WHY MONITOR THE EEG?

Until recently, EEG monitoring outside of the diagnostic laboratory has met with limited use despite its clear role in such areas as surgery, anesthesia, trauma, and intensive care. There are primarily three reasons why this is so (Levy, 1980): 1) the size and complexity of conventional EEG equipment, 2) the need for a trained technician to run the machine, and 3) the difficulty in interpreting the raw EEG.

Advances in electronic technology, however, have shown promise in reducing the magnitude of some of these difficulties and there has been a resurgence of interest in EEG monitoring in other clinical areas, particularly when the EEG is preprocessed by one of several automated techniques.

Monitoring the EEG during surgery has been found useful for the following reasons (Prior, 1979).

1. It provides continuous information about the onset, depth, and duration of anesthesia, allowing an optimal level to be obtained.

2. The effect of surgical stimuli that could lead to arousal may be observed.

3. The time of waking or recovery can be anticipated and therefore adjusted, if necessary.

4. Assessment of completeness of recovery can be aided by monitoring and this may be of value in outpatient or "day-case" surgery.

5. During special types of operations, for example during cardiopulmonary by-pass, carotid endarterectomy, or deliberate hypotension, EEG monitoring provides confirmation of satisfactory cerebral perfusion, indicates times of particular risk to the brain, and gives an early warning of the occurrence and degree of recovery from abnormal events.

6. EEG monitoring provides a permanent, objective, record both of the adequacy of the conduct of anesthesia and information about any complications. Documentation of these events is important for medico-legal reasons.

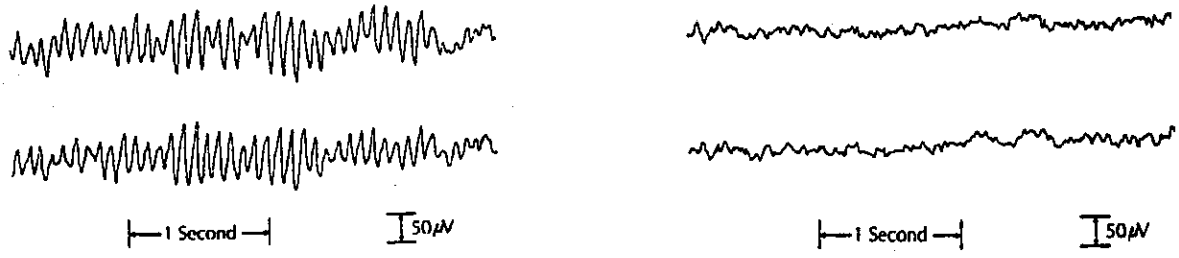
7. If any untoward incidents do occur, their effect on the brain can be immediately assessed, as can the efficiency of any corrective actions.

THE EEG

The electroencephalogram (EEG) is the minute, brain generated, voltage difference detected between two arbitrary sites on the scalp. The EEG varies both temporally and spatially, and with alterations in cerebral metabolism and function. Although the origin of the EEG is by no means clear, there is strong evidence that the signal measured on the scalp is a summation of the tens of billions of electrically active neurons comprising the brain. The present understanding of the underlying physiology and physics is that the cerebral cortex -- that convoluted, laminated sheet of cell bodies occupying about 600 cubic centimeters and containing about 100 billion neurons -- is the primary generator of the EEG (Cajal, 1909; Poggio, 1980; Kiloh, 1981).

The spontaneous EEG in normal, awake but relaxed adults often demonstrates a high degree of synchrony with the appearance of relatively large smooth waves over large areas of the scalp. This synchrony apparently represents a high degree of coordination in electrical activity among relatively idle cortical neurons or their subcortical afferents. The effect is particularly notable over the occipital, or visual cortex. When the eyes are closed there appears a large, almost sinusoidal oscillation at about 10 Hz in the EEG -- the alpha rhythm (Figure 1).

During periods of mentation or conscious effort, the EEG rapidly desynchronizes and is replaced by low amplitude, high frequency jumbles known as the beta rhythm (Figure 1). Low frequency patterns such as the delta or theta rhythms are usually not seen in alert adults except in the presence of pathology.



The Alpha Rhythm

The Beta Rhythm

Figure 1

OVERVIEW OF THE NEUROTRAC

The Neurotrac is an spectral analyzer specifically designed for monitoring applications. It was designed to be small and easy to use yet powerful and flexible in its EEG processing and display capabilities.

* 2 channel operation
* built-in amplifiers
* self-checking circuitry
* automatic electrode impedance checking
* automatic gain setting
* automatic detection of many common artifacts
* "hold screen" mode for stopping the screen update
* time, date, and patient ID on data displays
* event marker that can be displayed on CRT and printout
* optional external inputs for processing EEG from tape or other EEG machine output
* optional external digital output (to another computer) and analog output (to tape deck, video monitors, etc.)

The Neurotrac offers a variety of displays allowing the user full flexibility in choice of data presentation. The display options include:

* Raw EEG Waveform
* Compressed Spectral Array (CSA)
* Spectral Histogram
* Power Bands

All of the Spectral based displays (CSA, Spectrum, and Power Bands) features a variable update time allowing a range of display times: fast update times for critical surgical applications and slower update times for long term ICU monitoring. The spectral edge frequency can also be displayed to allow trending and observations of spectral changes.

The Neurotrac's optional printer provides a permanent record of the cerebral electrical activity throughout the monitoring session. The printer features:

* autoprint mode for continuous CSA printout
* patient ID number, date, and time recorded on printouts
* printout of the Neurotrac's configuration documenting all parameters under which the data was recorded and analyzed

NEUROLOGICAL APPROACH TO EEG INTERPRETATION

Patterns in the EEG are recognized by the human observer by analyzing the signal with respect to many different characteristics. Included in these characteristics are waveform morphology, frequency, amplitude, symmetry, reactivity, phase relation, repetition, distribution, and synchrony.

For the purposes of this discussion, only those characteristics most important to EEG Monitoring-- frequency, amplitude and symmetry -- will be defined below. This is a highly simplified discussion and, for more detail, the reader is referred to one of the textbooks listed in the bibliography.

Frequency

Frequency is a measure of the number of times a repetitive wave recurs in a one second interval. Most EEG activity occurs between 1 and 30 cycles per second (Hertz or Hz). The frequencies of EEG waves, traditionally, have been divided into four standard bands:

D	Delta	0 to 4 Hz
T	Theta	4 to 8 Hz
A	Alpha	8 to 13 Hz
B	Beta	13 to 30 Hz

It should be noted that the physiology generally associated with each band (beta = awake and alert, alpha = relaxed and eyes closed, delta and theta can indicate some pathology except in sleep) breaks down when under anesthesia as different agents can mimic any of these bands.

Amplitude

Amplitude refers to the height of the EEG wave as measured in microvolts (uV). EEG waves are loosely categorized as follows:

Low Amplitude	less than 20 uV
Medium Amplitude	20 to 50 uV
High Amplitude	greater than 50 uV

Symmetry

The EEG is generally symmetrical in the two hemispheres of the brain. Anesthetic agents usually affect the hemipsheres symmetrically also. Some surgical procedures, such as carotid clampings, can alter the normal EEG symmetry.

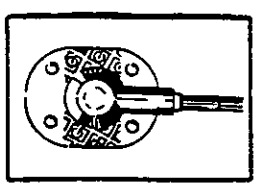
PRINCIPLES OF EEG RECORDING

An EEG measurement and recording system is analogous to that used for the ECG, varying principally in the EEG machine's additional flexibility. Since the character of the EEG is less well defined and more variable than the ECG, an EEG system must encompass a wider range of operation. The various components of an EEG system are outlined below.

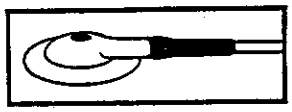
Electrodes

The purpose of electrodes is to provide an efficient interface between the scalp potentials and the EEG amplifiers. The key to electrode efficiency is to minimize the electrical resistance to current flow, including the frequency dependent resistance (impedance). Excessive electrode impedance leads to both decreased EEG signal applied to the amplifiers and frequency dependent distortion, and, in the case of asymmetric input impedance, an increase in the common mode (60 Hz power line hum) noise. Therefore a degree of care must be taken when applying the electrodes. The two major sources of excessive impedance are the stratum corneum (top layer) of the skin, and the skin-electrode interface. The impedance of the stratum corneum can easily be reduced by mildly abrading the skin with an abrasive gel (e.g. OMNI-PREP). The skin-to-electrode impedance can be reduced by using an electrode of appropriate design (particularly the type of metal used, and having sufficient surface area for contact) and using enough conductive paste. Nominal electrode impedance is below 5000 ohms (abreviated 5K ohms).

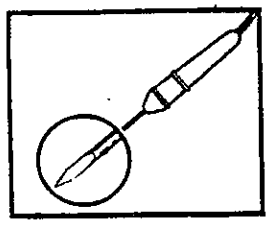
There are three types of electrodes in common use in the operating room and the intensive care: ECG pads, metal cups (silver-silver chloride, tin, or gold), and subdermal needles (figure 2).



ECG Pad



Metal Cup



Subdermal Needle

Figure 2. Electrodes used in EEG Recording

Electrode Placement

Standard neurological practice requires the precise placement of electrodes in relation to anatomic landmarks of the skull. A popular system for electrode placement is the International 10-20 system (Figure 3). Since the EEG is the potential difference observed between pairs of scalp electrodes, various electrodes must be paired for observation. The selection of pairs is referred to as the "derivation". Anatomical groupings of the derivations is called the "montage". A typical EEG montage compares anterior to posterior derivations between the right and left hemispheres.

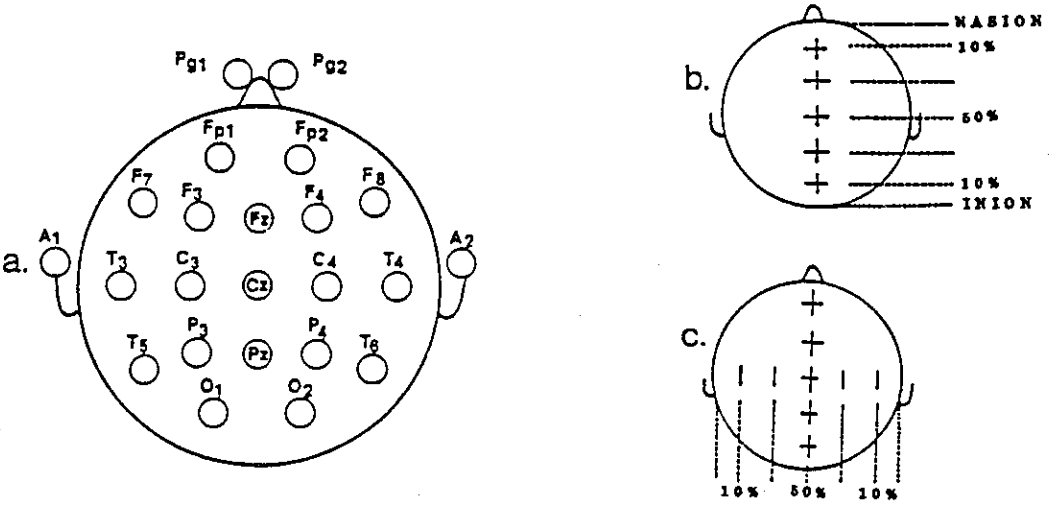


Figure 3. The 10-20 System of Electrode Placement (redrawn from Spehlmann)

Amplifiers

The EEG as detected on the scalp is of fairly low voltage and is almost overwhelmed by other physiological potentials (e.g. ECG or EMG) or extraneous interference or noise (e.g. 60 Hz). To minimize the effect of the extraneous potentials detected by the EEG electrodes a differential amplifier is used. A differential amplifier has two inputs which present the potentials at opposite polarities. This effectively cancels out any voltage signal common to both inputs (called common mode rejection), and amplifies only the voltage difference between the inputs.

The potential range of the EEG is quite variable -- it may be as small as 2 microvolts or as large as 1000 microvolts. To accomodate this variability the amplifiers need to have a gain (or sensitivity) which is adjustable to provide a display large enough to see the waveforms in detail without exceeding the limits of the electronics or of the display.

