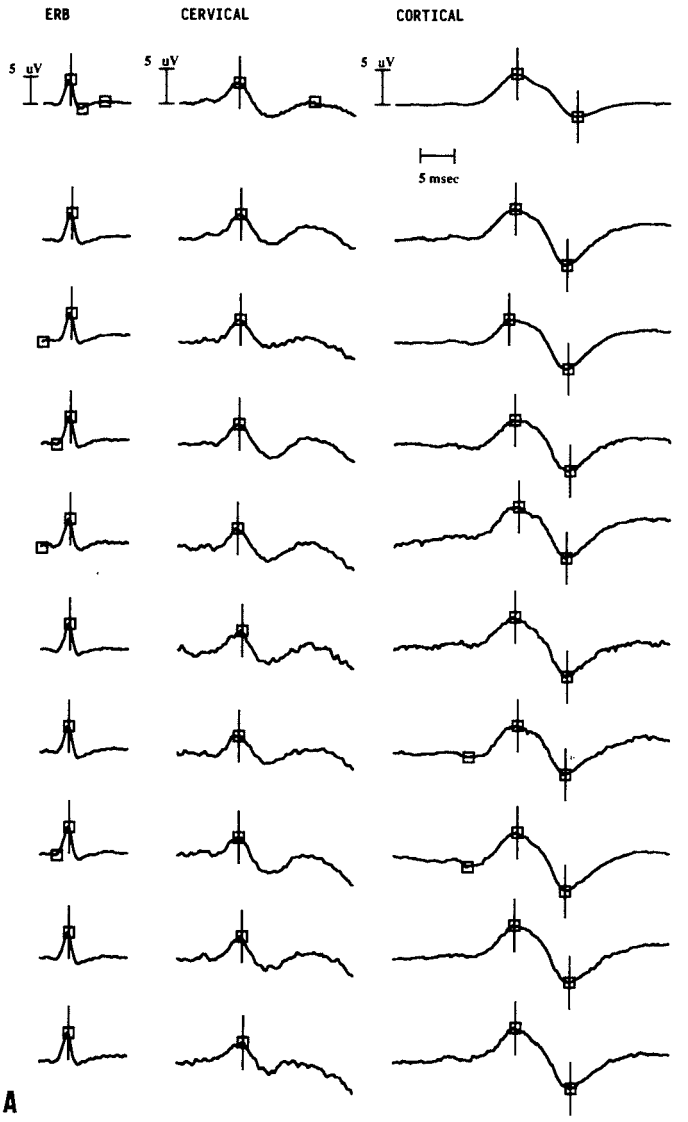
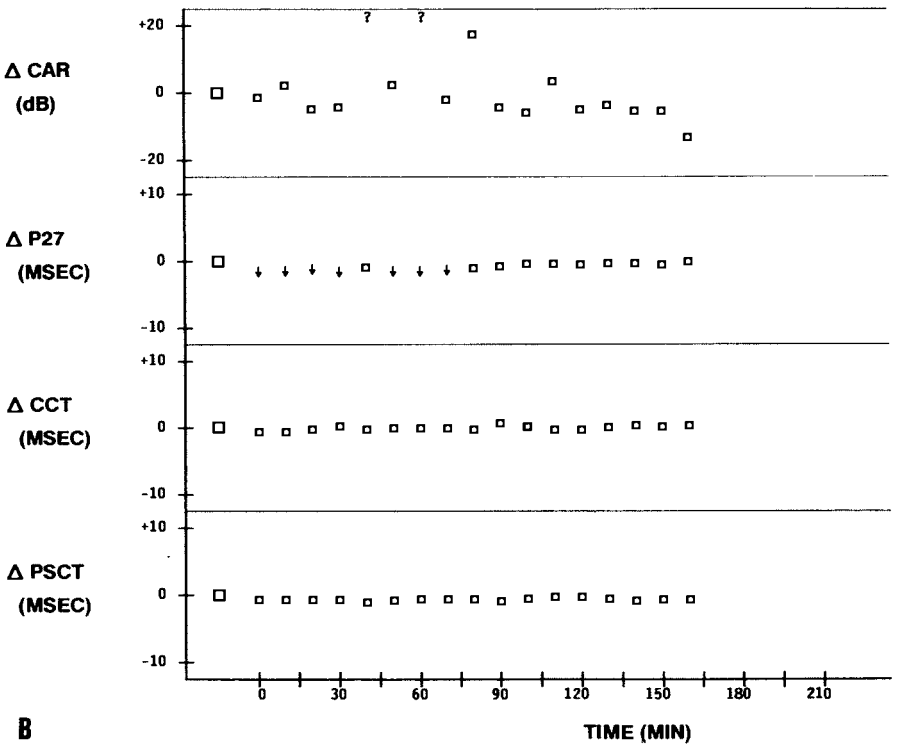


Fig. 3 A, B. Results of an intraoperative monitoring session in a patient undergoing resection of a cerebellar metastasis. **A:** Samples of responses taken at 10-minute intervals, show great stability of the response. The squares indicate the automatically identified peaks, and the vertical bars delineate those peaks needed for computation of the trend diagram in B. Each SSEP is the grand average of 8×32 (= 128) sweeps. **B:** Trend diagram, which is shown to the surgeon on a separate monitor. Plexus-spinal conduction time *(PSCT)*, central conduction time *(CCT)*, the latency of wave P_{27}, and the central amplitude ratio *(CAR)* are shown. The large square at the left of each row is the reference value; smaller squares represent intraoperative values not significantly different from the reference, arrows represent values which significantly differ, and question marks represent values that could not be calculated.



intraoperatively on a separate monitor that can be viewed by the surgeon. Different symbols are used to convey information on the trend diagram: the squares indicate that the value does not differ significantly from the control value, the arrows show statistically significant deviations from the control value, and the question marks indicate that these values could not be calculated. Thus, simply by glancing at the monitor, the surgeon can remain informed of the results of the monitoring. If values for a parameter are repeatedly missing, this will be signaled by an acoustic alarm, as well.

Results

A limited number of observations are available because these computer programs have been used to monitor only a small number of patients to date. During intraoperative monitoring we noted only completely uneventful courses such as that shown in Fig. 3. The patient whose results are shown in Fig. 3 underwent resection of a cerebellar metastasis. Fig. 3 B shows a statistically significant decrease in P_{27} peak latency, something we have seen occasionally in other patients, as well. This decrease is thought to reflect changes in the depth of anes-

thesia, which affect especially P_{27} and later cortical waves. Such decreases in latency are considered to be harmless and are therefore neglected. Postoperatively, however, this same patient developed severe complications and finally died, probably – because a postoperative CT-scan showed no abnormalities – as a result of brainstem infarction. Regrettably, an autopsy was refused. Fig. 4 shows recordings made during the postoperative course of this patient. Early deterioration is evidenced by increasing CCT and decreasing CAR. Because artificial ventilation was continued in the first hours after this patient's operation, the effects of anesthetic agents were not fully reversed and the patient's neurological status could not be assessed well by clinical examination. Thus, SSEP monitoring was the only way to detect the complication at that time.

Discussion

The design of the computer program we describe here was influenced by our intent to develop a practical tool that could be used even by non-experts in neurological monitoring after minimal instruction. To do this, it was necessary to extract the most important information from the complex pattern of the SSEP. Examination of the literature on evoked potential monitoring showed that most authors prefer to measure peak latencies and amplitudes or to calculate interwave latencies such as that represented by CCT (Markand et al. 1984; Prichep et al. 1985; Hume and Durkin 1986; Symon et al. 1986, Friedman et al. 1987; Little et al. 1987). In comparing the accuracy with which the trend parameters depicted in Fig. 3 (PSCT, CCT, P_{27} peak latency, and CAR) with the "global" evaluation procedures such as cross-correlating subsequent measurements (Walser et al. 1987), predicted neurological status it was found that the trend parameters are much more representative of the integrity of the central nervous system (Schwerdtfeger – unpublished results).

Other have developed automatic evoked potential evaluation algorithms (Billings 1981; Fridman et al. 1982; Boston and Deneault 1984; Boston et al. 1985; Bertrand et al. 1987). The present computer program, when compared with those developed previously, has the advantage that differences between the records are tested for significance. This is an important point, because recording conditions may vary considerably during a monitoring session, resulting in dramatic changes in the response. Increases in CCT of up to 2 ms, for example, occurred during our study. These were shown to be insignificant and were caused by alterations in background activity during recording. The variations we have seen to date have been temporary, and the patients' clinical courses have shown them to be harmless.

The technique of subaveraging allows one both to detect peaks and to assess their variability – prerequisites for statistical testing – and is essentially the same process used in visual evaluation in which evoked potentials are recorded several times to check the reproducibility of the presumed peaks. The computer can do this more often, more exactly, and – with a computation time of about 20 seconds, including the calculation of interwave latencies and the statistical tests –

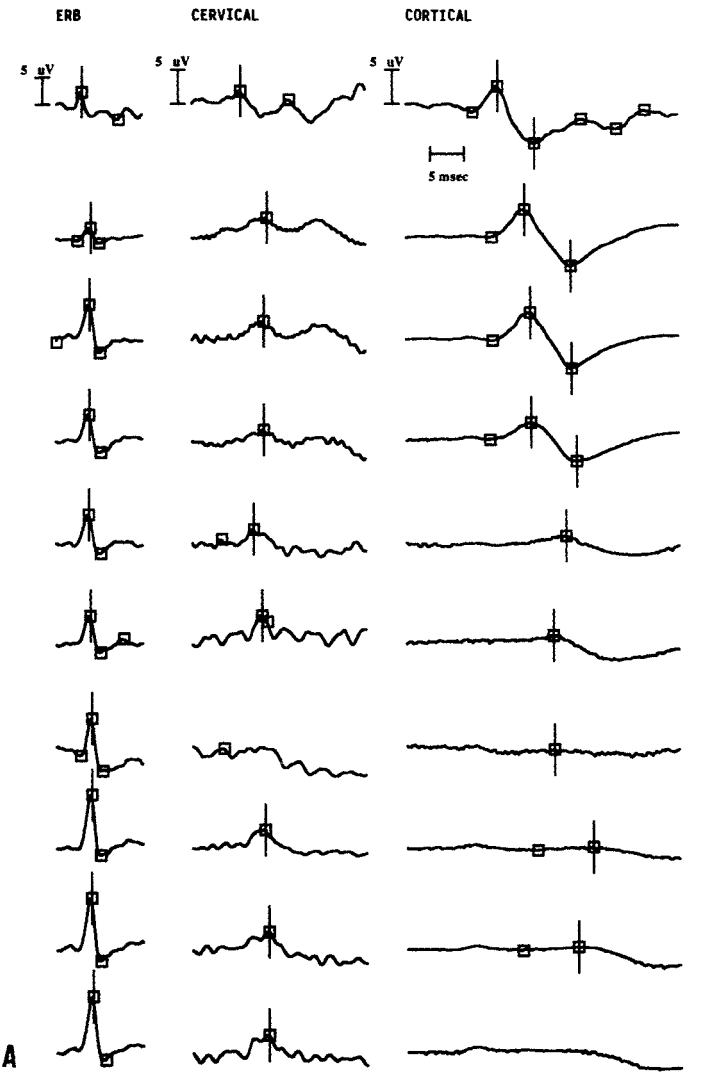
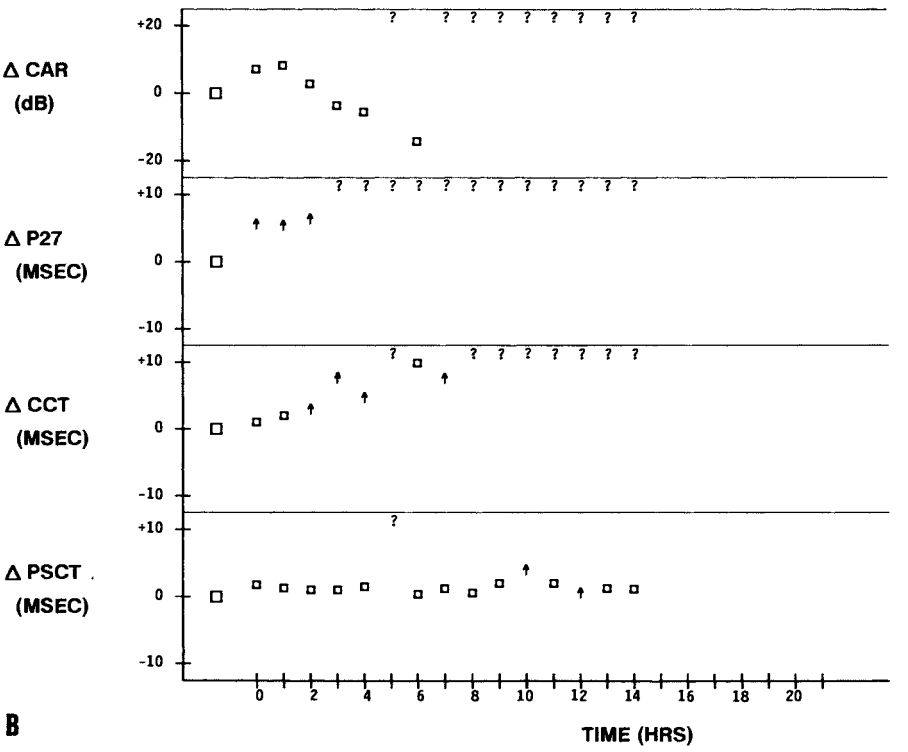


Fig. 4. Recordings made during the postoperative course of the patient whose intraoperative monitoring results are shown in Fig. 3. SSEP were recorded every half hour. **A:** The responses show a sudden deterioration and finally loss of the cortical SSEP. **B:** Corresponding trend diagram. Note that the decrease in CAR closely followed the increase in CCT and P_{27} latency; however, due to the great variability in CAR the differences did not reach statistically significant levels.



much more quickly even than an experienced electrophysiologist. Each subaverage must be only a rough estimate of the signal, and because in patients under anesthesia some noise sources such as EMG are suppressed, it is possible to run the program intraoperatively with 16 sweeps per subaverage, corresponding to 128 sweeps for the grand average. Intraoperatively, data can be obtained and computations performed to provide a fully evaluated record every 70 to 80 seconds. Postoperatively, there is usually more noise contamination, so more sweeps per subaverage are required. To limit the discomfort of the patient under study, the SSEP recording cycle was repeated only every half hour during this period. Nevertheless, this regimen seems to provide sufficient information for monitoring.

Finally, the limits of the computer programs should be discussed. Naturally, the major limiting factor in this automated system is the quality of the input signals. Sometimes the pathological process that has necessitated operation severely impairs the response and renders peak detection very difficult. However, even in recordings of good quality we have found 10 percent false-negative results, meaning that a peak present in the recording was not picked up by the automated monitoring program. Such failures are even more likely to occur in intraoperative monitoring than outside the operative setting. It seems that bipolar coagulation

during surgery is the cause of most of these failures. Recording during cautery usually is not possible, but coagulation sometimes induces an amplifier block that lasts several seconds and introduces several blank subaverages. This interferes with the reproducibility test. Despite these difficulties, however, our experiences with these computer programs are encouraging, and we believe that the software may contribute to a wider acceptance of evoked potential monitoring.

Acknowledgement. The help of Dipl.-Ing. H. Ludt in developing the software and of Prof. Dr. F. Loew in providing critical but encouraging advice is gratefully acknowledged. My special thanks to Prof. Dr. J. Schramm and Prof. Dr. R. Fahlbusch for the chance to present this paper at the Third International Symposium on Intraoperative Neurophysiologic Monitoring. This work was partly supported by a grant from the Kuratorium ZNS.

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Automatic Identification of Peak V in Brainstem Auditory Evoked Potentials

A. R. MØLLER[1]

Summary

The need for automatic interpretation of evoked potentials for use in intraoperative monitoring and for monitoring of critically ill patients in intensive care units is discussed. A computer algorithm for automatic identification and continuous tracking of peak V of brainstem auditory evoked potentials (BAEP) is described.

Intraoperative monitoring of evoked potentials demands continuous vigilance from the person who is conducting the monitoring. In many cases, although the recorded potentials show little change for many hours, nevertheless the person who performs the monitoring must respond promptly and correctly to those rarely occurring but sudden changes. The pool of personnel capable of meeting the requirements for intraoperative monitoring is limited, and there is therefore a need to automate the interpretation of intraoperatively recorded evoked potentials. That requirement is even greater when these techniques are used for monitoring in the intensive care units, where the events that require any action typically occur even more infrequently than they do in the operating room, yet the necessity for an appropriate and rapid response is just as important to the patient as it is in the operating room. The need for automated interpretation of responses applies to several modalities of evoked potentials.

In addition to being useful in monitoring the integrity of the specific neural pathways of a particular sensory system, sensory evoked potentials have been found useful in more general ways. Thus intraoperative monitoring of brainstem auditory evoked potentials (BAEP) has increasingly been found useful in operations where the integrity of the brainstem may be compromised. Monitoring of BAEP is therefore now routine in many neurosurgical operations. The usefulness of this method has become evident as well in the area of intensive care.

While it is realistic to assign qualified personnel the task of interpreting BAEP during neurosurgical operations that may last 5-10 hours, it is questionable whether it is possible to arrange to have such trained personnel present around-the-clock to interpret the recorded evoked potentials in the intensive care units. Because requiring such personnel to be present 24 hours each day is expensive,

[1] Department of Neurological Surgery, University of Pittsburgh School of Medicine, 230 Lothrop Street, Pittsburgh, PA 15213, USA.

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and also questionable because of limited resources, there is a need to develop computer programs that can interpret the complex patterns of evoked potentials such as BAEP and somatosensory evoked potentials (SSEP) and that can provide the essential information in a form that does not require skill in interpreting evoked potentials to understand and act upon.

We show in this chapter that it is possible to accomplish the goal just presented by appropriate pre-processing of the evoked potentials and then applying algorithms for automatic identification of the peaks that appear in the recorded waveform, the latency and amplitude of which are of clinical importance. Quality control of the recorded waveform is used in the determination of whether the recorded waveform is suitable for automatic processing.

We describe the use of these techniques for continuously tracking peak V of the BAEP, but similar methods can be implemented for automatic interpretation of other modalities of evoked potentials such as SSEP. Such methods are of value in situations where it is beneficial to monitor the BAEP over long periods of time (days), but where the conventional way of monitoring BAEP would be prohibited because of costs and scarcity of qualified personnel.

Description of an Automatic Peak V Detector for BAEP

We have earlier shown that aggressive spectral filtering using zero-phase digital filters can reduce the noise level of BAEP (Møller 1988a), and thus reduce the time that is necessary for averaging a sufficient number of responses to obtain an interpretable record. These filters also enhance the peaks in the BAEP in such a way that manual interpretation has been made easier. When such filtering is implemented it is also possible to identify individual peaks in the BAEP without human intervention (Møller 1988a). Such filtering thus makes it possible to achieve a high-quality record, which builds a base for the use of computer programs that automatically identify the various peaks in these records and determine the latencies of these peaks.

The algorithms used for digital filtering and peak detection are the same as those that have been in use in our clinic for more than 10 years (Møller 1988a). The strategy for identification of peak V in such enhanced records was developed and tested using recordings of BAEP that were obtained during routine intraoperative monitoring as well as in volunteer subjects.

The BAEP were recorded differentially between electrodes placed on the vertex and the contralateral earlobe and the responses were elicited by monaurally presented clicks of about 105 dB PeSPL at a rate of 20–30/s. The potentials were amplified 50,000x using filter settings of 10 Hz high-pass (6 dB/octave rolloff) and 3,000 Hz low-pass (12 or 18 dB rolloff) and averaged. The recorded potentials were sampled at 40-μs intervals and 256 samples were taken after the presentation of the stimulus. Traditional artifact rejection, based on a set amplitude, was used. If any of the sampled points exceeded that value, the entire response was discarded. If more than 10 successive responses exceeded that value, all recorded data were discarded for 2–5 seconds after the amplitude was again within the set

limits. This assured that the amplifiers had recovered from being overloaded by the artifact (Møller 1988b).

In addition to ordinary averaging, an average of the responses with every other response being inverted was obtained. This procedure of adding every other response and the inverse of the other responses cancels the response but leaves the noise that is uncorrelated with the stimulus unchanged (Hoke et al. 1984; Elberling et al. 1985). This ± average is therefore a measure of the noise level because the stimulus-related response is cancelled. The ratio between the root mean square (RMS) value of the ordinary averaged response and that of this ± average will normally increase as the number of responses that are included increases. Thus, this ratio can be used to determine when a satisfactory averaged response has been obtained. When implemented for automatic tracking of peak V, this ratio determines when a sufficient number of responses has been collected to obtain a reliable average.

When 500 responses had been added the averaged responses were digitally filtered. The filter used for filtering both the ordinary averaged responses and the ± average had an impulse response, the shape of which was a smoothed W (the W-50 filter; Møller 1988a), resulting in a bandpass characteristic. This filter enhanced peaks I, III, and V in the BAEP. If the ratio between the RMS value of the averaged response and the RMS value of the ± average was less than a set value, another run of 500 responses were added and the same processing was repeated. When the RMS values reached or exceeded the preset value the process was stopped and the individual peaks were identified.

The algorithm used to identify individual vertex-positive peaks was detected when the change in amplitude of the response changed sign, but in order to avoid mistaking small peaks that were caused by noise for real BAEP peaks the following constraints were introduced: 1) the difference between each of 6 adjacent samples prior to the one where the sign changed must have been (vertex) positive, and 2) the difference between the 6 adjacent samples that followed the one where the sign changed must have been negative. If these criteria were not met, the search for peaks continued from the point where the sign of the filtered average response first changed.

When recording was begun, a baseline was obtained by adding 3 averages, each of which fulfilled the set criteria for acceptability based on the value of the ratio between the root mean square (RMS) value of the regular averaged response and that of the ± averaged response. Peak V was then identified as the peak with the largest amplitude between 4.5 and 8.5 ms after the stimulus. In order to allow for slow changes in the latency of the selected peak, the baseline was constantly updated, so that the current baseline thus became the average of the 3 previous averages. It was, however, the deviation from the latency of peak V of the original baseline that was displayed.

Peak VI has a larger amplitude than peak V in some patients, and in some few instances the computer program tracked peak VI instead of peak V. In order to avoid the program alternating between tracking peak V and peak VI in cases where these 2 peaks have similar amplitudes, an additional algorithm was included. This algorithm detected the difference between the latency of the

selected peak and updated baseline. If this difference was more than 0.9 ms, the peak with the latency closer to the baseline was sought. If such a peak was found, that peak was selected, even though it might have a lower amplitude than the originally selected peak. If no such peak was found, the peak with the highest amplitude was selected, even if it had changed more than 0.9 ms since the last run.

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Some Common Problems and Pitfalls in Intraoperative Monitoring of Evoked Potentials

A. R. MØLLER[1]

Summary

This chapter reviews and suggests remedies for some of the common problems and pitfalls that may be encountered when sensory evoked potentials are recorded in the operating room to monitor the auditory and somatosensory system, and when electromyographic potentials are recorded intraoperatively for monitoring of cranial motor nerves.

In a new discipline such as that encompassing the use of evoked potentials in intraoperative monitoring, problems and pitfalls are many. Therefore this chapter naturally cannot provide complete coverage of what can go wrong in the operating room and how recordings can be misinterpreted, but it must instead focus on a small sample of the problems that can be encountered by the neurophysiologist performing intraoperative monitoring of evoked potentials.

General Problems and Pitfalls

Equipment failure is a problem that can never be eliminated totally, but most modern equipment has a very low rate of failure. The most important factor in managing equipment problems successfully is fast, effective trouble shooting. This requires in turn that qualified, experienced personnel be responsible for intraoperative monitoring. The availability of such personnel is a prerequisite for efficient resolution of technical problems, whether they be equipment failures or the appearance of interference. Only individuals with extensive knowledge and experience can identify and correct errors made in setting up the recording equipment or can replace a particular failed piece of equipment properly. Often, there is some redundancy in modern equipment, and this permits the neurophysiologist to substitute one amplifier or stimulator for another in the equipment, but it is normally important to have appropriate backup equipment available. In addition, the backup equipment must be readily available in the operating room if it is to be of any value because the time that is available for making equipment substitutions is usually very short. It is always helpful to check the equipment well

[1] Department of Neurological Surgery, University of Pittsburgh School of Medicine, 230 Lothrop Street, Pittsburgh, PA 15213, USA.

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before the start of the operation and to have spares of cables that are used to connect the patient to the equipment in the operating room, because these are the parts of the equipment that are most likely to fail.

Failure to obtain adequate recordings because equipment is not connected or is not switched on, or because dial settings are in error, can largely be eliminated by developing and using check lists for all typical situations. Check lists are used routinely in other fields such as aviation, where many tasks must be performed precisely and in sequence, and check lists can likewise serve as important aids in carrying out the tasks of intraoperative monitoring. When properly designed and followed, such check lists can eliminate many surprises in intraoperative monitoring, and are also very helpful in training personnel. We have therefore adopted the use of check lists in intraoperative monitoring of evoked potentials.

Sensory Evoked Potentials

Intraoperative monitoring is usually not started before the patient is anesthetized, and there is therefore not much time available to determine the cause of any failure to obtain satisfactory recordings; there is usually even less time available to correct any problem that may be identified. Thus, it is important that as many potential problems as possible are eliminated by appropriate planning for intraoperative monitoring.

A major problem is failure to obtain a response, when the appropriate stimulus is applied, from the system that is to be monitored in the operating room. There can be several reasons why it is not possible to obtain a response to a sensory stimulus when intraoperative monitoring of auditory evoked potentials or somatosensory evoked potentials (SSEP) is attempted. One reason is that the evoked potential(s) in question is not present before the operation. The absence of the response of interest can be related to a number of factors, but for the sake of intraoperative monitoring the cause is of minor importance. What is, however, imperative is that it is known before intraoperative monitoring is attempted that the response cannot be obtained or that it is impaired. Patients who are to undergo intraoperative monitoring of evoked potentials should therefore undergo appropriate studies preoperatively to ensure either that the type of evoked potential that is to be monitored intraoperatively is obtainable within normal limits or that the neurophysiologist is prepared before the operation to interpret intraoperative changes based on abnormal baseline potentials. If the evoked potentials in question cannot be obtained at all preoperatively there is little chance that they can be obtained intraoperatively. Before one engages in intraoperative monitoring of brainstem auditory evoked potentials (BAEP), it is thus important that the hearing of the patient in question is tested and that a preoperative BAEP recording is obtained. It is also important that the ear canals of the patients are examined and cleared of cerumen, if necessary, before the operation. If cerumen is present in the ear canal and insert earphones are to be used in the operating room, the cerumen may be pushed into the opening in the earphone and obstruct the sound.

Similar procedures should be followed routinely when other sensory modalities are monitored intraoperatively with recorded evoked potentials. Thus if SSEP are to be monitored intraoperatively, SSEP should be obtained preoperatively from the patient with the same recording and stimulation parameters as will be used intraoperatively.

One serious pitfall to avoid when monitoring BAEP during microvascular decompression (MVD) operations or during removal of acoustic tumors is monitoring the wrong component of the neural system at risk. Thus if the intracranial portion of the auditory nerve is at risk, one should not record from the ear, because the ear is distal to the portion of the auditory nervous system that may be injured. For instance, the auditory nerve can be injured severely during operations to manage acoustic tumors or MVD operations involving cranial nerves V, VII, VIII, or IX without any noticeable change occurring in the auditory nerve potentials that can be recorded from the nerve's distal portion, as is done when recording from the ear using the technique of electrocochleography (ECoG). This method, however, is perfectly adequate to detect impairment of the blood supply to the cochlea, such as could occur in an operation in the cerebellopontine angle (CPA) and which would naturally place hearing at risk (Levine et al. 1984; Ojemann et al. 1984).

Another situation in which the wrong portion of the system that is at risk is being monitored is stimulating the ear that is opposite to the side where the operation is being carried out. This can easily be done by mistake when both ears are equipped with earphones and when the potentials being monitored are BAEP recorded differentially between earlobe and vertex. This is because the BAEP recorded in the way just described are only slightly different when the opposite ear is stimulated. When earphones are placed in both ears, and the ear that is to be stimulated is selected by selecting a setting on the stimulus equipment, there are several ways in which the wrong ear can be selected to receive the stimulus. For example, the earphones might have been mistakenly switched – the left earphone could have been placed on the right ear – or if it is not clearly marked on the equipment which ear is to be stimulated, the ear opposite to the side being operated upon can be stimulated in the belief that it was the ipsilateral ear. The consequences of such mistakes can be catastrophic in that the auditory nerve can be injured and even severed without any change resulting in the BAEP because it is the opposite ear that is being stimulated. (Naturally, this is not a problem when one is recording from the eighth nerve directly.) It has been recommended that earphones be positioned routinely in the ear on the opposite side to facilitate checks of the equipment in case the response from the ipsilateral side should disappear. We feel that this introduces an unnecessary extra risk of mistaking the ear to be stimulated, and so, for operations that affect only one side (for example, operations in the CPA), we do not place an earphone in the opposite ear. If the purpose of the monitoring is to assess function of the brainstem and midbrain, we will place earphones in both ears but we will also take measures to help assure that the appropriate side is stimulated, including checking the sound after the earphones are placed in the ear and marking very clearly the switch used to select the earphone.

A similar situation can occur when recording SSEP for the purpose of monitoring neural conduction on one side. In these cases we use the same precautions as just discussed for monitoring BAEP.

One serious pitfall in connection with monitoring sensory systems is choosing an inappropriate stimulus. This problem arises in connection with monitoring visual evoked potentials (VEP). It is known from clinical studies that a reversing checkerboard pattern is the best stimulus to use in detecting pathologies affecting the optic nerve and optic tract. However, this type of stimulation cannot be used in the operating room because it requires that a pattern be focused on the retina of the patient. Therefore VEP are monitored intraoperatively using flash stimulation. However, it has been shown in the clinical setting that flash stimulation, although it produces a clear response, is unreliable in detecting injuries (Chiappa 1983). It has also been verified (Cedzich et al. 1988) that the changes in flash-evoked VEP observed intraoperatively are poorly correlated with postoperative visual function. This is just one example of how the use of an inadequate stimulus in intraoperative monitoring may lead to an incorrect assessment of intraoperative injuries caused by surgical manipulations.

Another problem that may occur during intraoperative monitoring of BAEP is accidental reduction in the intensity of the sound stimulus caused by fluid infiltrating the earphone. When earphones (such as miniature stereo earphones) that are placed directly in the ear become wet, a large stimulus artifact will appear in the recording, because electrical current from the earphone will leak to the body of the patient, where the electrical potentials that are generated by this current are then picked up by the recording electrodes. To protect the earphones used in the operating room from getting wet during the operation, the earphone must be sealed watertight in the ear (Møller 1988a).

The anesthetic regimen most often used has practically no influence on BAEP, but if the patient's body temperature drops, then the latencies of the various components of the BAEP become prolonged. It is therefore important for the neurophysiologist to be aware of the patient's temperature in order to interpret intraoperative BAEP correctly. Because it is usually the long-latency components of the SSEP that are observed intraoperatively, and because these potentials are affected by agents used in anesthesia such as inhalation anesthetics and barbiturates, deviations from the expected anesthetic regimen may cause changes in the latencies and amplitudes of these components of the SSEP that are used in intraoperative monitoring. The amplitudes of these components may decrease to values that are no longer discernible. These changes are similar to those caused by surgically induced injuries, and if the neurophysiologist is not aware of such changes in the anesthesia regimen, the changes produced in the recorded SSEP could be mistaken for surgically induced injuries that warrant modification in the surgical procedure.

Another common source of problems in intraoperative monitoring is related to the use of signal averaging for improving the signal-to-noise ratio of sensory evoked potentials. When an averaging technique is used to enhance a signal in noise, all the responses that are averaged must be identical and occur with the same latency. If the responses that are averaged (added) are not identical, the

waveshape of the averaged potentials may not resemble the shape that the response had at any time during the averaging. If the latency of the recorded potentials changes, the amplitude of the averaged response will be smaller than it would normally be if all responses had occurred with the same latency. This may lead to the erroneous interpretation that the waveform of the recorded potentials changed, when, in fact, it was the latency that changed. This situation often arises when evoked potentials are averaged during intraoperative monitoring and the potentials change as a result of surgical manipulations (Møller 1988a).

These problems can best be reduced by keeping the time over which responses are averaged as short as possible. One way of accomplishing this is to use optimal spectral filtering in order to reduce the number of responses that need to be averaged to obtain an interpretable record (Møller 1988a, b). The use of the highest possible stimulus rate that does not cause a noticeable reduction in the response amplitude or in the waveform of the response is also of great help in obtaining an interpretable record in as short a time as possible. Another effective way to avoid the problems with signal averaging just discussed is to select potentials that have larger amplitudes than the traditional farfield potentials used for monitoring. For example, compound action potentials (CAP) recorded directly from the eighth nerve (Møller and Jannetta 1983; Møller, 1988a) can be used to monitor neural conduction in the auditory nerve. Such potentials have large enough amplitudes to be interpreted without averaging or after adding only a few responses. Similarly, recordings from the exposed spinal cord contain potentials of similar amplitude and thus provide instant information about neural conduction (Lueders et al. 1982, 1983).

If a background noise of a periodic nature appears, and stimulation is performed at a constant rate, there is a risk that the periodic noise will be increased rather than decreased by the process of signal averaging. This can occur when the periodicity (frequency) of the noise is a whole-number multiple or fraction of the frequency of stimulus presentation. The periodic interference signal will then not be reduced by averaging many responses, which can give a false impression of the presence of a strong evoked potential. This problem can be remedied by changing the stimulus rate, or by making the rate of stimulus presentation vary slightly in a random fashion around its selected value (see Møller 1988a). By doing so, interference that has periodic components will not add in phase, as the components will if the stimulus repetition rate is constant and a whole-number fraction of the periodic interference components.

It is well known that the use of notch filters to reduce interference from the power line can create artifacts that may be mistaken for a real response. Therefore, notch filters should never be used in intraoperative monitoring of evoked potentials.

Monitoring Cranial Motor Nerves

Another chapter describes how monitoring of specific cranial nerves can be useful in a variety of different operations and how this can be done with the aid of

recording electromyographic (EMG) potentials from the muscles that are innervated by the nerves in question (Møller, this book, see Chapter 1, page 8). Such monitoring usually also involves electrical stimulation of the respective nerves intracranially. The problems that can make such monitoring troublesome are not usually related to electrical interference, as is the case for sensory evoked potentials. This is because the EMG potentials are usually of much larger amplitude than are sensory evoked potentials. The problems most often encountered during such monitoring are rather those of inability to apply a stimulus large enough to obtain a response without injuring the nerve and of anesthesia practices that interfere with monitoring (Møller and Jannetta 1984; Sekhar and Møller 1986; Møller 1987, 1988a).

A rather obvious reason for failure to obtain EMG potentials is paralysis of the patient. If intraoperative monitoring is being used to identify parts of a tumor in which there is no motor nerve present so that the tumor can be removed safely, then paralysis of the patient would be a serious problem because no potentials could be recorded from the paralyzed muscles, even with direct motor nerve stimulation. In such cases monitoring would fail to identify a motor nerve, and serious injury to the nerve would most likely occur without being noticed. This problem can occur because the anesthesia team was not informed of the need to use an anesthesia regimen that did not include paralyzing agents, or it may occur if there is a change in the anesthesia team and that information is not conveyed to the next team. Such problems usually do not occur when there is a good working relationship between the monitoring team and the anesthesia team.

Finally, problems can arise in intraoperative monitoring when one is recording from muscles and the recording electrodes pick up potentials that are generated by muscles other than those from which recording is intended (Møller 1988a). This can happen more easily when the distance between the two electrodes from which differential recordings are made is large. One such instance, which can arise when recordings are made from large groups of muscles, is presented in another chapter in this book (Møller, Chapter 1, page 9) in connection with recording from facial muscles to monitor facial function during removal of acoustic tumors. In this case, the response from the mastication muscles may be mistaken for a response from facial muscles. Similarly, when intracranial electrical stimulation is used to identify the facial nerve, the trigeminal motor nerve may be mistaken for the facial nerve.

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A Program for Automatic Brainstem Auditory Evoked Potential and Somatosensory Evoked Potential Monitoring Using the Pathfinder Software

E. WATANABE[1] and J. SCHRAMM[2]

Summary

A software program for the Nicolet Pathfinder was developed that allows semi-automatic monitoring of brainstem auditory evoked potentials (BAEP) and, alternatingly, somatosensory evoked potentials (SSEP) during posterior fossa surgery. A second offline program is described which connects the Pathfinder to an IBM PC-type personal computer, allowing for rapid plot-out of closely spaced curves with comments and without overlap. Both programs greatly facilitate monitoring in the operating room.

In an effort to protect neuronal functions during infratentorial surgery, such as operations to manage acoustic neurinomas or meningiomas in the cerebellopontine angle (CPA) and microvascular decompression (MVD) operations of cranial nerves V and VII, we have monitored the auditory pathways simultaneously with brainstem auditory evoked potentials (BAEP) (Schramm et al. 1989; Watanabe et al. 1989b) and somatosensory evoked potentials (SSEP) (Watanabe et al. 1989a). From our experiences, monitoring both types of potentials simultaneously provides neurosurgeons with much important information both intraoperatively and postoperatively (Schramm et al. 1989), including: 1) information necessary to make a prognosis regarding possible neuronal damage, 2) information that enables the surgeon to learn more about the effects on nerves of surgical manipulations, and 3) warnings that neuronal damage is occurring before the damage is irreversible. Because operations during which monitoring is performed may take quite a long time to complete, and because the monitoring requires intense concentration on the part of monitoring personnel, performing intraoperative monitoring can be very tiring to the monitoring personnel. To free these personnel for more sophisticated activities such as analyzing data waveforms, it would be useful to minimize the number of keystrokes and switches the monitoring personnel have to use.

Repetitive tasks are performed well by computers, and with recent advances in microprocessor technology, considerable improvements have been achieved in the design of averaging hardware so that this hardware can perform all switching

[1] Department of Neurosurgery, Tokyo Metropolitan Police Hospital, 2-10-41 Fujimi, Chiyoda-ku, Tokyo, Japan
[2] Department of Neurosurgery, University of Erlangen, Schwabachanlage 6, Erlangen, FRG

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functions necessary to intraoperative monitoring. We used Nicolet Pathfinder I hardware to develop a semiautomatic averaging program that guides monitoring during infratentorial surgery. This chapter presents the main functions of this software and discusses the future of this system, including important points to be taken into consideration in the design of automated monitoring equipment. Our discussion focuses on the technical aspects of this issues.

Methods

The system we developed consists of two parts. One is an on-line monitoring control program and the other is an off-line print-out routine.

On-Line Routine

Figure 1 shows how the computer program and operator interact. As may be seen from the diagram, the operator is required to select the next modality. As we usually monitor BAEP and SSEP alternately during surgery, the selection should include right and left BAEP and SSEP. For BAEP, the operator should also select the stimulation modality – i.e., "rarefaction" or "condensation" clicks. The operator must choose 1 of 6 alternatives by entering 2 characters, such as RR for right BAEP with rarefaction clicks or LC for left BAEP with condensation clicks. During the averaging procedure the computer requires no further attention or action from the monitoring personnel. Thus, the neurophysiologist can concentrate on interpreting the recorded potentials and monitoring the surgeon's video display of the operative field. After 2 averaging runs have been completed the neurophysiologist points the cursor to wave I, III, V, or N_{20} on the screen. The computer prints out a hardcopy of the time, modality, and wave latency for filing with the operative report and then sets the parameters for the next modality that the operator has selected.

The computer accepts 1) "next mode" or 2) "comment" for current data during averaging. As these functions are performed in background mode, there is no time lost from averaging, which proceeds in foreground mode. This method allows the operator more time to enter information and provides the results of 1 averaging run in less time than when standard procedures are used.

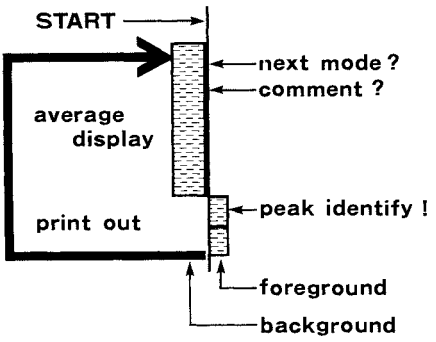

Fig. 1. Time schedule of computer-operator interaction

Off-Line Routine

The recorded potentials and other information pertaining to the actual recording are transferred in digital form through an RS 232-C line to a personal computer (IBM-PC type computer) that we connect to the Pathfinder I. The program to send the data is written in FORTRAN, and the receiving routine is written in BASIC. The IBM-PC plots out the waveform, placing successive waves close to each other but avoiding overlap of waves (Fig. 2). The routine we have just designed standardizes each monitoring session, making it easier to gain an over-

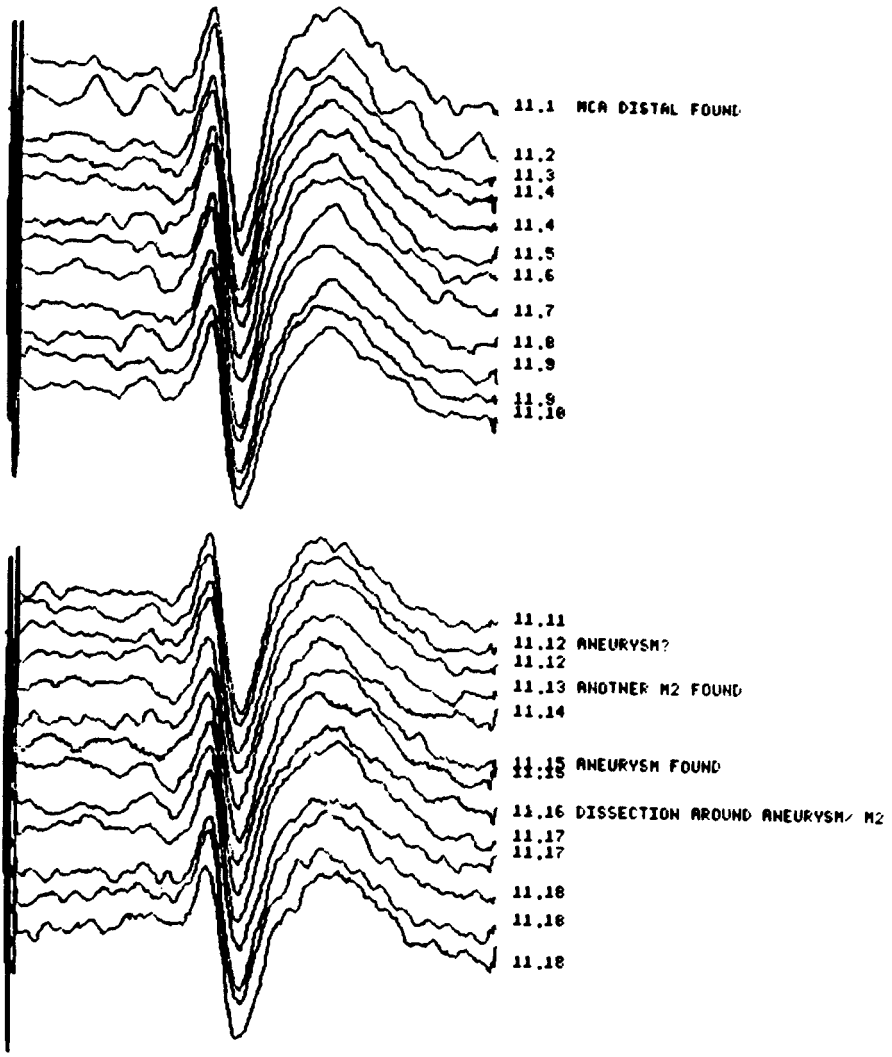

Fig. 2. Typical print-out obtained with the Pathfinder I-to-PC print-out program

view of longterm trends in evoked potential changes. The print-out mode may be used in the operating room during or between lengthy or difficult portions of the operation. Having the information available in graphic form greatly enhances our ability to recognize alterations in potentials.

Discussion

We decided to include the "next mode" feature rather than pre-programming all functions because the type of monitoring required will be different depending upon the operative procedure being performed. For example, continuous intensive ipsilateral BAEP monitoring is required during decompression of the internal meatus in acoustic neurinoma surgery. As yet, identifying when this function is needed is beyond the capabilities of computers, and such decisions must still be made by the individual performing the monitoring. However, it is obvious from the quality of the print-out shown in Figure 2 that using computers as described in this chapter can greatly enhance the results of and relieve the neurophysiologist of many routine tasks performed during intraoperative surgery.

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New Technique for Intraoperative Localization and Monitoring of Cranial Nerves – Preliminary Study

E. WATANABE[1] and Y. KOSUGI

Summary

In this chapter we describe a new stereotactic guiding device and the results of a preliminary study of its usefulness in predicting the location of the facial nerve during acoustic neurinoma surgery. The new device, the Neuronavigator, was developed to monitor the location of dissection during intracranial surgery. It consists of a structure with a multi-articulated arm and a 16-bit personal computer. The computer receives information about the angle of each joint in the arm and calculates the location of the tip of the arm. A computed tomography (CT) scan of the patient's head is obtained preoperatively with three reference marks placed on the patient's head so that the position of the head can be referenced to areas on the scan. When the Neuronavigator arm tip is placed at the operating site, the computer translates its position into coordinates on the CT scan and displays the location of the arm tip on the corresponding CT slice.

We conducted an animal experiment to determine how well the Neuronavigator might predict the nerve's location. An agar ball was placed on an isolated rat sciatic nerve, to serve as a model of an acoustic neurinoma. The nerve was electrically stimulated. The traveling volley was recorded from three pairs of electrodes placed on the surface of the ball and each 50 responses were averaged. The set of three averaged waves was compared to a set of theoretical waves that had been calculated by "best fit" with the recorded set. The predicted location determined by this method was very close to the actual location. These results suggest that the location of the facial nerve can be approximated very closely by recording the antidromic volley from the surface of an acoustic neurinoma. The technique tells us the angle and the location of the nerve – quantitative data that are easily translated into locations in the operating field with the help of the Neuronavigator. This device and the technique we describe for its use should be a powerful adjunct in operations to remove acoustic neurinomas.

With refinements in computer tomography (CT) and magnetic resonance imaging (MRI), it is possible to obtain large amounts of information about the location and morphology of intracranial lesions before the actual sugical approach is begun. Because the location of a lesion and its effect on neurological function are

[1]Department of Neurosurgery, Tokyo Metropolitan Police Hospital, 2-10-41 Fujimi, Chiyoda-ku, Tokyo, Japan

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closely correlated, being able to locate the lesion precisely *during* the operative approach to the lesion would be a great help to the surgeon, as well as a means of protecting nerve function. We developed a device that monitors the location of structures of interest within the operative site during intracranial operations. This new device, which we have named Neuronavigator, is used during the operation itself. It translates the position of a probe tip into CT-scan coordinates and projects the location of the probe tip onto an image of the appropriate CT scan obtained preoperatively (Kosugi and Ikebe 1988; Watanabe et al. 1987). In this chapter, we present the basic concepts underlying use of the device.

This chapter also presents preliminary results of using a nerve locator we developed to predict the location and course of the facial nerve during acoustic neurinoma operations.

Methods

Neuronavigator

Figure 1 is a diagram of the basic components of the Neuronavigator (Kosugi and Ikebe 1988; Watanabe et al. 1987). The system consists of a personal computer and an articulated arm structure. The arm is equipped with 6 potentiometers at each joint. Information about the angles of the joints is relayed to the microcomputer through an analog-to-digital (A/D) converter and the location of the arm's tip is calculated trigonometrically. CT films are scanned with an image scanner and the images are then fed into the computer.

Figure 2 shows the procedure used for navigation. Three metal markers are placed, 1 on the patient's nasion and 1 on each tragus, to serve as reference points for CT scanning. In the operating room, the patient's head is fixed in a Mayfield threepin headholder and the base of the Neuronavigator is attached securely to the clamp so that the position of the arm relative to the patient's head is fixed. The Neuronavigator tip is touched to each of the 3 reference points in turn to enter these coordinates into the computer. The computer then calculates three-dimen-

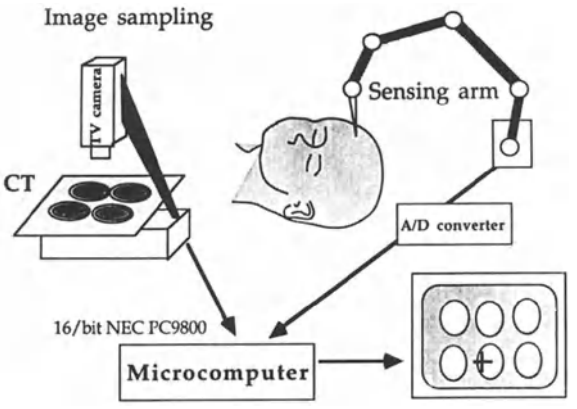

Fig. 1. Schematic diagram of the Neuronavigator system

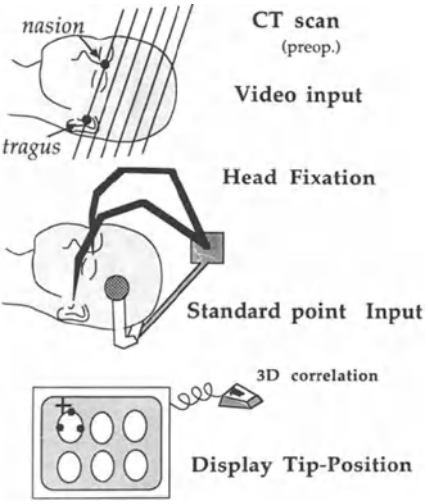

Fig. 2. Procedures before and during use of the Neuronavigator. A CT scan is obtained with 3 reference markers in place, 1 on the patient's nasion and 1 on each tragus. The angle of scanning is adjusted such that the 3 markers appear in a single plane. The sensing arm is fixed to the Mayfield skull clamp. Six CT images are displayed on the computer screen; the location of the Neuronavigator tip is indicated by the cursor cross.

sionally where the locator tip lies in the patient's head and relates this information to the CT scan images. Neuronavigator translates the location of the tip into coordinates on the CT scan using a conversion matrix, and places a cross over the calculated location on the corresponding CT scan.

We tested the accuracy of the Neuronavigator in locating structures in the skull by: 1) performing measurements on a phantom skull and 2) during an operation in which the skull base structures were clearly indentifiable on both CT images and in the operative field.

Nerve Locator

The ability of the nerve locator we developed to trace the course of the facial nerve during acoustic neuroma surgery was tested in the laboratory. A 20-mm diameter agar ball, which served as a model of the acoustic neurinoma, was placed on an isolated rat sciatic nerve. The nerve was stimulated by square wave constant current with a hook electrode. Compound action potentials (CAP) were recorded (50 averages/volley) from a matrix of 3 pairs of bipolar electrodes placed on the surface of the ball (Figure 3).

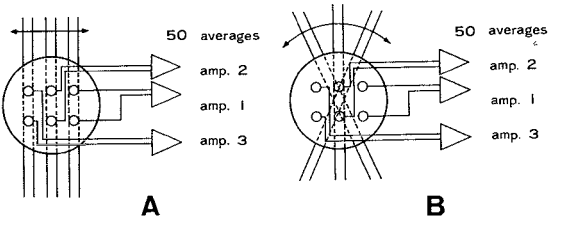

Fig. 3. Arrangement of the 3 points of recording electrodes on the agar ball

Results

Neuronavigator

When the mechanical accuracy of the Neuronavigator in locating structures was examined in a phantom skull, the device reproduced the actual measurements with only 3 mm error. When the Neuronavigator was used in the operating room to locate the anterior clinoid process and sphenoid ridge, it represented the locations of these structures to within 5 mm of their true locations as determined by observation and on CT scans.

Nerve Locator

The set of 3 averaged waves obtained using the method diagrammed in Figure 3 was compared with theoretical waves one would expect to obtain when recording at various distances from the center of the agar ball (Fig. 4). The theoretical waves were derived by the least standard error method.

The curves on the right side of Fig. 4 were obtained when the nerve was located on the right, in the center, or on the left side of the electrode matrix. It is notable that the response amplitude is greatest from the electrode nearest the nerve. On the left side of Fig. 4 the theoretical waveforms are shown. A phenomenon similar to that seen in the experimental recordings, in which the amplitude is largest at the nearest electrode, is apparent in the theoretical recordings. By calculating the

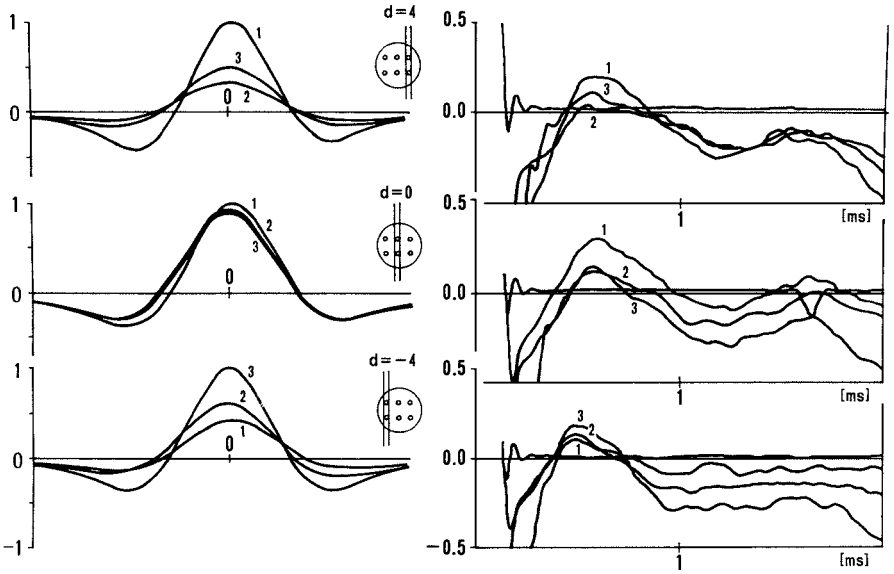

Fig. 4. Theoretical waves and experimental waves recorded when the nerve was located 4 mm, 0 mm, and -4 mm away from the center of the agar ball in a test of the nerve locator

relative amplitudes of the experimental and theoretical waves and comparing them, and from a knowledge of the distances involved in recording the theoretical waves, we can estimate the locations from which the experimental waves were recorded.

Figure 5 shows that the standard error of the mean of recorded waves is smallest when the nerve is in the center of the matrix and the recording is made from −1 mm away. When the nerve is 5 mm to the right of center, the error is smallest when the theoretical distance is 4 mm. The error is smallest with a theoretical distance of −5 mm when the nerve courses 5 mm to the left of center.

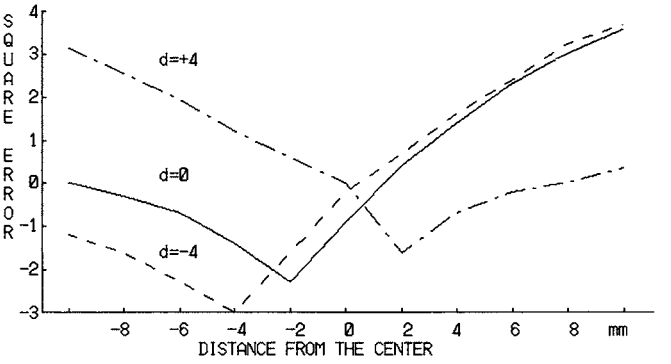

Fig. 5. The error between the experimental waves and theoretical waves at various distances from the center

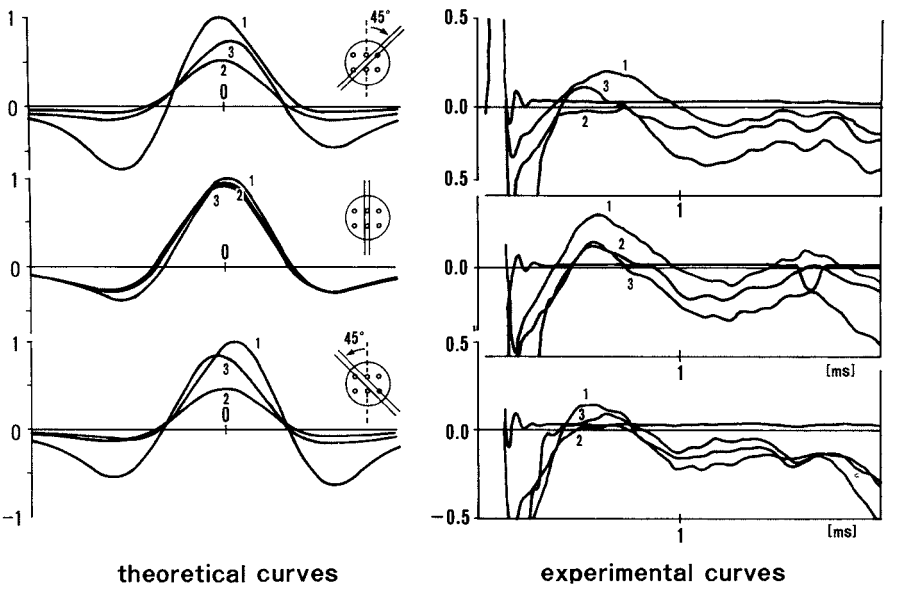

Fig. 6. Theoretical waves and experimental waves recorded with the nerve located 45, 0, and −45 degrees away from the center of the agar ball

The angle between the course of the nerve and the electrode matrix was also determined after the time scale was corrected (Fig. 6). The time sequence of the wave varied according to the angle. A theoretical set of waves was calculated at various angles, and the best fit was determined using the crosscorrelation method (Fig. 7).

When the angle was 0 degrees, the best fit was obtained when the theoretical angle was also 0 degrees (Fig. 7, middle). Figure 7, top, shows that when the experimental angle was set to 45 degrees clockwise, the best fit occurred with a theoretical angle of 50 to 60 degrees. Figure 7, bottom, shows the fit of the curve when the experimental angle was set to 45 degrees counterclockwise. The best fit was obtained with a theoretical angle of 40 to 50 degrees.

These results indicate that the distance and angle of the nerve can both be calculated reliably using the nerve locator.

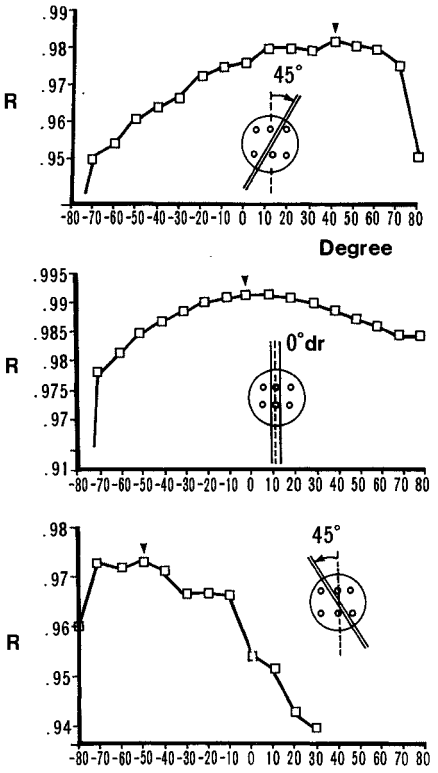

Fig. 7. Correlation between the experimental waves and theoretical waves recorded at various angles to the agar ball

Discussion

Morita et al. (1988) reported that the facial nerve can follow various courses in patients with acoustic neurinomas. They showed that the facial nerve runs ventral to the tumor in 56% of cases and ventrorostral to the tumor in 27% of cases. In this chapter we have reported on a method we developed to locate the facial nerve intraoperatively by recording antidromic action potentials from many points on the surface of the tumor. From the results of animal experiments we present here, it appears that it would be possible to predict the location of the facial nerve from some distance away by recording the antidromic volley from the surface of the acoustic neurinoma. Our method gives information about the angle and the location of the nerve – quantitative data that could easily be combined with information about the operative field obtained using the Neuronavigator we describe in this paper to locate the tumor and the nerve very precisely. The combination of these two systems could be a powerful adjunct to traditional techniques in operations to remove acoustic neurinomas.

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Monitoring of Motor Systems

Localization of Motor Cortex with Median Nerve Somatosensory Evoked Potentials

M. R. NUWER[1]

Summary

Motor cortex localization can be helpful during a craniotomy. Somatosensory evoked potentials (SSEP) can help with this task. The primary cortical SSEP from median nerve stimulation is generally easily and quickly defined on the exposed cerebral cortex. Several types of recording electrodes can be used. Interpretation is usually based upon several features, including the maximum N_{20} amplitude, phase reversal of N_{20}-P_{20} across the central fissure, and the presence of small step-like negative potentials leading up to the N_{20} over the primary sensory cortex. Localization of the motor cortex is most often applied in situations in which the surgeon can use some discretion in defining the limits of a resection or the location of a biopsy.

It can be advantageous to identify the motor cortex during a craniotomy. The complexities fo sulci and gyri anatomy are somewhat too variable and unpredictable to be reliable by themselves. Somatosensory evoked potentials (SSEP) provide a means of helping the surgeon identify the primary postcentral somatosensory cortex, thereby also establishing the location of the precentral motor cortex. This technique is relatively quick and simple, and it can be used without substantial problems or complications in many various circumstances.

Methods

Stimulation

The stimulus used is similar to that used for ordinary SSEP. The median nerve is usually chosen for stimulation. Needle electrodes can be placed over the median nerve at the wrist, with care being taken to place the needles subcutaneously so that they do not lie against the nerve or in the nerve itself. Disc electrodes can also be used for this purpose, again placed over the nerve at the wrist. However, disc electrodes can move or be dislodged during the several hours between their placement and the time of stimulation, so that they need to be secured carefully in

[1] Department of Neurology, University of California, Los Angeles, Reed Neurological Research Center, 710 Westwood Plaza, Los Angeles, CA 90024, USA.

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place. Also, disc electrodes are applied with paste, which can gradually dry over several hours. At our institution, we prefer to use needle electrodes because the location of the electrode stays constant over time and there is no paste that can dry. These electrodes are placed when the patient is draped for the operation, and connected to a cable that is carried from the operating room table to the place where the evoked potential (EP) machine is kept. When the time comes for stimulation, after the craniotomy flap has been turned, and impedence check can be made of the stimulating electrodes. This impedence check assures that the stimulating electrodes are still in place, and have not become loosened or unplugged during the several hours since they were put into place.

The stimulation rate is usually about 7/s. This is slightly faster than the traditional 5/s stimulation rate applied to the median nerve at the wrist. This faster rate is usually well tolerated, as shown by the fact that the recordings made from the cortex are usually particularly tall and are not sensitive to small increases in rate of stimulation. A 200-ms squarewave electrical pulse is used, with the more proximal electrode as the cathode.

When possible, stimulation is briefly performed around the time of draping, so that the response from the thumb can be observed directly. The stimulus should produce thumb movement, showing median nerve innervation of muscles of the thenar eminence. In the absence of neuromuscular junction blocking agents, the stimulus intensity should be set to produce a 1- to 2-cm thumb twitch. Unfortunately, in the presence of neuromuscular junction blocking agents this thumb twitch can be abolished, or some lesser degree of thumb twitch may be observed. If neuromuscular junction blockade is relatively complete and no thumb twitch can be obtained, a default setting of 20 mA stimulus intensity is generally used. Having ascertained that the stimulus intensity is appropriately set, the patient may be draped for the operation. This often means that the hand being stimulated is no longer visible during the rest of the operation.

Recording

The most convenient kind of recording electrode is the flexible strip. This type of electrode is a stainless steel or platinum-iridium electrode, such as is often used for recording from epileptic patients undergoing long-term electroencephalography (EEG) monitoring, embedded in Silastic. These kinds of electrodes are available commercially from several different vendors. Typically, they are discs 3 mm across, separated by seven mm from each other, so that the discs are separated by 1 cm center-to-center. These are available in various sizes. At our institution we usually use 1-by-4 or 1-by-8 strips, which are simple linear arrays of 4 or 8 electrodes. These strips are available with adaptor cables that can be plugged into a standard EEG jack. The adaptor cables are about 1 meter longer so the EP jackbox can be kept out of the sterile field.

Electrodes are usually positioned with the long axis in an anterior-posterior direction. They are placed onto the cortex at a location believed to be at or near the hand region of the sensory and motor cortex. One hopes that the strip of electrodes will span the central fissure itself. Once a location to be tested has been ten-

tatively identified, the electrode is weighed down with a few wet pieces of cottonoid strips.

Recordings are made from a minimum of 4 electrodes at a time, with use of more channels being preferable if available on the averager. At the present time, we only have 4 channels available on our averaging equipment. Recordings can be made either in a bipolar or a referential mode. Bipolar recordings are made by connecting the individual electrode contacts in sequence. Referential recordings are made with reference to a distant recording site. The reference electrode is often an alligator clip placed on dura or muscle.

Another alligator clip is placed on dura or muscle at some distance from the reference, to act as an isoground. This is appropriate only if the recording machinery has an isoground that does not serve as a true chassis ground. It is unsafe to have a true ground used in the recording apparatus unless the patient is not grounded at any other location. Patients usually have a separate, true ground already in place for use with the coagulation apparatus. If so, a second, true ground should be avoided.

The recording time base used is usually around 30–50 ms. The activity actually important for examination usually lies between 15 and 25 ms. We generally set our filters to 30 Hz and 3,000 Hz. A high setting of the high filter is useful because there are small components present at the beginning of the SSEP that we believe are important for identification in this process. Those small features often have a time course that is much less than 1 ms. A setting of the high filter to 1 kHz may blur or obscure those important small initial portions of the SSEP. The low filter is set to 30 Hz because that is traditional for most of our intraoperative SSEP recordings. A lower setting does not usually interfere with interpretation. A substantially higher setting may inappropriately attenuate some of the potentials in the primary EP, although settings as high as 100 Hz may be satisfactory in some instances.

One hundred trials can be completed quite easily in this paradigm, usually taking only about 15 seconds. In many patients, good SSEP can be recorded with as few as 20 trials, requiring only 3 seconds when using a 7/s stimulation rate. Artifacts are not usually a significant problem, since recording directly from the exposed cerebral cortex produces particularly high-amplitude, well-defined SSEP.

It is important to note that the surgeon will usually need to move the strip electrodes around before finally identifying the region from which the SSEP arise. Moving the 1-by-8 strip approximately a half-dozen times before the motor and sensory cortex regions are identified is typical at our institution. In some patients, it has been necessary to move the strip more than a dozen times. The strip is usually moved in whichever direction seems to be most likely to lie closer to the motor cortex than the one previously used. The strip can even be slipped under the skull and dura, to record from sites outside the immediate operative field. In some sense, this procedure is a survey of the quality of SSEP that can be obtained from many different regions close to each other. The SSEP changes in as little as 1 cm, so that the strip might be moved as little as 1 cm laterally or medially, or several centimeters in an anterior- posterior direction. For institutions that have

the ability to record EP on a large number of channels, the use of a 4-by-5 electrode array would be advantageous over the 1-by-4 strip, since a much larger area of cortex could be surveyed at one time. At one medical center, an 8-by-8 array is used, along with topographic computer mapping display of results (Wood et al. 1988).

Interpretation

The goal of EP monitoring is to identify when the phase of the earliest components of the SSEP reverses. Figure 1 shows the anatomy of the primary somatosensory cortex and the sign of the dipole that exists there at the time of the initial cortical SSEP peak. This peak is usually called N_{20}, and is believed to be generated from the posterior bank of the central fissure, corresponding to the primary somatosensory cortex itself. The positive-negative dipole there is oriented in an anterior-posterior direction. This results in an anterior-posterior dipole that also appears at the overlying cortex. This dipole usually has a prominent negative component N_{20} located maximally over the postcentral portion of this region, and a corresponding positive component P_{20} is usually seen over the motor cortex at the same time. The isoelectric point of the phase reversal may lie a few millimeters anterior or posterior to the central fissue itself, i.e., on the anterior or posterior lip of the central fissue. This is an important point to keep in mind when interpreting the location of the phase reversal. In many patients, the phase reversal occurs right at the central fissue itself, but a small amount of variation does occur and one must occasionally expect to see a dipole that might be slightly tilted and therefore will not precisely correspond to the central fissue.

The primary somatosensory cortex also often produces a series of small, step like negative potentials just before the primary N_{20} peak (see Fig. 2). The latter are important features to note, even if they are quite small and transient. These small potentials seem to occur in a region of cortex no larger than about 1 square

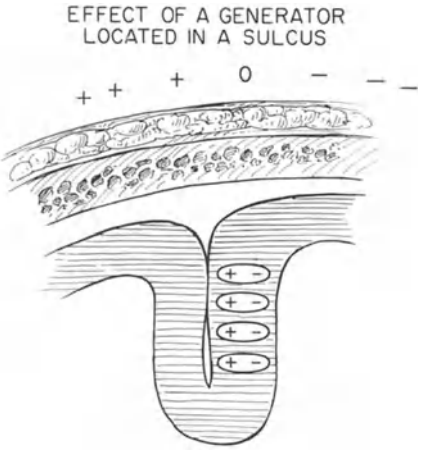

Fig. 1. The generators of the primary somatosensory cortex N_{20} EP lie along the posterior portion of the central fissure. They are oriented to cause an initial negative-polarity potential over the postcentral gyrus, which can be recorded from the scalp or directly from the overlying cortex. (From Nuwer 1986)

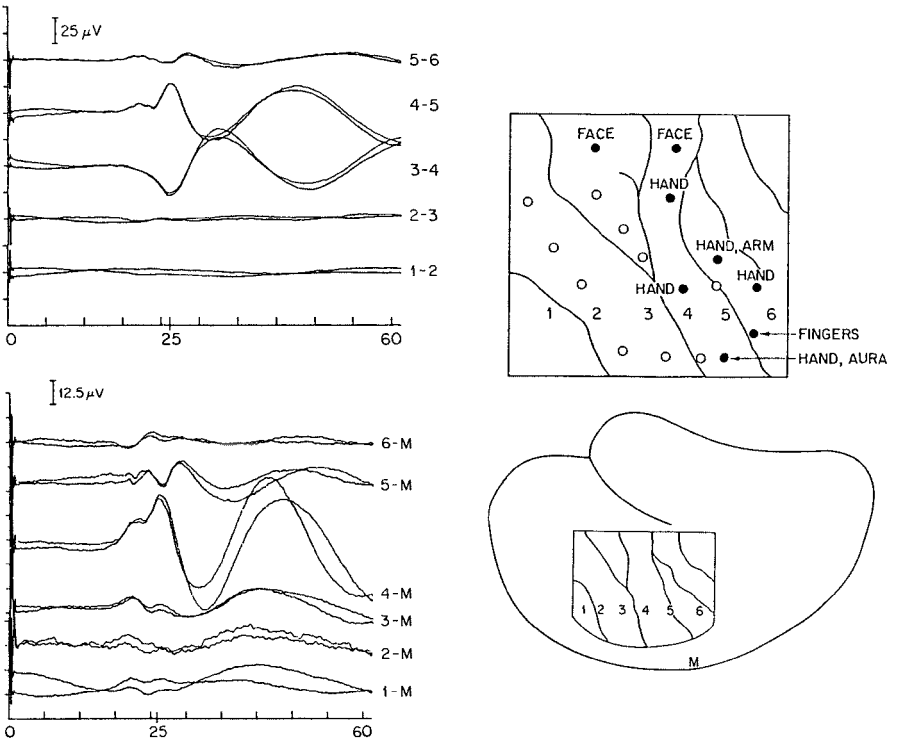

Fig. 2. SSEP recorded in a bipolar *(top left)* or referential *(bottom left)* manner, using a scalp muscle reference *(M)*. The orientation of electrodes is shown *(bottom right)*, as it would be seen by the surgical team. The more detailed diagram of electrode locations (top right) allows one to compare locations where direct cortical stimulation produced sensations *(closed circles)*. This patient was conscious during the operation. In one location, direct cortical stimulation also produced the patient's typical epileptic aura. At other locations, no sensation was caused by direct cortical stimulation *(open circles)*. Higher intensity stimulation probably would have caused motor responses from some of the anterior locations tested. At 25 ms an N_{20} negative potential was seen maximally around electrode 4. Just prior to that, at about 22 ms, a series of small negative potentials was seen, also maximal around electrode 4. These results confirm that electrode 4 lay in the primary sensory cortex, allowing us to deduce that electrodes 2 and 3 lay on the motor cortex. (From Nuwer 1987)

centimeter, corresponding to the thumb region of the somatosensory cortex itself. The primary N_{20} negativity itself can be recorded from a slightly larger area of somatosensory cortex.

Optimally, the following all will be present when the central fissure is reached:
(a) maximal amplitude of N_{20} over the sensory cortex,
(b) phase reversal of $N_{20} - P_{20}$ across the central fissure,
(c) small steplike negative potentials leading up to the N_{20} over sensory cortex.
It is particularly important to note that a series of positive and negative potentials often occur, beginning about 2 ms after the principal N_{20} peak. These potentials

can occur with substantial variability at different cortical regions, and can cause confusion about where the true N_{20} actually lies. It is commonplace to see these potentials occurring at 25 ms and thereafter. A peak P_{25} occurs over the sensory cortex itself. The potentials occurring at 25 ms and the P_{25} over the sensory cortex should not be misinterpreted as being the early primary potentials to be sought. The earliest peaks around 18 to 22 ms should be sought.

The latency of the N_{20} peak varies according to the patient's age, height, and gender. Height is one of the more important factors, and can substantially influence the time of the N_{20} peak. In persons who are particularly tall, older, or have a peripheral neuropathy causing peripheral delay, the N_{20} peak itself may well occur somewhat later than the usual 20 ms seen in persons of middle age and average height. This needs to be taken into account in deciding what N_{20} latency to expect for an individual patient.

Overall, the sequence of procedures involved in intraoperative monitoring of EP can be performed in a reasonably expeditious manner. An experienced team will set up the averager and recording strip quickly. The strip can be put into place, weighed down with wet cottonoid, and an EP can be obtained in a matter of relatively few seconds. This average can then be interpreted quickly to determine whether the N_{20} peak has been adequately identified. Usually a survey is then made of the general area by taking a new recording every 1 minute or so; in most cases the survey can be completed with 6 to 12 recordings. Developing a paradigm should only take 5 or 10 minutes in the usual operating room situation.

Clinical Situations

It is usual to choose this type of technique for patients in whom the location of the motor cortex will influence the actual surgical procedure itself. This happens often in several situations. The original situation in which this technique was used was epilepsy surgery. In that circumstance, it is particularly important to identify and remove the epileptic focus, sparing cortex, such as motor cortex, that is critical to ordinary daily functions. In that circumstance, the epileptic focus has often been identified by preoperative long-term monitoring to capture epileptic seizures on EEG recordings. For patients in whom the epileptic focus is believed to arise from the parietal or frontal regions, it is considered appropriate to identify the motor cortex with the greatest possible precision prior to actually carrying out the corticectomy. At some medical centers now, subdural arrays of electrodes are implanted surgically in order to perform this kind of long-term monitoring for epilepsy. Such subdural arrays of electrodes help identify the location of the motor cortex in outpatients in the same way they do in the operating room. When the subdural grids are not available, operating room identification of the motor cortex is the best way to localize the cortex prior to corticectomy for epilepsy.