Filters

Another technique for reducing the noise content of the EEG takes advantage of the fact that the frequency content of the noise is generally different from that of the EEG. Judicious use of frequency dependent filtering must balance reduction of noise against decreased fidelity of the "true" waveforms of interest. For standard clinical EEG, filters are usually set to attenuate signals slower than 0.3 Hz and faster than 70.0 Hz. In the operating room with a concommitant increase in noise, a better compromise is a frequency band pass of 0.5 Hz to 30.0 Hz.

Display Techniques

The predominant EEG display technique in clinical neurology is a paper strip chart written on by ink pens and moved by a motorized paper drive. The most commonly used paper speeds are 15, 30, and 60 mm/sec. At a paper speed of 30 mm/sec the machine produces 108 meters/hour (over 325 feet/hour) of recordings. For this reason, computerized techniques are increasingly being used to process and compress EEG data in monitoring applications.

COMPUTERIZED ANALYSIS

There are many methods of processing the EEG, but the most widely used method in intraoperative monitoring is frequency analysis. An EEG may be thought of as the summation of simple sine waves of different frequencies to produce a single complex waveform. Frequency analysis mathematically decomposes the complex EEG back into these simple frequency components. This technique appears to be the most attractive at present (Saunders, 1981; Levy, 1980). It has been shown to be a reliable indicator of cerebral ischemia (Chiappa, 1979; Rampil, 1983) and may, in many cases, be used to assess changes in the depth of anesthesia (Hudson et al, 1983).

A crude, but inexpensive, technique for performing frequency analysis is to measure the time period between two adjacent points where the signal crosses the zero voltage baseline. The reciprocal of this period measurement is called the instantanteous frequency. This type of analysis can be performed without a computer using a few circuit components, but is generally insensitive and inaccurate.

A more accurate method is Fourier analysis, which uses a computer to mathematically decompose the EEG waveform into all its frequency components. The most popular algorithm for doing this computation is the Fast Fourier Transform or FFT. For the FFT computation, the computer samples the continuous EEG voltage signal periodically and converts that sample to a digital number of finite precision. Then, using a fixed number of samples the distribution of energy (or power) at each interval of frequency is computed. This results in a frequency spectrum for each sample (Figure 4).

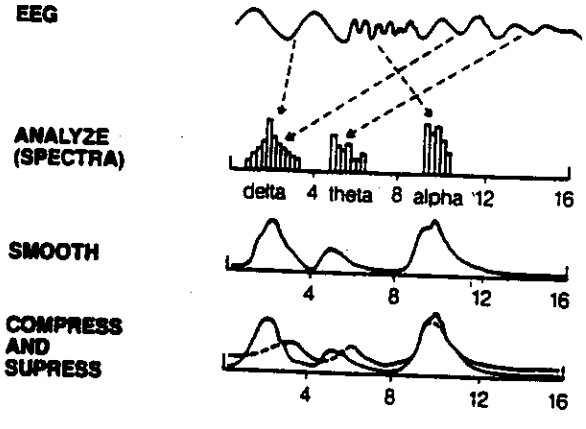


Figure 4. Calculation of the Frequency Spectrum (from Bickford, 1972)

The Compressed Spectral Array

Once the EEG signal has been processed by the FFT, there are a number of methods to display the frequency spectrum. The Compressed Spectral Array, CSA, format (Bickford, 1972), combines a high data compression factor and compact trending with the inherent formation of readily identifiable graphic patterns. The CSA provides a pseudo 3 dimensional display of frequency versus power distribution over time, where sequentially calculated spectra are drawn in a stack, one in front of the next. A hidden line suppression algorithm is usually implemented in the display to remove spectral information "behind" foreground features to add a three dimensional effect.

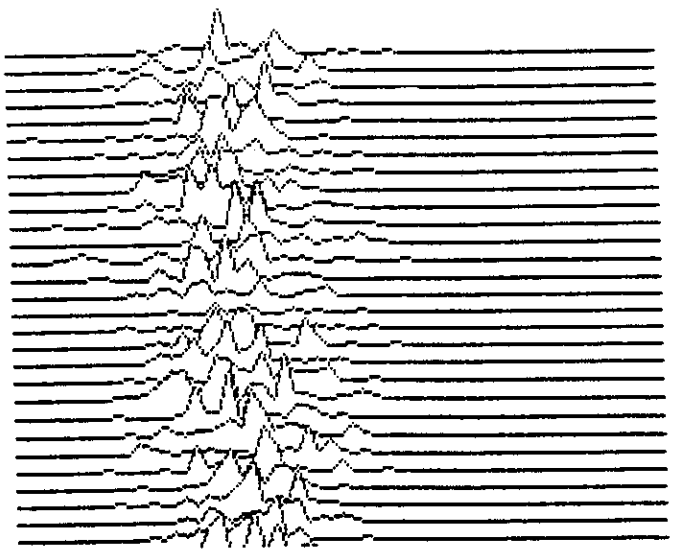


Figure 5. The Compressed Spectral Array

Other Methods of EEG Quantification

A further simplification of EEG interpretation may be made by having the computer quantify certain aspects of the computed spectra. Examples of these spectral parameters which have been investigated in the past include:

Peak Power Frequency (PPF)	The frequency at which the highest power is found to occur in the current spectrum.
Median Power Frequency (MPF)	The frequency in the current spectrum at which half the power is above, and half is below.
Spectral Edge Frequency (SEF)	The highest significant frequency present in the current EEG spectrum (usually at the frequency which represents 97% of the power).

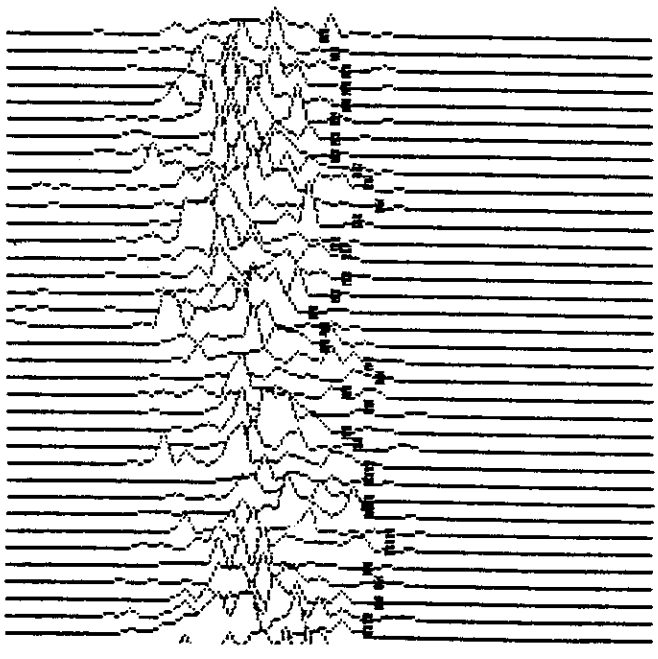


Figure 6. CSA with Spectral Edge Marked

EEG and Anesthetic Agents

The mechanism of action of anesthetic drugs is not well understood. Nonetheless, useful correlations have been noted between anesthetic dose, physiologic effect, and cerebral function as meaured by the EEG. Certainly, different classes of anesthetic agents have different mechanisms of action as well as possible common pathways.

Anesthetic effects form a continuum ranging from cortical excitation and hyperirritability through depression to isoelectricity. With the notable exceptions of the Ketamine class and Enflurane, most anesthetics cause a high frequency (beta) desynchronization (relatively low amplitude) of the EEG at subanesthetic doses, progressing to synchronization of lower frequencies (alpha) with higher amplitudes and then through depression with first lower frequencies (theta and delta) and then lower amplitudes to electrical silence (isoelectricity). Use of these patterns must be tempered by the ongoing clinical situation and the other factors which alter cerebral metabolism and hence EEG.

There is naturally a degree of inter-patient variability in EEG response to anesthetic agents, however, as pointed out by Faulconer and Bickford (1960), the large differences which exist between awake, alert people tend to disappear under the influence of anesthetics. Table 1 may be useful in the interpretation of the effects of these drugs. The table is limited to those EEG effects apparent in spectral analysis. Table 2 lists several common physiological parameters which may alter the EEG. These effects may be further modified by existing pathophysiology such as space occupying lesions.

TABLE 1

Effects of Anesthetic Drugs on the EEG

Drug	Dose	Freq(Hz)	Ampl.	Comments
Premedicants				
Diazepam	10 mg	13-25	low	In low doses, diazepam activates (desynchronizes) the EEG, in high doses it is a depressant.
Atropine	0.5	-	-	In clinical doses atropine is without EEG effect.
Droperidol	2.0	-	-	
Inhalationals				
Halothane	.75% .90% 1.3% 1.6%	10-20 10-15 5-10 1-5	low med high high	The frequency of anesthetic induced fast activity (as measured by the SEF) is inversely proportional to the end tidal concentration. The addition of N20 may further reduce the frequency.
Isoflurane	.75% .90% 1.3% 1.6%	15-20 5-10 0-4 0	low high low low	Isoflurane induces isoelectricity at a smaller dose than that causing cardiovascular collapse. Isoflurane otherwise resembles Halothane.
Enflurane	1.3% 1.8% 2.5% 3.6%	20-30 7-12 spikes spikes	low high	Enflurane seizure activity is accentuated by hypocapnia forming "spike and wave" with burst suppression.
Nitrous Oxide	70%	-	-	N20 alone seldom affects the EEG although it may cause slowing in combination with other drugs.
Parenterals				
Barbiturates				
Pentothal	50mg	25-35	low	In low doses barbiturates activate the EEG.
	250mg	1-4	high	High doses are potent EEG depressors
Narcotics				
Fentanyl	premed	-	-	Low dose narcotics do not affect the EEG.
	60ug/kg	1-3	high	High doses are potent EEG depressors
Ketamine		15-30	low	Overdose leads to seizures.

Table 2

Physiologic Conditions Which May Alter the EEG During Anesthesia

Condition	EEG Amplitude	EEG Frequency
Sensory Stimulation (1)	-	+
Hypoxia/Hypotension		
Very Early (2)	-	+
Persistant	+	-
Late	--	--
Hypocapnea	+	-
Hypercapnea (narcosis)	-	-
Hypothermia	-	-
Hypoglycemia		-
Hyperglycemia (3)		-
Hyponatremia (4)		-
Hypernatremia (5)		-
Epileptiform activity	++	++

Legend:

\+ increase
\- decrease

Notes:

(1) effect decreases with increasing depth of anesthesia
(2) stimulation of the Reticular Activating System by peripheral receptors
(3) hyperosmolar coma
(4) less than 120 mEq/L
(5) greater than 150 mEq/L

EEG AND ANESTHETIC DEPTH

The principal measure of the effect of anesthetics is seen on the electroencephalogram (Stockard & Bickford, 1975). Consistent changes can be seen in the EEG which correlate well with other assessments of anesthetic depth or with anesthetic dose (Egar, 1984). However, modern anesthetic poly-pharmaceutical techniques can make the interpretation of the EEG difficult when used to guage the depth of anesthesia. Computerized EEG, especially the compressed spectral array, helps to overcome this difficulty in interpretation (Stockard & Bickford, 1975). Although absolute levels of anesthetics are difficult to assess via the EEG when multiple agents are on board, shifts in the depth of anesthesia can almost always be seen.

The wide use of neuromuscular blocking agents has made much less useful the one universally accepted measure of aneshetic depth - movement in response to painful stimuli. The clinically desirable practice of dosing to effect is therefore difficult to obtain without new monitoring inputs such as the EEG.

This is an active field of research and with a trend toward the use of a single anesthetic agent such as fast acting narcotics (e.g. Fentanyl and Sufentanyl), EEG most certainly will play a major role in the assessment of depth of anesthesia.

THE EEG AND CEREBRAL ISCHEMIA

At the present time, perhaps the most significant contribution EEG monitoring may make to patient care is the early detection of cerebral dysfunction, especially that due to ischemia.

The functioning of the cerebral cortex is exquisitely sensitive to its environment. Insufficient cerebral blood flow or inadequate partial pressure of oxygen is reflected within seconds in the EEG (Sharbrough, 1973; Trojaborg, 1973; Rampil, 1983; Stockard, 1981; Sundt, 1981; Bauer, 1982; Niedermeyer, 1982). Although there may be a transient phase of excitation (following stimulation of peripheral chemoreceptors by hypoxia), the typical pattern (Figure 7) seen during cerebral ischemia is:

1. Reduction or loss of high frequency (beta and alpha) activity.

2. Appearance of large amplitude slow (mostly delta) waves.

3. Prolonged ischemia eventually results in a decreased amplitude and frequency leading to isoelectricity.

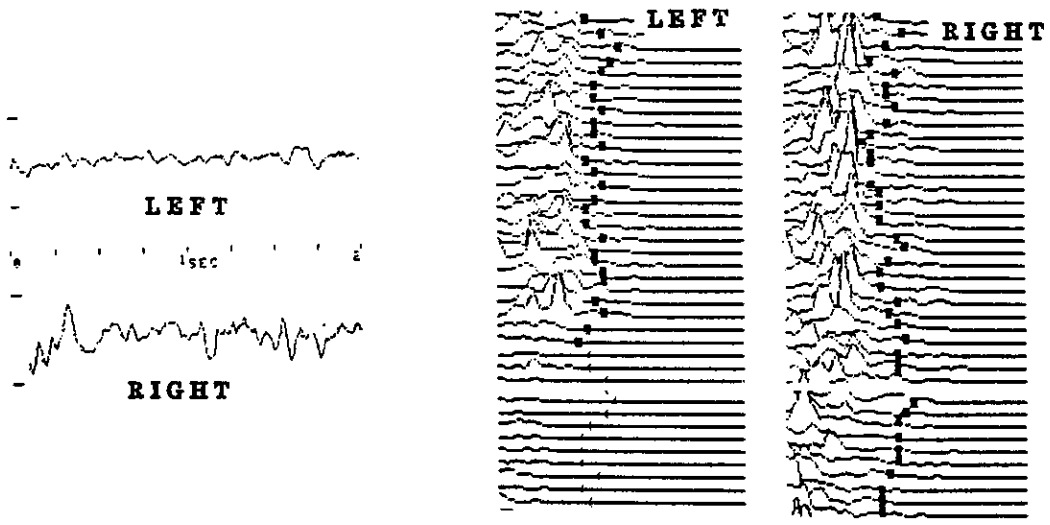


Figure 7. The EEG and CSA During Cerebral Ischemia

Interestingly, even a local decrease in cerebral blood flow, as occurs in an arterial embolism, results in an EEG change which is clearly visible in a spectral analysis of an EEG signal recorded some distance from the anatomic site of

ischemia (Rampil, 1983). This may be due to several factors including the very prominent "spatial smearing" caused by transmission of the EEG through the CSF, bone, and scalp. The computer analyzed spectra may even be more sensitive in detecting ischemia than manual interpretation because it is more sensitive to changes in high frequency activity.

There are, of course, several etiologies of cerebral ischemia which may occur in the operating room. These etiologies may be broadly categorized into global, hemispherical, or local phenomena:

1. Global

Hypotension	- volume depletion, pump failure or arrhythmias
Hypoxemia	- ventilator disconnect or malfunction, hypoxic mixture
Surgical Manipulation	- aortic clamping, and on occasion, carotid artery clamping

2) Hemispherical

Surgical Manipulation	- excess brain retraction, carotid artery clamping

3) Local

Embolic	- athermia, thrombi, air
Vasospasm	

The EEG appearance of ischemic episodes due to any of these factors appears to be similar in terms of the rapidity of onset, the change in frequency content, etc. Regardless of how similar the EEG patterns might appear, therapeutic interventions are best directed at the underlying factors. A differentiation may be made by observation of the clinical situation (e.g. the blood pressure, ECG, surgical maneuvers). Other concurrent factors which alter the intraoperative EEG such as the changing depth of anesthesia, temperature changes, and changes in CO_2 content may be recognized by the relatively slow onset of related EEG changes (several minutes as opposed to the changes of ischemia which generally occur within seconds). One should always keep in mind that the EEG may appear acutely depressed or ischemic following the rapid injection or parenteral agents which rapidly cross the blood-brain barrier. Again, attention must always be paid to

the clinical context in which the EEG is observed. Another key point in intraoperative EEG analysis is the fact that ischemia at different anatomical sites may induce different EEG patterns. The pattern described above (decreased SEF, increased amplitude) pertains particularly to cortical ischemia, whereas capsular ischemia may result in unremarkable EEG changes. Ischemia of the thalamus may produce alteration most prominent in the alpha rhythm of awake patient, and brainstem ischemia may result in EEG's appearing normal to EEG's showing widespread slowing and decreased amplitude (Niedermeyer, 1982; Kiloh, 1981).

Although there are certain maneuvers which increase the risk of cerebral ischemia -- such as carotid clamping -- clinical experience has demonstrated that ischemic EEG changes may occur at any time.

HOW DOES ONE TELL THE DIFFERENCE BETWEEN ISCHEMIC AND ANESTHETIC INDUCED EEG CHANGES?

The differentiation of ischemic and anesthetic-induced EEG changes can be made on a number of clinical and practical grounds. The simplest of these occur when the EEG changes are focal in nature. Such changes are seldom anesthetic-induced, but rather relate to intracerebral or cerebrovascular pathology. The situation is a bit more complex when global hypoxia must be differentiated from deepening anesthesia or hypothermia, but both the clinical situation and the EEG pattern itself provide important information allowing the determination of the etiology of the EEG changes.

Severe hypoxia often produces an isoelectric (flat-line) EEG. This may also be caused by deep hypothermia, large doses of barbiturates (much greater than those used to induce anesthesia), or isoflurane. It is almost inconceivable that either hypothermia or barbiturate dosage of sufficient magnitude to produce these EEG effects could occur without the awareness of the anesthesiologist.

Considering hypoxia which occurs as EEG slowing (rather than an isoelectric EEG), the pattern of development of the EEG slowing is important. Deep anesthesia requires time to develop, thus the onset of the EEG slowing is gradual. The transition between adequate and inadequate cerebral oxygen supply is abrupt thus the EEG slowing with hypoxia is quite

EEG, HYPOTHERMIA, AND CARDIOPULMONARY BYPASS

Cardiopulmonary bypass involves great physiological trespass, and therein lies the greatest potential for ensuing CNS damage. The onset of CPB is associated with sudden hemodilution, a change from pulsatile to non-pulsatile flow, often a reduction in mean arterial pressure, exposure of blood to foreign materials, a change in blood concentration of anesthetic agents and, as planned, a major reduction in body temperature (Silvay, 1983; Russell,1978). All of these have potentially important influences on the functional integrity of the CNS (Branthwaite, 1974), and indeed, investigators have found that the onset of CPB is associated with dramatic changes in the EEG (Kirtikon, 1977), although they may resolve spontaneously with time (Levy, 1984). The exact cause or causes of these changes have not been fully elucidated.

Hypothermia in itself has a dramatic effect on the EEG and even at stable concentrations of the anesthetic level, progressive hypothermia produces progressive slowing in the EEG frequencies with ultimate reduction in amplitude, intermittent suppression, and finally total suppression of EEG activity which occurs with rapidly developing hypothermia around 25 degrees centigrade (Sharbrough, 1984).

abrupt in onset. Hyperventilation can also produce EEG slowing; however this occurs in response to cerebral vasoconstriction (induced by hypocarbia) which in turn produces hypoxia (of a mild and inconsequential degree). These changes are also abrupt in onset, and the coexistance of hypocarbia as an etiology can be determined by blood gas measurements, end-tidal carbon dioxide determination or other types of analysis routinely available in the operating room.

Inhalational anesthesia of sufficient depth to produce an isoelectric EEG is likely to produce cardiovascular changes (usually hypotension) in a fashion which will alert the anesthetist to the presence of excessive anesthesia before, or together with, the EEG changes. This change in cardiovascular status provides further confirmation of the etiology of the EEG slowing. In addition, enflurane and isoflurane both produce burst-suppression in the EEG prior to, or together with, marked EEG slowing. Ischemia does not normally produce burst suppression. The presence of this pattern in the EEG preceeding or concurrent with slowing is another sign by which anesthetic depth may be differentiated from ischemia.

It should be noted that some intravenous anesthetics, particularly Fentanyl when used as a high-dose narcotic anesthetic and administered as a bolus, may produce the abrupt onset of EEG slowing. This would be likely to occur only at the start of a case; and again, under situations in which confusion as to the etiology of the EEG changes is unlikely. This anesthetic is somewhat unsuited for intraoperative EEG monitoring because of the EEG patterns produced by the anesthetic; however, severe hypoxia during a Fentanyl anesthetic still produces an isoelectric EEG, so that some degree of monitoring for hypoxia is possible. Furthermore, it is the responsibility of the anesthesiologist to select anesthetic techniques which allow him to use the monitoring available to its fullest potential; and if EEG monitoring is of paramount importance, then alternative techniques for the maintenance of anesthesia should be selected.

In summary, an EEG signal by itself does not allow the determination of the etiology of this signal any more than measuring a blood presure of 80/50 tells whether this was produced by bleeding or vasodilation. The clinical setting in which the EEG was recorded (like that of the blood pressure) allows for determination of the etiology, and thus permits differentiation of deep anesthesia from hypoxia in EEG monitoring.

APPLICATIONS IN INTENSIVE CARE AND TRAUMA

The Neurotrac may be used in the Intensive Care Unit or the Recovery Room setting for the same reasons outlined previously. In addition, EEG trend monitoring with the Neurotrac may be useful in the following situations:

1) As a noninvasive monitor in patients with head trauma, mass lesions, bleeds or other conditions in which the possibility exists that increased intracranial pressure may compromise cerebral circulation.

2) As a monitor of epileptic activity in unconcious patients, or those patients in status epilepticus managed with neuromuscular blockade and mechanical ventilation.

3) As a dosage guide in the management of induced barbiturate coma.

4) As an objective measure in the management of metabolic encephalopathies or drug overdoses.

5) As a screening monitor for situations in which "brain death" is suspected, prior to requesting a full 16 lead EEG examination.

Prior (1979) has also suggested that, in gravely ill patients, a continuous objective display of cerebral function provides an important degree of psychological reassurance to the ICU staff, the families and even to the patients themselves (when alert and interested).

CLINICAL EEG TRACES

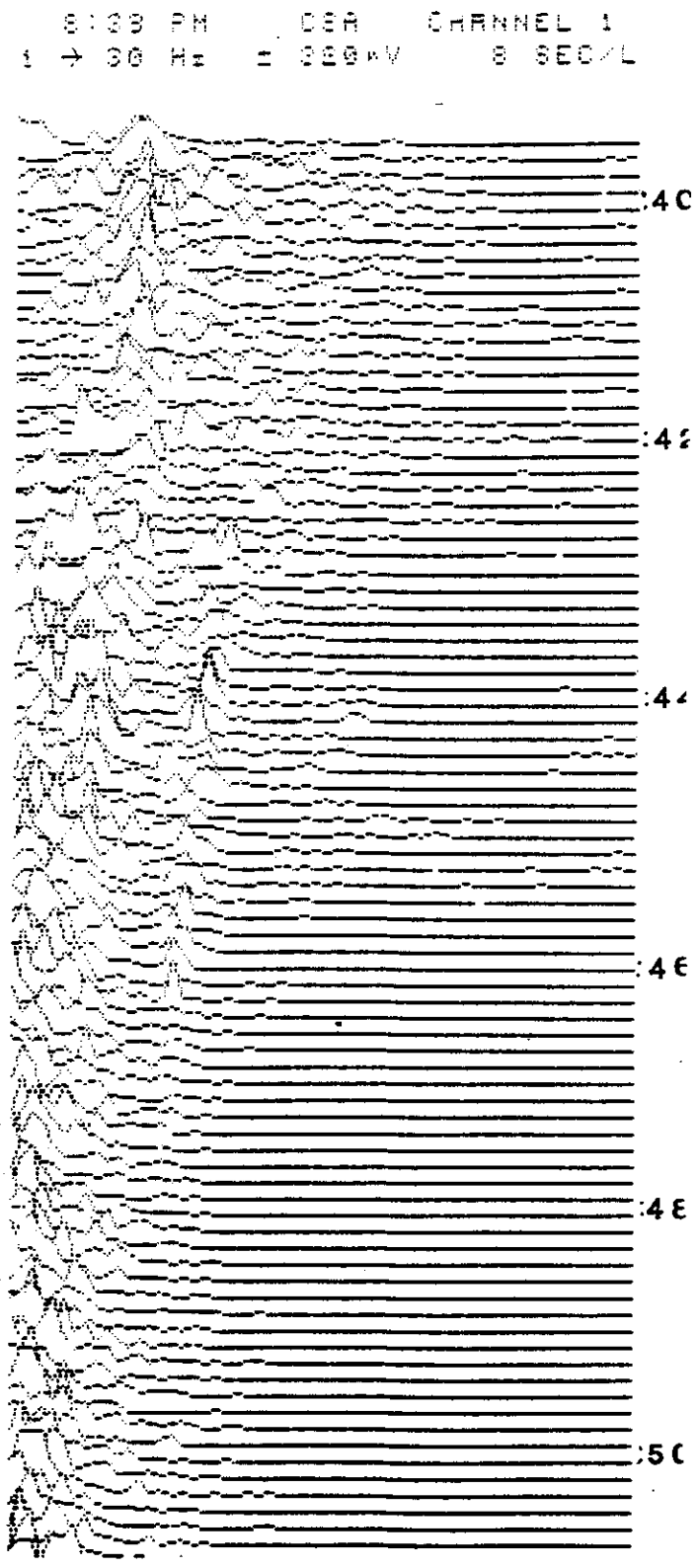
The following section presents a collection of EEG traces illustrating some of the applications of EEG monitoring intraoperatively and in the ICU. All cases were monitored using the Neurotrac EEG Monitoring System.