Similar principles apply to location of the motor cortex during an open biopsy. When a crainiotomy flap has been turned for biopsy of a possible tumor, most surgeons would prefer not to perform the biopsy in the motor cortex itself. Localizing the motor cortex can sometimes be useful in such cases. Similarly, if a tumor is to be removed partially, the surgeon will often prefer to know the location of

the motor cortex so that that region in particular can be spared, or at least spared as much as is possible given the constraints of the need to remove the tumor.

Occasionally, motor cortex localization is carried out at our institution for other purposes, such as resection of a vascular malformation or during other neurosurgical procedures.

The hand region is not the only one that can be detected in this manner. It is technically possible to record from the face or foot regions, using a technique analgous to the one described above. For the foot region, the electrode strip needs to be inserted into the interhemispheric fissure, which is somewhat more difficult to perform than placement in the hand region of the cortex, but certainly can be done in general without serious risk of bleeding, etc. It is somewhat more difficult, too, to move the strip around when it is in the interhemispheric fissure. The foot region of the motor cortex can often be located by following the motor gyrus from the hand region up to the interhemispheric fissure, so that it has only rarely been necessary actually to place recording strips and obtain test recordings to locate this cortex precisely.

The face region can also be used, but the stimulus artifact produced by face stimulation can be a confounding factor in interpretation of these EP. The recording of face stimulation SSEP is usually performed with the stimulator placed over one of the major portions of the trigeminal nerve, such as the supraorbital nerve or over the infraorbital nerve. Because the SSEP from direct cortical stimulation are of such high amplitude, one can often record adequate signals with a good-quality EP averager that is able to reduce stimulus artifact to manageable proportions. As was true for foot stimulation, it is often not necessary to place strip electrodes and obtain test recordings to locate the face region of the motor cortex, because the location of the face region of the motor cortex can be deduced by following the motor gyrus laterally from the thumb region to the face region. We rarely use these foot and face level recording techniques.

Anesthesia is not a critical factor when using this technique. It can be carried out essentially without regard to the kind of anesthesia used. Forane (isoflurane) is an anesthetic agent commonly used for this technique. The same agent often will abolish the cortical potentials used to monitor patients during spinal surgery, but it has not been a serious problem when using this motor cortex localization technique. The significant difference in these clinical settings probably lies primarily in the fact that the direct cortical recordings are so very high in amplitude and so easy to obtain. The isoflurane probably does cause an attenuation of significant proportions in the size of these direct cortical-recorded EP, but it does not cause an attenuation so severe as to abolish the potentials. At some medical centers, the concentration of enflurane or isoflurane is reduced just before and during SSEP recording (Wood et al., 1988). We have also recorded these potentials in patients given ordinary doses of halothane and in those anesthetized with a balanced nitrous oxide and narcotic technique.

Likewise, other patient-related factors do not significantly interfere with this recording technique in most circumstances. The effects of temperature, other medications, age, etc., are not usually problems of sufficient severity as to interfere with this technique. The one exception is the presence of a tumor directly

underlying the central region. In the case of patients who already have substantial loss of proprioception and serious weakness due to the presence of the tumor in the hemisphere, we have sometimes found it impossible to find any SSEP at the time of craniotomy. To some extent, we have been able to predict this in advance by trying to record SSEP prior to the time of the operation, and finding that the absence of a SSEP preoperatively frequently corresponds to a failure to find a SSEP intraoperatively. It is helpful to know this, since it can be frustrating for the intraoperative team to move the recording electrodes to a dozen or more locations around the proposed region of motor cortex, only to find no recordable EP intraoperatively. Wood et al. (1988) noted a similar loss only in patients with tumor, including 1 patient who did have a small but preserved preoperative cortical SSEP. Apart from patients who have significant preoperative impairment along this pathway, we have always been able to obtain SSEP from direct cortical recording sites.

Alternate techniques for identification of motor cortex include examination of the anatomy and direct cortical stimulation techniques. Examination of anatomy has not been found to be a particularly reliable tool, since it is often in error by one gyrus when compared to the results of the SSEP motor cortex localization technique (Wood et al. 1988). Preoperative magnetic resonance imaging (MRI) can help localize the central fissure, which appears on these images as a mirror-image pair of sulci extending to the interhemispheric fissure (Berger et al. 1990), but the relation of these structures on the image to actual intraoperative anatomy may be tricky and sometimes the usual markings are distorted by the pathology present. The direct cortical stimulation technique, as popularized by Penfield and Jasper (1954) has much in its favor and a long history. However, in our institution it is usually found to take significantly longer and, perhaps to be slightly more inaccurate than the localization technique we describe. Sensory evaluations can only be performed on awake patients. Motor responses can sometimes be hard to find. In some patients, stimulation at a pre-motor cortex level or sometimes at the somatosensory cortex can produce motor responses, so that the presence of a motor response does not always demonstrate that the stimulation electrode is located on the motor cortex itself. The direct cortical stimulation technique also makes it necessary to observe the patient, which can be somewhat awkward when the patient is fully draped for the operation. Finally, it takes some while to identify the proper amount of stimulation to be used with direct cortical stimulation, which lengthens the time necessary to carry out the technique appropriately. It should be pointed out that direct cortical stimulation is usually carried out in the presence of EEG recordings, which is a safety feature meant to avoid seizure-like discharges from the stimulation. The latter can cause motor responses to appear even from stimulation sites distant from motor cortex, and also can pose a safety problem in case the patient has a grand mal seizure on the operating room table. Direct cortical stimulation also can be difficult in some patients, especially in children. This is true even when the operation is performed with the patient under general anesthesia, as is the usual situation. Direct cortical stimulation is somewhat easier to carry out technically when the patient is awake during the operation, although that presents many other difficulties in itself.

Sometimes motor cortex localization is carried out along with electrocorticography. This is sometimes helpful in tumor cases, since the choice of which portion of cortex to biopsy or to remove needs to be decided with a great degree of clinical discretion. Identification of motor cortex is one step in being able to avoid biopsying or removing that critical portion of cortex. Electrocorticography can also be helpful as a supplement to other monitoring techniques, as the presence of greatly attenuated EEG waves, or lack of fast activity, or presence of increased slow activity can provide clues that certain areas of cortex are already substantially impaired, especially when compared to other nearby regions of cortex that fail to demonstrate those kinds of EEG abnormalities. A good clinical neurophysiologist should be able to help interpret SSEP in the light of electrocorticography results in these circumstances, helping to note which portions of cortex are still very functional and viable, and should be spared, as opposed to regions that are obviously impaired physiologically and would be good candidates for biopsy or removal. In many circumstances, the electrocorticography can be carried out using the same instrumentation, same electrodes, or at least the same personnel as would be brought into the operating room to manage the EP monitoring.

The reader is referred to discussions of related techniques reported elsewhere (Nuwer, 1986). Variations on these techniques include extraoperative localization with implanted electrode arrays (Lueders et al. 1983; Lesser et al. 1987, 1989), and direct cortical stimulation techniques for language and other functional localization (Ojemann et al. 1989).

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Recording of Myogenic Motor Evoked Potentials (mMEP) Under General Anesthesia*

M. TANIGUCHI[1], J. SCHRAMM, and C. CEDZICH

Summary

This chapter reviews possible reasons for the difficulties encountered obtaining well-delineated myogenic motor evoked potentials (mMEP) under general anesthesia. Three possible levels for anesthetic suppression are discussed: the cortical level, the spinal motor neuronal level, and the neuromuscular junction. A technique of facilitating mMEP at the spinal level is described. By combining peripheral stimulation, as for the H-reflex, with transcortical stimulation, the mMEP can be facilitated. The degree of facilitation is compared in 12 anesthetized and 8 nonanesthetized patients.

By combining transcranial electrical stimulation of the cortex with a well-timed peripheral stimulus, we were able to elicit reproducible muscle responses under general anesthesia and a typical surgical degree of muscle relaxation. Adding the peripheral stimulus resulted in a biphasic facilitatory effect in patients without anesthesia, but the second phase of increased excitation was missing in patients under anesthesia. In a small sub-group of patients the effects of transcranial magnetic and electrical stimulation were compared. In cases in which magnetic stimulation worked it provoked a more variable degree of facilitation than transcranial electrical stimulation.

The possible mechanisms of anesthetic suppression of mMEP are discussed, as are possible ways to circumvent suppression, including a change of stimulus parameters, the use of facilitation techniques, and a change in anesthesia regimen. It is concluded that, although there are limitations on the use of H-reflex facilitation in transcranial motor stimulation, this technique is useful to reduce the incidence of false-negative mMEP in comatose patients. Magnetic transcranial stimulation is not particularly useful in comatose patients.

Recording myogenic motor evoked potentials (mMEP) under general anesthesia is difficult. Nevertheless, to improve this recently introduced monitoring modality so that it might be used clinically in the future, we studied ways to facilitate the mMEP so that they can be recorded intraoperatively. It is not a new idea for neurosurgeons to utilize this muscle response to brain stimulation during

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[1] Neurochirurgische Universitätsklinik, Sigmund-Freud- Str. 25, W-5300 Bonn 1, FRG.

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neurosurgical operations. For example, the primary motor cortex, when exposed in the operative field, has been localized by applying direct cortical stimulation (Horsley 1909; Penfield and Boldrey 1937). But this response is known to be very susceptible to anesthetic suppression (Erickson 1949), and therefore, it was only used in patients under local anesthesia. There is no doubt that suppression of the mMEP by anesthesia has restricted wider use of intraoperative monitoring using these potentials: the benefits of using these potentials in intraoperative monitoring must be weighed against the risk of performing neurosurgical operations under local anesthesia.

Unfortunately, the introduction in the last decade of techniques for electrical and magnetic transcranial brain stimulation has not resolved the problem of using mMEP in monitoring of patients under general anesthesia. Transcranial stimulation, which is strong enough to evoke good muscle responses in awake patients (even those in a relaxed condition), is not usually effective in patients under general anesthesia, even when no muscle relaxant has been given (Zentner et al. 1989).

We undertook the work reported in this chapter to examine the following 2 questions: 1) what is the mechanism of anesthetic suppression of mMEP? and 2) what might possibly be done to reduce this suppression so that mMEP can be recorded in patients under general anesthesia?

Mechanism of Anesthetic Suppression

Both magnetic and electrical stimulation of the brain ultimately activate the corticomotoneuronal pathway, although the mechanism of action at the cortex may be different. The elicited neural activity travels down the spinal cord to motoneurons, the activation of which in turn leads to muscle contractions (Rothwell et al. 1989).

Anesthesia can suppress neuronal activation at any point in this pathway: 1) cortical stage, 2) spinal motoneuronal stage, or 3) neuromuscular junction. The completeness with which anesthesia suppresses mMEP seems to suggest that complete blockage of signal transmission occurs at one, or possibly more, of these stages.

However, complete blockage at the cortical stage is not likely, because spinal cord potentials (D-waves) are recordable following brain stimulation in man, even in patients under general anesthesia (Boyd et al. 1986; Katayama et al. 1988a). This shows that at least the brain is excitable, even when it is under the influence of surgical doses of anesthetics, although it is not excitable in the same way as it is in awake persons.

On the other hand, complete blockage of the neuromuscular junction is also not likely, because muscle activity can be recorded following peripheral nerve stimulation, even in patients under general anesthesia and in those under the influence of a certain amount of muscle relaxation (White et al. 1989).

Accordingly, the spinal cord seems to be the most likely location for interruption of the conduction of mMEP in patients under general anesthesia. It may thus be

assumed that motor signals resulting from brain stimulation do arrive at the presynaptic terminals of the spinal motoneurons, even in patients under general anesthesia. However, these motor signals seem to fail to bring motoneurons above firing threshold for some reason.

Several ways to overcome this anesthetic suppression of mMEP might be tried: 1) more effective brain stimulation, which would result in a stronger stimulus being passed to motoneurons, 2) facilitation of mMEP at the spinal level, i.e., sensitizing these motoneurons to corticifugal motor signals by combining the mMEP with additional excitatory postsynaptic potentials (EPSP) from another source, 3) a different method of anesthesia that would cause less suppression of motor systems, or 4) combinations of these methods.

Facilitation of mMEP at the Spinal Level – A Possible Solution

In this chapter, we report mainly our clinical experiences using the facilitation method. As noted by Milner-Brown et al. (1975) and Cowan et al. (1986), a well-timed combination of two stimuli – a peripheral nerve stimulus that elicits the H-reflex from the upper extremities and a stimulus to the motor cortex – resulted in larger compound muscle action potentials than those recorded when either stimulus was used alone. In other words, these 2 stimuli are known to facilitate each other's effects on motoneurons. In our study, peripheral nerve stimulation was used to produce a facilitation of motoneurons in order to compensate for the depression caused by anesthesia.

Using this technique, we first analyzed the mechanism of anesthetic suppression of mMEP. Then we compared briefly the effects of electrical and magnetic transcranial stimulation.

Methods and Patients

Cortical Stimulation

Cortical stimulation was performed transcranially with commercially available electrical and magnetic stimulators. Electrical stimuli were delivered using a Digitimer D-180D and standard ECG-pad electrodes. The anode was placed 2 cm anterior to C3 or C4, on the side contralateral to the extremity to be examined, with the cathode 2 cm anterior to Cz. Peak stimulus strength was expressed as a percent of the maximal voltage that could be delivered by the equipment (750 V). The time constant was 100 μs.

Magnetic stimulation was performed using a Novametrix Magstim 200 with a stimulating coil of 14 cm diameter (maximal magnetic field 2 Tesla, duration 2 ms). The stimulating coil was handheld with the center of the coil above Cz. The effects of clockwise and counterclockwise stimulation (as seen from above) were compared in each case, and the stimulation mode that gave the stronger effect was used. Stimulation intensity was expressed as a percent of maximal output.

Peripheral Stimulation

The method for peripheral stimulation was similar to that used to elicit the H-reflex from the m. flexor carpi radialis (Deschuytere et al. 1976; Garcia et al. 1979). A rectangular pulse of 1-ms duration was given to the median nerve at the cubital fossa using a Nicolet SM300 constant-current stimulator.

Combination of the Two Stimuli

The optimal interval between the two stimuli to obtain maximal facilitation was determined as follows: The H-reflex was recorded first and the stimulus strength that produced an H-reflex that was less than 50% of maximal was determined (Desmedt 1973; Meinck 1980). Then cortical stimulation was applied together with the median nerve stimulation and the interval between the two stimuli was varied, with the cortical stimulation appearing from 10 ms before to 10 ms after stimulation of the median nerve. The peak-to-peak amplitude of the facilitated H-reflex was expressed as a percentage of the size of the original H-reflex, and these amplitudes were plotted against the interstimulus intervals. From this plot, the time course and amplitude of changes in spinal excitability resulting from cortical stimulation could be determined. This procedure also permitted us to estimate the degree and duration of participation by each supraspinal synaptic component.

Data Acquisition and Stimulus Control

Data acquisition and storage as well as timing of the stimuli were performed with a Nicolet Pathfinder I, using specially written computer programs. Each individual response could be viewed on the screen, and normally 3 to 5 sequentially obtained responses were averaged.

Comparison of the Anesthetized and the Nonanesthetized State

In order to examine the mechanism of anesthetic suppression, we compared the time course and the amplitude of changes in spinal excitability caused by transcranial electrical stimulation in anesthetized and nonanesthetized patients. Eight comatose, nonanesthetized patients in an intensive care unit (ICU) and 12 anesthetized patients were examined. So that results for the two groups would be comparable, we included only those patients in whom a good baseline H-reflex was obtained, and for whom cortical stimulation intensity ranged between 15 and 30% of maximal output.

Patients studied intraoperatively were anesthetized by a modified neurolept anesthesia (NLA) method (a combination of nitrous oxide 66% and halogenated agents up to one half minimum alveolar concentration (MAC)). Fentanyl was administered intermittently (to a total maximal dose of 1 mg), as was a muscle relaxant (pancuronium bromide, 0.1–0.2 mg/kg bodyweight every hour). In this series of patients the level of anesthesia was kept "steady" as determined by mon-

itoring vegetative functions (blood pressure, heart rate, etc) in the usual manner. Muscle relaxation was monitored by the "train-of-four" technique, and was kept at between 1 and 2 responses to 4 test pulses. No comatose patient in the ICU received any sedative drugs during the 8 hours before the examination.

Comparison of Magnetic and Electrical Stimulation

In order to compare the availability of magnetic and electrical stimulation as a diagnostic tool, we used both types of stimulation on patients. The patients were typical of those cared for in the ICU and were in various stages of unconsciousness as a result of increasing intracranial pressure or administration of sedatives (flunitrazepam). Only patients who were clinically paretic on one side only were included in this part of the study. The unaffected side served as the control of each patient.

Both magnetic and electrical transcranial stimuli of various intensities were given to the patients, and the "direct" muscle responses were recorded. The peak-to-peak amplitude of the compound muscle action potentials (CAP) was measured, and stimulus response curves were obtained. These were used to estimate the equivalent stimulation intensities for the 2 ways of eliciting MEP.

In some cases in which direct muscle responses could only be obtained with electrical stimulation the magnetic stimulation of maximal intensity (100%) that we used together with electrical stimulation at an intensity lower than the threshold for direct muscle responses (approximately 2/3 to 4/5 of the threshold at the peak intensity) was used.

Results

Combination of Two Stimuli

The facilitation accomplished by stimulation of the median nerve made it possible to obtain a reliable muscle response, even in patients who were operated upon under general anesthesia. It is hypothesized that a single electrical shock of transcranial stimulation of normal intensity would give rise to EPSP at the motoneurons even in patients under general anesthesia. These EPSP, however, were not strong enough to bring the motoneurons above the firing threshold. As is shown in Fig. 1, a combination of cortical stimulation and a well-timed peripheral stimulation resulted in reproducible muscle responses even in patients under general anesthesia. A complete set of recording results is shown in Fig. 2. With increasing stimulation strength, the H-reflex (*H*) appeared first and then M waves (*M*) appeared. With adequate peripheral stimulation a portion of the motoneuron pool was excited, as indicated by the presence of an H-reflex, and another group of motoneurons was facilitated by the peripheral stimulation, but not quite to the firing threshold. These neurons reached their threshold for discharge from the addition of cortical stimulation, as evidenced by the facilitated H-reflex.

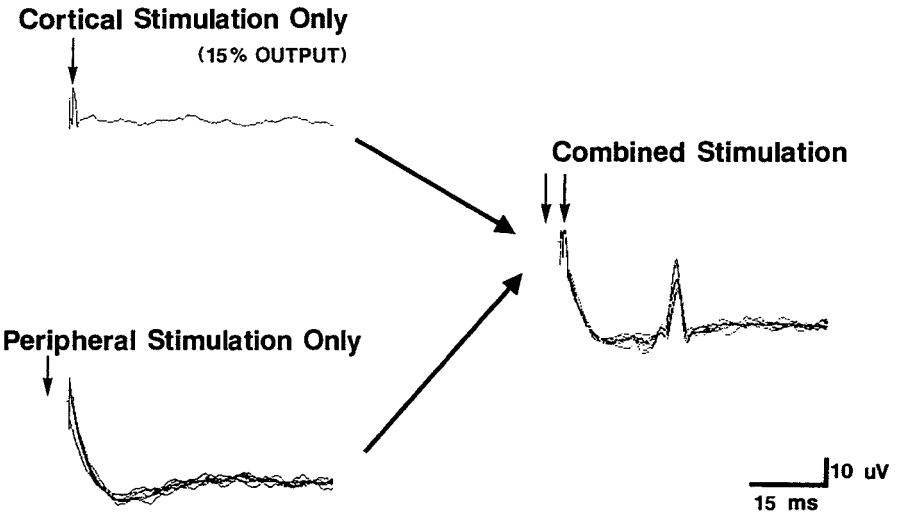


Fig. 1. mMEP recorded intraoperatively from forearm flexor muscles. Transcranial electrical stimulation (15% of maximal output, time constant 100 μs) and peripheral stimulation to elicit the H-reflex from the forearm flexor muscles (of subthreshold intensity) were combined. Interstimulus interval (ISI) was 4 ms. Recording was done under general anesthesia, about 20 minutes after administration of 2 mg pancuronium bromide

Influence of Anesthesia

The time course and the degree of change in spinal excitability caused by transcranial electrical stimulation were compared in anesthetized and nonanesthetized patients. In nonanesthetized patients (Fig. 3 A, B) the cortical stimulation caused a triphasic (short-lasting excitatory/inhibitory/long-lasting excitatory) spinal excitability change. In the anesthetized patients (Fig. 4A, B) the response was characterized by a monophasic, short-lasting excitatory effect and a lesser degree of facilitation. As may be seen readily by comparing Figs. 3 A and 4 B, transcranial electrical stimulation using a stimulus strength of 15 to 30% of the maximal output caused a triphasic change in motoneuron excitability in nonanesthetized patients (Fig. 3 B) that was present over a large range of interstimulus intervals (ISI), while only a monophasic, short-lasting change was seen in anesthetized patients (Fig. 4 B). The excitatory peak around the ISI of −4 ms was commonly seen in both patient groups. However, the degree of facilitation at this peak around an ISI of −4 ms was significantly less in anesthetized patients (231% of the control value on average, standard deviation 70) compared to nonanesthetized patients (591%, standard deviation 259).

Comparison of Magnetic and Electrical Stimulation

In 11 patients, mMEP were obtained with both magnetic and electrical stimulation in 5 cases, and with electrical stimulation only in 6 cases.

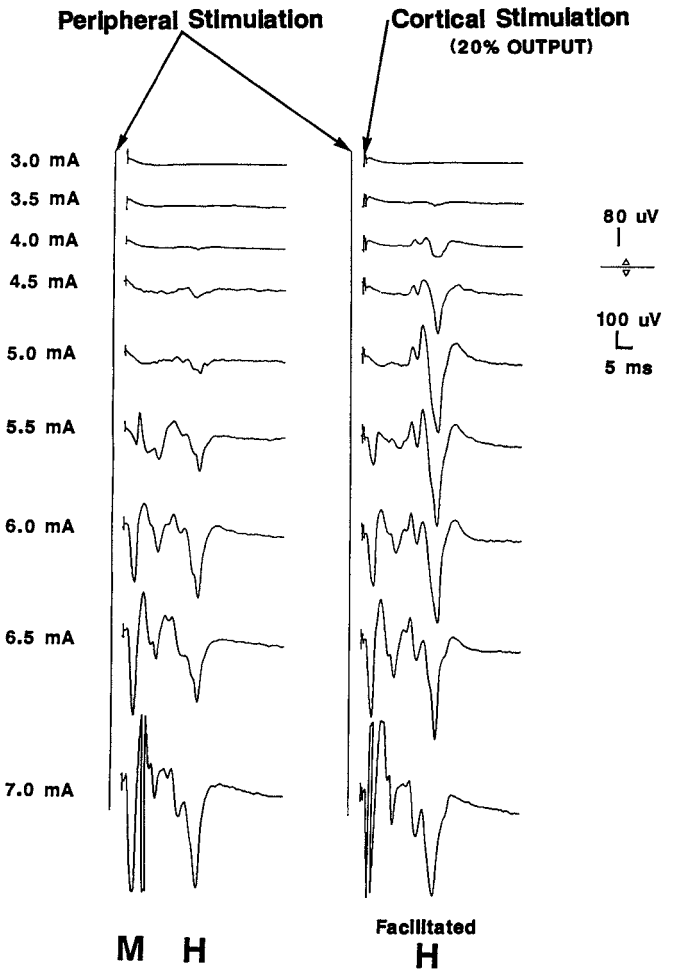
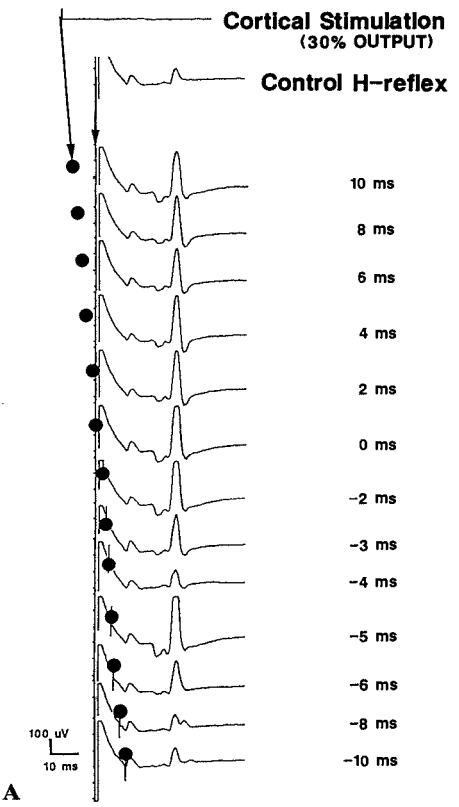


Fig. 2. Compound muscle action potentials recorded from forearm flexor muscles during spinal cord surgery. Results obtained from combination of peripheral stimulation at various intensities and constant cortical stimulation. Left column: results obtained using peripheral stimulation only. The recording started 4 ms after stimulation of the peripheral nerve. Right column: cortical stimulation was added 4 ms after the onset of the peripheral stimulation. The H-reflexes were obviously facilitated by this additional stimulation. M-waves remained stable

Intensity equivalents for the two types of stimulation were calculated from the results in the 5 patients who responded to both stimuli. Fig. 5 shows that 100% output of the magnetic stimulator was roughly equivalent to 25 to 30% output of the electrical stimulator (time constant 100 μs).

However, in the other 6 cases this relationship did not hold true; in these cases, magnetic stimulation at maximal intensity (100%) failed to evoke muscle contrac-



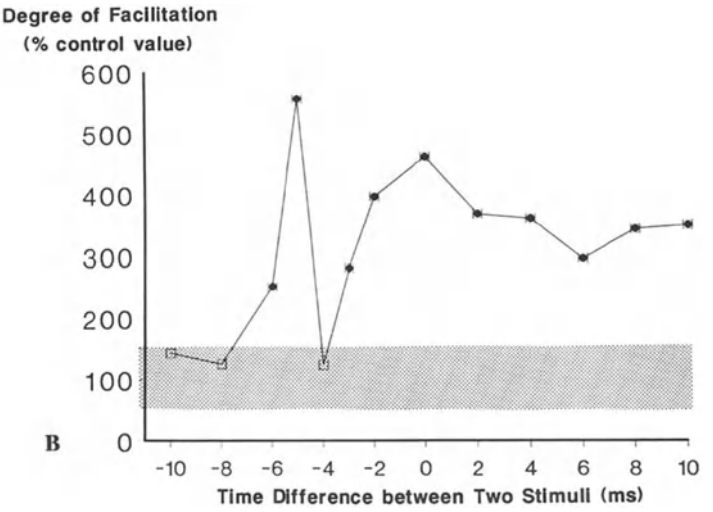


Fig. 3. A Typical compound muscle action potentials recorded from forearm flexor muscles in a nonanesthetized patient. The 2 stimuli were given with different ISI. These results are typical of recordings from the clinically unaffected side of nonanesthetized patients, and were obtained from a patient with bifrontal cerebral contusions. **B** Typical time course changes in the amplitude of the H-reflex response in nonanesthetized patients to show degree of spinal excitability changes for cortical stimulation of 30% intensity applied as a function of ISI. In this case, a stronger stimulus (40% of maximal output) alone led to a muscle response. A positive ISI means that the cortical stimulation precedes stimulation of the median nerve. Standard error of H-reflex at the settings we used was maximally 25%. More than 150% (over 2 standard deviation) facilitation was considered to be significant. The shaded area indicates 2 standard deviation (50%)

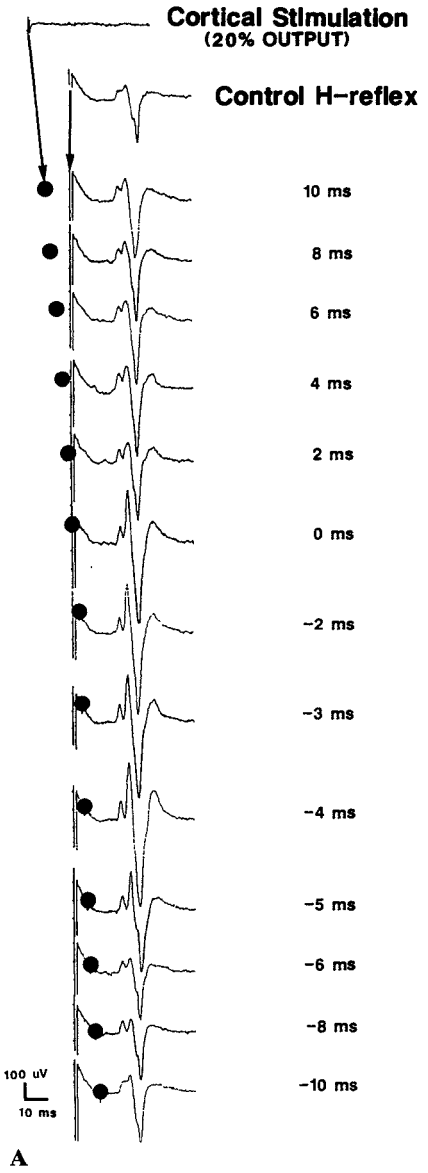
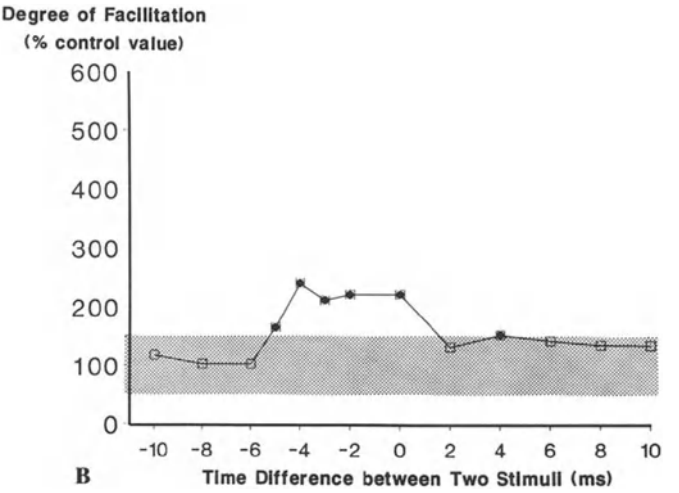


Fig. 4. A Typical results from an anesthetized patient. This patient had a small cavernous angioma in the frontal area. Cortical stimulation of 20% intensity was combined with peripheral stimulation in the same way as shown in Fig. 3 A. **B** Time course and degree of spinal excitatory changes for the patient described in Fig. 4 A, compared to nonanesthetized patients (Fig. 3 B).

tion, although the equivalent intensity of electrical stimulation (25 to 30% of maximal output) still gave reproducible muscle responses.

The mechanism by which these "false-negative" findings occurred was examined by the technique, already described, of H-reflex facilitation. In Figure 6, the "false-negative" results of stimulation in these 6 cases are shown. The effect of magnetic stimulation on motoneurons was characterized as follows: 1) magnetic



stimulation reached motoneurons at least 1 ms later than did electrical stimulation, 2) the time course of changes in spinal cord excitability caused by magnetic stimulation showed a monophasic, long-lasting excitatory pattern, 3) the facilitation caused by magnetic stimulation was more variable in degree than facilitation caused by electrical stimulation.

No definite cause for these false-negative findings can be established from examining so few cases. However, it should be noted that 3 of the 6 patients with false-negative results were sedated, and the other 3 patients were deeply comatose and had increasing intracranial pressure. In contrast, none of the 5 patients from whom responses were obtained to both types of stimulation was sedated or deeply comatose.

Discussion

Mechanism of Anesthetic Suppression of mMEP

Failure of transcranial electrical stimulation to bring motoneurons above firing threshold in patients under general anesthesia could be due to one or both of the following: 1) changed supraspinal inputs, i.e., the usual intensity of transcranial electrical stimulation causes, in patients under anesthesia, only a single excitation of the corticomotoneuronal pathways (D-wave), and 2) depressed EPSP generation at motoneurons, i.e., this single excitation of corticomotoneuronal pathways causes weaker EPSP at motoneurons in anesthetized patients.

The reduced excitation of corticomotoneuronal pathways alone may be sufficient to explain blockage of the mMEP in patients under anesthesia. Results obtained by Landgren et al. (1962) in experiments in the baboon suggest that repetitive firing of corticomotoneuronal pathways is necessary for enough motoneurons to be recruited to generate muscle responses. Analysis of peri-

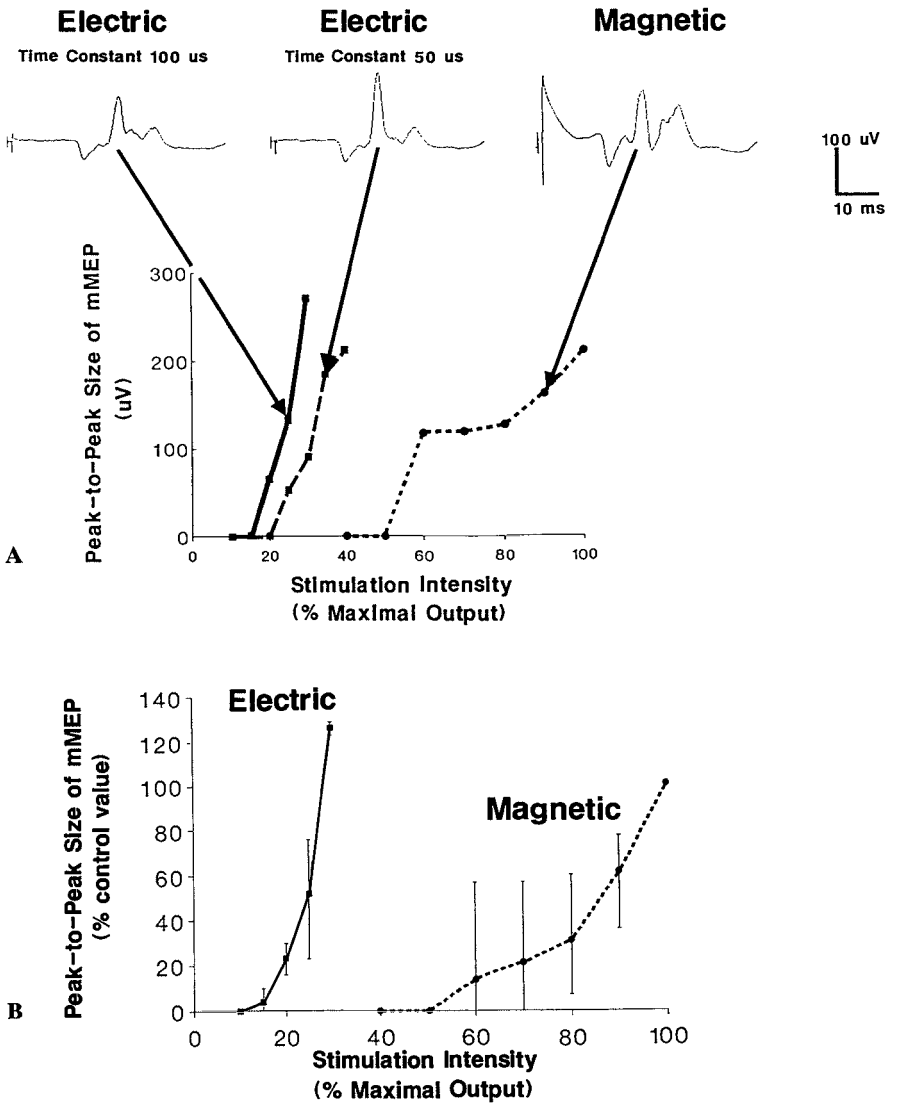


Fig. 5. A Rough comparisons can be made between magnetic and electrical transcranial stimulators from these results obtained in a patient in "alpha-coma" due to severe brainstem swelling after an operation to manage a giant acoustic neurinoma. The peak-to-peak size of the electromyographic recording from the forearm flexor muscles was plotted against the stimulation intensity (percent of maximal output) for each stimulator. The two types of stimuli generated muscle contractions of approximately the same shape and duration. **B** Comparison can be made of the two modes of stimulation from this presentation of the patients. In order to standardize results, the size of the muscle response obtained by magnetic stimulation at 100% output was set to 100%, and the results of stimulation by other modalities were expressed as percentages of this value. For these 5 patients, 100% output of the magnetic stimulator was approximately equivalent to 25 to 30% output of the electrical stimulator (time constant 100 μs)

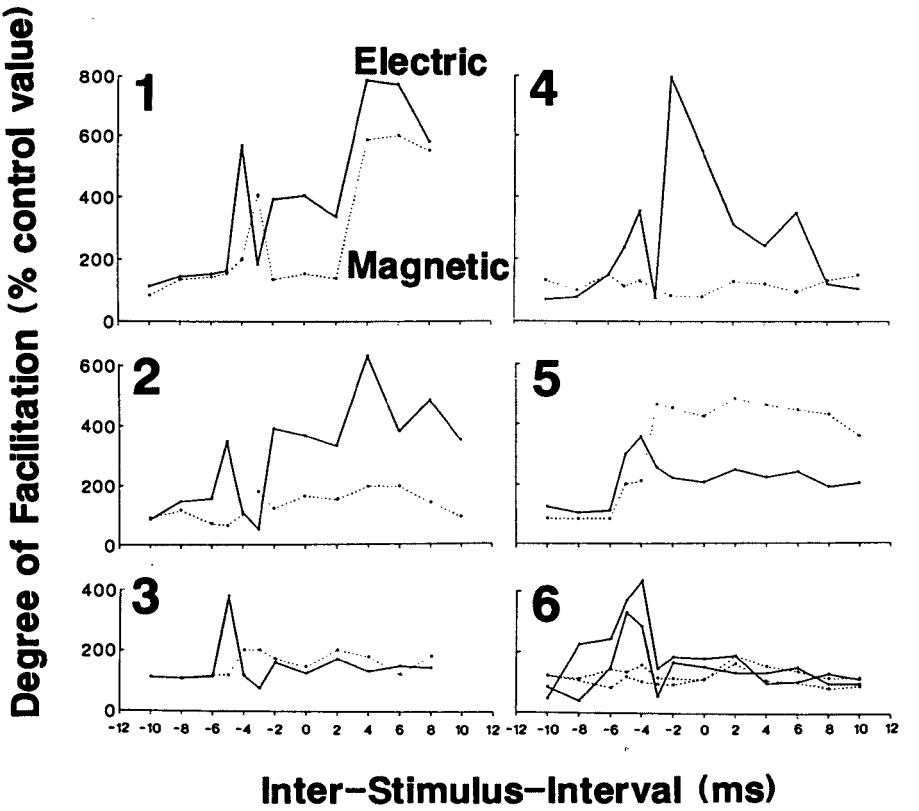


Fig. 6. Facilitation of the H-reflex response by stimulation of the median nerve in 6 patients who failed to show any response to magnetic stimulation alone at the maximal stimulus strength. Change in the amplitude of the H-reflex response with electrical *(continuous line)* and magnetic *(dotted line)* stimulation are shown. The output intensity of magnetic stimulation was set to 100%, and the output of the electrical stimulator was set to subthreshold intensity for direct muscle responses for each case (from 2/3 to 4/5 of the threshold at the peak intensity). Case 1: Patient with posttraumatic intracerebral bleeding, deep coma, intracranial pressure over 20 mmHg, and no sedation. Case 2: Patient with acute subdural hematoma and diffuse cerebral contusion, sedated with flunitrazepam. Case 3: Patient with giant cerebellar bleeding with marked hydrocephalus and no sedation. Case 4: Patient with high-grade subarachnoid hemorrhage and a huge intracranial hematoma, 6 hours before tentorial herniation due to brain swelling. No sedatives were given. Case 5: Patient with bifrontal traumatic contusion who was sedated with flunitrazepam. Case 6: Patient with acute subdural hemorrhage, sedated with flunitrazepam

stimulus-time-histograms obtained in man (Day et al. 1987) also suggest that a single excitation of corticomotoneuronal pathways is not of itself strong enough, even in awake relaxed patients, to bring motoneurons at rest above the firing threshold. Therefore, Day et al. (1987) concluded that repetitive excitation of corticomotoneuronal pathways is necessary to cause muscle contractions in relaxed patients. Because the anesthetized state can be regarded to be the equiva-

lent of an absolutely relaxed state, it is no surprise that a single excitation of corticomotoneuronal pathways fails to excite motoneurons.

The latter mechanism of depressed EPSP generation at motoneurons might be explained in 2 different ways: 1) anesthesia decreases the number of corticomotoneuronal fibers that are recruited by cortical stimulation, or 2) synaptic conductivity is depressed by anesthesia. Katayama et al. (1988a, b) recorded spinal cord potentials (D- and I-waves) caused by electrical stimulation of the brain and compared the results obtained before and after induction of anesthesia in the same patient. They reported that the D-wave recorded when the patient was under surgical anesthesia was not dramatically lower in amplitude than the D-wave recorded in the nonanesthetized patient. This result seems to support our second assumption that depressed synaptic conduction is the main cause of failure to record motor evoked potentials in response to electrical stimulation. However, the anesthesia used by Katayama et al. (1988a, b) (Thiopental) was different from ours, and therefore it is still uncertain which of the two explanations for depression of EPSP by anesthesia is correct.

Suggested Remedies

Three factors might be manipulated in order to overcome the problem of anesthesia suppression of mMEP: stimulation, spinal cord excitability (facilitation), and anesthesia.

1. Stimulation

The ideal stimulation would cause repetitive firing of corticomotoneuronal pathways in patients under the influence of general anesthesia. Unfortunately, this cannot be achieved by application of the single shock of an intensity in the range used for transcranial electrical stimulation. Two different modifications in stimulation seem possible: 1) use of a train of direct cortical stimuli with ISI shorter than 10 ms (Landgren et al. 1962; Milner-Brown et al. 1975), or 2) use of a strong stimulus, one that reaches enough of the cortex to evoke an intrinsic repetitive excitation of corticomotoneuronal pathways (Zentner et al. 1989).

The first modification, stimulation with a train of pulses, can only be implemented with a galvanic stimulator and therefore only when the motor cortex is directly exposed. At present, we know of no stimulator that can deliver a train of faradic stimuli at short intervals. Therefore, applying a train of transcranial electrical stimuli at high frequency remains a possibility but not one that can be implemented with current technology.

The second modification, activating a wider area or a larger volume of the cortex with each stimulation, could be achieved by applying more intense transcranial stimulus. However, it should be noted that, from the practical point of view, a very strong electrical stimulation applied transcranially causes strong artefactual contraction of muscles which would disturb the operative procedure. From our experience, a transcranial electrical stimulus stronger than 28% of maximum (time constant 100 μs) causes, even when the usual surgical doses of muscle relaxants have been given, a contraction of neck and shoulder muscles that is incom-

patible with a microsurgical procedure. In the patients not given a muscle relaxant, 20% of maximal output was strong enough to cause this motion artifact. It is mainly for this reason that we no longer use a strong transcranial electrical stimulus. Because direct stimulation of the cortical surface results in stimulation of a wider cortical area but not necessarily a stronger stimulus, this type of stimulus modification seems an attractive alternative.

2. Facilitation

Sensitization of motoneurons, in order to let them respond more easily to supraspinal input, could theoretically be achieved by adding any type of reflex input to the cortical stimulation protocol. However, until now we have only been successful in facilitating mMEP by combining cortical stimulation with the H-reflex from the m. flexor carpi radialis, and therefore, we would like to restrict our discussion to this combination. In another paper (Taniguchi and Schramm 1991), we discuss the possibility of applying this facilitation technique in the clinical setting in order to monitor mMEP intraoperatively. However, anesthetic suppression of the H-reflex itself makes intraoperative use of the technique more complex.

Inhalation anesthetics used in our series of patients suppressed not only mMEP but also the H-reflex itself. And, to make things more complicated, the degree of this suppression increased continuously for at least the first 60 to 90 minutes after induction before it reached a fairly steady state. In order to be able to assess the fine qualitative changes in mMEP that must be evaluated for monitoring, the motoneurons should ideally be potentiated at the same level during the whole, or at least during the important part, of the operation. This, however, could not be done with the anesthesia method used. An effort must be made, therefore, to find an anesthesia method that suppresses the H-reflex minimally and at a level that is constant during the whole operation. Unless this can be achieved, it would be difficult to use the facilitation method we describe for clinical intraoperative monitoring.

3. Anesthesia

At this time, we know of no combination of anesthetics that can supply, at the same time, both adequate level of surgical anesthesia and minimal suppression of mMEP. Therefore, a change in anesthesia regimen alone is not likely to be the total solution to the problem, although such a change could be a helpful adjunct to other modifications in technique.

Magnetic Versus Electric Transcranial Stimulation in Clinical Use

The magnetic stimulator, at least the one we used, which had a coil diameter of 14 cm and maximal output of 2 Tesla, is definitely not a reliable clinical "tool" for evaluating comatose patients in the ICU. From what we have learned to date, such stimulators appear equally unsuitable for use with anesthetized patients. Our comparison of the time course and the degree of spinal excitability changes caused by these two types of stimulation supported the results of Hess et al. (1987) and Rothwell et al. (1989), who concluded that magnetic transcranial stimuli act

mainly presynaptically. Thus, compared to electrical stimuli, magnetic transcranial stimuli must cross an additional synapse, which makes magnetic stimuli more likely than electrical stimuli to be affected by outside factors. This also explains the high proportion of false-negative findings with magnetic stimulation.

In conclusion: 1) magnetic transcranial stimulation is not particularly useful in comatose patients because it is less efficient in eliciting an mMEP than electrical stimulation and because it is more likely to be influenced presynaptically by many additional factors, and 2) the techniques of facilitating mMEP by adding peripheral nerve stimulation seems to help reduce the incidence of false-negative mMEP in comatose patients, in whom intentional contraction of the target muscles is impossible.

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Facilitation of Muscle Responses to Transcranial Magnetic Stimulation of the Motor Cortex by Trains of Afferent Electrical Impulses

U. D. Schmid[1], M. Date, J. Schmid, and C. W. Hess

Summary

Compound muscle action potentials elicited by magnetic stimulation of the cortex (CortStim) can be facilitated by voluntary background contraction of the target muscle, or, independent of the cooperation of the subject, by trains of peripheral afferent electrical impulses.

The differential effect of an afferent impulse train applied to the median nerve at the wrist on the responses from the M. abductor digiti minimi (ADM) and M. interosseus dorsalis I (IDI) following CortStim was investigated. Impulse trains of 10-ms duration, supramaximal stimulus intensities, and a train frequency of 400/s were used. The time interval between train start (TS) and CortStim was varied. The amplitudes of responses to CortStim with median nerve stimulation were compared to those obtained by CortStim alone.

When the train was started 10 ms prior to CortStim, mean amplitudes of compound muscle action potentials (CMAP) were enhanced by 300–1000%, and the amplitudes were enhanced by as much as 1500% with a TS of 50 ms prior to CortStim. With a TS of 20–30 ms, amplitudes decreased to below 100%. The differences were statistically significant by the Wilkoxon-Mann-Whitney u-test.

The facilitation seen with a TS of 10 ms may indicate events occurring in the spinal tracts, while the peak seen with a TS of 50 ms may reflect reflex events in the supraspinal or cortical areas. Preinnervation of the target muscles alone resulted in four times the degree of facilitation observed with an afferent impulse train alone.

Responses from the small hand muscles elicited by transcranial magnetic stimulation (Barker et al. 1985) can be enhanced by voluntary contraction of the target muscle, of a neighboring muscle of the same hand, or of a homologous muscle of the contralateral hand. This phenomenon is called facilitation and consists of a shortening of onset latencies and an increase of amplitudes of the muscle response. In general, facilitation depends on the intensity of magnetic stimulation used and on the strength of the voluntary muscle contraction (Hess et al. 1987a).

Where these facilitatory events take place in the central nervous system and by what mechanism they occur are controversial. Two main levels of motor path-

[1] Inselspital, Neurochirurgische Universitätsklinik, CH-3010 Bern, Switzerland.

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ways that influence the size of a motor response to magnetic cortex stimulation may contribute to the facilitation. When subthreshold cortical stimuli are presented, voluntary background muscle contraction facilitates the response and there is a linear increase in the amplitudes of muscle responses. The site of this effect has been traced to the spinal level.

When suprathreshold magnetic cortex stimuli are presented, background contraction of the target muscle facilitates the response exponentially; facilitation is at its peak when the target muscle contracts by 10% (Hess et al. 1987a), and appears as a shortening in the latency of the response and an increase in response amplitude. These effects are typically seen with transsynaptic cortical stimulation by magnetic impulses in awake subjects and during REM-sleep (Hess et al. 1987b); these effects have not been observed after electrical transcranial stimulation, and have been attributed to cortical mechanisms (Hess et al. 1987a).

Other methods must be used to facilitate muscle responses in patients in pathological coma or under general anesthesia. Facilitation effects similar to those caused by voluntary background muscle contraction have been observed in response to application of trains of afferent electrical stimulation of a peripheral nerve before electrical (Bergamasco et al. 1987) or magnetic stimulation (Schmid et al. 1991; also see chapter by Taniguchi et al. in this volume; Date et al. 1991) of the motor cortex. A similar effect has been observed when high-frequency vibration is applied to the tendon of the target muscle before the motor cortex is stimulated (Claus et al. 1988).

It was the aim of the present study to investigate the effects of supramaximal electrical stimulation of the median nerve on the responses from the M. Abductor digiti minimi (ADM) and M. interosseus dorsalis I (IDI) elicited by transcranial magnetic stimulation of the motor cortex with near threshold stimulus intensities.

Subjects and Methods

Eight healthy subjects (1 woman, 7 men) aged between 24 and 36 years (mean 26.9 years) volunteered for the experiments. The experimental methods complied with the standards of the local ethical committee.

Stimulation and Recording

Magnetic cortical stimulation was performed with the commercially available Magstim 200 (MAGSTIM Co Ldt, Whitland Industrial Estate, Whitland, Dyfed, SA OHR, UK). The stimulating coil consisted of concentric windings of copper wire; it had a mean diameter of 9.5 cm, a maximal internal resistance of 12 ± 5 Ohm, and an inductance of 35 ± 5 μH at maximal discharge. It had a total capacitance of 800 μF, a maximal charging energy of 2000 J, and a maximal output voltage of 2.8 kV, which corresponds to a maximal induced magnetic field of 2 Tesla in the center of the coil. For cortical stimulation, the coil was placed tangentially over the vertex, and the current flow was clockwise as seen from behind for the right hemisphere (Hess et al. 1987a). Threshold stimuli of about 1 Tesla were

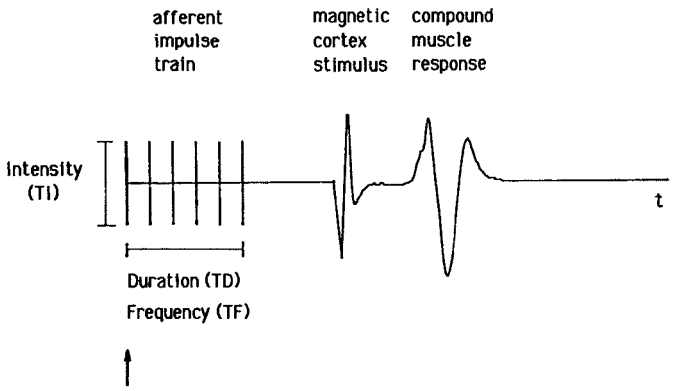


Fig. 1. Schematic representation of parameters varied in the afferent impulse train to the median nerve prior to the transcranial magnetic stimulation to the contralateral motor cortex. *t*, time

used in order to give a just-visible electromyographic (EMG) response of about 100 μV in the recorded muscles, when the subject was relaxed.

The peripheral afferent nerve stimuli that were applied before cortex stimulation were applied to the left median nerve at the wrist. Triggered trains of rectangular electrical impulses of 0.1 ms duration each were administered by surface electrodes (Tönnies). On the basis of the results of preliminary experiments in 5 subjects, 1) stimulus intensity was adjusted to give a supramaximal response from the M. abductor pollicis brevis (APB), 2) stimulus train duration (TD) was set at 10 ms, and 3) the frequency of the stimulus train (TF) was set at 400/s. The interval between the start of the train and the cortex stimulus (TS) was varied using a Digitimer D 4030 (Fig. 1).

Responses were recorded from the left ADM and IDI. Peak-to-peak amplitudes of the EMG responses were measured. Comparison of amplitudes to cortical stimulation with and without afferent impulse trains was done by expressing the amplitudes in percentages of the values obtained after cortical threshold stimulation alone ("no train"). The arithmetic mean of 6 to 12 amplitude measurements was taken to calculate mean amplitudes; thus, the mean amplitude after cortical threshold stimulation alone was set at 100%.

The *effect of a varying the start of the train* in relation to the cortical stimulus was evaluated by first administering a stimulus train of 10 ms prior to the cortical stimulus, and then successively increasing the interval between the train and the cortical stimulation to 20, 30, 40, and 50 ms. The effects of the various afferent impulse trains were statistically compared using the Wilkoxon-Mann-Whitney u-test (u-test).

Results

Fig. 2 A shows the responses from the ADM in 1 subject, and Fig. 2 B shows the relative amplitudes of responses from the ADM, as well as their mean values, in the same subject. Fig. 3 shows the results obtained with the same recording technique in 3 other subjects.

Increasing the time interval between train and cortical stimulation resulted in biphasic facilitation of responses in all subjects tested. When the train was started 10 ms before cortex stimulation, mean relative amplitudes of up to 500% could be observed, whereas when the train was started 50 ms before the cortex stimulation increases in mean amplitudes of up to 1400% of the control values were observed. Between these 2 maxima, i.e. when the trains were started 20 or 30 ms before cortex stimulation, significant – up to 100% – inhibition of the motor responses was observed in some cases.

Fig. 4 shows the mean of relative amplitudes in simultaneous recordings from the IDI and ADM muscles in 1 subject when the train to the median nerve was started 10–50 ms before the cortical stimulus. Fig. 4 also shows the statistical comparison between the values recorded from the IDI and ADM muscles. Fig. 4, as do Figs. 2 and 3, shows a facilitation phase followed by an inhibition phase, followed in turn by a second facilitation phase. Amplitudes of the IDI responses were significantly greater than those of the ADM responses in this case; when the train was started at 5 or 10 ms, IDI responses rose to as much as 1500% of the control value compared to 300% for the ADM. No difference in the sizes of the responses was found when the train was started 50 ms before cortical stimulation.

Discussion

We have shown earlier that afferent electrical impulse trains of 10-ms duration when applied to the median nerve at the wrist at supramaximal intensity (so as to give a maximal M-response from the APB) and with a train frequency of 300–400/s are able to facilitate or inhibit a cortically evoked response from the IDI and ADM muscles (Schmid et al. 1991).

The time that the train starts in relation to the onset of the cortex stimulus determines whether this impulse train exerts an inhibitory or a facilitatory effect on the cortically evoked muscle response. Two distinct peaks of facilitation of the muscle responses were observed consistently: one peak was seen when the impulse train preceded the cortical stimulation by 5–10 ms, and the second peak was seen when the train was presented 50 ms before magnetic cortical stimulation. Other studies have shown that latencies remain unchanged with TS at 10 ms, but shorten significantly with TS at 50 ms (Schmid et al. 1991; Date et al. 1991).

From the short time interval between the impulse train to the median nerve at the wrist and the cortex stimulus, it must be assumed that facilitation with a train start at 10 ms acts anatomically at a *spinal level.* In fact, the results of similar experiments, reported elsewhere in this book by Taniguchi et al. suggest that the monosynaptic Hoffmann-Reflex plays an important role in mediating this "early

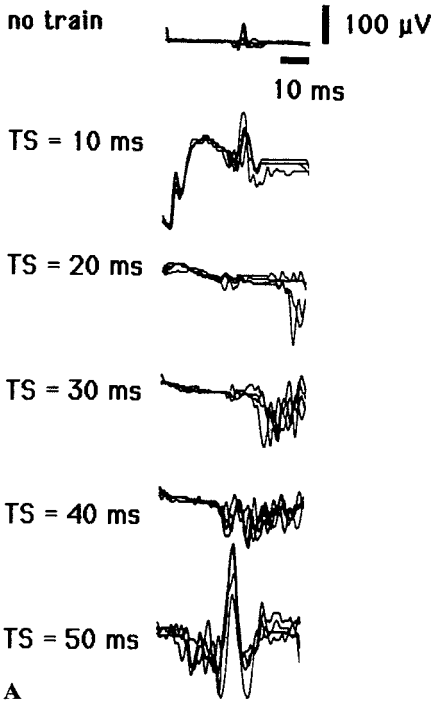
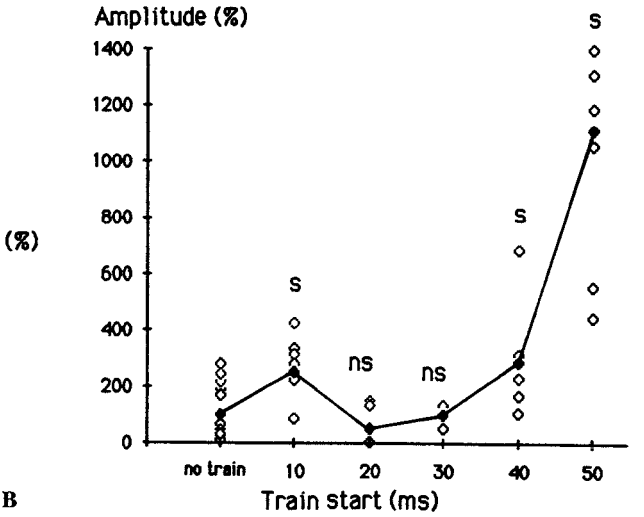


Fig. 2. A Responses from the ADM after magnetic cortex stimulation alone ("no train"), and after magnetic cortex stimulation with an afferent impulse train applied to the median nerve at the wrist beginning between 10 and 50 ms before the cortical stimulation.



B Relative amplitudes of ADM responses (open symbols) and their mean values (filled symbols) for the recordings shown in Fig. 2 A. The values obtained after cortical stimulation with afferent impulse trains were compared statistically to the values after cortical stimulation alone ("no train"). *s*, significant; *ns*, not significant (u-test)

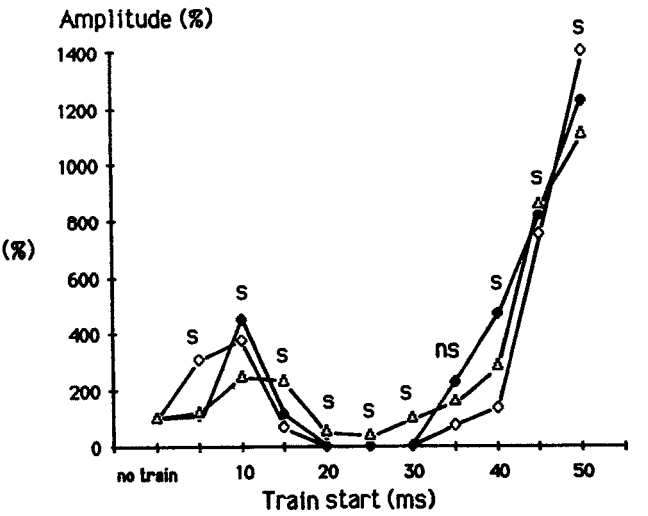


Fig. 3. Mean relative amplitudes of ADM responses obtained in 3 subjects under the same experimental conditions as described for Fig. 2. The values obtained after cortical stimulation and application of afferent impulse trains were statistically compared to the values after cortical stimulation alone ("no train"). *s*, significant; *ns*, not significant (u-test)

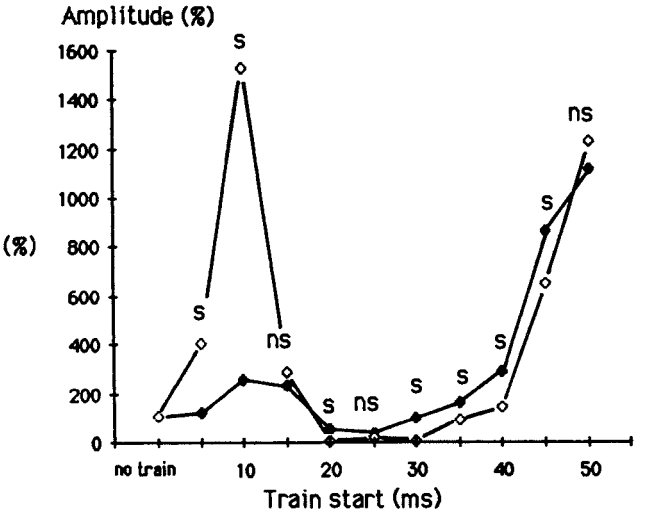


Fig. 4. Mean relative amplitudes of IDI and ADM responses recorded in 1 patient under the same experimental conditions described for Figs. 2 and 3. The values obtained for ADM and IDI were statistically compared for each train start. *s*, significant; *ns*, not significant (u-test)

facilitation," a finding that has been found also by others (Deutschl et al. 1989). However, such reflexes have been elicited predominantly after subthreshold afferent stimulation as opposed to the suprathreshold stimulation used in our study. Thus, it is likely that additional, probably multisynaptic, spinal reflex mechanisms involving other fibre populations may also contribute to this phenomenon. Another interpretation would be that the H-reflex elicited in the small hand muscles, as opposed to the lower limb, is not inhibited by increasing the stimulus intensity above the threshold for the maximal response from the APB.

In contrast, when the electrical stimulus train is started 50 ms prior to the cortex stimulus, there is, even for slow-conducting afferent fibres, enough time for a facilitation mechanism to act at as high as *supraspinal or cortical levels.* Responses after such "late facilitation" are therefore much more enhanced than those after "spinal facilitation" alone. The facilitation mechanisms that might play a role in such cases are involuntary movements occurring in response to the relatively painful stimulus train to the wrist in the awake test subjects, or, in general, transcortically conducted long-loop reflexes. In fact, experiments that approached this problem from the opposite direction (Deuschl et al. 1989) showed that the amplitude of a long-loop reflex response can be enhanced when a conditioning transcranial magnetic stimulus is given to the contralateral motor cortex. The fact that, in addition to the increased amplitudes, shorter onset latencies of responses were observed when the afferent train was started 50 ms before cortex stimulation (Schmid et al. 1991; Date et al. 1991) supports our presumption that cortical mechanisms play a role in facilitating the responses.

The transcortically mediated long-loop reflex is influenced by the state of consciousness and degree of cooperation of the subjects, and it diminishes when the subjects are asleep. We would therefore expect that the effect of such "late facilitation" would be reduced when cortical stimulation is performed on comatose patients. It remains to be seen whether "spinal facilitation" alone is sufficient to enhance muscle responses to magnetic cortical stimulation in comatose or anesthetized patients.

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Motor Evoked Potential Monitoring in Operations of the Brainstem and Posterior Fossa

J. ZENTNER[1]

Summary

In order to monitor descending pathways during neurosurgical operations on the brainstem and posterior fossa, motor evoked potentials (MEP) elicited by transcranial electrical stimulation were recorded from the thenar and anterior tibial muscles as well as from the epidural space of the cauda equina in a total of 29 patients. The aim was to test intraoperative recordability of potentials and their correlation with the patient's postoperative neurological condition. Overall, we were able to record MEP intraoperatively in 84.5% of cases, and there were no essential differences in recordability among recording sites used. When an acceptable level of variability in MEP amplitude from the baseline at the end of the operation was taken to be 50%, the changes in the recorded potentials correlated with the postoperative neurological condition in 79.6% of cases, with 20.4% false-positive results. There were no false-negative findings. Recordings from the cauda equina were slightly superior to those obtained at the other recording sites. Postoperative deterioration of MEP was observed in 3 patients and coincided in all cases with a permanent reduction in MEP amplitudes of 70 to 90% relative to the baseline. It is concluded that MEP elicited by transcranial electrical stimulation of the motor cortex and recorded from cauda equina as well as from muscles is a sensitive means to detect impending motor complications. However, in order to improve recordability of potentials and reliability of the technique, more experience must be gained in managing anesthesiological and patient-related aspects of monitoring, and acceptable limits for changes in potentials must be established.

Evoked potentials may be used in the operating room: 1) to identify local and systemic neurological impairment early enough to allow correction of its cause, and 2) to provide reassurance to the surgeon during the course of the operation that complications are unlikely to have occurred (Nuwer, 1986). To achieve these goals, intraoperative monitoring of somatosensory evoked potentials (SSEP) has been used for nearly two decades. However, motor impairment has always been the most feared complication of surgical manipulation in the vicinity of the descending pathways, and because motor and sensory pathways travel along

[1] Neurochirurgische Abteilung der Universität, Hoppe-Seyler-Str. 3, W-7400 Tübingen, FRG.

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separate tracts, each with its own vascular supply, it is possible in theory to injury one while leaving the other intact. In practice, acute changes usually affect both motor and sensory pathways. However, a few isolated cases have been documented in the literature in which motor impairment occurred even when the SSEP remained relatively stable (Ginsburg et al. 1985; Lesser et al. 1986; Levy 1983). As noninvasive motor cortex stimulation has been developed (Merton and Morton 1980; Barker et al. 1985), monitoring of descending pathways has become of increasing interest. This is especially true since animal studies of spinal cord trauma and ischemia confirmed the close relationship between changes in motor potentials and the patient's neurological condition (Fehlings et al. 1987; Konrad et al. 1987; Oro and Levy 1987; Patil et al. 1985; Simpson and Baskin 1987).

Several studies have shown that recording motor evoked potentials (MEP) is a reliable tool in assessing motor function during orthopedic and neurosurgical operations on the spinal cord (Boyd et al. 1986; Levy et al. 1984; Levy 1987; Pelosi et al. 1988; Katayama et al. 1988). Our experiences managing 83 patients during operations to remove spinal cord lesions confirm these results. Therefore, we began to use MEP monitoring in patients who were at risk of damage to motor function while undergoing neurosurgical treatment for brainstem and posterior fossa lesions. The goal of the work we report here was to evaluate both the recordability of MEP from the cauda equina and from muscles in response to transcranial electrical stimulation and to determine the correlation between such intraoperative recordings and the patient's postoperative motor status.

Patients and Methods

A total of 29 patients (18 males, 11 females) at ages between 5 and 79 years, and an average age of 48 years, were examined. Table 1 categorizes the patients by diagnosis. Disease was located around the fourth ventricle and pons in 10 cases (34.5%), in the craniocervical junction in 7 cases (24.1%), in the cerebellopontine angle (CPA) in 5 cases (17.2%), in the medulla oblongata in 4 cases (13.8%), and in the quadrigeminal region in 3 cases (10.4%). The lesion was tumorous in 23 cases (79.2%) and nontumorous in 6 cases (20.8%).

The motor cortex was stimulated transcranially with condenser discharges (constant voltage) using a Digitimer D 180 stimulator (Digitimer, Welwyn Garden City, Hertfordshire, England). The time constant of discharges was 200 μs. Single stimuli were applied at intervals of at least 3 s. Standard Ag/AgCl electrocardiogram (EKG) electrodes were used. The anode was placed over the foot of the precentral gyrus and the cathode at the bregma for stimulation of the motor hand area. When the motor leg area was stimulated the anode was placed at the bregma and the cathode was placed 6 cm posterior at the midline. In all patients electromyographic (EMG) responses were recorded from the anterior tibial muscle, and in 18 patients EMG responses were obtained from the thenar muscles on both sides as well. Standard EMG surface electrodes were used with one placed on the belly of the muscle and the other on the tendon. Furthermore, in 11

Table 1. Patients and diagnoses

Diagnosis	Patients	
	No.	%
Glioma	5	17.1
Ependymoma	4	13.9
Cavernoma	4	13.9
Neurinoma	3	10.3
Meningioma	2	6.9
Germinoma	2	6.9
Medulloblastoma	1	3.4
Epidermoid	1	3.4
Chordoma	1	3.4
Nontumorous	6	20.8
Total	29	100.0

patients we also recorded from the epidural space of the cauda equina through standard bipolar cardiac pacemaker electrodes, which were placed after induction of anesthesia by puncture at L3-4 using a Tuohy cannula.

Patients were premedicated with 20 mg trifluprom azine, 50 mg pethidine, and 0.5 mg atropine administered i.m. After an anesthetizing dose of 250 to 300 mg thiopental, 1 mg/kg succinylcholine was administered before intubation. No muscle relaxants were given thereafter. Usually, anesthesia was maintained by continuous administration of fentanyl and midazolam and the patient breathed an oxyen/air mixture. In addition, flunitracepam (single dose 0.02 mg/kg) or thiopental (single dose 2 to 3 mg/kg) was administered as required. Only a few patients received neurolept anesthesia based on nitrous oxide.

Baseline responses were recorded after the relaxation induced by drugs for intubation had waned. Stimulus strength was gradually increased until a clear response was obtained or the absence of any response to a stimulus strength of 750 V was documented. Once the stimulus strength that gave a clear response had been determined, the stimulus strength was kept constant at this level throughout the monitoring procedure. A Nicolet Compact 4 was used for the recordings with a time base of 100 ms, and the gain was adjusted to produce 10 and 20 μV gave a deflection of one division. The filter settings were 20 Hz lowpass and 3 kHz highpass. An upward deflection always represented negativity of the active electrode.

Stimulation was performed and responses recorded during major steps of the operation and at the end of the operation. Usually, the averages of 5 to 10 responses were evaluated.

Onset latencies of the initial negative wave as well as peak-to-peak amplitudes of the responses were measured. We evaluated intraoperative changes in potentials on the basis of how much the amplitudes of the responses varied at the end of the operation from the baselines obtained after induction of anesthesia. We regarded 50% or less change as acceptable and correlated the MEP findings with the patient's postoperative neurological status. Thus, we defined three categories: 1) good correlation of postoperative motor status and intraoperative

change in amplitudes such that baseline and postoperative MEP amplitudes differ by no more than 50% and pre- and postoperative motor function are unchanged or postoperative motor functions worse than preoperative function and MEP amplitudes differ by more than 50%; 2) false-positive permanent reduction in amplitudes of more than 50% with unchanged postoperative motor status; and 3) false-negative change in MEP amplitudes of less than 50% compared with the baselines but deteriorated postoperative motor status.

The patients were fully informed about and gave their consent to the procedure.

Results

The results of intraoperative monitoring were evaluated with regard to: 1) intraoperative recordability of potentials, and 2) correlation of intraoperative changes in potentials with postoperative motor function as established by clinical examination (Tables 2, 3).

MEP were recordable intraoperatively from the thenar muscle in 16 of 18 patients (88.9%), from the anterior tibial muscle in 24 of 29 (82.7%), and from the cauda equina in 9 of 11 (81.8%). The stimulus strength used was 450 to 750 V. On the whole, 49 of 58 recordings (84.5%) produced useful potentials (Table 2). In the remaining patients, no responses were obtained intraoperatively despite the use of a maximal stimulus strength of 750 V.

Using the criterion that changes in amplitudes at the end of the operation of up to 50% of baseline were acceptable, a postoperative motor status equal to

Table 2. Intraoperative recordability of potentials

Recording Site[a]	Patients No.	MEP No.	Recordable %
Thenar muscle	18	16	88.9
Tibial muscle	29	24	82.7
Cauda equina	11	9	81.8
Total	58	49	84.5

[a] In several patients more than one recording site was used.

Table 3. Correlation of potentials with postoperative motor function

Recording Site[a]	Patients No.	Correct No.	Correct %	False-Positive No.	False-Positive %	False-Negative No.	False-Negative %
Thenar muscle	16	13	81.3	3	18.7	–	–
Tibial muscle	24	18	75.0	6	25.0	–	–
Cauda equina	9	8	88.9	1	11.1	–	–
Total	49	39	79.6	10	20.4	–	–

[a] In several patients more than one recording site was used.

preoperative status was correctly predicted in 13 of 16 (81.3%) recordings from the thenar muscle and in 18 of 24 (75.0%) from the anterior tibial muscle, while false-positive results were obtained in 3 (18.7%) and 6 (25.0%) cases, respectively. Cauda equina recordings correlated with good results in 8 of 9 patients (88.9%) and a false-positive result was obtained in 1 case (11.1%). On the whole, 39 of 49 recordings (79.6%) correlated well with motor function, but 10 of 49 (20.4%) produced false-positive results. It is important to note that there were no false-negative results in our series (Table 3).

Motor function was the same postoperatively as preoperatively in 26 of our 29 patients. Although MEP amplitudes varied noticeably in most of these patients during the operation, in none was there a difference of more than 50% between values obtained at the end of the operation and the baselines obtained after induction of anesthesia. It is worth mentioning that amplitudes of responses recorded from the cauda equina were more stable than amplitudes of the EMG responses recorded from muscles. In 3 of our 29 patients, motor function deteriorated during the operation, and in all 3 the amplitudes of the responses were permanently reduced by 70 to 90% relative to the baseline values at the end of the operation. No total loss of potentials was observed intraoperatively, however, in our series. Figures 1 through 3 show examples of intraoperative potentials in individual cases.

Depending on the degree of pre-existing motor deficit, latencies of the MEP were quite different among individuals. This was the case both for the responses recorded invasively and those recorded noninvasively. Latencies of responses recorded from the thenar muscle varied between 19 and 25 ms, and latencies of responses recorded from the anterior tibial muscle were between 28.5 and 39 ms. Corresponding latency values for responses obtained from the cauda equina varied between 9.5 to 17 ms. Within each individual, however, there were no noticeable changes in the latencies of the potentials recorded epidurally, although latencies in some muscle recordings increased up to 2.5 ms during the course of the operation. However, because latencies did not correlate well with postoperative motor function, latencies were not used in assessing spinal cord function.

Discussion

Although the electrical irritability of the brain was discovered in the last century (Fritsch and Hitzig 1870; Ferrier 1875; Horsley and Schäfer 1884; Mills 1889), it was not until the 1950s that direct electrical stimulation of the cortex was used for intraoperative identification of the motor cortex in humans (Penfield and Rasmussen 1950). An essential step was taken in 1954 when Gualtierotti and Paterson succeeded in activating the motor cortex transcranially to cause movement of contralateral extremities. However, it was not until 1980 that Merton and Morton developed electrophysiological techniques based on the work of Hill et al. (1980) that could be applied to humans. Modifications of this method were reported by Levy et al. (1984), Hassan et al. (1985), Rossini et al. (1985), and Amassian and Cracco (1987), that resulted in less current being required to activate the motor

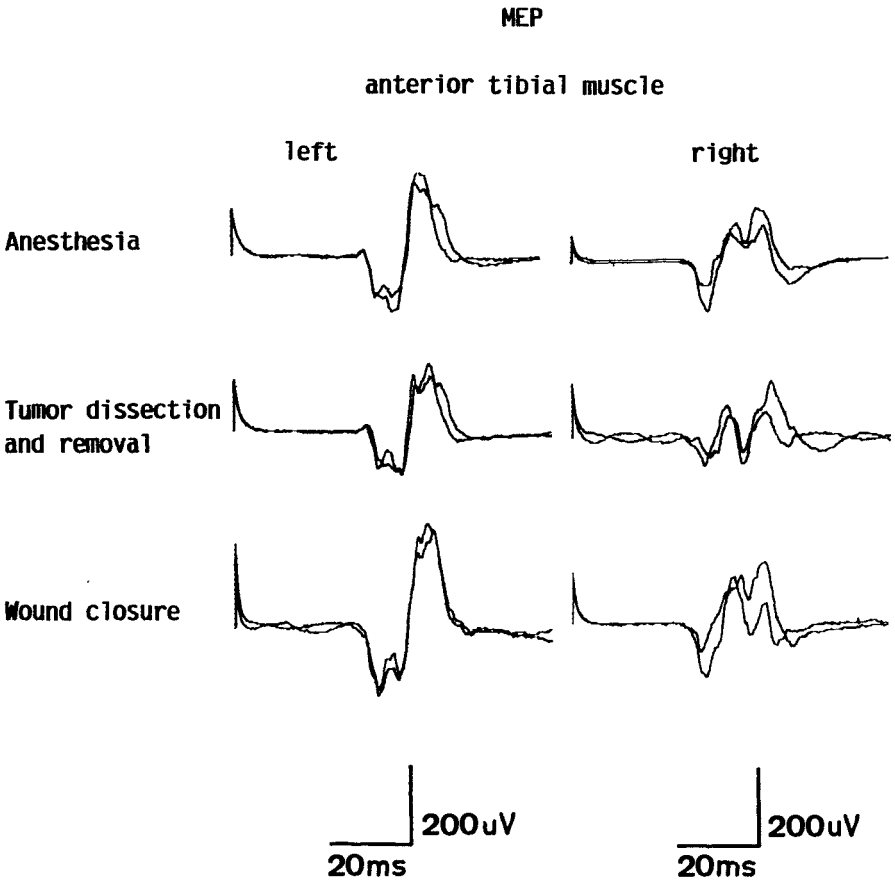

Fig. 1. Anterior tibial muscle MEP of a 76-year-old man recorded during removal of a right-sided craniocervical meningioma. Notice that the potentials of the affected side show lower amplitudes than those of the unaffected side in response to the same stimulus strength (650 V), while the amplitudes and latencies remained essentially the same during the surgical procedure. The postoperative motor status was the same as the preoperative status.

cortex. Once these modifications were reported, MEP began to be used clinically to assess the descending pathways in demyelinating and other neurological disorders (Cowan et al. 1984; Snooks and Swash 1985; Mills and Murray 1986; Rossini et al. 1987). Several reports have appeared regarding the use of MEP for intraoperative monitoring during orthopedic, vascular, and neurosurgical operations (Levy et al. 1984; Boyd et al. 1986; Tsubokawa 1985; Tsubokawa et al. 1986; Levy 1987, 1988; Katayama et al. 1988; Pelosi et al. 1988). Technical variations have been proposed: stimulation can be performed transcranially or the motor cortex may be stimulated directly; single or repeated stimuli may be used; and responses can be recorded from the muscles of the extremities, peripheral nerves, or the epidural space along the spinal cord and cauda equina. Although there is

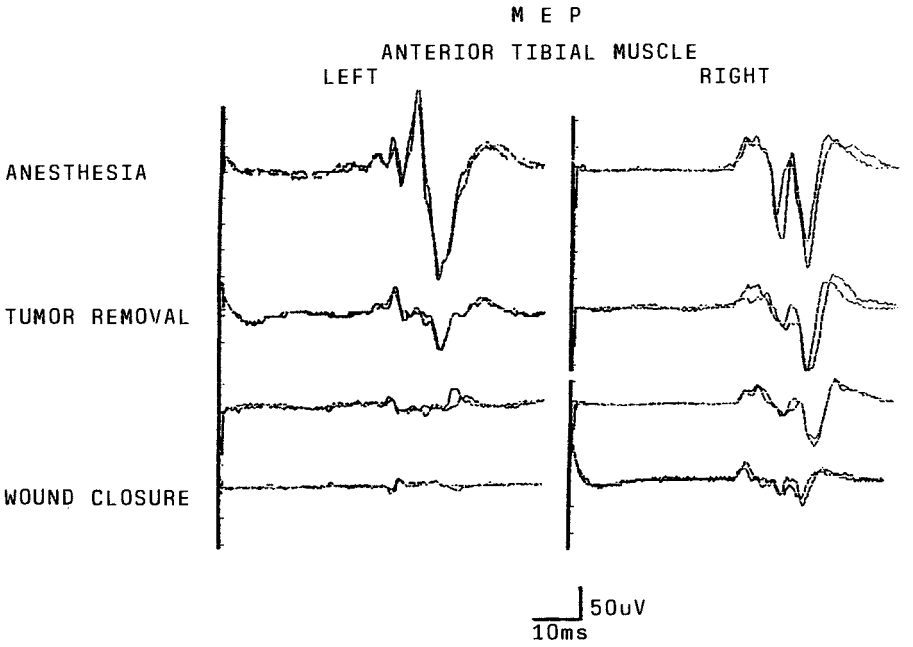

Fig. 2 Illustration of changes in anterior tibial muscle MEP of a 39-year-old woman with a brainstem glioma during tumor dissection and removal. The patient's postoperative motor status was worse than preoperatively.

no consensus on the most suitable stimulation and recording technique, all authors who have reported on such monitoring agree that recording MEP might prove useful in lowering the postoperative morbidity of operations affecting descending pathways.

Our results also indicate that recording MEP is an efficient way to detect impending motor deficits intraoperatively. When we have amplitudes 50% of baseline values obtained after induction of anesthesia as the acceptable limit for amplitudes at the end of the operations, we found good correlation between this degree of change in MEP amplitudes and no change in motor status or between larger MEP changes and a decrease in motor status in all cases. It is important that no false-negative results occurred in our series using this limit. Recordings from muscles of the upper and lower extremities showed similar correct results (81.3 and 75.0%, respectively) between changes in MEP amplitudes and motor status. Recording from the cauda equina seems to be slightly superior with regard to stability of potentials in that recording results correlated with motor function changes in 88.9% of cases. Nevertheless, a problem is that permanent reduction in amplitudes of more than 50% does not necessarily indicate deterioration of neurological condition, as we found false-positive results in 11.1 to 25% of recording situations. Postoperative deterioration of the motor status in our 3 patients was found to coincide with reduction in amplitudes of the intraoperative MEP of

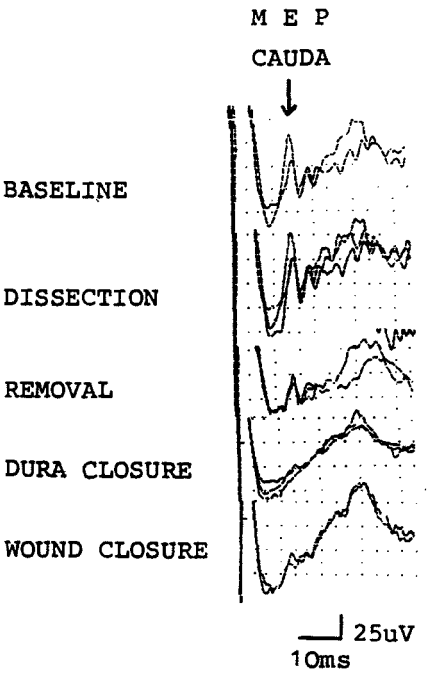

Fig. 3. Cauda equina MEP of a 34-year-old woman obtained during an operation to remove a craniocervical arteriovenous malformation. Only cauda equina potentials were obtained after induction of anesthesia. Notice the incomplete loss of potentials during removal of the lesion. The patient had a temporary postoperative tetraparesis.

70 to 90%. Therefore, there is still some ambiguity in interpreting amplitudes, and improved correlation between intraoperative MEP recording and postoperative status may be obtained if more reliable limits can be defined. Our experience with spinal cord monitoring shows that only intraoperative loss of potentials undoubtedly indicates poor outcome.

Although we found differences among individuals in the latencies of both muscle and spinal potentials, intraoperative changes in latencies did not correlate well with postoperative motor function, as shown by the finding that an increase in latency of up to 2.5 ms during an operation coincided in only one case with a postoperative deterioration of motor status. The differences in the latencies of the MEP are presumably results of pre-existing damage to motor pathways. In our opinion, increases in latencies are more likely to result from systemic changes that occur during operations, such as a drop in body temperature, than to damage of neural structures. Therefore, changes in the amplitudes of the MEP seem to be superior to changes in latencies for evaluation of intraoperative changes that are of importance in detecting injuries that may result in permanent neurological deficits. These findings are in accordance with the results of animal studies of acute and chronic spinal cord injury, in which changes in amplitudes of the recorded MEP were noted in the posttraumatic period but latencies were essentially unchanged (Fehlings et al. 1987; Patil et al. 1985).

In all of our patients, preoperative MEP were recordable, while intraoperative recordability of potentials was only 84.5% on the average, with no essential dif-

ferences being noted among the recording sites used. This indicates noticeable anesthesia-related suppression of MEP. Our experiences with the use of inhalational agents such as halothane, enflurane, or isoflurane have shown that EMG responses were completely abolished at concentrations beyond 1 MAC. It has also been shown that nitrous oxide is a major suppressor of EMG potentials (Zentner 1989; Zentner et al. 1989). In our experience, continuous infusion of intravenous narcotics such as fentanyl and midazolam and allowing the patient to breathe an oxygen/air mixture is the type of anesthesia most conducive to intraoperative MEP monitoring. The main disadvantage of this special anesthesia, however, is that in these patients ventilation usually must be controlled for 1 to 2 hours after the end of the operation. In addition to the use of sophisticated techniques for anesthesia, we believe these three possible ways to improve intraoperative recordability of potentials should be studied: 1) facilitation of responses by afferent electrical impulses, 2) the use of a higher stimulus strength, and 3) recording of neural activity along the spinal cord, which can be expected to be less influenced by anesthesia than is muscular activity.

We encountered no complications of electrical stimulation in our patients. The results of ultrastructural animal studies seem to show that applied charge density is most highly correlated with damage to neural structures, and it has been recommended that the charge density per phase not exceed 40 $\mu C/cm^2$ (Agnew and McCreery 1987). However, data on just what stimulation parameters – such as number of stimuli, stimulation frequency, charge per phase, and total applied charge – are acceptable must be elaborated in order for us to feel secure using this technique.

To conclude, intraoperative examination of MEP elicited by transcranial electrical cortex stimulation and recorded from extremity muscles and the epidural space of the cauda equina is a useful method for detection of impending motor deficits. Further experience with this technique is necessary for anesthesiological and patient-related influences on MEP such as body temperature, blood pressure, cerebral perfusion, and blood gases to be more precisely defined and for clear definition of acceptable limits for changes in amplitudes. Moreover, it must be clarified whether invasive or noninvasive recording techniques are preferable. When more experience has been gained in managing these problems, intraoperative MEP monitoring may become a feasible method for routine electrophysiological assessment of the descending pathways, and its use in combination with SSEP monitoring might provide surgeons greater securty in treating lesions affecting the descending and ascending pathways.

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Intraoperative Monitoring of Corticospinal Motor Evoked Potentials in Neurosurgical Operations

T. TSUBOKAWA[1]

Summary

1) The D-wave of corticospinal tract (CST) motor evoked potentials (MEP) recorded from the epidural space of the human spinal cord reflects synchronous impulses mediated by CST axons activated directly, without any intervening synapses.

2) The location of the motor cortex can be identified during intracranial procedures by recording corticospinal MEP. The functioning motor cortex is sometimes displaced in patients with brain tumors or arteriovenous malformations.

3) Corticospinal MEP are specific indicators of CST function. Therefore, intraoperative monitoring of corticospinal MEP provides useful information about possible damage to CST axons from manipulations during intracranial and spinal operations. This technique has been used in more than 300 cases to date, and no complications have yet been noted.

The possibility that a severe deficit in motor function may occur as a result of intraoperative damage to the corticospinal tract (CST) is a major concern when operations are performed on lesions located close to the CST. Because of this concern, neurosurgeons have long held the hope that intraoperative monitoring of CST function could help reduce such problems. Signals monitored for this purpose must have the following characteristics: 1) they must be recordable rapidly and constantly, 2) they must be minimally influenced by anesthesia or other drugs used in such operations, 3) intraoperative changes must be specific to changes in CST function, and 4) intraoperative changes must correlate well with postoperative deficits in motor function.

Electromyographic (EMG) responses to stimulation of the motor cortex obviously involve CST neurons (Rasmussen and Penfield 1947). Intraoperative monitoring of corticomyographic motor evoked potentials (MEP) for this particular purpose was, however, abandoned since none of the requirements mentioned above was met. There are two major problems. First, because EMG are affected by muscle relaxants, it is difficult to keep the amplitudes of corticomyographic MEP constant during general anesthesia sustained by muscle-relaxing agents. Second, corticomyographic MEP are responses relayed by spinal motoneurons.

[1] Department of Neurological Surgery, Nihon University School of Medicine, Itabashi-ku, Tokyo 173, Japan

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Because the excitability of spinal motoneurons is profoundly depressed by inhalation and intravenously administered agents used for general anesthesia, large stimulating currents are usually required to evoke corticomyographic MEP under general anesthesia, and the amplitudes of such corticomyographic MEP are greatly affected by the level of anesthesia (Katayama et al. 1988b). The excitability of spinal motoneurons is also under the influence of a number of neural pathways other than the CST. Therefore, changes in the amplitudes of the recorded MEP are not necessarily specific to changes in CST function. For these reasons, it is not appropriate to use corticomyographic MEP for intraoperative monitoring of CST function and an alternative method has long been sought.

Until a few years ago, no methods meeting the requirements just mentioned have been available clinically, although experiments in animals have demonstrated that impulses mediated by CST neurons in response to activation of the motor cortex can be recorded directly from the spinal cord (Patton and Amassian 1954). On the basis of these animal study results, we attempted to record this corticospinal MEP from the epidural space of the human spinal cord (Yamamoto et al. 1984; Tsubokawa 1985; Katayama et al. 1988c). After studying these potentials for 6 years, we were convinced that responses recorded in man to stimulation of the motor cortex when exposed during neurosurgical operations are analogous to corticospinal MEP observed in our animal experiments (Tsubokawa 1985; Katayama et al. 1988b), and that monitoring these potentials can be very helpful in evaluation of CST function (Tsubokawa 1985, 1987; Tsubokawa and Katayama 1987; Tsubokawa et al. 1987). We have applied this technique to the intraoperative monitoring of CST function and found that human corticospinal MEP have all the characteristics mentioned at the beginning of this article as being required of signals used to monitor CST motor function (Tsubokawa 1985; Tsubokawa et al. 1988; Katayama et al. 1988b). We have found intraoperative monitoring of corticospinal MEP to be an efficient method to detect when the CST was at risk from surgical manipulations and to avoid postoperative neurological deficits as a result of injury to this structure. This paper summarizes our technique and the results of using it routinely.

Methods

Stimulation Procedures

We use three different methods to stimulate the motor cortex. When the motor cortex is exposed intraoperatively, the motor cortex is stimulated directly with electrode arrays that consist of 4 plate electrodes, each 5 mm in diameter spaced 5 mm apart (Medtronic Co. M-3586, Minneapolis, Minnesota, U.S.A.) (Fig. 1). Two electrode arrays are placed on the hand, two on the trunk, and two on the thigh areas of the motor cortex. Although it is usually impossible to place electrodes on the leg area except when operations are performed in the interhemispheric space, stimulation with an electrode placed close to the edge of the hemisphere produces corticospinal MEP at as low as lumber level. The motor cortex may be stimulated monopolarly by using one of these electrode or bipolarly with

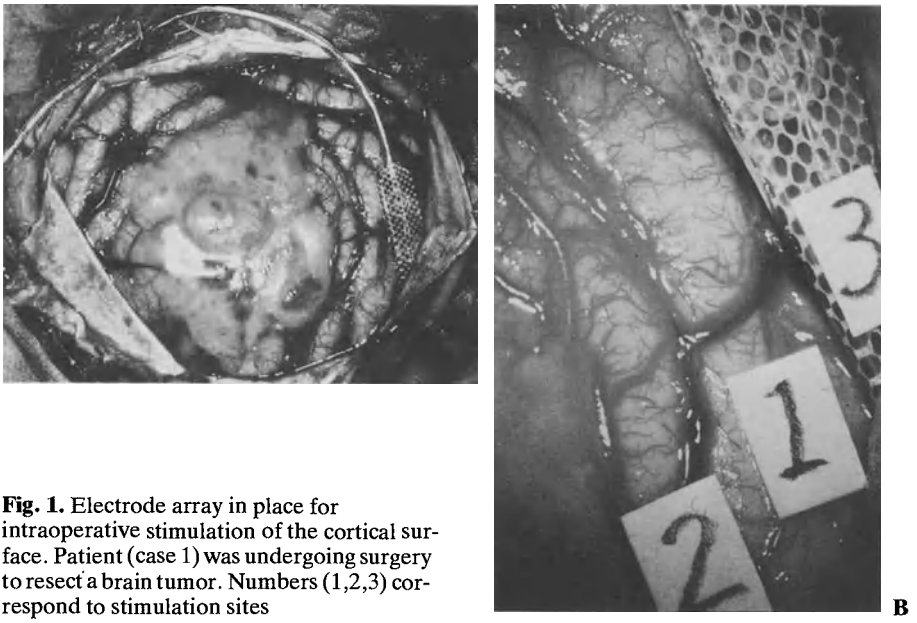


Fig. 1. Electrode array in place for intraoperative stimulation of the cortical surface. Patient (case 1) was undergoing surgery to resect a brain tumor. Numbers (1,2,3) correspond to stimulation sites

various pairs of electrodes. The interpolar distance for bipolar stimulation varied between 10 and 30 mm. The site for stimulation was approximated by the surgeon, using bony landmarks usually identified to locate structures during intracranial surgery. The location of the motor cortex, however, could not be defined precisely in any other way than recording of MEP. Therefore we always looked at the areas from which the corticospinal MEP with the lowest thresholds were recorded.

When the motor cortex was not exposed, such as when the procedure was in the posterior fossa or involved sterotaxic evacuation of an intracerebral hematoma, the electrode array was inserted through a burr hole into the intracranial epidural space overlying the motor cortex.For the monitoring of CST function during spinal procedures, the motor cortex was stimulated through a burr hole (Tsubokawa 1985; Katayama et al. 1988d) or epidurally through small electrodes screwed into the skull (Tsubokawa 1987; Tsubokawa and Katayama 1987; Tsubokawa et al. 1987). Stimuli were applied as monophasic square wave pulses of 0.2 to 0.5 ms duration delivered at 4 Hz, except when otherwise mentioned. Delivery of the stimuli was synchronized by electrocardiographic input so as not to compromise cardiac electrical function.

Recording Procedures

Under fluoroscopic control, 2 flexible platinum electrodes (Medtronic Co. M-8483, Minneapolis, Minnesota, U.S.A.) (Fig. 2) were inserted into the epidural space at the lower cervical or upper thoracic level and advanced rostrally. For

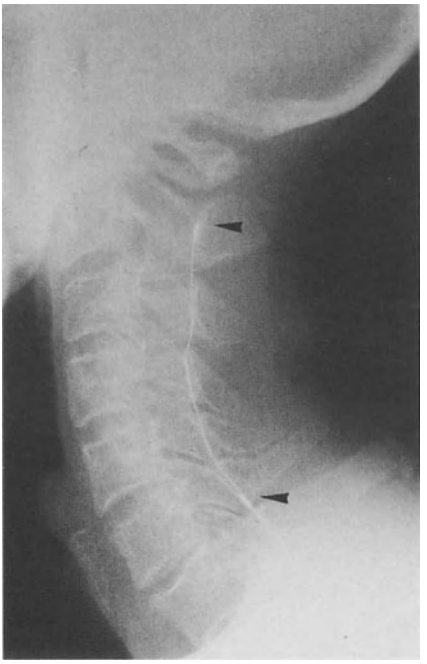


Fig. 2. Roentgenogram showing electrodes *(arrowheads)* inserted into the epidural space of the cervical spinal cord

intracranial procedures one electrode was placed at the level of C2 or C3 and the other at the level of C7 or T1. When operating on the spine, 1 electrode was placed above and the other below the presumed level of the lesion. Corticospinal MEP were recorded monopolarly from the epidural electrodes with a reference electrode placed at the paravertebral muscles, in response to motor cortex stimulation. When necessary, multiple recordings were obtained as the rostrally placed epidural electrode was withdrawn in stepwise fashion (Katayama et al. 1988a, 1989).

During spinal procedures, spinospinal responses were also recorded from the epidural electrode placed above the presumed level of the lesion, in response to stimulation through the epidural electrode placed below the presumed level of the lesion (Tsubokawa 1987; Tsubokawa and Katayama 1987; Tsubokawa et al. 1987). Potentials recorded from the electrodes were amplified with a bandpass range of 5 Hz to 5 kHz and 32 to 64 responses were averaged.

Physiological Characteristics

It has been demonstrated in experimental animals that synchronous impulses occurring in efferent axons of CST neurons in response to stimulation of the motor cortex can be recorded from the spinal cord (Patton and Amassian 1954). The response recorded from the contralateral CST shows an initial wave known

as the D-wave and later a sequence of volleys termed I-waves. The D-wave reflects impulses arising from direct activation of axons of CST neurons, and is induced most efficiently with anodal stimulation of the surface of the motor cortex (Patton and Amassian 1954). The series of I-waves that are seen in such recordings are thought to arise through indirect excitation of CST neurons, and these components of the response are better induced by cathodal stimulation of the surface of the motor cortex (see Amassian et al. 1987 for review). Responses identical in many respects to D- and I-waves can be recorded in human subjects from the epidural space of the spinal cord to stimulation of the motor cortex when the latter is exposed during intracranial procedures (see Tsubokawa and Katayama 1987 for review). Corticospinal MEP in experimental animals were originally recorded from an electrode inserted into the spinal cord and presented as single responses (Patton and Amassian 1954). Advances in techniques for signal averaging together with better placements of spinal epidural electrodes has permitted detection of the same response from the epidural space in man.

Direct Stimulation of the Exposed Motor Cortex

The initial response recorded from the epidural space to stimulation of the motor cortex is a large negative wave preceded by a small positive wave and followed by another small positive wave (Fig. 3). Since the amplitude of this triphasic response is relatively large (7 to 20 μV), averaging 32 to 64 responses is adequate to obtain a clear record. This response can be recorded regardless of whether the recording electrodes are placed dorsal or dorsolateral to the spinal cord. The 3 waves of such recordings had similar thresholds and refractory periods and they therefore appear to represent a single, synchronous volley that approaches,

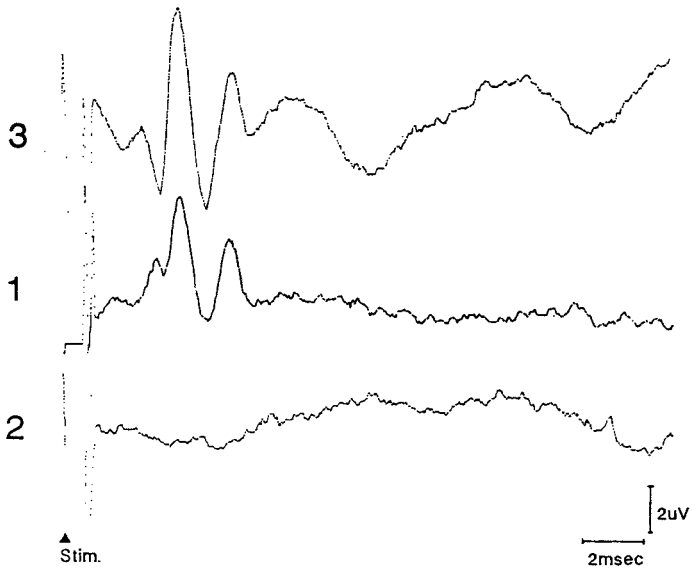


Fig. 3. Corticospinal MEP recorded from various stimulation sites (case 1). Numbers correspond to the stimulation sites shown in Figure 1

arrives at, and moves away from the recording site. As the recording site is moved caudally, the latency of the response increases and the amplitude decreases. These characteristics of the response are further indications that this large component of the response represents a volley mediated by a single fibre tract. The conduction velocity is usually calculated as the measured distance between the two recording sites divided by difference in latency of the negative peaks in the response that were recorded at two different sites. The conduction velocities calculated in earlier studies were within the range 50 to 75 m/s (Tsubokawa 1985; Katayama et al. 1988b).

Two lines of evidence indicate that the initial component of the response is mediated by fibres without any intervening synapses. Thus, it was demonstrated that the response is capable of following double pulse stimulation with interstimulus intervals of less than 2 ms (Tsubokawa and Katayama 1987; Katayama et al. 1988b). Only slight facilitation (5 to 15%) of the response to submaximal stimulation (approximately 50% maximal response) occurred with an antecedent conditioning stimulation when the interstimulus intervals ranged between 2 and 10 ms. The mechanism of this facilitation will be discussed later. Second, the response is clearly resistant to surgical doses of anesthetics.

The width of the response increases gradually as the recording site is moved caudally, indicating temporal dispersion. The decrease in amplitude of the response recorded from a caudal location, however, does not appear to be due only to temporal dispersion. The amplitude at the C7 level is at best approximately 50% of the amplitude at the C2 level. This indicates that a large proportion of the fibres mediating this response leave the fibre tract within the cervical level.

The threshold of the response to monopolar stimulation with anodal currents is generally lower than that with cathodal currents. The initial response can be evoked with stimulus intensities as low as 2 mA, when the stimulation electrodes are appropriately placed on the motor cortex. As the stimulus intensity is raised, the amplitude of the response gradually increases (Katayama et al. 1988b) and the latency decreases until a certain level of stimulus intensity is reached, at which the amplitude and the latency of the responses become constant. The conduction velocity calculated from the latency differences between two recording sites, however, is independent of the stimulus intensity. The mechanism by which the latency decreases will be discussed later.

Monopolar stimulation provides better spatial resolution for identifying the cortical area in which stimulation evokes a response. However, the response to monopolar stimulation during intracranial procedures often varies in amplitude and threshold over time and it sometimes contains artifacts that cannot be eliminated. These variations presumably are the result of changes in tissue impedance that occur with tissue manipulation. Bipolar stimulation is less efficient than monopolar stimulation in producing a response. A response can seldom be obtained with an interelectrode distance of less than 10 mm, but interelectrode distances of more than 10 mm definitely produces a clear initial response. Further increases in the interelectrode distance results in an increase in the amplitude of the response. When both stimulating electrodes are placed on the motor cortex, the amplitude of the response remains approximately the same after the polarity

of stimulation is reversed, and stimulation artifacts can be eliminated by averaging the responses to alternating polarity of the stimulus current. When only one of the stimulation electrodes is placed on the motor cortex, the amplitude is slightly larger when the anodal current rather than the cathode current is applied to the motor cortex.

The response can only be obtained to stimulation of a certain area, which corresponds roughly to the motor cortex as identified from bony landmarks. Furthermore, the response disappears only when surgical damage is caused to sites within this area (Tsubokawa 1985). These findings indicate that a fibre tract mediating this response originates in the motor cortex. No fibre tract other than the CST is known to have such a direct projection from the motor cortex to the spinal cord.

There are many similarities between the initial response recorded from the epidural space of the human spinal cord and the D-wave reported for experimental animals. The conduction velocity of the response, 50 to 77 m/s, is similar to that obtained for the D-wave in the cat and monkey (Patton and Amassian 1954; Philips and Porter 1977; Katayama et al. 1986). Because of the dispersion of the neural activity at lower levels of the spinal cord with subsequent broadening of the response, we estimated the conduction velocity from the onset latency of the response. However, estimating the conduction velocity from the onset latency yields a value that, at the cervical level, is probably not much different from the true value considering unavoidable inaccuracies in roentogenographic measurements of conduction distance. This range of conduction velocity is consistent for fast CST neurons (Takahashi 1965; Humphrey 1983).

CST neurons have been detected by retrograde axonal transport of horseradish peroxidase in other areas of the cortex in the monkey; however, the most dense labeling with horseradish peroxidase occurs in the motor cortex, and fast CST neurons are substantially restricted to the motor cortex (Toyoshima and Sakai 1982). The D-wave recorded from the spinal epidural space may be attributable to the synchronous activation of such dense corticospinal projections originating in the motor cortex. The results of these other investigators and the results of the present study thus directly demonstrate that a corticospinal response equivalent to the D-wave described for experimental animals (Patton and Amassian 1954) can be recorded in man as well.

The D-wave has been considered to result from activation of the cell bodies and/or axons of CST neurons rather than from activation of apical dendrites arborising in the superficial layer (Amassian et al. 1987). The results of studies in man (Katayama et al. 1988b) are in agreement with the results of earlier studies in experimental animals in showing that slight facilitation of the D-wave can occur with antecedent conditioning stimulation in man as well as in animals. The occurrence of facilitation supports the assumption that the D-wave results at least in part from excitation of the cell bodies or initial segments of corticospinal neurons. However, it does appear that the D-wave results largely from direct excitation of cortical or subcortical axons of CST neurons, especially when higher levels of stimulation intensity are used. As mentioned earlier in this article, when the stimulation intensity was raised, the latency of the D-wave decreased (Katayama et al. 1988b). Since the conduction velocity calculated from the difference in

latency of the response, as recorded at two different sites, did not change, the decrease in latency may have been due to spread of the stimulation currents to more distal parts of axons. A similar observation has been made in human subjects undergoing transcranial brain stimulation (Patton and Amassian 1954).

I-waves are sometimes seen following the D-wave, even in patients under general anesthesia. However, the I-waves seem to be easily affected by changes in the depth of anesthesia and the condition of the brain surface (Tsubokawa 1985; Katayama et al. 1988b). Because of this, I-waves appear to be of little value for intraoperative monitoring.

Transcranial Stimulation of the Motor Cortex

It has recently been reported that transcranial brain stimulation can activate the motor cortex through the intact skull and produce myographic responses (Merton and Morton 1980; Marsden et al. 1983; Rossini et al. 1985). Therefore, since 1984 we have been examining whether corticospinal MEP can be recorded with a similar technique of transcranial brain stimulation. Other authors have reported that when transcranial brain stimulation is used a CST response can be recorded from the spinal epidural space in man (Levy et al. 1984; Boyd et al. 1986). The reported physiological characteristics of spinal cord responses elicited with such a technique (Boyd et al. 1986) are in good agreement with our data from direct motor cortex stimulation. However, in our experience, selective activation of CST neurons in experimental animals is difficult with transcranial brain stimulation, presumably because of small brain volume in the animal species used (Katayama et al. 1986).

We have directly compared responses elicited from the spinal cord by electrical transcranial brain stimulation and corticospinal MEP elicited by epidural stimulation of the motor cortex. These responses were recorded in the same patient by leaving an electrode for stimulation of the motor cortex in the intracranial epidural space and recording after the patient is awake from anesthesia. Transcranial brain stimulation is performed with anodal current applied monopolarly to the scalp at the intact part of the skull overlying the motor cortex, employing a technique similar to that described previously (Rossini et al. 1985). Because the patient is not under anesthesia when these recordings are obtained, the corticospinal MEP include both the D- and I-waves. In such recordings, it may be seen clearly that if progressive doses of barbiturate (thiopental, 50 to 200 mg) are administered intravenously, the I-waves as well as the myographic responses are attenuated. In contrast, the D-wave shows only slight depression in amplitude (Katayama et al. 1988b).

The fact that spinal cord responses identical to the D- and I-waves recorded to epidural motor cortex stimulation can indeed be recorded with transcranial brain stimulation (Tsubokawa 1985; Katayama et al. 1988d) provides direct evidence that the human spinal cord responses to transcranial brain stimulation represent impulses occurring in CST neurons. Thus, in contrast to the situation observed in experimental animals (Katayama et al. 1986), it appears that in man transcranial brain stimulation can selectively activate the motor cortex, presumably because

of man's relatively large brain volume. One problem associated with the use of this technique, however, is that much of the applied current passes laterally through the scalp because of the high resistance of the skull. This current spread results in a large stimulus artifact, and it sometimes causes considerable difficulty in obtaining clear recordings of spinal cord potentials at the level of the cervical cord. The latency of the D-wave elicited by transcranial brain stimulation is slightly longer than a D-wave of the same amplitude recorded in response to epidural motor cortex stimulation (Katayama et al. 1988d). This observation suggests that the D-wave recorded in response to epidural motor cortex stimulation may be initiated from a slightly deeper layer of CST axons.

Clinical Applications

Motor Cortex Identification

The D-wave of corticospinal MEP is only seen in response to stimulation of the motor cortex. This fact helps in identifying the motor cortex during intracranial procedures on patients under general anesthesia. The traditional view of how the motor cortex is organized arose from studies in which the cortical surface was stimulated to evoke muscle movements. It has been shown in experimental animals that intracortical microstimulation can permit more discrete mapping of the cortex than can surface stimulation (Asanuma 1975). However, it is not easy to map the motor cortex by looking for evoked muscle movements when the patient is under general anesthesia with the use of muscle relaxants. In contrast, the D-wave of corticospinal MEP is resistant to the usual doses of anesthetics and is unaffected by muscle relaxants (Tsubokawa 1985). This is clearly one advantage of using the D-wave to identify the motor cortex in patients under general anesthesia.

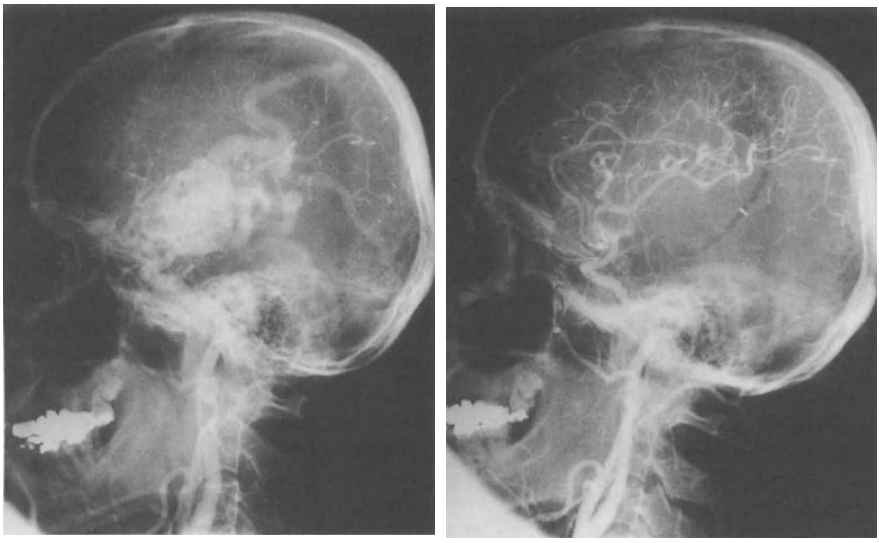
To identify the location of the motor cortex from the appearance of the D-wave, monopolar stimulation with anodal current is apparently preferable to bipolar stimulation. A large number of investigations in experimental animals have indicated that anodal current, more easily than cathodal current, activates deeply located segments or axons of CST neurons from which the D-wave originates (Amassian et al. 1987). However, monopolar stimulation during intracranial procedures gives quite variable results and recordings sometimes have artifacts that cannot be filtered out. This is the consequence of changes in impedance along pathways that vary as tissues are manipulated. In contrast, bipolar stimulation provides constant responses without large artifacts (Tsubokawa 1985; Katayama et al. 1988b). Nevertheless, it is very difficult to obtain clear responses in patients with bipolar stimulation when the interpolar distance is less than 10 mm. A similar observation has been made in experimental animals (Amassian et al. 1987). It has been argued that bipolar stimulation with such a short interpolar distance produces a current that spreads tangentially and thus preferentially activates superficially located neural elements. In contrast, responses obtained with bipolar stimulation with electrode distance of more than 10 mm may be considered to be the sum of responses to monopolar stimulation with anodal and

cathodal currents. Furthermore, the D-wave can only be obtained when an interelectrode distance of 10 mm is used by stimulating areas of the motor cortex that represent the hand, trunk, or thigh. Even with an interelectrode distance of 30 mm the D-wave can only be recorded when at least one electrode is located precisely on or very close to these areas of the cortex (Tsubokawa 1985; Katayama et al. 1988b).

The motor cortex may be displaced in patients who have space-occupying intracranial lesions. Also, representation of specific locations of the motor cortex may be altered in patients who have arteriovenous malformations that involve the motor cortex. Use of intraoperative monitoring of the response to stimulation of the motor cortex during intracranial procedures may therefore help to reduce neurological complications from injuries to the motor cortex. The following cases demonstrate how the technique just described can be used to locate the motor cortex.

Brain Tumor. In case 1, a tumor that was located within the frontal lobe adjacent to the motor cortex was partially visualized at the cortical surface after craniotomy (Fig. 1). The cortical gyri were obviously compressed and displaced by the growth of the tumor, and identification of the central sulcus and motor cortex was impossible by visual inspection. Bipolar stimulation of each gyrus with an interelectrode distance of 20 mm revealed that stimulation of the gyrus labeled 3 (Fig. 1) can elicit corticospinal low-threshold MEP (Fig. 3). The actual location of the motor cortex identified in this way was displaced more than 20 mm from the location estimated from bony landmarks.

Cerebral Arteriovenous Malformation. In case 2, an arteriovenous malformation thought to involve the areas of the motor cortex serving the face (Fig. 4) was



A B

Fig. 4A, B. Carotid angiograms showing arteriovenous malformation adjacent to the motor cortex (case 2) before **(A)** and after **(B)** surgery

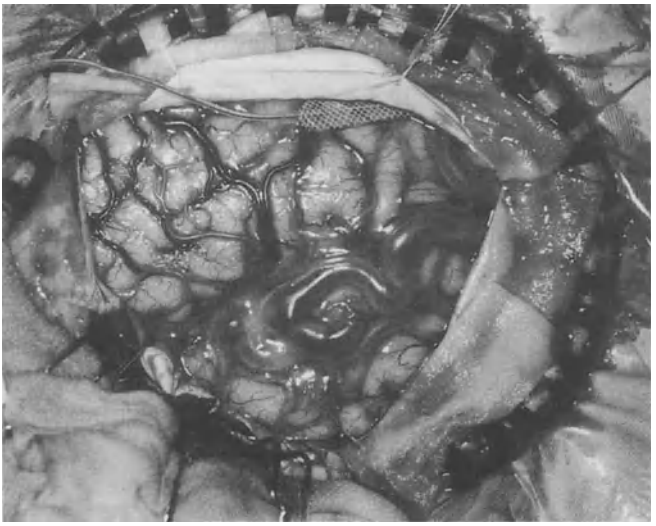


Fig. 5. Surgical field for resection of an arteriovenous malformation; the stimulation electrode array is in place on the cortical surface (case 2)

exposed by craniotomy (Fig. 5). It was found that bipolar stimulation (electrode distance of 10 mm) of the area presumed to serve the hand (Fig. 5), an area that was adjacent dorsally to the nidus of the arteriovenous malformation, elicited corticospinal MEP (Fig. 6). The area was actually located 15 mm posterior to the area identified from bony landmarks as representing the hand.

Intraoperative Monitoring

Recording corticospinal MEP intermittently during intracranial procedures is also of clinical value for determining whether or not damage caused by surgical manipulations extends into the white matter containing CST axons. The use of corticospinal MEP for this purpose requires that the D-wave be recorded to stimulation of multiple sites on the motor cortex. We usually place two electrode arrays over the areas representing the hand and trunk or thigh. When operating in the interhemispheric region, the electrode array is placed on the leg area of the motor cortex. Each array has 4 electrodes, each electrode 5 mm in diameter, placed 5 mm apart. This technique is especially useful for monitoring function during excision of brain tumors or arteriovenous malformations located within the white matter.

Resection of a Brain Tumor. In case 3, a small glioma was located adjacent to the CST (Fig. 7). The patient complained of slight weakness of the right hand before the operation. During the course of subtotal resection of this tumor, corticospinal MEP decreased in amplitude by 50% (Fig. 8). Since intraoperative histological examination of frozen sections revealed that there were a significant number of tumor cells within the tissue posterior to the resected area, the decision was made to resect further posteriorly. This additional small resection caused a sudden decrease in the amplitude of corticospinal MEP to 30% of the baseline

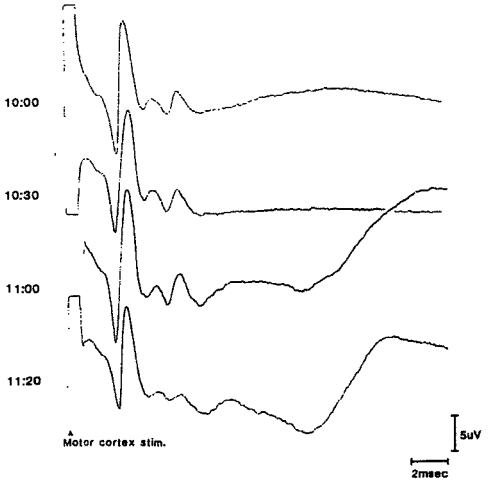
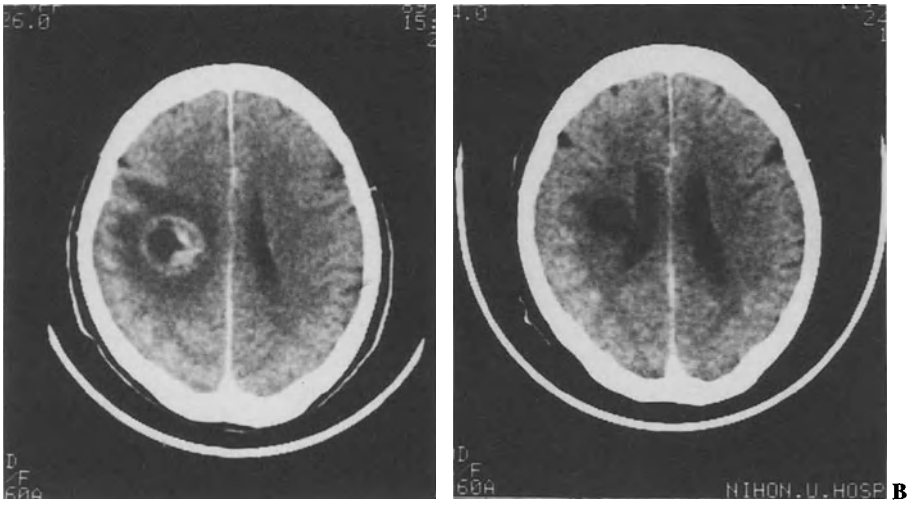


Fig. 6. Corticospinal MEP recorded sequentially during resection of an arteriovenous malformation (case 2). Stimulation sites are shown in Figure 5. The resection was started at 10 : 00 and completed at 11 : 20



A B

Fig. 7A, B. Computerized tomography scans with intravenous infusion of contrast medium show location of brain tumor within the white matter (case 3) before **(A)** and after **(B)** surgery

value (Fig. 8). It seemed likely that a large portion of the subcortical CST was destroyed by this subsequent resection. Corticospinal MEP finally disappeared, even though resection was discontinued (Fig. 8). This patient had hemiplegia postoperatively. A postoperative computer tomography (CT) scan showed that the tumor had been removed totally (Fig. 7B).

The results of intraoperative monitoring of corticospinal MEP and postoperative motor deficits in 26 patients with brain tumors are summarized in Table 1. It

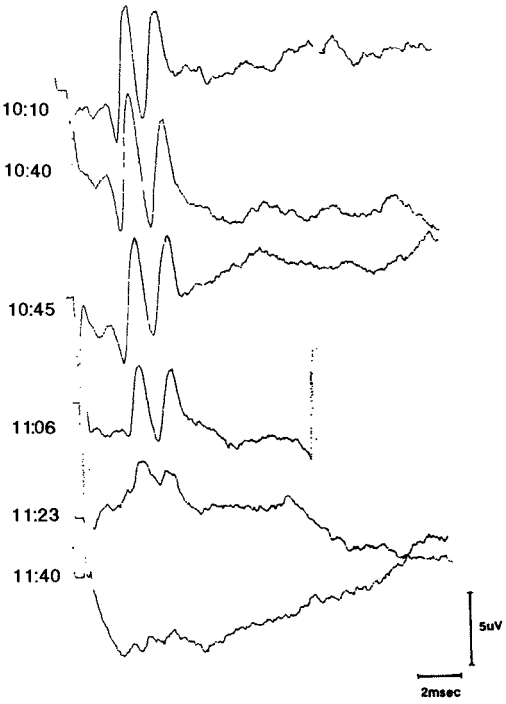


Fig. 8. Corticospinal MEP recorded sequentially during resection of a brain tumor (case 3). The resection was started at 10 : 40 and completed at 11 : 40. The initial resection (10 : 40 to 11 : 00) resulted in a 50% decrease in MEP amplitude; subsequent resection of a small area posterior to the first (11 : 23) resulted in a further decrease in MEP amplitude. Even though resection was discontinued, MEP disappeared altogether (11 : 40).

appears that a 50% decrease in the amplitude of corticospinal MEP during resection of the subcortical CST region is likely to cause hemiparesis, which renders the patient incapable of walking.

Resection of an Arteriovenous Malformation. Intraoperative monitoring of corticospinal MEP in case 2 (Fig. 4) was performed during resection of an arteriovenous malformation (Fig. 6). The total resection was accomplished without producing any motor deficits in the fingers and hand.

Intraoperative identification of the motor cortex and monitoring of CST function with this technique and postoperative motor deficits in patients with arteriovenous malformations is shown in Table 2. The arteriovenous malformations in these patients were all located close to the motor cortex. In a group of patients operated upon with recording of corticospinal MEP there was a significantly lower incidence of motor deficits (Muscle Maneuver Test – MMT – score less than 4) compared to a group of patients operated upon before the availability of intraoperative recording of corticospinal MEP. (The Muscle Maneuver Test is grading from full to no muscle power by scores from 5 to 1.)

Evacuation of Intracerebral Hematoma. The clinical benefit of evacuating the hematoma surgically in patients with hypertensive intracerebral hemorrhage is still a matter of controversy. In patients with putaminal hemorrhage who underwent the evacuation of the hematoma under general anesthesia through a small craniotomy with a transsylvian approach, the amplitude of corticospinal MEP

Table 1. Relationship between motor evoked potentials (MEP) and motor function in 26 cases

Classification	MEP		MMT	
	Before removal	After removal	Before surgery	After surgery (6 mos.)
Benign brain tumor (20 cases)	No response (4 cases)	No response (4 cases)	0 (4 cases)	0 (4 cases)
	Low ampl. Longer latency (6 cases)	No response	1 (1 case) 2 (5 cases)	0 (2 cases)
		Increased ampl. (4 cases)		3 (4 cases)
	Normal response (10 cases)	Decreased ampl. (2 cases)	5 (10 cases)	4 (2 cases)
		Normal response (8 cases)		5 (8 cases)
Malignant brain tumor (23 cases)	No response (6 cases)	No response (6 cases)	0 (6 cases)	0 (6 cases)
	Low ampl. Longer latency (8 cases)	No response (1 case)		0 (1 case)
		Low ampl. (2 cases)	1 (1 case) 2 (5 cases)	2 (2 cases)
		Increased ampl. → normal (5 cases)	3 (2 cases)	5 (5 cases)
Normal	(1 case) response (9 cases)	No response	(1 case) 5 (9 cases)	0
		Normal response (8 cases)		5 (8 cases)

MEP, motor evoked potentials; *MMT*, Muscle Maneuver Test; *ampl.*, amplitude

often increased immediately after the evacuation. The same observation was made when the hematoma was evacuated by CT-guided stereotaxic surgery without general anesthesia.

In case 4 a hypertensive putaminal hemorrhage had caused hemiparesis that was grade 3 by MMT. CT-guided stereotaxic evacuation of the hematoma was performed 6 hours after onset of the hemiparesis. The amplitude of corticospinal MEP increased immediately after the hematoma was evacuated (Fig. 9). When monitoring corticospinal MEP it is important to avoid introducing air subdurally through the burr hole, since the stimulation electrodes are inserted epidurally in cases of stereotaxic surgery. It is often difficult to place through a single burr hole electrodes to stimulate all of the hand, trunk, and thigh areas. Therefore, we usu-

Table 2. Results of surgical resection of cerebral arteriovenous malformations in unmonitored (1984–1986) versus monitored (1987–1988) cases [a]

	1984–1986	1987–1988
Total Cases	No. 37	No. 21
Results		
Excellent	9	9
Good Recovery	22	10
Moderately Disabled	6	1
Severely Disabled	0	0
Dead	0	1

[a]MEP were monitored in patients operated on during 1987 and 1988.

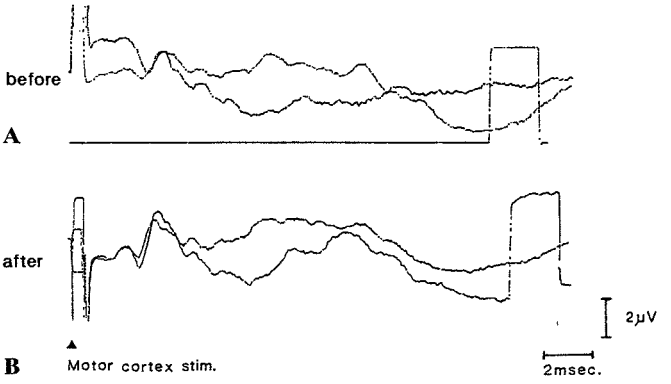


Fig. 9A, B. Corticospinal MEP recorded during CT-guided stereotaxic evacuation of an intracerebral hematoma (case 4)

ally place the electrode array so that the electrodes are in good contact with the trunk area, because our data from direct stimulation of the exposed motor cortex strongly suggest that good correlation between corticospinal MEP and postoperative ability to perform activities of daily living (ADL) is obtained by stimulation of the trunk area in patients with hypertensive putaminal hemorrhage.

Table 3 summarizes the relationship between the results of monitoring corticospinal MEP intraoperatively and the patient's ability to perform ADL 6 months postoperatively. The results in a series of patients who underwent craniotomy under general anesthesia suggest that corticospinal MEP recorded in response to stimulation of the trunk and thigh areas are well correlated with the ability to perform ADL, which is largely dependent upon the ability to walk. This finding was also confirmed in our study of a series of patients treated by stereotaxic surgery.

Resection of Spinal Cord Tumor. Intermittent repeated recordings of the corticospinal MEP are also useful in monitoring the function of CST axons during intraspinal surgery, such as excision of intramedullary spinal cord tumors.

In case 5 an intramedullary tumor extended from C1 to T4 (Fig. 10). The patient complained of paraparesis before surgery. When MEP are recorded dur-

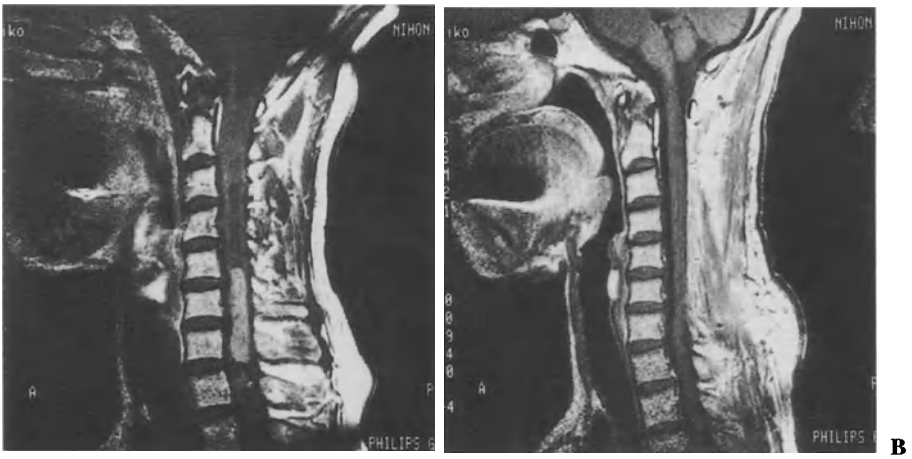
Table 3. Relationships between results of intraoprative MEP monitoring and postoperative ADL in patients with hypertensive putaminal hemorrhage

Case No.	*Stimulation Site* Hand/Face Area	Trunk/Thigh Area	ADL
	Result	*Result*	
1	+	+	II
2	+	+	II
3	+	+	D
4	+	+	II
5	+	+	III
6	+	+	III
7	+	+	III
8	+	+	III
9	+	+	III
10	+	+	III
11	−	−	IV
12	−	−	IV
13	−	−	V
14	−	−	V
15	−	−	V

ADL, Patients' abilities to perform activing of daily living (ADL) 6 months after onset of the hemorrhage; *II*, active life with instrument; *III*, useful life with someone's partial support; *IV*, largely confined to bed; *V*, vegetative state; *D*, dead

ing operations on the spine, some of the recorded potentials are to be expected because of changes in conductance in the tissue lying between the two recording electrodes. This is particularly the case for procedures such as dural incision and cyst puncture. The variations we saw in this case, however, did not prevent appropriate evaluation of CST function. In addition, the latency and the duration of the response were additional indicators of conduction disturbances in the CST. In this case only a slight prolongation of the latency was noted at the time of total resection of the tumor (Fig. 10 B). While the amplitude decreased to 78% of original amplitude during resection, the amplitude after the resection was even larger than what it was before the beginning of resection (Fig. 11). This patient was able to walk again 4 weeks after the surgery.

Embolization of Spinal Cord Arteriovenous Malformation. In case 6 an arteriovenous malformation of the spinal cord (Fig. 12) was treated by intravascular embolization. Since the patient was a child, embolization was performed under general anesthesia. Monitoring of corticospinal MEP was extremely useful in this instance because there was no other way to monitor changes in CST function during embolization. Several minutes after insertion of a catheter into the radicular artery, which fed this arteriovenous malformation (Fig. 12B), the amplitude of corticospinal MEP began to decrease gradually and progressively and the latency became prolonged (Fig. 13). These changes were reversed when the catheter was withdrawn from the radicular artery. Embolization material (Ivaron) was infused a small amount at a time. The catheter was withdrawn quickly from the radicular artery after each infusion and reinserted for the next



A B

Fig. 10A, B. Magnetic resonance imaging scans showing location of intramedullary spinal cord tumor (case 5) before **(A)** and after **(B)** surgery

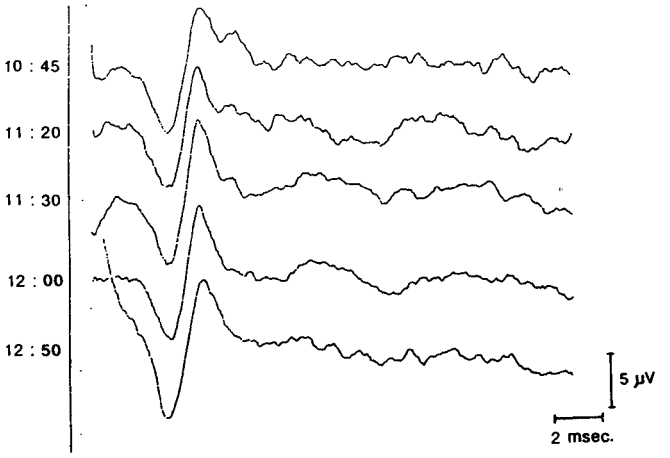


Fig. 11. Corticospinal MEP recorded sequentially during resection of a spinal cord tumor (case 5). The resection was started at 11 : 20 and completed at 12 : 50

infusion after the corticospinal MEP had recovered. When the arteriovenous malformation was completely embolized (Fig. 12C), the corticospinal MEP were not noticeably different from baseline value (Fig. 13). The patient evidenced no motor deficit after the embolization. Although it is possible, especially in children, to record corticospinal MEP without large artifacts using transcranial stimulation of the motor cortex (Levy et al. 1984; Boyd et al. 1987; Katayama et al. 1988d), epidural stimulation with the electrodes inserted through a burr hole was employed in this particular case. Corticospinal MEP recorded with epidural stimulation have been shown to be more clear than MEP recorded in response to transcranial stimulation (Katayama et al. 1988d). Epidural stimulation, although invasive, should be chosen when it is critical to have very clear recordings.

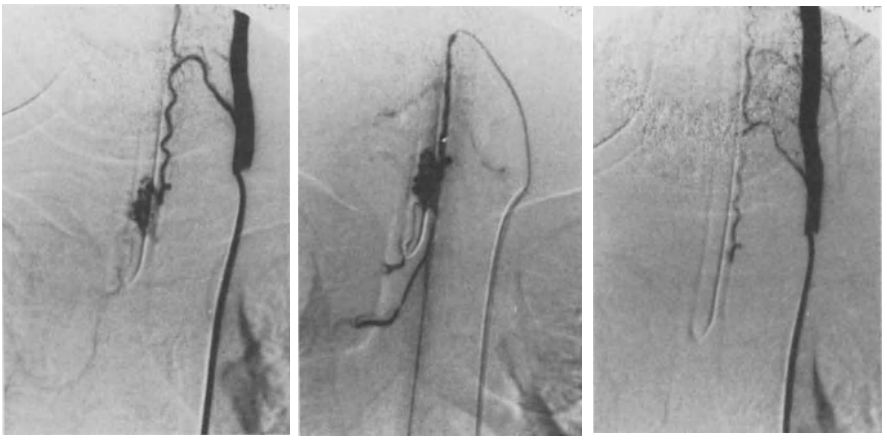


Fig. 12. Digital subtraction angiograms showing spinal cord arteriovenous malformation (case 6) before **(A)**, during **(B)** and after **(C)** embolization

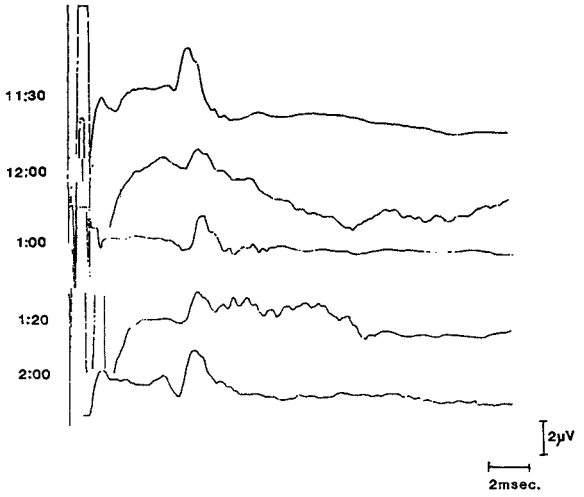


Fig. 13. Corticospinal MEP recorded sequentially during embolization of a spinal cord arteriovenous malformation (case 6). Initial insertion of a catheter into the radicular artery which fed this arteriovenous malformation occurred at 11 : 50. Embolization was completed at 1 : 20. Procedures described in the text

Perioperative Monitoring

Recordings of corticospinal MEP are of clinical value for evaluation of the extent and severity of spinal cord injury (Katayama et al. 1986; Tsubokawa 1987; Tsubokawa et al. 1987a,b, 1988). Corticospinal MEP can be recorded either by stimulating the motor cortex through an electrode screwed into the skull or by transcranial brain stimulation through the intact skull (Levy et al. 1984; Boyd et al. 1986; Katayama et al. 1988d).

A monophasic positive potential is recorded when a nerve impulse approaches but never passes beyond the recording site (Lorente de No. 1947; Deeke and Tator 1973; Cracco and Evans, 1978; Schramm et al. 1983). This type of potential, called the killed-end evoked potential (KP), has been seen in spinospinal responses recorded in patients with spinal cord injury (Tamaki and Yamane 1975; Kurokawa 1978; Katayama et al. 1988a). The KP of corticospinal MEP can also be recorded preoperatively from electrodes inserted percutaneously into the spinal epidural space and is of value for localizing lesions. Initially, the electrode is inserted caudal to the lesion and advanced rostral to the lesion. After normal corticospinal MEP have been recorded rostral to the lesion, the electrode is withdrawn in stepwise fashion, with MEP being recorded at each step. At the level of the lesion, both the D-waves and I-waves suddenly change to a monophasic positive response (Katayama et al. 1989).

The corticospinal MEP in experimental animals was originally described as a large KP (Patton and Amassian 1954). Where axons are injured they offer less resistance to current flow, and this results in a negative voltage gradient as the recording site is moved more distally. This pattern of potentials is the classic "demarcation potential" (Borgens 1988). The larger amplitude of the KP as compared to the normal evoked potential has been explained by this demarcation potential (Schramm et al. 1983). Thus, changes in the amplitude of the KP may reflect changes in transmembrane resistance and other changes in current due to the injury implicated in secondary processes of axonal degeneration, mainly through the influx of calcium ions. Furthermore, axonal degeneration has been shown experimentally to be inhibited by application of a "bucking" voltage, a procedure that has been proposed for clinical use (Borgens 1988). If the time courses of changes in KP in patients with spinal cord injury could be characterized, this information could lead to therapy to minimize the secondary processes of axonal degeneration.

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Monitoring of Spine Surgery with Evoked Potentials

J. R. DAUBE[1]

Summary

Several patterns of changes in SSEP and their correlation to postoperative neurologic function have been observed during surgery on the spine. Most of these changes are gradual in evolution, occurring in relation to distraction or other specific operative manipulations, and gradually resolve without neurologic sequelae. As these changes may develop gradually, it is thus important to continue monitoring until the patient is awake. These gradual changes in SSEP may also be caused by ischemia of the spinal cord or of peripheral nerves, as is observed during surgery on the thoracic or abdominal aorta. Less frequently, SSEP change abruptly, usually due to vascular injury, contusion of the spinal cord, or compression of the spinal cord by an epidural or subdural hematoma. When abrupt loss of SSEP occur, an attempt should be made to localize the site of injury using either epidural or direct spinal electrodes and to carefully inspect that site for the presence of hematomas or for other potentially reversible causes of spinal cord injury. Unfortunately, when the loss of SSEP is abrupt and non-reversible, postoperative paraplegia is likely to occur. When improvement in the SSEP occurs intraoperatively it is usually associated with improved neurological status postoperatively. However, a postoperative motor deficit can occur without an associated change in intraoperative SSEP. This can be caused by an anterior spinal artery syndrome or by direct injury to the ventral spinal cord, anterior horn cells, or nerve roots. Methods to monitor central and peripheral motor pathways are currently being developed. At present, the use of motor evoked potentials during surgery remains to be fully defined.

A small proportion, usually less than 0.5%, of patients undergoing corrective scoliosis surgery and other surgical procedures on the spine develop a persistent neurological deficit immediately postoperatively. Careful surgical techniques and stabilization of the spine during surgery have helped to keep the complication rate low. However, the procedure is nonetheless devastating for those individuals who awaken paraplegic from anesthesia for surgery on the spine. Thus, to further reduce the possibility of neurologic deficit the "wake up test" was devised (Hall et al. 1978). The patient is awakened during the operation to correct a spinal

[1] Mayo Clinic, 200 First Street S.W., Rochester, MN 55905/USA.

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deformity and the continuity of spinal cord pathways is tested by having the patient voluntarily move his or her feet. While the "wake up" test is some help, it is difficult to perform and presents problems because it requires changes in anesthesia level; also, the test is not useful for patients undergoing surgical procedures, such as resection of spine tumors, in which there is no well-defined time of major hazard.

Somatosensory Evoked Potentials

Monitoring of somatosensory potentials (SSEP) has therefore been used by a number of workers as an additional method to monitor spinal cord function intraoperatively (Nash et al. 1977; Engler et al. 1978; Allen et al. 1981; Grundy et al. 1982; Macon and Poletti 1982). Some investigators record SSEP within the operative field, some record SSEP outside the operative field, and some record motor evoked potentials (MEP). Electromyographic (EMG) potentials may be monitored along with SSEP. The particular combination of SSEP and EMG monitoring performed must be customized for each patient on the basis of preoperative clinical deficits and neural structures that may be traumatized during the operation.

Technical Factors

A number of technical factors must be considered when recording from the spine for intraoperative monitoring (Lesser et al. 1985). The rate of stimulation cannot be as fast as when recording from an awake patient, because anesthetic agents make the sensory system more susceptible to fatigue. While SSEP can be recorded at 5 or even 10 Hz in most awake patients, in patients under anesthesia the scalp SSEP will fatigue at rates over 3 Hz and some patients will require stimulation at rates as low as 0.5 or 1.0 Hz, especially when at deeper levels of anesthesia.

The number of responses that must be averaged varies with background noise level. Since most patients are paralyzed, muscle activity is minimal. When there are few sources of noise, clear responses can be obtained by averaging the responses to only 64 or 128 stimuli. However, most intraoperative recording situations have other sources of artifact than muscle potentials, so 286 or sometimes even 500 responses must be averaged to obtain reproducible traces.

Technical problems occur often in the operating room. Sixty-Hertz artifacts may arise from gas warmer/humidifiers, blood warmers, and some electric drills, which should be moved away from the operative field or even removed from the room. Wire movements of recording leads can also create noise and should therefore be eliminated. Recording leads can be disconnected, cut, dislodged, or damaged during the course of the operation, especially leads that run near or on the head or neck, and with movement of the torso. The recording leads must be firmly fixed at multiple points, and shielding of recording leads is sometimes necessary. Stimulating electrodes can also be displaced, requiring some type of

peripheral monitor to assure their function. Using constant-current, stimulus isolation units can decrease the risk of burns caused by electrical stimulation. Alterations in both the level of anesthesia and blood pressure will change the latency and amplitude of responses. Rarely, patients will have an enhanced scalp response after induction of anesthesia, but most scalp responses are reduced, and in a small number of cases the scalp response can be lost immediately after anesthesia induction. In a group of 140 patients undergoing thoracolumbar spine surgery at the Mayo Clinic, the mean scalp response amplitude decreased by 37% with induction of anesthesia, and 5% of subjects entirely lost the response, even some of those without underlying neurologic disease. Similarly there is a gradual reduction in amplitude and increase in latency with continued anesthesia (mean of 17%, with another 5% losing their responses). Latency was less variable, with only a 4 to 6% average increase during the course of anesthesia. Changes in the upper limb SSEP as a result of anesthesia are much less prominent. Alterations in blood pressure can also reduce the amplitude of the evoked response, especially when the mean blood pressures are under 70 mm/Hg.

Recording outside the operating field is by far the simplest technique for intraoperative monitoring and can be performed without the direct assistance of the surgeon, leaving the surgeon free for the surgical procedure. Stimulation is applied to a peripheral nerve, usually either the peroneal or tibial nerve in the leg, or the median or ulnar nerve in the arm. Recordings are most often made with electrodes placed on the scalp at CZ-FZ for leg stimulation and C3'(C4')-FZ for arm stimulation. Other sites for reference electrodes, such as the ears, are also used. Recordings can also be made from the spinal cord and peripheral nerves. Most of the early studies of intraoperative monitoring used peripheral stimulation to obtain scalp recordings, which generally gives a well-defined, although unstable response that may change with changes in blood pressure and anesthesia level.

In order to use SSEP during thoracic or lumbar surgery, supramaximal surface stimulation must be applied to each tibial nerve at the ankle and/or each peroneal nerve at the knee sequentially and continuously. Simultaneous bilateral stimulation of either peroneal or tibial nerves is used if the responses to unilateral stimulation are too low in amplitude to be recorded reliably. Stimulation is begun using a stimulus rate of 3 Hz and the rate is reduced if the amplitude of the response increases at lower stimulus rates.

Spinal cord function is best monitored by recording at 3 levels: 1) Peripheral electrodes record either over the cauda equina, near the sciatic nerve, or from peripheral muscles. Responses from these peripheral locations are used to monitor the adequacy of stimulation. 2) A needle electrode in the cervical paraspinal muscles at C7, referenced to the shoulder or FZ on the scalp, records the cervical spinal cord potential. Both the leg and cervical recording electrodes must be taped firmly in place using multiple wire loops to prevent dislodging. An electrode in the esophagus referenced to FZ can sometimes be used if the posterior cervical site is unavailable. 3) For scalp recording from CZ to FZ, surface electrodes should be applied with collodion so that they will remain firmly in place for many hours despite manipulation of the head and neck during anesthesia. Recordings are made sequentially before and during the operation.

To obtain SSEP during operations on the neck, stimulation is applied to the ulnar nerves on each side and responses are recorded from an esophageal electrode at the cervical spine (level $C_3 - C_7$) referred to FZ, and from C3' (or C4') referred to FZ locations on the scalp. Stimulation is also applied serially to the tibial nerves at the ankles while recording from the esophageal and scalp electrodes. Recordings of the responses from foot muscles or the sciatic nerve can be used for monitoring to ascertain whether adequate stimulation has been obtained when the tibial nerve is stimulated and recordings from ulnar hand muscles or the ulnar nerve serve the same purpose for stimulation at the ankles. Stimulation and recording parameters are similar to those for monitoring during lower spine surgery.

Patients undergoing operations on the spine are best selected for monitoring by surgeons on the basis of risk of neural damage. Patients of any age can be monitored, and usually the group of those monitored includes many patients under age 15. The largest group of patients whose SSEP are monitored are teenagers undergoing corrective surgery for scoliosis. Another large group is comprised of elderly individuals undergoing operation for cervical spondylosis. Patients undergoing operation for management of a bony spine tumor, thoracic aneurysm, traumatic spine damage, or spondylitis can also be monitored. Two-thirds of the surgical procedures are at the thoracic level.

A frequent problem during intraoperative monitoring is the variability of the evoked response from one recording run to the next because of artifacts, changes in blood pressure or anesthesia level, and other factors (Fig. 1). A significant change in SSEP therefore has to be identified as a consistent alteration in latency (2.0 ms more than baseline changes) and amplitude (50% less than baseline values) at both the neck and scalp sites with an intact peripheral response. In a few patients recordings are only obtainable at one of the two cephalad sites, usually the neck. In those cases only changes that are seen with stimulation of more than one nerve can be considered significant. No absolute change in amplitude can be considered evidence of spinal cord damage, because in some cases in which no damage occurred a scalp response appeared to be lost transiently although other responses were intact. In other cases in which damage did occur, there were smaller, but consistent alterations of the response to stimulation at 2 sites showing evidence of compression before the major changes occurred.

Applications

Those performing SSEP monitoring during operations on the spine are sometimes frustrated by their inability to record reliable potentials over the scalp, both in patients with and in those without preoperative neurological deficit. Over a 3-year period, intraoperative monitoring was requested for 379 patients undergoing operations on the spine at the Mayo Clinic. Twenty-eight of these patients had neurological deficits and could not be monitored because responses could not be recorded over the spine or scalp. SSEP monitoring, with multichannel recording during individual stimulation of tibial and peroneal nerves, was performed on 351 patients undergoing operations on the spine. In 24 patients with intact SSEP

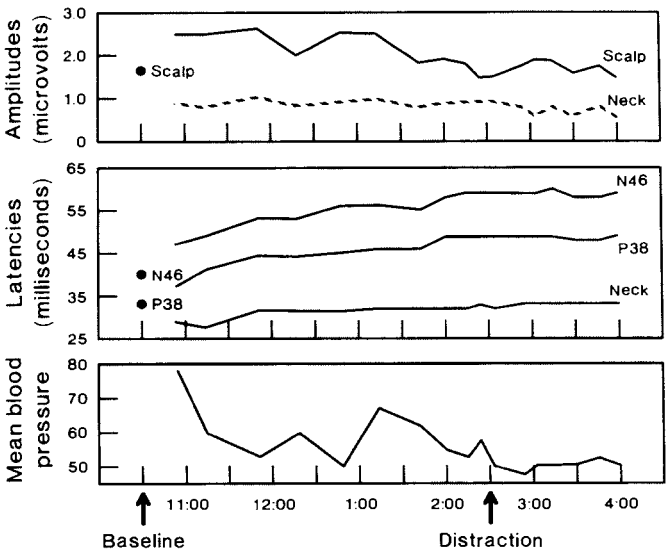

Fig. 1. Plot of the amplitudes and latencies of neck and scalp SSEP during thoracic spine surgery for scoliosis. There was a gradual reduction in amplitude and increase in latency of scalp SSEP before spine distraction. Note the increase in N_{20} amplitude immediately after induction of anesthesia

before anesthesia, the scalp potential either could not be recorded immediately after anesthesia or was lost within an hour of anesthesia; but well-defined neck potentials could still be recorded. In 17 patients neck potentials could not be recorded for technical reasons but scalp potentials were still obtained. In 3 patients scalp and neck potentials were lost in association with loss of sciatic potentials due to a failure in peripheral stimulation. The addition of a cervical spine and peripheral recording location to the usual scalp recording location resulted in reliable monitoring being performed on more patients.

SSEP monitoring during operations on the spine has proven of value in warning surgeons of potential damage to the spinal cord. Since such damage occurs infrequently, few cases are available to help us identify what changes in SSEP might be associated with spinal cord damage. Of the 351 patients who underwent monitoring of spinal cord sensory pathways during an operation on their spine in a 3-year period, we identified significant changes in SSEP intraoperatively in 6. In 3 cases the changes reversed with surgical intervention (removal of hematoma, removal of rods, and removal of spine wires) with no subsequent clinical deficit (Figs. 2, 3).

One patient developed a permanent postoperative deficit despite removal of rods. There was no deficit in 1 patient in whom the tibial SSEP decreased after spinal cord biopsy, but an L5 radiculopathy persisted in another patient in whom the amplitude of a peroneal SSEP fell without apparent cause; both of these patients recovered within 15 minutes during surgery. Two other patients had postoperative deficits due to anterior spinal artery syndrome not manifest by

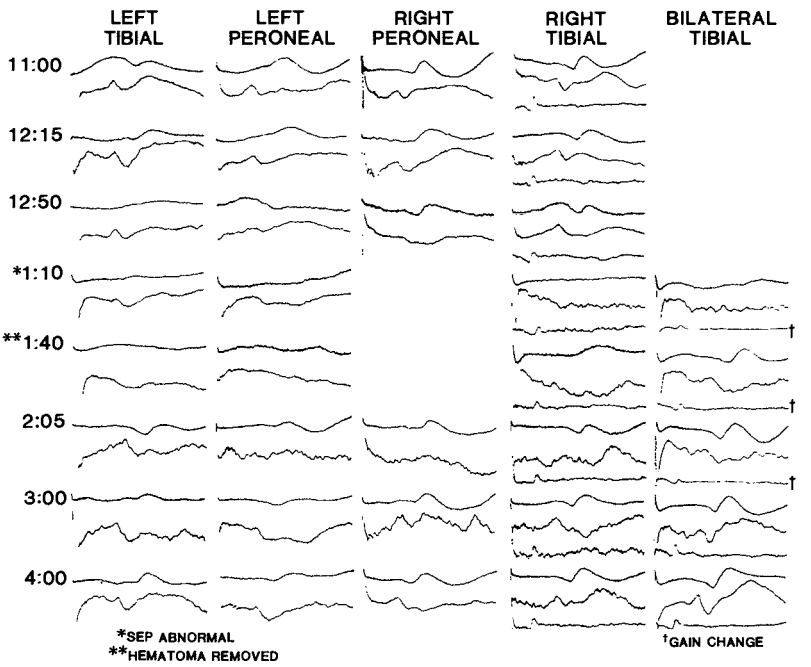

Fig. 2. SSEP recorded during thoracolumbar spine surgery for severe spondylitis. Gradual loss of SSEP was followed by return of potentials shortly after laminectomy and removal of an intraspinal hematoma. No postoperative neurologic deficit occurred

abnormal SSEP. Eight patients, only one of whom had the SSEP change noted above, had evidence of new lumbar radiculopathy after surgery. SSEP changes were similar in most patients. The amplitude reduction began 10 to 30 minutes after spinal cord injury. The amplitude of responses from the neck and scalp decreased gradually over 10 to 15 minutes, as the latency increased by up to 3 ms. In each of the 3 cases in which surgical action was taken to correct the situation that led to the SSEP loss, the SSEP recovered to baseline values within 5 to 10 minutes. More than 1000 patients would need to be tested to show a statistically significant difference in complication rate for those whose SSEP did not recover; nevertheless, the effectiveness of intervening when SSEP were lost in these 3 of 300 patients monitored shows that SSEP monitoring is of value in preserving function. In 1 patient the SSEP was lost abruptly with direct trauma to the spinal cord and no recovery of function occurred (Fig. 4). Abrupt loss of the SSEP has also been observed in patients undergoing vascular or stereotactic surgery of the brainstem. A few patients show improvement in SSEP during surgery (Fig. 5).