FENTANYL INDUCTION

Baseline alpha activity is prominent at time markers 40 to 42. A 250 ug/min infusion of Fentanyl was begun at 42, with the prompt development of EEG slow activity. As the infusion continues, a prominent peak occurs at about 12 Hz (marker 44). This activity slows slightly and then completely disappears after marker 46. Once established (42 to 44) the low frequency activity (0-3 Hz) continues at high amplitude, the characteristic pattern for high dose Fentanyl.

With the Neurotrac, inductions and recoveries may be monitored and documented.

Compliments of Dr. Warren Levy, University of Pennsylvania.



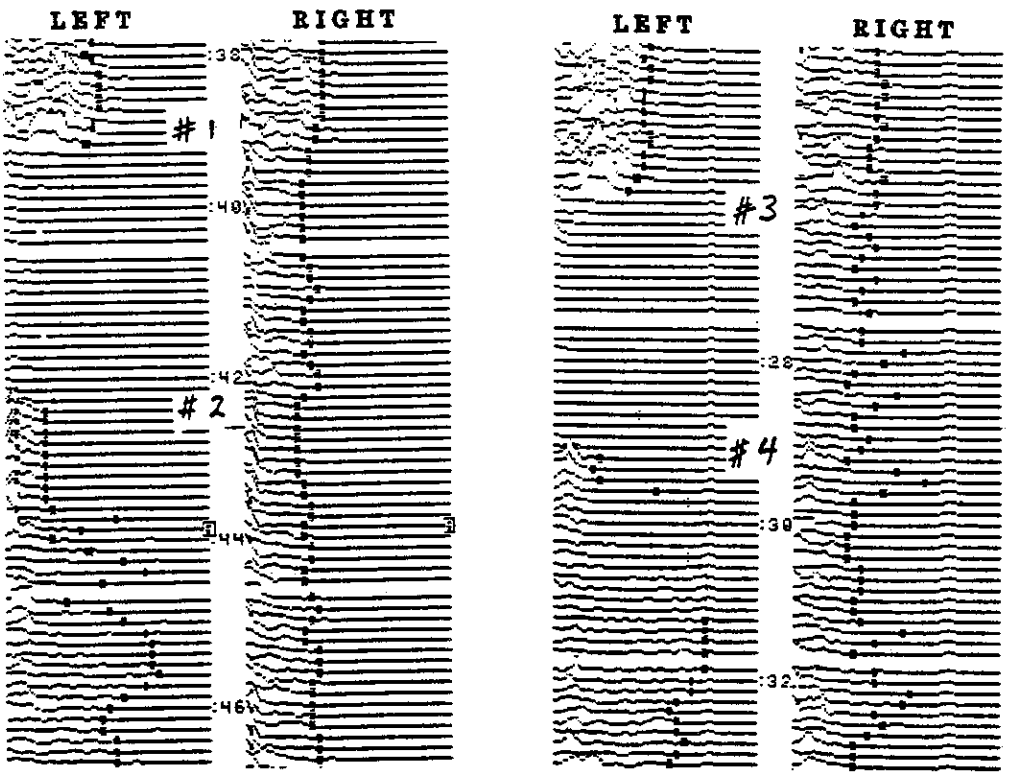
CAROTID ARTERY CLAMPING

The Compressed Spectral Array (CSA) recordings made during a carotid endarterectomy show two periods of marked ipsilateral cerebral ischemia during clamping of the artery for shunt insertions and removal. During these periods of ischemia the EEG frequency and amplitude dropped considerably. The severe amplitude drop is picked up by the Neurotrac and is indicated by the spectral edge "box" changing shape. During the first clamping, characteristic high amplitude delta waves appear causing the specral edge box to reappear. The "dynamic edge marker" aids in the identification of ischemic episodes.

The marker is also used to alert the user when the gain of the amplifiers is too low. These instances can occur during changes in anesthetic depth or cooling. These are differentiated from ischemic episodes by their gradual appearance and their correlation to changing temperature and anesthetic doses.

Legend:

#1 clamp on
#2 clamp off, shunt in
#3 clamp on
#4 clamp off, shunt removed



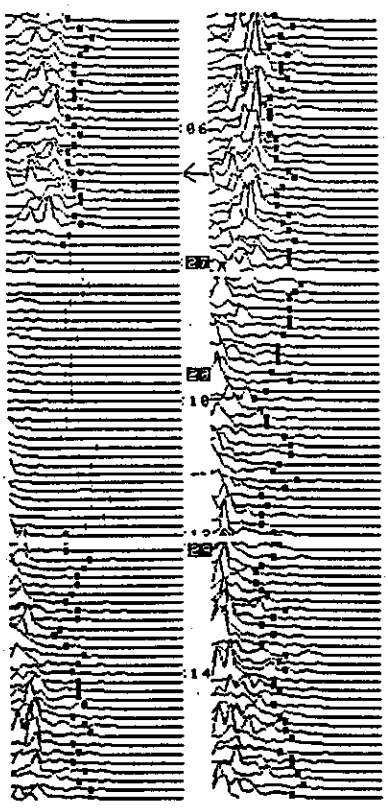
Compliments of Dr. Mark Zornow, Stanford University

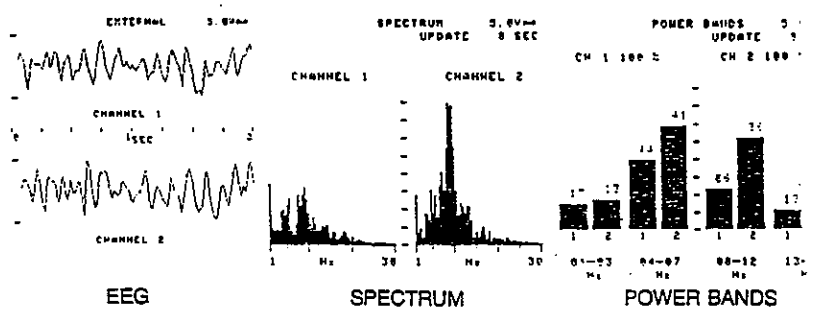
QUANTIFICATION OF AN ISCHEMIC EVENT

During this left carotid endarterectomy, an ischemic episode was seen following the cross clamping (at arrow). An almost total drop in power is seen in the left channel with a partial decrease in power and altered frequency distribution in the right channel. This is typical of a rather severe ischemic episode as presented in the EEG.

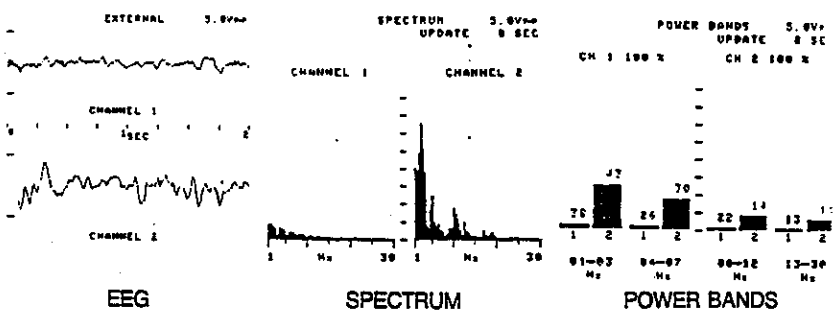
The Neurotrac can instantly quantify this frequency and amplitude difference between channels by switching to an alternate display mode. This spectral histogram shows the frequency distribution in half-hertz frequency units and the power band bar graphs quantify power in four user-selectable frequency bands.

Here we show these two displays both during baseline EEG immediately prior to the clamping and during the ischemic event. Note the disparity between channels and the overall reduction in amplitude during the clamping. The raw EEG can also be printed out to document changes or look for artifact.





BEFORE CLAMPING

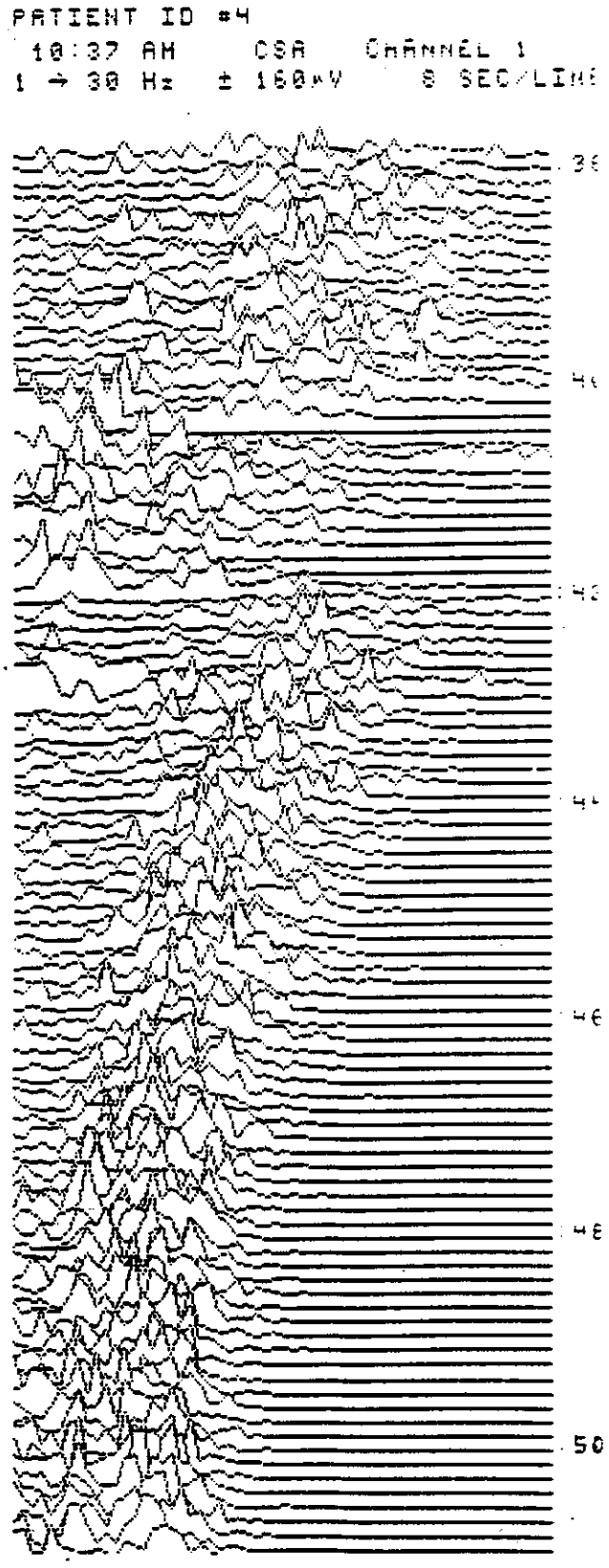


AFTER CLAMPING

Compliments of Dr. William Young, Columbia Presbyterian Hospital

COOLING OF CARDIOPULMONARY BYPASS

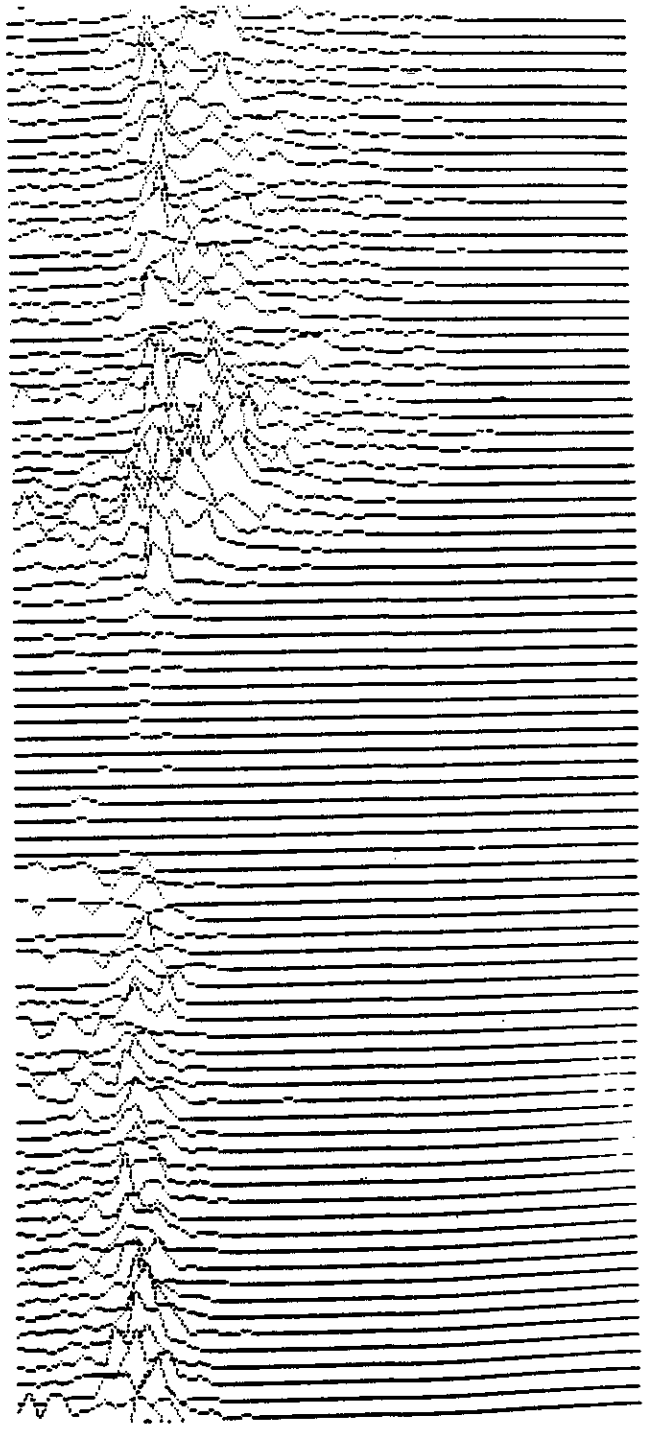
The baseline (time markers 38 to 40) shows high frequency activity (16 to 30 Hz) characteristic of light isoflurane anesthesia. At 40, cardiopulmonary bypass was begun, and EEG slowing is noted. This may reflect acute hemodilution, hypothermia or decreased arterial perfusion pressure resulting from the initiation of bypass. However, within two minutes (42) the EEG has been restabilized in a pattern almost identical to the baseline. Subsequent cooling from 36 to 26 degrees (marker 43 to 50) resulted in the gradual slowing of this band of activity as shown.



PUMP MALFUNCTION DURING CARDIOPULMONDARY BYPASS

This patient was being prepared for cardiopulmonary bypass. Upon start of bypass, a pump malfuction went unnoticed by the perfusionist, causing inadequate perfusion of the brain. The resulting ischemia was picked up on the EEG monitor as seen at the first arrow. Without adequate perfusion there is an abrupt loss of activity at all frequencies.

The establishment of cardiopulmonary bypass (second arrow) resulted in the prompt restoration of the EEG activity. A prolonged cerebral ischemic episode of this magnitude would surely have produced severe post-op neurological deficits.



EEG CHANGES DUE TO UNSUSPECTED AORTIC DISSECTION DURING CARDIOPULMONARY BYPASS

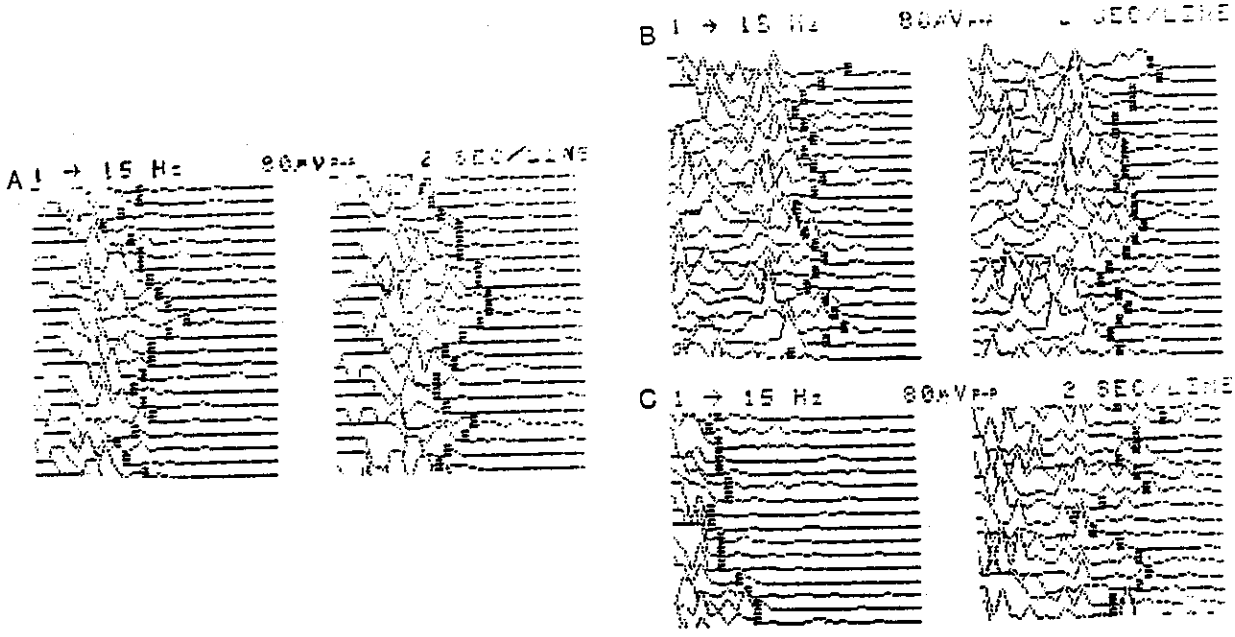
This patient was undergoing surgery for mitral valve replacement. The patient was being maintained on 50% N2O in O2, 20 ug/kg fentanyl and 0-0.5% enflurane. The EEG post-induction (A) showed symmetrical activity bilaterally. An arterial cannula was placed in the right iliac artery and a catheter in the right iliac vein. A second cannula was placed in the superior vena cava.

As bypass was begun, the EEG activity continued to be symmetrical bilaterally (B). Perfusion pressures in the thoracic aorta and the arterial line were normal.

30 minutes after institution of cardiopulmonary bypass, the urinary output was 30 ml. The EEG activity showed decreased activity in the left hemisphere (C). However, the perfusion pressures in both the arterial line and arterial cannula remained normal.

The patient could not be weaned from bypass. When an attempt was made to insert an Intra-Aortic Balloon Pump it was found that the aorta had dissected and that the cardioplegia solution had perfused into a false lumen. The patient was pronounced dead after 3 hours on bypass.

The EEG monitor was the first parameter to show that perfusion to the brain was not adequate.



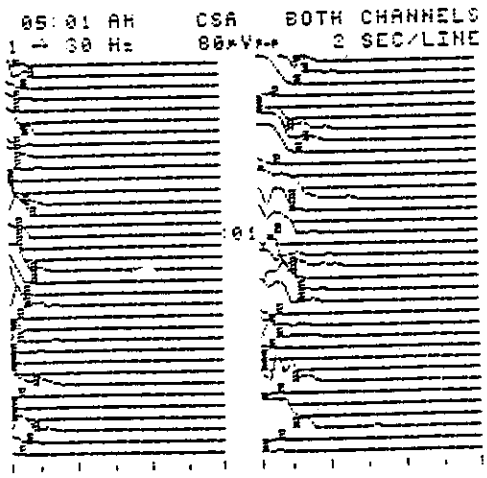
From the Clinical Report by Dr. Ira Michaels published in Anesthesia and Analgesia, September 1984.

USING THE CSA TO MONITOR BARBITURATE COMA

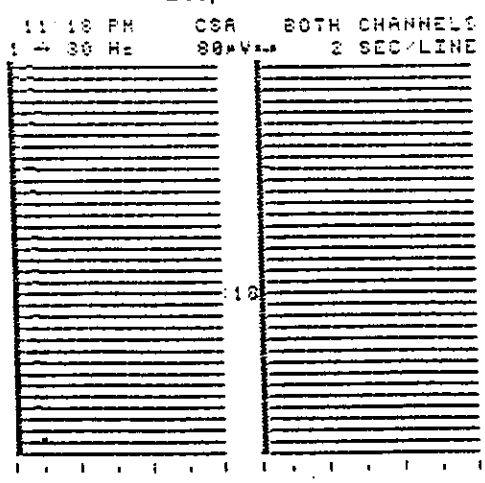
This patient was placed in a barbiturate coma following an attempted clipping of a large basilar artery aneurysm. Burst suppression ("bursts" of EEG activity followed by periods of no activity) seen in the compressed spectral array was used to monitor the depth of coma and the level of barbiturate necessary to keep this depth. The deeper the coma, the more widely spaced are the bursts of EEG activity. Burst suppression shows up clearly in the compressed spectral array format as isolated "mountains" of activity.

EEG monitoring serves as physiologic feedback that the dose of barbiturates (as measured by blood levels) is causing the desired results.

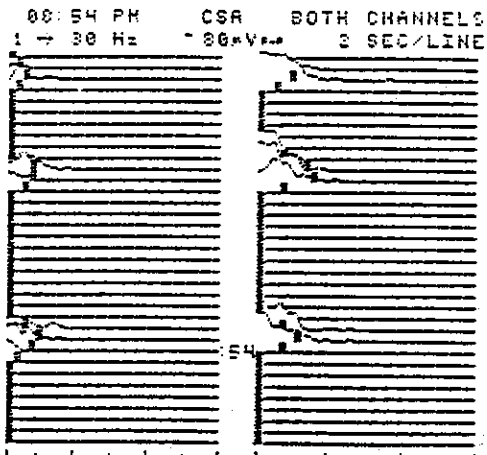
Light coma



Deep coma



Moderate coma



EVOKED POTENTIAL OVERVIEW

Evoked potentials are recordings of impulses (usually from the cortex) originating from some externally stimulated sensory nerve. They provide an objective physiological measure of the functional integrity of sensory nerve pathways and are used both as a clinical diagnostic procedure as well as for intraoperative monitoring.

An evoked potential differs from an EEG in these ways:

1. An EEG is the random, continuous signal which arises from the ongoing activity of the outer layers of the cortex. An evoked potential is the brain's response to a repetitive stimulus along a specific nerve pathway.

2. EEG signals range from 10 to 200 microvolts. Evoked potentials are smaller in amplitude (1 to 5 microvolts) requiring precise electrode positioning and special techniques (signal averaging) to extract the waveform from the EEG for analysis.