A number of methods have been developed to record closer to neural tissue in the operating field. These methods include recording from the subarachnoid or epidural space, the spinous process, or intraspinous ligaments (Sharbrough et al. 1973). Lueders et al. (1982) used needle electrodes placed between the spinous processes, but Jones et al. (1985) used epidural electrodes inserted between the

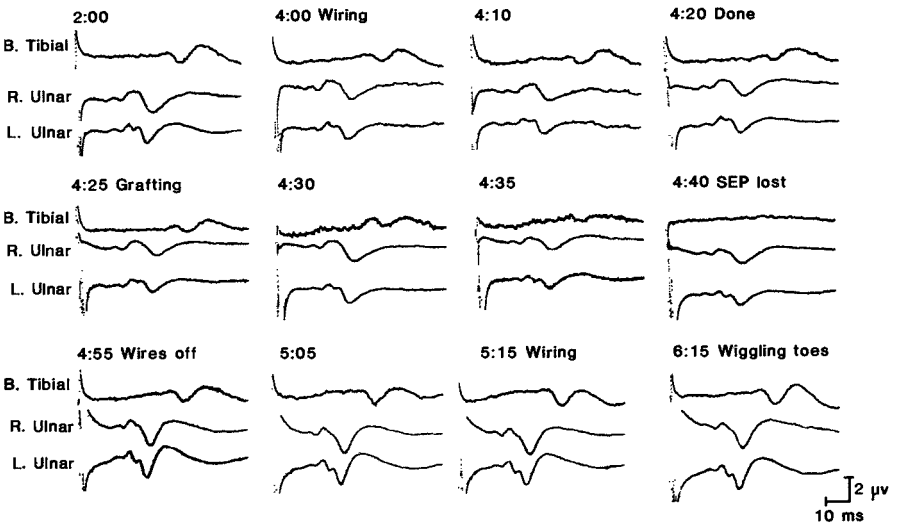

Fig. 3. Record of SSEP monitoring during cervical spine surgery for an unstable C6 fracture shows a gradual loss of SSEP (tibial more than ulnar), and rapid return with removal of wires. There was no change in anesthesia level during this period. No postoperative neurologic deficit occurred

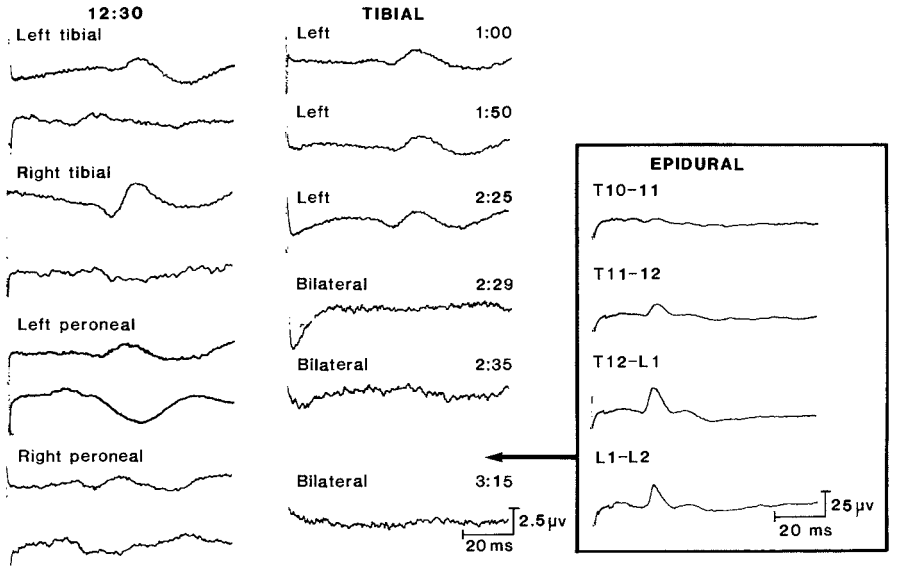

Fig. 4. Record of SSEP monitoring during rod placement and fusion after resection of a spine tumor shows an abrupt loss of SSEP with no return. Direct recordings from the dura demonstrated a localized area of SSEP loss. The patient was paraplegic postoperatively

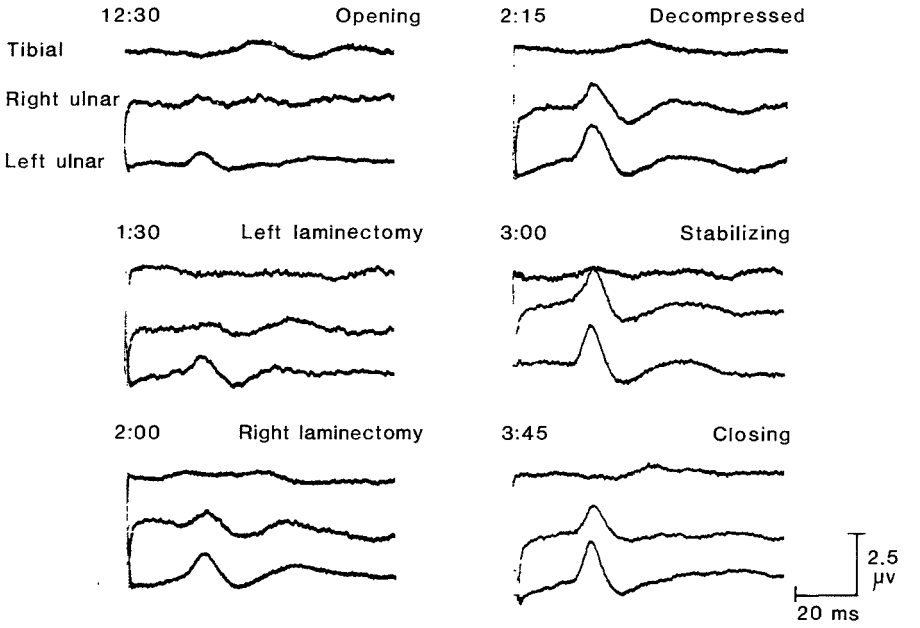

Fig. 5. Gradual improvement of SSEP occurred during upper cervical cord decompression for rheumatoid arthritis. Clinical and SSEP improvement continued postoperatively

spine and the dural sac to obtain large, readily recorded potentials. Similar recordings have been obtained of neural activity in descending spinal cord fibers by direct stimulation of and recording from the spinal cord (Tamaka et al. 1981).

While recording from the operative field can result in much larger responses, it has associated problems, including technical problems related to the surgical procedure, added risk of operative infection, subjectivity to mechanical artifact, and limitation to those surgical procedures in which the spine is opened to expose the dura. Obtaining satisfactory recordings from the operative field also generally requires more technical expertise and requires that the surgeon be familiar with and cooperative with the monitoring procedure. Recordings in the surgical field are most useful in operations on the spine, such as operations to treat tumors or arteriovenous malformations, in which the directly recorded potential can be used to localize the area of damage or record responses too small to record by other methods. Teflon-coated silver wire electrodes with a small cotton wick sutured onto 2 mm of exposed tip work well for this type of recording.

In summary, several patterns of change in SSEP have been observed during operations on the spine, and these patterns have been correlated with postoperative neurologic function. Most changes are gradual in evolution, occur in relation to distraction or other specific operative manipulation, and gradually resolve without neurologic sequelae. The change may develop gradually, emphasizing the importance of continuation of monitoring until the patient is awake. Gradual changes in SSEP may also be caused by ischemia of the spinal cord or peripheral

nerves, as has been noted during operations on the thoracic or abdominal aorta. Less frequently, the SSEP change abruptly, usually in relation to vascular injury, contusion of the cord, or compression of the cord by an epidural or subdural hematoma. When abrupt loss of SSEP occurs, an attempt should be made first to localize the site of injury by examining recordings made using epidural or direct spinal electrodes; this should be followed by careful inspection of that level for hematomas or other potentially reversible causes of cord injury. Unfortunately, when the loss of the SSEP is abrupt and nonreversible, postoperative paraplegia is likely to occur. When improvement in the SEP occurs intraoperatively it is usually associated with improved neurological status postoperatively. Finally, the occurrence of a postoperative motor deficit without an associated change in SEP has been well documented (Lesser et al. 1986). This can be caused by anterior spinal artery syndrome or direct injury to the ventral spinal cord, anterior horn cells, or nerve roots. Methods for monitoring central and peripheral motor pathways are currently under development (Levy et al. 1984; Boyd et al. 1986).

Motor Evoked Potentials

General

Two ways to stimulate the cerebral cortex directly through the intact skull have been developed over the past few years (Boyd et al. 1986; Edmonds et al. 1989; Halonen et al. 1988; Kitagawa et al. 1989; Shields et al. 1988; Zentner, 1989; Zentner et al. 1989). One of these methods uses high-voltage electrical impulses and the other uses high-energy magnetic stimulation. Both are being used clinically in Europe, but not yet in the United States. A small number of European centers have begun to use these methods for surgical monitoring. These methods are currently considered experimental in the U.S.A., and can only be used with patients under an investigational device exemption from the U.S. Food and Drug Administration (FDA).

Direct transcranial electrical stimulation of the motor cortex can be accomplished by placing an anode over the motor area to be stimulated, a cathode 5 cm anteriorly, and applying a 0.1-ms or shorter pulse with a voltage of 600 to 1000 volts. Brief twitches of muscles in the upper (and lower) extremities can be elicited. The responses are enhanced by minimal voluntary muscle contraction. Electrical stimulation with a lower voltage pulse can be performed by applying a series of 8 electrodes around the circumference of the head and linking these 8 electrodes to serve as the cathode with the anode over the motor cortex. Recording with this method is moderately uncomfortable for the awake patient, but can be accomplished without anesthesia. No complications have been known to occur, although there is, theoretically, the possibility of local tissue damage.

Extracranial magnetic stimulation can also activate the motor cortex through the skull. When 0.1-ms pulses at 2 to 3 tesla field strength are applied the motor cortex can be activated in a fashion similar to that described for electrical stimulation. Using magnetic stimuli to record motor evoked potentials (MEP) transcranially is less painful than using electrical stimuli.

Anesthesia reduces the amplitude of MEP, whether electrical or magnetic stimulation is used, so that MEP may be difficult to record in the legs of anesthetized patients. Attempts to record MEP directly from the spinal cord have been hampered by the slow rates of stimulation used, which preclude averaging. The value of this technique and ways to apply it intraoperatively have yet to be defined.

Mayo Testing

Over the past year, staff at the Mayo Clinic have studied the usefulness of recording magnetic MEP intraoperatively under an FDA-approved investigational device exemption. Healthy patients between 18 and 59 years who were undergoing corrective scoliosis surgery and who gave informed consent were tested with a defined protocol. Each patient's preoperative and postoperative evaluation included a neurologic examination; psychometric testing; determination of serum prolactin, TSH, and cortisol levels; electroencephalography (EEG); magnetic resonance imaging (MRI); and SSEP recording. Preoperative magnetic stimulation with a Novametrix stimulator and 7-inch coil was used to determine the optimal location and parameters for stimulation with the patient completely at rest and under EMG monitoring.

Latencies and amplitudes of maximal MEP recorded from proximal and distal muscles of both lower extremities were measured. All patients were tested with 100% output of the stimulator.

Anesthesia was induced with thiopental and maintained with a balanced mixture of isoflurane and nitrous oxide. Oxymorphone was given as needed. Recordings were also made from an electrode placed in the epidural space over the lumbar cord under direct visualization during the operation. Tibial SSEP were recorded simultaneously from the cervical spine and scalp. A maximum of 300 stimuli were applied to any one patient.

In the 7 patients enrolled in the study thus far, SSEP were normal and remained unchanged throughout the procedures. Reliable MEP were obtained in all patients preoperatively. MEP were markedly attenuated during anesthesia in all patients, precluding use of MEP to monitor motor pathways. MEP could not be recorded from the spinal cord. At follow-up, results of all clinical and laboratory studies were normal and unchanged from preoperative values. There was no evidence of untoward effects of the magnetic stimulation. Additional studies are under way to define an anesthesia regimen that will permit continued recording of MEP.

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Monitoring in Aneurysm Operations

Somatosensory Evoked Potential and Brainstem Auditory Evoked Potential Monitoring in Cerebral Aneurysm Surgery under Hypotension

A. Ducati[1] and E. Fava[2]

Summary

We report our experiences with somatosensory evoked potential (SSEP) monitoring during cerebral aneurysm operations under hypotension. In the first part of this study the surgical technique was intentionally not changed as a result of SSEP monitoring; in this way, we established what changes in intraoperative SSEP were consistent with preservation of function during surgical manipulations. We recorded both short-latency SSEP to computer central conduction time (CCT) and long-latency cortical SSEP, with a timebase of 200 ms, on admission of the patients to the hospital and intraoperatively. We found that 54% (7 of 13) patients with preoperative signs of arterial spasm, i.e., increased rate of blood flow in the middle cerebral artery, had ipsilateral prolongation of CCT and depression of the cortical late waves. During the operation a CCT exceeding 9 ms for at least 10 minutes and an irreversible loss of the cortical late waves were the only parameters predictive of postoperative deterioration in function. In addition, the patients whose neurological status deteriorated postoperatively had preoperative CCT equal to or longer than 7.5 ms and depression of the cortical late waves over the affected hemisphere. These patients are not good candidates for operations under hypotension. On the basis of these results, patients in the second part of this study who were not candidates for operation under hypotension were operated upon with the technique of temporary clipping under normotension, with evoked potential monitoring. No patients operated upon according to this protocol showed postoperative deterioration in function. Brainstem auditory evoked potentials (BAEP) were recorded, in addition to SSEP, when aneurysms of the vertebral artery were operated upon. Prolongation of the wave I-V interval was caused by brainstem retraction, but even more severe BAEP changes developed when hypotension was induced. These changes soon reversed when blood pressure was raised after the aneurysm had been clipped.

In conclusion, we found that for monitoring during cerebral aneurysm operations the late waves of cortical SSEP provide useful information to supplement that provided by CCT measurements. Moreover, SSEP were valuable in helping to identify those patients who were not suitable for surgery under hypotension because of the risk of brain ischemia, and in planning a safer technique for surgical management of the lesions in these patients.

[1] Chair of Neurosurgery – University of Ancona and Ospedale S. Raffaele – Milano
[2] Institute of Neurosurgery, University of Milano, Pad. Beretta Ovest, Ospedale Maggiore di Milano, via Francesco Sforza 35, I-20122 Milano, Italy

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Introduction

It has been suggested that inducing moderate to severe hypotension during operations to manage cerebral aneurysms will decrease pressure in the aneurysm and thus decrease the risks of rupture during surgery. Moderate hypotension is referred to as about 90 to 100 torr systolic blood pressure (SBP), usually corresponding to 70 to 75 torr mean blood pressure (MBP), while severe hypotension refers to a SBP of approximately 60 torr, corresponding to a MBP of about 40 torr. Such levels of deliberate hypotension have been chosen because, in the normal brain, autoregulation causes the cerebral blood flow to be maintained at a constant level down to a MBP of approximately 40 to 45 torr. However, the brain affected by subarachnoid hemorrhage is not a normal brain. In patients with subarachnoid hemorrhage, autoregulation is likely to be lost in some regions; moreover, in aged patients and in patients with atherosclerotic disease prolonged and/or deep hypotension may be dangerous for the brain as well as for other vital organs, i.e., the heart and kidneys. In fact, deliberate hypotension has been held responsible for several cases of postoperative clinical deterioration (Lassen and Tween 1975).

When reporting on induced hypotension, authors of surgical papers frequently make statements such as "...most patients tolerate this type of brief hypotension without postoperative sequelae..." (Jafar et al. 1988, p. 919). We wondered what "most" and "brief" mean in this context. Thus, we undertook to identify as correctly as possible the risk to the individual patient of decreasing blood pressure during operations to manage a cerebral aneurysm; specifically, we wished to find out how much the MBP could be reduced and how long a specific level of hypotension could be maintained without postoperative neurological deterioration.

This study was started 4 years ago, and it was conducted in 2 stages. First, a group of 50 unselected patients underwent intraoperative recording of somatosensory evoked potentials (SSEP) without any changes in any therapeutic protocol that previous experience with aneurysm management had suggested was correct and ethical. The reason that we did not want to change the management of these patients was that we did not know the clinical meaning of the SSEP changes such as those that were frequently observed intraoperatively; thus, we did not know the limits of tolerance to variation in SSEP consistent with normal postoperative neurological function. Moreover, the limits suggested by the literature were in some ways contradictory: for instance, Symon and Wang (1986) reported that a somatosensory central conduction time (CCT) of up to 10 ms may be associated with clinically normal results of surgery, whereas Kidooka et al. (1987) reported on a patient whose CCT never exceeded 8.1 ms but who died as a consequence of surgery. We think that this difference can be explained by differences in the anesthesiolological techniques of the 2 groups of investigators mostly as regards the kind of anesthesia gas chosen, whenever gas was used, and its concentration. The results of the first part of our study provided a definition of limits of tolerance to SSEP changes for our techniques of anesthesiological management (Ducati et al. 1988).

In the light of these preliminary results, we recorded SSEP from a second group

of 25 patients undergoing aneurysm surgery. We think we are now able to demonstrate that neurophysiological monitoring can indeed have a great impact on the management of clinical problems and even on the operative strategy for approaching cerebral aneurysms.

Patients and Methods

We recorded SSEP from 75 of a total of 265 patients who underwent operations for cerebral aneurysms over a 4-year period at the Institute of Neurosurgery of the University of Milano. Patients who underwent recording were not chosen consecutively: they were selected solely on the basis of availability of monitoring equipment and of trained personnel for monitoring at the moment of surgery. In most cases we operate under emergency conditions. This does not mean that we always perform early surgery, however: often patients are referred to us by other hospitals at different times after bleeding begins. According to the unmodified scale of Hunt and Hess (1968), aneurysms in 37 of our 75 patients were grade I or grade II, in 26 patients they were grade III, and in 12 patients they were grade IV before surgery.

All patients underwent transcranial Doppler sonography preoperatively, to determine the arterial blood flow, and SSEP were recorded to median nerve stimulation at the wrist. The side corresponding to the affected hemisphere or to the hemisphere from which the surgical approach would be made was tested most frequently, but at regular intervals also the contralateral hemisphere was tested. Both short-latency SSEP (slSSEP: 4 Hz stimulation frequency, 30 ms time basis, 10 to 2500 Hz filter bandwidth) and long-latency cortical responses (cSSEP: 1 Hz stimulation frequency, 200 ms time basis, 1 to 100 Hz filter bandwidth) were recorded with a midfrontal reference. On cSSEP the N_{20}/P_{25} complex was followed by "slow" waves of longer latency, also called late waves, whose pattern and amplitude have been found to be symmetrical over both hemispheres in most normal subjects. Active analog filters were used (12 dB/octave rolloff). Subdermal platinum needle electrodes were placed over the parietal regions (C3'-C4') and over the spinous process of the second cervical vertebra. The cervical N_{13} and the cortical N20 waveforms were analyzed by measuring their latencies and amplitudes. The CCT was computed as the latency difference of N_{20} and N_{13}, according to Hume and Cant (1978). The CCT value in a population of control subjects matched for age and sex was 5.7 ms (0.4 ms SD). Maximal normal CCT was 6.7 ms (mean + 2.5 SD).

Results

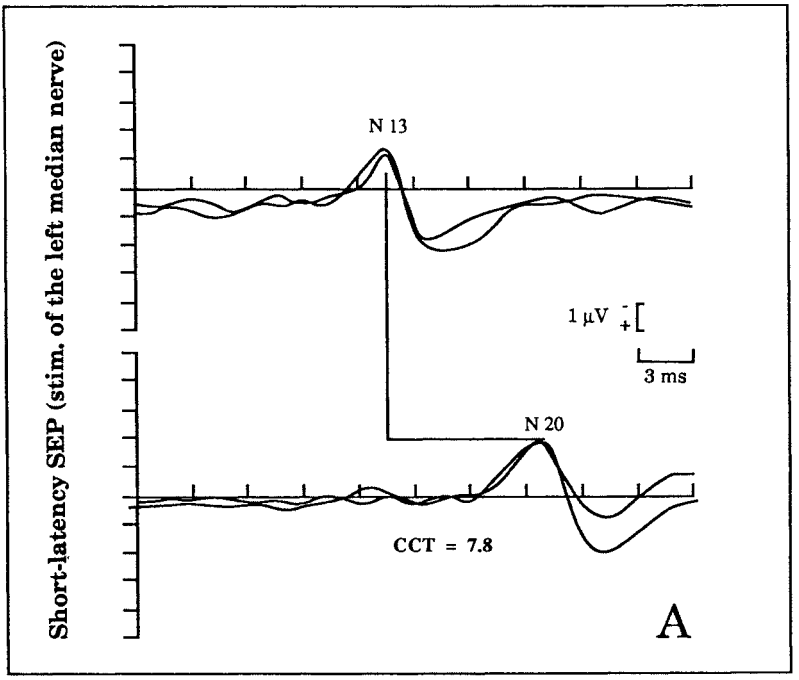
As has been well demonstrated by Symon et al. (1979), preoperative changes in CCT do not predict impending ischemia related to posthemorrhagic arterial spasm. However, we found it useful to compare preoperative CCT with the rate of blood flow in the middle cerebral artery (MCA), measured using transcranial

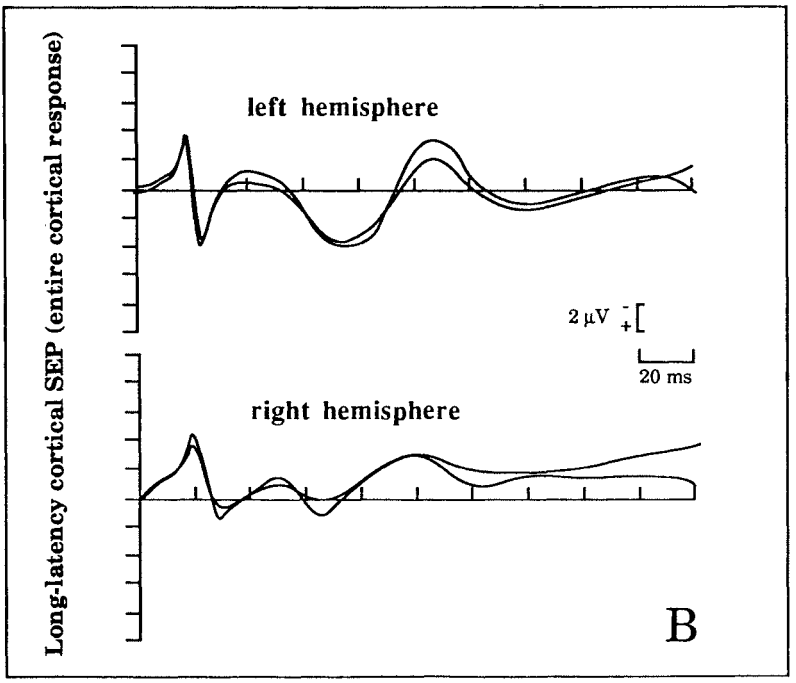
Doppler sonography. Patients who had normal rate of blood flow (i.e., less than 100 cm/s) invariably had normal CCT, whereas 7 of 13 patients who had increased MCA flow rate (above 150 cm/s) had ipsilateral prolongation of CCT and depression of the late waves. Sample SSEP records from 1 patient in this group are shown in Fig. 1. The occurrence of arterial vasospasm does not necessarily imply the presence of cerebral ischemia, although vasospasm is one prerequisite for ischemia. As concerns the relationship between an aneurysm's clinical grade and the patient's CCT, our results confirm that some correlation exists, as illustrated in Fig. 2. Figure 2 also shows that variability of CCT is somewhat greater among patients with low-grade aneurysms. The analysis of the entire cortical response yields further information about cortical perfusion: asymmetries in the amplitude of N_{20} and in the amplitudes and articulation, i.e., number of peaks, of later waves are highly suggestive of hemispheric hypoperfusion. Therefore, a patient with a low-grade aneurysm (see, for instance, Fig. 1) who has a prolonged CCT and reduced cortical response will probably be at utmost risk during surgery, particularly if hypotension is induced.

At our institution, anesthesia for neurosurgical procedures is usually induced with a short-acting barbiturate (thiopental 5 to 7 mg/kg) and maintained with enflurane 0.8 to 1% and nitrous oxide/oxygen (60%/40%). The level of anesthesia is held more or less constant throughout the operation. Deliberate hypotension is induced by continuous infusion of nitroglycerin (4 mcg/ml). The MBP at the time of skin incision is usually about 95 to 100 torr. Anesthesia, together with a slight reduction in body temperature, always affects slSSEP as well as the cSSEP: CCT increases by 0.5 to 0.7 ms over both hemispheres, independent of preoperative CCT, and the amplitude of the late cortical waves is reduced by approximately 20%. The amplitude of the N_{20}/P_{25} complex shows minimal if any change. These ranges of changes of CCT and of waveform amplitude are typical of the enflurane we use for neurosurgical procedures; we have noted that higher concentrations, used in different kinds of operations, have more severe effects on SSEP.

In our first series of 50 patients, 4 patients were paretic before the operation and did not improve afterwards. In the remaining 46 patients, when MBP was reduced to about 60 torr (80 torr SBP) by nitroglycerin, the CCT was further prolonged by approximately 1 ms. Four of these 46 patients suffered severe clinical deterioration postoperatively: 1 died, 2 were hemiparetic, and 1 patient was aphasic. In looking for the reasons for such postoperative deterioration we first investigated intraoperative changes in CCT, but it became evident that the value of this parameter alone is not predictive of the patient's postoperative clinical status. Indeed, some patients with a good clinical outcome had greater intraoperative prolongations of CCT than some patients whose clinical status deteriorated. It became apparent that only two conditions were predictive of

Fig. 1. SSEP recorded preoperatively from a patient with grade II aneurysm of the right middle cerebral artery. **A** Central conduction time *(CCT)* was delayed (upper normal CCT = 6.7 ms). **B** Amplitude of cortical late waves was reduced over the right hemisphere


Short-latency SEP (stim. of the left median nerve)
N 13
1 μV
3 ms
N 20
CCT = 7.8
A


Long-latency cortical SEP (entire cortical response)
left hemisphere
2 μV
20 ms
right hemisphere
B

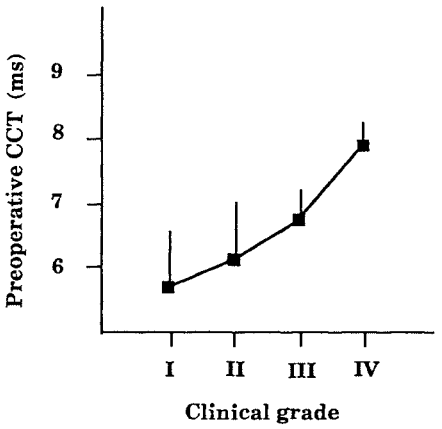


Fig. 2. Relationship between cerebral aneurysm clinical grade, according to the unmodified Hunt and Hess (1968) scale, and mean preoperative CCT. The vertical bars indicate the standard deviation. Note that the lower the grade, the higher the variability in CCT

postoperative deterioration: 1) an intraoperative CCT exceeding 9 ms maintained for 10 minutes or more, and 2) a complete and irreversible flattening of the late components of cSSEP. We never observed a flattening of N_{20} in this series (Ducati et al. 1988). These results are summarized in Table 1.

The patients whose clinical status deteriorated postoperatively could have been identified in advance on the basis of their preoperative CCT and cSSEP. Thus, they all had preoperative CCT equal to or longer than 7.5 ms in the affected hemisphere, and the amplitudes of their ipsilateral late waves were less than 50% of the amplitudes of waves in their contralateral hemispheres. Patients with such preoperative findings were not good candidates for surgery under hypotension.

These results led us to modify our operative strategy. If inducing hypotension would put a particular patient at risk for acquiring postoperative neurological deficits but blood pressure in the aneurysm needed to be reduced in order to be able to manipulate and clip the aneurysm safely, then the technique of temporary clipping under normotension was preferred. Sample SSEP recordings from a patient in this group are shown in Fig. 3. This patient showed signs of being at risk

Table 1. *SSEP changes in patients with poor outcome*

Aneurysm Location	Outcome	CCT Right (ms)	CCT Left (ms)	Late Waves Right	Late Waves Left
ACA	death	9.5	9.5	–	–
LMCA	R. hemiparesis	8.8	10.2	+	–
LMCA	R. hemiparesis	8.4	13.0	+	–
ACA	aphasia	7.8	8.3	+	–

ACA, anterior cerebral artery;
LMCA, left middle cerebral artery;
CCT, greatest value of central conduction time reached during surgery;
(–), irreversible loss of the late waves of cSSEP;
(+), persistence of late waves of cSSEP

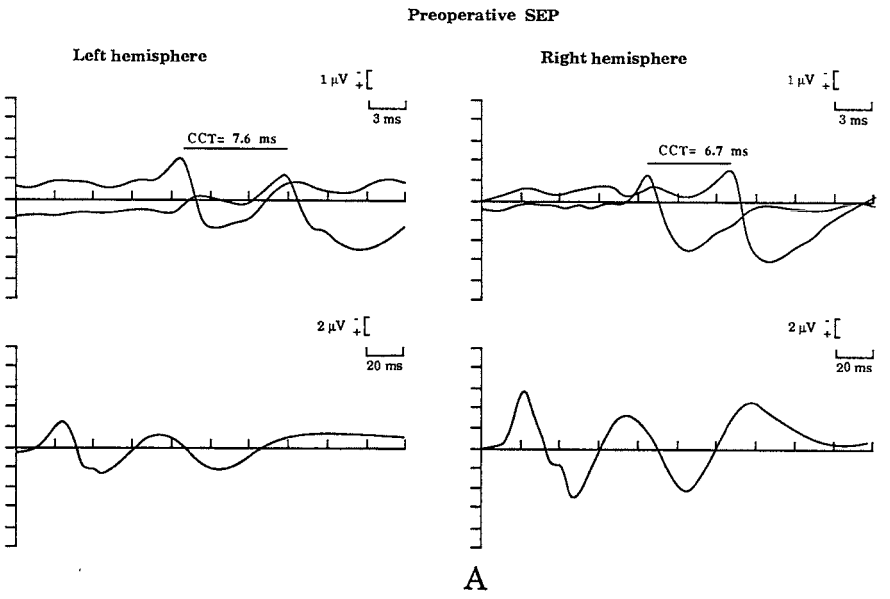
preoperatively, i.e., a CCT exceeding 7.5 ms and reduced late waves over the left hemisphere. Nevertheless, hypotension was induced during the operation, but when an MBP of 65 torr was reached the CCT as well as cortical late waves were severely affected. These changes were quickly reversed by raising the MBP to 90 torr. When the MBP was 100 torr a temporary clip was applied, and thereafter the SSEP remained within the limits defined by previous experience as being consistent with a good outcome. The operation was successfully completed and the patient awoke without any new neurological deficit. We have followed this procedure with subsequent patients, and no postoperative deterioration has occurred among patients undergoing operations to remove aneurysms using this technique and evoked potential monitoring. We have, of course, observed reversible SSEP changes as a consequence of excessive brain retraction, but this problem has already been discussed in detail by other authors (Grundy 1982; Symon et al. 1984; Wang et al. 1984; Schramm et al. 1989, 1990).

Finally, we noted how retraction and hypotension affect brainstem auditory evoked potentials (BAEP) recorded during operations to manage aneurysms of the vertebral artery (Ducati et al. 1988). A prolongation of the interpeak latency of wave I-V occurred after the retractors were placed; the BAEP partially recovered when retractors were repositioned, but an even more severe degeneration of the waveform developed when hypotension was induced just before the aneurysm was clipped. This change in the BAEP reversed quickly when the blood pressure was increased after clipping, and at the end of the operation the interpeak latency of waves I-V was the same as it was in the beginning. The interpeak latency of waves I-V at the start of the operation in this patient was 5.1 ms, and the maximal value reached 5.6 ms; these values are consistent with limits we obtained for BAEP recorded during operations in the posterior fossa.

Disussion

Judging by the literature, slSSEP are often used to monitor somatosensory pathways and the primary somatosensory cortex but long-latency cortical responses are seldom used for monitoring purposes. We found recording cSSEP to be very useful in intraoperative monitoring for two major reasons. First, the entire waveform of the cortical response is made up of the N_{20}/P_{25} complex (early response) and the following "slow" waves of longer latency that are recorded with a 200-ms analysis time. Because the amplitude of long-latency waves decreases when high rates of stimulation are used, we obtained cSSEP with a stimulation frequency of 1 Hz. Low-frequency stimulation evokes the larger N_{20}/P_{25} complex as well, and therefore emphasizes changes in waveform amplitude that result from changes in cerebral perfusion. Measurements of peak latencies for the purpose of determining CCT, however, are less accurate than are recordings of se SSEP.

Second, the entire cortical response includes the activity of cortical areas that are functionally related to the primary area but that extend over the posterior parietal regions. Thus, cSSEP also test the watershed regions of the middle cere-



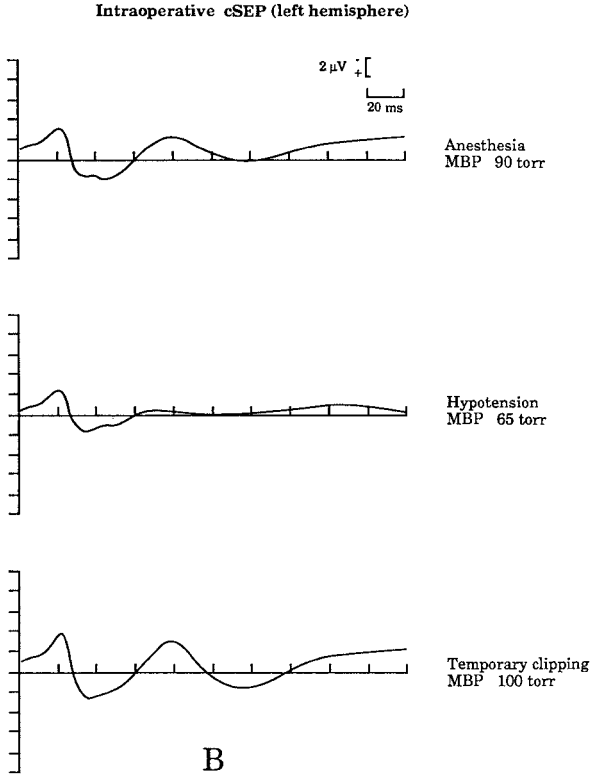


Fig. 3. SSEP of a patient with aneurysm of the left middle cerebral artery. **A** Preoperative SSEP evidenced CCT >7.5 ms *(left top)* and low-amplitude cortical late waves over the left hemisphere *(left bottom)*. Both CCT and late waves were normal over the right hemisphere *(right top and bottom)*. **B** Intraoperative SSEP recorded over the left hemisphere are shown. *Top tracing:* cSSEP recorded under anesthesia at the beginning of the operation. *Middle tracing:* significant changes developed when hypotension was induced. *Bottom tracing:* temporary clipping with normal blood pressure was associated with cSSEP within normal limits. This patient awoke from anesthesia without any new deficit

bral artery (MCA), where reduced perfusion is more likely to develop with induced hypotension. A commonly reported drawback of intraoperative monitoring of long-latency waves of cSSEP is their high variability. We, however, observed that as long as the anesthesia is kept constant for each patient, that patient's cSSEP are fairly reproducible and reliable.

It is obvious that patients whose cerebral circulation is compromised preoperatively are at a higher risk during operations to manage cerebral aneurysms because such patients do not tolerate anesthesia, brain retraction, and, particularly, hypotension, whether accidental or deliberate. As Thomas Huxley (1825–1895) stated in his Essays, "Science is nothing but welltrained and organized common sense." Perhaps Huxley knew that 50 years earlier Pierre-Simon de Laplace (1749–1827), in his "Introduction to the Analytic Theory of Probability," states, "...The theory of probability is basically common sense reduced to figures."

Without claiming to be scientists, we have attempted to arrive at "figures" that represent significant changes in clinical parameters, and to define those circumstances under which these "figures" make sense or, better, match values that might be arrived at by common sense. Our results show that, with balanced anesthesia (enflurane 0.8 to 1%, nitrous oxide 60%, oxygen 40%), a CCT equal to or longer than 9 ms together with flattening of cSSEP late waves are invariably associated with postoperative deterioration in neurological status. We did not see any deterioration with prolongation of the CCT alone, even with prolongation to or beyond the threshold value of 9 ms, when the above-described conditions of anesthesia and hypotension were not met. Analysis of the late components of cSSEP has proven to be useful for evaluating function of brain areas outside the primary somatosensory cortex. We suggest adding recording of this component to the traditional CCT measurement in order to obtain a measure of both subcortical neural conduction and of cortical responsiveness. When SSEP recordings suggest that the surgical or anesthesiological procedure in use implies a risk of permanent neurological deficits, alternative approaches should be chosen and the risk intrinsic to each of these should be tested. This increases the safety of the procedure for the patient.

Although intraoperative monitoring of functional aspects of the central nervous system with evoked potentials has been proven useful beyond any doubt, it is quite expensive in terms of time and personnel required. We have been able to record evoked potentials intraoperatively in no more than one third of all patients with cerebral aneurysms who were operated upon in our institution, and we frequently were un-able to monitor during operations carried out under emergency conditions, mostly because the equipment and trained personnel were already being used to monitor another patient. The solution may be to have the anesthesiologist include evoked potentials in the clinical parameters that are monitored intraoperatively. Monitoring of evoked potentials during neurosurgical operations is certainly as important as monitoring heart and ventilatory function.

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Value of Stable and Changing Somatosensory Evoked Potentials (SSEP) During Aneurysm Surgery*

J. SCHRAMM[1] and M. TANIGUCHI

Summary

Since it was established that monitoring somatosensory evoked potentials (SSEP) is a good method for evaluating the intactness of sensory afferent conduction, this method has been more and more frequently used during aneurysm surgery. This chapter describes the technical aspects of this method, including technical and anatomical limitations to its use, and the clinical value of this technique, with special consideration of those cases in which the preservation of SSEP or the disappearance of SSEP provided information considered helpful by the surgeon. The data presented were obtained during monitoring of 157 operations to manage 177 aneurysms out of a total of 190 cerebral vascular operations.

One of the remarkable findings in this study was that SSEP may be lost not only following impaired blood flow due to vascular occlusion, but also following retraction of the brain. The incidence of loss of potentials with occlusion of blood vessels is variable: in this study, 58 patients had vessel occlusions and in 19 of these evoked potentials changed. Other events associated with an evoked potential change were retraction of the cerebellum and splitting of the Sylvian fissure.

In an interesting subgroup of 17 cases, in which the vessel occlusion occurred accidentally, the loss of SSEP was the only sign of compromised blood flow in 2 cases. In the majority of these cases the surgeon detected the accidental vessel occlusion, either quickly or after some further dissection without the help of SSEP monitoring.

The lack of SSEP changes in cases where vessel occlusion was necessary was found to provide helpful information to the surgeon. Aneurysm dissection and clipping proceeded smoothly in several cases because the lack of SSEP change even when a major cerebral vessel was clipped signaled good collateral blood flow.

If the surgeon is sure that the supply area of the vessel is being monitored by the chosen SSEP monitoring modality, an absence of SSEP abnormalities during clipping of supratentorial aneurysms may be considered a sign that the blood supply to the brain regions being monitored is satisfactory. Monitoring SSEP during aneurysm surgery provides the surgeon with valuable information, especially dur-

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[1] Neurochirurgische Universitätsklinik, Sigmund-Freud-Str. 25, W-5300 Bonn, FRG.

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ing difficult procedures such as surgery on giant aneurysms; management of large, multilobed middle cerebral artery aneurysms, which require temporary clipping; and permanent vessel occlusion to treat unclippable aneurysms.

Despite all improvements in surgical technique during the last two decades, aneurysm surgery still carries a certain risk to the patient (Ljunggren et al. 1983). A considerable proportion of this risk comes from intraoperative reduction in cerebral blood flow, which may be caused by planned temporary vessel obstruction, by inadvertent encroachment on the vessel lumen, or by other factors. The intraoperative monitoring of somatosensory evoked potentials (SSEP) was introduced as a method to detect such disturbances in neural conduction in patients under anesthesia, and its use is based on the work by Hargadine et al. (1980), Branston et al. (1974), Symon (1973), and Hume and Cant (1978). Several reports have appeared that underline the value of this technique (Friedman et al. 1981; Grundy et al. 1982; Kidooka et al. 1987; Little et al. 1987; McPherson et al. 1983; Momma et al. 1987; Mooij et al. 1987; Schramm et al. 1990; Symon 1973; Symon et al. 1984; Thurner and Schramm 1986). Several of these authors have also pinpointed some limitations of this technique (Friedman et al. 1981; Little et al. 1987; McPherson et al. 1983; Schramm et al. 1990). From managing more than 170 aneurysms we have gained experience both with changing and with stable SSEP during aneurysm surgery; this chapter describes our experiences with and our conslusions regarding use of this monitoring technique for this type of operation.

Methods

The majority of our patients underwent operation with balanced anesthesia consisting of fentanyl in incremental doses in combination with 65% nitrous oxide or up to 0.5 minimal alveolar concentration (MAC) Ethrane (enflurane). Anesthesia was induced with a short-acting barbiturate, fentanyl, and pancuronium. Several patients were given midazolam or etomidate instead of the barbiturate for induction. In the last 17 cases in our series anesthesia was induced and maintained solely with intravenous administration of propofol.

In addition to the intraoperative SSEP monitoring, all patients underwent preoperative and postoperative SSEP testing. After induction of anesthesia and positioning of the patient on the operating table, electrodes were attached to the patient for bilateral stimulation and bilateral recording. The median nerve at the wrist and the posterior tibal nerve at the ankle were used, depending on the area to be monitored, as will be described later. The stimulation, recording, and data collecting were performed with a Nicolet Pathfinder I or a Nicolet CA 1000 averager. Data were stored on floppy diskettes, but with the use of a transfer program described elsewhere in this volume (Watanabe and Schramm, 1991), were also transferred to a personal computer and printed out during the operation. Square-wave electrical pulses of 20-mA intensity and 300-μs duration were used at a 5.3-Hz stimulation rate. Plate electrodes, 1 cm in diameter, were used for stimulation

and needle electrodes were used for recording. Recording electrodes were placed 2 cm inferior to C3, C4, and Cz in standard electrode positions according to the international 10-20 system. The reference electrode was situated at Fz. The sensitivity of the averager was set to $\pm 25-50$ μV, and a bandpass of 30 to 3000 Hz was selected. The cervical electrode was placed on the skin above the second cervical vertebra. An averaged evoked response consisted of at least 150 to a maximum of 500 responses. Two averaged responses were always superimposed to evaluate replicability.

The cervical and cortical SSEP were used to calculate the central conduction time (CCT). As previously described (Hargadine et al. 1980; Hume and Cant 1978), the CCT is the difference between the latency of the cortical N_{20} and latency of the cervical N_{14} of the median nerve SSEP. The presence or absence of the primary cortical complex ($N_{20}-P_{25}$), the value of the CCT, and the presence of the primary cortical complex of the tibal nerve SSEP ($N_{37}-P_{46}$) were used as parameters in the monitoring. Because the mean value for the CCT was found to be 5.82 ms with a standard deviation of 0.5 ms in a normal control population of 81 adults, the upper normal limit of CCT was taken to be 7.1 ms. A decrease to less than 50% of the intraoperatively established baseline in the amplitude of the primary cortical complex (baseline in this case was established after the patient was anesthetized but before the procedure began) was arbitrarily defined as the criteria of abnormality for cortical SSEP.

In all patients electrodes were placed bilaterally and the actual type of monitoring was selected according to which vessel was at risk during a particular part of the procedure. Monitoring median nerve SSEP is standard when an operation is performed to manage aneurysms in the territory of the internal carotid artery and the middle cerebral arteries. When the aneurysm is in the pericallosal artery, monitoring of posterior tibal nerve SSEP alone is sufficient. When surgery was performed to manage aneurysms of the posterior fossa or basilar tip, supplemental monitoring of brainstem auditory evoked potentials (BAEP) was performed.

When an aneurysm involved the anterior communicating artery complex, both hemispheres were monitored by placing recording scalp electrodes over the leg area and alternately stimulating the right and left posterior tibal nerves. The addition of median nerve stimulation to the monitoring protocol is often necessary when an aneurysm involves the anterior communicating artery complex because the recurring artery of Heubner, which supplies the basal ganglia, cannot adequately be monitored by posterior tibal nerve stimulation alone. During operations to manage a posterior communicating, internal carotid, or middle cerebral artery aneurysm adequate information is almost always obtained by recording SSEP while alternately stimulating the two median nerves. However, when a complex or giant aneurysm involves the anterior communicating artery complex inadvertent occlusion of Heubner's artery is possible, so that during disseciton of Heubner's artery ipsi- or contralateral median nerve SSEP should be monitored, and sometimes the median nerve SSEP should be monitored for a longer period of time. The actual decision as to which territory should be monitored during which period of an operation has to be made by the surgeon in close collaboration with the electrophysiologist monitoring the SSEP.

When monitoring is performed during surgical management of posterior fossa aneurysms, it should be kept in mind that the medial lemniscus crosses over in the brainstem approximately at the level of the obex, so that ipsilateral stimulation is necessary if the aneurysm is located caudal to the obex.

Patients

Of a total of 190 vascular operations, 157 patients were operated upon to manage 177 aneurysms. The other 33 cases were carotid endarterectomies. A few patients were operated on for incidental aneurysms. The distribution of the aneurysms by site did not differ significantly from the typical distribution reported in the literature, except that in 19 of the 157 patients the aneurysm was in the posterior fossa.

All patients were re-examined at discharge and later as out-patients. Each patient's neurological status at the "early" (predischarge) and "late" (out-patient) follow-up examinations was correlated with intraoperative SSEP changes. Details of most of these cases have been presented elsewhere (Schramm et al. 1990).

Results

Potential Changes Not Related to Surgery

In a small portion of cases, recording of median nerve SSEP was impaired by technical problems and in 3% of cases monitoring of median nerve SSEP was impossible. Due to technical problems with recording procedures and inhalational anesthesia, monitoring of tibal nerve SSEP was impossible in 23% of our initial cases. Since we began to use propofol intravenous anesthesia, the monitoring failure rate due to such technical problems has decreased to 6.5% for posterior tibial nerve stimulation. The typical changes related to anaesthesia that are described by most other authors (Friedman et al. 1981; Little et al. 1987; Momma et al. 1987; Mooij et al. 1987; Schramm et al. 1990; Symon et al. 1984) were also seen in our patients. Recordings obtained after the patient had been anesthetized and anesthesia-related effects had stabilized were used as baseline values. By comparing intraoperative recordings, made during periods of the operation when cerebral blood flow was at risk, to these baseline recordings, the significance of changes could easily be assessed. In nearly all cases continuous intraoperative infusion of nimodipine at 30 μg/kg/h resulted in no SSEP alterations. Monitoring the patient's systolic blood pressure at about 80 to 90 mm Hg during aneurysm dissection led to SSEP changes in only 3 cases; in 2 cases SSEP changes were associated with retraction (Fig. 1). In approximately 60% of cases aneurysm surgery was performed within the first 72 hours after identification of the lesion.

Vessel Occlusion

In 58 patients vessels were occluded: in 30 patients vessels were temporarily clipped intentionally, in 11 vessels were intentionally permanently occluded, and in

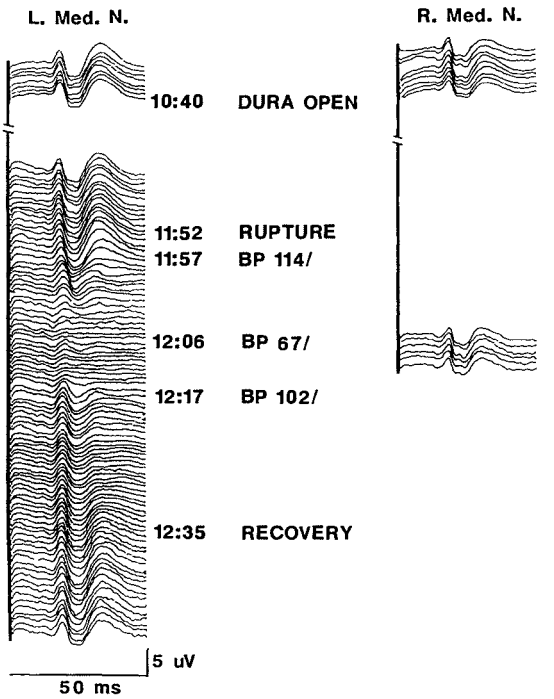

Fig. 1. The combined effects of induced hypotension plus vascular manipulation are demonstrated in this case. An internal carotid artery aneurysm ruptured during dissection and blood pressure was lowered significantly from 114 to 67 mm Hg systolic. While in the contralateral hemisphere SSEP remained normal during hypotension, median nerve SSEP disappeared on the affected side. After the rupture had been managed and blood pressure returned to normal, SSEP recovered in the same sequence they had deteriorated. The cortical N_{20} persisted longer and recovered earlier than the later components

17 temporary vessel occlusion occurred accidentally. In 19 of the 58 patients with vessel occlusions SSEP changes were noted; in 12 of these 19 cases SSEP were lost (Fig. 2), and in the other 7 cases SSEP were altered. The period of time from vessel occlusion until the SSEP change occurred was very variable, but usually the SSEP change occurred within 1 to 4 minutes of vessel occlusion. A slow, steady amplitude decrease that progressed to total loss of the potentials was the exception. Usually a more or less abrupt loss of the potentials was seen, often preceeded by a brief and slight decrease in amplitude. However, we have also seen SSEP changes occur much later after vessel occlusion, up to 22 minutes after the event. The main conclusion we draw from this finding is that although a patient may tolerate well a vessel occlusion of 2 to 3 minutes, that patient may not tolerate vessel occlusion indefinitely. (The usual period of vessel occlusion for the Matas test is only 2 minutes.)

SSEP Changes Associated With Nonoccluding Surgical Events

Of 6 patients in whom the brainstem was retracted, 1 lost SSEP (Fig. 3). In 2 patients in whom the cerebellum was retracted to a significant degree, changes in and loss of SSEP were seen once each. Vessel retraction led to one incident of SSEP change in each of 4 cases, but in 2 cases in which the vessel lumen was narrowed intentionally no SSEP alterations occurred. Three incidences of severe mechanical spasm in a major artery also were not associated with SSEP changes.

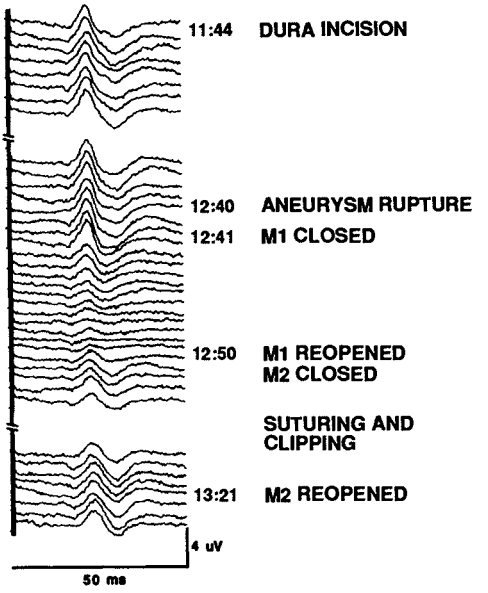

Fig. 2. Temporary placement of a clip on an avulsed middle cerebral artery aneurysm in a 58-year-old woman. After uneventful dissection of the aneurysm and its neck the first clip was not quite satisfactory and repositioned. During repositioning the neck of the aneurysm was partly avulsed at its base and the M1 branch of the artery was temporarily clipped. This led to an immediate marked reduction in amplitude of the SSEP. Repeated attempts to clip the aneurysm and at the same time occlude the partly avulsed neck failed, and the severe loss of amplitude progressed to near total loss of SSEP. Therefore the temporary M1 clip was positioned obliquely so that it mostly occluded one of the two M2 branches and allowed partial perfusion of M1 and the other M2 branch. This was followed by rapid recovery of the amplitude of the primary cortical complex. The defect was then sutured and the aneurysm clipped uneventfully. Postoperatively the patient had mild hemiparesis that resolved completely in approximately 36 hours. In this case, the surgeon was helped in a very difficult situation by good feedback from the electrophysiologist monitoring the patient

Reaction to SSEP Changes

One hundred fifty-seven patients (with 177 aneurysms) were monitored intraoperatively and in 23 cases SSEP changes were found, i.e., in approximately 15% of operations. In 19 of the 23 cases with SSEP changes the surgical course was altered in response to these monitoring results. Typical changes included repositioning the retractor, repositioning the aneurysm clip, early or repeated removal of a temporary vessel clip (Fig. 4), and elevation of blood pressure. In several cases no response from the surgeon was necessary because SSEP recovered spontaneously.

Significance of Lack of SSEP Changes

When the afferent pathway being monitored is in tissues supplied by the blood vessel being manipulated during aneurysm surgery, maintenance of normal SSEP can make a difference in how the surgeon conducts the operation. This was the case in about one fifth of our patients (Figs. 2, 5). For example, when SSEP remain stable with occlusion of an intracavernous giant aneurysm, the surgeon is helped in the decision to occlude the vessel permanently. In other cases, lack of changes in SSEP can help the surgeon in deciding to continue temporary clipping of blood vessels. In 3 cases in our series the surgeon decided against performing

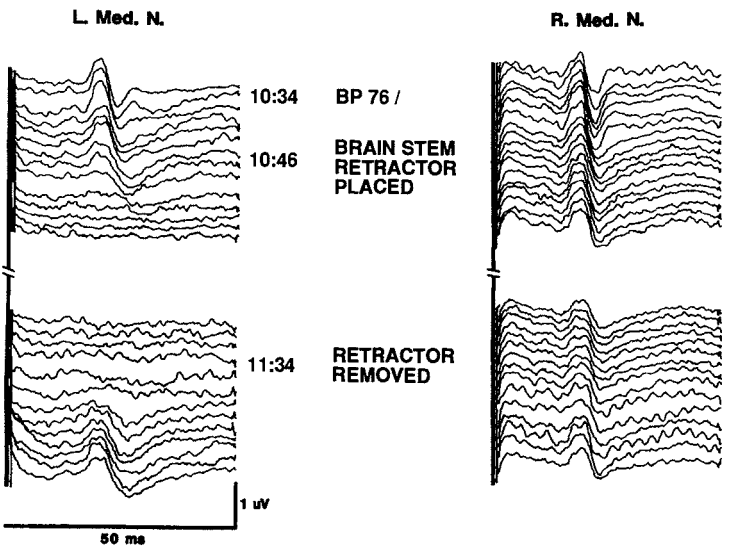

Fig. 3. Loss of potentials with brainstem retraction in a 28-year-old woman operated upon via a subtemporal approach for management of a small basilar tip aneurysm. A small-tipped malleable retractor placed over a cotton pledget on the right cerebral peduncle was used to retract the brainstem because the aneurysm was projecting posteriorly. None of the larger arteries was clipped, and none of the smaller perforating vessels was compromised. For this reason, and because of the anatomical location of the aneurysm, it was decided to continue with brainstem retraction despite loss pf SSEP. When the aneurysm was clipped, the retractor was removed and the potentials recovered within 3 minutes. It may safely be concluded that in this case the loss of the SSEP was mainly caused by peduncular retraction. The very low mean arterial blood pressure most likely was an additional factor in loss of SSEP. The patient's blood pressure was not raised, however, because of reports that even when SSEP are lost for periods up to 1 hour there may be no negative consequences

an extracranial-intracranial bypass because SSEP remained normal, indicating adequate perfusion. In other cases, when SSEP were unchanged by temporary vessel occlusion after premature aneurysm rupture, the surgeon was able to dissect the aneurysm neck calmly instead of moving quickly to reverse vessel occlusion.

Discussion

The sequelae of subarachnoid hemorrhage are severe, and may actually add to the morbidity of patients operated upon for aneurysms, as described by Ljunggren et al. (1983). The results of our study support previous reports that monitoring SSEP during such operations makes it possible to detect when impaired blood flow occurs (Friedman et al. 1981; Kikooka et al. 1987; Little et al. 1987; Momma et al. 1987; Mooij et al. 1987; Schramm et al. 1990; Symon 1973; Thurner and Schramm 1986). Our results also strengthen previous reports that

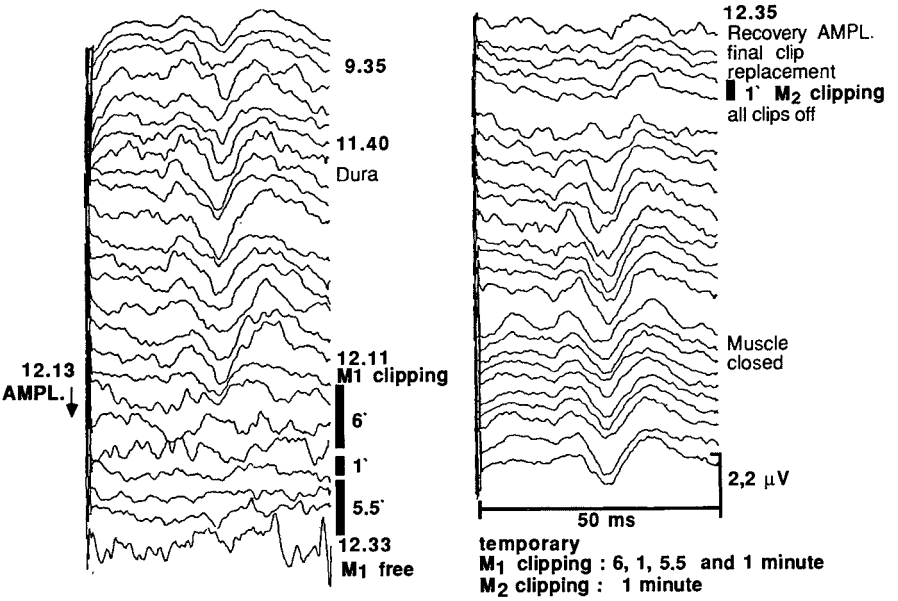

Fig. 4. Immediate loss of SSEP after temporary clipping of a giant, right-sided, middle cerebral artery aneurysm. Because of this loss, 3 periods (of 6, 1, and 5.5 minutes) of temporary M1 clipping were interrupted by 2 5-minute periods of blood flow in this vessel. Although potentials did not recover during those 5-minute periods of blood flow, the patient had no postoperative deficit. A period of 25 minutes with loss of SSEP and lack of blood flow might still have resulted in normal neurologic function postoperatively, but we believe it is justified to assume that the 10 minutes of re-established blood flow were helpful in avoiding a postoperative neurologic deficit in this patient

the loss or absence of SSEP also provides helpful information to the surgeon, provided certain reservations are kept in mind (Grundy et al. 1982; McPherson et al. 1983; Schramm et al. 1990).

The relatively high incidence of accidental occlusion of blood vessels results from the increasing acceptability of preliminary aneurysm clipping before the details of anatomy in that particular case have been clarified; this is often the case with large or giant anterior communicating or middle cerebral artery aneurysms. In these situations aneurysm clips may be used to reduce the bulk of the aneurysm so that the surgeon can more easily gain an overview of the area.

If a change occurs in SSEP, in many situations the operative technique may be modified in response to that change. Even if premature rupture of the aneurysm has necessitated temporary vessel clipping, the loss of SSEP may prove helpful: the time until disappearance of the SSEP may be used to proceed with dissection of the aneurysm and securing the leak. While continuous pressure is applied with a cotton pledget over the lead, temporary reestablishment of blood flow can be permitted to prevent permanent ischaemic damage (Fig. 4).

Possible actions by the surgeon in response to intraoperative SSEP changes, such as adjusting an aneurysm clip or performing extra-intracranial bypass sur-

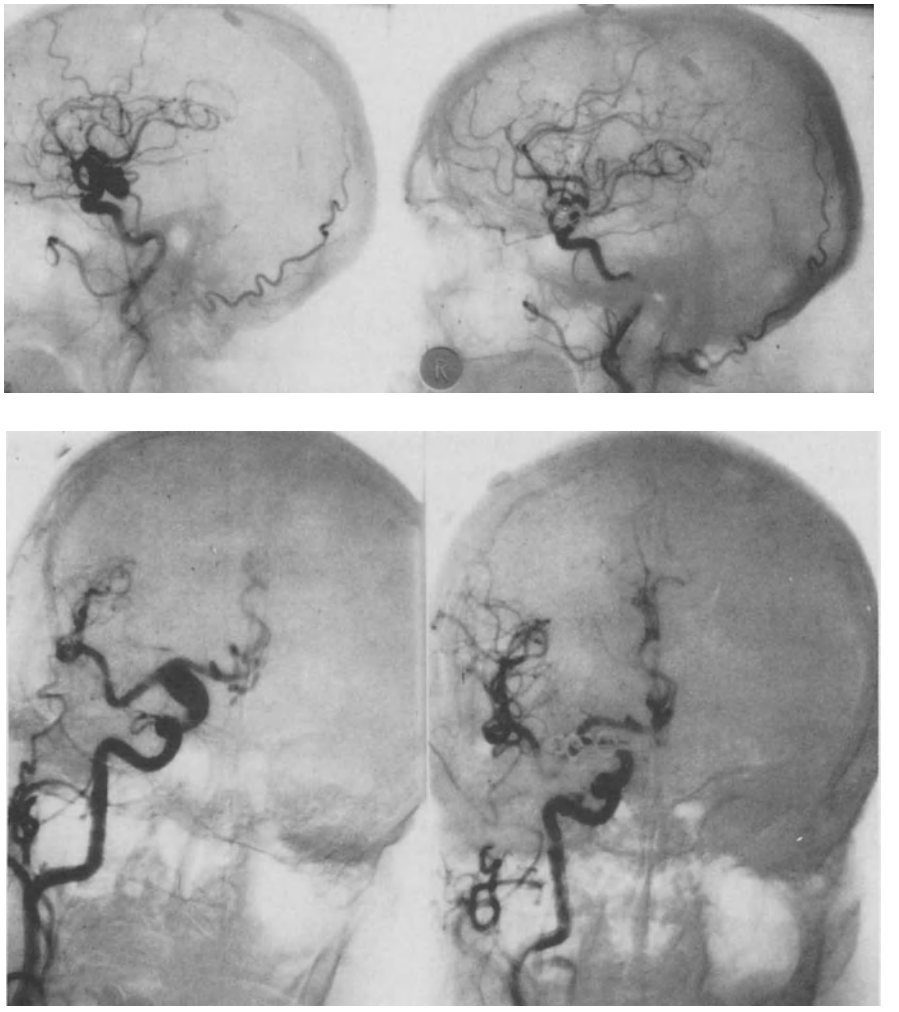

Fig. 5. Preoperative *(left)* and postoperative *(right)* carotid angiograms of a 72-year-old man with a giant internal carotid artery aneurysm. This broad-based aneurysm had no true neck and could only be clipped with several ring clips. Because the walls of such aneurysms are often atheromatous and thickened, it is difficult to assess by observation of the vessel whether the newly formed lumen is large enough to allow enough blood to pass through to maintain function of the cerebrum. In this case, the fact that SSEP remained stable for a long period of time after clips had been applied to the aneurysm satisfied the surgeon that it was not necessary to debulk the mass to ensure adequate blood flow. The patient had no neurologic deficit postoperatively

gery, or even the abortion of a proposed procedure, have previously been mentioned by Friedman et al. 1981.

The lack of a significant SSEP change when a difficult intraoperative maneuver is performed may provide useful information to the surgeon. Such maneuvers include long periods of temporary clipping, retraction of major cerebral vessels, intentional permanent vessel occlusion, and reconstruction of the vessel lumen affected by a giant aneurysm (Fig. 5). As pointed out in previous studies (Friedman et al. 1981; Little et al. 1987; Schramm et al. 1990), the lack of intraoperative changes in the SSEP in operations for aneurysms of the posterior circulation should not lead the surgeon to assume that no damage is being done to tissues. But if SSEP disappear during operation that affects the posterior circulation, it may rightly be assumed that ischaemia is affecting the brainstem.

From reports on several series of cases, it appears that intraoperative monitoring of SSEP is a reliable way to detect cerebral ischaemia if one pays attention to the anatomical areas served by the blood vessels being manipulated and the neural pathways being monitored. Both the preservation and the loss of SSEP proved helpful indicators of neural pathway integrity of patients undergoing aneurysm surgery. Thus, SSEP monitoring was definitely useful in our experience, and it often influenced the course of surgery in complicated cases or when difficult surgical maneuvers were being performed.

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Experiences with Intraoperative Temporary Vessel Occlusions and Monitoring in Aneurysm Surgery

L. SYMON[1] and D. A. JELLINEK

Summary

Acquisition of physiological data pertinent to the state of the function of the central nervous system during neurosurgical procedures is desirable wherever possible. For this information to be truly valuable it is necessary to have an experimental framework within which individual observations can be interpreted. Our clinic has studied the effects of controlled cerebral ischemia on electrophysiological cortical function in the primate and has established the concepts of "the ischemic penumbra" and "flow thresholds."

These concepts have subsequently been employed to interpret somatosensory evoked potential (SSEP) changes during temporary vascular occlusion in aneurysm surgery. Over the years it has become apparent that although some degree of prediction can be drawn from statistical analysis of surgical outcome and intraoperative disturbance of the SSEP during cerebral ischemia, one is often left to draw predictions as to the safety of a surgical manoeuvre from key cases. This paper discusses these problems and illustrates the significance of individual intraoperative experiences of temporary vascular occlusion.

It was Møller who said that neurophysiologists have a tremendous opportunity to study human physiology in the operating theatre. Data collection in the operating room is, however, often uncontrolled and recordings show enormous variability from one patient to another, depending on the original pathology involved. It is important therefore to have an experimental framework into which isolated clinical observations can be placed, hence more easily understood.

Perhaps the best model of human cerebrovascular physiology available is the primate, developed experimentally because the cerebrovascular anatomy and neurophysiology of primates most closely resemble those of man (Symon et al. 1974). By studying evoked potentials recorded during cerebrovascular operations one is really hoping to identify the relationships between cerebral blood flow (CBF), electrical activity, and, ultimately, cerebral function. Maintenance of normal brain metabolism depends upon maintenance of normal CBF. A disturbance of cerebral perfusion interferes with energy production by the brain. Energy is used by the brain in two ways: not only to maintain structure, but also

[1] Gough Cooper Department of Neurological Surgery, Institute of Neurology, The National Hospital, Queen Square, London WC1N 3BG, U.K.

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for interneuronal communication. The synchronized electrical discharge of the somatosensory cortex (SSEP) can be used as a reliable indicator of the brain's metabolism in the presence of energy starvation, such as occurs as a consequence of cerebral ischemia.

Our original work relating somatosensory evoked potential (SSEP) amplitude to CBF in the baboon (Branston et al. 1974) demonstrated a threshold relationship between CBF and the SSEP. The SSEP disappeared when CBF fell below 20 ml/100 g/min. Loss of the SSEP was not necessarily an immediate consequence of a decrease in cerebral perfusion: up to 20 minutes could elapse from the onset of loss of CBF to the time the evoked potential disappeared. In some baboons we found that the median nerve SSEP persisted despite middle cerebral artery occlusion, indicating that the decrease in blood flow over the relevant sensory cortex areas never reached a critical value because a good collateral blood supply was present.

We have also studied the relationships between CBF and motor cortex function by evaluating the direct cortical response and pyramidal tract motor evoked potentials (MEP) (Sabin et al. 1989) and we found the same relationship between function and perfusion: the early (D-wave) component of the MEP response was lost when cortical blood flow fell below 20 ml/100 g/min. Although we cannot be certain, it appears that many areas of the cerebral cortex have a similar threshold for tolerance to loss of blood flow, so that the cortex as a whole seems to lose function below about the same CBF threshold value.

An indication of the minimal CBF value that is necessary to maintain neuronal structure can be obtained by studying ionic gradients within the cortex (Astrup et al. 1977). If CBF falls to about 10 ml/100 g/min, the results of microelectrode recording make it apparent that ionic hemostasis is lost. For CBF between 10 and 20 ml/100 g/min, ionic hemostasis is maintained while electrical activity fails.

It follows therefore that a recording of electrical activity of the brain will show loss of electrical activity before there is disruption of ionic hemostasis. Further, there is a period of time in which decreased rate of flow causes loss of electrical function but ionic hemostasis is preserved. This is true not only for a single isolated cortical locus but also for different topographic areas, which can experience varying degrees of ischemia. In the experimental primate model, for example, there is a central cortical area that, when the middle cerebral artery is occluded, experiences very dense ischemia (this also applies to the basal ganglia); blood flow in this central cortical area may fall below 10 ml/100 g/min (compared to a normal of 50 ml/100 g/min) while in the somatosensory cortex CBF remains between 15 and 35 ml/100 g/min. There is rapid destruction of ionic hemostasis in the center of such an area of potential infarct, whils in the periphery, where ionic hemostasis is preserved, the degree to which the evoked potentials are impaired depends on the exact cortical blood flow. This is the topographic origin of what we have called the "ischemic penumbra" (Astrup et al. 1981).

This model must be kept in mind when one interprets evoked potentials during aneurysm operations (Symon et al. 1986). What one is looking at then is the electrical response from a specific cortical area below the recording electrode. The degree of ischemia during surgery may be more severe in areas of the cortex not

contributing to the evoked potential; if such is the case, then a significant ischemic event may be evolving without its being detected by monitoring the SSEP. A 100% correlation between the SSEP and neurological function cannot, therefore, be expected. For monitoring blood flow in the terminal carotid and middle cerebral arteries, however, we believe SSEPs from the hand area, which are easy to obtain, are good indicators of cortical function (Symon et al. 1988).

Blood flow thresholds exist in the central nervous system, and can be distinguished on the basis of their anatomic distribution and the functional losses and structural damage that may occur if they are crossed. These thresholds have a time component, meaning that if an area of cortex is exposed to a sufficient degree of ischemia both function and ionic hemostasis may be lost. What constitutes "a safe area" in terms of ischemia is failure of function but preservation of ionic hemostasis. Early reperfusion experiments in the baboon established that both functional and ionic thresholds could be crossed reversibly (Branston et al. 1976). The normal evoked response could be restored with reperfusion within half an hour, and ionic hemostasis restored after an hour long occlusion of the middle cerebral artery. Within the limits of an acute experiment one cannot restore the evoked response after an hour of temporary occlusion of the middle cerebral artery.

Clinical Studies

In our clinic we initially studied central conduction times as described by Hume and Cant (1978) and then began to search for methods to study central conduction in the operating room. Our initial studies showed that measurements of SSEP response amplitude alone were unreliable, partly because the potentials were unstable but also because our recording equipment was technically inadequate. However, it was possible to identify the N_{14} (neck) and the N_{20} (cortical) peaks, to measure their interpeak intervals accurately and, at the same time, to compare the response obtained from the operated side with that from the unoperated side, which then acted as a control (Symon et al. 1986).

This technique was satisfactory for monitoring during operations to manage aneurysms of the carotid and middle cerebral arteries, though there were problems using the technique during operations on anterior communicating and basilar artery aneurysms. The measured central conduction time among our patients has a very small standard deviation (Symon et al. 1986) and is close to values determined by others (Hume and Cant 1978; Desmedt and Cheron 1980; Fox and William 1984). Compared with errors in blood flow measurements, SSEP errors are fantastically low. In our clinic, intraoperative monitoring of SSEP is now performed routinely and with modern recording equipment (Nicolet 2000) that can be transferred easily between the operating room and the ward, obtaining good records is comparatively easy.

The advantage of recording farfield evoked potentials such as the SSEP recorded from scalp electrodes rather than recording directly from exposed neural tissue is the absence of electrodes in the operative field. The standard incision we use is

located well away from the site where recording electrodes are placed, and no time is wasted placing electrodes during the operation.

Our principal use for intraoperative monitoring of SSEP is during procedures to manage giant aneurysms (Symon 1990). The majority of giant aneurysms in our series have arisen from the distribution of the terminal carotid or middle cerebral artery. Monitoring of function in this area by recording evoked potentials is most valuable. Evaluations typical of such aneurysms by computer tomography (CT), magnetic resonance imaging (MRI) and angiography are shown in Figs. 1 to 3. As these aneurysms have large external diameters but small lumina, temporary vascular occlusion is often necessary while the aneurysm sac is opened to remove its clotted contents and define the neck prior to definitive clipping. It is often necessary when managing terminal carotid aneurysms to occlude the proximal middle cerebral and anterior cerebral arteries while manipulating the aneurysm. The closing pressure of the temporary clips used for this procedure should not exceed 80 gm to avoid damage to these vessels. When operating on terminal carotid aneurysms, we control circulatory flow proximally, ideally by temporary clipping of the carotid artery in the neck in case proximal control cannot be obtained in the head. Distal vascular control is also important to avoid exsanguination of the distal cerebral circulation, because this would lead to an unacceptable degree of distal circulatory hypotension. When both proximal and distal circulatory flow around the aneurysm are controlled, systemic hypotension is not necessary; one is seeking only to achieve focal hypotension within the operated field.

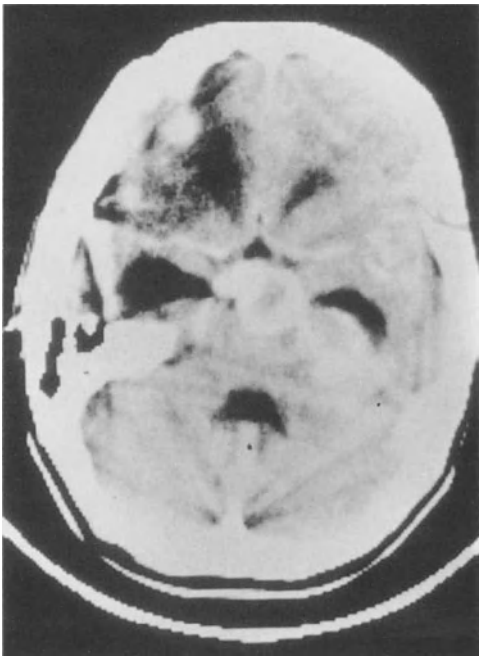

Fig. 1. CT scan showing giant basilar aneurysm

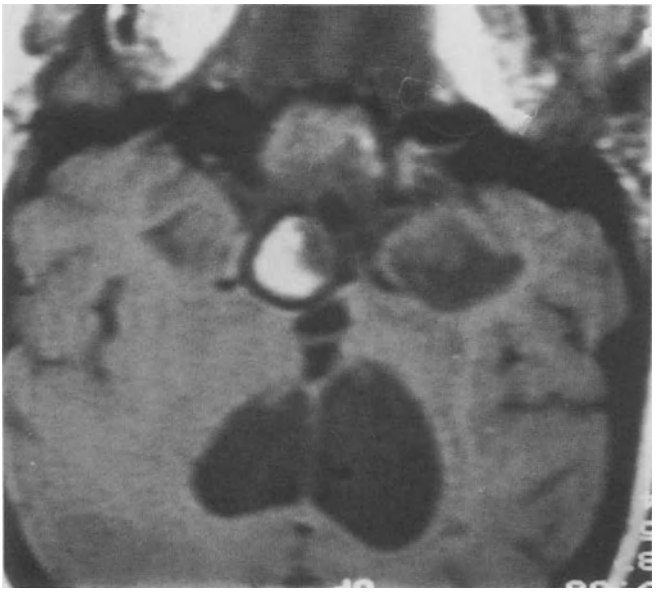

Fig. 2. MRI showing giant basilar aneurysm

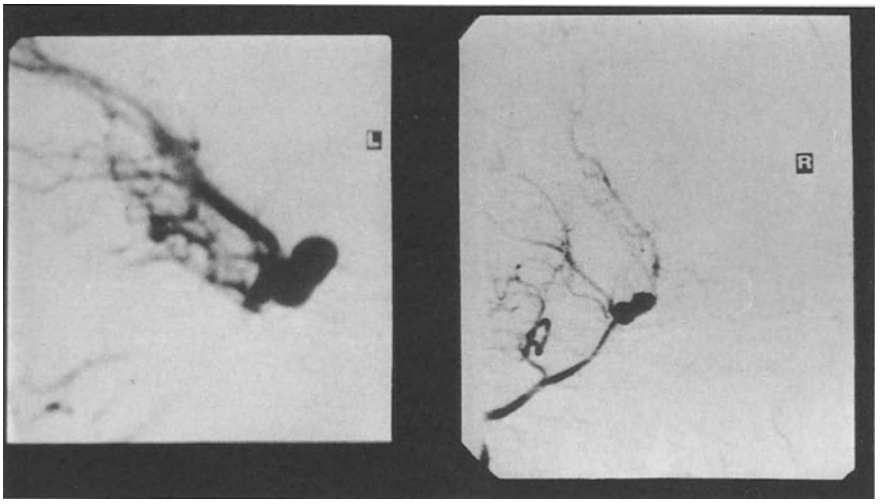

Fig. 3. Cerebral angiography demonstrating giant basilar aneurysm

One of our disappointments with SSEP monitoring has been with the results of its use during operations on anterior cerebral artery aneurysms (Symon and Murota 1989). Although one or two of our patients with anterior cerebral artery aneurysms showed median nerve SSEP slowing, presumably from involvement of Heubner's artery, we have found SSEPs to be quite unreliable indicators of anterior cerebral artery ischemia. We have been unable to approach Dr.