Classification

Evoked potentials (EP's) are broadly classified according to the sensory pathway which is stimulated. If the retina is stimulated (by a flash of light), it is called a visual evoked potential (VEP). If the auditory nerve is stimulated (by "clicks" from headphones on the patient), it is called an auditory evoked potential (AEP). The early part of the auditory AEP waveform (less than 10 msec) is called the brainstem auditory evoked potential (BAEP) since it reflects the passage of the impulse through that structure. If a nerve on the arm or leg is stimulated (by a small electric current applied to the overlying skin), it is called a somatosensory evoked potential (SEP).

Evoked potentials are used both as a diagnostic procedure and as a monitoring technique. As a diagnostic test, EPs are used in the diagnosis of such disorders as multiple sclerosis, acoustic nerve tumors, and optic neuritis. As a monitoring technique, EPs are used during surgical procedures which might compromise part of the brain or spinal cord.

PRINCIPLES OF SEP RECORDING

There are several differences between recording evoked potentials and recording the EEG for spectral analysis:

1. The electrodes must be placed in specific locations on the scalp necessitating the use of conventional EEG electrodes and not ECG electrodes.

2. The evoked potential waveform is produced by repeatedly averaging segments of EEG following a sensory stimulus. Thus a specific and repetitive stimulus must be provided and there will be a lag of up to a few minutes before the results of the test are known.

Electrode Types

In recording SEP's there are two sets of electrodes used. One set is for detecting and recording the impulse along the pathway. Since specific locations on the body and scalp are necessary, conventional EEG electrodes need to be used instead of ECG pad electrodes which may not stick well in areas which have hair. The other set is for providing the electrical stimulus to the nerve. The stimulus sites are over a peripheral nerve and the ECG pad electrodes work well.

Recording Electrodes

In recording SEP's each channel consists of a pair of electrodes. One of the electrodes is placed over the anatomical site which corresponds to the location along the sensory nerve pathway which is most likely to detect an impulse, and one is placed in a location not involved in the pathway response. This is called a referential montage.

When monitoring in surgical situations, it is important to monitor two anatomical sites along the nerve pathway. In one channel the active electrode site is over a peripheral nerve (over Erb's Point for median nerve stimulation, and over the Popliteal Fossa for posterior tibial nerve stimulation). In the other channel the active electrode site is over the sensory cortex site which corresponds to the nerve being stimulated. Generally, the peripheral nerve response serves as a control. For example, if only the cortical response were lost, it is likely due to the surgical procedure. If both responses were lost, it is most likely due to an improper stimulus.

The "inactive" electrode is placed on a point that is minimally involved in the sensory pathway. No electrode site on the body will ever be entirely inactive. Locations which may be used for the "inactive" electrode site include the forehead, mastoid, chin, shoulder, leg, etc. A larger plate electrode is used for the common or ground reference. This is usually placed around the limb being stimulated between the stimulation site and the first recording electrode. This will help decrease artifacts generated by the stimulus impulse.

Stimulating Electrodes

The most common nerves used for SEP stimulation are the median nerve (stimulated at the wrist) and the posterior tibial nerve (stimulated at the ankle). Since these are mixed nerves, both sensory and motor fibers are stimulated. The sensory response is what is recorded and used in the clinical evaluation of the patient's status. The motor response produces a twitch of the thumb (for median nerve stimulation) or toe (for posterior tibial nerve stimulation). This twitch is valuable for confirming proper stimulus location and for determining the stimulus intensity.

Averaging

The evoked potentials picked up on the scalp range from 1 to 6 microvolts compared to EEG potentials, whose amplitude ranges from 10 to 200 microvolts. For this reason, special techniques need to be employed to extract the evoked potential waveforms from the ongoing background EEG. This technique is called signal averaging. The EEG is a continuous signal, while the evoked potential signal is a specific, short duration response to a stimulus. The evoked potential is contained in the raw EEG signal.

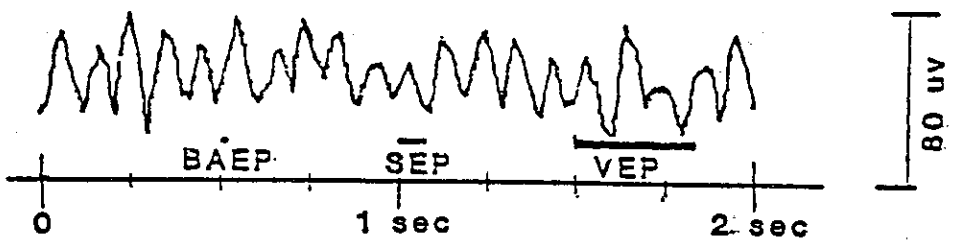


Figure 8. Relative Size and Duration of EP's compared to EEG

In averaging, the EEG activity during a specified time period immediately following the delivery of the stimulus is analyzed. For the analysis the continuous EEG signal is converted into a series of discrete numbers, each number proportional to the amplitude of the EEG at that point. The numbers are then stored in specific memory bins in the computer. This is called analog-to-digital conversion. With the delivery of the next stimulus the EEG signal during the specified time period is again digitized, and each point summed with the value previously stored in the corresponding bin, and then stored back in that bin.

The general principle of averaging is that if a sum is taken of values that are essentially random (in this case the EEG signal is considered to be random) the digitized values will be both positive and negative values, and their sum will approach zero. The nervous system, however, responds to a stimulus essentially the same each time, and the sum (average) of these digitized values in each bin will increase (either positively or negatively) to produce a consistent, recognizable waveform. The more samples used in obtaining the average, the greater the reduction of the background or random noise compared to the evoked response.

SEP WAVEFORMS

A characteristic waveform is generated as the impulse passes under each of the active electrodes placed along the nerve pathway. The resulting waveforms are named according to the polarity of the signal generated by the body (N for negative, and P for positive) and the approximate time (latency) in milliseconds it takes for that response to occur following the stimulus.

Normal Median Nerve SEP's

The diagram below shows traces obtained from stimulation of the Median Nerve at the wrist. The first electrode detecting the impulse is over the brachial plexus at Erb's Point, generating a nerve action potential (A), generally called N9. The impulse continues, entering the spinal cord and ascending in the dorsal columms to synapse in the brainstem nuclei. A second neuron originates in the brainstem and travels to the thalamus, where it synapses. A third neuron originates in the thalamus and projects to the sensory cortex resulting in the waveform (B) detected by the electrode placed over the sensory cortex. The thalamus/thalamocortical radiation response is initally negative, called N19, followed by a large physiologically positive potential, called P22.

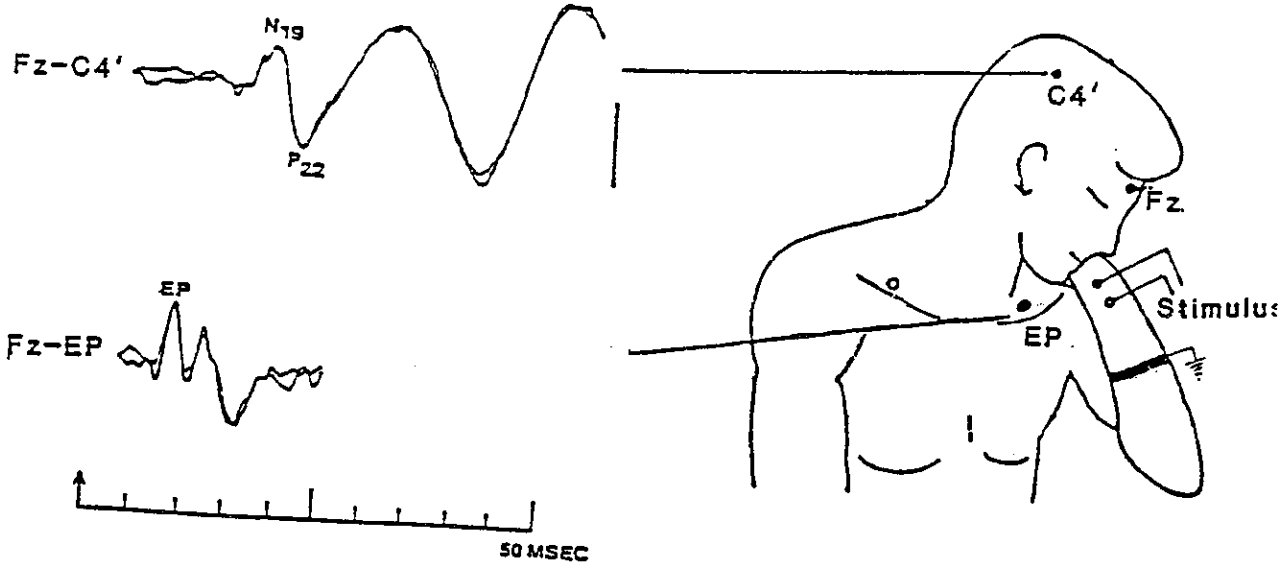


Figure 9. Normal Median Nerve SEP's

Normal Posterior Tibial Nerve SEP's

In recording the Posterior Tibial Nerve SEP's some technical problems arise if a forehead reference is used . The length of the sensory nerve pathway from the ankle to the brain introduces a lot of "artifact" from the electrical fields generated by other parts of the body making it more difficult to record the small potentials generated by the sensory nerve pathway. For this reason, to record the peripheral nerve action potential, the "active" electrode is placed over the Popliteal Fossa (PF) at the knee ipsilateral to stimulation and the "inactive" electrode is placed below and slightly to one side of the PF about 3 cm away.

The diagram below shows traces obtained from stimulation of the posterior tibial nerve at the ankle. As the impulse travels up the leg, the characteristic nerve action potential waveform (A) is generated as the impulse passes under the electrode at the Popliteal Fossa, usually designated N8. The impulse continues, entering the spinal cord ascending toward the brain in the dorsal columns. The sensory neuron synapses in the brainstem. A second neuron originates in the brainstem and ascends to the thalamus. From the thalamus a third neuron originates and is projected to the sensory cortex. The thalamus/thalamocortical radiation potential (B), is initally negative, called N33, followed by a large positive potential, called P37, with another large negative potential following, called N45.

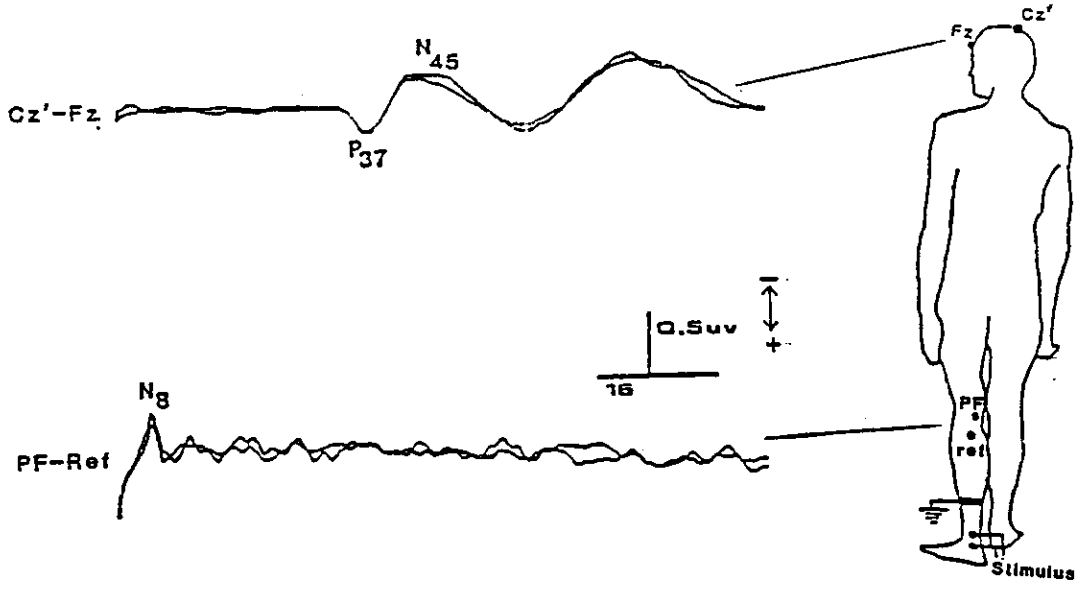


Figure 9. Normal Posterior Tibial SEP's

SURGICAL APPLICATIONS OF SEP MONITORING

In surgery, SEP monitoring finds use in many situations (Grundy, 1983) including:

1. Operative retraction and manipulation around the spinal cord and other CNS structures.
2. Orthopedic cases where the spinal cord may be damaged such as Harrington rod insertions and removals.
3. Manipulation of an unstable cervical spine.
4. Resection of peripheral nerve lesions.
5. Posterior fossa procedures.
6. Stereotactic thalamic procedures.
7. Identifaction of Rolandic fissure.
8. Epilepsy surgery.
9. Resection of parietal lesions.
10. As an indication of cerebral hypoxia.

WAVEFORM CHANGES SEEN IN SURGERY

There are several factors which will cause changes in the evoked responses which are unrelated to surgical manipulation. These include induction of anesthesia, increase of an anesthetic agent which has depressive effects on the cortex, cooling of the entire body, and/or cooling of the spinal cord at the surgical site.

With induction of anesthesia, an increase in latency of a few milliseconds is expected in the cortical response, but the peripheral response (ie. Erb's Point or Popliteal Fossa) should not change. The cortical response should stabalize once anesthesia is maintained.

Some of the anesthetic agents will have depressive effects on the cortex with increasing dosages. Most notable in their effects with increasing concentrations are the inhaled halogen agents (Forane, Isoflurane, Halothane, etc.), high doses of barbiturates (Pentothal), high doses of diazepam (Valium), and high doses of narcotics (Fentanyl). The peripheral evoked potentials will show no change, but the cortical potentials will show progressive increases in latency, broadening of the complex, and a decrease in amplitude related to the dose of the agent. These anesthetic effects reverse with decreases in the dosage of the agent.

Cooling of the entire body (cardiovascular procedures) will affect both the peripheral and cortical potentials -- showing a progressive increase in latency and a decrease in amplitude of all components. With severe hypothermia, total loss of all peripheral and cortical potentials can be expected. With warming, a progressive increase in amplitude and decrease of latency will occur.

Cooling of just the spinal cord at the surgical site will not affect the peripheral potential, but the cortical potential will show a progressive increase in latency and decrease in amplitude which reverses with warming.

Peripheral neuro-muscular blocking agents (Curare, Pavulon, etc.) will not affect the SEP responses in clinical dosages. However, spinal blocking agents (e.g. a saddle block) will prevent the impulses from traveling up the cord and prevent any cortical potentials.

Criteria for abnormalities for both median nerve and posterior tibial nerve evoked potentials include:

1. Shift to the right (increase) in latency of the cortical potential, related to the surgical procedure, which does not return to the surgical baseline when conditions are returned to pre-shift conditions.

2. Total loss of the cortical potential when related to surgical conditions.

SEP APPLICATIONS IN INTENSIVE CARE

In the Intensive Care Unit the task of neurologically evaulating central nervous system function in the severely head-injured patient is extremely difficult. Use of chemical paralysis or barbiturate coma in management of head injured patients has made the neurological examination unreliable. Even with the various physiological parameters measured at the bedside, there is no monitor which gives serial or continuous assessment of neuronal function. In the brain injured patient there is an acute phase during which the dynamic condition of the brain (increased ICP, ishcemia, cerebral blood flow, cerebral edema) influences survival of brain function. Modification of these critical conditions by medical or surgical management is greatly enchanced by serial monitoring of brain function using non-invasive procedures such as EEG and Evoked Potentials.

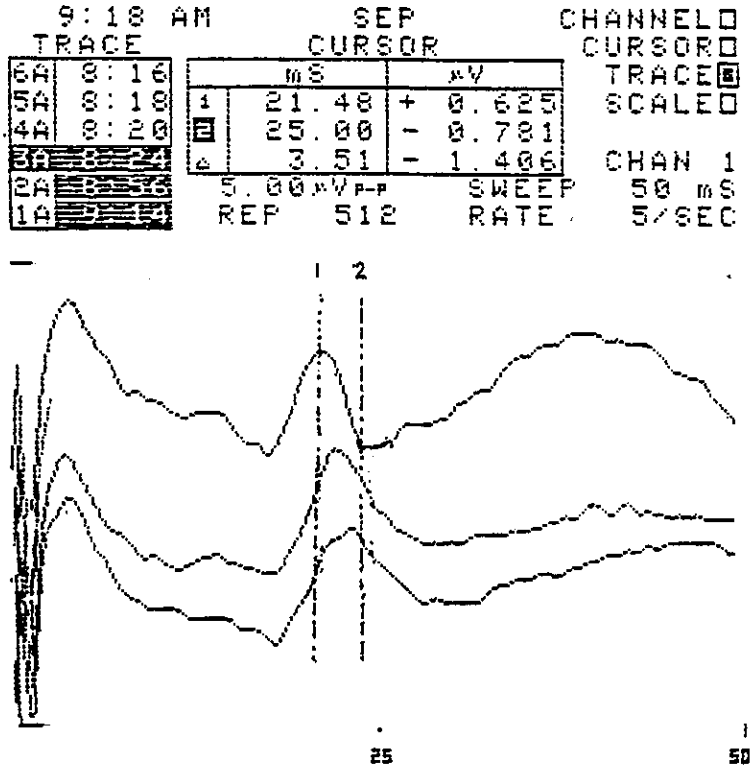
CLINICAL EP TRACES

The following section presents a collection of SEP traces illustrating some of the applications of SEP monitoring intraoperatively and in the ICU. All cases were monitored using the Neurotrac EEG Monitoring System.

EFFECTS-OF ETHRANE ON SOMATOSENSORY EVOKED POTENTIALS

The cortical response of the somatosensory evoked response is known to be altered by anesthetic agents. Most notable in their effect are the inhalational agents. In general, they cause an increase in latency or other waveform distortion. Nitrous oxide, however, has little or no effect on the waveforms. Short latency responses seem to be more immune to distortion by anesthetics than the longer latency components of a waveform.

The traces below show the shift in latency with increasing concentration of Ethrane. The response is from a median nerve stimulation. Cursors 1 and 2 are on the peak and trough of the trace taken at 0.5% Ethrane to give an idea of the amplitude of the response (1.406 uV). The total shift in latency is less than three milliseconds with a three-fold increase in anesthetic concentration.



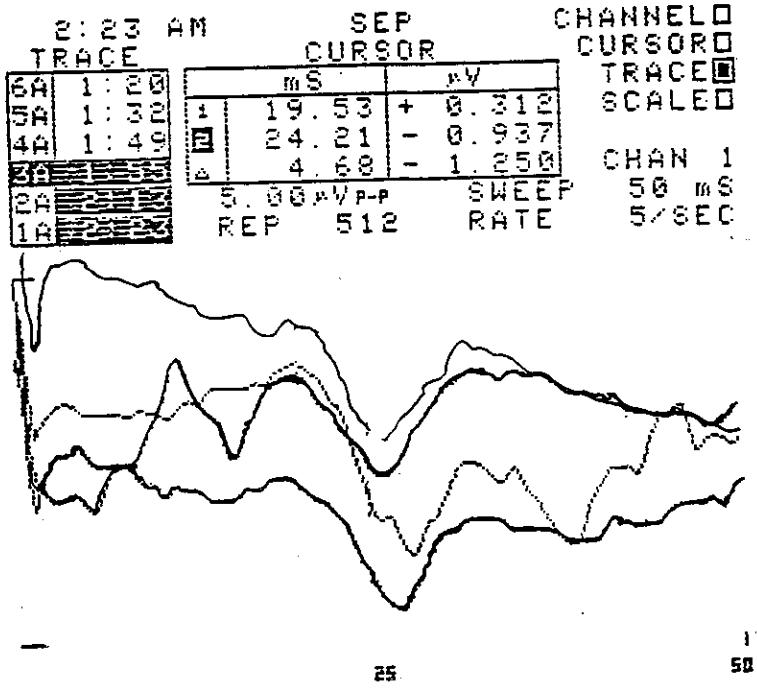
TRACE	ETHRANE
1	0.5%
2	1.25%
3	1.5%

Compliments of Dr. Michel Dubois, Georgetown University.

EFFECTS OF DELIBERATE HYPOTENSION ON SOMATOSENSORY EVOKED POTENTIALS

Many physiological factors affect the somatosensory evoked potentials including variations in body temperature, arterial blood pressure, intracranial pressure (ICP), tissue perfusion, arterial tensions of oxygen and carbon dioxide, and body chemistry. As with anesthetic agents, the longer latency components seem to be more sensitive to these changes.

The traces below show the changes in peak latency seen with changes in blood pressure. Ethrane and end tidal CO2 were held constant (1.25% and 30 respectively) while the mean arterial pressure (MAP) was varied. An increase in latency is seen as the MAP is decreased from 60 to 40 and a return to shorter latencies is seen as the MAP is brought back to 50.



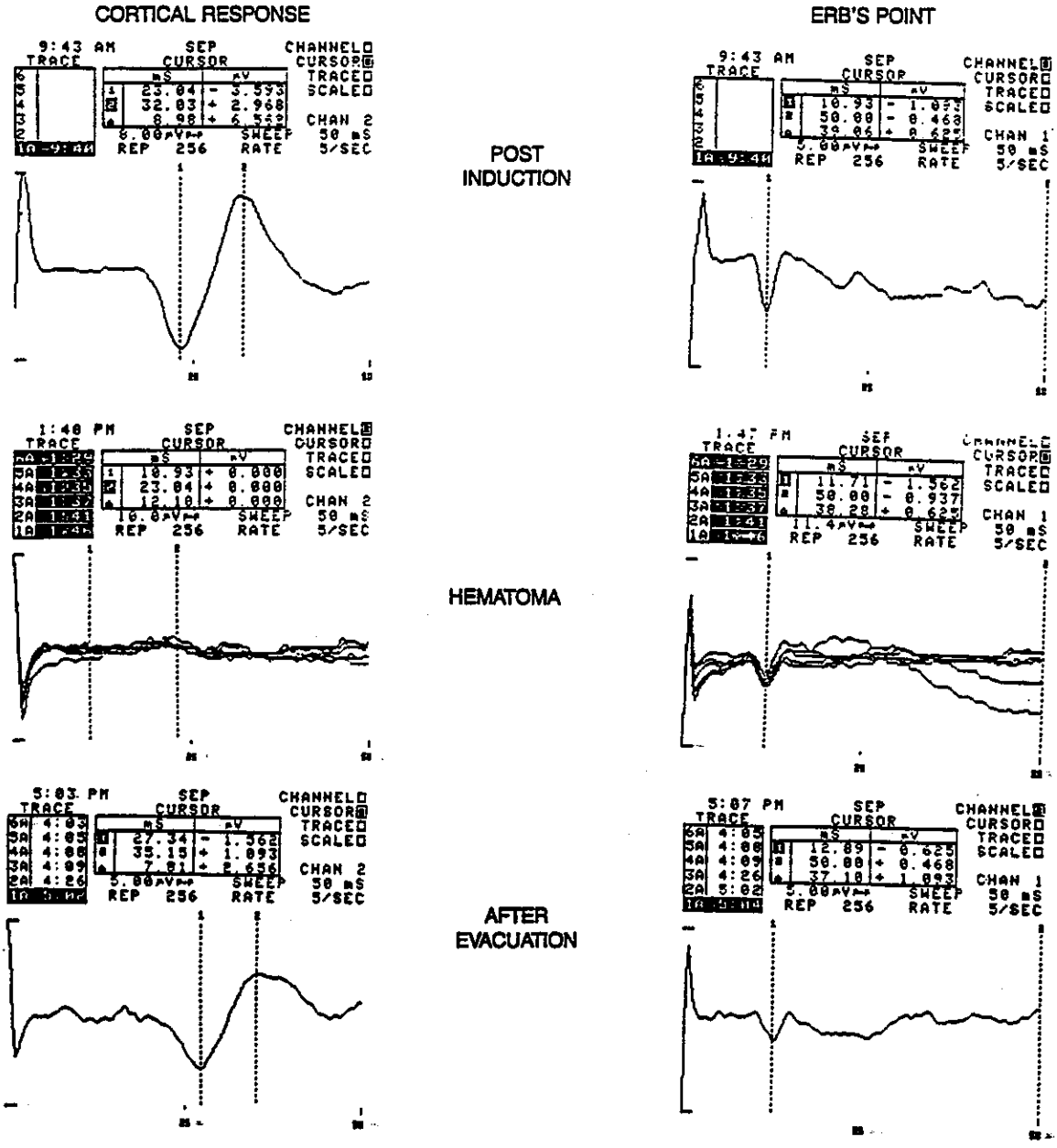
TRACE	ETHRANE	ETCO2	MAP
4	1.25%	30	60
3	1.25%	30	55
2	1.25%	30	40
1	1.25%	30	50

Note: Traces 3 & 1 bolded for identification purposes.