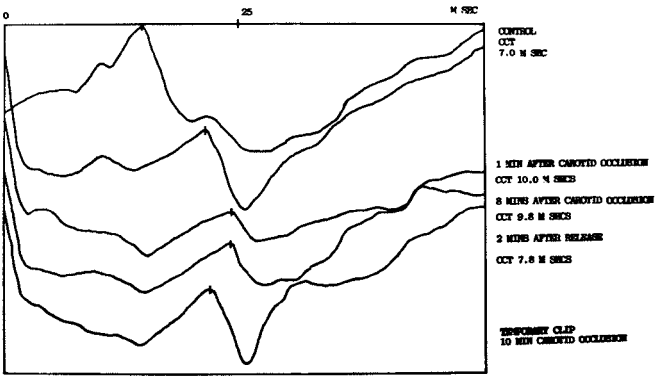

Fig. 4. SSEP recorded during an operation to manage a middle cerebral aneurysm. Temporary middle cerebral artery occlusion caused delay in N_{20} waveform and reduction in amplitude. These changes reversed within 10 minutes of reperfusion

Schramm's success rate of 60% (personal communication) with posterior tibial nerve stimulation. We have also had difficulty identifying from which hemisphere the SSEP is arising after a single anterior cerebral artery has been clipped.

Early in our study of SSEP monitoring during temporary vessel occlusion for aneurysm management we were able to compare the consequences of significant intraoperative changes in the SSEP with the patient's immediate and long-term postoperative clinical course (Momma et al. 1987). Figure 4 shows recordings from our first case in which there was considerable slowing of the SSEP intraoperatively. Ten minutes after temporary occlusion, the N_{20} peak became smaller and its latency increased. This reversed when temporary occlusion was discontinued and the patient awoke from anesthesia with no immediate deficit. Further, despite a postoperative course complicated by a small interhemispheric hematoma, the patient never developed a hemispheric defect. The lesson from this case being that preservation of the SSEP was a reasonable predictor of normal recovery. This case also demonstrates the advantages of using the results of recording from the nonsurgical hemisphere to assess the suppressant effects of anesthesia on electrophysiological parameters, because the effects of anesthesia are equal in both hemispheres. All anesthetic agents increase SSEP conduction time (Symon and Murota 1989), as do high concentrations of carbon dioxide (i.e., $PCO_2 > 50$) such as may occur with the resumption of spontaneous respiration at the end of surgery as practiced in our clinics.

Ideally one would like to use statistical analysis to predict outcome in terms of the effects on cerebral perfusion, as indicated by SSEP, of temporary vascular occlusion during aneurysm surgery. We have now recorded SSEPs from over 175 patients with aneurysms and have published results of managing 66 aneurysms by temporary occlusion versus 119 managed without occlusion (Jabre and Symon 1987). The outcome was the same in both groups, but statistical interpretation is blurred by the fact that temporary occlusion was only used in the more difficult cases. Despite this, the evoked potentials had disappeared in 20 of the patients in whom cerebral circulation was occluded.

Therefore one is left to draw from key cases. Figures 5 to 8 show results of preoperative studies, intraoperative recordings, and postoperative evaluation in

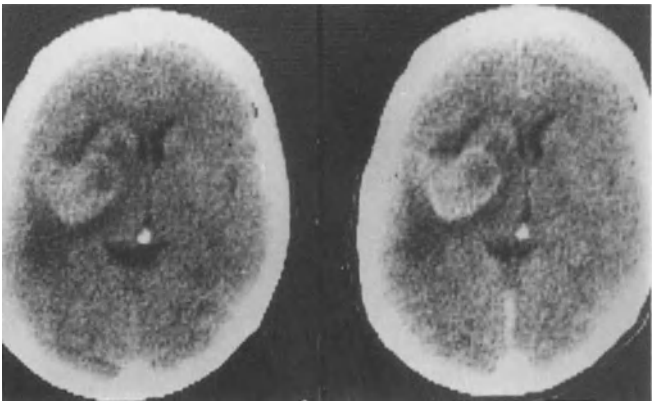

Fig. 5. This CT scan shows a giant middle cerebral artery aneurysm extending into the basal ganglia

such a key case. This patient, who presented with progressive deterioration of handwriting, was thought to have Parkinson's disease until computer tomography (CT) was performed and a giant aneurysm extending into the basal ganglia was identified (Fig. 5). Cerebral angiography was also performed to delineate the aneurysm (Fig. 6).

To clip the aneurysm successfully it was considered necessary to place proximal clips on MC 1 and all the MC axillary branches. The aneurysm sac needed to be opened so that a clot at the aneurysm's neck could be evacuated to permit a definitive clip to be positioned. During this time the lenticulostriate branches of the middle cerebral artery supplying the basal ganglia would have virtually no blood

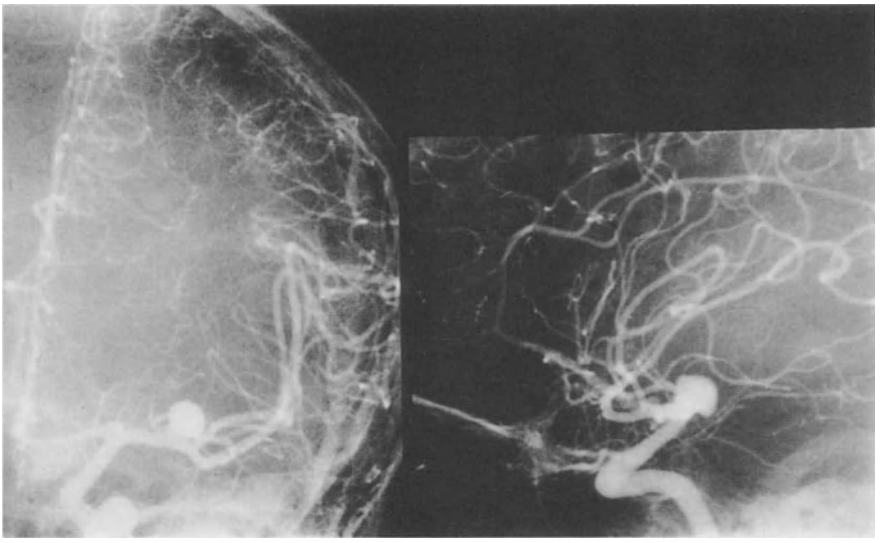

Fig. 6. In this cerebral angiogram of the same patient, the aneurysm sac appears at the bifurcation of the middle cerebral artery trunk

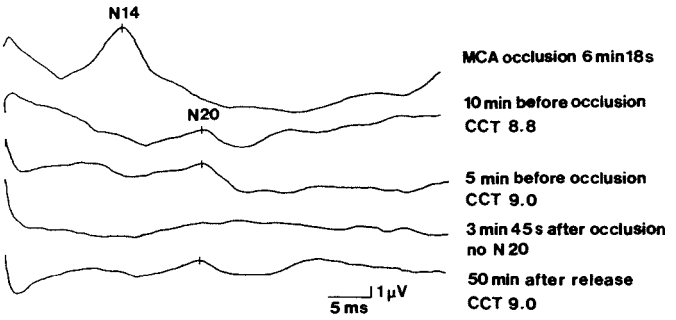

Fig. 7. Intraoperatively recorded SSEP of the same patient. The N_{20} response was lost 3 minutes 45 seconds after temporary middle cerebral artery occlusion. The N_{20} recovered 50 minutes after reperfusion

flow. In this area of poor collateral circulation, these manipulations resulted in blood flow falling below the critical rate of 20 ml/100 g/min. Figure 7 shows the SSEP recorded intraoperatively at this stage. There was rapid disappearance of the N_{20} with clipping – indeed within 2 minutes a flat trace resulted. Release of the temporary clips 6 minutes 30 seconds later (the time necessary to prepare and clip the aneurysm neck, Fig. 8) resulted in the gradual reappearance of the SSEP. Immediately postoperatively this patient was hemiplegic and aphasic, but within 2 hours was flexing the arm and leg on the affected side. Speech returned within 4 to 6 hours and full neurological recovery was apparent 48 hours postoperatively. Psychometric assessment 2 weeks after the operation established the patient's IQ as 140. Assessment of the SSEP recorded postoperatively showed that it took 5 days for the evoked response on the operative side to return to within 2 standard deviations of normal.

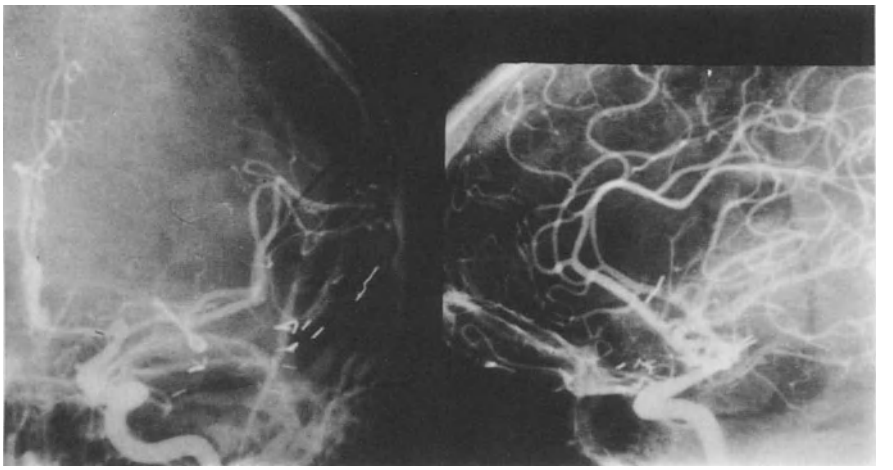

Fig. 8. Postoperative angiogram of the same patient showing successful occlusion of aneurysm neck

We have previously shown (Symon et al. 1981) that if the patient's SSEP returns to within 2 standard deviations of normal within 5 days after an operation for subarachnoid hemorrhage the patient is probably going to have reasonably good neurological function postoperatively. The critical point in the case of the patient just discussed is that 6½ minutes of temporary vascular occlusion and severe ischemia nearly caused a cerebral infarct. It took 48 hours for this patient to recover full neurological function, which is a good indication of what is the critical maximal period of temporary proximal middle cerebral artery occlusion. In this patient, it might have been safe to have continued clearing the aneurysm sac for a full 10 minutes, even though the evoked potentials had been lost. Nevertheless, we believe it vital to clear only enough of the clot within the aneurysm sac to identify the aneurysm neck and prepare it for definitive clipping.

In summary, it is our belief that if the SSEP returns within 10 minutes of temporary occlusion being released, then it is unlikely that a permanent deficit will occur as a result of the occlusion. Some patients will, however, have postoperative complications such as intracranial hematomas or vasospasm that cause neurological deterioration in the postoperative period. The most important intraoperative predictor of postoperative neurological function is the time interval between the onset of temporary clipping and loss of the SSEP. In our series of 22 patients (Momma et al. 1987) where the SSEP was lost intraoperatively, we noted that if the SSEP persisted after 3 minutes of temporary vascular occlusion of the internal carotid or middle cerebral artery and it was certain that the ischemic area was contributing to the loss of SSEP, a permanent neurological deficit never occurred. Loss of the SSEP within 3 minutes of temporary vascular occlusion does not inevitably result in a neurological deficit. Nevertheless, because the 3 patients who had permanent deficits all lost their SSEPs within 3 minutes of temporary vascular occlusion, we believe such loss a good predictor of poor postoperative neurological function.

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Somatosensory Evoked Potential Monitoring in Temporary Vascular Occlusion for Aneurysm Surgery under Moderate Hypothermia

A. Buchthal[1], M. Belopavlovic, and J. J. A. Mooij

Summary

Temporary vascular occlusion can be a useful technique for producing local hypotension to facilitate the dissection of technically difficult cerebral aneurysms. Hypothermia can be used to increase tolerance to occlusion, and cortical function in the territory of the occluded artery can be monitored during the occlusion using somatosensory evoked potentials (SSEP).

SSEP were monitored during temporary vascular occlusion for cerebral aneurysm surgery under moderate hypothermia in 55 patients: 25 with middle cerebral artery aneurysms, 10 with aneurysms of the internal carotid artery, and 20 with anterior communicating artery aneurysms. Tolerance to vascular occlusion at 28 to 30 °C, as manifested by the SSEP, varied widely, ranging from loss of the SSEP after 4 minutes of middle cerebral artery occlusion to preservation of the SSEP after 55 minutes of middle cerebral artery occlusion. Patients undergoing emergency surgery, those with a neurological deficit – either a transient deficit occuring at the time of subarachnoid haemorrhage or a persistant preoperative deficit – and those subjected to repeated vascular occlusion appeared to be prone to loss of SSEP during occlusion. The fact that at least 2 minutes of continuing occlusion after loss of the SSEP were tolerated in the pathway of the SSEP without permanent postoperative neurological deficit shows that changes in SSEP provide an effective early warning of ischaemic damage. On the other hand, severe ischaemia in areas not in the pathway of the SSEP, such as the internal capsule and Broca's area, could not be detected by monitoring median nerve or posterior tibial nerve SSEP.

We believe that moderate hypothermia has made a significant contribution to our patients' ability to tolerate relatively long cerebral artery occlusion times, as shown by the low postoperative morbidity in our cases, and that it deserves reappraisal for use in this situation.

Elective, temporary occlusion of the parent artery of an aneurysm has recently been used during aneurysm surgery in preference to prolonged or profound systemic arterial hypotension. Occlusion of the parent artery substitutes for the systemic hypotension a period of profound local hypotension, which may be needed

[1] Department of Anaesthetics, A.Z.G. Lenshoek Kliniek, Oostersingel 59, 9713 EZ Groningen, The Netherlands.

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Table 1. Hypothermia in cerebrovascular surgery – Benefits

Slack brain
Low heart rate
(decreased systemic arterial blood pressure, SABP)
Low metabolic rate
Reduced drug requirements
Increased arterial oxygen content ($C_a O_2$)
Increased tolerance to ischaemia

in some cases for technical reasons. Somatosensory evoked potentials (SSEP) can be used to monitor electrophysiological function of the territory of either the middle cerebral artery (MCA) or the anterior cerebral artery (ACA) and it is therefore useful to monitor SSEP during such occlusion.

At the Neurosurgical Clinic of the University Hospital in Groningen, we have used SSEP monitoring together with moderate hypothermia in 55 cases. Moderate hypothermia has been used in our Clinic for cerebrovascular surgery since Professor Beks introduced it 21 years ago. Core temperatures of 28 and 30 °C are achieved by surface cooling with water mattresses, assisted by pharmacologically induced vasodilatation. Table 1 shows some of the benefits such hypothermia offers. Metabolic rate is reduced to approximately one half at these temperatures.

Patients and Methods

Surgical Techniques

Vascular occlusion was elective in 55 cases, inadvertent in 2, and necessitated by haemorrhage in 3 cases (Table 2).

Thirteen patients underwent operation within 72 hours of subarachnoid haemorrhage (SAH). The operations were carried out through a trans-Sylvian approach with lumbar cerebrospinal fluid drainage and the use of microsurgical techniques. Dexamethasone cover was provided.

Table 2. Site of temporary occlusion in 55 cases

25 MCA	21 distal M1
	2 proximal M1
	1 inadvertent
10 ICA	
10 bilateral A1	
10 unilateral A1	

MCA, middle cerebral artery;
ICA, internal carotid artery;
MI, first segment of middle cerebral artery;
AI, first segment of anterior cerebral artery;

Anaesthesia

A standard pethidine-relaxant technique was used, following premedication with a lytic cocktail. Sodium nitroprusside was given as necessary to control systemic arterial blood pressure (SABP) and to promote peripheral vasodilatation to aid cooling. Haemodilution was achieved by administration of one half to one litre of 5% human albumin solution. Volatile anaesthetic agents other than nitrous oxide were never used. During the period of occlusion the SABP was raised to a high normal level in order to increase collateral perfusion. After the end of the operation, mechanical ventilation was continued until the patients' core temperature reached 36 °C.

Monitoring

SSEP in response to median nerve stimulation (MN-SSEP) were monitored in the MCA and internal carotid artery (ICA) occlusion cases, while responses to posterior tibial nerve stimulation (PTN-SSEP) were recorded when the ACA was involved. Nicolet CA 1000 equipment was used with the parameters shown in Table 3. Whenever possible the central conduction time (CCT), defined in the usual way, was used for evaluation and for comparing values in the two hemispheres. In most cases, it was possible to measure only sequential changes in one hemisphere during the occlusion and the number of responses averaged was reduced to 100 to 300 for the sake of rapid updating of the measurements. This could generally be done within 1 to 2 minutes.

A cervical peak could not usually be obtained during PTN stimulation. Further, bilateral A1 occlusion precludes comparison of an ischaemic with a nonischaemic hemisphere, so that evaluation then depends solely on sequential changes in the amplitude and latency of the first cortical wave (N_{70}). Nasopharyngeal, oesophageal, subcortical, and peripheral skin temperatures were monitored (Siemens).

Table 3. SSEP Parameters

		MN	PTN
Stimulus	Square wave	200 μs	25 μs
	Rate (regular)	3–7/s	3–4/s
Recording	Active	C_3', C_4'	C_z'
		C_{3-5}	C_7-T_2
	Reference	Linked mastoids	
	Time base	40 ms	120 ms
	Bandwidth	5–3000 Hz	
	Responses averaged	500	

MN, median nerve;
PTN, posterior tibial nerve

Results

MCA Cases

As may be seen from Table 4, there were 30 MCA occlusions in 25 cases; occlusion times ranged from 3 to 55 minutes, with 10 occlusions less than 10 minutes and 13 occlusions longer than 15 minutes. In all but 2 elective occlusions, the M1 segment was occluded about 5 mm proximal to its termination, distal to the origin of the proximal lateral striate arteries. In 2 cases the vessel had to be occluded more proximally. Occlusion was inadvertent in 2 cases, occurring during the approach to basilar artery aneurysms.

Loss of SSEP Intraoperatively

In 10 cases the N_{20} peak of the median nerve SSEP became unrecognizable during occlusions of 8 to 19 minutes. In all but 2 of these cases the patient 1) had a neurological deficit either immediately preoperatively or at the time of SAH, 2) was undergoing early surgery, 3) had evident vasospasm at operation, or 4) was undergoing a second or third occlusion. In the case of the 2 inadvertent occlusions, the SABP was lower than it would have been during an elective occlusion. Recordings of MN-SSEP from 1 of the 2 basilar artery aneurysm cases are shown in Figure 1. When the N_{20} disappeared, we first used a longer time base to search for it and then informed the surgeon that the peak was lost. The N_{20} reappeared within 9 minutes after the MCA was released.

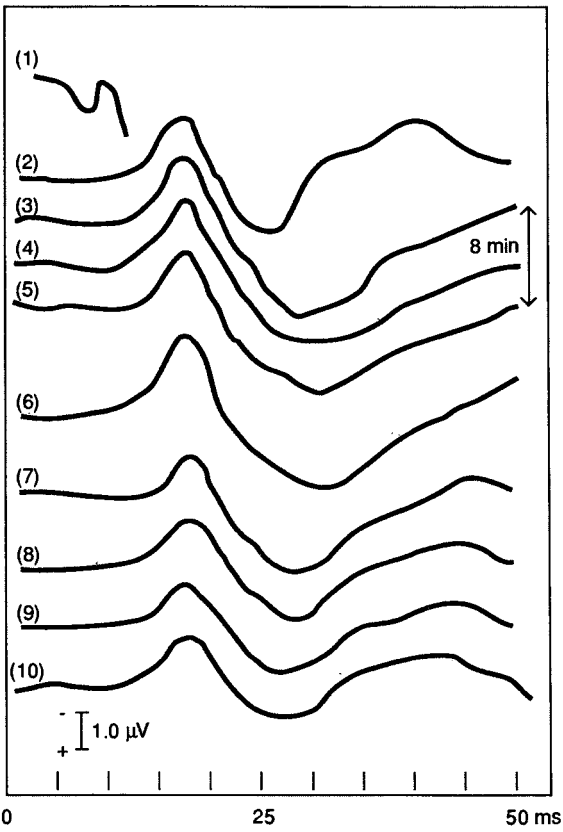
In 3 of the 10 patients in whom SSEP were lost during occlusion a new focal neurological deficit was present immediately postoperatively (Table 5). MCA occlusion lasted as long as 2 minutes after loss of the SSEP in 4 cases, and these patients showed no new deficit postoperatively. However, a new deficit was seen in 3 of 6 patients in whom occlusion lasted 4 minutes or more after loss of the SSEP.

One of these last 3 patients was in Hunt and Hess's Grade III preoperatively. The patient had experienced hemiparesis and dysphasia at the time of SAH and was left with some dysphasia that was evident preoperatively. Her dysphasia was exacerbated postoperatively, but it resolved completely within a year. This patient's intraoperative SSEP records are shown in Fig. 2. In this patient the MCA was occluded for about 15.8 minutes, with loss of the SSEP after about 7 minutes. An interhemispheric difference (IHD) of 4 ms in the CCT peristed at the end of the operation.

Table 4. 30 MCA occlusions in 25 cases

11 occlusions SSEP lost		
19 occlusions SSEP preserved:	{	3 at 50% amplitude
		4 CCT +> 2 ms
		12 CCT +< 2 ms

MCA, middle cerebral artery;
SSEP, somatosensory evoked potentials;
CCT, central conduction time



time	NP temp,°C	subdural temp,°C
16.25	29.0	27.5
16.34	28.8	27.3
16.38	28.8	27.2
16.42	28.8	26.4
MCA released		
16.45	28.6	26.8
17.07	28.8	
17.15	28.9	
17.20	29.1	
17.30	29.4	

Fig. 1. Intraoperative MN-SSEP recordings during the trans-Sylvian approach to a basilar artery aneurysm. Curve *1* was recorded over the upper cervical spine. The first cortical peak (N_{20}) seen at 41.2 ms in curve *2* is not seen in curves 3 to 6 due to compression of the middle cerebral artery by the retractor. It reappears in curve *7* at 46.8 ms, 22 minutes after release of the middle cerebral artery. All curves are on a 50-ms time base, except curve *6*, which is on a 60-ms time base. *NP temp,* nasopharyngeal temperature

Table 5. Tolerance to MCA occlusion: 10 cases MN-SSEP lost

No. of cases	Time to SSEP loss in minutes	Time SSEP absent in minutes	Sequelae
4	6–17	2	none
1	4	4.5	mild dysphasia 2 weeks
1	4	8	none
2	?	>10	none
1	7	9	increased dysphasia 1 year
1	6.5	4+11	paresis 24 hours

MCA, middle cerebral artery;
MN-SSEP; median nerve somatosensory evoked potentials;
SSEP, somatosensory evoked potentials

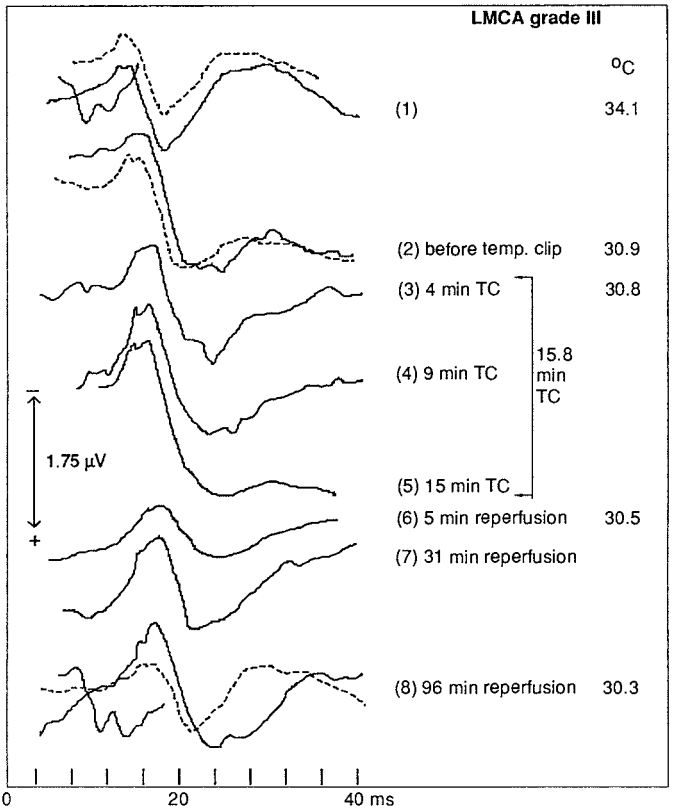


Fig. 2. Intraoperative MN-SSEP recordings in a patient undergoing removal of a left MCA aneurysm. *Solid lines* were recorded over the cervical spine and left cerebral hemisphere, *interrupted lines* over the right hemisphere. *TC*, temporary clip to left MCA. There is no clear N_{20} peak after 9 minutes' TC. An interhemispheric difference of 6 ms persists 96 minutes after removal of the TC. There was increased dysphasia postoperatively. Temperatures were measured nasopharyngeally

In the second patient who suffered neurological deficit after loss of SSEP, MCA occlusion continued for 4.5 minutes after loss of the SSEP. This was followed by a mild dysphasia lasting 2 weeks postoperatively. In the third case, marked vasospasm was evident at operation and the patient underwent proximal occlusion twice; occlusion was continued for 4 and 11 minutes after loss of the SSEP. This patient had a hemiparesis postoperatively that resolved within 24 hours.

SSEP Present Intraoperatively

In the other 15 MCA occlusion cases the SSEP were present throughout occlusions of up to 55 minutes (Table 6). In 3 of these patients the amplitude of the MN-

Table 6. 19 MCA occlusions in 15 cases: MN-SSEP preserved

No. of cases	SSEP results
3	amplitude 50%[a]
4	CCT +> 2 ms[a]
12	CCT +< 2 ms[a]

[a] Reocclusion.
[b] Increased dysphasia postoperative in 2 patients.

MCA, middle cerebral artery;
MN-SEP, median nerve somatosensory evoked potentials;
SSEP, somatosensory evoked potentials;
CCT, central conduction time

SSEP was reduced by more than 50% during occlusions of 4, 13, and 52 minutes, the last being a second occlusion. In 4 cases there was an increase in the CCT of more than 2 ms during the occlusion; none of these patients showed any postoperative deficit. In 8 cases the IHD was less than 2 ms during the occlusion. In one of these cases there were minimal or no changes in the CCT during an occlusion of 3 minutes, and in another of these cases there were minimal or no CCT changes during 3 occlusions totalling 8 minutes. Both of these patients had dysphasia postoperatively: in the first the dysphasia was new and in the second it was exacerbated even though the former was an acute case and in the latter, occlusion was employed only after rupture of the aneurysm.

ICA Cases

In 10 cases the ICA was temporarily occluded either intracranially or in the neck for 3 minutes to 3 hours (Table 7). In 1 case this was accompanied by loss of the MN-SSEP after 4 minutes' occlusion; reperfusion was instituted about 1 minute later and no deficit was noted postoperatively. A postoperative hemiparesis lasting 2 to 4 hours and responding well to intravascular volume therapy was seen in 1 of the early operation cases after an 8-minute occlusion, during which the N_{20}

Table 7. 10 ICA occlusion cases

No. of cases	Duration TC in minutes	SSEP	Sequelae
1	4.7	N_{20} lost at 4 min	none
1	8	N_{20} lost; persistent 1.2 ms CCT increase	paresis 4 hours
4	7–57	>1 ms CCT increase	none
3	5.5–180	no change	aphasia, hemiplegia

ICA, internal carotid artery;
TC, temporary clip;
SSEP, somatosensory evoked potentials;
CCT, central conduction time

Table 8. 20 cases of A1 occlusion

Occlusion:	*10 Unilateral*		*10 Bilateral*	
	No. of cases	Results	No. of cases	Results
	2	SSEP lost	3	SSEP lost
	1	amplitude <50%	2	amplitude <50%
	7	no change	3	no change

A1, first segment of anterior cerebral artery;
SSEP, somatosensory evoked potentials

of the MN-SSEP became poorly defined, and an IHD of 1.2 ms persisted after reperfusion. In another case no SSEP changes were detected during or after 3 ICA occlusions of 5, 2, and 1 minute, but this was followed by a severe hemiparesis and aphasia. A computerized tomography (CT) scan later showed infarction in the posterior limb of the internal capsule in this patient.

Anterior Communicating Artery (ACoA) Aneurysm Cases

The results in these cases are shown in Table 8.

Unilateral A1 Occlusion

In 10 ACoA aneurysm cases, unilateral A1 occlusion was performed. Occlusion times ranged from 2 to 97 minutes, with occlusions longer than 15 minutes in 4 cases. The N_{70} peak was lost in 2 of these cases: in 1 early surgery after 5 minutes of occlusion and after 24 minutes in another case. The occlusion continued for an additional 4 minutes in each case and neither patient showed a postoperative deficit. Reduction in the amplitude of the N_{70} wave of the PTN-SSEP to less than 50% during 9 minutes' occlusion in another early operation case also resulted in no postoperative sequelae.

In another patient, internal capsule infarction occurred after 13.56 minutes' unilateral A1 occlusion; no changes were detected in either the MN-SSEP or the PTN-SSEP. This patient had a second aneurysm located close to the anterior choroidal artery, and this aneurysm could not be clipped.

Bilateral A1 Occlusion

Bilateral A1 occlusion was employed in 10 cases, with occlusion lasting between 5.75 and 45 minutes, and longer than 15 minutes in 7 cases. The N_{70} peak was lost in 3 cases. In one of these 3 cases, in which the operation was performed as an emergency, the occlusion lasted 6 minutes. In another, 6 minutes of bilateral A1 occlusion followed 28 minutes of unilateral A1 occlusion. Recordings from the third case are shown in Fig. 3. No permanent postoperative sequelae were noted in these cases, although a paresis lasting about 6 hours was seen in one case in which the amplitude of the N_{70} wave was reduced to less than one half during the last of three occlusions that lasted 13.8, 7, and 5.75 minutes respectively.

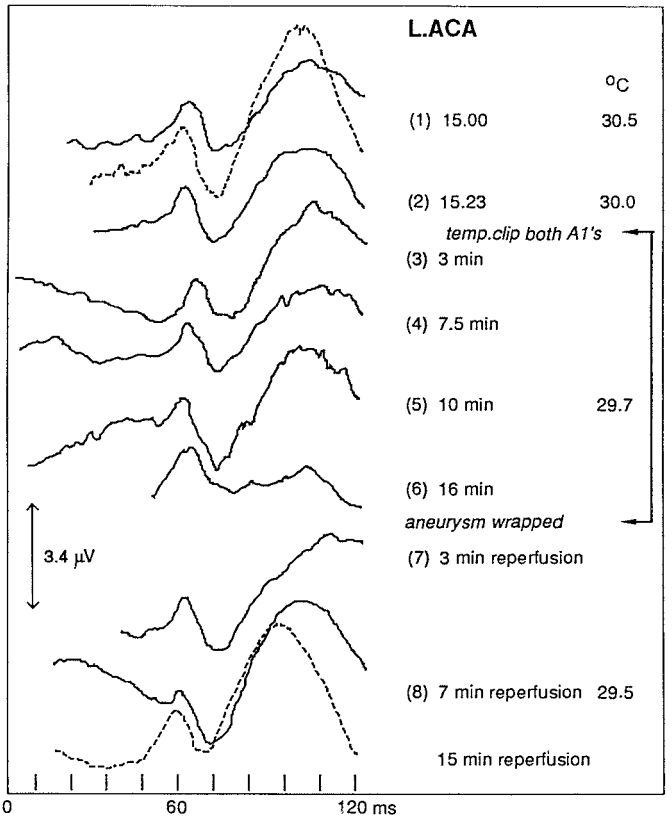


Fig. 3. Intraoperative PTN-SSEP recordings in an ACoA aneurysm case. Solid lines were recorded following left PTN stimulation, and interrupted lines following right PTN stimulation. Only the right hemisphere was monitored sequentially during occlusion of both A1s. The cortical wave was reduced to less than half its pre-clip amplitude after 16 minutes' occlusion, but recovered within 7 minutes of reperfusion. Temperatures were measured nasopharyngeally

Discussion

In reviewing these results, we note that in the majority of cases in which SSEP were lost during occlusion or the amplitude was reduced to less than one half, the patients had 1) undergone early surgery or 2) had a focal neurological deficit either immediately preoperatively or at the time of SAH, or 3) had repeated occlusion, or 4) had a combination of these factors (Table 9). All of these factors indicate some degree of ischaemia existing prior to the occlusion, and previous ischaemia is known to exacerbate the effects of a subsequent ischaemic episode. Thus, patients who have had a previous episode of ischaemia seem to be prone to loss of SSEP during occlusion and are likely to be more susceptible to ischaemic

Table 9. Loss of SSEP during occlusion

Site of occlusion	MCA	ICA	A1
	No. of cases	No. of cases	No. of cases
Total with SSEP loss	10	2	5
Predisposing factors:			
Early surgery	2		3
Deficit at SAH	3	1	1
Preoperative deficit	1		
Reocclusion	1		2
Inadvertent occlusion	2		
None	2	1	1

SSEP, somatosensory evoked potentials;
MCA, middle cerebral artery;
ICA, internal carotid artery;
A1, first segment of anterior cerebral artery;
SAH, subarachnoid haemorrhage

damage than are other cases. We now try to avoid temporary occlusion in early operations.

As to whether these results provide any indication that moderate hypothermia is of value in temporary vascular occlusion, our results show no obvious, simple correlation between clinical outcome and occlusion time. There was an extremely wide variation in tolerance to MCA occlusion, ranging from loss of MN-SSEP within 4 minutes of occlusion to preservation of MN-SSEP with little or no change after 55 minutes of occlusion. This is not surprising, considering that clinical studies have shown a wide range of variation in collateral blood supply to this area. In the absence of independent measurements of local blood flow in each case in our study, normal variations in collateral perfusion could easily account for a two-fold increase in tolerance to ischaemia, such as might be expected during moderate hypothermia. However, the SSEP changes we saw during occlusion and their correlation with new, postoperative neurological deficits do differ somewhat from SSEP and deficits which have been reported in normothermic patients.

In 4 of our patients relatively short periods of occlusion were accompanied by no detectable changes in the MN-SSEP or PTN-SSEP and yet were followed by focal neurological deficits. However, in none of these cases was there damage actually within the anatomical pathway of the SSEP: 2 of the 4 patients had internal capsule infarctions and 3 of the 4 developed dysphasia. Failure to detect SSEP changes in these latter 3 cases emphasises 1) the potential for significant inhomogeneity within the territory of a single cerebral artery such as the MCA, and that 2) SSEP provides information strictly limited to the specific pathway being monitored. Other monitoring techniques, perhaps electroencephalography (EEG), will need to be used in order to detect ischaemia intraoperatively in other areas.

Putting aside these 4 cases then, we can now look at the SSEP changes associated with the immediate, new, postoperative deficits seen in the other 51 cases in this study. A decrease in the amplitude of the first cortical peak of the

SSEP correlated well with neurological deficits resulting from brain retraction in a study in dogs (Bennett et al. 1977) and in a study of intraoperative brainstem auditory evoked potential (BAEP) monitoring (Schramm et al. 1988). Such a decrease was seen during occlusion in 7 of our cases, but in only 1 was a short-lasting paresis seen postoperatively, following 3 periods of bilateral A1 occlusion totalling 27 minutes.

MN-SSEP were lost in 10 MCA occlusion cases in our study (Table 5). No sequelae were noted when occlusion lasted no longer than 2 to 4 minutes after loss of the SSEP, and in 1 case with no preceding SAH as much as 8 minutes of occlusion after loss of the SSEP were tolerated without clinical sequelae. Occlusion lasted longer than 4 minutes after loss of the SSEP in 6 cases in our study and was followed by a postoperative deficit in 3 of these 6 cases: one of these patients had undergone earlier surgery, one was in grade III preoperatively, and one had a repeated occlusion in the presence of evident vasospasm. Similarly, in the 5 patients with A1 occlusions in whom SSEP were lost, there were no postoperative deficits: 4 minutes of occlusion after loss of the SSEP were tolerated in 3 cases, 2 minutes in 1 case, and about 1 minute in the last case.

These observations differ from those reported by Kidooka et al. (1987) for normothermic patients. These investigators found that prolongation of the CCT during vascular occlusion by more than twice its standard deviation, which might be considered abnormal on statistical grounds, was followed by a new postoperative neurological deficit in 8 of their 13 cases. Symon et al. (1984) found that prolongation of the CCT by more than 10 ms was likely to be associated with postoperative deficit, while Momma et al. (1987) regard disappearance of the cortical response within 3 to 4 minutes of ICA or MCA occlusion as likely to be followed by a permanent neurological deficit.

Conclusions

We conclude from the results of our present study that both MN-SSEP and PTN-SSEP are sensitive enough to provide early warning of significant ischaemia in the territories of the MCA and ACA, respectively, but that this sensitivity is limited strictly to the anatomic areas comprising the respective pathway.

Loss of SSEP during vascular occlusion occurs more readily in patients in grade III, those undergoing emergency surgery, during a repeat occlusion, or those who experienced a focal neurological deficit at the time of SAH.

At least 2 minutes of continuing occlusion after loss of the SSEP are tolerated by structures belonging to the pathway of that SSEP when core body temperature is maintained at 28 to 30 °C. A reduction in amplitude of the first cortical wave of more than 50% or an IHD persisting after reperfusion may indicate significant ischaemia.

In the absence of independent means of quantifying collateral perfusion during vascular occlusion, these results suggest that the use of hypothermia allows for longer periods of occlusion with greater safety when appropriate SSEP monitoring is also used than is the case for normothermia.

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Monitoring in Acoustic Neuroma Operations

Intraoperative Brainstem Auditory Evoked Potential (BAEP) Monitoring in Acoustic Neuroma Surgery

C. FISCHER[1] and G. FISCHER[2]

Summary

In a cohort of 260 patients with acoustic neuroma operated upon via the suboccipital approach, 61 were not deaf prior to surgery. Hearing preservation had been attempted in the 61 patients, regardless of the level of hearing and the size of the neuroma. Hearing was preserved in 19 of the 61 patients with acoustic neuromas, 8 of which had neuromas larger than 2 cm. Brainstem auditory evoked potentials (BAEP) were monitored throughout the operation in all 61 patients. This monitoring proved helpful in preserving hearing in these patients because the neurophysiologist was able to inform the surgeon immediately when a specific surgical manipulation was affecting and possibly damaging cochlear function.

Introduction

Intraoperative brainstem auditory evoked potential (BAEP) monitoring permits the recording of activity in the cochlear nerve and the ascending auditory pathways of the brainstem during surgery, thus allowing continuous evaluation of the functional status of the whole cochlear nerve and brainstem auditory system.

BAEP may be monitored in different ways, depending upon the objectives of the particular operation being performed. Monitoring the functional condition of the brainstem during operations to remove tumors, or during operations on posterior blood vessels, or monitoring the condition of the auditory nerve in acoustic neuroma operations may each be chosen or ruled out for one reason or another.

The actual objective of BAEP monitoring in acoustic neuroma operations is to reduce the risk of damage to the auditory nerve and to try to preserve some hearing function. When we started monitoring intraoperatively, we had been accustomed for years to recording in the laboratory all types of sensory evoked potentials, and intraoperative monitoring appeared to us as something new, with its own dynamics. We first had to learn the significance of the BAEP changes that we were recording, and then we had to learn to provide appropriate warning to the neurosurgeon and at the appropriate time.

[1] Service de Neurologie fonctionnelle Hôpital Neurologique, 59 Boulevard Pinel, F-69003 Lyon, France.
[2] Service de Neurochirurgie Hôpital Neurologique et Faculté Alexis-Carrel Lyon, France.

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Patients and Methods

From the time we began to monitor patients intraoperatively until March 1989, we performed BAEP monitoring during removal of 61 acoustic neuromas in 61 patients. None of the patients had been deaf prior to the operation, although their hearing levels and speech discrimination scores varied. In the same period of time, 260 neuromas were removed through the suboccipital approach. According to Koos' classification, the size of the neuroma in the 61 patients was as follows: grade I (intracanalicular), 2 patients; grade II (between 1 and 2 cm), 16 patients; grade III (between 2 and 3 cm), 23 patients; grade IV (>3 cm), 20 patients.

BAEP were recorded in all patients in the week before the operation. In all 61 patients, BAEP were abnormal with a delay of the I–V interval. We do not monitor BAEP intraoperatively when only wave I remains preoperatively or when all waves have been lost on the side of the neuroma. For recording of BAEP electrodes are placed at the frontal midline (Fz) and the earlobe ipsilateral to the stimulated ear (100 μs duration clicks), and the repetition frequency does not exceed 20 Hz. Most of our recordings have been made using analog on-line filtering (bandpass 160 to 1600 Hz). A minimum of 1500 trials are averaged. In 14 of the 61 intraoperative recording sessions, a system with adaptative digital filtering was used (Bertrand et al. 1987). This system allows automatic detection of peaks. When necessary, compound action potentials (CAP) were recorded from the eighth nerve using a sterilized Teflon-coated wire placed directly on the nerve by the surgeon. (For more details, see Fischer 1989.)

Results

Results are summarized in Table 1. Fig. 1 shows examples of recording during monitoring in a patient whose hearing was preserved.

Hearing was preserved in 2 patients (100%) with intracanalicular neuromas, in 9 of 16 patients (56%) with grade II neuromas, in 7 of 23 patients (30%) with grade III neuromas, and in only 1 of 20 patients (5%) with large neuromas (grade IV).

Hearing Loss

With time and the monitoring of increasing numbers of cases, we have learned about mechanisms of hearing loss during acoustic neuroma operations, because hearing was lost in 42 of the 61 patients who underwent operations with monitoring of BAEP. In none of these patients was peak V present at the end of the operation. Seven of them still had peak I at the end of the operation, but as soon as they had awaken from anesthesia they appeared to be deaf, and peak I had disappeared by the time BAEP were recorded again a few days later.

Some identified mechanisms of BAEP loss (and subsequent hearing loss) are listed in Table 2. For 6 of the patients whose hearing was lost, progressive loss of BAEP began immediately after the dura was opened, and even though no retraction of the cerebellum was done, loss of BAEP was complete in less than one

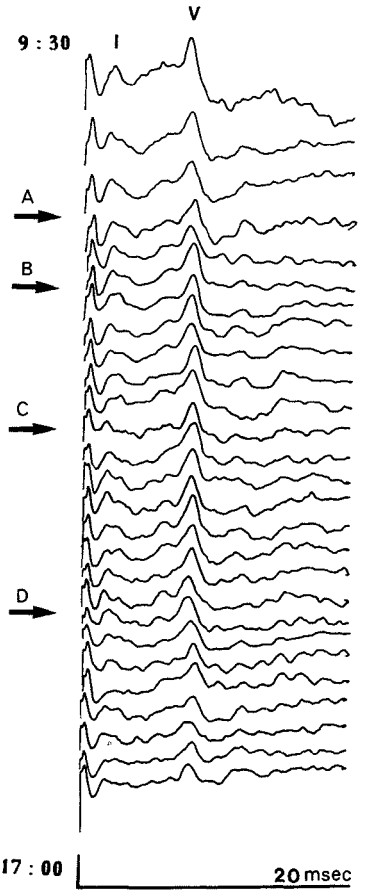

Fig. 1. Preservation of BAEP and hearing during removal of a grade II neuroma. 9:30 Baseline: Waves I and V are present, wave V is delayed. *A*, dura open; *B*, retractor on; *C*, end of drilling of the internal auditory canal; *D* removal of tumor in the lateral-most portion of the auditory canal. At the end of the operation wave V was preserved. Calibration 0.2 μV, positivity upwards

Table 1. Results of monitoring of BAEP in 61 patients during removal of acoustic neuromas. Hearing was preserved in 19 cases and lost in 42 cases. The neuromas were classified acccording to size (Koos) as: *I*, intracanalicular; *II*,1 to 2 cm; *III*, 2 to 3 cm; *IV*, > 3 cm.

Tumor Size	Total No. of Patients	Patients With: Hearing Preservation	Deafness
I	2	2 1 normal 1 serviceable	0
II	16	9 1 subnormal 4 serviceable 4 poor	7
III	23	7 1 subnormal	16
IV	20	1 1 poor	19

Table 2. Findings on BAEP monitoring during removal of 61 acoustic neuromas: cases with hearing loss

Patients with hearing loss: 42/61 0/42: peak V preserved 7/42: peak I preserved	
Some identified mechansms of BAEP loss:	
tumor vessel coagulation:	5
dura opening:	6
cerebellum retraction:	2
within internal auditory canal:	9
volume reduction:	9

Table 3. Findings on BAEP monitoring during removal of 61 acoustic neuromas: cases with hearing preservation

Patients with hearing preservation: 19/61	
13/19:	peak I and peak V preserved
3/19:	peak I only preserved
2/19:	total obliteration in the last 30 minutes
1/19:	no change

Mean latency increase: 0.8 ± 0.3 ms

hour. Loss of BAEP in these patients was not related to the operation itself; it might have been related to a change in the pressure balance in the cerebellopontine angle, leading to a stretching of the labyrinthine artery.

Hearing Preservation

We were able to preserve hearing in 19 of 61 patients (31%) who underwent BAEP monitoring during removal of a neuroma (Tables 2, 3). All patients who still had wave V at the end of the operation had some hearing preserved postoperatively. Three patients who had wave I postoperatively retained some hearing. Because 7 patients in whom wave I was preserved lost all hearing as a result of the operation, preservation of peak I does not seem to guarantee hearing preservation.

Discussion

Conduction of Monitoring

The utility of monitoring is greater when one has acquired more experience in using the technique for given indications, such as for acoustic neuroma operations. Those performing intraoperative monitoring must learn the significance of the BAEP changes that may be observed throughout the monitoring period, and must try to identify when a change warrants warning the surgeon, and the correct timing of this warning.

BAEP may change because of factors other than the surgical procedure, in particular the patient's temperature and pharmacologic agents. As far as we know, the anesthetic agents typically used for acoustic neuroma operations have little effect on intraoperative BAEP. But we do not have sufficient knowledge of the effects of drugs used during anesthesia to be certain that they do not affect BAEP, and we do not clearly know to what extent the effects of drugs on the BAEP depend on the functional status of the brainstem. Numerous factors beyond our control during such operations may have a cumulative effect on BAEP: drugs, changes in pressure in the middle ear, the core body temperature, and, above all, the local temperature, because the site for acoustic neuroma operations is very close to the generators of the evoked potentials which are being monitored. For all these reasons, a latency increase of as much as 1 ms from the beginning until the end of acoustic neuroma removal via the suboccipital route is often seen.

Some changes are related to the actual surgical procedure. When large neuromas are removed, the evoked response may disappear gradually, beginning immediately after the operation on the dura and before the actual removal of the neuroma has started. The mechanism for this observed disappearance could be disruption of pressure balance in the cerebellopontine angle. More related to the operation itself are the BAEP changes that may be seen after the retractor has been placed on the cerebellum. We now perform a systematic retraction test, that may lead to repositioning of the retractor by the surgeon, when the latency of wave V has increased by more than 0.5 ms for 10 minutes. In any case, retraction is performed as gently and moderately as possible. A warning is given to the surgeon as soon as a significant decrease in wave V amplitude or a latency shift is noted on the oscilloscope, so that the surgeon can stop temporarily the action that precipitated the BAEP change. The surgeon is notified when the BAEP amplitude or latency starts to return toward baseline, so that the operation can continue. Any sudden reduction in amplitude during dissection or debulking of the neuroma is signalled to the surgeon, who temporarily discontinues the activity that caused the amplitude change or moves dissection to another area of the tumor.

After the dura has been opened, a retractor is positioned and then adjusted if necessary according to the retractor test. After the dura has been opened and the retractor is positioned, the wall of the internal auditory canal is drilled to lessen stretching of the eighth cranial nerve. We prefer to drill the auditory canal wall in a number of very short sessions rather than in one long session. We first debulk the tumor, and then we dissect the tumor away from the nerve itself, using mostly a blunt hook and scissors. Bipolar coagulation in the tumor bed is avoided as much as possible, and when coagulation is performed it is very brief. The most crucial period is during removal of the tumor in the lateral-most portion of the auditory canal. A warning is expected from the neurophysiologist as soon as a significant decrease in amplitude or a latency shift is observed on the oscilloscope, so that the surgeon can stop activity temporarily, until the BAEP amplitude or latency starts to return toward baseline. Any sudden reduction in amplitude during dissection or debulking of the neuroma is also expected to be signalled to the surgeon, so that the surgeon can temporarily suspend or change activity related to tumor dissection (Watanabe et al. 1989).

From performing BAEP monitoring during acoustic neuroma operations, we have learned some other practical points:

1) Coagulation of the Dandy vein is not dangerous, but actually not always necessary in the procedure.

2) Also, it is not dangerous to coagulate the numerous anastomotic arteries between the dura and the tumor.

3) Because in 3 patients the BAEP disappeared within a few minutes after an intratumoral artery was injured by use of the cavitron (CUSA), use of CUSA in this type of operation has been limited. We do not use it any more when hearing preservation is attempted, especially during removal of small neuromas. Conversely, CUSA remains useful when the patient was already deaf before being operated upon.

Concluding Remarks

In summary, all patients in our study who still had wave V at the end of the operation retained their hearing, and all patients in whom hearing was preserved had at least peak I at the end of the operation. But the persistence of wave I is not a guarantee of hearing preservation.

We have used the results of intraoperative BAEP monitoring to identify steps in the surgical procedure to remove acoustic neuromas that place cochlear function at risk: placement of the retractor on the cerebellum, prolonged coagulation, removal of tumor in the auditory canal, and, above all, removal of tumor in the lateral-most portion of the auditory canal.

Further progress in hearing preservation in patients with acoustic neuromas requires early diagnosis of the condition, adequate removal surgically, and intraoperative BAEP monitoring by a trained neurophysiologist. Evoked potential monitoring may help to improve patients' safety in a general way. BAEP monitoring helps to preserve hearing during operations to remove acoustic neuromas provided that the neurophysiologist: 1) has a good understanding of the surgical steps involved in acoustic neuroma removal; 2) has learned the meaning of BAEP changes that may be recorded in that kind of operation; and 3) gives only the right warning to the surgeon and only at the right moment.

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Monitoring Auditory Evoked Potentials During Cerebellopontine Angle Tumor Surgery: Relative Value of Electrocochleography, Brainstem Auditory Evoked Potentials, and Cerebellopontine Angle Recordings

Robert A. LEVINE[1]

Summary

Recording short-latency auditory evoked potentials continuously throughout cerebellopontine angle (CPA) tumor surgery with electrocochleography (ECochG) and brainstem auditory evoked potentials (BAEP) monitors the status of the cochlea, auditory nerve peripheral to the tumor, and neural activity central to the tumor. ECochG is a nearfield potential and provides rapid feedback, whereas BAEP are farfield potentials and their feedback is slower. Whenever the later components (wave V) of the BAEP are detectable, an electrode positioned within the CPA can usually record a negative potential due to neural activity central to the tumor. This recording technique will often detect a positive component from the generator of N_1, but no cochlear potentials. The nearfield negative potential can provide much more rapid feedback than wave V, but it has the same predictive value for hearing as wave V: when present it predicts useful hearing, but when undetectable hearing outcome cannot be predicted. By contrast, when neural activity is present in the ECochG, hearing outcome cannot be predicted, but when it is undetectable, hearing is always lost. There is a fundamental limitation of electrophysiological monitoring: when neural activity can be detected peripheral to the tumor but not central to the tumor, the electrophysiological data cannot predict postoperative hearing.

Hearing loss is the major morbidity that occurs most frequently as a consequence of acoustic neuroma surgery. Yet, of the methods currently available for treating these benign neoplasms, surgical excision is the most effective means of preserving hearing while controlling tumor growth (Nadol et al. 1987). The effort to preserve hearing during operations to treat acoustic neuromas at first was hindered by the fact that the surgeon had no idea of the status of the patient's hearing throughout the procedure; only after the patient awoke from anesthesia could the patient's hearing ability be assessed. In an effort to improve this situation, the technique of monitoring auditory evoked potentials throughout surgery was developed (Levine et al. 1978).

[1] Department of Neuro-Otology, Eaton-Peabody Laboratory, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, MA 02114, USA.

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Of all the auditory evoked responses that may be recorded, the short-latency (<10 ms) potentials are the most useful for monitoring during surgery, because they change little when the patient is under anesthesia (Levine 1990). Initial attempts to monitor hearing during surgery by recording auditory evoked responses made use of farfield potentials, known as brainstem auditory evoked potentials (BAEP), because these potentials can be monitored noninvasively. Soon it became apparent that, although monitoring BAEP can provide feedback about the patient's hearing during surgery, at the same time use of these potentials has some major limitations. For instance, many times BAEP are undetectable, even when the patient has "useful" hearing. Moreover, when present, the BAEP's small size limits the rapidity with which feedback can be obtained to tens of seconds or even to minutes. Because nearfield potentials have larger amplitudes, and thus can be evaluated with less delay, techniques have been developed to monitor these potentials during acoustic neuroma operations.

Two basic approaches have been used to record nearfield (i.e. close to the sites where auditory evoked potentials are presumed to be generated). One approach is to place a recording electrode near the cochlea (hence, the name electrocochleogram, or ECochG, for recordings of these potentials); the other technique is to place an electrode in the cerebellopontine angle (CPA), somewhere in the vicinity of the intracranial portion of the auditory nerve, once the tumor has been exposed. This latter technique has been referred to as "direct auditory nerve recording" (Silverstein et al. 1986). Because it is not always possible to place the recording electrode directly on the auditory nerve, and, as will be shown, not all the potentials are generated "directly" or locally from the point of contact, these nearfield recordings are referred to in this paper as "cerebellopontine angle (CPA) recordings."

In this paper we compare intraoperative recordings of BAEP, ECochG, and from the CPA, with respect to their predictive value in assessing hearing and their ability to provide rapid feedback to the surgical team.

Methods

A block diagram of the current system we now use for intraoperative monitoring is shown in Fig. 1. Because we have been concerned that recording in the CPA could itself lead to morbidity, the only recording we have performed continuously throughout surgery has been of BAEP and ECochG; we have recorded from the CPA either just after tumor exposure and at the very beginning of any dissection, or immediately after removal of the tumor has been completed.

A detailed description of our technique has been published previously (Levine 1988). In brief, clicks were delivered at 29 per second to an ER-3A earphone, which introduced a 1-ms delay between the occurrence of the electrical pulse that generated the click and the arrival of the click at the tympanic membrane (this delay separated any electrical stimulus artifact from the physiological responses). The waveform of the sound pressure in the external ear canal was monitored with a miniature microphone attached to one end of a 3-inch tube, the other end of

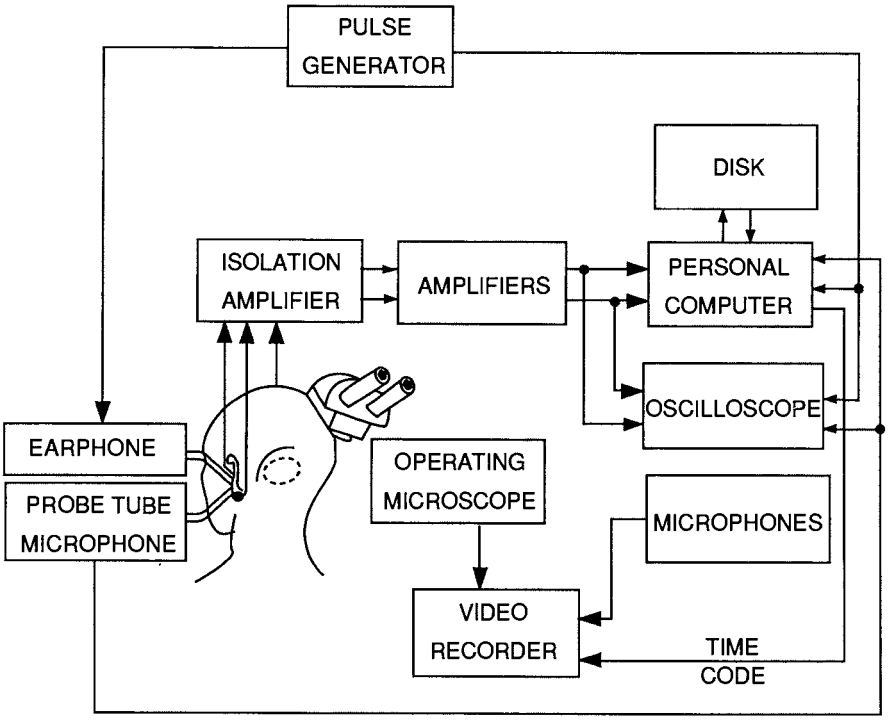

Fig. 1. Block diagram of system now in use for intraoperative monitoring

which was threaded (along with the tubing from the ER-3A earphone) through the middle of a sponge-type earplug. Broadband noise at 40 dB below the click level was presented through a second earphone to the contralateral ear to mask that ear and prevent contamination of our recordings with responses due to acoustic crosstalk. Click sounds were generally presented at 80 dB HL and the polarity was chosen to facilitate distinction between the cochlear and neural components of the ECochG. The duration of the pulse generating the click was generally 100 μsec.

The bandwidth of the recordings was generally 30 to 3000 Hz for ECochG and CPA recordings, and 75 to 3000 Hz for BAEP recordings. In addition, the BAEP were digitally lowpass filtered at 850 Hz. For ECochG, a transtympanic promontory electrode (a 3-inch long needle, Teflon-coated except at its tip) served as the active electrode and an electrode placed on the earlobe served as the reference electrode. For recording BAEP, input from the electrode on the earlobe was used as the negative input to the differential amplifier, with positive input coming from a vertex electrode. The intracranial recordings were made with a 0.3-mm silver ball as the active electrode and a clip on an exposed scalp muscle as the reference electrode. The position of the silver ball depended upon the exposure and it was not always possible to place the metal ball electrode on the auditory nerve. In general, we recorded from several positions in the CPA.

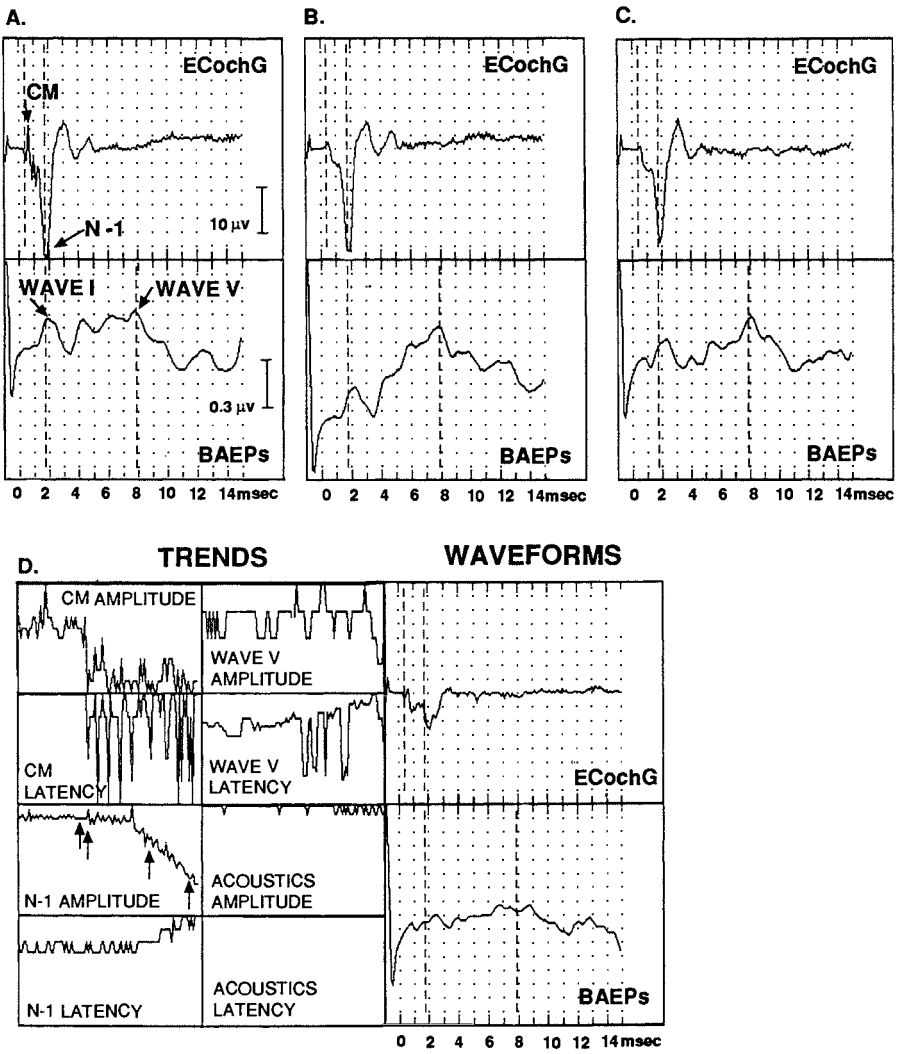

Fig. 2. Four sets of recordings from one patient undergoing removal of a 1.3-cm acoustic neuroma. The ECochG results, shown in the top half of each set, and BAEP, shown in the bottom half and obtained by exponential averaging, were recorded simultaneously. Panel D shows how our computer screen appears on-line. In addition to the waveforms, it includes the on-line trends for the amplitudes and latencies of the ECochG components, cochlear microphonic *(CM),* and compound action potential (CAP) of the auditory nerve *(N_1);* the BAEP component, wave V; and the first peak of the acoustic probe that measured the sound pressure in the external auditory canal (waveform not shown). Each time averaging is completed, the corresponding trend value is updated. The 4 arrows on the N_1 amplitude trend indicate points corresponding to the waveforms from panels A, B, C, and D, respectively. The BAEP were averaged over about 2 minutes, as compared to 7 seconds for the ECochG. A sudden loss of CM followed later by loss of N_1 can be seen. No recovery occurred. Postoperatively the patient was deaf in this ear. Stimulus: rarefaction clicks, 29/sec, 80 dB HL. Subject # 564.

Averaging was performed with a personal computer with an analog-to-digital converter and artifact rejection software. This system permits independent averaging for each channel. The number of sweeps needed to detect a response depends upon the signal size and the background noise, both of which may vary from patient to patient. In general, about 100 sweeps are needed for ECochG and CPA recordings, and about 1500 sweeps for BAEP recordings. Because a longer period of time is required to obtain an interpretable record of BAEP, they are monitored with an exponential averaging algorithm, which provides a continuously updated, "completed" average.

We are able to compare current responses to recordings obtained earlier in the operation, because the equipment we use allows the placement of horizontal or vertical cursors on the screen, and produces on-line trends of the amplitudes and latencies of peaks that have been specified for the waveforms being monitored (Fig. 2). The trends provided consist of plots of the amplitude (or latency) of the last 100 waveforms averaged. The trend is updated with each new completed waveform. By glancing at the trends, one can compare amplitudes and latencies of present peaks to those values for the previous 100 peaks.

Results

Electrocochleography and BAEP Recording

With the exception of the very first such operations, all the CPA tumor operations we have performed have included intraoperative monitoring by ECochG and BAEP recording, as an aid to the preservation of hearing. A total of 164 such procedures have been monitored in this way. In only one of these cases was there no detectable activity on the ECochG (Table 1). Nearly always N_1, the compound action potential (CAP) of the auditory nerve, was detectable; cochlear microphonic potentials (CM) were less frequently detected, although occasionally the CM was so large that it obscured the N_1. Wave V, the largest of the components of the BAEP that are generated after wave I (the equivalent of N_1 of the ECochG), was less frequently detected. It has been detected in about 60% of our cases, and it has often been of a very small amplitude and markedly delayed, compared to the wave V that can be recorded from normal ears.

Table 1. Detectability of ECochG responses and BAEP wave V during 164 cerebellopontine angle tumor operations

	Response Detectable	*Response Undetectable*
ECochG (CM or N_1)	163 (99%)	1 (1%)
BAEP (Wave V)	103 (63%)	61 (37%)

ECochG, electrocochleography;
BAEP, brainstem auditory evoked potentials;
CM, cochlear microphonic

The implications for hearing outcome are different for these 2 types of recordings (Table 2). In 41% of all cases N_1 was lost and was not recovered (it sometimes can be lost transiently (Levine et al. 1984)); in all cases no measurable hearing was detected postoperatively. Of the 59% of patients in whom N_1 was preserved, 16% of these had no measurable hearing postoperatively, 71% had "useful" (speech discrimination score >15%, and speech reception threshold <70 dB) hearing, and 12% had some measurable hearing, but it was not useful.

Even though wave V was frequently undetectable for extended periods during the operations reported on here, it was detectable by the end of the operation in 30% of the cases in which it was present at the beginning of the operation. In all but one of these cases "useful" hearing was present postoperatively. Wave V was never detected during the operation if it was undetectable at the beginning of the operation. Only in patients in whom wave V was unchanged was hearing always unchanged postoperatively. The outcome was unpredictable in the 70% of cases in which wave V was lost by the end of the operation (ignoring the status of N_1). In 21% of these cases useful hearing was retained; in 69% no measurable hearing was present; and 10% retained some hearing, but it was not useful. Of the cases in which wave V was lost but N_1 preserved, 47% had useful hearing, 22% lost hearing, and 31% had evidence of some residual hearing that was not useful.

While wave V was often lost well before N_1 was lost, wave V, if present, was lost at the same time as N_1 (see Figure 2). On the other hand, in most cases loss of wave V bore no relationship to the status of N_1.

Cerebellopontine Angle Recordings

CPA recordings were made either before or after tumor dissection. In 12 cases recordings were made at both times, in 6 cases recordings were made only before tumor removal, and in 18 cases recordings were made only after tumor removal. Thus, the observations made in this study are based on data obtained during 48 recording sessions in 36 patients.

The waveform of the CPA response often had an initial positive component that occurred with the same latency as the N_1 in the ECochG, followed by a nega-

Table 2. Hearing outcome as a function of evoked potentials recorded at end of 164 cerebellopontine angle tumor operations

Evoked Potentials		Hearing Outcome		
Wave V	N_1	"Useful"	Not "Useful"	No Hearing
Present	Present	31	1	0
Not Present	Present	38	11	16
i) Lost	Present	15	6	10
ii) Never Present	Present	23	5	6
Not Present	Lost	0	0	67
i) Lost	Lost	0	0	40
ii) Never Present	Lost	0	0	27
Totals		69 (42%)	12 (7%)	83 (51%)

tive peak, the latency of which varied according to the position of the recording electrode (Figs. 3 and 4). The positive potential was present in 37 of the recordings, the negative potential was detected in 29 of the recordings, and both were present in 25. For 6 recording sessions no potentials were detected, despite recording from multiple sites. N_1 was detectable in the ECochG, at the time of 3 of these CPA recordings, but wave V in the BAEP was not detectable during the recording of any of these CPA responses.

Whenever wave V in the BAEP was present during CPA recording, a negativity was recorded from the CPA. The converse was not true: wave V in the BAEP could be detected in only about two-thirds of the 29 records in which a negative potential was detected in the CPA recording.

On 19 occasions an attempt was made to follow more medially the negative potential that could be recorded in the CPA or in the internal auditory canal by recording at a few other points along the course of the eighth nerve all the way to the brainstem. In 8 instances this negativity could be recorded with increasing latency and decreasing amplitude as the electrode was moved away from the cochlea towards the brainstem (Fig. 3). In every one of these cases, wave V was present at the time the CPA recordings were obtained. In 8 cases the negativity could not be detected medial to the internal auditory meatus, and in 3 cases it could be followed medial to the internal auditory meatus but not all the way to the brainstem; in all 11 cases, wave V of the BAEP was undetectable.

A close relationship between the negativity in the CPA recordings near the brainstem and the presence of wave V in the BAEP is suggested by the observations that 1) whenever the negativity in the CPA response was recorded near the brainstem, wave V was detected in the BAEP, and 2) whenever CPA responses could not be recorded near the brainstem, wave V was not detected. Furthermore, these observations suggest that the negativity in the CPA recording represents the auditory nerve's CAP and that this negative peak is a result of a synchronous volley of action potentials of single auditory nerve fibers, moving past the nearby recording electrode. The increasing latency of this response with increasing distance from the cochlea is apparently related to the conduction time of the action potentials as they travel between the inner ear and the brainstem. In cases in which the negativity does not reach the brainstem, the point at which the negativity appears to stop propagating would correspond to a region of conduction block or at least desynchronization of the individual nerve fiber impulses. Such a block or desynchronization would lead to insufficient synchrony of the afferent impulses that reach the brainstem to permit generation of the brainstem evoked potentials, thus accounting for the absence of wave V in the BAEP recording from the brainstem.

The relationship between CPA recordings and wave V of the BAEP may even be stronger. In some cases the waveform of the CPA response recorded near the brainstem appears to be similar to the configuration of wave V in the BAEP (Fig. 3). This suggests that in a subject with a normally functioning brainstem the waveform of wave V is determined by the waveform of the negative component of the CPA potential recorded from the eighth nerve as it enters the brainstem.

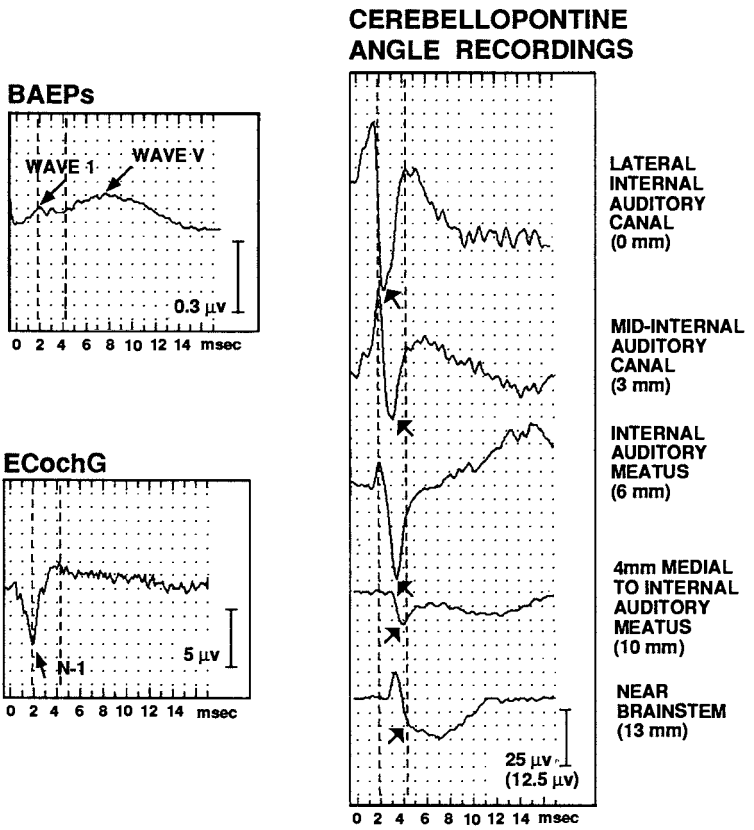

Fig. 3. Recordings from 5 locations in the CPA and the BAEP and ECochG responses obtained at about the same time after removal of a 1.2-cm acoustic neuroma. The internal auditory canal had been drilled open. Positivity is shown as an upward deflection in this and all subsequent figures. The major negative peak *(unlabeled arrows)* increased in latency and decreased in amplitude as the recording electrode was moved from the lateral internal auditory canal toward the brainstem (the distance of the recording site from the lateral internal auditory canal is shown in parentheses). Only near the brainstem did the waveshape of this peak become broad and resemble that of the BAEP. An initial positivity occurred at about the same time as N_1 of the ECochG in the recordings lateral to the internal auditory meatus, but this positivity is not apparent in the recording 4 mm medial to the internal auditory meatus. A positivity is present again in the recording near the brainstem, but at a longer latency than the positivity in the more medial recordings. The scale for the bottom CPA waveform is different from that for the top 4 and is shown in parentheses. Stimulus: alternating clicks, 29/sec, 80 dB HL. Postoperatively, a severe hearing loss was present with no measurable speech discrimination. Subject # 471.

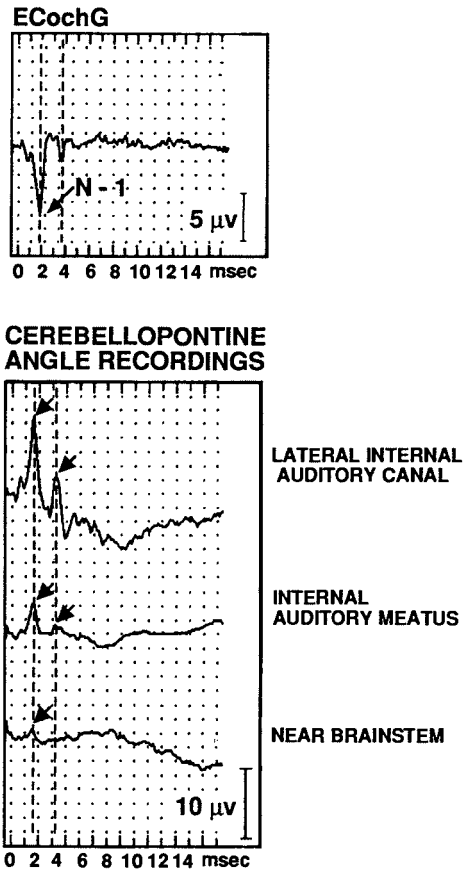

Fig. 4. Recordings from 3 locations in the CPA and ECochG responses (obtained at the same time after removal of a 0.9-cm acoustic neuroma). The BAEP were undetectable at the time of these recordings, with the exception of wave I. The major feature of these recordings is 2 positive peaks *(unlabeled arrows)* that occur at the same time as N1 and a later negative peak of the ECochG. The amplitudes decreased but the latencies did not change as the electrode was moved toward the brainstem. Stimulus: rarefaction clicks, 29/sec, 80 dB HL. Postoperatively, speech discrimination score was improved (76% versus 62%) and thresholds were unchanged (except at 8000 Hz). Subject # 499.

The positivity in the CPA response waveform usually precedes the negativity and occurs with the same latency as N_1 of the ECochG. Its amplitude decreases as the electrode is moved away from the cochlea, but, unlike the negativity, its latency generally does not change (Fig. 4). The CPA positivity often has a configuration similar to an inverted version of the patient's N_1, and the positivity was present in 85% of 44 recordings from the CPA for which an N_1 was present in the ECochG. In one instance the presence of N_1 in the ECochG was questionable, because very large amplitude CM were present; however, there was a positivity in the CPA recordings. Because CM do not appear to affect the CPA recordings, the finding of a positivity in the CPA recordings at the same time as the ECochG response is dominated by CM suggests that the CAP is likely present in the ECochG and would be detectable if the CM were not so large.

Discussion

Table 3 summarizes our observations from monitoring ECochG and BAEP during CPA tumor operations. A major advantage of intraoperative monitoring of the ECochG is the detectability of the responses. The ECochG potentials can always be recorded, whereas often the BAEP are undetectable. Rarely the CM component of the ECochG response can be so large that it obscures the neural component. A second major advantage of ECochG over BAEP monitoring is that the ECochG has a much larger amplitude. Thus, the ECochG responses can be detected clearly within a few seconds, but detection of BAEP in a typical patient requires minutes. On the other hand the 2 recording techniques complement each other in terms of the regions of the auditory system monitored. ECochG records cochlear activity as well as auditory nerve activity. The CM are generated by the hair cells of the inner ear and the summating potential is generated by multiple different elements in the cochlea (but not by the auditory nerve), whereas N_1 is generated by the auditory nerve, probably by the portion within or very close to the cochlea. The BAEP include components generated by the auditory nerve and brainstem. Wave I occurs with about the same latency as N_1 and probably has the same generator. In practice only wave V is monitored, because the other potentials are much smaller and usually undetectable. Wave V is generated from within the brainstem. By monitoring both ECochG responses and BAEP, the entire portion of the auditory system at risk during an operation to remove an acoustic neuroma can be monitored: (1) presence or absence of N_1 indicates the integrity of the auditory nerve peripheral to the tumor. (2) Wave V is an indicator of auditory nerve activity central to the tumor, and (3) the cochlear microphonic (CM) and summating potential indicate the status of the cochlea, which is at risk from interruption of its blood supply or from damage to other structures essential for cochlear function (such as the endolymphatic sac or the posterior semicircular canal).

In terms of what they indicate about hearing, these 2 types of potentials – ECochG and BAEP – also are complementary. N_1 in the ECochG is necessary

Table 3. Electrocochleography *(ECochG)* potentials versus brainstem auditory evoked potentials *(BAEP)* versus cerebellopontine angle *(CPA)* responses

	ECochG	BAEP	CPA Recordings
Detectability	Always Detectable	Often Detectable	Often Detectable
Feedback	Rapid	Slow	Rapid
Region Monitored	Cochlea & Distal Nerve	Distal Nerve & Brainstem	Distal Nerve & Proximal Nerve
Implications			
i) Absent	No Hearing	Indeterminate	Indeterminate
ii) Present	Indeterminate	Hearing	Hearing

Table 4. Hearing outcome as a function of presence of evoked potentials at end of 164 cerebellopontine angle tumor operations

	N_1 Present	N_1 Not Present
Wave V Present	Hearing (31)	**
Wave V Not Present	**Indeterminate** (66): "Useful" Hearing (38); Not "Useful" Hearing (12); No Hearing (16)	**No Hearing** (67)

BAEP, brainstem auditory evoked potentials;
ECochG, electrocochleography;
( ), number of operations;
** Loss of N_1 with preservation of wave V has never been observed.

but not sufficient as an indicator of hearing, whereas BAEP wave V is sufficient but not necessary as an indicator of hearing. As is shown in Table 4, if wave V was detectable then useful hearing was nearly always present postoperatively in this study; if N_1 was lost then hearing was always lost. If N_1 was present and wave V was lost or never present, then the outcome for hearing was unpredictable: useful hearing, hearing loss, or very poor hearing could occur.

Although CPA responses were not monitored continuously in this study, we can draw some conclusions about the usefulness of these responses (Table 3). When CPA responses were recorded central to the tumor (near the brainstem), the initial negativity provided the same information as did BAEP wave V and thus this wave has the same predictability: it is sufficient but not necessary for hearing. When the response was recorded more peripherally, the CPA response, like the N_1 wave, proved necessary but not sufficient for hearing. The initial positivity in the CPA response appears to have the same generator as the N_1 of the ECochG (although the CPA wave was less detectable than N_1 in the ECochG), and so the initial positivity in the CPA response has the same predictive value as N_1: its presence does not assure postoperative hearing. The major advantage of using these recordings versus BAEP recordings is the rapid feedback provided by monitoring CPA responses, which in turn is due to their large amplitude. By providing more rapid feedback, monitoring CPA responses may allow earlier detection of changes, and, thereby, earlier modification of the surgery and improved outcome.

Silverstein et al. (1986) monitored CPA responses during acoustic neuroma surgery with the electrode placed "at the cochlear nerve root entry zone." CPA responses were monitored in 8 of the 16 cases they report, but could not be obtained in 2 (like our results, wave V could not be detected in these two cases). They do not explain why CPA responses were not recorded in the other 6 patients. The results of Silverstein et al., generally appear to be in agreement with ours. One exception appears to be their case #6, for which they report a hearing loss despite preservation of an attenuated and delayed "eighth nerve potential."

However, it is unclear what these authors considered to be the eighth nerve potential; judging from their third figure, they may have considered the initial positive potential to be the eighth nerve potential. If this is the case then it would be consistent with our results, in that the initial positivity alone, like N_1, is not sufficient for hearing preservation.

From our experience, recording both ECochG potentials and BAEP is valuable for monitoring, because they are complementary in terms of predicting hearing preservation. Yet, as has been discussed, each has its limitations. When N_1 is present and wave V is not present, hearing outcome is unpredictable. Even when CPA responses are recorded this condition is still indeterminate for predicting the outcome for hearing. It appears to be a fundamental limitation of electrophysiologic monitoring that is related to how the individual nerve fibers can be injured. An injured nerve fiber may 1) stop conducting an impulse altogether or 2) conduct a modified impulse. The first case would account for the times when hearing is lost. In patients with some hearing preserved, some nerve fibers must still be conducting to the brainstem, yet wave V and the negativity in the CPA responses are undetectable. This suggests several possibilities: 1) the remaining nerve fibers are too few to generate a gross potential that can be detected; 2) the modified nerve impulses may be desynchronized with each other; 3) the waveforms of individual nerve fibers are no longer similar in configuration and so, even if they discharge synchronously to the stimulus, at the gross electrode the waveform of one fiber cancels out that of another; or 4) some combination of the first 3 possibilities.

Acknowledgements. It is a pleasure to thank the neurosurgeons (Drs. Ojemann and Martuza) and otological surgeons (Drs. Montgomery, Nadol, and McKenna) who have made this study possible, the consistent cooperation of the neuro-anesthesia and nursing staffs, and E. Carlisle for preparing the figures.

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Monitoring of Auditory Function in Acoustic Neuroma Surgery

L. SYMON[1] and D. A. JELLINEK

Summary

Our clinic now has a cumulative experience in operating on over 400 patients with acoustic neuromas. During the past 40 years it has been apparent that there has been a progressive reduction in the morbidity rate of patients being operated on for this condition. This prompted us to investigate means of maximizing the preservation of hearing after surgery for patients with an acoustic neuroma. As our department already had extensive experience in intraoperative electrophysiological monitoring, we explored brainstem auditory evoked potentials (BAEP), electrocochleography (ECoG) and direct eighth nerve recording in an attempt to resolve this problem.

This paper discusses our experiences with ECoG and direct eighth nerve recording. Despite the technical difficulties and shortcomings inherent in these techniques, we believe they are superior to BAEP in preserving useful postoperative hearing after surgery to remove acoustic neuroma.

This chapter describes our experience of preservation of hearing during operations to resect small acoustic neuromas. Being committed to study of the central nervous system, we are convinced that the study and application of electrophysiology in neurosurgery will enhance our understanding of the function of the nervous system. In addition we must consider how electrophysiological monitoring can help us maintain acceptable standards of patient care, especially medicolegal standards, without straining restricted budgets for allocation of our resources.

In our clinic we have to date managed acoustic neuromas in more than 400 patients (Symon et al. 1989). Over the last decade, the proportion of tumors we have seen that are less than 1.5 cm in size has increased (Fig. 1). During this period there has also been steady improvement in the patient's postoperative neurological status compared with the 1950s (Table 1). In addition, retrospective analysis has shown that we were able to preserve the eighth cranial nerve in a significant number of cases, particularly in patients with small tumors (Fig. 2), and that a proportion of patients still had useful hearing (Fig. 3). These findings prompted us, a neurosurgeon and otolaryngologist, to challenge ourselves as a

[1] Gough Cooper Department of Neurological Surgery, Institute of Neurology, The National Hospital, Queen Square, GB-London WC1N 3BG, U.K.

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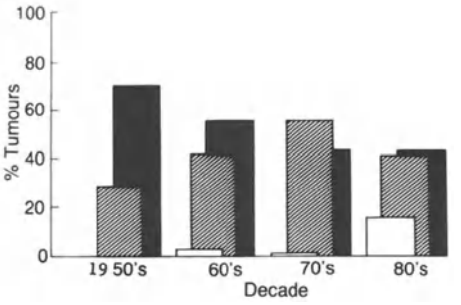

Fig. 1. Distribution of tumors by size by decade. We saw no small tumors in the 1950s. □ <1.5 cm; ▨ 1.5 to 3 cm; ■ >3 cm

Table 1. Changes in outcome of operations to manage acoustic tumors, by decade, at one clinic

	Decade			
	1950s	1960s	1970s	1980s
No Tumors	41	72	137	147
Perioperative death (%)	9.7	4.2	4.6	1.4
Complicatons				
CSF leak	2.4	2.8	6.8	18.4
Others	4.9	12.5	12.8	4.1
Outcome				
Good/Excellent	75.6	79.2	87.1	93.9
Poor	14.6	16.6	7.5	4.1

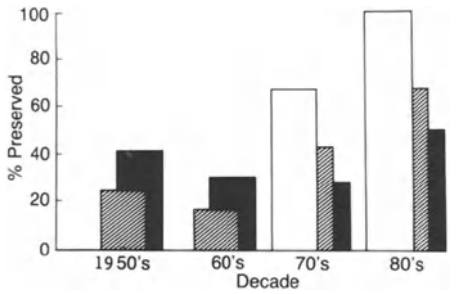

Fig. 2. Anatomical preservation of the facial nerve in patients treated over 4 decades. □ <1.5 cm; ▨ 1.5 to 3 cm; ■ >3 cm

professional team to preserve hearing in a larger proportion of patients with small tumors and good preoperative auditory function (Compton et al. 1989).

The advent of high-resolution imaging has enabled us to identify the anatomical features of small acoustic neuromas that would help us significantly in our efforts to preserve the auditory nerve in these operations. Fig. 4 is an example: the air meatogram demonstrates that this tumor is really intracanalicular. The patient had presented with tinnitus and slightly reduced speech discrimination.

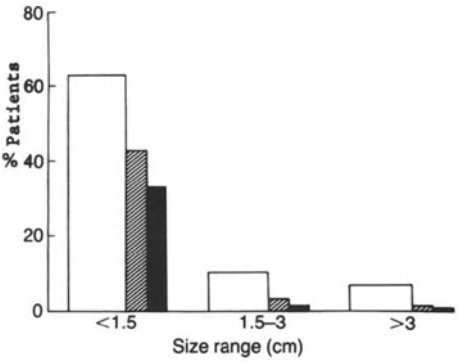

Fig. 3. Preservation of the cochlear nerve and hearing, by size of tumor, in patients operated upon to resect acoustic neuromas. Percentage of patients with anatomically intact cochlear nerve postoperatively □; percentage of patients with some hearing postoperatively ▨; proportion with useful hearing ■

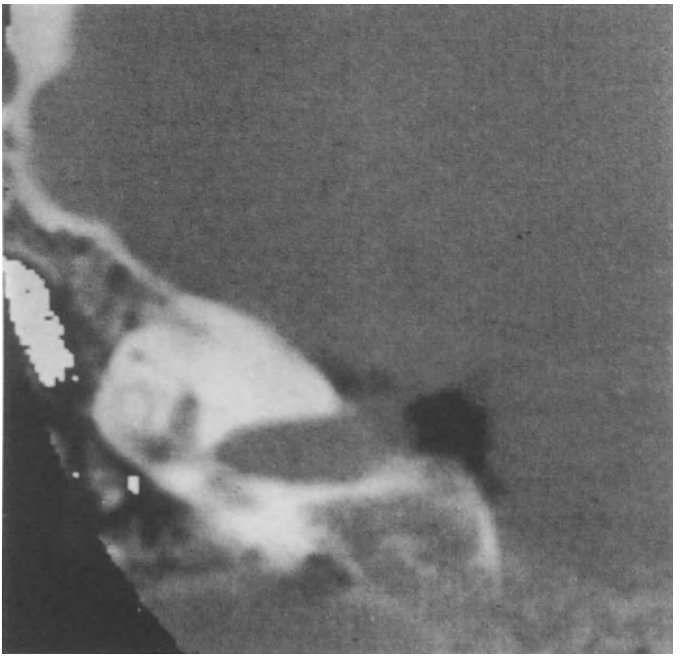

Fig. 4. Air meatogram showing small intracanalicular acoustic tumor

One must ask if removing this tumor and thereby causing deafness (even though the facial nerve would be preserved) is really offering this patient a good service?

We turned to electrophysiological monitoring for possible means to preserve auditory nerve function. Two electrophysiological techniques are available to monitor eighth cranial nerve function intraoperatively, brainstem auditory evoked potential (BAEP) and electrocochleography (ECoG) (Levine et al. 1984; Ojemann et al. 1984). We owe a great debt to Drs. Ojemann and Levine, because it was they who directed our attention to ECoG in preference to BAEP recording (Ojemann et al. 1984).

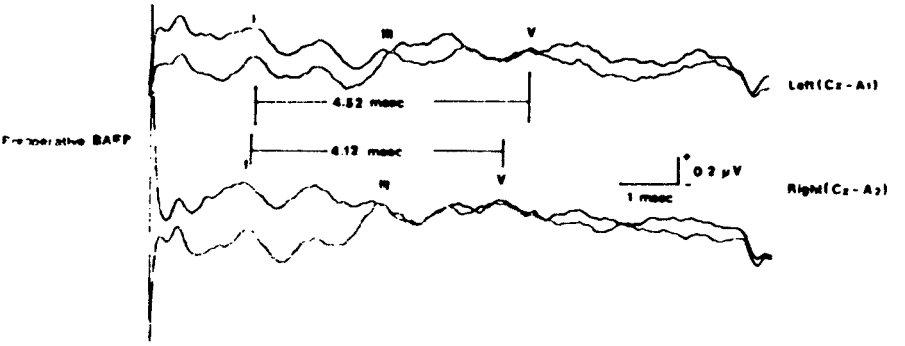

Fig. 5. Preoperative BAEP of patient with right temporal malignant glioma. Note that the I-V interpeak latency is prolonged on the opposite side

We already had considerable experience in recording BAEP intraoperatively and knew that delay of wave V could be reversed by removal of a subtentorial mass, for example. However, changes in BAEP appeared to be quite nonspecific as regarded a tumor's location, as the BAEP changed regardless of whether surgery was performed ipsi- or contralateral to the side from which recordings were being made. We also found that BAEP could change in the presence of pathology quite remote from the posterior fossa (Fig. 5) (Tsutsui et al. 1986). Further, changes in BAEP could occur during almost any manoeuvre in the posterior fossa, for example positioning of a retractor during acoustic tumor operations or simply with draining of cerebrospinal fluid (CSF). It seemed that we were obtaining nonspecific information that alarmed and did not really help the surgeon.

Therefore we turned our attention to ECoG, prompted by the experiences of Ojemann et al. (1984). ECoG has two advantages over BAEP monitoring: because the amplitude of the recorded potentials are much larger than that of the BAEP, 1) the need for averaging is minimized, and 2) recorded potentials can be interpreted much more quickly. Against this are a number of technical problems (Fig. 6) (Sabin et al. 1987). It is necessary to position a recording electrode against the promontorium of the middle ear; this necessitates using microscopic guidance to puncture the tympanic membrane. We position a 1-mm ball electrode on the promontorium in the region of the round window (this is preferable to a needle electrode as the larger contact area of the ball gives a higher signal-to-noise ratio). We then use a 15-cm length of tubing to deliver the click stimuli to the ear. A venting tube adjacent to this tube enables one to suction CSF from within the middle ear cavity should this become flooded as a consequence of opening mastoid air cells during tumor resection (fluid within the middle ear may impair sound transmission to the inner ear and dampens the ECoG response in a manner indistinguishable from dampening due to eighth cranial nerve damage). The whole stimulating array is held in place with ear impression putty reinforced with collodion glue.

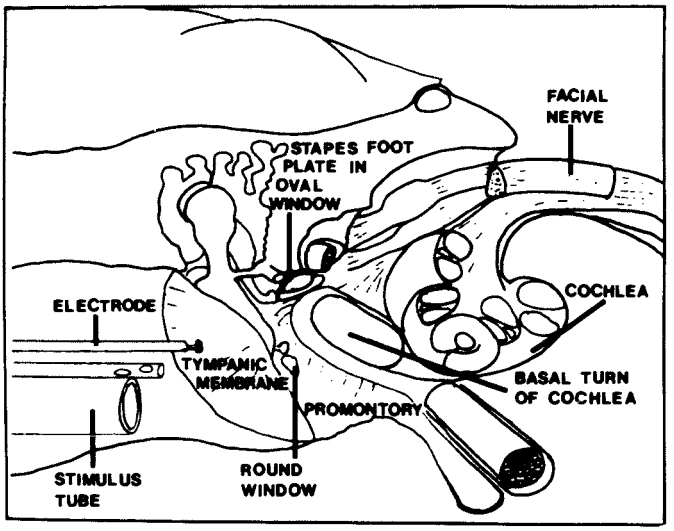

Fig. 6. Schematic coronal section of the ear, showing stimulus tube and electrode positions for ECoG

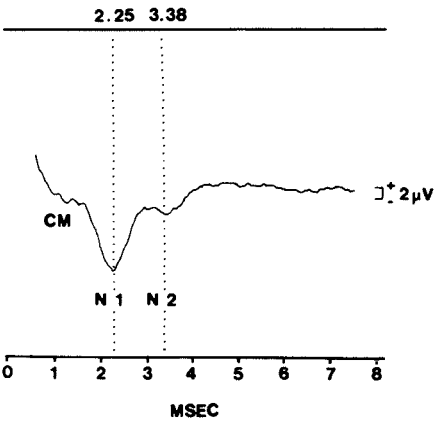

Fig. 7. Normal ECoG waveform showing cochlear microphonics *(CM)*, main negative peak *(N_1)*, and second negative peak *(N_2)*

More recently we have been recording responses directly from the eighth cranial nerve. The best way to place the recording electrode is to hook it into the lateral recess of the fourth ventricle with the aid of a fold of arachnoid; here, it will be well medial to the operative field.

All patients on whom we operate to manage cerebellopontine angle (CPA) lesions are placed in a contralateral-lateral (or "park bench") position. This position provides the advantage that with lumbar CSF drainage no cerebellar retraction is necessary. Fig. 7 shows a normal ECoG waveform. The latency of N_1 and N_2 will increase if the stimulus intensity is reduced. We were generally able to recognize wave N_1 clearly after averaging the response to only 10 to 20 stimuli owing to the large amplitude of the response. When we started using ECoG we applied

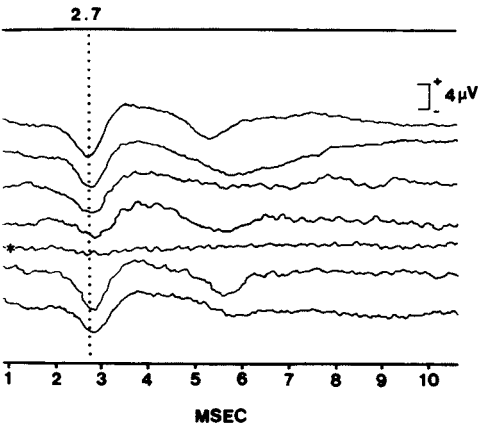

Fig. 8. Loss of N_1 due to tumor manipulation with subsequent return when the tumor's large central cyst was decompressed (*)

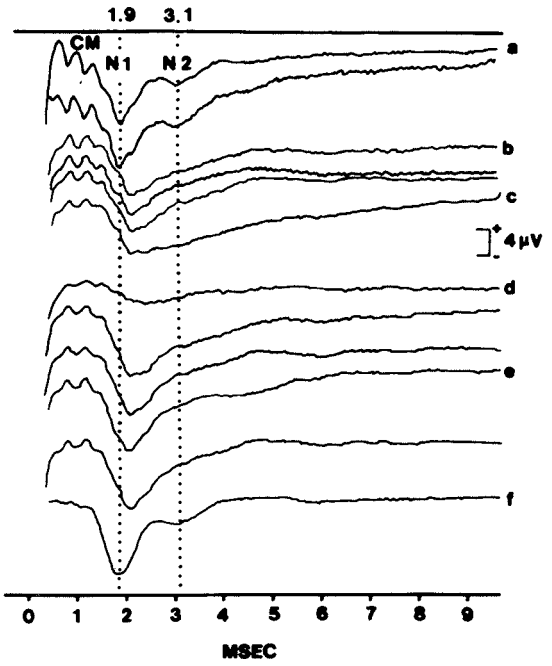

Fig. 9. Intraoperative N_1 latency changes in a patient with good postoperative hearing. Tracings of interest have been detailed: *a*, before skin incision; *b*, initial tumor manipulation; *c*, drilling porus acusticus; *d*, dissecting tumor from cochlear nerve; *e*, end of tumor dissection; and *f*, end of operation. It can be seen that N_1 latency increased but returned to normal by the end of the operation. The CM do not appear clearly in the final trace because an alternating-polarity stimulus was used

it to every patient undergoing acoustic tumor operations. Experience has subsequently taught us to restrict its application to patients with a speech discrimination score of 50% or more and better than 60 dB nHL (Sabin et al. 1987).

Fig. 8 shows a typical intraoperative record. A large cyst was present in association with the tumor. As the acoustic tumour was manipulated the N_1 response was temporarily lost, although it returned subsequently. Fig. 9 shows a similar pattern. The tumor had filled the internal meatus, and extensive drilling was neces-

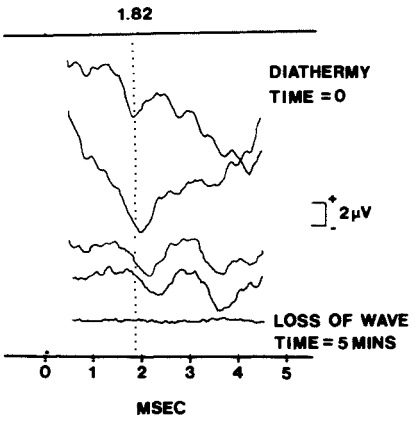

Fig. 10. Gradual loss of N_1 and CM following diathermy to the internal auditory artery. The tumor had been excised and no other nerve manipulation was occurring. The recordings span a 5-minute period

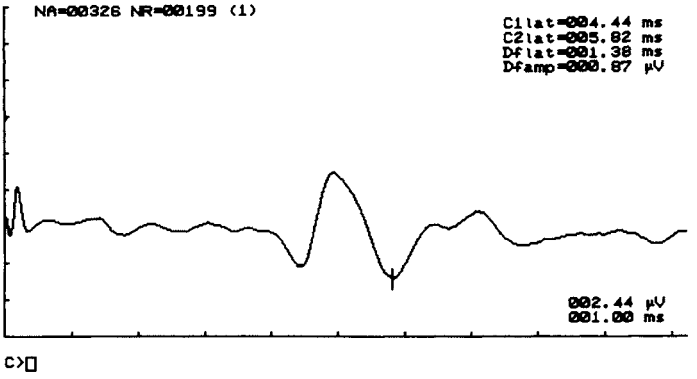

Fig. 11. Direct recording from the eighth nerve. The 1-ms delay in waveform was due to technical problems

sary to mobilize it. The amplitude of N_1 decreased and there was an increase in latency during dissection, but the potentials returned to normal at the end of the operation.

Fig. 10 demonstrates the consequences of diathermy within the internal auditory canal. These effects were particularly unfortunate in this patient because a good N_1 potential had been present after the tumor had been removed. In retrospect it is probably wiser to place Surgicel or Tisseel around the internal auditory artery to promote hemostasis than to use diathermy.

Loss of the N_1 wave was an inevitable herald of postoperative deafness. Of greater concern, 2 patients who still had an N_1 at the end of the operation were deaf postoperatively. In 1 case the N_1 was still present until 48 hours after the operation. Our interpretation of this occurrence is either that the site of conduction failure was proximal to the segment generating the N_1 potential, or that damage had occurred to central auditory pathways.

Information about the function of the medial portion of the eighth nerve can be obtained by placing an electrode in the CPA (Møller and Jannetta 1983). This has

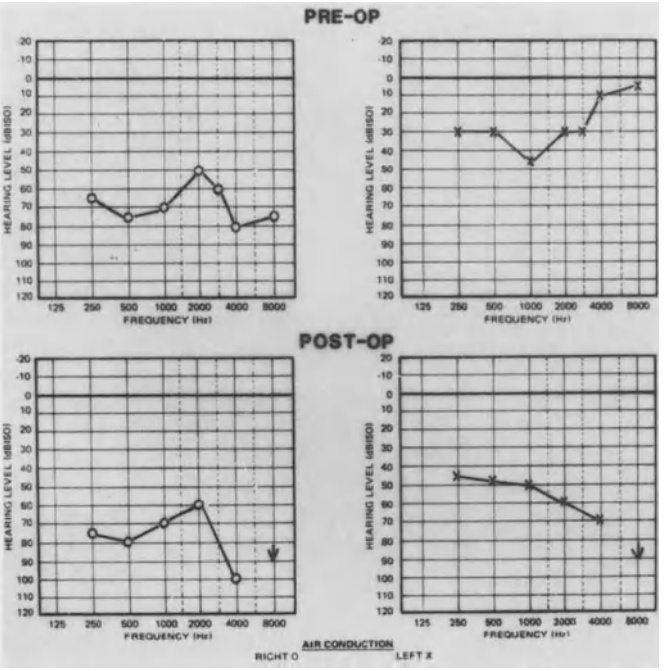

Fig. 12. Pre- and postoperative audiograms of patient with a second (left sided) acoustic tumor

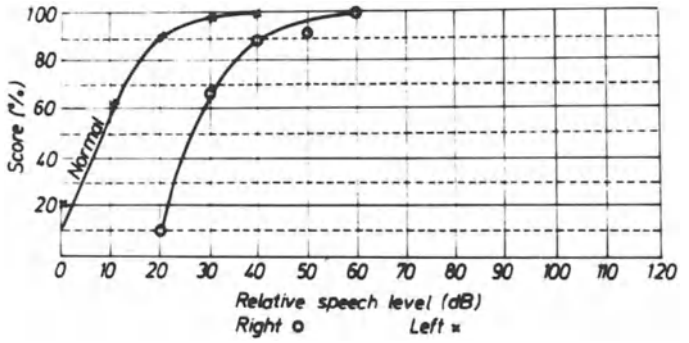

Fig. 13. Postoperative speech discrimination scores; the patient retained useful hearing in the operated (left) ear

the disadvantage that one has no preoperative recordings to use as baseline, and thus is "blind" while the craniedomy is being performed. However, such an electrode is easy to position and keep out of the operating field. Direct eighth nerve recording also saves the time that would be needed to prepare for ECoG recording. We have little experience in this technique to date (Fig. 11). In patients already deaf in the contralateral ear (for example, those with a second acoustic neuroma) in whom one is particularly anxious to preserve hearing, ECoG is valuable. The audiograms of such a patient are shown in Fig. 12; this patient was left with enough auditory nerve function to provide useful hearing with amplification.

To summarize, we have preserved useful hearing in about half of our patients with acoustic tumors less than 1.5 cm in diameter. Postoperative hearing loss has been considerable in others, but in many speech discrimination has remained at useful levels (Fig. 13).

From these results we consider that, despite its technical difficulties and 10% failure rate in our clinic, ECoG is of greater value than BAEP recording in preserving hearing in patients undergoing operations to manage acoustic neuromas.

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Intraoperative Monitoring of Cochlear and Auditory Nerve Potentials in Operations in the Cerebellopontine Angle: An Aid to Hearing Preservation

D. W. Rowed[1], J. M. Nedzelski, M. Z. Cashman, and S. G. Stanton

Summary

Intraoperative monitoring of cochlear compound action potentials (CAP) by transtympanic electrocochleography (ECoG) and by direct recording from the cochlear nerve intracranially, when possible, was performed during 59 operations in the cerebellopontine angle. Thirty operations were performed to remove acoustic neuromas by a suboccipital hearing-preservation approach, 24 were vestibular neurectomies (VN) to treat Meniere's disease, and 5 were nervus intermedius (NI) sections to relieve chronic cluster headaches. Interpretable responses were obtained in most patients (93%).

We prefer to record cochlear CAP rather than brainstem auditory evoked potentials (BAEP) because the larger amplitude of CAP obviates the time delay required for summation of responses, and therefore makes reversible changes in neural function more likely to be detected promptly.

Monitoring of CAP has also proven helpful in cranial nerve procedures in which reversible changes in neural function were recognized; CAP monitoring has helped to preserve serviceable hearing postoperatively in all 29 patients in whom we have used this technique.

In patients with acoustic neuromas who evidenced major CAP threshold shifts (mean 29 dB) or disappearance of the CAP during operations to remove the tumor, postoperative hearing was consistently not serviceable. Changes in the CAP, however, have been harder to correlate with events in the operative field in patients undergoing tumor resection versus those undergoing cranial nerve section, and our success rate for hearing preservation following acoustic neuroma operations has so far failed to improve since we began routine intraoperative monitoring of these patients.

Early diagnosis of acoustic neuromas, and their complete removal with virtually no mortality and with preservation of facial nerve function, have become almost routine. Hearing preservation, particularly with removal of small tumours, has become a realistic goal in the management of many patients but thus far is not achievable in the majority (Cohen et al. 1986; Jannetta et al. 1984; Silverstein et al. 1986; Ojemann et al. 1984). Monitoring of cochlear nerve function has been

[1] Division of Neurosurgery, Sunnybrook Medical Centre, University of Toronto, 2075 Bayview Avenue, Toronto, Ontario, M4N 3M5 Canada.

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seen as a way of identifying potentially reversible nerve injury and improving the rate of successful hearing conservation, in the way electromyography of the facial muscles is used to decrease the incidence of facial nerve injury (Ojemann et al. 1984; Rowed et al. 1988; Stanton et al. 1989; Levine et al. 1984; Møller and Jannetta 1984; Silverstein et al. 1985a).

We monitor cochlear compound action potentials (CAP) by either transtympanic electrocochleography (ECoG) or by direct recording from the cochlear nerve intracranially, believing this to be the most reliable and relevant technique for assessing cochlear function intraoperatively (Rowed et al. 1988; Stanton et al. 1989). The large amplitude response requires little or no averaging and is therefore available in virtually real time, making possible the timely detection of potentially reversible perturbations of cochlear or eighth nerve function (Silverstein et al. 1985a, b; Levine et al. 1984; Stanton et al. 1989; Rowed et al. 1988; Møller and Jannetta, 1984).

Cochlear monitoring is also useful during other cerebellopontine angle (CPA) procedures such as vestibular neurectomy (VN) (Silverstein et al. 1985b; Rowed et al. 1988), nervus intermedius (NI) section, and cranial nerve microvascular decompression (MVD) procedures (Møller and Jannetta 1983) when minimal morbidity is essential.

Patients and Methods

At the Sunnybrook Hospital, University of Toronto, a total of 331 acoustic neuromas have been excised to date. A translabyrinthine approach was employed for 281 of these cases, and a suboccipital hearing conservation approach in 50.

We believe that hearing conservation is a realistic goal in patients with good preoperative hearing and relatively small tumours, and an unlikely possibility in patients with large tumours (Silverstein et al. 1985a, 1986; Jannetta et al., 1984). The cutoff point between 'larger' and 'smaller' is somewhat arbitrary, but preservation of *useful* hearing when the tumour is larger than 2 cm in diameter is infrequently possible (Cohen et al. 1986; Silverstein et al. 1985a). Our policy has been to consider patients with tumours of approximately 1.5 cm diameter *medial to the porus acusticus* as potential candidates for hearing conservation. Thus, we classify as small tumours those up to 1.5 cm in diameter, and these constitute 44% of our series (increasing slightly in proportion in recent years). Medium tumours are those up to 3 cm in diameter and make up 30% of our population, and large tumours are >3 cm in diameter and comprise 26% of our population.

Using a translabyrinthine rather than a suboccipital approach to larger tumours is optional, but the former approach offers a less deep operative field with little or no cerebellar retraction, and has yielded a high rate of facial nerve preservation at our institution, particularly for tumours larger than 1.5 cm (Table 1).

Using this method to select the operative approach, 281 patients with acoustic neuromas have been operated on to date by a translabyrinthine approach, and 50 by a suboccipital hearing conservation (SHC) approach. Thirty of the suboccipital procedures were performed with cochlear nerve monitoring.

Table 1. Success rate for preservation of the facial nerve with complete Excision of 284 acoustic neuromas of various sizes

Ascoustic Neuroma	Successes/Total	Percent Success
Small	121/125	(97%)
Medium	77/83	(93%)
Large	67/76	(88%)

Intraoperative cochlear nerve monitoring has also been carried out on 24 patients undergoing VN to treat Meniere's disease and on 5 of 7 patients undergoing NI section and MVD procedures to manage chronic cluster headache.

Operative Procedure

Patients undergoing acoustic neuroma resection with hearing preservation are positioned prone with the head flexed, rotated 30 degrees to the side of the tumour, and stabilized by three-point fixation of the calvarium. A Y-shaped retroauricularly based scalp flap is used, extending from the mastoid tip to 1.5 cm above the transverse sinus and to a point approximately 2 cm lateral to the midline. The suboccipital muscles are divided in the line of the incision down to the pericranium, and a 3-cm disc of bone is removed from the squamous occipital bone; the disc extends to the sinus angle and exposes the inferior edge of the transverse sinus and the extreme posterior margin of the sigmoid sinus (Fig. 1).

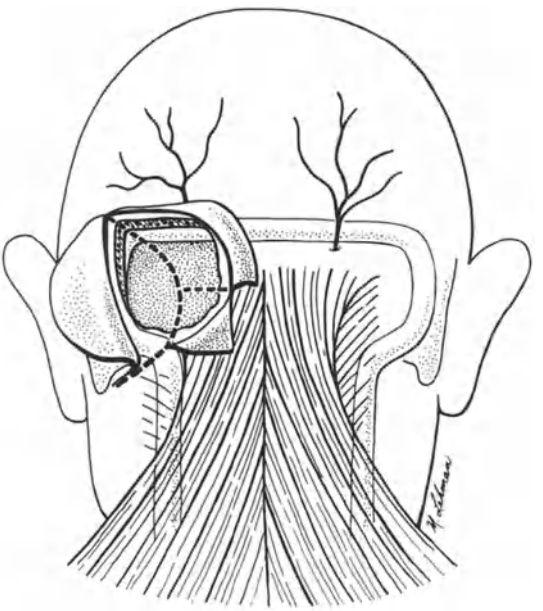

Fig. 1. Operative exposure for suboccipital hearing conservation approach. Postoperative discomfort is less than with the commonly employed vertical or sigmoid incisions

This approach minimizes neck muscle dissection and therefore postoperative pain, and, when used in conjunction with lumbar drainage of cerebrospinal fluid, allows exposure of smaller CPA tumours without any need for cerebellar retraction, thus lessening the potential for postoperative morbidity.

A precisely similar approach has been used for NI sections and for MVD procedures. For VN a similar approach is appropriate, although we have usually employed a retrolabyrinthine approach, anterior to the sigmoid sinus (Silverstein and Norrell 1982).

Monitoring Technique

Stimulation: Unfiltered 100-μs clicks of alternating polarity, and tone pips at 1, 2, 4, and 8 kHz were applied to Nicolet Tubal Insert Phones (T1P-10). Rise and fall times for 1-kHz tone pips were 2 ms with no plateau, and for the other frequencies were 1 ms with a 1-ms plateau. After thresholds had been determined, a stimulus of approximately 20 dB above threshold was used for intraoperative monitoring. Stimuli were presented at a rate of 11.2/sec, usually either 200 or 500 responses were averaged to establish thresholds, although a response could frequently be seen after 10 responses or less were averaged. All thresholds were replicated.

Recording: Prior to beginning the operative exposure, a transtympanic electrode was placed so that ECoG could be performed during exposure. The electrode consisted of a Teflon-coated silver wire with a small cottonoid pledget sutured to the tip. This was placed against the middle ear promontory via a small myringotomy, and stabilized by the extension tube that delivered acoustic stimuli from the adjacent receiver. Myringotomy was not necessary when the retrolabyrinthine approach was used because the recording electrode was placed via a small incision to expose the facial recess. Whenever possible recordings were also made directly from the ipsilateral cochlear nerve, using a similar electrode fashioned from finer wire. Intracranial recordings have been obtained from the proximal cochlear nerve during removal of small tumours, from the pons near the root entry zone, and from the cochlear nerve in the internal auditory canal, either extra- or intradurally. Needle electrodes were used in the forehead scalp for reference and on the contralateral shoulder for ground.

Click and tone pip thresholds were measured in 5- to 10-dB steps before and after tumour removal or nerve section. When possible, promontory and direct nerve recordings were carried out simultaneously.

Changes in recorded potentials were reported immediately to the surgical team so that they could be correlated closely with events in the surgical field.

Preoperative and Postoperative Audiological Assessments

Pure tone audiometry with determination of air and bone conduction thresholds and speech audiometry with determination of speech reception thresholds (SRT) and speech discrimination scores (SDS) were performed preoperatively and postoperatively, with the mean interval between pre- and postoperative testing being

1.3 months for patients with tumours and 1.7 months for those undergoing nerve section. We define serviceable hearing as a speech reception threshold (SRT) <50 dB and a speech discrimination score (SDS) >60% (Rowed et al. 1988). We believe this to be a suitable standard that is comparable to although not precisely the same as that reported by other investigators (Cohen et al. 1986; Silverstein et al. 1986).

Results

Reliability

Good-quality recordings were consistently obtained from the cochlear promontory (ECoG), and from the cochlear nerve intracranially (8NP) when the latter was accessible (Fig. 2).

CAPs were recordable intraoperatively in 55 of 59 patients, including 30 patients with tumours (3 failures, 10%), 24 VN patients (1 failure, 4%), and 5 NI section patients (no failures). In 1 patient failure to record CAP was attributable to a broken electrode wire, and the reasons for failure in the other cases are unclear. The overall success rate for obtaining intraoperative CAP was thus 93%.

Detection of Cochlear Nerve Dysfunction

In acoustic neuroma patients, presence of a CAP at the conclusion of the procedure without any major threshold shift accurately predicted postoperative preservation of hearing. The cochlear CAP was present at the conclusion of the operation in 17 of 30 tumour patients. Five of the 17 patients who had a CAP at the end of the operation demonstrated major intraoperative shifts in threshold (mean shift 29 dB) and none of these had serviceable hearing postoperatively according to our previously stated criteria. The remaining 12 patients in whom CAPs were

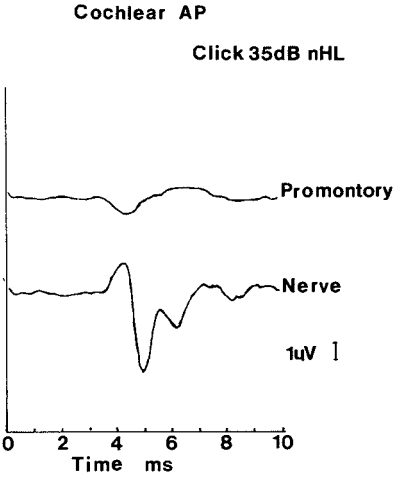

Fig. 2. Recordings of CAP from the cochlear promontory by means of a transtympanic electrode (electrocochleography, ECoG) and from the cochlear nerve intracranially using a similar electrode placed directly on the nerve. Positivity is indicated by an upward deflection

present at the conclusion of the operation, at relatively low thresholds, had serviceable hearing postoperatively. However, none of the 13 patients whose CAP disappeared during the operation had hearing postoperatively. In 2 of these 13 patients the CAP disappeared during cranial nerve exposure without apparent reason, and those 2 patients did not have serviceable hearing postoperatively. In 1 additional patient in this group of 13 the CAP disappeared when there was no activity in the surgical field. In this case an apparently inconsequential artery in the tumour capsule had been coagulated a few minutes earlier, and the cause of hearing loss may have been ischemia to the auditory nerve or the cochlea, although this remains unproven.

All of the VN patients had easily detectable CAP at the conclusion of the operation, without large shifts in threshold, and all of these patients had serviceable hearing postoperatively. Five of 7 NI section patients had CAP at the conclusion of the operation and serviceable hearing postoperatively, whereas the other 2 had no hearing postoperatively. These 2 patients, however, were not monitored intraoperatively for logistical reasons, so no correlation could be determined.

Detection of potentially reversible cochlear nerve dysfunction is of paramount importance. On occasion this has proved possible. One such case is illustrated (Fig. 3).

In this instance manipulation of the cochlear nerve during vestibular neurectomy resulted in sudden loss of the cochlear CAP. Immediate cessation of the operation, followed by irrigation of the nerve with warm physiologic saline solution, was followed by return and gradual increase in amplitude of the CAP. The patient's postoperative audiogram was similar to the preoperative audiogram.

More frequently, however, sudden loss of the CAP, as just described, has not correlated well with events in the operative field, and cessation of the procedure has not been followed by return of the CAP, or by conservation of hearing postoperatively.

Postoperative Hearing

The quality of postoperative hearing, when present, was nearly as good as or better than preoperative hearing in 16 of 23 acoustic neuroma patients (Fig. 4). These patients with postoperative hearing conservation had postoperative SRT within 20 dB and SDS changes <30% of preoperative values. Twelve of these 16 patients had been monitored intraoperatively, and 4 were from the group of 20 who were operated on before monitoring became routine.

Thus, the success rate for preserving serviceable hearing during the 20 suboccipital hearing preservation procedures performed at our institution prior to the inception of cochlear nerve monitoring was 4/20 (20%), and the success rate since monitoring has become routine is 12/30 (40%). The difference in success for the 2 groups is not statistically significant ($p = 0.12$).

Three VN patients experienced an SRT decrease of >20 dB (25, 25, and 30 dB), and none of the 5 NI patients showed an SRT change of >20 dB.

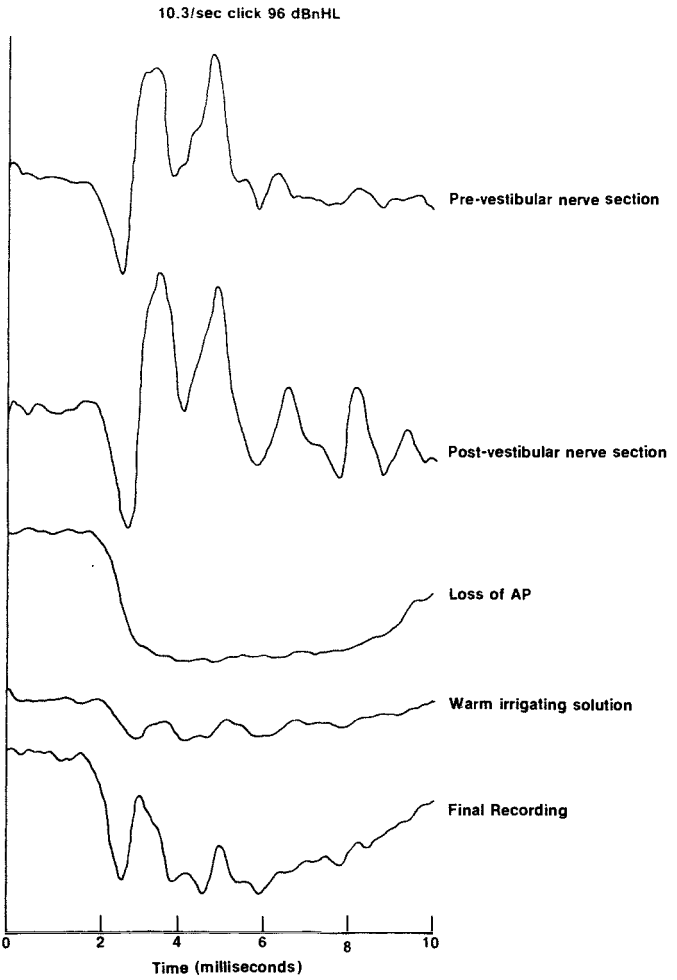

Fig. 3. The CAP recorded directly from the cochlear nerve disappears abruptly and without warning after manipulation of the nerve in the course of vestibular neurectomy. Cessation of the operation at that point was followed by return of the CAP, and the patient had serviceable hearing postoperatively

Discussion

The overall success rate of 93% for obtaining either ECoG, direct eighth nerve action potentials, or both, establishes the reliability of the methodology reported. We record ECoG from the middle ear promontory in all patients, and direct intracranial recording from the cochlear nerve is carried out whenever possible. During the operative exposure we have to rely on the promontory recording. Thereafter, we can obtain direct intracranial recordings during cranial nerve pro-

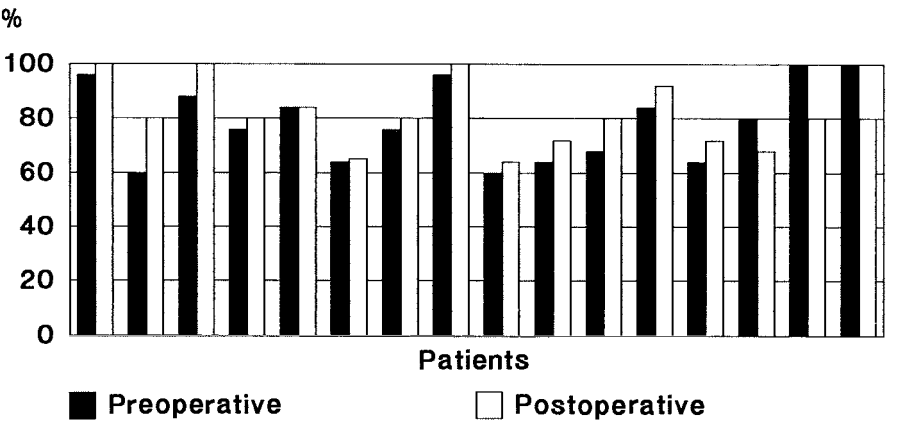

Fig. 4. Pre- and postoperative speech discrimination scores of 16 acoustic neuroma patients whose cochlear CAP and thresholds changed little intraoperatively

cedures in most instances, whereas in acoustic neuroma patients we are often unable to obtain direct recordings because of difficulty with electrode placement.

We would prefer to have both promontory and cranial nerve recordings in all cases, but are not able to accomplish this. Nevertheless, either response is consistent and easy to interpret, and eighth nerve CAP monitoring has been more reliable in our hands than is monitoring of ipsilateral brainstem auditory evoked potentials (BAEP), even though others report BAEP monitoring to be equally reliable (Schramm et al. 1988). Certainly BAEP have been more widely used than cochlear compound action potentials (CAP) for monitoring of acoustic function intraoperatively (Schramm et al. 1988; Grundy et al. 1981; Jannetta et al. 1984). Peaks I and II of the BAEP appear to receive contributions only from the cochlear nerve (Mller, 1988; Mller et al. 1982). Monitoring BAEP also has the theoretical advantage of allowing one to follow events in the more central part of the auditory pathway in the brainstem, but, at least during removal of smaller tumours, this is probably not particularly important.

Although the results that are available in the literature are not uniformly supportive of our impression that cochlear CAP changes are a more sensitive indicator than BAEP changes of injury to the cochlea/eighth nerve complex (Levine et al. 1984), most reports suggest that this is the case (Ojemann et al. 1984).

We believe that the greatest potential advantage of cochlear CAP over BAEP is the detection of changes quickly. The relatively high amplitude CAP can be seen without averaging or with summation of only a few sweeps so that changes are apparent in virtually real time, allowing correction of potentially reversible perturbations (Rowed et al. 1988; Levine et al. 1984; Møller and Jannetta 1984). As indicated by our results, this appears to have been possible on occasion (Fig. 3), an observation reported also by others (Levine et al. 1984; Ojemann et al. 1984).

In other instances, manipulation of the eighth nerve has been stopped when changes in the CAP are first apparent, and the changes have progressed either to

complete disappearance or to marked amplitude decrease with major thresnold shift. This has usually occurred when a tumour capsule is dissected from the eighth nerve. Lateral-to-medial stretching of the nerve is known to be particularly dangerous, damaging the nerve either by injury to the anterior internal carotid artery (AICA)/internal auditory artery complex, with hemorrhage or ischemia, or causing axon interruption (Sekiya et al. 1985; Sekiya and Møller 1987a).

In some cases, as we have indicated, changes in the CAP have occurred during times when no surgical manipulations took place, making correlation with intraoperative events less certain. The case in which disappearance of the CAP followed soon after coagulation of the capsular artery makes ischemia to the eighth nerve or cochlea a likely explanation of the loss of CAP, but an explanation that we cannot confirm. Delayed ischemia is believed to occur, and a process akin to the no-reflow phenomenon (Ames et al. 1968) has been suggested to account for the irreversibility of CAP changes (Sekiya et al. 1985). Other investigators have also reported difficulty in correlating operative manipulations with changes in the action potential in some instances (Levine et al. 1984). This is probably due to more than one mechanism of eighth nerve injury being possible (Levine et al. 1984; Sekiya et al. 1985; Sekiya and Møller 1987a, b).

There appears to be little doubt that the presence of an eighth nerve CAP at the conclusion of the operation, without a major shift in threshold, is an accurate prognostic indicator for serviceable postoperative hearing as shown by pre- and postoperative SDS (Fig. 4), and that, conversely, the intraoperative disappearance of the CAP accurately predicts absence of hearing postoperatively. Similar results have been reported by others (Silverstein et al. 1985a).

Delayed deterioration of hearing, that occurring after the immediate postoperative period, has been reported by others (Levine et al. 1984; Palva et al. 1985) but has not occurred in our experience.

Whether hearing conservation in operations on acoustic tumours is made more successful by intraoperative cochlear monitoring is uncertain. Certainly we are not able to state from our own experience that intraoperative monitoring improves the success rate. We have not randomized the selection of patients for monitoring, but our success rate, as we have shown, is not significantly better since the advent of monitoring than it was previously. Most other groups reporting the results of intraoperative monitoring during acoustic neuroma surgery have also been unable to conclude that success is improved by monitoring (Schramm et al. 1988; Silverstein et al. 1985a; Mller and Jannetta, 1984; Levine et al. 1984), although all are agreed that, at least in some instances, the rapidly available feedback from intraoperative monitoring of cochlear potentials is of value in protecting the nerve.

As regards reporting of postoperative hearing results, we agree with Silverstein et al. (1986) that detailed audiometric data must be presented and that a uniform classification of results is desirable. Most reports utilize similar criteria (Silverstein et al. 1986; Rowed et al. 1988; Cohen et al. 1986), although in some cases the audiometric criteria for preservation of hearing would not generally be considered to represent serviceable hearing postoperatively (Ojemann et al. 1984).

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Monitoring in Operations in the Posterior Fossa

Somatosensory Evoked Potential Monitoring in Tumor and Brainstem Surgery

A. WITZMANN[1], H. BERAN, A. HUBER, and J. FISCHER

Summary

This chapter describes general observations made and conclusions drawn regarding the prognostic value of intraoperative SSEP monitoring during tumor and brainstem surgery. Detailed analysis of the results of monitoring and neurological status evaluations of 97 patients suffering from tumors (cerebral hemisphere: N = 45; midline: N = 13; posterior fossa: N = 21; base of the skull: N = 6), or from cerebellar hemorrhage and infarction (N = 4), or undergoing functional neurosurgical procedures in the posterior fossa (N = 8) revealed recovery of central conduction time (CCT) at the end of surgery to be the most important prognostic factor for postoperative neurological status. Patients whose neurological status was the same as preoperatively (N = 79) did not show any significant change in CCT at the end of surgery compared with the CCT patterns after anesthesia induction. Patients whose neurological status deteriorated postoperatively (N = 18) showed significant CCT prolongation or even loss of the N_{20} component at the end of the procedure. When patients were grouped according to whether they had undergone infratentorial surgery, hemisphere tumor removal, or midline tumor removal, to determine if there were differences in CCT that were related to differences in lesion locations, the results were similar. Changes in amplitude of the N_{20} component also had prognostic value, but such changes were less significant because they were very variable and thus standard deviations were large.

To obtain additional insight into whether the location of the tumor in the hemisphere and the extent of the tumor affected the prognostic value of SSEP, 17 patients with hemisphere tumors were studied. Distances from the tumor midpoint to the central sulcus, distance to the midline, and to the base of the skull, and the width and length of the tumor were correlated with SSEP patterns to determine what relationship these had to prognosis. CCT recovery was found to be the only factor that differed significantly between patients whose postoperative neurological status was unchanged and those whose neurological status deteriorated postoperatively. The predictive capacity of CCT recovery was calculated by means of discriminant analysis, which revealed that CCT recovery is a better indicator of prognosis (15 correct classifications) than were all of the tumor

[1] Abteilung für Neurochirurgie, Wagner-Jauregg- Krankenhaus, Wagner-Jauregg-Weg 15, A-4020 Linz, Austria.

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parameters together (14 correct classifications). These findings suggest that SSEP monitoring is not only useful in predicting neurological function during removal of lesions located in a specific somatosensory area, but also during removal of lesions remote from this area. The reasons are discussed in this chapter.

Tibial nerve evoked SSEP were found to be less sensitive and less prognostic of injury occurring during intracranial surgical procedures than were median nerve evoked components. The former do, however, provide important additional information, especially during surgical procedures that involve midline structures.

Recently, we have obtained bilateral frontal recordings simultaneously with bilateral parietal recordings in response to median nerve stimulation. Our experience with this type of intraoperative SSEP recording has been limited to date because of the small number of patients (6 with brain tumors and 4 with aneurysms) qualifying for this study. For all patients, postoperative neurological status was the same as preoperative status, and in all patients stable frontal as well as parietal components were found. In one patient with a glioma of the right posterior frontal lobe extending into the prerolandic area, the P_{22} component was lost during dissection of the posterior parts of the tumor. Dissection was discontinued and the P_{22} component completely recovered. The patient awoke without any signs of hemiparesis.

Intraoperative monitoring of evoked potentials (EP) is used in various kinds of surgical procedures involving contact with nervous tissue, in particular, operations on the spine or spinal cord and on peripheral nerves, and intracranial surgical procedures (Møller 1988; Desmedt 1989). Neuromonitoring of EP has been performed during carotid endarterectomy (Markand et al. 1984; De Vleeschauwer et al. 1985), open heart surgery (Coles et al. 1984), and interventional radiology procedures (Hacke et al. 1985). The main goal of intraoperative monitoring of EP is to detect when an injury or functional disturbance of the nervous tissue has resulted from surgical manipulations so that steps may be taken to prevent permanent neurological deficits. To achieve this goal, modalities for stimulation and recording must be chosen so as to best indicate the status of the part of the nervous system the surgical procedure affects. Visual evoked potentials (VEP), although they reflect the function of visual pathways, to date have proved inappropriate for intraoperative monitoring of these pathways because the results of monitoring are highly variable and may not accurately reflect clinical status (Cedzich et al. 1988; Schramm 1989). Brainstem auditory evoked potentials (BAEP) are widely used to monitor neural function during infratentorial surgery, including tumor removal, and other neurosurgical procedures in which nerve function may be affected (Grundy et al. 1982; Raudzens and Shetter 1982; Fischer 1989). Somatosensory evoked potentials (SSEP) are the EP most widely used; they are used intraoperatively to evaluate neural function during operations on the spine and spinal cord, carotid endarterectomy, during operations involving cranial and peripheral nerves, open heart operations (Coles et al. 1984), and during different intracranial neurosurgical procedures (Desmedt, 1989). Because of the well-studied and universally accepted correlation between

SSEP changes and alterations in cerebral blood flow (Branston et al. 1974, 1976; Symon et al. 1979), SSEP are usually monitored during neurosurgical procedures to clip aneurysms. SSEP, however, have not been as widely used during removal of tumors and procedures involving the brainstem. However, we have developed a technique for using SSEP to monitor function in these areas, and will now describe our method and the results of its use.

In addition to monitoring SSEP in patients undergoing neurosurgical procedures to manage aneurysms and angiomas, we have monitored SSEP in more than 150 patients operated upon for lesions in the cerebral hemisphere, in the midline, or in the posterior fossa, or for relief of a functional cranial nerve problem.

Methods

All patients were examined neurologically before and after operation by one of us (A.W.). The results of pre- and postoperative examinations were compared with the intraoperative SSEP pattern and conclusions were drawn about the prognostic value of intraoperative changes in the SSEP pattern.

In the first years of our work we obtained SSEP preoperatively from the patients the day before the operation. Later on, we found that baseline recordings to be used intraoperatively were more appropriately obtained after induction of anaesthesia because the SSEP are influenced by anaesthesia. We, therefore, discontinued SSEP recording from conscious patients. However, we recently began recording preoperative EP profiles again for these reasons: 1) the laboratory setting provides the time needed to determine the most suitable recording site in relation to the planned skin incision; 2) patients who have lost response components (i.e., as the result of Rolandic area involvement by a tumor) can be identified; because intraoperative monitoring would not be of any help in these cases, finding this out preoperatively shortens the duration of the operation for these patients; 3) anaesthesia and muscle relaxation hinders optimal placement of stimulating electrodes due to lack of muscle response; when recordings are made preoperatively, the optimal recording sites can be identified and marked on the skin.

At the time of induction of anaesthesia and placement of intravascular lines, puncturing of the radial artery would interfere with median nerve stimulation. The anaesthetist is therefore asked to use the brachial artery for arterial pressure monitoring. After the patient's head has been fixed in position, needle or cup recording electrodes and surface stimulating electrodes are placed on the sites identified during the preoperative recording that was performed in the awake patient the day before. Next, the electrode impedances are checked. Impedances for cup electrodes should be between 1 and 3 kOhm and those for needle electrodes are usually between 3 and 5 kOhm. For 3 months we have been using a 4-channel Axon Sentinel averager which records well-defined traces even with impedances up to 9 kOhm. If recording and stimulating electrode impedances are acceptable, monitoring is begun and is continued until skin closure.

Stimulation

SSEP were elicited by standard transcutaneous electrical stimulation of the median nerve at the wrist or the posterior tibial nerve at the ankle. Stimulation of the peroneal nerve at the knee is increasingly used in our department instead of posterior tibial nerve stimulation because we have been able to elicit better-defined motor twitches in awake patients using lower stimulation intensities with peroneal nerve stimulation compared to posterior tibial nerve stimulation. Further, in many cases the optimal site for recording posterior tibial nerve responses was only a few millimeters away from the optimal stimulation site, and the motor response was inadequate for interpretation. As the stimulation area of the common peroneal nerve is larger, this problem does not arise when the peroneal nerve is used. Contrary to the findings of Chiappa (1983), we have found that the amplitudes of SSEP recorded form the scalp in response to tibial and peroneal nerve stimulation are equal, although the threshold of stimulation for a supramaximal motor response in the anaesthetized patient is twice that in the unanaesthetized patient.

Both median nerves are stimulated simultaneously, but the posterior tibial or peroneal nerve stimulation is performed alternately (right stimulation followed by left) because we use a single electrode placed in the midline to record from both hemispheres. Lately, we have used the following procedure to obtain baseline recordings: 2 recordings are obtained in response to simultaneous median nerve stimulation, then recordings are made to right and left tibial nerve stimulation, respectively. In some cases when a procedure will involve sites near or in the brainstem, BAEP monitoring is also carried out; in these cases a baseline BAEP recording is obtained before median nerve monitoring is begun. This schedule can easily be modified, if required, according to where the surgeon is working. For example, when the surgeon is dissecting lateral portions of a large falx meningioma or a meningioma of the olfactory groove, monitoring is performed with continuous stimulation of both median nerves. As dissection progresses medially, possibly to involve an adherent pericallosal artery, monitoring is performed with continuous stimulation of the contralateral posterior tibial or peroneal nerve.

Another way to modify the monitoring procedure is to reduce the number of sweeps, thus shortening the time between recordings. This modification in technique is particularly helpful when the dissection puts neural structures at great risk of injury because it provides event-related results that enable the surgeon to alter surgical manipulations as soon as the SSEP pattern deteriorates. For example, in one woman we operated upon for a pontine glioma, about half of the tumor was removed with median nerve SSEP monitoring based on averages of 50 responses at a rate of 4.8 per second. When central conduction time (CCT) became prolonged by 2 ms and the amplitude of N_{20} decreased by more than 50% from baseline, dissection was stopped immediately and the SSEP pattern showed no further deterioration until the end of the operation. The patient was kept in a barbiturate-induced coma for 2 days to reduce the risks of cerebral edema; when she awoke she had mild hemiparesis, cranial nerve dysfunction, and dizziness.

These symptoms gradually improved during the postoperative period and finally she was able to walk unaided and to care for herself independently until the tumor recurred. This case emphasizes the importance of close cooperation between the surgeon and those performing intraoperative monitoring. The neurosurgeon must detail which systems are to be monitored and those performing the monitoring must inform the neurosurgeon immediately when the recorded potentials change. Loss of components of the recorded potentials should always be followed by a careful search for the cause of the loss. In many cases of tumor removal, SSEP changes of variable extent have been noted to occur with excess retraction in the direction of the Rolandic area, the thalamocortical fibres, or the thalamus itself. In our experience, CCT prolongation and an interhemispheric difference (IHD) of up to 2 ms do not predict a postoperative neurological deficit, but changes of more than 2 ms should be followed by a search for the causes of the change because of the risks of permanent deficits.

When both median nerves are stimulated simultaneously, recording simultaneously from both hemispheres provides concurrent information about the operated side and the non-operated side. When lower extremity nerves must be stimulated (i.e., to monitor supratentorial midline processes), particular attention must be paid to the results of stimulation of the contralateral nerve. Occasionally it is useful to monitor ipsilateral responses as well, however, as they indicate the patient's overall status and are needed to calculate the IHP. Thus, bilateral monitoring may be advantageous even during periods when the nature of the operation would seem to place only one side at high risk of injury.

Recording

Standard recording sites are used. Needles or cup surface electrodes placed 2 cm behind C3, C4 (C3′, C4′/Fpz) are used for recordings of the response to median nerve stimulation, and Cz (Cz′/Fpz) are used when lower extremity nerves are stimulated. When these placements would interfere with the skin incision (i.e., when a mass is to be removed from the temporal or parietal region), the skin incision is relocated, as described in detail elsewhere (Witzmann and Reisecker 1989). One electrode is placed at the spinous process of the C7 vertebra and the reference electrode is placed at Fpz. We prefer the C7 site instead of C2 because the N_{13} component in a C2 recording is likely to overlap the N_{14} component (Prior and Maynard 1986). This is because N_{13} is generated in the dorsal horn of the cervical spinal cord (Desmedt and Nguyen 1984) and N_{14} reflects summation of synaptic activity and the lemniscal volley in the brainstem (Desmedt and Cheron 1980). Also, C7 derivation is more remote from scalp incisions over the posterior fossa.

We recently began recording from additional, frontal sites (F3, F4/sternum) in patients being operated upon for frontal or pre-Rolandic lesions. As was the case for parietal recording sites, when the F3, F4 position interferes with placement of the skin incision another recording site is chosen. A noncephalic reference is necessary to obtain well-defined components. We prefer a sternal reference, because this location is convenient and easily accessible during the operation. The

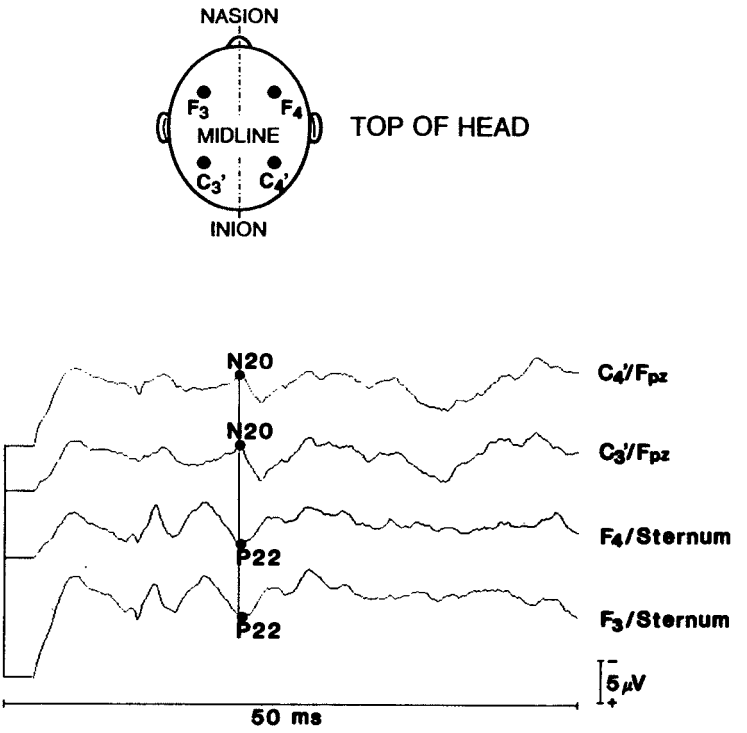

Fig. 1. Bilateral simultaneous median nerve stimulation and resultant simultaneous 4-channel scalp recording in an unanaesthetized person without neural deficits. The N_{20} component was recorded at C3′ and C4′ with a reference at Fpz. The P_{22} component was recorded at frontal sites with a sternal reference

use of a noncephalic reference leads to considerable contamination with artifacts, but we have been able to obtain continuous recordings of clearly defined and reproducible components in the overwhelming majority of cases by careful fixation of electrodes with adhesive tape, covering the recording electrode sites with double layers of self-adhesive drapes, twisting the cables together, ensuring that the electrocautery apparatus is well-shielded, and grounding all machines. To day, we have used frontal recordings in relatively few cases, and we omitted tibial nerve stimulation and Cz' recording. We recorded scalp SSEP simultaneously on 4 channels, from C3′, C4′/Fpz, F3, and F4/sternum in response to bilateral median nerve stimulation (Fig. 1). This type of recording cannot be performed in conjunction with C7 recordings, because most commercially available recording apparatuses offer no more than 4 channels. However, not being able to record from C7 is not a major disadvantage because in our experience the N_{13} latency and amplitude change only minimally and insignificantly during the operation. Thus, latency changes of cortical components are very closely correlated to CCT changes. Furthermore, when scalp SSEP are recorded simultaneously from the operated and nonoperated hemispheres it is possible to detect an IHD

immediately. As we show later in this paper, an increase in the IHD indicates a disturbance in cerebral function in almost the same way as does CCT prolongation on the operated side.

Monitoring Parameters

During the first years of our work, a 2-channel Interspec Neurotrac averager was at our disposal. The CCT (calculated from the latency of N20 and of the cervical N13 of the median nerve SSEP; Hume and Cant 1978), as estimated for the operated side, and the $N_{20} - P_{27}$ amplitude were the only parameters that could be estimated in the response to contralateral median nerve stimulation. Later on, we began to use a 4-channel Nicolet CA 2000 for monitoring. When this averager was used, electrodes were placed at each stimulation or recording site. First, we recorded bilaterally from the parietal region and C7 to bilateral simultaneous median nerve stimulation, either alone or alternating with Cz'recording in response to lower extremity nerve stimulation. This arrangement allowed for monitoring of the nonoperated as well as the operated side, and thus also permitted estimation of IHD in CCT and amplitudes. Then, by interchanging the cables to the preamplifier and stimulating lower extremity nerves instead of the median nerve, we could record posterior tibial or peroneal nerve evoked potentials and estimate P_{40} latency and $P_{40} - N_{50}$ amplitude.

For the last 3 months we have used an Axon Sentinel recording equipment with 4 channels. The curves are displayed on a screen that, when all 4 channels are used, is divided into 4 columns. Each column displays the responses from one channel. N_{20} and P_{22} latency and amplitude values for each hemisphere are, therefore, displayed simultaneously.

General Intraoperative SSEP Observations

The induction of anaesthesia using 150–250 mg thiopental and 80 mg succinylchloride almost always leads to N_{20} latency delay and amplitude depression compared with preoperative recordings. The impact of anaesthetic agents on later SSEP components has been well documented (Abrahamian et al. 1963; Clark and Rosner 1973). Although the influence of most narcotic agents on subcortical components and even on the $N_{20} - P_{27}$ complex is much less profound (Grundy 1982; Sutton et al. 1982; Reisecker and Witzmann 1984; Drummond et al. 1985), bolus injection of thiopental or other anaesthetics leads to at least minor latency delay and amplitude depression. After induction, bolus injections of anaesthetic agents should, therefore, be avoided (Grundy 1982). Anaesthesia is maintained by 0.5–1.5% isoflurane after induction. On this dosage schedule, SSEP recover after 20–30 minutes to near the levels seen in the conscious patient. Despite high variability among individuals (Witzmann et al. 1990) the SSEP pattern remains constant in a given patient under this anaesthesia regimen.

Confounding components that appear in SSEP recorded after induction of anaesthesia are often the result of artifacts. Alternating current artifacts, in par-

ticular, are likely to occur when electrodes are not well secured or electrode cables are allowed to move. Thus, electrodes are fixed firmly in place, watertight drapes are secured over recording electrodes, and the operative field is cleaned with an alcohol solution and draped before the baseline recording is made. These preparations can usually be completed in about 30 minutes after induction of anaesthesia.

SSEP patterns exhibit only minimal fluctuations until the beginning of the "main procedure" as we call the operative maneuvers that are the aim of the operation, i.e., tumor removal, vessel dissection, or insertion of the sponge in cranial nerve decompression. The amplitude of the various components of the SSEP may decrease and/or CCT become prolonged. Sometimes, the SSEP waveform becomes so distorted that identification of components is not possible. When this happens, the operative maneuver that preceded the change is modified in an attempt to elicit SSEP recovery, which may occur after a period ranging from a few seconds to more than 1 hour. In some cases, SSEP do not recover at all. There seems to be no clear correlation between the type and duration of SSEP changes during the main procedure and the clinical outcome. On the other hand, we gained the impression that there was a correlation between the SSEP pattern observed at the end of the operation and the patient's clinical outcome.

Prognostic Value of SSEP Monitoring

To obtain more detailed information about just how reliably changes in SSEP during neurosurgical procedures predict postoperative function, we analyzed retrospectively the intraoperative SSEP patterns of 97 patients and compared these patterns with the patients' neurological status postoperatively (Witzmann and Reisecker 1989). These patients underwent operations to manage tumors (cerebral hemisphere, N = 45; midline, N = 13; posterior fossa, N = 21; base of the skull, N = 6), cerebellar infarction and haemorrhage (N = 4), or functional defects in the posterior fossa (N = 8). We actually evaluated recordings from 116 patients, not including those operated on for aneurysm or angioma, but only 97 patients' results could be used in this study. This was because only artifact-free traces could be used, and only when monitoring was continuous and surgical or anaesthesiological events were described and related in time to the events on the recording could we infer how surgical or anaesthesiological events affected the SSEP.

Median Nerve Stimulation

All 97 patients underwent contralateral median nerve stimulation and ipsilateral scalp and C7 recording. Some patients also underwent bilateral median nerve stimulation with bilateral scalp recordings, alternating tibial nerve stimulation with Cz′ recording, and BAEP monitoring. In 79 patients whose postoperative status was the same as their preoperative status, no significant difference was seen between CCT baseline values and CCT values at the end of the operation. The

same held true for their "R values" (amplitude N_{20} – P_{27}/amplitude N_{13}), although 1 of these 79 patients had an inexplicable loss of N_{20} that persisted to the end of the operation. Neurological deterioration occurred in 18 patients. The mean CCT values in 13 of these patients were significantly prolonged at the end of the operation compared to their baseline values. R values were also considerably decreased in these cases, with the N_{20} – P_{27} complex being lost altogether in 5.

To study differences in how SSEP recordings were affected by the operative site, we made separate evaluations of the SSEP recorded from patients undergoing operations for infratentorial lesions, hemisphere tumors, and midline tumors. The mean CCT values and R values were determined when the operation started and at the end of the operation (Figs. 2 and 3). Neither CCT nor R values differed significantly as a function of operative site for patients whose neurological status postoperatively was unchanged from their preoperative status. On the other hand, in patients whose neurological status had deteriorated postoperatively CCT were significantly prolonged intraoperatively. This prolongation was seen for all operative sites, although not with the same frequency. In the midline tumor group, only 1 patient suffered a decrease in neurological status during the operation. This patient had, however, much greater CCT prolongation at the end of the operation than the mean value of the 21 patients in this group whose neurological

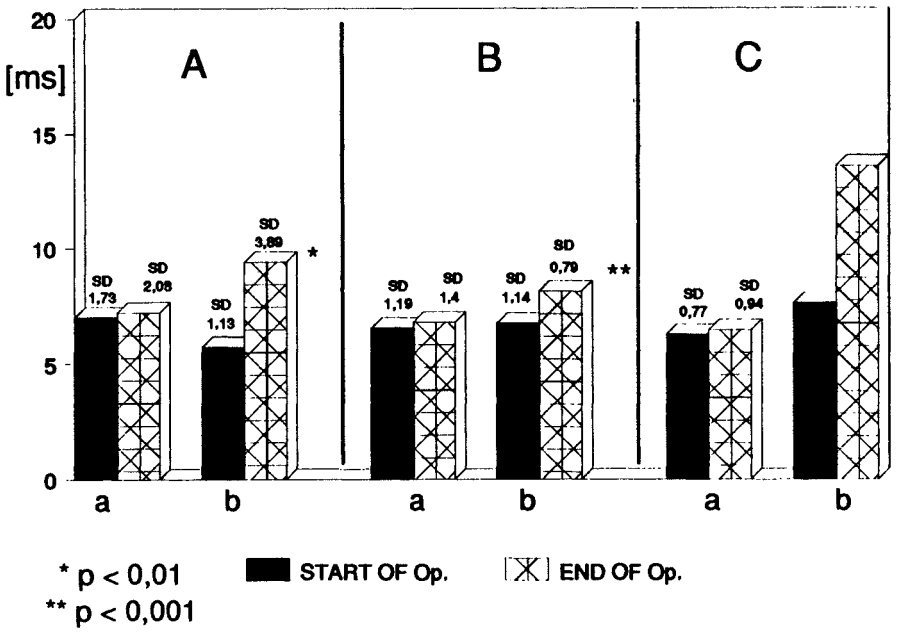

Fig. 2. Central conduction time (CCT) (mean values) for the operated side, measured intraoperatively in operations for infratentorial lesions (N = 30) *(A)*, hemisphere tumors (N = 45) *(B)*, and midline tumors (N = 22) *(C)* for patients whose neurological status was unchanged after the operation *(a)* and patients whose neurological status deteriorated after the operation *(b)*

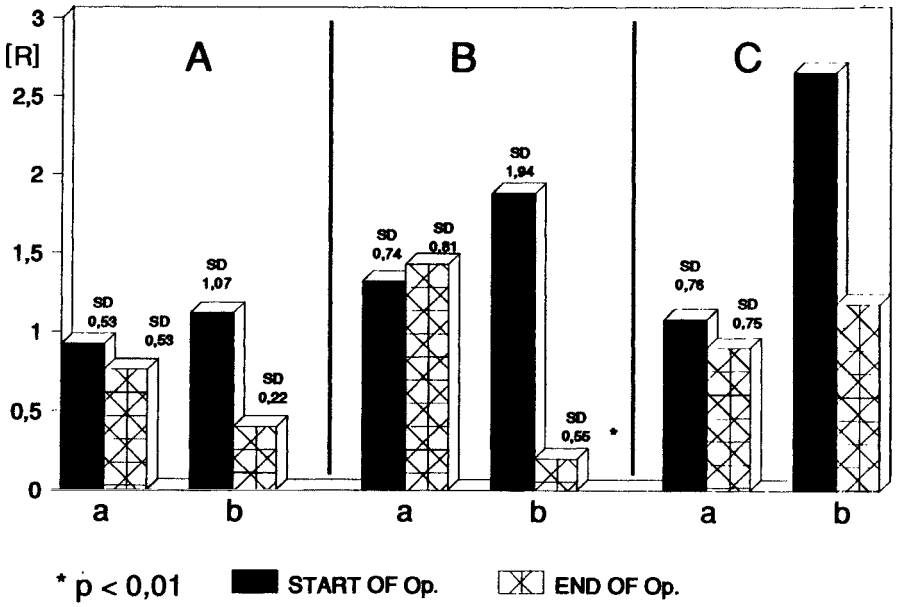

Fig. 3. R value (amplitude N_{20} – P_{27}/amplitude N_{13}; mean values) during surgery for infratentorial lesions (N = 30) *(A)*, hemisphere tumors (N = 45) *(B)*, and midline tumors (N = 22) *(C)* for patients whose neurological status was unchanged after the operation *(a)* patients whose neurological status deteriorated after the operation *(b)*

status remained unchanged. The amplitudes (R values) for patients in this group showed similar changes, but the prognostic value of these changes in amplitude was, however, diminished by their great variability, as evidenced by the large standard deviations for the patient with changed and those with unchanged neurological status.