Compliments of Dr. Michel Dubois, Georgetown University

EFFECTS OF AN UNNOTICED INTRACRANIAL BLEED DURING THE CLIPPING ON AN ANEURYSM

During the clipping of a posterior communicating artery aneurysm an intracranial bleed went unnoticed producing a large left temporal epidural hematoma. The hematoma was seen in the somatosensory evoked potentials as an obliteration of the cortical response. After evacuation of the hematoma, the cortical response returned. The Erb's point response remained intact throughout the procedure.

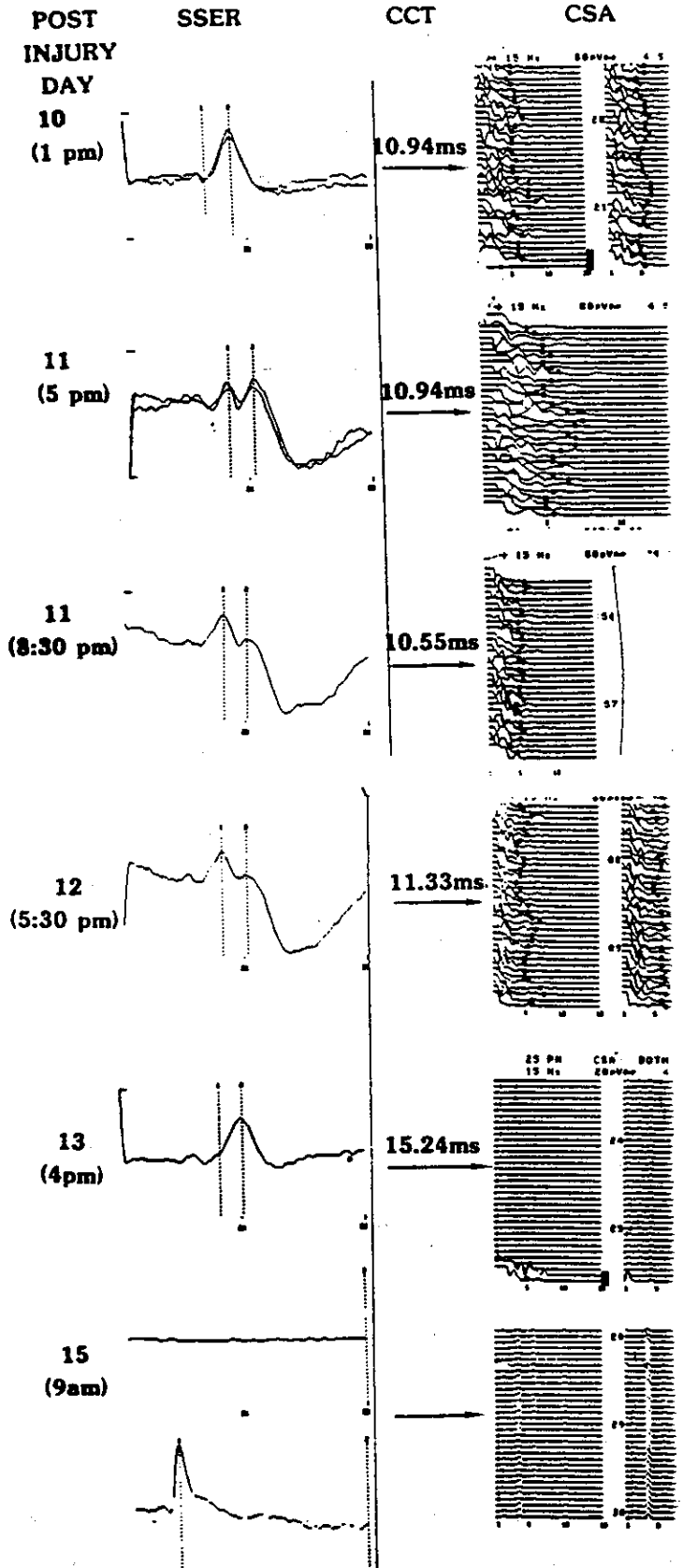


Compliments of Dr. Michel Dubois, Georgetown University

INTRACRANIAL PRESSURE MONITORING IN THE ICU

This 30 y.o. male was admitted to the ICU following a motor vehicle accident. An ICP monitor registared an opening pressure of 13 cm H2O. The patient was placed in a barbiturate coma. A series of SEP and CSA recordings were used to monitor the patient during barbiturate coma. 10 days post-injury a reproducible SEP cortical response was recorded with a mild latency delay. The next day, the ICP increased and the SEP cortical developed an abnormal morphology. The ICP was treated agressively over the next four hours. As the ICP was controlled the cerebral perfusion pressure (CPP) increased, and the cortical response morphology and latency began to return to normal. The patient continued to do poorly. Two days later the CPP was 8 mmHg, and no cortical activity was recorded with either the CSA or the SEP. The patient was pronounced dead.

In cases where the patient is chemically paralyzed and in a barbiturate coma, the classical neurological exam for brain death does not apply. Increased use of evoked responses to assess electrical function has been of particular value in these paitents.



Compliments of Dr. J. Hargadine, Univ. of Texas, Houston.

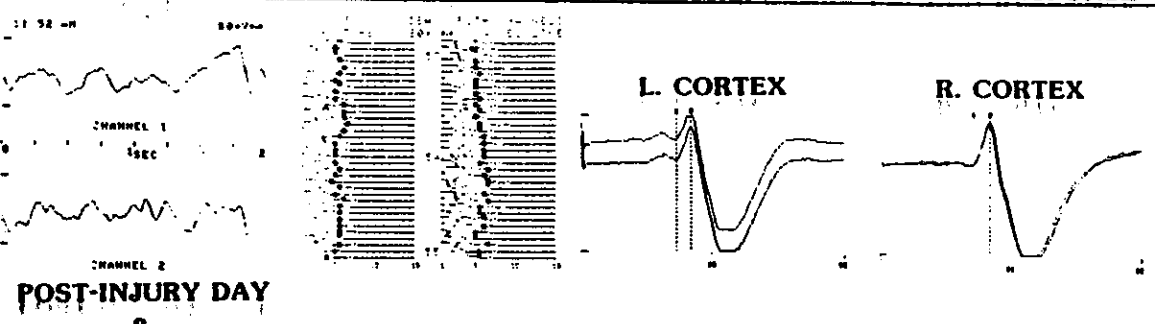
SEP AND CSA IN BARBITURATE COMA

This 28 y.o. male was involved in an auto-pedestrian accident. He was admitted to ICU from the operating room following evacuation of a subdural hematoma and placement of an ICP monitor. Intracranial pressure (ICP) at opening was 10 cm H20, and cerebral perfusion pressure (CPP) was 90 mm Hg). The patient was first monitored with CSA and SEP's eight days post-injury. The ICP was 31 cm H20 and the CPP was 89. The SEP's were normal bilaterally and the CSA demonstrated activity of 80uV in the 4 to 7 Hz range. The following day, CSA studies demonstrated activity of 80uV in the 8 to 12 Hz range. The SEP studies were unchanged. The patient was placed in a barbiturate coma later that day. With a pentobarbital level of 26ug/ml, reliable SEPs were recorded bilaterally. The CSA and power bands demonstrated the effects of pentobarbital on cortical activity. It was evident that an optimum barbiturate level had not been reached as electrical activity was still recorded in the 4 to 7 Hz range at low voltage (40uV).

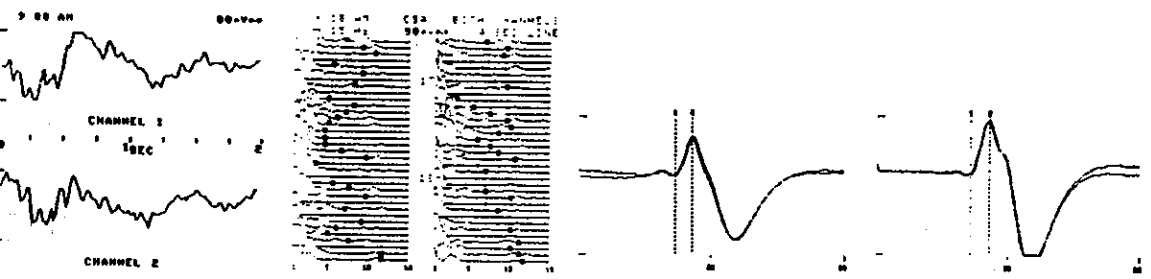
CSA recordings can be extremely useful in determining the level of pentobarbital necessary to suppress cortical activity, with the goal of preserving brain function during increased ICP. Since high barbiturate levels cause unwanted effects on blood pressure and CPP, titration of the barbital level against cortical activity is extremely important. Once an isoelectric EEG with burst suppression is obtained, maximum benefit of the pentobarbital therapy is obtained. When cortical activity is suppressed on the CSA, SEP's can provide a measure of cortical perfusion, because the SEP can be recorded in many cases when the EEG is isoelectric.

Compliments of Dr. J. Hargadine, Univ. of Texas, Houston

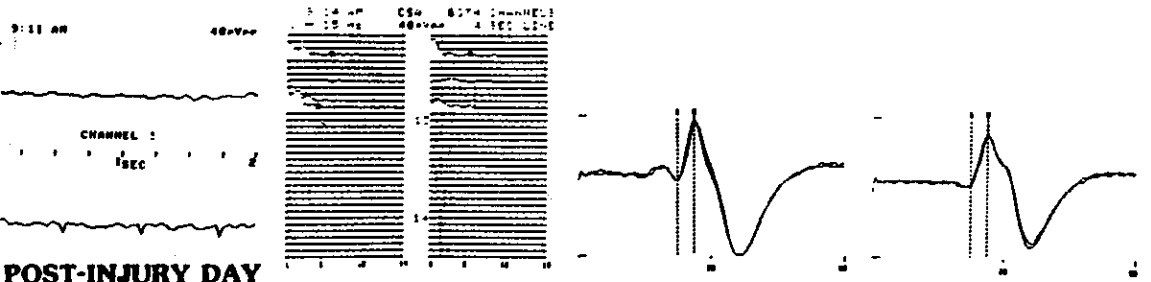
EEG CSA SOMATOSENSORY EVOKED RESPONSE



POST-INJURY DAY 8



POST-INJURY DAY 9



POST-INJURY DAY 10

Barb. level µg/ml	CPP mm Hg	CCT(L) msec.	CCT(R) msec.
0	89	5.86	5.47
26	79	5.86	5.86
29	64	5.86	5.86

OVERVIEW OF THE NEUROTRAC

The Neurotrac is an spectral analyzer specifically designed for monitoring applications. It was designed to be small and easy to use yet powerful and flexible in its EEG processing and display capabilities.

* 2 channel operation
* built-in amplifiers
* self-checking circuitry
* automatic electrode impedance checking
* automatic gain setting
* automatic detection of many common artifacts
* "hold screen" mode for stopping the screen update
* time, date, and patient ID on data displays
* event marker that can be displayed on CRT and printout
* optional external inputs for processing EEG from tape or other EEG machine output
* optional external digital output (to another computer) and analog output (to tape deck, video monitors, etc.)

The Neurotrac offers a variety of displays allowing the user full flexibility in choice of data presentation. The display options include:

* Raw EEG Waveform
* Compressed Spectral Array (CSA)
* Spectral Histogram
* Power Bands

All of the Spectral based displays (CSA, Spectrum, and Power Bands) features a variable update time allowing a range of display times: fast update times for critical surgical applications and slower update times for long term ICU monitoring. The spectral edge frequency can also be displayed to allow trending and observations of spectral changes.

The Neurotrac's optional printer provides a permanent record of the cerebral electrical activity throughout the monitoring session. The printer features:

* autoprint mode for continuous CSA printout
* patient ID number, date, and time recorded on printouts
* printout of the Neurotrac's configuration documenting all parameters under which the data was recorded and analyzed