The question arises whether the prognostic value of intraoperative SSEP monitoring differs according to localization and extent of hemisphere tumors. In other words, might the site and extent of at least some of these lesions have a greater impact on the prognosis than the intraoperative changes in SSEP pattern? To investigate this, we studied 17 patients suffering from tumors in various areas of the cerebral hemisphere (Witzmann et al. 1987, 1990). The number of patients studied was limited to those who had CT scans performed in our hospital, a limitation imposed by the software we use to evaluate tumor localization and extent. The tumor parameters (determined by our computer programs) were distance from the tumor midpoint to the central sulcus, distance from the tumor midpoint to the midline, and distance from the tumor midpoint to the base of the skull. Tumor size was also determined using computer programs. Next, we calculated the change in CCT and R values between the end and the start of the operation (Δ CCT1, Δ R1), and also the differences in CCT and R values between the end and the most substantial changes during the main procedure (Δ CCT2, Δ R2). Analysis of variance was performed to determine if these changes were signifi-

cantly different for patients whose postoperative neurological status had deteriorated versus those whose status was the same. The only value that differred significantly between these two groups of patients was Δ CCT1.

Discriminant analysis (Donchin and Herning, 1975; Weber, 1973) was performed in order to test how well tumor parameters or intraoperatively recorded SSEP predicted the postoperative changes in the patients' neurological status. Using tumor data and SSEP parameters together, all 17 patients were correctly classified. SSEP results as a whole classified 15 patients correctly and tumor parameters as a whole classified 14 patients correctly.CCT1 alone classified 15 patients correctly, thus a high level of predictability no other single parameter achieved. These results demonstrate the importance of CCT recovery for outcome.

Recently, we analyzed IHD in CCT in another 22 patients (Fig. 4). In these patients the tibial nerve was stimulated in addition to the median nerve. The patients were operated upon for hemisphere tumors (N = 11), midline tumors (N = 5), posterior fossa tumors (N = 5), or facial nerve decompression for hemifacial spasm (N = 1). Twenty of the 22 patients had unchanged neurological status and 2 had deteriorated status after the operation. There was only small shifts of the IHD from baseline during the main procedure in the patients whose neurological status was unchanged postoperatively, but complete recovery was observed

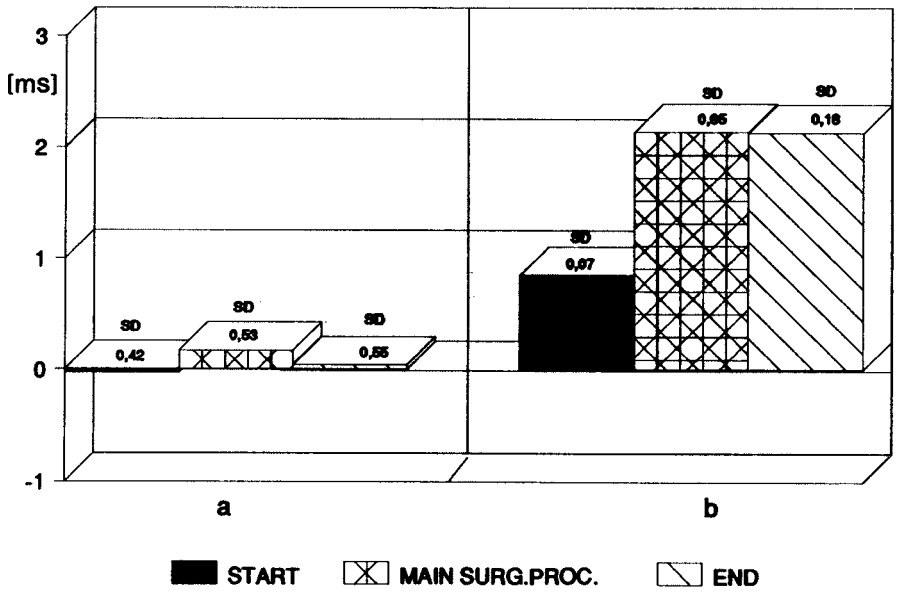

Fig. 4. Intraoperative interhemisphere differences in central conduction (CCT) time for 22 patients with hemisphere tumors (N = 11), midline tumors (N = 5), posterior fossa tumors (N = 5), or undergoing facial nerve decompression (N = 1). *a*, mean values for patients whose neurological status was unchanged after the procedure (N = 20); *b*, mean values for patients whose neurological status deteriorated after the procedure (N = 2)

at the end. The 2 patients whose neurological status deteriorated postoperatively both suffered from large hemisphere tumors and had remarkable IHD, even at the beginning. This finding was in accordance with hemiparesis of some degree noted on preoperative examination. Removal of the mass led to further increase in these patients' IHD, which did not recover at the end of surgery. These findings underline the fact that IHD changes have almost the same significance as do CCT alterations recorded from the operated side.

Our experiences in the use of results of frontal recordings to predict postoperative neurological status are limited, because we have so far only performed this type of monitoring in 6 patients suffering from brain tumors and 4 patients with aneurysms. These patients did not have any signs of severe hemiparesis preoperatively and all had unchanged neurological status after the procedure. In all patients frontal as well as parietal SSEP components were unchanged after the operation. In 1 patient with a glioma of the right posterior frontal lobe extending into the precentral area, the P_{22} component was lost during dissection of the posterior parts of the tumor. The N_{20} component recorded parietally remained unaffected (Fig. 5). Because of the risk of postoperative hemiparesis, dissection was discontinued and the P_{22} component had recovered completely at the end of the operation. The patient had no neurological deficit after surgery.

Posterior Tibial Nerve Stimulation

In an earlier study we examined 18 patients who had undergone posterior tibial nerve stimulation in addition to median nerve stimulation (Witzmann and Reisecker 1989). These patients had had hemispheric tumors in regions supplied by the pericallosal artery (N = 7) or tumors at the brainstem level (N = 9). Two patients underwent tractonucleotomy. All patients had the same neurological function after the operation as before. We looked for changes in P_{40} latency, and during the main procedure SSEP changes were noticed in 16 patients. In 9 patients these changes reversed, but in 7 patients the P_{40} latency delay had not recovered completely at the end of the operation. Loss of components was not observed in any patient. These findings indicate that lower extremity SSEP are useful when recorded to median nerve stimulation. Such SSEP seem to be, however, less sensitive than median nerve evoked potentials to changes that may cause neurological deficit, as their latency delay has a wider range than N_{20} latency delay or CCT prolongation. These findings are in accordance with studies performed in conscious patients (Vogel and Vogel 1982).

The IHD of the P_{40} component in the 22 patients in whom also the median nerve was stimulated (Fig. 4) are shown in Fig. 6. During the main procedure, IHD rose slightly and recovered at the end of the operation in the 20 patients whose neurological status did not change. The 2 patients who had deteriorated neurological status postoperatively had a higher P_{40} IHD than CCT elevations before the procedure even began. The further rise in P_{40} IHD that occurred during the main procedure was, on the other hand, less than the increase during this time in CCT IHD. The P_{40} IHD further increased slightly from the main procedure to the end of the operation. These findings again support the assumption

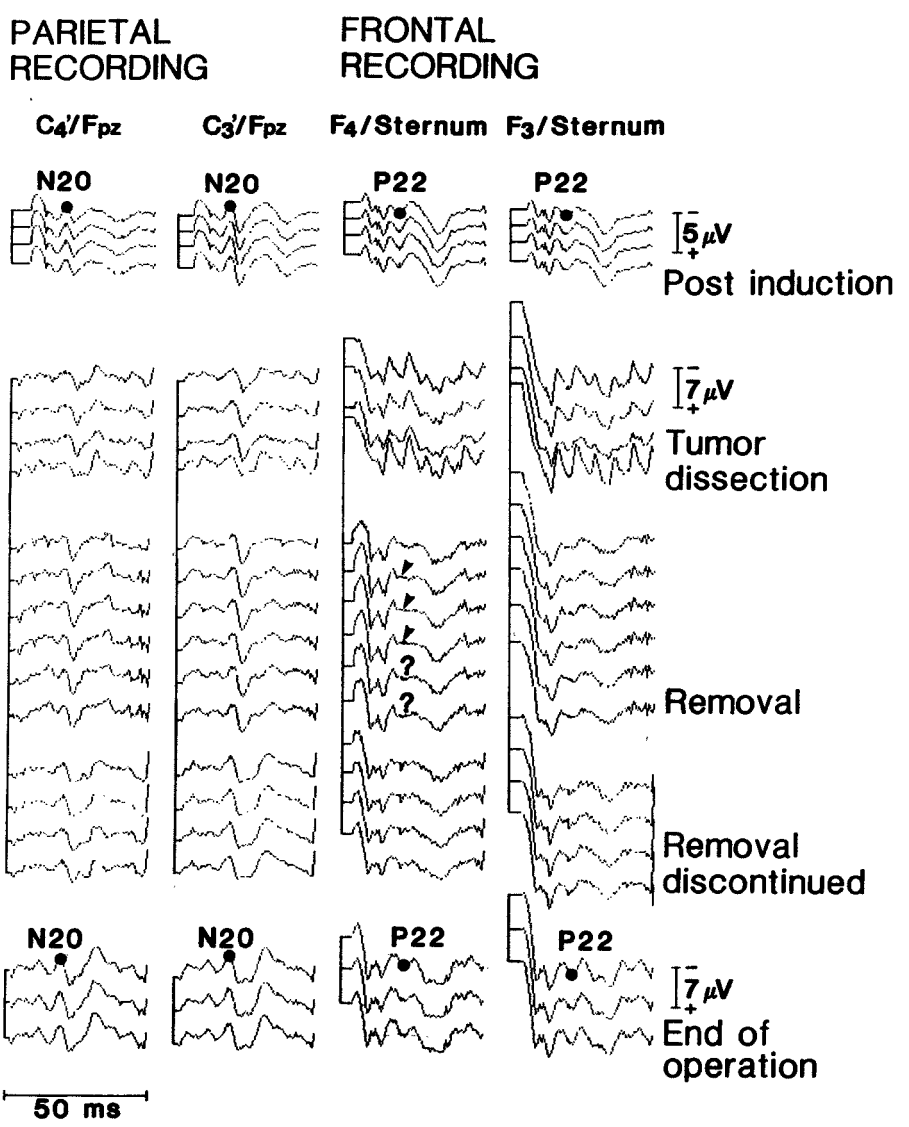

Fig. 5. SSEP patterns during removal of a large, right-sided frontal glioma. The median nerves were stimulated bilaterally simultaneously and responses were recorded simultaneously from the parietal and frontal scalp over both hemispheres. Note loss of P_{22} over the operated side during tumour removal. After resection was discontinued, the P_{22} component was indistinct for a time (arrows indicate P_{22} loss). At the end of surgery, the P_{22} component reappeared. As is evident in the parietal recordings, components in both hemispheres remained unaffected by tumour removal. The left frontal recordings show that P_{22} was unchanged during the entire operation. The patient awoke without signs of neurological deficit

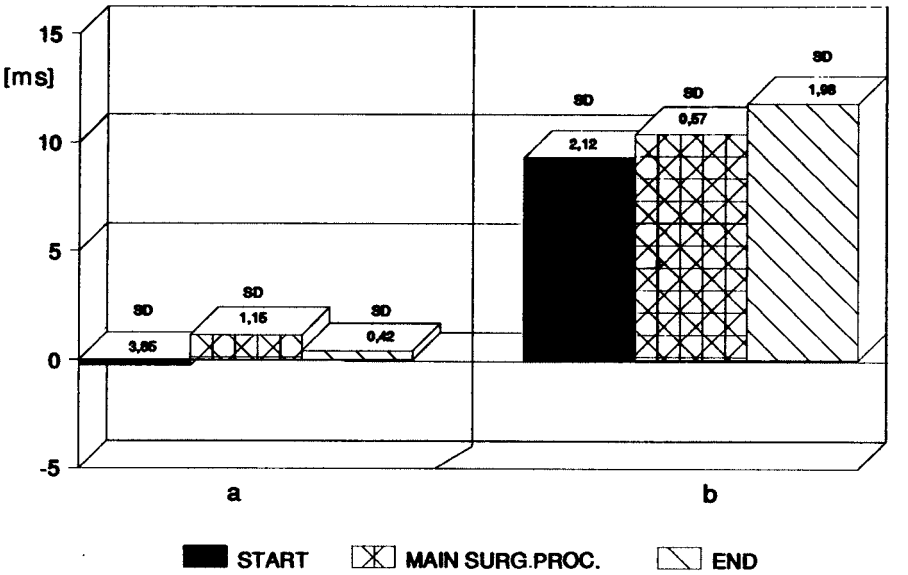

Fig. 6. Intraoperative differences of interhemispheric P_{40} for the same patients whose results are shown in Fig. 4. *a*, mean values for patients whose neurological status was unchanged after surgery (N = 20); *b*, mean values for patients whose neurological status deteriorated after surgery (N = 2)

that the responses to lower extremity nerve stimulation are less sensitive and have less prognostic power than those to median nerve stimulation during intracranial procedures. In some instances, however, monitoring lower extremity SSEP provides important additional information.

Fig. 7 shows the SSEP evoked by median nerve and posterior tibial nerve stimulation during removal of a left-sided acoustic neuroma. Both the median nerve and the tibial nerve SSEP remained unchanged during the procedure, and the patient awoke without neurological deficit.

The patterns of median nerve and posterior tibial nerve SSEP recorded during removal of a large left-sided falx meningioma with severe brain edema are shown in Fig. 8. The patient had right-sided hemiparesis, particularly affecting right leg function, preoperatively. Both the N_{20} and the P_{40} components were pathologically delayed before the start of the operation. The N_{20} component did not change significantly during the entire procedure, but P_{40} was lost after the mass was removed. This coincided exactly with a sudden increase in brain edema. After the operation the patient had paralysis of the right leg.

Discussion

There are relatively few studies that describe using intraoperative monitoring of SSEP in large series of operations to remove tumors or in other surgical proce-

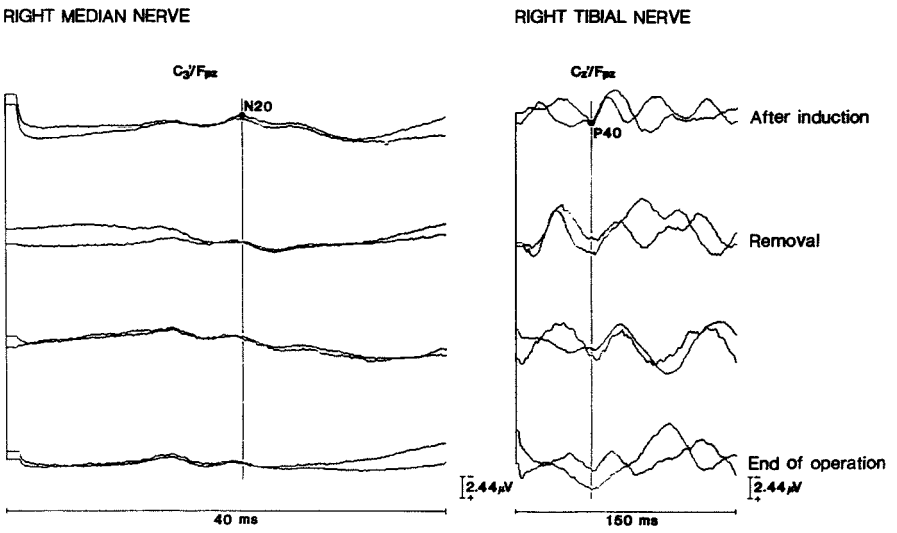

Fig. 7. Removal of left-sided acoustic neuroma. Patterns of median nerve and posterior tibial nerve SSEP on the operated side remained unchanged during the entire procedure. This patient had an uneventful postoperative course

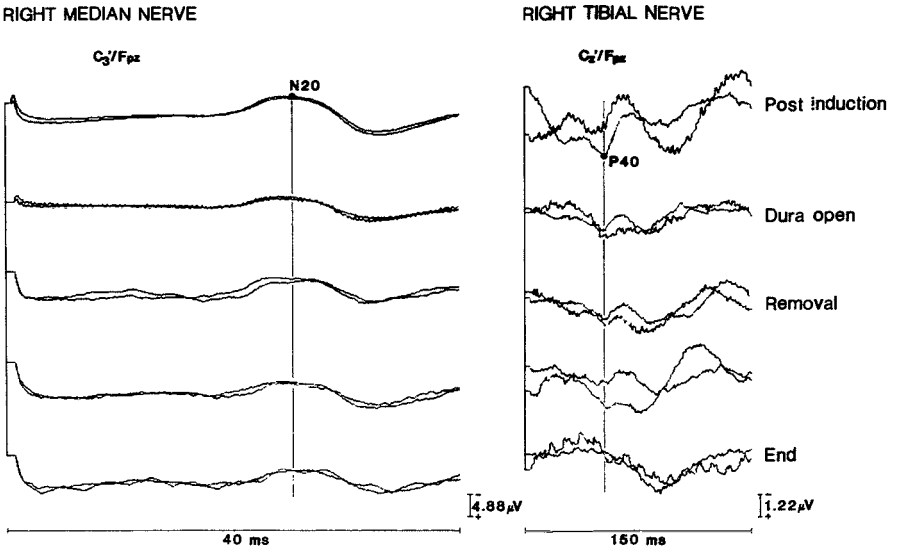

Fig. 8. Median nerve and posterior tibial nerve SSEP pattern over the affected side during removal of a large left falx meningioma. A right hemiparesis (worse for the leg) was present preoperatively. Pathological delay of both the N_{20} and the P_{40} components was present before the start of the operation. N_{20} was unchanged during surgery. Loss of P_{40} at the end of the procedure was due to edema. The patient awoke with complete paralysis of the right leg

dures in or around the brainstem (Gentili et al. 1985; Witzmann et al. 1985, 1987, 1990). There are obvious reasons why intraoperative monitoring of SSEP has found limited use in operations in and around the brainstem. Thus, because the neural generators of the BAEP are located in the brainstem, intraoperative monitoring of BAEP has been the technique of choice in operations in which the brainstem is involved. In a series of 10 patients who underwent operations to remove infratentorial tumors (Witzmann and Reisecker 1989), loss of waves III and V of the BAEP without recovery occurred in 2 patients whose neurological status deteriorated after the operation. Latency changes and interpeak latency prolongations were seen in all of the 8 patients who had unchanged neurological status postoperatively. Thus, only the loss of 1 or more components of wave I to V seemed to indicate a poor prognosis. Although BAEP monitoring can provide more useful results when performed in large series of patients by experienced staff (Fischer 1989), helpful information can be obtained, even for patients undergoing operations for posterior fossa lesions, when BAEP and SSEP are monitored alternately (Schramm 1989).

The situation is somewhat different regarding supratentorial tumor removal. A large number of reports have documented that SSEP abnormalities in conscious patients can be related to exactly defined morphological lesions involving specific sensory pathways and synapses (Noel and Desmedt 1975; Mauguiére et al. 1983a,b). However, morphological lesions in areas other than specific sensory areas do not, as a rule, lead to EP alterations, although there have been a few reports of SSEP abnormalities associated with focal brain lesions remote from the primary sensorimotor areas (Obeso et al. 1980; Reisecker et al. 1986). One may, therefore, assume that the information obtained from intraoperative monitoring of SSEP during removal of a mass lesion that is not localized to a specific sensory area will not adequately reflect actual brain function. Furthermore, the fact that lesions involving specific pathways as well as cortical and subcortical gray matter may affect the SSEP preoperatively does not mean that SSEP will change intraoperatively when these regions are injured by surgical manipulations, or that the intraoperative changes may be interpreted to show such injury. One should, however, not forget that intraoperative monitoring is a dynamic situation, quite distinct from the static situation encountered in diagnostic testing of the awake patient. During operations the brain may be disturbed in several ways. Thus, removal of an expanding mass deep in the frontal white matter might be accompanied by applying pressure on surrounding brain tissue by retraction. This could affect the thalamocortical fibres or the internal capsule. Removal of a mass in the opercular region may require retraction in a posterocentral direction, which could result in compression of an important branch of the middle cerebral artery. All these events are likely to lead to SSEP alterations that signal brain dysfunction and imminent neurological deficit.

The findings of our studies indicate that permanent intraoperative loss of the $N_{20} - P_{27}$ components is a sensitive predictor of neurological deficit. Persistant CCT prolongation and/or an IHD of more than 2 ms can be followed by at least temporary hemiparesis in the postoperative period, especially when amplitude depression is also present. One of our patients had, however, N_{20} loss without

neurological deficit. On the other hand, hemiparesis may occur despite a lack of change in SSEP, because SSEP recorded from the parietal scalp reflect the function of the sensory system – the motor system is only monitored indirectly. The best solution to this problem is, of course, to monitor the motor tracts by means of cortex stimulation. There is, however, strong evidence that the frontal recorded P_{22} component has a neural generator in the pre-Rolandic area, so that the P_{22} component represents directly the response from the motor cortex (Mauguiére et al. 1983a; Desmedt and Bourguet 1985; Rossini et al. 1987). Thus, performing frontal recordings with noncephalic references and parietal leads may provide important information about pre-Rolandic and thus be of value when monitoring motor disturbances. The preliminary results in our small series of patients who underwent intraoperative monitoring using frontal recordings are encouraging, but further studies and larger series are necessary. Lower extremity SSEP show somewhat greater fluctuation than do upper limb SSEP. The fluctuations in SSEP elicited by stimulation of lower extremities are not always associated with severe brain dysfunction. Nevertheless, this type of monitoring provides important information, especially during operations to remove midline tumors.

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Intraoperative Monitoring of Cranial Motor Nerves

J. R. DAUBE[1]

Summary

Multimodality recordings from a combination of various structures provide a new and effective method to reduce morbidity associated with intracranial or extracranial surgery in the region of the third and twelfth cranial nerves. Electromyography and compound muscle action potentials have been particularly helpful, especially when monitoring facial nerve function in patients with acoustic neuromas. Brainstem auditory evoked potentials (BAEP) and somatosensory evoked potential (SSEP) should also be recorded when there is a compromise of the brainstem or auditory nerve.

The utility of electrophysiologic testing as a diagnostic tool in routine clinical neurological practice has been well established. The use of similar electrophysiological techniques to assess the integrity of the nervous system intraoperatively is also not a new concept. For many years, surgeons have used the presence or absence of a visible muscle twitch following electrical stimulation of a peripheral nerve as an indicator of peripheral nerve integrity. Over the past 10 years these techniques have been adapted for use in patients undergoing a variety of surgical procedures involving the cerebral cortex, brainstem, spinal cord, and cranial or peripheral nerves.

General Considerations for Intraoperative Monitoring

The purpose of intraoperative monitoring is to preserve function and prevent injury to vital neural structures at a time when clinical examination is not possible. The ideal monitoring system should allow rapid detection of a reliable and useful signal in the electrically hostile environment of the operating room while interfering minimally with the course of the operation. The identification of reversible changes in the recorded potentials allows the surgeon or anesthesiologist to take appropriate steps to prevent permanent damage to neural structures. Even recognition of irreversible changes can be useful when they teach the surgeon about the mechanism of injury and help predict the nature and severity of the postoperative deficit.

[1] Mayo Clinic, 200 First Street S.W., Rochester, MN 55905, USA.

Intraop. Neurophysiol. Monitoring
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Electrophysiologic monitoring provides surgeons direct and immediate feedback about the function of neural structures that may be injured inadvertently during the operation. The value of these techniques in many different types of operations is just being realized. Many different methods have been tried to measure neural function electrically in patients under anesthesia, and most have been found helpful.

Cortical function can be monitored with electroencephalography (EEG) and surface recording of somatosensory evoked potentials (SSEP). Thalamic nuclei can be located by direct recording during stereotactic surgery. Eighth cranial nerve and brainstem function can be monitored by studying brainstem auditory evoked potentials (BAEP). Peripheral and central sensory pathways are monitored by recording SSEP. Peripheral and cranial motor nerve function can be monitored with electromyography (EMG) and by studying compound muscle action potentials (CMAP). Nerve action potentials (NAP) can be recorded directly from peripheral nerves, the trigeminal nerve, and the eighth cranial nerve. Use of visual evoked potentials (VEP) for monitoring has been limited because of problems in identifying an adequate stimulus and appropriate applications. Motor evoked potentials (MEP) from the spinal cord can be used to evaluate central motor pathways. Blink reflexes and F-waves become too variable in patients under anesthesia to be used for monitoring.

The selection of which potentials to monitor and which monitoring technique(s) to use is best made on the basis of each individual patient's clinical status and the structures most at risk in each case. Often some combination of methods is needed. Patients undergoing operations in the posterior fossa to manage large tumors may need to have EMG potentials, CMAP, BAEP, and SSEP monitored, whereas only one of these types of responses may be monitored in patients with smaller lesions. In patients undergoing operations involving peripheral nerves or a nerve plexus, SSEP, NAP, or CMAP may be monitored. SSEP alone may be monitored in patients undergoing stereotactic biopsy or stereotactic resection or cortical resection. Patients undergoing carotid endarterectomy are best monitored by EEG, but at times will benefit from EMG monitoring as well.

During the course of surgery, damage to neural structures may occur rapidly and irreversibly. Therefore, it is desirable to have a way of learning whether the patient is at risk of such damage before the damage occurs, even if the technique used to identify the risk does not give information about whether the damage will result in a clinical deficit. This approach to intraoperative monitoring carries with it the understanding that changes will be identified that are not serious in themselves. The rate of change in neural function will vary with the type and severity of damage. Some damage will occur abruptly or may develop gradually over 20-30 minutes. Abnormalities in the potentials monitored can occur at different times in an operative procedure. Some abnormalities may be seen immediately after an injury; others, particularly those indicative of mild nerve compression, may not become manifest for up to an hour after the step in the procedure that caused injury. Monitoring must therefore be carried on throughout the operative procedure, even after a so-called "critical period" in the operation has passed.

As have most other techniques in medicine, intraoperative monitoring has its limitations, the greatest of which is the rare occurrence of a postoperative deficit in the absence of electrophysiologic warning signs. These so-called false-negative results may occur for a number of reasons, the most common being selective damage to pathways or areas that were not directly monitored by the technique used.

The appearance and reproducibility of potentials recorded intraoperatively are affected greatly by the type and level of anesthesia, blood pressure, temperature, and other physiological variables. Therefore, a coordinated effort by a team, made up of the clinical neurophysiologist, anesthesiologist, and surgeon, is necessary to ensure that the monitoring protocol is tailored to provide optimal results for each patient. Recording equipment that can record several different evoked potentials and EEG and/or spontaneous EMG simultaneously, and that can store, display, and report results efficiently, is preferred. Careful attention should be paid to electrical safety when using equipment purchased commercially or made "in house."

The surgeon is always working under pressure to complete the operation smoothly and quickly, and he or she must be able to proceed efficiently with this work. In selecting techniques for intraoperative monitoring, the avoidance of operative delays engendered by waiting for monitoring results should be paramount. The surgeon must learn as quickly as possible whether a step in the operation that could cause damage has done so.

Equally important to the success of intraoperative monitoring is the reliability with which changes in recorded potentials are identified. Identifying changes requires that a well-defined set of baseline values be obtained during the initial, low-risk portions of the surgery. The variations due to extraneous factors must be identified so that the surgeon can be assured that, if he or she is told of a change, it is related to the surgical procedure and not to changes in blood pressure, artifacts, technical problems, or other factors.

As monitoring methods are refined, the need to minimize interference with the surgical procedure should always be kept in mind. Monitoring techniques that entirely avoid the operative field are preferred, as long as they provide adequate information. Such techniques are less affected by "noise" due to surgical manipulations and also add less complexity to the surgical procedure.

Despite these limitations, there is a growing body of data supporting the overall accuracy and utility of intraoperative monitoring. In this chapter we outline some techniques used for intraoperative monitoring. These methods have evolved, and will continue to evolve, as experience is gained. Therefore, this chapter can only be considered a summary of current monitoring procedures, and not a description of acceptable monitoring methods.

Electromyography

General Considerations

Electromyographic monitoring involves recording the potentials generated by muscles. Many different types of potentials can be recorded intraoperatively, but

those of interest are 1) neurotonic discharges, potentials that occur in response to mechanical or metabolic irritation of the nerve that innervates a muscle, and 2) motor unit potentials, the reflex activity of anterior horn cells. Neurotonic discharges are distinctive discharges of a motor unit that appear as rapid, irregular bursts. Motor unit potentials, in contrast, are the result of firing that occurs in a semirhythmic pattern. Neurotonic discharges and motor unit potentials must be distinguished from a variety of other activities, both physiologic and artifactual.

Electrical interference of many types is present in the operating room, and must be distinguished from neurotonic discharges. Among the most frequently seen and distressing types of electrical interference are movement artifacts. These are irregular, triangular waves. Once recognized they can be differentiated readily from evoked potentials, but they may be mistaken for neurotonic discharges (Fig. 1). Electrical equipment and fluorescent lights in the operating room also can contribute to electrical artifacts. Sixty-cycle interference from gas humidifiers, lights, and heating blankets can be eliminated by proper shielding and grounding of equipment. The artifacts from nerve stimulators, Cavitrons, and respirators can be recognized easily. Interference from cautery is best suppressed with a switch attached directly to the cautery control switch. EMG activity cannot be monitored during cautery.

Muscle activity that must be distinguished from neurotonic discharges includes motor unit potentials that may occur in patients not deeply anesthetized so that their reflexes remain active, fibrillation potentials that may occur if a muscle has been partially denervated, myokymic discharges, muscle end-plate noise and spikes, and complex repetitive discharges (Fig. 2). Each of these types of activity

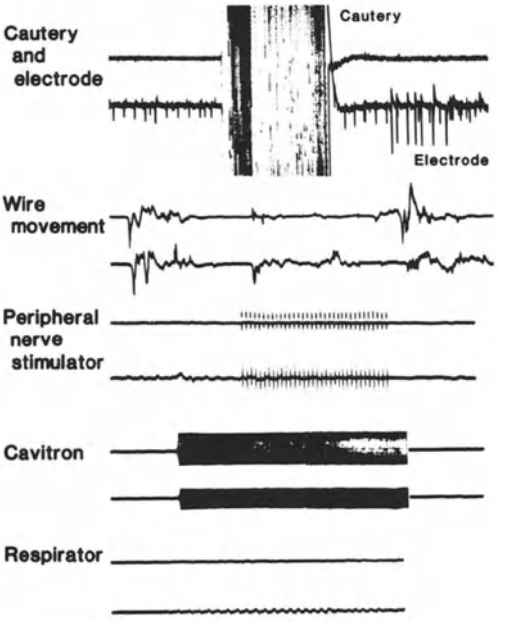

Fig. 1. Artifacts recorded with EMG wire electrodes during intraoperative monitoring

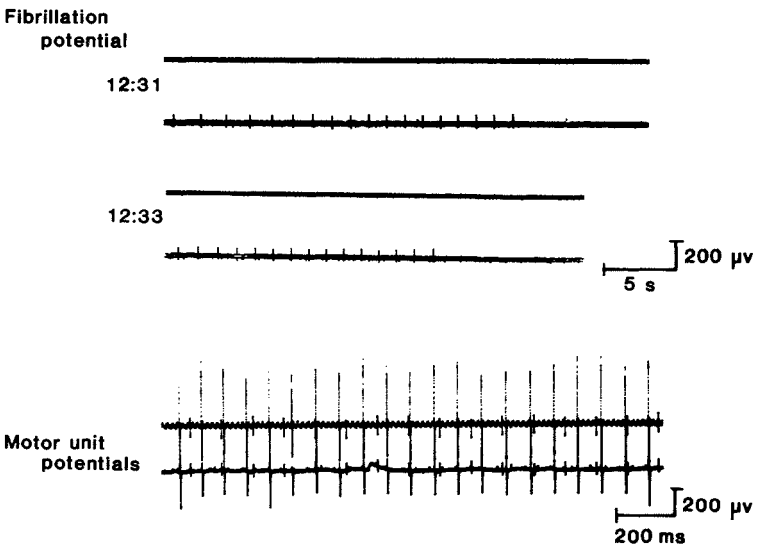

Fig. 2. Spontaneous activity recorded from facial muscle during resection of an acoustic neurinoma

has been seen intraoperatively, but can be distinguished readily from neurotonic discharges by the firing patterns and action potential characteristics typical of each. Neurotonic discharges may take a variety of forms, but all are rapid bursts that occur irregularly (Figs. 3 and 4).

They may last less than 100 ms or persist for many seconds. Long bursts are seen more often after nerve stretching or after irrigation of the nerve. Typically, multiple neurotonic discharges occur independently in each muscle. Often the different discharges occur at the same time in all muscles innervated by one nerve, but not in muscles innervated by another nerve. Once familiar with the characteristics of neurotonic discharges, the surgeon can readily recognize the discharges as soon as they are elicited by surgical manipulation of a nerve. Nerve section may result in dense, long bursts of neurotonic discharges. Neurotonic discharges are very sensitive indicators of nerve irritation and occur in virtually all patients who have been monitored. Neurotonic discharges occur more with cranial nerve manipulation than with peripheral nerve manipulation. Irritation of spinal nerves, especially dorsal roots, often results in a burst of motor unit potentials rather than neurotonic discharges. Central motor unit potentials that occur with cranial or peripheral nerve monitoring are usually due to reflex contractions. The presence of these discharges warns the surgeon that a nerve is being affected by surgical activity, and absence of these discharges can reassure the surgeon that the nerve remains unaffected. The extent of postoperative loss of function is related to the density and frequency of the neurotonic discharges that occur during an operation (Harner et al. 1987).

A variety of EMG recording electrodes have been tested. Surface and subcutaneous electrodes may record some of the neurotonic discharges, but cannot

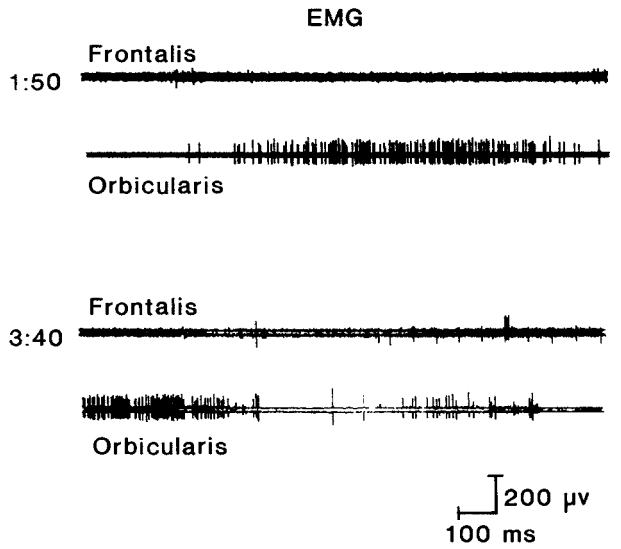

Fig. 3. Neurotonic discharges recorded on EMG of facial muscles during monitoring for acoustic neurinoma resection. There was an incomplete facial palsy after surgery

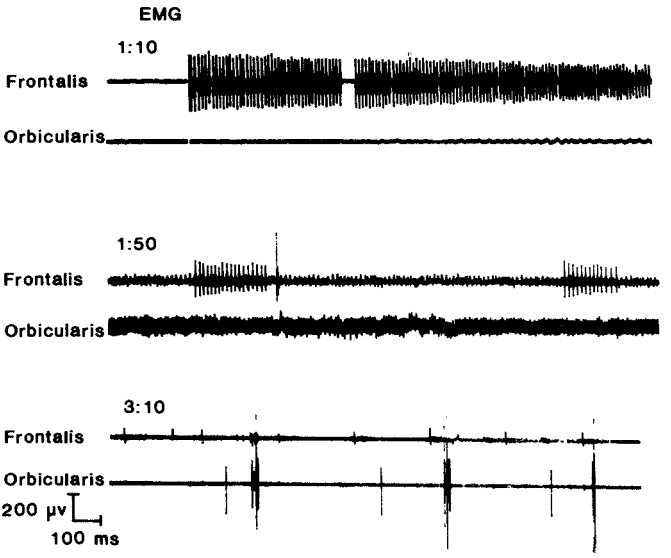

Fig. 4. Results of monitoring acoustic neurinoma resection with EMG. Dense neurotonic discharges occurred. No residual facial function was present after the operation

reliably record the discharges of deep or sparsely distributed motor units; they are also noisier and less stable at the gains needed for EMG recording.

Standard concentric and monopolar EMG needle electrodes provide excellent-quality EMG signals, but are somewhat uncomfortable to apply, and because they are bulky, they also may get in the way of the anesthetist. The most satisfactory electrodes have proven to be fine, insulated wires with 2 mm bared at the tips that are inserted 5 mm apart through a 26-gauge needle, which is then removed. Once the patient has been scrubbed and draped, there is no longer any opportunity to replace or reconnect wires that are dislodged. Thus, the wires are taped to the skin in loops to prevent dislodging of the wires during surgical manipulations. Perfect placement and stabilization is required initially to assure high quality of the recording throughout the surgical procedure.

The fine wires can be connected to the input of the preamplifiers either through small, hooked probe connectors or through modified integrated circuit connectors held in their sockets by integrated circuit pins. The leads from the probes to the preamplifier must be kept as short as possible to reduce interference from external sources.

EMG recordings are made with standard gain, sweep, and filter settings (gain, 200 or 500 volts; low filter, 32 Hz; high filter, 16 KHz; oscilloscope sweep speed, 10 ms/cm). Recordings can be made from any somatic muscle, including extraocular, facial, laryngeal, intercostal, abdominal, anal sphincter, and any limb muscles. EMG recordings from multiple muscles can be presented simultaneously over a loudspeaker as well as on an oscilloscope. EMG activity of interest is photographed and recorded on magnetic tape.

The effect of neuromuscular blocking agents must be reduced if EMG monitoring is to be performed, but neurotonic discharges can be recorded with a 50% neuromuscular block (approximately 2.0 g/kg) administered by continuous intravenous (IV) infusion. Although such a level of muscle relaxation increases the possibility of unwanted movement during the operation, movements of the patient can be prevented with adequate levels of narcotic inhalation anesthesia. At times additional agents such as fentanyl or Versed (midazolam) must be administered to reduce background muscle contractions and associated motor unit potentials.

Applications

These general methods of monitoring can be used during a wide variety of neurosurgical procedures involving central and/or peripheral nerves. They can be of importance in most neurosurgical procedures involving the spinal cord, posterior fossa, and peripheral nerves. In particular, any procedure that requires manipulation or dissection of a nerve can be performed more safely and effectively with intraoperative monitoring. Most tumors involving these areas are best removed with the help of intraoperative monitoring during the procedure (Figs. 5, 6). It should be remembered that not only does using a number of different monitoring procedures simultaneously give more readily interpretable results, it will also provide information about the function of a number of different structures at the same time.

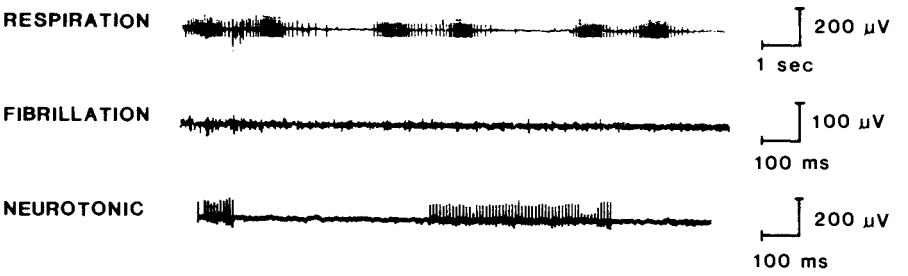

Fig. 5. Spontaneous EMG activity recorded, via electrodes placed in the vocalis muscle, during neck dissection for metastatic thyroid cancer

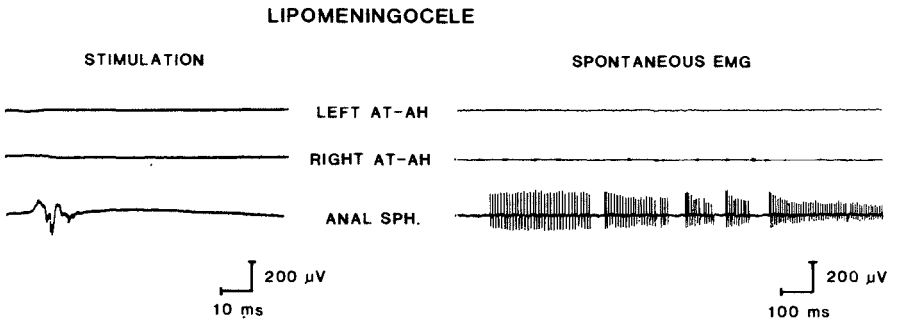

Fig. 6. Bipolar intramuscular responses recorded from the anterior tibia to stimulation of the abductor hallucis muscles *(AT-AH)* and the anal sphincter *(ANAL SPH)*. Stimulation of lower sacral roots produced a CMAP in the anal sphincter *(left)*, whereas traction on the same roots produced spontaneous neurotonic discharges *(right)*

Compound Muscle Action Potentials

Monitoring of compound muscle action potentials helps to:
1) identify the facial nerve during acoustic neuroma operations,
2) identify the superior laryngeal nerve during thyroid surgery,
3) locate conduction blocks or segmental slowing in operations on the ulnar nerve, and 4) provide information about the amount of damage occurring to a nerve during a surgical procedure.

The integrity of motor axons can be tested directly by stimulating the nerve in the surgical field and recording the summated activity of the muscle fibers (CMAP) in one or more of the muscles innervated by the nerve. The responses to nerve stimulation can be recorded from muscle with either intramuscular wire electrodes such as are used for EMG monitoring or with surface electrodes (Fig. 7). The responses obtained with the wire electrodes are usually complex, multispike waveforms that cannot be measured easily or reliably. Therefore, they cannot be used for quantitative measurements. Surface electrodes produce reproducible, biphasic compound muscle action potentials (CMAP) that can be mea-

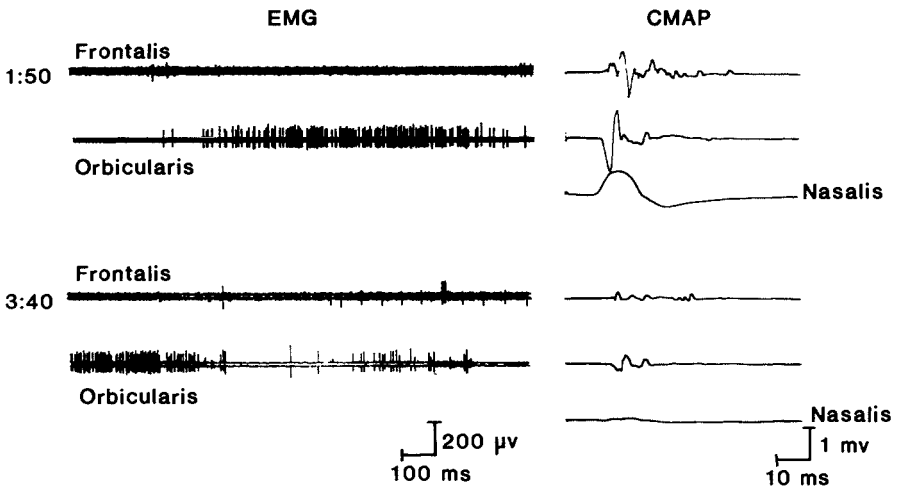

Fig. 7. Results of monitoring EMG and CMAP of facial muscles during acoustic neurinoma resection. Neurotonic discharges recorded from intramuscular wire electrodes are shown on the left. On the right, evoked potentials recorded from the wire electrodes and from the surface electrodes over the nasalis muscle are shown. There was an incomplete facial palsy after the operation

sured and compared with potentials obtained later in the surgical procedure. The optimal surface electrodes are 5-mm tin discs that are applied to the skin with collodion to assure firm adherence to the skin. The CMAPs are recorded at the sweep speed and gain that are appropriate for the response that is to be measured. Sweep speeds of 1 to 10 ms/cm are usually satisfactory. The amplification of the response that is required also varies, but between 500 μV and 5 mV/cm is usually suitable. Filter settings similar to those used for routine motor nerve conduction studies – 1.6 Hz low and 16 kHz high – can be used.

Surface electrodes record the CMAP from any muscle in the region of the electrode, not only the muscle immediately under it. Thus, ulnar nerve stimulation will produce a CMAP over the thenar muscles. Therefore, CMAP must be interpreted with care.

Stimulation

A number of different stimulators are available for use in the operating room; each has advantages and disadvantages. All of the stimulators should be handheld by the surgeon to ensure the proper location of the stimulus and for ease of moving the stimulator during the operation. It should be remembered that the most effective stimulus occurs when the electrical current passes along the length of the nerve. Therefore, the cathode and anode should be placed directly along the length of the nerve. The cathode is the active or depolarizing pole and the electrode that acts as a cathode should be placed so that it is closest to the recording electrode. With this electrode arrangement, the stimulator is referred to as a

bipolar stimulator. The bipolar stimulator may be 2 1-mm wires, insulated except for their tips; a pair of insulated forceps; or a pair of insulated, curved forceps with the insulation removed from the inner surfaces of the hooked ends.

Stimulation of the nerve may also be performed by placing a single-pronged cathode on the nerve and placing the anode some distance from the nerve. This is referred to as a "monopolar" stimulator. The advantage of the bipolar stimulator is that it can provide a focal stimulus, thus ensuring that only the target nerve is stimulated. The disadvantage is that if the nerve is distant from the stimulator or there is too much fluid in the operative field there may be inadequate activation of the nerve and thus a low-amplitude response. A monopolar stimulator reduces this likelihood, but it increases the possibility of current spread to other nerves and shock artifact.

The size of the stimulating electrodes will vary depending on the nerve that is stimulated. Small cranial nerves will require stimulator tips that are as small as 1 mm, while larger peripheral nerves may require electrodes with 2–3 mm tips to provide an adequate stimulus. A surgical forceps modified to be used as a bipolar stimulator allows the surgeon to dissect tissue with the stimulator.

The stimulation is applied at rates of 1 to 5 Hz with a stimulus duration of 0.05 ms. The stimulus strength is gradually increased until a maximal response is obtained. Maximal responses are obtained from normal nerves with currents of less than 2 mA (15 to 25 V). If the nerve is damaged, if there is tissue between the nerve and the stimulating electrodes, or if there is excess fluid in the stimulating field, then a higher voltage will be necessary to activate the nerve. To avoid current spread to nearby nerves it is best to avoid stimulating with currents greater than 3 to 4 mA (30 to 50 V).

Measurement Parameters

The amplitude of the CMAP is proportional to the number of axons that are able to conduct a response. Therefore, when the purpose of monitoring is to determine the number of intact axons, the amplitude of the maximal response should be measured and compared to the responses obtained prior to surgery and earlier in the procedure. The amplitudes of responses along the length of the nerve should also be compared. To demonstrate localized abnormalities, both the latency of the response and the amplitude of the potential should be measured (Fig. 8).

Technical Problems

An absent CMAP may indicate that there has been significant damage to the nerve. However, technical problems may have caused loss of the CMAP, and such problems should be sought and corrected if present before there is undue concern. Failure to record a response may be due to the use of a muscle relaxant with the anesthesia, shorting of the stimulus current, stimulating electrodes not being placed on the nerve or not connected to the stimulator, amplifiers not being

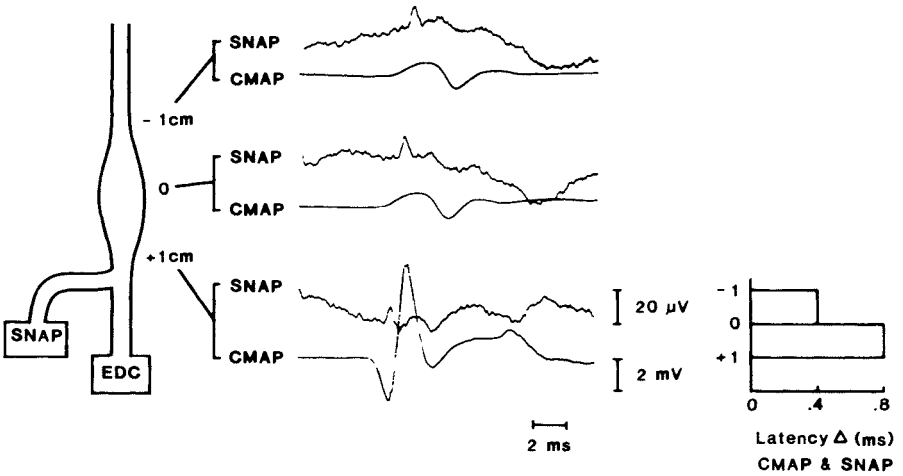

Fig. 8. Intraoperative recording of extensor digitorum communis CMAP *(EDC)* and responses to the superficial radial nerve stimulation *(SNAP)* at 1-cm intervals. The "0" indicates the point of maximal nerve swelling. The location of the lesion was confirmed by a change in latency of the CMAP and SNAP and by a change in the amplitude of the CMAP

turned on, recording electrodes having been disconnected, and/or recording electrodes not working properly.

In other situations a response may be obtained that is larger than expected or that is not expected. This may be the result of the stimulator being placed on the wrong nerve, or of too strong a stimulus causing current spread to adjacent nerves.

In order to understand how intraoperative monitoring can help prevent neurological deficits postoperatively, a brief review of the pathophysiology and electrodiagnostic manifestations of focal nerve lesions will be presented. When a peripheral nerve is injured, there are several possible consequences and each has distinctive pathological and neurophysiological characteristics. The mildest form of peripheral nerve dysfunction is neurapraxia. In this condition, a temporary block in conduction along one or more axons results from metabolic factors or changes in the structure of the perinodal myelin (demyelination and/or invagination of adjacent nodes). Because these injuries are not associated with structural changes in the axon, the prognosis for complete recovery is excellent. In cases of pure neurapraxia, surgical exploration and decompression of the nerve are required only if there is an obvious source of entrapment or failure of improvement with prolonged conservative therapy.

The electrophysiological hallmark of neurapraxia is a conduction block, manifested as a lower-amplitude CMAP or NAP with proximal as compared to distal stimulation during nerve conduction studies. In some cases the lesion(s) may be localized to a 1- to 2-cm segment with a technique called "inching." Slowing of segmental conduction velocities may also occur with focal demyelination or axonal narrowing. When a lesion is purely neurapractic, the CMAP and sensory

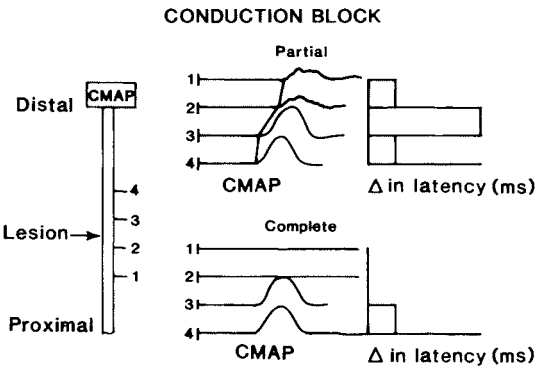

Fig. 9. Results of recording CMAP during stimulation proximal and distal to a lesion causing partial *(top)* and complete *(bottom)* conduction block. The location of the lesion is indicated by changes in CMAP amplitude and latency

nerve action potential (SNAP) obtained in response to stimulation and recording along the distal segment of nerve are normal. Standard nerve conduction studies can often define the number and location of neurapractic lesions when proximal stimulation and inching are feasible. Similar electrophysiological studies involving direct stimulation of the peripheral nerve intraoperatively can be used to confirm the precise location of a conduction block, to rule out the presence of additional lesions, and to identify localized abnormalities not found on standard testing. Continuous EMG monitoring for neurotonic discharges can prevent further trauma to the peripheral nerve during dissection and manipulation. Fig. 9 illustrates the technique and results of intraoperative monitoring when the predominant lesion is a conduction block.

Monitoring Cranial Nerve Surgery

Operations that involve the structures in the posterior fossa are often performed, particularly for the removal of acoustic neurinomas. This approach is also used for operative management of hemifacial spasm, trigeminal neuralgia, foramen magnum lesions, and a variety of brainstem lesions. The most frequently used techniques for intraoperative monitoring of posterior fossa procedures are those that monitor the cranial nerves arising from the brainstem in the posterior fossa.

Although in many cases monitoring during posterior fossa procedures involves only monitoring of cranial nerves, there are situations when other types of electrophysiological monitoring such as SSEP and BAEP monitoring may be of value. SSEP monitoring can be helpful when the patient is undergoing resection of a large tumor in the posterior fossa or in an operation to treat a vascular lesion that involves the brainstem. BAEP monitoring is often performed during the removal of acoustic neurinomas or other cerebellopontine angle tumors, and during microvascular decompression procedures to treat trigeminal neuralgia and hemifacial spasm.

Cranial nerve involvement is common with posterior fossa tumors, especially those over 2 cm in diameter. Electrophysiologic signs of facial nerve damage are

Table 1. Frequency of monitoring cranial nerve function during surgery

Cranial nerve	Operations	Neurotonic discharges present	CMAP tested
Oculomotor	8	4	4
Trochlear	2	0	1
Abducens	9	4	4
Trigeminal (motor)	305	44	25
Facial	335	306	272
Vagus	17	7	4
Accessory	18	6	5
Hypoglossal	16	4	0

present in a high proportion of patients, often without clinical symptoms or signs (Rossi et al. 1979). In 6% of patients with acoustic neurinomas, facial nerve conduction study results are abnormal, in 44% the blink reflexes are abnormal, and in 78% needle EMG are abnormal (Harner et al. 1986). The extent of abnormality is proportional to the size of the tumor, and is an excellent predictor of the extent of postoperative deficit. While large tumors may impinge on the accessory and hypoglossal nerves, clinical symptoms or signs have not been reported (Table 1).

Facial Nerve (VII)

As surgical techniques to manage facial nerve problems have improved, there has been increasing emphasis on the preservation of facial nerve function after removal of acoustic neurinoma. The outcome depends mainly on tumor size and location and, to a lesser extent, on specific surgical techniques. Of patients with large tumors (over 4 cm), close to 100% have complete facial paralysis postoperatively. The nerve is intact after surgery in 85% of patients with medium-sized tumors and in all patients with small (less than 2 cm) tumors. Nonetheless, 95% of patients with medium-sized tumors have some postoperative weakness, and 50% have complete paralysis. Of patients with small tumors, 21% have complete paralysis and 68% have some weakness after surgery. Luckily, if the nerve is left anatomically intact, recovery of function occurs eventually in up to two-thirds of patients (Harner and Laws 1981).

In 1979 Sugita et al. reported an improvement in the rate of preservation of facial nerve function when electrophysiologic monitoring was used during operations to resect acoustic neurinomas. In their series of 22 patients, 20 had lesions more than 4 cm in diameter. Loss of facial function was present preoperatively in 64% of patients and facial weakness in 55%. In addition to using the surgical microscope for identification and dissection of the nerve from the tumor, Sugita et al. applied direct electrical stimulation to the nerve using insulated bipolar coagulating forceps. The surgeon applied stimulation of 1 to 3 V for 1 ms at rates of 2 to 4 Hz and monitored the movement of eyelid and lip muscles with a mechanical monitor of movement. The response was amplified and broadcast through a

loudspeaker to give the surgeon immediate feedback when he was stimulating the nerve. With these techniques the authors preserved anatomical and physiologic function in 86% of their patients.

Three complementary methods can be used to obtain different types of information for monitoring the facial nerve. 1) Preoperative EMG, evaluation of blink reflexes, and facial conduction studies define the amount of preoperative nerve damage and identify any spontaneous discharges. These reliably predict the likelihood of further loss of function during surgery. 2) Inadvertent mechanical stimulation of the motor nerves during the surgical procedure is monitored by visual and auditory monitoring of EMG activity in muscles innervated by the cranial nerve of interest. 3) The location and function of the nerve in the operating field is monitored by recording the CMAP over the appropriate muscles in response to direct electrical stimulation of the nerve by the surgeon (Harner et al. 1986; Møller and Jannetta 1984).

EMG recording electrodes are placed directly within the muscles of interest (Fig. 10). For facial nerve monitoring this usually includes the orbicularis oculi and orbicularis oris muscles and occasionally other facial muscles such as the frontalis and mentalis. To assure proper electrode placement and satisfactory recordings, the insertion is best performed as for standard EMG, before the patient is anesthetized. Proper electrode placement is confirmed when voluntary EMG

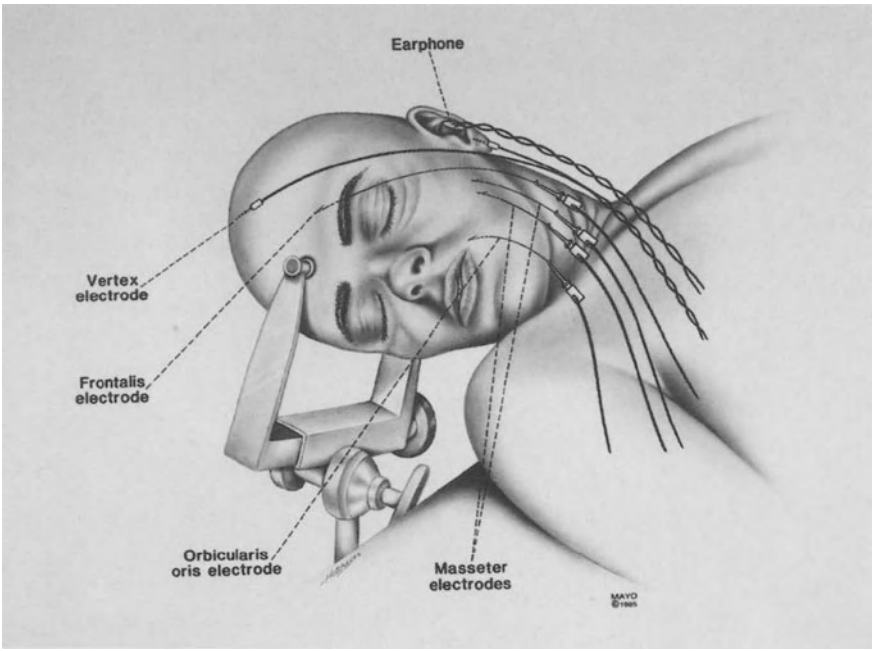

Fig. 10. Placement of recording electrodes used in monitoring operations in the posterior fossa. Hooks represent intramuscular EMG wire electrodes. Vertex and ear needles are used to record BAEP. (By permission of Map Foundation)

activity can be recorded. Two wire electrodes can be inserted together through a single needle if the tips are exposed at different locations. This results in focal recording from a single muscle. Placement of 2 wires separately in a muscle monitors a broader area of muscle, but may show more artifact. It is also possible to place wires in 2 different muscles, which would then be monitored simultaneously without indication of which muscle was giving rise to neurotonic discharges. If the muscles have the same innervation, this may be satisfactory and allow monitoring of more muscles with fewer recording channels.

For CMAP monitoring (Fig. 11), a baseline response is recorded from the facial nerve preoperatively with routine nerve conduction techniques and used for comparison with responses obtained intraoperatively. The mentalis muscle has been found to give the most reliable, easily measurable responses in most patients, but the nasalis muscle is also occasionally used. Other facial muscles provide less reliable results. Selective stimulation of individual nerves should be attempted (Fig. 12).

If possible, the surgeon stimulates a distal segment of the nerve early in the procedure to determine the threshold for activation, the voltage needed to obtain a supramaximal response, and a baseline response to compare the later responses. Stimulation is then applied at intervals, either to localize the nerve or to determine if it is still intact (Figs. 7, 13). During resection of a large tumor involving multiple nerves, the individual nerves can be identified and distinguished by elec-

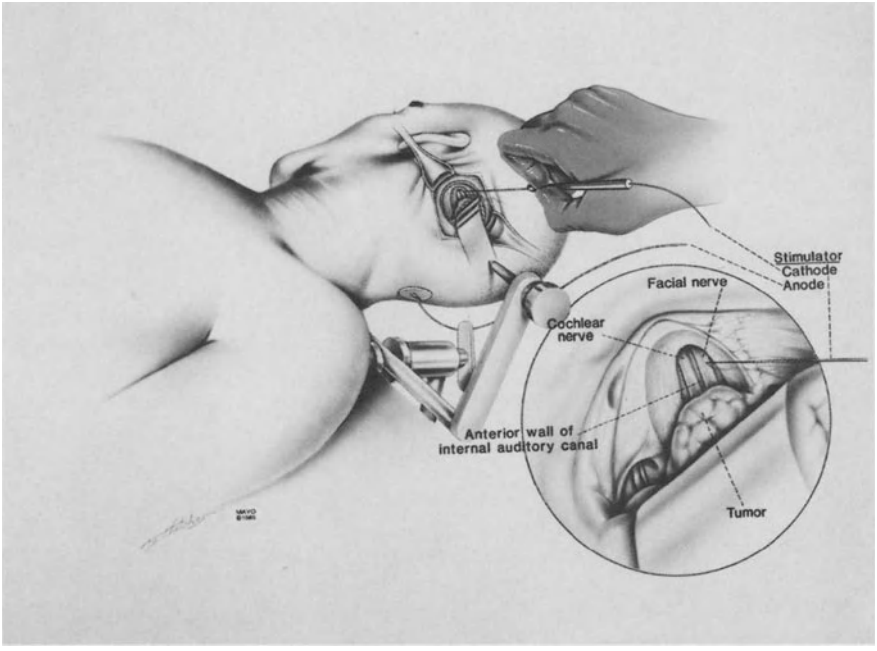

Fig. 11. Representation of direct stimulation of cranial nerves during posterior fossa surgery. Monopolar stimulation is shown. (By permission of Map Foundation)

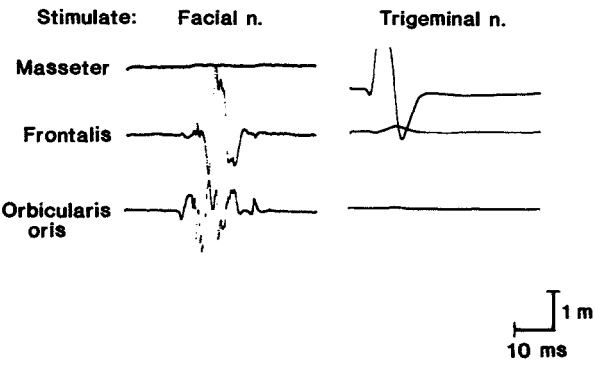

Fig. 12. Results of intraoperative stimulation of selected cranial nerves during an operation in the posterior fossa for meningioma. Some volume-conducted responses from the nearby masseter and frontalis muscles are seen with facial and trigeminal nerve stimulation, respectively

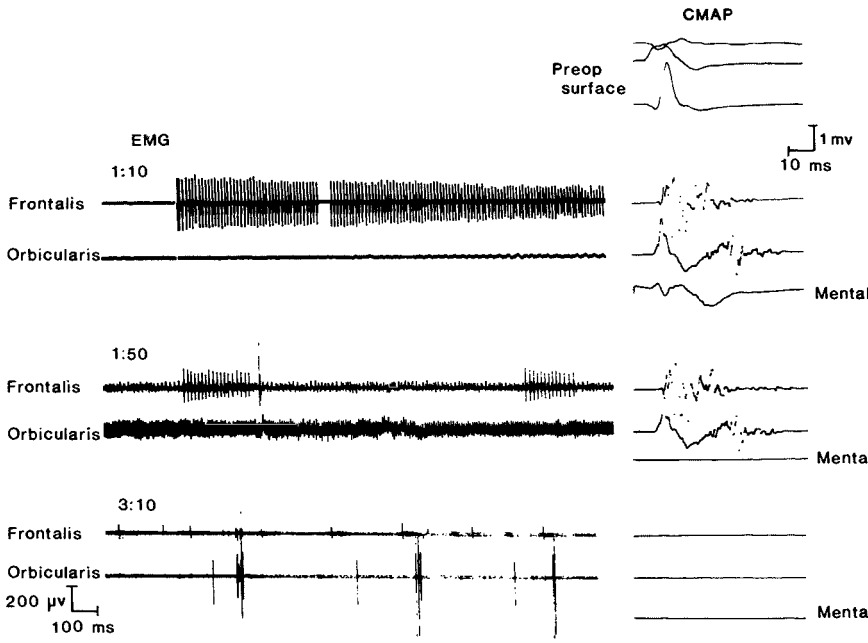

Fig. 13. Results of monitoring EMG and CMAP during acoustic neurinoma resection. Dense neurotonic discharges occurred and the CMAP was lost. No residual facial function was present after surgery

trical stimulation. If the nerve has been ruptured during dissection, it can still be activated distally, but no response is obtained from proximal portions (Fig. 14). There has been excellent correlation between intraoperative preservation of an evoked response and preservation of facial function postoperatively. None of our patients who retained an evoked response had total facial paresis, and all of those who lost the response had complete paralysis (Harner et al. 1986).

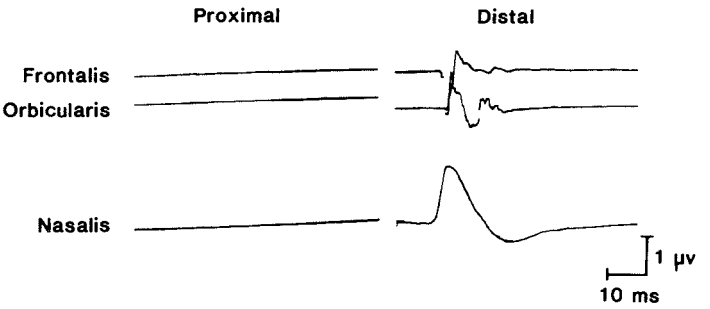

Fig. 14. Results of facial nerve stimulation at proximal and distal sites along the facial nerve after resection of an acoustic neurinoma show total loss of function proximally. There was no residual facial nerve function postoperatively

In patients with hemifacial spasm (HFS) an additional form of stimulation and recording provides valuable information to the surgeon about the adequacy of microvascular decompression (MVD) (Møller and Jannetta 1985).

Most facial nerve monitoring has been performed in patients undergoing resection of acoustic neurinomas. Some patients with large tumors who were monitored had better preservation of facial function than those who were not monitored (Harner et al. 1986). As well as improving the outcome for individual patients, intraoperative monitoring has also helped improve surgical techniques more generally by demonstrating to surgeons what surgical maneuvers cause damage to the nerve. Patients with other tumors, mainly meningiomas, had similar results.

This monitoring involves applying a stimulus to the lower face, preferably over the marginal mandibular branch of the facial nerve at the chin. A standard 50 to 150 V (5 to 15 mA) stimulus can be applied for 0.05 to 0.1 ms, either with surface or needle electrodes. Responses are recorded outside the surgical field from the orbicularis oculi muscle through either surface or wire electrodes. The same wire electrodes can be used that are in place for monitoring neurotonic discharges. The sites of stimulation and recording and appropriate parameters are best determined by preoperative EMG and a nerve conduction study.

In patients with HFS a response occurs in the orbicularis oculi muscle with stimulation of the marginal mandibular branch of the facial nerve. The response, not seen in normal individuals, is a brief, 1 to 5 ms burst of spikes that follows the stimulus by 7 to 12 ms. This response has been called the "lateral spread response" by Møller and Jannetta (1985) because it may arise by ephaptic activation of one group of axons by action potentials in another. The lateral spread response is somewhat variable in its appearance from one stimulus to the next. When the offending vessel has been removed from the nerve, the lateral spread response disappears completely, almost immediately. If the vessel is allowed to touch the nerve again, the lateral spread response quickly returns. At times manipulation of the nerve may also shut the response off temporarily.

In these patients standard EMG and CMAP monitoring are used as described earlier in this chapter. It is also important to perform BAEP monitoring in these

cases because of the occurrence of postoperative hearing loss in up to 15% of patients operated upon to treat HFS.

Other surgical procedures involving other cranial nerves can also be monitored with these techniques. Patients undergoing section of the trigeminal nerve for severe cluster headache can be monitored to be certain that the motor branch of the fifth cranial nerve is not sectioned as well. In patients undergoing resection of chemodectomas the eleventh and twelfth cranial nerves can be monitored.

Trigeminal Nerve (V)

EMG monitoring of trigeminal nerve motor function is similar to monitoring of the facial nerve, with responses being recorded from nichrome wires placed in the temporalis and/or masseter muscles. Potentials of these muscles are readily recorded, as large, easily identified potentials with fewer artifacts than facial muscle potential recordings.

CMAP can be recorded through surface electrodes placed over the masseter muscle, but because of the proximity of facial muscles to this site, "masseter muscle" responses may be composed of both facial responses and masseter responses (Fig. 10). CMAP are therefore usually adequately recorded from wire electrodes in the masseter or temporalis muscles. Direct stimulation of the trigeminal nerve in the operative field is performed by the surgeon in a way similar to that used for facial nerve activation. Nerve action potentials (NAP) can also be recorded directly from the trigeminal nerve in the surgical field via small wick electrodes placed on the nerve and referenced to a distant scalp electrode. Stimulation of the trigeminal nerve with small needle electrodes adjacent to the nerve in the supraorbital or infraorbital foramina can elicit NAP that can be recorded from the intracranial portion of the trigeminal nerve with a minimum of artifact, if the stimulus voltage (current) can be kept low. Antidromic NAP can also be recorded with stimulation of the nerve in the surgical field while recording from the peripheral nerve. The latter technique is more often marred by excess stimulus artifacts, however, precluding its being used to obtain a reliable measurement of the NAP. Trigeminal monitoring is most often used in combination with facial monitoring during removal of large posterior fossa tumors such as acoustic neurinomas or meningiomas. The trigeminal nerve is also monitored during sectioning of that nerve for intractable pain.

Spinal Accessory Nerve (XI)

The accessory nerve is monitored in the same way as the facial nerve is monitored, with wire electrodes placed in the trapezius and/or sternocleidomastoid muscles. CMAP can be recorded from the wire electrodes as readily as from surface electrodes, precluding the need for the latter. The spinal accessory nerve is less often directly involved in tumors requiring direct dissection, and therefore is less often the source of neurotonic discharges. When this nerve does produce such discharges, the discharges are similar to those of other nerves. Eleventh cranial nerve monitoring is most often performed during removal of large meningiomas

Table 2. Indications for monitoring of cranial nerve XI in 18 patients

Indication	No. Patients
Glomus jugulare tumor resection	3
Clivus chordoma/meningioma resection	3
Posterior fossa meningioma resection	2
Acoustic neurinoma (NF) resection	3
Arnold Chiari syndrome management	1
Glossopharyngeal neuralgia management	2
Torticollis management	4
Type of Monitoring	**No. Patients**
Combined recordings	11
Trigeminal motor nerve	6
Facial nerve	8
Vagus nerve	2
Hypoglossal nerve	5
SSEP/BAEP	6

and glomus jugulare tumors, although resection of some carcinomas of the neck may be an indication for monitoring this nerve (Table 2).

Hypoglossal Nerve (XII)

Neurotonic discharges arising in the twelfth cranial nerve can be recorded readily from the tongue muscles with nichrome wires inserted using a submandibular approach near the midline to a depth of 3 to 5 cm. Wires can be placed selectively in the right and/or left sides of the tongue from this location. Appropriate placement of these wires should be tested by asking the patient to produce a voluntary response before anesthesia is induced. CMAP are recorded from the wire electrodes because recording from surface electrodes is not feasible. Monitoring of the hypoglossal nerve is only rarely necessary. It is usually performed only during resection of large posterior fossa meningiomas, clivus tumors, or foramen magnum tumors. It may be of help during resection of some intraventricular tumors (Table 3).

Glossopharyngeal and Vagus Nerves (X)

There are few surgical procedures during which monitoring of the IX and X cranial nerves is needed. Although recordings could theoretically be made from posterior pharyngeal muscles to monitor the IX cranial nerve, we are not aware of any examples of this application. Vagus nerve monitoring is most readily performed by evaluating EMG responses from the cricothyroid or vocalis muscles. Wires to monitor cricothyroid responses can be placed directly into the muscle from externally, but placement of an electrode in the vocalis muscle requires either puncture of the cricothyroid membrane or placement of long electrode wires through the mouth via direct laryngoscopy (Table 4).

Table 3. Monitoring cranial nerve XII in 16 patients

Indication	No. Patients
Foramen meningioma resection	3
Fourth ventricular ependymoma resection	4
Medullary glioma resection	2
Clivus chordoma resection	2
Acoustic neuroma (NF) resection	2
Glomus jugulare tumor resection	2
Arnold Chiari syndrome management	1
Type of Monitoring	**No. Patients**
Combined recordings	16
Trigeminal motor nerve	4
Facial nerve	8
Vagus nerve	6
Accessory nerve	5
SSEP	12

Table 4. Monitoring cranial nerve X in 17 patients

Indication	No. Patients
Fourth ventricular ependymoma resection	3
Medullary glioma resection	3
Clivus chordoma resection	1
Foramen magnum meningioma resection	1
Posterior fossa meningioma resection	3
Glossopharyngeal neuralgia management	2
Thyroid carcinoma management	4
Type of Monitoring	**No. Patients**
Combined recordings	11
Facial nerve	6
Accessory nerve	3
Hypoglossal nerve	5
SSEP (Air and Infarct)	9

Fig. 5 illustrates the use of this technique to monitor the recurrent laryngeal nerve during an operation to resect an invasive thyroid carcinoma in the neck. With the patient anesthetized and intubated, recording wires were placed into the vocalis muscle. Spontaneous EMG activity was observed and included fibrillation potentials and rhythmic bursts of motor unit activity related to respirations. These types of spontaneous activity and artifacts secondary to wire movement and cautery could readily be distinguished from neurotonic discharges, which occurred intermittently during dissection in and around the recurrent laryngeal nerve.

Fig. 15 illustrates the value of selective peripheral nerve stimulation intraoperatively. In this patient undergoing a ninth cranial nerve section for glos-

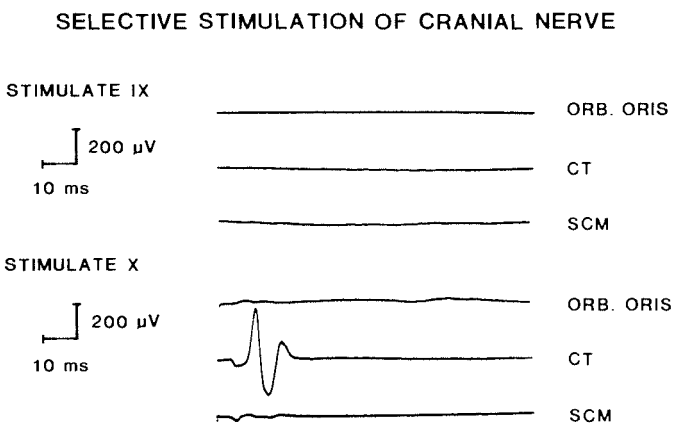

Fig. 15. Results of stimulating the glossopharyngeal *(IX)* and vagus *(X)* nerves in the posterior fossa and recording from intramuscular electrodes in the orbicularis oris *(ORB. ORIS)*, cricothyroid *(CT)*, and sternocleidomastoid *(SCM)* muscles

Table 5. Monitoring cranial nerve III and VI in 10 patients

Indication	No. Patients
Petroclival	
Meningioma resection	4
Epidermoid cyst resection	1
Cavernous sinus	
Meningioma resection	2
Cylindroma resection	1
Arterial venous malformation	1
Clivus	
Chordoma resection	1
Type of Monitoring	**No. Patients**
Combined recordings	8
Trigeminal nerve	7
Median nerve SSEP	3

sopharyngeal neuralgia, spontaneous and stimulus-evoked EMG responses were recorded from the cricothyroid muscle in order to avoid sectioning of any nerve rootlets contributing to the vagus nerve.

Oculomotor and Abducens Nerves (III and VI)

There is rarely a need to monitor the oculomotor nerves, but it can be done. Tumors in the region of the cavernous sinus or intraventricular tumors are the indication for this type of monitoring. Fine wire electrodes are inserted per-

cutaneously into the inferior oblique and/or lateral rectus muscles to monitor the EMG and CMAP from the third and sixth cranial nerves, respectively (Table 5).

For most surgical procedures in the posterior fossa or the neck, monitoring of multiple channels of electrical activity is necessary to evaluate function in the specific nerves put at risk by the operation. It is usual to monitor a number of different cranial motor nerves on different channels. These recordings are often combined with recording of median nerve SSEP to monitor the central pathways and/or BAEP to monitor the eighth cranial nerve. It must be remembered that each patient's monitoring needs must be selected based on the surgeon's assessment of the structures at risk during the operation on that individual patient. No single protocol can be used to define the monitoring that will be optimal for all patients.

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Transcranial Magnetic and Intraoperative Electrical Stimulation of the Trigeminal and Facial Nerves: Sites and Mechanisms of Excitation

U. D. Schmid[1], K. M. Rösler, C. W. Hess, and H. J. Reulen

Summary

Currents induced by a rapidly varying magnetic field were applied to the parieto-occipital scalps of patients undergoing neurosurgical operations to stimulate simultaneously the ipsilateral facial and trigeminal nerve, as well as other caudal motor cranial nerves. The magnetic stimulation was assumed to generate electrical currents in the cerebrospinal fluid (CSF). The stimulating currents were assumed to pass through the foramina of the respective cranial nerves distal to the CPA, where the nerves are still surrounded by CSF. Thus, the point of excitation for the facial nerve was within the labyrinthine segment of the facial canal; and for the trigeminal nerve was probably within Meckel's cave.

A technique for transcranial magnetic stimulation of the facial nerves was reported upon recently by Murray et al. (1987) and Schriefer et al. (1988), and a technique for such stimulation of other motor cranial nerves was reported by Benecke et al. (1988). When responses were recorded from respective muscles ipsilateral to the site of magnetic stimulation, the responses showed no inherent variability in amplitude and did not appear to be influenced by pre-innervation. We believe that the lack of change in amplitude may indicate that the stimulation was supramaximal. Further, the onset latencies of the ipsilateral responses were found to be about 5 ms shorter than the onset latencies of the responses to contralateral stimulation of the motor cortex (Rösler et al. 1989; Benecke et al. 1988). These characteristics of the responses to magnetic stimulation led to the conclusion that the effective sites of stimulation were the peripheral nerves, rather than the motor cortex or the brainstem.

However, on the basis of hypothesized conduction velocities and supposed lengths of these nerves, excitation sites within the cerebellopontine angle (CPA) or even at the root exit zones (REZ) near the brainstem have been proposed (Benecke et al. 1988; Maccabee et al. 1988).

It was the aim of the present study to further elucidate the sites and mechanisms of magnetic stimulation by performing direct intraoperative electrical stimulation of these nerves and examining the results in patients with known sites of facial and trigeminal nerve injury.

[1] Inselspital, Neurochirurgische Universitätsklinik, CH-3010 Bern, Switzerland.

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Patients and Methods

Patients

Magnetic transcranial stimulation was performed on a total of 19 patients. In 6 patients transcranial magnetic stimulation was performed immediately before the skin incision was made and direct electrical stimulation of the trigeminal and facial nerves was performed intraoperatively after surgical exposure of these nerves. Patients were positioned for the operation in a prone position and anesthesia was administered without muscle relaxants, except for intubation as described previously (Schmid et al. 1988). The cerebellopontine angle (CPA) was exposed by a suboccipital retromastoid approach.

Stimulation and Recording

The technique used for transcranial magnetic stimulation of the facial nerve was described in detail by Schriefer et al. (1988) and Rösler et al. (1989), and it was similar to that reported by others (Benecke et al. 1988; Maccabee et al. 1988). A magnetic coil was placed tangential to the parietooccipital surface of the scalp, ipsilateral to the site of recording. The mean diameter of the coil was 8.1 cm and maximal stimulus intensity was 2 Tesla at the rim of the coil. The direction of current in the coil was clockwise as viewed from behind the patient's head for stimulation of the left side, and vice versa for stimulation of the right side. For comparison, the stylomastoid segment of the facial nerve was stimulated by surface electrodes.

Intraoperative stimulation was performed in 6 patients (Table 1). For intracranial electrical stimulation bipolar handheld electrodes with an interelectrode distance of about 0.5 cm were used. Currents of 0.2–5 mA (constant current) were utilized. These electrodes were used to stimulate the trigeminal or facial nerves, and the cathode was placed distal to the anode.

We recorded the responses from the nasal muscle through surface electrodes in 17 subjects and from the masseter muscle through needle electrodes in 2 subjects. Latencies and baseline-to-peak amplitudes of the compound muscle action potentials (CMAPs) recorded to magnetic stimulation were compared either with those obtained in response to direct electrical stimulation (intraoperative experiments) or with normal values obtained from 15 healthy subjects (Rösler et al. 1989). The study complied with standards of the local ethical committee, and subjects gave informed consent to participation in the study.

Results

Using the technique just described for transcranial magnetic stimulation, we readily evoked CMAPs from masseter and nasal muscles in normal subjects as well as in patients (Fig. 1). The CMAPs were of similar shape and amplitude in response to extra- or intracranial electrical stimulation of the facial or the trigeminal nerves.

Table 1. Intraoperative stimulation of the facial or trigeminal nerves in 6 patients

Pt. No.	Diagnosis	Nerve stimulated	Clinical findings before surgery	Clinical findings after surgery
1.	Intra/extrameatal acoustic neurinoma, 3 cm	CN V CPA and at tentorium	CN V normal	CN V normal
2.	Plexus papilloma lateral recess, 3 cm	CN VII CPA	CN VII normal	CN VII normal
3.	Extrameatal acoustic neurinoma, 1.2 cm	CN VII CPA and intrameatal	CN VII normal	CN VII normal
4.	Negative exploration of the CPA and meatus, no lesion found	CN VII CPA and intrameatal	CN VII normal	CN VII normal
5.	Intra/extrameatal acoustic neurinoma, 1 cm	CN VII CPA and intrameatal	CN VII normal	CN VII paretic (lesion in CPA)
6.	Intra/extrameatal acoustic neurinoma, 3 cm	CN VII CPA	CN VII normal	CN VII paretic (lesion in CPA)

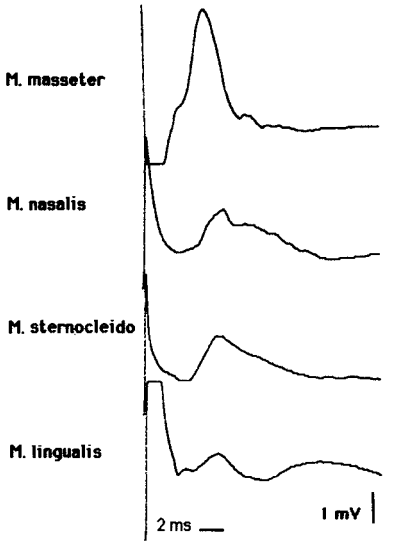


Fig. 1. Responses recorded from the masseter, nasal, sternocleidomastoid, and lingual muscles to transcranial magnetic stimulation over the ipsilateral parieto-occipital scalp

Trigeminal Nerve

Case 1: This patient was operated upon to remove an acoustic neurinoma of about 3 cm in diameter. Transcranial magnetic stimulation was performed immediately before the operation and direct electrical stimulation was performed intraoperatively. After removal of the tumor, the motor portion of the undamaged trigeminal nerve was stimulated electrically 1) within the cerebellopontine angle (CPA)

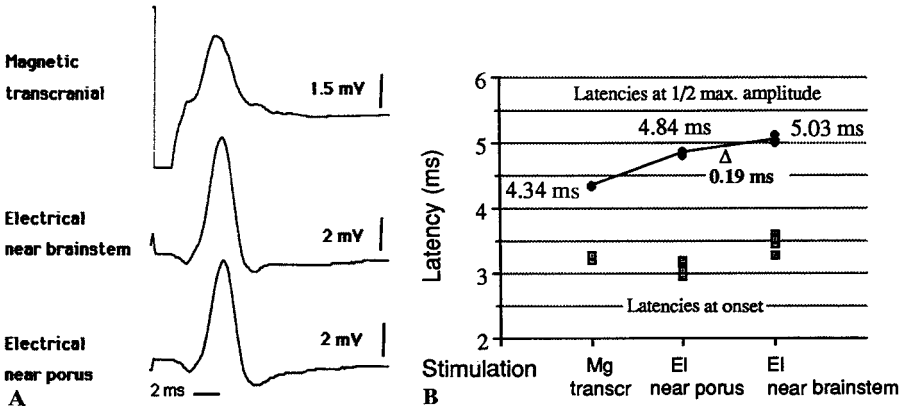


Fig. 2. A Responses recorded from the masseter muscle to transcranial magnetic stimulation and intraoperative electrical stimulation of the trigeminal nerve in a patient with normal trigeminal nerve function, who underwent an operation to remove an acoustic neurinoma in the posterior fossa (Case 1). **B** Latencies to half-peak amplitude of the responses from the masseter muscle in the patient whose responses are shown in Fig. 2A

near the brainstem and 2) at the location where the nerve exited the CPA through the porus trigeminalis.

Because high magnetic stimulation intensities resulted in more stimulus artifacts, we used the latencies measured from the stimulus to a point one half of the peak amplitude to the response, instead of using the onset latencies. These latencies were consistently shorter to magnetic stimulation than to direct electrical stimulation of the nerve 1) just distal to its exit from the brainstem (Δ 0.5 ms) or 2) just proximal to its entrance into the porus trigeminalis ($\Delta = 0.19$ ms) (Fig. 2).

Case 2: This patient at operation was found to have a meningioma 2 cm in diameter within Meckel's cave. Preoperatively the patient presented with slight paresis of the ipsilateral masseter muscle. No response could be recorded from this muscle to transcranial magnetic stimulation, although contralateral stimulation and recording resulted in a response with an amplitude of 1.1 mV and an onset latency of 4.0 ms.

Facial Nerve

Intraoperative Recordings

In 5 patients who underwent operations within the CPA, magnetic stimulation was performed after sterile draping of the patient but before the skin incision was made. The recording electrodes were left in place for intraoperative monitoring of nerve function, and after the facial nerve had been exposed it was stimulated electrically at various points along its intracisternal and labyrinthine course. Examples of the responses to this stimulation are shown in Fig. 3.

Figure 4 shows the onset latencies of all 5 patients' responses to preoperative transcranial magnetic and intraoperative intracranial electrical stimulation at four different stimulation sites. Comparison of onset latencies of responses to

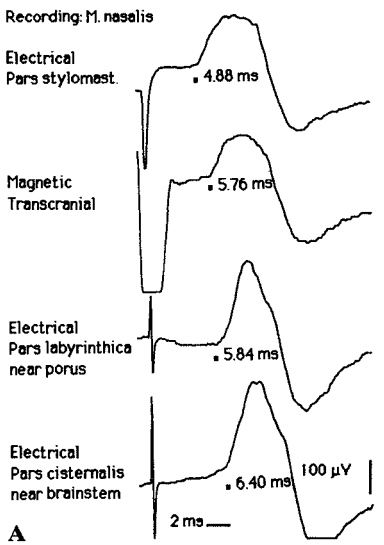
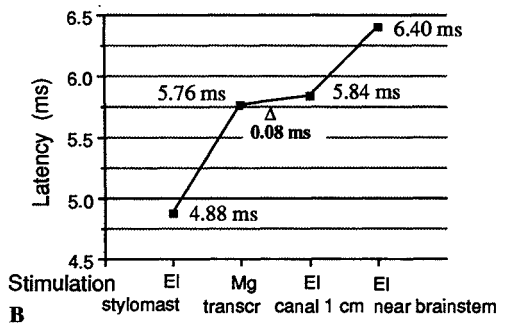


Fig. 3. A Responses recorded from the nasal muscle to transcranial magnetic and direct electrical stimulation of the facial nerve in a patient with normal facial nerve function who underwent an operation to remove a small acoustic neurinoma in the posterior fossa.



B Onset latencies of nasal muscle responses in the patient whose results are shown in Fig. 3A

magnetic stimulation and of responses to electrical stimulation in the CPA shows that responses to magnetic stimulation were always shorter than responses to electrical stimulation within the CPA, regardless of where in the CPA the facial nerve was stimulated. However, the latencies of the responses to transcranial stimulation were longer by up to 1.2 ms than the latencies of the responses to stimulation of the stylomastoid segment of the facial nerve. In 3 patients the undamaged facial nerve was stimulated electrically deep within the facial canal (intrameatal stimulation during the operation, Table 1). The onset latencies were identical or 0.2 ms shorter than the latencies to magnetic transcranial stimulation.

To study the role of cerebrospinal fluid (CSF) in mediating the magnetically induced electrical stimulus, the facial nerve was stimulated magnetically after removal of an acoustic neurinoma but before the dura was closed. Ringer solution was instilled and removed, then instilled and removed again, with magnetic stimulation being performed under each circumstance. The corresponding recordings from the nasal muscle are shown in Figure 5. In the presence of Ringer's solution, the onset of the responses was more clearly discernible,

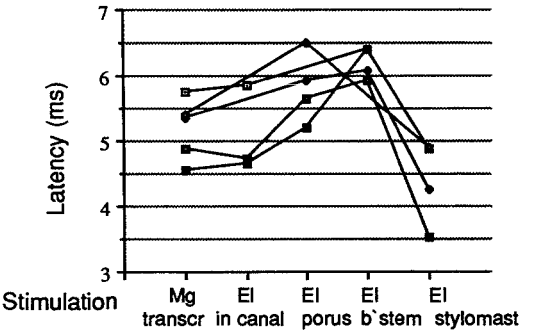


Fig. 4. Onset latencies of nasal muscle responses to transcranial magnetic and intraoperative direct electrical stimulation of the facial nerve in 5 patients who underwent posterior fossa surgery (see also Table 2)

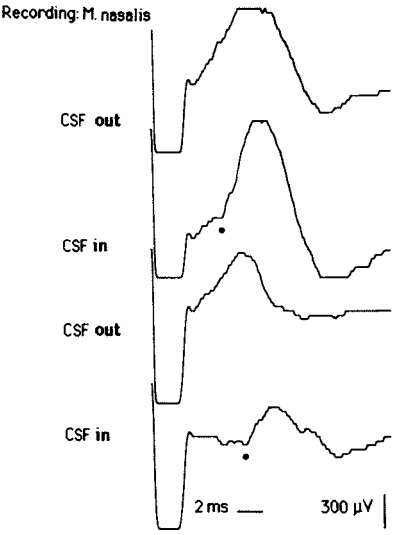


Fig. 5. Nasal muscle responses to intraoperative transcranial magnetic stimulation of the facial nerve. Traces 1 *(top)* and 3: When CSF and Ringer solution surrounded the nerve within the CPA, the clear onset of responses *(points)* indicates a rather focal excitation of the nerve. Traces 2 and 4 *(bottom)*: When CSF was removed, the nerve was excited less focally

although the amplitudes of the responses did not differ consistently with either instillation or removal of solution.

Investigations in Patients with Acute Facial Nerve Palsies

Transcranial magnetic and electrical stimulation was performed in 12 patients with acute facial nerve palsies of various etiologies and known sites of lesions in the facial nerve between the brainstem and the petrous bone. The patients' diagnoses and the results of stimulation are listed in Table 2.

In a patient suffering from a demyelinating focus in the brainstem, nasal muscle responses to transcranial magnetic and stylomastoid electrical stimulation were preserved. Two patients who experienced transient incomplete facial nerve palsy after surgical removal of acoustic neurinoma still had magnetically evoked nasal muscle responses 1 day postoperatively, but in both patients the responses to both transcranial magnetic and stylomastoid electrical stimulation disappeared within

Table 2. Results of stimulation in 12 patients with acute facial nerve palsy

No. Pts.	Diagnosis	Site of lesion transcranial[a]	Magnetic stylomastoid[a]	Electrical
1	Demyelinization	Brainstem	Normal	Normal
2	Acoustic Tumors, Postoperatively	Intracisternal CN VII	Normal	Normal
8	Bells Palsy	Labyrinthine CN VII	Pathological	Normal
1	Trauma	Petrous CN VII	Pathological	Normal

[a] The values were compared to the normative data obtained in 14 subjects from the same laboratory (Rösler et al. 1989).

some days after the operation, although the function of the facial nerve was partially preserved.

In 8 patients with acute Bell's palsy, and in 1 patient with complete facial palsy after petrous bone fracture, there was no response from the nasal muscle to magnetic stimulation, although responses were preserved in the early stage of illness to electrical stimulation of the nerve at its exit from the stylomastoid foramen.

Discussion

The recent introduction of a method for transcranial magnetic stimulation of motor cranial nerves (Murray et al. 1987; Schriefer et al. 1988; Benecke et al. 1988) had made it possible to study the neural conduction in the intracranial portions of cranial motor nerves. The discomfort caused by such magnetic stimulation is minimal, and subjects preferred magnetic stimulation to electrical stimulation when the facial nerve was stimulated where it leaves the stylomastoid foramen.

Transcranial magnetic stimulation of nerves opens new avenues in electroneurography permitting noninvasive assessment of the intracranial portion of the motor cranial nerves. Stimulation of these nerves intracranially can be used to assess central motor conduction time by motor cortex stimulation, where the peripheral nerve conduction time must be subtracted from the total latency of the responses. Additionally, peripheral motor conduction time may be used to assess intracranial lesions of these nerves. For example, in acute Bell's palsy, no muscular response can be evoked from the nasal muscle by transcranial magnetic stimulation (Rösler et al. 1989; Schriefer et al. 1988), but in the early stage of the disease stylomastoid stimulation of the facial nerve yields normal responses.

When determining the practical applications of this technique, the crucial question that must be asked is whether the actual site of excitation of the nerve fibres can be determined adequately. Because the magnetic coil is remote from the nerve fibers it excites, the principles that apply to intracranial stimulation differ from those that apply to stimulation of subcutaneous nerves. Whereas all intact axons of subcutaneous nerves are excited at a well-defined point beneath the

cathode, the conductivity of tissue over the parieto-occipital scalp and within the posterior fossa is far from homogeneous, so that the density and direction of the magnetically induced, transcranial stimulating current are difficult to predict.

When the magnetic coil that is used for transcranial stimulation is placed over the parieto-occipital scalp and the current flows clockwise in the coil as viewed from behind the patient's head for stimulation of the left side (and vice versa for stimulation of the right side) (Rösler et al. 1989), the facial and trigeminal nerves are stimulated simultaneously. Our intraoperative recordings provide evidence that the magnetically induced electrical stimulus is confined to the *intracranial segments* of these nerves. However, the latencies of the responses we measured indicate that the excitation of these nerves from such magnetic stimulation occurred *at a location that was distal to the CPA*. The results of both our intraoperative recordings and recordings in patients with acute lesions of the *facial nerve* at known locations support these findings and suggest that the actual site of magnetic stimulation lies within the labyrinthine part of the facial nerve rather than in its intracisternal course in the CPA. This is in contrast to the interpretation of the results of others (Maccabee et al. 1988; Benecke et al. 1988), who reached the conclusion that the actual site of neural excitation from magnetic stimulation occurs in the intracisternal course of the facial nerve.

The results of our study lead to similar preliminary conclusions regarding the *trigeminal nerve*. Thus, the latency of the masseter muscle response to transcranial magnetic stimulation was 0.19 ms shorter than the latency of the response to direct electrical stimulation of the nerve where it exits the CPA. Additionally, the patient with a meningioma within Meckel's cave had no response to magnetic stimulation in the masseter muscle, which was only slightly paretic. This is taken to indicate that the trigeminal nerve was not excitable at the focus of compression by the tumor. From this we concluded that transcranial magnetic stimulation of the motor portion of the trigeminal nerve occurs within Meckel's cave rather than more proximally.

Our experimental results provide evidence that cerebrospinal fluid (CSF) with its high electrical conductivity plays an important role in transmitting the magnetically induced currents to the respective nerves. Our conclusion, that magnetic stimulation of both the facial and trigeminal nerves occurs distal to their exits from the CPA, is based on the fact that both nerves are surrounded by CSF after as well as before they exit the CPA and enter their respective intradural canals. Anatomical studies have shown that CSF extends 7 to 16 mm (Lang 1981) into the facial canal, i.e., into its labyrinthine part. Similarly, contrast radiological studies performed during glycerol infiltration for treatment of tic douloureux show that the trigeminal nerve is surrounded by CSF also within Meckel's cave. Thus, stimulating currents generated by transcranial magnetic stimulation seem to be focused by the anatomical structures just discussed, resulting in excitation of the most distal portion of the nerves where they are still surrounded by CSF.

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Intraoperative Monitoring of Evoked Potentials in Microvascular Decompression Operations: Influence on Surgical Strategy

P. J. JANNETTA[1]

Summary

Neurophysiologic monitoring of patients during microvascular decompression (MVD) operations to treat cranial nerve disorders in the cerebellopontine angle (CPA) has become essential not only to the decrease in morbidity of these operations that now makes them a desirable option for patients with disorders such as hemifacial spasm and trigeminal neuralgia, but in some cases to the efficient completion of the procedure itself.

Because the surgeon must devote primary attention to events in the operative field, monitoring of evoked potentials during operations in the CPA must be the primary responsibility of another professional on the surgical team. When the electrophysiologist and the surgeon establish a collaborative relationship so that they communicate well and coordinate their efforts, solutions can be found to the many problems that arise during the performance and monitoring of surgical maneuvers in the CPA.

Specific situations in which intraoperative monitoring facilitates the surgical goals of MVD, and ways in which the surgical strategy may be altered because of monitoring, are discussed.

Monitoring of evoked potentials during operations in the cerebellopontine angle (CPA) has been of great benefit to patients; it has also had a significant influence on how the surgery is performed. This chapter reviews why and how surgeons may use intraoperative monitoring, including specific ways in which such monitoring has altered surgical strategy.

Development of Monitoring from the Surgeon's Perspective

The primary monitoring technique used by surgeons is nearly constant observation of the surgical wound. The effective surgeon tries constantly to visualize both the normal – and possibilities for the pathological – anatomy and physiology in the operative area. This view of anatomy must be three-dimensional, and it must constantly be revised with retraction, displacement, removal, or other alterations in

[1] Department of Neurological Surgery, University of Pittsburgh School of Medicine, 230 Lothrop Street, Pittsburgh, PA 15213, USA.

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tissues. Before operating microscopes came into routine use, operations in the CPA were associated with very high morbidity, and although use of the operating microscope helps the surgeon monitor the operative site visually, morbidity for these operations continued to be relatively high until the advent of intraoperative electrophysiologic monitoring of cranial nerve function. For example, before we began to use eighth nerve monitoring there was significant risk that the patient might awaken from anesthesia after an operation to relieve hemifacial spasm (HFS) with complete hearing loss. Electrophysiologic monitoring in these situations can result in significant decreases in operative morbidity because monitoring can alert the surgeon to the fact that activities seemingly unrelated to the surgical goal of the procedure, such as retraction, are causing changes in cranial nerve function that might result in severe postoperative deficit (Grundy et al. 1981; Møller and Jannetta 1983; Møller and Møller 1989). The use of appropriate monitoring techniques for patients undergoing operations in the CPA can make the difference between relief of a debilitating condition such as trigeminal neuralgia or hemifacial spasm with preservation of cranial nerve function and loss of hearing or facial function and possibly also failure to relieve the condition for which the patient underwent the operation.

Despite the importance of intraoperative monitoring of evoked potentials to the success of operative procedures in the CPA, the surgeon's attention must be focused primarily on the operative field. The surgeon can spare little time to watch monitor screens for evoked potential changes. For this reason, another professional must be primarily responsible for monitoring evoked potentials intraoperatively, and the surgeon's role in monitoring becomes collaborative with the individual whose primary function in the operating room is to perform the monitoring. The surgeon and electrophysiologist must agree on the goals of monitoring and must be committed to it; they must communicate well so that they can work as a team; and they must develop protocols for monitoring so that the technique is used for all patients in whom it is appropriate.

Intraoperative Monitoring in Practice

The surgeon may participate in monitoring in various ways. A simple but important consideration is where to place the recording electrode for monitoring of compound action potentials (CAP) from the auditory nerve so that optimal results are achieved with minimal interference in the surgeon's activities. In some instances this task can be problematic. Such an electrode must be positioned to provide a clear recording, but far enough away from the center of the operative field that it does not interfere with the surgeon's activities. In addition, the electrode must be secured well (we often twist the electrode wire around one of the dural stay sutures to achieve this).

Other circumstances will now be discussed with reference to specific procedures in the CPA.

Decompression of the Trigeminal Nerve

The distribution of pain in patients with trigeminal neuralgia gives clues to the location of vascular compression of the fifth cranial nerve (Jannetta 1977, 1981a). Thus, lower facial pain is caused by compression of the rostral portion of the nerve, central facial pain by lateral or medial compression, and upper facial tic by caudal compression. In addition, trigeminal nerve pain is often caused by multiple blood vessels, and all should be treated for the microvascular decompression (MVD) to be effective.

Brainstem auditory evoked potentials (BAEP) should be monitored to prevent damage to the eighth cranial nerve during decompression of the fifth cranial nerve via a superolateral approach over the cerebellum. Bennett developed the first usable method for recording trigeminal evoked potentials and found that changes do occur in these potentials – 83% of patients with trigeminal neuralgia have abnormal trigeminal evoked potentials in the area of their pain (Bennett and Jannetta 1980, 1983). Nevertheless, monitoring of these potentials provides no information about whether the trigeminal nerve has been decompressed or not. These potentials return to normal quite rapidly after the operation in 86% of patients.

Because we cannot identify from evoked potential monitoring when the fifth cranial nerve has been fully decompressed, the surgeon must search diligently for any sites of nerve compression and must relieve as many as possible. However, caution should be exercised in this regard, because transection of small perforating arterioles can lead to postoperative focal deficits, as these may be end arteries, especially in the lower pons and upper medulla. Small surface veins can be taken with impunity, but these have a tendency to recollateralize in the postoperative period (four to six months).

One monitoring technique we have found helpful during MVD to treat trigeminal neuralgia is Doppler ultrasound. Although we have not had a problem with air emboli since we began placing patients in the lateral rather than a modified sitting position for this operation, we have often noted temporary bradycardia during this procedure.

Although the mechanism is unknown, when a trigeminal nerve that has been severely compressed by blood vessels for a long time, such that the nerve is very distorted, is manipulated during MVD, the patient often becomes bradycardic. For this reason, it is valuable to use Doppler ultrasound to monitor the heart rate and rhythm in such patients. During one procedure, as a large implant of shredded Teflon felt was being placed between the trigeminal nerve and the superior cerebellar artery lying rostral to it, the patient's heart stopped. When the implant was removed, the heart started to beat again immediately. Several small pieces of felt were placed in this patient with no sequelae.

Monitoring During Microvascular Decompression for Hemifacial Spasm

Contrary to what was described for the fifth cranial nerve, where trigeminal neuralgia is often found to be caused by multiple blood vessels compressing the nerve, it is uncommon for more than one vessel to compress the seventh cranial

nerve to cause HFS (Jannetta 1981b). When the facial nerve is seen to be compressed by multiple vessels, they are most often the posterior inferior cerebellar artery (PICA) and the anterior inferior cerebellar artery (AICA), and these arteries can be moved as a unit.

The most highly developed system for monitoring function in a specific cranial nerve during MVD of that nerve is electromyography (EMG) for HFS. This technique not only provides early warning that the facial nerve is being injured, a warning that it is essential to heed because there is a direct relation between duration of injury due to retraction or compression of a nerve and the severity of nerve damage that results, but it also makes it possible to determine when the facial nerve has been adequately decompressed (Møller and Jannetta 1987).

Cranial nerves can be retracted medial-to-lateral with far less likelihood of injury occurring than when they are retracted lateral-to-medial. Likewise, the brainstem is at risk if the cerebellum is compressed into it during neurological retraction, but if the cerebellum is lifted posteriorly, gently and slowly so that retraction occurs by gravity and the retractor is merely used to support the cerebellum, the possibility of damage will be minimal and cerebellar hemorrhagic infarctions will be very rare.

A problem that may arise during MVD is secondary compression of the nerve that is not noticed during the operation and that causes reappearance of symptoms. For example, the surgeon may remove a large vessel, such as the vertebral artery, from the nerve and place an implant between the larger vessel and the nerve. However, a small vein or artery (such as a perforating vessel from the AICA) may have been resting lightly across the nerve in the brainstem, and placement of the implant may cause this smaller vessel to exert pressure on the facial nerve in the lower pons (Jannetta 1984). When this has happened, the patient's symptoms characteristically improve in the initial postoperative period, but then improvement plateaus and the symptoms become worse again over time. The compression of the nerve caused by the implant being pushed into the nerve can be found by reexploration of the area. The intraoperative monitoring of the abnormal muscle response (Møller and Jannetta 1987) during the initial procedure can identify such secondary compression of the facial nerve.

Another situation in which intraoperative monitoring can be of use to the surgeon occurs infrequently but may still be of interest. An occasional patient will present with HFS and trigeminal neuralgia ("tic convulsif"). In one such patient in whom the trigeminal nerve was compressed by a vein to cause leftsided trigeminal neuralgia, the compression of the facial nerve did not become apparent until the cerebellar retraction was released, at which time it was seen that the PICA pressed into the caudal side of the facial nerve. The site of such compression in typical HFS can be as far caudal as the pontomedullary junction.

Monitoring Auditory Nerve Function

In a review of the first 595 patients in whom BAEP were monitored by a number of different neurophysiologists, we found that permanent hearing loss occurred in only 6 patients, 2 of whom actually had acoustic tumors rather than injury. Thus,

BAEP monitoring to identify when the eighth cranial nerve may be at risk of injury is one of the most valuable monitoring techniques available.

Although others may not agree with us, our operating team believes that any change in the BAEP that occurs intraoperatively is significant, and so should be communicated to the surgeon.

We also monitor auditory nerve CAP (Møller and Jannetta 1983; Møller, 1988) when the nerve becomes exposed during MVD. CAP respond quickly to any change in status of the auditory nerve, and the alterations in potentials are easy to interpret. This makes CAP monitoring ideal in situations in which the site of compression is not immediately apparent. We also use CAP monitoring to help train resident neurosurgeons in the techniques for MVD.

Finally, intraoperative monitoring of evoked potentials is of benefit because it helps surgeons learn more about the anatomy and physiology of the cranial nerves and the effects of surgical manipulation on the nerves. The amount of information that can be gained about normal and abnormal nerve function from studying evoked potentials during MVD operations is incredible compared to the information gained by traditional means.

For example, we have learned from intraoperative monitoring that when a vein is coagulated a significant amount of heat is transmitted to surrounding tissues. We noted that when a vein overlying or near a nerve was coagulated without protecting the nerve from this transmitted heat, the nerve was likely to be injured. Also, because heat is transmitted more readily through fluid, a suction tip should be placed beside the nerve if the field is very wet and a low coagulating current should be used to minimize current spread to the nerve. Coagulating a vessel in stages – coagulating a portion, dividing the vessel, and coagulating further as needed – can also be performed to protect a nerve in close proximity to the blood vessel being coagulated.

Conclusions

A common misconception regarding intraoperative monitoring of evoked potentials is that it prevents injury. Monitoring BAEP, for example, is not a means of monitoring hearing, and it certainly cannot prevent damage to the auditory nerve – rather it only indicates when damage might have occurred. But because we have learned that any alterations in evoked potentials may signal increased risk to the nerve, we now retract and move instruments more slowly and gently, allowing time for the effects of minimal surgical manipulations to be reflected in changes in recorded potentials, and altering the surgical technique accordingly should changes be noted.

Thus, surgeons are, in a way, practicing "preventive surgery" when they perform operations in the CPA with intraoperative monitoring of evoked potentials. Not only does monitoring help surgeons perform the operation more safely and effectively in an individual patient, it helps us expand our knowledge of anatomy and physiology and the limitations of what can be accomplished by the surgical technique itself. The feedback provided to the surgeon by intraoperative monitoring is an invaluable teaching tool.

Because it enables the surgeon to alter surgical technique to decrease morbidity, and helps the surgeon learn more about the anatomy and physiology of the area being operated upon, intraoperative monitoring is one of the most valuable tools in the surgeon's armamentarium.

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Brainstem Auditory Evoked Potential Monitoring Related to Morbidity and Mortality in Basal and Posterior Fossa Tumors

C. B. LUMENTA[1], M. KRAMER, W. VON TEMPELHOFF, and J. HAMACHER

Summary

In the past 7 years at our institution, intraoperative brainstem auditory evoked potential (BAEP) monitoring was performed during 114 operations in the posterior fossa. Twenty-seven patients had a midline tumor, 17 a cerebellar hemispheric tumor, 62 a cerebellopontine angle (CPA) or basal tumor, and 8 patients had vascular disease of the posterior fossa. The short term outcome (3 weeks postoperative) of these 114 patients was excellent in 48 cases (42.1%), good in 44 (38.6%), and poor in 10 (8.8%); 12 patients (10.5%) died.

Analysis of the BAEP recorded in the 22 patients in the last two outcome groups from the time dissection of the mass lesions began until it was completed showed that changes in the interpeak latencies of waves I to V (BAEP, Grade II) are very important and sensitive indicators of brainstem dysfunction. However, conclusive evidence of dysfunction is a decrease in amplitude of wave III and/or wave V to less than 0.08 μV or 0.09 μV, respectively (BAEP, Grade III). Our findings suggest that when changes last more than 120 minutes the patient is very likely to die.

In the early 1980s many authors, including Allen et al. (1981), Grundy et al. (1981), and Hashimoto et al. (1980) demonstrated the usefulness of brainstem auditory evoked potential (BAEP) monitoring during posterior fossa operations, especially in patients with mass lesions affecting the eighth cranial nerve or the brainstem. Although complicated and difficult surgical procedures can be performed with less morbidity and mortality when a microsurgical technique is used, posterior fossa operations in the vicinity of the brainstem may cause severe neurological complications.

For this reason, we compiled data regarding morbidity and mortality of patients who had undergone operations in the posterior fossa with the aid of intraoperative BAEP monitoring. We analyzed the results of all patients who suffered brainstem morbidity (defined as coma, respiratory failure, vegetative dysregulation, and positive pyramidal signs) or who died within 3 weeks after the operation. We compared the changes that occurred in the BAEP of these patients during dissection of the pathologic processes until the end of the operation with their postoperative neurological status.

[1] Neurochirurgische Universitätsklinik, Moorenstraße 5, W-4000 Düsseldorf 1, FRG.

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Patients and Methods

In the past 7 years at our institution, intraoperative monitoring of BAEP was performed during 114 operative procedures in the posterior fossa. Fifty-one of the patients were male and 63 were female. The youngest patient was 3 years old and the oldest 73, with an average age of 46.7 years. We used the same BAEP monitoring technique we described previously (Lumenta et al. 1984).

Of the 114 patients, 27 had a midline tumor (6 pinealomas, 10 medulloblastomas, 5 astrocytomas, 3 ependymomas, and 3 metastatic tumors), and 17 had cerebellar hemispheric tumors (4 hemangioblastomas, 1 benign intracerebellar cyst, 8 metastatic tumors, and 4 astrocytomas). The largest group of patients were those with cerebellopontine angle (CPA) and skull base tumors. Of the 62 patients in this group, 45 had neurinomas, 10 meningiomas, 2 chordomas, 3 hematomas, 1 metastatic tumor, and 1 epidermoid cyst. Of the final 8 patients, 4 had an aneurysm of the basilar artery, 2 an arteriovenous malformation (AVM), and 2 a microvascular compression of the trigeminal nerve (Table 1).

The short term outcome determined 3 weeks postoperatively for the 114 patients was excellent in 48 (42.1%), good in 44 (38.6%), and poor in 10 (8.8%); 12 patients (10.5%) died within 3 weeks after the operation.

Table 1. Lesions in 114 patients who underwent intraoperative BAEP monitoring at the Department of Neurosurgery, University of Düsseldorf, between 1982 and 1988

Lesion	No. Patients
I. Midline (n = 27)	
1. Pinealoma	6
2. Medulloblastoma	10
3. Astrocytoma	5
4. Ependymoma	3
5. Metastatic tumor	3
II. Hemispheric (n = 17)	
1. Hemangioblastoma	4
2. Benign cyst	1
3. Metastatic tumor	8
4. Astrocytoma	4
III. Cerebellopontine angle/basal (n = 62)	
1. Neurinoma	45
2. Menigioma	10
3. Chordoma	2
4. Hematoma	3
5. Metastatic tumor	1
6. Epidermoid	1
IV. Others (n = 8)	
1. Basilar aneurysm	4
2. Arteriovenous malformation	2
3. Cranial nerve compression	2

We analyzed intraoperative BAEP findings of all 22 patients in the groups with poor outcome or death from the beginning of dissection of the tumor or aneurysm until the end of the procedure. According to the classification of BAEP findings developed by Anderson et al. (1984) for patients with severe head injuries, we selected four categories for the BAEP findings in our patients:

Grade I: Normal BAEP
Grade II: Interpeak latency of waves I through V more than 4.07 ms (+ 1 SD)
Grade III: Amplitude of wave III less than 0.08 μV and/or amplitude of wave V less than 0.09 μV (− 2 SD)
Grade IV: Loss of BAEP

Results

Poor Outcome

Table 2 and Fig. 1 show the results for the 10 patients (6 males and 4 females, aged 6 to 68 years) in whom brainstem function was still impaired 3 weeks after the operation. Six of the 10 had undergone operations for a tumor in the midline, 3 for a tumor in the CPA, and 1 for a tumor in the petroclival region.

The time from the beginning of dissection of the tumor until the end of the operation ranged between 130 minutes (patient No. 2, medulloblastoma) and 375 minutes (patient No. 3, medulloblastoma) with an average of 218.5 minutes. The number of BAEP recordings made during these procedures ranged between 18 (patient No. 2, medulloblastoma) and 58 (patient No. 8, acoustic neurinoma).

Increased interpeak latency of waves I through V of more than 4.07 ms (BAEP, Grade II) also occurred in 9 patients, in from 1 of the 26 recordings (3.8%, patient No. 7, acoustic neurinoma) to 13 of 40 recordings (32.5%, patient

Table 2. Results in 10 patients who had poor outcome

Patient No.	Sex	Age yrs.	BAEP No.	Time min.	Diagnosis
1	M	50	23	150	ependymoma
2	F	22	18	130	medulloblastoma
3	M	16	48	375	medulloblastoma
4	F	6	35	225	ML-astrocytoma
5	M	49	40	300	pinealoma
6	M	52	32	250	pinealoma
7	M	52	26	160	acoustic neurinoma
8	M	26	58	305	acoustic neurinoma
9	F	58	28	120	acoustic neurinoma
10	F	68	26	170	meningioma

BAEP, number of BAEP measurements;
F, female;
M, male;
ML, midline;
Time, time from beginning of dissection of the tumor or aneurysm to the end of the operation.

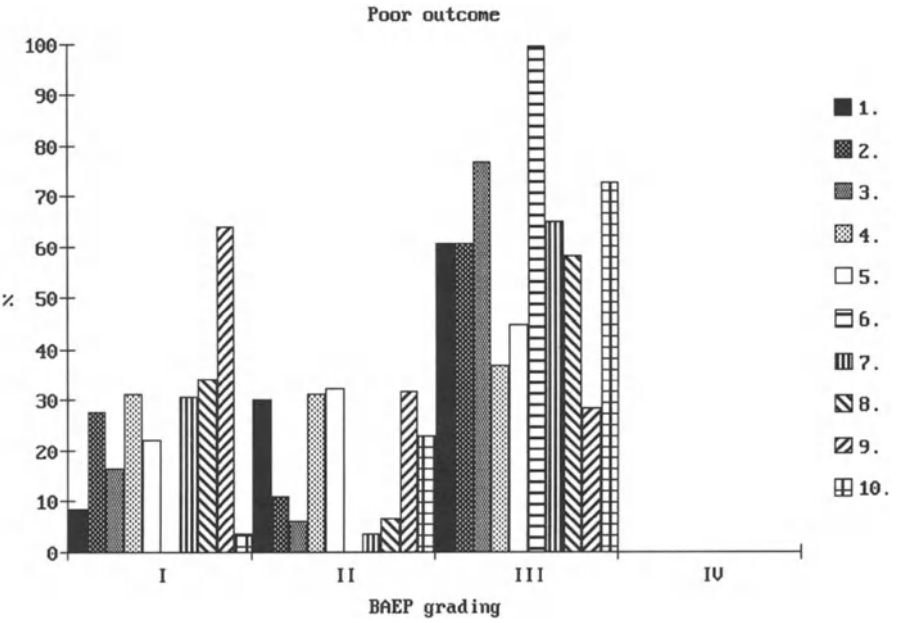

Fig. 1. Frequency of occurrence (%) of different BAEP changes in patients with poor outcome

No. 5, pinealoma). Because this type of BAEP change was found to occur at any stage of the dissection, it could not be attributed to a specific surgical manipulation.

A decrease in the amplitude of wave III and/or wave V of more than 2 SD (BAEP, Grade III) was noted in all 10 of these patients. This finding was noted in from 8 of 28 recordings (28.6%, patient No. 9, acoustic neurinoma) to 32 of 32 recordings (100%, patient No. 6, pinealoma). These BAEP changes were recorded mainly during dissection and removal of tumor from the brainstem or midbrain. Loss of BAEP (BAEP, Grade IV) was never seen during intraoperative recording from patients in this study.

Outcome Death of the Patient

Table 3 and Fig. 2 show the results for the 12 patients who died within 3 weeks after undergoing posterior fossa surgical procedures. There were 6 men and 6 women between 25 and 73 years old in this group. Two patients had an aneurysm of the basilar artery, 2 a midline tumor (astrocytoma and ependymoma), 1 a recurrence of a hemangioblastoma, 3 an acoustic neurinoma (1 a recurrence), 1 a trigeminal neurinoma, and 3 a meningioma (1 a recurrence at the craniocervical junction, 1 a petroclival lesion, and 1 a lesion at the tentorial notch).

The shortest time needed for removal of the lesion was 95 minutes (patient No. 22, tentorial meningioma) and the longest time was 315 minutes (patient No. 21,

Table 3. Results in 12 patients who died

Patient No.	Sex	Age yrs.	Dissection min.	Diagnosis
11	F	58	160	basilar aneurysm
12	M	61	240	basilar aneurysm
13	M	25	240	ML-astrocytoma
14	M	53	220	ependymoma
15	F	49	135	hemangioblastoma[a]
16	M	73	265	acoustic neurinoma
17	M	52	180	acoustic neurinoma
18	M	65	190	trigeminal neurinoma
19	F	48	215	acoustic neurinoma[a]
20	F	61	230	meningioma[a]
21	F	69	315	meningioma
22	F	68	95	meningioma

F, female;
M, male;
ML, midline;
[a] recurrence

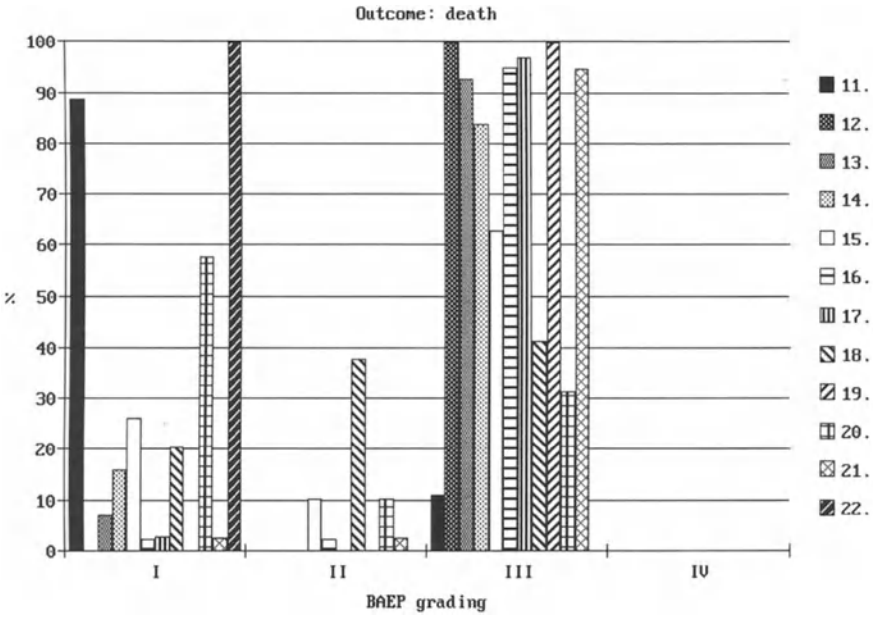

Fig. 2. Frequency of occurrence (%) of different BAEP changes in patients who died

petroclival meningioma). The number of BAEP recordings made varied between 11 (patient No. 22, tentorial meningioma) and 42 (patient No. 16, acoustic neurinoma). Grade II BAEP were seen in only 5 patients with a frequency of from 1 of 42 recordings (2.4%, patient No. 16, acoustic neurinoma) to 11 of 29 recordings (37.9%, patient No. 18, trigeminal neurinoma). In 11 patients, Grade

III BAEP occurred in from 2 of 18 recordings (11.1%, patient No. 11, basilar aneurysm) to 25 of 25 recordings (100%, patient No. 12, basilar aneurysm), and 18 of 18 recordings (100%, patient No. 19, recurrence of an acoustic neurinoma). As in those with poor outcome, loss of BAEP (BAEP, Grade IV) was not seen in any of the patients who died.

Case Reports

Patient No. 4: A 6-year-old girl was admitted to our service with complaints of headache, nausea, and vomiting. Physical examination revealed typical signs of cerebellar dysfunction, including ataxia and nystagmus, and signs of increased intracranial pressure were evident on fundoscopy. Computed tomography (CT) showed a large midline pathologic enhancement in the posterior fossa, with compression of the fourth ventricle and occlusive hydrocephalus. A ventriculoperitoneal shunt was inserted. One week later, the tumor was totally excised by a microsurgical technique using an ultrasonic aspirator (CUSA).

BAEP were normal before and at the beginning of tumor dissection. Later in the procedure, the interpeak latencies of waves I and V became prolonged and the amplitude of wave V, and later that of wave III, became abnormal. These pathologic changes persisted until the end of the operation (Fig. 3).

For 4 days after the operation, the girl was comatose and in respiratory failure. Neurological examination showed spasticity of all four limbs with positive pyramidal signs on both sides. CT scan at that time showed no signs of bleeding. This patient's spasticity and pyramidal abnormalities resolved 3 months after the operation, and at the time of this report 17 months after the operation the girl has only mild cerebellar symptoms and attends a school for normal children.

Patient No. 18: This 65-year-old man visited his physician because of symptomatic facial pain on the right side. CT scan showed that a large tumor was

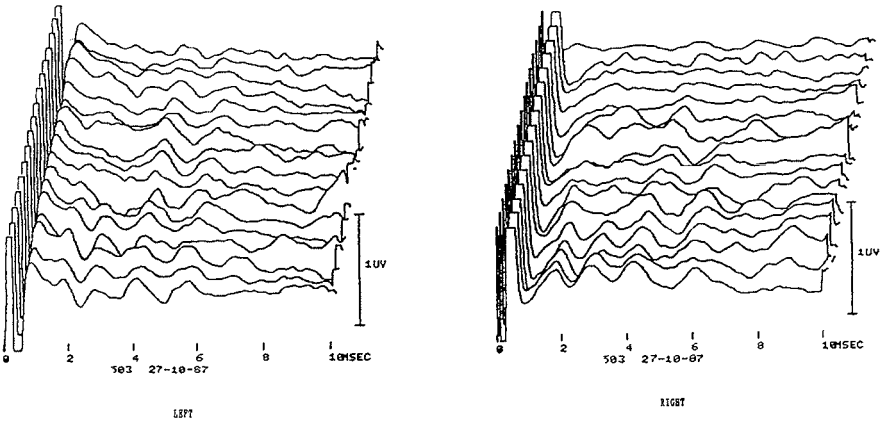

Fig. 3. BAEP recordings in a patient (patient No. 4) with a midline astrocytoma who had a poor outcome of surgery (baseline recordings are at the bottom)

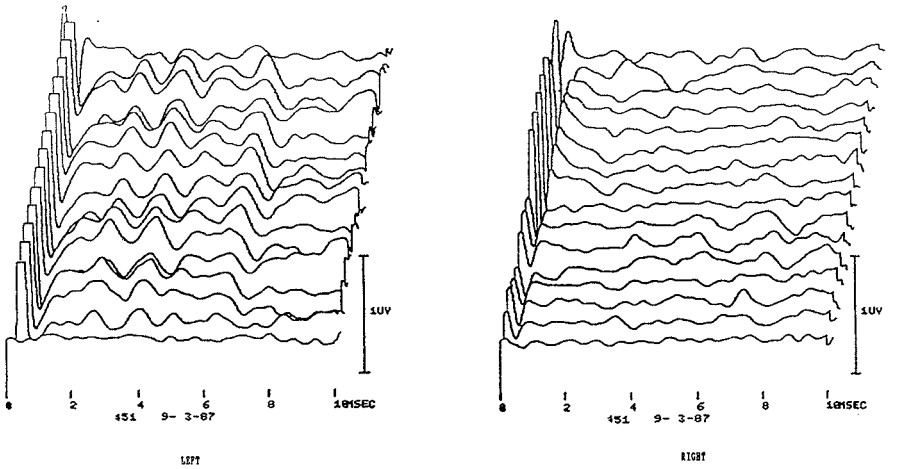

Fig. 4. BAEP recordings in a patient (patient No. 18) with a trigeminal neurinoma who died within 3 weeks after surgery (baseline recordings are at the bottom)

present in the CPA on the right side. On admission to hospital the patient had hypalgesia of the right trigeminal nerve, greater for the maxillary and mandibular branches. He had no hearing deficit, but did complain of vertigo. The tumor was removed totally through a right suboccipital craniectomy. At the beginning of tumor dissection, BAEP were abnormal on both sides, possibly because of the position of the retractor. After the retractor was repositioned, the BAEP on the left side became normal and remained normal until the end of the procedure. The BAEP on the right, however, showed decreased amplitude of wave I, although this wave was never lost, and the interpeak latencies of wave I through V were prolonged. Later the amplitudes of both waves III and V decreased. These changes were stable until the end of the procedure (Fig. 4).

The patient remained comatose postoperatively. A CT scan showed severe subarachnoid hemorrhage and general brain edema, and the patient died 6 days postoperatively.

Discussion

Intraoperative BAEP monitoring has been established as a routine procedure during operation in the posterior fossa (Lumenta et al. 1984, 1986; Schramm et al. 1986, 1988). In the current study of 114 patients who underwent posterior fossa operations with intraoperative BAEP monitoring, brainstem function decreased in 8.8%, and 10.5% of patients died during the 3 weeks after surgery.

To establish a possible correlation between the BAEP changes and postoperative neurological status of the patients in our study, the data were analyzed statistically. The mean frequency of each type of BAEP finding (Grade I, II, or III) was estimated for the two groups of patients, those who had poor neurological out-

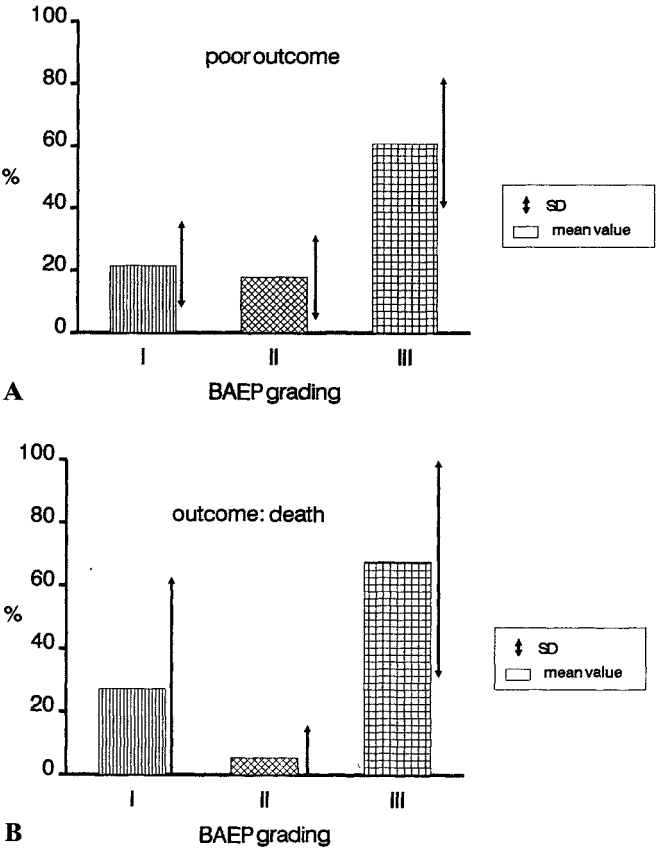

Fig. 5. Mean values and standard deviation *(SD)* of different BAEP changes in patients **A** with poor outcome, and **B** who died

come and those who died. This estimation established that standard deviations for the BAEP changes in the group of patients who died were significantly higher for Grade I and Grade III than were those in the group with the poor outcome. There was no significant difference in the mean values for the two groups. The frequency of Grade II BAEP changes was significantly higher ($P < 0.01$) in the group with poor outcome than in those who died (Fig. 5A and B).

Of the patients who died, patient No. 11 had normal BAEP in 88.9% of 18 recordings, and patient No. 22 had 100% normal BAEP. Although these patients had normal BAEP intraoperatively, they died within 3 weeks postoperatively. Such false-negative findings were not found in any other outcome group. There were in our study, however, no false-positive findings on BAEP monitoring such as Allen et al. (1981) described.

In an unpublished study we found that reversible changes occurred in the BAEP in all cases in the "excellent" and "good" outcome groups. These changes in BAEP were mostly of Grade II, consisting of prolonged interpeak latencies of waves I through V. Grade III BAEP changes occurred less than 10% of the time in these patients' recordings.

Other investigators such as Grundy et al. (1981) and Schramm et al. (1986) described the occurrence of BAEP changes associated with retractor positioning, suctioning, tumor dissection, and other surgical manipulations and noted that these changes reversed after the manipulation ceased. In a previous study (Lumenta et al. 1986) comprising 59 patients who had undergone posterior fossa operations we found that pathological BAEP changes lasting for more than 2 hours caused impairment in brainstem function.

The results of the present study showed that Grade II BAEP changes in patients with poor outcome lasted between 6.1 and 97.5 minutes, with a mean of 36.7 minutes; Grade III BAEP changes lasted between 34.3 and 250 minutes, with an average of 132.8 minutes. In patients who died, Grade II BAEP changes lasted between 6.3 and 72.1 minutes, with an average of 12.6 minutes, and Grade III BAEP changes lasted between 17.8 and 240 minutes, with a mean of 149.3 minutes. Thus, in both the groups of patients with poor outcome and those who died, Grade III BAEP changes lasted on average longer than 120 minutes, with the mean duration in those that died being longer than in those with poor outcome.

The classification of BAEP changes just described was arbitrary. However, the advantage of using the modified classification system, described by Anderson et al. (1984), in monitoring intensive care patients with severe head injuries (Link 1988) had already been established.

The results of our present study show that changes in interpeak latency of waves I and V are very sensitive indicators and thus very important in the detection of brainstem dysfunction; however, decreased amplitude of wave III and/or wave V is conclusive evidence that brainstem dysfunction is highly likely. These results confirm those of Schramm et al. (1988), who suggested that during intraoperative BAEP monitoring a reduction in amplitude is more important than an increase in latency.

In our opinion, it is mandatory to monitor BAEP during operative procedures in the posterior fossa because more complicated surgical procedures are now being undertaken due to advances in microneurosurgery techniques. It is helpful to use the BAEP grading system we described in this report because such information is more valuable to the surgeon than raw data on the changes in individual waves. We found that reduction of the amplitude of wave III to less than 0.08 μV or of wave V to less than 0.09 μV (BAEP, Grade III) is a sign of a poor prognosis, and one of which the surgeon should be informed immediately because our results suggest that if these changes last more than 120 minutes death of the patient is likely.

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Lessons from Brainstem Auditory Evoked Potential Monitoring During Microvascular Decompression for Trigeminal Neuralgia and Hemifacial Spasm

M. Sindou[1], D. Ciriano[1], and C. Fischer[2]

Summary

Continuous monitoring of brainstem auditory evoked potentials (BAEP) during microvascular decompression (MVD) operations in the cerebellopontine angle (CPA) provides the surgeon with information regarding the integrity of the auditory pathway. The most dangerous moments in this type of surgery are during cerebellar retraction (especially if the CPA is approached laterally) and during vascular manipulation, especially of the labyrinthine artery, which can generate vasospasm. To avoid stretching the eighth nerve, a supracerebellar approach should be made under the tentorium for operations to relieve trigeminal neuralgia, and an inferolateral approach should be made along the ninth and tenth nerves for operations to relieve hemifacial spasm.

A close relationship between severe intraoperative BAEP changes and permanent postoperative hearing loss could be demonstrated, which would provide further evidence to the value of intraoperative monitoring of BAEP.

Introduction

Continuous monitoring of brainstem auditory evoked potentials (BAEP) has been shown to be a useful tool in detecting operative damage to auditory pathways during microvascular decompression (MVD) of cranial nerves in the posterior fossa (Chiappa 1983; Fischer 1989; Fischer et al. 1985; Friedmann et al. 1985; Grundy et al. 1982; Jacobson and Tew 1987; Møller and Møller 1985, 1989; Radtke et al. 1989; Raudzens and Shetter 1982; Schramm et al. 1989; Watanabe et al. 1989). As a matter of fact, changes in electrophysiological events arise before irreversible anatomical lesions occur. Beyond indicating direct injury to the auditory nerve, which is infrequent and easily recognizable, BAEP monitoring helps prevent irreversible deficits due to excessive traction on the nerve or aggressive manipulation of its vascular supply, as has been stressed by several authors of reports on experimental studies (Sekiya and Møller 1987, 1988). Investigators who had used BAEP monitoring in large series of patients undergoing MVD were able to demonstrate a decrease in operative morbidity associated with

[1] Department of Neurosurgery, Hôpital Neurologique, UFR Grange blanche Université Claude Bernard, 59 Bd Pinel, 69003 Lyon, France.
[2] Service de Neurologie fonctionnelle et épileptologie, Hôpital Neurologique, 59 Bd Pinel, 69003 Lyon, France.

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the use of this technique (Grundy et al. 1982; Møller and Møller 1989; Radtke et al. 1989).

Because it would be impossible to monitor the approximately 3800 patients with various pathologies operated upon each year in our institution who are eligible for intraoperative evoked potential monitoring, we selected for BAEP monitoring only the patients at high risk of hearing loss.

This chapter reports on a retrospective study of 34 individuals who underwent BAEP monitoring during MVD: 17 of the 325 and 17 of the 25 who underwent the procedure for trigeminal neuralgia (TN) and hemifacial spasm (HFS), respectively, over the last 4 years. The fact that there were the same number in each group is totally fortuitous. The 17 patients with TN were monitored at the beginning of the study period. Monitoring patients undergoing MVD for TN was then abandoned, after the surgical risks had been identified and the technical means to avoid them were incorporated. Conversely, the 17 patients with HFS were studied more recently, as BAEP monitoring for patients with this pathology seemed more useful to us as time went on.

The aim of our work was to correlate changes in BAEP: 1) with the surgical maneuvers that might be responsible for these alterations, so that we could modify our technique to minimize the risks of the procedure in nonmonitored patients; and 2) with postoperative hearing disturbances, so that we could define prognostic criteria based on the results of BAEP monitoring.

Patients and Methods

Patients

The TN group consisted of 9 men and 8 women, 24 to 72 years old (55.6 on average), and the HFS group consisted of 3 men and 14 women, 30 to 75 years old (51 on average). An audiogram was obtained for each patient preoperatively, and BAEP were recorded from 12 with TN and 14 with HFS. All 34 patients underwent CT scanning preoperatively, and 26 underwent angiography. For all patients with HFS, both pure tone audiometry and BAEP recordings were obtained postoperatively. In the TN group, audiograms were obtained in only 7, BAEP were obtained in 3, and 4 had both postoperatively, but the 3 patients who complained of permanent or even transient auditory disturbances postoperatively underwent a complete evaluation with pure tone audiometry and BAEP recording.

Surgical Technique

Our technique was progressively modified as new information was obtained from BAEP recordings.

Originally, patients were operated upon in the sitting position, through a retromastoid craniotomy 4 cm in diameter. Access to the Vth (for TN) or the VIIth (for HFS) cranial nerve was gained through a lateral cerebellar approach. Once the area of vascular compression was identified and the artery detached and transposed, a piece of Dacron was inserted between the artery and the nerve.

Table 1. Stimulus and recording parameters for brainstem auditory evoked potentials

Parameter	Description
Stimulation	
site	ipsilateral, insert transducers
stimulus	alternating compression and rarefaction clicks
intensity	90 dB HL
duration	100 μs
rate	20 Hz
contralateral masking	wideband pseudorandom noise, 70 dB
Recording	
site	channel 1, Cz–A1; channel 2, Cz–A2; common isolation ground
electrodes	hypodermic needles
impedance	less than 3000 ohms
filters	160 to 1600 Hz
sensitivity	± 25 μV
sweep time	20 ms
repetitions per average	at least 1280, usually 2000

Later in the study, the patient's position was changed to a lateral one and the craniotomy was reduced to a small triangle (2 cm base and 1.5 cm height), so as to avoid abundant leakage of cerebrospinal fluid and excessive cerebellar retraction. The advantages of these technical changes are analyzed in detail elsewhere (Sindou et al. 1990).

After intraoperative BAEP recordings had shown that avoiding lateral retraction of the cerebellum reduced stretching of the VIIIth cranial nerve, the lateral cerebellar approach was replaced by a superolateral approach to the Vth cranial nerve and by an inferolateral one to the VIIth cranial nerve.

BAEP Monitoring

Stimulation and recording parameters used in our series are summarized in Table 1.

Changes in electrophysiological events were interpreted intraoperatively by the neurophysiologist (C.F.), who was simultaneously watching the surgical procedure on the videoscreen that showed the view through the microscope. The surgeon was warned every time the latency or amplitude of peak V of the BAEP changed significantly. (We regarded an increase in latency of 0.5 ms in 10 minutes, or a sudden change during an operative manipulation, or a 50% decrease in the wave I/V amplitude ratio to be significant.)

Results

Relief of TN or HFS

Trigeminal neuralgia (TN) was totally relieved in 12 patients, partially in 2, and not at all in 3 patients (failure rate of 17.6%). The follow-up period ranged from

2.5 to 4.6 years, with an average of 3.5 years. Hemifacial spasm (HFS) was totally relieved in 15 patients, partially in 1, and not at all in 1 patient (i.e., failure rate of 5.8%). The follow-up period ranged from 1 to 4.5 years, with an average of 2 years, for HFS patients.

Effects on Hearing

Postoperative auditory function was evaluated by pure tone audiometry at 500, 1000, 2000, and 4000 Hz. A mean threshold increase of 20% or more over preoperative levels was considered significant. Three patients with TN and 2 with HFS suffered a transient hearing loss, and 1 patient in each group suffered a permanent hearing loss.

Intraoperative Changes in BAEP

During intraoperative monitoring several changes in the pattern of BAEP were noticed, namely 1) an increase in latency, 2) a decrease in amplitude (with or without wave morphology degradation), and 3) even abolition of responses. These changes were categorized according to their degree of severity, using a system that we modified from Grundy et al. (1982) (see Table 2).

Figure 1 shows the distribution of patients in our TN and HFS series according to category of intraoperative BAEP findings. More pronounced intraoperative changes have been observed in patients with HFS.

Correlations Between BAEP Changes and Surgical Maneuvers

In order to identify surgical maneuvers during the operation that pose a risk of hearing loss, the latency of peak V was determined during each step of the MVD procedure (approach, cerebellar retraction, decompression itself, and closure). To help in visualization of the surgical risks, the values of peak V latencies at each step of the procedure were averaged for the 17 patients in each group, and these values are represented graphically in Fig. 2.

The curves showing the average latencies for TN and HFS patients have approximately the same shape, but the curve for the HFS patients indicates more

Table 2. Categories of intraoperative BAEP findings

Category	Characteristics
1	Minimal changes
2	Increase in latency returning to normal befor end of operation
2a	Increase in latency without normalization before end of operation
3	Abolition or partial decrease of responses returning to normal before end of operation
3a	Abolition or partial decrease of responses without complete normalization before end of operation
4	Total loss of responses lasting through the entire operation

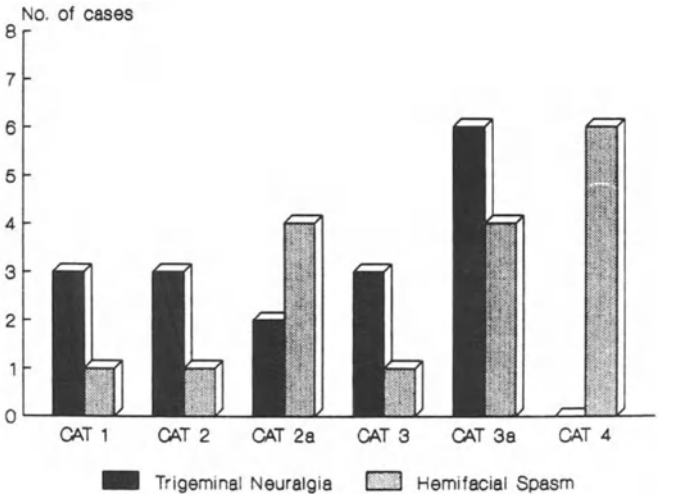

Fig. 1. Distribution of patients by category of intraoperative BAEP findings. Categories *(CAT)* are defined in Table 2

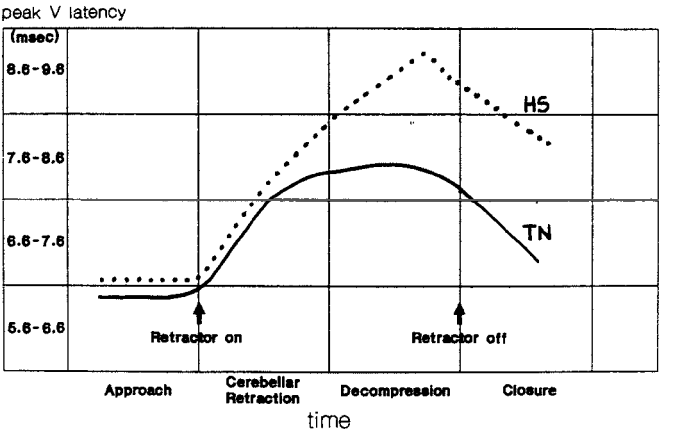

Fig. 2. Peak V latencies as a function of surgical maneuvers; averaged results of 17 patients with trigeminal neuralgia *(TN)* and 17 patients with hemifacial spasm *(HS)* are shown

severe changes. In all patients, latency of peak V increased significantly and rapidly after placement of the retractor on the cerebellar hemisphere. Latency continued to increase but more slowly, until the surgeon had stopped dissection of neurovascular structures, which requires a minimal degree of positive retraction of the cerebellum. Then there was a marked decrease in latency as soon as the retractor had been removed.

In patients with TN, abolition of peak V occurred once and was due to a difficult vascular dissection in the vicinity of the VIIIth cranial nerve. In patients

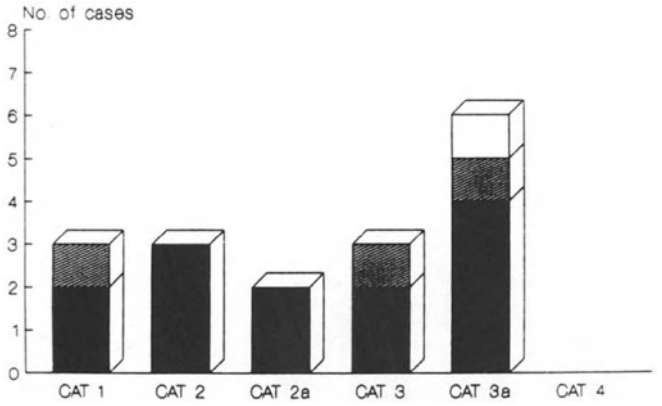

Fig. 3. Postoperative changes in pure tone thresholds in patients who were operated upon for TN as a function of intraoperative BAEP findings. Categories *(CAT)* are defined in Table 2. (*black* normal hearing; *hatched,* transient loss; white, permanent loss)

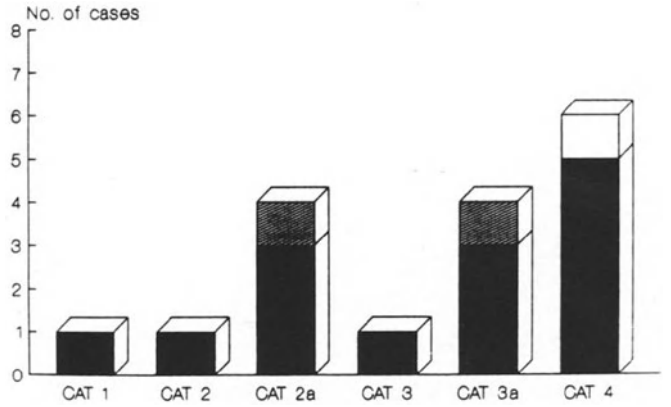

Fig. 4. Postoperative changes in pure tone thresholds in patients who were operated upon for HFS as a function of intraoperative BAEP findings. Categories *(CAT)* are defined in Table 2. (*black,* normal hearing; *hatched,* transient loss; *white,* permanent loss)

with HFS, abolition of peak V was noticed in 7 cases and was related to the direct approach and manipulation of the facial and acoustic nerves.

Prognostic Value of Intraoperative BAEP Changes

Intraoperative changes in BAEP could be used to define prognostic criteria for postoperative auditory function.

In our series of TN patients (Fig. 3), 75% of the patients who suffered a hearing loss (2 cases of transient hearing loss and 1 case of permanent hearing loss) belonged to categories 3 and 3a regarding intraoperative BAEP changes.

Sixty-six percent of the patients with HFS (Fig. 4), who suffered a hearing loss (1 case of transient hearing loss and 1 case of permanent hearing loss) belonged to categories 3a and 4 regarding BAEP changes.

These results indicate that there is a higher probability of postoperative hearing loss in those patients whose BAEP show significant changes during the operation.

Discussion and Conclusion

I. Intraoperative recording of BAEP makes it possible to identify the steps of MVD operations that pose the greatest risks of injuring the auditory pathways. We found that the greatest risk of hearing loss occurred during cerebellar retraction and vascular manipulation.

A supracerebellar approach under the tentorium is preferable to a lateral approach in MVD operations for TN because the former presents less risk of stretching the VIIIth cranial nerve. By preserving the venous affluents to the superior petrosal sinus excessive retraction of the cerebellum can be prevented and by not opening the arachnoid membrane around the VIIth and VIIIth cranial nerves, MVD operations for TN can be performed with greater safety.

Lateral retraction of the cerebellum during MVD operations for HFS carries a greater risk of harming auditory function because it stretches the VIIIth cranial nerve both directly and also indirectly stretches the cerebellar flocculus attached to the VIIIth nerve. The risk of stretching the VIIIth cranial nerve can be reduced by using an inferolateral approach, moving along the IXth and Xth cranial nerves to the VIIth cranial nerve from below. Manipulation of the labyrinthine artery and/or even coagulation of the tiny arterial branches that supply the VIIIth nerve can produce severe BAEP changes; therefore special care must be taken to avoid aggressive maneuvers.

II. In our series, good correlation could be demonstrated between the absence or presence (and the kinds) of intraoperative BAEP changes and postoperative auditory function. Permanent hearing loss was observed only when severe BAEP changes, i.e., categories 3a and 4, occurred. Figures 3 and 4 show how intraoperative BAEP changes can be used to predict postoperative function.

The results of intraoperative BAEP monitoring in our series of patients as well as in series in the literature prove that such monitoring is an important – if not always necessary – tool for this type of operation. However, when monitoring is not available routinely, priorities have to be established for its use.

Changes in surgical technique have reduced the frequency of postoperative hearing loss in MVD operations for TN to such an extent that we decided not to monitor these patients, but monitoring became a priority for our HFS patients because of consistently great vulnerability of the VIIIth cranial nerve to injury when the VIIth cranial nerve is being manipulated.

We also consider monitoring a priority for surgeons less experienced in performing MVD operations, since monitoring serves to warn the surgeon that a technical modification may be needed.

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